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THE EFFECTS OF 10 WEEKS OF RESISTIVE TRAINING ON THE LIPOPROTEIN PROFILE OF MEN

by

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ABSTRACT

Amongst the many risk factors, a high level of serum cholesterol has been consistently associated with coronary heart disease. Although aerobic exercise has been shown to decrease the cholesterol level by increasing the high density lipoprotein (HDL) concentration, little is known with respect to the effect of anaerobic training on the lipoprotein profile. This study assessed the effect of 10 weeks of resistive training on the lipoprotein profile of sedentary men; 10 men served as experimental subjects and 4 men were control subjects. Body composition and muscular endurance were measured pre- and post-study. Blood samples were taken prior, at week 6 and at the completion of the study. The triglycerides, total cholesterol, LDL-C, VLDL-C, HDL-C, HDL2-C and HDL3-C concentrations were analyzed. The results indicate that body composition did not vary during the course of the study. The training increased significantly the muscular endurance in the experimental subjects. The total cholesterol, the triglyceride, LDL-C, VLDL-C, HDL-C and HDL2-C values did not vary during the course of the study in either group. HDL3-C level was significantly greater after the 6th week of the study for the combined mean of both groups. The HDL2-C/HDL-C ratio began to increase significantly in the experimental group prior to the 6th week of training. Since the control group had a significantly low post-study HDL2-C value, the HDL2-C/HDL-C ratio decreased significantly during the course of the study in their group. The greater HDL2-C/HDL-C ratio in the training group offers a favorable lipoprotein profile.

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I

INTRODUCTION

1.1 INTRODUCTION

In recent years, a number of studies have recognized the prophylactic effects of exercise in the prevention of coronary heart disease (CHD) (Paffenbarger and Hale, 1975; Paffenbarger et al., 1978) and its influence in modifying the coronary risk factors (Morris et al., 1980; Garcia-Palmieri et al., 1982). Amongst the risk factors, a high level of serum cholesterol (Castelli et al., 1977; Kannel et al., 1979) and particularly its low density lipoprotein fraction (LDL) (Mahley et al., 1980; Fainaru et al., 1982; Brown and Goldstein, 1984) have been associated with the development of atherosclerosis. As such, much research has been directed towards finding the possible effect of exercise on lipoproteins to reduce total serum cholesterol. Exercise has been shown to increase high density lipoprotein (HDL) and to provide a protective role against CHD (Miller and Miller, 1975; Rhoads et al., 1976; Castelli et al., 1977; Gordon et al., 1977). Its level has been shown to rise with increased physical activity (Lehtonen et al., 1979; Hartung et al., 1980; Peltonen et al., 1981).

HDLs have been attributed a protective role toward CHD by influencing the lipoprotein metabolism in such a way as to reduce the extra hepatic concentration of serum cholesterol. One of the beneficial effects of HDL is accomplished through its scavenger role of lipids during very low density lipoprotein (VLDL) lipolysis. HDL accepts on its

surface free cholesterol to transport it to the liver to be degraded (Miller and Miller, 1975; Reichl et al., 1980; Miller et al., 1985). A second possible effect is that HDL may interfere competitively with the uptake of LDL cholesterol by tissues (Carew et al., 1976).

There are many possible pathways by which the HDL-cholesterol (HDL-C) level may rise with exercise. The increased activity of the enzyme lipoprotein lipase (LPL) in the adipose tissue (Nikkila et al., 1978A and 1978B; Marniemi et al., 1980; Stubbe et al., 1983), the increased secretion of nascent HDL (Nikkila et al., 1978B), or the greater concentration of apoprotein A-I which activates lecithin-cholesterol acyltransferase (LCAT) to convert HDL3 into HDL2 (Krauss et al., 1977; Lehtonen et al., 1979) could contribute to a higher concentration of HDL-C.

The majority of the studies concerning exercise and lipoproteins have evaluated the modifications of lipoproteins, especially HDL, through aerobic exercise. Many researchers have shown that aerobic exercise increases the concentration of HDL-C (Lehtonen et al., 1979; Hartung et al., 1980; Peltonen et al., 1981), and more specifically HDL2 (Krauss et al., 1977; Miller et al., 1979; Nye et al., 1981; Stubbe et al., 1983; Herbert et al., 1984). However, little is known with respect to the effect of anaerobic training on the lipoprotein profile. Most comparative studies (Berg et al., 1980; Lehtonen and Viikari, 1980; Clarkson et al., 1981; Farrell et al., 1982) have been unable to reveal an increased HDL-C concentration in anaerobically trained athletes as compared to aerobically trained, mixed trained, untrained or sedentary subjects. However, in long term studies of 12 to 16 weeks (Lopez-S. et al., 1980; Johnson et al., 1983; Goldberg et al., 1984), the HDL-C concentration increased following resistive training.

None of the studies of anaerobic exercise measured the level of HDL's subfractions but a few researchers have assessed the effects of aerobic exercise on HDL2 and HDL3 concentrations. As an example, Nye et al. (1981) assessed the effects of 10 weeks of

group calisthenics on sedentary men. Although total HDL-C level was not modified, HDL2 and HDL3 concentrations changed significantly; the HDL2 level increased after the second week and the HDL3 level fell. The level of HDL's subfractions seems to be a more relevant lipoprotein parameter than total HDL-C since HDL2 reflects an efficient lipolytic process.

1.2 STATEMENT OF THE PROBLEM

The purpose of this study was to determine the effects in sedentary men (eleven experimental and four control subjects) of a 10 week resistive training program (anaerobic) on the lipoprotein profile, and more specifically the HDL subfractions.

1.3 SCOPE OF THE STUDY

This study was initiated to determine the effects of anaerobic exercise, more specifically resistive training, on the lipoprotein profile of previously sedentary men aged 25 to 45 years as compared to a control group. The total cholesterol (TC), triglyceride, total HDL-C, HDL2-C, HDL3-C. LDL-C and VLDL-C levels were measured from venous blood drawn after fasting at the pre-training level, 6th week of training, and post-training level for all subjects. Anthropometric measurements, muscular endurance and strength tests were administered pre- and post-training. The effect of anaerobic exercise throughout the training session on the lipoprotein profile was assessed. The resistive training program consisted of the following exercises for 3 sets of 10 reps: seated

overhead press, bench press, leg curl, leg press, leg extension, lat machine pulldown, and seated shoulder row. The subjects performed at 60% of 1RM (single repetition maximum) for the first week of training and gradually increasing to 75% of 1RM by the fourth week. The presence of significant differences in the results of the lipoprotein profile was detected by an analysis of variance with repeated measures and the Tukey test was used for the post-hoc analysis.

1.4 DEFINITION OF TERMS AND ABBREVIATIONS

1RM:

A single repetition maximum.

Aerobic Exercise:

Physical activity requiring the oxydative breakdown of carbohydrates, fats, and even proteins to provide energy for ATP resynthesis.

Anaerobic Exercise:

Physical activity requiring the phosphagen system (phosphocreatine) and lactic acid system (anaerobic glycolysis) to provide short-duration energy.

Apoprotein (also known as apolipoprotein or simply apo):

These proteins regulate lipoprotein metabolism by transporting lipids, acting as cofactors for enzymes of lipid metabolism, and maintaining the structure of lipoproteins.

Chylomicrons :

They are the largest lipoproteins which transport dietary triglyceride and cholesterol from the intestinal epithelium.

High Density Lipoproteins (HDL):

These lipoproteins of a density of 1,063-1,21 g/ml consist of an apolar core surrounded by a more polar coat of phospholipids and globular apoproteins. Its primary function is to transport cholesterol from the peripheral tissues to the liver for excretion or to be synthetized into bile acids. HDLs are subfractionned by their density: HDL2 (density = 1,063-1,125 g/ml) and HDL3 (density = 1,125-1,21 g/ml).

Hepatic Lipase (HL):

This enzyme, found on the surface of liver cells, has phospholipase and triacylglycerol hydrolase activities.

Lecithin-Cholesterol Acyltransferase (LCAT):

This enzyme catalyzes the conversion of cholesterol and lecithin to cholesterol ester and lysolecithin. The apoprotein A-I, found on HDL2, activates the enzyme.

Low Density Lipoproteins (LDL):

The LDLs (density = 1,006-1,063 g/ml) are the most important cholesterol carriers and represent the end product of VLDL.

Lipoproteins :

These water-soluble spherical macromolecules contain neutral lipids, triglyceride and/or cholesteryl esters, and are surrounded by a shell of phospholipids, unesterified cholesterol and apoproteins. They are divided by the density at which they float by ultracentrifugation. Lipoproteins are transport vehicles in circulation for endogenously synthesized and exogenous lipids.

Lipoprotein Lipase (LPL):

This key enzyme hydrolyses the triglycerides of chylomicrons and VLDLs into remnants and LDLs.

Repetitions (Reps):

A single repetition of any individual exercise.

Resistive Exercise:

Physical activity done with a resistance (weights) which combines the endurance (aerobic) and isometric (anaerobic) exercises.

Set:

A series of repetitions (reps) performed continuously.

Triglycerides :

These are esters of fatty acids with glycerol which are not soluble in water.

Very Low Density Lipoproteins (VLDL):

These lipoproteins of a density less than 1,006 g/ml transport triglycerides and cholesterol from the liver.

II

REVIEW OF LITERATURE

2.1 INTRODUCTION

The purpose of this study was to assess the effects in men of resistive training on the lipoprotein profile, and more specifically the HDL subfractions. The following literature will be reviewed: the structure, role and metabolism of lipoproteins, atherosclerosis and HDL, the influences on HDL-C levels, the effects of exercise on lipoprotein metabolism, and the effects of anaerobic training on the lipoprotein profile.

2.2 STRUCTURE AND ROLE OF LIPOPROTEINS

Lipids are a group of organic substances which are insoluble in water. Cholesterol, cholesteryl esters, triglycerides, phospholipids and fatty acids are known as lipids and they cannot circulate freely in the blood. Fatty acids are carried as a complex with albumin while the other lipids require a lipoprotein complex for transportation. Plasma lipoproteins facilitate the transport of lipids in the soluble form. These macromolecular complexes are spherical; their core contains neutral lipids, triglyceride and/or cholesteryl esters and is surrounded by a layer of phospholipids, unesterified cholesterol, and apoproteins (Patsch and Patsch, 1984). According to their lipid and protein contents, lipoproteins are classified by their densities.

The chylomicrons, the largest of the lipoproteins, have the lowest density (less than 0,95 g/ml) followed by VLDL (0,95-1,006 g/ml), LDL (1,006-1,063 g/ml), and HDL (1,063-1,210 g/ml) (Table 1). Due to their content, the chylomicrons and VLDLs are known as triglyceride-rich lipoproteins.

In addition to having different densities, lipoproteins also vary by their content of apoproteins. As well as maintaining the structure of lipoproteins, the apoproteins transport and redistribute lipids among tissues and act as a cofactor to the enzymes of the lipid metabolism. Apo A-I, an important particle in cholesterol transport, is found mostly in HDL and activates LCAT (Soutar et al., 1975; Jonas et al., 1984) whereas apo A-II is a structural component of HDL that may inhibit the activation of LCAT by apo A-I (Soutar et al., 1975). As regulators of the metabolism, apo B and apo E are recognition sites for lipoproteins on the surface of particles (Innerarity et al. 1981; Brown and Goldstein, 1984). Lipoprotein receptors, a primary control of lipoprotein homeostasis, bind lipoproteins to internalize them. By radiation inactivation, Innerarity et al. (1981) derived that one receptor can bind one apo E-HDL or four LDL which contain only apo B. Apo C-I activates LCAT (Soutar et al., 1975; Jonas et al., 1984), apo C-II acts as co-factor to activate LPL (LaRosa et al., 1970; Breckenridge et al., 1978) whereas apo C-III has been attributed many possible roles: the inhibition of the hepatic uptake of chylomicrons in rats (Shelburne et al., 1980), the inhibition of tissue LPL (Kashyap et al., 1979), and the activation of LCAT (Jonas et al., 1984). Apo D may transfer cholesteryl ester from HDL to other lipoproteins (Fielding and Fielding, 1980) but its function remains unclear. Tables 1 and 2 show these constituents and the physicochemical properties of the lipoproteins and apoproteins.

TABLE I

Characteristics of Lipoproteins

Variables	Chylomicron	VLDL	LDL	HDL
Density (g/ml)	0,95	0,95-1,006	1,006-1,063	1,063-1,210
Electrophoretic Mobility	Origin	Pre-beta	Beta	Alpha
Lipid-Protein Ratio	99:1	90:10	80:20	50:50
Major Lipids	Exogenous Triglycerides (90%)	Endogenous Triglycerides (50,4 - 51,5%)	Esters (37,5%) Free-cholesterol (7,5%)	Phospholipids (21-26%) Cholesterol (22%)
Sites of Production	Intestine	Liver	Metabolic end product of VLDL catabolism	Liver, Intestine, Metabolic end product of chylo. catabolism
Apoproteins	A-I * B * C-I ** C-II ** C-III** E *	B ** C-I ** C-II ** C-III** E *	B **	A-I ** A-II** D * E *

* minor
** major

(adapted from Voutilainen and Hietanen, 1982, p.3; Rifai, 1986, p.695; Roheim, 1986, p. 4C-5C)

TABLE 2

Characteristics of Apoproteins

Apoprotein	Site(s) of Synthesis	Function(s)	Major Lipoprotein Component
A-I	Intestine, Liver	- LCAT activation - reverse cholesterol transport	HDL
A-II	Intestine, Liver	- inhibit LCAT?	HDL
B-48	Intestine	- cholesterol clearance	Chylomicron
B-100	Liver	- cholesterol clearance	Chylomicron, VLDL, LDL
C-I	Liver	- activates LCAT	Chylomicron, VLDL
C-II	Liver	- activates LPL	Chylomicron, VLDL
C-III	Liver	- inhibits hepatic uptake of lipoproteins - inhibits LPL - activates LCAT	Chylomicron, VLDL
D	Liver?	- part of cholesterol ester transfer complex	HDL
E	Liver, Macrophages	- cholesterol clearance	Chylomicron, VLDL, IDL, HDL

(adapted from Voutilainen and Hietanen, 1982, p. 3; Rifai, 1986, p. 695; Roheim, 1986, p. 5C)

2.3 METABOLISM OF LIPOPROTEINS

The metabolic relationship of the lipoproteins has been researched extensively. Dietary fat is carried from the intestines by chylomicrons. These lipoproteins enter the venous circulation via the thoracic duct and acquire additional apo C from HDL when transferring from lymph to blood (Havel et al., 1973). The endogenous lipids are transported by VLDLs from the liver. Attached to endothelial cells, LPL is activated by apo C-II located on the surface of the chylomicrons and hydrolyzes the triglycerides of the chylomi- crons and VLDLs to form remnants, smaller particles of lipids enriched with cholesteryl esters (Higgins and Fielding, 1975). The chylomicron remnants contain apo E which permits quick removal from circulation by receptors in the liver (Sherrill et al., 1980). LPL hydrolyzes VLDL remnants (Eisenberg and Rachmilewitz, 1975), also known as intermediate density lipoproteins (IDL), into LDL. Figure 1 schematizes this lipoprotein metabolism.

Two pathways are possible for the LDLs. The LDL receptor pathway, which is saturable, permits the delivery of cholesterol to cells needing it, suppressing at the same time the rate-limiting enzyme in cholesterol biosynthesis, 3-hydroxy-3-methylglutaryl coenzyme A reductase (Brown et al., 1974). Acting as the recognition site, the apo B in the LDL is derived from VLDL catabolism (Eisenberg and Rachmilewitz, 1975). When this pathway is saturated, the excess LDL follows the scavenger pathway (Goldstein et al., 1980). After a certain prolonged time, this excess LDL is physically and chemically modified in such a way as to be taken by the scavenger pathway receptors. These receptors are found mostly in the reticuloendothelial system. Goldstein et al. (1980),

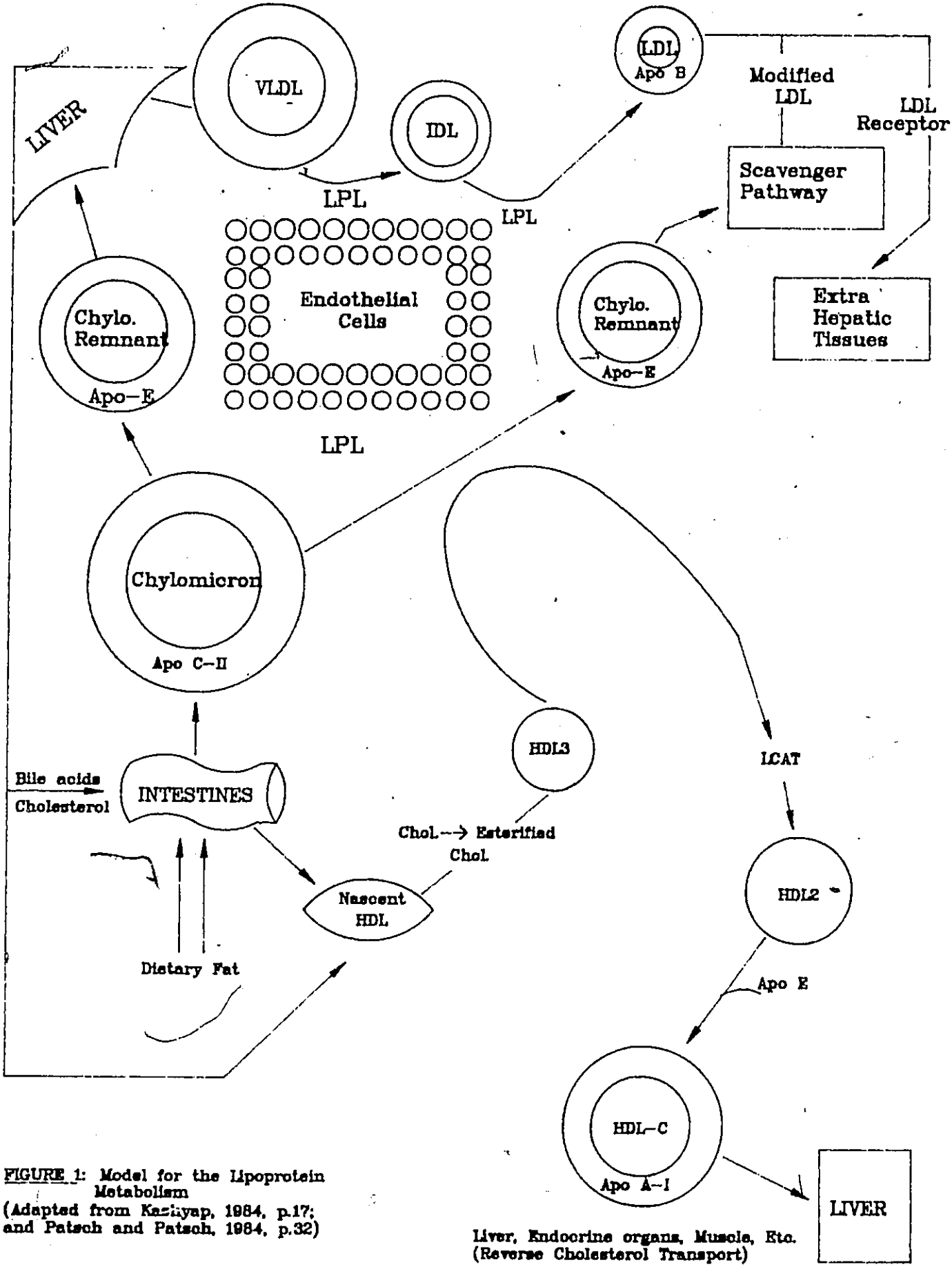

FIGURE 1: Model for the Lipoprotein Metabolism
(Adapted from Kashyap, 1984, p.17; and Patsch and Patsch, 1984, p.32)

Mahley et al. (1980) and Fainaru et al. (1982) have identified these modified lipoproteins in cholesterol-fed animals as B-VLDLs which deposit cholesteryl esters in macrophages to become foam cells, precursors of atherosclerotic lesions. Since B-VLDL contains apo B48 and apo B100, it indicates that B-VLDL consists of a mixture of chylomicrons remnants and VLDLs from the liver (Fainaru et al., 1982; Campbell et al., 1985). Van Lenten et al. (1985) found that the receptors of cholesterol-loaded cells recognize chylomicron remnants and B-VLDLs.

As LPL hydrolyzes triglycerides from the lipoproteins, an excess of surface components appears and is transferred to HDL or forms a disk-like lipid complex known as nascent HDL. Rubinstein et al. (1979) found a shift of apo C and apo E from VLDL to HDL following lipolysis in vitro. In Patsch et al. 's (1978) in vitro study of VLDL lipolysis, apoproteins, phospholipids, and cholesterol were transferred to HDL3 to form a stable "HDL2" similar to native HDL2 from human plasma. Compatible data was derived from Taskinen and Nikkila's (1981) in vivo study of the conversion of HDL3 to HDL2 through the lipolysis of triglyceride-rich lipoproteins. Therefore HDL3 becomes enriched with phospholipids and unesterified cholesterol. This cholesterol is then transformed into cholesteryl esters by LCAT, transforming the discoidal shape of HDL into a spherical particle. Analysing the interconversion of HDL3/HDL2 in vitro, Schmitz et al. 's (1981) found that LCAT promoted this interconversion irrespective of the presence or absence of triglyceride-rich lipoproteins or LPL. According to Dieplinger et al. (1985), the combination of LCAT and triglyceride-rich lipoproteins offers a better conversion of HDL3 to HDL2 in vitro. Since the cholesteryl ester moves into the core of HDL, this lipoprotein enlarges in size, forming more surface to bind cholesterol. Figure 2 illustrates this mechanism where layers of cholesteryl esters are added within the core of HDL.

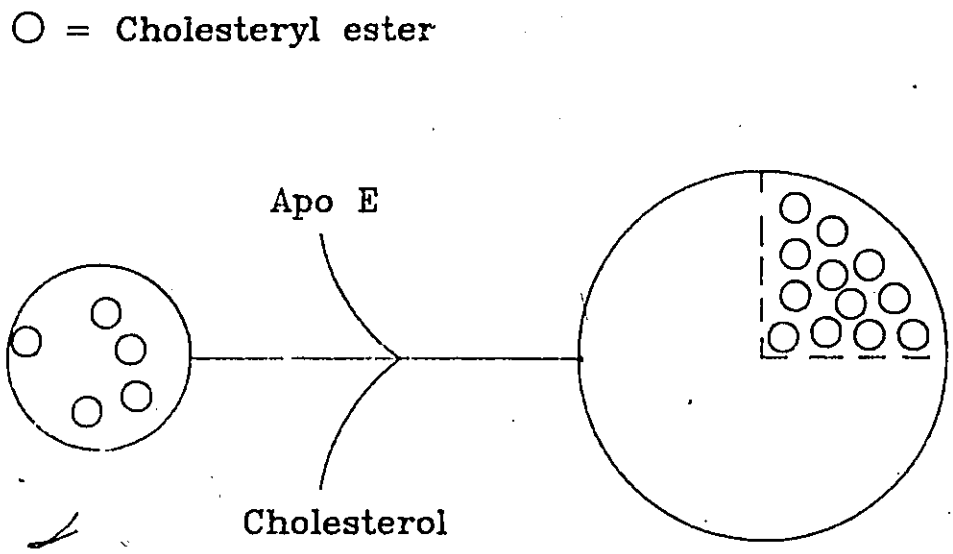

FIGURE 2: Schematic Illustration of the Formation of HDLs. Layers of Cholesteryl esters are added within the core of HDL.

(Adapted from Gordon et al., 1983, p. 6211)

As reviewed by Mahley and Innerarity (1983), it is suggested that HDL with apo E is formed from HDL without apo E (typical HDL) that has received cholesterol from various cells. Then, apo E is added and the HDL increases in size and floats at lower densities. Then, HDL can be taken up by receptors in the liver cells where the hydrolyzed cholesterol esters are excreted as neutral sterols in the bile. In the review of lipid markers for atherosclerosis by Kottke (1986), apo A-I is also given a major role in regulating the rate of removal of cholesterol esters. Situated on the surface of HDL, apo A-I picks up free cholesterol and transfers cholesterol esters from HDL to other particles; the rate of removal is dependent on the presence of apo A-I. All these processes result in the reverse transport of cholesterol from the periphery to the liver for bile acids or to endocrine organs for steroid synthesis.

It seems that the enzyme hepatic lipase (HL) may help to degrade and remove circulating HDL2. Kuusi et al. (1980) studied the relationship between HDL and HL on a group of young men; the HDL2 constituents (cholesterol, phospholipid and protein concentrations) showed a very significant negative correlation with postheparin HL activity ($p<0,001$). When rat hepatoma cells were exposed to HL-modified HDL in Bamberger et al 's (1983) study, the uptake of HDL-C increased; therefore, HDL exposed to HL delivers cholesterol to cells.

2.4 HDL AND ATHEROSCLEROSIS

Although many theories have attempted to explain the event initiating the formation of atheromatous plaque, it seems that the response of the smooth muscle cell, more specifically the irreversible synthetic-state phenotype of the smooth muscle cell (Campbell et al., 1985), to agents causing atherosclerosis is a key factor. LDL may be able to

provoke arterial smooth muscle cells to proliferate and to accumulate lipids (Scanu, 1978). By decreasing the effect of the agents promoting lipid accumulation or foam cells, the regression of plaques may be possible.

In a 15-year study of the lipoprotein profile of 6457 men, Goldbourt et al. (1985) found a J-type response of mortality from coronary heart disease (CHD) with increasing cholesterol level (by percentile) where little or no association is observed below a concentration of 240 mg/100 ml. Since HDL was inversely related in a linear fashion to mortality from CHD, a low HDL was determined to be more predictive of mortality from CHD than a high total cholesterol. In a case-control study of 6859 men and women in five populations, Castelli et al. 's (1977) study indicated that subjects with CHD had a mean HDL-C level lower than those subjects without CHD. As shown by other researchers (Miller and Miller, 1975; Rhoads et al., 1976; Gordon et al., 1977), HDL appears to provide some form of protection against atherosclerosis and CHD.

A few studies do not attribute HDL with such protective powers against CHD. Reed et al. (1986) found that total cholesterol predicted CHD as well as HDL in the 2122 men of the Honolulu Heart Program who participated in the Cooperative Lipoprotein Phenotyping Study. Although the HDL level was inversely asssociated with only nonfatal myocardial infarction (MI) and total CHD, total cholesterol and LDL levels were significantly associated with all clinical types of CHD (fatal CHD, nonfatal MI, angina/coronary insufficiency and total CHD); a dose-response pattern was more consistent with total cholesterol. With values from the World Health Organization data bank, Simon's (1986) calculated the interrelations of the lipid profile with coronary artery disease (CAD) mortality in 19 countries. Variation in the total cholesterol explained 45% of the interpopulation variation in CAD mortality. In a similar manner, 32% of interpopulation variation for CAD mortality was explained by HDL-C levels and 55% of this variation by total choles-

terol/HDL-C. Although this study had a a few methodological problems (lipid data gathered within a decade, no biochemical cross-standardization, and regional lipid data compared with national mortality rates), the high variation value of total cholesterol/HDL indicates that this ratio may be the best predictor of CAD mortality. From 7415 middle-aged men participating in the British Regional Heart Study, Pocock et al. (1986) compared the total cholesterol and HDL concentrations between ischaemic heart disease (IHD) cases and the rest of the group. The HDL level was significantly lower in the IHD cases but when the data was adjusted for other risk factors (age, blood pressure, body mass index and cigarette smoking), the HDL concentration had lost its statistical significance. Therefore, HDL was not a negative predictor of IHD.

Only one study analyzed the level of HDL and its subfractions (HDL2 and HDL3) in CHD subjects. Separating and quantitating HDL subclasses on "normal" subjects and on patients with CHD with his double precipitation method, Gidez et al. (1982) found a significantly lower level of HDL, HDL2 and HDL3 ($p<0,001$) in the latter group; in men with CHD, the HDL level was 28% lower, HDL2 was 44% lower, and HDL3 was 19% lower. The data was only age-adjusted. Since HDL2 showed proportionately greater changes than HDL3 in other abnormal conditions (i.e. hypercholesterolemia), HDL2 might be a more meaningful lipid component to study.

Some studies correlated angiographic images with lipoprotein profiles and demonstrated more obliterated coronaries with lower HDL-C levels. HDL-C levels were negatively correlated to a coronary atherosclerosis score ($p<0,01$) (Jenkins et al., 1978; Barboriak et al., 1979) or were lower as the severity and extent of coronary artery disease increased ($p<0,05$) (Kanamori et al., 1984) while the number of affected major coronary arteries was linearly correlated to serum apo B levels ($P<0,001$) (Luknarova et al., 1985). Wallentin and Sundin (1985) found a lower HDL2-C level in 74 CAD patients as

compared to controls ($p<0,05$), even when the influences of obesity and triglyceride level were taken into account. In Miller et al. 's (1981) study, subjects with lower HDL2 levels had more stenoses ($p<0,002$) and the best predictor for coronary artery disease was the HDL2/LDL2 ratio ($p<0,02$) where LDL2 is the LDL level without the IDL subfraction. Brook et al. (1982) compared the lipid profiles of 10 normolipidemic CHD patients (greater than 70% occlusion of at least one major coronary artery) and 10 normolipidemic controls. The groups did not differ in total cholesterol, triglycerides, VLDL or LDL values but all the components of HDL and its subfractions varied significantly. In the CHD patients, HDL-C was 39% lower, HDL-triglyceride 40% lower, and HDL-protein 53% lower ($p<0,01$). HDL3 had a slightly greater difference than HDL2: HDL3-C was reduced by 46% ($p<0,01$), HDL3-triglyceride by 36% ($p<0,05$) and HDL3-protein by 55% ($p<0,01$). The CHD patients also had a higher cholesterol/protein ratio for both HDL2 and HDL3 ($p<0,01$). Therefore, it seems that the concentration and composition of HDL and its subfractions are important in the pathogenesis of CHD.

In the Leiden Intervention Trial (Arntzenius et al., 1985), the effect of lowering cholesterol by diet on coronary atherosclerosis was assessed. Of the 35 male and 4 female patients who followed the diet for 2 years, 18 subjects that did not have a progression of disease exhibited lower values of total cholesterol ($p<0,01$) and total cholesterol/HDL-C ($p<0,001$), and higher values of HDL-C ($p<0,01$). Total cholesterol/HDL-C ratio correlated positively with lesion growth at the baseline level ($r=0,55$, $p<0,001$) and after 2 years of following the diet ($r=0,39$, $p=0,01$). A few other studies using angiography (Zampogna et al., 1980; Swanson et al., 1981; Kanamori et al., 1984; Luknarova et al., 1985; Schmidt et al., 1985) also found the HDL-C/total cholesterol ratio a good predictor of atherosclerosis; Swanson et al. (1981) determined that this ratio was predictive of the presence of CHD but not its severity.

Many mechanisms for HDL's protective role have been suggested. As indicated by Steinberg (1978), HDL is a better negative predictor for CHD than LDL is as a positive predictor. Glomset (1968) hypothesized that LCAT activity transforming free cholesterol to cholesterol ester in HDL would permit receiving additional free cholesterol; therefore, HDL, being the preferred substrate for LCAT, could remove cholesterol from peripheral tissues. Miller and Miller (1975) proposed that HDL carries cholesterol from the peripheral tissues to the liver to be degraded and excreted. In in vitro studies, HDL (Stein et al., 1976) and HDL3 (Ho et al., 1980) helped to remove free cholesterol from fibroblasts, smooth muscle cells, and macrophages. Ho et al. (1980) suggested that the HDL constituent apo A-I could remove the macrophage cholesterol. By studying HDL metabolism following injection of acetylated or native human LDL (8,4 times the circulating LDL pool) in rabbits, Miller et al. (1985) provided in vivo direct evidence that HDL is involved in reverse cholesterol transport.

Another protective role for HDL would be to reduce the cellular uptake and degradation of LDL by competing for its binding sites. Carew et al. (1976) measured surface binding of HDL and LDL in the cultured porcine smooth-muscle cells. With the presence of HDL, the binding, internalisation and degradation of LDL were reduced. This phenomenon was a function of the concentration of HDL present. Therefore, HDL may directly reduce the atherogenic process. According to the availability of HDL, the quantity and quality of the components leading to atherosclerosis accepted by the reticuloendothelial cells (such as macrophages) may vary.

2.5 INFLUENCES ON HDL-C LEVELS

Because it is difficult to study one isolated variable in human subjects, it becomes important to analyze the possibility of the influences of other factors. When assessing the effect of anaerobic exercise on the lipoprotein profile, one may encounter the influences of gender, age, body composition, dietary pattern and androgen use.

2.5.1 Gender

Many studies have consistently found a higher level of HDL, more specifically HDL2, in pre-menopausal women. In Shepherd et al. 's (1980) study of 13 men (mean age = 29 years) and 12 women (mean age = 30 years), the protein and lipid composition of HDL were analyzed. The only gender-related differences in the lipoprotein profile were the apo A-I and HDL-C levels which were significantly higher ($p<0,01$) in the women than in the men. The level of HDL2 in the plasma of women was 2,4 times higher than for the men (0,75 vs 0,31 g/l, $p<0,01$) while the level of HDL3 was similar for both genders. In 1978, Shepherd et al. determined that the gender-related differences in the HDL profile were not due to changes in the apo A-I and apo A-II metabolisms. In the Framingham study (Gordon et al., 1977), the protective role of HDL against CHD was analyzed in 2815 men and women aged 49 to 82 years. The HDL-C level was higher in women than in men, even though the women's values decreased slightly from the ages of 50 to 80. Mjos et al. (1977) also noted a higher HDL-C level for women after adolescence.

Investigating the relationship between triglyceride-rich lipoproteins and HDL2 and HDL3 in the post-prandial phase of 6 men and 6 women, Baggio et al. (1980) found a significantly higher level of HDL2 (11,4 vs 7,5 mg/100 ml, $p<0,05$) in women as compared to men. Utilizing the double precipitation method to determine HDL2 and HDL3 levels in 273 men and 180 women, Gidez et al. (1982) detected higher concentrations of HDL2 (by 40%) (20,0 vs 14,3 mg/dl) and total HDL-C (55,5 vs 45,8 mg/dl) in women ($p<0,001$) as compared to men.

From 14 male and 14 female subjects participating in an acute, moderate-intensity bicycle exercise, the results of Lennon et al. (1983) indicated that the female subjects had higher HDL-C levels ($p<0,05$) at rest and at 40 minutes of exercise. Because the women's HDL-C levels returned to the rest values more rapidly than the men's, no gender-related differences in HDL-C existed at 15 minutes post-exercise. Therefore, it is well recognized that women have higher resting values of HDL and HDL2 than men.

2.5.2 Age

Studying the lipoprotein profiles of families in Northern Norway with and without a history of myocardial infarction, Mjos et al. (1977) observed a gender and age differences of the HDL-C level. Women had higher HDL-C concentrations; this gender difference occurred at puberty, reached its peak (difference of 7,4 mg/dl) between the ages of 30-39 years ($p<0,01$) and fell slightly by 40-49 years. Further age groups and the use of contraceptive pills were not analyzed in this study.

The Lipid Research Clinics Program is an international study of different geographical populations to analyze the determinants of lipids and lipoproteins and their cardio-

vascular consequences. Heiss et al. (1980) reported the characteristics of the lipids and lipoproteins of this Program as related to gender, age and the use of gonadal hormones by women of 4756 white participants aged 20 to 59 years. As shown in Figure 3, TC in men increased from 162 mg/dl at 20-24 years old to a peak of 215 mg/dl at 50-54 years. The slope of this increase was greatest for 20 to 34 years and then fell to a plateau at 50-59 years. As indicated in Figure 4, the LDL-C increased from 20-24 years (103 mg/dl) to 40-49 years (144 mg/dl) and reached a plateau at 50-59 years. HDL-C remained stable (44 mg/dl) from 20 to 54 years but rose by 4 mg/dl from 55 to 59 years. VLDL-C demonstrated a parabolic relationship where the maximum value was at 40-44 years (26 mg/dl). For women not using exogenous gonadal hormones, a curvilinear increase of TC from 20-24 years (162 mg/dl) to 50-59 years (231 mg/dl) was observed. LDL-C increased with age (from 98 to 150 mg/dl) with a steeper slope during middle age, VLDL-C also increased (especially after 25 years old), and HDL-C showed a moderate linear rise from 20-24 years (52 mg/dl) to 55-59 years (60 mg/dl). Wo- men using exogenous gonadal hormones demonstrated a linear increase with age for all parameters: TC (from 178 to 222 mg/dl), LDL-C (from 108 to 133 mg/dl), HDL-C (from 55 to 71 mg/dl), and VLDL-C (from 15 to 19 mg/dl). These data are shown in Figures 3 and 4.

As determined by the double precipitation procedure, the Gidez et al. 's (1982) study had findings similar to the previous study. The effect of age was non-significant on HDL2 for men and women. HDL3 levels were lower for men aged less than 25 years than those 25-54 years old ($p<0,05$). After 45 years old, the HDL3 values of women were higher than those younger ($p<0,05$). For the women, this study did not differentiate between users and non-users of exogenous gonodal hormones. Ferns et al. (1986) reported similar variations of HDL subfractions in men to Gidez et al. 's (1982) study. Table 3 indicates the cholesterol values of HDL, HDL2, HDL3 and plasma lipids of "normal" male subjects as analyzed by the double precipitation method.

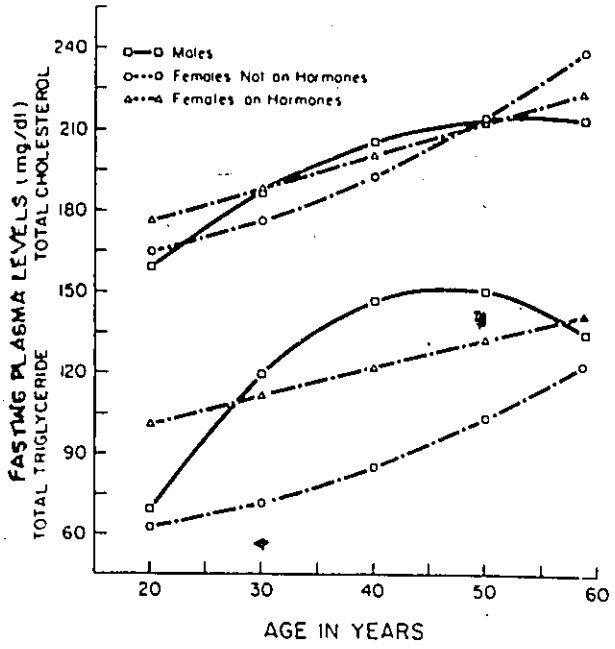

Figure 3: Regression Estimates of Triglyceride & TC Levels. Lipid Research Clinics Prevalence Study. (from Heiss et al., 1980, p. 307, by permission of the American Heart Association, Inc.)

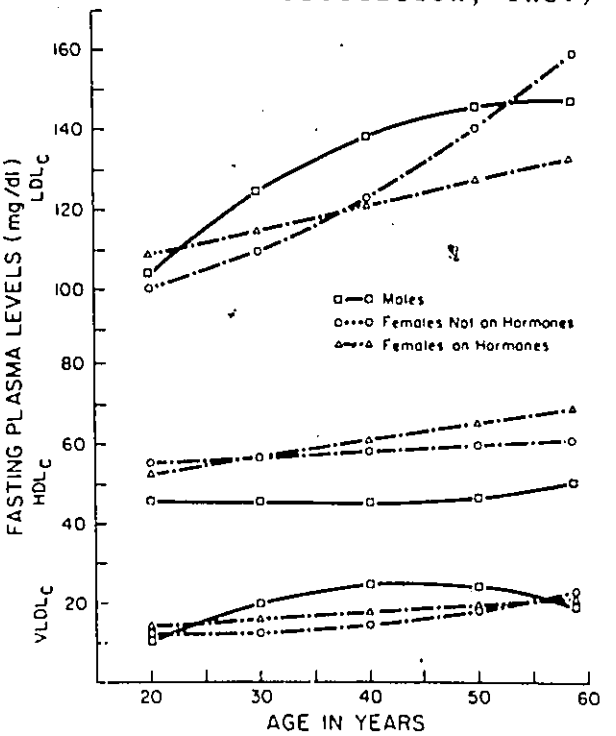

Figure 4: Regression Estimates of LDL-C, HDL-C and VLDL-C Levels. Lipid Research Clinics Prevalence Study. (from Heiss et al., 1980, p. 307, by permission of the American Heart Association, Inc.)

TABLE 3

Normal Concentrations of HDL, HDL2, HDL3, and Lipids

Age	HDL	"HDL2"	"HDL3"	TG	Chol
	cholesterol, mg/dl			mg/dl	
Males					
25	44.7 ± 9.9	15.1 ± 7.9	29.6 ± 5.9	69 ± 34	164 ± 30
25-34	47.0 ± 12.6	13.3 ± 6.5	33.7 ± 8.5	90 ± 60	181 ± 33
35-44	44.9 ± 11.6	12.1 ± 7.6	32.8 ± 7.5	105 ± 55	196 ± 33
45-54	48.0 ± 13.3	14.8 ± 8.3	33.2 ± 8.1	96 ± 54	206 ± 29
55-64	46.0 ± 12.7	15.6 ± 8.0	30.4 ± 7.2	104 ± 54	223 ± 30

(adapted from Gidez et al., 1982, p. 1215)

2.5.3 Body Composition

Many studies do not assess the effect of weight loss or gain on lipoprotein levels. The following investigations discuss the influence of body composition on lipoprotein profiles. Using 36 exercising men who were previously sedentary and 28 sedentary control men, Williams et al. (1983) analyzed the correlation between HDL-C concentrations and body weight changes. After one year, the experimental group of runners had lost significantly more body mass (-1,4 vs +1,1 kg) and percent body fat (-1,3 vs +2,8 %) ($p<0,001$) than the control group. The most significant relationship was the inverse association between weight changes and HDL-C levels and HDL2 mass and not the higher HDL-C and HDL2 levels in the runners as compared to the control group. Sopko et al. (1985) studied the effect of exercise and weight loss on the lipid profiles of young obese men. Independently, exercise ($p=0,009$) and weight loss ($p=0,01$) increased HDL-C levels but both interventions together produced a summation of exercise and weight loss effects on HDL-C. Only the TC/HDL-C ratio varied with weight loss alone ($p=0,03$) while a synergistic effect of both treatments reduced the TC ($p=0,03$), triglyceride ($p=0,008$) and VLDL-C ($p=0,019$) levels. Using meta-analysis, Tran and Weltman (1985) analyzed ninety-five studies investigating changes in serum lipoprotein profiles following exercise. The studies were grouped in three categories where subjects gained, lost or maintained their body weight following training. The triglyceride, TC, HDL-C, LDL-C levels and TC/HDL-C ratio varied in the three groups. The data indicated that the combination of exercise and body weight loss produced the greatest reduction in TC and LDL-C levels.

When studying the relationship of serum lipoproteins and CHD in Hawaii Japanese men, Rhoads et al. (1976) found a strong negative correlation between HDL and the

sum of skinfolds. Since HDL may be formed from the surface components of the lypolysis of triglyceride-rich lipoproteins, a decreased activity of LPL in adipose tissue of obese subjects might explain the negative correlation between HDL-C and body mass. Castelli et al. (1977), Sauar et al. (1980), Neel et al. (1984), Chang et al. (1985) and Sopko et al. (1985) showed an inverse relationship between triglycerides and HDL-C concentrations. Chang et al. (1985) found a decreased HDL particle size and proportion of HDL2 in men and women having an increasing plasma triglyceride concentration. Poapst et al. (1985) have indicated by four different methods that a variation in plasma triglyceride concentration causes a change in the number, and not in the size, of triglyceride-rich lipoproteins.

Glueck et al. (1980) assessed the relationship of HDL-C levels and anthropometry of 3517 males and 3348 females participating in the Lipid Research Clinics Program. The weight and measures of ponderosity (weight/height, weight/height2, weight/height3) were significantly and inversely related to HDL-C levels and positively related to plasma triglyceride concentrations; the Quetelet index (weight/height2) provided the best correlation for adults of both genders (regression coefficients ranged from -6,33 to -10,93). Even after covariance adjustments considering age, smoking habits and use of exogenous gonodal hormone, the Quetelet index correlated significantly to the HDL-C level in the adult population aged 20 to 64 years ($p<0,0001$). The differences in the HDL-C levels between the 10th, 50th and 90th percentiles of the Quetelet index were within the range reported as highly significant differences for CHD subjects over 50 years old.

2.5.4 Dietary Effects

In most longitudinal studies, it is difficult to supervise and control the dietary intake and pattern of the subjects. The best solution seems to try to maintain the quality of the nutrients throughout the study. The two following studies indicate the minor effects of dietary factors on HDL-C.

Studying the relationship between the lipoprotein profiles and the diet of 59 marathon runners, 85 joggers, and 74 inactive men, Hartung et al. (1980) assessed the food-intake, the lipoprotein profile and the distance run for each subjects. The HDL-C levels for the marathon runners (65 mg/dl), the joggers (58 mg/dl), and inactive men (43 mg/dl) showed a positive relationship to the distance run. Although the marathon runners and joggers indicated that they ate less red meat, bacon and sausage than the inactive men, dietary factors did not modify HDL-C concentrations.

Twenty-three male physical education instructors participated in Kiens et al. 's (1981) study. While maintaining their body weight and physical activity level, the experimental group followed a fat-rich diet (54% fat) for 4 weeks and then a fat-poor diet (29% fat) for another 4 weeks. HDL-C levels did not change significantly following these large dietary changes; the levels of apo B and the triglyceride-content of HDL decreased with both diets ($p<0,05$) and the levels of apo A-I and A-II decreased with the fat-poor diet ($p<0,05$). The only changes that occurred between both diets are the reduced levels of apo A-I and A-II. Therefore, variations in the intake of neutral fat have little influence on the HDL-C concentration.

Studying the effect of saturated (S) and polyunsaturated (P) fat diets on HDL-C levels, Shepherd et al. (1977) administered to 4 men isocaloric diets containing 300 mg

cholesterol per day and fat where diet S had a P/S ratio = 0,25 and diet P had a P/S ratio = 4,0. Diet P caused a 31% fall in HDL2 ($p<0,05$), 21% decrease in apo A-I ($p<0,01$) and 26% diminution in the rate of synthesis of apo A-I (15,6 vs 21,0 mg/kg/d, $p<0,01$) as measured by electroimmunoassay. Therefore, a diet containing mostly polyunsaturated fat would reduce the synthesis of apo A-I which would be reflected by a fall in HDL2 level. Sacks et al. (1985) studied the influence of dairy products on the lipoprotein profile of 75 adult lactovegetarians. Being the major source of saturated fat and cholesterol, the dairy products correlated positively with TC and LDL-C levels. Although the influence of physical activity was not considered, HDL-C did not correlate significantly with any of the nutrients. Therefore, the LDL-C level may be more sensitive to nutrient intake than the HDL-C concentration is.

In the Lipid Research Clinics Program Prevalence Study, Ernst et al. (1980) did not find a significant association between HDL-C levels and fatty acids (saturated, monounsaturated, and polyunsaturated). Total carbohydrate, sucrose and starch intakes negatively affected HDL-C concentrations. The strongest correlation ($p<0,001$) was the positive relationship between HDL-C and alcohol consumption of any kind; the 20-29 years age group had the weakest association while the 60 years and over group demonstrated the strongest association. The multiple linear regression used for this cross-sectional data did not consider the influence of smoking and physical activity on HDL and was based on a 24-hour dietary recall for the nutrient intake and alcohol consumption (as well as an interview of a 7-day recall for the alcohol use only). Williams et al. (1985) studied the effects of diet and alcohol on HDL subfractions of 77 middle-aged sedentary men. The HDL3-mass concentration, analyzed by a schlieren curve, was associated negatively to nutrients intake (total carbohydrates ($p<0,01$) and starch ($p<0,0001$)) and positively throughout the range of alcohol consumption levels ($p<0,0001$). HDL2 showed little relationship to these nutritional components.

Therefore, the longitudinal studies indicate little influence of dietary fat on HDL-C level while Ernst et al. 's (1980) cross-sectional study and Williams et al. 's (1985) investigation detected the stronger influence of alcohol and the effects of carbohydrate, sucrose and starch. Goldberg and Schonfeld (1985) reviewed the studies assessing the effects of diet on lipoprotein metabolism. In general, a greater intake of saturated fats increased the TC, LDL-C and IDL-C levels; dietary cholesterol elevated the TC, LDL-C and HDL-C levels; all plasma cholesterol levels were reduced with polyunsaturated fats; carbohydrates increased the triglyceride level but reduced the HDL2 concentration; soy protein lowered non-significantly the plasma cholesterol levels as compared to animal protein which produced higher cholesterol levels.

2.5.5 Androgen Use

With some weight-training programs, athletes will self-administer anabolic-androgenic steroids to improve their performance. Many studies investigated the effect of steroids on the lipoprotein profile and analyzed its effect in combination with weight-training.

Haffner et al. (1983) studied the mechanism whereby an anabolic steroid, stanozolol, reduces the HDL-C and apoproteins levels. Six subjects, 4 postmenopausal women aged 56-79 years and 2 men aged 46 and 56 years, were treated with the steroid (6 mg/day). HDL turnover and postheparin plasma lipolytic activity were analyzed. The LDL-C level increased by 13% (from 160 to 181 mg/dl, $p<0,025$), the HDL-C concentration decreased by 51% (from 59 to 29 mg/dl, $p<0,005$), the apo A-I synthesis was reduced, the hepatic triglyceride lipase activity and the HDL catabolism were increased. Therefore, this anabolic steroid is detrimental to the lipoprotein profile.

Hurley et al. (1984B) assessed the effect of androgen use on strength-trained athletes, bodybuilders and powerlifters. Eight body-builders and four powerlifters self-administered exogenous androgens (of various dose equivalents ranging from 0,8 to 27,75) for 4-6 weeks. Body weight increased ($p<0,05$) without alterations in body fat. In the lipoprotein profile, TC (from 185 to 232 mg/dl, $p<0,05$) and LDL-C (from 117 to 188 mg/dl, $p<0,0001$) levels rose while the HDL-C concentration decreased by 55 +/- 4% (from 51 to 23 mg/dl, $p<0,001$) as did HDL2-C by 55 +/- 5% (from 11 to 5 mg/dl, $p<0,001$). Therefore LDL-C/HDL-C ratio increased by 280 +/- 40% (from 2,5 to 9,5, $p<0,001$). Kantor et al. (1985) also studied the effects of androgen use on HDL subfractions of 5 weightlifters as compared to non-users. The users of androgenic hormones had a lower HDL-C (26 vs 50 mg/dl, $p<0,05$) and HDL2-C (6 vs 22 mg/dl, $p<0,05$) than non-users, possibly by stimulating the activity of HL which was 3 times greater in users (27,99 vs 11,15 umol FFA/ml/hr, $p<0,001$) and correlated with HDL-C and HDL2-C ($r=-0,82$ and $-0,81$ respectively, $p<0,01$).

To examine the effects of steroids on the lipoprotein profile, 11 male body builders and 3 male power lifters were studied by Webb et al. (1984) with and without the administration of exogenous anabolic-androgenic steroids. These drugs reduced HDL-C levels by 50% (28,5 vs 60,9 mg/dl, $p<0,01$), elevated LDL-C levels (150 vs 125 mg/dl, $p<0,05$), and almost tripled the LDL-C/HDL-C ratio (6,0 vs 2,2, $p<0,01$). These variations in HDL-C did not correlate to changes in body weight or the Quetelet index (weight/height2). Some residual effects of the steroids on HDL-C were still present at an average of 7,3 months after cessation of their use. The longer the athletes were off the steroids, the higher was their HDL-C level.

Alen and Rahkila (1984) assessed the effects of androgenic steroids and strength training on serum lipids of power athletes. The seven athletes trained for 8 weeks with

the use of anabolic steroids (average of 45 mg/day) while seven other athletes served as a control group. Both groups followed a similar diet. None of the athletes had used steroids for the previous 3 months but, as indicated in the previous study of Webb et al. (1984), a residual effect might still have been present. In the experimental group, the HDL-C level decreased by 54% (from 1,42 to 0,67 mmol/l, $p<0,001$).

Serum lipoproteins can also be influenced by vitamins. Serfontein et al. (1983) demonstrated that alpha-tocopherol (vitamin E) increased LDL-C by 11,7% (2,56 to 2,86 mmol/l, $p<0,01$), TC by 6,3% (4,26 to 4,53 mmol/l, $p<0,05$), and HDL3 by 7,4% (0,81 to 0,87 mmol/l, $p<0,05$). Therefore, not only steroids but also vitamins can produce an atherogenic lipoprotein profile.

Steroid use reduces HDL-C levels and increases LDL-C levels, producing a very unfavorable lipoprotein profile. Therefore, it becomes important to always assess any previous drug use in studies examining weight-training.

2.5.6 Summary

In order to investigate the isolated effect of one independent variable such as weight-training on the lipoprotein profile, a number of factors must be assessed. One such factor is age; therefore, all subjects should be within a similar age-group of the same gender. Since steroids unfavorably influence the lipoprotein profile, the previous or current use of androgens must be monitored as well as the period elapsed since the cessation of its use. Although dietary factors seem to be of only slight importance to the cholesterol level, a dietary pattern should be established and/or maintained throughout the study. Body composition should also be assessed since weight changes influence the lipoprotein profile.

2.6 THE EFFECTS OF EXERCISE ON LIPOPROTEIN METABOLISM

Since physical activity appears to be beneficial in reducing CHD, recent research is concentrating on the effect of exercise on HDL, the favorable lipoprotein. Most studies have directed their attention to aerobic activities where the concentrations of HDL-C (Lehtonen et al., 1979; Hartung et al., 1980; Peltonen et al., 1981) and HDL2-C (Krauss et al., 1977; Miller et al., 1979; Nye et al., 1981; Stubbe et al., 1983; Herbert et al., 1984) increased. Many hypotheses are proposed for this elevation of HDL-C through exercise.

2.6.1 Lipoprotein Lipase

The most popular theory for the increased HDL-C associates it with an increased activity of LPL in adipose tissue. The pioneers of this hypothesis are Nikkila et al. (1978A) who measured by biopsies the LPL activity in the adipose tissues and the skeletal muscles of 21 male and 20 female subjects. In the adipose tissue, LPL activity was higher in women than in men but LPL activity in the skeletal muscle was similar for both sexes. Although it was not related to skeletal muscle LPL activity, the HDL-C level was positively correlated with the LPL activity in the adipose tissue, more significantly for women ($r=+0,57$, $p<0,01$) than for men ($r=+0,43$, $p<0,05$). It was therefore proposed that HDL-C is elevated when the catabolism of triglyceride-rich lipoproteins is increased.

Pursuing this theory, Nikkila et al. (1978B) then studied the LPL activity in the adipose tissues and skeletal muscles of male competitive sprinters and long distance runners of both sexes as compared to physically less-active control subjects. The female

($p<0,05$) and male ($p<0,01$) long distance runners had higher HDL-C values than the sprinters, whose levels were similar to those of the control group. In the adipose tissue, the LPL activity of the sprinters ressembled that of controls. In long distance runners, the LPL activity was 2,7 times higher than the controls for men ($p<0,05$) and similar to control subjects for women. For the male runners as a group, the adipose tissue LPL activity correlated significantly with the average amount of kilometers run weekly ($r=+0,55$, $p<0,05$). In the skeletal muscle, the long distance runners of both sexes had 1,7 times higher LPL activity than the controls ($p<0,01$). This enzymatic activity did not correlate with the distance run or the fiber composition of the skeletal muscle. The best correlation was found between adipose tissue LPL activity per tissue weight and HDL-C levels ($r=+0,72$, $p<0,001$) in males. These researchers suggested that LPL activity increases in adipose tissue to replenish more rapidly fat stores from circulating chylomicrons and VLDL triglycerides. To maintain their weight constant, the runners increased their dietary intake. Nikkila et al. (1978B) also suggested that this elevated nutrient flux could induce LPL activity.

Krauss et al. (1979) also observed an increased LPL activity in 12 male runners ($p<0,05$) as compared to 20 control subjects (5,0 vs 3,6 mEq fatty acid/ml/hr); this enzymatic activity in the runners correlated negatively to VLDL ($r=0,58$, $p<0,05$) and positively to HDL ($r=0,51$, $p<0,01$). To study the acute effect of exercise on the LPL activity, Taskinen et al. (1980) analyzed the muscle and adipose tissue biopsies and blood of 10 male subjects (17 to 42 years) running 80 to 120 kilometers per week. After a similar meal and a 10-hour fast, the subjects ran 20 km. After exercise, the LPL activity was higher in the skeletal muscle of all the subjects (by 112%, from 1,70 to 3,60 umol FFA/h/g, $p<0,01$) and in the adipose tissue of 8 subjects (by 20%, from 2,33 to 2,80 umol FFA/h/g, $p<0,05$). Although there was no significant correlation between the lipoprotein levels and LPL activity, the runners had a LPL activity in the skeletal muscle twice

as high as that found in normal men. The authors suggested that this acute elevation of LPL activity during exercise liberates free fatty acids from VLDL triglycerides to replenish muscle triglycerides.

Marniemi et al. (1980) examined the LPL activity in postheparin plasma and adipose tissue in regularly training subjects with different intensities of training. The 28 male subjects were divided into 4 groups: very active group (heavy exercise - 4 times per week), active group (heavy exercise - 3 hours per week), light exercise (4 hours per week), and a less-active group. The first two groups were categorized as actively training (n=17) while the others were the non-training control group (n=11). The controls had a higher hepatic lipase activity in postheparin plasma ($p<0,02$) while the LPL activity in adipose tissue was 70% higher in the trained subjects (1,8 vs 3,0 umol FFA/h/g w.wt., $p<0,10$). This LPL activity was related to the intensity of training ($r=0,38$, $p<0,05$) of the 4 groups. The lower HL activity in the trained subjects would permit an increased level of HDL2.

In a longitudinal study of 15 weeks of moderate aerobic training, Peltonen et al. (1981) analyzed the serum lipids and lipase activities of 20 men in the experimental group and 7 male controls. In addition to increasing their performance, the experimental group increased by 7% their HDL-C level (from 1,20 to 1,28 mmole/l, $p<0,01$) and by 11% the HDL/total cholesterol ratio (from 0,215 to 0,238, $p<0,001$), and decreased their LDL-C ($p<0,05$) and insulin ($p<0,05$) values. Postheparin plasma LPL activity was increased ($p<0,001$) in the experimental group but it was not correlated to HDL-C. LPL activity in the adipose tissue did not vary significantly as observed in the previous studies but this discrepancy may be due to different measurement techniques.

Lithell et al. (1981) investigated the lipid metabolism of 6 elite soldiers during a 10 day march with a heavy pack at varying intensities of physical effort. As compared to

control days, the free fatty acids (FFA) and muscle LPL levels increased following days of heavy work ($p<0,01$), the triglyceride values were lowest after 3 days of heavy work ($p<0,05$), and the 24 hour epinephrine excretion rates were directly related to the muscle LPL activities. The data seemed to indicate that heavy prolonged exercise increased the muscle LPL activity, which was related to the epinephrine urine excretion, and with a higher FFA level, could explain the variation of plasma triglycerides.

A Swedish study by Stubbe et al. (1983) assessed the lipolytic activities during cycling training of 18 sedentary men: 12 men in group A who trained for 6 weeks (3 times per week) at heavy intensity (at 85% of maximal heart rate) and 6 men in group B where the training intensity was moderate (at 70% of maximal rate) but the training lasted 12 weeks. The decreased LDL-C and increased HDL-C levels were more pronounced in group B but HDL-C levels fell in the last 6 weeks. Although LPL activity did not vary in the skeletal muscle, it did increase by almost 50% (from 101,0 to 153,0 pkat/g lipid, $p<0,05$) in the adipose tissue of group A and was correlated to HDL-C levels ($r=0,50$, $p<0,05$). The dietary fat intake did correlate with HDL-C levels ($r=0,65$, $p<0,01$) for all subjects.

Hurley et al. (1984A) compared the LPL and HL activities in bodybuilders, powerlifters, and runners. The activity of both enzymes did not differ between the groups. HL correlated with HDL-C ($r=-0,72$, $p<0,001$) and HDL2-C ($r=-0,67$, $p<0,001$); LPL was not related to these lipoprotein parameters. The authors concluded that HL may influence HDL metabolism in exercise more than LPL does.

Amongst the eight studies concerning the effect of exercise on the LPL activity, four of them indicated an increased LPL activity in the adipose tissue. The elevated value of this key enzyme could explain the lower VLDL and LDL levels in athletes due to a rapid turnover of triglyceride-rich lipoproteins (Nikkila et al., 1978B). Therefore, the triglycer-

ide removal in the circulation is accelerated. As indicated by Nikkila et al. (1978B), it is always possible that an increased secretion of nascent HDL could explain the elevated HDL-C levels following exercise.

2.6.2 Lecithin-Cholesterol Acyltransferase

Since LCAT esterifies the cholesterol from HDL3 to HDL2, the increased activity of this enzyme could explain the elevated level of HDL2 following physical training. However, studies on the effect of exercise on LCAT activity are scarce. Lopez-S. et al. (1974) seem to be the first to investigate this hypothesis. Amongst 13 subjects exercising 4 times per week at an intensity of 7 Mets for 7 weeks, only 4 subjects showed a tendency for an increased LCAT activity. In a 4 month cross-over study of two groups of rats swimming daily in either the first 2 months or the last 2 months, Simko and Kelley (1979) found an elevated LCAT activity in the exercising rats ($p<0,05$) as well as a decreased red blood cell cholesterol and phospholipids. The increased enzymatic activity could be related to the fall of the red blood cell lipids.

Glomset (1968) postulated that LCAT is a transport mechanism for cholesterol from the peripheral tissues to the liver; therefore, HDL-C levels should correlate with LCAT activity. Studying the relationship between the rate of cholesteryl ester formation and HDL-C levels of 2 groups of men with HDL-C levels under the 10th or above the 90th percentiles, Stokke and Enger (1979) could not demonstrate this hypothesis. This study, being of a comparative nature, does not necessarily reflect the LCAT activity following physical training.

After 15 weeks of training (running and skiing), an increased LCAT activity (from 13,9 +/- 2,2 to 31,2 +/- 3,2 nmol/ml/h, $p<0,001$) was observed in 19 subjects as compared to 6 controls (Peltonen et al., 1980). Athough there was no difference at baseline level between the two groups for body weight, age and physical fitness, the experimental subjects increased their physical fitness ($p<0,001$). It was proposed that an increased apo A-I level could explain the greater LCAT activity.

Marniemi et al. (1982) assessed the effect of 2 months of military training of 35 conscripts, as compared to 26 sportsmen (10 hours/week of aerobic activity), on the LCAT activity. At baseline values, the serum lipids levels did not differ between the two groups although the LCAT activity was higher in the sportsmen. With the training, the conscripts increased their VO2max (41,2 +/- 1,26 to 53,1 +/- 1,39 ml/min/kg, $p<0,001$), their HDL-C level (1,22 +/- 0,03 to 1,34 +/- 0,04 mmol/l, $p<0,001$) and their LCAT activity (17,1 +/- 0,53 to 18,6 +/- 0,56 % cholesterol esterified, $p<0,01$). When analyzing the different correlations, the authors found that the association between the LCAT activity and total cholesterol was due to variations in HDL-C levels. Therefore, LCAT may be responsible for the changes in the serum lipids observed with physical activity, such as increasing HDL-C values. In a study of the acute variations of LCAT activity following exercise, Dufaux et al. (1986) investigated the delayed effects of a 3 hour run on the serum lipoproteins. Paradoxically, the acivity of LCAT was increased 3 hours after the race (57,9 to 71,0 mmol/ml/hour, $p<0,05$) while the unesterified cholesterol to cholesteryl esters ratio was also elevated (0,224 to 0,245, $p<0,05$). Therefore, the role of LCAT at exercise remains obscure.

2.6.3 Apoproteins

Since the subfractions of HDL contain apoproteins, it is interesting to investigate if the apo A-I and apo A-II concentrations will increase with exercise as does HDL-C levels. In runners, as compared to controls, the HDL2 concentration is increased as is the apo A-I ($p<0,001$) level but not the apo A-II (Krauss et al., 1977). In Miller et al. 's (1979) study of 11 young males exercising regularly at varying intensities, aerobic capacity was positively correlated with HDL-C concentrations ($r=0,81$, $p<0,01$) but not significantly with the increased apo A-I level.

When examining the effect of exercise on HDL's apoproteins, Lehtonen et al. (1979) observed a higher apo A-I (2,16 vs 1,65 g/l, $p<0,001$) and HDL-C (1,77 vs 1,42 mmol/l, $p<0,01$) levels in 23 athletes as compared to the controls. Since the apo A-II concentration did not differ between the groups, the apo A-I/apo A-II ratio was higher in the athletes ($p<0,01$). No association was reported between apo A-I and HLD-C levels in the trained group but a correlation ($p<0,001$) was observed in the control group. Nagao et al. (1984) also found a greater apo A-I level in exercising Japanese adults as compared to a control group; the concentration of apo A-I increased with the amount of training. In a similar study of 216 subjects (74 endurance trained, 87 variably trained and 55 sedentary), Kullmer and Kindermann (1985) oberved that only apo A-I had a strong relation to physical performance when adjusted for the influences of age and body weight.

Following 12 weeks of physical conditioning, the HDL-C ($p<0,002$) and apo A-I ($p<0,02$) levels of 24 sedentary non-obese men (30 to 44 years old) increased in Kiens et al. 's (1980) study; the changes in apo A-I and HDL-C levels were not related. In this experiment, the factors possibly influencing HDL-C concentrations were controlled and

analyzed: dietary and alcohol intakes, smoking and body composition. Although no relationship between the apo A-I level and the dietary intake was observed, the authors cautionned that a higher fat intake during training could have stimulated the synthesis of apo A-I in the intestine.

Pursuing this possible effect of fat intake on apo A-I levels, Kiens et al. (1981) investigated the effect of a fat-rich (54% fat) and fat-poor (29% fat) diets on 25 physical education instructors. Although the diet did not modify HDL-C concentrations and only slightly influenced apo A-I levels, the apo A-I concentrations correlated with HDL-C ($r=0,82$, $p<0,001$) throughout the study.

Therefore, in physically active persons, the increased HDL-C concentration is accompanied by a rise in the apo A-I level. Apo A-I activates LCAT which in turn promotes the formation of cholesteryl ester in HDL. Table 4 summarizes the results of the studies on the effects of exercise on lipoprotein metabolism.

2.7 PHYSIOLOGICAL ADAPTATIONS CONSEQUENT TO RESISTIVE TRAINING

In most studies on the effects of exercise on the lipid profile, the subjects were aerobically trained. Since the oxidation of free fatty acids was the energy source in the mild to moderate aerobic training, the lipolysis of muscle triglyceride explains the increased LPL activity, higher levels of HDL-C and its subfractions, and lower triglyceride, total cholesterol and LDL-C values. The increase in circulating free fatty acids levels is an important factor affecting fatty acid oxidation. Since resistive training is mostly an anaerobic exercise, the association between this physical activity and the lipid metabolism must be considered.

TABLE 4

Effects of Exercise on the Lipoprotein Metabolism

Parameter	Metabolic Activity with Exercise	Study
LPL	- increased in adipose tissue ($p<0,05$) and skeletal muscle ($p<0,01$) and correlated to distance run ($p<0,05$) and HDL-C ($p<0,001$) for adipose tissue only	Nikkila et al., 1978B
	- increased in runners ($p<0,05$) and correlated negatively to VLDL ($p<0,05$) and positively to HDL ($p<0,01$)	Krauss et al., 1979
	- (acute effect) increased in skeletal muscle by 112% ($p<0,01$) and adipose tissue by 20% ($p<0,05$)	Taskinen et al., 1980
	- increased by 70% in adipose tissue ($p<0,10$) and related to intensity of training ($p<0,05$)	Marniemi et al., 1980
	- postheparin plasma LPL activity increased ($p<0,001$)	Peltonen et al., 1981
	- muscle LPL levels increased ($p<0,01$)	Lithell et al., 1981
	- increased by 50% in adipose tissue ($p<0,05$) and correlated to HDL-C ($p<0,05$)	Stubbe et al., 1983
	- LPL did not correlate with HDL-C but HL correlated inversely with HDL-C and HDL2-C ($p<0,001$)	Hurley et al., 1984A
LCAT	- increased in 4 subjects (out of 13)	Lopez-S. et al., 1974
	- increased ($p<0,05$) in rats	Simko and Kelley, 1979
	- no correlation between HDL-C and rate of cholesterol esterification	Stokke and Enger, 1979
	- increased ($p<0,001$)	Peltonen et al., 1980
	- increased ($p<0,01$)	Marniemi et al., 1982
	- increased ($p<0,05$) and unesterified cholesterol/ cholesteryl esters increased ($p<0,05$)	Dufaux et al., 1986
Apoproteins	- Apo A-I increased ($p<0,001$)	Krauss et al., 1977
	- Apo A-I increased (NS)	Miller et al., 1979
	- Apo A-I increased ($p<0,001$)	Lehtonen et al., 1979
	- Apo A-I increased ($p<0,02$)	Kiens et al., 1980
	- Apo A-I correlated with HDL-C ($p<0,001$)	Kiens et al., 1981
	- Apo A-I increased with amount of training	Nagao et al., 1984
	- strong relation to physical performance	Kullmer and Kindermann, 1985

As reviewed by Gettman and Pollock (1981) and observed by others (MacDougall et al., 1977; Gettman et al., 1978; Wilmore et al., 1978; MacDougall et al., 1979), weight training and circuit weight training (40-60% of 1RM for 3 circuits) improve strength. Circuit weight training compared favorably with traditional weight training; its energy cost was similar to walking on a flat surface at 4 miles per hour. Weight training elicited no change in VO2max (Gettman et al., 1978; Wilmore et al., 1978) or only a slight increase in VO2max, 4,3% in average in men participating in circuit weight training (Gettman and Pollock 1981).

As for the biochemical adaptations consequent to weight training; MacDougall et al. (1977) found a significant increase in the resting concentrations of creatine (by 39%), creatine phosphate (by 22%), ATP (by 18%) and glycogen (by 66%) ($p<0,05$) in 9 men training at 8-10 reps for 3-5 sets for 5 months. During the training, the long rest periods between sets (2 minutes) probably permitted the creatine phosphate and ATP to be replenished. Therefore, weight training utilizes high-energy phosphates and glycogen reserves as energy sources. Tesch et al. (1986) also found similar results in nine bodybuilders (four subjects were taking steroids). The intense, heavy-resistance exercise session produced the breakdown of high-energy phosphates and the modest utilization of glycogen, including the accumulation of high lactate levels. To investigate the muscle oxidative capacity through weight training, MacDougall et al. (1979) then studied the mitochondrial volume density of 6 men weight-training for 6 months (same training program as in his above-mentionned study). Following the training, the mitochondrial volume density was reduced by 26% ($p<0,05$) and the fibre area was increased by 33% for FT fibres and 27% for ST fibres ($p<0,05$). Consequently, the mitochondrial volume density decreased through a greater myofibrillar size with hypertrophy but without a proportional increase in mitochondrial volume.

With weight training, strength and myofibrillar size increase as do the high energy phosphates and glycogen levels. The muscle oxidative capacity does not seem to increase, as reflected by a reduced mitochondrial volume density and minimal VO2max changes. There seems to be a general correlation between the mitochondrial content of the working muscle and the contribution of lipid oxidation in performing moderately intense prolonged exercise (review by Terjung and Kaciuba-Uscilko, 1986); therefore, lipids do not seem to participate in the metabolism of weight training.

2.8 EFFECTS OF ANAEROBIC TRAINING ON LIPOPROTEIN PROFILE

Many investigators have examined the effects of aerobic training on the lipoprotein profile, more specifically HDL for its beneficial anti-atherogenic effect. Research on anaerobic activities, such as weight training, have been scarce. The studies dealing with anaerobic training use two types of analyses: 1) comparative studies in which anaerobically trained athletes are compared to sedentary, untrained and/or aerobically trained individuals, and 2) longitudinal studies where an anaerobic training program was followed.

2.8.1 Comparative Studies

Lehtonen and Viikari (1980) examined the lipoprotein profiles of Finnish top-class football and ice-hockey players during different training programs as compared to a control group. The ice-hockey training was considered as anaerobic. The endurance-

trained football players had the highest HDL-C level while the hockey players, who weighed more, had the lowest value (64,3 vs 46,0 mg/100 ml, $p<0,001$) but HDL-C/TC ratio increased with the addition of aerobic training. Because of the negative correlation between body weight and HDL-C, the greater relative weight of ice-hockey players could account partly for the lower HDL-C levels. Ice-hockey training was associated with a less favorable lipoprotein profile than football.

Berg et al. (1980) also studied the lipoprotein profiles of athletes with different training regimens. Group A was composed of 109 athletes training in endurance, 88 men had mixed training in group B, group C had 44 athletes power training, and group D had 55 control subjects. The power trainers had the lowest HDL-C levels at 0,88 +/- 0,32 mmol/l ($p<0,001$) while the endurance athletes had the lowest VLDL-C (0,27 +/- 0,13 mmol/l, $p<0,001$) and LDL-C (2,92 +/- 0,59 mmol/l, $p<0,05$) levels. No correlation existed between the HDL-C concentration and the relative body weight. As indicated by the authors, the lipoprotein profiles of the power trainers need further research since pharmacological data on these top athletes was difficult to obtain. Therefore, the use of steroids or vitamins could have contaminated the lipoprotein profile. Clarkson et al. (1981) also found similar results where weight lifters had the highest values of TC/HDL-C ratio (3,44 vs 2,32 vs 3,37) and body weight (85,99 vs 68,94 vs 72,86 kg) as compared to runners and untrained subjects.

Farrell et al. (1982) compared sedentary men, weightlifters (anaerobic training) and speed skaters (aerobic and anaerobic training). The sedentary subjects were fatter ($p<0,05$), the weightlifters were older ($p<0,05$) and the latter group was lighter ($p<0,05$) but only two significant differences prevailed in the lipid profile: the speed skaters had the highest HDL-C levels (53,7 mg/dl, $p<0,05$) while the weightlifters had the highest TC/HDL-C ratio (4,13, $p<0,05$). It was suggested that exercise intensity is critical to

modify HDL-C, especially to stimulate LPL activity. The weightlifters' HDL-C values might have been lowered by their weight: they were fatter than the speedskaters ($p<0,05$).

Hurley et al. (1984B) also examined the type of weight training (8 bodybuilders and 8 weightlifters) and its effects on the lipid profile as compared to runners (n=8). Bodybuilders train with moderate resistance, high repetitions and short rest intervals while weightlifters and powerlifters exercise with heavy resistance, few repetitions and longer rest intervals. The bodybuilders' training provided a TC level, LDL-C level and LDL-C/HDL-C ratio comparable to the runners'. Although body builders had the highest HDL-C level (55 vs 47 mg/dl, $p<0,05$, as compared to runners) and a lower VO2max than the runners (49 vs 60 ml/kg/min), their HDL2 level was not significantly different from the runners' but was higher than the powerlifters' (12 vs 6 mg/dl, $p<0,05$). The maximal aerobic capacity did not seem to be the determining factor in the lipoprotein levels. The percent body fat was similar among the groups but a residual effect of androgens could have been present in the body builders and the powerlifters since there was abstinence from these drugs for only 10 weeks prior to the study. Therefore, moderate-resistance and high repetition weight training would seem to positively influence the lipoprotein profile. When comparing the lipid profile and LPL activity between 9 body builders, 8 weight-matched controls and 8 normal-weight controls, Yki-Jarvinen et al. (1984) found no significant difference in the HDL-C, HDL2-C and HDL3-C levels and the LPL activity in the adipose and skeletal tissues between the groups. The body builders, who had never used steroids, had less body fat (5 +/- 1 % vs 10 +/- 1 %, $p<0,005$) and the three groups had a comparable VO2max (from 52 +/- 6 to 54 +/- 3 ml/kg/min). Although the body builders did not show an increased HDL2-C concentration, their low LDL-C value (2,48 +/- 0,23 vs 3,27 +/- 0,34 mmol/l) still reflected a positive lipid profile.

Investigating the effect of rapid weight loss on the lipid profile of 15 weightlifters, 19 wrestlers, 16 boxers and 12 judokas, Jauhialnen et al. (1985) established that the weightlifters had a very high level of total cholesterol (5,07 +/- 0,74 as compared to boxers 4,56 +/- 0,80 mmol/l), the lowest HDL-C concentration (1,02 +/- 0,24 as compared to wrestlers 1,35 +/- 0,32 mmol/l), a high triglyceride value (1,42 +/- 0,46 as compared to wrestlers 1,10 +/- 0,57 mmol/l), and high levels of apo A-I (1404 +/- 130 mg/l) and apo B (712 +/- 93 mg/l). Other than the apo A-I level, the weightlifters reflected an atherogenic lipid profile.

As shown in Table 5, these comparative studies indicate a lower HDL-C level or a higher TC/HDL-C ratio for anaerobically trained athletes, except for bodybuilders (Hurley et al., 1984B; Yki-Jarvinen et al., 1984). The relative weight and previous or present drug use of these subjects may influence the lipoprotein profile. Like many comparative studies, the research design prevents a definite conclusion that the differences in the lipoprotein profile are caused solely by the intensity or type of training.

2.8.2 Longitudinal Studies

In 1980, Lopez-S. et al. investigated the effect of resistive (anaerobic) training for 12 weeks (3 days per week) on 14 men as compared to 8 sedentary control subjects and 9 joggers (mean age for all subjects = 42 years). The experimental group decreased significantly their serum TC (200 to 193 mg%) and LDL-C (169 to 122 mg%) levels and increased significantly their HDL-C (46 to 53 mg%) concentration. Their body weight did not change but their percent body fat decreased whereas the lean body mass increased.

In a similar study, Johnson et al. (1983) examined the effect of 12 weeks of resistive training on the lipid profiles and testosterone levels of previously sedentary middle-aged men (mean age 42 years). The improved physical fitness of the experimental group caused a linear ($p<0,01$) increase in the mean testosterone level; a less significant linear increase ($p<0,05$) was observed in the control group. The weight training also linearly decreased the TC (201 to 192 mg/100 ml, $p<0,05$) and LDL-C (169 to 122 mg/100 ml, $p<0,05$) levels while the increase in HDL-C concentration was linear and quadratic (46 to 53 mg/100 ml, $p<0,05$), the HDL-C level falling slightly at the 6th week. Weight data was not indicated. The authors expected to observe an increased testosterone level following a weight training program which would in turn reduce the HDL-C level. The increased levels of HDL-C and testosterone are paradoxical and indicate a need for further research on the effect of weight training on the lipoprotein profile.

Pursuing this subject, Goldberg et al. (1984) studied the effect of 16 weeks of weight-training on the lipid profiles of previously sedentary men (n=6, mean age = 33 years) and women (n=8, mean age = 27 years). The women reduced their TC by 9,5% (198 to 179 mg%, $p<0,05$), LDL-C by 17,9% (107 to 87 mg%, $p<0,01$) and triglycerides by 28,3% (70 to 50 mg%, $p<0,05$) while the HDL-C level rose insignificantly by 4,8% (77,4 to 81,1 mg%). The men reduced the TC by 6,8% (209 to 194 mg%, $p=0,07$) and LDL-C by 16,2% (142 to 119 mg%, $p<0,05$) while the triglyceride concentration was unchanged and the HDL-C level increased by 15,8% (50,6 to 58,6 mg%, $p>0,052$). Both men and women also decreased significantly ($p<0,05$) the TC/HDL-C (4,22 to 3,31 and 2,59 to 2,22 respectively) and LDL-C/HDL-C (2,84 to 2,02 and 1,38 to 1,10 respectively) ratios. The tendency toward an increased HDL-C level could be related to the reduced percent body fat and increased lean body mass. No possible metabolic mechanism for the changes in the lipoprotein profile following the weight training was indicated.

In the three longitudinal studies (refer to Table 5), the resistive training (moderate weights and 3 to 8 reps with short rest periods) produced a favorable lipid profile for previously sedentary subjects. It seems evident that body composition might influence the lipoproteins' cholesterol levels.

2.9 SUMMARY

The comparative and longitudinal studies of the effects of resistive training on lipid metabolism appears controversial. With the comparative studies (Berg et al., 1980; Lehtonen and Viikari, 1980; Clarkson et al., 1981; Farrell et al., 1982; Hurley et al., 1984B; Jauhialenen et al., 1985), a lower HDL-C level or a higher TC/HDL-C ratio prevails for anaerobically trained athletes. Many factors could have influenced the lipoprotein cholesterol level: increased body fat and use of androgens and vitamins can decrease the HDL-C content. Another inherent factor was the age of the subjects. All the subjects of the comparative studies, except for Hurley et al. 's (1984B) body builders (where the lipid profile was favorable) and for Farrell et al. 's (1982) weight lifters, were in their early 20's; the greatest slope of an increased HDL3 (Gidez et al., 1982), LDL-C and VLDL-C (Heiss et al., 1980) levels in men is found in the 20 to 30 age group.

Longitudinal studies indicate a favorable lipid profile for previously sedentary men (mean age of 33 and 42 years in the three studies) following weight training although no metabolic mechanism for these changes was revealed. The loss of body fat following such training may play an important role. It would be more revealing to follow throughout a weight training program the changes of the lipid profile in men, more specifically the HDL subfractions since HDL2 is considered as being "anti-atherogenic".

TABLE 5

Effects of Anaerobic Training on the Lipoprotein Profile

Study	Effect of Anaerobic Training on Lipids	Comments of Anaerobic Training
COMPARATIVE		
- Lehtonen and Viikari, 1980 (ice-hockey vs football and controls)	- lowest HDL-C	- players had highest relative weight
- Berg et al., 1980A (power training vs controls, endurance and mixed trainings)	- lowest HDL-C ($P<0,001$)	- no reliable pharmacological data for these athletes
- Clarkson et al., 1981 (weightlifters vs runners and untrained subjects)	- highest total cholest./- HDL-C ratio ($p<0,01$ when compared to runners)	- also highest body weights
- Farrell et al., 1982 (weightlifters vs speedskaters and sedentary men)	- highest total cholest./- HDL-C ratio ($p<0,05$)	- were fatter than speedskaters ($p<0,05$) - other groups were younger ($p<0,05$)
- Hurley et al., 1984B (bodybuilders and powerlifters vs runners and sedentary controls)	- Body builders had lipid-profile similar to runners (TC, LDL-C, and LDL-C/HDL-C) and better than powerlifters (HDL2, $p<0,05$)	- possible residual effect of androgen use
- Yki-Jarvinen et al., 1984 (body builders vs controls)	- no difference in the HDL-C HDL2-C, HDL3-C levels and the LPL activity - decreased LDL-C	- lowest body fat
- Jauhiainen et al., 1985 (weightlifters, wrestlers, boxers and judokas)	- weightlifters had a high total cholest. and the lowest HDL-C	- no information on the training regimen, body weight or androgen use.
LONGITUDINAL		
- Lopez-S. et al., 1980 (resistive training vs sedentary and joggers) - 12 weeks -	- HDL-C increased - total cholesterol and LDL-C decreased	- percent body fat decreased and lean body mass increased
- Johnson et al., 1983 (resistive training vs sedentary) - 12 weeks -	- HDL-C increased (linear and quadratic $p<0,05$) - total cholesterol and LDL-C decreased (linear $p<0,05$)	- no weight data available
- Goldberg et al., 1984 (weight training of men and women) - 16 weeks -	- Men:-total cholest. decreased ($p=0,07$) -LDL-C decreased ($p<0,05$) -HDL-C increased ($p>0,052$) -cholest./HDL-C and LDL-C/HDL-C decreased ($p<0,05$)	- reduced percent body fat and increased lean body mass

III

METHODOLOGY

3.1 INTRODUCTION

The purpose of this study was to determine the changes in the lipid profile, and more specifically the HDL subfractions, in men aged 25 to 45 years during the course of a 10-week resistive training program.

3.2 SUBJECTS

Fifteen healthy and sedentary subjects, males aged 25 to 43 years (mean age of 32,1; experimental = 33,3 years; control group = 29,0 years), participated in this study. None of the subjects had a condition preventing them from participating in the training program, as assessed by the Physical Activity Readiness Questionnaire (PAR-Q) and health questionnaire (Appendix A).

The following criteria were applied in the selection of the subjects for this study:

1) be a male, aged between 25 and 45 years;

2) have a satisfactory body weight, less than 35% body fat;

3) have a triglyceride level less than 400 mg%:

4) have a normal resting blood pressure with a diastolic pressure lower than 95 mmHg and a systolic pressure lower than 140 mmHg;

5) not participate in a physical activity on a regular basis;

6) respond negatively to all questions of the PAR-Q;

7) agree to participate in this study as a control or experimental subject.

Eleven subjects formed the experimental group and 4 subjects served as controls. All the experimental subjects were employees of the Bank of Canada and their training and testing were done at the Bank of Canada Employee Fitness Centre. The controls were tested under similar conditions at the University of Ottawa, Kinanthropology Department. Information on smoking habits, diet, alcohol intake and physical activity pattern was obtained by a lifestyle questionnaire (Appendix B). The subject gave his medical history in the health questionnaire (Appendix B). All the subjects were requested to maintain their dietary pattern and alcohol consumption and not to change their habitual level of physical activity throughout the ten-week period (other than the training for the experimental group).

In the experimental group, one subject dropped out of the study after 4 weeks due to a conflict between his holidays and the training period. This left 10 subjects in the experimental group and 4 control subjects. One subject in the experimental group was absent for the blood samples in the 6th week of training.

3.3 SEQUENCE OF EVENTS

The subjects were seen individually to have the purpose of the study explained to them and to obtain the subject's informed consent. The PAR-Q, health and lifestyle questionnaires were completed. Then the pre-study testing of anthropometric measurements, resting heart rate, resting blood pressure, strength tests and blood profile was undertaken.

The experimental group then started the supervised 10-week training program while the control subjects were only seen for the fasting blood samples. Venous blood was drawn from the subjects of both groups in the 6th week of the program. After the 10-week program, the initial testing protocol was readministered as a post-test.

The following schedule details the sequence of events.

3.3.1 Pre-Training Phase

The subjects who volunteered to participate in this study were met individually to be informed of the purpose of the study. If still interested in the study, the subjects completed the PAR-Q, health and lifestyle questionnaires and were scheduled for a pre-study test. In this session, anthropometric measurements, resting heart rate and blood pressure, muscular endurance tests, and a blood sample were taken to ensure that the subjects met the criteria outlined above (refer to testing procedures for the specific protocol). Within the next two days, a second blood sample was taken to determine an averaged pre-study lipoprotein profile. After analysis of the blood samples, the subjects not meeting the selection criteria were screened out from the study. Arrangements for

weekly training times were made with the experimental subjects meeting the selection criteria.

3.3.2 Training Study Phase

After the pre-study testing was completed for all subjects, the experimental group started their training sessions (the training program is described further in the training section). In the first week, each exercise was performed at 60% of 1RM, increasing to 65% of 1RM in the second week, 70% of 1RM in the third week, and 75% of 1RM in the fourth week. All the subjects had venous blood drawn twice within three days in the sixth week.

3.3.3 Post-Training Phase

In the 11th week of the study, all the subjects were retested in a post-study test identical to the pre-study test. The lifestyle questionnaire was readministered to verify the absence of changes in exercise pattern, food and alcohol consumptions and smoking habits.

3.4 TESTING PROCEDURES

3.4.1 Anthropometric Measurements

The height, weight, girths (chest, waist and gluteal), and skinfolds (triceps, biceps, subscapular and supra-iliac) were measured according to the Canadian Standardized Test of Fitness (Fitness and Amateur Sport, 1983). Using the skinfold measurements, the body density was calculated using the linear regression equations of Durnin and Womersley (1974) and the percentage of body fat was derived according to Siri 's (1956) equation: ((4,95/body density) - 4,50) X 100.

3.4.2 Heart Rate and Blood Pressure

After five minutes of rest in the sitting position, the apical heart rate was taken. Then, the blood pressure was measured with an anaeroid manometer.

3.4.3 Muscular Endurance Test

The maximal number of repetitions of three weight training exercises (seated overhead press, leg press and bench press) performed at 70% of 1RM served as a muscular endurance test. The sum of the number of repetitions of the three exercises gave a score referred to as the muscular endurance index.

The control group performed these exercises on the Universal system (Universal Equipment Inc., Cedar Rapids, Iowa) where the weight is expressed in pounds while the experimental group used the Global system (Global Gym and Fitness Equipment Limited, Weston, Ontario) to do the same exercises as expressed in kilograms.

3.5 BLOOD SAMPLING

On the assigned blood sampling day, the subjects came to the laboratory between 7:30 and 9:00 a.m. The subjects were asked if they had fasted for 12 hours; if not, an alternate day was scheduled. Blood was drawn from an antecubital vein into 3 Vacutainer tubes containing 1,0-1,4 mg of dry EDTA per ml of blood for the analysis of the triglyceride, TC, HDL-C, HDL2-C, HDL3-C, LDL-C, and VLDL-C levels.

The blood was centrifuged for 10 minutes and the plasma was decanted, pipeted into glass tubes, and frozen for later analysis.

For each subject, sample 0 was the averaged value of the two venous blood samples drawn at the pre-study test. Sample 1 corresponded to the averaged value of the two blood samples taken in week 6. Sample 2 was the averaged value of the two post-study blood samples.

3.6 BLOOD ANALYSIS

The frozen fasting plasma was analyzed within 3 weeks of the blood sampling day. Frozen samples stored from 3 to 18 weeks have shown no changes in the estimated values for HDL-C, TC and triglyceride (Stokes et al., 1986). The triglyceride, the TC, HDL-C, HDL2-C, HDL3-C, LDL-C and VLDL-C levels were assessed from the plasma drawn in the Vacutainers containing 1,0-1,4 mg of dry EDTA per ml of blood.

To verify the consistency of the techniques, aliquots of pooled plasma were analyzed for triglycerides, TC, HDL-C, HDL2-C, HDL3-C, LDL-C and VLDL-C. Then, for each assay, an aliquot of this pooled plasma was used and reported in conjunction with the results.

3.6.1 Triglyceride Analysis

The triglyceride concentration was analyzed by an enzymatic triglyceride assay after saponification with ethanolic potassium hydroxide using the Test-Combination Triglycerides (Neutral Fat) kit by Boehringer Mannheim. The ethanolic potassium hydroxide transformed the triglycerides in the EDTA plasma sample into glycerol and fatty acids. With the addition of ATP, glycerol-3-phosphate and ADP were formed through the action of glycerol kinase (GK). Phosphokinase changed the resulting ADP and phospho-enol-pyruvate into pyruvate and ATP. Then lactate and NAD+ were formed from the pyruvate, NADH, H+ and lactate deshydrogenase.

The intensity of colour of glycerol (the series of reactions without the GK) was quantitated by photometry at 365 nm (Absorbance 1). Then, photometry was used a second time with the addition of GK (Absorbance 2). The triglyceride concentration was calculated as the result of Absorbance 1 - Absorbance 2 - Absorbance of the reagent blank.

In each assay, an aliquot of the pooled plasma was analyzed and the concentrations were reported in conjunction with the triglyceride results.

3.6.2 Total Cholesterol (TC) Analysis

The TC concentrations were determined by an enzymatic colorimetric method using the Cholesterol C-system, CHOD-PAP method from Boehringer Mannheim. The cholesterol ester combined with water was split by cholesterol esterase into free cholesterol to be oxidized by cholesterol oxidase into cholest-4-en-3-one and peroxidase. The POD transformed this peroxidase in the presence of 4-aminophenazone and phenol into a p-quinone imine dye. The intensity of the colour was proportional to the concentration of TC and could be measured quantitively by photometry at 500 nm. The calibration curve was established with six standard TC concentrations (50, 100, 150, 200, 300 and 400 mg%) of the Preciset Cholesterol Kit by Boehringer Mannheim. For each assay, two standards were analysed and reported in conjunction with the TC results.

3.6.3 HDL-C, HDL2-C and HDL3-C Analyses

Gidez et al. (1982) used a simple precipitation method to analyse HDL-C level and developed a second precipitation procedure to further assess the HDL3-C concentration. The HDL2-C level is determined as the difference between HDL-C and HDL3-C concentrations.

In the HDL-C analysis, the apo B-containing lipoproteins (LDL and VLDL) were precipitated with heparin-manganese to form an HDL-C supernatant. The precipitation reaction still unclear, it might be caused by an interaction between the negatively charged heparin with the protein moieties of the lipoproteins. VLDL and LDL form insoluble complexes that can be sedimented at low speed centrifugation. This HDL-C supernatant was then measured photometrically for its cholesterol content as described in the TC analysis. As control for accuracy in the HDL-C assays, a Special Control Serum Kit (a lyophilized control serum based on human serum) from Boehringer Manheim was used for each assay.

From this HDL-C supernatant, HDL2-C was precipitated by dextran sulfate (mol. wt. 15 000) to form a HDL3-C supernatant which was removed after centrifugation and analyzed for its cholesterol level as described in the TC analysis.

The difference between HDL-C (heparin-manganese supernatant) and HDL3-C (dextran sulfate supernatant) represented the HDL2-C level. This double precipitation method to determine HDL2-C correlated well in Gidez et al. 's (1982) study as compared to preparative ultracentrifugation (n=295, r=0,91) and analytical ultracentrifugation (n=17, r=0,92).

In each assay, an aliquot of the pooled plasma was analyzed and the concentrations were reported in conjunction with the results.

3.6.4 LDL-C and VLDL-C Analyses

The LDL-C and VLDL-C concentrations were estimated as described by Friedewald et al. (1972). The VLDL-C level was calculated as being equal to the total triglyceride concentration divided by 5. This estimation of VLDL-C may cause large percentage errors but the ensuing LDL-C calculation reduces percentage errors to an acceptable level.

The LDL-C concentration is calculated as follows: LDL-C = TC - VLDL-C - HDL-C. This LDL-C estimation assumes that the chylomicrons are absent since the subject has fasted and that the triglyceride concentration does not exceed 400 mg/100ml. As compared to ultracentrifugation with samples from different populations, this LDL-C estimation method yielded correlation coefficients ranging from 0,94 to 0,99 (Friedewald et al., 1972).

In each assay, an aliquot of the pooled plasma was analyzed and the concentrations were reported in conjunction with the LDL-C and VLDL-C results.

3.7 TRAINING PROGRAM

The weight training was performed 3 times per week on the Global system. One day of rest was scheduled between training sessions. The initial session determined the 1RM (single repetition maximum) lifted for the following exercises: seated overhead press (shoulders), bench press (chest and triceps), leg curl (hamstrings), leg press (quadriceps and gluteal), lat machine pulldown (back), seated shoulder row (biceps), and leg extension (quadriceps).

For the first week of training, the subjects performed 3 sets of 10 reps of each exercise at 60% of the 1RM. The intensity of 1RM was increased to 65% for the 2nd week, 70% for the 3rd week and 75% for the fourth week. Then, whenever the subject accomplished 2 sets of 10 reps in any exercise, the weight was increased in increments of 5,0 or 7,5 kg, according to the weights available on the machine.

The warm-up consisted of flexibility exercises, curl-ups, and push-ups. During training, the exercises were performed with rest intervals of 1 minute between exercises and 30 seconds of rest between sets. A 5-minute session of flexibility exercises (flexion/extension of the differents joints, and trunk rotation and lateral flexion) served as a cool-down.

3.8 STATISTICAL ANALYSES

All variables were analyzed by a T-test and tests of homogeneity to verify the presence of significant differences between the experimental and control groups at the pre-

training level. To detect any significant difference in the results, an analysis of variance (ANOVA) with repeated measures was done for all variables in the experimental and control groups. The dependent variables (triglycerides, TC, HDL-C, HDL2-C, HDL3-C, LDL-C, VLDL-C, TC/HDL-C, and HDL2/HDL) included 3 (pre-training, week 6, and post-training) repeated measures while the remaining dependent variables (percentage of fat and muscular endurance index) had 2 repeated measures (pre- and post-trainings). The resistive training was the independent variable.

The data was analyzed with the Bio-Medical Data Processing (BMDP) Statistical package. A probability level of $p<0,05$ was considered as a significant difference. From the two-way ANOVA, three F ratios with a corresponding probability level were calculated. The F ratio entitled "groups" represented the degree of difference between the experimental and control groups' means throughout the study. The "weeks" F ratio revealed the degree of difference between the total mean of both groups combined at the three different intervals (pre, 6th week and post). The "G*W" (weeks * groups) F ratio expressed the interaction of both main effects combined. The interaction effect was the joint effect of the groups and weeks on the dependent variable that could not be accounted for by the main effects alone. When the F ratio was significant, the difference between the means was greater than could be expected by chance.

When the interaction (G*W) F ratio was significant, simple main effects were calculated. In the presence of significant differences ($p<0,05$), the Tukey's technique served as the post-hoc analytical procedure to locate the differences between the means.

When both main effects and the interaction produced a significant F ratio, a related measure of the relationship between the independent and dependent variables, known as omega-squared, was calculated. The omega-squared estimated the proportion of the total variance of the dependent variable that could be predicted in the population from the independent variable.

IV

RESULTS

4.1 INTRODUCTION

This study was initiated to assess the effects of resistive training on the lipoprotein profile of men. In addition to the lipoprotein profile (triglycerides, TC, HDL-C, HDL2-C, HDL3-C, LDL-C, VLDL-C, TC/HDL-C, and HDL2/HDL), the percentage of body fat and the muscular endurance index were measured. This chapter presents the results and statistical analysis of the study.

4.2 RESULTS

All the results were analyzed by a two-way ANOVA with repeated measures. First, the results of the dependent variables with two repeated measures (percentage of body fat and muscular endurance index) were analyzed, followed by the dependent variables with three repeated measures (the lipoprotein profile).

The independent T-tests and tests of homogeneity revealed no significant difference between the training group and the control subjects at the pre-training level for all

the dependent variables. Therefore, the ANOVA was the proper statistical analysis since both groups were homogeneous at the baseline level.

By the responses to the lifestyle questionnaires (appendix B), it appeared that the smoking habits, diet and alcohol intake did not vary during the study. The experimental group modified their physical activity pattern solely through the added resistive training program.

Appendix C contains the raw scores for all variables and appendix D indicates the coefficient of variation for the pooled plasma.

4.2.1 Dependent Variables with Two Repeated Measures

The percentage of body fat and muscular endurance index were measured at the pre- and post-training levels. Table 6 contains the means and standard deviations for both these variables.

TABLE 6

Statistics for Body Fat and Muscular Endurance Index

		Body Fat (%)		Muscular Endurance Index	
		Experimental Group	Control Group	Experimental Group	Control Group
Mean	Pre	28,23	26,55	56,29	51,00
	Post	28,26	26,50	104,29	54,25
Marginal		28,24	26,53	80,29	52,63
Standard Deviation	Pre	4,11	2,18	13,98	10,98
	Post	3,17	3,01	24,45	12,45

Percent Body Fat

In the results of the two-way ANOVA (table 7), the two main effects (groups and weeks) and their interaction (G*W) were non significant at $p<0,05$. Therefore, the percentage of body fat did not vary between and within the groups throughout the study.

TABLE 7

Results of the Two-Way ANOVA for Percent Body Fat

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	16,905	1	16,905	0,79	0,3918
Error	257,019	12	21,418		
Weeks	0,001	1	0,001	0,00	0,9876
G*W	0,009	1	0,009	0,00	0,9503
Error	27,136	12	2,261		

Muscular Endurance Index

As indicated in table 8, the groups main effect was significant for the muscular endurance index at a level of probability of 0,0165. At the completion of the study, the experimental group had a higher muscular endurance index (80,29 vs 52,63), as observed in table 6.

TABLE 8

Results of Two-Way ANOVA for Muscular Endurance Index

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability	Omega Squared
Groups	3895,13	1	3895,13	8,64*	0,0165	21,76%
Error	4058,23	9	450,91			
Weeks	3342,90	1	3342,90	19,67*	0,0016	20,41%
G*W	2548,72	1	2548,72	15,00*	0,0038	15,30%
Error	1529,38	9	169,93			

* significant at the respective level

The weeks main effect was also significant (p=0,0016) but the main effects had to be calculated since their interaction (G*W) was significant (p=0,0038). When the simple main effects were calculated, a significant F ratio was revealed between the groups at the post-study interval and between the two intervals (pre- and post-study) for the experimental group. Therefore, the experimental group at the post-study interval had a higher muscular endurance index (104,29) as compared to the pre-level (56,29) and to the control group at the post-level (54,25).

From the two main effects and their interaction, the strength association (omega squared) was estimated. The groups main effect reflected 21,76% of the variance in the muscular endurance index, the weeks main effect accounted for 20,41% of this variance and their interaction (G*W) indicated 15,30% of this variance.

The results of two-way ANOVA of these dependent variables indicate that the resistive training did not modify the body composition (percent body fat) but increased the muscular endurance (index).

4.2.2 Dependent Variables with Three Repeated Measures

The lipid profile (triglycerides, TC, LDL-C, VLDL-C, HDL-C, HDL2-C, HDL3-C, HDL2-C/HDL-C and TC/HDL-C) was analyzed by a two-way ANOVA with three repeated measures (pre, week 6 and post). The means and standard deviations of the lipid profile are found in table 9.

Triglycerides

As shown in table 10, the two main effects (groups and weeks) and their interaction (G*W) were non significant at $p<0,05$. Throughout the study, the triglyceride levels did not vary between and within the groups.

TABLE 9

Means and Standard Deviations for the Lipoproteins

Lipids Lipids		Mean PRE	Mean W6	Mean POST	Marginal Mean	Standard Deviation PRE	Standard Deviation W6	Standard Deviation POST
Triglyceride mg %	E*	121,18	106,11	131,71	119,67	61,46	26,05	36,12
	C*	107,90	108,23	104,40	106,85	26,50	28,03	43,57
TC mg %	E*	219,01	225,13	262,97	235,70	44,59	27,57	23,26
	C*	249,01	228,99	214,51	230,84	63,18	34,44	45,75
LDL-C mg %	E*	155,02	162,59	192,65	170,09	45,19	28,29	22,29
	C*	186,47	168,43	151,44	168,78	62,05	35,28	43,20
VLDL-C mg %	E*	24,24	21,22	26,34	23,93	12,29	5,21	7,22
	C*	21,58	21,65	20,88	21,37	5,30	5,60	8,71
HDL-C mg %	E*	39,76	41,32	43,97	41,68	6,74	5,43	6,79
	C*	40,96	38,91	42,19	40,69	3,77	7,55	5,64
HDL2-C mg %	E*	10,46	14,02	13,64	12,71	3,03	3,37	3,94
	C*	10,86	10,29	6,96	9,37	2,80	4,00	2,26
HDL3-C mg %	E*	29,36	27,43	30,89	29,23	4,51	3,59	3,44
	C*	29,65	28,62	35,36	31,21	3,75	4,12	3,88
HDL2-C/HDL-C	E*	0,26127	0,33721	0,30621	0,30156	0,04413	0,04975	0,05496
	C*	0,26492	0,25912	0,16275	0,22893	0,06094	0,05274	0,03309
TC/HDL-C	E*	5,627	5,512	6,092	5,744	1,291	0,791	0,966
	C*	6,220	6,110	5,230	5,853	2,150	1,697	1,660

* E-= Experimental group
C = Control group

TABLE 10

Results of the Two-Way ANOVA for Triglyceride Levels

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	1365,57	1	1365,57	0,37	0,5575
Error	41057,10	11	3732,46		
Weeks	683,93	2	341,96	0,50	0,6160
G*W	1200,11	2	600,06	0,87	0,4332
Error	15189,23	22	690,42		

Total Cholesterol (TC)

The results of the two-way ANOVA in table 11 showed no significant difference for the two main effects (groups and weeks) but their interaction (G*W) indicated a significant F ratio at a level of probability of 0,0003. This interaction indicated that the effect of one factor was not the same for all levels of the other factor. Since the TC concentration increased in the experimental group and decreased in the control group throughout the study, an interaction (G*W) was found.

TABLE 11

Results of the Two-Way ANOVA for TC Concentrations

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	196,60	1	196,60	0,05	0,8208
Error	40184,84	11	3653,17		
Weeks	764,33	2	382,17	1,06	0,3639
G*W	8839,33	2	4419,67	12,24*	0,0003
Error	7940,76	22	360,94		

* significant at the respective level

Low Density Lipoprotein - Cholesterol (LDL-C)

The two main effects, groups and weeks, showed no significance at $p<0,05$ but their interaction revealed a significant F ratio at $p=0,0006$ (table 12). As observed for the TC variable, the interaction was created when the LDL-C concentration rose in the experimental group and fell in the control group from pre- to post-training sessions.

TABLE 12

Results of the Two-Way ANOVA for LDL-C Concentrations

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	14,18	1	14,18	0,00	0,9512
Error	39826,59	11	3620,60		
Weeks	265,08	2	132,54	0,38	0,6914
G*W	7523,68	2	3761,84	10,65*	0,0006
Error	7768,56	22	353,12		

* significant at the respective level

Very Low Density Lipoprotein - Cholesterol (VLDL-C)

From the ANOVA results in table 13, no significant F ratio was revealed for the two main effects nor their interaction at $p<0,05$. Throughout the study, the VLDL-C levels did not change between and within the groups.

TABLE 13

Results of the Two-Way ANOVA for VLDL-C Concentrations

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	54,62	1	54,62	0,37	0,5575
Error	1642,25	11	149,30		
Weeks	27,32	2	13,66	0,49	0,6164
G*W	48,01	2	24,01	0,87	0,4331
Error	607,42	22	27,61		

Total High Density Lipoprotein - Cholesterol (HDL-C)

From the two-way ANOVA analysis in table 14, the groups and weeks main effects and their interaction produced no significant F ratio at $p<0,05$. Therefore, the HDL-C concentrations were not modified between the groups throughout the study.

TABLE 14

Results of the Two-Way ANOVA for HDL-C Concentrations

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	8,21	1	8,21	0,08	0,7768
Error	1069,53	11	97,23		
Weeks	59,96	2	29,98	3,18	0,0614
G*W	20,63	2	10,32	1,09	0,3527
Error	207,63	22	9,44		

High Density Lipoprotein 2 - Cholesterol (HDL2-C)

From the ANOVA results in table 15, the groups and weeks main effects revealed no significant difference but their interaction (G*W) indicated a significant F ratio at a probability level of 0,0005. During the course of the study, the HDL2-C level of the control group decreased. In the experimental group, the pre-HDL2-C value was slightly lower than the control's but then increased at week 6 to fall slightly at the post-study interval. Therefore, an interaction between the experimental and control groups was produced at the pre-study interval.

TABLE 15

Results of the Two-Way ANOVA for HDL2-C Concentrations

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	92,59	1	92,59	3,34	0,0949
Error	305,05	11	27,73		
Weeks	21,37	2	10,68	3,36	0,0532
G*W	69,85	2	34,93	10,99*	0,0005
Error	69,88	22	3,18		

* significant at the respective level

High Density Lipoprotein 3 - Cholesterol (HDL3-C)

As indicated in the results of the ANOVA in table 16, the groups main effect and the G*W interaction had a non-significant F ratio at $p<0,05$ but the weeks main effects (repeated measures) had a significant F ratio at p=0,0001.

TABLE 16

Results of the Two-Way ANOVA for HDL3-C Concentrations

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	32,74	1	32,74	0,92	0,3570
Error	389,52	11	35,41		
Weeks	152,40	2	76,20	15,32*	0,0001
G*W	26,97	2	13,48	2,71	0,0887
Error	109,45	22	4,98		

* significant at the respective level

Tukey's post-hoc technique was used following the ANOVA to locate at which interval the combined mean of both groups differed significantly. The post-study HDL3-C concentration of both groups (32,26 mg%) was significantly greater than the pre-study level (29,45 mg%, $p<0,05$) and the 6th week of study level (27,80 mg%, $p<0,01$) (table 17). Therefore, both groups (combined mean) at the post-study interval differed from the pre-study and 6th week of study intervals.

TABLE 17

Tukey's Results on the Combined Group's HDL3-C Means

WEEKS		PRE	6th WEEK	POST
MEANS (mg%)		29,45	27,80	32,26
PRE	29,45	-----	1,65	2,81*
6th WEEK	27,80		-----	4,46**
POST	32,26			-----

* $p<0,05$
** $p<0,01$

High Density Lipoprotein 2 - Cholesterol to Total High Density Lipoprotein - Cholesterol Ratio (HDL2-C/HDL-C)

As shown in table 18, the two main effects and their interaction had a significant F ratio. Since the groups effect was significant at p=0,0117, the experimental group had a higher HDL2-C/HDL-C ratio than the control group (0,30156 vs 0,22893), as indicated in table 9.

The weeks main effect (repeated measures) was also significant at a probability level of 0,0019 but the main effects had to be computed since the interaction (G*W) also produced a significant F ratio at p=0,0004.

TABLE 18

Results of the Two-Way ANOVA for HDL2-C/HDL-C Ratio

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability	Omega Squared
Groups	0,044	1	0,044	9,10*	0,0117	21,24%
Error	0,053	11	0,005			
Weeks	0,023	2	0,011	8,45*	0,0019	11,04%
G*W	0,030	2	0,015	11,28*	0,0004	20,18%
Error	0,029	22	0,001			

* significant at the respective level

The calculation of the simple main effects produced 4 significant F ratios: between the groups 1) at the 6th week of study (p=0,0261) and 2) at the post-study level (p=0,0006), and between the three intervals for 3) the experimental group (p=0,0009) and 4) the control group (p=0,0009). Therefore, the HDL2-C/HDL-C ratio of the experimental group was greater than the control group's at the 6th week of study (0,33721 vs 0,25912) and at the post-study level (0,30621 vs 0,16275). Furthermore, both groups individually revealed significant F ratios between the three intervals. The results from Tukey's post-hoc technique (table 19) for the experimental group indicated significant differences at the pre-study interval (0,26127) which varied from the 6th week of study (0,33721, $p<0,01$) and post-study (0,30621, $p<0,05$) ratios.

TABLE 19

Tukey's Results on the Exp. Group's HDL2-C/HDL-C Ratio

WEEKS	MEANS	PRE 0,26127	6th WEEK 0,33721	POST 0,30621
PRE	0,26127	-------	0,07594**	0,04494*
6th WEEK	0,33721		---------	0,03100
POST	0,30621			-------

* p<0,05; ** p<0,01

As for the control group, the HDL2-C/HDL-C ratio at the post-study interval (0,16275) differed significantly from the pre-study (0,26492, p<0,01) and the 6th week of study (0,25912, p<0,01) levels (table 20).

TABLE 20

Tukey's Results of Control Group's HDL2-C/HDL-C Ratio

WEEKS	MEANS	PRE 0,26492	6th WEEK 0,25912	POST 0,16275
PRE	0,26492	-------	0,00580	0,10217*
6th WEEK	0,25912		-------	0,09637*
POST	0,16275			--------

* p<0,01

The estimation of the strength association is represented by omega squared. The groups effect accounted for 21,24% of the variance in the HDL2-C/HDL-C ratio, the weeks effect produced 11,04% of this variance while their interaction (G*W) explained 20,18% of this variance.

Total Cholesterol to High Density Lipoprotein - Cholesterol Ratio (TC/HDL-C)

In table 21 of the ANOVA results, the two main effects revealed a non-significant F ratio but their interaction effect (G*W) was significant at a probability level of 0,0117.

TABLE 21

Results of the Two-Way ANOVA for TC/HDL-C Ratio

Source	Sum of Squares	Degrees Freedom	Mean Square	F Ratio	Level of Probability
Groups	0,10	1	0,10	0,02	0,8834
Error	48,72	11	4,43		
Weeks	0,38	2	0,19	0,54	0,5923
G*W	3,92	2	1,96	5,49*	0,0117
Error	7,86	22	0,36		

* significant at the respective level

The TC/HDL-C ratio of the experimental group fell slightly from the pre-study to week 6 intervals but then increased sharply to the post-study session. The control's ratio followed a similar trend from pre-study to week 6 but their ratio dropped at the post-study interval. An interaction was produced between week 6 and post-study intervals.

4.3 SUMMARY

Table 22 summarizes the two-way ANOVA results for all the variables. From the two dependent variables with two repeated measures, only the muscular endurance varied: the experimental subjects increased significantly their muscular endurance index at the completion of the study. As for the lipid profile measured at the three different intervals (pre, week 6 and post), the two-way ANOVA provided certain significant ($p<0,05$) results. The only lipoprotein variable to show significant F ratios for both main effects and their interaction was the HDL2-C/HDL-C ratio; the experimental group's ratio was greater than the control group's at week 6 and at the completion of the study. The HDL3-C levels reflected a significant F ratio for only the repeated measures (weeks) where both groups at the completion of the study differed significantly from the pre-training and week 6 intervals. The triglyceride, VLDL-C and HDL-C concentrations produced non-significant F ratios for the main effects and their interaction. Only the interaction effect (G*W) was significant for the TC, LDL-C, HDL2-C levels and TC/HDL-C ratio.

TABLE 22

Summary of the Statistical Analyses

VARIABLES	ANOVA (Probability Level) Groups	Weeks	G*W	SIMPLE MAIN EFFECTS Groups	Weeks	TUKEY'S TECHNIQUE Sign. Diff.
Percent Body Fat (Table 7)	NS	NS	NS	-	-	-
Muscular Endurance Index (Table 8)	0,0165	0,0016	0,0038	post	Exp.	-
Triglyceride (Table 10)	NS	NS	NS	-	-	-
TC (Table 11)	NS	NS	0,0003	-	-	-
LDL-C (Table 12)	NS	NS	0,0006	-	-	-
VLDL-C (Table 13)	NS	NS	NS	-	-	-
HDL-C (Table 14)	NS	NS	NS	-	-	-
HDL2-C (Table 15)	NS	NS	0,0005	-	-	-
HDL3-C (Tables 16 & 17)	NS	0,0001	NS	-	-	Combined: Pre vs Post* Week 6 vs Post*
HDL2-C/HDL-C (Tables 18 to 20)	0,0117	0,0019	0,0004	week 6 & post	Exp. & Control	Exp.: Pre vs Week Pre vs Post* Control: Pre vs Post* Week 6 vs Post*
TC/HDL-C (Table 21)	NS	NS	0,0117	-	-	-

Note: NS = Not Significant
* = p 0,05
** = p 0,01

V

DISCUSSION

5.1 INTRODUCTION

This study assessed the effects of 10 weeks of resistive training on the lipoprotein profile of fourteen men aged 25 to 45 years. The four control subjects were used in order to reflect the normal seasonal variations and lifestyle changes that may occur in the lipoprotein profile. At baseline level, both groups were homogenous for all variables studied. By the responses to the lifestyle questionnaires, it appeared that the smoking habits, diet and alcohol intake did not vary during the study.

5.2 BODY COMPOSITION AND MUSCULAR ENDURANCE

The resistive training did not alter the body composition, as reflected by the percentage of body fat. It was not expected that this 10 week resistive training study would modify this variable. Although two longitudinal studies on 12 to 16 weeks of resistive training (Lopez et al., 1980 and Goldberg et al., 1984) produced a decreased percent body fat, a 10-week program (Wilmore et al., 1978) and a 20-week program (Gettman et al., 1978) of circuit weight training did not change the relative body fat of men.

As was expected from resistive training, a significantly higher muscular endurance in the experimental subjects was observed. The muscular endurance index increased by 85,3% from 56,29 to 104,29 in the experimental group. In a similar study, Goldberg et al. (1984) reported a 147% increase in the performance of men following 16 weeks of weight training.

5.3 LIPOPROTEIN PROFILE

This study was initiated to determine the effects of resistive training on cholesterol transport metabolism. Only three longitudinal studies (Lopez et al., 1980; Johnson et al., 1983; and Goldberg et al., 1984) had quantified the lipid levels pre- and post-training of a resistive training program. Although, total cholesterol (TC), triglycerides, LDL-C and HDL-C were measured, none of these authors studied the HDL-C subfractions.

In this study, all the lipoprotein values, except for one TC concentration, were within the normal range for the different age groups as observed by Gidez et al. (1982). The TC post-study level for the experimental group (262,97 +/- 23,26 mg%) was slightly higher than the Gidez et al. 's (1982) value of 181 to 196 +/- 33 mg%.

The non-significant changes in the triglyceride concentrations observed here reflect the findings of Goldberg et al. (1984) who reported no triglyceride change following 16 weeks of training. Lopez et al. (1980) and Johnson et al. (1983) did not assess the triglyceride parameter in their studies. The triglyceride values may not have varied because fatty acids were not the main energy source for this anaerobic activity, as reflected by the unchanged percent body fat throughout the study. Possibly, a critical

exercise intensity threshold was not attained during training to induce adipose-tissue lipolysis.

The total cholesterol variable produced only a significant interaction; the experimental's level increased while the control's concentration decreased throughout the study. The non-significant elevated total cholesterol in the experimental group cannot be fully explained. This higher post-study value could result from experimental errors or a rise in the LDL-C level which is a by-product of the LPL activity. The LDL-C concentration also showed a similar trend: a significant interaction was produced when the LDL-C level rose in the experimental group but fell in the control group from the pre- to post-study intervals.

The relation between the total cholesterol and LDL-C concentrations was expected since the LDL-C value is estimated from Friedewald et al. 's (1972) equation which formulates that LDL-C = TC - VLDL-C - HDL-C. Consequently, the non-significant rise in total cholesterol level accompanied by the unchanged HDL-C values determined much of the LDL-C value. Although the three longitudinal resistive training studies also denoted a similar association between total cholesterol and LDL-C, all of these studies reported a decreased total cholesterol and LDL-C concentration following the training. As one of the influences on the lipoprotein levels, the combination of exercise and weight loss produces the greatest reduction in TC and LDL-C levels (Tran and Weltman, 1985). The reduced body fat in conjunction with the weight training noted in Lopez et al. 's (1980) and Goldberg et al. 's (1984) studies could explain the decreased TC and LDL-C concentrations; the third longitudinal study (Johnson et al., 1983) did not report any weight data. Since this present study did not observe any variation in body composition, it could explain the absence of the reduction of these two lipoprotein levels. A non-significant increase in the TC and LDL-C concentrations could have resulted from

a) experimental measurement errors; b) a modified physical activity pattern for the study period which could have reduced a certain amount of aerobic exercise; or c) a combination of the two above-mentionned factors. Even though the subjects were sedentary previous to the study, the experimental subjects could have been more conscious of their physical activity pattern and inadvertently limited their aerobic efforts to comply to the requisites of this study. Some subjects reported working for a short period of time on home projects where the energy requirements could not be verified statistically.

Since the VLDL-C value is expressed as a fifth of the triglyceride level and the triglyceride concentrations did not produce significant differences, it is not surprising that the VLDL-C levels did not significantly vary during the study.

In this study, much importance was put on the HDL-C, and more specifically its subfractions, because none of the previous studies assessed the HDL2-C and HDL3-C concentrations. The total HDL-C level did not significantly vary in this study. Goldberg et al. (1980) and Johnson et al. (1983) reported elevated HDL-C values after training. The HDL-C concentration in this study may not have varied because a) the exercise stimulus was not appropriate to such a change; b) the percentage of body fat remained the same throughout the study; c) the total triglycerides was not significantly reduced; and d) its subfractions varied by interconversion without a noticeable change in the total HDL-C. The exercise intensity may not have been adequate to modify the HDL-C level but its subfractions may show such changes by interconversion between HDL2-C and HDL3-C. The body composition remained unchanged and HDL-C has been inversely related to weight loss (Rhoads et al., 1976; Glueck et al., 1980; Williams et al., 1983; Sopko et al., 1985). Since HDL may be formed from the surface components of the lypolysis of triglyceride-rich lipoprotein, a constant triglyceride level throughout the study could result in an unchanged HDL-C concentration.

As for the HDL-C subfractions, the HDL2-C did not produce significant differences in the main effects but the interaction was found significant between the experimental and control groups at the pre-study interval. An experimental error could reflect the slight decline in HDL2-C concentration from week 6 to the post-study session in the control group. Since the number of subjects in the control group was restricted, an experimental error concerning one subject can easily affect the group's mean.

The HDL3-C concentration of both groups (combined mean) at the post-study interval (32,26 mg%) differed significantly from the baseline value (29,45 mg%, $p<0,05$) and the 6th week of study session (27,80 mg%, $p<0,01$).

The non-significant increased HDL2-C in the experimental group and significantly higher HDL3-C concentrations in the combined groups could result from a) an increased enzymatic activity, namely LPL and LCAT; b) an increased level of apo A-I; c) a reflection of a greater total cholesterol level; d) an experimental error; or e) any combination of the above-mentionned factors. As summarized in table 4, physical activity may enhance a greater LPL and LCAT enzymatic reaction and/or increase the apo A-I levels which activate LCAT. Since the total cholesterol concentration did increase (not significantly) in the training group , the HDL pool may have been enriched. More cholesterol in the bloodstream required a greater enzymatic activity which would then be reflected by an elevated HDL2-C and HDL3-C concentrations in the experimental subjects. This concept would also produce more LDL-C due to an increase cholesterol catabolism. Since HDL3-C levels increased in the combined group and the HDL2-C did not vary, it would be expected that total HDL-C value would also rise. Because HDL-C levels remained unchanged throughout this study, experimental errors may come into play.

Since the concentration of HDL2 shows greater variability than HDL3 (Gotto and Rifkind, 1982), HDL2 is considered a better predictor of CHD than HDL3. However, it is the increased total HDL (Miller and Miller, 1975; Rhoads et al., 1976; Castelli et al., 1977; Gordon et al., 1977; Goldbourt et al., 1985), as reflected by HDL2 and HDL3, that is correlated to a reduced risk of CHD. Therefore, high HDL3-C levels may still reflect an efficient lipid metabolism.

These results of the HDL-C and its subfractions show a similar trend to Nye et al. 's (1981) study where the total HDL-C level was not modified but the HDL2-C and HDL3-C concentrations changed significantly; in this study, the HDL2-C did not vary. The HDL subfractions seem to be very relevant lipoprotein parameters to study. Therefore, the HDL2-C/HDL-C ratio would be an important indicator of the percentage of cholesterol that is transported by the beneficial lipoprotein.

Since the HDL2-C increased (non-significantly) following training and the HDL-C remained unchanged, the HDL2-C/HDL-C ratio produced significant differences. The experimental group had a higher ratio at week 6 and at the end of the study than the control group. Therefore, the ratio increased before the 6th week of training. The post-hoc analysis verified this variation early in training for the experimental group when the pre-study ratio produced significant differences from week 6 ($p<0,01$) and the post-study interval ($p<0,05$). Therefore, more cholesterol was transported by the HDL2 lipoprotein at the end of the training than at baseline level. Unexpectedly, the ratio was found also significant ($p<0,01$) in the control group: the post-study ratio (0,16275) was lower than the pre-study ratio (0,26492) and the 6th week of study ratio (0,25912). This trend in the control group reflects the decline found in the HDL2-C levels at the post-study interval of the control group.

The TC/HDL-C ratio is the most popular ratio reported in studies related to aerobic training. Only one resistive training study (Goldberg et al., 1984) measured this ratio and revealed a significant decrease by 21,6% due to a reduced TC concentration and an unchanged HDL-C level. In this study, the TC/HDL-C ratio remained unchanged because both lipids varied little throughout the study.

Since the HDL-C level was significantly lower in CHD patients (Miller and Miller, 1975; Rhoads et al., 1976; Castelli et al., 1977; Gordon et al., 1977; Goldbourt et al., 1985), many authors regard the TC/HDL-C ratio a good predictor of atherosclerosis (Zampogna et al., 1980; Swanson et al., 1981; Kanamori et al., 1984; Arntzenius et al., 1985; Luknarova et al., 1985; Schmidt et al., 1985; Simons, 1986). A few studies analyzed the HDL subfractions in the CHD population; the HDL2-C concentration was lower in these patients (Miller et al., 1981; Gidez et al., 1982; Wallentin and Sundin, 1985). Gidez et al., (1982) observed proportionately greater changes in the HDL2-C than in the HDL3-C subfraction while Brook et al. (1982) reported the opposite trend. This study observed changes only in the HDL3-C subfraction. If the HDL2-C is a more meaningful lipid component to analyze (responsible for the reverse cholesterol transport), then the HDL2-C/HDL-C ratio is a very relevant ratio to study. This observation was found in this study where total HDL-C remained unchanged and one of its subfractions varied significantly but the HDL2-C/HDL-C ratio proved itself more dicriminatory by producing significant differences which were absent in the TC/HDL-C ratio.

5.4 SUMMARY

Most epidemiological and longitudinal studies have supported the concept that aerobic physical activity reduces the incidence of CHD. Within the anaerobic exercise training, only three longitudinal studies (Lopez et al., 1980; Johnson et al., 1983; Goldberg et al., 1984) demonstrated a positive lipid profile in the experimental subjects, as reflected by an increased HDL-C level and a reduced TC and LDL-C concentrations. These lipoprotein variations could have resulted from a reduction in the percent body fat. When analyzing the role of physical activity and the coronary risk in longshoremen, Paffenberger and Hale (1975) found a lower coronary mortality among the cargo handlers, persons working with repeated bursts of high-energy resistance exercise. Therefore, a critical treshold level of exercise intensity may be the important factor in predicting CHD. Resistance exercise may then offer a protective influence against CHD by modifying the lipoproteins into a favorable lipid profile.

This study assessed the effect of 10 weeks of resistive training in men (10 experimental subjects and 4 controls). Body composition and muscular endurance were measured pre- and post-study. Blood samples were taken prior, at week 6 and at the completion of the study. Triglycerides, total cholesterol, LDL-C, VLDL-C, HDL-C, HDL2-C and HDL3-C concentrations were analyzed. The results indicate that body composition did not vary during the course of the study. The training increased significantly the muscular endurance in the experimental subjects. At the completion of the training, the total cholesterol and LDL-C levels increased slightly non-significantly (NS) but decreased (NS) in the control group. The triglyceride, VLDL-C and HDL-C values did not vary during the course of the study. The HDL2-C concentration increased slightly (NS) in the experimental group. HDL3-C level was significantly greater after the 6th week of

the study for the combined mean of both groups. The HDL2-C/HDL-C ratio began to increase significantly in the experimental group prior to the 6th week of training. Since the control group had a significantly low post-study HDL2-C value, the HDL2-C/HDL-C ratio decreased significantly during the course of the study in their group. Although the high TC and LDL-C concentrations increase the incidence of CHD, the greater HDL2-C/HDL-C ratio in the training group (when the post HDL2-C value in the control group is ignored) offers a favorable lipoprotein profile. Possibly, the resistance exercise increased the enzymatic activity of LPL and LCAT which promotes greater HDL-C and HDL2-C concentrations but these high levels could simply reflect an increased total cholesterol value which would be redistributed amongst all its lipoprotein components.

It is possible that with a greater number of subjects, especially in the control group, and a longer resistive exercise training period, a more substantial change in the lipoproteins of the experimental group would have occurred and the variations observed in the control group would have disappeared.

To fully explain the variations in the lipoprotein concentrations following resistive training, all the cholesterol metabolism components would have to be analyzed. It was beyond the limitations of this study to measure the different enzymes (LPL, LCAT and HL) and apoproteins (A-I, A-II, B, C-I, C-II, C-III, D and E) which regulate the cholesterol transport mechanism.

VI

CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

The effects of 10 weeks of resistive training on the lipoprotein profile of 14 men were studied. Within the scope of this study, the following may be concluded with respect to the effects of resistive training on the variables measured:

1. Muscular endurance increased by 85,3% in this study through training.

2. Body composition was not significantly influenced by the resistive training program.

3. Total cholesterol, total triglyceride, LDL-C, VLDL-C, total HDL-C and HDL2-C levels were not significantly influenced by the resistive training program.

4. The HDL3-C was not influenced by the resistive training; its level was significantly greater after the 6th week of the study for the combined mean of both groups.

5. The HDL2-C/HDL-C ratio rose significantly in the training group. Since the control group produced a low post-HDL2 value, these subjects showed a significantly decreased HDL2-C/HDL-C ratio.

6. Because the total cholesterol and HDL-C levels remained unchanged during the course of the study, the TC/HDL-C ratio did not change significantly.

7. The resistive training program provided a greater HDL2-C/HDL-C ratio. Also, all these changes cannot be attributed only to the training since the control group showed sometimes unexplainable significant changes.

6.2 RECOMMENDATIONS

To explain more fully the results indicated in this study, the following changes are recommended:

1. To explain the metabolism transport mechanism, the measurement of the most important enzymes (LPL and LCAT) and apoproteins (A-I, B and E) is required.

2. More accurate blood lipid results would be obtained by the ultracentrifugation method, the gold standard.

3. The number of participants should be increased, especially in the control group, to better discriminate the changes occurring during the course of the training. For the same reason, the number of "weekly" intervals should be greater to locate more adequately the changes in the lipoproteins.

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Appendix A

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

Physical Activity Readiness Questionnaire (PAR-Q)

Family Name _____ Age _____

Given Name _____ Date _____

For most people, physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

	YES	NO
1. Has your doctor ever said you have heart trouble?	☐	☐
2. Do you frequently suffer from pains in your heart or chest?	☐	☐
3. Do you often feel faint or have spells of serious dizziness?	☐	☐
4. Has a doctor ever said your blood pressure was too high?	☐	☐
5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?	☐	☐
6. Is there a good physical reason not mentioned here why you should not follow an activity programme even if you wanted to?	☐	☐

Appendix B

HEALTH AND LIFESTYLE QUESTIONNAIRES

Health Questionnaire

Family Name _____ Given Name _____

Date of Birth _____

1. MEDICAL HISTORY

Have you ever had or are presently suffering from any of the following health problems?

	YES	NO	If yes, please describe briefly:
Heart Disease	____	____	_____
Rheumatic Fever	____	____	_____
Angina (Chest Pain)	____	____	_____
High Blood Pressure	____	____	_____
Varicose Veins	____	____	_____
Asthma	____	____	_____
Emphysema	____	____	_____
Epilepsy	____	____	_____
Arthritis	____	____	_____
Diabetes	____	____	_____

2. FAMILY HISTORY

Has anyone in your family suffered from any of the following conditions?

	YES	NO	If yes, please describe briefly:
Heart Disease	____	____	_____
Stroke	____	____	_____
High Blood Pressure	____	____	_____
Diabetes	____	____	_____
Nervous Disorder	____	____	_____
High Cholesterol	____	____	_____
Arthritis	____	____	_____

3. MEDICATION

Are you on any type of medication? _____

Name of Prescription	Reason	Dosage

Lifestyle Questionnaire

Family Name _____ Date of Birth _____

Given Name _____

Address _____

Telephone (home) _____ (business) _____

Height _____ Weight _____

SMOKING

1. Do you smoke cigarettes, cigars or a pipe? _____

   Cigarettes: Number per day _____ Number of years smoked _____

   Cigars: Number per day _____ Number of years smoked _____

   Pipe: Amount smoked per day _____ Number of years smoked _____

2. If you have stopped smoking, how long ago was this? _____

ALCOHOL

1. Do you consume alcoholic beverages ? _____

2. If yes, do you consume alcoholic beverages
   Monthly _____ Weekly _____ Daily _____

NUTRITIONAL HABITS

1. Do you regularly eat:

	YES	NO
Breakfast	___	___
Lunch	___	___
Supper	___	___
Snacks	___	___

2. Do you diet? _____
   If yes, why? _____

   _____

   Weight loss _____

   Do you feel yourdieting is successful?

   _____

3. Are you a vegetarian? _____

PHYSICAL ACTIVITY

1. In which physical activity do you participate ? _____

   _____

2. For each activity, how many sessions per week?

Activity	Sessions per week

Appendix C

RAW SCORES

Percent Body Fat and Muscular Endurance Index Values

SUBJECTS		%FAT		MUSCULAR ENDURANCE INDEX	
		Pre	Post	Pre	Post
Exp.	1	35,8	31,3	039	079
	2	30,2	29,2	045	114
	3	24,2	23,2	153	110
	4	29,9	30,7	034*	063*
	5	27,7	30,4	056	103
	6	23,4	27,2	045*	075*
	7	25,4	25,3	051	085
	8	26,3	27,4	027*	041*
	9	33,7	33,0	077	088
	10	25,7	24,9	073	151
Cont.	1	27,7	29,9	045	047
	2	23,7	22,7	040	042
	3	26,1	25,9	065	070
	4	28,7	27,5	054	058

* Due to injury not related to training, only 2 exercises (not the usual 3 exercises) were used to calculate the index.

The Pre-Study Lipoprotein Values

Sub	TRI	TC	LDL	VLDL	HDL	HDL2	HDL3	TC/HDL	HDL2HDL
E1	84,46	248,98	198,83	16,89	33,26	7,33	25,93	7,49	0,2204
2	76,48	197,08	147,42	15,30	34,36	8,03	26,94	5,74	0,2337
3	91,44	192,63	122,95	18,29	51,39	17,29	34,10	3,75	0,3364
4	133,67	319,13	247,85	26,73	44,55	11,80	32,75	7,16	0,2649
5	112,39	196,94	134,94	22,48	39,52	9,73	29,79	4,98	0,2462
6	271,66	187,31	103,42	54,33	29,56	9,39	20,17	6,34	0,3177
7	87,12	191,33	133,67	17,42	40,24	8,02	32,22	4,75	0,1993
8	147,63	246,67	177,36	29,53	39,78	10,98	28,80	6,20	0,2760
9	85,79	191,04	128,70	17,16	45,18	11,60	33,58	4,23	0,2568
10	90,78	146,06	97,86	18,16	30,04	5,08	24,96	4,86	0,1691
C1	104,74	194,06	129,40	20,95	43,71	13,63	34,81	4,44	0,3118
2	124,03	289,37	223,20	24,81	41,36	12,82	27,36	7,00	0,3100
3	131,01	316,40	254,68	26,20	35,52	9,08	26,44	8,91	0,2556
4	71,82	196,22	138,59	14,36	43,27	7,89	29,98	4,53	0,1823

The 6th Week Lipoprotein Values

Sub	TRI	TC	LDL	VLDL	HDL	HDL2	HDL3	TC/HDL	HDL2HDL
E1	119,37	219,07	159,41	23,87	35,79	12,35	23,44	6,12	0,3451
2	70,83	225,98	170,76	14,17	41,05	11,08	29,01	5,50	0,2699
3	109,73	204,13	133,12	21,95	49,06	20,23	30,94	4,16	0,4124
4	102,41	290,23	224,12	20,48	45,63	17,27	28,36	6,36	0,3785
5	104,41	208,01	141,12	20,88	46,01	17,11	28,90	4,52	0,3719
6	156,28	213,04	149,05	31,26	32,73	10,57	22,16	6,51	0,3229
7	79,80	233,60	173,46	15,96	44,18	11,44	32,74	5,29	0,2589
8	86,45	234,60	176,43	17,29	40,88	13,30	27,58	5,74	0,3253
9	125,69	197,51	135,83	25,14	36,54	12,79	23,75	5,41	0,3500
C1	75,48	195,93	130,96	15,10	49,87	16,19	33,68	3,93	0,3246
2	141,65	244,95	184,04	28,33	32,58	8,92	23,66	7,52	0,2738
3	117,04	269,67	209,75	23,41	36,51	7,35	29,16	7,39	0,2013
4	98,76	205,42	148,98	19,75	36,69	8,69	28,00	5,60	0,2368

The Post-Study Lipoprotein Values

Sub	TRI	TC	LDL	VLDL	HDL	HDL2	HDL3	TC/HDL	HDL2HDL
E1	123,69	247,83	184,31	24,74	38,78	11,28	27,50	6,39	0,2909
2	88,45	247,83	187,34	17,69	42,80	13,81	30,70	5,79	0,3227
3	156,61	282,90	196,90	31,32	54,68	20,98	33,70	5,17	0,3837
4	100,42	311,22	246,99	20,08	44,15	16,06	28,09	7,05	0,3638
5	145,97	277,30	199,75	29,19	48,36	12,71	35,65	5,73	0,2628
6	204,16	244,09	173,16	40,83	30,10	7,31	26,07	8,11	0,2429
7	94,77	242,94	176,53	18,95	47,46	13,26	34,20	5,12	0,2794
8	140,98	263,06	190,82	28,20	44,04	10,69	33,35	5,97	0,2427
9	130,34	249,53	178,09	26,07	45,37	16,65	28,72	5,50	0,3670
10	98,09	230,43	170,66	19,62	40,15	11,58	28,57	5,74	0,2884
C1	61,18	172,50	110,43	12,24	49,83	10,17	39,66	3,46	0,2041
2	110,72	255,73	197,34	22,14	36,25	5,60	30,65	7,05	0,1545
3	162,26	252,42	178,98	32,45	40,99	6,89	34,10	6,16	0,1681
4	83,46	177,39	119,01	16,69	41,69	5,18	37,05	4,25	0,1243

Appendix D

COEFFICIENTS OF VARIATION FOR POOLED PLASMA

HDL-C

POOL 1	Within Day (n=9)	50,44 mg% (3,05)	c.v.=6,05%
	Day to Day (n=2 days)	50,57 mg% (1,65)	c.v.=3,26%
POOL 2	Within Day (n=8)	57,05 mg% (1,76)	c.v.=3,09%
	Day to Day (n=2 days)	57,03 mg% (0,12)	c.v.=2,10%

HDL3-C

POOL 1	Within Day (n=9)	34,27 mg% (0,85)	c.v.=2,48%
	Day to Day (n=2 days)	34,32 mg% (0,69)	c.v.=2,02%
POOL 2	Within Day (n=8)	38,71 mg% (1,16)	c.v.=3,01%
	Day to Day (n=2 days)	38,92 mg% (0,85)	c.v.=2,17%

Note: ( ) denotes standard deviations
c.v. denotes coefficient of variation

Appendix E

PERMISSION FOR COPYRIGHTED MATERIAL

February 25th, 1987

The American Heart Association,
7320 Greenville Ave.,
Dallas, TX 75231

To whom it may concern,

I am completing a thesis on the effect of resistive training on the lipid profile of men for my Masters degree in Kinanthropology at the University of Ottawa. In my review of literature I would like to use two figures from one of your articles. I am referring to figures 7 and 8 on page 307 of your article entitled "Lipoprotein-Cholesterol Distributions in Selected North American Populations: The Lipid Research Clinics Program Prevalence Study" by Heiss et al. (1980) in Circulation 61, No. 2, February 1980. These figures would be used solely in my thesis and not for publication in any journal. Therefore, I am requesting for permission to reproduce these two figures for my thesis.

Thanking you for your attention in this matter,

Sincerely,

J. Quenneville

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