THE EFFECTS OF EXERCISE INTENSITY ON PLASMA LIPOPROTEINS DURING A STATIONARY BICYCLE TRAINING PROGRAM

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> The reasonable man adapts himself to the world.
> The unreasonable man persists in trying to adapt the world to himself.
> Therefore, all progress depends upon the unreasonable man.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Atherosclerosis, the progressive blockage of the arteries due to the buildup of atherosclerotic plaques, has become a major concern during the last half of the twentieth century. Preclinical lesions have been observed in humans since the beginning of recorded medical history, so it should not be classified as a disease of modern man (Leibowitz, 1970). When post-mortem examinations became common in nineteenth century Europe, lesions due to atherosclerosis were frequently recognized. However, it was not until 1913 that myocardial infarctions were clinically defined as damage or death to an area of the heart muscle (myocardium) resulting from a reduction in blood supply to that area. At that time an association was drawn between ischemic necrosis of the myocardium, coronary atherosclerosis, and thrombosis (the formation of blood clots). After this period there was a steady increase in the reported incidence of myocardial infarction until the middle of the twentieth century. This was probably a reflection of both an enhanced awareness and improved diagnostic methods.

It is not clear ". . . whether the dramatic increase in the incidence of clinically manifested disease from 1900 until 1960 was caused by an increase in thrombotic and ischemic complications in the presence of a relatively constant severity of atherosclerosis" (National Institute of Health [NIH], 1981). Recently, an important new trend has been observed.

There has been a decrease in mortality from coronary heart disease (CHD) in the United States which is associated with a reduction in coronary atherosclerosis (Strong, Guzman, Tracey, Newman, & Salman, 1979). This is observed in the overall downward trend for atherosclerosis-related deaths in the 1970's as compared to the period between 1940 to 1960, when there was an increase. However, one should not be deluded into thinking that CHD has reached endemic levels. In 1978, there were 873,390 deaths which were related to atherosclerosis (U.S. Department of Health, Education and Welfare [DHEW], 1978).

Coronary heart disease was responsible for placing 33 percent of the 500,000 individuals receiving disability benefits on the permanently disabled rolls (NIH, 1981). There are another 52,000 permanently disabled on the rolls due to atherosclerotic-related diseases. The number one cause of permanent disability for individuals over 40 years of age is atherosclerosis. There are almost nine million individuals between the ages of 20 to 64 suffering some form of disability due to atherosclerosis.

The health care cost from 1970 to 1980 increased from $75 billion (NIH, 1981) a year to $245 billion (estimation), which was a 227 percent increase. More meaningful is the fact that health care cost increased from 7.5 percent of the national gross product in 1970 to 9.5 percent in 1980. In 1980, Americans spent approximately $1,080 per person per year on health care. The estimated cost of those with CHD was 17 percent of the cost for total health care in 1980. This amounted to almost twice the cost spent on respiratory diseases, which is the second major disease. Atherosclerotic diseases account for 70 percent of the cost of all

cardiovascular diseases (NIH, 1981). It is then no wonder there is a national concern about atherosclerotic-related diseases.

Atherosclerosis is a multifactorial health problem with its genesis remaining in doubt. Epidemiological studies have identified approximately 40 risk factors that are statistically related to the development of atherosclerosis. The Framingham and other studies isolated major factors that are believed to be atherosclerotic agents. Those factors are: elevated blood cholesterol, the cigarette habit, hypertension, measures of carbohydrate tolerance, and several ECG abnormalities (Kannel, 1979).

Several studies have identified cholesterol as being one of the major factors in the development of atherosclerosis; but what is the role of cholesterol and is it all bad as the popular literature leads one to believe?

Cholesterol is found in the diets of people who consume animal products. It is slowly absorbed from the gastrointestinal tract within the intestinal lymph system (Guyton, 1976). Cholesterol that is absorbed from the diet is referred to as exogenous cholesterol, which accounts for 10 percent of the total body cholesterol. Ninety percent of the body's cholesterol is manufactured in the body by the liver and is referred to as endogenous cholesterol (Stare, 1974). Both sources circulate in the lipoproteins of the plasma.

Circulating cholesterol may be greatly increased through diets high in saturated fat. This is believed to result from fat deposition in the liver where cholesterol is produced (Guyton, 1976).

Cholesterol, by association with CHD and the development of atherosclerosis, has received a great deal of negative attention. However, cholesterol serves several vital functions within the body. It is a

precursor for vitamin D, for steroids which are important in the production of sex hormones, and for some hormones from the adrenal cortex (Hole, 1978). Bile acids are derived from cholesterol and it serves as a part of the protective coating of nerves and the spinal cord (Calloway & Carpenter, 1981). "Cholesterol modulates the fluidity of all membranes and is, therefore, partly responsible for regulating permeability across the cells and the normal immune response and defense system of the body" (Oliver, 1982). The female sex hormones (estrogens) decrease blood cholestrol, whereas the male sex hormones (androgens) increase blood cholesterol (Hole, 1978). It is interesting to note that females are less prone to heart disease before menopause, but after menopause their susceptibility is near that of men (Kannel, 1979). One of the several reasons for this is the reduction in estrogens.

Cholesterol is removed from the body by bile into the small intestine, where some is reabsorbed for use within the body again and the rest is excreted in the feces. One of the cholesterol reduction methods makes use of drugs that bind it within the small intestine which prevents its reabsorption (Calloway & Carpenter, 1981). Several authors have noted that there is a direct relationship between the blood serum cholesterol level (measured in mg/dl) and the susceptibility or potential for developing coronary heart disease (Albrink, Meigs, & Man, 1961; Carlson & Botteger, 1972; Gofman, Young, & Tandey, 1966; Kannel, Castelli, & Gordon, 1971). However, Oliver (1982), writing in the Executive Health Newsletter, states that ". . . it is not expected that a strong, consistent relationship should exist between raised blood cholesterol and coronary heart disease." To support this, he cited a study that compared healthy individuals in Stockholm with others in Edinburgh, aged 40,

who had identical cholesterol levels. However, CHD was three times higher in Edinburgh than in Stockholm.

Oliver believed that cholesterol then, as a variable in identifying CHD, was population dependent. It was his opinion that other factors may enter into the heart disease problem which have little influence on increased cholesterol. As further evidence, he pointed out that the United States Pooling Project Research Group found ". . . no neat, continuous relationship . . ." between CHD and cholesterol levels (Oliver, 1982).

As early as 1856, Virchow advanced the theory based on animal studies that ". . . atherogenesis is largely determined by the rate of influx of cholesterol from plasma into the artery wall" (Virchow, cited in Miller, 1978). Since that period, research has indicated that atherosclerosis is the major cause of death related to CHD in the United States and Western Europe (Ross & Glomset, 1976).

The recent ten-year Primary Coronary Prevention Trial (Langone, 1984) has found, contrary to Oliver's opinion, a consistent relationship between elevated blood cholesterol and CHD. This research indicated that for every one percent decrease in plasma cholesterol, there was a two percent decrease in the likelihood of a coronary event.

Oliver may have concluded that the relationship between elevated serum cholesterol and CHD is not a strong one; however, research from epidemiological studies (Carlson & Botteger, 1972; Kannel, 1979) and the recent ten-year studies (Clark, 1984; Langone, 1984) certainly indicate the opposite.

Cholesterol and the lipids that are associated with it are found in several forms. The lipids that circulate in the blood after digestion

are known as chylomicrons. During the post-absorptive state, when these particles are not present, over 95 percent of the lipids found in the plasma circulate in the form of lipoproteins (Guyton, 1978). These molecules are smaller than the chylomicrons but are similar in nature. The lipoproteins contain a mixture of triglycerides, phospholipids, cholesterol, and proteins. The proteins make up 25 to 33 percent of the lipoprotein mixture, with the remainder being composed of lipids. There are three major classes of lipoproteins: (1) very low density lipoproteins which contain a high concentration of triglycerides and moderate concentrations of both phospholipids and cholesterol; (2) low density lipoproteins which are low in concentration of triglycerides but high in cholesterol; and (3) high density lipoproteins with a concentration of about 50 percent protein and a small fraction of lipids (Guyton, 1976).

The three classes of lipoproteins have each been identified with the development of coronary heart disease. Their full involvement relative to coronary risks will be discussed in Chapter II. Presently it is important to point out that a direct relationship has been identified between low-density lipoprotein (LDL) and the increased incidence of CHD, whereas there is an inverse relationship between high-density lipoprotein (HDL) and CHD.

Pathogenesis of Atherosclerosis

Atherogenesis begins early in life, since it has been observed in pre-adolescents (Thorland & Gilliam, 1981) and among young soldiers (average age of 22.1 years) killed during the Korean War (Enos, 1953).

Atherosclerosis is a lesion within the arterial wall. The basic unit for it is the atheromatous plaque, which is made up of lipids

(cholesterols and other fats) and has a covering or cap of fibrous (scar) tissue (Ross & Glomset, 1976). As the plaque increases in size and number, it develops inward on the inner wall of the vessel and may eventually impede or cut off blood within the affected arteries and thus cause damage to the organs and tissues that are supplied by these arteries Ross et al., 1976).

The specific mechanism through which the lesions grow remains in doubt. Currently there are three working hypotheses involved in the pathogenesis of atherosclerosis, each involving lesions to the artery; these are: fatty streak, fibrous plaque, and the so-called complicated lesion (Ross et al., 1976).

The development of the atherosclerotic artery involves smooth-muscle cell proliferation and changes of the intima, the innermost layer of cells within the artery. The fatty streak ". . . is characterized by a focal accumulation of relatively small number of intimal smooth-muscle cells, containing and surrounded by deposits of lipids" (Ross et al., 1976). The lesion does not protrude into the artery; rather, it is flat and causes no obstruction and no clinical symptoms. Its color is yellow, which indicates the presence of lipid deposits which are primarily found within the smooth-muscle cells and in microphages. Macrophages are cells which have the ability to ingest and destroy particulate substances. The lipids are mostly in the form of cholesterol and cholesterol ester. Esters are compounds which no longer contain water. The majority of cholesterol within the fatty streak is absorbed from the blood plasma and is probably re-esterified upon being taken up by intimal layers of smooth-muscle cells (Ross et al., 1976).

Fatty streaks are found at various anatomical sites within the arterial tree. The age of appearance differs with the anatomical site; however, they are found in the aorta of virtually every child regardless of race, sex, or environment by the age of ten years (Ross et al., 1976). The areas of the intimal surface covered in the aorta are approximately 10 percent at age 10 years and progress to 30 to 50 percent by age 25 (Ross et al., 1976).

The second type of lesion is referred to as a fibrous plaque which is most characteristic of advancing atherosclerosis; however, this plaque is not evenly distributed as the fatty streak is among the world's population. The appearance of this plaque is whitish and is elevated so that is protrudes into the lumen of the artery. The principle constituents are lipid-laden smooth-muscle cells, cholesterol, and cholesterol esters. Lipids, collagen, elastic fibers, and proteoglycans surround the cells. A fibrous cap is formed by the cells and extracellular (collage, elastic fibers, proteoglycan) matrix. The fibrous cap covers ". . . a large, deeper deposit of free extracellular lipids intermixed with cell debris" (Ross et al., 1976).

The third lesion is referred to as a complicated lesion. It has been altered as a result of hemorrhage, calcification, cell necrosis, and mural thrombosis (a blood clot formed on the wall of the vessel) (Ross et al., 1976). This lesion is distinctively characterized by the presence of calcification which is often present in occlusive diseases.

The three focal lesions discussed and the site of the disease process are identified by three fundamental phenomena: ". . . proliferation of smooth-muscle cells, deposition of intracellular and extracellular

lipids, and accumulation of extracellular matrix components including collagen, elastic fibers and proteoglycans" (Ross et al., 1976).

The possible atherosclerotic processes discussed indicated that the lipid, cholesterol, was absorbed from the plasma into the intimal layers of the artery. There are several theories concerning the process of passage of lipoproteins from the plasma into the smooth-muscle cells. The endothelial cells of the artery are unlike those of capillaries in that they normally preclude the passage of blood constituents into the artery wall. However, recent studies have indicated that constituents may pass through by means of vesicular transport or by transiently formed channels (Ross et al., 1976). Vesicular transport is by means of small blister-like structures. The vesicular and the transiently formed channels are highly specialized so they will only transport molecules of specific sizes. It is believed that passage would occur for small high-density lipoprotein molecules, but not the larger, very low density lipoproteins or chylomicrons. The relationship between the passage of the different size molecules of lipoproteins, metabolism of lipids within the arterial wall, and CHD remains to be fully elucidated.

As mentioned previously, there are several fractions of lipoproteins. The HDL and LDL components both play important but opposite roles in the pathology of CHD.

The low density lipoprotein has been identified as contributing directly to atherosclerosis through being deposited by means not fully understood within the inner layers of the arterial wall, the intimal region (Kare & Borresen, 1976; Miller & Miller, 1975). Through its deposition, the lumen's diameter is reduced, along with a reduction in the vessel's compliance. The reduced diameter decreases the vessel's ability

to transport nutrients to those tissues and organs which are supplied by the affected vessel with a concomitant reduction in their function. The lack of the vessel's compliance is exhibited through a loss of elasticity.

It is postulated that high levels of HDL may inhibit LDL from being deposited but low levels may allow it to build up (Miller, 1978; Yoshikazu, Noboru, Yasuhiko, & Motoo, 1980). It is presently believed that the HDL bonds to LDL and transports it to the liver where catabolic metabolism of the LDL takes place (Glomset, 1968). The LDL is then excreted in the bile as a salt. Thus it can be understood why HDL is an important fraction of the total lipoprotein profile and the importance of high levels as a safeguard against the development of CHD.

Although the importance of the total lipoprotein profile is now realized, early investigators focused on the role of total cholesterol and triglycerides in the development of atherogenesis. However, Miller and Miller (1975) point out that clinical investigation in the early 1950's of the total lipoprotein profile was available. Investigation in the 1950's revealed that ". . . hypercholesterolaemia commonly present in coronary victims reflected an increase in the plasma concentrations of very low density (VLDL) and/or low density (LDL) lipoproteins" (Miller, 1978). The same investigation found that HDL concentration was relatively low. For reasons not known, ". . . these findings (about HDL) failed to arouse interest, and the attention of epidemiologists, clinicians and experimental pathologists continued to be focused on the roles of VLDL and LDL in atherogenesis" (Miller, 1978).

The literature shows that while HDL was not investigated, important research was conducted concerning the relationship between CHD and total cholesterol. Epidemiological evidence suggests that total cholesterol

was elevated in individuals who experienced a coronary event (Kannel, 1979). Furthermore, it was established that individuals who were more physically active were less susceptible to heart disease. These and other epidemiological studies may have been the spawning grounds for the idea that physical activity may have a prophylactic effect on CHD. With this information, researchers in the 1950's and 1960's conducted studies on the relationship between physical exercise and several physiological parameters. It was shown that through exercise programs, individuals reduced their body weight and total cholesterol; however, with cessation of exercise both increased (Mann, 1955). Research of this nature continued for several years and emphasized different types of exercise protocols. The major finding showed that a negative caloric balance, or loss of weight, paralleled a decrease in serum cholesterol.

Beginning in the early 1970's, analysis of the total lipoprotein profile was more common. Studies of these profiles revealed that individuals with CHD had low levels of HDL (Gofman, Young, & Tandey, 1966); those with no overt signs of atherosclerosis had high levels of HDL (Rhoads, Gulbrandsen, & Kagan, 1976). This suggested the idea that HDL may act as a protective agent against CHD (Gordon, Castelli, & Hjortland, 1977).

Data from cross-sectional and longitudinal studies that investigated chronic physical exercisers showed HDL levels higher than sedentary populations. Investigations of runners (Martin, Haskell, & Wood, 1977; Wood, Haskell, Klein, Lewis, Stern, & Farquhar, 1976), cross-country skiers (Carlson & Mossfeldt, 1964; Enger, Herbjornsen, Eriksen, & Fretland, 1977), and tennis players (Vodak, Wood, Haskell, & Williams, 1980) showed higher levels of HDL than matched controls.

The aforementioned studies indicate that individuals who are physically active have a lower incidence of CHD and higher levels of HDL. Thus a reverse relationship exists between these two factors. However, it is not presently known how much exercise or physical activity is necessary to increase the HDL concentration to a protective level. It has been established that HDL levels may be increased through running approximately 11 miles per week (Cooper, 1981). Another investigation determined that this occurs at 10 miles per week after nine months of running (Williams, 1979). However, neither investigation quantified running in intensity, duration, or frequency of training necessary for these changes to occur.

The relationship between coronary heart disease, physical activity, and HDL is an inverse one. In addition, the relationship between HDL and total cholesterol is one that should not be overlooked. Cooper (1981) has suggested that absolute high levels of HDL or low levels of total cholesterol alone are not always an indication of protection from heart disease. He cited an example of an individual who was a regular runner with a high HDL level, yet he required bypass surgery due to major obstruction of his coronary arteries. His thesis in this example was that CHD does not depend upon one or two factors "alone." Cooper pointed out that a more important factor than low total cholesterol or high levels of HDL is their ratio. He believed this ratio should be at least 4.5 or smaller.

Purpose of the Study

This study investigated the effects of exercise using stationary bicycles on HDL plasma levels. The choice of bicycles was a logical one, since many individuals have them in their homes or can find them at health

clubs. They are used in rehabilitation programs and, more importantly, bicycling does not cause trauma to joints as may occur in running. Also, some individuals are not capable of walking at intensities necessary to facilitate an improvement in their functional capacity. Swimming programs may be used to increase range of motion for older individuals or as a basic fitness program with the handicapped or normal populations. Swimming, however, requires a degree of coordination, skill, and feeling of "at ease" before it can be used to develop an aerobic base. These prerequisites often eliminate many individuals, along with the inaccessibility of swimming facilities. Therefore, bicycling may be the avenue for many through which fitness may be achieved.

It is believed that for a minimum aerobic training effect to occur, the exercise intensity threshold is 60 percent of the heart rate range, provided this occurs a minimum of three times per week (Pollack, Wilmore, & Fox, 1978). However, it is not known what the threshold intensity is for an increase in the HDL level in relation to the heart rate range. Specifically, the review of literature indicates there have not been any studies that compared different exercise intensities while bicycling to change plasma levels of HDL and total cholesterol. Therefore, this study will investigate three exercise intensities while bicycling in order to determine if an independent exercise threshold intensity exists.

Significance of the Study

Several studies have indicated that physical activity and CHD are inversely related. The majority of the research has focused on running as a means of changing risk factors that are associated with the development of CHD.

There are many individuals who, like runners, are interested in improving their health through physical exercise, but who cannot run due to orthopedic and related problems. A viable alternative for these individuals are stationary bidycles which are used as a therapeutic medium in hospitals and university rehabilitation and physical fitness programs. Additionally, stationary cycling is not weather dependent. There are several models which are inexpensive enough for the homeowner to purchase. However, the less expensive models may render less than satisfactory service. Therefore, caution and judgment need to be applied before purchasing a stationary cycle.

For individuals who use bicycles as runners enjoy the roads, there should be a method to quantify the intensity level necessary to alter their cardiovascular risk factors. The importance of this study rests with the identification of specific exercise intensities as quantified through the exercise heart rate, which may identify an exercise threshold which alters the lipoprotein profile, specifically elevation of HDL.

Problem Statement

The purpose of this study was to determine the effects of three different exercise intensities on high-density lipoprotein during a twelve-week stationary bicycling exercise program. The intensities were 50, 60, and 70 percent of the heart range.

Null Hypotheses

The three hypotheses below were tested using analysis of covariance in which percent fat was used as the covariable. The same three hypotheses were tested without the covariable.

1. There is no interaction among intensity levels and weeks for HDL, ratio between HDL and total cholesterol, and total cholesterol.

2. There is no effect due to intensity level for HDL, ratio (HDL/total cholesterol) and total cholesterol, and total cholesterol.

3. There is no effect due to time for HDL, ratio (HDL/total cholesterol), and total cholesterol.

Subproblems

Assumptions

1. The pulse measuring device was valid.

2. The HDL and T.C. assay methods were valid.

3. The subjects were willing and enthusiastic to participate in the investigations.

Limitations

1. The subjects were volunteers. There was no insurance of participation.

2. The subjects' diets were not controlled.

3. The subjects' weight gains or losses were not controlled.

Delimitations

1. The subjects were all males between 20 and 45 years of age.

2. The subjects were all members of the Oklahoma State University Police Department.

Definitions of Terms

Aerobic exercise--an exercise which uses the large muscle groups in a continuous rhythmic isotonic fashion. Examples are bicycling, walking, running, swimming, cross-country skiiing, and skating.

Arteriosclerosis--commonly called hardening of the arteries. This broad term includes a variety of conditions that cause the artery walls to become thick and hard.

Atherogenesis--the chain of events leading to the development of atheromatous lesions in the arterial wall.

Atherosclerosis--a form of arteriosclerosis in which the inner layer of the artery wall is made thick and irregular by deposits of fatty substances. These deposits (called atheromata) project above the surface of the inner layer of the artery, and thus decrease the diameter of the internal channel of the vessel.

Atheroma--a deposit of fatty (and other) substances in the inner lining of the artery wall characteristic of atherosclerosis. Plural form of the word is atheromata. See atherosclerosis.

Cholesterol--a sterol widely distributed in animal tissues and occurring in the yolks of eggs, various oils, fats, and nerve tissue of the brain and spinal cord. It is important as a precursor of various hormones (sex hormones, adrenal corticoids) and in repair of cell membranes.

Electrocardiogram--often referred to as EKG or ECG. A graphic record of the electric currents produced by the heart.

Heart rate range--the maximum heart rate minus the resting heart rate.

Intima--the innermost layer of a blood vessel.

Lipid--substances soluble in organic solvents but poorly soluble, or not at all, in water.

Lipoproteins--a complex of lipids and protein molecules. Lipoproteins are classified according to their density (high, low, very low) or electrical charge (alpha, beta, and pre-beta).

Lumen--the passageway inside the tubular organ. Vascular lumen is the passageway inside the blood vessel.

Saturated fat--a fat so constituted chemically that it is not capable of combining with any more hydrogen. These are usually the solid fats of animal origin such as the fats in milk, butter, meat, etc. A diet high in saturated fat tends to increase the amount of cholesterol in the blood.

Risk factor--attribute of human populations or their environments which is associated with a greater than average incidence of a specified disease.

CHAPTER II

REVIEW OF THE LITERATURE

Coronary heart disease (CHD) is one of the major medical and health-related problems of the twentieth century (Altekruse, 1973; NIH, 1981). The development of CHD is related to atherosclerosis, which may be thought of as multifactorial and closely related to the levels of one major variable--cholesterol (Glueck, 1976; Langone, 1984). Other variables that contribute to the development of heart disease have been identified through epidemiological studies and are collectively referred to as risk factors (Gordon, Castelli, & Hjortland, 1977; Paffenbarger, Wing, & Robert, 1978; Paffenbarger, Laughlin, & Gima, 1970).

In 1949, a 16-year multiphasic screening program, known as the Framingham study, was begun. Initially, 5,208 men and women between the ages of 30 and 59 were screened. They were studied for 16 years to determine those health-related factors which contributed to cardiovascular diseases (Gordon et al., 1977). The hallmark of this study was the identification of risk factors for a variety of American populations which aid in the prediction of the probability of having the first coronary event. The risk factors are not appropriate once vascular disease has developed (Kannel, 1979).

Initially, a large number of risk factors were identified which were believed to contribute to the development of vascular disease (Table 1). Viewing this list gives the impression that the problem is too complex

Table 1

Factors Related to Atherosclerosis and Ischemic Heart Disease[1]

Age	Income, living standards
Radioactivity in water	Lipid fractions, serum lipoprotein
Being a British medical practitioner	Magnesium deficiency
Blood group other than O	Manganese deficiency
Carbon disulfide	Obesity, overweight
Carboxyhemoglobin	Pectin consumption (P)[2]
Cholesterol, elevated	Personality type
Chromium deficiency	Physical activity (P)
Climate	Polyunsaturated fatty acids (P)
Coagulation disorders	Respiratory impairment
Diabetes, glucose tolerance	Saturated fats
ECG abnormalities	Sex
Education	Smoking
Family history, genetic factors	Stress
Fat intake, total	Sugar intake
Heart rate, resting	Triglyceridemia, elevated
Heavy meals	Uric acid in serum
Hematocrit values	Vanadium deficiency
Hypertension	Water softness
Hypothyroidism, latent	

[1]Kannel, 1979. [2]Protective factor.

for prophylactic procedures. However, there are only a few risk factors which have been established as major ones. These include: serum total cholesterol, hypertension, measures of carbohydrate tolerance, the cigarette habit, and several ECG abnormalities (Kannel, 1979). Each of these factors has been shown to make a sizable independent contribution to the risk of developing cardiovascular diseases (Kannel, 1979). The cigarette habit and high blood pressure have also been identified in other studies as cardiovascular risk factors (Paffenbarger, Wing, & Robert, 1978; Paffenbarger, Laughlin, & Gima, 1970). The recent ten-year Coronary Primary Prevention Trial found an unequivocal relationship between heart disease and abnormally high cholesterol levels (Langone, 1984).

Research prior to the Coronary Primary Prevention Trial (Langone, 1984) developed norms which indicated a risk of developing CHD. The Framingham study suggested that a cholesterol level of 224 mg/dl or greater as being associated with three times greater incidence of CHD than is associated with cholesterol levels of 210 mg/dl or less (Altekruse, 1973). Schilling (1964) identified the upper units for desirable total cholesterol for anyone beyond adolescence as being 180 to 220 mg/dl. Jolliffe (1962) believes that the maximum limit should not be beyond 200 mg/dl. Cooper (1981) identified levels relative to fitness categories. He has indicated total cholesterol levels above 224.5 mg/dl as a very poor level of fitness and levels of 211.2 mg/dl or below as a high level of fitness. These ranges represent different aspects of research with almost 20 years separating them. However, these norms for health are all very close, since the normal range for total cholesterol is 110 to 300 mg/dl (Altekruse & Wilmore, 1973).

Cholesterol is only one fraction of the lipoproteins which is transported in the plasma. Lipoproteins, which transport cholesterol, also transport the triglycerides, phospholipids, and proteins (Guyton, 1976). Protein makes up about 25 to 33 percent of the total; the remainder is lipids (Guyton, 1976). The lipoproteins are broken into three major classes: (1) very low-density lipoproteins which contain high concentrations of triglycerides and moderate concentrations of both phospholipids and cholesterol; (2) low-density lipoproteins which are low in concentration of triglycerides but high in cholesterol; and (3) high-density lipoproteins with a concentration of about 50 percent protein and a small fraction of lipids (Guyton, 1976).

The three lipoprotein fractions are each associated with the coronary risk profile. The very low-density lipoprotein (VLDL), or the triglycerides it carries, is related to CHD through its association ". . . with higher than average relative weight and cholesterol and blood sugar values, and with lower-than-average values of high-density lipoproteins" (Kannel, 1979). The VLDL does not make a strong independent contribution to CHD as do some of the other risk factors.

Low-density lipoproteins (LDL) are the principle constituents of atheromatic plaques which appear in the subintimal layers of the arteries. The development of these plaques may eventually lead to the development of atherosclerosis or hardening of the arteries (Guyton, 1976). Thus LDL has been identified as making a sizable independent contribution to CHD.

High-density lipoproteins (HDL) appear to have a protective effect against CHD. It is believed that they inhibit the accumulation of LDL in the intimal layers of the arteries (Bondjers & Bjorkervds, 1975). The

importance of the HDL level is realized by the fact that individuals with low levels of HDL are at a higher risk for developing CHD than those with higher levels. As a result of one portion of the Framingham study, it was determined that individuals with HDL levels of 35 mg/dl or less had an eight times greater chance of developing CHD than those with HDL levels of 65 mg/dl or greater (Gordon et al., 1977). Cooper (1981) has identified HDL levels of 37.0 mg/dl and 49.3 mg/dl as representing very poor levels of fitness and high levels of fitness, respectively. He believes that the single greatest predictor of heart disease is the ratio between total cholesterol and HDL. According to Cooper, that ratio should be less than 5.0 and preferably less than 4.5.

It has been well established in both humans and experimental animal studies that elevated blood pressure ". . . accelerates atherogenesis and increases the incidence of coronary heart disease and other forms of atherosclerotic diseases, particularly cerebral vascular disease" (Pooling Research Group, 1978). It has been further established that hypertension may act independently of the other known major risk factors.

The risk of developing CHD is directly related to the number of cigarettes smoked per day. It is about 60 to 70 percent greater for cigarette smokers than nonsmokers (NIH, 1977). Paffenbarger, Wing, and Robert (1978) found in both the longshoremen and Harvard alumni studies that the cigarette habit increased the risk of having a heart attack. It is encouraging to note that smokers who gave up smoking reduced their risk to one-half that of those who continued to smoke.

Obesity is associated with hyperlipidemia, hypertension, and diabetes. Obese individuals may also have a high level of LDL, triglyceride, and a low level of HDL (NIH, 1977).

A family history of premature coronary events has also been identified as an added risk factor. However, as Kannel (1979) points out, ". . . families share more than genes." Lifestyles are transmitted as readily as genes; however, this can be changed. Sedentary living, overeating, and a lack of or too little exercise can contribute to increased risks through the development of hypertension and elevated serum cholesterol.

Physical inactivity has been thought to have little to do with the development of atherosclerosis. However, its effect upon other risk factors, especially cholesterol and its HDL fraction (Wood & Haskell, 1979), triglycerides (Altekruse & Wilmore, 1973), and its possible role in prevention of CHD has been shown to be important (Gordon et al., 1977; Miller & Miller, 1975).

Although a number of major factors have been identified, their cause within the general population remains unclear and controversial (Kannel, 1979). A study much more recent than Framingham has identified a positive link between cholesterol and heart disease. The study also identified diet as one of the causes for high levels of plasma cholesterol, and suggested methods for reducing it (Langone, 1984). This relationship has been assumed by the medical community but never conclusively proven until now. The 10-year Coronary Primary Prevention Trial ". . . showed that the greater the reduction of cholesterol toward normal levels (none were identified), the greater the reduction of heart attack events" (Langone, 1984).

The study placed 3,806 men who were free of heart disease but considered high risk due to their high cholesterol levels on a combination diet and drug program. Initially, all subjects were placed on a moderate

diet that reduced their cholesterol by 3.5 percent. The subjects were then divided into two groups, one receiving a ". . . cholesterol-lowering resinous drug cholestyramine while the others received a placebo" (Langone, 1984). The results showed a drop in total cholesterol of 13.4 percent and a 20.3 percent drop in LDL within the experimental group. This was a significantly greater drop than the one shown by the group taking the placebos. More importantly, there was a 19 percent drop in the risk of heart attacks. Both groups experienced coronary events, with the experimental group recording 155 compared to 185 within the controls. Additionally, those taking the drug developed fewer symptoms of incipient heart disease and had fewer coronary by-pass operations (Clark, Hager, & Witherspoon, 1984).

Dr. Basil Rifkind, the director of the study, believes that a change in the typical American high fat animal diet to one that is low in animal fat can bring cholesterol levels down by 10 to 15 percent. Individuals with extremely high levels of cholesterol, especially LDL, should receive drug therapy in addition to diet modification. He also emphasizes that attention must be directed toward reducing all the risk factors, especially the cigarette habit and blood pressure, since all risk factors are additive.

Several studies have indicated that the chance of developing CHD is greatly increased when risk factors are combined (Kannel, 1979; Paffenbarger, Wing, & Robert, 1978; Paffenbarger, Laughlin, & Gima, 1970). In other words, there is an accumulative effect. That is one reason why it is so important to eliminate as many risk factors as possible. By use of the major risk factors, a risk profile may be developed that will help ". . . identify a tenth of the population from which 25% of the

coronary disease, 40% of the occlusive peripheral arterial disease, and 50% of the strokes and cardiac failure will emerge" (Kannel, 1979).

Since the major risk factors are known to be associated with the development of cardiovascular diseases, the next logical step would be the modification of these. It has been shown that a reduction of hypertension (Boyer & Kasch, 1970), the cigarette habit (Kannel, 1979), and a reduction of total cholesterol and LDL (Langone, 1984) will all contribute to a reduced risk profile.

When risk factors are artificially induced, there is an increase in atherosclerosis in animal experiments; and it is known to occur at higher levels in humans afflicted with vascular disease. Furthermore, individuals who are exposed to risk factors tend to develop more disease than those less exposed. Additionally, it is known that these factors operate independently, and the risk increases in exponential fashion when more than one factor is present. This is evidenced in several studies that have looked at risk factors independently and additively. It is a well established fact that atherosclerosis and related vascular diseases are a leading health hazard and a major cause of disabilities in affluent societies. However, "this is not necessarily an inevitable consequence of aging and genetic make up" (Kannel, 1979). This statement by Kannel, one of the principle researchers of the Framingham study, was made after investigating data of CHD around the world.

Although it is possible to estimate the risk of having a coronary event over a wide variety of populations within the United States, it does not entirely explain the variance of risks within or among various populations. This is demonstrated by comparing the Framingham, Puerto Rican, and Hawaiian-Japanese populations. The major risk factors are

evident in each of these populations; however, ". . . the incidence of disease in Framingham is roughly twice that of the other populations" (Gordon, Garcia-Palmieri, & Kagan, 1974).

There are many factors, both environmental and genetic, that help to determine the level of plasma cholesterol in different individuals (NIH, 1977). Genetic factors cannot be altered but mental ones can. The major environmental contribution to plasma cholesterol which individuals have control over is the amount of saturated fat within the diet. There is evidence that reduction in dietary saturated fats will result in decreased total serum cholesterol (Hall & Barnard, 1982; Pritikin, 1982).

The Relationship Between Diet, Cholesterol, and CHD

The relationship between diet, cholesterol, and heart disease is an area that has received considerable attention. Dietary lipids have been considered by several investigators to be a major environmental agent responsible for the development of severe atherosclerosis and related diseases among populations of highly technologically developed and affluent societies (Glueck, 1979; Mahley, 1979; McGill, 1979). The diets of populations in far Eastern countries are low in saturated fats which results in a decreased total serum cholesterol (National Diet-Heart Study Research Group, 1968; Keys & Parlin, 1966; Hartley, Sherwood, & Hert, 1981). As a consequence, their mortality rates from CHD is substantially lower than in the United States (DHEW, 1978).

Early in the twentieth century, dietary cholesterol was shown to produce artherosclerotic-like lesions in rabbits and was discovered to be the main lipid found within human atherosclerotic lesions (Guyton, 1976). The case against saturated fats in helping to cause atherosclerosis

was strengthened when it was observed that there was a concomitant rise in the concentration of serum cholesterol with an increase in dietary saturated fats.

Initial dietary studies showed a relationship between the amount of dietary cholesterol ingested and the concentration of plasma cholesterol in the serum. The morbidity and mortality rates for the atherosclerotic diseases were also influenced by the amount of dietary cholesterol ingested. One problem in trying to isolate cholesterol as a causative factor was its close chemical and dietary association with saturated fats. For this reason, it was not possible to show ". . . an exclusive and independent association of dietary cholesterol with arterial lesions or atherosclerotic diseases in humans" (NIH, 1981).

Another problem with the early dietary cholesterol studies was an assumption that dietary cholesterol directly affected atherogenesis through an increased concentration of serum cholesterol. Later it was determined that cholesterol affects atherogenesis by alteration of the lipoprotein profile. This occurs through a change in the structural or functional properties of specific lipoproteins. It is now known that dietary cholesterol increases the low-density lipoproteins (LDL-C), but does not significantly alter the HDL (NIH, 1977). The typical American diet which is high in saturated fats has been named as the principle culprit in heart disease and abnormally high cholesterol levels by Pritikin (1982) for several years. Pritikin, who is the founder and director of the Pritikin Longevity Center in California, pointed out that cultures ascribing to diets low in saturated fats have low or almost nonexistent heart disease rates. For example, the Japanese, who are heavy smokers--which is a known risk factor--have the lowest incidence of heart disease

among the developed nations. This is believed to be due to their low daily fat intake of about 10 percent, which keeps their cholesterol levels at an average of 150 mg/dl; however, the average cholesterol level in the United States is 210 to 220 mg/dl (Pritikin, 1982).

A population that is almost immune to heart disease are the Tarahumara Indians of Northern Mexico. Connor, Cerqueira, Connor, and Wallace (1978) studied this population and found no evidence of deaths relating to cardiovascular diseases. They found their diet to be similar to populations that have a low death rate due to cardiovascular diseases. As with the Japanese, the Tarahumara Indians' diet averaged 10 percent fat and the cholesterol levels of the adults were between 100 to 140 mg/dl.

Kannel (1979) indicated that it had not yet been conclusively proven that an ". . . alteration in the diet does in fact reduce the potential for (heart) disease. . . ." At the time of his writing, the previously mentioned population studies indicated that diets other than the typical American high fat diet do, in fact, reduce the potential for heart disease.

Another approach to the problem of CHD has been to study the relationship between diet, cholesterol, and CHD in native populations and migrant populations in their adopted land. Yoshitazu, Noboru, Yasuhiko, and Motoo (1980) compared plasma HDL levels in native Japanese and Westerners living there with norms for Westerners. It was observed that native Japanese and Westerners who ate the traditional Japanese diet low in saturated fats had significantly higher HDL levels than Westerners. To determine if this was genetic or environmental, the HDL blood levels of neonates for Westerners and Japanese were compared. It was found that

the levels for both populations were nearly the same. The authors concluded that the higher levels of Japanese HDL was environmentally related, possibly owing to the traditional Japanese diet which is low in saturated fats. They did not report on physical activity, which has been shown to influence HDL levels (Williams, 1979).

Along the same line of study, Toor, Katchalsky, Agmon, and Allalouf (1957) found that when healthy immigrants from comparatively uncivilized countries moved to more technologically advanced ones, they developed cholesterol levels comparable to those of their adopted country. This is further demonstrated by Keys and Parlin (1966), who studied Japanese living in the United States, Hawaii, and Japan. It was found that the Japanese living in America had the highest levels of cholesterol and incidence of CHD. Another investigator (Groom, 1959) compared autopsy data of American and Haitian Negroes. It was reported that the American Negroes had twice the degree of atherosclerosis than the Haitians who ate a diet that was low in saturated fats. Groom postulated that diet alone was not the cause. He believed that factors such as a stressful environment, mechanization, competition, and lack of physical activity may have all had a contributing effect.

An approach to reducing cholesterol that combines exercise and a low fat diet has been developed by Pritikin (Barnard, Guzy, Rosenberg, & O'Brien, 1982). At the Pritikin Longevity Center, 23 men and 6 women, ages 31 to 82 years, all with CHD, participated in a 26-day rehabilitation program. The subjects were placed on a walking program and attended exercise classes five days per week. The walking program started with one-half mile and increased to 5.8 miles. All subjects ate a high complex carbohydrate and low fat diet.

The subjects' serum cholesterol decreased from 236 to 179 mg/dl, the triglycerides dropped from 174 to 147 mg/dl, and mean weight loss was 6.61 pounds. There was a low correlation between serum lipid decreases and weight loss, daily walking, and MET capacity. The investigators concluded that the drop in serum cholesterol was due to a decrease in dietary cholesterol. Evidence from the recent 10-year Coronary Primary Prevention Trial study pointed to the importance of lower total cholesterol. Results from the study found ". . . that every one percent drop in the cholesterol level in the blood stream means a two percent decrease in the likelihood of a heart attack" (Langone, 1984).

The controversy should be settled, since evidence is now in from the medical community that shows unequivocally that a decrease in total blood cholesterol results in a decrease in coronary events. This combined with epidemiological studies of populations eating a low fat diet, which results in both low total cholesterol values and decreased CHD, should resolve any question of the importance of a low fat diet with regard to decreasing total cholesterol and the resultant probability of decreasing CHD.

Coronary Heart Disease and Its Relation to Various Occupations

Several epidemiological studies have advanced the idea of an inverse relationship between coronary heart disease and occupational physical activity. Morris, Raffle, Roberts, and Parks (1953) investigated the cause of death between 31,000 bus drivers and bus conductors who were previously employed by the London Transport System. It was determined that fatal heart attacks were more frequent among drivers than conductors. Similar

results were noted when comparing postmen and postal clerks. These differences were attributed to the physical activity that conductors and postmen did in performing their jobs. It was reported that lack of physical activity may have been the cause of the higher rates of CHD in a study of a North Dakota community (Zukel & Lewis, 1959). Taylor, Klepetar, Keys, and Parlin (1962) reported that the CHD mortality is inversely related to job activity in a study of railroad clerks, switchmen, and section men. Although the above studies found a significant difference between physical exertion on the job and CHD, others have not. Chapman (1957) and Stamler (1960) found no significant relationship between physical exertion on the job and the incidence of CHD between Los Angeles civil servants and Chicago utility workers. Spain and Bradess (1960) analyzed the autopsy records of individuals who died of violent deaths. They found no significant difference in the extent of the occlusion of coronary arteries on those with active as compared to those with sedentary occupations.

The previously mentioned cross-sectional studies yield important information regarding the particular effects of an occupation upon the health status of its employees. However, limitations are inherent since investigators are not able to determine if it was the occupation that exerted its influence upon their health or some predisposing factor that qualified them for their job. In order to fill this void, longitudinal studies investigate both initial physical characteristics and long-term effects of the environment upon the individual.

Paffenbarger, Laughlin, and Gima (1970) studied the incidence of CHD among San Francisco longshoremen over a 16-year period. Beginning in 1951, 3,263 longshoremen, ages 35 to 64 years, were put through a

multiphasic screening program to assess job assignments, cigarette habits, blood pressure, and weight/height ratio, all of which were believed to be associated with coronary heart disease.

Before the advent of containers, longshoremen as a group, expended more kilocalories per work week than most other occupational groups. The cargo handlers expended approximately 6.7 kcal/min as compared to approximately 2.8 kcal/min for the less active individuals. During an eight-hour day, the less active expend about two and one-half times less energy than the more active cargo handlers, which translates to 925 fewer kilocalories per day.

The follow-up study in 1967 revealed there was a total of 888 deaths from heart attacks and 67 from strokes. The less active workers had a death rate one-third higher than the cargo handlers. With respect to age, there was a more favorable association for younger active workers than older individuals. The older workers usually move from active jobs of handling cargo to sedentary work. This is believed to be reflected in the increased death rate among the older workers.

In addition to the relationship between work and CHD, other factors were also investigated. Those individuals who smoked one or more packs of cigarettes per day increased a risk of CHD that was double that of light smokers. Those with high systolic blood pressure had an 89 percent greater chance of having a heart attack than those with normal levels. Individuals with body weight above normal had a 35 percent greater death rate.

Physical activity seems to have a mediating effect upon heart disease even when risk factors are taken into account. The more active

workers suffered a lower death rate at both levels of the risk scale than the less active workers.

A classic longitudinal study was conducted by Paffenbarger, Wing, and Roberts (1978) on 36,500 male Harvard alumni who entered college between the years of 1916 and 1950; this represents a life style span of between 16 to 50 years since matriculation. Information on athletic participation and personal background was obtained through the alumni office. Information on blood lipids and diet was not obtainable. Questionnaires were mailed to alumni to obtain information on adult exercise habits and doctor-diagnosed disease. Official death certificates furnished information on mortality.

Initial questionnaires were mailed in 1962 and 1966, depending on the year of matriculation. A second questionnaire was mailed in 1972, in order to identify the nonfatal and fatal heart attacks that had occurred between 1962 and 1966.

Energy expenditure groups were identified for those individuals who were free of diagnosed heart attacks. These individuals were investigated until a heart attack occurred, death from any cause, age 75, or the end of the study in 1972. Person years of observation were compiled by single years of age so that both the qualitative and quantitative roles of physical activity could be assessed as the population aged during the six to ten years of follow-up.

The initial questionnaires (1962 and 1966) inquired about alumni physical activity patterns. They addressed the number of flights of stairs climbed each day, number of city blocks walked daily, and type of sports activities and hours per week of participation. Sports were divided into light and strenuous categories. Light sports such as bowling

required little energy output; strenuous sports such as running required considerable energy expenditure.

A physical activity index was developed, which was composed of stair climbing, city blocks walked, and sports. Energy expenditure for the activities was calculated from norms rather than actual measurement. The index was divided at 2,000 kcal/week, which resulted in a 60 to 40 percent grouping in the low and high energy categories.

Heart attack patterns were inversely related to energy expenditure. A sharp reduction was observed in both nonfatal and fatal heart attacks with an increase in weekly energy output, 2,000 to 2,999 kcal/week. Energy expenditure beyond this rate did not indicate further protection; instead, there seemed to be a plateau effect rather than a continued decrease.

Involvement in strenuous sports seemed to be the key to reduction in heart attacks. This is indicated by the fact that those who engaged in light sports activity, stair climbing, and walking had a 54 percent higher rate of heart attack than the corresponding rate of 36.8 percent for men who reported strenuous sports play. Another way of viewing this is those men who did not participate in strenuous sports were at a 38 percent greater risk of heart attacks than those who did engage in these activities. It is of interest to also note that men who reported not playing any sports were at no increased risk of heart attack over those who did engage in light sports. When the age factor was considered, it was observed that there was a lower rate of heart attacks with increasing age and increasing energy expenditure. Thus physical activity, when considered independently in relation to heart attacks, appears to have a protective quality.

The relationship between energy expenditure, fatal, and nonfatal heart attacks appeared to be directly related to the amount of energy expended per week. It was observed that as the energy expenditure rose, there was a decrease in the number of heart attacks. Thus physical activity may have resulted in some type of protection against heart attacks.

In addition to lack of physical activity, there were other factors which appeared to influence the risk of having a heart attack. To assess these in relation to physical fitness, information was gathered on student and adult characteristics from alumni records and questionnaires. Eight predictors of an increased risk for heart attacks were identified. Those risks were: cigarette smoking, systolic and diastolic blood pressure, body mass, body stature, early parental death, physical inactivity, and varsity sports. Univariate analysis of these characteristics showed that the differential rate of heart attacks which was associated with the high and low physical activity index (<2,000 kcal/week and >2,000 kcal/week) persisted. When the eight factors were taken separately, they were predictive of heart attacks. However, apparently strenuous activity (2,000 kcal/week) had an ameliorating effect, for when it was taken into account there was a reduction in the risk of heart attacks with or without the specific risk factor. This indicated that the level of energy expenditure had an independent influence on the risk of heart attacks. In other words, the relative risk of having a heart problem was decreased at both ends of the scale of risk factors when strenuous exercise was included.

When looking at risk factors independently, hypertension was considered to have the greatest independent risk for a potential heart attack, while cigarette smoking and physical inactivity were next. One student

characteristic, physical activity or varsity sports participation in college, was initially found to have an inverse relationship between heart disease in middle aged men (35 to 54 years). However, this relationship did not continue unless physical activity was continued during the adult years. In other words, participation in sports during college was unrelated to the risk of a heart attack during the adult years. It is encouraging to note that those alumni who were not active during their college days, but active during their adult years, showed a reduction in heart attacks.

Strenuous physical activity also has a protective effect for those who had heart attacks. Paffenbarger, Wing, and Roberts' (1978) data and data from the Framingham study (Kannel, Sorlie, & McNamara, 1970) suggest there was no difference in risk of sudden death between those who were active versus those less active; however, there was a lower risk of a lethal outcome for the more active men.

While physical activity does not prevent the development of atherosclerosis, it does contribute to the development of a superior cardiovascular system. This occurs through the development of collateral circulation of the myocardium which helps to compensate for a compromised coronary circulation system (Kannel, Sorlie, & McNamara, 1970). However, the amount and intensity of exercise that is necessary to promote collateral circulation remains in doubt. Although it is recognized that for collaterals to develop, it is necessary first that the vessel be partially blocked (Eckstein, 1957). It is recognized that physical training will increase the myocardial stroke volume, decrease the resting pulse, increase the cardiac output, and increase the A-VO_2 difference. Each of these and in combination will decrease the work load placed upon the

myocardium. However, for these to occur requires training of athletic intensity that would not be found in the normal adult population.

In summary, Paffenbarger, Wing, and Roberts (1978) found that those individuals who were more physically active had a lower incidence of CHD than their sedentary counterparts. More importantly, the survival rate from a coronary event was higher for the more active alumni, which may be due to a superior cardiovascular system. Thus, according to Kannel, Sorlie, and McNamara (1970), the intensity of exercise for protection to occur may be quite modest.

Deaths related to coronary vascular diseases are decreasing in number; however, it still remains the largest medical and health care problem in the United States (NIH, 1981). Its cause is complex and multifactorial in nature. Several epidemiological studies have identified major risk factors which are associated with the development of CHD. Elevated levels of plasma cholesterol, hypertension, and the cigarette habit are the principle risk factors involved. Risk profiles using these and other factors may be developed to indicate the likelihood of developing heart disease. Through the use of risk profiles, it may be observed that risks are additive.

Dietary cholesterol has been strongly implicated in the development of atherosclerosis and CHD. The American diet, which is high in saturated fats, has been identified as one of the principle culprits in this disease. Dietary studies of the Tarahumara Indians of Northern Mexico and the Japanese show their diets to be composed of 10 percent fat versus the American diet which is 30 to 40 percent fat. Death among the Tarahumara Indians from heart disease is virtually unknown. Likewise, the Japanese have a low incidence of coronary vascular-related deaths.

The dietary implications are evident: lowering the percent of fat in the diet may lead to a reduction in heart disease.

It has been postulated that there is an inverse relationship between CHD and physical activity. Epidemiological studies of occupational groups which are physically active have indicated there is a lower incidence of CHD than with sedentary groups. It is believed that the benefits of physical activity are a superior cardiovascular system, rather than the prevention of atherosclerosis which is the leading cause of CHD (Kannel, Castelli, & Gordon, 1971).

In order to identify relationships that may exist between CHD and physical activity, controlled research needed to be conducted. However, this did not occur until the late 1950's and early 1960's. Most of the early studies concentrated on weight reduction, vegetable oil, diet, and drug medication as methods of reducing total cholesterol and thus preventing CHD (Rochelle, 1961).

Physical Exercise and Its Relation to Coronary Heart Disease

One of the earlier studies on cholesterol and its relation to exercise was Rochelle's (1961). He studied twelve normal adults, ages 20 to 36 years; six acted as controls. His program was divided into three phases. During Phase I, a pretraining period, and Phase II, a five-week exercise period, blood plasma was drawn four times daily from two experimental subjects and three times daily from two control subjects. During Phase III and during eight weeks of detraining, blood samples were drawn twice daily, every second week. The eight remaining subjects had samples drawn weekly during each phase except Phase I, two two-week

pretraining periods, when daily determinations were run. Blood samples for cholesterol determination were drawn at 7:00 a.m., 12:00 noon, before exercise; 12:30 p.m., after exercise; and 10:00 p.m. The 7:00 a.m., 12:00 noon, and 12:30 p.m. were premeal samples, whereas the 10:00 p.m. sample was taken on the average of four to five hours after the evening meal. Blood was drawn from any finger and collected in four 0.05 ml heparinized capillary tubes. The samples were analyzed by the Duboff-Stevenson ultramicro method. The training program was a daily two-mile run for time. Individuals were encouraged to improve their running time. The subjects kept daily food consumption charts which showed they ingested between 35 to 40 percent saturated fat through the study. The results showed that: (1) mean cholesterol decreased significantly between the pre- and post-test; (2) cholesterol increased following exercise, which may indicate fat mobilization and utilization during physical exercise; and (3) cholesterol levels during the detraining phase returned to pretraining levels within four weeks.

The changes in body weight and skin fold thickness were investigated but not statistically controlled for. It was observed that most individuals decreased their total cholesterol regardless of their change in body weight and skin fold thickness.

This rather lengthy discussion points out two facts. First, all individuals did not receive the same pre- and post-treatments, which was a major flaw in the experimental design. Second, it is important to note that the investigator did not account for changes in body weight and skin fold thickness in the statistical analysis. Without accounting for this, it is hard to determine if exercise or weight loss was

responsible for the decrease in cholesterol. Unfortunately, this problem continues in more recent studies.

Milesis (1974) reported that inconsistent results appeared in the literature concerning the effects of exercise training on serum cholesterol and triglycerides. He suggested that programs not only vary in method, but more importantly, they could not be accurately compared since the total caloric cost of exercise was lacking. Milesis attempted to correct for this by determining the caloric output for one subject and then using his energy expenditures for all subjects for a given exercise session.

The subjects for this study were 22 adult males ranging in age from 28 to 54, with total cholesterol within the normal range. The training sessions consisted fo a modified Cureton's rhythmic endurance exercise four times weekly for 35 minutes. Modification was necessary to include more running and jogging. However, the 35-minute session was still broken up and did not include continuous exercise.

The subjects showed an overall nonsignificant decrease in serum cholesterol from pre- to post-analysis. It is interesting to note that Rochelle's (1961) subjects showed a significant decrease in serum cholesterol while exercising at high intensity daily for 12 to 15 minutes while Milesis' subjects exercised intermittently for 35 minutes. Additionally, both groups reduced body fat. The difference in total cholesterol reduction then might be explained by the type of exercise and/or the method of analysis.

It is now known that the lipoprotein subfractions, pre-beta (VLDL), beta (LDL), and alpha (HDL) may change with no overall lowering in total cholesterol. It has been shown that exercise may increase the alpha-

fraction and decrease the pre-beta fraction. If earlier studies would have investigated these fractions or the total lipid profile, different results may have been found. Thus if exercise programs are to be effectively evaluated, the total lipoprotein profile needs to be evaluated.

Altekruse and Wilmore (1973) were one of the first investigators to examine the effect of exercise on the total lipid profile. The exercise program consisted of having 39 sedentary male subjects walk, jog, or run a prescribed distance three times a week for ten weeks. The subjects were encouraged to either increase the distance run or decrease the time to run a given distance in a prescribed time.

Fasting venous blood samples were drawn two days prior to the beginning of the program and three days after its termination. The blood was analyzed for total cholesterol by the Lieberman-Burchard method and for lipoproteins (alpha, pre-beta, beta, chylomicrons) by electrophoresis.

Total cholesterol from the pre-test value of 224.10 changed to a post-test value of 200.45 mg/100 ml, which was a significant change at the 1 percent level of significance. The subject's body mean weight also decreased from 81.42 to 80.42 kg. A decrease in body weight has been found by other investigators to parallel a decrease in total cholesterol. However, other investigators have found that when the subjects increase their caloric intake during exercise, thus maintaining body weight, no significant change occurs in total cholesterol (Holloszy, 1964; Mann, 1955). Further evidence for a decrease in total cholesterol during exercise comes from other investigators (Golding, 1961; Naughton & Balke, 1964). In general, they found that when the subject's body weight decreased, so did their total cholesterol. However, when exercise was

terminated, their total cholesterol increased as their body weight approached pretraining levels.

Altekruse and Wilmore (1973) concluded that a decrease in serum cholesterol may be an expected phenomenon that is associated with a weight decrease when a sedentary individual becomes involved in a physical conditioning program.

Analysis of the lipoproteins showed a significant increase in HDL (alpha) and a significant decrease in both LDL (beta) and VLDL (prebeta). It is interesting to compare Altekruse and Wilmore's research in the early 1970's, where little mention is made of the role of lipids in CHD and their metabolism, to researchers in the late 1970's and early 1980's who spent considerable time in the discussion of lipids. Altekruse and Wilmore's study is confusing because they did not truly control the exercise conditions. Subjects were allowed to walk, jog, or run at their own pace. Although findings were significant for both total cholesterol and HDL-C, little can be stated about the quantity and quality of exercise necessary to produce these changes.

An investigation was performed on the effect that running had on the blood chemistry of middle-aged runners (Wood, Haskell, Klein, Lewis, Stern, & Farquhar, 1976; Wood, Klein, Lewis, & Haskell, 1974). They observed that runners have significantly lower blood levels of triglycerides and total cholesterol than nonrunners who were matched by age. They also found that HDL as well as the HDL/total cholesterol ratio was higher in runners than nonrunners.

Enger, Herbjornsen, Eriksen, and Fretland (1977) investigated trained cross-country skiers the day before competition and found they had significantly higher HDL/total cholesterol ratios than untrained skiers.

Lopez, Vidal, Balart, and Arroyane (1974) investigated young men with normal serum lipids. It was found that after seven weeks of intense physical activity, four 30-minute sessions per week, there was a significant decrease in serum triglycerides and pre-beta (VLDL). There was also a significant decrease in the absolute concentration of beta lipoprotein (LDL), with alpha lipoprotein (HDL) concentration increasing significantly.

Deshaies and Allard (1982) investigated the lipid profile of Olympic athletes at the 1976 Games in Montreal, Canada. It was observed that both men and women had ". . . approximately 20 per cent more HDL than levels reported in the literature for the general population of North Americans."

One of the major problems in comparing cholesterol-related studies has been the lack of control for body weight difference, since some investigators have shown that body fat is positively related to serum cholesterol decreases. This being the case, it has been postulated that HDL and body fat are inversely related (Hagan & Gettman, 1983).

Hagan & Gettman (1983) controlled for this by having no significant difference in the sedentary controls and active distance runners for body weight, lean body mass, and body fat. There was also no significant difference between the two groups with respect to cholesterol, LDL-C, and VLDL-C. However, the runners had significantly higher HDL-C and HDL-C/cholesterol ratios. They concluded that ". . . distance running does not affect cholesterol and LDL and VLDL levels but is associated with significantly lower triglyceride levels and significantly higher HDL-C levels."

Hagan and Gettman (1983), Mann (1955), Milesis (1974), and Rochelle (1961), to mention a few, did not control for weight loss or variation. It is important to do so, for the loss of weight represents a negative caloric balance which is a major factor in lowering serum cholesterol (Anderson, Lawler, & Keys, 1957; Hatch, 1966).

Holloszy's (1964) research over a six-month physical conditioning period showed a nonsignificant gain in serum cholesterol. The interesting point here is that the cholesterol gain was accompanied by an increase in body weight, which is indicative of a positive caloric balance.

Further evidence is presented by Campbell (1967) who worked with obese and control individuals. The subjects walked a motor-driven treadmill at the same absolute work load for three hours per week for ten weeks. At the end of the ten weeks, the obese subjects recorded a weight loss and significant decrease in serum cholesterol. This did not occur with the control subjects. Golding (1961), working with four overweight subjects, trained them for one hour per day, five days per week for 25 weeks. At the termination of the program, serum cholesterol levels had decreased at an average of 30 percent, which was consistent with body weight losses. Both of these studies indicate that a negative caloric balance, or a weight loss, is consistent with a decrease in total serum cholesterol. However, it is difficult to determine in these studies, as others, which caused a reduction in serum cholesterol: the exercise program or weight loss.

Dalderup, de Voogd, Meyknecht, and Hartog (1967) exercised male subjects for several weeks, five days a week, for 80 minutes a day, and found a slight decrease in serum cholesterol. The subjects maintained body weight with an increase in lean body mass.

Milesis (1974), in an article discussed previously, found a reduction in skin fold measurement while body weight remained constant and serum lipids decreased. Research of the nature of Milesis that does not differentiate between body composition changes may lead to confusing retults. Dalderup et al. (1967) may be interpreted as suggesting that weight loss has little or no effect upon decreases in serum cholesterol. However, Milesis' research indicated that a decrease in serum cholesterol may parallel weight loss.

Much of the research about cholesterol has centered on running. It is encouraging to note that walking has also been considered. Leon (1977) exercised six obese men, aged 19 to 31 years. The subjects walked five days per week on a motor-driven treadmill, beginning at 1.5 mph and progressing to 3.2 mph at a constant grade of 10 percent. The sessions increased from 15 minutes to 90 minutes after two weeks. The subjects progressed to a final work load of 5,000 to 6,000 kcal per week or about 1,000 to 1,200 kcal per day. The participants lost from 11 to 13 pounds of fat weight and showed a cardiorespiratory training effect. The plasma lipid profile showed that HDL increased significantly along with the HDL/LDL ratio. However, total cholesterol and triglycerides did not show a significant change. It is difficult to determine what was responsible for the change in the lipoprotein profile and cholesterol: exercise or weight loss. If these factors would have been controlled for, one or the other could have been isolated as a possible causative factor.

Some researchers have quantified the change in lipoproteins and miles run per week (Cooper, 1981; Williams, 1979). Cooper determined the threshold level for change in HDL levels to be approximately 11 miles per week. Williams found that LDL and HDL did not decrease and increase

until the subjects had been running 10 miles per week for nine months. Neither of these investigators determined the intensity, duration, or frequency of training that was necessary for the changes to occur. Therefore, mileage alone may not be the precipitating factor.

The foregoing research is encouraging for those who run. But what about the many individuals who are concerned about their health and want to change but cannot run for medical or bio-mechanical reasons, or simply do not enjoy it? For those individuals who enjoy walking, bicycling, or swimming, there should be a method to quantify these activities in relation to the threshold level for a significant increase in the HDL level. However, most research does not address those areas.

Summary

Epidemiological data indicate a strong relationship between cholesterol and lipoprotein levels, and the incidence of heart disease. Heart disease increases in proportion to the levels of total cholesterol and is inversely related to HDL levels.

Cholesterol levels are governed by genetic and environmental factors (NIH, 1981). One environmental factor found in the diet and known to increase total cholesterol is saturated fat, which is found in food products derived from animals. In an attempt to identify its importance in the development of CHD, investigators have compared populations that consume diets high in saturated fat with those who derive their fat mainly from vegetables. Cross-sectional studies have indicated that CHD is more prevalent in Western populations who consume high saturated fat diets than in far Eastern cultures who consume little saturated fat and have a low incidence of CHD.

Another approach to finding underlying factors for heart disease has been to investigate occupations. Morris (1953) and Taylor et al. (1962) found that the more active occupations had a lower incidence of heart disease. Zukel and Lewis (1959) observed that the lack of physical activity may have been the cause for higher death rates. On the other hand, Chapman (1957), Stamler (1960), and Spain and Bradess (1960) found no relationship between physical activity on the job and CHD. However, the idea was still spawned that physical exercise might have a prophylactic effect on CHD.

In an attempt to verify this, research began in the early 1960's on physical conditioning and its effect upon cholesterol. This continues to be one of the most heavily researched topics today. Since the beginning of research in this area, findings have been mixed due to inadequate research designs and inconsistencies from study to study. Thus it is difficult to compare results. The actual effectiveness of exercise programs as a method of reducing serum cholesterol and altering the lipoprotein profile is questionable because changes in body weight, skinfold thickness (body composition), and diet have not been adequately considered in the experiments.

CHAPTER III

METHODOLOGY

The study began with 24 male employees of the Oklahoma State University Police Department. Eleven of the subjects were unable to complete the twelve-week program due to police training schools which they were required to attend. Two of the subjects left the program after eight weeks of training, thus leaving eleven participants.

Personal Data

Prior to any testing, all subjects were required to sign an informed consent form (Appendix A). In addition, those individuals over forty years of age were also required to have a signed physician's approval form (Appendix A). The health status was evaluated by asking them several health-related questions (Appendix A).

Physiological Measurements

The resting electrical activity performance of each subject's myocardium was assessed through a resting twelve lead EKG to determine if any abnormalities existed; none was observed. The following pre- and post-tests were administered: (1) a supervised submaximal functional EKG bicycle stress test, (2) a four site, skinfold measurement, and (3) venous blood was analyzed for total cholesterol and high density

lipoprotein. In addition to the pre- and post-blood test analysis, blood was also analyzed at the end of week eight.

The blood samples were drawn by a registered nurse or this investigator from the cephalic vein at the anti-cubital area following a twelve-hour fast. The blood samples were analyzed by the magnesium-phosphotungstate enzyme method (Staff, 1982) for cholesterol and HDL utilizing a Bausch-Lomb Spectrometer 20.

Body Composition

Several investigators have shown that change in body composition is related to alteration of lipoprotein profile (Campbell, 1967; Golding, 1961). For this reason, body composition was assessed by measuring skinfolds at four sites: tricep, bicep, liac crest, and sub-scapular. Total measurement in millimeters was then used to predict the subject's body composition (Durnin, 1967). Ideal body composition for adult males is 15 percent fat weight with acceptable range for a healthy individual between 15 to 20 percent. Each subject was informed of his body composition and told how much weight it would be necessary to lose in order to reach his ideal weight. In the event that weight loss did occur, and hence change in body composition, total skinfolds were held as a covariant in order to segregate out the effect of change in body composition upon the effect exercise had upon the total lipoprotein profile.

Lipoprotein Profile

Total cholesterol has been associated with the occurrence of CHD. The Framingham study and the ten-year Coronary Primary Prevention Trial indicated that there is a direct relationship between total cholesterol and the incidence of CHD and an inverse relationship between HDL and CHD.

Initially, total cholesterol was the major variable associated with heart disease. In the mid-1970's, the importance of the total lipoprotein profile was realized. Investigators began to be concerned with its fractions in relation to CHD. It was determined that the low density lipoprotein (LDL), which has been referred to as the "bad cholesterol," was responsible for the build-up of fatty plaques within the walls of the coronary arteries which may lead to a reduction in blood supply to the myocardium. On the other hand, HDL has been shown to prevent the LDL from being deposited and to aid in the removal of the same.

To assess total cholesterol and high-density lipoprotein, the magnesium-phosphotungstate analysis method was employed. This investigator's accuracy in making these assessments was checked by having an experienced laboratory technician test three samples independently. The results are shown in Table 2.

Table 2

Cholesterol and HDL Lipoprotein Analysis

Total Cholesterol ($mg \cdot dl^{-1}$)		HDL-Cholesterol ($mg \cdot dl^{-1}$)	
A	B	A	B
142.8	149.7	38.2	39.4
137.1	127.8	39.6	40.9
205.7	212.0	50.5	53.9

A = Experienced laboratory technician.

B = Investigator.

Submaximal Bicycle Ergometer Test

The most reliable measurement of work capacity (functional capacity) is the assessment of maximal oxygen consumption (Wilmore & Norton, 1975). There is a linear relationship between the amount of oxygen consumed, heart rate, and work performed. The more oxygen per unit of time an individual is capable of consuming, the greater the work that can be performed.

A maximal oxygen consumption test may be performed on either a bicycle ergometer or motor-driven treadmill. During a maximal test, expired gas is collected and analyzed for its oxygen and carbondioxide content. This is a very strenuous test for the participant and time consuming for the investigator. To circumvent these factors, reliable and valid submaximal tests have been developed to predict VO_2 max. Predicted VO_2 is determined from nomograms or calculations from the data collected during the submaximal test.

The instrument used for testing the subjects of this study was a Monarch bicycle ergometer model 668. Since bicycling uses different muscle groups than walking or running, local muscle fatigue may develop earlier. For this reason, bicycle tests may produce lower VO_2 results than by walking or running. However, since the subjects of this study exercised on stationary bicycles for the twelve weeks of the study, the bicycle became the natural instrument for testing. The submaximal bicycle test used to test the subjects of this study (Appendix B) required that the subject pedal at 50 revolutions per minute for three minutes at three progressive work loads. The subject initially warmed up by pedaling at a zero work load for one minute. The resistance was then moved to one

kilopond to begin the test. At the end of the first three-minute bout, the resistance was increased according to the subject's heart rate during the last half of the second and third minutes. The heart rate at these times should not have differed by more than five beats per minute. If the subject's heart rate had not stabilized and the rate differed by more than five beats per minute, the test remained at the same work load for an additional minute or until the heart rate stabilized (Pollock, Wilmore, & Fox, 1978).

To determine the predicted maximal oxygen consumption, the heart rates during the second and third work loads were graphed along with the predicted maximal heart rate. The maximal oxygen consumption (VO_2 max) in liters per minute ($L\text{-}min^{-1}$) was then interpolated from the line connecting the three heart rates (Appendix B).

Oxygen uptake expressed in liters per minute indicates the amount of oxygen an individual would use for a given unit of work. As the work load increases, so does the amount of oxygen needed. Individuals with a larger body mass would consume more oxygen per minute than individuals with a smaller one at rest as well as during work.

In order to allow all individuals to be compared equally as to their ability to consume and use oxygen per unit of time, regardless of their total body mass, another means of expressing oxygen consumption was used. By converting liters of oxygen consumption to milliliters and expressing it per kilogram of body weight per minute ($ml^{-1}/kg^{-1}/min^{-1}$), individuals of differing body masses were compared as to their aerobic capacity.

Exercise Prescription

In order for this exercise program to be beneficial, the participants

needed to bicycle for the specified time, three days per week. The importance of this was verbally explained to the participants during the initial phases of the program. Additionally, two communications during the 12-week period were written to encourage them to participate (Appendix C).

An exercise prescription was developed for each individual based on the results of the battery of physiological tests. The prescription consisted of a warm-up period, progressive target heart rate (THR), and cool-down period. The conditioning periods progressed as follows: week one, 10 minutes; week two, 12 minutes; week three, 17 minutes; week four, 20 minutes; week five, 25 minutes; week six and thereafter, 30 minutes.

Target heart rates were developed based on their submaximal functional capacity tested and were expressed in METS. A MET is the subject's resting metabolic rate or heart production. One MET is considered to be 3.5 $ml^{-1}/kg^{-1}/min^{-1}$. Thus if an individual's functional capacity was 11.6 METs, 50 percent of that would be 5.8 METs. This information was applied to a heart rate graph from the submaximal bicycle test to determine the heart rate at 50 percent of functional capacity. The same method was used to determine heart rates for 60 and 70 percent (Appendix D).

Each group exercised three times per week for twelve weeks on either a Monarch bicycle ergometer which was used during the pre-test, or on a standard ten-speed bicycle which was mounted on a stand known as a racermate. The 50 percent group's intensity did not change during the twelve weeks; however, the 60 and 70 percent groups increased their intensity after two weeks of exercise. The 60 percent group made one increase,

while the 70 percent group made another and final increase after a total of five weeks of exercise.

The exercise pulse rates were monitored by a Carolina electronic pulse rate meter which was validated at the beginning of the exercise program and periodically revalidated. The exercise protocol was as follows:

1. Warm-up period--five minutes.
2. Exercise period at THR for specified period.
3. Cool-down period for five minutes.

Assignment of Subjects to Groups

Each subject in the study was assigned a number which was used in conjunction with a random number table to determine assignment to one of three exercise treatment groups. The treatment groups were based on a percentage of the heart rate range: 50, 60, and 70 percent.

Experimental Design

The experiment was set up in a 3 x 3 factorial arrangement in which there were three levels of intensity and three testing periods. The array below shows the number of observations in each combination of intensity and time:

Treatment[1] Intensity	Pre-Test	8 Weeks	12 Weeks
50	3[2]	3	3
60	4	4	4
70	4	4	4

[1]Percent of heart rate range. [2]Number of subjects per group.

The variables are presented below:

1. Dependent Variable: Total Cholesterol (TC)
    a. High density cholesterol (HDC)
    b. HDL/Total cholesterol ratio
2. Independent Variable: Intensity
    a. 50 percent of HRR
    b. 60 percent of HRR
    c. 70 percent of HRR
3. Covariable: Body Composition
    a. Percent fat
4. Significance Level: Alpha was set at 0.05 percent.

Analysis of Variance

The data were analyzed as a split plot over time in which the main plots (intensity, 50, 60, and 70 percent of heart rate range) were in a completely randomized design. The subplots (pre-test, 08 weeks and 12 weeks) were set up so that each individual had his own control over time.

Analysis of Covariance

The analysis of covariance was used to eliminate the effects of potential change in body composition, which may have an effect on the statistical analysis.

CHAPTER IV

RESULTS AND DISCUSSION

Eleven sedentary adult male volunteers from the Oklahoma State University Police Department completed this study as subjects. The pre- and post-parameters of the 11 subjects are presented in Table 3.

Table 3

Pre-Test Characteristics of the Subjects

Subjects	Age	Weight	Percent Fat	Total Chol.	HDL	Ratio HDL/TOT-C	VO_2 max
1	30	175	15.2	133.0	26.9	20.2	27.8
2	29	181	20.0	212.0	53.9	25.4	37.0
3	26	251	24.0	157.1	28.9	18.4	20.8
4	40	194	20.3	243.5	47.6	19.5	23.3
5	39	155	19.1	181.8	52.0	28.6	34.6
6	32	194	26.1	251.4	16.4	6.5	37.0
7	23	238	29.1	212.0	32.9	15.5	31.6
8	23	216	24.3	193.9	35.9	18.5	33.0
9	49	174	25.3	254.5	60.3	23.7	23.0
10	46	229	26.2	220.0	46.0	20.9	17.3
11	32	232	26.2	209.1	20.4	9.8	26.2

The analysis of variance was applied to the groups for total cholesterol, HDL, and their ratio (HDL/total cholesterol). Neither HDL nor

the ratio showed a significant change at the .05 level of significance among groups or weeks over time. However, there was a highly significant ($P < .01$) linear change in total cholesterol over time. Figure 1 shows that total cholesterol decreased for each of the three groups when moving from 0, to week 8, to week 12. The greatest incremental decrease over time appeared to be in the 70 percent group (Table 4) which might be expected since that was the heaviest work load. However, the statistical analysis showed that the linear change over time in all three groups was essentially the same.

Table 4

Post-Test Characteristics of the Subjects

Subjects	Age	Weight	Percent Fat	Total Chol.	HDL	Ratio HDL/TOT-C		VO_2 max
1	30	170	13.2	106.0	37.0	34.9	27.0	1.42
2	29	176	18.0	206.0	44.0	21.4	33.0	1.25
3	26	246	22.8	151.0	39.2	25.9	31.4	2.30
4	40	193	17.7	256.3	49.1	19.1	36.6	1.92
5	39	159	16.8	150.0	48.3	32.2	48.6	1.83
6	32	182	21.6	196.7	18.5	9.4	35.9	1.92
7	23	233	29.1	187.0	35.0	18.7	25.7	1.17
8	23	217	24.1	206.0	30.8	14.9	30.1	1.25
9	49	169	22.8	162.5	37.1	22.8	29.4	3.00
10	46	224	22.3	187.5	32.9	27.6	26.8	1.58
11	32	226	22.8	168.8	45.5	26.9	29.3	1.25

Since body fat has been shown to be related to total cholesterol (Altekruse et al., 1973), body fat was analyzed to see if it could be

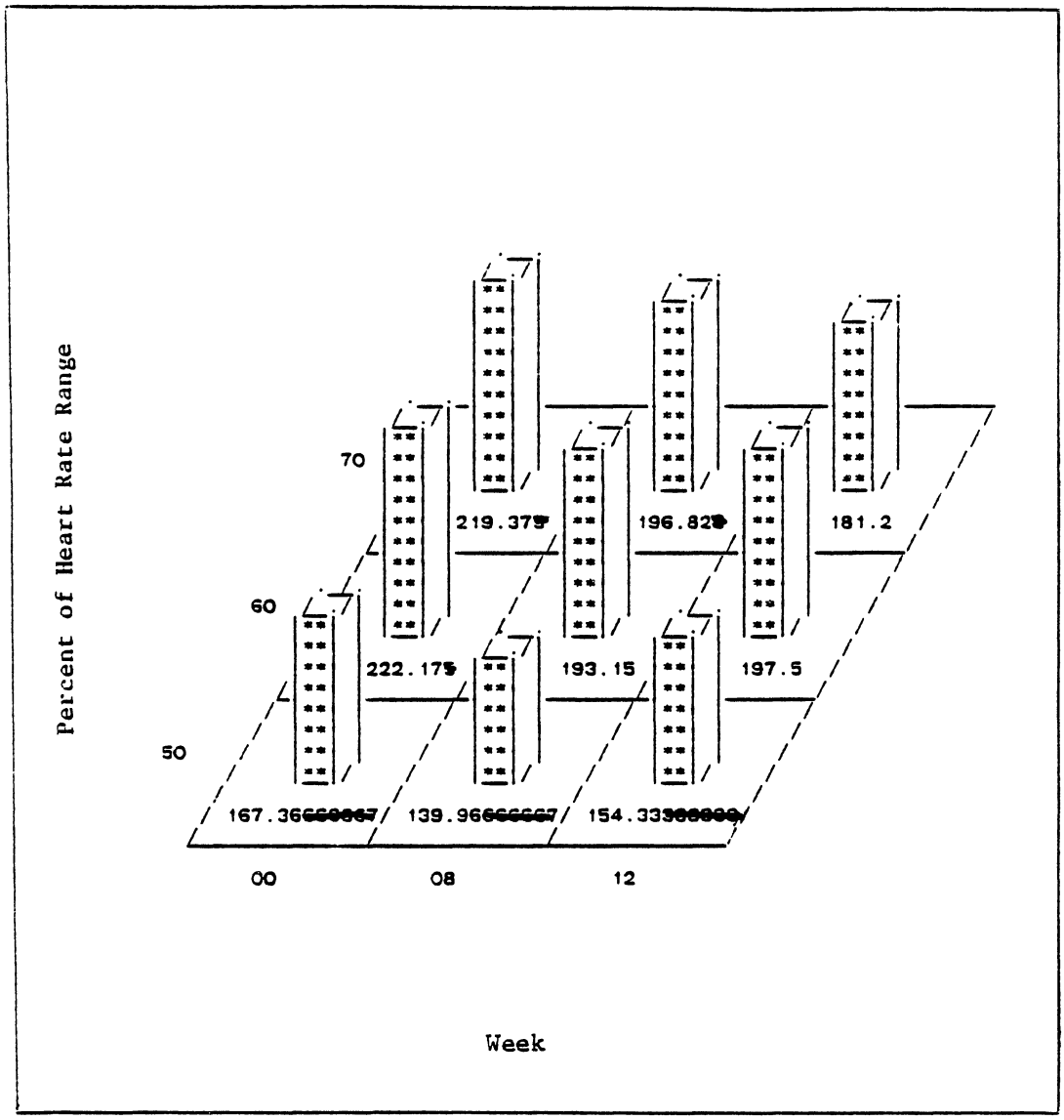

Figure 1. Mean total cholesterol for the twelve-week training program.

used as a covariable to adjust the total cholesterol value to the same body fat composition. These data showed that there was no relationship between body fat and total cholesterol. Therefore, body fat was not used as a covariable. The reason it did not occur in this experiment may have been due to the small numbers of individuals for each treatment group. The same results occurred for the relationship between body fat and HDL, body fat and the rates of HDL to total cholesterol (HDL/Total Cholesterol) (Table 5). In other words, the slope of the line for fat versus total cholesterol, fat versus HDL, and fat versus their ratio was essentially zero. Therefore, change in fat was not the major reason for a decrease in total cholesterol. Thus it was difficult to determine what the main reason for a decrease in total cholesterol was, other than just exercising over time.

Table 5

Table of Means for the Lipoprotein Profile

Percent HRR	Week	N	Total Chol.	HDL	Ratio HDL/Total-Chol.
50	0	3	167.36	36.56	21.34
50	8	3	139.96	38.26	27.70
50	12	3	154.33	40.06	27.40
60	0	4	222.17	37.22	17.54
60	8	4	193.15	38.85	20.29
60	12	4	197.50	37.72	19.86
70	0	4	219.37	40.65	18.21
70	8	4	196.82	33.07	16.71
70	12	4	181.20	36.57	20.57

An important thrust of this investigation was the possible determination of a point where HDL would significantly increase. This study did not identify a level where HDL increased. Other studies have indicated that exercisers in general have higher HDL levels than their sedentary counterparts (Altekruse & Wilmore, 1973; Lopez, Vidal, Balart & Arroyane, 1974). In another study, Cooper (1981) found that when runners maintained eleven miles per week, their HDL levels show a significant increase. Cooper may have quantified the mileage, but he did not identify the length of time necessary to reach this level. Williams (1979) found that runners needed to maintain twelve miles per week for nine months before their HDL levels significantly increased. The main factor in both of these studies was consistent exercise patterns which was lacking in this present investigation, as shown in Figure 2.

It is important to point out that even though the only significant change observed was for total cholesterol (percent HRR versus weeks), trends were noticeable for the other variables.

Figure 3 shows that maximal oxygen consumption increased in all three groups. Cooper (1981) found a positive relationship between max VO_2 and HDL levels in runners. However, it is difficult in the present study to explain the lack of consistency between increases in max VO_2 and the fluctuation in HDL levels between and within groups.

It can be observed from Figure 4 that HDL levels increased in the 50 and 60 percent groups from week 00 to week 08. The 50 percent group showed another increase from week 08 to week 12, while the 60 percent group decreased to near pre-exercise levels. The 70 percent group shows an initial decrease from week 00 to week 08 and then a slight increase

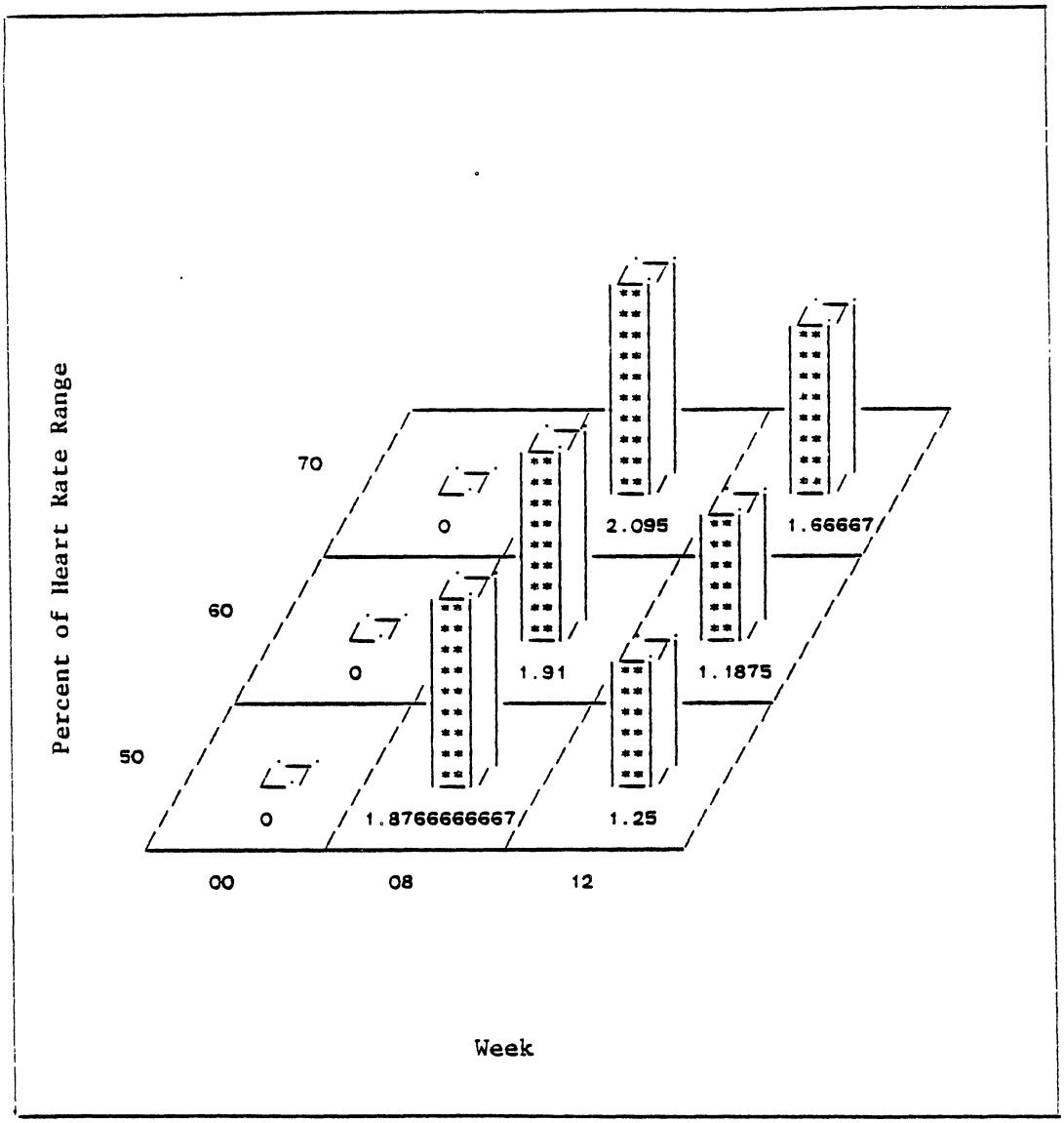

Figure 2. Mean number of exercise sessions attended per week.

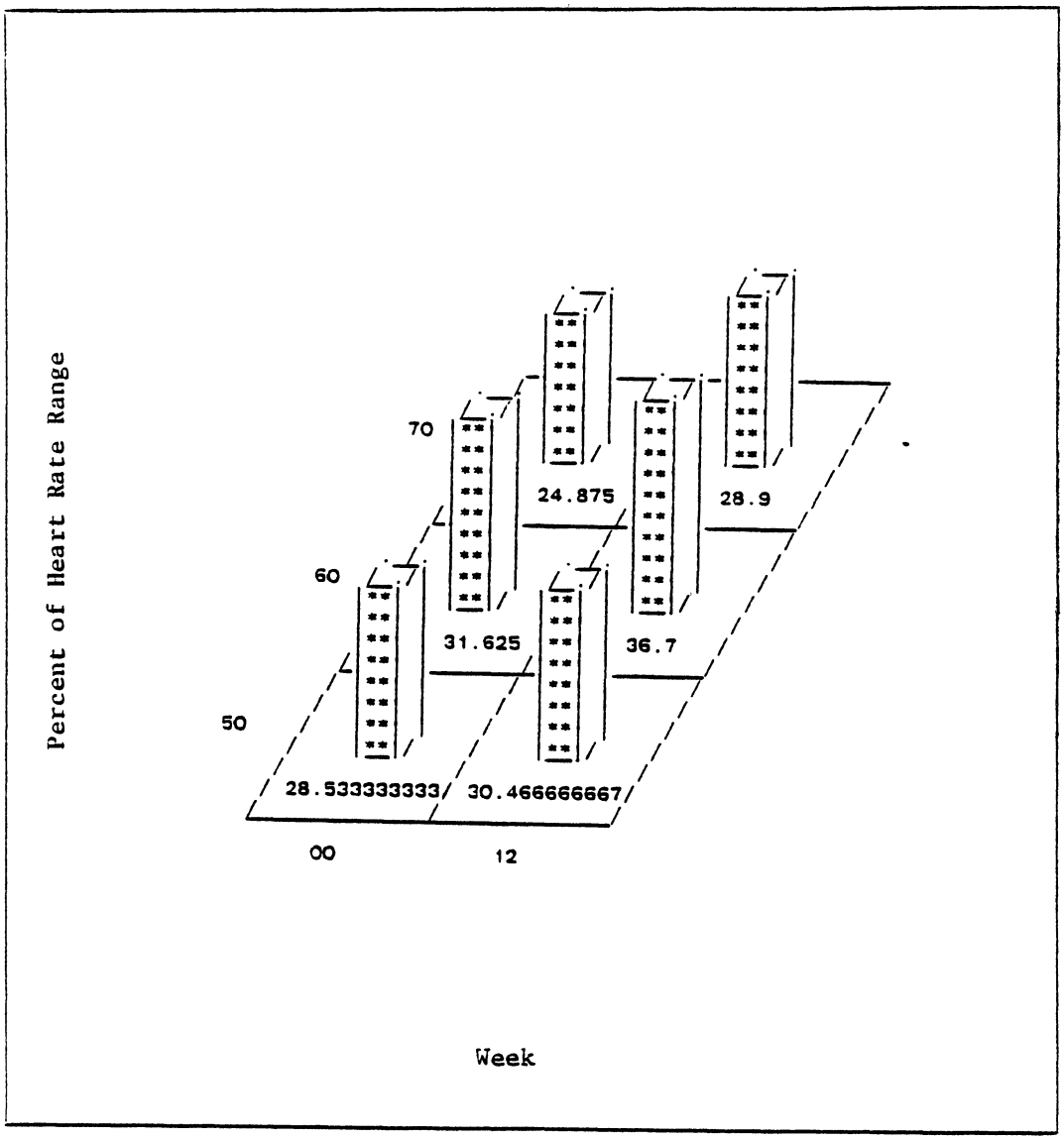

Figure 3. Mean maximal oxygen consumption.

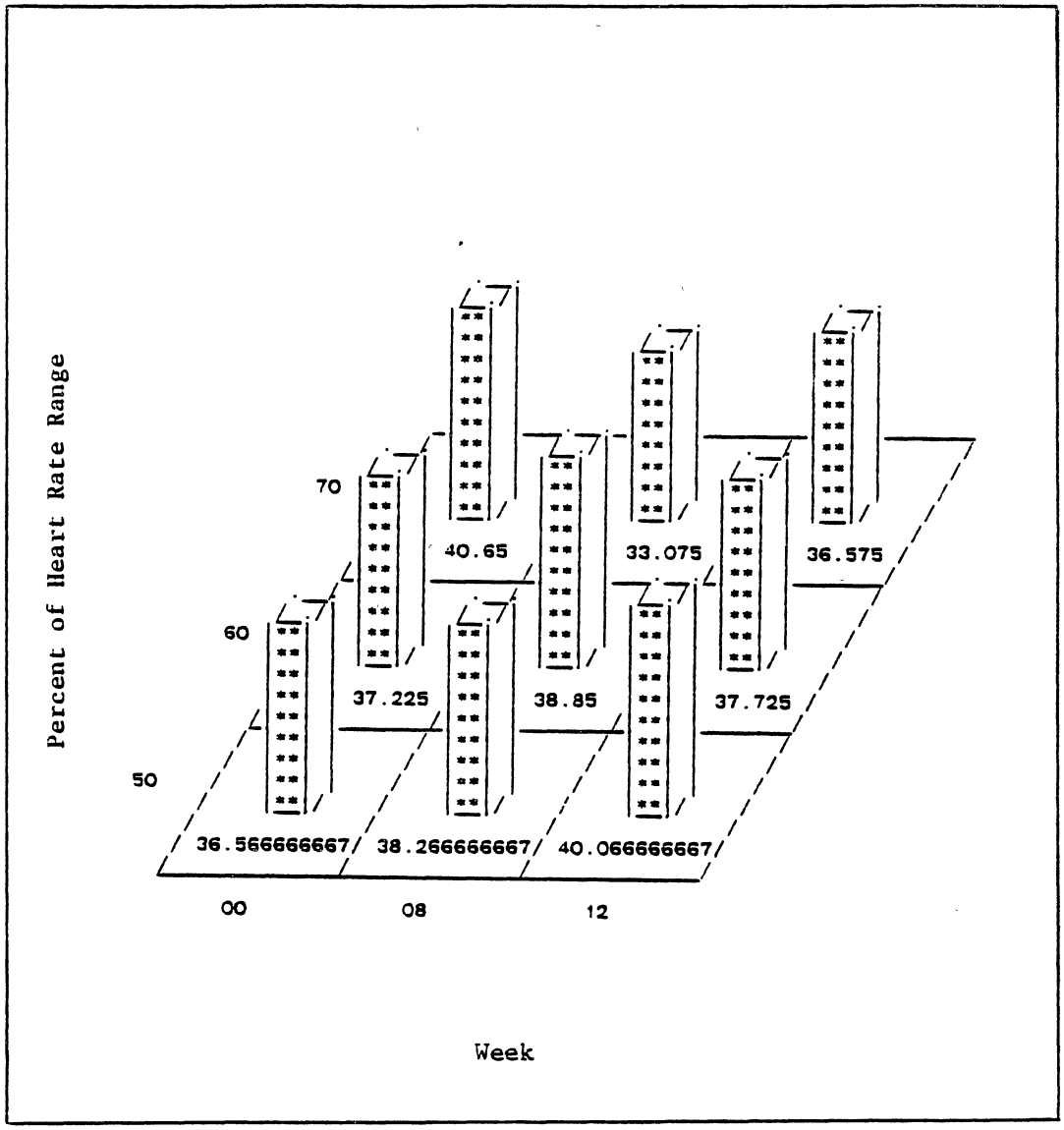

Figure 4. Mean HDL concentration.

at week 12. However, the final HDL level was still below the pre-exercise level.

The mean exercise sessions as shown in Figure 2 indicate a lack of any specific relationship to the overall fluctuation in HDL levels between or within groups.

One of the indicators of developing CHD is the ratio between HDL and total cholesterol (Cooper, 1981), as shown in Figure 5. There was an overall nonsignificant ($P < .10$) increase in the ratio, which reflects the significant decrease in total cholesterol and the nonsignificant increase in HDL.

In summary, it may be stated that total cholesterol decreased significantly over time, but showed no relationship to a negative caloric balance as other investigations have found. Other researchers have found that a relationship exists between consistent exercise patterns and increases in HDL levels. It is this investigator's opinion that the HDL levels and the ratio between HDL and total cholesterol did not increase significantly in this study because of inconsistent exercise habits.

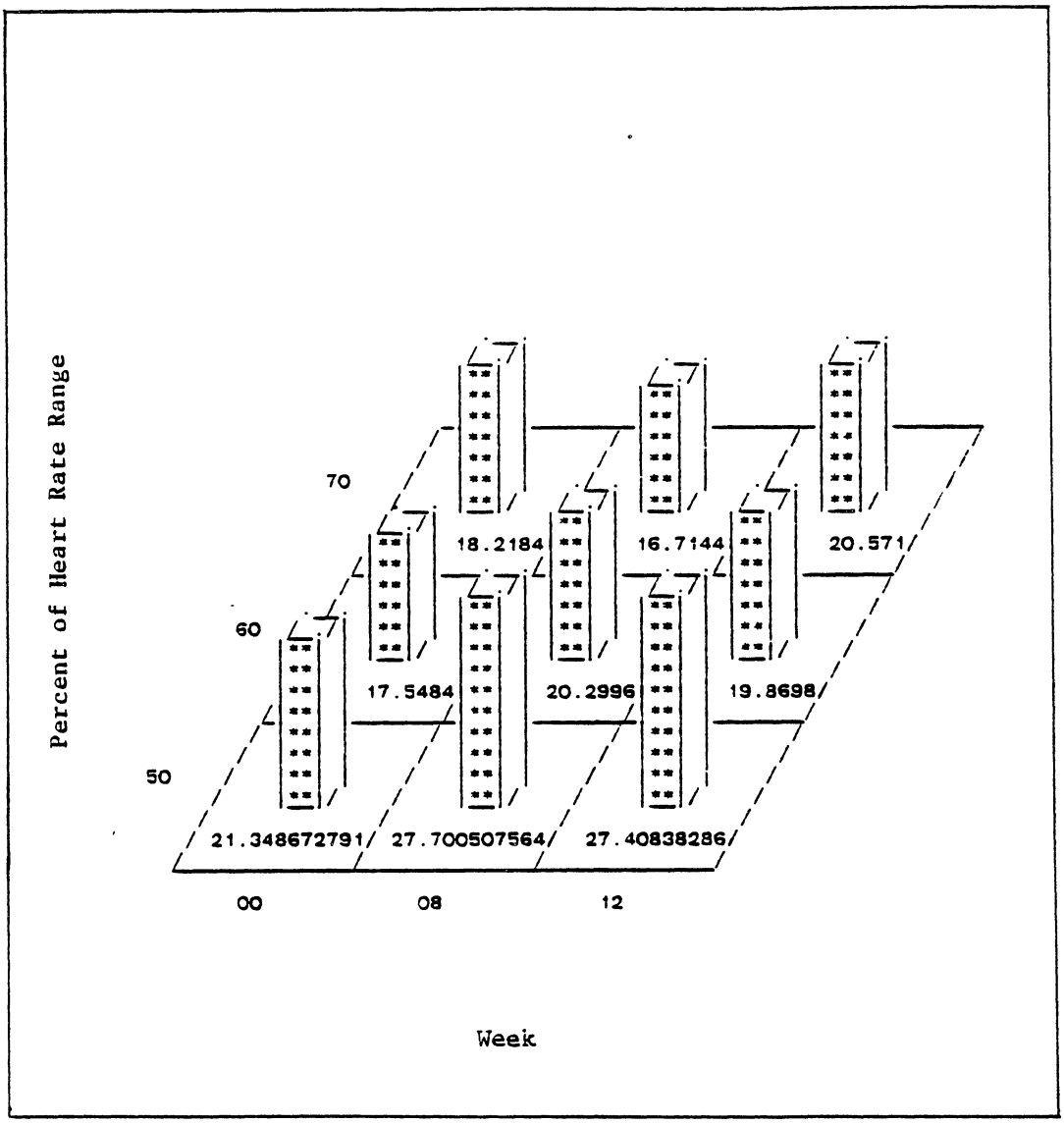

Figure 5. Mean ratio of HDL/total cholesterol.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

Coronary heart disease is one of the major medical and health concerns of the twentieth century. Through epidemiological studies, several major risk factors have been identified as being associated with the development of CHD. Epidemiological studies have also identified characteristics that are inversely related to the development of CHD.

Of the several risk factors which have been identified, cholesterol --which is carried by the lipoproteins in the plasma--has been singled out as one of the major contributors to atherogenesis. Cholesterol is divided into two major fractions: low-density lipoprotein and high-density lipoprotein. It has been observed that those individuals who suffer from a coronary event have high levels of LDL and low levels of HDL. The recent 10-year Primary Coronary Prevention Trial found that for every two percent decrease in blood cholesterol, there was a one percent decrease in the probability of developing CHD.

Coronary heart disease has also been found to be more prevalent in certain occupations. An inverse relationship has been found between physical activity level of the occupation and the occurrence of coronary events. The more sedentary the occupation, the greater the incidence of CHD.

Research in the 1950's and 1960's began to investigate the relationship between physical exercise and CHD. Initial investigations found

that those who exercised regularly had lower levels of total cholesterol. The mid-1970's found investigators looking at not only the total cholesterol levels of exercisers, but also at the high density lipoprotein level.

Most of the research concerning exercise and HDL levels has been performed on runners. The research has indicated that when individuals consistently run 11 or more miles per week, there may be a significant increase in their HDL levels. However, the intensity for these changes to occur has not been identified. Therefore, the purpose of this study was to determine the effect of intensity of bicycling on HDL levels.

Conclusions

The analysis of covariance showed no effect due to the adjustment of all subjects to the same percent body fat. Therefore, the conclusions were made using the analysis of variance.

1. The analysis of data showed there was no interaction (intensity by weeks) for the variables of HDL, ratio of HDL/total cholesterol, and total cholesterol. The hypothesis of no interaction was accepted.

2. There was no effect due to intensity for the variables of HDL, ratio of HDL/total cholesterol, and total cholesterol. The hypothesis of no effect due to intensity was accepted.

3. There was no effect due to time for HDL and ratio of HDL/total cholesterol.

    a. The hypothesis for HDL and the ratio was accepted.

    b. The hypothesis for no effect due to time for total cholesterol was rejected at the $P < .01$ level.

Subproblems

The subproblems are as follows:

1. In regard to total body fat, it was found that there was a decrease over time. This was a nonsignificant decrease.

2. In regard to maximal oxygen consumption, it was found that there was an increase over time. This was a nonsignificant increase.

3. In regard to body weight, it was found that there was a decrease over time. This was a nonsignificant decrease.

Recommendations

This study found that through participation in a stationary bicycle exercise program, the total cholesterol decreased significantly. However, the major emphasis of this study was to determine an exercise intensity for a significant change in HDL; this was not found. It is believed by this investigator that the lack of adherence to the exercise program was a major contributing factor in not finding a significant change in HDL.

Since consistent exercise habits are a vital portion of any exercise study, it is imperative that the volunteers, if not personally motivated to exercise, be externally motivated to participate. Therefore, it is recommended that the subjects be given released time from the jobs to participate; and/or if funds are available, pay them at the end of the study, providing they meet minimum participation criteria which were established at the beginning of the study. It is suggested that the subjects should not be retested unless they attend a minimum of 80 percent of the exercise sessions.

This investigator believes that a minimum participation is an essential ingredient for the success of a physical exercise study. However, a major problem may occur: the minimum participation requirement may reduce the retest number to a point where no subjects are qualified for retesting. To counter this, two suggestions are offered. The first, as previously dealt with, is motivation. Second, it is important to start with a sufficient number of participants, realizing that some may drop out. Recruitment of subjects is possible from beginning adult fitness classes, by advertising through local newspapers, and through speaking to civic clubs and faculty organizations. Whatever process is used, the important point is to gather a sufficient sampling of adults who are committed to exercising for the specified period.

Since stationary bicycles are becoming popular vehicles for exercising, more studies should be conducted using them to quantify physiological variables that are used in exercise prescription for American populations. The major problem with using large numbers of bicycles in research is the expense in obtaining them. The recent emphasis on bicycling as an avenue for exercising and health has led to the development of stands that support a standard ten-speed bicycle. It is suggested that future studies use stands to hold bicycles that could possibly be secured at a low cost through the manufacturers. Bicycles could be obtained from the pool of lost or stolen ones that police departments usually possess.

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APPENDIX A

PHYSICAL EXERCISE PROGRAM FORMS

OKLAHOMA STATE UNIVERSITY

HEALTH AND FITNESS CENTER

INFORMED CONSENT FORM

Subject's name_____ Date_____

I hereby authorize Dr. A. B. Harrison and/or such assistants as may be selected by him to perform the following procedure(s) and investigation(s):

A laboratory physical fitness evaluation including electrocardiogram, phonocardiogram, pulse waves, blood pressure, weight analysis, respiratory capacities and function and a treadmill walking test to predict maximal oxygen intake capacity,

on _____.
subject

The procedure(s) and investigation(s) has (have) been explained to me by Dr. A. B. Harrison or his assistant.

I understand that the procedure(s) and investigation(s) involve the following possible risks and discomforts:

All tests except the treadmill walk are resting tests and involve no unusual risk or discomfort. The treadmill test involves walking at a gradually increasing grade up to a target heart rate. The target heart rate is determined by age level, medical and physical condition. The EKG is monitored during the treadmill walk and the test is terminated upon signs of cardiac distress. The subject is free to terminate the test at any time at his own discretion.

I also understand that all test records will be kept confidential and will not be released to anyone without permission of myself or family. Test results will be tabulated for research purposes as group data and in no case will a subject's personal identity be associated with his test results without his express permission.

I understand that the potential benefits of the investigation are as follows:

The results of the test battery will give the subject an in depth view of his current fitness status. Test results will be explained and interpreted to the subject. Guidance concerning exercise programs will be given. Subjects will be encouraged to engage in a systematic exercise program to produce favorable changes in test scores.

Subject's signature_____

Witness_____

HEALTH AND FITNESS CENTER
Oklahoma State University

PHYSICIANS' APPROVAL FORM

To the physician of _____ Date_____

This subject has indicated a desire to participate in the physical fitness evaluation program offered by our Center. At the conclusion of this evaluation, we will make exercise recommendations based on the results plus your recommendations, if any. We expect the subject to have no medical conditions which would contraindicate participation in light to moderate exercise, and therefore, ask the subject to have this form signed by you.

Our evaluation consists of the following: (a) some resting tests, (b) some tests requiring minimal effort and (c) a progressive treadmill walk test to predict maximal oxygen uptake capacity. The exercise test is the Balke treadmill procedure with the speed being a constant walk of 3.0-3.4 mph and the elevation starting at 0% and being raised 1% each minute until a target heart rate is reached. The EKG is monitored continuously throughout the exercise test and the test is terminated upon signs of ST elevation, depression or unusual arrythmias. Maximal oxygen uptake is then predicted from a regression equation developed by Balke. Target heart rates are always sub-maximal heart rates. For deconditioned subjects, we use the suggested target heart rate published by Dr. Kenneth Cooper, M.D. The age-fitness targets are generally higher for a person in a good physical condition.

To the best of my knowledge, this patient is not suffering from any of the following:

1. manifest circulatory insufficiency ("congestive heart failure")
2. recent acute myocardial infarction
3. active myocarditis
4. recent embolism, either systemic or pulmonary
5. dissecting aneurysm
6. acute infectious disease
7. thrombophlebitis
8. ventricular tachycardia and other dangerous dysrhythmias (multifocal)
9. severe aortic stenosis
10. uncontrolled metabolic disease (diabetes, thyrotoxicosis, myxedema)

Copies of the subjects test results will be made available to you at the request of you or your patient.

THIS SUBJECT HAS MY APPROVAL TO PARTICIPATE IN THE PHYSICAL FITNESS EVALUATION. I UNDERSTAND THAT BY AFFIXING MY SIGNATURE BELOW, I DO NOT ASSUME ANY LEGAL LIABILITY FOR INCIDENTS ARRISING FROM THE FITNESS EVALUATION.

Signed,

_____, M.D.

_____
(City)

On the back of this sheet, please comment on any medical condition which would contraindicate participation in any phase of this evaluation or in a subsequent exercise program.

HEALTH AND FITNESS CENTER
Oklahoma State University

The following information is needed for our records and in assessing your current health and fitness status. By providing as much of this information as possible in advance, time will be saved during the evaluation. All information provided will be held in strict confidence.

NAME _____ DATE _____

ADDRESS: Street _____ City _____ State _____ ZIP_____

HOME PHONE _____ EMPLOYER _____

OCCUPATION _____ BUSINESS ADDRESS _____ PHONE _____

AGE LAST BIRTHDAY _____ BIRTH YEAR _____ Does your job require physical activity? _____

Do you currently smoke? _____ If so, what? _____ number/day _____
If not, have you ever smoked?_____ If yes, what? _____ no/yrs_____ yrs. quit_____

Do you ever drink alcoholic beverages? _____ If yes, approx. no.: less than 1/day _____
1-2 per day _____ 3 or more per day _____

Do you currently participate in any form of exercise on a regular basis?_____
Indicate no. of times/weekly of participation: walking _____ jogging _____ swim _____
golf _____ basketball _____ handball/racquetball _____ tennis _____ other (name)_____
If you walk, job or swim, please indicate distance and time covered each session and approximate pace _____

What is your estimate of your current medical condition? ex.___ good___ fair___ poor___
What is your estimate of your current physical fitness? ex.___ good___ fair___ poor___

Circle the number of blood relatives (parents, grandparents, brothers, sisters, that have been diagnosed as having some form of heart disease:
Under 60 years of age: 1 2 3 4 5 6 7 8 9 Over 60 years of age: 1 2 3 4 5 6 7 8 9

Have you ever been told that you have any form of heart disease? _____
Have you ever been told that you have diabetes? _____
Do you have blood relatives with diabetes? _____ If so, how many? _____

Do you consider yourself to be overweight? _____ If so, approx. how many lbs.? _____
Do you have any medical conditions (other than heart disease or diabetes) that might affect your exercise performance? _____ If so, please list _____

Who is your family physician? _____ City _____
Address, if known _____ date last medical exam _____
Would you like your stress test records sent to this physician? _____
If you would prefer to have your records sent to another physician, please list name and address _____

Are you currently taking any kind of medication? _____
If yes, is it non-prescription? _____ If so, name _____
If yes, is it prescription? _____ If yes, give name if possible _____

Have you ever been told that you had high cholesterol or high triglyceride levels in the blood? Cholesterol: yes___ no___ Triglyceride: yes___ no___

If you know your cholesterol and/or triglyceride levels, please list

Cholesterol _____ Triglyceride _____

APPENDIX B

SUBMAXIMAL BICYCLE TEST FORM

Table 6

Worksheet for Use in Determining Maximum Oxygen Uptake on Submaximal Test

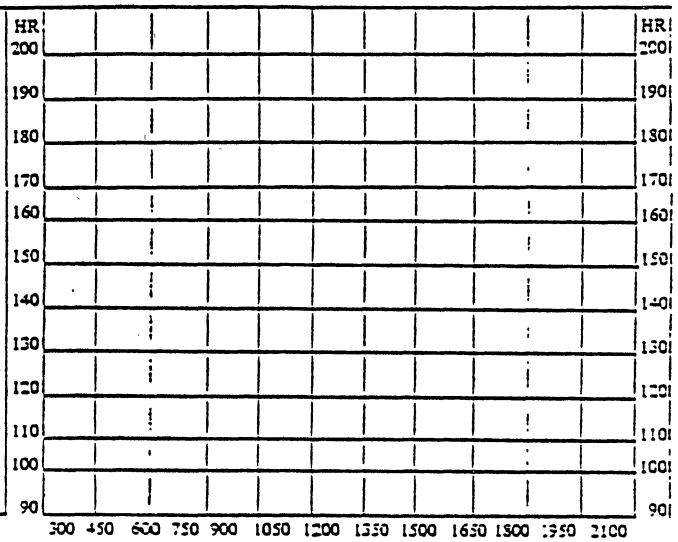
NAME _____ AGE _____ WEIGHT _____ LB _____ KG

DATE

Second Load HR[a]	Third Load HR	Maximum Work Load	Maximum Oxygen Uptake (L/mn)	Maximum Oxygen Uptake (ml/kg)
Test 1 _____ /	_____ /	_____	_____	_____
Test 2 _____ /	_____ /	_____	_____	_____
Test 3 _____ /	_____ /	_____	_____	_____

Directions

1. Plot the heart rate of the second and third loads versus the work (kpm/mn)
2. Determine maximum heart rate line from information on Table A.18.
3. Draw a line through both points and extend to the maximum heart rate line for age.
4. Drop a line from this point to the baseline and read the maximum oxygen uptake.



WORKLOAD (kpm/mn)	300	450	600	750	900	1050	1200	1350	1500	1650	1800	1950	2100
MAXIMUM OXYGEN UPTAKE (L/m)	0.9	1.2	1.5	1.8	2.1	2.4	2.8	3.2	3.5	3.8	4.2	4.6	5.0
KILOCALORIES USED (Kcal/m)	4.5	6.0	7.5	9.0	10.5	12.0	14.0	16.0	17.5	19.0	21.0	23.0	25.0

[a]Heart rate.

DIRECTIONS

1. Set the first work load at 300 kilopond meters per minute (1.0 kilopond).
2. If heart rate in third minute is: Less than (<) 90, set second load at 900 kilopond meters per minute (3 kilopond).
   Between 90 and 105, set second load at 750 kilopond meters per minute (2.5 kilopond).
   Greater than (>) 105, set second load at 600 kilopond meters per minute (2.0 kilopond).
3. Follow the same pattern for setting third and final load.

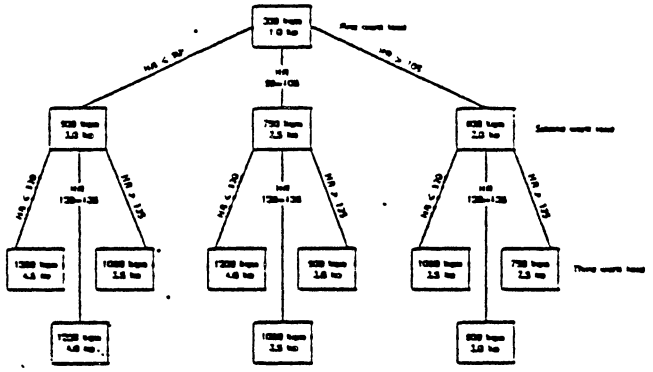


Figure 6. Guide to setting work loads for submaximal bicycle test.

APPENDIX C

COMMUNICATIONS TO PROGRAM PARTICIPANTS

Dear Exercisers:

Poor communications may be the beginning of many problems; problems that need not exist. Hopefully, this communication will clear up and answer questions that you may have or that may arise.

The major purpose of this exercise program is to determine what effect different exercise intensities have upon the total cholesterol (TC) and its fraction, high density cholesterol (HDC). Additionally, the ratio between TC and HDC is being investigated. The reason for the interest in cholesterol is its relationship to coronary heart disease and related ailments. It is believed that HDC is associated with a low incident of heart disease. Exercise is one factor that will increase HDC although the intensity is not exactly known.

In an attempt to determine at what exercise intensity HDC is increased, you will be divided into three separate exercise intensity groups. Exercise intensity is determined as a percentage of your heart rate range. The heart rate range is your resting rate minus your maximum rate. Therefore, your exercise groups will be set at 50, 60, and 70 percent of your heart rate range.

Exercise may help change your cholesterol along with your aerobic capacity if it is performed a minimum of three times per week for 20 to 30 minutes. Therefore, you can understand the importance of exercising three times per week (Monday, Wednesday, Friday) during this 12 week program. Of course it is probable that you will have to miss a session during the week. If this happens, please try and make it up on Tuesday, Thursday, Saturday, or Sunday. Additionally, the exercise will be incrementally increased from 10 minutes to 30 minutes, hopefully by week 6 or 8, depending on your adaptability.

So far it has been a fun learning experience working with you all. I can only expect things to get better. If I can be of any help or answer questions, please call me at 624-8736 (H) or 624-7556 (O).

Thanks again,

Tom Gibson

Dear Exercising Cyclist:

This is it, the final week of your exercise program. You will continue to exercise normally this week. Next week, post tests will be given.

On Monday, 20 June '83, you will have a 12-hour blood sample drawn between 7-8 a.m. It is imperative that you have your blood drawn on Monday as post bicycle tests will be given on Tuesday, 21 June. This test will be the same as the initial bike test which you participated in. Results will be placed in your mail boxes in approximately two weeks.

I realize that some of your work schedules may conflict with the above test periods. I will certainly work around your schedule as you have worked with me for the past twelve (12) weeks.

There will be a sign-up schedule posted on the bulletin board for blood drawing times on Monday (other than between 7-8 a.m.) and for bicycle testing times on Tuesday.

Again, thank you for your help and I hope you have gained some important knowledge concerning exercise and your health status. I will be happy to work with each of you in developing a continuing exercise program for your specific needs and lifelong goals.

As a final note, please remember to turn your fee cards and locks in at the conclusion of the program.

Tom Gibson

APPENDIX D

HEART RATES FOR EXERCISE PRESCRIPTION

Heart Rate Range

Exercise intensity may be prescribed by use of the heart rate range method. The basis for this is that a linera relationship is normally present between the intensity of exercise and the heart rate. Deviation from this relationship will occur due to environmental conditions, psychologic stimuli or disease (American College of Sports Medicine, 1980).

Calculation of Heart Rate Range

Maximal Heart Rate	=	190
Resting Heart Rate		-60
		130
Exercise Intensity		.60%
		78
Resting Heart Rate		+ 60
Exercise Training Heart Rate		138

APPENDIX E

RAW DATA

Table 7

Raw Data

Wk	Subj	Group Stress Level (HRR)	Age	Wt	Percent Fat	TOT Chol	HDL	Ratio HDL/ TOT	MaxVO$_2$	Exercise Session
0	1	50	30	175	15.2	133.0	26.9	20.22	27.8	0
0	2	50	29	181	20.0	212.0	53.9	25.42	37.0	0
0	3	50	26	251	24.0	157.1	28.9	18.39	20.8	0
8	1	50	30	.	.	113.3	35.0	30.89	.	1.15
8	2	50	29	.	.	166.6	42.0	25.10	.	1.63
8	3	50	26	.	.	140.0	27.8	17.00	.	1.25
12	1	50	30	170	13.2	106.0	37.0	34.90	27.0	1.42
12	2	50	29	176	18.0	206.0	44.0	21.35	33.0	1.25
12	3	50	26	246	22.8	151.0	39.2	25.96	31.4	2.30
0	1	60	40	194	20.3	243.5	47.6	19.54	23.3	0
0	2	60	39	155	19.1	181.8	52.0	28.60	34.6	0
0	3	60	32	194	26.1	251.4	16.4	6.52	37.0	0
0	4	60	23	238	29.1	212.0	32.9	15.51	31.6	0
8	1	60	40	.	.	246.6	50.4	20.43	.	2.13
8	2	60	39	.	.	166.0	51.8	31.20	.	1.25
8	3	60	32	.	.	180.0	23.8	13.22	.	2.88
8	4	60	23	.	.	180.0	29.4	16.33	.	1.38
12	1	60	40	193	17.7	256.3	49.1	19.15	36.6	1.92
12	2	60	39	159	16.8	150.0	48.3	32.20	48.6	1.83
12	3	60	32	182	21.6	196.7	18.5	9.40	35.9	1.92
12	4	60	23	233	29.1	187.0	35.0	18.71	25.7	1.17
0	1	70	23	216	24.3	193.9	35.9	18.51	33.0	0
0	2	70	49	174	25.3	254.5	60.3	23.69	23.0	0
0	3	70	46	229	26.2	220.0	46.0	20.90	17.3	0
0	4	70	32	232	26.2	209.1	20.4	9.75	26.2	0
8	1	70	23	.	.	220.0	35.0	15.90	.	1.25
8	2	70	49	.	.	201.0	42.0	20.89	.	3.00
8	3	70	46	.	.	193.3	31.5	16.29	.	2.00
8	4	70	32	.	.	173.0	23.8	13.75	.	2.13
12	1	70	23	217	24.1	206.0	30.8	14.95	30.1	1.25
12	2	70	49	169	22.8	162.5	37.1	22.83	29.4	3.00
12	3	70	46	224	22.3	187.5	32.9	27.54	26.8	1.58
12	4	70	32	226	22.8	168.8	45.5	26.95	29.3	1.25

VITA

Thomas Livingston Gibson

Candidate for the Degree of

Doctor of Education

Thesis: THE EFFECTS OF EXERCISE INTENSITY ON PLASMA LIPOPROTEINS DURING A STATIONARY BICYCLE TRAINING PROGRAM

Major Field: Higher Education

Minor Field: Health, Physical Education, and Recreation

Biographical Data:

Personal: Born in Oklahoma City, Oklahoma, January 18, 1942.

Education: Graduated from John Marshall High School, Oklahoma City, Oklahoma, in 1960; received the Bachelor of Science degree in Forestry from Oklahoma State University in 1969; received the Master of Science degree in Recreation from State University of New York, Cortland, New York, in 1976; completed requirements for the Doctor of Education degree at Oklahoma State University in December, 1985.

Professional Experience: I worked in the field of outdoor adventure education from 1974, until returning to Oklahoma State University in 1979 to begin working toward the Doctor of Education degree in Exercise Science. At Oklahoma State University, I was involved in working in the Human Performance Laboratory as an intern and as a graduate assistant. I belong to the American College of Sports Medicine, and I am certified as an Exercise Test Technologist.