Total Blood Cholesterol, Blood Triglyceride, and Blood HDL Correlation

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TOTAL BLOOD CHOLESTEROL, BLOOD TRIGLYCERIDE, AND BLOOD HDL CORRELATION

by

Rebecca Lynn Babler

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
Degree of Master of Arts
Department of Health, Physical Education, and Recreation

Western Michigan University
Kalamazoo, Michigan
June 1991
The purpose of this investigation was to determine the relationship between total blood cholesterol levels, blood triglyceride levels, and blood HDL levels in men and women 20 to 80 years of age. All subjects fasted for 12 hours prior to having their blood drawn.

The computer program SPSSX (SPSSX, Inc., 1988) was used along with subprograms CONDESCRIPTIVE, CORRELATION, and ANOVA. Pearson Product Moment Correlations were calculated for all subjects, for male subjects, and for female subjects.

The study revealed a low correlation coefficient between blood cholesterol levels and blood triglyceride levels for all subjects, female subjects and male subjects. There was an interaction effect between age group and gender using cholesterol as the dependent variable and there was an interaction effect between age group and gender using triglyceride as the dependent variable.
ACKNOWLEDGEMENTS

I wish to thank my advisor, Dr. Mary Dawson, for her time in making this thesis possible. Very special thanks go to my mother for her time and assistance.

Rebecca Lynn Babler
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Total blood cholesterol, blood triglyceride, and blood HDL correlation

Babler, Rebecca Lynn, M.A.

Western Michigan University, 1991
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CHAPTER I

INTRODUCTION

The National Institute of Health (NIH) has enacted a campaign that encourages people to know their blood cholesterol levels. The NIH's National Cholesterol Education Program is a nationwide cholesterol screening crusade. "Those with high levels," the NIH has stated, "should take steps to reduce their blood cholesterol level because of its atherogenic implications" (Parke Davis, 1986, p.7). The American Heart Association (AHA) recommends measuring not only total blood cholesterol but also triglycerides, possibly low density lipoproteins (LDL), and high density lipoproteins (HDL) to determine one's risk of developing coronary heart disease (Parke Davis, 1986, p.7). Recent studies have demonstrated that the issue of lipids and atherosclerosis is clearly more complicated than simply high total blood cholesterol levels (e.g., Grundy, 1966; Kannel, 1987).

Problem Statement

The purpose of this investigation is to determine the relationship between total blood cholesterol levels, HDL
levels and blood triglyceride levels and their relationship to age group and gender. Men and women 20 to 80 years of age were tested.

Since 1984, the National Heart, Lung and Blood Institute has encouraged cholesterol testing for the general population because of its implications for coronary artery disease (Marwick, 1986). It has been hypothesized that other lipid fractions such as triglycerides are also (independent) risk factors for coronary artery disease and atherosclerosis (Genest, McNamara, Ordovas, & Schaeffer, 1988). A low correlation coefficient between blood total cholesterol and triglycerides would indicate a need for a more in-depth blood screening procedure.

Presently, a national effort exists to inform people about blood total cholesterol levels and their atherosclerotic implications. Due to this national effort, people may be led to believe that total cholesterol is the only lipid fraction associated with atherosclerosis and coronary artery disease.

Delimitations

Subjects were limited to men and women tested during the past five years at the South Bend Medical Foundation, South Bend, Indiana. Subjects were within the 20 to 80 year age range at the time of testing.
Limitations

A limiting factor in this study was that the subjects' cholesterol and triglyceride levels were intended to represent the status of a normal healthy population. It was possible that some patients may have been undiagnosed victims of atherosclerotic disease.

Assumptions

The assumptions of the study were that: (a) subjects would maintain a fasting state for twelve hours prior to testing, and (b) the laboratory tests would be performed accurately.

Hypotheses

The study included the following research hypotheses:

1. A low positive correlation was expected between total cholesterol levels and triglyceride levels.
2. A low positive correlation was expected between total cholesterol levels and HDL levels.
3. A high negative correlation was expected between triglyceride levels and HDL levels.
4. HDL levels will be higher in females than in males.
5. Triglyceride levels will be higher in males
compared to females.

6. Total cholesterol will increase with age for females.

7. Total cholesterol levels will increase with age for males.

Definition of Terms

1. Antithrombogenic: an anti-clot producing action.

2. Apolipoproteins: proteins that combine with lipids to form lipoproteins.

3. Atherosclerosis: a form of arteriosclerosis in which there are localized accumulations of lipids containing material within or beneath the intima surface of blood vessels.

4. Cholesterol: a steroid widely distributed in animal tissue and found in various oils and fats.

5. Chylomicrons: small particles of fat in the blood after digestion and absorption of fat.

6. HDL: high density lipoproteins which are a fraction of total cholesterol.

7. IDL: intermediate density lipoproteins which are a fraction of total cholesterol.

8. LDL: low density lipoproteins which are a fraction of total cholesterol.
9. Lipids: another name for fat.

10. Lipoproteins: conjugated proteins consisting of simple proteins combined with lipid components.

11. Phospholipids: conjugated proteins consisting of simple proteins combined with lipid components.

12. Triglycerides: lipid substances that contain phosphorus and fatty acids.

13. VLDL: very low density lipoproteins which are a fraction of total cholesterol.
CHAPTER II

REVIEW OF LITERATURE

The review of literature pertinent to this study is divided into eight main areas: (1) atherosclerosis, (2) plaque accumulation, (3) lipids, (4) lipid biochemistry, (5) lipid fractions, (6) lipid profiles, (7) hypertriglyceridemia, and (8) atherosclerosis and diabetes.

Atherosclerosis

Atherosclerosis is a complex process affecting a number of arteries. This process initially involves the internal layer of the arterial wall in which there is focal accumulation of a variety of complex lipids, proteins and carbohydrates. Atherosclerosis involves cellular components such as smooth muscle cells and macrophage-like cells. In more advanced lesions, blood constituents and minerals, such as calcium, are present (St. Clair, 1987).

The first pathological change in atherosclerosis is the fatty streak. Fatty streaks contain both foam cells and extra-cellular lipids principally located in the intima, the innermost coat of the blood vessel. In the
beginning stages of atherosclerosis, there is a yellow streaked appearance observable on the inter-arterial wall.

A comprehensive study regarding atherosclerosis is the Geographic Pathological Study conducted in the 1950s and 1960s (St. Clair, 1987). The study reviewed the extent and severity of atherosclerosis in 19 countries in individuals of different ethnic and socioeconomic background. The study revealed that fatty streaks developed in all populations during the first two decades of life. Juvenile fatty streaks consisted of cholesterol ester laden foam cells, while more advanced lesions in older individuals had fibrous plaque formation.

**Plaque Accumulation**

Initially, cholesterol esters accumulate within foam cells, but as the lesions become more fibrous and necrotic, large amounts of extracellular cholesterol esters are found (St. Clair, 1987). The cholesterol of the atherosclerotic lesion is derived from the plasma. The cellular elements of the arterial wall play an essential role in the formation of the lesion. Cholesterol esters accumulate within foam cells as a result of the esterification of plasma derived cholesterol. Since fatty acids are required for esterification to cholesterol, fatty
acid synthesis is also increased. Changes in phospholipid and triglyceride synthesis by the atherosclerotic lesion also occur. Endothelial cells, macrophage cells and smooth muscle cells all interact in a complex manner in the formation of an atherosclerotic plaque.

Healthy endothelia, the vasculature cell lining, plays an important role in limiting permeability of macromolecules and in providing an antithrombogenic effect, which prevents blood clotting on its surface. It may also alter the lipoproteins that pass near the endothelia (St. Clair, 1986). When the endothelia is injured by plaque, metabolic dysfunction, altered permeability and antithrombogenic effects occur.

Macrophages have the ability to take up abnormal lipoproteins and become foam cells. Macrophages also produce a powerful mitogen, an agent that stimulates proliferation of endothelial cells and smooth muscle cells. This proliferation of cells also stimulates LDL receptor synthesis and may promote LDL uptake. If the rate of LDL cholesterol uptake exceeds the rate of cholesterol efflux, then the smooth muscle cells may become foam cells. The interaction of the cellular components of the arterial wall and lipids is a complex process. The final product is atherosclerotic plaque.

The close link between cholesterol blood levels and
atherosclerosis appears indisputable (Assman, 1982; Grundy, 1986; Stamler, Wentworth & Neaton, 1986; and Tzagournis, 1987). This theory is widely accepted even though the mechanism for disposition of the plasma cholesterol in the atherosclerotic plaque is not clear. Elevated blood triglyceride levels may be an important risk factor for premature cardiovascular disease. It is, however, accepted that elevated cholesterol alone does not signify the propensity for atherosclerosis for every patient. Several other biochemical processes contribute as well.

Dyslipidemia is lipid dysfunction, an imbalance of lipids and lipoproteins, which are the substances that carry lipids to and from body tissues by way of the blood stream. Lipoproteins are water soluble complexes of high molecular weight. They are composed of lipids such as cholesterol, triglycerides, and phospholipids and one or more specific proteins called apolipoproteins. The lipids and proteins bond in a fixed ratio. Lipoproteins represent the functional unit of transport for water insoluble lipids in the blood.

Lipids are important because of their physiological function as well as their presumed association with vascular disease. Cholesterol and phospholipids make up important structural portions of cells, especially cell
membranes. Triglycerides serve as a source of calories (Tzagournis, 1987). Imbalances, especially the increase in certain lipids, cause biochemical disorders such as coronary heart disease and atherosclerosis.

Lipids

The terms "fats" and "lipids" are synonymous. Cholesterol and triglycerides are both lipids, but have different chemical entities. The lipids in the bloodstream, cholesterol and triglycerides, have two origins. They are produced by the body or come from the diet. The liver produces most of the cholesterol and triglycerides in the body, and the intestine contributes the rest. The cholesterol we consume is found exclusively in animal products. Cholesterol is used in the production of cell membranes, the production of adrenal glands and the production of sex glands. Unlike cholesterol, triglycerides are found in animal and vegetable fats. Most triglycerides from vegetable sources contain polyunsaturated fats. In contrast, triglycerides from animal fat are rich in saturated fats.

Dyslipidemia, which is an abnormal lipid balance in the blood, may be due to one of many causes. Some types of dyslipidemia are due to digestive disorders, liver disorders, or thyroid disorders. Other types may be due
to diseases such as diabetes mellitus. Still other types of dyslipidemia are hereditary or due to abnormal diets or an abnormal response to normal diet. The unusual buildup and breakdown of lipids may cause other physiological changes that may result in atherosclerosis (Assman, 1982).

Lipid Biochemistry

The understanding of the biochemistry of lipids and lipoproteins is paramount in understanding the contribution each provides to dyslipidemia. The triglyceride molecule is composed of three fatty acids bonded to the glycerol backbone which serves as the main transporter of fatty acids to tissues and stored adipocytes. The fatty acid composition of triglycerides in plasma and adipose tissue is determined in large part by the composition of dietary fats. The major fatty acids in plasma are linoleic acid, and γ-linolenic acid. These are essential fatty acids that cannot be synthesized in the body and must be obtained from the diet. Linoleic acid is converted by intermediates to arachidonic acid. Derivatives of linoleic acid and γ-linolenic acids serve as precursors for prostaglandins which have significant biologic effects, including effects on blood pressure, smooth muscle cells, reproduction platelets, red blood cells,
and immune response.

Most of the steps of the enzymatic biosynthesis of cholesterol are now known as a result of the research of Bloch in the United States, Lynen in Germany and Papjak and Conforth in Great Britain in the 1940s (cited in Lehninger, 1975). The last stereochemical details of the structure of cholesterol were not known until 1955. Two German chemists, Windaus and Willant, were responsible for outlining the structure of cholesterol. Cholesterol is a steroid with a unique combination of eight chiral centers. The complex synthesis requires 25 reaction steps (cited in Lehninger, 1975).

Biosynthesis of cholesterol in the liver is suppressed by dietary cholesterol and by fasting, an effect that is caused by depression of the biosynthesis of beta-hydroxy-methylglutaryl-CoA reductase in the liver. However, cholesterol itself appears not to be the inhibitor. It has been postulated that cholesterol containing lipoprotein, a bile acid, or a specific protein found in bile is the true inhibitor. Fasting also inhibits cholesterol biosynthesis, but high fat diets accelerate the process (Lehninger, 1975).

Lipid Fractions

The transporter of triglycerides is lipoproteins
synthesized by the intestine. These lipoproteins, chylomicrons, also contain cholesterol, phospholipid, and various apolipoproteins. Also contained in the chylomicrons are the fat soluble vitamins A, D, E, and K, (Genest, et al., 1988).

After entry into the plasma, spare chylomicrons and triglycerides are rapidly hydrolyzed to form free fatty acid and glycerol by the action of lipoprotein lipase. Lipoprotein lipase is activated by apo-C-II, and its activity is inhibited by apo-C-III. The acceptor for free fatty acids is albumin and there are two tight binding sites for long chain fatty acids per albumin molecule. Free fatty acids are also produced after hydrolysis of triglyceride within adipocytes through the action of hormone-sensitive lipase. This enzyme is regulated by cyclic adenosine monophosphate. Free fatty acid release from adipose tissue is enhanced by exercise, stress, and fasting as well as in uncontrolled diabetes. Free fatty acids have a resistance time of approximately five minutes in plasma and are rapidly taken up by muscle and other tissues as well as the liver (Genest et al., 1988).

Chylomicrons are lipoprotein particles with a density of 0.95 g/ml (Assman, 1982). These particles are formed exclusively in the mucosal cells of the duodenum.
and jejunum and serve to transport triglycerides, as well as phospholipids, 5%, cholesterol, 3%, and cholesteryl esters and protein, 2% (Steiner, 1986). The principal apolipoprotein components are those apolipoproteins synthesized in the intestinal mucosa apo A-I, apo A-II, apo A-IV and apo B-48. After entering the general circulation via the thoracic duct, the chylomicrons have a half-life of only a few minutes. They are degraded to core remnants and enriched in cholesteryl esters, apolipoprotein E, surface remnants, phospholipids, cholesterol, and apolipoprotein C by the action of lipoprotein lipase found in muscle and adipose tissue. Consequently, in the absence of metabolic disease, chylomicrons are only found in serum after a high-fat meal and not in serum of a fasting patient.

Over 90% of the triglyceride present in fasting blood is synthesized in the liver and secreted into the blood as a component of VLDL (very low density lipoprotein). VLDL particles do not represent a single substance, but are a heterogeneous mixture of macromolecules that float in the 0.95 to 1.00 g/ml density range (Steiner, 1986). Triglycerides compose 60-70% of the VLDL mass, 10-15% is phospholipids and cholesterol, and about 10% is protein. VLDL particles undergo degradation in the plasma under the action of lipoprotein
lipase to IDL (intermediate density lipoprotein) and further to LDL (low density lipoprotein).

Approximately 65-70% of the total cholesterol is transported in the LDL. The LDL particles float in the density range between 1.019 and 1.063 g/ml upon ultracentrifugation. The LDL particles are composed of about 75% lipid (primarily cholesterol esters, cholesterol and phospholipids) and 25% protein. Cholesterol esters and triglycerides form the hydrophobic core which is surrounded by a surface coat of apolipoprotein B and phospholipid components (Assman, 1982).

The plasma of fasting subjects with plasma triglyceride rich lipoproteins is found in the IDL intermediate density lipoproteins fraction. IDLs are considered a distinct fraction of lipoproteins. Individuals with an extremely elevated triglyceride level have an increase in numbers of IDL particles, rather than an increase in size of the triglyceride-rich lipoprotein. IDL is a remnant of VLDL. They are relatively rich in triglycerides and when triglyceride levels are elevated because of increased production rates of VLDL, this excessive input of VLDL can be a cause of increased concentration of IDL (Grundy & Vega, 1988). Since there is a positive correlation between plasma total triglyceride levels and concentrations of IDL, hypertriglyceridemia often denotes
the presence of elevated IDL concentrations and in turn may reflect an increased risk for coronary heart disease. HDL does not represent a single unified substance, but consists of a heterogeneous mixture of macromolecules differentiated by particle size (Assman, 1982). HDL floats at a density of 1.063-1.21 g/ml. Approximately 50% of the mass of HDL is protein, 30% phospholipid, 10-20% cholesterol and cholesterol esters and 5% triglyceride. It is generally recognized that HDL exists as globular particles with symmetry. HDL is formed essentially in the plasma. The biosynthesis of HDL is directly associated with the lipolysis of chylomicrons. It is believed that during the hydrolysis of chylomicrons discarded HDL particles of lipid bilayer membrane associated with apolipoprotein A-I are formed (Assman, 1982).

**Lipid Profiles**

When a physician requests a blood cholesterol level, it is actually the total amount of HDL, LDL, and VLDL that is measured. One value, a sum of the fractions, is reported. It is only when the physician requests a lipid profile that a fractionated result of the lipids and a triglyceride level is reported. The fractionated report renders information valuable for the prediction of
coronary artery disease. High levels of LDL are often present in persons with early heart attacks, whereas high levels of HDL are associated with a decreased risk of coronary heart disease (Assman, 1982; Grundy, 1986; Tzagournis, 1987).

The relationship between VLDLs, which contain large amounts of triglycerides, and coronary heart disease is not as well defined as the link between cholesterol and coronary heart disease. There is a positive correlation between high blood triglyceride levels and persons with coronary heart disease. The level of triglycerides in the blood has an inverse relationship with HDL which only consists of cholesterol. Individuals with the highest concentration of triglycerides have the lowest concentration of HDL. Therefore, individuals with HDL have more protection against coronary heart disease. It is believed that HDL transports cholesterol out of all tissues, including the arterial wall. The cholesterol is then carried to the liver and excreted into the bile.

The relationship between VLDLs, which contain large amounts of plasma triglycerides, and coronary heart disease has been a subject of continuing debate. Many epidemiologic studies have shown a positive correlation between plasma triglyceride concentrations and rates of coronary heart disease (Grundy & Vega, 1988). This
observation has led some authorities to claim that elevated triglyceride concentrations are a risk factor for a coronary heart disease. Others believe that it is not the high levels of triglycerides that cause the risk but, rather, the low levels of HDL, that are associated with them (Grundy & Vega, 1988). Although this view may be analytically correct, it fails to take into account the possibility that abnormalities in triglyceride metabolism and the metabolism of each of the lipoprotein species is worthy of consideration. High triglyceride levels recently have been implicated in epidemiologic studies as a risk factor for coronary heart disease (Grundy & Vega, 1988).

If similar phenomena occur in vivo, these abnormal cellular interactions could represent basic cellular mechanisms to explain the association of elevated plasma triglycerides with premature atherosclerosis. Endothelial injury and the accumulation of lipid filled foam cells in the arterial intima are thought to be involved in the initiation of atherosclerosis. Endothelial injury exposes the intima. The blood components such as lipoproteins, platelets, and coagulation factors are involved in the initiation of plaque. Another initiating event is the accumulation of foam cells in the intima of the arterial wall. Arterial foam cells are primarily mono-
cyte-derived macrophages that are engaged with cholesterol esters.

Hypertriglyceridemia

According to a study published in seminars on thrombosis and hemostasis, hypertriglyceridemic VLDL but not normal VLDL are toxic to cultured bovine aortic endothelial cells (Bradley & Gianturco, 1988). After 48 hours of exposure to low levels of hypertriglyceridemic VLDL, the number of viable cells was reduced by approximately 60%. The toxicity persisted for 72 hours in culture without refeeding the cells. The same concentration of normal VLDL or LDL had no significant effect on viability of the endothelial cells. The levels of hypertriglyceridemic VLDL found injurious to the cells were those that are present in normal humans in the fasting state, far lower than the levels present in hypertriglycericemic subjects; therefore, the injury to their endothelial cells may be greater than those with normal triglyceride levels. It is conceivable that circulation levels of VLDL in hypertriglyceridemic subjects could perturb the endothelium in vivo (Bradley & Gianturco, 1988).

The foam cells that are thought to be involved in the initiation of atherosclerosis are formed into
abnormal triglyceride rich lipoprotein. Macrophages develop large numbers of visible lipid inclusions when incubated with human chylomicrons or larger VLDL from certain hypertriglyceridemic subjects (Bradley & Gianturco, 1988).

Similar lipid inclusions are produced when the cells are incubated with cholesteryl ester rich VLDL from cholestrol fed animals. By contrast normal VLDL and native LDL from normal or hyperlipidemic subjects fail to include appreciable lipid accumulation in macrophage even during prolonged incubation. Uptake of hypertriglyceride rich VLDL and chylomicrons by macrophages is mediated by cell surface receptors. The lipid that accumulates in the macrophyage receptor initially reflects the lipid composition of the lipoprotein. Macrophages incubated in vitro in the absence of lipoproteins contain only a small amount of triglyceride and cholesterol, with virtually no cholesterol ester. Macrophages incubated with normal LDL or with normal VLDL do not accumulate appreciable lipid. The only human lipid accumulation in macrophagis in vitro are VLDL and chylomicrons from hypertriglyceridemic subjects (Assman, 1982).

Certain observations made in human subjects with hypertriglyceridemia suggest that these potentially atherogermic events observed in vitro may occur in vivo.
In contrast to VLDL catabolism in normal persons, the catabolism of VLDL in subjects with hypertriglyceridemia is abnormal. Most of the VLDL in hypertriglyceridemic subjects disappears directly from the plasma without being converted to VLDL, IDL, or LDL. This direct disappearance appears to be due to the removal of hypertriglyceride VLDL by receptors (Assman, 1982).

Reports from the Framingham study, which has followed subjects since the late 1940s, indicated that the higher the VLDL, or triglyceride level, the higher the subsequent coronary heart disease rate (cited in Kannel, 1987). The study associated elevated triglyceride levels with a higher risk of coronary heart disease. This relationship was much stronger in women than in men. Triglycerides were a powerful predictor of coronary heart disease in women over the age of 50 years. Using multivariate analysis, triglycerides were a statistically significant risk factor for women (Kannel, 1987). A multi-variate analysis, which is a mathematical comparison of the significant risk factors, was performed to determine the likelihood of triglycerides as a significant risk factor of coronary heart disease. Triglyceride levels were second only to HDL and various combinations of lipid measures and were better than LDL in predicting subsequent coronary heart disease. More
recent analysis, which used a projected shape analysis in men, indicated that triglycerides play an independent role in coronary heart disease risk in men over the age of 50 years, and that this role has been masked previously by the association of triglycerides with other risk factors that are themselves related to coronary heart disease (Castelli, 1986). Also, information from the Framingham study revealed that in both men and women triglyceride has little impact on the risk of heart disease in people who have average or high HDL (Kannel, 1987). An increase in coronary heart disease is seen only when the HDL concentration was below 40 mg/dl. The study also revealed a new syndrome, characterized by a high triglyceride level, a normal cholesterol level, and a low HDL (Kannel & Levy, 1988). People who were overweight tended to develop diabetes mellitus at twice the rate of the general population, and had elevated serum uric acid levels.

Atherosclerosis and Diabetes

Atherosclerosis is the most frequent complication of diabetics (Steiner, 1986). Most diabetics today are hyperinsulinemic because the amount of injected insulin surpasses physiologic requirements or because the pancreatic B cells are secreting insulin in an attempt to
compensate for insulin resistance. Examination of subfractions of triglyceride rich lipoprotein in diabetic patients supports the possibility that subjects have increased LDL levels. This finding is consistent with some pathophysiologic evidence that indicates that hyperlipidemia is associated with hyperinsulinemia (Steiner, 1986). Hyperinsulinemia is a reaction of the pancreatic B cells to attempt to compensate for insulin resistance. The resistance may be caused by mild obesity that is frequently associated with hypertriglyceridemia.

The process of hyperinsulinemia and increased triglyceride production may be a cycle (Steiner, 1986). Hyperinsulinemia may cause hypertriglyceridemia which may in turn lead to insulin resistance, for which the pancreas then attempts to compensate, resulting in further hyperinsulinemia. Increased VLDL turnover results in increased production of the catabolic remnants of VLDL on LDL fractions which can be atherogenic.

The atheroschertotic process for diabetic patients is a continual cycle with hyperinsulinemia. This group represents a population that may need to be studied separately from the normal healthy popoulation (Steiner, 1986).
EXPERIMENTAL PROCEDURES

The purpose of this investigation was to determine the relationship between blood total cholesterol levels and blood triglyceride levels in men and women 20 to 80 years of age. The experimental procedures were divided into three main areas; (1) subjects, (2) instrumentation, and (3) statistical analysis.

Subjects

The subjects were males and females 20 to 80 years of age. All subjects were required to fast for 12 hours prior to having their blood drawn. The subjects were not hospital patients. The laboratory blood test may have been part of a prehospital admittance testing procedure. Some of the subjects were assessed as diabetic because of their elevated fasting blood glucoses that accompanied the cholesterol and triglyceride reports. The subjects included in the study were tested for total blood cholesterol, blood triglyceride levels, and HDL levels from August 1988 to November 1988. Subjects were included only if a fasting state was declared. A total
of 1,808 subjects comprised the sample.

Dr. Louis Galup, M.D., pathologist and President of the South Bend Medical Foundation, South Bend, Indiana, gave consent for the use of the data for this study. Bette Gae Dart, Manager of the Automated Chemistry Department of the South Bend Medical Foundation, South Bend, Indiana, gave written consent for the use of data for the study (see Appendix B).

Instrumentation

The triglyceride measurement was performed using the Technicon Sequential Multi-channel Analyzer Computerized (SMAC) System from the Automated Chemistry Department of the South Bend Medical Foundation, South Bend, Indiana. The SMAC is a high speed computer-controlled biochemical analyzer. Preparation for analysis began with the patients' triglyceride levels being tested with an enzyme lipase. Two additional reactions were required to produce a pyruvate product which was then analyzed using the NADA-NAD Nicotinamide adenine reaction catalyzed by LDH (lactate dehydrogenase). The reaction steps were fast and easily controlled by the Technicon SMAC system.

The Automated Chemistry Department of the South Bend Medical Foundation also performed the testing of the cholesterol levels. The cholesterol levels were tested
on the Technicon SMAC. The Technicon SMAC Cholesterol method uses cholesterol esterase to hydrolyze the cholesterol esters in seven steps to free cholesterol. The free cholesterol is then oxidized to produce hydrogen peroxide that in turn is used to form a quinoneimine dye. Since the reaction takes place quantitatively, the concentration of the dye, measured colorimetrically, is directly proportional to the cholesterol content in the serum sample.

The sensitivity of the triglyceride methods is 0.000043 absorbence unit per unit of concentration (mg/dl). The sensitivity of the total cholesterol method is 0.00103 absorbence unit per unit of concentration (mg/dl). The accuracy of these methods was determined by a correlation with a previous Technicon method with a regression value of 0.9999.

The HDL values were measured on the Hitachi 717 from The Automated Chemistry Department of the South Bend Medical Foundation. Quantitation of cholesterol bound to the high density lipoprotein fraction was measured after separation of the HDL from LDL and VLDL fractions in serum. LDL, VLDL and the cholesterol associated with these lipoproteins are precipitated by dextran sulfate and magnesium chloride. The precipitate was removed by centrifugation and the HDL cholesterol in the supernate
is measured by an enzymatic cholesterol method. A comparison of this method with the manual procedure using cholesterol standards on an SMA 12/60 resulted in a linear regression equation of $y = 0.991x - 0.70$ and a correlation coefficient of 0.997. The accuracy of this method was also determined by the South Bend Medical Foundation to have a correlation of near 0.999 with other methods.

Statistical Analysis

The Computer Program used was SPSSX (SPSSX Inc., 1988) with subprograms, CONDESCRIPTIVE, CORRELATIONS and ANOVA Western Michigan University, 1990). Pearson Product Moment Correlations were calculated for all subjects, male subjects and female subjects. For each subject, correlations were calculated between the variables cholesterol, triglyceride, HDL, and age. The subjects were divided into six age groups. The six age groups were as follows: Group One included subjects 20 to 29 years of age, Group Two included subject 30 to 39 years of age, Group Three included subjects 40 to 49 years of age, Group Four included subjects 50 to 59 years of age, Group Five included subjects 60 to 69 years of age, and Group Six included subjects 70 to 80 years of age.
Three two way ANOVA's were run for gender and age group as the independent variables and triglycerides, cholesterol and HDL as the dependent variables. A $p < .001$ was used.
CHAPTER IV

RESULTS AND DISCUSSION

The purpose of this investigation was to determine the relationships among blood total cholesterol levels, HDL levels and blood triglyceride levels in men and women 20 to 80 years of age.

Results

Subjects

The subjects were 800 males and 1008 females 20 to 80 years of age. The mean age of the subjects was 56.6 years. All subjects fasted for 12 hours prior to drawing their blood samples. The subjects were not hospital patients, although the laboratory test may have been part of a prehospital admittance testing procedure. Some of the subjects were assessed as diabetic because of their elevated fasting blood glucose that accompanied the cholesterol and triglyceride reports. The subjects included in the study were tested for SMAC and HDL levels from August 1988 to November of 1988. Subjects were included in the sample only if a fasting state was declared. A fasting state consisted of no food consumption 12 hours prior to testing. The mean cholesterol for
The mean cholesterol level for all subjects was 227.7 mg/dl and the standard deviation was 44.1 mg/dl. The mean triglyceride level was 167.8 mg/dl and the standard deviation was 112.7 mg/dl. The mean HDL level was 49.0 mg/dl and the standard deviation was 13.6 mg/dl. Refer to Table 1 for a summary of the descriptive statistics.

Correlation

The computer program used to analyze the relationship between variables was SPSSX (SPSSX Inc., 1988) with CORRELATION. The formula used was the Pearson Product Moment Correlation. Correlations were calculated for all subjects, male and female. For each subject correlations were calculated among variables cholesterol, triglycerides, HDL and age.

Table 1

Mean and Standard Deviation for Age, Cholesterol, Triglyceride and HDL

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>56.6 years</td>
<td>13.1</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>227.7 mg/dl</td>
<td>44.1</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>167.8 mg/dl</td>
<td>112.7</td>
</tr>
<tr>
<td>HDL</td>
<td>49.0 mg/dl</td>
<td>13.6</td>
</tr>
</tbody>
</table>
All Subjects

The correlation between cholesterol and triglycerides for all subjects was significant ($r = .34$, $p < .001$). The correlation between cholesterol and age for all subjects was significant ($r = .18$, $p < .001$). The correlation between cholesterol and HDL was significant ($r = .11$, $p < .001$). The correlation for all subjects between age and triglyceride was not significant ($r = .05$, $p < .001$). The correlation between age and HDL was not significant ($r = .05$, $p < .001$). The correlation between triglyceride and HDL was significant ($r = -.40$, $p < .001$). Table 2 presents a correlation matrix for age, cholesterol, triglyceride and HDL for all subjects.

Table 2
Pearson Product Moment Correlation Coefficients for All Subjects

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Cholesterol</th>
<th>Triglyceride</th>
<th>HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.0</td>
<td>0.18*</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>0.18*</td>
<td>1.0</td>
<td>0.34*</td>
<td>0.11*</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>0.05</td>
<td>0.34*</td>
<td>1.0</td>
<td>-0.40*</td>
</tr>
<tr>
<td>HDL</td>
<td>0.05</td>
<td>0.11*</td>
<td>-0.40*</td>
<td>1.0</td>
</tr>
</tbody>
</table>

* $p < .001$
**Male Subjects**

The relationships of age, triglyceride, cholesterol, and HDL were compared for male subjects. The correlation between cholesterol and age was not significant ($r = -0.01, p < .001$). The correlation between cholesterol and triglyceride was significant ($r = 0.34, p < .001$). The correlation between cholesterol and HDL was not significant ($r = 0.06, p < .001$). The correlation between age and triglyceride was not significant ($r = -0.03, p < .001$). The correlation between age and HDL was not significant ($r = 0.05, p < .001$). The correlation between triglyceride and HDL was significant ($r = -0.39, p < .001$). Table 3 illustrates the correlation matrix for age, cholesterol, triglyceride and HDL levels for males.

**Table 3**

*Pearson Product Moment Correlation Coefficients for Males*

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Cholesterol</th>
<th>Triglyceride</th>
<th>HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.0</td>
<td>-0.01</td>
<td>-0.03</td>
<td>0.05</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>-0.01</td>
<td>1.0</td>
<td>0.34*</td>
<td>0.06</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>-0.03</td>
<td>0.34*</td>
<td>1.0</td>
<td>0.39*</td>
</tr>
<tr>
<td>HDL</td>
<td>0.05</td>
<td>0.06</td>
<td>-0.39*</td>
<td>1.0</td>
</tr>
</tbody>
</table>

* $p < .001$
Female Subjects

The relationships among age, triglyceride, cholesterol and HDL were compared for female subjects. The correlation between cholesterol and age was significant ($r = .31, p < .001$). The correlation between age and triglyceride was significant ($r = .16, p < .001$). The correlation between age and HDL was not significant ($r = .01, p < .001$). The correlation between cholesterol and triglyceride was significant ($r = .38, p < .001$). The correlation between cholesterol and HDL was not significant ($r = .07, p < .001$). The correlation between triglyceride and HDL was significant ($r = - .40, p < .001$). A correlation matrix for age, cholesterol, triglyceride and HDL levels for female subjects is presented in Table 4.

ANOVA

The computer program used to compare dependent variables with selected independent variables was SPSSX using the subprogram ANOVA. The dependent variables for each of the three ANOVA's were cholesterol, triglyceride, and HDL. The independent variables used in each of the three ANOVA's were gender and age groups. The six age groups were as follows: Group 1 included subjects' 20
Table 4
Pearson Product Correlation Moment Correlation Coefficients for Females

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Cholesterol</th>
<th>Triglyceride</th>
<th>HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.00</td>
<td>0.31*</td>
<td>0.16</td>
<td>-0.01</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>0.31*</td>
<td>1.00</td>
<td>0.38</td>
<td>0.07</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>0.16*</td>
<td>0.38</td>
<td>1.00</td>
<td>-0.40*</td>
</tr>
<tr>
<td>HDL</td>
<td>-0.01</td>
<td>0.07</td>
<td>-0.40*</td>
<td>1.00</td>
</tr>
</tbody>
</table>

* p < .001

to 29 years of age, Group 2 included subjects 30 to 39 years of age, Group 3 included subjects 40 to 49 years of age, Group 4 included subjects 50 to 59 years of age, Group 5 included subjects 60 to 69 years of age, and Group 6 included subjects 70 to 80 years of age. The alpha level was set at .01 for all ANOVA's.

Cholesterol

The mean cholesterol values for female subjects, Groups 1 through 6, were, 205, 205, 210, 239, 242 and 246 mg/dl, respectively. The mean cholesterol value for male subjects, Groups 1 through 6, were, 197, 221, 226, 219, 222 and 217 mg/dl, respectively. The ANOVA indicated a significant difference between age groups, F (5,1804) =
12.88, $p < .01$. For females across age groups, the mean value, $M=233$ mg/dl, was higher than the reference range, $< 180$ mg/dl, for cholesterol. For males across age groups, the mean value, $M=221$ mg/dl, was also higher than the reference range, $<180$ mg/dl (South Bend Medical Foundation, 1987). The analysis of variance indicated a significant difference between genders, $F (1,1804) = 31.81, p < .01$. The interaction effect, age groups by gender, was also significant, $F (5,1804) = 17.81, p < .01$. Refer to Table 5 for descriptive statistics. See Table 6 for an ANOVA summary.

Table 5

<table>
<thead>
<tr>
<th>Group</th>
<th>Female mg/dl</th>
<th>Male mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>205</td>
<td>198</td>
</tr>
<tr>
<td>2</td>
<td>205</td>
<td>222</td>
</tr>
<tr>
<td>3</td>
<td>210</td>
<td>227</td>
</tr>
<tr>
<td>4</td>
<td>239</td>
<td>220</td>
</tr>
<tr>
<td>5</td>
<td>242</td>
<td>222</td>
</tr>
<tr>
<td>6</td>
<td>246</td>
<td>217</td>
</tr>
<tr>
<td>All Groups</td>
<td>233</td>
<td>221</td>
</tr>
</tbody>
</table>
Table 6
ANOVA Summary Table for Cholesterol

<table>
<thead>
<tr>
<th>Source</th>
<th>ss</th>
<th>df</th>
<th>ms</th>
<th>f</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main Effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age Groups(A)</td>
<td>114505.3</td>
<td>5</td>
<td>22901.1</td>
<td>12.88</td>
</tr>
<tr>
<td>Gender(G)</td>
<td>56583.0</td>
<td>1</td>
<td>56583.0</td>
<td>31.81</td>
</tr>
<tr>
<td>Interaction</td>
<td>131748.8</td>
<td>5</td>
<td>26349.8</td>
<td>14.81</td>
</tr>
<tr>
<td>(AxG)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Residual</td>
<td>3208923.2</td>
<td>1804</td>
<td>1778.8</td>
<td></td>
</tr>
</tbody>
</table>

p < .01
* F (5,1804) = 3.32
** F (1,1804) = 6.63

Triglyceride

The mean triglyceride values for female subjects, Groups 1 through 6, were 173, 106, 133, 163, 169 and 175 mg/dl, respectively. The mean triglyceride values for male subjects, Groups 1 through 6, were 148, 199, 172, 192, 174 and 169 mg/dl, respectively. The ANOVA did not indicate a significant difference between age groups and triglyceride levels, $F (5,1803) = 2.73 \ p < .01$. For females, the mean triglyceride value, 158 mg/dl, was higher than the reference range < 150 mg/dl. For males, the mean triglyceride value, 180 mg/dl, was also higher.
than the reference range, < 150 mg/dl (South Bend Medical Foundation, 1987). The analysis of variance indicated a significant difference among gender and triglyceride levels, $F,(1,1803) = 18.71, p < .01$. The interaction effect age groups by gender was significant, $F (5,1803) = 6.13, p < .01$. Refer to Table 7 for the descriptive statistics for triglyceride levels for age groups and gender. See Table 8 for a summary of the ANOVA.

Table 7

<table>
<thead>
<tr>
<th>Group</th>
<th>Female (mg/dl)</th>
<th>Male (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>173</td>
<td>148</td>
</tr>
<tr>
<td>2</td>
<td>106</td>
<td>199</td>
</tr>
<tr>
<td>3</td>
<td>133</td>
<td>172</td>
</tr>
<tr>
<td>4</td>
<td>163</td>
<td>192</td>
</tr>
<tr>
<td>5</td>
<td>169</td>
<td>174</td>
</tr>
<tr>
<td>6</td>
<td>175</td>
<td>169</td>
</tr>
<tr>
<td>All Groups</td>
<td>158</td>
<td>180</td>
</tr>
</tbody>
</table>

HDL

The mean HDL values for female subjects, Age Groups 1 through 6 were 50, 52, 53, 55, 54, and 51 mg/dl, respect-
Table 8
ANOVA Summary Table for Triglyceride

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age Group (A)</td>
<td>168105</td>
<td>5</td>
<td>33621.0</td>
<td>2.73*</td>
</tr>
<tr>
<td>Gender (G)</td>
<td>230843</td>
<td>1</td>
<td>230843.0</td>
<td>18.71**</td>
</tr>
<tr>
<td>Interaction (AxG)</td>
<td>378048</td>
<td>5</td>
<td>74609.6</td>
<td>6.13*</td>
</tr>
<tr>
<td>Residual</td>
<td>2224570</td>
<td>1803</td>
<td>12338.2</td>
<td></td>
</tr>
</tbody>
</table>

\( p < .01 \)
* \( F(5,1803) = 3.32 \)
** \( F(1,1803) = 6.63 \)

Groups One through Six were 41, 43, 42, 44, and 45 mg/dl, respectively. A significant difference was found between the means of age groups HDL levels, \( F(5,1803) = 49.7, p < .01 \). For females, the mean HDL level, 53 mg/dl, was greater than the reference range, > 45 mg/dl. For males, the mean HDL level, 43 mg/dl, was lower than the reference range > 45 mg/dl (South Bend Medical Foundation, 1987).

The analysis of variance showed no significant difference between the HDL levels of males and females, \( F(1,1803) = 1.63, p < .01 \). Also, the interaction effect

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Table 9

HDL Means for Gender and Age Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Female (mg/dl)</th>
<th>Male (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>41</td>
</tr>
<tr>
<td>2</td>
<td>52</td>
<td>43</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>44</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>42</td>
</tr>
<tr>
<td>5</td>
<td>54</td>
<td>44</td>
</tr>
<tr>
<td>6</td>
<td>51</td>
<td>45</td>
</tr>
<tr>
<td>All Groups</td>
<td>53</td>
<td>43</td>
</tr>
</tbody>
</table>

of age groups by gender for HDL was not significant, $F(5,1803) = 2.74, p < .01$. Refer to Table 9 for descriptive statistics for HDL. See Table 10 for a summary of the ANOVA results for HDL.

Discussion

The mean value for cholesterol was 227.7 mg/dl which is higher than the reference range of <200 mg/dl stated by the South Bend Medical Foundation. The triglyceride mean value of 167.8 mg/dl was also higher than the reference range of 0-150 mg/dl stated by the South Bend Medical Foundation (1987, p. 192). The mean HDL value
Table 10
ANOVA Summary Table for HDL

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>DF</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Groups (A)</td>
<td>46807.3</td>
<td>5</td>
<td>7801.0</td>
<td>49.69*</td>
</tr>
<tr>
<td>Gender (G)</td>
<td>1281.9</td>
<td>1</td>
<td>256.4</td>
<td>1.63</td>
</tr>
<tr>
<td>Interaction (AxG)</td>
<td>2464.6</td>
<td>5</td>
<td>429.9</td>
<td>2.74</td>
</tr>
<tr>
<td>Residual</td>
<td>283048.0</td>
<td>1803</td>
<td>157.0</td>
<td></td>
</tr>
</tbody>
</table>

* $F (5,1803) = 3.32$

was 49.0 mg/dl which is higher than the 45 mg/dl indicating a risk factor for coronary heart disease. These values correspond with the American Heart Association's prediction for the need to lower cholesterol and triglyceride levels for adults (Parke Davis, 1986).

The relationships among cholesterol, triglyceride, HDL, and age were compared for all subjects. Age was not related to cholesterol, triglycerides, and HDL. Cholesterol was not related to HDL levels. The relationship between cholesterol and triglyceride was significant but low coefficient $r = .34$. Because of the low correlation between cholesterol and triglyceride, $r = .10$ and the lack of correlation between cholesterol and HDL, $r = .11$,
cholesterol should not be used to relate other lipid fractions. This follows the American Heart Association's recommendation to measure not only cholesterol, but also measure triglyceride and possibly LDL and HDL to determine the risk of developing coronary heart disease (Parke Davis, 1986).

The relationships among cholesterol, triglyceride, HDL and age were compared for male subjects. The correlations for male subjects agreed with the correlations for all subjects. Cholesterol should not be used to relate to other lipid fractions.

The relationships among cholesterol, triglyceride, HDL, and age were compared for female subjects. Age was related to cholesterol and triglyceride. The correlations for female subjects agreed with the correlations for all subjects. Cholesterol should not be used to relate other lipid fractions.

The ANOVA design for cholesterol with age groups and gender as independent variables demonstrated a significant difference between age groups and cholesterol means. There was also an interaction effect between age groups and gender. The mean cholesterol values increased with age for females after 40 years of age. The mean cholesterol values for females greatly increased after 50 years of age which may be due to the onset of menopause.
The mean cholesterol values for men did not vary significantly between 30 and 80 years of age. See Figure 1 for an illustration of the interaction effect of age groups and gender.

The ANOVA design for triglyceride with age groups and gender as independent variable demonstrated a significant difference between the genders' triglyceride levels. There was an interaction effect between age groups and gender for triglyceride levels. Triglyceride levels cannot be a predictor by age or gender. The mean triglyceride value for females was significantly lower at 30 to 39 years of age than the mean triglyceride value of females 20 to 29 years of age. The lower value may be due to better eating habits of older females. The mean

![Graph image](https://via.placeholder.com/150)

**Figure 1. Interaction Between Age Groups and Gender for Cholesterol.**
values of triglycerides increased at every age group after 39 years of age. The mean triglyceride values for males were significantly higher at 40 to 49 years of age than at 30 to 39 years of age. See Figure 2 for an illustration of the effect of age groups and gender.

The ANOVA design for HDL with the independent variables, age groups and gender, showed a significant difference between HDL levels for males and females. The female subjects at every age group had higher HDL levels which corresponds to a lower risk factor for coronary heart disease (Assman, 1982). The interaction effect of age groups by gender for HDL was not significant. The mean of age groups and gender. See Figure 3 for an illustration of the effect of age groups and gender for HDL.

![Graph showing interaction between age groups and gender for triglyceride levels.](image-url)

**Figure 2. Interaction Between Age Groups and Gender for Triglyceride.**
Due to the low correlation coefficient found between cholesterol and HDL, and cholesterol and triglycerides, it becomes apparent that cholesterol should not be used as a predictor for other lipid fractions. Low coefficients were found in all correlations evaluated: all subjects, male subjects and female subjects. Thus, all correlations support this conclusion.

Age cannot be used as a predictor for lipid fractions. The relationships among age, triglyceride, cholesterol and HDL were not significant for all subjects and male subjects. Therefore, gender cannot be used as a predictor for triglycerides and HDL.

The ANOVA designs showed an interaction effect for cholesterol, age groups and gender. Each lipid fraction

![Graph showing interaction between age groups and gender for HDL.](image)

Figure 3. Interaction Between Age Groups and Gender for HDL.
must be measured to determine the patient's risk factor for coronary heart disease. Cholesterol testing alone cannot predict the values for the other lipid fractions.
CHAPTER V

SUMMARY, FINDINGS, and CONCLUSIONS

The purpose of this investigation was to determine the relationship between blood total cholesterol levels and triglyceride levels in men and women 20 to 80 years of age. Chapter V is divided into four sections: (1) summary, (2) findings, (3) conclusions, and (4) recommendations.

Summary

It was hypothesized that blood total cholesterol and blood triglycerides would have a low relationship. The cholesterol levels were tested by the South Bend Medical Foundation, South Bend, Indiana on the Technicon SMAC. The triglyceride levels were also tested on the Technicon SMAC. The HDL levels were measured on the Hitachi 717 by the Automated Chemistry Department of the South Bend Medical Foundation.

Subjects were males and females 20 to 80 years of age. All subjects fasted for 12 hours prior to drawing their blood samples. The subjects included in the study were tested for total cholesterol, triglyceride and HDL.
levels. The testing was performed from August to November 1988.

The computer program used to calculate the relationship between variables was SPSSX (SPSSX, Inc., 1988) with subprogram CORRELATIONS. The computer program used to compare dependent variables with selected independent variables was SPSSX using subprogram ANOVA. The means and standard deviations were also calculated for age, cholesterol, triglyceride, HDL, and gender.

Findings

The study produced the following findings:

1. The relationship between cholesterol and age for all subjects was significant ($r = .18$, $p < .001$).

2. The relationship between cholesterol and HDL for all subjects was significant ($r = .11$, $p < .001$).

3. The relationship between triglyceride and HDL for all subjects was significant ($r = -.40$, $p < .001$).

4. The relationship between cholesterol and triglyceride for all subjects was significant ($r = .34$, $p < .001$).

5. The relationship between cholesterol and triglyceride for males was significant ($r = -.34$, $p < .001$).

6. The relationship between triglyceride and HDL for males was significant ($r = -.39$, $p < .001$).
7. The relationship between cholesterol and age for females was significant ($r = .31, p < .001$).

8. The relationship between age and triglyceride for females was significant ($r = .16, p < .001$).

9. The relationship between cholesterol and triglycerides for females was significant ($r = .38, p < .001$).

10. The relationship between triglyceride and HDL for females was significant ($r = -.40, p < .001$).

11. The analysis of variance for cholesterol indicated a significant difference between genders, $F(1, 1804) = 31.81, p < .01$.

12. The analysis of variance for cholesterol indicated a significant difference between age groups, $F(5, 1805) = 12.88, p < .01$.

13. The analysis of variance for demonstrated a significant interaction effect, age groups by gender, $F(5, 1804) = 14.81, p < .01$.

14. The analysis of variance for triglyceride indicated a significant difference between genders, $F(1, 1803) = 18.71, p < .01$.

15. The analysis of variance for triglycerides did not indicate a significant difference for age groups, $F(5, 1803) = 2.73 p < .01$.

16. The analysis of variance for triglycerides indicated an interaction effect, age groups by gender, $F$
(5, 1803) = 6.13, \( p < .01 \).

17. The analysis of variance for HDL showed a significant difference between HDL levels of males and females, \( F(1, 1803) = 282.74, p < .01 \).

18. Total cholesterol levels increased with age for females.

19. HDL levels were higher in females.

20. The mean triglyceride value was higher in males.

21. Total cholesterol levels increased with age for females.

22. Total cholesterol levels did not increase with age for males.

Conclusions

The following conclusions were drawn based on the data analysis.

1. A low positive correlation coefficient was calculated between total cholesterol levels and triglyceride levels (\( r = .34, p < .001 \)).

2. A low positive correlation coefficient was calculated between total cholesterol levels and HDL levels (\( r = .11, p < .001 \)).

3. A negative correlation coefficient was calculated between triglyceride levels and HDL levels (\( r = -.40, p < .001 \)).
4. Cholesterol should not be used as a predictor for other lipid fractions.

5. Age cannot be used as a predictor for lipid fractions.

6. Gender cannot be used as a predictor for triglyceride levels.

7. Gender cannot be used as a predictor for HDL levels.

8. Cholesterol testing alone cannot predict the values for other lipid fractions.

Recommendations

Further investigation comparing total cholesterol levels, triglyceride levels, and HDL levels of coronary heart disease patients to noncoronary diseased people is needed. Other variables of interest in future research on blood lipids could include age, gender, and their relationship to other types of diseased populations such as diabetics.
Appendix A

Approval Letter: Human Subjects
Institutional Review Board
TO: Rebecca Armstead
FROM: Ellen Page-Robin, Chair
RE: Research Protocol
DATE: December 3, 1988

This letter will serve as confirmation that your research protocol, "Total Cholesterol and Triglyceride Correlation" has been approved as exempt by the HSIRB.

If you have any further questions, please call me at 387-2547.
Appendix B

Data Approval Letter:
South Bend Medical Foundation
January 10, 1991

To Whom It May Concern:

Becky Bablar had my consent and support to utilize HDL Cholesterol data generated by Automated Chemistry from August, 1988 through November, 1988.

Bette Gae Dart  MT(ASCP)
Manager, Automated Chemistry
BIBLIOGRAPHY


