Running Head: WAIST SIZE

The Relationship of Waist Size to Blood Pressure and Cholesterol Among College Students

Stephanie Miller

A Senior Thesis submitted in partial fulfillment
of the requirements for graduation
in the Honors Program
Liberty University
Spring 2007

Acceptance of Senior Honors Thesis

This Senior Honors Thesis is accepted in partial fulfillment of the requirements for graduation from the Honors Program of Liberty University.

_____

Vance J. Pickard, Ed.D.
Chairman of Thesis

_____

David Horton, Ed.D.
Committee Member

_____

James A. Borland, Th.D.
Committee Member

_____

Judy R. Sandlin, Ph.D.
Assistant Honors Program Director

_____

Date

Abstract

The relationship between food consumed and the resulting body size is not easy to understand. Considering the vast array of nutritional input, numerous hypotheses have been tested for connections among diet constituents and the shape of the human body. The purpose of this study was two fold: to determine the correlation between the level of blood cholesterol and waist size among college students and to determine the correlation between blood pressure and waist size among college students. Volunteers from an upper level Kinesiology class at Liberty University were measured around the waist, screened for blood pressure, and administered a cholesterol blood test. Three groups were formed based on waist size measurements. Significant differences were found between waist size and blood pressure. Results of this study extended the research of Roizen and Oz (2006) using college students and should be used for preventative purposes.

The Relationship of Waist Size to Blood Pressure and Cholesterol Among College Students

Sadly, obesity now affects all ages of the human population, and is progressing into the number one health problem in America. In a society so obsessed with food and fast dining, it becomes increasingly difficult to manage body fat. In fact, according to the National Center for Health Statistics (2007), over 30% of adults over 20 years in age are obese. The health problems associated with obesity are countless, including, but not limited to: increased morbidity, diabetes, cardiovascular disease, hypertension, gallstones, and certain types of cancer. Persons with obesity also suffer from psychological, social, and even economic consequences (Wyatt, Winters, & Dubbert, 2006). Despite minor genetic implications involved, becoming overweight can be avoided; ergo, eliminating the array of complications upon the human body.

The problem of obesity is strongly related to the diet. Foods high in saturated and trans fats affect the level of blood cholesterol. Complications with blood cholesterol contribute to a rise in blood pressure, a serious health risk. The destructive trail of a poor diet induces a variety of health risks, all of which can be prevented.

Excess calories, whether containing fat or cholesterol, will all be converted into fat and stored in various places throughout the body, mainly the peritoneal cavity. The location of this cavity is at the belly button and is, therefore, related to an increase in waist size. Within the peritoneal cavity lies the omentum, an organ designed to help store food processed by the body (Roizen & Oz, 2006). The larger the excess of food, the more the omentum must store; thus, creating a path to obesity and the variety of complications

it produces. Roizen and Oz (2006) suggest controlled saturated and trans fat intake will reduce blood cholesterol, waist size, obesity and its effects on the human body.

Cholesterol is positioned throughout the human body, and is found in abundance throughout the human diet. Additionally, cholesterol is the product of two other causes inside of the body: derived from bile and secreted by intestinal epithelial cells. Similar to fat, cholesterol has a bad rap despite how useful it is for the body: repairing arteries and fueling energy production for the liver (Jensen, 1976). Just as energy can be stored as fat, the storage form of cholesterol is also fat; thus, the supposed link between fat and cholesterol. Still, long before cholesterol is stored as fat, it is broken down and used for the use and removal of fat. Furthermore, there are two major components of cholesterol which influence the blood level of cholesterol, and are the focus of this research.

The additional elements connected to cholesterol are low-density lipoproteins (LDL) and high-density lipoproteins (HDL). LDL is responsible for transporting cholesterol and lipids to tissues; and HDL is vital for transporting cholesterol to the liver for removal (Sizer & Whitney, 2006). The purpose and function of both LDL and HDL cholesterols are not bad. In fact, if the body can digest and synthesize healthy amounts of each, it will remain in a comfortable state of homeostasis. This is the struggle, keeping the balance.

The misconception would be to simply lower the intake of dietary cholesterol to maintain this balance. While dietary cholesterol is positively related to cholesterol throughout the body, a blood test is used to measure the specific levels of both LDL and HDL cholesterol in the body. Conversely, there is a vast difference between dietary cholesterol and blood cholesterol. It is necessary to repeat that cholesterol is not the

prime culprit in raising the level of blood cholesterol. Rather, Sizer & Whitney (2006) affirm high amounts of saturated and trans fats are the dietary components which contribute to elevated blood cholesterol. In short, the greatest influence over cholesterol levels is by monitoring saturated and trans fat intake.

In addition to elevating blood cholesterol, obesity can also aggravate blood pressure. Bragg (1975) defines blood pressure as the amount of force or pressure exerted on blood vessels and arteries to pump blood throughout the body. It is determined by measuring the amount of pressure during two phases of the heart, contraction and relaxation. Systolic blood pressure is related to the contraction phase of the heart; greater pressure is required. The relaxation phase of the heart correlates to diastolic blood pressure; less pressure is required. Being overweight puts undue strain on the heart (Bragg, 1975). Fat cells require blood; thus, the heart strains to supply blood to excess fatty tissue in addition to the life supporting vital organs. Maintaining a youthful waist will decrease excess fat, thus limiting increased levels of blood cholesterol and blood pressure.

Statement of the Problem

The problem of this study was to investigate the relationship between the cholesterol level, blood pressure, and waist size of college students. More specifically, the study examined the relationship between waist size, HDL, LDL, systolic blood pressure, and diastolic blood pressure.

Purpose of the Study

If there was a relationship between waist size, cholesterol, and blood pressure, college students would need to monitor more closely foods related to increased blood

cholesterol, or those containing high levels of saturated and trans fats. Based upon a positive or negative correlation, persons of all ages would be able to manage their waist size by also monitoring such foods. Furthermore, if waist size was a more accurate measure of health and wellness, education concerning the contribution of cholesterol to a well-balanced diet would enable college students to make wise decisions when consuming food. The result of this proper nutrition would be a trimmer waist line and the reduced likelihood of disease and disorders later in life.

Hypothesis

The following hypotheses were tested by the data:

1. College students who recorded a smaller waist size would have a higher level of HDL cholesterol, when compared to students who recorded medium or large waist sizes.
2. College students who recorded a smaller waist size would have a lower level of LDL cholesterol, when compared to students who recorded medium or large waist sizes.
3. College students who recorded a smaller waist size would have a lower systolic blood pressure, when compared to students who recorded medium or large waist sizes.
4. College students who recorded a smaller waist size would have a lower diastolic blood pressure, when compared to students who recorded medium or large waist sizes.

Delimitations

This study is delimited to:

1. college students, ages 20 to 26.
2. a sample consisting of members from an upper level kinesiology class of Liberty University's 2006-2007 academic year.

3. the use of a fasting cholesterol blood test to measure both LDL and HDL cholesterol levels.

4. the use of a sphygmomanometer to measure blood pressure.

5. administration of the cholesterol data collection instruments during individual testing conducted on the participant's own time.

Limitations

The study is limited by:

1. the sample size was small.

2. the investigator was unable to personally conduct the cholesterol test. To ensure standardization, participants were instructed to visit a doctor's office.

3. the investigator was unable to personally measure the subjects' blood pressure.

4. the collected data was reported to the investigator based upon the honor system.

Assumptions

1. Extra fat is stored largely in the omentum.

2. All subjects recorded their data honestly and correctly.

Review of Literature

Over half of the American population is overweight or obese. The most recent surveys conducted by the National Center for Health Statistics (2003-04) reveal 66.3% of adults 20 years and older are overweight, and 32.2% are obese. From the mid seventies, the statistics for obesity indicate over a one hundred percent increase rate. Obesity is measured using a mathematical computation to determine the body's Body Mass Index (BMI), using the formula weigh in kilograms divided by height in meters squared. The result is a BMI number that correlates with an approximate amount of body fat. Overweight is defined by a BMI of 25 to 29.9, and a BMI over 30 is defined obese (American Obesity, 2002). Obesity is of great concern due to the amount of health problems associated with the disease. A variety of serious health concerns may surface, including, but not limited to: diabetes, cardiovascular disease, hypertension, gallstones, and certain types of cancer. Obese individuals are also subject to increased mortality rates (Wyatt, Winters, & Dubbert, 2007). In addition to these physical conditions, persons with obesity also suffer from psychological, social, and economic consequences. Although heredity can be a minor cause of weight gain, becoming overweight or obese can be avoided. Thus, the complications upon the human body and associated consequences can be eradicated.

The relationship between food consumed and the resulting body size is not easy to understand. To grasp the concept entailing the digestion of nutrients is difficult, especially considering the plethora of elements involved. Beginning with the human psyche, and ending with a figure reflecting the choices of such a thought process, the following investigates the daunting traveling process in between. Hour upon hour the

brain sends chemical messages to the stomach. While this is a slight exaggeration, it is precisely the manner in which the human body was so intrinsically designed. In fact, Mason (1960) reports hunger is an instinct of survival; it entices the human to find food. Although there are several reasons for experiencing hunger, there is only one reason for consuming food: for energy.

Every food consumed contains calories, and each calorie must be transformed into energy, otherwise it is stored (Hafen, 1981). There are three possible paths for food. Food is broken down by various organs and can be used as energy, stored as fat, or processed as waste (Roizen & Oz, 2006). The time scale for determining which course food will travel is brief. Since the body is working full time all of the time, there is a high demand for a constant supply of energy. The most available source will be used first for energy, and the remainder must be processed immediately thereafter. Regardless of the type of food or nutrient consumed, if not utilized quickly, all remainders will be converted into fat for storage (Roizen & Oz, 2006).

The stored fat forms fat tissue which is useful for a variety of beneficial measures. Fat tissue functions to insulate the body, regulate body temperature, and protect vital organs. The purpose of an individual fat cell is to store surplus glucose that has been converted to fat. Glucose is the most common substance converted into energy; thus, excess glucose represents idle energy. Consequently, Hafen (1981) proposes stored energy is represented by the amount of fat in the body. Stored energy is vital; it is used during long-term periods in which the body is void of food consumption. On average, there is enough stored energy to suffice two months of total starvation (Hafen, 1981); which is an advantageous use for food stored as fat. However, the body only requires a

certain amount of fat for stored energy, and the previously mentioned functions of fat tissue. The remaining fat must somehow be digested; otherwise it will continue to be stored on top of the existing reserves.

Several functions of the small intestine are to assist the decomposition fat. Lipases excreted by the pancreas assist this decomposition. These small particles mix with bile from the liver, and are easily absorbed by cells throughout the body (Roizen & Oz, 2006). Still, the question remains what the destiny is of remaining fat particles, those not yet absorbed into cells. When fat has been adequately distributed to cells in need, excess fat particles gravitate to existing fat cells. Roizen and Oz (2006) avow that at no point during maturation does the body produce additional fat cells. In fact, an adult body has the same number of cells as it did in adolescence. While the number of cells does not increase, the size of the cells does. As more fat is stored, an increase in size occurs among fat lobules, which are found within each fat cell. Ergo, the existing cells are not increasing in number, but in size. While it has been stated several times that fat deposits or tissues are not negative, an excess of deposited fat can lead to becoming overweight or obese (Hafen, 1981). Additional examination will explore more closely a specific avenue of fat—its role in affecting the level of cholesterol in blood.

The relationship between fat and its decomposition has been established; the application being to monitor saturated and trans fat intake (Roizen & Oz, 2006). What has not been assessed, however, is the relationship between the cholesterol levels in the blood and the waist size of the human body. Excess calories, whether in the form of carbohydrates, proteins, or fats, will all be converted into fat and stored in various places throughout the body. This fat, or stored energy, has been designed to be used as a reserve,

and Jensen (1976) suggests there is no maximum amount of space or limit for fat storage. The human body is a highly expansible storage container for fat. Provided the increasing number of obese individuals, his hypothesis could be correct.

Further research by Jensen (1976) indicates the fat is stored in several areas. Fat is deposited along the subcutaneous layer and inside the peritoneal cavity. It is placed around skeletal muscles and organs, such as the kidneys and ovaries. Naturally, only so much fat is necessary to cover the kidneys, ovaries, and skeletal muscle. Hence, the majority of fat is neither stored there, nor just below the skin in the subcutaneous layer. With the limited amount of space in each area, fat must be deposited elsewhere. This leaves one section, the peritoneal cavity.

The location of this cavity is at the belly button. Observe various overweight individuals. Notice that although there may be increases in size around the perimeter of the body, the largest proportion of size gain is around the stomach. This peritoneal cavity contains an organ called the omentum. Organs like the small intestine assist processing food, while the omentum facilitates a storage place (Roizen & Oz, 2006). The larger the excess of food, the more the omentum must store. This contributes to another cause for an increase in waist size; yet, it is not the only explanation.

The digestion of fat demands an extensive process initiated in the mouth. Due to the complex structure of fat, it undergoes the slowest digestion (Hafen, 1981). Several actions and reactions occur at the beginning of the digestive tract; however, the major components of fat digestion occur in the small intestine. Upon its entrance into the small intestine, fat triggers the release of pancreatic enzymes. The enzymes break down and separate the fat into three fatty acids and glycerol.

At this point the fatty acids are insoluble; therefore, they cannot break through the layer of water surrounding the jejunum (middle section of the small intestine). To reach the absorptive cells located in the jejunum, fat must puncture this membrane. The fatty acids coalesce with bile to become soluble; and the molecules can pass freely into the cell membrane where glycerol and the fatty acids are reformed. Finally, cholesterol and protein combine with the glycerol and fatty acids so the substances can enter lymph vessels and blood vessels, thereby being transported throughout the body (Hafen, 1981). These two functions—the decomposition of fat by pancreatic enzymes and the fusion of fat with bile to become soluble—are vital to a healthy and properly functioning small intestine.

If either of these functions fail or becomes compromised, McMurray (1983) affirms insufficient fat decomposition and devastation to the intestines would occur. The alarming consequence of such intestinal derangement is the inflammation of the intestine and abdominal area. The inflammation leads to a variety of medical concerns, one of which is an increase in waist size. The increase in waist size contributes to the likelihood of becoming overweight or potentially obese. This manner of size increase is the result of insufficient fat decomposition and damage to the small intestine. When fat is broken down correctly and the small intestine is functioning properly, the waist can still increase in size. This occurs as digested fat is continually stored in fat tissue; a problem attributed to overloading individual fat cells.

Cholesterol is positioned all over the human body (Jensen, 1976). Cholesterol is such a necessity that it is both consumed and produced by the body. Cholesterol is found in abundance throughout the human diet. However, despite what Jensen calls the

"ubiquitous occurrence" (p.867) of cholesterol in the diet, it is also the consequent of two internal processes as well. It is derived from bile, and also by secretions from intestinal cells and tissues. Since cholesterol is so important it is necessary to discuss the various uses and functions of the element found throughout the human body.

There are quite a variety of functions of cholesterol. Cholesterol is vital to repair arteries in the body (Roizen & Oz, 2006). In seeming contradiction, it can also be the cause of fatal damage to the same arteries. Prior to discussing the relationship between cholesterol and the arteries, several foundational functions of the constituent are noteworthy.

Cholesterol is present during both development and maintenance of billions of cells that form the body. It shapes the membranes for cell plasma and organelles traveling in and between cells (McMurray, 1983). Dr. McMurray also reports cholesterol is used as a chief fuel for the production of energy in several organs, such as the heart and liver. Skeletal muscles also use cholesterol for energy. Cholesterol is the precursor for prostaglandins—essential substances for regulation. Like fat, cholesterol is used for the production of energy. It is comprised of chemical and physical properties comparable to fat, and metabolized similarly. For these reasons, cholesterol is therefore incorporated as a dietary fat (Jensen, 1976). In fact, the storage form of cholesterol is fat!

Just as energy can be stored as fat, cholesterol can be also stored as such. Therefore, a few more functions of cholesterol are provided, given its similarity to fat in property and functionality. Analysis of the functions related to fat and size increase is most important. Cholic acid in the liver is formed by cholesterol, which in return contributes to the synthesis of bile salts, which are significant for digesting and absorbing

fat (Strand, 1978). Long before cholesterol is stored as fat, it is broken down and used for the removal of fat. Beginning in the liver, this process also utilizes the small intestine. The small intestine is where much of fat absorption occurs. Within the intestine, cholesterol can be absorbed when mixed with fat, bile salts, and a cholesterol esterase (found in pancreatic juice). The esterase hydrolyzes cholesterol esters found in the abdominal area. The cholesterol is then released to be absorbed along with other lipids in the body (Jensen, 1976). This concludes the basic functions of cholesterol. There are, however, two other elements that combine with cholesterol in the blood to assist the body with further overall functioning.

Additional elements associated with cholesterol are LDL and HDL. The two lipoproteins are responsible for transporting cholesterol. LDL carry cholesterol and lipids *toward* tissues. HDL move cholesterol *away* from tissues and cells. HDL also carry cholesterol to the liver for removal (Sizer & Whitney, 2006). Recall the possible disastrous effect of cholesterol upon the arteries. How can the element that appears to repair arteries can also be the cause of their destruction? Roizen and Oz (2006) refer to it as the domino effect of fat, a process requiring many infractions to occur. An accumulation of LDL piles up forming a plaque, and eventually makes its way into the middle layers of an artery or arteries. Due to this interruption to the normal anatomy of an artery, the environment responds as if there is an injury, stimulating the protection of the body's immune system. White blood cells surface to soothe and smooth out the festering cholesterol.

During the process, the white blood cells release other toxicities which ordinarily break down infection; but here, cause inflammation in arterial walls. This is the

beginning of a very hazardous route to problematic heart conditions. Continuing to defend and attempting to protect the body, the immune system consistently sends agents to remove the toxins and cholesterol. The unfortunate side effect is an even larger inflammation, and rough surface on the arterial wall. As the body continues the normal healing process, platelets are attracted to the site and form clots. White blood cells and platelets pile up rapidly, expanding the inflammation and clotting. The filled artery cannot allow blood to pass through; therefore, the blood supply to the heart is eliminated (Roizen & Oz, 2006). The final result is a heart attack. However, this is not the natural design of the body. What went wrong? The problem lies between the levels of LDL and HDL.

The purpose and functions of both low density lipoproteins and high density lipoproteins are not bad. In fact, if the body can digest and synthesize healthy amounts of each, it will remain in a comfortable state of homeostasis. Keeping this balance is crucial. The purpose of LDL is to bind to and transport cholesterol to the tissues. As explicated by the domino theory of fat, an abundance of LDL cholesterol begins building plaque in the arteries of a tissue. At this point, HDL purposes to rescue the suffering tissue, by carrying away the extra cholesterol for excretion by the liver.

Sizer & Whitney (2006) assess the difference between the two lipoproteins is the route in which they transport cholesterol, and the magnitude of lipids being transported. Since LDL are responsible for transporting cholesterol toward tissues and can foster the build up leading to heart attacks, LDL is often coined “lousy” cholesterol. Yet, there is no difference in the type of cholesterol transported by LDL and HDL. Both LDL and HDL carry the same cholesterol, it is simply a matter of what is done with the cholesterol, or

where it is taken. It is important to note that cholesterol by itself is not bad. The problem associated with cholesterol is low levels of HDL and high levels of LDL (Roizen & Oz, 2006). When LDL and HDL cholesterol are in proper proportion work together, the body remains balanced.

It would appear the key to maintaining a balanced cholesterol level would solve all previously mentioned problems. To do this, it must determined how to keep LDL and HDL levels in balance. Lowering the intake of dietary cholesterol is not sufficient. Dietary cholesterol is positively related blood cholesterol; however, the relationship is complex (Hafen, 1981). A blood test is used to measure the levels of both LDL and HDL cholesterol in the body. Conversely, there is a vast difference between dietary cholesterol and blood cholesterol. Research identifies elevated levels of blood cholesterol as a risk factor for both atherosclerosis and coronary heart disease. Additional evidence distinguishes the role of specific fats on blood cholesterol levels.

Hafen (1981) studies indicate saturated fats increase blood cholesterol, and polyunsaturated fats can decrease the level of blood cholesterol. It is necessary to repeat dietary cholesterol is not the prime culprit in raising the level of blood cholesterol. Rather, saturated fat and trans fat are the core dietary factors linked to the elevation of blood cholesterol (Sizer & Whitney, 2006). Furthermore, Sizer and Whitney (2006) assert cholesterol itself is produced by saturated fat found in the diet. The greatest influence in maintaining balanced cholesterol levels is by monitoring saturated and trans fat intake.

Previously, the concern over piling up of LDL cholesterol was mentioned. The result of the body naturally clearing out the excess LDL leads to piles of white blood cells and platelets. The result of this hostile environment is called inflammation. Similar to

what occurs in arteries, Roizen and Oz (2006) report inflammation can also arise in the small intestine and omentum. The bloodstream undergoes the chemical reaction producing inflammation. A malfunctioning small intestine and expanding omentum further complicate inflammation, and often become the primary cause for weight gain. The inflammation in the body occurs almost entirely because of poor food choices, most of which are linked to the way the body responds to food allergies, saturated and trans fats, and toxins like stress, cigarettes, and alcohol. Roizen and Oz (2006) have discovered how initial inflammation responds by causing hypertension and insulin resistance. Such responses contribute to the inflammation of arteries throughout the body; thus, leading to heart disease. Sizer and Whitney (2006) report visceral fat, which surrounds vital organs within the abdominal region, is released readily into the bloodstream. The release of this additional fat may contribute to the burden of LDL, also increasing the risk of heart disease. The alarming effect of inflammation is the ability for fat and toxins to continue to enter the bloodstream. Clearly, inflammation can be problematic; and several additional consequences follow.

While many systems throughout the human physiology revolve around a cycle, so does the process of inflammation. The brain plays an integral role in total body functioning, assume its presence. To ensure the proper functioning of the brain, it must be nourished. The brain feeds off of glucose in the body; inflammation prohibits glucose from getting to the body's cells. As a result, the body desires more glucose. A human will consume more sugary foods to meet this need, and also increase inflammation; the cycle continues (Roizen & Oz, 2006). The only way to stop the cycle is to prevent the inflammation.

It can be hypothesized to best decrease the amount of inflammation in the body, simply monitor the blood cholesterol levels in the body. While heredity is an influential component of the blood cholesterol level, physical activity and diet are more significant. The level of physical activity, saturated and trans fat intakes, and the overall amount of calories consumed by the human body dictate the level of blood cholesterol (Roizen & Oz, 2006). To reemphasize, this has little to do with dietary cholesterol, and everything to do with saturated and trans fats. As the body begins losing fat, Roizen and Oz (2006) suggest blood-related numbers in the body, such as cholesterol and blood pressure, will return to healthful norms. The final result is a decrease in the body's inflammation, and also in waist size.

The relationship between cholesterol and waist size indicates a direct correlation. In order to understand this perplexing relationship, the basics about fat and its components need be addressed. From the functions to the digestive route of fat, much is learned. It can also be compared to cholesterol. While all cholesterol in the body is the same, it is the varying functions of cholesterol that remain to be seen. The difference between dietary cholesterol and the blood cholesterol level accounts for an excessive accumulation of fat deposits in the omentum, located in the abdominal region (Roizen & Oz, 2006). All of these factors combine to impact the size of the human body waist.

Excess fat accumulations can also have substantial effects on blood pressure. Blood pressure refers to the amount of force exerted on blood vessels and arteries to transport blood throughout the body. Normal blood pressure varies by activity and age. DeBakey and Gotto (1977) denote slight variations can also occur during changes in tension, exercise, movement, injury, and pain of an individual. Blood pressure can also

fluctuate with age. An older individual may necessitate higher normal blood pressure, as the heart must work harder as it ages.

Blood pressure is determined by measuring the amount of pressure during two phases of the heart, contraction and relaxation. DeBakey and Gotto (1977) correlate these phases of the heart with two phases of blood pressure, systole and diastole. Systolic blood pressure is related to the contraction phase of the heart; greater pressure is required due to changes in activity, stress, and other previously mentioned factors. Systolic blood pressure is the higher number. The lower number is diastolic blood pressure. The relaxation phase of the heart is associated with diastolic blood pressure; less pressure is required. The lower pressure is necessary when the heart is relaxed, or not beating; otherwise, the blood would retract to the heart (DeBakey & Gotto, 1977).

Blood pressure is measured in millimeters of mercury. A sphygmomanometer is the common device used to determine blood pressure (DeBakey & Gotto, 1977). One form includes an inflatable cuff which is placed around the arm. To constrict the brachial artery, an inflatable bulb is squeezed. As the bulb is squeezed, the pressure inside the cuff rises, and an examiner listens with a stethoscope below the cuff on the arm. Blood is able to flow through the vessel while the pressure in the cuff is lower than pressure in the artery; the sound of the blood flow is heard in the stethoscope. The cuff is pumped enough to stop the circulation of blood, and the sound stops. As air is gradually released from the cuff, arterial pressure is reduced. The sound of blood flow resumes; diastolic pressure is measured. Average ranges for systole are 110-140. During diastole, ranges fall to 70-90 (DeBakey & Gotto, 1977).

Hypertension is the medical term for high blood pressure. An elevation of normal blood pressure is caused by the narrowing of small blood vessels. To get blood through the vessels, the heart must work harder; thus, the pressure must rise (D'Alonza, 1961). DeBakey and Gotto (1977) suggest a standard definition of high blood pressure is systolic over 150 millimeters of mercury and diastolic over 90 millimeters.

High blood pressure causes strain on the body; thus weakening the heart and blood vessels. Void of many noticeable symptoms, hypertension is referred to as a silent killer. Hypertension may damage tissues because the transportation of oxygen is prohibited (D'Alonza, 1961). Vital organs and other areas in the body can also be affected. An enlarged heart is one of the major results of consistent high blood pressure. The broadest effect of hypertension is the development of atherosclerosis, a disease damaging arterial walls (Marx & Kolata, 1978).

There are several patterns of atherosclerosis, including those of which the disease is widespread among arteries throughout the body, or confined to regions of arteries in the brain, heart, or kidneys. In some instances, collateral circulation occurs to compensate obstructed blood flow. Small arteries from above and below an obstruction combine to provide smoother blood flow. As a result, circulation is provided to allow some function, but not enough is provided for normal function (DeBakey & Gotto, 1977).

Blood pressure is dependent upon cardiac output and the resistance of blood flow through arteries (DeBakey & Gotto, 1977). Ross and O'Rourke (1976) conclude variations in blood pressure over extended periods of time are primarily due to high resistance to blood flow. Atherosclerosis results from increased LDL levels, and the plaque that builds on arterial walls, hence blood flow is restricted. The resistance to

proper blood flow thereby increases blood pressure to keep blood moving through the vessels. Thus, blood cholesterol and blood pressure are conversely linked.

Obesity aggravates blood pressure and the level of blood cholesterol, two of the three primary risk factors present in developing cardiovascular disease. DeBakey and Gotto (1977) further suggest obesity is such a factor in raising blood pressure that changes in diet are the beginning of hypertension treatment. Being overweight also increases the strain on the heart. A slim, youthful waist line will decrease excess fat, thus limiting increased levels of blood cholesterol and blood pressure.

Methods

Subjects

A sample of 42 undergraduate students (30 female and 12 men, *M* age = 21.6 years, *SD* = 1.3), was used. As a required assignment of an upper level kinesiology class at Liberty University, college students completed a cholesterol blood test (Horton, 2007). The investigator visited the class to recruit students to volunteer the release of their cholesterol and blood pressure data. Students gave verbal consent to participate in the study.

Measurements

Instrument. Descriptive information was collected on a seven item questionnaire (see Appendix). Questions were directed at the subject's personal information (age, gender), cholesterol levels, and blood pressure. Waist size was also documented on this questionnaire.

Waist measure. Subjects were measured around the waist with the use of a standard sewing measurement tape. Measurements were taken at the naval and one inch above the outer iliac border. Subjects were to stand with a relaxed posture. Sucking in was not permitted. Waist size was measured in inches for each subject. Female subjects were measured by the investigator, and male subjects were measured by Dr. David Horton, professor of Kinesiology at Liberty University.

Cholesterol measurement. A fasting blood test was used to determine total, HDL, and LDL cholesterol levels. Subjects fasted for a minimum of twelve hours prior to the test. They did not eat the night before or morning of the blood test. Subjects arrived to class at 7:50 a.m., and reported as a group to Light Medical: Liberty University Health

Services. Dr. Richard A. Lane and registered nurses on staff drew blood for each individual. A coronary risk panel was used for the drawn blood. The samples were sent to an outside laboratory for testing.

Blood pressure. Subjects were instructed to have their blood pressure taken by visiting a physician's office or using a self-use instrument. If subjects chose a self-use instrument, they were instructed to use the instrument three times. The average of the three scores was to be recorded.

Results

Descriptive

Forty-two subjects participated in the study. A seven item questionnaire was used to collect descriptive information. Descriptive data for the subjects is provided in Table 1.

Table 1

Means for Descriptive Data

	Overall		Male		Female	
	M	*SD*	*M*	*SD*	*M*	*SD*
Age	21.64	1.3	21.67	1.70	21.69	1.16
Height	67.32	3.43	70.64	2.34	66.1	2.94
Waist	32.17	3.66	32.73	3.45	31.95	3.78
HDL	58.95	25.79	49.75	20.04	62.63	27.19
LDL	94.43	27.59	86.75	18.96	97.5	30.09
Systolic BP	113.92	10.67	117.09	5.42	112.63	12.10
Diastolic BP	70.26	20.9	69.55	16.35	70.56	22.29

Multivariate Analysis of Variance

A one-way multivariate analysis of variance (MANOVA) was used to determine differences among the four groups for the four dependent variables. Subjects were divided into three groups based on waist size: small, medium, and large. Female subjects classified as having a small waist size were those with waist sizes below 29.25 inches.

Male subjects classified as having a small waist were those with waist sizes below 30.5 inches. Female subjects classified as having a medium waist were those with waist sizes between 29.5 and 34 inches. Male subjects classified as having a medium waist were those with waist sizes between 31.5 and 33 inches. Female subjects classified as having a large waist were those with waist sizes above 35 inches. Male subjects classified as having a large waist were those with waist sizes above 33.5 inches. Based on these groups the hypotheses were tested.

The global F-test was significant, Pillai's Trace = .032, $F(8, 66) = 2.276$, p .032. Follow up univariate analyses were conducted and results are given by hypothesis number.

Hypothesis I. A single factor ANOVA was used to determine differences in HDL among the three groups. There was no significant difference in Waist Size x HDL, $F(2, 38) = 1.21$, $p = .31$.

Hypothesis II. A single factor ANOVA was used to determine differences in LDL among the three groups. There was no significant difference in Waist Size x LDL, $F(2, 38) = .38$, $p = .68$.

Hypothesis III. A single factor ANOVA was used to determine differences in systolic blood pressure among the three groups. A significant difference was observed among the groups with regard to systolic blood pressure, $F(2, 35) = 7.73$, $p = .002$. Subsequent post hoc analysis using Duncan's test indicated that systolic blood pressure was lower for the small waist group when compared to the medium and large waist groups.

Hypothesis IV. A single factor ANOVA was used to determine differences in diastolic blood pressure among the three groups. A significant difference was observed among the

groups with regard to diastolic blood pressure, $F(2, 35) = 7.40$, $p = .002$. Subsequent post hoc analysis using Duncan's test indicated that diastolic blood pressure was lower for the small waist group when compared to the medium and large waist groups.

Table 2

Means and Standard Deviations for Small, Medium, Large Waist Groups

	Small Waist		Medium Waist		Large Waist	
	M	*SD*	*M*	*SD*	*M*	*SD*
HDL	57.93	15.9	66.64	40.3	51.15	9.61
LDL	98.71	25.4	92.5	30.7	89.54	27.2
Systolic BP	107	11.0	119.38	9.60	118.82	5.17
Diastolic BP	64.57	7.27	74.15	8.72	73.82	5.09

Table 2 contains the means and standard deviations of the four variables for each of the waist size groups. As hypothesized, the small waist group recorded lower systolic and diastolic blood pressure when compared with the medium and large waist groups.

Discussion

The results of this study do not support waist size as an indicator of LDL or HDL cholesterol levels among college students. Roizen and Oz (2006) suggested that adults with a smaller waist size have lower blood cholesterol. The hypothesis was not confirmed by the college student subjects in this study. This may be explained by aspects that may or may not be under the subjects' control. Each subject was in control of his own diet and exercise routine. A diet low in saturated and trans fat along with a steady exercise routine reduces elevations in blood cholesterol. Heredity, on the other hand, directs its own course. Although subjects may not have control of heredity, Roizen and Oz (2006) assert diet and exercise contribute much more in managing blood cholesterol.

One additional factor might contribute to the lack of significant correlations between waist size and LDL and HDL levels. Due to the financial expenses of completing a coronary risk panel, the investigator was limited by the number of subjects. Thus, the study may be limited by pooling only upper-class kinesiology majors. Since a small pool of subjects (n=42) participated, the method to divide groups by waist size was driven by data. A greater supply of subjects would allow criteria for small, medium, and large waist size groups to be previously established. However, this did not retract from the purpose of the study, to investigate a correction between waist size, blood cholesterol, and blood pressure.

The findings do indicate a strong relationship between waist size and blood pressure among college students. Again, the controlled and uncontrollable factors of diet, exercise, and heredity are important here. Furthermore, the first step doctors prescribe to reduce blood pressure is to lose weight. The evidence of losing weight is a smaller waist

size. Whether the result of lost weight or maintenance of an ideal weight, this study supports a smaller waist size is related to lower blood pressure among college students.

Further research should be directed at more subjects, non-kinesiology students, older subjects (ages 25-35), monitoring diet and exercise routines, and screening heredity factors. Continued examination of this population is necessary to provide comprehensive data of the relationship between waist size, blood cholesterol, and blood pressure.

References

American Obesity Association. (2002). *Development of the weight wellness profile.* Retrieved January 24, 2007, from American Obesity Association Web site: http://www.obesity.org/subs/News/Vol2_No2_cover.shtml

Appel, L.J., Moore, T.J., Obarzanek, E., Vollmer, W.M., Svetkey, L.P., Sacks, F.M., et al. (1997). A clinical trial of the effects of dietary patterns on blood pressure. *The New England Journal of Medicine.* 336:1117-1124.

Bodanis, D. (1984). *The body book: a fantastic voyage to the world within*. Boston: Little, Brown and Company.

Bragg, P.C. (1975). *How to keep your heart healthy and fit.* Desert Hot Springs, CA: Health Science.

D'Alonza, C.A. (1961). *Heart disease, blood pressure, and strokes*. Houston: Gulf Publishing Company.

DeBakey, M. & Gotto, A. (1977). *The living heart*. New York: David McKay Company.

Hales, D. (2005) *An invitation to health* (4th ed.) Belmont, CA: Thomson & Wadsworth Publishing.

Hafen, B.Q. (1981). *Nutrition, food, and weight control* (Expanded ed.). Boston: Allyn and Bacon.

Jacob, S.W. & Francone C.A. (1976). *Elements of anatomy and physiology*. Philadelphia: W.B. Saunders Company.

Jensen, D. (1976). *The principles of physiology*. New York: Appleton-Century-Crofts.

Macfarlane, R.G. & Robb-Smith, A.H.T. (1961). *Functions of the blood*. New York: Academic Press.

Marx, J.L. & Kolata, G.B. (1978). *Combating the #1 killer: the science report on heart research.* Washington D.C.: American Association for the Advancement of Science.

Mason, S.A. (1960). *Health and hormones*. Baltimore, MD: Penguin Books.

McMurray, W.C. (1983). *Essentials of human metabolism: the relationship of biochemistry to human physiology and disease* (2nd ed.). Philadelphia: Harper and Row, Publishers.

National Center for Health Statistics. (2007). *Prevalence of overweight and obesity among adults: United States, 2003-2004*. Retrieved January 24, 2007, from http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overweight/overwght_adult_03.htm

Roizen, M.F. & Oz, M.C. (2006). *You on a diet: the owner's manual for waist management*. New York: Free Press.

Singer, S. & Hilgard, H.R. (1978). *The biology of people*. San Fransisco: W.H. Freeman and Company.

Sizer, F. & Whitney, E. (2006). *Nutrition concepts and controversies* (10th ed.). Belmont, CA: Thomson Wadsworth.

Strand, F.L. (1978). *Physiology: a regulatory systems approach*. New York: Macmillan Publishing.

Stearns, P.N. (2002). *Fat history: bodies and beauty in the modern west*. New York: New York University Press.

Turner, J.D., Le, N.A., & Brown. W.V. Effect of changing dietary fat saturation on low-density lipoprotein metabolism in man [Electronic version]. *American Journal of Physiol: Endocrinology & Metabolism*. 241.

Vander, A.J. (1976). *Human physiology and the environment in health and disease*. San Francisco: W.H. Freeman and Company.

White, A., Handler, P. & Smith, E.L. (1964). *Principles of biochemistry* (3rd ed.). New York: McGraw-Hill Book Company.

Wyatt, S., Winters, K. & Dubbert P. Overweight and obesity prevalence, consequences, and causes of a growing public health problem [Electronic version]. *The American Journal of the Medical Sciences*, 331(4).

Appendix

Data Collection Form

Gender: M F
Age:
Height:
Waist:
HDL:
LDL:
Blood Pressure: