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The effects of Bios Life and exercise on total cholesterol, serum low-density lipoprotein, and high-density lipoproteins

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THE EFFECTS OF BIOSLIFE AND EXERCISE ON TOTAL CHOLESTEROL, SERUM LOW DENSITY LIPOPROTEIN, AND HIGH DENSITY LIPOPROTEINS

by

Lori Jan Inderlied-Rucks

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A thesis submitted in partial fulfillment
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ABSTRACT

The Effects of Bios Life and Exercise on Total Cholesterol, Serum Low Density Lipoprotein, and High Density Lipoprotein

by

Lori Jan Inderlied-Rucks

Lawrence A. Golding, Ph.D. Examination Committee Chair, Distinguished Professor of Kinesiology, University of Nevada, Las Vegas

The present study was designed to determine the effects of Bios Life, a non-prescription dietary fiber supplement, on serum lipoproteins and determine whether exercise accentuates this effect. Fifty male and female subjects (ages 30-60) participated in the study. Twenty-five subjects were exercising regularly in a supervised exercise program and the other twenty-five were sedentary. All participants where required to have a LDL-C of 130 mg/dl or higher and none were on any cholesterol lowering medications. Blood was drawn three times at baseline, before the study started, 45 days into the program and at 90 days which was the end of the study. Total cholesterol, HDL and LDL's were analyzed by the Cholestech L.D.X. system. A mixed model ANOVA was used to analyze the data. The results indicated significant decreases in total cholesterol from baseline to 45 to 90 days ($F = 18.29$, $p<0.05$) with no significant difference between exercise and non-exercise groups ($F = 0.20$, $p>0.05$). The results also indicated significant decreases in LDL's from baseline to 45 to 90 days ($F = 21.60$, $p<0.05$) with no significant difference between exercise and non-exercise groups ($F =$

1.59, $p>0.05$). The analysis of the HDL's yielded no significant difference between exercise and non-exercise groups ($F = 1.07$, $p>0.05$) nor was there a significant difference from baseline to 45 to 90 days ($F = 1.67$, $p>0.05$). These results suggest that whether or not one participates in an exercise program Bios Life will decrease total cholesterol and LDL's equally without changing HDL's, which may help reduce the risk of coronary heart disease.

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CHAPTER 1

INTRODUCTION

Coronary heart disease (CHD) is the leading cause of death in the United States. The American Heart Association (AHA) statistics show that in 1993 there were 489,970 deaths from CHD. In 1998, as many as 1,500,000 Americans had a new or recurrent heart attack and about one third died. In 1993 artherosclerosis was the underlying cause of many of the 639,710 heart attack and stroke deaths. In the U.S. approximately 96 million adults have total blood cholesterol levels of over 200mg/dl and 37.8 million have levels greater than 240 mg/dl according to the AHA. These high levels are a major contributor to artherosclerosis and other types of coronary artery disease. Due to its lifestyle etiology, CHD is expected to remain the leading cause of death for future years. These statistics prove that CHD is a major public health problem and concern (Anderson, Zettwach, Feldman, Clark, Oeltgen & Bishop, 1988).

It has been well documented that both high levels of total serum cholesterol and specifically low-density lipoproteins (LDL-C cholesterol) are linked with coronary heart disease (Bridges, Anderson, Deakins, Dillon & Wood, 1992). From the epidemiological data from the Framingham Heart Study elevated total serum cholesterol was determined to be a major coronary risk factor. Larosa and associates (1990) studied 2,282 men and 2,845 women investigating CHD history and total cholesterol levels and concluded that there was definite evidence that high levels of total cholesterol were associated with a

high incidence of CHD and that low levels of total cholesterol were associated with a low incidence of CHD (Larosa, Becker & Fitzgerald, 1990).

The National Cholesterol Education Program (Expert Panel, 1993) recommends the following values for cholesterol in individuals to be:

Total Cholesterol	< 200mg/dl
HDL	> 35mg/dl
LDL	< 130mg/dl

Individuals with 200-239 mg/dl are considered borderline risk and those with total cholesterol greater than 240 are at abnormal risk. Increasing HDL and decreasing LDL reduces the risk of CHD (Anderson, Riddell-Mason, Gustafson, Smith & Mackey, 1992). A lowfat diet was initially the main method of reducing total cholesterol. New cholesterol lowering drugs are now commonly prescribed. Cholesterol lowering drugs were classified into bile acids, sequestrates, nicotinic acids, statins, fibric acids and probucol (Anderson et al., 1988). The goals of drug therapy was to lower total cholesterol, raise HDL's and lower LDL's. However, there were several major side effects. The side effects can range from itching of the skin, gastrointestinal distress, liver toxic effects, and muscular damage due to interaction between drugs and blood clots (JAMA, 1993).

Dietary therapy remains the first choice for treatment of high cholesterol. There is evidence indicating that dietary fiber has important lipid lowering effects and may reduce risk of CHD (Bell, Hectorn, Reynolds & Hunninghake, 1990). Increasing Dietary fiber intake is an important therapeutic treatment for other specific conditions besides CHD such as diabetes, hyperlipidemia, hyperglyceridemia, hypercholesterolemia and

intestinal disorders (Anderson, Gilinsky, Deakins, Smith, O'Neal, Dillion & Oeltgen, 1991). Individuals consuming a diet high in fiber tend to have lower incidence of myocardial infarction and sudden cardiac death (Haskell, Spiller, Jensen, Ellis & Gates, 1992).

Dietary fiber is defined as "the endogenous components of plant materials in the diet that are resistant to digestion by enzymes produced by man" (Anderson, 1987). There are two main types of fibers soluble and insoluble. In experimental studies, soluble fiber has been shown to reduce blood cholesterol levels. Soluble fiber increases the fecal excretion of bile acids, which may alter the quantity of cholesterol absorbed by the intestine. The fibers from fresh fruits, vegetables, legumes, oat bran and barley appear to have the most significance for reducing cholesterol levels (Haskell et al., 1992). Insoluble fiber, such as, wheat bran have essentially no effect on serum cholesterol levels.

The general purpose of dietary fiber as therapy is to reduce elevated serum cholesterol levels. This is achieved primarily by reducing fat intake and consuming a healthy diet, rich in fiber. Dietary therapy in conjunction with regular exercise is considered an essential element in therapy of elevated serum cholesterol (Ready, 1996). Exercise has been shown to help in the promotion of weight reduction which enhances LDL cholesterol lowering, reducing triglyceride, raising HDL, reduces blood pressure and decreasing the risk for diabetes mellitus (Grundy, 1986). Therefore, the combination of regular exercise and diet rich in fiber should lower total cholesterol, increase HDL, decrease LDL and consequently reduce the risk of CHD.

The purpose of this study was to investigate the effects of Bios Life, a non-prescription dietary fiber supplement, on Total, LDL cholesterol and determine whether

exercise accentuates this effect. The null hypothesis is that exercise does not accentuate the effect of Bios Life on Total Cholesterol, Low-Density Lipoproteins and High-Density Lipoproteins and the alternate hypothesis is that exercise accentuates the effect of Bios Life on Total Cholesterol, Low-Density Lipoproteins and High-Density Lipoproteins.

CHAPTER 2

LITERATURE REVIEW

An adult male weighing 70kg would have approximately 140g of cholesterol present in the body with 8g of this in the plasma (Sabrine, 1977). The average synthesis of cholesterol in the body is about 1000mg per day. The daily production is approximately 400mg from intestinal absorption and 600mg synthesized in the cells and the liver. An adult consuming a normal American diet ingests about 1000mg per day. Every cell of the body, except a mature red blood cell, produces cholesterol (Sabrine, 1977). The liver is a major organ in cholesterol synthesis. The liver must synthesize and maintain control over the enzymes that convert acetyl coenzyme A to cholesterol. More than ninety percent of the cholesterol in the body is found in the cell membrane.

Chemical Nature of Cholesterol

At the end of the eighteenth century a French chemist de Fourcroy described the compound now known as cholesterol by isolating a crystalline substance from the alcohol-soluble fraction of human gallstones (Sabrine, 1977). About the same time another French chemist Chevreue detected it in human and animal bile (Sabrine, 1977). After the turn of the century another French chemist Lecanu found cholesterol in the blood of humans (Sabrine, 1977). De Fourcroy's gave the name cholesterine from the Greek words Chole meaning bile and Steros meaning solid (Sabrine, 1977). The correct

empirical formula ($C_{27}H_{46}O$) was published in 1888 by Reinitzer (Newsholme & Leech, 1983).

Cholesterol is probably the best known sterol, which is essential to the human body. "Cholesterol is found associated with the fats, but chemically it is not related to them. Cholesterol, a white waxy solid, is the principal sterol found in animal organisms" (Smolin & Grosvenor, 1997). Most of the cholesterol found in the body occurs in free form, the unesterified alcohol, which is precipitable by digitonin. The so-called bound cholesterol is found in much smaller portions; it is present as the ester of long chain fatty acids. The largest amount of cholesterol is found in muscle, nervous and connective tissue. It is needed to synthesize vitamin D, cholic acid which is part of bile, some hormones, and cortisol which promotes glucose synthesis in the liver (Smolin & Grosvenor, 1997).

Production, Absorption, Transportation and Excretion

Cholesterol is not soluble in water therefore it can not enter the blood stream directly. Lipoproteins called chylomicron are formed to help cholesterol enter the blood stream (Williams, 1997). Chylomicrons are formed by combining triglycerides with cholesterol, free fatty acids, phospholipids, traces of fat soluble vitamins, steroid hormones and lipoproteins (Williams, 1997). Once chylomicrons are produced they will carry lipids from the intestines and deliver triglycerides to body cells. On the surface of cells, lining the blood vessels is lipoprotein lipase which breaks down the triglycerides too fatty acids which can be either used as fuel or resynthesized into triglycerides for storage. After the breakdown of chylomicron cholesterol and protein remain and returned to the liver (Smolin & Grosvenor, 1997).

Crystalline cholesterol administered orally is absorbed in only small amounts unless some fatty material is also present in the intestine. This is not true with colloidal or amorphous cholesterol, which can be absorbed in the absence of dietary fat. Bile and pancreatic juices are said to be two agents, which aid in the absorption of cholesterol, because the combination of cholesterol with bile acids increase the solubility of cholesterol in intestinal fluids. Pancreatic and intestinal enzymes hydrolyze cholesterol esters, which are later re-synthesized before reaching the lymph stream, the main route by which cholesterol is absorbed. Some cholesterol is absorbed directly into the blood stream; part of the absorbed free cholesterol, but not the ester, is excreted in the bile; part is changed to coprosterol and eliminated later in the feces (Smolin & Grosvenor, 1997).

Lipids are produced in the liver, by breaking down excess protein, carbohydrate or alcohol to produce either triglycerides or cholesterol. In the liver triglycerides, cholesterol, fatty acids and returned chylomicron are formed into very-low-density lipoprotein (VLDL) (Smolin & Grosvenor, 1997). VLDL carries a large lipid content, but also contains about ten to fifteen percent cholesterol formed in the liver from endogenous fat stores (Williams, 1997). VLDL transport lipids out of the liver and delivers triglycerides to body cells. They must be broken-down by the enzyme lipoprotein lipase so that fatty acids can be taken up by the cells (Smolin & Grosvenor, 1997).

Triglycerides are removed from VLDL leaving a smaller intermediate density lipoprotein (IDL) which contains mostly cholesterol. These IDL's are either transported to the liver or formed into low-density lipoproteins (LDL's). LDL's are lipoproteins that transport cholesterol to cells. For LDL's to be taken up by the cells, apoliprotein B, must

bind with the LDL receptor. A LDL receptor is a protein on the surface of all cells, which binds to LDL particles and allows their contents to be taken up for uses by the cell (Smolin & Grosvenor, 1997).

The body can not breakdown cholesterol esters, so it must be transported back to the liver where then it will be eliminated from the body as bile. This process is done by high-density lipoprotein (HDL). HDL's are manufactured in the intestinal tract. They are then transported through blood to pick up cholesterol from cells so that the body can excrete it (Smolin & Grosvenor, 1997).

Metabolism

The synthetic process of cholesterol takes place in the liver and may occur in other organs. In all tissues except in the brain, cholesterol is continually regenerated. If any organ can be singled out as the single most important to metabolize cholesterol and some amount of plant sterols, it is the liver. It can both synthesize and destroy cholesterol. The breakdown and lose of cholesterol can occur in four ways:

1. By direct reduction to dihydrocholesterol or by passage through the intermediate cholesterone to dihydrocholesterol or coprosterol.
2. By loss of cholesterol in the feces.
3. By conversion into steroid hormones.
4. By conversion to bile acids in the liver.

Cholesterol and Age

There are a number of variables that are important in determining or changing the level of blood cholesterol in the body. Throughout the day the level of serum cholesterol remains for the most part constant, even though there is a large variation in the rate that

cholesterol enters and leaves the body (Winkel, Stratland & Bokelund, 1974). Over an extended period of time the amount of exdogensis cholesterol entering the body compared to cholesterol degradation will rise significantly (Hollister & Wright, 1956). This significant rise will eventually reach abnormal level of cholesterol (Hollister & Wright, 1956).

Females at birth have slightly higher levels of plasma cholesterol than males. During childhood, adolescence and early adulthood there is very little difference in blood cholesterol between men and women (Syerberg & Hjorne, 1973). In later life males tend to have higher serum cholesterol levels due to higher concentrations of VLDL and LDL. Serum cholesterol levels tend to be lower in premenstrual women. After the age of 50 male cholesterol levels tend to lower as females tend to raise slightly (Adlersberg, Schaefer, Steinberg & Wang, 1956). This rise in serum cholesterol levels in women is due to the fact that women going through menopause have lower levels of estrogen. Estrogen is a cholesterol-lowering agent and the decrease of estrogen during menopause is the cause of rising serum cholesterol levels in women.

At birth plasma levels of cholesterol are usually low around 80mg/dl (Darmady, Fosbrooke & Llyod, 1972). Glueck, Heckman, Schoenfeld, Steiner and Pearce, (1971) studied the umbilical cord blood cholesterol in 1800 consecutive unselected live births and found it to be about 80mg/100 ml. After birth cholesterol rises rapidly to around 183 mg/100ml for the average male 20 to 29 years of age depending mostly on diet (Fredrickson, Levy & Lees, 1967). Throughout life, cholesterol levels continue to rise slowly (Darmady, Fosbrooke & Lloyd, 1972). At about 50 years of age serum

cholesterol levels begin to plateau off. In men the level begins to drop slightly and in women blood cholesterol may continue to rise after 50 (Fredrick, Levy & Lees, 1967).

Adlerberg and associates (1956) studied approximately 1,200 healthy males and females between the age of 2 and 77 years. Their blood serum was analyzed for cholesterol in order to establish average lipid levels. It was found that in the age groups 3-12 and 53-57, the females have significantly higher cholesterol levels than men, whereas, in age groups 28-42 males have significantly higher levels than women. Adlersberg also compared the serum cholesterol levels between males and females at different age groups. The total serum cholesterol level of the males remained constant from age 2 throughout 19. From the age 20 to 33 there was a significant increase of total cholesterol level. No change was seen until after age 60. The total serum cholesterol level of the females did not change significantly from age 2 through 32. From age 33 to 58 a significant increase of 3.2 mg per 100cc per year was seen. The difference in the serum cholesterol levels at the different age-trends between males and females may be the result from evidence showing that males at a young age start to show signs of coronary artery disease and among women they tend to show signs of coronary artery disease after the age of 50 around the time of menopause (Adlersberg, Schaefer, Steinberg & Wang, 1956).

Diurnal and Seasonal Variations

Several studies have examined seasonal fluctuations of serum cholesterol in man. Fyfe, Dunnigan, Hamilton and Rae (1968) studied the seasonal variation of 5,630 serum cholesterol and serum triglyceride on patients with confirmed or suspected ischemic heart disease. Serum cholesterol levels were at their peak in the spring, and fell progressively

to the lowest level in autumn. Serum Triglyceride did not exhibit this kind of seasonal fluctuation. This study concluded that the highest level of serum cholesterol occur in the spring and the lowest in the autumn (Fyfe et al., 1968).

Cholesterol and Heart Disease

Atherosclerosis is the major disease affecting the heart and blood vessels, and is the leading cause of death in the United States (Bierman & Chait, 1988). According to the International Atherosclerosis Project; observations were made on 19 different countries and race groups concluding that "practically all persons have some degree of atherosclerosis when examined at autopsy" (McHill, 1968). Atherosclerosis is a progressive disease throughout life. The severity which it develops seems to be due primarily to environmental conditions (Gresham, 1972). Since the 1960's the number of deaths from coronary artery disease has been declining at a fast rate but still nearly half of the deaths in this country are from blood vessel diseases (Lipid Research Clinics Program, 1984).

Atherosclerosis is the result of fatty cholesterol deposits in blood vessel walls; reducing elasticity and eventually blocking blood flow (Smolin & Grosvenor, 1997). It is a disorder of the coronary arteries, cerebral arteries, iliac and femoral arteries and aorta that is responsible for coronary heart disease, stroke and peripheral arterial disease (Bierman & Chait, 1988). Atherosclerosis is characterized by the collection of lipids in smooth muscle cells, macrophages of the inner lining of the walls of arteries (Bierman & Chait, 1988).

In 1833 Lobstein introduced the word atherosclerosis, athero meaning gruel or musk and scleroses meaning hardening of the arteries (Katz & Stamler, 1953). Michael

Brown and Joseph Goldstein showed the connection between the discovery of LDL receptors on cells with the development of atherosclerosis (Smolin & Grosvenor, 1997). LDL receptors bind to LDL particles and help them to be absorbed by the cell. A high level of LDL means there is too much cholesterol or not enough LDL receptors cells, this leads to cholesterol deposits in the artery walls causing atherosclerosis (Smolin & Grosvenor, 1997).

Even though extensive studies have been done on atherosclerosis, there is still no known cause of the exact events which initiate the buildup of cholesterol in arterial walls (Grundy, 1990). One theory is an injury to the arterial wall is caused by high blood pressure, viruses, chemicals or some other factor (Brown & Goldstein, 1984). Another theory suggests that the presence of high levels of blood cholesterol causes injury to the arterial wall (Brown & Goldstein, 1984). Inside the artery wall, as the injury occurs, oxidized LDL cholesterol binds to scavenger receptors located on the surface of the white blood cells. The white blood cells fill up with the oxidized LDL cholesterol and burst depositing cholesterol to form a fatty streak inside the artery wall. This process leads to an excessive amount of cholesterol, smooth muscle cells and fibrous tissue called plaque. The plaque will collect calcium causing it to became hard. Blood clots will form around the plaque causing the artery to narrow and lose it's elasticity. The artery becomes blocked and will no longer allow blood flow to supply oxygen and nutrients to the cell, leading to the death of cells (Smolin & Grosvenor, 1997).

Atherosclerotic lesions in the coronary arteries lead to CHD, which is the most common and most serious of cardiovascular diseases in middle-aged adults (National Research Council, 1989). If atherosclerosis causes interference with blood flow to the

myocardium a Myocardial Infarction results. One third of all myocardial infarction cases, coronary artery occlusion is the cause of death in myocardial cells (National Research Council, 1989). Patients may also suffer from failure of the heart to pump sufficient blood, known as congestive heart failure, or irregular heart beats, known as arrhythmias. The presence of severe lesions in the coronary arteries will often cause angina pectoris, especially on exertion (National Research Council, 1989).

Cholesterol has received much attention, especially in view of the well known clinical fact that certain diseases and syndromes often associated with hypercholesteremia, including diabetes mellitus, the nephrotic syndrome, myxedema, and familial hypercholesteremia, predispose to premature atherosclerosis. In spite of the fact that cholesterol levels are often above 260 mg in persons with atherosclerosis, there is evidence that a high proportion of people develop atherosclerosis with blood levels in the normal range of 125 to 260 mg (Barry, 1997).

Incidence of CHD varies widely from one country to another, which sparked the interest of researchers to answer the question of why cholesterol in blood and coronary heart disease is so different between countries (Frayn, 1996). Researchers started by looking at international coronary artery disease mortality data and autopsy records from cross-population studies which provided evidence supporting a significant association between CAD and dietary fat intake (Caggiula & Mustad, 1997). The Seven-Country study and the Japan-Honolulu-San Francisco study established the importance of the fatty acid composition of dietary fats with the link of increased rates of CHD.

In the "Seven Country Study" dietary intake of the participants was done by a 7-day food record and then a sample of each food consumed was chemically analyzed. The

results showed in Finland and the United States total fat intake was 40% of energy and in Japan it was 20% of energy. Saturated Fatty Acid (SFA) intake ranged from 20% in the United States to less than 10% of energy in Japan. This study was the first to show a strong correlation between SFA, coronary artery disease and death. This study showed that coronary artery disease rates were low despite moderately high total fat intakes, especially when SFA intakes were high (Keys, 1970).

Three middle-aged men of Japanese ancestry living in either Japan, Honolulu or San Francisco participated in a study observing the relation between SFA, cholesterol and heart disease. The intake of total fat differed significantly among the three different cultures. In Japan the percent of energy from fat was 15%, in Honolulu it was 33% and San Francisco participants SFA intake ranged from 7%, 13% and 38% (Kato, Tilloston, Nichaman, Rhodes & Hamilton, 1973). Coronary artery disease mortality was 1.7 times higher in Hawaii and 2.8 times higher in San Francisco than in Japan. Although no single dietary factor existed that is associated with coronary artery disease between and within all different countries the mortality in these populations SFA and cholesterol are generally positively correlated with coronary artery disease (Kato et. al., 1973).

There are specific differences in death rates from coronary artery disease among most industrialized countries (Shils & Young, 1988). The highest rate of CAD in males between 35 and 74 years is seen in Finland and the United States. Eastern Europe and Japan are about one fifth of that in the United States. These differences in CAD maybe due to genetic factors, but as seen with Japanese migrants to the United States they have rapidly accumulated the risk of artherosclerosis through western diet and lack of exercise (Shils & Young, 1988). Evidence suggests that cultural and environmental factors

including diet may have an important part in CAD, but genetics probably explains the difference in CAD among individuals from the same ethnic and cultural background (Shils & Young, 1988).

There are a number of conditions and habits that are correlated with the incidence of CAD and these have been termed "coronary risk factors" (Dawber, 1975). Non-reversible risk factors or otherwise known as primary risk factors are aging, male sex and genetic traits: i.e. positive family history of premature atherosclerosis (Dawber, 1975). Potentially reversible risk factors or Secondary risk factors are: cigarette smoking, hypertension, obesity, hyperlipidemia, hypercholesterolemia, hyperglycemia, diabetes mellitus, low levels of HDL's and sedentary lifestyle (JAMA, 1993). Other possible risk factors are body build, emotional stress and personality type (JAMA, 1993).

Hyperlipidemia consists of increased plasma levels of cholesterol and/or triglycerides (Dawber, 1975). The elevated plasma lipid levels result from one or more abnormalities of lipid metabolism or transport (Dawber, 1975). Hypercholesterolemia and hypertriglyceridemia seem to play an important role in the development of atherosclerosis.

Hypercholesterolemia is associated with increased incidence of premature CAD, but it's importance varies in relation to age. The incidence of myocardial infarction in individuals between the ages of 30 and 49 with cholesterol levels greater than 260 mg/dl was three to five times higher than in individuals with cholesterol levels less than 200 mg/dl (Bierman & Ross, 1977).

Hypertriglyceridemia has a significant relationship to CAD due to increased triglycerides and very low density lipoproteins (VLDL) (Assmann & Schulte, 1992).

Individuals with elevated VLDL levels may have an increased risk for premature atherosclerosis due to the fact that high levels of VLDL will be transformed into LDL and an even higher risk if the individual has other risk factors such as diabetes, smoking and hypotensive (Assmann & Schulte, 1992).

Goldstein (1973) studied the role of genetics of hyperlipidemia in atherosclerosis in 500 survivors of myocardial infarctions. The study showed that approximately one half of the males and two thirds of females below the age of 50 had either hypertriglycerdemia, hypercholesterolemia or both. In the individuals over 70 the presence of atherosclerotic coronary disease was high, but no males and one fourth of the females had hypertriglycerdemia or hypercholesterolemia. It appears that hyperlipidemia is more of a risk factor in individuals below the age of 50, and for men and women over the age of 65, however, there is no evidence to support a correlation between hyperlipidemia and atherosclerosis (Goldstein, Hazzard, Schrott, Bierman & Motulsky, 1973).

Cholesterol and Fiber

Treatment of high total blood cholesterol begins with dietary therapy. The goal of dietary therapy is to lower LDL-cholesterol to levels under 130 mg/dl if definite CHD or two other CHD risk factors are present (Expert Panel, 1993). The general purpose of dietary therapy is to reduce elevated cholesterol levels while maintaining a healthy adequate diet. Modification of the patient's diet is an essential element of therapy (Expert Panel, 1993). One of the important modifications to help reduce high levels of cholesterol through diet is to increase the intake of dietary fiber (Anderson et al., 1991).

The effect of fiber in various disease conditions has been studied for several years. British scientists in the 1960's concluded that many diseases and disorders of the Western society were related to a low intake of dietary fiber (Council on Scientific Affairs, 1989). They compared Western cultures to rural African blacks and found that cancer of the colon and gastrointestinal disorders were rare in this society due to high-fiber diets and production of soft stools in large volume (Council Scientific Affairs, 1989). In Western cultures where life-styles and diet contain high amounts of saturated fat and low amounts of fiber the incidence of heart disease is much higher (Council Scientific Affairs, 1989).

Dietary fiber has been shown to have an important lipid lowering effects and may reduce risk for coronary artery disease (Anderson & Tietyen-Clark, 1986). Dietary fibers, are the endogenous components of plant material in the diet that are resistant to digestion by enzymes produced by man (Pilch, 1987). Dietary fibers can be classified according to their water solubility. Most water insoluble fibers such as cellulose, cellulose-rich products and lignin (i.e. wheat bran) do not lower serum cholesterol concentrations of humans or animals (Anderson, 1987). Water-soluble fibers (pectins, gums, and mucilages) and diets high in water-soluble fiber from oats and bran decrease the glycemic response to foods and lower serum cholesterol concentrations (Anderson, 1987).

The theories of how fiber lowers cholesterol levels include the following: (1) Soluble fibers bind bile acids and other lipids and may interfere with micelle formation in the small intestine resulting in alterations in the quantity of cholesterol or fatty acids absorbed or altering the size of lipoprotein particles formed by intestinal mucosa (Council

on Scientific Affairs, 1989). (2) Soluble fibers increase the fecal excretion of bile acids and may interfere with cholesterol and bile acid homeostasis sufficiently to affect hepatic secretion of lipoproteins. (3) Soluble fibers fermented by colonic bacteria form gases and short chain fatty acids (Council on Scientific Affairs, 1989). Short Chain fatty acids are almost completely absorbed into the portal vein and could effect hepatic cholesterol synthesis (Council on Scientific Affairs, 1989). However various types of dietary fiber have different effects on serum cholesterol (Anderson & Chen, 1979). Soluble fiber (Pectin, guar gum, barley and oat bran) have been shown to reduce blood cholesterol levels, when given in large amounts (Life Sciences Research Office, 1987). The fibers from fresh fruits, vegetables, legumes, oat bran and barley appears to have the most potential for reducing cholesterol levels (Life Sciences Research Office, 1987). Insoluble fibers such as wheat bran have a very small or no effect on serum cholesterol levels (Nutrition Committee, American Heart Association, 1988).

Anderson and associates (1991) studied 20 hypercholesterolemic men admitted to a hospital metabolic ward. They were randomly divided into either an oat bran or wheat bran group for 21 days after a 7-day control-diet period. Both the control and treatment diets were designed to have the same energy content and nutrients, the only difference is in the amounts of soluble fiber. After the 21 day of treatment oat bran significantly decreased total cholesterol by 12.8% and low density-lipoprotein cholesterol by 12.1%. Wheat bran had no significantly change. In both groups high density lipoprotein did not change significantly. They concluded that oat bran could have been effective in reducing the risk for CHD, since they found a significant reduction in total cholesterol and low density lipoproteins (Anderson et al., 1991).

Kestin, Moss, Clifton and Nestel (1990) had similar findings on 24 mildly hypercholesterolemic men on the effects of adding 11.8g dietary fiber per day from each of three cereal brans (wheat, rice and oat) to a low-fiber diet for 4 weeks each. The subjects were placed on a low-fiber diet for 3 weeks and randomly allocated to consume the wheat, rice, or oat-bran supplement for 4 weeks in a double-blind crossover design. Altogether there were six orders of treatment and the study lasted 90 days. At the end of each period a fasted blood draw was done. Plasma total and low-density lipoprotein cholesterol concentrations were significantly lowered by oat bran. Oat bran significantly lowered the plasma total cholesterol concentration by 5.6%. This decrease was seen mainly in the LDL-cholesterol fraction. In both the rice and oat bran a slight increase was seen in the HDL cholesterol concentration of 2.9% in rice bran and 4.0% in the oat bran when compared with the wheat bran. They concluded that an increase intake of fiber-rich foods from several sources might help prevent coronary heart disease and cancer. However, the consumption of a single food source of fiber such as oat bran or rice bran is unlikely to help lower plasma lipoprotein concentrations (Kestin, Moss, Clifton & Nestel, 1990).

Oat-bran is a palatable cereal which is rich in water-soluble fiber (Kestin, Moss, Clifton & Nestel, 1990). Oat products, have important hypocholesterolemic properties as demonstrated in both humans and animals (Judd & Truswell, 1988). A 6 week study looking at oat-bran supplementation of 50g per day in the diets of 12 healthy young people showed a decrease of serum cholesterol by 12% (Storch, Anderson & Young, 1984). In studies using larger intakes of oat bran serum total cholesterol lowered by 13-

19% and LDL cholesterol lowered by 14-23% with no change seen in HDL cholesterol (Anderson, Story, Sieling, Chen, Petro & Story, 1984).

Anderson et al. (1990) studied 12 men with high serum total cholesterol concentrations to determine the effects of a ready-to-eat oat bran cereal on lipid concentrations. Subjects were randomly assigned to either 56g of oat bran cereal or corn flakes. Both diets were 43% of energy from carbohydrate, 41% fat, 16% protein. After completing the first diet, subjects completed 2 weeks on the alternate diet. The oat bran cereal diet compared with the corn flakes diet lowered serum total cholesterol by 5.4%. LDL cholesterol decreased by 8.5% and HDL cholesterol decreased by 3.3% on the oat bran diet (Anderson, Spencer, Hamilton, Smith, Tietyen, Bryant & Oeltgen, 1990).

Kirby, Anderson, Sieling, Rees, Chen, Miller and Kay (1981) had similar findings on eight men who were fed control and oat bran diets with previously documented hypercholesterolemia. The men were fed two identical solid diets that only differed in the inclusion of 100g of oat bran per day provided in muffins and hot cereals. Each 100g of the oat-bran preparation contained 26.4g of plant fiber including 14.8g of water-soluble fiber. The diets were randomized alternating each group to the other diet after 10 days. Serum concentrations of total cholesterol, triglycerides and glucose were measured daily after a 10 hour fast. They measured plasma HDL cholesterol concentrations on 2 or 3 days at the end of each dietary period and calculated values for LDL cholesterol. Patients tolerated both diets well and ate 94% of the oat bran served. The non-bran control diets had a 5% increase on serum total cholesterol concentrations and in the oat bran group an 18% decrease in serum cholesterol concentrations. The decrease in total serum cholesterol was from a 14% reduction in LDL cholesterol whereas the cholesterol

concentrations did not change. Concluding that palatable and inexpensive high fiber foods such as oat bran may have an important role in treating hypercholesterolemia.

Anderson et al. (1984) examined 20 hypercholesterolemic men who were randomly allocated to oat-bran or bean supplemented diets for 21 days in a hospital metabolic ward. They developed control and experimental diets that were virtually identical in nutrient content and differed only in the amount of plant fiber. The oat-bran diet provided 100g of oat bran per day served as hot cereal and five oat-bran muffins a day. Oat-bran diet supplied approximately 47g total plant fiber and 17g soluble fiber per day. The bean diet contained 115g of dried bean per day, which provided approximately the same amount of total plant fiber and soluble fiber as did the oat-bran diet. After the 21 days a 10 hour fasting blood draw was taken to measure serum cholesterol, triglyceride and HDL cholesterol. The subjects ate 98% of the oat bran provided and an average of 88% of the beans served. Subjects consumed the oat-bran without difficulty. The subjects who consumed the beans complained of mild abdominal distension and gas production. Oat-bran diets decreased serum cholesterol concentrations by 19% and low-density lipoprotein cholesterol by 23%. Bean diets decreased serum cholesterol concentrations by 19% and low-density lipoprotein cholesterol by 24%. Oat-bran supplements and bean supplements had almost identical effects on serum total cholesterol by reductions of approximately 19% and LDL cholesterol concentrations by about 24%. Neither diet was accompanied by substantial reductions in HDL cholesterol concentrations. They concluded that oat-bran or bean supplement might have substantial therapeutic benefits in the long-term management of selected patients with hypercholesterolemia.

These studies document the hypocholesterolemic effect of soluble-fiber rich foods (Anderson & Chen, 1979). The lipid lowering effects of soluble-fibers such as guar gum, locust bean gum, pectin, oat bran, legumes and psyllium have all shown significant cholesterol lowering effects (Anderson et al., 1992). Considering the side effects of many cholesterol lowering drugs adding soluble fiber to the diet has been suggested as a safe, practical and cost effective alternative to help reduce serum cholesterol concentrations and may have a substantial impact in lowering the risk for CHD (Kinosian & Eisenberg, 1988; Witztum 1989, Anderson, Deakins, Floore, Smith & Whitis, 1990).

Cholesterol and Exercise

Individuals who engage regularly in cardio-respiratory activities are leaner and more physically fit in general than sedentary individuals. They also are reported to have higher plasma concentrations of high-density lipoprotein cholesterol (HDL-C) and lower concentrations of total cholesterol, low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), and triglycerides than sedentary control subjects (Williams, Wood, Haskell & Vranizan, 1982). It is still uncertain whether physical activity can favorably influence the process of coronary artery disease and coronary heart disease in human beings (Kramsch, Aspen, Abramowitz, Kreimendahl & Hood, 1981). Recent studies appear to support the belief that regular aerobic exercise may protect against premature cardiovascular disease (Kramsch et al., 1981). Observations from early investigations indicated lower triglyceride concentration in endurance-trained subjects while total cholesterol was either not changed or only slightly different. However, it became evident that physical activity had an impact on the lipoprotein lipid distribution, and later investigations have focused on the measurement of

both the cholesterol and protein content associated with these various lipoproteins (Kendler, 1997). In most cases, regular participation in physical activity is associated with lower plasma triglyceride concentrations. Generally, when a person's base line plasma triglyceride concentration is elevated endurance exercise training usually can reduce the high concentration (Thompson, Cullianane, Sady, Glynn, Chenevert & Herbert, 1991). The amount of the concentration that is reduced is related to the pre-training concentration and the volume of exercise completed during the training program (Guntelberg, Brennan, Holloszy, Schonfeld, Rennie & Weidman, 1977). Researchers believe that reduction in triglyceride concentrations result from both regular exercise and diet. Even though changing ones dietary intake can help with lowering triglyceride concentrations alone. It does not seem to be the absolute reason seen in people who are physically active (Kiens, Gad, Lithell & Vessby, 1981). This is portrayed especially in endurance athletes, extreme leanness is related to lower triglyceride concentrations (Hagan & Gettman, 1983).

Holloszy, Skinner, Toro and Cureton, (1964) studied the effects of a six month program of endurance exercise on serum lipids of middle-aged men. Two groups of subjects were involved in this study. In group A. 15 men who all led sedentary lives for three or more years before the study participated in an organized exercise program consisting of pushups, sit-ups and distance running (2 to 4 miles) on an average of 3.35 times per week for six months. In group B. 12 subjects did not exercise together with an instructor, but participated in a program of distance running geared to their individual capacities and increasing progressively in intensity. Three fasting blood samples were taken on each subject over a seven day period to establish base line values. Total serum

cholesterol levels were determined once a month, and phospholipids along with triglycerides measured every other month. All three samples were taken over a seven day period after the study. The results of the serum triglycerides fell from a mean pre-training of 208 ± 127 to 125 ± 78 mg. in both groups. The mean serum cholesterol and phospholipid levels did not change significantly with training. This reduction in serum triglycerides appears to occur within two to three hours after exercise and lasts for approximately two days. From their results they concluded that it would appear that serum triglyceride levels can be kept significantly lower by means of regularly performed endurance exercise. This finding may represent one mechanism by which exercise possibly could protect against coronary heart disease. (Holloszy, Skinner, Gelson & Cureton, 1964).

Cholesterol is an important part of cell membrane and synthesis of steroid hormones however; excess (high levels of) cholesterol is also associated with development of coronary artery disease. Studies have concluded differently on whether or not plasma cholesterol concentrations are lower in endurance-trained male and female athletes when compared to inactive control groups (Williams, Krauss, Wood, Lindgren, Giotas & Vraganizan, 1986).

Factors such as body weight, percentage of body fat and differences in food intake are important considerations when evaluating the effects of physical activity on plasma cholesterol concentration. Factors such as age and gender can play an important role in the difference of plasma concentrations, which can effect whether or not physically active individuals have lower plasma concentrations than do inactive individuals (Seals, Hagberg, Hurley, Ehsani & Holloszy, 1984).

Studies comparing LDL-C concentrations in men and women athletes from various sports with those of inactive subjects have produced mixed results (Thompson, Callinane, Sady, Flynn & Bernier, 1988). Tsopanakis, Kotsarellis and Tsopanakis (1986) found that athletes participating in power or speed-related events have LDL-C either similar or lower than those of inactive controls. Stein, Michielli, Glantz, Sardy, Cohen, Goldberg and Brown (1989) found similar findings as Tsopanakis when studying the effects of different exercise training intensities on lipoprotein cholesterol fractions in healthy middled-aged men. They reported that LDL-C concentrations are lower for men and women following endurance training. However, in one study conducted by Marti, Suter, Riesen, Tschopp, Wanner and Gutzwiller (1990) that looked at only men and another study conducted by Nikkila, Kuusi and Myllyen (1980) looked at both men and women long distance runners reported lower LDL-triglyceride concentrations in men but not in women when compared with inactive controls. However, a study conducted by Kuusela, Vautilainen, Kukkonen and Raurammaa (1980) compared Scandinavian lumberjacks with sedentary workers and concluded that lumberjacks have higher LDL-triglyceride concentration.

Endurance-exercise training has been associated with increased HDL-C concentration, associating it with reduced CAD risk. There is a consensus, among cross-sectional studies that HDL-C concentrations are elevated typically 20-30% in endurance trained athletes when compared to inactive groups (Thompson, Lazarus, Cullianane, Henderson & Musliner, 1983). Some researchers have suggested a dose-response relationship between the amount of exercise performed and how much HLD-C will increase (Rotkis, Cote, Coyle & Wilmore, 1982). Some researchers do not believe that

one bout of exercise can have an effect on HDL-C increasing. Reasons for the discrepancy may be related to several factors: the length of the training period, the volume of training period, the volume of training completed, changes in body composition, dietary intake, weight loss, and the pre-training HDL-C concentrations (Weltman, Matter & Stanford, 1980).

Brownell, Bachorik and Ayerle (1982) studied the effect of a 10 week exercise program on changes in plasma lipid and lipoprotein levels in 37 women and 24 men. Thirty-minute sessions were held three times weekly for 3 different groups of 15-25 subjects. The class was conducted by certified fitness instructors and focused on cardiopulmonary conditioning, flexibility, muscular strength and endurance. By the fourth week, the subjects were exercising at approximately 70% of maximal heart rate for 15-20 minutes. The remaining time was used for warm-up, cool-down and stretching. Blood samples were obtained before and after the 10-week program. Results from this study demonstrate that men and women differ in their lipid and lipoprotein responses to an exercise program. Men showed significant reductions of 4.4% in cholesterol 6% in LDL cholesterol and 9.5% in triglycerides and a 5.1% increase in HDL cholesterol. Women showed a 4.1% decrease in cholesterol with no significant changes in HDL cholesterol or LDL cholesterol and a 14.5% decrease in triglycerides. The results from the study concluded that moderate exercise might have different effects on men and women (Thompson, Lararus, Cyllianane, Henderson & Musliner, 1983).

Studies on exercise and lipid changes have proposed conflicting results between men and women. Studies of men have shown consistent increases in HDL cholesterol during programs of moderate or intensive exercise and have shown either a decrease or

no significant change in triglyceride, The HDL/LDL ratio, an important predictor of coronary heart disease, increased significantly in men, but did not change significantly in women when participating in an exercise program. Conclusions of this matter suggests that a short-term exercise program in a work setting can improve plasma lipid and lipoprotein patterns more in men than in women (Brownell et al., 1982).

In women, some studies showed no change in HDL cholesterol and a decrease in LDL cholesterol after moderate exercise (Lee, Nieman, Raval, Blankenship & Lee, 1991). In addition, it is becoming increasingly apparent from studies that the quantity and intensity of physical activity required to have a positive change in lipoprotein levels may be varied (Leon, Connet, Jacobs & Rauramaa, 1987). For instance, it has been shown recently that more moderate levels of activity can improve the lipoprotein profile by increasing HDL cholesterol levels and lowering LDL cholesterol levels (Duncan, Gordon & Scott, 1991). Researchers thought one would need to endurance train to show significant changes in cholesterol levels.

King, Haskell, Young, Oka and Stefanick (1995) studied 149 men and 120 postmenopausal women to determine the two-year effects of differing intensities and formats of endurance exercise on exercise participation rates, fitness, and plasma HDL cholesterol levels among healthy older adults. The subjects were randomly assigned to one of four groups. (1) higher-intensity group-based exercise training (2) higher-intensity, home-based exercise training (3) lower-intensity home-based exercise training (4) a 1-year delayed treatment control condition that received an exercise training program during the second year. The primary purpose of the second study year was to evaluate the maintenance of changes that had occurred during the first year in the three

experimental conditions. It also allowed the researchers to look at long-term effects of the three different exercise-training conditions on variables such as lipoprotein levels that had not changed significantly during the first year. For the higher-intensity exercise training, three 40-minute endurance training sessions per week were prescribed at 73% to 88% of peak treadmill max heart rate. For lower-intensity exercise, five 30-minute endurance training sessions per week were prescribed at 60% to 73%. Subjects assigned to the year delayed treatment control condition were requested not to change their activity habits during the initial 12-month study period. Changes in total cholesterol, HDL, LDL cholesterol and plasma triglycerides did not differ significantly between the three exercise training conditions versus the one-year wait-listed control group at the end of 1 year. By the end of the second year subjects in the two home-based training conditions showed small but significant HDL cholesterol increase from baseline. The increases were the greatest for subjects in the lower-intensity condition, whose exercise prescription required more frequent exercise sessions per week. In all exercise conditions, increases in HDL cholesterol were associated with decreases in waist-to-hip ratio in both men and women. Concluding that frequency and length of time needed to achieve HDL cholesterol change may be longer for older populations (King, Haskell, Young, Oka & Stefanick, 1995).

Stein et al. (1989) studied the effects of different exercise intensities on lipoprotein cholesterol fractions in healthy middle-aged men. Forty-nine healthy men were randomly divided into four different groups of exercise training. The subjects trained at either 65%, 75% or 85% of their measured maximal heart rate. The fourth group was a 12 week non exercise control group. Fasting morning blood samples were

obtained from all subjects before and immediately after the exercise training or control period. Results of the lipid profiles showed that total cholesterol did not change significantly in any of the groups HDL cholesterol increased significantly in the 75% and 85% maximal heart rate training groups, but not in the other two groups. LDL cholesterol decreased significantly only in the 75% group. No significant differences were seen with in any of the groups for VLDL cholesterol or triglyceride levels. Concluding that an aerobic exercise at 75% maximal heart rate, 30 minutes per session, 3 days a week for 12 weeks results in a 19% increase in HDL cholesterol and an 22% decrease in LDL cholesterol (Stein et al., 1989).

Elevated levels of plasma high-density-lipoprotein (HDL) may be associated with a lowered risk of coronary heart disease (Gordon, Castelli, Hjortland, Kannel & Dawber, 1977). Several studies have indicated that vigorous exercise training may result in elevated HDL cholesterol levels in young and middle aged men (Lopez, Vial, Balart & Arroyave, 1974). The concept of elevated HDL levels resulting from exercise has been combined with the idea of the effect which dietary modification may have on HDL levels (Sacks, Castelli, Donner & Kass, 1975). Some researchers have suggested that moderate alcohol intake and a reduction caloric intake will increase HDL levels. Hartung, Foreyt, Mithcell, Vlasek and Gotto (1980) studied 59 healthy middle aged marathon runners, 85 joggers and 74 inactive men and observed the relation between diet and plasma lipids and lipoprotein levels in all of these men. The inactive subjects were men selected randomly from the general population who indicated that they did not exercise regularly. They participated in a diet-education study and were randomized into a control group. The marathon runners had all completed a 26.2 mile marathon during the 12 months before

the study and continued to train heavily. They reported an average of 40 miles per week. The joggers ran at least 2 miles three times per week. Blood was drawn in the morning after a 12 to 16 hour fast. Height, weight, tricep skinfold and blood pressure were also measured and a comprehensive questionnaire was completed during the visit on the food-intake-record section the subject was asked how many servings of each food he ate daily weekly, monthly or yearly. The marathon runners and joggers did not differ substantially from the inactive subjects in their reported dietary habits, although they had significantly higher HDL-cholesterol levels. The positive relation between distance running and HDL cholesterol was significant. The distance ran was the best predictor of both HDL cholesterol and the ratio of HDL to total cholesterol. Concluding that it is primarily the jogging and running, rather than diet that elevated HDL to a level associated with significant reduction of coronary risk (Hartung, Foreyt, Mitchell, Vlasek & Gotto, 1980).

Hartung, Squires and Gotto (1981) found similar results in studying the effects of chronic exercise training on plasma HDL cholesterol on 18 male coronary patients. Exercise training was conducted three times weekly for 20 minutes, increasing to 40 minutes per session at an intensity utilizing 70% to 85% of the maximal heart rate for a period of 3 months. None of the patients were placed on a specific diet. Blood was drawn at the start of the study following a 12 to 14 hour fast and then again at the end of the 3 months. Hartung found similar results from the previous study he conducted a year earlier. Moderate physical activity for 3 months can contribute to increases in HDL-C in patients with coronary artery disease without dietary intervention or significant change in total cholesterol, triglycerides and body weight (Hartung, Squires & Gotto, 1981).

Regular participation in physical activity as well as single exercise session can alter lipoprotein metabolism, plasma lipid and lipoprotein concentrations and lipid transport (Durstine & Haskell, 1994). The understanding of the precise mechanisms responsible for these changes is not totally clear. There is evidence indicating that other factors including diet composition adiposity, weight loss, plasma volume change, and hormone and enzyme activity interact with exercise to alter the rates of synthesis, transport and clearance of lipid and lipoproteins from the blood (Durstine & Haskell, 1994).

Lipoprotein Lipase (LPL) is responsible for delipidation of chylomicron and VLDL molecules and promotes the clearance of fatty acids and glycerol from the vascular compartment for either storage or use as substrate in energy metabolism (Durstine & Haskell, 1994). Cross-sectional studies indicate that endurance-trained runners at rest have higher plasma concentrations of LPL activity than less active controls (Thompson et al., 1991). Inactive men, after undergoing endurance-exercise training, usually have significantly lower adipose tissue and LPL activity than compared to their adipose tissue when they were inactive. (Peltonen, Marniemi, Hietanen, Vuori & Ehnholm, 1981).

Studies evaluating a single exercise session indicates that it increases LPL activity. Depletion of intramuscular triglyceride stores by endurance exercise may promote secretion and/or synthesis of LPL by muscle cells (Oscari, Essig & Palmer, 1990). Kanter, Cullinane, Sady, Herbert and Thompson (1987) studied 21 trained and untrained men on the effect of a single exercise session on lipid and lipoprotein concentrations and on plasma lipoprotein lipase. After the trained and untrained subjects

performed a single prolonged session of endurance cycling exercise both groups had higher LPL activity.

Studies show that increased LPL activity results in increased HDL synthesis and also indicates that exercise training also prolongs the survival of HDL (Durstine & Haskell, 1994). The survival time of HDL protein was 27% longer in the circulation of physically active men, compared with inactive men (Herbert, Bernier, Cullinane, Edelstein, Kantor & Thompson, 1984). Endurance training increases the half-life of apoliproteins A-I and A-II in active men (Thompson et al., 1988). Researchers have concluded the increase of HDL associated with endurance training is a result of both increased synthesis and survival.

Other factors should be considered when evaluating the impact of a single session of exercise or exercise training on lipoprotein metabolism. Controversy exists about the effect of dietary modification on lipoproteins (Sacks et al., 1975). Some researchers have suggested that moderate alcohol intake or caloric restriction resulting in weight loss may elevate HDL (Hulley, Cohen & Widdowson, 1977). Reduced body fat and increased leanness are important outcomes of exercise training. Leanness has been associated with lower hepatic lipase activity in physically active people (Wood & Stefanick, 1990). Increasing one's exercise and changing one's diet will help reduce body fat and prevent the incidence of high cholesterol and occurrence of heart disease.

The adipose distribution shown by waist-to-hip girth ratio (WHR) is associated with altered lipoproteins (Durstine & Haskell, 1994). Abdominal adiposity with a WHR greater than 1.0 in nonobese males is associated with lower HDL-C concentration (Barakat, Burton, Carpenter, Holbert & Israel, 1980). Studies have shown subjects with

abdominal adiposity to have plasma LDL characteristics that look almost identical to the LDL plasma of subjects with CAD (Peeples, Carpenter, Israel & Barakat, 1989). This may suggest, as a result of endurance training, a selective loss of abdominal fat versus other areas (gluteal), a reduction in the WHR could perhaps reduce CAD risk (Peeples et. al, 1989).

Physical activity has a positive effect on the lipid and lipoprotein concentrations. In normal men and women increased physical activity is associated with lower plasma triglyceride concentrations (Durstine & Haskell, 1994). The dominant changes in lipoproteins associated with endurance training are increased HDL-C, HDL2-C and apoliprotein A-I concentration (Durstine & Haskell, 1994). Exercise-induced reductions in LDL-C are only minor without change in adiposity or dietary fat cholesterol intake (Durstine & Haskell, 1994).

Cholesterol Lowering Drugs

Patients whose LDL cholesterol levels remain high even with extensive dietary therapy and exercise should be considered for drug treatment. Dietary therapy should be at least six months long before drug treatment is started (The Expert Panel, 1988). Individual with elevated LDL cholesterol levels above 220 mg/dl and/or with confirmed coronary heart disease, diet and exercise should not be the only therapy considered (The Expert Panel, 1988). After the required six months of dietary therapy, to establish adequate baseline values, drug therapy should be initiated. (The Expert Panel, 1988).

There are about fourteen types of cholesterol lowering drugs, which are grouped into categories based on their action. Bile acid sequestrates: cholestyramine and colestipal action binds acids in the GI tract, forming an insoluble complex resulting in

increased clearance of cholesterol binded to bile acids eliminated in the feces. Some of the most common side effects are nausea, constipation, abdominal discomfort, rashes and skin irritations (Hopfer, 1995).

Nicotininc acid generally known as Niacin helps lower cholesterol levels by decreasing lipoprotein and triglyceride synthesis by inhibiting the release of free fatty acids from adipose tissue and decreasing hepatic lipoprotein synthesis. Generally it decreases blood lipids. Some major side effects are nausea and flushing of the face and neck (Hopfer, 1995).

Fibric acids: clofibrate, fenofibrate, gemfibrozil and benzafibrate works by decreasing triglyceride production by the liver and increases HDL lipoproteins. Major side effects consist of diarrhea and abdominal pain (Hopfer, 1995).

Probucol is one of the most common cholesterol drugs prescribed to date. It may decrease transport of cholesterol from intestine or interfere with cholesterol synthesis. Also it may increase fecal excretion of cholesterol and bile acids. The most common side effects are diarrhea, bloating, abdominal pain, nausea and vomiting (Hopfer, 1995).

There are four HMG-CoA reductase inhibitors available on prescription in the United States. Pravastin (provachol), Fluvastatin (lescol), and two of the newest and most common prescribed Lovastatin (mevacor) and Simvastatin (zocor). Also known as "statins" these agents slow the progression of coronary artery disease and have actually been shown to induce regression of artherosclerotic lesions in CAD patients (Pedersen, 1995). These drugs act by inhibiting the hepatic enzyme HMG-CoA reductase, which leads to an increase in the hepatic production of cholesterol receptors. These receptors pull cholesterol out of the blood stream, thus reducing serum cholesterol levels

(Thompson et al.,1995). Statins have been associated with some skeletal muscle complaints (myositis and rhabdomyolysis). The most common side effects include headache, abdominal pain, constipation, and diarrhea (Kobashigawa et al., 1995).

Patients should incorporate diet, weight control, exercise and stop smoking to lower cholesterol levels and the risk of CHD before any drug therapy is considered (Expert Panel, 1993). If these methods fail and cholesterol levels are not lowered drug therapy should then be considered.

Summary

Cholesterol is probably the best known sterol, which is essential to the human body. It is found associated with the fats, but chemically it is not related to them. Cholesterol, a white waxy solid, is the principal sterol found in animal organisms. The largest amount of cholesterol is found in muscle, nervous and connective tissue. It is needed to synthesis vitamin D, cholic acid which is part of bile, some hormones, and cortisol which promotes glucose synthesis in the liver. The daily production is approximately 400 mg from intestinal absorption and 600mg synthesized in the cells and the liver. In the human body every cell, except a mature red blood cell, produces cholesterol.

The synthetic process of cholesterol takes place in the liver and may occur in other organs. In all tissues expect in the brain, cholesterol is continually regenerated. If any organ can be singled out as the single most important to metabolize cholesterol and some amount of plant sterols, it is the liver. It can both synthesize and destroy cholesterol. The breakdown and lose of cholesterol can occur in four ways:

1. By direct reduction to dihydrocholesterol or by passage through the intermediate cholesterone to dihydrocholesterol or coprosterol.
2. By loss of cholesterol in the feces.
3. By conversion into steroid hormones.
4. By conversion to bile acids in the liver.

There are a number of variables that are important in determining or changing the level of blood cholesterol in the body. Throughout the day the level of serum cholesterol remains for the most part constant, even through there is a large variation in the rate that

cholesterol enters and leaves the body. Females at birth have slightly higher levels of plasma cholesterol than males. During childhood, adolescence and early adulthood there is very little difference in blood cholesterol between men and women. In later life males tend to have higher serum cholesterol levels due to higher concentrations of VLDL and LDL. Serum cholesterol levels tend to be a lower in premenstrual women. After the age of 50 male cholesterol levels tend to lower whereas females tend to rise slightly.

Artherosclerosis is the major disease affecting the heart and blood vessels, and is the leading cause of death among middle-aged adults in the United States. Artherosclerosis is the result of fatty cholesterol deposits in blood vessels walls, reducing elasticity and eventually blocking blood flow. It is a disorder of the coronary arteries, cerebral arteries, illac and femoral arteries and aorta that is responsible for coronary heart disease, stroke and peripheral arterial disease.

Treatment of high cholesterol begins with dietary treatment. There is evidence indicating that dietary fiber has important lipid lowering effects and may reduce risk of CHD. Increasing dietary fiber intake is an important therapeutic treatment for other specific conditions besides CHD, such as diabetes, hyperlipidemia, hyperglyceridemia, hypercholesterolemia and intestinal disorders. Soluble fiber has been shown to reduce blood cholesterol levels. Soluble fiber increases the fecal excretion of bile acids, which may alter the quantity of cholesterol absorbed by the intestine. The fibers from fresh fruits, vegetables, legumes, oat bran and barley appear to have the most significance for reducing cholesterol levels.

Individuals who engaged regularly in cardio-respiratory activities are leaner and more physically fit in general than sedentary individuals. They also cholesterol (HDL-C)

and lower concentrations of total cholesterol, low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C) and triglycerides than sedentary control subjects. Regular participation in physical activity as well as a single exercise session can alter lipoprotein metabolism, plasma lipid and lipoprotein concentrations and lipid transport. The understanding of the precise mechanisms responsible for these changes is not totally clear. There is evidence indicating that other factors including diet composition adiposity, weight loss, plasma volume change, and hormone and enzyme activity interact with exercise to alter the rates of synthesis, transport and clearance of lipid and lipoproteins from the blood.

Patients who cholesterol levels do not lower with dietary and exercise therapy drug intervention should be considered as a method of lowering these high levels. There are fourteen types of cholesterol lowering drugs which are grouped into categories based on the action they perform in the body; bile acid sequestrants, nicotininc acids, Fibric acids, probucol and HMG-CoA. The goals of drug therapy are to lower total cholesterol, raise HDL's and lower LDL's. However, there are several major side effects. They range from itching of the skin, gastrointestinal distress, liver toxic effects, muscular damage due to interaction between drugs and blood clots.

In summary, coronary heart disease (CHD) is the leading cause of death in the United States (JAMA, 1995). While factors such as obesity, diabetes, smoking, sedentary living and hypertension have been shown to influence CHD risk, serum cholesterol seems to have the strongest correlation with CHD (Genest & Cohn, 1995). The real aim of any cholesterol management intervention is to lower circulating cholesterol fractions. The National Cholesterol Education Program Guidelines state that

exercise along with a high fiber diet, weight loss and medication should be included in the management of high cholesterol fractions. It is important to know not only the interactive effect of exercise and diet on lipoprotein metabolism but also the interactions between exercise and various classes of lipid medications now used in treatment.

CHAPTER 3

METHODS

Subjects

Fifty (50) male and female subjects volunteered to participate in this study. Twenty-five were exercising regularly in a supervised exercise program and the other 25 were sedentary. The twenty-five exercising subjects belonged to two formal exercise programs. Twenty were from the Adult fitness program at the University of Nevada Las Vegas (UNLV). This program starts at a low level of intensity and progresses in intensity each week. Subjects meet as a group five days a week Monday through Friday. The supervised exercise program consists of four components: 1) warm-up and stretching, 2) strength and muscular endurance, 3) cardiovascular or aerobic, and 4) cool down. The warm-up period includes 5 to 7 minutes of flexibility exercises, consisting of bending, stretching and twisting. Exercises for muscular strength and endurance include push-ups, sit-ups and chest raising. For the aerobic portion of the class the women do bench steps and jogging, and the men do bench steps and swimming. The final part of the program is a cool down period; these exercises are similar to the warm up exercises. The other five were participating in their own exercise program at least 3 times a week for duration of 30 minutes and at an intensity of 70 % or more of their max heart rate. The twenty-five sedentary subjects were recruited from the Las Vegas community and the UNLV faculty

and staff. All subjects had an LDL-C of 130 mg/dl or higher and were not on any cholesterol lowering medications. This study was approved by the Institutional Review Board (See Appendix B).

Research Design

The study was divided into a pre-experimental period, an experimental period and a post experimental period.

1). The 7 day pre-experimental period was for collecting baseline data.

2). The experimental period was 90 days in duration during which time the subjects ingested the experimental fiber.

3). The post-experimental period was when final testing was done.

There were three testing periods. Test 1 (T1) during the pre-experimental period. Test 2 (T2) 45 days after starting the experimental period and Test 3 (T3) test during the post-experimental period.

Statistical Design

This study was a 2 (Group) by 3 (Time) mixed model with repeated measures design with exercise being between subjects and time being within subjects. The two levels of exercise were exercise and non-exercise. The three levels of time were baseline, 45 days and 90 days. The dependent measures of interest were Total Cholesterol, Low-Density lipoproteins (LDL's) and High-Density lipoproteins (HDL's).

Procedure

Pre-experimental Period

During this period of approximately one week the testing (T1) included a 12-hour fasting blood sample. A lipid profile determined Total Cholesterol, HDL-C, LDL-C,

VLDL-C, Cholesterol/HDL Ratio and Triglycerides. This lipid profile served as their baseline blood profile if it was greater than 130 mg/dl. Since an LDL-C greater than 130 mg/dl was required to be in the study. If the LDL-C was less than 130mg/dl they were eliminated from the study. During this pre-experimental period, subjects were briefed on the study, questions were answered and they read and signed an informed consent form (Appendix B). Written information about Bios Life 2 was distributed to subjects (Appendix A). Height and weight was measured and Body Mass Index was calculated. Waist girth and hip girth were measured from which a waist to hip ratio was calculated. Body fat was determined by bioimpedance analysis. A six-minute walk test was administered. The six-minute walk test was used to determine "functional exercise capacity", which is defined as a patient's ability to undertake physical demanding activities encountered in everyday life that are not reflected by conventional exercise testing.

Experimental Period

During this 90-day experimental period the subjects in the exercise group exercised a minimum of three times a week and took the prescribed dosage of Bios Life 2. The non-exercise group took the prescribed amount of Bios Life 2 and did not change their regular routine or eating habits. After 45 days all tests (T2) were repeated exactly as performed in T1. This was Test 2 (T2).

Post-experimental Period

During this period all tests were again administered: Test 3 (T3). Test T3 was administered as soon after the experimental period as possible. However, the subjects continued the experimental protocol until they were tested.

BiosLife 2

BiosLife 2 is a dietary fiber in the form of a powder and packaged in 4.5-gram packets. It was mixed with eight-ounces of water or fruit juice and one packet was taken twice per day. It was taken 15 minutes prior to breakfast and dinner. (See appendix A for ingredients). A dietary survey was completed on all subjects to determine the amount of fiber in their normal diet. (Appendix C). If the diet was low in fiber subjects were introduced to the Bios Life supplement gradually by taking one-half the normal dose for the first three to four days. Subjects reported to the laboratory every two weeks during the experimental period. During this time weight was taken and questions were asked about the subject's adherence to the experiment protocol and reported any side effects of the Bios Life. Every two weeks the subjects brought back their used and (any unused packages) from the prior two weeks. This was used to determine compliance.

Six-minute Walk Test

To determine a level of cardio-respiratory fitness a submaximal six-minute walk test was given. The six-minute walk test was done on a measured course. The distance covered in six minutes was recorded and compared to national norms. The number of stops (or rest periods) during the walk were also recorded. The outdoor course, where the test was administered, was on a concrete walkway and consisted of 283 feet and 8 inches per lap. In the center of the course was a courtyard, which was surrounded by concrete benches. These benches not only served as course markers they also provided a place to sit if a rest was needed. The subjects were encouraged to walk as far as they could in the 6-minute period but were allowed to stop and rest if necessary. The distance and the number of stops were recorded for each subject.

Anthropometrical Measurements

Height was taken without shoes with an anthropometer to the nearest quarter of an inch. Subjects were instructed to stand as tall as possible, feet together and looking straightforward. Weight was measured, without shoes and minimal clothes, on a physicians balance scale to the nearest quarter of a pound. Body Mass Index was computed from formula of body weight in kilograms divided by height in meters squared (BW (kg)/HT $(m)^2$). Men with a BMI greater than 27, and women with a BMI greater than 26.9 were considered overweight. (Appendix E). Waist girth was measured in centimeters at the narrowest circumference and hip girth was taken at the largest circumference. A waist to hip ratio of greater than .9 was considered obese (See appendix E for risks).

BMI and waist to hip ratio are commonly used in epidemiology studies to describe the degree of fatness in a population. Although these measurements were taken at T1, T2 and T3 they are measurements that do not usually reflect body composition changes in an individual over time, but were used to describe the population.

Bioimpedance Analysis (BIA)

Bioimpedance analysis was done using the Bio Analgesics system. A low-grade electric current is passed through the body, which is resisted, by body water. This resistance is measured in Ohms. This resistance is used in an equation to determine total body water. Total body water is then used to calculate per cent body fat.

Cholestech LDX

The cholestech LDX Analyzer is a small size (5"x 5" x 8") lightweight (2lb) instrument designed for point of service lipid analysis. The analyzer uses a disposable

cassette that needs only 35 uL blood samples, which can easily be obtained by a fingerstick. The cassette contains a well into which the blood is pipetted, a glass fiber screen, four optical windows, a reagent impregnated bar allowing four separate reactions and a magnetic code bar to communicate with the analyzer. The blood is separated by the glass fiber screen that allows only the plasma into a reservoir. The system than uses reflectance photometry to obtain lipid results. The instrument has a display window that guides the user and displays the results, which are available within four minutes (Drimmer et al., 1995).

Optics Check Cassette Test Procedure

Before any procedure is performed the analyzer must be tested. The Cholestech Optics Check Cassette should be ran once each day before patient samples are tested and after the cholestech L.D.X System has been moved or serviced. One should never use the Cholestech L.D.X Optics Check Cassette that has become damaged or altered in anyway. The cassette is placed in the analyzer and the test is run. The analyzer will automatically perform the optics check. Once the test is complete four numbers will appear on the screen and if they are within the acceptable range (80-105) a blood sample can be ran.

Performing a Fingerstick

The patient was asked to sit quietly for five minutes before the blood sample was taken. Gloves were worn while working with the blood sample and the samples were placed into the biohazard box when the test was completed. A capillary plunger was placed into the end of a cholestech capillary tube with the red mark and set aside. To help increase blood flow, the fingers and hands were warm to the touch. If not the capillary puncture was done on center fingers that has been thoroughly cleaned with an

alcohol swab. The area was thoroughly dried with a gauze pad before pricking the finger. Then the center fingers were firmly pricked with the lancet. The finger was squeezed gently to obtain a large drop of blood. The first drop of blood was wiped away due to the fact it may contain tissue fluid. The finger was gently squeezed again while holding it downward until a second large drop of blood forms. The puncture should provide a free-flowing drop of blood. The capillary tube was held horizontally by the end with the plunger, then touched to the drop of blood without touching the skin. The tube will fill by capillary action up to the black mark. A very important step is not to collect air bubbles. The excess blood was wiped off and then the patient applied pressure to the puncture until the bleeding stopped.

Performing the Test

The fingerstick sample was analyzed within 5 minutes after the collection or the blood will clot. The cassette was taken out of the refrigerator and warmed up to room temperature for 10 minutes before opening. The Analyzer is plugged in and warmed up. The cassette was removed from its pouch and held by the short sides only. Do not touch the black bar or the brown magnetic stripe. The cassette was placed on a flat surface. Press run on the analyzer first so that a self-test can be ran. Once a Self-Test is ok the cassette drawer will open and load cassette will appear on screen. Use the Cholestech capillary tube to place sample into the test cassette sample well. Keep the cassette level after the sample has been applied. Immediately place the cassette into the drawer of the analyzer. The black reaction bar must face toward the analyzer. The brown magnetic strip must be on the right. By pressing run the drawer will close and the testing begins. When the test is completed, the analyzer will beep and the screen will display the data.

Everything that came into contact with blood including the cassette was placed into a biohazardous container. The results were recorded on the appropriate form.

CHAPTER 4

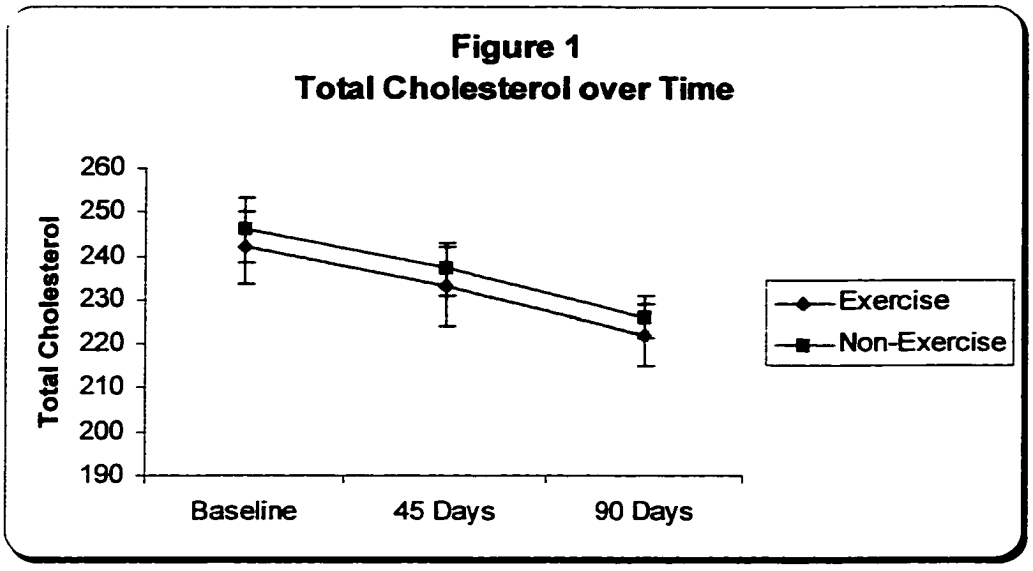
RESULTS & DISCUSSION

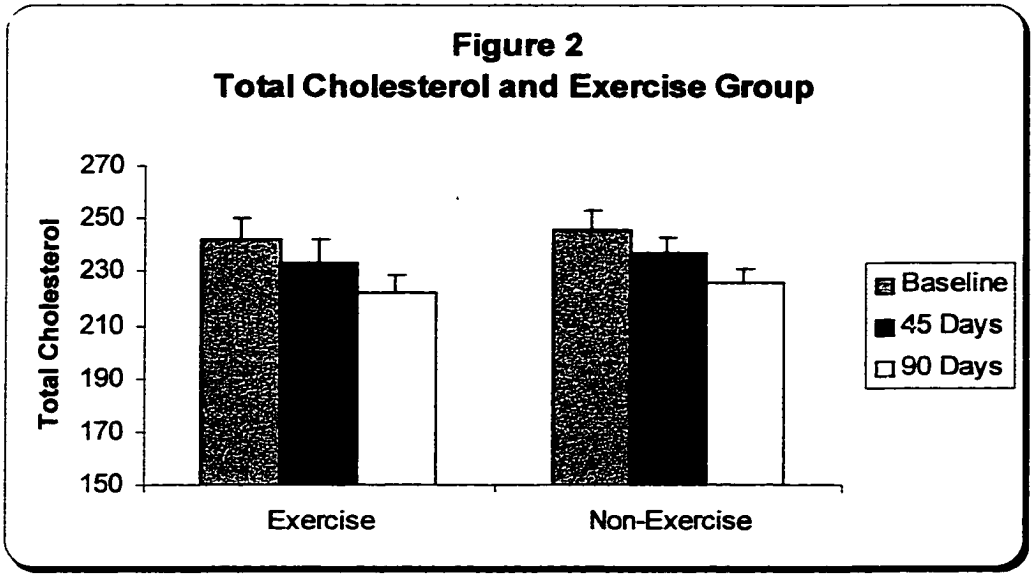
Mean total cholesterol and low-density lipoproteins were analyzed using a 2 (group) by 3 (time) analysis of variance (ANOVA) procedure with repeated measures on the second factor. All statistical analysis was done using the SAS system.

Total Cholesterol

The test for interaction was not significant $F = 0.00$, $p = .9985$. The ANOVA did however yield a significant main effect for time, ($F = 18.29$, $p = .0001$), with means for the exercise group being 242, 233, 222 and the means for the non-exercise group being 246, 237, 226 for baseline, 45 days and 90 days, respectively (see figure 1). Tukey's test was used to determine the nature of the difference. This indicated a significant decrease in total cholesterol at each measure baseline, 45 and 90 days in both exercising and non-exercising groups.

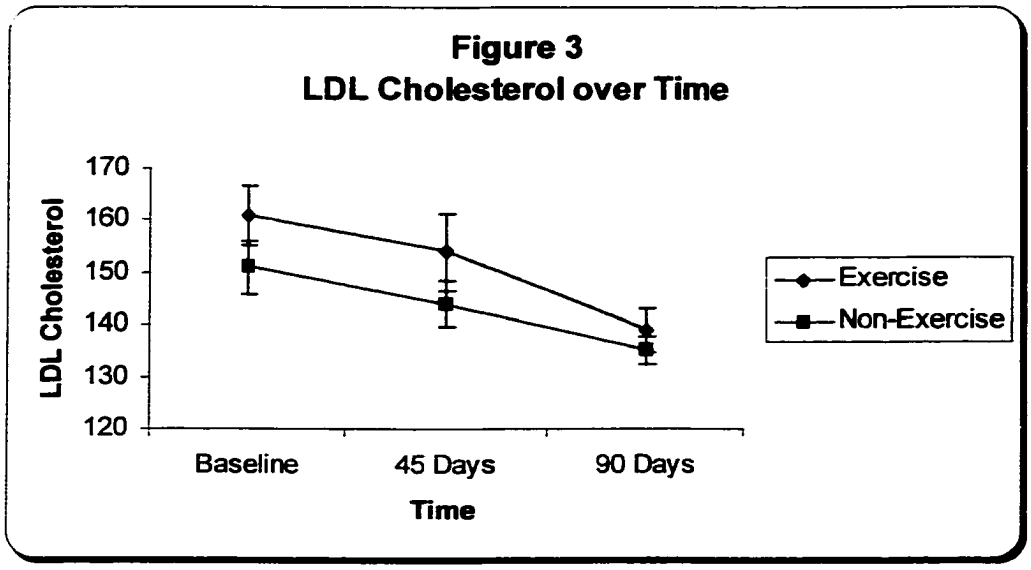
The ANOVA did not yield a significant main effect for group, ($F = 0.20$, $p = .6540$), with means being 232, 236 for exercise and non-exercise, respectively (see figure 2). Therefore, there was a significant decrease in total cholesterol for the exercise and non-exercise groups, but there was no significant difference between exercise and non-exercise.



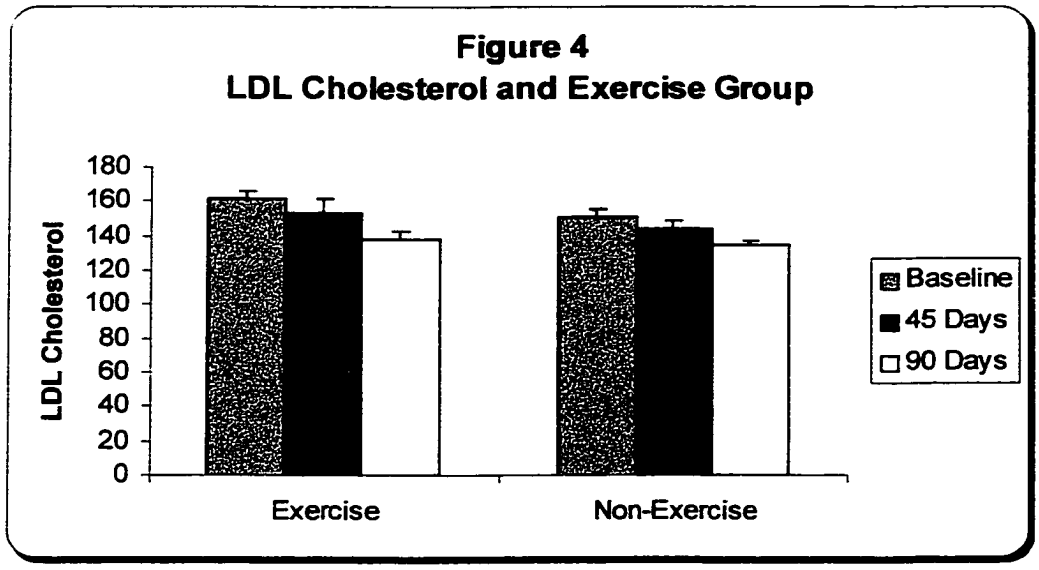


Low Density Lipoprotein Cholesterol

The test for interaction was not significant $F = 0.72$, $p = .4896$. The ANOVA did however yield a significant main effect for time, ($F = 21.60$, $p = .0001$), with means for the exercise group being 161, 154, 139 and the means for the non-exercise group being 151, 144, 135 for baseline, 45 days and 90 days, respectively (see figure 3). Tukey's test was used to determine the nature of the difference.

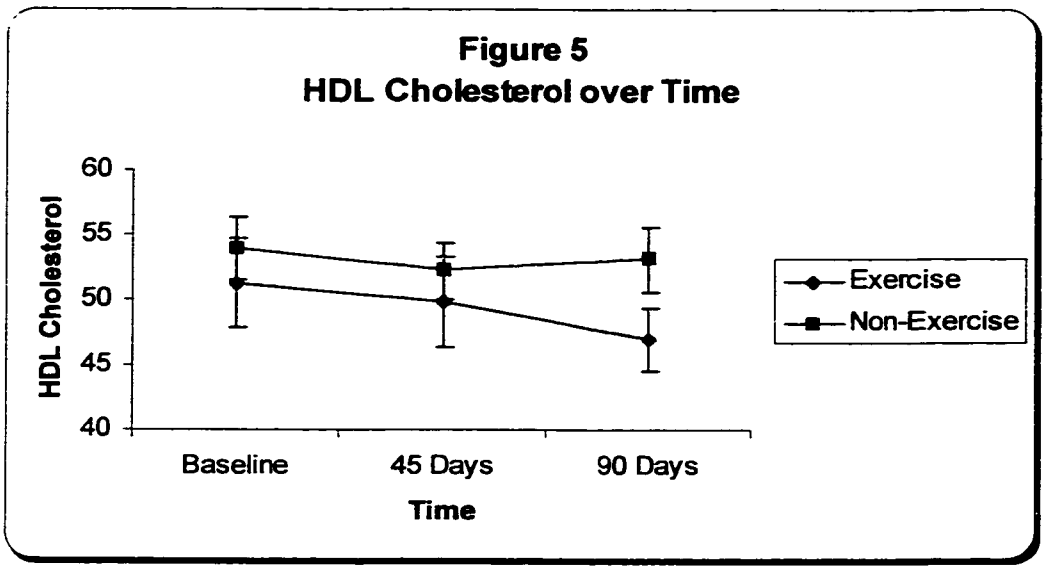


This indicated a significant decrease in LDL cholesterol at each measure from baseline to 90 days in both exercising and non-exercising groups. The ANOVA did not yield a significant main effect for group, ($F = 1.59$, $p = .2128$), with means being 151, 158 for exercise and non-exercise respectively (see figure 4). Therefore, there was a significant decrease in LDL cholesterol for the exercise and non-exercise groups, but there was no significant difference between the exercise and non-exercise.

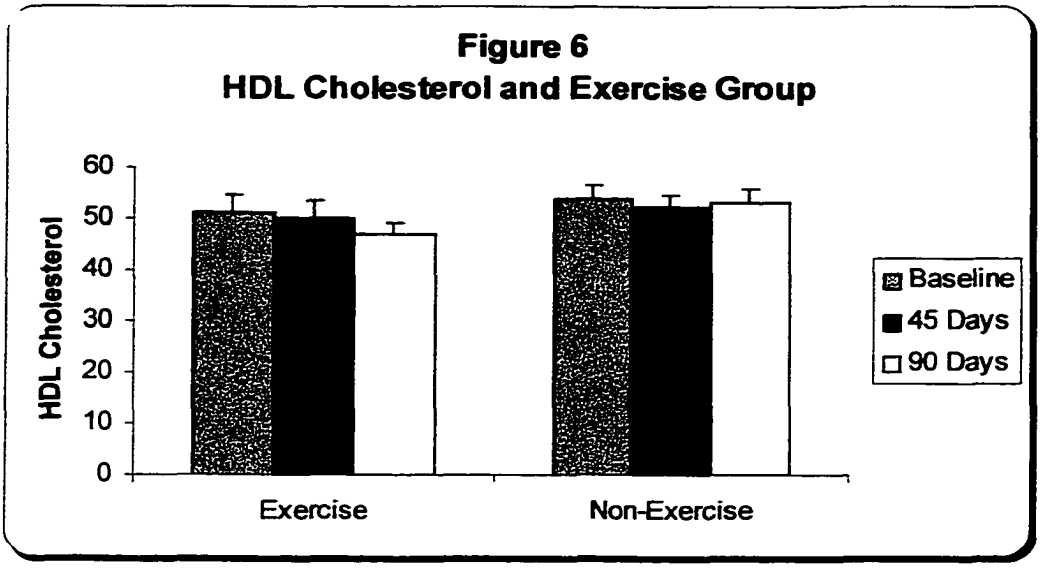


High Density Lipoprotein Cholesterol

The test for interaction was not significant ($F = 1.06$, $p > 0.05$). The ANOVA did not yield a significant main effect for time, ($F = 1.67$, $p>0.05$), with means for the exercise group being 51, 49, 46 and the means for the non-exercise group being 54, 52, 53 for baseline, 45 days and 90 days, respectively (see figure 5).



Therefore, there was no significant difference in the HDL cholesterol levels from baseline to 45 days, from 45 days to 90 days and from baseline to 90 days in both exercising and non-exercising groups. The ANOVA did not yield a significant main effect for group, ($F = 1.07$, $p>0.05$), with means being 49, 53 for exercise and non-exercise, respectively (see figure 6). Therefore, there was no significant difference in HDL cholesterol for the exercise and non-exercise groups, nor was there a significant difference between exercise and non-exercise.



In the present study, serum total cholesterol and low-density lipoprotein cholesterol was significantly reduced in both exercise and non-exercising groups after taking three months of the dietary fiber supplement. Total serum cholesterol concentrations in the exercise group were reduced by 7.96%, and in the non-exercise by 7.84%. Low-density lipoprotein concentrations in the exercise group were reduced by 13.3% and in the non-exercise group decreased by 10.46%. Similar reductions in total cholesterol have been reported in other studies using insoluble fibers, such as pectin, guar gum, psyllium, legumes, oat bran and locust bean gum. Van Horn, Liu, Parker, Emidy, Liao, Pan, Giumetti, Hewitt and Stamler (1986) supplemented the diet of healthy volunteers on a low-fat diet with 4g soluble fiber from either oatmeal or oat bran. Serum total cholesterol was reduced by 5% after 6 weeks on just a low fat diet and an additional 3% reduction after 6 more weeks after adding either oatmeal or oat bran (Van Horn et al., 1986). Jensen, Spillar, Gates, Miler and Whitman (1993) also found similar findings with patients who took a mixture of guar gum, pectin, psyllium and locust bean gum

three times a day and the subjects experienced a 10% reduction in serum TC and a 14% reduction in serum LDL (Jensen et al., 1993).

Total cholesterol and serum LDL cholesterol were lowered in both the exercise and non-exercise groups without significantly affecting serum HDL cholesterol levels. In the exercise group serum HDL levels were reduced by 8.42% and 1.63% in the non-exercise group. This was also observed by Anderson and associates (1992) who studied 44 hypercholesterolemic male and female participants. After one week baseline period, subjects were assigned to consume either 114g per day of a psyllium-flake or wheat bran-flake cereal for 6 weeks. Fasting HDL cholesterol levels did not change significantly during the six weeks (Anderson et al., 1992). In a similar study Anderson (1984) also found that in thirty-five hypercholesterolemic men with total cholesterol higher than 260 mg/dl who were placed in either a oat bran diet, dried bean diet or canned bean diet for 21 days. Twelve participants were placed in the oat-bran diet which the consumed between 50 to 100 g of oat bran daily as hot cereal or oat bran muffins. Eleven participants were placed in the dried bean diet group, which they consumed 100 g of navy or pinto beans served in soup. The last twelve participants were placed in the canned bean diet in which they consumed 120 g of canned beans from Campbell's Pork and Beans. HDL cholesterol concentrations had a non-significant decrease of 6% as for the dried and canned beans there was a significant decrease in HDL concentrations.

In this study the effect of the exercise on the participant's total, low-density lipoprotein, and high-density lipoprotein serum cholesterol levels was also of interest. It has been well documented that individuals who engaged in regular cardiovascular concentrations that place them at low risk for developing coronary artery disease. Studies

demonstrating this lower risk include the results of world class marathon runners having exceptionally high plasma HDL cholesterol and relatively low total cholesterol, LDL cholesterol and triglyceride concentrations. These findings indicate that individuals who participate in regular strenuous exercise achieve increases in HDL levels helping reduce the risk of cardiovascular disease. Huttunen, Lansimis, Voutilainen, Ehnholm, Hietanen, Penttila, Siitonen and Raurama (1990) reported a significant increase in HDL cholesterol by 19%, whereas plasma total cholesterol, LDL cholesterol, and triglyceride levels were decreased by 10% in a group of middle-aged men who performed aerobic exercise 3 to 4 days a week for 16 weeks.

Analysis of data from the present study shows that participants performing regular aerobic exercises three times a week for a duration of 30 minutes and at an intensity of 70% or more of their predicted max heart rate had no greater decrease in lipid serum concentrations then those in the non-exercising participants. As shown in figure 1 and 2 total cholesterol decreased 7.96% in the exercising group and 7.84% in the non-exercising group and LDL decreased 13.3% in the exercise group and 10.46% in the non-exercising group. HDL concentration decreased 8.42% in the exercise group and 1.63% in the non-exercise group (see figure 3). Generally, when a person's lipid levels are high, regular exercise training can usually reduce high levels of total and LDL cholesterol. The exercise group's lack of response to have an even larger decrease in total and LDL cholesterol concentrations when compared to the non exercise group might be explained by the fact that each participant in the exercise group had been exercising for 6 months or longer before entering the study. One might hypothesis that they may have already lowered their cholesterol levels to the extent in which exercise can affect them. Dietary

therapy in conjunction with regular exercise is considered an essential element in therapy of elevated serum cholesterol levels. In this study, patients at risk for coronary heart disease exhibited significant reductions in serum cholesterol after using Bios Life.

CHAPTER 5

SUMMARY AND CONCLUSIONS

Summary

An increase in the incidence of coronary heart disease has lead to an increased interest in preventive measures. Since there is a strong relationship between high fiber diet, regular exercise and coronary heart disease, health professionals support a high fiber diet and regular exercise as a contributing factor in reducing this incidence.

It has been well documented that both high levels of total serum cholesterol and specifically low-density lipoproteins are linked with coronary heart disease. Epidemiological data from the Framingham Heart Study indicate that elevated total serum cholesterol is a major coronary risk factor. Larosa and associates (1990) studied CHD history and total cholesterol in 2,282 men and 2,845 women and concluded that there was definite evidence that high levels of total cholesterol were associated with an increase in the incidence of CHD and that conversely low levels of total cholesterol were associated with a low incidence of CHD (Larosa et al., 1990).

The general purpose of dietary fiber supplementation as therapy is to reduce elevated serum cholesterol levels. This is achieved primarily by reducing fat intake and consuming a healthy diet, rich in fiber. Dietary therapy in conjunction with regular exercise is considered an essential element in therapy of elevated serum cholesterol

(Ready, 1996). Exercise has been shown to help in the promotion of weight reduction, which enhances LDL cholesterol lowering, reducing triglyceride, raising HDL, reduces blood pressure and decreasing the risk of diabetes mellitus. Therefore, the combination of regular exercise and diet rich in fiber can lower total cholesterol, increase HDL, decrease LDL and consequently reduce the risk of CHD.

The purpose of this study was to investigate the effects of Bios Life, a non-prescription dietary fiber supplement, on Total and LDL cholesterol and determine whether exercise accentuates this effect. Fifty (50) male and female subjects volunteered to participate. Twenty-five were exercising regularly in a supervised exercise program and the other 25 were sedentary. A 12-hour fasting blood sample was taken prior to the study to obtain a baseline value and again at 45 and 90 days. Subjects had to have LDL-C greater than 130 mg/dl to participate in the study. Both groups took 4.5 gram packets of Bios Life twice a day. Neither group changed their regular routine or eating habits.

Mean total cholesterol, low-density lipoproteins and high-density lipoproteins were analyzed using a 2 (group) by 3 (time) analysis of variance (ANOVA) procedure with repeated measures on time. All statistical analysis was done using the SAS system.

The results of this study showed that total cholesterol and low-density lipoproteins concentrations decreased significantly from baseline to 90 days. This was true whether or not the subject was in the exercise group or non-exercising group.

Conclusions

The following conclusions can be drawn from this study:

1. Exercise did not accentuate the effect of Bios Life.

Recommendations

The following are recommendations for future studies:

1. Since there was no significant difference between the exercise and non-exercise groups it is suggested that the participants in the exercise group not be involved in an exercise program prior to the study. Both groups should take the Bios Life, but the participants in the exercise group start an exercise regimen at the beginning of a study.
2. Bios Life did decrease total cholesterol and LDL levels over the 90 days but not to the same extent as some of the other drugs like probucol, niacin, pravastin, and zocor. As reported in the existing literature, further research over a greater amount of time may be needed to see if time would allow for a greater decrease in total cholesterol and LDL levels with Bios Life users.

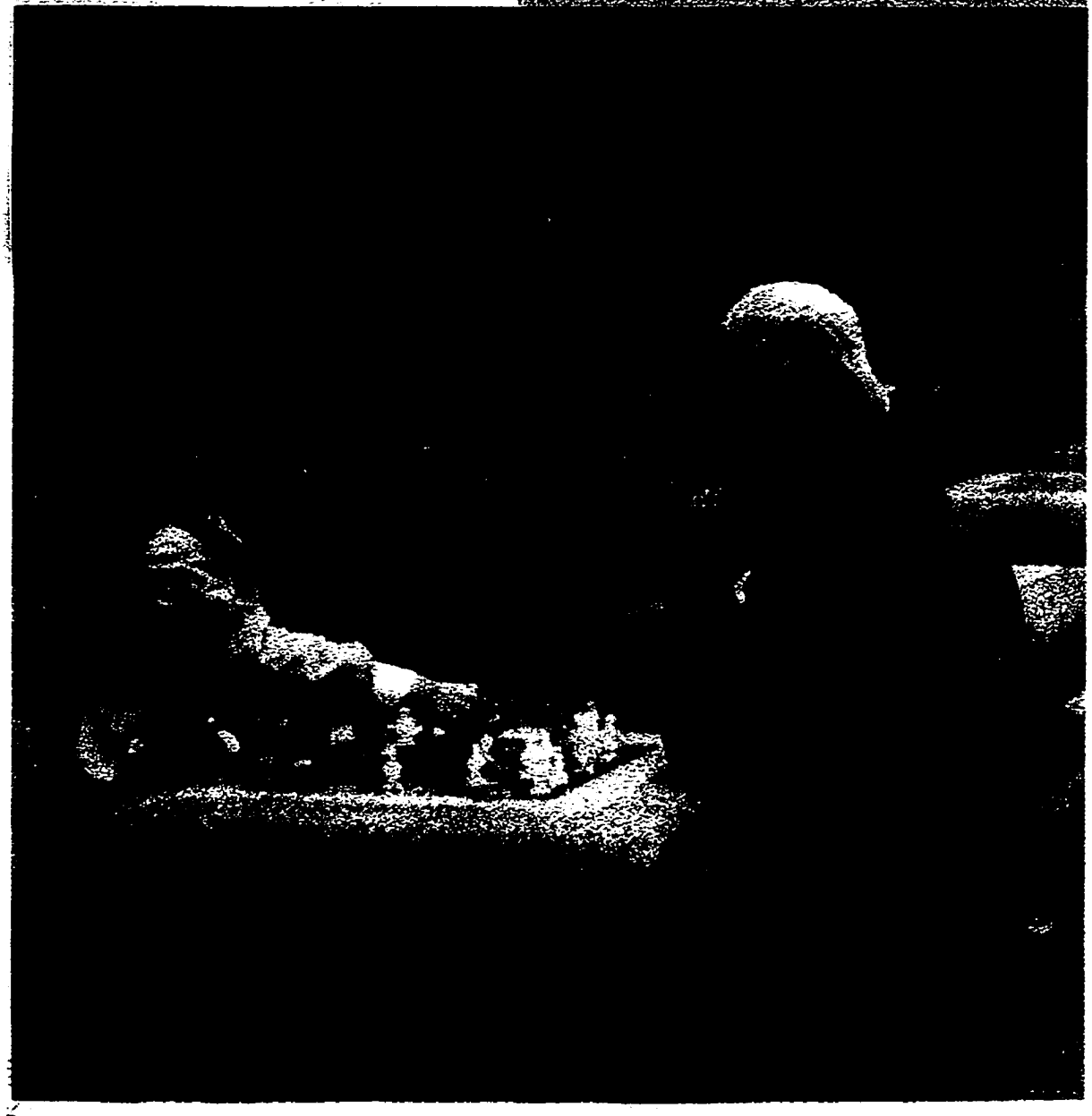
APPENDIX A

INSTRUCTIONS TO SUBJECTS

MEMORANDUM TO SUBJECTS

Bio Life 2 Natural

Research Study



PURPOSE

It has been documented that high levels of total serum cholesterol have been linked with coronary heart disease. However, reductions in the low-density lipoprotein fraction of total cholesterol are linked with reduction in myocardial infarction and sudden cardiac death. There is evidence indicating that dietary fiber can lower serum cholesterol. Individuals consuming a diet high in fiber tend to have a lower incidence of myocardial infarction and sudden cardiac death. This study will the effects of Bios Life 2 a dietary fiber supplement on serum lipoproteins, and determine whether exercise potentates this effect.

PRODUCT

The natural dietary fiber supplement consisted of a mixture based on a patented formula (# 4,824,672) and (# 4,883,788) titles "Method and Composition for Reducing Serum Cholesterol' manufactured by Rexall Showcase International under the trade name Bios Life Natural. This patented formula consisted of pectin, guar gum, gum arabic, oat fiber, and locust bean, gum. This product also contains various vitamins (beta carotene, vitamin E, vitamin C, niacinamide, vitamin B-12, pyridoxine HCl, riboflavin, thiamine HCl, biotin, and folic acid). In addition, chromium polynicotinate and selenomethionine were added to enhance the metabolic aspects of the product. To improve palatability, Stevia, a natural fiber with known natural sweet effects, was added to the product mix.

METHODS

As a volunteer for this study your LDL cholesterol level must be greater than 130 mg/dl and keep participating in a formal exercise program at least three times per week.

You will give a 12-hr. fasting blood sample baseline, 30, 60 and 90 days. Also at these time periods height and weight will be taken and Body Mass Index computed. Waist girth and hip girth will be measured and waist to hip ratio will be computed. Body fat will be determined by bioimpedance analysis. A six-minute walk test will be administered.

For the next 90 days you will mix Bios Life 2 a dietary fiber with an eight-ounce glass of water, fruit juice or Gatorade. It will be taken 15 minutes prior to breakfast and dinner. Weekly questioning to monitor adherence to the fiber supplement will administered.

UNIVERSITY OF NEVADA, LAS VEGAS
COLLEGE OF HEALTH SCIENCES

DEPARTMENT OF KINESIOLOGY
EXERCISE PHYSIOLOGY
BOX 453034
LAS VEGAS, NV 89154-3034

MEMORANDUM

DATE: 9/15/98

TO: Subjects for Cholesterol lowering study

FROM: Lawrence A. Golding. E-mail: LAGolding@aol.com and Lori Inderlied

SUBJECT: Requirements for the study

The Exercise Physiology Laboratory is about to start a study on the effect of a non-prescription fiber preparation, which is claimed to reduce LDL Cholesterol. Total cholesterol is made up of low-density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C). The popular literature refers to LDL as the "bad" cholesterol and the HDL as the "good" cholesterol.
This study will investigate the effect of taking this supplement in people who are exercising and in people who are sedentary. All subjects must have a LDL cholesterol of more than 130mg/dl.
You have been identified as someone who is exercising regularly (the adult exercise program) but who has a LDL cholesterol greater than 130mg/dl. We would like you to volunteer to be a subject.
The details of the study are as follows:

1. **The study will be 90 days in duration.**
2. **A lipid profile will be done prior to the study starting, after 45 days and again at the end of the study.**
3. **During those same 3 times the following measurements will be taken:**
   - **i. Percent fat by Bioimpedance. This only requires two electrodes to be placed on your wrist and ankle. A very low-grade electric current (from a 9volt battery, which you will not feel) will pass between the electrodes and impedance will be measured from which percent fat is calculated. This is done in street clothes only requiring the right shoe and sox to be removed.**
   - **ii. Height and weight.**
   - **iii. Waist and hip girth**
   - **iv. A 6minute walk will be done for time.**
   - **v. Every two weeks you will be asked to come to the laboratory to pick up your two week supply of Bioslife and return the empty packages from the previous two weeks. This is the way we document that you have taken the Bioslife. If you miss a dose, simply bring back the full package and we can**

Telephone; (702)-895-3766
FAX, (702)- 895-4191

record what you missed. When you come in weight will be taken and a modified percent fat through bioimpedance will be done. This modified procedure only requires you to hold and instrument in your hand.
vi. Prior to starting, you will be asked to complete a simple questionnaire to determine how much fiber you are presently consuming in your normal daily diet.

Enclosed is an informed consent form which explains the above. If you are willing to participate please sign the form, have your signature witnessed and return it in the enclosed, self addressed stamped envelope.

Telephone; (702)-895-3766
FAX, (702)- 895-4191

APPENDIX B

APPROVAL LETTER FROM INSTITUTIONAL REVIEW BOARD

INFORMED CONSENT

UNLV

DATE: May 11, 1998

TO: Lori J. Inderlied (KIN)
M/S: 3034

FROM: Dr. William E. Schulze
for Director, Office of Sponsored Programs and
Member, Biomedical Sciences Committee
UNLV Institutional Review Board

RE: Status of Human Subject Protocol entitled:
"The Effect of Bios Life, a Non-prescription Cholesterol-Lowering Substance and a Combination of Bios Life and Exercise on Serum Lipids:"

OSP #504s0498-013b

This memorandum is official notification that the protocol for the project referenced above has been approved by the Biomedical Sciences Committee of the Institutional Review Board. This approval is approved for a period of one year from the date of this notification, and work on the project may proceed.

Should the use of human subjects described in this protocol continue beyond a year from the date of this notification, it will be necessary to request an extension.

If you have any questions or require any assistance, please Marsha Green at 895-1357.

cc: L. Golding (KIN-3034)
OSP File

Office of Sponsored Programs
4505 Maryland Parkway • Box 451037 • Las Vegas, Nevada 89154-1037
(702) 895-1357 • FAX (702) 895-4242

CONSENT TO PARTICIPATE IN RESEARCH
UNIVERSITY OF NEVADA, LAS VEGAS
EXERCISE PHYSIOLOGY LABORATORY

TITLE OF THE STUDY

The effect of Bios Life and a combination of Bios Life and exercise on total cholesterol, serum low density lipoprotein, and serum high density lipoprotein will be investigated.

PURPOSE

It has been well documented that high levels of total serum cholesterol have been linked with coronary heart disease. However, reductions in the low density lipoprotein fraction of total cholesterol are linked with reduction in myocardial infarction and sudden cardiac death. There is evidence indicating that dietary fiber can lower serum cholesterol. Individuals consuming a diet high in fiber tend to have a lower incidence of myocardial infarction and sudden cardiac death. This study will investigate the effects of Bios Life, a dietary fiber supplement on serum lipoproteins, and determine whether exercise potentiates this effect.

PROCEDURES

If you decide to participate in this study you will report to either the Exercise Physiology Laboratory or the Cardiovascular Center of Southern Nevada for an explanation of the study. A six minute walk test will be given as well as weight and body percent fat taken. Four times during the study you will report to the blood lab for a 12 hour fasting blood sample to be collected. There is no cost to you as a participant for any tests done in this project. The details of the study areas followed:

Pretreatment Period: A 12 hour fasting blood sample will be taken from each participant to determine if they fulfill the cholesterol and lipoprotein criteria. The criteria to participate is ones LDL must be 130 mg/dl or greater. Once 50 participants are identified body fat and weight will be taken. A six minute walk will be administered to determine aerobic capacity.

Treatment Period: The group will be randomly divided in two groups: The control group will not be exercising and will remain on their current diet. The experimental group will continue, their regular exercise program at least three times per week.

Each group will be instructed to consume 4.5g of Bios Life before breakfast and dinner by stirring the contents of one packet into an 8oz glass of half water and half juice and drink immediately. This will be done 15 minutes prior to breakfast and dinner everyday for the length of the study. Participants will have a fasting lipid panel completed 30, 60 and 90 days later. Also weight, aerobic capacity and body percent fat will be measured at 30, 60 and 90 days.

RISKS

The risks of taking Bios Life are minimal. This is an non-prescriptive dietary supplement administered commonly be cardiologists. However, using this product may result in mild gastrointestinal distress, increases stool volume, diarrhea, constipation, abdominal

cramping or appetite suppression. If you experience any of these side affects, please report to the researcher as soon as possible and also consult your physician if necessary.

BENEFITS

The benefits include a reduction in LDL-C and increase in HDL-C. The exercise group will benefit from the physical effect of exercise, and the value of exercise combined with ingestion of Bios Life will be known.

CONFIDENTIALITY

Your participation in and the results of this study will remain confidential. Only those parties directly related with the collection and analysis of data will have access to your file. If the data is presented in a scientific journal or conference you will be referred to by a participant number not your name.

RIGHT TO REFUSE OR WITHDRAWAL

Your participation in this study is strictly voluntary. You may withdraw consent or refuse to participate at any time.

QUESTIONS

Any questions you have about the study it's purpose, design, methodology, procedures and significance will be addressed to your satisfaction. If you have additional questions please feel free to contact Lori Inderlied or Dr. Golding at 895-3766 or Cherri Epstein Director of the Cardiac Rehabilitation program at 254-2400.

For questions regarding the rights of research participants you may contact the Office of Sponsored Programs at 895-1357.

You will be given a signed and dated copy of this form for you records.

I UNDERSTAND THE RESEARCH PROCEDURE AND HAVE DECIDED TO VOLUNTEER AS A RESEARCH SUBJECT. I HAVE READ THE PROVIDED INFORMATION AND ALL QUESTIONS REGARDING THE EXPERIMENT HAVE BEEN ANSWERED TO MY SATISFACTION. I UNDERSTAND THAT I HAVE THE RIGHT TO WITHDRAW FROM THIS STUDY AT ANY TIME WITHOUT RECRIMINATION.

Name of Participant (print)	Signature	Date
Name of Witness (print)	Signature	Date

APPENDIX C

INSTRUCTIONS OF DIETARY SURVERY

HELPFUL MEASURING GUIDE

DIETARY SURVEY

EXPLANATION OF DIETARY SURVEY

LIST OF MINERALS AND VITAMINS

UNLV
University of Las Vegas Nevada
Inderlied Cholesterol/Bios Life Study
Instructions on Dietary Survey

For three days you will write down everything you eat including beverages in the first column. In the second column you will put down the amount of your servings. Attached to the dietary survey there is a helping guide, this allows you to figure out the exact serving size. The third column you will write down how you prepared the food. For example: You baked chicken in the oven. **Remember** to count what you might have marinated the chicken in and also how you coated the baking pan. The last column is for any comments that you might want to add for the nutritionist who will be analyzing the survey.

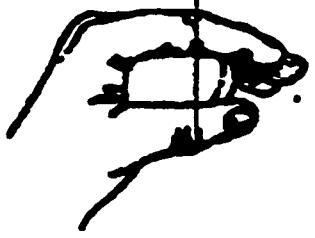
GIVE YOUR HEART A HEALTHY BEAT!

A "Helping" Hand

What's a Food Guide Pyramid serving?

A half-cup of cooked cereal, rice, or pasta is 1 serving. For raw leafy greens, such as lettuce, a serving is a cup. A 1/2 cup of cooked or chopped raw vegetables or fruit equals 1 serving.

A fist or cupped hand = 1 cup
Because hand size varies, compare your fist with an actual measuring cup.



A handful = 1 or 2 ounces of snack food
One handful equals one ounce of nuts and small candies. For chips and pretzels, 2 handfuls equal 1 ounce.

2-1/2 or 3 ounces is a serving of meat, fish, shellfish, or poultry. You are allowed two 3-ounce servings or a single 6-ounce each day.

1 palm = 3 ounces
2 palms = 6 ounces

Thumb tip = 1 teaspoon

Tip of the index finger = half a teaspoon

A thumb = 1 ounce of cheese

One thumb-size chunk of cheese equals approximately one ounce. 1-1/2 to 2 ounces of low-fat cheese counts for one of the 2 to 3 dairy servings recommended daily.

North Carolina Cooperative Extension Service

UNLV Nutrition Program
3 Day Food Record
(For Exercise Physiology Cholesterol Study)

Name: ______________________ Age: __________ Gender: ___________

Today's Date: ____________ Weight: _____________ Height: ___________

Food or Beverage	Amount Eaten	Method Prepared	Comments

UNLV

June 1, 1999

Mrs.
Participant
Exercise and Diet Research Project

Dear Mrs.

Enclosed you will find report of your food intake from the three days you recorded. Your intake was compared to the latest Recommended Dietary allowances or the appropriate dietary goal currently recommended for that nutrient. This intake indicates only the nutrients in the foods you ate for the three days you recorded and does not include any vitamin, mineral or dietary fiber supplements that you may take.

We have highlighted the nutrients in your diet which are below recommended amounts. A list of selected vitamin and mineral food sources is also enclosed for your knowledge.

If you have any questions regarding your results, please call Dr. Susan L. Meecham at 702-895-1169 or leave a message at 702-895-4328

Sincerely,

Susan L. Meecham

Susan L.. Meecham, Ph.D., R.D.
Assistant Professor

sec

Department of Nursing
4505 Maryland Parkway • Box 453018 • Las Vegas, Nevada 89154-3018
(702) 895-3380 • FAX (702) 895-4807

May 28, 1999

Gender:	Female
Activity Level:	Lightly Active
Height:	5 ft 8 in
Weight:	161 lbs
Age:	50 yrs
BMI:	24.48

Basic Components		Recommended Daily Nutrients			
Calories	2100.07	Riboflavin-B2	1.26 mg	Calcium	800.00 mg
Protein	58.42 g	Niacin-B3	13.86 mg	Chromium	125.00 mcg
Carbohydrates	304.51 g	Niacin Equiv.	13.86 mg	Copper	2.50 mg
Dietary Fiber	21.00 g	Vitamin-B6	1.60 mg	Fluoride	2.75 mg
Fat - Total	70.00 g	Vitamin-B12	2.00 mcg	Iodine	150.00 mcg
Saturated Fat	21.00 g	Biotin	65.00 mcg	Iron	15.00 mg
Mono Fat	25.67 g	Vitamin C	60.00 mg	Magnesium	280.00 mg
Poly Fat	23.33 g	Vitamin D IU	200.00 IU	Manganese	3.50 mg
Cholesterol	300.00 mg	Vitamin D mcg	5.00 mcg	Molybdenum	163.00 mcg
Vitamins		Vit E-Alpha Equiv.	8.00 mg	Phosphorus	800.00 mg
Vitamin A IU	4000.00 IU	Folate	180.00 mcg	Potassium	3750.00 mg
Vitamin A RE	800.00 RE	Vitamin K	75.00 mcg	Selenium	55.00 mcg
Thiamin-B1	1.05 mg	Pantothenic Acid	7.00 mg	Sodium	2400.00 mg
		Minerals		Zinc	12.00 mg

May 28, 1999

Serving Size: 2471.53 g (87.18 oz.)
Serves: 3
Cost: --

% comparison to:

Multi-Column

Basic Components			Vitamin A RE	928.33 RE	116%	[illegible]	[illegible]	40%
Calories	1466.19	70%	A - Carotenoid	220.04 RE		Fluoride	– mg	
Protein	43.36 g	74%	A - Retinol	58.89 RE		[illegible]	[illegible]	[illegible]
Carbohydrates	250.27 g	82%	Thiamin-B1	1.07 mg	102%	Iron	14.36 mg	96%
Dietary Fiber	15.76 g	75%	[illegible]	[illegible]	[illegible]	Magnesium	256.44 mg	92%
Soluble Fiber	3.95 g		[illegible]	[illegible]	[illegible]	Manganese	4.25 mg	121%
InSoluble Fiber	6.74 g		[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Sugar - Total	147.41 g		[illegible]	[illegible]	[illegible]	Phosphorus	533.81 mg	67%
Monosaccharides	18.41 g		[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Disaccharides	15.38 g		Vitamin C	320.18 mg	534%	Selenium	43.90 mcg	80%
Other Carbs	55.00 g		[illegible]	[illegible]	[illegible]	Sodium	1777.85 mg	74%
[illegible]	[illegible]	[illegible]	Vitamin E mg	5.23 mg		[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	Folate	183.53 mcg	102%	Other Fats		
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Omega 3 Fatty Acids	0.63 g	
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Omega 6 Fatty Acids	4.84 g	
Trans Fatty Acids	0.06 g		Minerals			Other		
[illegible]	[illegible]	[illegible]	Boron	0.10 mg		Alcohol	0.47 g	
Water	2014.45 g		[illegible]	[illegible]	[illegible]	Caffeine	101.79 mg	
Vitamins			[illegible]	[illegible]	[illegible]			

Minerals

Calcium
Milk, cheese, related dairy products, dark green leafy vegetables, legumes (dried beans, peas), some tofu, almonds

Chromium
Mushrooms, prunes, nuts, asparagus, meat, organ meat (heart, kidney, liver), whole grains, cheese

Copper
Seafood, shellfish, organ meat (heart, kidney, liver), whole grains, nuts, seeds, vegetables, water from copper pipes

Iodine
Iodized salt, some seafood, plant & animal products from fertilizers and feed, dairy products from feed additives and disinfectants, breads through the iodates in dough conditioners

Iron
Lean meats, fish, poultry, organ meat (heart, kidney, liver), legumes, nuts, seeds, whole grains, green leafy vegetables

Magnesium
Whole grains, legumes, nuts, seeds, chocolate, dark green vegetables, bananas

Manganese
Whole grains, cereal products, tea, fruits, vegetables

Phosphorus
Protein-rich foods (especially animal protein), cereal grains, milk, milk products, meat

Potassium
Fresh fruits: peaches, pears, cantaloupe; fresh vegetables: winter squash, potatoes, spinach, legumes, yogurt

Selenium
Seafood, meats, eggs, whole grains, brazil nuts, legumes

Sodium
Sodium intake may be reduced by eating more whole grains, fresh fruits and vegetables

Zinc
Seafood, meats, whole grains, legumes. Shellfish are higher in zinc concentration than white fish, and darker poultry meat has more than light meat.

Dietary Fiber

Fresh fruits & vegetables, legumes, nuts, seeds, whole grains, whole grain products

Vitamins

Vitamin A
From plant sources (carotenoids): broccoli, swiss chard, kale, spinach, romaine lettuce, endive, carrots, sweet potatoes, winter squash, apricots, peaches, cantaloupes, papayas (yellow/orange vegetables and fruits). From retinol (animal sources): liver, fish liver oils, margarine, milk, milk products, butter, eggs

B1- Thiamin
Whole grains, legumes (beans and peas), seeds, pork, organ meats (heart, kidney, liver), brewer's yeast, breads made with enriched white wheat flour, fortified cereals

B2-Riboflavin
Dairy products, eggs, whole grains, fortified cereals, baked goods made with enriched white wheat flour, broccoli, asparagus, turnip greens, spinach, liver

B3 - Niacin
Meat, fish, poultry, breads made with enriched flour, fortified cereals, mushrooms, baked potatoes, peanuts

Vitamin B6
Chicken, fish, pork, eggs, liver, (whole grains) brown rice, oats, whole wheat products, soybeans, peanuts, avocados, bananas

Vitamin B12
Meats, dairy products, eggs

Biotin
Kidney, liver, egg yolk, soy flour, cereal grains, yeast

Vitamin C
Fresh fruits & vegetables, especially citrus fruits, broccoli, cauliflower, sweet & hot peppers, strawberries

Vitamin D
Fortified processed cow's milk and infant formula, egg yolks, butter, liver, some fatty fish, fortified margarine, sunshine

Vitamin E
Whole grains, vegetable oils, margarine, salad dressings, foods high in unsaturated fats, nuts, seeds, poultry, fish, eggs

Folacin/Folate
Fresh, green, leafy vegetables, liver, legumes, oranges, peanuts, sunflower seeds, whole grains

Vitamin K
Green leafy vegetables, members of the cabbage family, milk, soybean oil, egg yolks

Pantothenic Acid
Whole grains, legumes, some vegetables and fruits, organ meats (heart, kidney, liver), yeast, egg yolk

APPENDIX D

DATA SHEET

RAW DATA

Baseline

UNLV

INDERLIED CHOLESTEROL/BIOSLIFE STUDY
DATA SHEET

TESTER_____ **DATE**_____

SUBJECT_____ **SUBJECT #**_____

TESTING LOCATION_____ **SEX M F**

HEIGHT_____ **INS.** **WEIGHT**_____ **LBS.**

BMI_____

HIP GIRTH_____ **INS.** **WAIST GIRTH**_____ **INS.**

WAIST/HIP RATIO_____

BIOIMPEDANCE_____ **OHMS.** _____**% FAT**

BLOOD PANEL:

TOTAL_____ **mg/dl** **HDL**_____**mg/dl**

LDL_____ **mg/dl** **HDL/RATIO**_____ **mg/dl**

TRIGLYCERIDES_____ **mg/dl**

6-MINUTE WALK _____ **# LAPS**

NUMBER OF PACKETS OF BIOSLIFE RETURNED

FULL_____ **EMPTY**_____

DATE TEST STARTED _____

NEXT SCHEDULED APPOINTMENT _____

COMMENTS:

_____
_____
_____
_____
_____

College of Health Sciences
Department of Kinesiology
4505 Maryland Parkway • Box 453034 • Las Vegas, Nevada 89154-3034
(702) 895-0996 • FAX (702) 895-1500

45 Days

UNLV

INDERLIED CHOLESTEROL/BIOSLIFE STUDY
DATA SHEET

TESTER_____ **DATE**_____

SUBJECT_____ **SUBJECT #**_____

TESTING LOCATION_____ **SEX M F**

HEIGHT_____ **INS.** **WEIGHT**_____ **LBS.**

BMI_____

HIP GIRTH_____ **INS.** **WAIST GIRTH**_____ **INS.**

WAIST/HIP RATIO_____

BIOIMPEDANCE_____ **OHMS.** _____**% FAT**

BLOOD PANEL:

TOTAL_____ **mg/dl** **HDL**_____**mg/dl**

LDL_____ **mg/dl** **HDL/RATIO**_____ **mg/dl**

TRIGLYCERIDES_____ **mg/dl**

6-MINUTE WALK _____ **# LAPS**

NUMBER OF PACKETS OF BIOSLIFE RETURNED

FULL_____ **EMPTY**_____

DATE TEST STARTED _____

NEXT SCHEDULED APPOINTMENT _____

COMMENTS:

_____

_____

_____

_____

_____

College of Health Sciences
Department of Kinesiology
4505 Maryland Parkway • Box 453034 • Las Vegas, Nevada 89154-3034
(702) 895-0996 • FAX (702) 895-1500

90 Days

UNLV

INDERLIED CHOLESTEROL/BIOSLIFE STUDY
DATA SHEET

TESTER_____ **DATE**_____

SUBJECT_____ **SUBJECT #**_____

TESTING LOCATION_____ **SEX M F**

HEIGHT_____ **INS.** **WEIGHT**_____ **LBS.**

BMI_____

HIP GIRTH_____ **INS.** **WAIST GIRTH**_____ **INS.**

WAIST/HIP RATIO_____

BIOIMPEDANCE_____ **OHMS.** _____**% FAT**

BLOOD PANEL:

TOTAL_____ **mg/dl** **HDL**_____**mg/dl**

LDL_____ **mg/dl** **HDL/RATIO**_____ **mg/dl**

TRIGLYCERIDES_____ **mg/dl**

6-MINUTE WALK _____ **# LAPS**

NUMBER OF PACKETS OF BIOSLIFE RETURNED

FULL_____ **EMPTY**_____

DATE TEST STARTED _____

NEXT SCHEDULED APPOINTMENT _____

COMMENTS:

_____

_____

_____

_____

_____

College of Health Sciences
Department of Kinesiology
4505 Maryland Parkway • Box 453034 • Las Vegas, Nevada 89154-3034
(702) 895-0996 • FAX (702) 895-1500

Raw Data Total Cholesterol

Exercise Group				Non-Exercise Group			
	Baseline	45 Days	90 Days		Baseline	45 Days	90 Days
Subject 1	284	270	250	Subject 26	240	237	228
Subject 2	196	196	180	Subject 27	205	200	200
Subject 3	186	190	180	Subject 28	200	200	200
Subject 4	204	203	200	Subject 29	260	240	210
Subject 5	316	239	277	Subject 30	220	210	200
Subject 6	246	250	248	Subject 31	359	313	253
Subject 7	249	230	225	Subject 32	283	255	230
Subject 8	279	272	273	Subject 33	280	270	240
Subject 9	197	225	199	Subject 34	221	211	190
Subject 10	227	227	228	Subject 35	250	240	250
Subject 11	218	211	190	Subject 36	210	210	200
Subject 12	246	220	235	Subject 37	228	240	237
Subject 13	209	188	183	Subject 38	265	265	260
Subject 14	230	209	200	Subject 39	264	250	244
Subject 15	287	378	293	Subject 40	234	246	212
Subject 16	217	218	200	Subject 41	246	236	212
Subject 17	210	209	200	Subject 42	246	246	246
Subject 18	246	230	200	Subject 43	290	290	280
Subject 19	230	230	200	Subject 44	210	210	210
Subject 20	288	270	270	Subject 45	222	200	214
Subject 21	213	200	200	Subject 46	240	237	237
Subject 22	222	177	200	Subject 47	205	205	205
Subject 23	269	279	280	Subject 48	283	255	255
Subject 24	222	200	205	Subject 49	284	270	250
Subject 25	359	313	253	Subject 50	210	210	210
Mean	242	233.36	222.76	Mean	246.2	237.84	226.92
Std. Dev.	41.560197	44.598094	35.18910816	Std. Dev.	36.924472	29.762504	23.891979
Std. Error	8.3120395	8.9196188	7.037821633	Std. Error	7.3848945	5.9525009	4.7783958

Raw Data LDL Cholesterol

Exercise Group				Non-Exercise Group			
	Baseline	45 Days	90 Days		Baseline	45 Days	90 Days
Subject 1	206	197	160	Subject 26	138	129	137
Subject 2	133	133	120	Subject 27	132	140	130
Subject 3	132	130	125	Subject 28	130	130	130
Subject 4	141	139	130	Subject 29	180	170	130
Subject 5	189	165	140	Subject 30	146	136	126
Subject 6	142	173	149	Subject 31	236	196	139
Subject 7	162	150	140	Subject 32	158	114	114
Subject 8	177	173	150	Subject 33	160	160	140
Subject 9	173	142	140	Subject 34	131	124	120
Subject 10	154	154	172	Subject 35	140	135	135
Subject 11	154	150	130	Subject 36	140	141	130
Subject 12	143	113	120	Subject 37	137	138	129
Subject 13	155	117	136	Subject 38	180	180	175
Subject 14	139	124	112	Subject 39	139	139	130
Subject 15	219	296	211	Subject 40	140	143	136
Subject 16	134	133	120	Subject 41	149	146	131
Subject 17	134	136	129	Subject 42	138	138	138
Subject 18	140	136	129	Subject 43	158	158	154
Subject 19	140	140	130	Subject 44	141	140	141
Subject 20	171	161	161	Subject 45	138	137	149
Subject 21	172	162	146	Subject 46	138	129	129
Subject 22	140	123	126	Subject 47	132	132	132
Subject 23	170	155	145	Subject 48	158	114	114
Subject 24	174	164	131	Subject 49	206	197	160
Subject 25	236	196	139	Subject 50	141	140	141
Mean	161.2	154.48	139.64	Mean	151.44	144.24	135.6
Std. Dev.	27.971712	36.766289	20.605177	Std. Dev.	25.270338	21.6781	13.400871
Std. Error	5.5943424	7.3532578	4.1210355	Std. Error	5.0540677	4.3356199	2.6801741

Raw Data HDL Cholesterol

Exercise Group				Non-Exercise Group			
	Baseline	45 Days	90 Days		Baseline	45 Days	90 Days
Subject 1	58	54	50	Subject 26	60	60	82
Subject 2	49	49	46	Subject 27	54	56	50
Subject 3	25	20	54	Subject 28	54	54	54
Subject 4	48	51	30	Subject 29	60	60	48
Subject 5	56	66	53	Subject 30	54	54	50
Subject 6	61	61	71	Subject 31	56	55	59
Subject 7	64	54	64	Subject 32	64	63	50
Subject 8	62	48	44	Subject 33	56	56	50
Subject 9	23	27	45	Subject 34	54	43	54
Subject 10	31	31	36	Subject 35	54	54	50
Subject 11	31	30	31	Subject 36	54	52	50
Subject 12	82	84	58	Subject 37	82	60	60
Subject 13	23	31	29	Subject 38	34	34	29
Subject 14	59	51	50	Subject 39	61	61	61
Subject 15	43	60	39	Subject 40	32	34	36
Subject 16	50	45	47	Subject 41	39	39	41
Subject 17	58	58	51	Subject 42	60	60	60
Subject 18	45	45	45	Subject 43	64	64	62
Subject 19	65	65	50	Subject 44	50	50	50
Subject 20	70	60	60	Subject 45	22	19	27
Subject 21	54	50	46	Subject 46	60	60	60
Subject 22	55	31	29	Subject 47	54	54	54
Subject 23	86	94	60	Subject 48	64	63	63
Subject 24	28	26	27	Subject 49	60	60	82
Subject 25	56	55	59	Subject 50	48	40	46
Mean	51.28	49.84	46.96	Mean	54	52.2	53.12
Std. Dev.	17.145262	17.747488	11.890052	Std. Dev.	12.086494	11.25833	12.804036
Std. Error	3.4290523	3.5494976	2.3780104	Std. Error	2.4172988	2.251666	2.5608072

APPENDIX E

PHYSICAL CHARACTERISTICS

BODY COMPOSTION TABLES

BODY MASS INDEX

WAIST/HIP CIRCUMFERENCE

WAIST/HIP RATIO PERCENTILE FOR HEART DISEASE

Physical Characteristics of Participants

Exercise Group	Sex	Age Years	Height Inches	Weight Pounds	BMI	Waist to Hip Ratio	Bioimpedance Ohms	% Fat	6-Minute Walk Test
Subject 1	Male	50	70	215	31	0.8	400	21.3	7 laps
Subject 2	Female	32	63	138	24.5	0.8	478	24.2	8 laps
Subject 3	Male	58	69	170	25	0.9	486	19.7	8 laps
Subject 4	Male	53	73	250	31	0.6	486	36.8	8 laps
Subject 5	Female	50	68	163	24	0.75	557	29.7	6 laps
Subject 6	Male	36	71	156	22	0.8	477	13.9	8 laps
Subject 7	Female	63	71	186	26	0.75	586	33.1	6 laps
Subject 8	Male	50	73	246	33	1	403	23	6 laps
Subject 9	Male	59	71	178	25	0.9	462	17.6	7 laps
Subject 10	Female	59	64	120	21	0.8	400	21	5 laps
Subject 11	Female	53	63	166	29	0.9	586	37.8	6 laps
Subject 12	Male	56	72	200	28	0.8	496	29	7 laps
Subject 13	Male	59	69	177	26	0.8	496	27	7 laps
Subject 14	Female	47	64	171	29	0.75	483	32.7	6 laps
Subject 15	Male	47	70	188	27	0.85	451	19.8	6 laps
Subject 16	Male	64	68	131	20	1.1	531	13.8	6 laps
Subject 17	Male	54	61	153	29	0.8	464	23.8	6 laps
Subject 18	Male	60	69	190	28	0.8	470	24.2	6 laps
Subject 19	Female	52	67	155	23	0.8	581	30.5	7 laps
Subject 20	Female	48	66	162	26	0.9	580	31	6 laps
Subject 21	Female	50	60	132	26	0.9	581	21	6 laps
Subject 22	Male	59	68	156	23	0.9	467	16.1	8 laps
Subject 23	Female	30	67	142	22	0.8	389	16.1	7 laps
Subject 24	Male	65	72	257	30	1.1	486	36	5 laps
Subject 25	Male	45	71	166	23	0.75	478	16.1	9 laps
Mean		51.96	68	174.72	26.06	0.842	490.96	24.608	6.68
Standard Dev.		9.18	3.71	36.25	3.41	0.11	60.47	7.37	1.03
Standard Error		1.84	0.74	7.25	0.68	0.02	12.09	1.47	0.21

Physical Characteristics of Participants

Non-Exercise Group	Sex	Age Years	Height Inches	Weight Pounds	BMI	Waist to Hip Ratio	Bioimpedance Ohms	% Fat	6-Minute Walk Test
Subject 26	Female	57	67	141	22	0.8	648	28.8	7 laps
Subject 27	Male	56	62	131	24	0.8	648	28.8	6 laps
Subject 28	Female	60	70	180	26	1	423	16.1	8 laps
Subject 29	Male	59	68	140	21	0.8	477	13.9	7 laps
Subject 30	Male	50	72	205	28	0.9	486	28	6 laps
Subject 31	Female	46	60	120	23	1	300	18	6 laps
Subject 32	Female	40	62	130	24	1	400	23	7 laps
Subject 33	Female	30	70	221	30	1.1	535	15	7 laps
Subject 34	Female	60	70	174	25	1.1	386	17.1	6 laps
Subject 35	Male	56	74	250	30	1.1	586	27	6 laps
Subject 36	Male	51	66	140	24	0.8	486	23	6 laps
Subject 37	Male	40	62	140	25	0.9	424	12	4 laps
Subject 38	Female	48	68	131	35	0.9	648	28.6	5 laps
Subject 39	Male	57	72	186	25	0.9	500	26	5 laps
Subject 40	Female	36	64	140	24	0.7	480	25	8 laps
Subject 41	Female	38	62	146	27	0.9	464	28.6	4 laps
Subject 42	Male	45	70	200	29	1	500	24.6	7 laps
Subject 43	Female	55	61	205	35	1	486	31.1	4 laps
Subject 44	Female	39	58	150	30	1	486	26.7	7 laps
Subject 45	Male	36	67	166	25	0.9	423	16.1	8 laps
Subject 46	Female	57	64	154	26	0.7	646	28	7 laps
Subject 47	Male	52	67	283	40	1.2	537	34.4	7 laps
Subject 48	Female	44	64	126	21	0.7	648	28.8	8 laps
Subject 49	Female	30	67	142	22	0.8	389	16.1	7 laps
Subject 50	Male	59	68	156	23	0.9	467	16.1	8 laps
Mean		48.04	66.2	166.28	26.56	0.916	498.92	23.232	6.44
Standard Dev.		9.69	4.16	41.28	4.71	0.13	94.86	6.36	1.26
Standard Error		1.94	0.83	8.26	0.94	0.03	18.97	1.27	0.25

-١. BODY COMPOSITION (% BODY FAT) FOR MEN*

	Age				
% ile	20–29	30–39	40–49	50–59	60+
99	≤2.4	≤5.2	≤6.6	≤8.8	≤7.7
95	5.2	9.1	11.4	12.9	13.1 S
90	7.1	11.3	13.6	15.3	15.3
85	8.3	12.7	15.1	16.9	17.2
80	9.4	13.9	16.3	17.9	18.4 E
75	10.6	14.9	17.3	19.0	19.3
70	11.8	15.9	18.1	19.8	20.3
65	12.9	16.6	18.8	20.6	21.1
60	14.1	17.5	19.6	21.3	22.0 G
55	15.0	18.2	20.3	22.1	22.6
50	15.9	19.0	21.1	22.7	23.5
45	16.8	19.7	21.8	23.4	24.3
40	17.4	20.5	22.5	24.1	25.0 F
35	18.3	21.4	23.3	24.9	25.9
30	19.5	22.3	24.1	25.7	26.7
25	20.7	23.2	25.0	26.6	27.6
20	22.4	24.2	26.1	27.5	28.5 P
15	23.9	25.5	27.3	28.8	29.7
10	25.9	27.3	28.9	30.3	31.2
5	29.1	29.9	31.5	32.4	33.4 VP
1	≥36.4	≥35.6	≥37.4	≥38.1	≥41.3
N =	1342	5611	5724	3275	984

TOTAL N = 16936

*Data provided by the Institute for Aerobics Research, Dallas, TX (1994). S, superior; E, excellent; G, good; F, fair; P, poor; VP, very poor.

BODY COMPOSITION (% BODY FAT) FOR WOMEN*

	Age				
% ile	20–29	30–39	40–49	50–59	60+
99	≤5.4	≤7.3	≤11.6	≤11.6	≤15.4
95	10.8	13.4	16.1	18.8	16.8 S
90	14.5	15.5	18.5	21.6	21.1
85	16.0	16.9	20.3	23.6	23.5
80	17.1	18.0	21.3	25.0	25.1 E
75	18.2	19.1	22.4	25.8	26.7
70	19.0	20.0	23.5	26.6	27.5
65	19.8	20.8	24.3	27.4	28.5
60	20.6	21.6	24.9	28.5	29.3 G
55	21.3	22.4	25.5	29.2	29.9
50	22.1	23.1	26.4	30.1	30.9
45	22.7	24.0	27.3	30.8	31.8
40	23.7	24.9	28.1	31.6	32.5 F
35	24.4	26.0	29.0	32.6	33.0
30	25.4	27.0	30.1	33.5	34.3
25	26.6	28.1	31.1	34.3	35.5
20	27.7	29.3	32.1	35.6	36.6 P
15	29.8	31.0	33.3	36.6	38.0
10	32.1	32.8	35.0	37.9	39.3
5	35.4	35.7	37.8	39.6	40.5 VP
1	≥40.5	≥40.0	≥45.5	≥50.8	≥47.0
N =	638	1336	1175	708	250

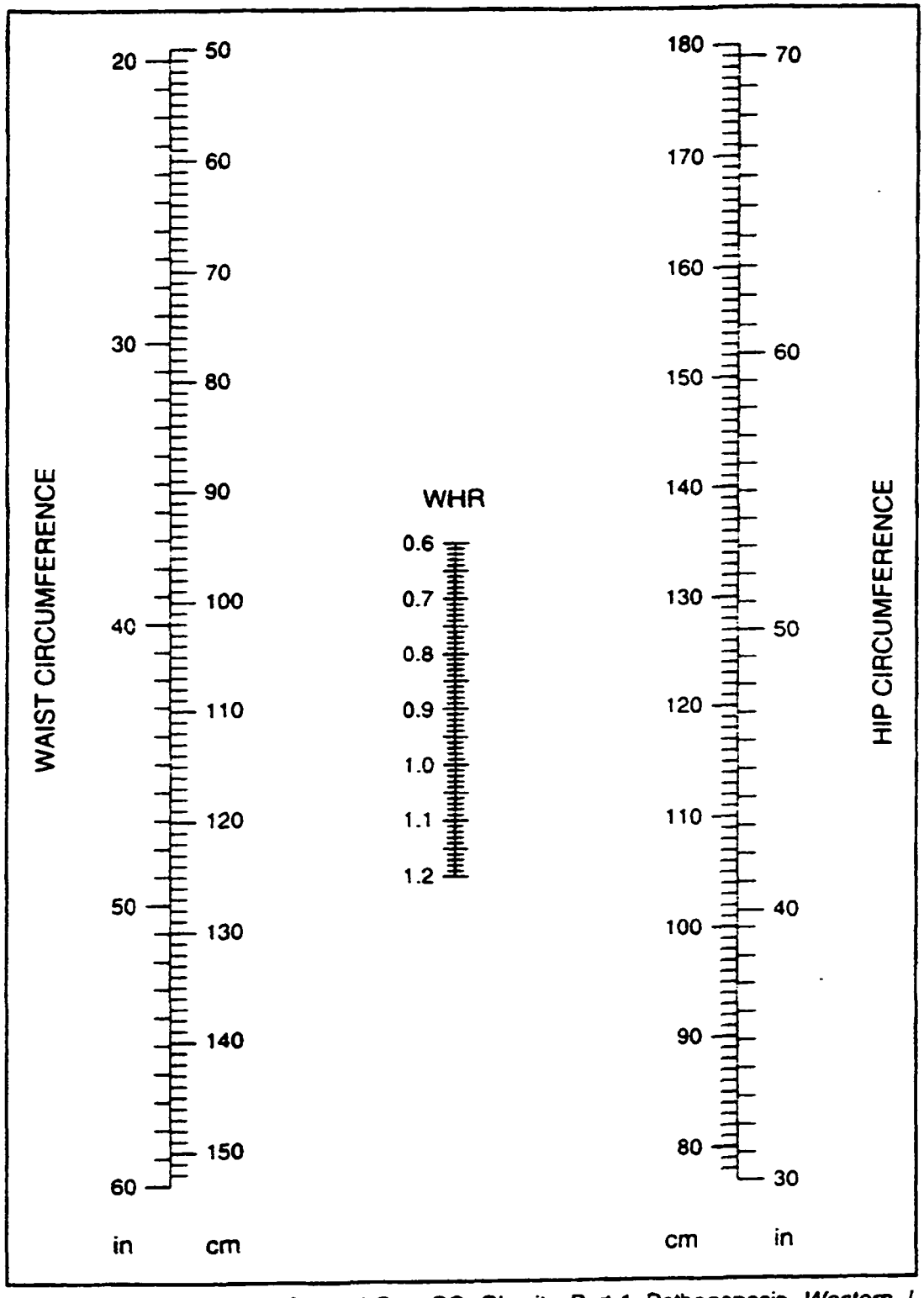
TOTAL N = 4107

*Data provided by the Institute for Aerobics Research, Dallas, TX (1994). S, superior; E, excellent; G, good; F, fair; P, poor; VP, very poor.

BODY MASS INDEX

	BODY MASS INDEX													
	19	20	21	22	23	24	25	26	27	28	29	30	35	40
Height (ins)	Weight (lbs)													
58	91	96	100	105	110	115	119	124	129	134	138	143	167	191
59	94	99	104	109	114	119	124	128	133	138	143	148	173	198
60	97	102	107	112	118	123	128	133	138	143	148	153	179	204
61	100	106	111	116	122	127	132	137	143	148	153	158	185	211
62	104	109	115	120	126	131	136	142	147	153	158	164	191	218
63	107	113	118	124	130	135	141	146	152	158	163	169	197	225
64	110	116	122	128	134	140	145	151	157	163	169	174	204	232
65	114	120	126	132	138	144	150	156	162	168	174	180	210	240
66	118	124	130	136	142	148	155	161	167	173	179	186	216	247
67	121	127	134	140	146	153	159	166	172	178	185	191	223	255
68	125	131	138	144	151	158	164	171	177	184	190	197	230	262
69	128	135	142	149	155	162	169	176	182	189	196	203	236	270
70	132	139	146	153	160	167	174	181	188	195	202	207	243	278
71	136	143	150	157	165	172	179	186	193	200	208	215	250	286
72	140	147	154	162	169	177	184	191	199	206	213	221	258	294
73	144	151	159	166	174	182	189	197	204	212	219	227	265	302
74	148	155	163	171	179	186	194	202	210	218	225	233	272	311
75	152	160	168	176	184	192	200	208	216	224	232	240	279	319
76	156	164	172	180	189	197	205	213	221	230	238	246	287	328

Less than 20	20 - 25	26 to 27	Higher than 27
Weight gain may be advisable	Acceptable range for most people but potential health problems with weight gain	Escalating Health risks	Dramatic risk of health problems



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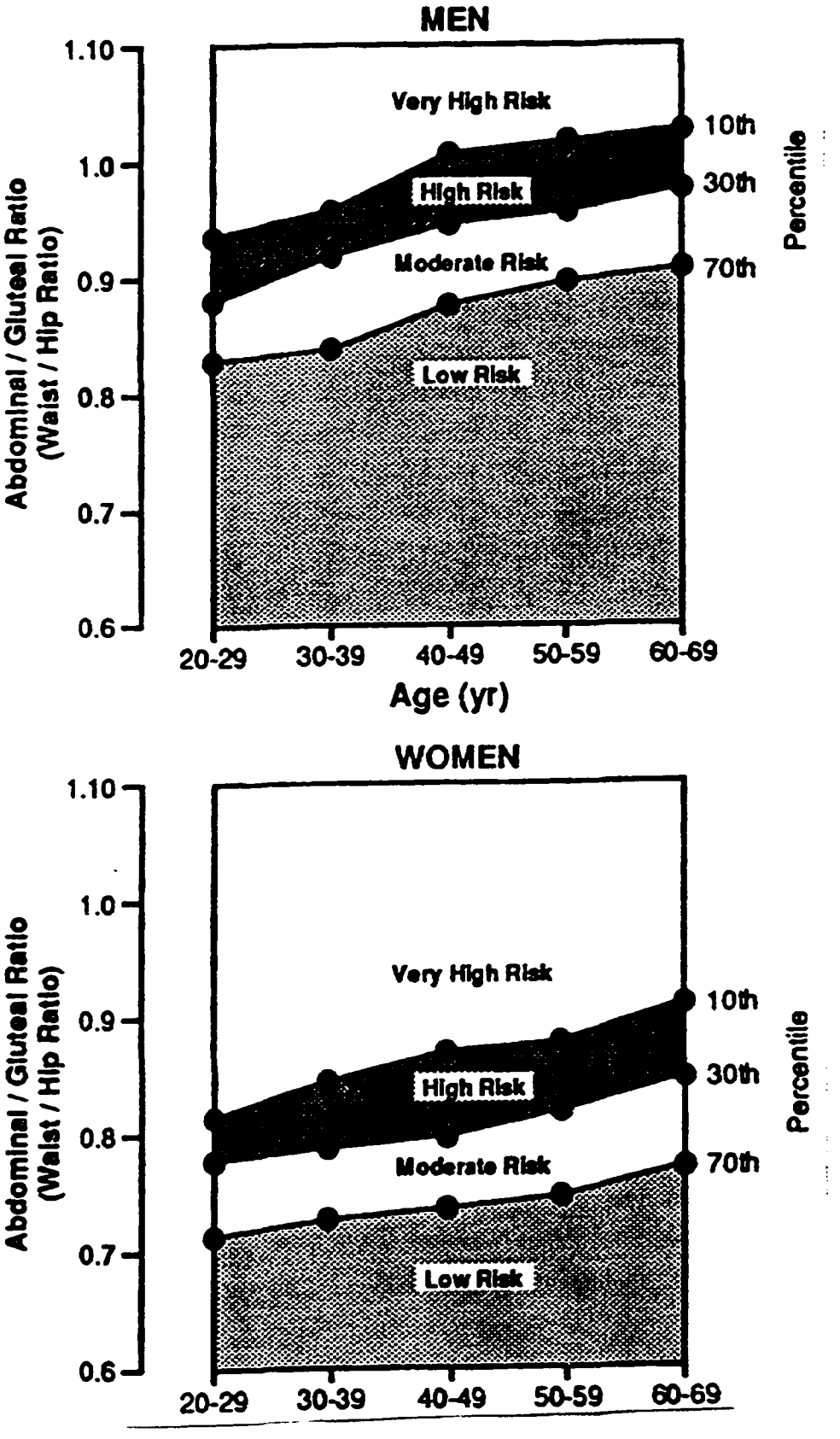

BIBLIOGRAPHY

Adlersberg, D., Schaefer, L., Steinberg, A. & Wang, C. (1956). Age, sex, serum lipids, and coronary artherosclerosis. The Journal of the American Medical Association, 162, 619-622.

Anderson, J., & Chen, W. (1979). Plant fiber: carbohydrate and lipid metabolism. The American Journal of Clinical Nutrition, 32, 346-363.

Anderson, J., Story, L., Sieling, B., Chen, W., Petro, M. & Story, J. (1984). Hypocholesterolemic effects of oat-bran or bean intake for hypercholesterolemic men. The American Journal of Clinical Nutrition, 40, 1146-1155.

Anderson, J., & Tietyen-Clark, J. (1986). Dietary fiber hyperlipidemia, hypertension and coronary heart disease. The American Journal Gastroenterol, 81, 907-919.

Anderson, J. (1987). Dietary fiber, lipids and atherosclerosis. The American Journal of Cardiology, 60, 17G-22G.

Anderson, J. & Bridges, S. (1988). Dietary fiber content of selected foods. The American Journal of Clinical Nutrition, 47, 440-447.

Anderson, J., Zettwoch, N., Feldman, T., Tietyen-Clark, J., Oeltgen, P. & Bishop, C. (1988). Cholesterol-lowering effects of psyllium hydrophilic mucilloid for hypercholesterolemic men. Archives Internal Medicine, 148, 292-296.

Anderson, J., Deakins, D., Floore, T., Smith, B., & White, S. (1990). Dietary fiber and coronary heart disease. Critical Review Food Science Nutrition, 29, 95-147.

Anderson, J., Spencer, D., Hamilton, C., Smith, S., Tietyen, J., Bryant, C. & Oeltgen, P. (1990). Oat-bran cereal lowers serum total and LDL cholesterol in hypercholesterolemic men. The American Journal of Clinical Nutrition, 52, 495-499.

Anderson, J., Gilinsky, N., Deakins, D., Smith, S., O'Neal, D., Dillion, D. & Oeltgen, P. (1991). Lipid response of hypercholesterolemic men to oat-bran and wheat-bran intake. The American Journal of Clinical Nutrition, 54, 678-683.

Anderson, J., Riddell-Mason, S., Gustafson, N., Smith, S. & Mackey, M. (1992). Cholesterol-Lowering effects of psyllium-enriched cereal as an adjunct to a prudent diet in the treatment of mild to moderate hypercholesterolemia. The American Journal of Clinical Nutrition, 56, 93-8.

Assmann, G. & Schulte, H. (1992). Relationship of high density cholesterol and triglyceride to incidence of artherosclerosis artery disease. American Journal of Cardiology, 70, 733-737.

Barakat, H., Burton, D., Carpenter, J., Holbert, D., & Israel, R. (1980). Body fat distribution, plasma lipoproteins and the risk for coronary heart disease of male subjects. Int. J. Obes., 12, 423-430.

Barrett, A. (1964). Adventitious factors affecting the concentration of free fatty acids in the plasma of rats. British Journal of Pharmacology, 22, 577-584.

Barry, S. (1997). Recent nutritional approaches to the prevention and therapy of cardiovascular disease. Prevention, 12, 3-23.

Bell, L., Hectorn, K., Reynolds, H. & Hunninghake, D. (1990). Cholesterol-lowering effects of soluble-fiber cereals as part of a prudent diet for patients with mild to moderate hypercholesterolemia. The American Journal of Clinical Nutrition, 52, 1020-1026.

Bierman, E., & Ross, R. (1977). Aging and atherosclerosis. In Atherosclerosis Reviews. Raven Press: New York.

Bierman, E., & Chait, A. (1988). Nutrition and diet relation to hyperlipidemia and atherosclerosis. Diet and Nutrition in the Prevention and Treatment of Disease, Philadelphia: Lea & Febiger, 1283-1297.

Bioanalogics User Manual. Bioanalogics, Beavelton, OR 1994.

Blincoe, C., & Brody, S. (1951). The influence of temperature on blood composition of cattle. Mo. Agric. Exp. Sta. Res. Bull, 488.

Bridges, S., Anderson, J., Deakins, D., Dillion, D. & Wood, C. (1992). Oat bran increases serum acetate of hypercholesterolemic men. The American Journal of Clinical Nutrition, 56, 455-459.

Brown, M., & Goldstein, J. (1984). How LDL receptors influence cholesterol and atherosclerosis. Science American, 251, 58-66.

Brownell, K., Bachorik, P. & Ayerle, R. (1982). Changes in plasma lipid and lipoprotein levels in men and women after a program of moderate exercise. Circulation, 65, 477-484.

Caggiula, A., & Mustad, V. (1997). Effects of dietary fat and fatty acids on coronary artery disease risk and total and lipoprotein cholesterol concentrations: epidemiologic studies. The American Journal of Clinical Nutrition, 65, 1597S-1610S.

Cassidy, M., & Calver, R. (1993). Effects of dietary fiber on intestinal absorption of lipids. CRC Handbook of Dietary Fiber in Human Nurtrition, 2nd edition, CRC Press, Inc., New York, 1993.

Council on Scientific Affairs. (1989). Dietary fiber and health. The Journal of the American Medical Association, 262, 544-546.

Darmady, J., Fosbrooke, A. & Lloyd, J. (1972). Prospective study of serum cholesterol levels during first year of life. British Medicine Journal, 2, 685-686.

Dawber, T. (1975). Risk factors for atherosclerotic disease. In Current Concepts, Missouri: Upjohn.

Dietary guidelines for healthy Americans: a statement for physicans and health professionals by the Nutrition Committee, American Heart Association. (1988). Arteriosclerosis, 8, 218A-221A.

Dyerberg, D. & Hjorne, N. (1973). Plasma lipid and lipoprotein levels in childhood and adolescence. Scand. J Clin Lab, 31, 473-479.

Duncan, J., Gordon, N. & Scott, C. (1991). Women walking for health and fitness: how much is enough? The Journal of the American Medical Association, 266, 3259-3299.

Durstine, L. & Haskell, W. (1994). Effects of exercise training on plasma lipids and lipoproteins. Exercise and Sports Science Reviews, 22, 477-522.

Edgren, R. (1963). Seasonal variations in rat blood cholesterol concentrations and its responsiveness to estrone. Journal of Atherosclerosis Research, 3, 206-209.

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

Expert Panel on Detection, Evaluation, and Treatment of High blood Cholesterol in Adults. (1993). Summary of the Second Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). The Journal of the American Medical Association, 16, 3015-3023.

Fernandez, M. (1995). Distinct mechanisms of plasma LDL lowering by dietary fiber in the guinea pig: specific effects of pectin, guar gum, and psyllium. Journal of Lipid Research, 36, 2394-2404.

Frayn, K. (1996). Metabolic Regulation A Human Perspective, London: Portland Press.

Fyfe, T., Dunnigan, G., Hamilton, E. & Rae, R. (1968). Seasonal variation in serum lipids, and incidence and mortality of ischemic heart disease. Journal of Atherosclerosis Research, 8, 591-596.

Fredrickson, D., Levy, R., & Lees, R. (1967). Fat transport in lipoproteins-an integrated approach to mechanisms and disorders. England Journal of Medicine, 276, 148-156.

Genest, J. & Cohn, J. (1995). Clustering of cardiovascular risk factor: targeting high-risk individuals. American Journal of Cardiology, 76, 8A-20A.

Glueck, C., Heckman, F., Schoenfeld, M., Steiner, P. & Pearce, W. (1971). Neonatal familial type II hyperlipoproteinemia: Cord blood cholesterol in 1,800 births. Metabolism, 20, 597-608.

Goldstein, J., Hazzard, W., Schrott, H., Bierman, E., & Motulsky, A. (1973). Hyperlipidemia in coronary heart disease. I. Lipid levels in 500 survivors of myocardial infarction. Journal of Clinical Investigation, 52, 1533-1543.

Gresham, G. (1972). Atherosclerosis in man: Natural history effects. Proc. Nutr. Soc., 31, 303.

Grundy, S. (1986). Cholesterol and coronary heart disease: A new era. Journal of the American Medical Association, 256, 2849-2858.

Grundy, S. (1990). Cholesterol and coronary heart disease: future directions. Journal of the American Medical Association, 264, 3053-3059.

Gordon, T., Castelli, W., Hjortland, MC., Kannel, W., & Dawber, TR. (1977). High density lipoproteins as a protective factor against coronary heart disease: The Framingham Study. American Journal of Medicine, 55, 767-72.

Gorski, J., Oscai, L., & Palmer, W. (1990). Hepatic lipid metabolism in exercise and training. Med. Sci. Sports Exerc., 22, 213-221.

Guntelberg, F., Brennan, R., Holloszy, J., Schonfeld, G., Rennie, M., & Weidman, S. (1977). Plasma triglyceride lowering by exercise despite increased food intake in patients with type IV hyperlipoproteinemia. American Journal of Clinical Nutrition, 30, 716-720.

Hagan, R., & Gettman, L. (1983). Maximal aerobic power, bodyfat, and serum lipoproteins in male distance runners. Journal of Cardiac Rehabiliation, 3, 331-337.

Hartung, H., Foreyt, J., Mitchell, R., Vlasek, I., & Gotto, A. (1980). Relation of diet to high-density-lipoprotein cholesterol in middle-aged marathon runners, joggers, and inactive men. The New England Journal of Medicine, 302, 357-361.

Hartung, H., Squires, W., & Gotto, A. (1981). Effect of exercise training on high-density lipoprotein cholesterol in coronary disease patients. American Heart Journal, 101, 181-184.

Haskell, W. L. (1984). The influence of exercise on the concentrations of triglyceride and cholesterol in human plasma. R.L. Terjung (ed). Exercise and Sport Sciences Reviews. Lexington, MA: Collamore Press, 205-244.

Haskell, W., Spiller, G., Jensen, C., Ellis, B., & Gates, J. (1992). Role of water-soluble dietary fiber in the management of elevated plasma cholesterol in healthy subjects. The American Journal of Cardiology, 69, 433-439.

Herbert, P., Bernier, D., Cullinane, E., Edelstein, L., Kantor, M., & Thompson, P. (1 984). High-density lipoprotein metabolism in runners and sedentary men. The Journal of the American Medical Association, 252, 1034-1037.

Hollister, L. & Wright, A. (1956). Diurnal variation of serum lipids. Journal of Atherosclerosis Research, 5, 445-450.

Holloszy, J., Skinner, J., Toro, G. & Cureton, T. (1964). Effects of a six-month program of endurance exercise on the serum lipids of middle-aged men. The American Journal of Cardiology, 14, 753-760.

Hulley, SB., Cohen, R., & Widdowson, G. (1977). Plasma high-density lipoprotein cholesterol level: influence of risk factor intervention. The Journal of the American Medical Association, 238, 2269-2271.

Huttunnen, J., Lansimies, E., Voutilainen, E., Ehnholm, C., Hietanen, E., Penttila, I., Siitonen, O., & Rauramaa, R. (1979). Effect of moderate physical exercise on serum lipoproteins. Circulation, 60, No. 6, 1220-1229.

Jensen, C., Spillar, G., Gates, J., Miler, A. & Whitman, J. (1993). The effects of acacia gum and a water-soluble dietary fiber mixture on blood lipids in humans. Journal of American College of Nutrition, 12, 147-154.

Judd, P. & Truswell, A. (1988). The effect of rolled oats on blood lipids fecal steroid excretion in man. The American Journal of Clinical Nutrition, 118, 144-151.

Kanter, M., Cullinane, E., Sady, S., Herbert, P. & Thompson, P. (1987). Exercise acutely increases high density lipoproteins-cholesterol and lipoproteins lipase activity in trained and untrained men. Metabolism, 36, 188-192.

Kato, H., Tillotson, J., Nichaman, JZ., Rhoads, GG. & Hamilton. (1973). Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California. American Journal of Epidemiology, 97, 372-85.

Katz, L., & Stamler, J. (1953). Experimental Artherosclerosis. Illinosis: Charles C. Thomas.

Kendler, Barry. (1997). Recent nutritional approaches to the prevention and therapy of cardiovascular disease. Progress in Cardiovascular Nursing, 12, 3-23.

Kestin, M., Moss, R., Clifton, P. & Nestel, P. (1990). Comparative effects of three cereal brands on plasma lipids, blood pressure, and glucose metabolism in mildly hypercholesterolemic men. The American Journal of Clinical Nutrition, 52, 661-666.

Keys, A. (1970). Coronary heart disease in seven countries. Circulation, 41, I162-83.

Kiens, B., Gad, P., Lithell, H., & Vessby, B. (1981). Minor dietary effects on HDL in physically active men. European Journal of Clinical Investigation, 11, 265-271.

King, A., Haskell, W., Young, D., Oka, R. & Stefanick, M. (1995). Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50-65 years. Circulation, 91, 2956-2604.

Kinosian, B. & Eisenberg, J. (1988). Cutting into cholesterol: cost-effective alternative for treating hypercholesterolemia. The Journal of the American Medical Association, 259, 2249-2254.

Kirby, R., Anderson, J., Sieling, B., Rees, D., Chen, W., Miller, R. & Kay, R. (1981). Oat-bran intake selectively lowers serum low-density lipoprotein cholesterol concentrations of hypercholesterolemic men. The American Journal of Clinical Nutrition, 34, 824-829.

Kobashigawa, J. (1995). Effect of pravastatin on outcomes after cardiac transplantation. New England Journal of Medicine, 333, 621-627.

Kramsch, D., Aspen, A., Abramowitz, B., Kreimendahl, T. & Hood, W. Jr. (1981). Reduction of coronary atherosclerosis by moderate conditioning exercise in monkeys on an atherogenic diet. New England Journal of Medicine, 305, 1483-1489.

Kuusela, P., Voutilainen, E., Kukkonen, K., & Rauramaa, R. (1980). Lipoprotein patterns in lumberjacks. Scadd Journal of Sports Science, 2, 13-16.

LaRosa, J.H., Becker, D.M., & Fitzgerald S. (1990). Elevated blood cholesterol. A risk factor for coronary heart disease. American Association of Occupational Health Nurses, 38, 211-215.

Leon, A.S., Connet, J. & Raurama, R. (1987). Leisure-time physical activity levels and risk of coronary heart disease and death. The Journal of the American Medical Association, 258, 2388-2391.

Lee, R., Nieman, D., Raval, R., Blankenship, J. & Lee, J. (1991). The effects of acute moderate exercise on serum lipids and lipoproteins in mildly obese women. International Journal of Sports Medicine, 12, 537-542.

Life Sciences Research Office, Federation of American Societies for Experimental Biology. (1987). Physiological effects and health consequences of dietary fiber. Washington, DC.

Lipid Research Clinics Program: The lipid research clinics coronary primary prevention trial results. (1984). The Journal of the American Medical Association, 251, 351-364.

Lloyd, J., & Wolff, O. (1969). A pediatric approach to the prevention of atherosclerosis. Atherosclerosis Research, 10, 135.

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

Marti, B., Suter, E., Riesen, W., Tschopp, A., Wanner, H., & Gutzwiller, F. (1990). Effects of long-term, self-monitored exercise on the serum lipoprotein and apolipoprotein profile in middle-aged men. Atherosclerosis, 81, 19-31.

Marti, B., Knobloch, M., Riesen, W., & Howard, H. (1991). Fifteen year changes in exercise, aerobic power, abdominal fat, and serum lipids in runners and controls. Medical science of Sports Exercise, 23, 115-122.

McGill, H. (1968). Introduction to the geographic pathology of artherosclerosis. Lab. Invest., 18, 465-467.

Montoye, H., Block, W., & Gayle, R. (1978). Maximal oxygen intake and blood lipids. Journal of Chronic Disease, 31, 111-118.

National Cholesterol Education Program (NCEP). (1988). Report of the national cholesterol program expert panel on detection. Archives Internal Medicine, 148, 36-69.

National Institutes of Health Consensus Development Conference on lowering blood cholesterol to prevent heart disease. (1985). The Journal of the American Medical Association, 253, 2080-2086.

National Research Council. (1989). Diet and Health Implications for Reducing Chronic Disease Risk, Washington: National Academy Press.

Newsholme, E., & Leech, A. (1983). Biochemistry for medical sciences. Chichester: John Wiley.

Nikkila, E., Taskinen, M., Rehunen, S., & Harkonen, M. (1978). Lipoprotein lipase activity in adipose tissue and skeletal muscle of runners: relation to serum lipoprotiens. Metabolism, 27, 1661-1671.

Nikkila, E., Kuusi, T., & Myllynen, P. (1980). High-density lipoproteins and apolipoprotien A-I during physical inactivity. Artherosclerosis, 37, 457-462.

O' Kelly, J. (1972). Seasonal variations in the plasma lipids of genetically different types of cattle: grazing steers. Comparative Biochemistry Physiology, 43B, 283-294.

Parhofer, K., Richter, W., & Schwandt, P. (1996). Drug therapy of severe hypercholesterolemia in patients with coronary artery disease. Artificial Organs, 20, 286-291.

Pedersen, T. (1995). Lowering cholesterol with drugs and diet. New England Journal of Medicine, 333, 1350-1351.

Peeples, L., Carpenter, J., Israel, R., & Barakat, H. (1989). Alterations in low-density lipoproteins in subjects with abdominal adiposity. Metabolism, 38, 1029-1036.

Peltonen, P., Marniemi, J., Hietanen, E., Vuori, I., & Ehnholm, C. (1981). Changes in serum lipids, lipoproteins, and heparin releasable lipolytic enzymes during moderate physical training in man: a longitudinal study. Metabolism, 30, 518-526.

Physiological effects and health consequences of dietary fiber. (1987). Life Sciences Research office, Federation of American societies for Experimental Biology. Washington, DC.

Pilch, S. (1987). Physiological effects and health consequences of dietary fiber. LifeSciences Research office, Federation of American societies for Experimental Biology. Bethesda, Maryland.

Oscari, L., Essig, D., & Palmer, W. (1990). Lipase regulation of muscle triglyceride hydrolysis. Journal of Applied Physiology, 69, 1571-1577.

Ragsdale, A., Worstell, D., Thompson, H., & Brody, S. (1949). Influence of temperature 50^0-0^0 F and 50^0-95^0 F on milk production, feed and water consumption and body weight in Jersey and Holstein cows. Mo. Agric. Exp. Sta. Bull., 449.

Ready, A. E. (1996). Walking program maintenance in women with elevated serum cholesterol. Behavioral Medicine, 22, 23-31.

Rotkis, T., Cote, R., Coyle, E., & Wilmore. J. (1982). Relationship between high-density lipoprotein cholesterol and weekly running mileage. Journal of Cardiac Rehabilitation, 2, 109-112.

Sabine, John. Cholesterol. (1997). New York: Marcel Dekker Inc.

Sacks, F., Castelli, W., Donner, A. & Kass, E. (1975). Plasma lipids and lipoproteins in vegetarians and controls. New England Journal of Medicine, 292, 1148-1151.

Seals, D., Hagberg, J., Hurley, B., Ehsani, A., & Holloszy, J. (1984). Effects of endurance training on glucose tolerance and plasma lipid level in older men and women. The Journal of the American Medical Association, 252, 645-649.

Shils. M., & Young, V. (1988). Modern Nutrition In Health and Disease. 7th ed. Philadelphia: Lea & Febiger.

Smolin, L., & Grosvenor, M. (1977). Nutrition Science and Applications. 2nd ed. Fort Worth: Saunders College Publishing.

Steele, B. (1996). Timed walking tests of exercise capacity in chronic cardiopulmonary illness. Journal of Cardiopulmonary Rehabilitation, 16, 25-33.

Stein, R., Michielli, D., Glantz, M., Sardy, H., Cohen, A., Goldberg, N., & Brown, C. (1989). Effects of different exercise training intensities on lipoprotein cholesterol fractions in healthy middle-aged men. American Heart Journal, 119, 277-283.

Storch, K., Anderson, J., & Young, V. (1984). Oat bran muffins lower serum cholesterol of healthy young people. Clinical Research, 34, 740A.

Thompson, G. (1995). Familial hypercholesterolaemia regression study: a randomized trial of low-density-lipoproteins apheresis. Lancet, 345, 811-816.

Thompson, P., Lazarus, B., Cullinane, E., Henderson, O., & Musliner, T. (1983). Exercise, diet, or physical characteristics as determinants of HDL-levels in Endurance athletes. Atherosclerosis, 46, 333-339.

Thompson, P., Cullinane, E., Sady, S., Flynn, M., & Bernier, D. (1988). Modest changes in high-density lipoprotein concentrations and metabolism with prolonged exercise training. Circulation, 78, 25-34.

Thompson, P., Cullinane, E., Sady, S., Glynn, F., Chenevert, C., & Herbert, P. (1991). High density lipoprotein metabolism in endurance athletes and sedentary men. Circulation, 84, 140-152.

Throp, J., & Waring, W. (1962). Metabolism and distribution of lipids of ethyl Chlorophenoxyisobutyrate. Nature, 194, 948-949.

Tsopanakis, C., Kotsarellis, D. & Tsopanakis, D. (1986). Lipoprotein and lipid profiles of elite athletes in Olympic sports. International Journal of Sports Medicine, 1, 316-321.

VanHorn, L., Liu, K., Parker, D., Emidy, L., Liao, Y., Pan, W., Giumetti, D., Hewitt, J., & Stamler, J. (1986). Serum lipid response to oat product intake with a fat-modified diet. Journal of American Diet Association, 86, 759-758.

Weltman, A., Matter, S., & Stamford, B. (1980). Caloric restriction and/or mild exercise Effects on serum lipids and body composition. American Journal of Clinical Nutrition, 33, 1002-1009.

Williams, P., Wood, P., Haskell, W. & Vranizan, K. (1982). The effect of running mileage and duration on plasma lipoprotein levels. The Journal of the American Medical Association, 247, 2674-2679.

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

Williams, Sue. (1997). Nutrition and Diet Therapy. St. Louis: Mosby.

Winkel, P., Stratland, B., & Bokelund, H. (1974). Factors contributing to intra individual variation of serum constituents: Short-term day-to-day and within-hour variation of serum constituents in healthy subjects. Clinical Chemistry, 20, 1520-1527.

Witztum, J. (1989). Current approaches to drug therapy for the hypercholesterolemic patient. Circulation, 80, 1101-1114.

Wood, P., & Stefanick, M. (1990). Exercise, fitness, and atherosclerosis. Bouchard, C., Shephard, J., stephens, T., Sutton, J., & McPherson, B. (ed). Exercise, Fitness, and Health. Champaign IL: Human Kinetics Books, 409-423.

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