A COMPARISON OF BLACK AND WHITE MALE FACULTY MEMBERS' SELECTED CARDIOVASCULAR RISK FACTORS

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CHAPTER I

INTRODUCTION

The leading cause of death among Americans is heart disease, as indicated by data (1979) released by the United States Public Health Service.[1] Death rates for specified causes by race and sex in the United States, established by the National Center for Health Statistics, indicated that the leading cause of death is major cardiovascular disease, diseases of the heart, and hypertension. According to this source, the non-white population suffered more from this condition than the white population.

Numerous cardiovascular risk factors are contributors to heart disease. The purpose of this study was to explore a selected number of these risk factors, especially those that are most prevalent in males. The subjects used in this study were black faculty members from Langston University and white faculty members from Oklahoma State University. A comparison of the cardiovascular risk factors from each group was made to determine if there was a high incidence of hypertension among black faculty members compared with white male faculty members and to find explanations about other risk factors that are contributors to cardiovascular diseases. These risk factors included smoking habits,

exercise habits, lipoproteins (triglycerides, HDL and LDL), and fat as determined by skinfold measurements.

Hypertension is a condition found more in males than in females with the black male population leading the list. Hypertension is better known as high blood pressure, which is an elevation of the pressure that blood exerts against the inner walls of the blood vessels. This condition with all of its complications may strike its victims without their knowledge of the seriousness of the problem because there may be few or no symptoms that are detectable.[2]

Studies have been conducted comparing males and females and the incidence of coronary heart disease as related to blood pressure or hypertension. In the Tecumseh study, 1457 men and 1607 women, age groups ranging from 20 years of age to past 55 were studied. The age groups were separated into young, middle, and old age categories. The results showed that blood pressure and serum cholesterol levels were consistently correlated with incidence of coronary heart disease.[3]

Heredity seems to be related to hypertension and to play an important role in the problems that are associated with this condition. If both parents have high blood pressure, their offspring stands a 50 percent chance of developing this condition. If only one parent has hypertension, there is a 25 percent chance that the off-springs may develop high blood pressure.[4] Hypertension is a health problem that may be attributed in part to heredity, but

there are some controversies over the so-called racially inherited genetic phenomena. It is hard to say or predict that hypertension in Blacks is transferred through the genetical makeup, but there is a tendency to label the disease as highly familial among black people in America.[5]

Obesity is another contributing risk factor of cardiovascular disease that is associated with all races, ages, and sexes of people. Any individual who is 30 percent overweight is considered as obese. Although there is very little information about the relationship of obesity and hypertension, health problems are numerous with those individuals who suffer from being just plain fat. When obesity is dealt with as the only variable, it appears to contribute very little to the coronary problems. As indicated by Ostrander and Lamphlear, "Obesity is relatively unimportant as an independent precursor of coronary event, and is often associated with other antecedent conditions."[6] There is little evidence as to the direct or major role obesity plays in the development of coronary heart disease, but its association has been too conspicuous to be ignored. The following quote supports this idea:

> There are various studies that have shown that excessive weight increases the risk of coronary disease. In the government's Framingham Study it was stated that, 'Men who were 30% overweight were 2.8 times more likely to develop coronary heart disease in ten years than men who were 10% or more overweight.' Particularly marked in the overweight group was the likelihood of developing angina pectoris.[7]

There are simple tests that individuals can use to assess their fatness. One such test is the skinfold fat test. This is where skinfold calipers are used to measure the skinfold thickness on various parts of the body, such as triceps, biceps, abdomen, and subscapular. The skinfold measurements suggested by Best include the triceps, abdomen, and the chest.[8] This method uses a nomogram to determine the specific gravity and percent body fat. By using the age, height, and observed body weight of men, the Consolazio method finds the relative body weight percent for individuals.[9] This method uses the nomogram to determine the specific gravity and percent body fat from the skinfold measurements of the chest, subscapular, and triceps. Underwater measurement is the most accurate method for determining total body weight. This method is

> . . . based on the density of the whole body which is the total body weight in air divided by the total body volume. The estimation of the latter is determined from its displacement of water.[10]

This method is not widely used for determining body fat because it is very time-consuming.

Blood lipids have been established as a factor when observing cardiovascular problems particularly in the stages of acute myocardial infarction. This condition is associated with ischemic heart disease, which develops as a consequence of inadequate performance of a portion of the myocardium. It is characterized by chest pains which are usually frequent and severe enough to be described as the worst pain a victim has ever experienced. It is a deep visceral pain

that is characterized by a heavy, squeezing, and crushing sensation. It is more severe than the pain of angia pectoris. It lasts longer and is usually relatively constant.[11] This condition occurs with a high frequency in individuals with high serum cholesterol.

Atherosclerosis contributes vastly to cardiovascular problems and receives a great deal of publicity in the news media as well as in the medical and scientific world. Pritikin indicated that "Atherosclerosis is a disease of the arteries which causes most kinds of heart disease and most kinds of stroke."[12]

Practicing physicians often have difficulty in deciding on the proper treatment for this cardiovascular problem. This difficulty is due to the controversies that exist in developing areas of scientific investigation. One such problem is pointed out by the Select Committee on Nutrition and Human Needs of the United States, which was chaired by Senator George McGovern. This Committee recommended that Americans could control their weight by reducing their fat consumption to 30 percent of calories, saturated fat to at least 10 percent, and cholesterol intake to about 300 mg per day.[13]

Pritikin advocated a theory in his book, based upon research by the Longevity Foundation of America, which indicated that individuals can live longer with proper diet and exercise. There are claims made in the book that patients may experience reverses in atherosclerosis by combining low

fat, no cholesterol diet, and exercise. This claim was based upon research by Armstrong and his associates with rhesus monkeys that had been fed a high fat and high cholesterol diet which generated a high degree of atherosclerosis in the animals over a period of time. Some of these animals were fed a very little fat and no cholesterol diet which was supplemented with unsaturated fat. It was found that the animals had their plaques reversed substantially, which indicated that a diet low in both fats and cholesterol can reverse atherosclerosis. This same experiment was done by Tucker and his associates who had the same results. Although this experiment has not been reproduced in humans, Pritikin claims that with all of the advanced knowledge about atherosclerosis, it can be reversed in humans by switching to a low fat and low cholesterol diet.[14] With every type of recommendation, some individuals will challenge the authenticity in changing dietary habits of the general public as a means of controlling or decreasing cardiovascular risk factors.[15]

Smoking contributes significantly to the development of coronary artery problems and associated death rates. There is some speculation that smoking also is a factor in excess levels of blood fat.[16] Smoking increases the heart rate, and it produces a temporary rise in blood pressure.[17] Recent studies have shown that smoking even low-nicotine filter cigarettes raises systolic blood pressure by eight points and produces a nine-beats-a-minute increase in heart

rate. Other studies have shown that cigarette smoking stimulates release of hormones from the adrenal gland which has powerful effects on the heart and circulation and influences the levels of fats circulating in the blood stream.[18] In another recent study, blood cells of smokers were found to have less affinity for oxygen. As much as 26 percent of the blood that is pushed around by the heart of the smoker is not working as far as carrying oxygen is concerned. Since the heart has the highest oxygen requirement per unit weight of any tissue, any change in the supply of oxygen could affect the heart first and thereby increase the risk of an attack for the smoker.[19]

Individuals who participate in exercise regularly may reduce their chances of acquiring cardiovascular problems. However, studies have shown that exercise alone cannot dictate the limitations of risk factors such as elevated cholesterol levels, hypertension, and smoking that are contributors to cardiovascular problems.[20]

Exercise is beneficial at all age levels, but for the middle aged and older persons, it is most profitable. There are reasons to believe that through regular exercise with proper intensities and duration, much can be done to postpone deterioration of the body. With all of the available data on cardiovascular disease, there are still some unanswered questions about the disease. It appears that more research should be conducted to answer some of the questions about this health problem.[21]

Statement of the Problem

The problem was to determine the incidence of cardiovascular risk factors in the black male faculty of Langston University and to compare this incidence with that of the white male faculty members from Oklahoma State University. These risk factors are hypertension, blood lipids, percent body fat, smoking, and exercise habits.

The black male faculty members were the entire black male faculty personnel at Langston University. The white male faculty members were volunteers from a group of male faculty members who have been participating in an annual physical fitness evaluation program at Oklahoma State Universty.

Hypotheses

1. There is no significant relationship between percent body fat and blood pressure in the black, white, and combined populations in this study.

2. There is no significant relationship between exercise habits and blood pressure in the black, white, and combined populations in this study.

3. There is no significant relationship between smoking habits and blood pressure in the black, white, and combined populations in this study.

4. There is no significant relationship between lipoproteins (total cholesterol HDL, LDL, and triglycerides) and blood pressure in the black, white, and combined

populations in this study.

5. There is no significant relationship between body fat and blood pressures in black, white, and the combined populations in this study.

6. There is no significant relationship between race and blood pressures.

7. There is no significant relationship between race and lipoproteins (total cholesterol HDL, LDL, and triglycerides).

8. There is no significant relationship between race and smoking habits.

9. There is no significant relationship between race and exercise habits.

10. There is no significant relationship between lipoproteins (total cholesterol HDL, LDL, and triglycerides and percent body fat.

11. There is no significant relationship between lipoproteins (total cholesterol, HDL, LDL, and triglycerides) and exercise habits.

12. There is no significant relationship between lipoproteins and smoking habits.

Limitation of Study

1. There was some apprehension of the black subjects due to the unfamiliarity of the testing equipment, even though the black subjects were oriented to the testing equipment.

2. Only a selected few risk factors related to heart disease were compared in this study.

3. The OSU faculty subjects were volunteers from a group of 93 faculty members who started in 1972 on an annual physical fitness evaluation program. This group started out with 20 non-exercisers. At the present time (1980), the number is down to 55 to 60 members with only 4 to 5 non-exercisers. This group cannot be recognized as a typical OSU male faculty member, but they are non-random volunteer participants in this study.

Assumptions

1. There was a wide range of physical conditioning represented in the subjects for both the black and white populations.

2. Blood pressures which were measured by Dr. F. Cooke at Langston University were valid and comparable to OSU Laboratory blood pressure measurements, and the instruments were properly calibrated.

3. The questionnaire form was valid in determining the smoking and exercise habits of the subjects.

4. Valid lipoprotein levels were produced through analysis at Stillwater Medical Center for both the black and white populations.

5. Percent body fat was validly predicted from skin-fold fat measures.

Significance of the Study

A sound body and a sound mind go hand in hand as tools for a healthy person. With awareness of this statement, the university faculty members should be concerned with their mental and physiological well-being. The male population is more susceptible to cardiovascular risk factors than his female counterpart. They may be suffering from the so-called "silent killer" without anyone being aware of the potential dangers. If persons who are working with young people can be alerted to this national health problem, then this study will be worthwhile.

The significance of the study is based partially upon the population from which the study is designed. With various data already available which have established a high rate of cardiovascular risk factors in the male population, there is a definite need for expanding research with diverse variables that are related to the cardiovascular problems. Specifically, can it be found that cardiovascular risk factors are more prevalent in black male faculty members than in white male faculty members with the population studied? Also, this study attempted to determine if cardiovascular risk factors are as prevalent in university faculty members as they are in the general population. The uniqueness of this study lies in the fact that few studies have been attempted with this population.

With hypertension being one of the major health problems among Blacks, and especially the male, it is imperative

the Blacks be concerned with this health risk. As a black male faculty member working in a predominantly black institution, it would seem that the stress factor is a significant variable which may contribute to hypertension. This stress my be caused primarily by two conditions. In the last ten years, the institution has been plagued with a continual change in administrators. This could be mainly due to inadequacies or political forces.

Langston University depends largely upon federal subsidies for most of its programs. About 90 percent of the students who attend the institution are on financial aid or some type of governmental grant. With the new conservative movement where cuts appear to be aimed at funding for education, many of these programs will be drastically reduced or deleted. Faculty and staff jobs are at stake. The financial aid picture is bleak. Therefore, the stress factor continues to be a pertinent possibility for the faculty at Langston University.

When comparing Langston University faculty and the OSU faculty, it would appear that the OSU faculty would not be as stressed due to the obvious stability of its administration and financial resources. According to Dr. Harrison's findings, OSU faculty personnel do not have as much hypertension as other professionals, such as businessmen, bankers, and lawyers. Therefore, this study's significance depends upon comparing these and other factors which have not been previously explored.

Definition of Terms

1. Hypertension: High blood pressure; an elevation of the pressure which the blood exerts against the sides of the blood vessels.

2. The Silent Killer: Hypertension; where many of the people who are suffering from the affliction do not know they have the disease.

3. Physiograph: A multi-channel ink-writing recorder used to monitor and record blood pressure.

4. Cardiovascular risk factors: Those factors related to heart attacks and other concerns of cardiac arrest.

5. Sphygomomanometer: An inflatable cuff with a gauge used to indirectly measure blood pressure.

6. Obesity: A condition in which 30 percent or more excess weight occurs.

7. Skinfold fat: Thickness of fat and skin as measured with a caliper used to predict body fat.

8. Lipoprotein: A conjugated protein that is a complex protein and lipid.

9. Myocardial infarction: A sudden irreversible ischemic injury due to coronary arterial narrowing or occlusion with sustained damage to a segment of the myocardium.

10. Cholesterol: A steroid alcohol which is present in body cells and body fluids; an important physiological process and implicated experimentally as a factor in atherosclerosis. The primary element in the fatty atherosclerotic deposits are found in the arteries.

11. High Density Lipoproteins (HDL): A plasma protein which appears to serve as a reservoir for small apoproteins involved in the transportation of triglycerides. It is thought of as a scavenger because it picks up excess cholesterol inside of the arteries and has the highest amount of protein of the lipoproteins and the lowest amount of cholesterol.

12. Very Low Denstiy Lipoproteins (VLDL): A plasma protein which carries most of the triglycerides.

13. Low Density Lipoproteins (LDL): A plasma protein, where the smaller the lipoprotein particle, the greater its protein content and density. It correlates the amount of cholesterol in the blood stream and after metabolism, it leaves cholesterol inside the blood vessels.

14. Atherosclerosis: An arterial disease, whereby the openings of the arteries are narrowed by the accumulation of fat in the arterial walls. The narrowing or blocking of the coronary arteries by these masses of fat or theromas, is by far the most common cause of coronary artery disease.

15. Familial Hypercholesterolemis (FH): The genetically classical determined hyperlipidemias. Most typical of this disorder are an association with a high incidence of premature arteriosclerosis.

16. Coronary Heart Disease (CHD): The blocking or obstruction of one or more of the arteries that supply oxygen to the heart muscles. The blocking or obstructions are caused by masses of fat or atheromas which is the most

common cause of coronary artery disease.

17. Systolic Blood Pressure: The pressure required to occlude the trachial artery.

18. Diastolic Blood Pressure: The pressure at which the sounds resulting from occlusion become muffled or disappear.

19. Percent Body Fat: The amount of body fat exceeds the amount that is considered to be desirable for the sex and weight of the individual.

20. Risks: One or more identifiable characteristics which causes death or disabling conditions in people who exhibit some form of atherosclerosis.

21. Aerobic Points: A system developed by Kenneth H. Cooper, M.D. Having determined that Aerobic exercise is most beneficial from the standpoint of cardio-vascular conditioning and having established a goal for fitness based on oxygen utilization, this is a system people could use to achieve these goals.

22. Triglycerides: Lipoproteins which contain protein components known as apoproteins.

ENDNOTES

[1]Time, The 1979 Hammond Almanac (New York: Hammond Almanac, Inc., 1979), p. 270.

[2]William Manger and Irvine Page, "Hypertension: The Silent Killer," Harper's Bazaar (October, 1977), pp. 94-202.

[3]Jack Slater, "Hypertension: Biggest Killer of Blacks," Ebony, Vol. 28 (June, 1973), pp. 74-82.

[4]Ibid.

[5]Ibid.

[6]Ibid., p. 76.

[7]William Likoff, Bernard Segal, and Lawrence Galton, Your Heart: Complete Information for the Family (Philadelphia: Lippincott Co., 1972), p. 176.

[8]Ibid.

[9]Herbert A. deVries, Physiology of Exercise for Physical Education and Athletes (Dubuque, Iowa: Wm. C. Brown, 1972), pp. 282-293.

[10]Richard H. Cox and Jack K. Nelson, "AAHPERD Research Consortium," Symposium Papers 1980 (Washington, D. C.: AAH Pero Publication, 1980), p. 76.

[11]Eugene Braunwald, "Disorders of the Heart," Principles of Internal Medicine (New York: McGraw-Hill Co., 1970), pp. 1080-1081.

[12]N. Pritikin, J. L. Hofer, and N. Leonard. Live Longer Now (New York: Grossett and Dunlap, A Filmways Company, 1974), p. 18.

[13]Antonio M. Gotto and Rodolfo Paoletti, Atherosclerosis Review (New York: Raven Press, 1979), pp. 1-80.

[14]Pritikin, Hofer, and Leonard, p. 19.

[15]Gotto and Paoletti, p. 60.

[16]D. H. Beece, ed., "Tobacco Consumption in Various Countries," Research Paper No. 6 (London, Tobacco Research Council, 1972).

[17]Ibid.

[18]Ibid.

[19]Ibid.

[20]Robert S. Levine, "Families and Hypertension," Primary Cardiology, Vol. 4, No. 6 (June, 1978), pp. 26-29.

[21]Ernst Jokl, "Nutrition, Exercise, and Body Composition." American Lectures in Sports Medicine (Springfield, Illinois: Charles C. Thomas, 1964), pp. 13-71.

CHAPTER II

REVIEW OF RELATED LITERATURE

The review of literature will be divided into several categories: (A) Heart Disease: Statistical Data; (B) Hypertension: Statistical Data, Effects and Complications, Contributing Factors, Lowering the Risk; (C) Atherosclerosis: High Density Lipoproteins, Lipid Metabolism and Dietary Source, Pathway of LDL Metabolism, High Density Lipoprotein-Ratio and Total Cholesterol; (D) Obesity: Body Fat as Related to Age and Sex, Exercise and Body Fat, Obesity a National Problem; (E) Smoking Habits: Diseases Associated with Cigarette Smoking, The Effects of Smoking; (F) Exercise Habits: Effect on Cardiovascular Problems, Coronary Heart Disease and Exercise, Summary.

Heart Disease

The literature that is available on heart disease indicates that it is most often referred to as the result from the narrowing of coronary arteries due to the hardening where fatty tissue builds up (atherosclerotic process). This process is where the coronary vessels go through a progressive reduction in their internal diameter. Whenever the supply of blood to the myocardium falls below the critical

level, symptoms and complications of coronary heart disease may appear.

When the coronary function is impeded, the blood supply is limited. As a result of this low blood supply, an individual who has this problem will experience frequent chest pains often associating them with indigestion.

The reduced pumping ability of the heart commonly leads to weakness and fatigability, or when severe, it produces blue jaundice where enough air is not getting to the blood (cyanosis, or it may lead to hypertension and fainting better known as syncope). Elevated intravascular pressure upstream to a failing ventricle often leads to breathing difficulties and an accumulation of fluids.[1]

Cardiac arrhythmias often develop suddenly, and in the resulting complaints - palpitations, dyspnea, suffocation, hypertension or syncope - generally occur abruptly and may disappear as rapidly as they develop.[2] Patients with cardiocirculatory disease may also be entirely asymptomatic, both at rest and during exertion, but cardiac arrhythmias may be present with an abnormal physical finding, such as heart murmurs, elevated systematic arterial pressure, or an abnormality of the electrocardiogram, "or of the cardiac silhouette on the chest roentgenogram."[3]

The literature on heart disease also indicates that a widespread fear of this disease in the United States is so deepseated that frequent development of systems of this organic disease can be observed in individuals with normal

cardiovascular systems. In order to get a clear picture of individuals who are suffering from the disease or those who show symptoms of the disease, suggestions have been made by the New York Heart Association which more clearly define heart symptoms.[4]

Statistical Data

In 1976, there were 1,909,440 American deaths from heart disease according to The 1979 Hammond Almanac. Death rates for specified causes, by race and sex in the United States in 1976 (rate per 100,000 population in specified group) from cardiovascular disease for both sexes was 454.0; for males, 490.0; for females, 419.7; for Whites both sexes, 470.4; white males, 507.0; white females, 435.6; for Blacks both sexes, 345.8; black males, 376.8; and black females, 317.8.[5]

Hypertension

"The Silent Killer," a phrase often used in describing hypertension, is really high blood pressure. This is the most common chronic health problem that is facing the entire world because it rarely produces any symptoms. If this disease is not checked or treated, it can cause death to the individual who suffers from it. Hypertension can be controlled with drugs prescribed by a physician if detected in time.

Hypertension is "an elevation of the pressure which

your blood exerts against the sides of your blood vessels."[6] If one can imagine a steady increase of water pressure against the inner walls of a fire hose, then one can imagine high blood pressure pounding against the inner walls of arteries. This pounding is so constant that the inner walls of these blood vessels thicken to protect themselves. Finally, some portion of the vessels or the walls may blow out like a tube in an automobile tire.

Because hypertension produces no symptoms, it can cause irreversible organ damages before it can be detected. Blood pressure at 140/90 or higher has also been called various names such as "The Silent Disease," "The Quiet Assassin," and a fooler of a disease. But for individuals with hypertension all of these various names for the disease add up to the same thing, a deceptive sense of perfect health. Dr. Cooper, Director of the National Heart and Lung Institute in Bethesda, Maryland, pointed out that an individual can feel fine, "but those who have hypertension are a part of the largest single public health problem in this country."[7]

When one's blood pressure rises and is allowed to remain above normal for a substantial period of time, an individual should be concerned. There is a risk factor that is involved with this condition, which ultimately will develop serious cardiovascular disorders, such as heart failure, hardening of the arteries, strokes, kidney damages, heart attacks, and a good chance of shortening the life of an individual. "This unrelenting pressure can also cause

permanent loss of vision and the senility of old age."[8]

The risk factor that is associated with this problem of hypertension is the greatest threat to black people in the form of strokes, with kidney disease close behind. "For black people, stroke is the number one complication resulting from hypertension," says Dr. Saunders, Chief Cardiologist at Provident Hospital in Baltimore, Maryland, "and in spite of the fact high blood pressure is common, we do not see an extremely high rate of hypertensive heart attacks causing deaths among Blacks."[9] Evidently there is a paradox that occurs that protects black people from heart attacks but makes them exceptionally vulnerable to strokes.

Statistical Data

According to Dr. Manger and Dr. Page, today's hypertension afflicts 35,000,000 Americans; another 25,000,000 are borderline cases.[10] It is the underlying cause of 1,500,000 million deaths each year and costs the nation 20,000,000,000 dollars annually in medical bills, lost wages, etc. It is responsible for 52,000,000 million working days of productivity lost to business each year, and it cripples or disables more than 1,500,000 million people each year.[11]

Not all of the cases of hypertension are alike. The causes can vary, and in 90 percent of patients, the condition must be labeled as primary or essential hypertension because the physician does not know why the pressure has risen.

Drs. Manger and Page further stated that heredity plays an important role in essential hypertension. If both parents have it, perhaps 50 percent of their children will develop it; if only one parent has it, it may afflict about one-fourth of their offspring. In the United States, essential hypertension occurs more frequently, more severely, and earlier in Blacks than in Whites. Thirty percent of adults who are black and 15 to 20 percent of white adults develop it. Men have it more often than women. Recent studies have shown that high blood pressure affects a significant number of children even at a very young age - and a higher percentage of black than white children.[12]

The 1979 Hammond Almanac showed death rates for specified causes by color and sex in the United States in 1976, hypertension was 2.0 for both sexes. The rate per 100,000 population in each specified group was males, 2.8; female, 2.9; white, both sexes, 2.6; white male, 2.5; white female, 2.6; non-white, both sexes, 4.6; male, 4.8; female, 4.4.[13]

Dr. Johnson, a heart specialist from Washington, D.C., was deeply involved in a study of hypertension when he died. As 5,000,000 or 6,000,000 other Blacks (no one knows the exact figure), he suffered, maybe because of his race, a greater severity of hypertension. According to studies made by Dr. Johnson, high blood pressure kills black males, age 25-45, at a rate 15.5 times more frequently than it does white males. For the black female in this same age group,

it kills 17 times more frequently than it does the white females. It is currently estimated that about 25 percent of all black Americans and 15 percent of all white Americans, age 18 years and above, are afflicted with hypertension.[14]

Effects and Complications of Hypertension

The sufferers of hypertension in the advanced stages may experience throbbing headaches, strokes, and pain. The pain is so severe and intense that it seems to the sufferer that it is pressing downward and so sharp that it feels like an incision is being made into the brain. With this kind of suffering, the vision can become blurred. Because hypertension produces no symptoms or warnings until it has caused irreversible damage to various organs of the body, many people are unaware they are so close to sudden death.[15]

Contributing Factors to Hypertension

Hypertension causes seem to vary from time to time, and in the majority of instances the cause is virtually unknown. There are phases of hypertension whose conditions are known as "essential or primary."[16] It was thought by some doctors that it was essential for blood pressure to be elevated in order to maintain a degree of adequate circulation. But this is not considered to be the case in today's medical world. Any elevation of the blood pressure is considered to be a health problem, or at least the beginning of one.

Hypertension is a health problem that may be attributed to heredity. Dr. Calloway, Director of Harlem's Hospital Stroke Clinic indicated that "hypertension seems to run in families."[17] If the parents have the disease, there is a good chance that their offspring will have it also. There are some controversies over the so-called racially inherited genetical phenomena. It is hard to say or predict that hypertension in Blacks is transferred through the genetic make-up, but there is a tendency to label the disease as highly familial among black people in America. Of all the contributing factors related to hypertension, heredity is by far the most important.[18]

If a person is black and also obese, that individual has an increased chance of having hypertension. Obesity is one of the contributing factors associated with the elevated blood pressure for all ages. However, no primary causal relationship between the two has been established.

According to Dr. Bonnano:

> Obesity has long been implicated as a major contributing factor to coronary heart disease, hypertension and hyperlipidemia. However, there is little firm evidence to support this contention. When obesity per se is considered as an independent variable, it appears to be only a minor determinant of blood pressure or lipid level, and has little demonstrable effect on the development of coronary heart disease. Nevertheless, the association between obesity and coronary heart disease has been too conspicuous to ignore.[19]

Dr. Saunders indicated that

> Of all the contributing factors involved in hypertension, heredity is definitely the most important one, but the physicians don't really know enough about high blood pressure to identify a specific genetic abnormality that causes the disease in the black population.[20]

But they do know that it is passed down from one generation to the next generation.

Dr. Saunders also stated that Blacks with hypertension or even those with a good chance of having hypertension, use a lot of salt or highly salted foods which aggravates hypertension. There is a positive correlation between salt intake and high blood pressure. It is further suggested that a diet of the so-called soul food with high salt contents may be suicidal for black people. Therefore, it would be advisable for black people to select and limit their intake of heavily salted greens, spicy bar-be-que sauces or bacon, salt pork and ham hocks which in some circles are considered as soul food specialties.[21]

Living in the United States of America is a contributing factor that cannot be overlooked as related to hypertension and heart attacks in people. For Blacks the visible and not so visible caste and class systems, the sociological range, the emotional stress, economic battles, and the daily psychic battles for self-respect, may all play an important part in inducing high blood pressure in the black person. In addition, a study conducted by the International Cooperative Study on the Epidemiology of Cardiovascular Disease over a 10 year period, including such countries as Finland, Greece, Italy, Japan, the Netherlands, the United

States, and Yugoslavia, compared the incidence of heart disease among 12,000 men between 40 and 59 years of age. The results indicated that heart disease varied fourfold with the highest rates in the United States and Finland; the lowest was in Japan.[22] In this study diet was also found to have a significant effect on the incidence of heart disease.

Stress, anxiety, fear, frustration, rage, and hostility will elevate pressure and especially in people who have a history of hypertension. Some people will experience high blood pressure when a physician takes their blood pressure for a reading. A physician who recognizes that this could be a problem will try to relax the individual so that the pressure will return to normal. Most physicians view hypertension as a highly complex disease resulting from many forces impinging cumulatively on people.[23]

For the past few years, scientists have been focusing their attention on an enzyme produced by the kidney, renin, as being a possible cause of blood pressure problems. Laragh and his colleagues, first at the Columbia-Presbyterian Medical Center and more recently at the Cornell University Medical Center in New York City, have produced the following results in their studies:

> Patients with high plasma renin activity have the highest incidence of heart attacks, strokes, and kidney failure, whereas patients with low activity have lower risks of the serious consequence of high blood pressure . . . .[24]

There is also a great deal of interest in the relationship between hypertension and the nervous system. Reis and Amer of the Mead Johnson Research Center made the following observations:

> Certain neurotransmitters released by neurons of the autonomic nervous system in response to stress and other stimuli are thought to produce their effects by causing increases or decreases in the concentrations of these cyclic nucleotides in the target cells, including smooth muscle cells. The changes observed by the investigators are in the direction that would be expected if the smooth muscle of the vessel walls were becoming more rigid and more resistant to blood flow. If they were sustained, the result might be an increase in blood pressure.[25]

According to Marx, it is difficult to pinpoint specific causes of hypertension because there are several systems which appear to interact to control blood pressure. Each of these systems seem to influence each other by such experimental conditions as the water and salt balance of the subjects. There are still a number of problems to be solved in determining just what causes high blood pressure.[26]

Lowering the Risk of Hypertension

Many of the American people who suffer from hypertension and those who do not know that they have this condition, can be consoled by the fact that there is a constant fight to control this dreaded disease. In 1975, according to investigators at the National Center for Health Statistics, 979,180 people died of the major cardiovascular diseases. This is the first time since 1967, that the number of such deaths was less than 1,000,000.[27] A greater

awareness of the problem by the American people is a major factor in the decline. Various drugs on the market with fewer side effects enables victims to survive for longer periods of time.

Often people with hypertension do not know that they have the condition, but with regular physical check-ups by the family physician the condition can be detected to deter further damage to the patient.[28] A person's blood pressure may vary considerably. Factors such as age, general health, and emotional make-up will also affect blood pressure. If the pressure is repeatedly measured higher than 140/90, it is considered as very dangerous and treatment is required immediately.[29] Recent data compiled by the National High Blood Pressure Education Program of the NHLBI indicate that between 1971 and 1974, there was a significant increase in the percentage of cases of hypertension that were diagnosed, but the death rate dropped 28 percent from 1970 to 1975.[30] Many investigators believe this downward trend in deaths is not only due to the advanced treatment with drugs, but also to the frequent check-ups received by most Americans.

A reduction of sodium intake or a reduction of salt in the diet can be a factor that has contributed to the decline of hypertension in this country. Reisin and his associates did a study with 24 patients (16 obese hypertensive men and 8 women) who had essential hypertension noting the effects of weight loss without salt restriction.

> The authors contend that weight loss per se leads to reduction in blood pressure, independent of

> salt intake. Their study of a reduction in hypertension causes serious questions among investigators.[31]

For one thing, whenever one goes on a 1000 calorie per day diet there is an automatic salt reduction taking place. Parijs and his co-workers designed a crossover experiment with 16 patients on a restricted salt diet. The investigators estimated that "NaCl intake of 5 g a day instead of 10 g a day in patients with mild to moderate hypertension can lower the systolic blood pressure by 10 mm Hg."[32] Magnani and associates in Italy support the reduction in salt intake as being responsible for lowering the risk of hypertension.[33]

The importance of weight reduction in treating hypertension in obese patients has received a great deal of attention and helped to lower the risk. Long term epidemiologic studies have revealed that there is a relationship between obesity and essential hypertension.

> In the United States and other Western countries, approximately 60 percent of patients with essential hypertension are more than 20 percent above their normal weight.[34]

A study conducted in Evans County in 1960 with 60 subjects between the ages of 15 and 20 years old over a seven year period found that, only "overweight and weight gain determined which individuals would develop sustained hypertension and which would return to normotensive levels without therapy."[35] American's increased interest in staying thin may be a contributing factor in the decline of deaths due to hypertension in this country.

The American Cancer Society indicates that the recent

decreases in cigarette smoking and the shift to low-tar, low-nicotine cigarettes may help people to live longer. Dr. Stamler said in a recent interview that young adults can look forward to a long life if they keep a nice low blood pressure and avoid smoking too many cigarettes.[36]

According to Ashley and Rankin, there is a relationship between alcohol consumption and hypertension, and "that such hypertension may be at least partially reversible upon cessation of drinking."[37] In this source it was stated that:

> An increased prevalence of hypertension associated with heavy drinking has been suggested from studies of ambulatory heavy drinking populations from alcoholic inpatient populations, and from several general population studies including the Framingham study, the Los Angeles heart study, and the Kaiser-Permanente study. The range of intake associated with increased blood pressure was from 45 to more than 400 g of ethanol per day. In the Kaiser-Permanente study, the association of drinking 45 g or more of ethanol daily with increased blood pressure was independent of age, sex, race, smoking, coffee use, former heavy drinking, educational attainment, and adiposity; this strongly suggests that the regular consumption of this amount of alcohol is a real risk factor for hypertension.[38]

Reduction in the intake of alcohol could reduce the risk of hypertension as well as other cardiovascular diseases.

Moderate exercise, sufficient rest, learned relaxation techniques, and meditation can reduce the risk factors in hypertension. Sometimes in mild cases of hypertension, these methods alone may control the condition.[39] Some data suggest that small acute risk of cardiovascular events for adults participating in vigorous exercise may exist;

therefore, a caution is given for a trend in moderate exercise. Dr. Gibbons conducted a study over a 5 1/2 year period studying 2,935 adults (mean age, 37 years), from August 1973 through December 1978. Exercise records were maintained for persons who regularly used an exercise facility in Dallas. Each person was given a physical with a cardiovascular examination. Two of the persons suffered from cardiovascular complications. It was concluded that there is slight risk from exercise that is too vigorous.[40] However, some moderate and regular exercise such as walking is recommended for reducing blood pressure.

Atherosclerosis

Atherosclerosis contributes vastly to cardiovascular problems and receives a great deal of publicity in the news media as well as in the medical and scientific world. Practicing physicians often have difficulty in deciding on the proper treatment for controlling or reducing this cardiovascular problem. This difficulty is due to the controversies that exist developing areas of scientific investigation. One such problem is pointed out by the Select Committee on Nutrition and Human Needs of the United States, which was chaired by Senator McGovern. This committee recommended that Americans could control their weight by reducing their fat consumption to 30 percent of calories, saturated fat to at least 10 percent, and cholesterol intake to about 300 mg per day. With every type of recommendation, some individuals

will challenge the authenticity in changing dietary habits of the general public as a means of controlling or decreasing cardiovascular risk factors.[41]

A study conducted by Mahley on dietary lipid and atherosclerosis in 1979, summarized important findings associated with a high incidence of cardiovascular disease among people of many countries of the world. Mahley's results from laboratory and epidemiological animal studies indicated that dietary saturated fat and cholesterol are factors which developed accelerated atherosclerosis. With the consistency of data as related to human atherosclerosis and the acceleration of dietary induced atherosclerosis in experimental animals, it is very revealing as to a possible cause of this cardiovascular problem.[42]

Accelerated atherosclerosis can be the results of an elevation of plasma cholesterol which can be a risk factor for this heart problem. However, methods where dietary induced hypercholesterolemia causes atherosclerosis, the problem is not completely clear or well-defined. There could exist some alterations in lipoprotein, lipoprotein stability and possibly some arterial endothelial integrity. There is a risk factor involved with the consumption of high cholesterol and saturated fats without an excessive plasma cholesterol accumulation.[43] However, experiments with animals indicate that diets, regardless of the plasma cholesterol level, may cause an increase of risk factors upon the individual.[44]

In a government funded health program, a Framingham Study revealed that people who were overweight, smoked cigarettes or those with high blood pressure would most likely develop heart disease when compared with other groups.[45] If the persons had high blood cholesterol levels, they were most likely to suffer from heart attacks when compared with those who had lower levels of cholesterols. As a result of this finding, the Framingham Study convinced many doctors to advise their patients to lower their cholesterol levels.

To understand the concern for lowering the cholesterol levels, it is wise to know something about the physiological make-up. The two fatty substances in the human body that are needed are triglycerides and cholesterol. Cholesterol is the raw material in the cell walls from which bile acids and sex hormones are made. Triglycerides are forms of energy that are stored in the fatty tissues. These two substances will combine with protein as minute particles called lipoproteins, which are distributed throughout the body by the blood stream.

The cholesterol in the arteries comes in three different forms of minute lipoproteins, and they are classified by their density. The largest and the least are the very low density lipoproteins, or VLDL. Next comes the middle-sized low density lipoproteins, or LDL, and finally the small high density lipoproteins, or HDL. All three particles contain protein.

High Density Lipoproteins

The large VLDL carries most of the triglycerides; therefore, a high blood triglyceride level means a high VLDL level. On the other hand, cholesterol is carried by both LDL and HDL particles so that a high blood cholesterol can mean a high LDL, a high HDL or both. A small amount of cholesterol is carried in the VLDL particles. Research has shown clearly that the three particles have a different effect.[46] The health consequences of high levels of VLDL are not very clear, but the available evidence suggests low levels are preferable. The middle-sized LDL particles have a longer and more sobering medical history. It has been known for many years that high levels of LDL particles in the blood are associated with (and probably cause) hardening of the arteries or atherosclerosis, which eventually leads to heart attacks.[47]

The LDL receptors are important because of their involvement in the cholesterol metabolistic mechanism. When parenchymal cells outside of the liver need a supply of cholesterol, LDL produces receptors on their surfaces. These receptors act as openings through which LDL enters the cells and splits into cholesterol and proteins (free cholesterol and amino acids). When cholesterol needs are met, LDL receptors are no longer produced and the unused cholesterol is returned to the plasma. During this time, the Hepatic cells monitor the plasma levels of LDL and slow its production by the liver. The hepatic LDL receptors are part of a

feedback system that keeps plasma LDL at a normal level and furnishes cholesterol to cells when needed.[48]

Finally there is HDL, which is the smallest and most dense cholesterol carrying particle. In a study comparing high density lipoprotein cholesterol in the black and white populations, it was found HDL cholesterol was higher in Blacks than in Whites. The study compared 191 Blacks and 1,341 white males ages 5 through 44 years. The findings of higher levels of HDL cholesterol in black males was consistent even after additional adjustment for education levels, smoking habits and alcohol consumption were made. These findings illustrated that adult males in the United States who are black have lower coronary heart disease mortality rates than Whites.[49]

The Evans County Study showed a lower rate of acute myocardial infarction in black males, which indicated that there were possibly protective mechanisms that needed identifying. It was thought that since there were higher levels of high density lipoprotein (HDL) cholesterol in black males compared to white males, this could be a form of protection for them against atherosclerosis. The Evans County Study showed a higher HDL cholesterol and lower low density lipoprotein (LDL) cholesterol and very low density lipoprotein (VLDL) cholesterol in Blacks compared to white adults.[50]

Higher plasma HDL cholesterol and lower levels of triglycerides in black adult males were found in a Cincinnati

Lipid Research Clinic. High density lipoproteins (HDL) is considered as the good cholesterol and can be acquired by participants who are involved in vigorous physical activities if only for a few days in a week. This good cholesterol can prevent or even protect individuals from strokes or heart attacks.

In a study that was prompted by questions relating to the expected increase in total cholesterol with advancing age and an equal increase in all of the components of cholesterol such as HDL and LDL, it was found that there was an increase in total cholesterol with advancing age.[51] This study involved 2,000 healthy men who were evaluated at the Cooper Clinic for Complete Cholesterol Data. The HDL remained constant while the LDL continued to advance along with age. An increased total cholesterol HDL ratio was from 4.1 to 4.8.[52] The younger men's percent body fat was much less than that of the men past 40 years old, which indicated that as men get older their percent body fat increases although the body weight may remain the same. Through physical fitness at a high level, the percent body fat will decrease while the muscles will increase. A consistent physical fitness program is beneficial to men as they grow older in order to keep their total cholesterol HDL, LDL, and cholesterol HDL ratio at a normal level.[53]

In a study by Hartung at Baylor College in Houston, Texas, a comparison was made of HDL levels of marathon runners and joggers with those of inactive men. It was

found that marathoners had the highest HDL levels and the joggers had higher levels of HDL than the inactive men. Hartung indicated that normal HDL for men should be around 45 mgs per 100 mgs of blood while the marathoners had HDL levels of 64.8, joggers 58, and the inactive men showed 44.3 mgs per 100 mgs.[54]

Research has suggested that HDL helps protect people from heart attacks when it appears to interfere with the cells' ability to take in LDL, which stops the build-up of the fatty deposits that can cause hardening of the arteries and heart attacks. HDL will also aid the body in excreting excess cholesterol. Castelli indicated that women will have a higher level of HDL than men which may account to some degree the lower incidence of heart attacks in women.[55] In 1975, Miller and Miller in England found evidence that indicated that high levels HDL seemed to be a good indicator for fewer heart attacks. They found that populations such as the Scottish people who had low average HDL, were susceptible to a high rate of heart attacks; and those populations, such as Jamaican farmers and Greenlander Eskimos with high averages of HDL were not prone to have this problem. It is evident that victims of heart attacks will have a lower than average HDL levels.[56]

LDL levels in the blood can be lowered by the reduction of cholesterol and saturated fats in the diet or by drugs. With strenuous exercise and a diet of fish, low cholesterol, and drugs, HDL can be increased.[57] As can be readily seen,

there is a lot of contradiction in the literature. However, exercisers do have blood lipoprotein that possess characteristics that are associated with lower risk for developing heart disease or heart attacks.[58]

In different species of animals (dogs, rats, and swine), it was observed by Mahley that the plasma lipoproteins changed in response to high levels of dietary cholesterol. There is a difference in various diet-induced lipoproteins, although the type of plasma lipoproteins among various animal species are associated with accelerated atherosclerosis.[59]

The lipoprotein changes in hyperlipidemia and atherosclerosis should be represented by an increase in LDL and changes in the HDL. Mahley found that changes in HDL of the various animal species included a decrease in HDL and the appearance of HDL_c (high density lipoprotein with more cholesteryl ester-rich). Mahley also found that in man, "Cholesterol feeding will result in changes in the HDL which indicate the HDL_c will occur with or without an elevation of the plasma cholesterol levels."[60]

Mahley's intent was to bring together data from experimental animals of various species to get a picture of the role that saturated fat and cholesterol levels play in the development of atherosclerosis. However, there are still many questions to be answered that are related to the specific dietary compositions which will increase or decrease the development of atherosclerosis.[61]

Victims who suffer from coronary artery disease who will ultimately have heart failure, have in addition to myocardial ischemia, a severe focal or general destruction of myocardium. The destruction of myocardium will likely be characterized by some previous myocardial infarction. However, some victims are insensitive to cardiac pain when they are in situations or conditions that produce stress, but they can experience severe primary depression of cardiac function with pulmonary edema and without previous myocardial damage.[62]

In the civilized western countries, atherosclerosis has been the major cause of heart diseases. There are four major reasons for the development of this cardiovascular condition: (1) high blood pressure (140/90), (2) smoking habits, (3) diabetes, and (4) an increase in plasma cholesterol above 220. There is usually no spreading of atherosclerosis for many years after the process begins. Yellow nodules or plaques on the elbows, knees, achilles tendons or forehead may be the only clues to this cardiac problem. These nodules are called xanthomatas which are associated with lipoprotein metabolism inside the walls of the blood vessels. The levels of LDL (low-density lipoprotein) in the blood correlates directly with the incidence of atherosclerosis. When cholesterol accumulates from the metabolism of VLDL (very low density lipoprotein) in the vascular wall due to excess VLDL, there is a decrease in the metabolic activity of the smooth muscle of the vascular

wall. This leads to decreased metabolism of cholesterol and increased blood levels of cholesterol of ten times, ending in the development of atherosclerosis. High density lipoprotein (HDL) is instrumental in "scavenging" excess cholesterol depositing in the vascular wall.

Lipid Metabolism and Dietary Source

When discussing lipid metabolism from its dietary source, it can be started with:

1. Pancreatic lipases which degrade the triglycerides (TG) into diglycerides (DG), monoglycerides (MG), free fatty acids (FFA) and glycerol.
2. Bile acids are responsible for assemblying the DG, MG, FFA, and some TG in the micelles.
3. Lipids from the micelle are absorbed by intestinal mucosal cells.
4. Mucosal cells reconstruct the micelle lipids into chylomicrons (CHYLO) which enters the blood stream through the lymphatics.
5. CHYLO binds the endothelial surface of blood vessels. Lipoprotein lipase is an enzyme on the endothelial surface that breaks the TG, DG, and MG in the CHYLO into FFA and glycerol.
6. The circulating FFA are taken up by different tissues.
   A. Some of the FFA are taken up by adipose

tissue and is stored as energy.

B. FFA may be taken up by muscles to be used as a direct energy source.

C. FFA can also bind albumin, enter the liver and be resynthesized into endogenous TG. These TG are bound to lipoproteins and released as VLDL (very low density lipoprotein.)[63]

VLDL has a triglyceride rich core which is degraded by the same lipoprotein lipose as it relate to CHYLO degredation. The TG core of VLDL is degraded and eventually becomes the cholesterol core of the LDL particle. The VLDL core is surrounded by a surface which is rich in phospholipids and free cholesterol. This surface is ultimately degraded by the enzyme lecithin cholesterol acyltransferase (LCAT). As stated before, the TG rich VLDL particle is degraded by the endothelial bound lipoprotein lipase enzyme and organized to form the cholesterol ester rich core of the LDL particle.

VLDL ---------- LDL

High in TG Low cholesterol	Low TG High in cholesterol

Serum levels of VLDL and CHYLO do not correlate with the incidence of atherosclerosis. Serum levels of LDL correlate directly with the disease. The knowledge of LDL degradation has contributed significantly to the understanding of atherogenesis.

Pathway of LDL Metabolism

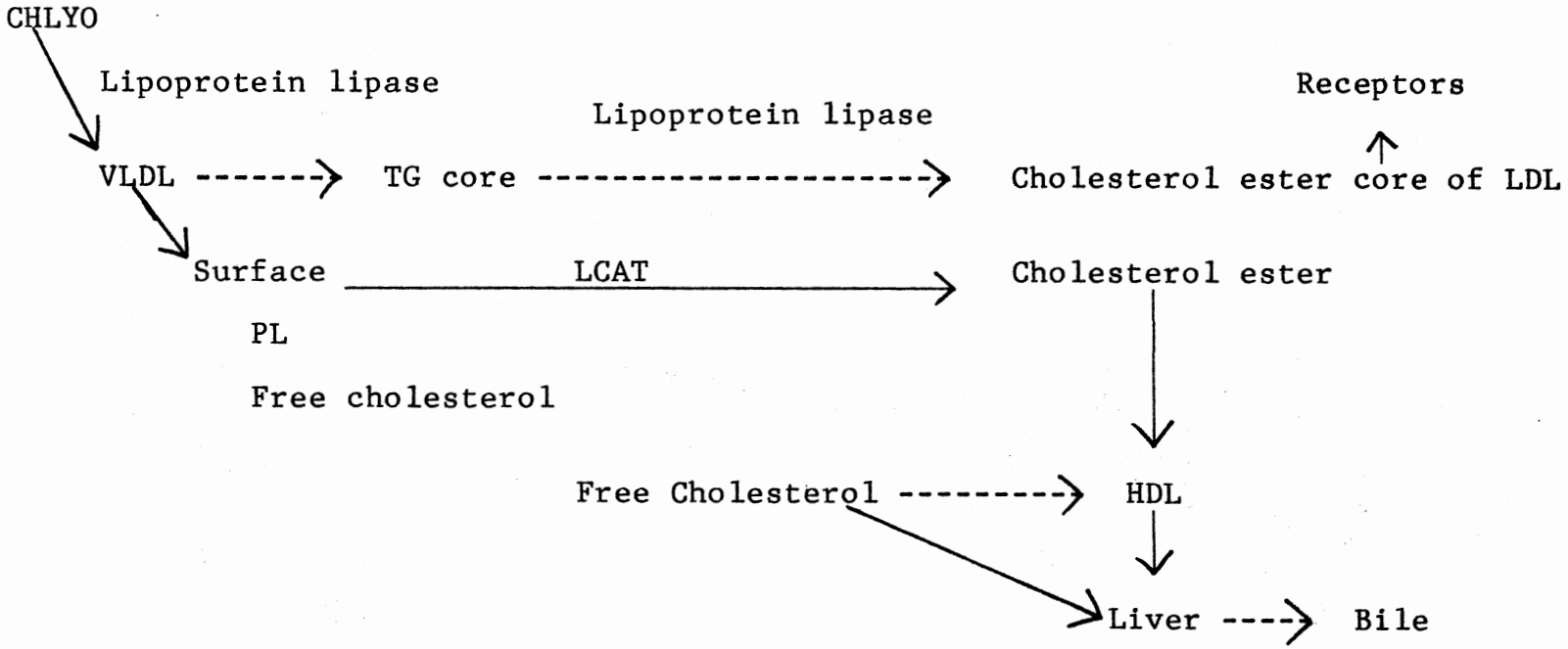
In the LDL metabolism circulating LDL particles are metabolized by cells in arterial walls which are exposed to plasma. Receptors of these cells are specific for apoliproprotein B, one of the protein constituents of LDL. The receptors are responsible for the recognition and uptake of LDL. Following its uptake, the LDL particle is broken down by lysosomal hydrolyzed to free cholesterol and FFA.

These cells also synthesize cholesterol de novo. Cholesterol released from LDL breakdown inhibits this synthesis by inhibiting HMG CoA reductase, the rate limiting enzyme for cholesterol synthesis.

cholesterol release	---- HMG CoA activity ----	cholesterol synthesis

This regulation is very important in certain types of hypercholesterolemias.

The surface of the VLDL particle contains phospholipids (PL) and free cholesterol. Physiologically this material accumulates when the VLDL core is removed. The accumulation of this surface constitutes a special problem, especially with respect to free cholesterol. Cholesterol is a stiff molecule. When it is incorporated into certain membranes such as an arterial wall, the surface becomes rigid. This deposition decreases the metabolic activity in these areas. Under normal conditions a certain amount of cholesterol is taken up by arterial smooth muscle. Its removal from the



Source: William Sodeman and Thomas Sodoman, Pathologic Physiology Mechanisms of Disease (Philadelphia, W. B. Saunders Co., 1979), p. 271.

Figure 1. Pathway of Serum Levels of VLDL and CHLYO

tissue is controlled by the enzyme lecithin cholesterol acyl transferase (LCAT). LCAT removes one fatty acid from the PL molecule and attaches it to the free cholesterol component of the surface material.

The results of the LCAT reaction include:

1. The removal of two water insoluble components, PL and free cholesterol.
2. The formation of a cholesterol ester which forms the core of the HDL particles.
3. The formation of lysolecithin, a water soluble product transported bound to albumin.

A decrease in LCAT activity leads to an accumulation of surface material, a substance that is not easily handled by the vessels.[64]

There are several factors involved in the metabolism of HDL:

1. HDL is taken up by the liver and its cholesterol core can have two fates:
   A. Most of the cholesterol is converted into bile acid and becomes part of the enterohepatic circulation.
   B. Cholesterol can be excreted directly in the bile.
2. These mechanisms constitute the major pathways for cholesterol excretion.
3. HDL is vitally important in correcting accumulation of cholesterol.

4. HDL can also take up free cholesterol from the surface of peripheral cells and incorporate it into its membrane. LCAT can then be activated, esterify the free cholesterol, which is incorporated into the core of HDL.[65]

High Density Lipoprotein-Ratio and Total Cholesterol

Cooper at the Aerobics Center in Dallas indicates the following in regard to cholesterol levels and HDL ratios:

> If the acceptable upper limits of normal total cholesterol is 250 mg% (although I would prefer that total cholesterol be under 200 mg%), then roughly one-fifth of that should be HDL or good cholesterol. In men, above 45 is considered good; whereas in women, the HDL cholesterol should be above 55. As you may recall from previous articles, it is not the fact that we are born with this 1 to 5 ratio; it is the fact that we tend to lose it. Newborn children have HDL cholesterol levels which are one-half of the total cholesterol. Yet, possibly more important than either the total cholesterol or the HDL cholesterol is the total cholesterol-HDL ratio. It must be less than 5, preferably less than 4.5.[66]

Obesity

Major contributing factors to coronary heart disease are obesity, hyperlipidemia, and hypertension. However, when obesity is dealt with as the only variable, it appears to contribute very little to the real cause of coronary problems. There is very little evidence as to the direct or major role obesity plays in the development of coronary heart disease, but its association has been too conspicuous

to be ignored.

There are many studies that have shown that excessive weight increases the risk of coronary heart disease. In the government's Framingham study,

> Men who were 30 percent overweight were 2.8 times as likely to develop coronary heart disease in ten years as men who were 10 percent or more underweight. Particularly marked in the overweight group was the likelihood of developing angina pectoris.[67]

Any individual who is 30 percent overweight is considered as obese. Even if they are not that much overweight, it is important that they should lose the first 10 pounds of excess fat. Most likely when a person gets older, he will normally gain more weight. This is usually accomplished by replacing muscles with fat as well as adding new fat tissue. A reduction of this weight is accompanied by a decrease in the mortality risks and contributes to the improvement in blood pressure and blood fat levels.

There are simple tests individuals can use to assess their actual fatness. The ruler test, which is based upon the fact that the abdominal surface between the flare of the ribs and the front of the pelvis is normally flat, can be used to determine the fat that causes the abdominal area to protrude. In this ruler test, the individual can lie flat on his back and place a ruler on his abdomen along the midline of the body. The ruler should point upward at the midsection. If it does, then the person needs to slim down.

The pinch or skinfold test calls for grasping a pinch of skin with the thumb and forefinger at the side, abdomen,

upper arm, buttock and calf. About one-half of body fat lies directly just beneath the skin surface which is measured when it is pinched. The pinch or grasp should be between one-fourth and one-half inches thick, but if the fold is over one inch, it is a good indication that there is excess fatness.[68]

Body Fat as Related to Age and Sex

In surveys of various populations carried on by scientists and related to obesity, body fat has been estimated from measuring skinfold thickness. The skinfold is composed of a double layer of sub-cutaneous fat, which should reflect the level of obesity in the body. The apparatus used to test the skinfold thickness is rather simple and portable, with a series of measurements (three or more). The folds' dimension can be rapidly acquired. When this technique is used, it presents a number of problems, because readings can differ from one observer to another. Also an absolute interpretation of the data collected is difficult unless the technique has been standardized in some fashion. There is evidence that shows sub-cutaneous and total body fat do have a relationship that varies with age. Most of the body fat will appear in some areas of sub-cutaneous tissues as the individual ages.

In a study by Malina evidence was found that indicates there are differences in skinfold and body weight between blacks and whites as early as elementary school. In

measuring skinfold and body weight, children and adolescents between the ages of 3 to 19, it was found that the mean correlation between skinfold thickness and body weight was higher for white than for black children.[69]

This skinfold thickness and a variety of other body measurements were observed periodically over a year of a sample of Philadelphia black and white children. There was a total of 825 children involved in the study with 242 black boys, 244 black girls, 191 white boys, and 148 white girls All were from the ages of 6 through 13 years of age. There was no consistent difference between black and white boys in the weight and height, except the 10 to 11 year old white boys who were heavier. The black girls were taller and heavier than their white counterparts from 8 through 12 years of age. Their skinfold measurements were taken from the left side of the body; triceps (at the back of the upper arm), subscapular (below the tip of the scapula), and midaxillary (at the level of the junction of the xiphoid process and sternum). All of the thicknesses were measured with a Lange skinfold caliper and read to the nearest one-half millimeter. It was found that white children of both sexes had consistently more subcutaneous fat in all measurements than did the black children. The most apparent difference was in the triceps skinfold site. It was found that all three skinfolds measured were generally highly correlated with each other, but the velocities for subcutaneous fat development were moderately correlated. As far as sex or race related to

trends in the patterns or even the magnitude of correlations among skinfolds and velocities for subcutaneous fat, there were no apparent differences.[70]

It has been established that skinfold fat and body weight are highly related, although the correlations for skinfold velocities and weight are a little lower than those observed at each time period. Correlation values between skinfold measured on the trunk and body weight have a tendency to be higher than those between the triceps skinfold and body weight. This does not hold true for weight and skinfold velocities. But during the adolescent period, there is a tremendous impact on subcutaneous fat development which could reduce the correlation values. The data collected indicated that a fat loss on the extremities was present in males (black and white) during the adolescent periods. Girls (black and white) showed a reduction in the rate at which fat was accumulated. The magnitude of skinfold-weight correlation in black and white children was somewhat similar for all skinfold sites when observed. However, the correlations for white boys were slightly higher than black boys, but not necessarily true for the girls. The correlations also indicated a slightly higher value between gains and losses in weight and subcutaneous fat in white girls when comparing them to that of black girls. This lower correlation was due to the thinner skinfolds of black children when observation was made between skinfold thickness and body weight.[71]

Exercise and Body Fat

In a study by Skinner and others, the rate of perceived exertion scale was evaluated. They observed a large group of middle-aged American males and made efforts to determine the relationship between heart rate and rate of perceived exertion; the correlations among heart rate, systolic blood pressure and rate of perceived exertion; and some physiological and perceptual indicators of submaximal work level. They also compared the subjective reponses to exercise of conditioned and unconditioned middle-aged men to examine the relationship between body fatness and exercise perception both on the treadmill and on the bicycle. There were 70 middle-aged males picked from a group of 167 (ages 41-60) to participate in this study on the effects of increased physical activity on some factors that are associated with the development of coronary heart disease. The subjects were basically healthy, but each had two or more characteristics which were similar such as smoking one or more packs of cigarettes per day, diastolic blood pressure between 90-99 mmhg, level of serum cholesterol of about 250-349 mg percent, from 25 to 40 percent overweight and with a similar ST segmental depression.[72]

The subjects were divided into three groups according to their percent of body fat. To estimate this, skinfold thickness was measured in the subscapular, triceps and abdomen. Although all of the subjects were or had been

fairly sedentary until a few months before the study was started, they were divided into active and non-active groups. Regardless of the possession of similar factors tha are associated with coronary heart disease, these men showed a need for weight reduction when participating in exercise tests. There was no physiological or perceptual difference that could be detected between the lean and the overweight groups when they were exercising on the bicycle, but a significantly lower workload values were obtained for the overweight subjects when they exercised on the treadmill. No difference was found in the heart rate and rate of perceived exertion relationship of the lean and overweight groups. Neither the degree of obesity of the overweight group or level of conditioning of the active group was very high.[73]

In Montoye, Mikkelsen, Metzer, and Keller's study on physical activity, fatness and serum uric acid, their skinfold measures were taken with a Lange caliper, which was calibrated by a small spring of known force-compression. This method was a measurement of body fatness known as the sum of fours. The purpose of the study was to identify the factors which are important in the maintenance of health or the development of disease. In a community of 10,000, the serum uric acid, body fatness, and physical activity were measured in the males from the ages of 16-65. With the relationship of serum uric acid to gout and to coronary heart disease and with the trend toward regular exercise, it seems to be important to study the relationship between

serum uric acid and regular physical activity. It was found that body fatness (sum of four skinfolds), was positively related to less physical active individuals and body fatness was related to serum uric acid.[74]

Obesity a National Problem

Obesity is one of the nation's most important health problems. It lends itself to significant cardiovascular diseases, diabetes, and it can shorten the life span of individuals who suffer from it. There are a number of common causes of death, but it appears that they will occur in individuals who are obese more often than those who are lean. In order to prevent this unnecessary mortality rate, it is necessary to detect early causes of obesity and try to prevent this crippling or even killing condition. After obesity has been developed, it is rather hard to treat because of its diverse nature as it relates to individual differences.

It is possible to predict what individual is most likely to become obese or even develop it at an early age. There could be genetic factors involved, if one or both parents of an offspring suffer from obesity. When this occurs, the child is most likely to suffer the same fate. It could develop during the prenatal period depending upon the type of nutrition the fetus is exposed to. In adolescence a familial and probable genetic possibility develops into obesity with normal food intake especially

with girls. Of course, the body type could play an important role in the development of this condition (endomorph, ectomorph or mesomorphic body types.) When middle aged individuals have the problem of obesity, it does not matter how much they eat or how much they do not eat, they will usually gain some body fat especially if they have an endomorphic body type. However, age, sex, and occupation could be factors that are important if the obese condition is advanced or is to be held to a minimum. It can be stated that endomorphics are inclined to deposit fat even though they apparently are consuming a normal diet with regard to their age, sex, and height.[75]

Regardless of the body type, age, sex, or height, if individuals suffer from obesity, they must be concerned about this condition because of its detrimental effect on health. To overcome or control this problem, various methods have been tried. One that seems to help many people to solve their problems is exercising. One purpose or aim of exercise is to cause changes in body composition. This can be accomplished only if the fat content is reduced by a combination of energy expenditure and caloric intake. It is understandable that a balance could be affected between the gain in lean weight through exercise and the loss of body fat through mobilization of the adipose tissues. However, exercise may or may not change the total body weight, so it is important to have a careful evaluation of the body composition. Some former athletes are proud to announce

that their weight has been the same as when they were in athletic competition many years ago. The weight may be the same, but they stand a good chance of having that weight redistributed, and the ratio of fat to lean content may have altered considerably.

With the implementation of the President's Physical Fitness Program in schools and to the general public, it is the consensus by researchers in the field that regular physical exercise has a favorable effect on body composition for individuals of all ages. Athletes in general, are known to have a great deal of lean tissue, and less body fat than the average person. Distance runners are recognized by their lean appearance and less body fat than men of their same age level.[76]

It is evident that the most efficient method to reduce body fat is by regular exercise, employed in a manner where the longer duration endurance activities are used to burn calories. This can be accomplished by vigorous walking (especially if the age is over 30) or in the so-called middle aged men. However, this is not confined to the older persons; the young can benefit from this exercise.[77]

If individuals adhere to a regular exercise program there will be a change in body composition which will cause a significant increase in active tissue and a decrease in excess fat. This effort could reduce the chances of cardiovascular risk factors.

Smoking Habits

Coronary artery disease from numerous clinical reports indicate that smoking is a major risk factor and is associated with peripheral atherosclerosis. In younger people and those who suffer from hypertension and hyperlipidemia, the risk of developing coronary problems are threefold if these victims use cigarettes.[78] There are other effects of coronary atherosclerosis which include myocardial infarction, intra myocardial fibrosis associated with arrhythmias, and depression of ventricular function. This is the most common form of heart disease in which the American man is afflicted, and is characterized by chest pains and angina pectoris which are brought on mostly by stress.

A major blow was struck to the cigarette industry in 1953, when the first reports on the health hazards of cigarette smoking were communicated to the general public. However, the cigarette industry could be optimistic and rather smug about the reports because they knew that most people are not going to give up a habit just because it was bad for them. Usually smokers do not wish to read or hear about the diseases and death risks involved in the use of cigarettes. There are some people who want to know all about the risk factors and their effect on health, even though they will continue to smoke.

In 1959, the Public Health Service, headed by the Surgeon General, evaluated all available evidence linking smoking habits to health. President Kennedy asked the

Surgeon General to develop a report on their findings and communicate it to the general public. The report indicated that cigarette smoking was a major cause of many health problems such as bronchitis, lung cancer, emphysema, coronary heart disease, and many other diseases. Further evidence indicated that men who started smoking before they were 20 years old had a higher death rate than those who began after age 35. When comparing non-smokers with cigarette smokers, the mortality risk was greater in the cigarette smokers. It was also found that the mortality risk factor was greater among those who quit smoking after the age of 25 than those who stopped earlier. In studies that reported on the degree of inhalation, the mortality ratio for a given amount of smoking was greater among the inhalers than for non-inhalers. The ratio of death was greater for smokers than non-smokers from the ages of 40 to 50.[79]

Diseases Associated with Cigarette Smoking

Coronary heart disease, lung cancer, and numerous other respiratory diseases are related to cigarette smoking. Smoking and especially cigarette smoking, is most serious in its contribution to various health disorders. Smoking is found to be the cause of exerted stress on blood vessels and narrowing of arteries, a condition called "Buerger's disease." An individual who has this problem in the leg arteries may experience cramping and severe pain of the leg

muscle while walking. Smoking has been found to be instrumental in the progression of this disease which can develop to the point of setting up gangrene which could lead to amputation of the lower extremity.[80]

Studies have been demonstrated that freedom from coronary heart disease (atherosclerosis) is much more common in non-smokers than in smokers. The progression of atherosclerosis has been related to the number of cigarettes smoked per day. In an investigation by Dr. Frank of 110,000 adults who were enrolled in a health insurance plan of greater New York, it was found that previously healthy men and women who smoked cigarettes would run two times the risk of a heart attack of non-smokers. Studies by Dr. Spain of Brooklyn on a postmortem examination of 291 subjects who had died suddenly of coronary heart disease found that they were heavy smokers. He also indicated that heavy smokers survived a shorter time after a coronary attack and was on an average of 16 years younger than non-smokers.[81]

Smoking contributes to the development of coronary problems and to death from it. There are some speculations that it also is a factor in excess levels of blood fat. It increases the heart rate; it produces a temporary rise in blood pressure. Recent studies have shown that smoking even low-nicotine filter cigarettes raises systolic blood pressure by eight points and produces a nine-beat-a-minute increase in heart rate. Other studies have shown that cigarette smoking stimulates the release of hormones from the

adrenal gland which has powerful effects on the heart and circulation and influences the levels of fats circulating in the blood stream.[82]

Another recent study indicated that the red blood cells of smokers have less affinity for oxygen. As much as 20 percent of the blood that is pushed around by the heart of the smoker is not working so far as carrying oxygen is concerned. Since the heart has the highest oxygen requirement per unit weight of any tissue, any change in the supply of oxygen could affect the heart first and thereby increase the risk of an attack for the smoker.[83]

There is a definite distinction between cigarettes, pipe or cigar smoking as they relate to heart disease. The cigar smoker usually does not inhale the smoke even if he does smoke a cigarette every now and then. Therefore, if he does not inhale with a cigar, he will not inhale as deeply as a cigarette smoker. This is also true for the pipe smoker who spends a lot of time packing, tampering, lighting, and relighting his pipe.[84]

In a study by Heyden et al., concerning body weight and cigarette smoking as risk factors, in Evans County, Georgia, it was indicated that:

> In males the incidence rate of coronary heart disease (CHD) white non-cigarette smokers was 52.7/1,000 and among Blacks 9.8/1,000; among white cigarette smokers the rate was 101/1,000 but in Blacks only 32.5/1,000. The incidence rate of CHD in the leanest Whites was 95.5/1,000, among the leanest Blacks 24.1/1,000; however, in the most obese White, the rate was 137/1,000 and among the

> Blacks, 53.6/1,000. When comparing white smokers with non-smokers in the leanest and most obese smokers run a substantial risk of developing CHD, increasing with increase in overweight (80, 90 and 150/1,000, respectively). The risk of non-smokers developing CHD does not increase from the leanest to the moderately overweight and the most obese group (51, 30 and 64/1,000, respectively). Therefore, obesity in white males seems to enhance the risk of CHD among cigarette smokers but not among non-smokers.[85]

In the follow-up study it was found that the rate of new coronary heart disease (CHD) there was a higher rate at each age in white males than was found in Blacks. This was "due to variation in diagnostic, criteria, missing black cases, or to competing causes of death."[86] The differences were possibly attributed to the distribution of some of the standard risk factors in Whites and Blacks. It was shown that black male levels of blood pressure (both systolic and diastolic) and cholesterol levels were lower than those of white males. It was further determined that both black and white male smokers had higher rates of CHD than did non-smokers, but white males had higher rates of CHD than black whether they were smokers or not.

The Effects of Smoking

Smoking has long been a "no, no" of coaches for athletes, because of the respiratory problems that this habit can develop. Scientific evidence found by Nadel and Comroe in a controlled study, demonstrated "that 15 puffs of a cigarette smoke in five minutes caused an average decrease in airway conductance of 31 percent in 36 normal subjects."[87]

The finding was for both smokers and non-smokers, and indicated that a rapid change occurred one minute after smoking began and lasted from 10 to 80 minutes. The change in inhalation was due to submicronic particles rather than nicotine.[88]

There are about 4,000 known compounds in tobacco smoke; carbon monoxide, ocides of nitrogen, hydrogen cyanide, and carbon disulfide, just to name a few. All of these may play a part in developing cardiovascular disease.[89]

Nicotine is a stimulant which affects the human system somewhat like amphetamines. It increases the blood pressure and heart rate. According to Girdano and Girdano:

> Nicotine directly affects cholinergic nerve synaspes by mimicking acetycholine. This does not only elicit greater excitability, but also blocks out meaningful impulses that would normally be directed by acetylcholine. After initially exciting these nerve fibers, nicotine "overloads" the ability of the nerve cells to respond, and a blocking effect takes place at the synapse. The second way in which nicotine affects the nervous system is through its action on the adrenal glands. It causes these endocrines to release adrenal hormones, which circulate in the blood, causing excitation of the sympathetic nervous system.[90]

There are several types of histologic classification of lung cancer, but the one that is most important at this point is the incidence of small cell lung cancer. The small cell lung cancer is highest in the age range of 50 to 70 years, and it occurs about three to four times more often among men. It has been established that there is a high correlation between cigarette smoking and lung cancer and

especially in small cell lung cancer (small cell carcinomas).[91] Individuals who smoke low nicotine and low-tar cigarettes should stand a better chance of being protected against small cell lung cancer than they would from coronary artery disease or arteriosclerosis. It is a possibility that this is due to the exposure to carbon monoxide when it is not eliminated or reduced. The carbon monoxide is the gaseous phase of cigarette smoke which is believed to significantly decrease cardiac work capacity. The hemoglobin in the blood will pick up the carbon monoxide from the lungs. This chemical reaction will alter the myocardial metabolism which interferes with oxygen transportation and, therefore, causes a myocardial problem.[92]

When comparing the life expectancy between men who smoke and those that are non-smokers, the study by Hammond best describe this comparison. Hammond discovered that out of over 1,000,000 men and women, men at the age of 35 and over who smoked two or more packs of cigarettes a day would have a life expectancy of from 20-25 percent less than those men who were non-smokers. He found that light smokers or those who smoked less than 10 cigarettes per day had two to four fewer years to live than non-smokers. These facts are an indication that cigarettes should be condemmed in this country to reduce the number of people who develop cardiovascular problems.[93]

Exercise Habits

Individuals who participate in exercise regularly may reduce their chances of acquiring cardiovascular problems. However, studies have shown that exercise alone cannot dictate the limitations of risk factors that are associated with heart disease. This could be due to other risk factors such as elevated cholesterol levels, hypertension, and smoking that are contributors to cardiovascular problems.

Cardiovascular problems are numerous and take on many forms such as atherosclorosis congestive heart failure and coronary artery disease, that it would be hard to prescribe an exercise that would be appropriate to prevent or delay one or all of these conditions. Exercises like cardiovascular problems are numerous; therefore, it would be difficult to choose the right type, frequency, intensity or duration to get the best results as related to heart disease.[94]

In the circulatory system the normal heart becomes more efficient through regular exercise. Exercise will have a tendency to improve the tone of the heart muscles which will increase the flow of blood. However, individuals who exercise to the point of exhaustion may do more harm than good. This can only be a problem to untrained persons, because trained individuals will most likely discontinue the exercise performance based upon psychological reactions long before the exerciser reaches the physiological limitations.

Exercise is beneficial at all age levels, but for the middle aged and older persons, it is most profitable. In physiological research, there are reasons to believe that with regular exercise of proper intensities and duration, much can be done to delay or postpone deterioration of the body. With new discoveries in physiology of muscular activities, helpful information is available to help determine the types and duration of physical exertion that will benefit people to a maximum degree. These physiological discoveries will allow the individuals to avoid stress, strain, and fatigue which may result in temporary or permanent damage to the heart. This scientific information can serve to help plan sound individual exercise programs and adapt them to individual needs through varying age levels.

When comparing diet and exercise to the cardiac status of an individual, it appears that the status of the person's heart seems to be influenced by regular and appropriate exercise much more than a regular and appropriate diet. Blood lipids concentrations are influenced by a proper diet, but its effectiveness on the cardiovascular system is not nearly as effective on a long term basis as regular exercise. Individuals could eat a fat rich diet as long as they exercise regularly, and they would not be affected by hardening of the arteries as those persons who are inactive.[95]

Effect on Cardiovascular Problems

Recent studies indicate that athletes or individuals

who continue to exercise "live six-eight years longer than individuals who lead a sedentary life."[96] Studies that are related to the incidence of ischemic heart disease found that exercise is the most significant factor when observing "known non-genetic modifiers of the pathogenesis of ischemic heart disease."[97]

Inactive individuals who consume a fat rich diet will produce a high concentration of blood lipids. This is due to the overload of lipids in the circulatory system, which is deposited through the system. With exercise, this condition can be minimized because the lipids will be metabolized. Although exercise may reduce or metabolize the blood lipid concentrations, it is not clear as to the influences that exercise have on this metabolistic phenomenon over a long period of time.[98] However, exercise does increase the efficiency of blood distribution throughout the circulatory system.

When physical activity is increased, it is effective in controlling some of the risk factors that are related to heart diseases and especially coronary heart disease. Serum cholesterol levels are reduced when exercise duration is up to about 45 minutes per day and at least five days a week. The resting and exercise blood pressures in coronary victims are lowered, especially those who suffer from hypertension. Cardiac function as a whole can be improved, and its stroke volume cardiac output and oxygen consumption will show an increase in its proficiency.[99]

Coronary Heart Disease and Exercise

When exercise is correctly administered, it may help to prevent a heart attack or, if a heart attack does occur, it is possible to improve the chances of surviving it. Exercise is not a remedy for CHD, because there is really no precise remedy for such a condition. There are many factors which contribute to the disease, and with the most diligent attention given to just one of these factors would not prevent the disease from occurring. Still properly used and prescribed exercise can be an important aid for the health of the heart circulation. Most Americans live a soft and sedentary existence with this age of automation which actually contributes to the reduction of physical activity. In recent studies evidence is provided that inadequate physical activity will most likely promote coronary heart disease. One study by Kahn of the National Heart Institute that was conducted with more than 2,000 post office workers in Washington, D.C., showed that the risk of developing coronary heart disease was as much as 1.9 times greater among the clerks than it was among the mail carriers. Dr. Brunner of Israel's Tel Aviv University has studied 10,000 sedentary and non-sedentary workers who had heart diseases. He found that over a 15 year period the death rate was four times greater among the sedentary workers.[100]

As indicated by these studies and others, it is clear that exercise tends to reduce the risk of coronary heart

disease (CHD), which is a fact in helping the body to maintain a normal level of cholesterol regardless of the amount of fat intake. Exercise will help individuals lose weight and maintain or reduce blood pressure elevation. In a study at Vanderbilt University, a group of men, 21 to 40 years of age who had at least one coronary disease contributory factor such as high blood pressure, high cholesterol, or obesity, participated in a six week conditioning program. The program started with mild calisthenics and built up to a mile long run. There were no restrictions on diet as each individual ate whatever he wanted. At the end of the program, the average weight loss was six pounds, and the cholesterol and blood pressures were significantly lower.[101]

It has been established that exercise can have a conditioning effect on the heart. When a body is at rest, the muscles use only about 1/30 of the oxygen they use during maximum effort. As exercise increases, the muscles need more oxygen, the heart pumps harder to get more blood into the circulation system, and the pumping efficiency of the heart becomes greater over a period of time for supplying more blood with each stroke.[102]

With proper exercise, the heart may become more efficient. When the body is at rest, it will beat more slowly, function more economically, and will require an amount less than normal for pumping activity by the heart muscle for any physical activity undertaken. It has been noted that physical activity is a form of conditioning for

the heart muscles which allows them to adjust to a period of stress or strain. This is especially true for the victim of a heart attack. Exercise increases the blood supply to the heart, and the network of capillaries and blood vessels are increased that send blood to the heart. Therefore, if a blood clot occurs that shuts off one of the coronary artery branches, the effect on the heart would be less damaging.

Alcohol Consumption

Alcohol has certain physiologial effects on the human body. It acts specifically on the central nervous system. When taken in excess, it may become habit-forming. This leads to a condition called alcoholism. Organs such as the brain, liver, and kidneys may be damaged by excessive indulgence in alcohol. Mental impairment may also result from over indulgence in alcohol consumption.

Moderate drinking can hasten the progress of atherosclerosis in some individuals, according to a study by a group of Pennsylvania investigators.[103] Although alcohol has not been singled out as a specific causal factor in the genesis of atherosclerosis, it appears to play an important role in sustaining a persistent and perhaps contributing factor of arterial deterioration.

Preliminary observations by Dr. Kuo, M.D., Professor of Medicine at the Hospital at the University of Pennsylvania, suggest that alcohol restriction is important in the successful control of the majority of atherosclerosis and

arteriosclerosis prone individuals. Dr. Kuo based his hypothesis upon two related studies that led to this result. One study dealt with 125 randomly selected chronic alcoholics. The other study dealt with 120 hyperlipidemic control-study patients. It was observed that when the control group stopped drinking, their triglycerides were very low. Although abstinence from alcohol resulted in a significant reduction in triglyceride in the control group, it was observed that within two weeks after they started drinking again, there was a rapid rise in the concentration of triglycerides in their arteriole system.

Summary

With the growing knowledge about the place of lipids in coronary artery disease, the cause of atherosclerosis is still not clearly defined. There are many factors known to contribute to this condition. These factors include aging, smoking habits, hypertension, sex (male), diabetes, obesity, high serum cholesterol, and triglycerides. More or less measurable risk factors are exercise habits and the so-called Type A personality. Some of these risk factors are uncontrollable such as age and sex. However, hypertension is still thought to be the most important risk factor for coronary heart disease (CHD).

It is estimated that between the ages of 25 to 45 years, high blood pressure kills black males 15 times more frequently than it does white males. Hypertension kills the

black female in the same age group about 17 times more frequently than it does the white female. It is the chief cause of death among black adults in America between the ages of 40 to 49. It is estimated that about 25 percent of all black Americans and 15 percent of all white Americans, age 19 years and above are afflicted with hypertension. However, a study by Heyden in Evans County Georgia diasgreed with the above stated statistics, because according to his findings the rate of CHD was higher at each age in white males than was found in Blacks. This was due to variation in diagnostic criteria, missing black cases, or to competing causes of death. The differences were possibly attributed to the distribution of some of the standard risk factors in Whites and Blacks.

Because hypertension produces no symptoms until it causes irreversible amounts of damage, it has been called the fooler of disease or the silent killer. Efforts are being made to find anti-hypertension drugs and a number of other efforts are being made to combat this dreaded disease. Even today there are means of controlling high blood pressure through effective diet, early detection, and prescription drugs. The future looks bright for control of hypertension with new and better drugs and a greater awareness of the dangers.

In the second place, perhaps triglycerides and elevated cholesterol fit the slot of being one of the most important risk factors for coronary heart disease. Cholesterol is

very important to the human body's cell walls and is the raw material from which bile acids are made and sex hormones are produced. Triglycerides are forms of energy which are stored in fat tissues. These materials need to be moved through the body by some transporting system, this system being the circulatory system. The blood in the circulatory system is made up largely of water; and since cholesterol and triglycerides cannot dissolve in water, they are combined together with proteins as minute particles called lipoproteins. An elevated cholesterol will develop a condition in the arteriole system known as atherosclerosis which is the principle disease underlying most cardiovascular diseases.

Cholesterol comes in the form of lipoprotein particles, which are classified by their minute sizes, the largest and and the least dense are the very low density lipoproteins (VLDL). The VLDL carries most of the triglycerides in the blood, and a small amount of cholesterol is also carried in the VLDL particles. These particles tend to embed themselves in the wall of the arteries, although high levels of VLDL effect on health is not very clear by researchers. It is recommended that the levels be kept low.

The middle-size low density lipoproteins (LDL) carries cholesterol which has been known to be associated with hardening of the arteries (atherosclerosis) and eventually heart attack. The small high density lipoproteins (HDL) is a cholesterol carrying particle but is considered as the

"good cholesterol." Researchers have pulled together evidence which indicate that high levels of HDL are associated with fewer heart attacks. Heart attack victims have lower than average HDL levels in their blood stream. A low incidence of heart disease is associated with high levels of high density lipoproteins (HDL) which might actually be a protection against CHD. In older people it appears that HDL cholesterol is the most predictive of all lipoprotein measurements for developing CHD. It is not known or well defined as to how HDL prevents atherosclerosis.

Although studies in the United States and abroad show some strong relationship between high triglycerides and CHD, it is still not clear or well defined as a significant risk factor. Obesity has long been implicated as a major contributing factor to coronary heart disease, hypertension and hyperlipidemia. Obesity is one of the most important medical hazards in the United States today. It shortens the life span, particularly increasing the incidence of cardiovascular disease. Many common causes of death occur at an earlier age in those individuals who are obese than in those who are lean. Many factors leading to the development of obesity are well known; most of them can be anticipated and prevented. After obesity has developed, treatment is much more difficult, because its nature is to perpetuate itself. The controversial the role obesity plays as a risk factor to CHD may hinge largely upon whether an elevated triglyceride level is considered primary or secondary as risk factors.

Smoking habits are very important as related to risk factors for coronary heart disease. Heavy cigarette smokers may experience an increase in risk of myocardial infarction when compared with non-cigarette smokers. Increased risk of myocardial infarction or death is associated with cigarette smoking in all combinations of high and low systolic blood pressures and cholesterol levels. Cigarette smoking could possibly be more important than the serum lipids as a risk factor for CHD.

Exercise conditioning programs have been recommended in medical literature as a means of decreasing prevalence, severity or mortality of coronary heart disease. Physical training has become a vital part of American society, especially when reports are made by researchers that improvement in cardiovascular functional capacity can be acquired through proper and vigorous exercises. However, a cause and effect relationship between physical inactivity and CHD has never been firmly demonstrated. There is no conclusive proof that physical exercise will prevent this disease or reduce its mortality rate. If a regular exercise training program can favorably modify some coronary risk factors, its potential for primary prevention of coronary disease is reasonable and a possibility.

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CHAPTER III

METHOD AND PROCEDURES

The purpose of this study was to compare black and white male faculty members' selected cardiovascular risk factors such as the blood pressure, blood lipids, body fat, smoking, exercise habits, and alcohol consumption. This chapter will discuss the procedures and methodology used in measuring blood pressures with the sphygmonanometer attached to the multi-channel physiograph and measuring the skinfold fat with calipers. Questionnaires were used for collecting data on subjects' smoking and exercise habits (Appendix A).

Selection of Subjects

The subjects in this study consisted of 60 black male faculty members, ages 27 to 60 at Langston Universtiy and 60 white male faculty members ages 27 to 60 at Oklahoma State University. It was just chance that the age range of the groups were identical. All male members of the Langston University faculty participated, while volunteers were solicited from OSU who participated in the OSU Faculty Fitness Research Project.

Selection of Tests

Blood pressures for the OSU faculty were taken by a sphygmomanometer attached to the multi-channel physiograph for recording blood pressures by a lab technician at OSU. Blood pressures were taken manually by Dr. Cooke, M.D., (a physician at Langston University) for the instructors at Langston University. Blood pressures were taken lying and standing (Appendix A).

In order to determine the subcutaneous body fat, the same Lange Skinfold Fat Calipers were used for all subjects in the study. They are calibrated to provide a constant tension throughout their range of motion. It measured the thickness of a double layer of skin and the interposed layer of fat. Although it is small, the resulting value from the measurements is an indirect estimation of individual differences in the thickness of subcutaneous fat (Appendix B).[1]

When measuring the skinfold thickness, it was essential to determine the precise location of the site to be measured. It was important to grasp the skinfold firmly and maintain a constant distance between the caliper, and the thumb and the finger holding the site being measured. The skinfold site was taken from the right side only as suggested by Behnke. According to his instructions:

> The triceps site will be midway between the acromion and olecranon processes on the posterior aspect of the arm, the arm held vertically, with the fold running parallel to the length of the arm. The biceps will be measured midway between the acromion and olecranon processes on the anterior aspect of the arm, the arm held vertically.

> The chest will be measured over the lateral border of the pector-alis major, just medial to the axilla, fold running diagonally between the shoulder and the opposite hip. The subscapular which is located at an inferior angle to the scapula will be measured with the fold running parallel to the axillary border.[2]

The measurements for the skinfold fat included measuring the supra-illiac where the verticle fold on the crest of the ilium at the mid-axillary line and the abdominal measurement where the horizontal fold is adjacent to the umbilicus (about one inch).[3] Five measurements were taken on each site by the investigator. The five were averaged for use in predicting body fat. In predicting body fat, two formulas were used: the Nomogram by Best and the Sum of Fours by Durin and Womersley.[4,5]

Data for the white subjects came from that collected during the current year (1980-81) at the OSU Physiology of Exercise Laboratory by Dr. Harrison and his staff.[6] The investigator did not participate in the collection of the data on the white subjects.

A pilot study on skinfold measurements was conducted at the OSU Physiology of Exercise Laboratory. Five black male subjects' (from Langston Universty) skinfold measures were taken by the investigator and compared to those collected by Dr. Harrison. This study was conducted to determine the validity of measurements taken by the investigator.

The black subjects in the study reported to the Health Center at Langston University dressed in their regular clothing. Data such as age, height, weight, smoking, and

exercise habits were recorded. Exercise was quantified according to the Cooper's Aerobic Point Scale.

All of the subjects followed the same instruction as they were tested. Blood pressures were recorded in the lying and standing positions. Subjects were told to lie quietly for one minute before a reading was taken.

Blood pressures for the black males were manually taken by Dr. Cooke, M.D. The subjects' blood pressures were taken before the blood serum was extracted from their arms as well as after the extraction. A comparison of the pressures was conducted to see if there was any change in the pressure readings.

Each subject at OSU had a sample of his blood drawn by an experienced medical technician at the OSU Physiology of Exercise Laboratory. The blood samples for both groups were analyzed at the Stillwater Medical Center to determine the levels of HDL, LDL, cholesterol, and triglycerides.

The Risko chart used was designed by the Michigan Heart Association, which is based on data from the Framingham Study. This Risko chart quantifies risk factors based upon age, heredity, smoking habits, weight, exercise, cholesterol or percent fat in the diet, blood pressure, and sex (Appendix C). The total collection of points by an individual indicated the level of risk.

Statistical Analysis

1. A contingency coefficient was used to test for

significant relationships between race and the following variables: blood pressures, lipoproteins (HDL, LDL, total cholesterol, HDL/total cholesterol and triglycerides), percent body fat, exercise, Risko, and smoking habits.

2. To determine relationships between lying blood pressure and the following: smoking, exercise points, body fat, triglycerides, HDL, LDL, total cholesterol, and HDL/total cholesterol, a coefficient of contingency was used.
3. To determine the contribution of each of the above variables to blood pressure - a step-wise regression analysis was used with each race and the total group.
4. To determine the relationship between lipoprotein levels and total body fat, a coefficient of contingency was used.
5. To determine the relationship between lipoprotein level and exercise habits, a coefficient of contingency was used.
6. To determine the relationship between lipoprotein level and smoking habits, a coefficient of contingency was used.
7. To determine the relationship between lipoprotein level and alcohol consumption, a coefficient of contingency was used.

ENDNOTES

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[5]J. V. Durnin and J. Womersley, "Body Fat Assessed From Total Body Density and Its Estimation from Skinfold Thickness: Measurement of 481 Men and Women Aged From 16 to 72 Yrs." British Journal of Nutrition, Vol. 32 (1974), pp. 77-97.

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CHAPTER IV

ANALYSIS OF DATA AND DISCUSSION OF RESULTS

The purpose of this study was to compare cardiovascular risk factors of black male faculty at a small midwestern predominately black university with white male faculty members at a large midwestern predominately white university. This chapter relates to the findings of the physiological measurements utilized, questionnaires, and discussion of the implication of the results.

The majority of people, who die or are disabled because of atherosclerosis, exhibit one or more identifiable characteristics called risk factors. Lipoprotein deposits or build-ups in the arteriole system, especially in the coronary areas are associated with high levels of plasmalipoproteins and an increased incidence of clinical atherosclerosis. The average concentration of cholesterol in American adults ranges from 205 to 225 mg/100 ml. The groups in this study had mean scores of 218 and 210 mg/100 ml which fell within the normal range of concentration.

Literature pertaining to blood pressure readings in adult males during a quiet reclining position indicate a normal range from 110 to 135 mm hg, for systolic and 60 to

90 for diastolic. Individuals' blood pressure may be considered to be normal if it comes within the minimum-maximum range, provided the relative pulse pressure is maintained. The subjects in this study had a mean score for systolic of 128 to 127 and diastolic of 77 to 79 which fell within the normal range.

Table I shows the means and standard deviation for selected cardiovascular risk-factors for black male faculty members.

The breaking points used to characterize the high and low levels for cardiovascular risk factors were as follows: systolic blood pressure 130 mm hg; diastolic 80 mm hg; percent body fat 20; HDL 50 mg/100 ml; LDL 140 mg/100 ml; triglycerides 90 mg/100 ml; total-cholesterol 190 mg/100 ml; HDL/total cholesterol 20 mg/100 ml; smoking habits, exercise, alcohol consumption, (yes-no); Risko 25.

In determining the relationship between race and cardiovascular risk factors, the subjects were categorized into two groups; those who were black male faculty and those who were white male faculty members.

Table II shows the means and standard deviation for selected cardiovascular risk factors for white male faculty members.

When comparing the two groups, there were very few Blacks participating in a regular exercise program. Only 10 black faculty members exercised and obtained aerobic points while there were 58 white faculty who exercised and obtained

aerobic points. With a mean score of 1.48 in weekly aerobic points, the black faculty members were way below the 45.06 mean score attained by the white faculty members.

TABLE I

MEANS OF BLACK MALE FACULTY RISK FACTORS

Variable	N	Mean	Standard Deviation
Systolic-Lying mm hg	64	128.55	7.26
Diastolic-Lying mm hg	64	77.84	3.64
%Body Fat, Nomogram	64	22.17	4.21
%Body Fat, Sum of 4's	64	21.71	5.25
HDL/Tot. Chol.	46	0.23	0.07
HDL mg/100 ml	46	48.37	13.44
LDL mg/100 ml	46	145.85	33.63
Tot. Chol. mg/100 ml	46	218.61	36.34
Risko	46	25.17	4.02
Aerobic Points/week	64	-1.48	1.21
Triglycerides mg/100 ml	46	122.26	61.78

The triglycerides levels may also be a direct consequence of the exercise habits of the faculty members. The black faculty members had a mean sore of 122.26 while the white faculty members had a score of 107.96.

Total cholesterol was slightly higher in Blacks, but HDL/total cholesterol ratio was about equal. This is considered by Dr. Cooper to be the most significant predictor of heart disease of all the lipoprotein measurements.

TABLE II

MEANS OF WHITE MALE FACULTY RISK FACTORS

Variable	N	Mean	Standard Deviation
Systolic-Lying mm hg	72	126.71	12.37
Diastolic-Lying mm hg	72	79.11	10.37
%Body Fat, Nomogram	71	20.21	4.21
%Body Fat, Sum of 4's	71	19.99	4.47
HDL/Tot. Chol.	46	0.23	0.06
HDL mg/100 ml	46	47.76	13.10
LDL mg/100 ml	46	140.78	28.79
Tot. Chol. mg/100 ml	46	210.09	32.40
Risko	72	25.10	5.10
Aerobic Points/week	72	50.52	7.31
Triglycerides mg/100 ml	46	107.96	64.50

A contingency coefficient was used to test for significant relationships between black and white male faculty members and cardiovascular risk factors.

Table III shows the relationship of cardiovascular risk factors to race.

There was a significant relationship between Risko measurements of white male faculty and black male faculty members. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 4.05, there was a significant relationship between the Risko score and race. With a breaking point of 25, the black males showed a higher percentage for low Risko than the white males. Risko means for

TABLE III

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS FOR BLACK AND WHITE MALE FACULTY MEMBERS (N=127)

Cardiovascular Risk Factors	Breaking Point	(Hi) N	(Lo) N	χ^2	X^2 Prob	Contingency Coefficient
Systolic B.P. mm hg	130	60	67	5.61	0.02	0.21*
Diastolic B.P. mm hg	80	54	73	2.63	0.10	0.14
%Body Fat, Nomogram	20	79	48	4.33	0.04	0.18*
HDL mg/100 ml	50	31	96	0.02	0.88	0.01
LDL mg/100 ml	150	41	86	1.00	0.32	0.09
Trig. mg/100 ml	90	49	78	3.14	0.08	0.16
Tot. Chol. mg/100 ml	190	64	63	0.97	0.33	0.09
HDL/Total Chol.	20	52	75	0.03	0.88	0.01
Smoking Habits	Yes-No	87	40	7.39	0.01	0.23*
Risko	25	67	60	4.05	0.04	0.18*

P > .05

* = Significant (.05 or greater)

both groups were approximately equal. This relationship occurred in spite of near equal means due to the fact that a few in the black faculty scored extremely high on Risko. There were five black faculty members scoring 32 points or more indicating "dangerous" risk while no white faculty members scored this high.

There was a significant relationship between systolic blood pressure and race. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 5.61, there was a significant relationship between race and systolic blood pressure.

The systolic blood pressure in the Blacks was higher than the systolic blood pressure in the Whites although the difference was smaller than that generally found in large population studies. The significant relationship found in this study indicates that a higher percentage of Blacks than Whites fell in the higher systolic pressure cell. This relationship is in agreement with most literature found on the subject.

There was a difference between percent body fat measurements of white male faculty and black male faculty members. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 4.33, there was a significant

relationship between race and percent body fat. The Blacks tended to have more body fat than the Whites.

Table IV and V show the smoking and alcohol habits of Blacks and Whites. Table IV shows the smoking and alcohol consumption for black males and Table V shows the smoking and alcohol consumption for white males. There were more black smokers than white smokers. This may indicate that Whites are more conscious of the health hazards involved in smoking.

There was a difference between smoking habits of white male faculty and black male faculty members. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 7.39, there was a significant difference between white male faculty and black male faculty members. This indicated a significant relationship between smoking and race.

The literature indicated that one to two ounces of alcohol per day tends to raise the HDL levels. In this study there was no significant relationship between alcohol consumption and the various combinations of lipoproteins.

Smoking tends to lower the HDL levels. The Blacks in this study had a higher incidence of smoking than the Whites, but this did not seem to have affected HDL levels.

A contingency coefficient was used to determine relationships between systolic blood pressure and cardiovascular risk factors in the total group. Table VI shows these relationships.

TABLE IV

SMOKING AND ALCOHOL CONSUMPTION

Smokers	Non-Smokers	Alcohol Consumption		
		None	Less than 1 a day	More than 1 a day
27	37	29	17	18

Black Males N=64

TABLE V

SMOKING AND ALCOHOL CONSUMPTION

Smokers	Non-Smokers	Alcohol Consumption		
		None	Less than 1 a day	More than 1 a day
15	56	22	35	14

White Males N=71

Shown on Table VI is the relationship of cardiovascular risk factors to systolic blood pressure.

There was a significant relationship between systolic blood pressure and exercise habit measurements. For one degree of freedom at the .05 level of significance, an

TABLE VI

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS TO SYSTOLIC BLOOD PRESSURE

Cardiovascular Risk Factors	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	31	96	0.02	0.88	0.01
LDL mg/100 ml	150	41	86	0.06	0.81	0.02
Trig. mg/100 ml	90	49	78	1.08	0.29	0.09
Tot. Chol. mg/100 ml	190	64	63	0.01	0.93	0.01
HDL/Tot. Chol.	20	52	75	0.03	0.88	0.01
Smoking Habits	Yes-No	87	40	0.65	0.42	0.07
Exercise Habits	Yes-No	61	66	4.29	0.04	0.18*
%Body Fat, Nomogram	25	79	48	12.58	0.00	0.30*

Total Group: N=127

P > .05

* = Significant (.05 or greater)

obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 4.29, there was a significant relationship between systolic blood pressure and exercise habits. This relationship indicates that the exercisers tended to have lower systolic blood pressure.

There was a significant relationship between systolic blood pressure and percent body fat measurements in the total group. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 12.58, there was a significant relationship between systolic blood pressure and percent body fat. The systolic blood pressure tended to be high when the percent body fat was high. When systolic blood pressure was low, the percent body fat was low. This was to be expected as blood pressure tends to rise with excessive fat.

Table VII lists the relationships between systolic blood pressure and risk factors among the black faculty members.

There was a significant relationship between percent body fat and systolic blood pressure for the black male faculty. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square falue was 5.18, there was a significant relationship between systolic blood pressure and percent body fat in the

TABLE VII

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS TO SYSTOLIC BLOOD PRESSURE

Cardiovascular Risk Factors	Breaking Point	(Hi) N	(Lo) N	χ^2	X^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	15	45	0.21	0.65	0.06
LDL mg/100 ml	150	22	38	0.40	0.53	0.08
Trig. mg/100 ml	90	28	32	0.03	0.86	0.02
Tot. Chol. mg/100 ml	190	33	27	0.02	0.90	0.02
HDL/Tot. Chol.	20	25	35	0.10	0.76	0.04
Smoking Habits	Yes-No	34	26	0.01	0.93	0.01
Exercise Habits	Yes-No	7	53	0.01	0.95	0.01
%Body Fat, Nomogram	25	43	17	5.18	0.02	0.28*

Black Male Faculty N=60

P> .05

* = Significant (.05 or greater)

black male faculty. When systolic blood pressure was high, the body fat was high. When the systolic blood pressure was low, the percent body fat was low. This indicates that reduction of body fat may lower the blood pressure.

Table VIII lists the relationships between systolic blood pressure and risk factors in the white faculty members.

There was a significant relationship between systolic blood pressure and percent body at among the white male faculty. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained chi-square value was 5.35, there was a significant relationship between percent body fat and systolic blood pressure among the white male faculty. When systolic blood pressure was high, percent body fat was high. When systolic blood pressure was low, the percent body fat was low. This was as expected since most research tends to support the fact that systolic blood pressure and excessive body fat are highly related.

A contingency coefficient was used to determine relationships between cardiovascular risk factors to diastolic blood pressure.

Table IX lists the relationships between diastolic blood pressure and risk factor in the total group.

There was a significant relationship between diastolic blood pressure and LDL measurements in the total group. For one degree of freedom at the .05 level of significance, an

TABLE VIII

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS TO SYSTOLIC BLOOD PRESSURE

Cardiovascular Risk Factors	Breaking Point	(Hi) N	(Lo) N	$\chi 2$	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	16	51	0.37	0.54	0.07
LDL mg/100 ml	150	19	48	0.37	0.54	0.07
Trig. mg/100 ml	90	21	46	1.39	0.24	0.14
Tot. Chol. mg/100 ml	190	31	36	0.08	0.77	0.04
HDL/Tot. Chol.	20	27	40	0.23	0.63	0.06
Smoking Habits	Yes-No	53	14	0.23	0.63	0.06
Exercise Habits	Yes-No	54	13	0.54	0.46	0.09
%Body Fat, Nomogram	25	36	31	5.35	0.02	0.27*

White Male Faculty N=67

P>.05

* = Significant (.05 or greater)

TABLE IX

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS TO DIASTOLIC BLOOD PRESSURE

Cardiovascular Risk Factors	Breaking Point	(Hi) N	(Lo) N	$\chi 2$	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	31	96	0.83	0.36	0.08
LDL mg/100 ml	150	41	86	8.14	0.00	0.25*
Trig. mg/100 ml	90	49	78	1.99	0.16	0.12
Tot. Chol. mg/100 ml	190	64	63	3.50	0.06	0.16
HDL/Tot. Chol.	20	52	75	0.16	0.69	0.04
Smoking Habits	Yes-No	87	40	0.00	0.99	0.00
Exercise Habits	Yes-No	61	66	0.15	0.70	0.03
%Body Fat, Nomogram	25	79	48	2.66	0.10	0.14

Total Group: N=127

P>.05

* = Significant (.05 or greater)

obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 8.14, there was a significant relationship between those in the total group measured for diastolic blood pressure and LDL. When diastolic blood pressure was high, the LDL tended to be low. For those in the high diastolic pressure group (N=54), over 51 percent fell in the low LDL category. This unexpected relationship has no apparent explanation. This may not be a valid relationship since one or more of the cells had less than 5 subjects.

Table X lists the relationships found between risk factors and diastolic blood pressure for the black male faculty.

There was a significant relationship between diastolic blood pressure and LDL among black male faculty. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 6.97, there was a significant relationship between those found with diastolic blood pressure and LDL. This is the same relationship found in the total group. Of those in the high diastolic blood pressure category, over 85 percent fell in the low LDL category.

There was a significant relationship between diastolic blood pressure and trigylcerides among black male faculty. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for

TABLE X

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS TO DIASTOLIC BLOOD PRESSURE

Cardiovascular Risk Factor	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	15	45	0.61	0.43	0.10
LDL mg/100 ml	150	22	38	6.97	0.01	0.32*
Trig. mg/100 ml	90	28	32	4.25	0.04	0.26*
Tot. Chol. mg/100 ml	190	33	27	6.13	0.01	0.30*
HDL/Tot. Chol.	20	25	35	2.28	0.13	0.19
Smoking Habits	Yes-No	34	26	0.24	0.62	0.06
Exercise Habits	Yes-No	7	53	1.50	0.22	0.16
%Body Fat, Nomogram	25	43	17	1.37	0.24	0.15

Black Male Faculty: N=60

P>.05

* = Significant (.05 or greater)

rejection of the null hypothesis. Since the obtained Chi-square value was 4.25, there was a significant relationship between those with diastolic blood pressure and triglycerides. When diastolic blood pressure was high, triglycerides were low. When diastolic blood pressure was low, triglycerides were high. This is the reverse of the relationship generally expected and is difficult to explain. The black group did have several subjects who were extremely high on triglyceride level.

There was a significant relationship between diastolic blood pressure and total cholesterol among black male faculty. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 6.13, there was a significant relationship between diastolic blood pressure and total cholesterol. When diastolic blood pressure was high, then total cholesterol was low. When diastolic blood pressure was low, total cholesterol was high. This relationship was also the reverse from that to be expected on the basis of previous research.

Table XI lists the relationships of cardiovascular risk factors to diastolic blood pressure for white male faculty. There were no significant relationships between the risk factors and diastolic blood pressure for white males.

To determine the contribution of each cardiovascular

TABLE XI

RELATIONSHIP OF CARDIOVASCULAR RISK FACTORS TO DIASTOLIC BLOOD PRESSURE

Cardiovascular Risk Factors	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	16	51	0.26	0.61	0.06
LDL mg/100 ml	150	19	48	1.64	0.20	0.15
Trig. mg/100 ml	90	21	46	0.12	0.73	0.04
Tot. Chol. mg/100 ml	190	31	36	0.02	0.90	0.02
HDL/Tot. Chol.	20	27	40	0.72	0.40	0.10
Smoking Habits	Yes-No	53	14	0.00	0.95	0.01
Exercise Habits	Yes-No	54	13	0.14	0.71	0.05
%Body Fat, Nomogram	25	36	31	2.57	0.11	0.19

White Male Faculty: N=67

risk factors to systolic and diastolic blood pressure, a maximum improvement for dependent variable from a step-wise regression analysis was used for the total group.

Table XII shows the contribution of each of the variables measured to systolic blood pressures. When observing the maximum R-square it was found that no significant amount of relationship existed between the common variances with each combination and blood pressure. The most important contribution to blood pressure was aerobic points. The R-square accounted for only .03. When adding the effects of all the variables, only .08 could be gained from these combinations; therefore, .92 of systolic blood sure was accounted for by something else other than these variables.

TABLE XII

MAXIMUM IMPROVEMENT FOR DEPENDENT VARIABLE SYSTOLIC BLOOD PRESSURE FROM STEP-WISE REGRESSION

No. of Variables in Model	Variable	R^2
1.	Aerobic Points/week	.031
2.	Triglycerides mg/100 ml	.048
3.	LDL mg/100 ml	.051
4.	Smoking	.056
5.	Drinking	.062
6.	Skinfold Fat	.065
7.	% Body-Fat Sums of 4's	.068
8.	% Body-Fat, Nomogram	.070
9.	HDL mg/100 ml	.080
10.	Risko	.085
11.	Total-Cholesterol mg/100 ml	.086

Table XIII shows contributions that each of the variables made to distolic blood pressure. The maximum R-square was found to have no significant relationship existing between the common variances when combined. The R-square accounts for only .04 of the total variance in blood pressure. When adding the ten variances to aerobic points, only .10 is contributed from these combinations; therefore, .90 of diastolic blood pressure is accounted for by something else.

TABLE XIII

MAXIMUM IMPROVEMENT FOR DEPENDENT VARIABLE DIASTOLIC BLOOD PRESSURE FROM STEP-WISE REGRESSION

No. of Variables in Model	Variable	R^2
1.	Aerobic Points/week	.040
2.	%Body Fat Sum of 4's	.058
3.	Smoking	.070
4.	Triglycerides mg/100 ml	.079
5.	Total-Chol. mg/100 ml	.084
6.	%Body Fat, Nomogram	.086
7.	HDL mg/100 ml	.090
8.	LDL mg/100 ml	.102
9.	Drinking	.106
10.	Risko	.107

The cardiovascular risk factors are not very good predictors of blood pressure. Something other than these risk

factors is more important to blood pressure. Blood pressure in this group cannot be predicted by these risk factors. Aerobic points was the best predictor of systolic and diastolic blood pressure.

A contingency coefficient was used to determine relationships between cardiovascular risk factors and total body fat in the total group.

Table XIV lists the relationships of total body fat with lipoprotein levels for the total group.

There was a significant relationship between total body fat and HDL measurements in the total group. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 5.07, there was a significant relationship between percent body fat and HDL. When the total body fat was high, the HDL was low.

There was a significant relationship between total body fat and HDL/total cholesterol measurements. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 5.58, there was a significant relationship between white male faculty and black male faculty. For percent total body fat and HDL/total cholesterol, when percent body fat was high, then HDL/total cholesterol expressed as a percent was

TABLE XIV

RELATIONSHIP OF LIPOPROTEIN LEVELS TO TOTAL BODY FAT FOR TOTAL GROUP (NOMOGRAM)

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	$\chi 2$ Prob	Contingency Coefficient
HDL mg/100 ml	50	31	96	5.07	0.02	0.20*
LDL mg/100 ml	150	41	86	0.95	0.33	0.09
Trig. mg/100 ml	90	49	78	1.75	0.19	0.12
Tot. Chol. mg/100 ml	190	64	63	0.44	0.51	0.06
HDL/Total-Chol.	20	52	75	5.58	0.02	0.21*

N=127

P> .05

* = Significant (.05 or greater)

low. When the percent body fat was low, the HDL/total cholesterol expressed as a percent was high.

Table XV lists the relationships of lipoprotein levels to total body fat for black male faculty. There was no significant relationship between lipoprotein levels and total body fat for the back male faculty members.

Table XVI lists the relationships of lipoprotein levels to total body fat for white male faculty. The HDL/total cholesterol was significantly related to total body fat. As body fat increased the ratio went up (improved).

A contingency coefficient was used for determining the relationships between lipoprotein levels and exercise habits. Table XVII shows the relationship between lipoprotein levels and exercise habits for the total group. There was no significant relationship between lipoprotein levels and exercise habits for the total group.

Table XVIII shows the relationship between lipoprotein levels and exercise habits for the black male population. There was no significnat relationship between lipoprotein levels and exercise habits for the black male population.

Table XIX shows the relationships of lipoprotein levels to exercise habits for the white male population.

There was a significant relationship between exercise habits and HDL levels. However, this relationship cannot be considered valid since there were no subjects in the low exercise-high HDL cell of the table.

TABLE XV

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO TOTAL BODY FAT (NOMOGRAM)

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	X^2 Prob	Contingency. Coefficient.
HDL mg/100 ml	50	15	45	3.31	0.07	0.23
LDL mg/100 ml	150	22	38	0.02	0.89	0.02
Trig. mg/100 ml	90	28	32	0.00	0.97	0.01
Tot. Chol. mg/100 ml	190	33	27	0.90	0.34	0.12
HDL/Tot. Chol.	20	25	35	1.24	0.27	0.14

Black Male Faculty: N=60

TABLE XVI

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO TOTAL BODY FAT (NOMOGRAM)

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	16	51	2.23	0.14	0.18
LDL mg/100 ml	150	19	48	0.95	0.33	0.12
Trig. mg/100 ml	90	21	46	2.06	0.15	0.17
Tot. Chol. mg/100 ml	190	31	36	0.10	0.75	0.04
HDL/Tot. Chol.	20	27	40	5.07	0.02	0.27*

White Male Faculty: N=67

P> .05

* = Significant (.05 or greater)

TABLE XVII

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO EXERCISE HABITS

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	31	96	2.89	0.09	0.15
LDL mg/100 ml	150	41	86	0.41	0.52	0.05
Trig. mg/100 ml	90	49	78	0.31	0.58	0.05
Tot. Chol. mg/100 ml	190	64	63	0.07	0.79	0.02
HDL/Tot. Chol.	20	52	75	0.53	0.46	0.07

Total Group: N=127

TABLE XVIII

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO EXERCISE HABITS

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	15	45	1.35	0.25	0.15
LDL mg/100 ml	150	22	38	0.13	0.72	0.05
Trig. mg/100 ml	90	28	32	0.35	0.55	0.08
Tot. Chol. mg/100 ml	190	33	27	0.02	0.90	0.02
HDL/Tot. Chol.	20	25	35	0.01	0.95	0.01

Black Male Faculty: N=60

TABLE XIX

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO EXERCISE HABITS

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	16	51	5.06	0.02	0.27*
LDL mg/100 ml	150	19	48	0.05	0.83	0.03
Trig. mg/100 ml	90	21	46	0.51	0.47	0.09
Tot. Chol. mg/100 ml	190	31	36	0.40	0.53	0.08
HDL/Tot. Chol.	20	27	40	1.99	0.16	0.17

White Male Faculty: N=67

P>.05

* = Significant (.05 or greater)

A contingency coefficient was used for determining the relationships between lipoprotein levels and smoking habits.

Table XX shows the relationship between lipoprotein levels and smoking habits for the total group.

There was no significant relationship between any of the risk factors and smoking habits in the total group.

Table XXI shows the relationships between lipoprotein levels and smoking habits for the black male faculty group.

There was a significant relationship between smoking habits and LDL levels measurements. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 6.01, there was a significant relationship between smoking habits and LDL.

Among smokers there were equal numbers in the high and low LDL cells. Among non-smokers, a majority fell in the low LDL category.

There was significant relationship between smoking habits and total cholesterol level measurements. For one degree of freedom at the .05 level of significance, an obtained Chi-square value of 3.84 is required for rejection of the null hypothesis. Since the obtained Chi-square value was 7.70, there was a significant relationship between smoking habits and total cholesterol measurements for black faculty. When smoking was high, then the total cholesterol was also high. When smoking was low, the total cholesterol was also low.

TABLE XX

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO SMOKING HABITS

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	31	96	0.62	0.43	0.07
LDL mg/100 ml	150	41	86	2.56	0.11	0.14
Trig. mg/100 ml	90	49	78	0.32	0.57	0.05
Tot. Chol. mg/100 ml	190	64	63	1.46	0.23	0.11
HDL/Tot. Chol.	20	52	75	0.29	0.59	0.05

Total Group: N=127

TABLE XXI

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO SMOKING HABITS

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	15	45	0.81	0.37	0.12
LDL mg/100 ml	150	22	38	6.01	0.01	0.30*
Trig. mg/100 ml	90	28	32	2.68	0.10	0.21
Tot. Chol. mg/100 ml	190	33	27	7.70	0.01	0.34*
HDL/Tot. Chol.	20	25	35	0.19	0.66	0.06

Black Male Faculty: N=60

P >.05

* = Significant (.05 or greater)

Table XXII shows the relationship between lipoprotein levels and smoking habits for the white male faculty group.

There was no significant relationship between the risk factors and smoking habits for the group.

A contingency coefficient was used for determining the relationship between lipoprotein levels to alcohol consumption. Table XXIII shows the relationship of lipoprotein levels to alcohol consumption for the total group. There was no significant relationship shown.

Table XXIV shows the relationship between lipoprotein levels and alcohol consumption for the black male faculty group. There was no significant relationship between the risk factors and alcohol consumption for the black group.

Table XXV shows the relationship between lipoprotein levels to alcohol consumption for the white male faculty group. There was no significant relationship when comparing the risk factors with alcohol consumption.

The results of these correlations point out the specificity of all of the components of lipoprotein levels separately as well as in combination.

Discussion of Results

Most literature suggests that high blood pressure is a major consideration for black Americans, because this is their number one health problem. This study revealed that the black faculty, at Langston University have several of

TABLE XXII

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO SMOKING HABITS

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	16	51	0.06	0.80	0.03
LDL mg/100 ml	150	19	48	0.00	0.98	0.00
Trig. mg/100 ml	90	21	46	0.16	0.69	0.05
Tot. Chol. mg/100 ml	190	31	36	0.84	0.36	0.11
HDL/Tot. Chol.	20	27	40	0.16	0.69	0.05

White Male Faculty: N=67

TABLE XXIII

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO ALCOHOL CONSUMPTION

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	31	96	0.54	0.46	0.07
LDL mg/100 ml	150	41	86	1.87	0.17	0.12
Trig. mg/100 ml	90	49	78	0.90	0.34	0.08
Tot. Chol. mg/100 ml	190	64	63	0.01	0.94	0.01
HDL/Tot. Chol.	20	52	75	0.02	0.90	0.01

Total Group: N= 127

TABLE XXIV

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO ALCOHOL CONSUMPTION

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	15	45	0.09	0.76	0.04
LDL mg/100 ml	150	22	38	1.88	0.17	0.17
Trig. mg/100 ml	90	28	32	2.68	0.10	0.21
Tot. Chol. mg/100 ml	190	33	27	0.03	0.88	0.20
HDL/Tot. Chol.	20	25	35	0.01	0.93	0.01

Black Male Faculty: N=60

TABLE XXV

RELATIONSHIP OF LIPOPROTEIN LEVELS
TO ALCOHOL CONSUMPTION

Lipoprotein Levels	Breaking Point	(Hi) N	(Lo) N	χ^2	χ^2 Prob	Contingency Coefficient
HDL mg/100 ml	50	16	51	0.59	0.44	0.09
LDL mg/100 ml	150	19	48	0.51	0.47	0.09
Trig. mg/100 ml	90	21	46	0.00	0.95	0.01
Tot. Chol. mg/100 ml	190	31	36	0.01	0.93	0.01
HDL/Tot. Chol.	20	27	40	0.01	0.94	0.01

White Male Faculty: N=67

the traditional risk factors for heart disease. They are almost all non-exercisers and a high proportion of them are smokers. It is assumed that they eat a high fat diet and live under certain job stresses related to financial and administrative problems at the school. Based on these factors, one would expect a relatively high incidence of high blood pressure and heart attacks among the Langston faculty. However, in this study of white and black male faculty members, there was an exception to this national trend. The black male faculty members showed very little incidence of high blood pressure with a mean score of only 2 mm hg greater than the white male faculty members. It is difficult to make assumptions about causal relationships in this particular situation. Possibly there is less stress in the rural setting. Then, it could be that with the continuous stressful situations found in this environment, the Blacks have adjusted and developed mechanisms of coping skills which tend to minimize threatening and non-threatening events. Since both populations displayed little hypertension, perhaps university teaching in Oklahoma is less stressful.

The subjects utilized in this study demonstrated few significant relationships between cardiovascular risk factors and blood pressure. The exception was the systolic blood pressure which was significant between the races with mean scores of 128.55 for black males and 126.71 for white males. As expected, the black males' blood pressure was

slightly higher than the white males. In percent body fat, the mean scores for black males was 22.17 and for the white male, it was 20.21. This indicates that black males had a tendency to have more body fat than the white males. Percent body fat generally increases with age, according to Dr. Kenneth Cooper, and standards require males to be 19 percent fat or less. Studies show that as the percent body fat increases, the blood pressure measurements tend to increase.

Weekly mean aerobic point scores for blacks was 1.48, and the mean point score for whites was 45.06. These scores indicated that the whites tended to be participants in regular exercise programs, while only a few of the blacks exercised.

LDL mean scores of 145.85 to 140.78 suggest that white males had less build-up of cholesterol in their arterial system.

In general there were few significant relationships found between lipoprotein levels and exercise, smoking or drinking. The results in this study indicated similarities between black and white male faculty members in most factors; however, the findings cannot be generalized to other or larger populations, but only to the subjects in this study.

There seemed to be a consistent pattern of significant relationships between diastolic blood pressure and lipoproteins in the Blacks. There were significant relationships

with LDL, triglycerides, and total cholesterol. All of these relationships are in the opposite direction from that expected from previous research. Previous studies would indicate that as these lipoproteins go up, the blood pressure would also go up. That did not happen in this case in relation to diastolic pressure among the Blacks. Both Blacks and Whites fell pretty much in the normal range for diastolic blood pressure. Only one black subject would be classified as diastolic hypertensive (above 90 mm hg). The validity of the relationships of diastolic blood pressure with HDL, LDL, and the sums of 4's are all subject to question since one or more of the cells in each case had less than 5 subjects.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The purpose of this study was to compare selected cardiovascular risk factors between black male faculty of a small predominately black university with that of a white male faculty from a large predominately white university. The results of the cardiovascular risk factors compared in the study indicated the following conclusions:

1. Subjects utilized in this study demonstrated a low risk for developing coronary heart disease as a group.
2. Blood pressure recordings fell within the range of normal limits for both groups.
3. The measurements for reclining systolic blood pressures showed normal values for both groups, but it was slightly higher in the black male faculty members.
4. The LDL readings were within the normal range for both racial groups, but the black males showed a higher concentration of this cholesterol.
5. There was a significant association shown between race and percent body fat with the Blacks showing

a higher percent of fat.

6. Regular exercise habits were found to be at a higher degree among the white male faculty members.
7. A significant relationship between smoking habits and race was shown; there was a much higher percentage of Blacks who were smokers.
8. In general there were no significant differences in most of the cardiovascular risk factors when compared by race.

In relation to the hypothesis as stated in Chapter I, the following conclusions are made:

1. There is no significant relationship between percent body fat and blood pressure in black, white, and combined populations in this study.
   A. There was a significant relationship between percent body fat and systolic blood pressure in all three groups.
   B. There was no significant relationship between percent body fat and diastolic blood pressure in all three groups.
2. There is no significant relationship between exercise habits and blood pressure in black, white, and combined populations in this study.
   A. There was no significant relationship between exercise habits and systolic blood pressure in black and white faculty.
   B. There was a significant relationship between

exercise habits and systolic blood pressure in all three groups.

C. There was no significant relationship between exercise habits and diastolic blood pressure in all three groups.

3. There is no significant relationship between smoking habits and blood pressure in black, white, and combined populations.

A. There was no significant relationship between smoking habits and systolic and diastolic blood pressure in all three groups.

4. There is no significant relationship between lipoproteins (total cholesterol, LDL, HDL, and triglycerides) and blood pressure in the black, white, and combined populations in this study.

A. There was no significant relationship between total cholesterol and systolic blood pressure in all three groups.

B. There was a significant relationship between total cholesterol and diastolic blood pressure in the black male faculty.

C. There was no significant relationship between total cholesterol and diastolic blood pressure in the total group and white male faculty.

D. There was no significant relationship between HDL and systolic and diastolic blood pressure in all three groups.

E. There was no significant relationship between LDL and systolic blood pressure in all three groups.

F. There was a significant relationship between LDL and diastolic blood pressure in the total group and black male faculty.

G. There was no significant relationship between LDL and diastolic blood pressure in the white male faculty.

H. There was no significant relationship between triglycerides and systolic blood pressure in all three groups.

I. There was a significant relationship between triglycerides and diastolic blood pressure in the black male faculty.

J. There was no significant relationship between triglycerides and diastolic blood pressure in the total group and white male faculty.

5. There is no significant relationship between body fat and blood pressure in black, white, and combined populations in this study.

A. There was a significant relationship between body fat and systolic blood pressure in all three groups.

B. There was no significant relationship between body fat and diastolic blood pressure in all three groups.

6. There is no significant relationship between black and white faculty.
   A. There was a significant relationship between black and white faculty's systolic blood pressure.
   B. There was no significant relationship between (race) black and white faculty's diastolic blood pressure.
7. There is no significant relationship between (race) black and white faculty's lipoproteins (total cholesterol, LDL, HDL, and triglycerides).
   A. There was no significant relationship between (race) black and white faculty's lipoproteins.
8. There is no significant relationship between (race) black and white faculty's smoking habits.
   A. There was a significant relationship between (race) black and white faculty's smoking habits.
9. There is no significant relationship between (race) black and white faculty's exercise habits.
   A. There was a significant relationship between (race) black and white faculty's exercise habits.
10. There is no significant relationship between lipoproteins (total cholesterol, HDL, LDL, and triglycerides) and percent body fat in black, white, and combined populations in this study.

A. There was no significant relationship between total cholesterol and percent body fat in all three groups.

B. There was no significant relationship between HDL and percent body fat in the black and white male faculty.

C. There was a significant relationship between HDL and percent body-fat in the combined population.

D. There was no significant relationship between LDL and percent body fat in all three groups.

E. There was no significant relationship between triglycerides and percent body fat in all three groups.

11. There is no significant relationship between lipoproteins (total cholesterol, HDL, LDL, and triglycerides) and exercise habits in black, white, and combined populations in this study.

A. There was no significant relationship between total cholesterol and exercise habits in all three groups.

B. There was no significant relationship between HDL and exercise habits in the combined population and the black male faculty.

C. There was a significant relationship between HDL and exercise habits in the white faculty.

D. There was no significant relationship between

LDL and exercise habits in all three groups.

E. There was no significant relationship between triglycerides and exercise habits in all three groups.

12. There is no significant relationship between lipoproteins and smoking habits in black, white, and combined populations in this study.

A. There was a significant relationship between total cholesterol and smoking habits in the black faculty.

B. There was no significant relationship between total cholesterol and smoking habits in the white faculty and the combined population.

C. There was no significant relationship between HDL and smoking habits in all three groups.

D. There was a significant relationship between LDL and smoking habits in the black faculty.

E. There was no significant relationship between HDL and smoking habits in the white faculty and the combined population.

F. There was no significant relationship between triglycerides and smoking habits in all three groups.

In relation to the hypothesis as stated in Chapter I, the following conclusions are made:

1. There is no significant relationship between percent body fat and blood pressure in black, white, and combined populations in this study.

A. There was a significant relationship between percent body fat and systolic blood pressure in all three groups.

B. There was no significant relationship between percent body fat and diastolic blood pressure in all three groups.

2. There is no significant relationship between exercise habits and blood pressure in black, white, and combined populations in this study.

A. There was no significant relationship between exercise habits and systolic blood pressure in black and white faculty.

B. There was a significant relationship between exercise habits and systolic blood pressure in the combined population.

C. There was no significant relationship between exercise habits and diastolic blood pressure in all three groups.

Recommendations

The primary concern of this study was to compare cardiovascular risk factors between black and white male faculty members. These groups represent a highly educated group of people - one group from a small university population and the other from a large university population.

It would be extremely important to the black male faculty to increase their awareness of cardiovascular risk

factors, since hypertension is one of the major health problems in black people, especially the black male. According to the results in this study, it is evident that black male faculty members obviously are not aware of the importance of exercise and frequent check-ups on their cholesterol levels. It would be to the black male faculty's advantage to become involved in a regular exercise program and yearly check-ups on his cholesterol level.

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APPENDICES

APPENDIX A

HEALTH AND FITNESS CENTER

HEALTH AND FITNESS CENTER
Oklahoma State University

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

NAME_____DATE_____

ADDRESS: Street_____City_____State____Zip_____

HOME PHONE_____EMPLOYER_____

OCCUPATION_____BUSINESS ADDRESS_____

PHONE_____AGE LAST BIRTHDAY_____BIRTH YEAR_____

Does your job require physical activity?_____

Do you currently smoke?____If so, what?_____no./day_____
If not, have you ever smoked?_____If yes, what?_____
no/yrs._____yrs. quit_____

Do you ever drink alcoholic beverages?____If yes, approx. no.: less than 1/day___ 1-2/day___ 3 or more/day___

Do you currently participate in any form of exercise on a regular basis?_____Indicate no. of times/weekly of participation: walking___jogging___swim___golf___basketball___handball/racquetball___tennis___other (name)____

If you walk, jog or swim, please indicate distance and time covered each session and approximate pace _____
_____

What is your estimate of your current medical condition?
ex.____good____fair____poor____
What is your estimate of your current physical fitness?
ex.____good____fair____poor____

Circle the number of blood relatives (parents, grandparents, brothers, sisters, that have been diagnosed as having some form of heart disease:

Under 60 years of age: 1 2 3 4 5 6 7 8 9

Over 60 years of age: 1 2 3 4 5 6 7 8 9

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

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

Who is your family physician?_____
City_____Address, if known_____
Date last medical exam_____
Would you like your stress test records sent to this physician?_____
If you would prefer to have your records sent to another physician, please list name and address_____
_____

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

Have you ever been told that you had high cholesterol or high triglyceride levels in the blood? Cholesterol: yes___ no___ Triglyceride: yes___no___
If you know your cholesterol and/or triglyceride levels, please list. Cholesterol_____Triglyceride_____

RISKO

Age	10 to 20 (1)	21 to 30 (2)	31 to 40 (3)	41 to 50 (4)	51 to 60 (6)	61 to 70 and over (8)
Heredity	No known history of heart disease (1)	One relative with cardiovascular disease over 60 (2)	Two relatives with cardiovascular disease over 60 (3)	One relative with cardiovascular disease under 60 (4)	Two relatives with cardiovasuclar disease under 60 (6)	Three relatives with cardiovascular disease under 60 (7)
Weight	More than 5 lbs. below standard (0)	Standard weight (1)	5-20 lb. overweight (2)	21-35 lb. overweight (3)	36-50 lbs. overweight (5)	51-65 lbs. overweight (7)
Tobacco Smoking	Non-user (0)	Cigar and/or pipe (1)	10 cigarettes or less a day (2)	20 cigarettes a day (4)	30 cigarettes a day (6)	40 cigarettes a day or more (10)
Exercise	Intensive occupational and recreational exertion (1)	Moderate occupational and recreational exertion (2)	Sedentary work and intense recreational exertion (3)	Sedentary occupational and moderate recreational exertion (5)	Sedentary work and light recreational exertion (6)	Complete lack of all exercise (8)
Cholesterol or % fat in diet	Cholesterol below 180 mg; diet contains no animal or solid fats (1)	Cholesterol 181-205 mg; diet contains 10% animal or solid fats (2)	Cholesterol 206-230 mg; diet contains 20% animal or solid fats (3)	Cholesterol 231-255 mg; diet contains 30% animal or solid fats (4)	Cholesterol 256-280 mg; diet contains 40% animal or solid fats (5)	Cholesterol 281-330 mg; diet contains 50% animal or solid fats (7)
Blood Pressure	100 upper reading (1)	120 upper reading (2)	140 upper reading (3)	160 upper reading (4)	180 upper reading (6)	200 or over upper reading (8)
Sex	Female under 40 (1)	Female 40-50 (2)	Female over 50 (3)	Male (5)	Short, stocky male (6)	Bald, short, stocky male (7)

IF YOU SCORE:

6-11:	Risk well below average
12-17:	Risk below average
18-24:	Risk generally average
35-31:	Risk moderate
32-40:	Risk at a dangerous level
41-62:	Danger urgent. See your doctor now!

TOTAL SCORE:

APPENDIX B

DATA COLLECTION FOR ESTIMATION OF BLOOD PRESSURES, SKINFOLD MEASUREMENTS AND LIPOPROTEIN LEVELS

Data Collection for Estimation of Blood Pressures, Skinfold Measurements and Lipoprotein Levels

Name_____Birth Date_____

Ht._____Wt._____

Blood Pressure:

Lying	Systolic							
	Diastolic							
Standing	Systolic							
	Diastolic							

Skinfold Measure:

	1	2	3	4	5	Total/Average			
Biceps									
Triceps									
Chest									
Abdomen									
Illium									
Scapulae (Back)									

Specific Gravity _____

Percent Body Fat _____

Lipoprotein Levels:

Cholesterol_____Triglycerides_____

High Density Lipoprotein Levels: (HDL)_____

Low Density Lipoprotein Levels: (LDL)_____

MEASURING BLOOD PRESSURE

The instrument used to measure blood pressure is called a Sphygmomanometer. It consists of a cuff and a pressure measuring device (mercury or aneroid). You can get these at most drug stores and many department stores. Costs will range from $15 to $50 and the cheaper models are generally as good as the more expensive ones. Be sure to obtain an adult size cuff. The cuff should cover two-thirds of the arm above the elbow. The bladder (inside the cuff) should go at least half way around the arm but not overlap. The usual position for taking blood pressure is to have the subject sitting. Pressure may be measured on either arm and it may vary slightly from one arm to the other. A general practice is to take the measurement on the right arm. The cuff should be at approximately heart level when wrapped around the upper arm with the bottom of the cuff about one inch above the elbow. Place the lower arm on a table or arm of a chair with the palm of the hand up. Place a stethoscope over the artery (in center of arm) just below the bend in the elbow and hold firmly in place while measuring pressure.

Measuring technique: Inflate the cuff rapidly but smoothly to around 180 mm hg. with pressure valve closed (full clockwise). Immediately open pressure valve (counterclockwise) slightly so that pressure would fall back to zero in 14-20 seconds. (Do not leave cuff inflated for over 30 sec. on subjects are as this acts as a tourniquet.) When you hear the first rhythmic thumping sound through the stethoscope, note the pressure reading. This is the systolic or upper pressure number. Continue to allow the pressure to drop until the rhythmic thumping sound disappear or become muffled. Read the pressure again at this point as this is the diastolic or bottom number. Allow air pressure to rapidly return to zero.

Blood pressure readings are reported as systolic over diastolic. If you consistently have readings of higher than 150 systolic or 90 diastolic you should consult your physician.

With continued practice you can become proficient at reading blood pressures.

INTERPRETATION OF SKINFOLDS: 50 yrs. and over

		MALE	FEMALE
Tricep	Lean	2- 8	2-12
	Good	9-16	13-21
	Avg.	17-25	22-31
	Obese	26-over	32-over

		MALE	FEMALE
Illiac	Lean	2-10	2-14
	Good	11-19	15-20
	Avg.	20-26	21-31
	Obese	27-46	32-46

		MALE	FEMALE
Abdominal	Lean	4-14	4-20
	Good	15-24	21-26
	Avg.	25-36	27-37
	Obese	37-48	38-48

		MALE	FEMALE
3 combined	Lean	10-32	10-45
	Good	33-60	46-68
	Avg.	61-88	69-96
	Obese	89-over	100-over

Inexpensive fat calipers can be ordered from:

Fat Control Inc.
P. O. Box 10117
Towson, MD 21204

"Fat O Meter"
Health Education Services
7N015 York Road
Bensenville, IL 60106

APPENDIX C

BODY COMPOSITION FROM SKINFOLD MEASUREMENTS IN MEN

BODY COMPOSITION FROM SKINFOLD MEASUREMENTS IN MEN

TABLE 19-1

Data Collection for Estimation of Body Composition from Skinfold Measurement

Name_____Age_____Ht._____in._____cm._____

Wt._____lbs._____kg._____

Date_____

Skinfold	Observer 1			Observer 2		
	First	Second	Mean	First	Second	Mean
1. Chest						
2. Abdominal						
3. Arm						

Specific Gravity_____Specific Gravity_____

Percent Body fat_____Percent Body fat_____

NOMOGRAM FOR CONVERSION OF SKINFOLD THICKNESS TO SPECIFIC GRAVITY AND PERCENT FAT IN YOUNG MEN

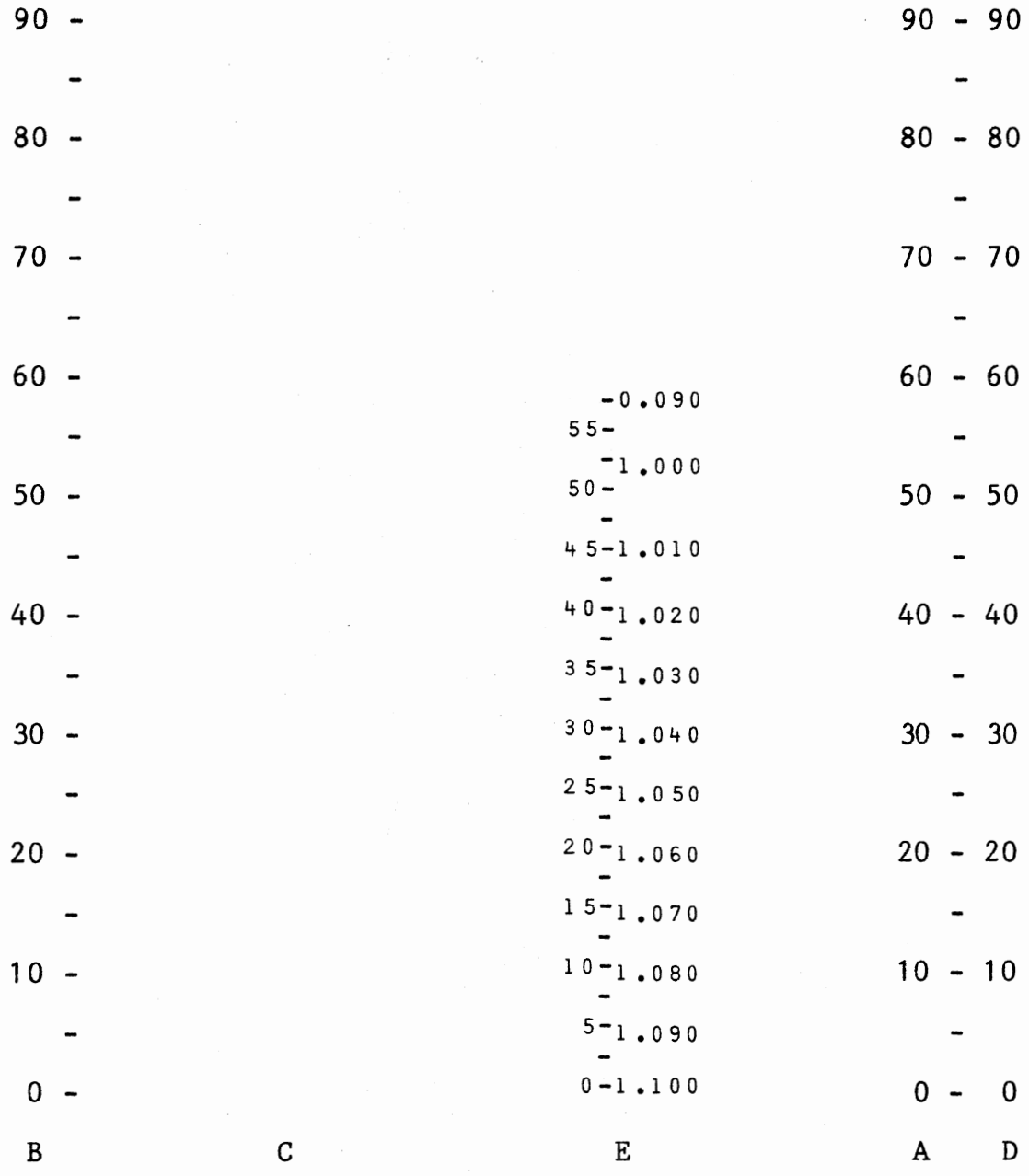

Figure 19-2. Nomogram for conversion of skinfold thickness to specific gravity and percent fat in young men. (From W. R. Best USAMRNL Report no. 113. August, 1953.)

Data Collection for Estimation of Body Composition from Skinfold Measurement

Name_____Age_____Ht._____in._____cm._____
Wt._____lbs._____kg._____
Date_____

Skinfold	Observer 1			Observer 2		
	First	Second	Mean	First	Second	Mean
1. Chest						
2. Abdominal						
3. Arm						

Specific Gravity_____Specific Gravity_____
Percent Body fat_____Percent Body Σ4's_____

Bi_____	Bi_____	Bi_____	Bi_____
Tri_____	Tri_____	Tri_____	Tri_____
Iliac_____	Iliac_____	Iliac_____	Iliac_____
Back_____	Back_____	Back_____	Back_____
Σ4's_____	Σ4's_____	Σ4's_____	Average Σ4's_____

Name_____Age_____Ht._____in._____cm._____
Wt._____lbs._____kg._____

Skinfold	Observer 1			Observer 2		
	First	Second	Mean	First	Second	Mean
1. Chest						
2. Abdominal						
3. Arm						

Specific Gravity_____Specific Gravity_____
Percent Body fat_____Percent Body Σ4's_____

Bi_____	Bi_____	Bi_____	Bi_____
Tri_____	Tri_____	Tri_____	Tri_____
Iliac_____	Iliac_____	Iliac_____	Iliac_____
Back_____	Back_____	Back_____	Back_____
Σ4's_____	Σ4's_____	Σ4's_____	Average Σ4's_____

SKINFOLD FAT MEASURES

Dr. Harrison/Schoats

_____Bi_______

_____Tri______

_____Iliac____

_____Back_____

_____Abd._____

_____Chest____

_____Arm______

Dr. Harrison/Schoats

_____Bi_______

_____Tri______

_____Iliac____

_____Back_____

_____Abd._____

_____Chest____

_____Arm______

Dr. Harrison/Schoats

_____Bi_______

_____Tri______

_____Iliac____

_____Back_____

_____Abd._____

_____Chest____

_____Arm______

Dr. Harrison/Schoats

_____Bi_______

_____Tri______

_____Iliac____

_____Back_____

_____Abd._____

_____Chest____

_____Arm______

Dr. Harrison/Schoats

_____Bi_______

_____Tri______

_____Iliac____

_____Back_____

_____Abd._____

_____Chest____

_____Arm______

Dr. Harrison/Schoats

_____Bi_______

_____Tri______

_____Iliac____

_____Back_____

_____Abd._____

_____Chest____

_____Arm______

ESTIMATE OF % FAT - DURMIN & RAHAMAH, BRIT. J. NUTR., 21:681 (1967)

% FAT-MALE	SUM OF 4SF	% FAT-FEMALE
0.9	10	5.0
2.0	11	7.0
3.0	12	8.2
3.9	13	9.2
4.7	14	10.2
5.5	15	11.1
6.3	16	12.0
7.0	17	12.3
7.6	18	13.6
8.3	19	14.3
8.9	20	15.0
9.4-	21	15.7
10.0	22	16.3
10.5	23	17.0
11.0	24	17.5
11.5	25	18.1
11.9	26	18.7
12.4	27	19.2
12.8	28	19.7
13.2	29	20.2
13.6	30	20.6
14.0	31	21.1
14.4	32	21.6
14.8	33	21.0
15.1	34	22.4
15.5	35	22.8
15.8	36	23.2
16.2	37	23.6
16.5	38	24.0
16.8	39	24.3
17.1	40	24.7
17.4	41	25.1
17.7	42	25.4
18.0	43	25.7
18.2	44	26.1
18.5	45	26.4
18.8	46	26.7
19.1	47	27.0
19.3	48	27.3
19.4	49	27.6
19.8	50	27.9
20.0	51	28.2
20.3	52	28.5
20.5	53	28.7
20.8	54	29.0
21.0	55	29.3
21.2	56	29.5
21.4-	57	29.6
21.6	58	30.1
21.8	59	30.3
33.0	60	30.5
22.3	61	30.8
22.5	62	31.0
22.7	63	31.3
22.8	64	31.5
23.0	65	31.7
23.2	66	31.9
23.4	67	32.2
23.6	68	32.4
23.8	69	32.6
24.0	70	32.8
24.1	71	33.0
24.3	72	33.2
24.5	73	33.4
24.7	74	33.6
24.8	75	33.8
25.0	76	34.0
25.2	77	34.2
25.3	78	34.4
25.5	79	34.6
25.6	80	34.8
25.8	81	35.0
25.9	82	35.1
26.1	83	35.3
26.2	84	35.5
26.4	85	35.7
26.5	86	35.9
26.7	87	36.0
26.8	88	36.2
27.0	89	36.4
27.1	90	36.5
27.3	91	36.7
27.4	92	36.9
27.5	93	37.0
27.7	94	37.2
27.8	95	37.3
27.9	96	37.5
28.1	97	37.7
28.2	98	37.8
28.3	99	38.0
28.4	100	38.8

% FAT-MALE	SUM OF 4SF	% FAT-FEMALE
28.6	101	38.3
28.7	102	38.4
28.8	103	38.6
28.9	104	38.7
29.1	105	38.9
29.2	106	39.0
29.3	107	39.1
29.4	108	39.3
29.5	109	39.4
29.7	110	39.6
29.8	111	39.7
29.9	112	39.8
29.0	113	40.0
30.1	114	40.1
30.2	115	40.2
30.3	116	40.4
30.4	117	40.5
30.6	118	40.6
30.7	119	40.8
30.8	120	40.9
30.9	121	41.0
31.0	122	41.1
31.1	123	41.3
31.2	124	41.4
31.3	125	41.5
31.4	126	41.6
31.5	127	41.7
31.6	128	41.9
31.7	129	42.0
31.8	130	42.1
31.9	131	42.2
32.0	132	42.3
32.1	133	42.4
32.2	134	42.6
32.3	135	42.7
32.4	136	42.8
32.5	137	42.9
32.6	138	43.0
32.7	139	43.1
32.8	140	43.2
32.9	141	43.3
32.9	142	43.5
33.0	143	43.6
33.1	144	43.7
33.2	145	43.8
33.3	146	43.9
33.4	147	44.0
33.5	148	44.1
33.6	149	44.2
33.6	150	44.3
33.7	151	44.4
33.8	152	44.5
33.9	153	44.6
34.0	154	44.7
34.1	155	44.8
34.2	156	44.9
34.2	157	45.0
34.3	158	45.1
34.4	159	45.2
34.5	160	45.3
34.6	161	45.4
34.6	162	45.5
34.7	163	45.6
34.8	164	45.7
34.9	165	45.8
35.0	166	45.9
35.0	167	46.0
35.1	168	46.1
35.2	169	46.1
35.3	170	46.2
35.4	171	46.3
35.4	172	46.4
35.5	173	46.5
35.6	174	46.6
35.7	175	46.7
35.7	176	46.8
35.8	177	46.9
35.9	178	47.0
36.0	179	47.0
36.0	180	47.1
36.1	181	47.2
36.2	182	47.3
36.2	183	47.4
36.3	184	47.5
36.4	185	47.6
36.5	186	47.6
36.5	187	47.7
36.6	188	47.8
36.7	189	47.9
36.7	190	48.0
36.8	191	48.1
36 9	192	48.1
36.9	193	48.2
36.0	194	48.3
37.1	195	48.4
37.1	196	48.5
37.2	197	48.5
37.3	198	48.6
37.3	199	48.7
37.4	200	48.8

VITA

Albert H. Schoats

Candidate for the Degree of

Doctor of Education

Thesis: A COMPARISON OF BLACK AND WHITE MALE FACULTY MEMBERS' SELECTED CARDIOVASCULAR RISK FACTORS

Major Field: Higher Education

Minor Field: Health, Physical Education, and Recreation

Biographical:

Personal Data: Born in Muskogee, Oklahoma, July 4, 1927, son of Jesse and Ida B. Schoats; married to Dr. Virginia L. Schoats with four children, Jacqueline, Janice, Jennifer, and Jerry Lynn.

Education: Graduated from Manual Training High School, Muskogee, Oklahoma, in May, 1947; received Bachelor of Science degree in Physical Education from Langston University in May, 1951; received Master of Science degree from Oklahoma State University in July, 1957; received certification in Drivers Education from Northwestern State University, Alva, Oklahoma, July, 1957; received certification from the National Science Foundation at Grambling University, July, 1959, 1960, 1961; completed requirements for the Doctor of Education degree at Oklahoma State University in December, 1981.

Professional Experience: Employed as high school principal, science teacher and basketball coach in Paris, Arkansas, September, 1952-May, 1953; employed as science and mathematics teacher and basketball coach at Wellston, Oklahoma, September, 1953-May, 1954; employed as science teacher, basketball, baseball, track and football coach at Hugo, Oklahoma, September, 1954-May, 1956; employed as driver education instructor, physical

education teacher, assistant basketball coach, assistant football coach in the Muskogee, Oklahoma, school system, September, 1956-May, 1962; employed as driver education instructor, physical education teacher, head basketball coach, assistant football coach, and science teacher, September, 1962-May, 1966; employed as head football coach, driver education instructor, physical education teacher, and biology teacher, September, 1966-February, 1970; employed as physical education teacher and head football coach at Langston University, February, 1970-May, 1977; employed at Langston University as physical education teacher, June, 1977 to the present.

Professional Organizations: Oklahoma Education Association; National Education Association; Oklahoma Coaches Association; Oklahoma Health and Physical Education Association; American Alliance for Health, Physical Education, Recreation and Dance; Kappa Alpha Psi Fraternity Life Member.