CORONARY RISK FACTORS IN PRE-TEENAGE SWIMMERS

By

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ABSTRACT

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Coronary artery disease risk has been positively associated with elevated serum lipids and decreased high density lipoprotein (HDL) cholesterol. Significantly favorable lipid profiles have been seen in elite youth runners but research on normal children placed on exercise training programs has been inconclusive. This study investigated the relationships of exercise duration and intensity, adiposity, diet, blood pressure, family history of premature cardiovascular disease, and work capacity to serum lipids and lipoproteins in young swimmers (9 to 12 years of age) and in a control group of children who were matched with the swimmers by age and sex. Eighteen children, evenly divided between gender, composed each comparison group. Pre-evaluation questionnaires were used to obtain activity history and family history of cardiovascular disease. Dietary history was collected using three-day diaries. Laboratory evaluations included serum lipid

profiles, a physical examamination, hydrostatic and skinfold body fat determinations, and a maximal oxygen uptake evaluation using a continuous treadmill test to exhaustion. Significant differences ($P<.05$) between the swimmers and the controls were found in hydrostatic body fat, systolic blood pressure, maximal heart rate, maximal oxygen uptake, exercise duration and intensity, and in the ratios of low density lipoprotein (LDL) cholesterol to HDL-cholesterol and total cholesterol to HDL-cholesterol. Differences in HDL-cholesterol, LDL-cholesterol, and caloric intake were located in the reserve judgement region. No differences were seen in total cholesterol and in triglycerides. Exercise duration, exercise intensity, and family history were the variables investigated which were associated most highly with the serum lipoprotein variables. The results imply that regular long-term participation in swimming benefits children in terms of their coronary risk factors.

DEDICATION

To my mother;

my wife, Mitzi;

my sister, Karen;

and to the memory of my father.

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CHAPTER I

THE PROBLEM

Coronary artery disease is the leading killer of adults in the United States today. This disease is a byproduct of a chronic, degenerative process called atherosclerosis which is the pathological formation of fibrous plaques in the vascular network inducing a narrowing and eventual closure of the blood conduits. The result of atherosclerosis in the coronary arteries is diminished coronary blood flow in a portion of the heart muscle which may give rise to ischemia, myocardial infartion and death.

Coronary artery disease commomly is believed to begin developing during one's childhood. Autopsies of American servicemen killed in Korea (37) and in Vietnam (102) revealed advanced necrotic lesions in the coronary arteries of these late teenagers and young adults. Fibrous plaques have been reported in persons as young as fifteen years of age (82).

Hypertension, abnormal serum lipid and lipoprotein concentrations and distributions, inadequate stress management, smoking, adiposity, genetic predisposition and chronic diseases such as diabetes all have been implicated in contributing to coronary artery disease.

Most epidemiological studies indicate that serum lipids are strongly associated with the risk of developing coronary artery disease (18,19,55,65). Elevated concentrations of high density lipoprotein (HDL) cholesterol (8,19,55,77) as well as reduced ratios of low density lipoprotein (LDL) cholesterol to HDL-cholesterol (18) and total cholesterol to HDL-cholesterol (18) have been negatively associated with coronary artery disease risk.

One factor postulated to have a positive, causal association with both the total amount and the percentage of HDL-cholesterol in the serum is aerobic exercise (109,156). Endurance running has been the exercise selected most often for research on the effects of physical activity on serum lipids and lipoproteins (59,60,94,100,104,158). This predominance of running studies may be attributed to the popularity of running with adults. However, other physical activites such as swimming, cross-country skiing, and cycling apparently can produce similar aerobic benefits. These activities have been explored minimally in terms of their ability to elicit modifications in serum lipids and lipoproteins.

While there has been extensive research conducted on serum lipids and other risk factors in adults, few studies are available on children (82). In the past, research related to the effects of exercise on serum lipids in normal children addressed either general physical activity (150) or a prescribed exercise program built into the study (34,48,89,91). However, recent research on elite age-group endurance runners (136) has revealed a dramatically high level of HDL-cholesterol. The same results have been reported in elite adult endurance runners (1,100). There has been no research conducted up to this time on the effects of swimming on serum lipids and lipoproteins in pre-teenage boys while the only study that investigated young girl swimmers (161) reported no differences in serum lipids when those subjects were compared to a group of youngsters who hardly participated in sports.

Purpose of the Study

The purpose of this study was to investigate the relationships of exercise duration and intensity, adiposity, diet, blood pressure, and work capacity to serum lipids and lipoproteins in pre-teenage swimmers and control subjects of the same age and sex.

Hypotheses

The following hypotheses were tested during the conduct of the study:

1) There is no difference in the level of total cholesterol between young swimmers and control subjects.

2) There is a significant difference in the HDL-cholesterol and LDL-cholesterol fractions between the two groups.

3) Age-group swimmers between the ages of 9 and 12 years have higher maximal oxygen uptakes than do equivalently aged active controls.

4) There is a meaningful positive relationship between maximum oxygen uptake and HDL-cholesterol combining subjects in the two groups.

5) There is no difference in adiposity between youth swimmers and active control subjects between the ages of 9 and 12 years.

6) There is no relationship between adiposity and HDL-cholesterol combining subjects in the two groups.

7) There is no difference in the percentage contributions of caloric intake from proteins, fats and carbohydrates between the two groups.

8) There is no difference in the ratio of polyunsaturated fat to saturated fat or in the amount of dietary cholesterol ingested between the two groups.

9) There is no difference in resting blood pressure between the two groups.

10) There is no relationship between resting blood pressure and HDL-cholesterol pooling subjects in the two groups.

Research Plan

Eighteen swimmers ranging from 9 to 12 years of age were compared to eighteen active controls selected from the Motor Performance Study at Michigan State University. The subject pool was equally distributed in terms of gender. Activity histories, family histories of cardiovascular disease, medical histories of the participants, and normal dietary regimens were obtained using questionnaires and diaries.

Each subject was required to have venous blood drawn after an overnight fast for determination of serum lipids and lipoproteins. Other laboratory evaluations included medical screening with basal physiological measurements, hydrostatic weighing, anthropometric assessment of adiposity, and maximum work capacity. A continuous treadmill

run to exhaustion was used in determining maximum oxygen uptake.

The serum lipids and lipoproteins evaluated were HDL-cholesterol, LDL-cholesterol, total cholesterol and triglycerides. Repeat samples were acquired if established ranges of normative values were exceeded.

Group differences were analyzed using one- and two-tailed Student t-tests, Chi-square contingency tests, and Mann-Whitney U-tests. Correlational analyses were performed using Pearson product-moment correlations, point-biserial correlations, and Spearman rank-order correlations. A probability level of .05 was set for determining significance in all circumstances.

Limitations of the Study

The conduct of this study necessitates consideration of the following limitations for proper evaluation:

1) An insufficent sample size, 36 subjects, was obtained which required the calculation of a reserve judgement region for some of the statisical analyses. Forty subjects were calculated to be necessary and sufficient for the study.

2) It was not possible to supervise the fasting period before blood samples were drawn. Reliance was placed on the word of the subjects.

3) The data of four of the control subjects were gathered one year prior to the start of this study for another investigation. This resulted in the following limitations relative to these four subjects:

a) Dietary information collected in the previous investigation was insufficient for the current study. Therefore, a current three-day diet diary was required of the four subjects. This diet was assumed to be similar to the diet consumed one year ago.

b) An intermittent treadmill test to determine maximum oxygen uptake was performed in the previous investigation. Results from that test were used in the current study.

c) Resting blood pressure and resting heart rate were not measured in the other investigation and therefore were unavailable for these subjects.

4) The results of this study are applicable only to children between the ages of 9 and 12 years.

5) The pre-test questionnaire and diary data collected were assumed to be accurate and complete. Reliance was placed on the word of the subjects and their parents.

Significance of the Study

Age-group swimming in the United States is participated in by more youths than is age-group endurance running. Yet it is unconfirmed that the potential benefits exhibited in the lipid profiles of elite age-group runners occur in pre-teenage children who take part in age-group swimming. With today's children being tomorrow's candidates for coronary artery disease, this study can make an initial important contribution to understanding and possibly retarding the course of this disease at an early age.

CHAPTER II

REVIEW OF LITERATURE

For simplicity and clarity, the review of relevant literature for this investigation will be discussed in the following sequence:(a) Serum Lipid Structure and Origin, (b) Lipoprotein Structure and Origin, (c) Lipoprotein Metabolism, (d) Lipids and Atherosclerosis, (e) Dietary Influence on Serum Lipids and Lipoproteins and (f) Effects of Exercise on Serum Lipids and Lipoproteins.

Serum Lipid Structure and Origin

Lipids represent one of the major molecular components of all living cells. In humans, lipids are important for metabolic energy storage, the formation of steroid hormones and membrane structure. While many types of lipids are required by the human body, only two lipid forms are

recognized by the majority of the general public: triglycerides and cholesterol.

Triglycerides (triacylglycerols as preferred in systematic nomenclature) are the primary storage form for fatty acids in the body. The general structure of a triglyceride is three fatty acid molecules attached to a glycerol backbone through a series of acylations (Figure 2.1).

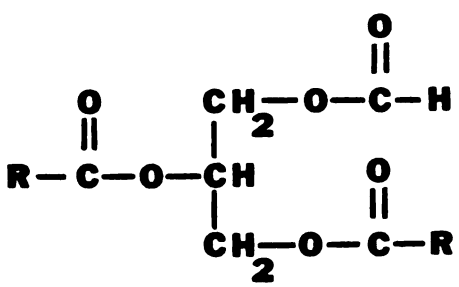

Figure 2.1. Triglyceride Structure (81)

The triglyceride can possess three identical fatty acids or contain a mixture of different fatty acids. The fatty acids can be saturated or unsaturated.

The bulk of the fat contained in the American diet is composed of triglycerides. During intestinal digestion, the triglycerides are hydrolyzed by pancreatic lipases into monoglycerides and/or free fatty acids and glycerol. The monoglycerides and long-chain fatty acids are incorporated with other lipids including cholesterol into micelles. The contents of the micelles are passively absorbed into the intestinal cells of the duodenum and the jejunum by pinocytosis.

In the intestinal mucosal cell, the monoglycerides (carriers of the long-chain fatty acids) and fatty acids of greater than twelve carbons in length are resynthesized into triglycerides within the endoplasmic reticulum (49). These triglycerides then are incorporated into chylomicrons for release to the lacteals and eventual transport into the systemic circulation. As for the fate of the smaller-chained fatty acids, they are absorbed directly into the portal blood.

Endogenous triglycerides play a critical role in the body's aerobic energy utilization. These triglycerides are stored in the adipose cells. Increased need for free fatty acids in the circulation results in hydrolysis of all forms of triglycerides. In contrast, excess free fatty acids are

removed from the circulation for storage as triglycerides in the adipose tissue.

Cholesterol is an ubiquitous molecule in the human body. The structure of cholesterol includes a cyclophenanthrene ring to which a hydroxyl group is attached at carbon position 3, methyl groups are attached at positions 10 and 13 and an eight-membered branched hydrocarbon group is attached at position 17 (143) (Figure 2.2).

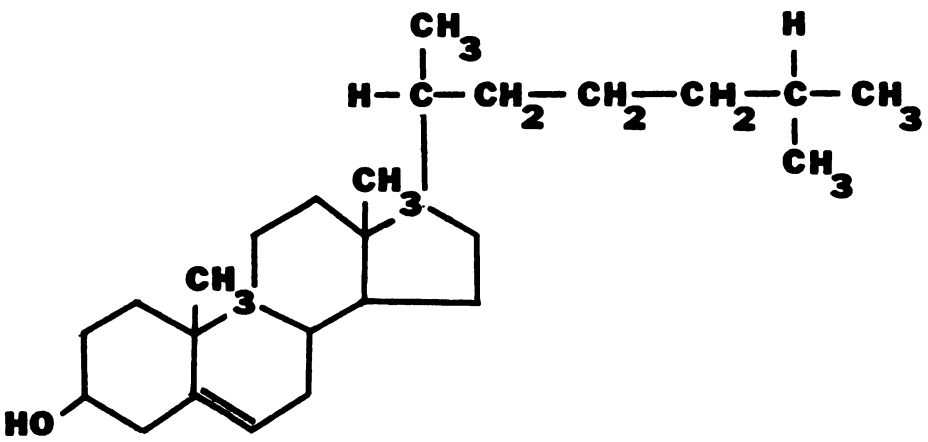

Figure 2.2. Cholesterol Structure (143)

Each cell in the body has the capacity to synthesize cholesterol. This ability results from the requirement for cholesterol as a constituent of all cell membranes. Certain areas of the body either have a large demand for cholesterol, such as the adrenal cortex and reproductive tissues for hormone production and the liver for bile acids and lipoprotein production, or play a significant role in the processing of cholesterol as in the case of the intestine which removes cholesterol from the diet for incorporation into the peripheral circulation.

Endogenous cholesterol is synthesized in the cytoplasm by only one mechanism. The entire carbon skeleton is derived from acetyl coenzyme A (49,143). Control of the mechanism for cholesterol synthesis depends on the enzyme 3-hydroxy-3-methyl-glutaryl coenzyme A reductase (HMG Co-A reductase) which catalyzes the formation of mevalonate with the help of reducing equivalents generated from the hexose monophosphate shunt (49,143).

Exogenous cholesterol is absorbed in the intestinal tract via the micelles and is released to the circulation in the same manner as triglycerides are. However, the majority of the cholesterol ingested is in the form of cholesterol ester. The hydroxyl group on position 3 has been altered to an ester linkage with a long-chain fatty acid. Cholesterol ester cannot be absorbed. Therefore, an esterase from the pancreas catalyzes removal of the fatty acid from

cholesterol in order for both cholesterol and the fatty acid to be absorbed.

The amount of exogenous cholesterol that is absorbed depends on the quantity of cholesterol in the diet. For the first 300 mg ingested daily in the normal human, nearly all of the cholesterol is absorbed. After 300 mg, the body's absorption rate becomes progressively less efficient until a plateau is reached at around 500 to 600 mg of cholesterol ingested (21).

The liver and the intestine are the organs of the body responsible for serum cholesterol levels. The liver is the major site of endogenous cholesterol production. Many control systems are involved with liver cholesterol synthesis. The concentration of cholesterol in the liver cell functions as a negative feedback mechanism of liver cholesterol synthesis by inhibiting the production of HMG Co-A reductase. In this regard, the serum cholesterol level is an important variable in regulating liver cell cholesterol.

Not only can the synthesis of HMG Co-A reductase in the liver be controlled, but the activity of the enzyme can be modified as well. Pancreatic hormones have antagonistic effects on HMG Co-A reductase activity via cyclic-AMP. Insulin release will reduce cyclic-AMP which increases the HMG Co-A reductase activity (46,108). An opposite effect is observed with increased glucagon release (46,108).

The ratio of saturated fatty acids in the diet to polyunsaturated fatty acids can influence HMG Co-A reductase activity. A reduced saturated fat to polyunsaturated fat ratio will lower HMG Co-A reductase activity (21). Furthermore, polyunsaturated fatty acids are involved with increased cholesterol esterification which lowers the free cholesterol concentration (21).

The main control of cholesterol uptake in the intestine is the amount of fat one consumes in the diet (21). The more fat consumed, the more cholesterol taken up by the mucosal cells (108). Therefore, the lack of control of intestinal cholesterol uptake cannot be fully compensated by tight controls on cholesterol synthesis in the liver. This explains why some normal individuals, though not all, who consume excess dietary cholesterol may exhibit elevated serum cholesterol.

Lipoprotein Structure and Origin

A problem exists in the transport of lipids such as cholesterol and triglycerides throughout the body since the body's transport medium, blood, is an aqueous solution. By complexing lipids with protein noncovalently to form water-soluble lipoproteins, transport for lipids via the blood is assured (84).

The structure of a lipoprotein consists of a hydrophobic inner core of the nonpolar lipids, triglycerides and cholesterol esters, and a hydrophilic outer capsule comprised of proteins and polar lipids such as phospholipids and unesterified cholesterol (49,57). There are four distinct classes of lipoproteins: chylomicrons, very low density lipoprotein (VLDL or pre-beta) , low density lipoprotein (LDL or beta) and high density lipoprotein (HDL or alpha). Another lipoprotein entity, intermediate density lipoprotein (IDL) has special characteristics that will be described later (142).

Lipoproteins are classified by electrophoretic and ultracentrifugation techniques. Size and density of the lipoproteins are inversely related. The lipoprotein classes are established by the percentage of lipid and protein the molecule possesses. Not only do the percentage of lipid and protein vary among the classes but the composition of the lipids and proteins in the various classes differs as well (49)(Table 2.1).

The protein portion of the lipoprotein is called an apoprotein. There are five categories of apoproteins, A through E, as shown in Table 2.2. These proteins are vital in maintaining the integrity of the lipoprotein structure, especially in terms of water-solubility. Apoproteins also help regulate the metabolic functioning of the

TABLE 2.1. Composition of Lipoproteins (49).

	Lipoprotein Type			
	Chylo	VLDL	LDL	HDL
Average % Composition				
Triglycerides	86	55	8	5
Phospholipids	8	20	20	30
Cholesterol	2	10	10	5
Cholesterol Esters	2	6	37	15
Protein	2	9	25	45

Chylo = Chylomicrons

VLDL = Very Low Density Lipoproteins

LDL = Low Density Lipoproteins

HDL = High Density Lipoproteins

TABLE 2.2. Classification of Apolipoproteins (9).

Apo	Density Class	Mr	Function
A-I	HDL	28,000	LCAT Activator
A-II	HDL	17,000	unknown
B	VLDL,LDL,Chylo	250,000	unknown
C-I	" " "	6,500	LCAT/LPL Activator
C-II	VLDL,LDL, ? ,HDL	~10,000	LPL Activator
$C\text{-}III^{0-2}$	" " ? "	10,000	? LPL Inhibitor
D	HDL_3	~20,000	? LCAT Activator
E_{1-2}	VLDL,LDL,HDL	32-39,000	?Chol. Transport

Apo = Apolipoprotein

Mr = Molecular Weight

VLDL = Very Low Density Lipoprotein

LDL = Low Density Lipoprotein

HDL = High Density Lipoprotein

Chylo = Chylomicron

LCAT = Lecithin:Cholesterol Acyltransferase

LPL = Lipoprotein Lipase

Chol. = Cholesterol

lipoproteins by activating two important enzymes: lipoprotein lipase (LPL) and lecithin-cholesterol acyltransferase (LCAT).

Chylomicrons are the largest of the lipoproteins and are the least dense of the lipoprotein moieties due to the high percentage of lipid incorporated. The exclusive location of chylomicron assembly and secretion is the mucosal cells of the intestine.

Dietary triglycerides comprise the bulk of the lipid contribution of chylomicrons. Cholesterol, cholesterol ester and phospholipid are present too. Of these components, only phospholipid is primarily endogenous in origin (45,46). Just two percent of the chylomicron is composed of protein (49). All five classes of apoproteins are contained in chylomicrons with apo-C and apo-B being in greatest quantity (9).

Intestinal chylomicrons reach the circulation via the lymphatic system emptying into the thoracic duct. Apoprotein C is not added to the chylomicron until it reaches the plasma circulation (45). The intestine does not possess the ability to synthesize apo-C (108). Therefore, the chylomicron must obtain apo-C from a circulating source which is HDL (46).

Very low density lipoprotein is synthesized from endogenous substrates primarily in the hepatic parenchymal cells (46). There is evidence that VLDL can be produced in

the intestinal mucosal cell as well (45,46). Apoproteins B,C and E predominate in the VLDL with apo-C obtained from HDL just as with chylomicrons (46,137). The VLDL is the primary carrier of endogenous triglycerides.

The intermediate density lipoprotein (IDL) is a transient state for a VLDL in its degradation to LDL (142). The density and size of these lipoproteins are intermediate between the VLDL and LDL, hence the name.

Low density lipoproteins consist of spherical particles of diameter 200-250 A and molecular weight of 2.0-2.5 million (142). The major constituents of LDL are cholesterol and cholesterol ester. The only apoprotein incorporated into LDL is apo-B and it accounts for twenty-five percent of the mass of the lipoprotein (142). The LDL originates in the plasma as the degradation product of VLDL (142).

High density lipoprotein (HDL) is the smallest lipoprotein in diameter but also the densest. High density lipoprotein comprises the only class of lipoproteins to be divided into subgroups, HDL_2 and HDL_3, on the basis of density and molecular weight. Schaefer and Levy (129) state that HDL_3 is made up of approximately fifty-five percent protein and forty-five percent lipid, while the protein/lipid proportions in HDL_2 are 40/60. The subgroups of HDL differ in the ratio of free cholesterol to esterified cholesterol with HDL_2 possessing the higher ratio (129).

The primary apoproteins contained in HDL are apo-AI and

apo-AII with lesser contributions from apo-C, apo-D and apo-E (45,46,142). Apoprotein AI is the primary stimulator of LCAT activity (46,141) and for this and other reasons, HDL is placed in a pivotal situation to affect lipoprotein metabolism.

The formation of HDL arises from two generally accepted sources: parenchymal cells of the liver (46,144) and absorptive cells of the small intestine (56,142). Some authors also report that HDL can be produced from the surface remnant particles of chylomicrons following hydrolysis of triglycerides (142).

Lipoprotein Metabolism

The primary enzymes of lipoprotein transport and metabolism are lipoprotein lipase (LPL) and lecithin-cholesterol acyltransferase (LCAT). Other enzymes of importance for this study include acyl-coenzyme A:cholesterol acyltransferase (ACAT), hormone-sensitive lipase and 3-methyl-3-methyl-glutaryl coenzyme A reductase (HMG-Co A reductase).

Lipoprotein lipase is manufactured in parenchymal cells of adipose tissue, heart muscle and skeletal muscle (152). Nilsson-Ehle (113) has proposed that the protein enzyme is not only synthesized in the parenchymal cell but is stored

and modified for activation there as well.

By some unknown mechanism, LPL is transported from the parenchymal cells to the luminal endothelial surface of extrahepatic capillary walls to which it is bound by high affinity receptors for LPL (10,112,137). Robinson (137) suggests that glycosaminoglycans may be involved in the binding.

Lipoprotein lipase catalyzes the hydrolysis of triglycerides from plasma chylomicrons and VLDL into free fatty acids, monoacylglycerol and monoglycerides. The hydrolysis products are removed by the adipose tissue for storage via reesterification to triglycerides, or are removed by the heart and skeletaal muscles for oxidative substrates. The hydrolysis and uptake processes are required to regulate plasma triglyceride levels, especially after feeding.

Characteristic physical properties of LPL are a cofactor requirement of apo-CII, inhibition by high salt and activation by heparin (10,137). A 1/1 ratio of LPL to apo-CII provides the maximal rate of trigylceride hydrolysis (137).

Lipoprotein lipase activity varies from tissue to tissue in response to changing hormonal and nutritional situations. Insulin seems to be the primary hormonal agonist of LPL (10,137), but this has been witnessed in adipose tissue LPL exclusively (10).

Lecithin-cholesterol acyltransferase is synthesized primarily in hepatic parenchymal cells (33,115). Upon secretion into the plasma, LCAT's half-life is approximately 4.6 days (116). In the plasma, LCAT is found attached to HDL which is the only lipoprotein on which LCAT acts (116).

Lecithin-cholesterol aclytransferase is responsible for catalyzing the plasma esterification of free cholesterol to cholesterol ester. Another closely related enzyme, ACAT, displays similar biological action but is located intracellularly. The esterification process involves the transfer of fatty acid from the C-2 position of lecithin to cholesterol (50).

Activation of LCAT requires apoprotein cofactors. Apoprotein AI, the major apoprotein of HDL, has been identified as the most potent activator of LCAT (56,57). Apoprotein CI has been shown to activate LCAT too (33,50,116).

The action of LPL on chylomicrons and VLDL provides the necessary substrates for LCAT, cholesterol and lecithin. These remnants are esterified and incorporated into HDL_3, converting the HDL_3 into HDL_2 (50).

The importance of LCAT encompasses cholesterol removal and transport between the circulating lipoproteins and the extrahepatic tissues. Glomset (52) has proposed that LCAT catalyzes a reaction between HDL and free cholesterol on the arterial wall surface. High density lipoprotein would

transport the newly formed cholesterol esters back to the liver for derivatization of the sterol ring (52). This theory has been demonstrated in rats (29) but not in humans.

Norum, Berg and Drevon (116) have suggested that not only HDL can transport cholesterol ester to the liver, but VLDL and LDL can as well. This concept is consistent with the report of a protein which apparently transfers plasma cholesterol among the lipoproteins (20). However, this proposed process would allow the cholesterol ester from HDL to be available for entry into the arterial endothelial cells via the LDL receptor (11) as well as for transport to hepatocytes. An unresolved question that relates to this theory is the destination of the newly formed cholesterol ester carrier: arterial cell or hepatocyte or both.

Hormone-sensitive lipase is located in the adipose cells and its biological action is to hydrolyze the stored triglycerides to free fatty acids and glycerol. The lipase derives its name from its activation by epinephrine, which initiates a cyclic-AMP protein kinase response.

The interaction between the various lipoprotein moieties and their corresponding enzymes results in a complex scheme for the transport of cholesterol and triglyceride to extrahepatic tissues (Figure 2.3). Brown, Kovanen and Goldstein (11) have proposed a transport mechanism which is divided into an exogenous pathway and an endogenous pathway.

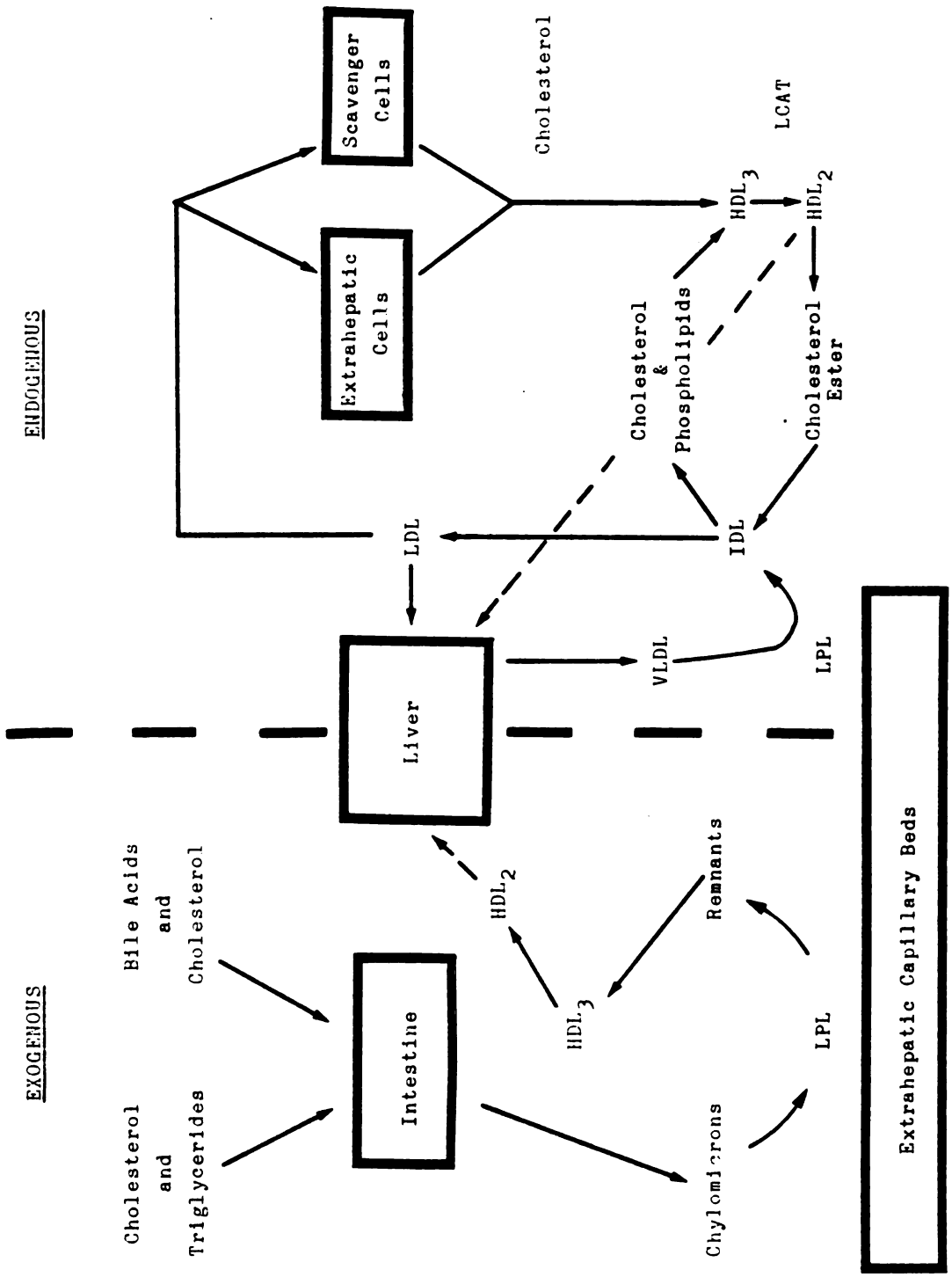

Figure 2.3. Proposed Mechanisms for Cholesterol Transport (11)

Cholesterol synthesis is under the regulation of HMG-Co A reductase. This enzyme determines the rate of the reductiion of HMG-Co A to mevalonic acid (105). Plasma levels of cholesterol are not directly affected by HMG-Co A reductase since it's located intracellularly. However, dietary intake of cholesterol inhibits the hepatic synthesis of cholesterol by inhibiting HMG-Co A reductase (105). This feedback inhibition is exhibited in the capillary endothelial cell with high levels of LDL-cholesterol.

The exogenous pathway incorporates dietary cholesterol and triglycerides into the plasma circulation (11). As mentioned earlier, chylomicrons enter the plasma circulation via the thoracic duct. Apoproteins C and E are donated from HDL. The apo-CII activates LPL, to which the chylomicron has been bound, releasing free fatty acids, monoglycerides and glycerol for peripheral uptake as energy substrates. The excess surface components, cholesterol and phospholipids, are picked up by HDL_3 (11). This process, catalyzed by LCAT, transforms the HDL_3 into HDL_2 (57,141). The HDL_2 can be delivered to the liver for degradation (139), or the cholesterol ester core can be transferred to either IDL or LDL (11,20). The remainder of the chylomicron, referred to as remnants, is transported via the circulation to the liver (11,57). At the liver, the remnants bind to specific receptors and are internalized for catabolism (11).

The other side of the transport picture involves

endogenous lipids. The liver utilizes dietary cholesterol obtained from the remnants and HDL_2 as well triglycerides converted from carbohydrates and free fatty acids to assemble VLDL for the transport of these neutral lipids (11). The VLDL attaches to LPL when released into the circulation and triglycerides are hydrolyzed. The surface components are transferred to HDL_3. HDL_2 is formed which follows the same fate as HDL_2 formed in the exogenous scheme.

The VLDL has been transformed into IDL and is released from the LPL receptor to the circulation (11). The transient IDL has two possible fates: conversion to LDL thru further loss of free cholesterol, triglycerides and phospholipids with concomitant uptake of cholesterol ester, or direct uptake by the liver for catabolism (11,57).

The unsettled fate of LDL was touched upon when LCAT was discussed earlier. Low density lipoprotein receptors are found on both the hepatocytes and the extrahepatic endothelial cells (11,57,115).

The reverse cholesterol transport theory proposed by Glomset (50,51,52) involves the uptake of free cholesterol from the capillary cell surface by HDL_3 with the subsequent esterification by LCAT to form HDL_2. Brown, Kovanen and Goldstein (11) argue, "In the steady state, tissues excrete cholesterol into the plasma in amounts equal to the amounts taken up from LDL." This concept lends support for reverse

cholesterol transport. However, the fate of HDL_2 is controversial. Glomset (52) postulates that the HDL_2 formed will be transported to the liver for degradation, whereas others (11,113) support the transfer of cholesterol ester from HDL_2 to IDL, thereby creating a futile cycle for cholesterol transport.

Lipids and Atherosclerosis

In the United States, atherosclerotic heart diseases is one of the leading causes of death. Atherosclerosis can be described as a silent, chronic killer. Autopsies on soldiers in their late teens and early twenties who were killed in Korea (37) and Vietnam (102) revealed advanced atherosclerotic lesions. Fibrous plaques have been viewed in persons as young as fifteen years of age (82).

"Atherosclerosis is a disease that results from a complex interplay of many physiologic, genetic and enviromental factors," say Lee and Lauer (82). Many risk factors for coronary atery disease (CAD), an endproduct of atherosclerosis, have been identified. The most common risk factors include serum lipids and lipoproteins, hypertension, smoking, family history of CAD, stress, obesity, inadequate physical activity and diabetes mellitus (82,83,90,126,157).

Serum lipids and lipoproteins have been well documented

as being a primary, if not the primary, risk factor for CAD. When the risk factor concept for CAD was introduced, serum cholesterol was implicated as the chief lipid measure for determining CAD risk (69). After reviewing thirty-two epidemiological studies on CAD, Stamler (140) said, "The positive findings demonstrating a relationship of serum cholesterol to CAD incidence are of a scope and consistency rare in medical research on chronic, non-infectious diseases."

As more research was conducted on the relationship of serum cholesterol and CAD, it became clear that the distribution of serum cholesterol in the lipoprotein fractions was a strong indicator of CAD risk (55,70). Various investigators have shown LDL-cholesterol to be positively correlated with CAD (19,115), while HDL-cholesterol was found to be negatively associated with the development of CAD (30,54,70,115). Pometta et al. (120) found that the only risk factor that differed between children of parents with CAD and children whose parents did not have CAD was lowered HDL-cholesterol in the former.

A recent report by Castelli, Abbott and McNamara (18) has indicated that ratios of total cholesterol to HDL-cholesterol and LDL-cholesterol to HDL-cholesterol are useful measures for determining CAD risk. "However, neither ratio by itself may be as informative about the risk of developing CAD as the information contained in the

configuration of the specific values of cholesterols," explained the authors (18).

When lipoprotein fractions have been evaluated, the value of the triglyceride measure as a predictor has been questioned (55). Originally, reports of the Framingham study (147) had identified triglycerides as a risk factor. Further work with the Framingham group suggested that triglycerides made no significant contribution to the prediction of CAD (55).

The epidemiological work on children and CAD risk is less complete than it is for adults. However, according to Linder and DuRant (90), there is sufficient evidence to indicate that CAD risk can be and should be monitored in children since these factors seem to persist into adult life. A study by Ibsen, Lous and Anderson (68) indicates that hyperlipidemia is the only major risk factor which is seen frequently in children of CAD victims. Vartainen, Puska and Salanen (153) surveyed 996 thirteen-year old Finnish children and found high mean serum cholesterol levels as a result of which they concluded that the prevention of CAD should start in childhood. Studies in the mid-1970's of U.S. schoolchildren indicated abnormal levels of serum cholesterol in nearly ten percent of the children evvaluated (31,41,80). Wilmore and McNamara (157) evaluated 95 boys, eight to twelve years of age, and found obesity in thirteen percent, elevated serum cholesterol in twenty percent, and

hypertriglyceridemia in eight percent of these youngsters.

Most of the research on controlling serum lipids and lipoproteins in humans has focused on dietary modification and exercise prescription. These areas are interrelated with many risk factors previously mentioned and therefore require a thorough review.

Dietary Influence on Serum Lipids and Lipoproteins

The influence of diet on the synthesis and transport of serum lipids has been extensively researched. The strength of association between dietary cholesterol and the incidence of atherosclerosis has been controversial because of a lack of direct evidence (90,101). McGill (101) states, "Most cross-sectional studies of individuals within population groups have failed to show associations between cholesterol intake and plasma cholesterol levels or between cholesterol intake and the incidence of atherosclerotic disease." An example of this is the Tecumseh study (110). Knuiman, Hermus and Hautvast (76) support McGill's view by saying that the association between dietary cholesterol and plasma cholesterol is more apparent between population comparisons. However, Shekelle *et al*. (133) determined that dietary cholesterol was significantly associated with death from CAD in a longitudinal study of 1900 men. They contend that

dietary cholesterol can be associated with atherosclerosis via many mechanisms other than serum cholesterol such as lipoprotein structure and distribution alterations (133).

Dietary cholesterol is not the only dietary parameter which is thought to influence plasma lipoproteins. Excess calories, carbohydrates, and fats have been implicated in various lipoprotein changes. Originally, other dietary constituents such as proteins (23) and fiber (124,160) were believed to have no effect on serum lipids and lipoproteins. Research by Carroll et al. (16) and Sirtori et al. (135) showed that soy protein reduces serum cholesterol, while Anderson and Chen (3,21) found that soluble fiber can reduce serum cholesterol too.

Caloric excess is the most common cause of hypertriglyceridemia according to Thompson (148). The lipoprotein fractions affected are increased VLDL and IDL (22,148). The excess calories provide extra substrate for triglyceride synthesis by the liver (22). Another problem for persons with hypertriglyceridemia is that the swollen adipose cell becomes insulin-resistant which limits the activity of LPL on the adipose cell surface (22,148).

Caloric restriction has been shown to have a lowering effect on VLDL-cholesterol (155) while lowering HDL-cholesterol as well (148,155). A concomitant change in LDL-cholesterol does not accompany the reduction in HDL-cholesterol (155). The result is elevated

LDL-cholesterol to HDL-cholesterol and total cholesterol to HDL-cholesterol ratios (155) which indicates increased CAD risk (17). A combination of caloric restriction and mild exercise does not decrease the levels of HDL-cholesterol (155).

Carbohydrates have a transient elevating effect on plasma triglycerides (22). Populations which subsist on high-carbohydrate diets do not display elevated triglycerides and have a low incidence of CAD (23).

There are some conflicting reports on the effects of a high-carbohydrate diet on HDL-cholesterol. Most of the literature supports the view that persons who consume a high-carbohydrate diet have lowered levels of HDL-cholesterol (38,117,130,131,148). However, endurance athletes who eat high-carbohydrate diets have been shown to exhibit elevated HDL-cholesterol (149). Of course, the effects of diet and exercise cannot be differentiated in athlete populations.

The effects of dietary fat on serum lipids and lipoproteins have been studied intensely. Both the amount and the composition of dietary fats are important factors in lipoprotein metabolism (22,148,154). The primary effect of excess fat intake is hypertriglyceridemia due to excess chylomicron production and absorption (22,71). Plasma cholesterol levels may rise, but the composition of the fat is the determining factor (22,148).

Saturated fats raise plasma cholesterol while polyunsaturated fats lower serum cholesterol (22). A neutral effect on cholesterol is obtained from ingesting monounsaturated fats (22). Keys (72) has shown that the correlation of saturated fatty acids with CAD is approximately the same as the correlation of serum cholesterol with CAD.

Dietary changes in fat primarily affect LDL-cholesterol (148). When LDL-cholesterol is reduced from increasing the polyunsaturated to saturated fat ratio, the reduction can be attributed to decreased cholesterol carried by the LDL (87).

The literature related to the effects of dietary fat on HDL-cholesterol is confusing. In the Lipid Research Clinics' Prevalence Study (38), the total fat intake as well as the composition of fat absorbed from saturated, polyunsaturated and monosaturated fat were unrelated to HDL-cholesterol. These findings were substantiated by Oster et al. (117) who gave a normal fat diet with an elevated polyunsaturated to saturated fat ratio to type II and type IV hyperlipoproteinemic patients.

A study by Hjermann et al. (63) reports that lowering the total fat contribution of the diet along with increasing the polyunsaturated to saturated fat ratio results in increased levels of HDL-cholesterol. Kiens et al. (75) suggests that physical activity may induce an adaptation in lipoprotein metabolism such that the effects of dietary

modification are minimized.

In summary, the effects of diet on serum lipids and lipoproteins are controversial, especially with regard to HDL-cholesterol. Schlierf, Arab and Oster (130) believe that HDL-cholesterol is influenced by diet but think that the most dominant effect of diet is on LDL-cholesterol. Wood (58) says, "Diet appears to be the most significant factor in causing short-term changes in serum lipid levels and that exercise probably accounts for long-term changes."

Dietary survey techniquess probably are responsible for a portion of the conflicting results. Keys (73) says, "Collection of good data on food intake is time consuming for both subject and surveyor, and the more data the more expensive is the analysis." One-day diet recalls give meaningless estimates of a person's usual diet according to Todd, Hudes and Calloway (151). Keys (73) suggests that a seven-day recall avoids erroneous results. Marr (99) claims that three-day and seven-day surveys are closely correlated and that a three-day survey is appropriate to use.

Effects of Exercise on Serum Lipids and Lipoproteins

There exists substantial epidemiological evidence that exercise modifies CAD risk factors (17,43,118). Hooper and Eaton (66) believe that exercise reduces CAD risk by

altering multiple factors such as weight, cardiovascular dynamics, cigarette smoking, and serum lipids and lipoproteins.

Even with the epidemiological evidence, there exists much disagreement as to what the role of exercise is in lipoprotein metabolism. This controversy is due to some research failing to exhibit significant changes in serum lipids, inability to control for confounding variables that may affect lipoprotein metabolism, and unsubstantiated mechanisms explaining the effect of exercise on serum lipids.

Extensive evidence indicates that endurance athletes exhibit reduced serum triglycerides when compared to sedentary controls (59,60,85,106,158). This pattern has been witnessed in comparisons of joggers and marathon runners (59) or high mileage runners (60). A dose-dependent effect of exercise is suggested. Lowered triglycerides have been seen in low-active adults that have engaged in an aerobic exercise program of four months (67) duration.

Serum triglyceride hydrolysis is believed to be under the control of LPL. Increased physical activity has been shown to raise LPL activity in well-trained men (145). Costill et al. (26) showed increased LPL activity in normal untrained men after participation in a training program.

In endurance exercise, the muscle preferentially utilizes free fatty acids as an oxidative substrate due to

having limited carbohydrate stores. Serum free fatty acid concentration is increased to meet the elevated demand by the muscles (121). Possible sources for these free fatty acids are triglyceride depots in the adipose tissue which are under the control of hormone-sensitive lipase, circulating VLDL, and chylomicrons which are hydrolyzed under the control of LPL. Terjung et al. (146) has shown increased uptake of chylomicron triglyceride in the muscles of dogs during exercise.

According to Mackie et al. (97), little direct evidence is available to weigh the contribution of plasma triglycerides during exercise. This statement takes on greater meaning when one is presented with the research that contradicts the role of exercise in lowering triglycerides. In exercise training studies of six weeks (94), eight weeks (39), fifteen weeeks (119) and sixteen weeks (124) no significant changes were seen in serum triglycerides from pre-testing to post-testing. In comparisons of male (1) and female (28) distance runners to controls, no significant differences in serum triglycerides were noted.

Summarizing, the triglyceride research suggests that habitual, endurance exercise has a lowering effect on serum triglycerides. The variability of triglyceride measurement (the normal range is 10-190 mg/dl) along with modifying factors such as caloric intake (23), alcohol consumption (59), body weight (85), adiposity (85), diet composition

(23) or a genetic component (60) may mask or limit the proposed exercise adaptation.

The most controversial lipid fraction to medical clinicians is total cholesterol. Exercise has been associated with lowered total cholesterol (59,60,100,105,158), but some studies (86,136,139,149) report no change in total cholesterol-- only a redistribution of cholesterol in the various lipoprotein fractions. Other studies (1,28,106,138) indicate only significant changes in HDL-cholesterol with no change in total cholesterol. These inconsistencies reveal that total cholesterol is probably a poor choice as a primary indicator for the effects of exercise on serum lipids and lipoproteins and should be interpreted carefully.

The bulk of research on exercise and lipoproteins has focused on HDL and the cholesterol it carries. There is overwhelming evidence that participation in endurance activities such as running (1,59,60,85,86,106,119,149, 158,159), cross-country skiing (67,85,106), cycling (67) and swimming (138) is associated with increased HDL-cholesterol. The few articles which refute this finding in adults are studies utilizing short-term training programs of six weeks (94,105) and nine weeks (125) respectively.

One controversial issue that remains is: What duration and intensity of aerobic exercise is required to elicit changes in plasma lipoproteins and/or changes in cholesterol

transport? Farrell and Barboriak (39) reported that four weeks of training at 70% of VO_2 max for three to four days per week were required before a improved lipid profile could be seen. Williams et al. (156) states that a positive, causal relationship exists between running and HDL-cholesterol but that nine months of running for at least ten miles per week is required. If the claims of Williams et al. (156) are correct, this would explain why no HDL-cholesterol changes were detected in some of the training programs.

The LDL-cholesterol fraction is usually lowered in individuals who exercise (59,100,136,158). Studies by Hartung et al. (59) and Martin, Haskell and Wood (100) displayed identical patterns of elite runners having significantly lowered LDL-cholesterol when compared to either joggers or sedentary controls. There was no difference in LDL-cholesterol between joggers and controls. These findings could indicate self-selection or could support the explanation of Williams et al. (156) that a threshold exists for affecting LDL-cholesterol similar to the one postulated to affect HDL-cholesterol. Diet may be involved with delaying the exercise response.

Both LPL and LCAT are believed to be involved in the adaptation of lipid transport to exercise. Nikkila et al. (112) has suggested that increased LPL activity due to exercise would result in a greater number of remnant and

surface particles in the circulation from increased triglyceride metabolism. The surface particles from VLDL and chylomicrons would be incorporated into the HDL_3 forming the HDL_2 (52) catalyzed by the exercise-stimulated action of LCAT (98). Marnieni et al. (98) have stated that the actions of LPL work in parallel with the actions of LCAT. With increased VLDL catabolism, fewer LDL particles should remain in the circulation and become attached to the endothelial surface receptors.

Another possible mechanism could be that exercise somehow promotes an increase in the reverse cholesterol transport phenomenon proposed by Glomset (52). This would increase the amount of cholesterol carried by HDL_2. For this process to take place, more cholesterol would have to be released from the cell into the circulation than is taken up from the LDL at the LDL receptor since no HDL receptor has been discovered on the endothelial cell surface to pick up unesterified cholesterol. Furthermore, this newly formed HDL_2 would have to avoid the cholesterol ester transfer protein (20) so that increased transport to the liver for catabolism could occur.

The research on exercise and serum lipids and lipoproteins in children is very limited. Linder, DuRant and Mahoney (91) studied the effect of an eight-week aerobic interval training program on white male adolescents and found no significant changes in serum lipids and

lipoproteins. DuRant et al. (34) found no association of physical activity with lipid profiles in black children. Gilliam and Burke (48) conducted a study to ascertain the effects of a six-week physical activity program on serum lipids in 14 girls aged 8 to 10 years. They found significantly increased HDL-cholesterol and a significantly decreased total cholesterol to HDL-cholesterol ratio. A study by Nizankowska-Blaz and Abramowicz (114), comparing control children with children having ten 45-minute activity periods per week, showed that the more active children displayed significantly higher HDL-cholesterol and lower triglycerides. However, no differences were found in LDL-cholesterol or total cholesterol. Thorland and Gilliam (150) divided a group of pre-adolescent males into two activity groups based on an activity questionnaire. The results indicated no differences between groups in terms of HDL-cholesterol or total cholesterol but significantly lowered triglycerides and total cholesterol to HDL-cholesterol for the more active children. A recent study by Smith et al. (136) indicates that elite youth-age runners have lower triglycerides and LDL-cholesterol, higher HDL-cholesterol and lower, more favorable lipid ratios than do active controls.

The literature contains only one study on swimming and lipid profiles in children. Zonderland et al. (161) compared the apolipoprotein and lipid profiles of 20 female swimmers

aged approximately 12 years old who swam at the club level roughly 4.6 hours per week to 12 controls who participated infrequently in sports. The results indicated no difference in the ratios of LDL-cholesterol to HDL-cholesterol and total cholesterol to HDL-cholesterol between the two groups. These findings contradict an earlier study by Smith et al. (138) which showed increased HDL-cholesterol and a lowered total cholesterol/HDL-cholesterol ratio in collegiate female competitive swimmers when compared to synchronized swimmers and sedentary controls.

To summarize, there is overwhelming evidence to support aerobic exercise eliciting a change in lipoprotein metabolism in adults. The contradictory evidence can usually be explained by insufficient training. The mechanisms postulated for the favorable alterations in the serum lipids and lipoproteins are as yet unproven.

In children, the evidence supporting the effects of exercise on serum lipids and lipoproteins is sketchy and more contradictory. However, this could be due to the limited number of studies which were conducted either with insufficient training or under restricted conditions.

CHAPTER III

RESEARCH METHODS

This study was designed to determine if differences exist in two age- and sex-matched comparison groups (pre-teenage swimmers and active control subjects) and to investigate the factors associated with possible serum lipid and lipoprotein alterations. The results of this retrospective study were analyzed to identify the reasons for the differences observed.

Subjects

Eighteen caucasian youngsters from 9 to 12 years of age were invited from swim clubs in the Lansing, Michigan area to participate in the study. Each swimmer had to have been swimming 3,500 yards per day, at least four days per week

for a minimum of 35 weeks, to be included in the swimmers group. The control subjects were selected from the Motor Performance Study conducted by the Michigan State University Department of Health and Physical Education. The subjects were equally distributed in terms of sex.

The subjects had no recent history of medication known to affect serum lipids and lipoproteins, no prior history of smoking, and no prior history of alcohol consumption. The subjects and their parents were informed of the nature, purpose and possible risks of the study and were required to sign informed consent and release documents.

Preliminary Data Collection

The parents of the subjects received activity history questionnaires with detailed instructions for proper completion (Appendix A). The questionnaires asked the subjects to list the organized sporting activities they were currently participating in, the length of participation, year of first participation, and a subjective perception of the intensity of the average workout on a scale from 1 to 20. Data sheets to record a three-day food intake diary on the Wednesday, Thursday and Friday before the scheduled test date were mailed with instructions for completion (Appendix B). A medical history questionaire was sent to the parents

to assist medical screening (Appendix C). All questionnaires and diaries were collected at the time of testing.

Laboratory Data Collection

The subjects came to the Center for the Study of Human Performance after an overnight fast of twelve hours. All subjects were instructed not to participate in strenuous physical activity during the twelve hours immediately prior to testing.

Upon entering the laboratory, each subject had blood drawn from an antecubital vein. Blood was collected in serum separator vacutainers of 7 ml capacity. The samples were cooled immediately to 4° C and the cells were removed by centrifugation within one hour.

The subjects proceeded randomly to stations for activity history follow-up screening, diet diary follow-up screening, medical screening, and anthropometric evaluation. Activity history and diet diary follow-ups consisted of collecting pre-test questionnaires and diaries and making sure all items asked were answered according to the directions. A pediatric cardiologist conducted the medical screening by reviewing the medical history questionnaire and conducting a physical examination for disqualifying abnormalities. Resting blood pressure and resting heart rate

were determined with the subject in a seated position using an Olympic Accu-Sphyg[1] sphygmomanometer. All blood pressures were recorded by the cardiologist to minimize technician error. Anthropometric evaluation involved measuring the subjects' standing heights and body weights. Skinfold measurements were collected from the subscapular, suprailiac, triceps and biceps skinfolds using a set of Lange[2] skinfold calipers. The skinfold measurements were used in a prediction equation for percentage of body fat as developed by Durnin (35).

Each subject then proceeded to the hydrostatic weighing station. Underwater weighing was performed in a seated position on a chair assembly which is supported by a strain gauge. Signals from the strain gauge were sent to a chart recorder[3] via a Wheatstone bridge. Residual lung volume underwater was determined using a modification of the closed-circuit method of Lundsgaard and Van Slyke (96) which has been further modified by Rahn, Fenn and Otis (122). This method involves oxygen rebreathing from which a sample is analyzed for nitrogen content with a MedScience 505 Nitralyzer[4]. A modification of Buskirk's formula (14) was used to obtain body density. Body fat was calculated using the Siri formula (134). A sample worksheet is included in Appendix D.

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[1]Olympic Medical, Seattle, WA.

[2]Cambridge Instrument Co., Ossining, NY.

[3]Model 2115M, Allen Datagraph Corp.

[4]Med-Science Electronics, St. Louis, MO.

The final station for each subject was work capacity. A continuous treadmill run to exhaustion was administered to obtain respiratory variables necessary for determining maximum oxygen uptake. The treadmill speed was fixed at five miles per hour. The protocol began with the treadmill set at zero percent grade. At the end of each minute, the treadmill grade was increased by one percent until the subject could not continue. Heart rate was monitored and recorded throughout the test using an electrocardiographic[5] recording of the CM-5 lead (36,74).

Maximum oxygen uptake was determined by the open-circuit Douglas bag method (25). The subject inspired through a two-way Daniels respiratory valve[6] which was connected to a four-way automated switching valve[7] by two feet of corrugated tubing (1 and 1/4 inch I.D.). Expiratory gases were collected continuously in neoprene weather balloons (44). Bags were changed every 30 seconds. There was no gas collection during recovery.

Expired gases were analyzed for percentages of CO_2 and O_2 using an infrared CO_2 analyzer (Applied Electrochemistry CD-3A[8]) and an electrochemical O_2 analyzer (Applied Electrochemistry S-3A[8]). The gas volumes were measured using a DTM-115[9] dry gas meter through which the gas was pumped at

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[5]Model VS4S, Cambridge Instrument Co.

[6]R-Pel Company, Los Altos, CA.

[7]Van Huss-Wells Automated Switching Valve.

[8]Applied Electrochemistry, Sunneyvale, CA.

[9]American Meter Co. (Singer).

a constant rate of 50 l/min. An inert gas, helium, was used for zeroing the analyzers. Calibration of the analyzers was conducted using room air and a known standard gas sample verified for CO_2 and O_2 with a Haldane Chemical Analyzer[10].

The following work capacity variables were determined: pulmonary ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}O_2$) and maximum oxygen uptake (Max $\dot{V}O_2$). These values were calculated by the equations of Consolazio, Johnson and Pecora (25).

Post-Test Analyses

Lipid and lipoprotein determinations were performed at the Laboratory of Clinical Medicine, Lansing, Michigan. Triglycerides and total cholesterol were determined enzymatically using a Technicon SMAC[11]. The SMAC uses a triglyceride specific lipase and protease to determine serum triglycerides (12) and uses a cholesterol oxidase for determination of serum cholesterol (2,88). The techniques have been almost perfectly correlated (r=.997) with the results obtained by the Lipid Standardization Laboratory, Center for Disease Control, Atlanta, Georgia (93) which uses an AutoAnalyzer II[11]. High density lipoprotein cholesterol was calculated using a DuPont Automatic Clinical Analyzer[12]. The procedure involved selective precipitation of LDL

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[10]Arthur H. Thomas Co., Philadelphia, PA.

[11]Technicon Instruments, Tarrytown, NY.

[12]DuPont Company, Wilmington, DE.

and VLDL from the serum separated supernate using sodium phosphotungstate and magnesium ion reagent at pH 5.7 (12). The HDL-cholesterol was analyzed enzymatically using cholesterol esterase, cholesterol oxidase and horseradish peroxidase (123). Results obtained by this method have been very highly correlated (r=.95) with those of the heparin-Mg^{2+} method employed by the Lipid Standardization Laboratory (93). Low density lipoprotein cholesterol was determined indirectly using the formula derived by Friedewald, Levy and Fredrickson (42).

A repeat sample was drawn from the subject if: a) triglycerides were greater than 140 mg/dl or b) total cholesterol was greater than 220 mg/dl and LDL-cholesterol was greater than 164 mg/dl (79). The results of the repeat sample replaced the results of the original sample for all lipid measures irrespective of the findings.

Diet diaries were coded for fifty-seven nutrients using the Michigan State University Nutrient Data Bank (107).

Statistical Analyses

The subjects in the two comparison groups were matched by age and by sex. However, subsequent pairwise correlations for each of the dependent variables yielded values which were not only statistically nonsignificant but trivial

(r=0.032 to 0.066). Consequently, the assumption of dependence was rejected, and, to maximize the power of the statistical analyzes, the scalar data were analyzed for group differences using one- and two-tailed Student t-tests for independent groups (62). A significance level of .05 was set for all analyses. The maximum acceptable probability of making a type II error was set at the .20 level. The reserve judgement region for the t-statistic calculated for the one-tailed test was $1.546<t<1.691$ and for the two-tailed test the calculated range was $1.972<|t|<2.032$. The actual probability of making a type II error (62) and the power of the t-test (62) were calculated for each t-value contained in the reserve judgement region. Group differences in the nominal data related to family history of premature cardiovascular disease were analyzed using two-sample Chi-square contingency tests with alpha set at .05. Group differences in the ordinal data related to activity intensity were analyzed using Mann-Whitney U-tests (62) with alpha set at .05. Correlations of the lipid variables with the remaining scalar variables were performed using Pearson product-moment coefficients of correlation (62). Correlations of the lipid variables with the family histories of premature cardiovascular disease were conducted using point-biserial correlation coefficients (62). Correlations of the ordinal intensity variables were performed using Spearman rank-order correlation coefficients

(62). Significance was set at the .05 level for all correlational analyses. Scattergrams of the correlations found to be significant were plotted to check linearity and homoscedasticity.

The raw data are stored in the Human Energy Research Laboratory, 3 IM Sports Circle, Michigan State University, East Lansing, MI 48824. Inquiries should be addressed to William W. Heusner, Ph.D.

CHAPTER IV

RESULTS AND DISCUSSION

The findings of this investigation are presented initially in terms of group differences (swimmers versus controls). The order of presentation is as follows: (a) chronological age, (b) activity history in organized sports, (c) family history of premature cardiovascular disease, (d) body composition, (e) basal physiological measures, (f) maximal physiological measures, (g) diet and (h) serum lipids and lipoproteins.

Correlational analyses were performed on the combined data of the swimmers and controls. The results of the Pearson, Spearman, and point-biserial correlations follow the group difference presentations. These presentations are in the order of: (a) HDL-cholesterol, (b) LDL-cholesterol, (c) total cholesterol to HDL-cholesterol, and (d) LDL-cholesterol to HDL-cholesterol. A discussion section which addresses both the intergroup differences and the combined group correlations follows the presentation of results.

Group Differences Results

Chronological Age

The mean ages and standard errors of both groups were 11.5 ± 0.2 years. The swimmers ranged in age from 9.5 to 12.9 years while the controls varied in age from 9.7 to 12.9 years.

Activity History in Organized Sports

The activity histories were organized into three categories for the swimmers: (a) organized activites other than swimming, (b) swimming activity and (c) total organized activities. Each category was compared to the total organized activities of the controls. Thirteen of the swimmers took part in organized sports other than swimming. These sports included basketball, cycling, soccer, golf, tennis, football, ballet, baseball, and gymnastics. Of the controls, only three children were not participating currently in organized sports. Activities the controls listed were basketball, soccer, football, gymnastics, cycling, wrestling, tennis, ballet, swimming, folk dance, track, softball, baseball, and karate. Comparisons of the average number of years of participation revealed no

difference between the two groups after comparing all three swimmers' categories (Table 4.1).

TABLE 4.1. Years of Participation in Organized Sporting Activity

	Mean	SEM	Range	P
Other Activity				
Swimmers	2.50	0.5	0 - 6	.802
Controls	2.67	0.4	0 - 6	
Total Activity				
Swimmers	3.22	0.4	1 - 7	.305
Controls	2.67	0.4	0 - 6	

Other Activity = Activities Other Than Swimming.

Figure 4.1, which shows exercise duration as determined in hours per year, reveals that the controls participate significantly (P=.019) more in their activities than the swimmers participate in organized activities other than swimming. The total number of hours that the swimmers devote to swimming is more than three times that which the controls spend in all their activities combined. This comparison, along with the total activity comparison between the groups shown in Figure 4.1, is statistically significant (P<.001).

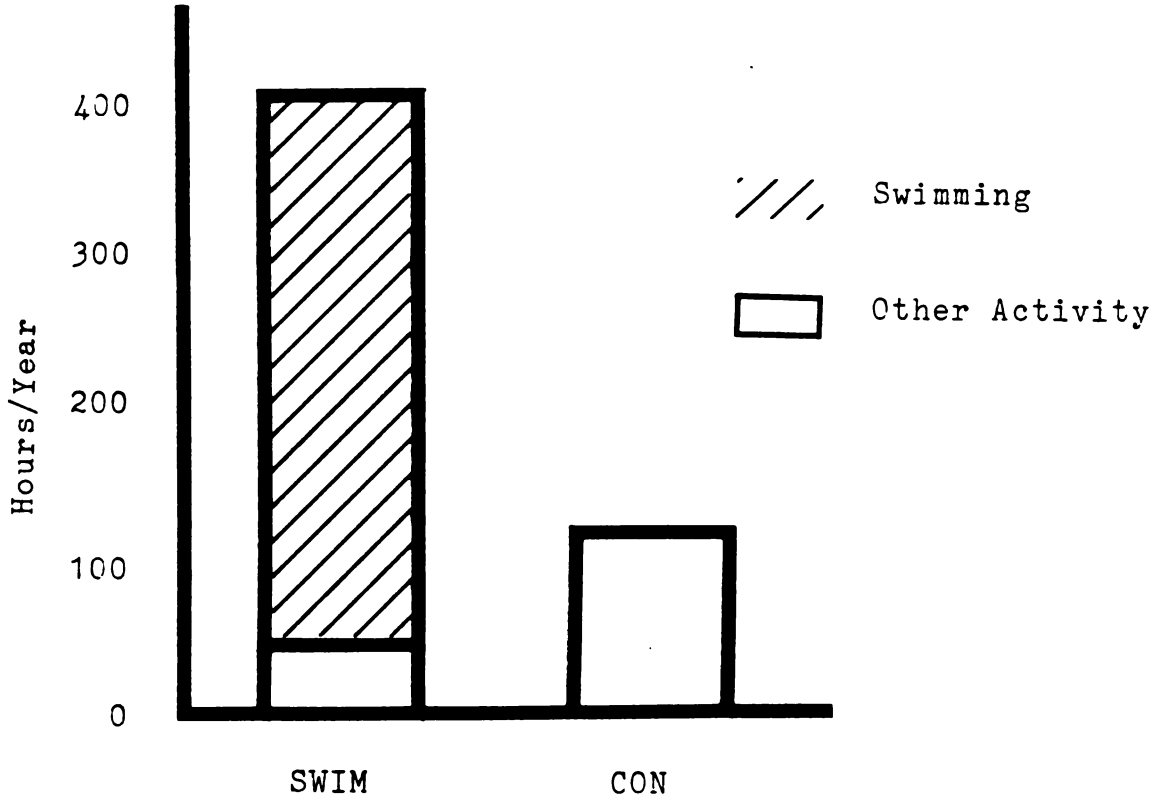

Figure 4.1. Exercise Duration Measured by Hours of Organized Sporting Activity For the Current Year.

In Figure 4.2a, it can be seen that the swimmers perceived their activities other than swimming to be about as intense as the controls' activities ($P>.05$). The perceived intensity of the average swimming workout, as shown in Figure 4.2b, was significantly ($P<.05$) higher than the average workout for the controls. Total perceived intensity comparisons revealed that the swimmers work at a significantly ($P<.05$) greater intensity in their activities than do the controls (Figure 4.2c).

Family History of Premature Cardiovascular Disease

Premature cardiovascular disease refers to the incidence of heart attack, stroke, hypertension, or diabetes mellitus occurring in a parent, grandparent, or sibling of the subject before the age of 60 years. There were no reports of any siblings having premature cardiovascular disease. Therefore, Figure 4.3 shows comparisons of the incidence of premature cardiovascular disease in just the parents and the grandparents between the two groups. No significant ($P>.05$) differences were seen in heart attacks (Figure 4.3a), strokes (Figure 4.3b), hypertension (Figure 4.3c) or diabetes mellitus (Figure 4.3d). The results must be judged cautiously because information known to be relative to the development of cardiovascular disease was

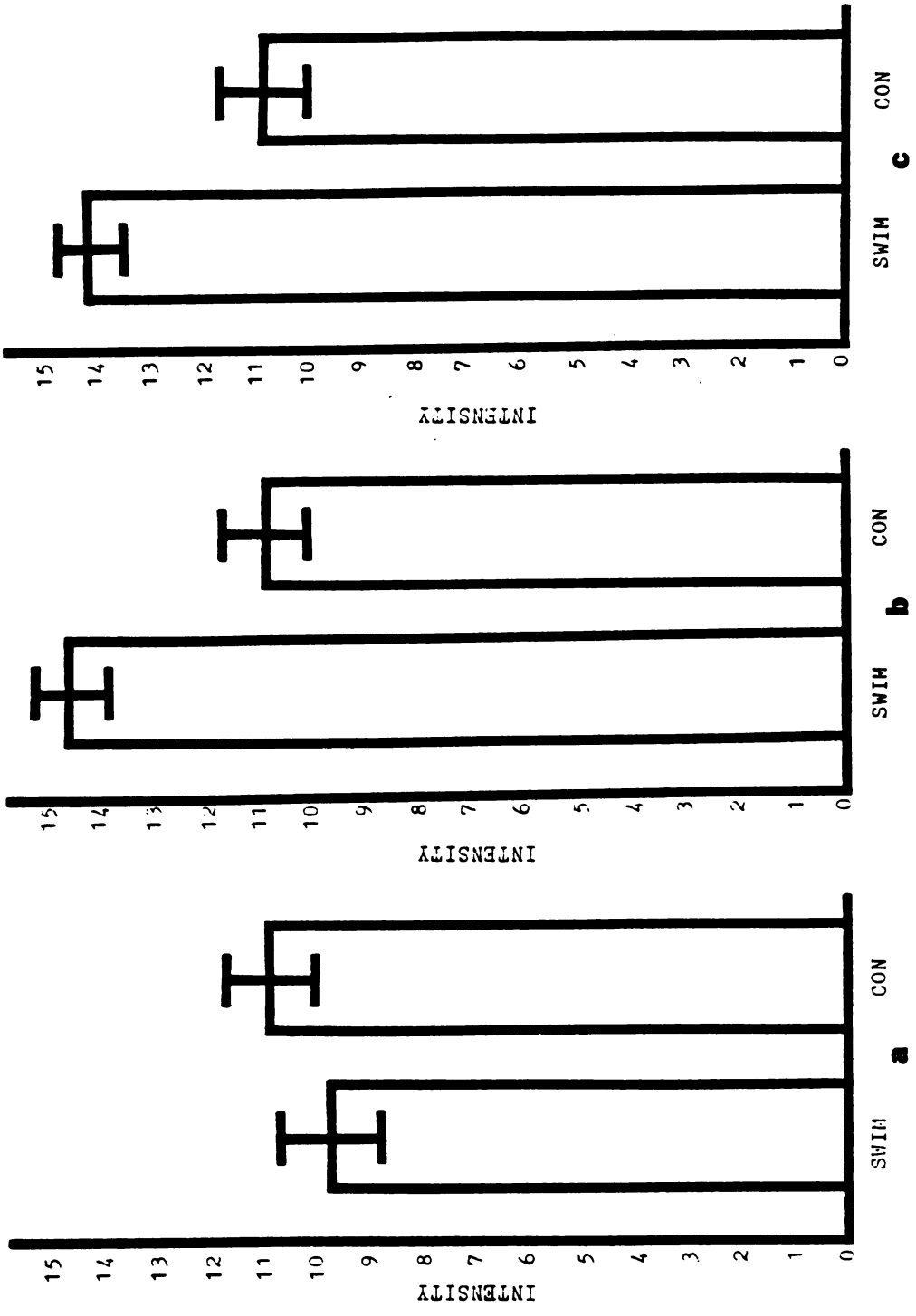

Figure 4.2. Perceived Exercise Intensity: (a) Other Activities Besides Swimming, (b) Swimming vs Controls' Total Activities, (c) Total Activity

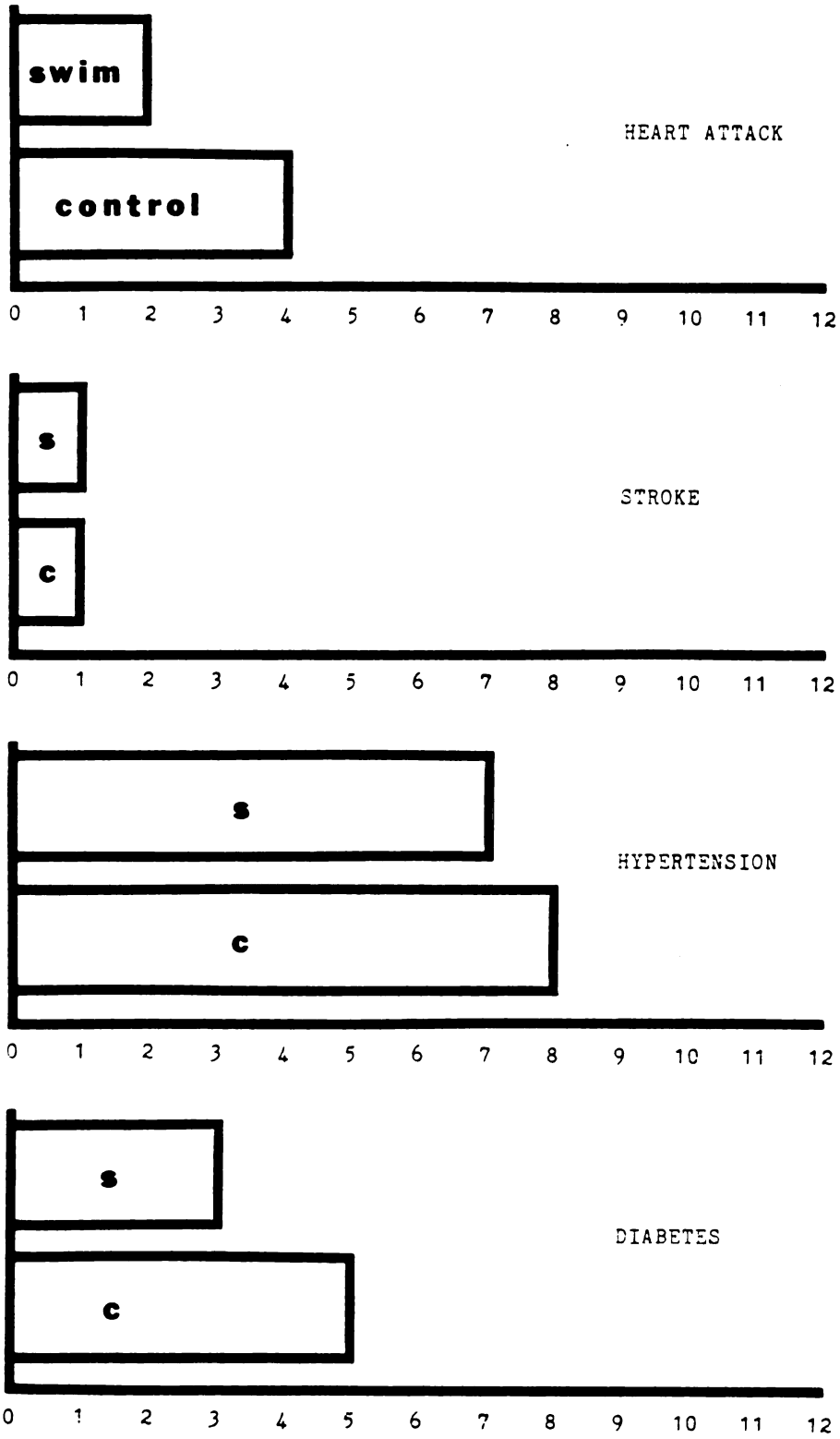

Figure 4.3. Families (Parents and Grandparents) Indicating Premature Cardiovascular Disease: (a) Heart Attack, (b) Stroke, (c) Hypertension, (d) Diabetes Mellitus.

not collected from the family members. Such data would have helped to determine whether the comparison groups came from equivalent families. Also, reliance of the accuracy and completeness of the data was placed on the available information and memories of the parents.

Body Composition

In Table 4.2, the statistcal results of standing height and body weight for the two subject groups are presented. There were no significant (P>.05) differences between the groups.

TABLE 4.2. Standing Height and Body Weight

	Mean	SEM	Range	P
Height (cm)				
Swimmers	149.0	1.8	137.0 - 163.2	.688
Controls	148.0	1.8	134.5 - 163.5	
Weight (kg)				
Swimmers	38.4	1.4	30.6 - 50.6	.803
Controls	37.9	1.5	27.7 - 50.8	

Table 4.3 shows the results of body fat percentage and body density calculated from hydrostatic weighing as well as body fat percentage calculated using Durnin's (35) skinfold technique. The swimmers exhibited a significantly (P=.031) lower percentage of fat than did the controls when hydrostatic weighing was used as the basis of comparison. There was no significant (P>.05) difference between groups in the present study using Durnin (35) skinfold measurements.

TABLE 4.3. Body Composition Results

	Mean	SEM	Range	P
Density (g/ml)				
Swimmers	1.054	.002	1.037 - 1.073	.053
Controls	1.048	.003	1.023 - 1.062	
Percent Fat (Hydro)				
Swimmers	19.8	1.0	11.2 - 27.6	.031
Controls	22.8	1.2	16.1 - 33.9	
Percent Fat (Durnin)				
Swimmers	18.5	1.2	10.8 - 28.1	.160
Controls	20.1	1.0	12.8 - 27.8	

Hydro = Hydrostatic Weighing

Durnin = Durnin Skinfolds

The t-value associated with the body density results (P=.053) falls in the reserve judgement region. The power of the statistical analysis was calculated to be .723. Since the percentage of fat calculated from hydrostatic weighing is a linear transformation of body density, rounding error may be assumed to be the cause for the difference in significance between percentage of fat obtained from hydrostatic weighing and body density.

Resting Physiological Measures

The results of the resting physiological measures are shown in Table 4.4. The swimmers displayed significantly (P=.035) higher resting systolic blood pressures than the controls. No significant (P>.05) group difference was observed in terms of resting diastolic blood pressure. All blood pressure means were in the normal range for this age distribution (92).

There was no significant (P>.05) difference in resting heart rate between the groups.

Maximal Physiological Measures

Table 4.5 contains the results for maximal heart rate and maximal oxygen uptake. The results indicate that swimmers had significantly (P=.009) higher maximal heart rates. In terms of maximal oxygen uptake, the swimmers exhibited a significantly (P<.002) higher $\dot{V}O_2$ max.

TABLE 4.4 Resting Blood Pressures and Heart Rates

	Mean	SEM	Range	P
Systolic BP (mm)				
Swimmers	99.2	1.4	86 - 109	.035
Controls	93.6	2.3	81 - 104	
Diastolic BP (mm)				
Swimmers	67.4	1.6	63 - 82	.103
Controls	62.9	2.3	51 - 76	
Heart Rate (beats/min)				
Swimmers	79.1	2.6	62 - 108	.078
Controls	85.1	3.2	64 - 108	

TABLE 4.5. Maximal Heart Rates and Oxygen Uptakes

	Mean	SEM	Range	P
Heart Rate (beats/min)				
Swimmers	216.3	2.0	200 - 232	.009
Controls	205.9	3.2	163 - 215	
$\dot{V}O_2$ (ml/kg/min)				
Swimmers	52.9	1.5	40.6 - 63.9	.002
Controls	45.5	1.8	33.8 - 60.3	

$\dot{V}O_2$ = Maximal Oxygen Uptake

Diet

The caloric results of the dietary investigation are located in Table 4.6. The dietary fat ratios and dietary cholesterol are presented in Table 4.7.

The t-value for caloric intake falls in the reserve judgement region (P=.062). The power of the t-test was .705. The percentages of calories divided into fats, proteins, and carbohydrates were virtually identical between the two groups. The breakdown of 35% fat to 14% protein to 51% carbohydrate was more favorable than the American average of 40 to 15 to 45 (23).

The swimmers ingested slightly more cholesterol than the controls but the difference was not significant

TABLE 4.6. Dietary Caloric Results

	Mean	SEM	Range	P
Caloric Intake				
Swimmers	2288	123	1549 - 3218	.062
Controls	1988	145	564 - 3552	
Percent Protein				
Swimmers	14.0	0.5	10.9 - 18.9	.304
Controls	14.8	0.6	10.2 - 19.6	
Percent Fat				
Swimmers	36.2	1.1	10.6 - 46.0	.403
Controls	34.8	1.4	29.4 - 46.7	
Percent Carbohydrate				
Swimmers	51.2	1.2	37.8 - 58.2	.966
Controls	51.2	1.3	40.6 - 60.6	

TABLE 4.7. Dietary Cholesterol and Dietary Fat Ratio

	Mean	SEM	Range	P
Cholesterol (mg)				
Swimmers	256.4	37.2	120.7 - 821.4	.466
Controls	219.8	32.8	47.4 - 716.0	
Poly/Sat Fat				
Swimmers	0.42	0.04	0.19 - 0.82	.787
Controls	0.40	0.04	0.15 - 0.69	

Poly/Sat Fat = Polyunsaturated Fat vs. Saturated Fat

(P=.466). For the ratio of polyunsaturated fat to saturated fat, there were nearly identical values of 0.4 for each group. A dietary fat intake ratio of 0.4 equals the American norm (23), but is substantially lower than the American Heart Association's recommended ratio of 1.0 to 1.1 (23).

Serum Lipids and Lipoproteins

The results related to serum lipids are presented in Table 4.8, while the results of the lipoprotein cholesterol analyses and the corresponding ratios are exhibited in Table 4.9.

There was no significant (P>.05) difference in serum triglycerides between the two groups. In fact, the means were nearly identical. The controls exhibited a slightly higher mean total cholesterol value than did the swimmers, but the difference was insignificant (P>.05) (Table 4.8).

In terms of HDL-cholesterol, the swimmers displayed a higher mean concentration than did the controls. The P-value was .051 which places the results in the reserve judgement region. The calculated power of the t-test was .749. The t-value for LDL-cholesterol is located in the reserve judgement region too. Swimmers exhibited a somewhat lower (P=.054) concentration of LDL-cholesterol than did the controls. The calculated power of the t-test was .738.

TABLE 4.8. Serum Lipid Results

	Mean	SEM	Range	P
Cholesterol (mg/dl)				
Swimmers	173.2	7.0	134 - 248	.325
Controls	182.8	6.7	135 - 240	
Triglycerides (mg/dl)				
Swimmers	71.0	6.3	44 - 136	.986
Controls	70.8	6.7	40 - 131	

TABLE 4.9. Lipoprotein Cholesterol and Ratios

	Mean	SEM	Range	P
HDL-C (mg/dl)				
Swimmers	64.6	2.7	48 - 90	.051
Controls	58.4	2.5	38 - 79	
LDL-C (mg/dl)				
Swimmers	93.0	6.1	62.0 - 157.6	.054
Controls	107.8	6.6	65.0 - 160.8	
LDL-C/HDL-C				
Swimmers	1.47	.10	0.82 - 2.43	.009
Controls	1.91	.15	0.94 - 3.23	
Cholesterol/HDL-C				
Swimmers	2.69	.12	1.97 - 4.00	.013
Controls	3.18	.17	2.06 - 4.74	

HDL-C - High Density Lipoprotein Cholesterol

LDL-C - Low Density Lipoprotein Cholesterol

Cholesterol - Total Serum Cholesterol

The redistribution of HDL-cholesterol and LDL-cholesterol in the swimmers results in significantly lower ratios of LDL-cholesterol to HDL-cholesterol (P=.009) and total cholesterol to HDL-cholesterol (P=.013).

Correlations Results

Correlations were performed on those lipid variables, which were significantly different between groups or had t-values located in the reserve judgement region, against the non-lipid variables. The data from both comparison groups were combined for the correlation analyses. The correlation technique employed depended on the type of data to be analyzed.

HDL-Cholesterol

Table 4.10 shows the non-lipid variables which were significantly correlated with HDL-cholesterol. A negative association (P=.040) between adiposity determined by hydrostatic weighing and HDL-cholesterol can be seen. Positive correlations of HDL-cholesterol were observed with perceived swimming intensity (P=.047) and total activity duration (P=.050). Of the physiological variables

TABLE 4.10 Significant Correlations with HDL-Cholesterol

	r	P
Perceived Swimming Intensity	.306	.047
Percent Fat (Hydro)	-.296	.040
Total Activity Duration	.279	.050

Hydro - Hydrostatic Weighing

TABLE 4.11 Selected Non-Significant Correlations with HDL-Cholesterol

	r	P
Maximal Oxygen Uptake	.034	.423
Resting Systolic BP	-.187	.153
Resting Diastolic BP	-.221	.112

BP - Blood Pressure

measured, none showed a significant (P<.05) correlation with HDL-cholesterol. The correlations of HDL-cholesterol with maximal oxygen uptake, and the resting determinations of systolic and diastolic blood pressure are presented in Table 4.11.

LDL-Cholesterol

Variables from the categories of family history, diet, physiological measures and exercise duration correlated significantly (P<.05) with LDL-cholesterol (Table 4.12). Family history of hypertension had the strongest correlation with LDL-cholesterol and was the only family history variable which was significantly (P=.001) correlated. Dietary cholesterol intake (P=.030) and resting heart rate were both positively correlated with LDL-cholesterol.

All three exercise duration determinations, as measured by hours of activity for the current year, were negatively associated with LDL-cholesterol. Swimming activity showed the strongest correlation (P=.032) while activities other than swimming gave the weakest correlation (P=.043).

Total Cholesterol to HDL-Cholesterol

Two measures of exercise duration, total activity time and the swimmers' swimming time with the controls' total activity time, exhibited negative correlations with the cholesterol ratio of total cholesterol to HDL-cholesterol (Table 4.13). A significant (P=.037) correlation was observed with resting heart rate too.

LDL-Cholesterol to HDL-Cholesterol

Table 4.14 shows the significant correlations with the ratio of LDL-cholesterol to HDL-cholesterol. Significant, negative associations of the LDL-cholesterol to HDL-cholesterol ratio were seen with both total activity (P=.005) and swimming activity (P=.006). Resting heart rate exhibited a significant (P=.037) positive correlation.

TABLE 4.12 Significant Correlations with LDL-Cholesterol

	r	P
Family History of Hypertension	.439	.001
Dietary Cholesterol	.316	.030
Resting Heart Rate	.303	.046
Swimming Activity	-.313	.032
Total Activity	-.299	.038
Other Activity	-.290	.043

TABLE 4.13 Significant Correlations with the Ratio of Total Cholesterol to HDL-Cholesterol

	r	P
Total Activity	-.407	.007
Swimming Activity	-.387	.010
Resting Heart Rate	.320	.037

TABLE 4.14 Significant Correlations with the Ratio of LDL-Cholesterol to HDL-Cholesterol

	r	P
Total Activity	-.426	.005
Swimming Activity	-.417	.006
Resting Heart Rate	.336	.030

Group Differences Discussion

The absence of any group differences in family history for premature cardiovascular disease, diet, standing height, and body weight supports the assumption that the two comparison groups are relatively equivalent in most of the factors believed to elicit chronic changes in serum lipids and lipoproteins.

In this investigation, significant ($P<.05$) group differences were observed in the ratios of LDL-cholesterol to HDL-cholesterol and total cholesterol to HDL-cholesterol with the swimmers exhibiting a more favorable lipid profile for lowering coronary risk. These ratios confirm the findings of Smith *et al.* (138) who studied female collegiate swimmers. Pediatric studies by Thorland and Gilliam (150) using physical activity, Gilliam and Burke (48) using running, and Smith *et al.* (136) using running revealed

similar lipid results.

The current findings appear to contradict those of Zonderland et al. (161) who studied youth-aged club swimmers. A possible explanation for why Zonderland did not detect lower serum lipid ratios for the club swimmers could be that the club swimmers trained only about 50% as much as the swimmers in the current study. In the two studies (136,150) which investigated highly active or trained children and found lowered lipid ratios, the volume of activity or training was approximately equivalent to that of the swimmers in this study.

The current lipid ratios take on greater meaning when the comparison groups in previous studies are examined. In the current study, a non-elite but trained group of young swimmers was compared to an age- and sex-matched group of active controls. Only Smith et al. (136) previously used an active control group for comparison. The other retrospective studies (150,161) of the effects of exercise on serum lipids in children used relatively sedentary control groups for comparison. The use of less active comparison group should increase the probability of detecting significant group differences. Smith et al. (136) studied an elite group of young athletes which could increase the probability of detecting group differences due to possible self-selection factors.

In terms of triglycerides, most studies

(59,60,85,106,136,158) have shown aerobically trained individuals to have lower serum triglycerides than their control counterparts. However, there have been exceptions (39,94,117,125). Considering that elite age-group runners (136) exhibited a low mean value for triglycerides of 68.3 mg/dl, an activity threshold for eliciting a lowering in serum triglycerides may have been met by a majority of both swimmers and controls in this study.

The lack of a difference in serum cholesterol supports previous findings by Smith et al. (136), Nizankowska-Blaz and Abramowicz (114), and Linder, DuRant and Mahoney (91). The current results further support the claim of Smith et al. (136) that serum total cholesterol is a futile method for screening lipid extremes in children.

Most previous investigations of serum lipids in children did not determine body composition (48,91,161) or did not use hydrostatic weighing to determine body fat percentage (150). Smith et al. determined percentage of body fat when studying elite age-group runners but found a larger group difference than was seen in the current study. Part of the difference between the two studies could be due to runners supporting their own weight during running, while swimmers can use water buoyancy to support their weight during swimming (136).

Thorland and Gilliam (150) found no difference in the percentage of body fat in their study of physically active

children who did not differ in body weight. An explanation for why Thorland and Gilliam`s results seem to contradict the present findings can be found by examining their procedure. The percentage of fat estimates in Thorland and Gilliam's (150) investigation were obtained using skinfold measurements. Using Durnin (35) skinfold measurements in the current investigation, no group difference in percentage body fat was observed indicating that for this age group, hydrostatic weighing and Durnin (35) skinfold techniques do not measure the same thing. Therefore, one can only speculate whether a true difference in adiposity occurs between groups in the current study.

The resting physiological measures of heart rate and blood pressure in the current study displayed unusual patterns as compared with the typical adaptations in aerobically-trained individuals. Aerobic training commonly is believed to lower resting heart rate in adults (6) and this phenomenon has been demonstrated in elite age-group runners (136). However, swimming studies by Robinson (127) and Caffrey (15) of youngsters in the same age range as the ones in the current study show no difference in resting heart rate between trained swimmers and controls. These observations support the current findings.

The systolic blood pressure adaptation of the swimmers is the reverse of what aerobic exercise training is believed to cause. However, Faulkner (40) mentions this

swimming-related phenomenon and the current findings are supported by the work of Giese (47) on pre-pubescent swimmers. Holmer (64) states that arterial blood pressures are higher during swimming than running and that this is due possibly to the hydrostatic force and increased peripheral vascular resistance incurred from being in the water.

Giese (47) found no difference in diastolic blood pressure between pre-pubertal swimmers and controls of equivalent ages. The current findings confirm this result. Why the pattern seen with the systolic blood pressure measures does not occur with diastolic blood pressure could be because the range for diastolic blood pressure is less than for systolic blood pressure and there is more difficulty in pinpointing diastolic blood pressure as compared to systolic blood pressure.

Astrand and Rodahl (6) have stated that no difference in maximal heart rate exists between trained and nontrained adults. Caffrey (15) and Robinson (127) found the same lack of a training effect in observing age-group swimmers between 10 and 16 years of age. These results are contrary to the significant (P=.009) difference obtained in the present study. However, Astrand and Rodahl (6) have said that a 10 beat/min deviation is common in the determination of maximal heart rate. Bar-Or (6) has stated that the normal range for maximal heart rate for children is between 195 and 215 beats/min. Therefore, the difference observed in this study

may or may not represent a real physiological phenomemon. If one assumes the observed difference is not a measurement artifact, a possible explanation for the high maximal heart rates in the swimmers can be hypothesized from the work of Andrew et al. (4). In their study of heart and lung functions of young swimmers over a three-year period, the authors found no difference in stroke volume between swimmers and controls and no difference in heart rate for a given oxygen uptake in girls (4). In the current study, a possible lack of difference in stroke volume and heart rate for a given oxygen uptake could explain the need for higher maximal heart rates in the swimmers who must meet cardiac output requirements necessary for high maximum oxygen uptakes.

The difference between the swimmers and the controls in terms of maximal oxygen uptake is not surprising considering the volume of aerobic training that is routinely imposed upon the swimmers. The swimmers' $\dot{V}O_2$ max of 52.9 ml/kg/min compares quite closely with the values for child swimmers of both sexes reported by Cunningham and Eynon (27) at 49.7 ml/kg/min, by Robinson (127) and Caffrey (15) at 53.0 ml/kg/min, by Kramer and Lurie (78) for girls only at 49.5 ml/kg/min, and by Astrand et al. (5) for girls only at 51.5 ml/kg/min.

Correlations Discussion

Exercise duration, as measured by hours per week of participation in organized sports, was the only variable studied that was associated with each of the selected lipid variables. Interestingly, maximal oxygen uptake displayed no association with any of the lipid variables shown to exhibit group differences. Since epidemiological studies involving large numbers of subjects usually do not include maximal oxygen uptake for reasons of practicality, there are almost no references comparing maximal oxygen uptake and serum lipids. Smith et al. (136) found a modest positive correlation between HDL-cholesterol and maximal oxygen uptake (r=.384). Why this pattern was not seen in the current study is unknown. However, self-selection as an extraneous factor, could not be controlled in either study.

In the Lipid Research Clinics' Prevalence Study conducted by Haskell et al. (61), no correlation of HDL-cholesterol with treadmill exercise test performance was shown. However, a significant correlation of reported activity with HDL-cholesterol was found (61). Other investigators (60,86) have shown positive correlations between HDL-cholesterol and chronic activity.

Variables that are influenced by chronic exercise, such as resting heart rate and percentage of fat, are

correlated with some of the lipid variables. This appears to be physiologically consistent with the association between exercise and serum lipid changes. However, it must be mentioned that heredity should be considered as a possible influencing factor on both resting heart rate and percentage of body fat.

LDL-cholesterol appears to be correlated with more unique variables, e.g., diet, family history, and exercise, than any of the other lipid measures. The positive association between dietary cholesterol and LDL-cholesterol supports the belief of Conner and Conner (23) that dietary cholesterol has a stronger influence on LDL-cholesterol than on HDL-cholesterol.

Hypertension was the only family history variable related to premature cardiovascular disease to correlate with any of the lipid variables. This is consistent with our observation that LDL-cholesterol appears to be influenced by more risk factors than HDL-cholesterol.

CHAPTER V

SUMMARY, CONCLUSIONS, and RECOMMENDATIONS

Summary

The purpose of this retrospective study was to investigate the relationships of exercise duration and intensity, adiposity, diet, blood pressure, and work capacity to serum lipids and lipoproteins in pre-teenage swimmers and control subjects of the same age and sex. Eighteen competitive swimmers from the Lansing, Michigan area who ranged in age from 9 years to 12 years were compared to eighteen active control youngsters selected from the Motor Performance Study conducted by the Michigan State University Department of Health and Physical Education.

Methods

All of the subjects and their parents were informed of the aims and the possible risks of participation in the study, and each subject and his/her parent(s) gave their written consent.

Pre-test questionnaires for activity history and family history of cardiovascular disease were collected. A three-day dietary diary was required of each subject to determine the child's normal dietary regimen.

Laboratory evaluations began with a venous blood sample drawn after an overnight fast and hiatus from activity. The blood sample was enzymatically analyzed for serum trigylcerides (12), serum cholesterol (2,88), and HDL-cholesterol (12,88). LDL-cholesterol was determined indirectly (42).

The subjects were all physically examined by a pediatric cardiologist who determined the subjects' resting blood pressures and resting heart rates. Anthropometric evaluation followed, which involved skinfold measurements of the biceps, triceps, suprailiac, and subscapular skinfold sites as well as determinations of body weight and standing height. Adiposity was assessed with a closed-circuit hydrostatic weighing apparatus (14,134) which uses oxygen rebreathing for determination of the underwater lung volume (96,122).

The final laboratory evaluation was the determination of work capacity using a continuous treadmill run to exhaustion. The test protocol consisted of a fixed treadmill speed of five miles per hour beginning initially at 0% grade. At the end of each minute, the treadmill grade was increased by one percent. The subject was monitored continuously with an electrocardiograph. Maximum oxygen uptake was determined by the open-circuit Douglas bag method (25).There was no gas collection during recovery.

Group differences for exercise duration, adiposity, diet, work capacity, anthropometry, basal physiological measures of heart rate and blood pressure, and serum lipids and lipoproteins were analyzed using one-tailed and two-tailed Student t-tests for independent groups (62) with significance set at the .05 level. A reserve judgement region was calculated since a sample size that was less than necessary and sufficient was collected. Group differences of family history of premature cardiovascular disease were evaluated using two-sample Chi-square contingency tests with alpha set at .05. Group differences of activity intensity were determined using Mann-Whitney U-tests (62) with alpha set at .05.

Correlations of HDL-cholesterol, LDL-cholesterol, total cholesterol to HDL-cholesterol, and LDL-cholesterol to HDL-cholesterol with the remaining scalar variables were performed using Pearson product-moment correlation

coefficients (62) with significance set at the .05 level. Point-biserial correlations (62) were used to compare the aforementioned lipid variables to the family histories for premature cardiovascular disease. Spearman rank-order correlations (62) were performed on the activity intensites to determine the strength of association with the aforementioned lipid variables.

Results

No statistically significant ($P<.05$) differences were observed in chronological age; height; weight; resting diastolic blood pressure; resting heart rate; serum triglycerides; total cholesterol; family histories of premature heart attack, stroke, hypertension, and diabetes mellitus; percentage of caloric intake represented in terms of proteins, fats, and carbohydrates; dietary cholesterol ingested; polyunsaturated fat to saturated fat ratio; years of participation in organized sporting activities other than swimming; years of participation in all organized sporting activities; or perceived intensity of organized sporting activities other than swimming.

The group differences of HDL-cholesterol ($P=.051$) and LDL-cholesterol ($P=.054$) were located in the reserve judgement region. However, significantly lower ratios of

LDL-cholesterol to HDL-cholesterol (P=.009) and total cholesterol to HDL-cholesterol (P=.013) were seen for the swimmers.

At rest, the swimmers displayed significantly (P=.035) increased systolic blood pressures. Adiposity determinations revealed a significant (P=.031) group difference using hydrostatic weighing, while no group difference was seen using Durnin (35) skinfold measurements. Treadmill testing revealed significantly higher (P=.002) maximal oxygen uptakes for the swimmers along with significantly (P=.009) higher maximal heart rates.

Inspection of exercise duration as measured by activity history, shows that controls participated in a significantly (P=.019) greater number of organized sporting activities than did the swimmers. However, measured in hours per year, the swimmers took part in age-group swimming more than three times (P=.001) as much as the controls participated in all of their activities combined. A comparison of total activity revealed that swimmers took part in significantly (P=.001) more activity than did the controls.

Correlational analyses for HDL-cholesterol showed significant relationships with swimming intensity (r=.306,P=.046), adiposity determined by hydrostatic weighing (r= -.296,P=.04), and total activity time (r=.279,P=.05). Significant correlations with LDL-cholesterol were seen with family history of premature

hypertension (r=.439,P=.01), dietary cholesterol (r=.316,P=.03), resting heart rate (r=.303,P=.046), and all three exercise duration comparisons: other activity (r= -.313,P=.043), swimming activity (r= -.299,P=.032), and total activity (r= -.290,P=.038). Both the ratios of LDL-cholesterol to HDL-cholesterol and total cholesterol to HDL-cholesterol had significant associations with total activity (r= -.426,P=.005) (r= -.407,P=.007), swimming activity (r= -.417,P=.006) (r= -.387,P=.01), and resting heart rate (r=.336,P=.03) (r=.320,P=.037).

Conclusions

1. There was no difference in the concentration of total cholesterol between age-group swimmers and active controls ranging in age from 9 to 12 years.

2. Definite conclusions cannot be drawn from the current investigation concerning the differences in the HDL-cholesterol and LDL-cholesterol fractions between the two groups. Judgement should be reserved until additional data can be obtained.

3. Age-group swimmers between the ages of 9 and 12 years had significantly (P<.05) higher maximal oxygen uptakes than did equivalently aged active controls.

4. There was no meaningful relationship between maximum oxygen uptake and HDL-cholesterol combining subjects in the two groups.

5. There was a significant ($P<.05$) difference in adiposity between the groups as determined by hydrostatic weighing but there was no difference in adiposity as determined using Durnin (35) skinfold measurements.

6. There was a negative relationship between adiposity determined by hydrostatic weighing and HDL-cholesterol combining the comparison groups but there was no relationship between adiposity determined with Durnin (35) skinfold measurements and HDL-cholesterol across the comparison groups.

7. There were no differences in the percentage contributions of caloric intake from proteins, fats, and carbohydrates between the two comparison groups.

8. There were no differences in the ratio of polyunsaturated fat to saturated fat or in the amount of dietary cholesterol ingested between the two groups.

9. Age-group swimmers ranging in age from 9 to 12 years had higher resting systolic blood pressures than active children of equivalent ages, but there was no difference between groups in terms of resting diastolic blood pressure.

10. There was no relationship between resting blood pressure and HDL-cholesterol pooling age-group swimmers and active controls ranging in age from 9 to 12 years.

11. The use of total cholesterol and/or triglycerides as determinants of coronary artery disease risk are unjustified in age-group swimmers and active controls from 9 to 12 years of age.

12. There was a modest positive relationship between exercise duration and the ratios of LDL-cholesterol to HDL-cholesterol and total cholesterol to HDL-cholesterol pooling age-group swimmers and active controls between 9 and 12 years of age.

13. There were significant ($P<.05$) differences in the ratios of total cholesterol to HDL-cholesterol and LDL-cholesterol to HDL-cholesterol between the two groups.

Recommendations

1. A larger sample size should be used in future studies to avoid limitations related to statistical analyses.

2. Family history should be carefully examined in a large, epidemiological study to determine relationships with the various lipid variables.

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APPENDICES

APPENDIX A

ACTIVITY HISTORY QUESTIONNAIRE

Name _____

Today's Date _____

Date of Birth _____

ACTIVITY HISTORY

A. Have you participated in organized sports within the last year? Yes or No If so, please list the primary sport you participated in, the number of years you have been participating, the number of weeks per year in which you participated per week (both practice and game competition) and the amount of time spent at each workout session. Also, circle the intensity of the average workout.

Sport	Total Yrs	Yr Began	Wks/Yr	Days/Wk	Min/Workout

Intensity of Average Workout 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20

mild average heavy

B. Have you ever participated in any other organized sports? If so, please list the sport, the number of years you have participated, the number of weeks per year in which you participated per week (both practice and game competition) and the amount of time spent at each workout session. Also, circle the intensity of the average workout.

Sport	Total Yrs	Yr Began	Wks/Yr	Days/Wk	Min/Workout

Intensity 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20

mild average heavy

Sport	Total Yrs	Yr Began	Wks/Yr	Days/Wk	Min/Workout
________	_____	______	____	_____	_______

Intensity 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
mild average heavy

Sport	Total Yrs	Yr Began	Wks/Yr	Days/Wk	Min/Workout
________	_____	______	____	_____	_______

Intensity 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
mild average heavy

Sport	Total Yrs	Yr Began	Wks/Yr	Days/Wk	Min/Workout
________	_____	______	____	_____	_______

Intensity 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
mild average heavy

C. For each sport listed in A and/or B, please give your coach's name and phone number if possible.

Sport	Coach's Name	Phone Number
1.		
2.		
3.		
4.		
5.		

D. Please think about your present level of physical conditioning. Are you in good shape? How good? Pretend that you have just accepted an invitation to swim in an international championship meet exactly two weeks from today. Assume that you will be entered in only one event, the one in which you swam the single most outstanding time in your life. Assume further that you have nothing to do in the next two weeks but eat, sleep and get ready for the meet. Finally. assume that your favorite coach will do everything possible to prepare you for the event. Please check the statement below which <u>best</u> describes how you feel you would do in the event two weeks from today:

___ a. **I believe my present level of conditioning is good enough so that my time two weeks from now could be faster than my best previous time.**

___ b. **I believe my present level of conditioning is good enough so that my time two weeks from now could be equal to my best previous time.**

___ c. **I believe my present level of conditioning is not as good as it might be so that my time two weeks from now would be slower than my best previous time.**

APPENDIX B

DIET DIARY WORKSHEET AND INSTRUCTIONS

FORM FOR TWENTY-FOUR HOUR DIET RECALL OR FOOD DIARY

Is this a typical diet? ____ yes ____ no

If not, what makes it different? _____

_____

Subject _____

Date of Interview _____

Interviewee/s _____

Date _____

Food	Amount	How Prepared	Where consumed
Breakfast			
Snack			
Noon			
Snack			
Evening Meal			
Snack			

Subject: _____

Ingredients in Nutrient Supplement

Name	Chemical formula	Quantity of Nutrient

DIRECTIONS FOR COMPLETING FOOD DIARY

General:

Identify food by name.

List the amount eaten in cups, ounces, servings, bottles, etc.
Under preparation, describe how product was made or identify by brand name.
Record whether eaten at home, at school, away from home or not at all.
List the time of day the food was eaten for each snack or meal.
Record whether or not vitamins or minerals were taken on the back of the sheet. Record by brand name and number of pills taken.

Specific:

Breakfast:

Cereals

List cereals by brand name and amount in cups or ounces. Indicate whether or not sugar was added. Record by "teaspoon". Do the same for brown sugar or honey. Indicate how much milk was added. Indicate whether milk was whole, 2% or skimmed.

Breads, pancakes, rolls, donuts

Indicate whether bread was white or whole wheat. Indicate if toasted and the number of slices.
Record butter if added and brand name of margarine if used on bread.
Record pancakes or waffles by brand name and size.
Indicate syrup, jelly, margarine/butter if used.
Record roll/donut size, whether it is frosted (chocolate, caramel, white). For donuts indicate if cake or glazed.
Indicate if rolls have nuts or raisins

Snacks:

Record amounts and products by brand name. Indicate if raw fruits are small, medium or large. If cereal is eaten, record as described for breakfast.

Lunch and Dinner:

Indicate recipe contents for mixed dishes.
Record fast foods eaten at home or away from home by brand name such as McDonalds, Pizza Hut, etc.
Record pop, Kool-Aid, etc. as regular or sugar free, or caffeine free if cola beverage.
For sandwiches, describe the bread and filling. Include butter, mustard/catsup, mayonnaise. tartar sauce if used.
Record fruits and vegetables as canned, frozen or fresh and amounts in cups or number such as 2 canned peach halves.
Describe salads. Indicate kind of dressing and brand name. If no salad dressing is used, please indicate "No dressing".

Cakes and Cookies

List kind such as chocolate cake with chocolate icing or chocolate chip cookies.

If not home made, indicate brand name under "preparation" on food diary form.

APPENDIX C

MEDICAL HISTORY QUESTIONNAIRE

MICHIGAN STATE UNIVERSITY

Center for Elite Athletes

Pretest Medical Screen

Name:_____ Date:___/___/___

Address:_____

_____

Phone:_____

Sex:_____

Birthdate:___/___/___

Medical History

Please answer the following questions as carefully as possible. If you answer yes to any question, please give an explanation in the space provided.

	No	Yes	Explanation
1. Are you taking any medication?	____	____	
2. Are you allergic to any food or medication?	____	____	
3. Do you wear glasses or contact lenses?	____	____	
4. Do you smoke - how much per day?	____	____	
5. Have you ever had an operation?	____	____	
6. Have you ever been hospitalized?	____	____	
7. Have you ever had an accident or injury that required medical attention?	____	____	
8. Have you ever been refused permission to participate in an athletic event for medical reasons?	____	____	

Please indicate below if you or any member of your family have suffered from any of the following conditions.

	Yourself			Family Member	
	No	Yes	Never Examined	No	Yes
Heart murmur	____	____	____	____	____
* Other heart problems	____	____	____	____	____
* High blood pressure	____	____	____	____	____
Rheumatic fever	____	____	____	____	____
Chest pain or discomfort	____	____	____	____	____
Shortness of breath	____	____	____	____	____
Asthma	____	____	____	____	____
Coughing upon exertion	____	____	____	____	____
* Diabetes mellitus	____	____	____	____	____
Epilepsy	____	____	____	____	____
Fainting or dizziness	____	____	____	____	____
Passing blood in urine	____	____	____		
Abdominal pain	____	____	____		
Severe headaches	____	____	____		
Back injuries	____	____	____		
Bone problems	____	____	____		
Joint problems - Knee	____	____	____		
Ankle	____	____	____		
Hip	____	____	____		
Shoulder	____	____	____		
Elbow	____	____	____		

* If you answered yes to any of these, please answer on the next page

_____
Signature of parent or guardian

Please fill out the entire page. Family members considered should include parents, siblings, grandparents, and subject.

Heart Attack

Family Member	Age at Occurence(s)	Death Resulted (yes or no)

Stroke

Family Member	Age at Occurence(s)	Death Resulted (yes or no)

High Blood Pressure

Family Member	Age at Occurence

Hyperlipidemia

Family Member	Age at Occurence	Type

Diabetes Mellitus

Family Member	Age at Occurence

APPENDIX D

HYDROSTATIC WEIGHING WORKSHEET

MICHIGAN STATE UNIVERSITY -- CENTER FOR THE STUDY OF HUMAN PERFORMANCE

Underwater Weighing Calculations

Subject (name) _____ No. _____ Study _____

Age _____yr/mo Date of Birth ___/___/___ Height _____cm Time of Day _____am/pm Date ___/___/___

Turn HP-67 ON and set to RUN. Load sides 1 and 2 of Underwater Weighing program card No. 4.

Press C To Start Program

Enter:

Bar. Pres. _____mm Hg | Room Air Temp. _____°C | Rel. Hum. _____% | Tank Water Temp. _____°C

Record:

N_2 Meter Calib. Point _____%

Press A To Start New Subj.

Enter:

Syringe Gas Temp. _____°C | Belt Weight In H_2O _____lb | Body Weight In Air _____kg

Notes:

a. Siri equation used for % fat.

b. °C = (°F - 32)/1.8

c. kg = 0.4536(lb)

Press E To Start Replicate Measure

Enter:

Bag Volume Used (l)	N_2 in Bag Before Rebreath. (%)	Mouth-piece Depth (cm)	Body Weight In H_2O (lb)	N_2 in Bag After Rebreath. (%)

Record:

*Lung Gas Vol. After Exhalation (l)	Body Density (g/ml)	% Fat	Fat (kg)	Fat (lb)	Body Weight (lb)

*This is residual volume (RV) only if subject submits complete