Association of aerobic capacity with selected CHD risk factors in males ages 16-18 years

Kerstin A. Stoedefalke

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ASSOCIATION OF AEROBIC CAPACITY WITH SELECTED
CHD RISK FACTORS IN MALES AGES 16-18 YEARS

Kerstin A. Stoedefalke
B.S. Penn State University, 1983
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Master of Science
University of Montana
1985

Approved by:

[Signatures]
Chairman, Board of Examiners
Dean, Graduate School

3/12/85
Date
The purpose of this study was to detect whether total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL), body weight, and percent body fat were different between the aerobically fit and aerobically less fit groups in males ages 16-18 years. A second purpose of this study was to determine the relationship of aerobic fitness to TC levels, HDL levels, LDL levels, body weight and percent body fat. A final purpose was to detect which factors correlated to the greatest amount of variation in VO₂ max (ml/kg.min), HDL and LDL among the subjects tested. To obtain a VO₂ max, each subject did a maximal graded exercise test. Performance in the test along with the subject's current activity level were used as the criteria in placing the subject in either the aerobically fit or aerobically less fit group. Percent body fat of each subject was estimated from their individual weights under water and their residual volume. Blood samples were drawn from the subjects and analyzed for TC, HDL and LDL levels at the Western Montana Clinic. Correlation coefficients were computed to assess associations. Stepwise multiple regression equations were used to predict variation among selected variables.

The results of this study showed a significant (p<0.05) difference between the aerobically fit group and aerobically less fit group in percent body fat, VO₂ max, TC level, LDL level, TC:HDL ratio and the LDL:HDL ratio. VO₂ max was significantly correlated to HDL percent body fat, body weight, TC:HDL ratio and the LDL:HDL ratio. The factor correlated with the greatest variation in VO₂ max was percent body fat followed by LDL level. Body weight was the only factor to significantly correlate to the variation in HDL while TC was the only factor to significantly correlate to variation in the LDL level.
ACKNOWLEDGEMENTS

The author wishes to thank Dr. Brian Sharkey for his guidance during this study. The author is also indebted to Charlie Ward at the Western Montana Clinic for his assistance in the subjects' blood sampling and analysis.

A special thanks to my husband, Joe, and my family for their constant support and encouragement.
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CHAPTER 1

Introduction

Cardiovascular heart disease (CHD), which accounts for more than 600,000 deaths a year, is the leading cause of death in the United States (5). Risk factors for CHD in the adult population established by the American Heart Association are: high blood pressure, increased total cholesterol (TC), increased low density lipoprotein (LDL) levels, lowered high density lipoprotein (HDL) levels, cigarette smoking, elevated uric acid levels, inactivity, excessive emotional stress, obesity and heredity.

The probability of manifestation of CHD is increased exponentially with additive numbers of risk factors (11, 14). In the past, research on CHD and CHD risk factors has been aimed at the adult population with the primary focus being middle-aged men. The population for which CHD risk factors have not been established is young adults under the age of eighteen years. Researchers have hypothesized that CHD begins in early childhood. In order to catch the disease at its earliest stages, scientists need to establish risk factors for the pediatric population. Crittenden supported childhood screening of CHD risk because:

the genesis of the atherosclerotic lesion appears to begin in childhood. Efforts to prevent this process should therefore be directed at children since substantial and potentially irreversible damage may already exist by the third or fourth decade of life before any symptoms have occurred (11).

It was the purpose of this investigation to study the relationship of serum cholesterol, HDL, LDL and percent body fat to the aerobic fitness of males between the ages of 16-18 years.
Exercise

Exercise has been postulated in the literature for years as an effective mode of decreasing CHD risk factors. One method of evaluating how the body has responded to regular exercise is to examine the individual’s aerobic fitness level. A common measure of aerobic fitness is VO\textsubscript{max} in ml/kg.min. The correlation between aerobic fitness, expressed as VO\textsubscript{max}, and decreased CHD risk factors is significantly high (23,25). In an investigation by Hartung, et al., eighteen coronary patients showed a beneficial increase in serum HDL with an increase in VO\textsubscript{max} as a result of exercise (13,20). Similarly, Cooper, et al., found a decrease in TC, LDL, and percent body fat and a beneficial increase in HDL which was positively correlated to an increase in VO\textsubscript{max} following an exercise training program (10). In the preceding experiments, an increase in the activity level of the subject resulted in an increased aerobic fitness level or VO\textsubscript{max}. There is little research which has tried to use the VO\textsubscript{max} of adolescents, without a training regime, as a reflection of blood lipid levels.

HDL

Exercise tends to have the greatest impact on raising HDL levels. HDL is a carrier lipoprotein of cholesterol. Although the role of HDL is not thoroughly understood, evidence suggests HDL carries cholesterol from the arterial wall to the liver for catabolism and excretion (40). Epidemiological data indicates that
individuals with increased levels of HDL have a decreased incidence of CHD (7). Some researchers believe HDL levels to be the most powerful indicator of risk for CHD (2,17,39). The lower the serum HDL level, the greater the severity of CHD lesions (50). Although there is disagreement in the literature as to whether diet or exercise is more effective in lowering serum TC levels, researchers tend to agree exercise is more effective than diet in raising HDL levels (20,24). Hartung, et al., suggested that a high correlation between HDL and VO$_2$ max indicated the possibility of a direct fitness related mechanism in elevation of HDL levels (7). Exercise also appears to decrease serum LDL levels.

**LDL**

As mentioned earlier, an increased level of LDL is considered a risk factor for CHD. LDL is a carrier lipoprotein for cholesterol. Unlike HDL, LDL carries cholesterol to the atherosclerotic process (63). Exercise and a corresponding increase in aerobic fitness has been positively correlated to lower serum LDL levels (36). Conversely, caloric restriction has not been characteristic of lower LDL levels (62). Simpson, however, found that obesity was inversely related to HDL levels. He suggested fatness may contribute independently to the lowering of HDL levels beyond the effect of activity and dietary saturated fat in the subjects (56).
Obesity

Although obesity is considered a risk factor for CHD, obesity is not always indicative of increased serum TC levels. Weltman, et al., noticed no decrease in serum LDL in subjects who had lost weight due entirely to caloric restriction. He did notice a decrease in HDL in the caloric restricted subjects (62). A similar response to caloric restriction and body composition change was recognized by other researchers (12,66).

The previously mentioned CHD risk factors have been clearly established and studied in the adult population. Adults who have low fat diets, low percent body fat, decreased TC levels, increased HDL levels and regular exercise have a lower risk of CHD (10). Currently, the literature has very little information on CHD related risk factors in young adults 18 years and younger. There is even less research on the effect exercise or diet intervention has on the possible risk factors for CHD in the young population. Several articles have postulated the need for CHD related research on children, but few studies have been reported (10,37,46,49,51).

The proposed research examined 16-18 year old males from the Missoula Area School District and three subjects from Browning, Montana. Aerobic fitness was correlated with percent body fat, serum TC, HDL and LDL levels to determine whether the relationships in young adults are similar to those in the adult population.
The American Heart Association and related health organizations are focusing attention on prevention and intervention in the CHD disease process. Because of the age related increase in LDL and TC levels and decrease in HDL levels, the medical profession should research the feasibility of early intervention in young adults. If scientific procedures can identify CHD risk factors in young adults, there is a high probability that the disease process can be interrupted in successive generations before the disease becomes a serious problem for the individual.

**Purpose of the Study**

The purpose of this study was to detect whether TC, HDL, LDL, body weight and percent body fat are different between the aerobically fit and aerobically less fit groups in males ages 16-18 years. A second purpose of this study was to determine the relationship of aerobic fitness to TC levels, HDL levels, LDL levels, weight and percent body fat. A third purpose was to detect which factors correlated best with the greatest amount of variability in VO max (ml/kg.min), HDL and LDL among the subjects tested.

**Limitations of the Study**

The number of subjects was limited to 18 male volunteers, ages 16-18 years, from the Missoula Area School District and three subjects from Browning, Montana. Because of the use of volunteers
and thus a lack of random sampling, the results of this investigation may relate to only these subjects and not to the universe.

Definitions

Aerobic fitness—the ability of an individual to take in, transport and utilize oxygen. In this study, VO\text{max} in \text{ml/kg.min}^{2} was used to assess the subjects' aerobic fitness level.

Aerobically fit youth—youth who have a VO\text{max} at or above 60 \text{ml/kg.min} and are currently exercising and utilizing 3000 kcal or more a week.

Aerobically less fit youth—youth who have a VO\text{max} below 60 \text{ml/kg.min} and are not currently involved in a regular exercise program.

High density lipoproteins (HDL)—carrier proteins which transport cholesterol to the liver for catabolism.

Low density lipoproteins (LDL)—carrier proteins which transport cholesterol to peripheral cells for deposition.

Percent body fat—the fraction of the total body weight that is fat.

Total serum cholesterol (TC)—the sum of all the lipoprotein fractions.
CHAPTER 2

Review of Related Literature

Cardiovascular heart disease (CHD) is the leading cause of death in America. By the age of sixty, one-third of the American male population will suffer from CHD. To combat the disabling conditions of CHD, the medical profession has identified several CHD risk factors in the adult population. Such risk factors include: elevated TC levels, elevated lipoprotein levels (LDL and VLDL), low HDL levels, elevated triglyceride levels, obesity and hypertension.

In studies dealing with postmortem examinations of servicemen killed in combat, 45-75% of the men showed aortic fibrous plaques by the second or third decade of life (37). Atherosclerosis leading to CHD is now believed to begin early in life (6,9,31,38,39). Few extensive studies have been conducted to show whether these same CHD risk factors appear in the pediatric population ages eighteen years and younger.

Total Cholesterol

Increased total cholesterol levels have a high correlation with the incidence of CHD (10,11,37,49). As TC levels increase, the risk for CHD also increases (10,27,29). Excess cholesterol in the blood can result in cholesterol deposition in the arteries which forms fatty streaks. If cholesterol continues to be deposited in the arterial walls, the fatty streaks can eventually form the fibrous plaques of atherosclerosis.

Lauer, et al., conducted a fourteen month study on 4,329 school children ages 6-18 years (31). They found the mean TC levels of all
ages and sexes in the study to be 182 mg/dl + 29. In adults, the American Heart Association has established a TC level of 220 mg/dl or greater as being a CHD risk factor. By extrapolating from the adult CHD risk factor of 220 mg/dl, Lauer, et al., concluded a TC of 180 mg/dl or greater required therapeutic intervention. It was concluded fifty percent of the subjects studied were already at risk for future development of CHD (31).

Thorland and Gilliam conducted a study on cholesterol levels in high and low active pre-adolescent males. Fifty-five subjects were evaluated. The higher active group had a mean TC level of 148.8 + 3 mg/dl and the lower active group had a mean TC of 155.3 + 5 mg/dl (60). Interestingly, the lower active group's diet consisted of less cholesterol (3.112 mg/dl) than the higher active group (324.0 mg/dl). Thorland and Gilliam attributed the difference in dietary cholesterol to the greater caloric demand in the bodies of active boys as compared to the inactive boys.

Other studies have shown TC does not significantly correlate with the level of activity of an individual (7, 33, 46, 55). Nataro found no change in the TC of 16 adolescent boys as a result of an exercise training program (46). Similarly, Montoye, et al., reviewed thirteen studies and found no significant correlation between TC and activity level once body fatness was removed from the analysis (43). To further examine the role cholesterol plays in the atherosclerotic process, researchers have looked towards the individual blood lipid fractions as possible indicators of CHD.
**Blood Lipoproteins**

Lipoproteins are carrier molecules which transport cholesterol between the intestine, adipose tissue, the liver and other target organs. The four major lipoproteins are chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL). The concentration of cholesterol is the largest in LDL (see Figure 1). LDL carries cholesterol to the arterial wall for deposition and thus is believed to contribute to the atherosclerotic process (63). Conversely, HDL is believed to carry cholesterol from peripheral cells to the liver for metabolism and excretion (63). The key enzyme in the catabolism of VLDL and LDL is thought to be lipoprotein lipase (LPL) (26,47,58).

LPL is located in both adipose and skeletal muscle as well as other body tissues. The concentration of LPL is greater in adipose tissue of women than of men. Conversely there are no differences in the concentration of LPL in skeletal muscle tissue between males and females. When the catabolic rate of chylomicrons and VLDL is high, HDL level is increased. Nikkila, et al., found plasma HDL levels and LPL activity correlated in adipose tissue \((r=0.66, \ p \ 0.001)\) but found no correlation between HDL and skeletal muscle LPL (47). Nikkila suggested exercise may induce insulin sensitivity in the tissues, which would enhance LPL activity and result in increased HDL levels (47).
FIGURE 1

The Approximate Composition of the Major Lipoproteins (2)
Epidemiological data indicates populations with increased levels of HDL have a decreased incidence of CHD (8,53). Low HDL levels have been positively related to increased severity of CHD lesions (3,7,18,28,50). Active individuals have higher HDL levels than inactive people and women have higher HDL levels than men (47). Nikkila, et al., suggested the greater LPL concentration in adipose tissue of women may account for the known sex difference in HDL levels (47).

Miller reported that the size of the total pool of body cholesterol was unrelated to the mean plasma concentrations of LDL, VLDL, HDL or triglycerides (41). Pool size of LDL, however, showed a strong negative correlation with the plasma level of HDL. Some researchers have also suggested recently that the ratio of TC to HDL and the ratio of LDL to HDL may be more important determinants of the risk for developing CHD than TC, LDL or HDL are independently (1,12).

Effect of Exercise on Blood Lipid Levels

Several investigators have studied the effect of exercise on risk factors associated with CHD (5,10,13,20,28,45,50). Paffenbarger and Hale, in an investigation of 6,351 long shoremen, found that workers classified in a high-caloric-output-job task had significantly lower death rates from CHD (48). Morris, et al., who studied the leisure-time habits of 16,000 men ages 40-64 years concluded vigorous exercise apparently protected them from CHD.
Cooper, et al., conducted a study of 3,000 men in an attempt to quantify the status of cardiorespiratory fitness and risk factor variables (10). Cooper, et al., supports the hypothesis that protection from CHD appears to be associated with a higher level of fitness. Simpson, in his study of 53 male forest service employees, found HDL to be significantly correlated with intense activity ($r=0.37$, $p<0.01$) (56). The body's use of more than 7 kcal/min is defined by Simpson as intense activity.

Numerous investigators have found a beneficial rise in HDL and decrease in LDL levels following an aerobic exercise training regime (26,35,45,59). Huttunen, et al., investigated the effects of exercise on the HDL and LDL levels of 110 men ages 40-45 years (26). One group participated in a four month exercise program and the other group was used as the control. Huttunen, et al., found an increase in HDL and decrease in LDL in the exercise group (see Figures 2, 3). Lopez, et al., found significant reductions in VLDL and LDL and increased HDL in a study involving 13 men in a seven week exercise training program (35). Hartung, et al., found a high post training correlation between HDL and VO$_2$ max in 18 coronary patients following an aerobic training program (19). Other researchers have found similar results in coronary patients as a result of exercise (13,23,57). Thorland and Gilliam studied the activity level of 55 adolescent males. They found the higher active group had a characteristically higher HDL level (48.4 mg/dl) than the lower active group (42.9 mg/dl) (60).
FIGURE 2. Fasting LDL concentration in the exercise and in the control group during the trial. The shaded area represents the time period before the beginning of the exercise intervention (26).

Ⅰ Ⅱ Ⅲ Ⅳ

TIME POINT

FIGURE 3. Fasting serum HDL concentration in the exercise and in the control group during the trial. The shaded area represents the time period before the beginning of the exercise intervention (26).

○ - exercise group

● - control group

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Although cross sectional studies do not provide a cause and effect relationship, exercise has been shown to be a positive correlate to an improved lipoprotein profile. Similar research needs to be done on individuals 18 years and younger. After all, Americans tend to be lower than other countries in physical fitness and at a higher risk for CHD (49).

**Effect of Diet and Body Weight on Blood Lipoprotein Levels**

Obese individuals (greater than 25% fat for men and 30% fat for women), tend to exhibit more CHD risk factors than their non-obese counterparts (4). Thompson, et al., found percent body fat to be inversely proportional to HDL levels (59). Several other studies have also shown a strong inverse correlation between HDL and percent body fat (15,16,21,35,42,52,61). Research on childhood obesity (greater than 20% fat) shows obese children have higher TC levels than their non-obese counterparts (30,31,60). Although obesity has not been proven to be a cause of CHD, obese individuals often suffer from CHD (5). Exercise and diet are two means of controlling obesity.

Dietary restriction has been used as a means of combating obesity. Dietary modification has also been considered a therapeutic means of improving the blood lipid profile. Modifications in diet such as decreased consumption of fat and cholesterol have been shown to lower total plasma cholesterol (15,61). Reports using only total serum cholesterol changes are
limited since they do not reveal changes in the lipoprotein cholesterol fractions (62).

Several studies have shown that diet is less important than the amount of exercise in determining HDL levels (21, 24, 59). Thompson, et al., studied the ten day dietary records of 20 male distance runners and 14 sedentary controls (59). The runners had significantly higher HDL levels (66±12 mg/dl) as compared to the sedentary group (46±10 mg/dl) despite the fact that the runners consumed 20 percent more calories including more fat than the sedentary group. Hartung, et al., studied the effect of diet to HDL levels in marathon runners, joggers and inactive men (21). There were no major dietary differences among the marathon runners, joggers and sedentary men: however, the marathon runners and joggers had higher HDL levels (20). Hartung also noted LDL levels were lower in the marathon runners, but LDL levels did not differ between the joggers and sedentary men. Weltman, et al., placed 58 sedentary men into four groups in order to study the relationship between caloric restriction, mild exercise and lipoprotein levels (62). For the ten week session, they placed the men into either a caloric restriction group (CR), mild exercise group (ME), mild exercise and caloric restriction group (CR+ME) and a control group. Their data shows ME and CR+ME as effective treatments for lipoprotein alteration (see Figure 4).
FIGURE 4

Total cholesterol and lipoprotein changes during the ten week experimental period (62).

- Pre-experimental measure
- CR (caloric restriction)
- ME (mild exercise)
- CR+ME
- CONTROL

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The literature indicates that there is conflicting information as to whether a low fat diet alone increases or decreases HDL levels. Hartung, et al., found no change in HDL levels following a low fat diet intervention program in his subjects after a ten week diet intervention period (21). Weltman, et al., found a decrease in HDL levels following caloric restriction in eight subjects tested (62). Other reports suggest weight loss must accompany dietary restriction for there to be an increase in HDL levels (23,20).

The effects of alcohol on HDL levels is also a controversial topic. Several investigators have found moderate amounts of alcohol elevate HDL levels (8,25,64). Others have found alcohol consumption does not modify HDL levels (4,16,53).

**Summary**

Increased TC levels, increased LDL levels, decreased HDL levels and obesity have all been identified as CHD risk factors. Although the effect of exercise on blood lipid levels remains unclear, exercise and a correspondingly increased aerobic fitness level has been shown to beneficially alter the lipoprotein fractions in the adult male population. Few studies have been done on the relationship of aerobic fitness and TC, HDL and LDL on the under 18 age group.

Khoury, et al., found obesity to be an indication of a lower HDL level when compared to the non-obese child (30). Thorland and Gilliam observed a similar phenomena among the more active
subjects of their study (60). The more active children had a mean HDL level of 46.4 mg/dl while the non-active subjects had a mean HDL value of 42.9 mg/dl. They also found obesity (20% or more in children) to be positively correlated to decreased HDL levels (60).

Research on the effects of alcohol on HDL remains unclear; however, most studies have shown alcohol consumption raises HDL levels in adults. Because of the possible legal implications, there is no research on the effects alcohol has on the HDL levels of minors.

Research indicates that exercise appears to be the most effective means of altering the blood lipid profile. Dietary modification alone does not appear to have as much of an impact on altering blood lipid levels as does exercise. Several researchers have found aerobic fitness to be the single best indicator of HDL levels and possibly LDL levels. Therefore, the present study was undertaken to determine if CHD characteristics, normally shown in an adult male population, were also observable in an adolescent population.
CHAPTER 3

Methodology

I investigated the relationship of aerobic fitness with selected CHD risk factors. Aerobically fit and aerobically less fit were the two subcategories of the aerobic fitness category. The subjects consisted of thirteen males ages 16-18 years. Eight of the subjects were from Missoula, Montana, and five subjects were from Browning, Montana.

The first two purposes of the study were to test the proposed hypothesis. The main hypothesis was that one or more of the variables of VO max, TC, HDL, LDL, percent body fat and body weight would be significantly different between the two groups. A second hypothesis was that VO max would be significantly correlated to HDL; and VO max would be inversely, but significantly correlated to TC, LDL, percent body fat and body weight. A third purpose of the study was to determine which variables correlated with the greatest amount of variation in VO max, HDL and LDL. The ratios of TC to HDL and LDL to HDL were also used in the statistical analysis of data. The ratios are hypothesized by Heath, et al., as being an important indication of coronary risk since the delivery, deposition and clearance of cholesterol from the tissues is modulated by these lipoproteins (23).

Subjects

Male volunteers (N=13), 16 to 18 years of age, participated in the study. Testing occurred from May 1984 to July 1984. The letter sent to the subjects prior to testing appears in Appendix B.
informed consent was signed by the parents of each subject prior to testing. The informed consent I read subject prior to testing. A sample informed consent appears in Appendix A. The subjects were placed into one of two groups.

The first group (A) consisted of eight subjects who were currently training to compete in distance running events for their high school track team. The second group (B) consisted of five subjects who were not currently involved in any type of structured or supervised training program. Demographic data of the subjects appears in Table 1 and Table 2.

<table>
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<td>Physical Characteristics of Group A (Trained)</td>
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<tr>
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<td>16</td>
<td>55.68</td>
</tr>
<tr>
<td>D.B.</td>
<td>17</td>
<td>59.09</td>
</tr>
<tr>
<td>P.M.</td>
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<td>64.10</td>
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<tr>
<td>G.C.</td>
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<td>M.P.</td>
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<td>69.55</td>
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<tr>
<td>D.T.</td>
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<td>69.55</td>
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<td>D.J.</td>
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<tr>
<td>A.K.</td>
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<td>Means</td>
<td>16.6</td>
<td>64.09</td>
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Table 2

Physical Characteristics of Group B
(Untrained)

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<tr>
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<td>60.45</td>
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<td>F.R.</td>
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<td>S.W.</td>
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<tr>
<td>E.G.</td>
<td>16</td>
<td>83.63</td>
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</table>

Means 16.4 70.50

Blood samples were drawn by a nurse at the Western Montana Clinic at eight a.m. on either May 16, 1984, or June 11, 1984. The subjects were instructed by the researcher not to eat anything after eight p.m. the night prior to the blood sampling. Vena punctures from the cubital fossa provided 5 cc. of blood for analysis at the Western Montana Clinic. TC, HDL and LDL were measured in the subjects' blood sample.

Body densitometry was obtained through the hydrostatic weighing procedure technique described by Siri (1956). A sample of the body composition worksheet appears in Appendix C. The protocol used in the underwater weighings was developed by the University of Montana Physiology Laboratory. A sample of the instructions given to the subjects prior to the underwater weight measurement appears in Appendix D.
Tests and Instrumentation

1. **Maximal Graded Exercise Test** VO max test results were used as an indication of the subjects' aerobic fitness level. The protocol used in the test was identical to the one used to evaluate the U.S. Jr. Ski Team members in the fall of 1984. The five minute warm-up period consisted of (1) walking for 2.5 minutes at a 4% grade and 3.5 mph and (2) running 2.5 minutes at 8 mph at a grade of 4%. If the subjects' heart rate exceeded 160 bpm, indicating more than 75% of their age predicted maximal heart rate, a walking protocol was used. If the subjects' heart rate at the conclusion of the warm-up period did not reach 160 bpm, a running protocol was administered.

The walking protocol was started at a 6% grade and 3.5 mph. The speed remained constant throughout the test, while the grade increased 2% every 2 minutes until the subjects indicated they could no longer continue.

The running protocol was started at a 2% grade and 8 mph. The speed remained constant throughout the test, while the grade increased 2% every 2 minutes until the subject could no longer continue.

**Quinton Treadmill** A motor driven treadmill was used as the exercise mode for the graded exercise test. Both speed and elevation were controlled by the experimenter. Each subject was indoctrinated to the treadmill prior to test initiation. The subject was shown, by the experimenter, the proper technique for getting onto a treadmill set at 3.5 mph. A thumbs up signal was used by the subject to indicate he was okay and a thumbs down signal...
was used by the subject to indicate test termination. The subjects were also informed they could stop the treadmill at any time by pushing a red button on the treadmill which was in the subjects' reach at all times.

Beckman Metabolic Measurement System was used to determine the subjects' expired O₂, CO₂ and the total volume of the expired air. From these measurements, VO₂ max was calculated by the Beckman computer. All VO₂ max values are accurate to ±0.10 ml/kg.min.

QM-11 is the oxygen analyzer used by the Beckman metabolic cart. After calibration with a known standard, the QM-11 is accurate to ±5% of full scale. Full scale is 1 to 100%.

Medical Gas Analyzer LB-2 is the CO₂ analyzer contained in the Beckman metabolic cart. After calibration with a known standard, the LB-2 is accurate to ±2% of full scale. Full scale is 1 to 10%.

Turbine meter is a volume meter contained in the Beckman metabolic cart. After calibration, the turbine meter is accurate to ±0.04 l/min.

Avionics Stress Test Monitor Model #2900B displayed a digital readout of the subjects' heart rate in beats per minute. The heart rate was used at each stage of the test as an indication to the experimenter, of how close the subject was to his age related maximal heart rate. Astrand has determined that there is a positive, linear relationship between heart rate and VO₂ max (86). The age related maximum heart rate is equal to 220 minus the age of the subject.
2. **Percent Body Fat** As mentioned previously, hydrostatic weighing was the technique used to determine the subjects' percent body fat. An autopsy scale was used to record the subjects' weight underwater.

   **Residual Volume** A dry gas spirometer was used to obtain the subjects' forced vital capacity. The mouthpiece on the spirometer was changed for each subject to insure sanitary conditions. The subjects were instructed to take one deep breath and to then expire all of their air into the spirometer through the mouthpiece. The subjects were given three trials before a final reading was made by the experimenter. A widely accepted age related correction factor of 0.25 was used to estimate the subject's residual volume from his forced vital capacity (65). All values obtained from the spirometer are accurate to ±0.10.

3. **Air Weight Scale** A Detecto-Medic balance scale was used to obtain the weight of the subjects prior to their underwater weight test. Each subject was weighed wearing only a swimsuit. The Detecto-Medic balance is accurate to ±0.50 kg.

4. **Blood Analysis** A 5 ml sample of blood was drawn from each subject the morning following a 12 hour fast. The blood sample was analyzed for TC, HDL and LDL content at the Western Montana Clinic.

   **The Isolab LDL-Direct** An American Monitor Micro KDA cholesterol audit system which analyzed all of the blood samples. The method of
analysis is ion selective chromatography. TC, HDL and LDL values obtained from the LDL-Direct are accurate to ±0.02 ml/dl.

5. Statistical Analysis of Data The University of Montana’s Dec 2065 computer was used to analyze all of the data. The SPSS system of analysis was the program used. A TI59 hand calculator was also used to confirm the means and standard deviations obtained through the Dec 20-65 computer. Data on each subject appears in Appendix E.

Means and standard deviations were calculated for each variable. Analysis of variance was utilized to study the differences among variables between the two groups. Pearson Product Moment Correlation Coefficients were computed to assess the association among the variables. The variables tested were: fitness level (VO\text{max} in ml/kg.min), HDL, LDL, TC, percent body fat, body weight, TC:HDL ratio, and LDL:ratio. The ratios of TC to HDL and LDL to HDL were included in the analysis because of their clinical use in CHD risk factor analysis. Stepwise multiple regression equations were computed using fitness level, HDL and LDL as the dependent variables. Independent variables used in the regression equations were selected based on the results from the Pearson Product Moment Correlations. To be included in the regression equation, a variable had to show a significant correlation with the criterion variable.

The 0.05 level was selected as the minimum level of significance for all statistical results.
CHAPTER 4
Analysis and Discussion of Data

Introduction

This chapter presents the data on the various tests performed: means and standard deviations of all variables sampled; Pearson Product Moment Correlations among all variables with special emphasis on the VOmax variable; analysis of variance between the 2 aerobically fit group and aerobically less fit group; and stepwise multiple regression equations with aerobic capacity, HDL and LDL as the dependent variables.

Sample Population Means and Standard Deviations

The population means and standard deviations appear in Table 3. Means and standard deviations for the aerobically fit group and the aerobically less fit group appear in Table 4 and Table 5 respectively. The population mean percent body fat is 2 to 3 percent lower than the age related norm of 16 percent body fat in males 16-18 years. Mean total serum cholesterol levels are all within the normal range. Subject E.G. was the only subject to reach the upper limits of the normal HDL and LDL level ranges.
Table 3
Subject Means and Standard Deviations (N=13)

<table>
<thead>
<tr>
<th></th>
<th>Means</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>16.60</td>
<td>0.80</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66.30</td>
<td>7.26</td>
</tr>
<tr>
<td>Percent body fat</td>
<td>10.63</td>
<td>3.99</td>
</tr>
<tr>
<td>VO\textsubscript{max} (ml/kg.min)</td>
<td>63.40</td>
<td>4.27</td>
</tr>
<tr>
<td>Total serum cholesterol (mg/dl)</td>
<td>128.01</td>
<td>24.34</td>
</tr>
<tr>
<td>High density lipoprotein (mg/dl)</td>
<td>42.26</td>
<td>8.18</td>
</tr>
<tr>
<td>Low density lipoprotein (mg/dl)</td>
<td>85.75</td>
<td>24.50</td>
</tr>
<tr>
<td>TC:HDL ratio</td>
<td>3.05</td>
<td>1.04</td>
</tr>
<tr>
<td>LDL:HDL ratio</td>
<td>2.10</td>
<td>0.99</td>
</tr>
</tbody>
</table>

Table 4
Aerobically Fit Means and Standard Deviations (N=8)

<table>
<thead>
<tr>
<th></th>
<th>Means</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>16.60</td>
<td>0.74</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>63.66</td>
<td>6.14</td>
</tr>
<tr>
<td>Percent body fat</td>
<td>8.91</td>
<td>3.34</td>
</tr>
<tr>
<td>VO\textsubscript{max} (ml/kg.min)</td>
<td>69.75</td>
<td>4.27</td>
</tr>
<tr>
<td>Total serum cholesterol (mg/dl)</td>
<td>116.10</td>
<td>15.92</td>
</tr>
<tr>
<td>High density lipoprotein (mg/dl)</td>
<td>44.55</td>
<td>6.60</td>
</tr>
<tr>
<td>Low density lipoprotein (mg/dl)</td>
<td>71.55</td>
<td>11.98</td>
</tr>
<tr>
<td>TC:HDL ratio</td>
<td>2.48</td>
<td>0.42</td>
</tr>
<tr>
<td>LDL:HDL ratio</td>
<td>1.56</td>
<td>0.30</td>
</tr>
</tbody>
</table>
Table 5

Aerobically Less Fit Means and Standard Deviations (N=5)

<table>
<thead>
<tr>
<th></th>
<th>Means</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>16.60</td>
<td>0.77</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.50</td>
<td>8.88</td>
</tr>
<tr>
<td>Percent body fat</td>
<td>13.40</td>
<td>3.61</td>
</tr>
<tr>
<td>VO$_2$ max (ml/kg/min)</td>
<td>53.30</td>
<td>9.35</td>
</tr>
<tr>
<td>Total serum cholesterol (mg/dl)</td>
<td>147.10</td>
<td>24.44</td>
</tr>
<tr>
<td>High density lipoprotein (mg/dl)</td>
<td>38.60</td>
<td>9.86</td>
</tr>
<tr>
<td>Low density lipoprotein (mg/dl)</td>
<td>108.50</td>
<td>22.38</td>
</tr>
<tr>
<td>TC:HDL ratio</td>
<td>3.96</td>
<td>1.13</td>
</tr>
<tr>
<td>LDL:HDL ratio</td>
<td>2.96</td>
<td>1.13</td>
</tr>
</tbody>
</table>

Aerobically Fit versus Aerobically Less Fit

The results of the analysis of variance for the difference among means obtained from the aerobically fit and aerobically less fit groups are found in Table 6. These results indicate there was a significant difference in VO$_2$ max (L/min) and percent body fat at the 0.05 level of confidence. VO$_2$ max (ml/kg.min), TC, LDL, TC:HDL ratio and LDL:HDL ratio were significantly different at the 0.01 level of confidence. Age, HDL and weight were not significantly different between the two groups. The results support that two distinct groups did exist with regard to aerobic capacity.
Table 6
Analysis of Variance Between the Two Groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>MS*</th>
<th>F**</th>
<th>SIG.***</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.002</td>
<td>0.003</td>
<td>0.9574</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>144.451</td>
<td>2.743</td>
<td>0.1259</td>
</tr>
<tr>
<td>Percent body fat</td>
<td>61.143</td>
<td>5.168</td>
<td>0.0440</td>
</tr>
<tr>
<td>VO max(ml/kg.min)</td>
<td>836.677</td>
<td>43.275</td>
<td>0.0000</td>
</tr>
<tr>
<td>VO max(L/min)</td>
<td>2.104</td>
<td>5.659</td>
<td>0.0366</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>2953.109</td>
<td>7.806</td>
<td>0.0175</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>108.931</td>
<td>1.728</td>
<td>0.2154</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>273.603</td>
<td>15.338</td>
<td>0.0024</td>
</tr>
<tr>
<td>Total cholesterol:HDL</td>
<td>6.672</td>
<td>11.430</td>
<td>0.0061</td>
</tr>
<tr>
<td>LDL:HDL</td>
<td>6.009</td>
<td>11.415</td>
<td>0.0062</td>
</tr>
</tbody>
</table>

* Mean Square  ** F-ratio  *** Significance

Correlations Among Variables Measured

Correlation coefficients for aerobic capacity \( (\text{VO max(\text{ml/kg.min})})^2 \) with selected variables are given in Table 7. Significant correlations were found with all variables except TC \((p=0.19)\) and LDL \((p=0.059)\). All significant correlations were negative except for the correlation with HDL which was positive \((p=0.02)\).
Table 7
Correlations Between VO\(_{\text{max}}\) (ml/kg.min) and Other Variables (N=13)

<table>
<thead>
<tr>
<th></th>
<th>ml/kg.min</th>
<th>SIG.</th>
</tr>
</thead>
<tbody>
<tr>
<td>HDL</td>
<td>0.600</td>
<td>0.020</td>
</tr>
<tr>
<td>LDL</td>
<td>-0.476</td>
<td>0.059</td>
</tr>
<tr>
<td>TC</td>
<td>-0.269</td>
<td>0.199</td>
</tr>
<tr>
<td>TC:HDL</td>
<td>-0.702</td>
<td>0.005</td>
</tr>
<tr>
<td>LDL:HDL</td>
<td>-0.724</td>
<td>0.004</td>
</tr>
<tr>
<td>Pat</td>
<td>-0.743</td>
<td>0.003</td>
</tr>
<tr>
<td>Weight</td>
<td>-0.975</td>
<td>0.000</td>
</tr>
</tbody>
</table>

Correlation coefficients between serum cholesterol and other variables are given in Table 8. No significant correlations involving TC were found. Negative, significant correlations between HDL and percent body fat (p=0.03) and weight (p=0.01) are evident. LDL was significantly correlated with only one variable which was weight. The correlation was positive and significant at the 0.01 level of significance.

Correlation coefficients between the cholesterol ratios and other variables appear in Table 9. Both the TC:HDL ratio and LDL:HDL ratio were significantly correlated with VO\(_{\text{max}}\) (ml/kg.min)\(^2\) (p=0.01) and weight (p=0.01). The TC:HDL ratio and LDL:HDL ratio were positive correlates with weight, while VO\(_{\text{max}}\) (ml/kg.min)\(^2\) was negatively correlated with the TC:HDL ratio and the LDL:HDL ratio.
### Table 8
Correlations Between Plasma Cholesterol and Other Variables (N=13)

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>HDL</th>
<th>LDL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VO₂ max (ml/kg.min)</strong></td>
<td>-0.269 (0.199)</td>
<td>0.600 (0.020)</td>
<td>-0.476 (0.059)</td>
</tr>
<tr>
<td><strong>Percent body fat</strong></td>
<td>-0.114 (0.365)</td>
<td>-0.657 (0.027)</td>
<td>0.087 (0.393)</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>0.158 (0.312)</td>
<td>-0.657 (0.010)</td>
<td>0.386 (0.013)</td>
</tr>
<tr>
<td><strong>VO₂ max (L/min)</strong></td>
<td>-0.3005 (0.159)</td>
<td>-0.0084 (0.489)</td>
<td>-0.2955 (0.163)</td>
</tr>
</tbody>
</table>

**NOTE:** ( ) = significance

### Table 9
Correlations Between Cholesterol Ratios and Other Variables (N=13)

<table>
<thead>
<tr>
<th></th>
<th>TC: HDL</th>
<th>LDL: HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VO₂ max (ml/kg.min)</strong></td>
<td>-0.7022 (0.005)</td>
<td>-0.7246 (0.004)</td>
</tr>
<tr>
<td><strong>VO₂ max (L/min)</strong></td>
<td>-0.4954 (0.051)</td>
<td>-0.4577 (0.067)</td>
</tr>
<tr>
<td><strong>Percent body fat</strong></td>
<td>0.3640 (0.122)</td>
<td>0.436 (0.078)</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>0.6816 (0.007)</td>
<td>0.713 (0.005)</td>
</tr>
</tbody>
</table>

**NOTE:** ( ) = significance
Stepwise Multiple Regression Equations

Multiple regression coefficients with \( VO_{\text{max}}(\text{ml/kg.min}) \) as the dependent variable are given in Table 10 and Table 11. Because TC:HDL is not independent from HDL, two equations were necessary to test the relative strength of TC:HDL. TC was not used in either equation because it did not correlate significantly with \( VO_{\text{max}}(\text{ml/kg.min}) \). The \( t \)-ratio is a measure of the relative strength of the independent variable.

Table 10

Regression Coefficients for Specified Variables on \( VO_{2,\text{max}}(\text{ml/kg.min}) \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>( B )</th>
<th>( t )-ratio</th>
<th>SIG-( t )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat</td>
<td>-1.57579</td>
<td>-3.016</td>
<td>0.0117</td>
</tr>
<tr>
<td>LDL</td>
<td>-0.16669</td>
<td>-2.272</td>
<td>0.0464</td>
</tr>
<tr>
<td>HDL</td>
<td>0.05846</td>
<td>0.209</td>
<td>0.8384</td>
</tr>
</tbody>
</table>

\[ R=0.67279 \quad R^2=0.45265 \]

The equation reached the 0.05 level of significance upon entry of both fat and LDL. Adding HDL to the equation exceeded the accepted level of confidence and thus HDL cannot be used as an accurate predictor of \( VO_{\text{max}} \) in the sample population.
### Table 11
Regression Coefficients for Specified Variables on \( \text{VO}_2\max \text{(ml/kg.min)} \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>t-ratio</th>
<th>SIG-t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat</td>
<td>-1.57579</td>
<td>-3.016</td>
<td>0.0117</td>
</tr>
<tr>
<td>TC: HDL</td>
<td>-0.43801</td>
<td>-2.045</td>
<td>0.0680</td>
</tr>
</tbody>
</table>

\[ R = 0.67279 \quad \text{and} \quad R^2 = 0.4265 \]

The entry of TC: HDL to the regression equation made no significant contribution to the variation of \( \text{VO}_2\max \text{(ml/kg.min)} \).

Multiple regression coefficients with HDL as the dependent variable are given in Table 12.

### Table 12
Regression Coefficients for Specified Variables on HDL

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>t-ratio</th>
<th>SIG-t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>-0.66513</td>
<td>-2.703</td>
<td>0.0205</td>
</tr>
<tr>
<td>Fat</td>
<td>-0.21482</td>
<td>-0.645</td>
<td>0.5335</td>
</tr>
<tr>
<td>LDL</td>
<td>-0.01771</td>
<td>-0.070</td>
<td>0.9459</td>
</tr>
</tbody>
</table>

\[ R = 0.63175 \quad \text{and} \quad R^2 = 0.39911 \]
Multiple regression coefficients with LDL as the dependent variable are given in Table 13.

Table 13
Regression Coefficients for Specified Variables on LDL

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>T-ratio</th>
<th>SIG-T</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>0.95004</td>
<td>9.489</td>
<td>0.0000</td>
</tr>
<tr>
<td>VO₂max (ml/kg.min)</td>
<td>-0.18542</td>
<td>-1.907</td>
<td>0.0857</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>-0.25755</td>
<td>-1.118</td>
<td>0.2897</td>
</tr>
</tbody>
</table>

\[ R=0.77320 \quad R^2=0.59784 \]

Discussion

Although there are some few studies have been done examining the possibility of aerobic fitness as a criterion in CHD risk factor analysis in individuals under the age of 18 years. In adults, Cooper, et al., found a higher level of fitness to be negatively correlated to risk factors of CHD in his study of 3,000 men (10). Thorland and Gilliam found physical activity levels of normal children to be predictive of serum lipid levels (59). Durant and Linder's study found the relationship between physical activity and lipoprotein levels to be more pronounced in active adolescents than among less active adolescents (34). In this study I found the aerobically fit individuals to have lower risk for CHD in TC level,
LDL level, TC to HDL ratio, LDL to HDL ratio and percent body fat. These findings suggest that CHD risk factors are present in the adolescent population and there is less prevalence of CHD risk factors in the aerobically fit individuals. Therefore, it would be beneficial for individuals to be physically active during childhood as well as when they are adults.

Comparison of the Two Groups

In this study the hypothesis was that there would be a significant difference in selected CHD risk factors between the aerobically fit group and the aerobically less fit group. The analysis of variance showed there was a significant difference in TC, LDL, TC:HDL, LDL:HDL and percent body fat between the two groups. The finding of no significant difference between the aerobically fit group and aerobically less fit group in HDL levels is in agreement with results from both Haskell, et al., research and Heath, et al., research (22,23). In this study, there are a few possible reasons why HDL did not differ significantly between the two groups. First, the sample size was small. It is possible that the sample was not indicative of the population as a whole and therefore the unusual finding with HDL would not be in agreement with most of the literature. Secondly, the activity level of the aerobically fit group may not have been high enough to significantly change their HDL level. Some research has indicated changes in HDL levels appear to be correlated with the intensity of exercise rather
than the aerobic capacity of the subject (22,43,47). Finally, the difference in aerobic capacity between groups was not large (aerobically less fit mean = 53.30 ml/kg.min versus 69.75 ml/kg.min for the aerobically fit). Perhaps a greater range in aerobic capacity would have yielded a significant difference between the two groups.

Haskell, et al., found that the level of performance achieved during a treadmill exercise test was not significantly related to HDL in 2,319 men and 2,036 women tested (22). They did not suggest any reasons for these results. They also did not test any subjects who were currently following a rigorous training schedule.

Heath, et al., collected data on ten coronary artery disease patients who were participating in an outpatient exercise training program. They found LDL, TC:HDL ratio and the LDL:HDL ratio to significantly correlate inversely to VO\textsubscript{max}(ml/kg.min). Although 2 HDL did increase significantly with exercise, HDL did not significantly correlate with VO\textsubscript{max}.

In this study HDL did correlate significantly with VO\textsubscript{max}. The majority of research, however, has indicated increased HDL levels are significantly correlated to increased aerobic capacity (10,12,20,60). Therefore, exercise and a correspondingly high VO\textsubscript{max} would probably decrease an individual's risk for CHD by 2 maintaining a high HDL level.

\textbf{Relationship of VO\textsubscript{max} with Other Variables}

As previously mentioned, HDL was significantly correlated to VO\textsubscript{max} (p=0.02). LDL, on the other hand, was significantly
different between the two groups; but, LDL was not significantly correlated to VO\textsubscript{max}. The relationship of LDL to VO\textsubscript{max} is also indecisive in the literature.

On one hand, Heath, et al., found significant, inverse correlations between VO\textsubscript{max} and LDL (23). Conversely, Williams, et al., found significant increases in aerobic capacity were achieved at lower exercise levels than were required to alter LDL levels in their year long experiment involving men, ages 30-55 years (64). Possibly LDL level alteration is dependent on exercise intensity as is similar to a hypothesis held by some researchers with regard to HDL level alteration. They believe the level of exercise intensity must reach 7 kcal/min before there will be a beneficial alteration in HDL level (46,48,54).

The TC:HDL ratio (p=0.005) and the LDL:HDL ratio (p=0.004) were both inversely, negatively correlated to VO\textsubscript{max}. Hartung, et al., found a similar inverse correlation in their study involving marathon runners and sedentary controls (21). The two mentioned ratios are being used more often than in the past as identification of at risk individuals. This study has indicated two reasons why the ratios should be used in CHD risk factor analysis. The first reason is in regard to HDL. As mentioned, this study did not show a significant difference between the two groups in HDL levels. However, the study did show a significant difference between the two groups in the TC to HDL ratio and the LDL to HDL ratio. Both of these ratios include HDL. Therefore, HDL does show a significant
difference between the two groups when it is included in the ratios. Similarly, LDL does not significantly correlate to VO max by itself; but, when LDL is included in the LDL to HDL ratio, it does significantly correlate to VO max. Therefore the use of ratios may be more indicative of risk for CHD than the lipoproteins are alone.

TC was the only variable that was not even close to having a significant correlation with VO max which is another indication that the lipoprotein ratios may be a more accurate means of screening for CHD than TC level alone. Once again the use of ratios can be supported by the findings of TC alone and TC as part of the TC to HDL ratio. The TC to HDL ratio showed a significant difference between the two groups as well as a significant correlation to VO max. Therefore, TC can be more beneficial in a CHD screening test if it is used as part of the TC to HDL ratio than if it were used alone.

Body fat (p=0.003) and body weight (p=0.001) were both inversely, significantly correlated with VO max. Both percent body fat and body weight have been recognized by researchers as inverse correlates to VO max for several years (5,10,16,31). Therefore, a decreased body weight and decreased percent body fat would seem to decrease an individual's risk for CHD.
VO\text{\textsubscript{max}}(ml/kg/min)

The factor correlating with the greatest amount of variation in VO\text{\textsubscript{max}} was percent body fat \((p=0.01)\). Several researchers have suggested the use of percent body fat as a risk factor in CHD screening procedure \((15,17,21,36,43,52)\). Similarly, this study has indicated percent body fat may also be used as a predictor in the VO\text{\textsubscript{max}} level of an individual. In a practical sense, this means that decreasing an individual’s percent body fat could cause an increased fitness level. As mentioned earlier, a high fitness level is significantly correlated to a decreased risk for CHD. Therefore, decreasing an individual’s percent body fat would increase his fitness level and thus decrease his risk for CHD.

LDL was the next variable correlated with a significant amount of variation in VO\text{\textsubscript{max}} when entered with percent body fat \((p=0.05)\). This particular finding is not apparent in the research of other investigators.

None of the other variables correlated with a significant amount of variation in VO\text{\textsubscript{max}}. In the sample population, percent body fat and LDL could be used as accurate predictors of VO\text{\textsubscript{max}}. Therefore if any of the subjects had a decrease in percent body fat or LDL level, I would expect a similar increase in VO\text{\textsubscript{max}}.

HDL

In the regression equation using HDL as the dependent variable, body weight correlated with the greatest variation in HDL \((p=0.02)\).
Body weight has been suggested as an accurate predictor of HDL levels (22,56). As mentioned earlier, obese individuals are thought to be more at risk for CHD than their non-obese counterparts. This study would support the mentioned hypothesis because HDL correlated with greatest amount of variation in body weight. Therefore the less extra body weight an individual is carrying, the less at risk he is for CHD.

LDL

TC correlated with the greatest variation in LDL (p=0.001). This was expected since LDL level is the highest of the lipoprotein fractions of TC. Although not significant, VO max (p=0.08) turned out to be a greater correlate to LDL variation than did body weight (p=0.28). In the straight correlations analysis, body weight (p=0.01) showed a stronger correlation to LDL than did VO max (p=0.06). This is in support of the idea that strong correlates do not always act as strong contributors to the variation of a selected variable.

Although research has failed to demonstrate that an aerobic exercise program alters serum lipid levels in normal children and adolescents, several studies have shown that habitual physical activity levels of normal children were predictive of serum lipid levels (12,34,60). Although correlational studies do not show a cause and effect relationship, this study strongly supports the continually growing body of evidence which suggests regular exercise
decreases an individual's risk for CHD. In particular, aerobic fitness is correlated to a decreased risk for CHD in adolescent boys. Therefore, it would be beneficial for children and adolescents to start a regular exercise program which could have the possible effect of reducing their risk for CHD.
CHAPTER 5
Summary, Conclusions and Recommendations

Summary
The main purpose of the study was to detect whether TC, HDL, LDL, body weight and percent body fat were different between the two test groups. The two study groups were: an aerobically fit group and an aerobically less fit group. Each group consisted of males, ages 16-18 years. A second purpose of this study was to determine the relationship of aerobic capacity (VO\textsubscript{max}) to TC levels, HDL levels, LDL levels, body weight and percent body fat of the subjects. A final purpose of the study was to identify which variables measured correlated with the greatest amount of variation in VO\textsubscript{max} (ml/kg.min), HDL levels and LDL levels in an attempt to predict those levels among the subjects tested. The TC to HDL ratio and the LDL to HDL ratio were also included in the statistical analysis of data.

Ten male students from the Missoula, Montana area and three males from Browning, Montana volunteered for the research project. Letters containing both an explanation of test procedures and the purpose of the study were mailed to each parent. The parents were also sent informed consents which they were required to sign before their son could participate in the study.

Several different measurements were taken from the subjects in order to obtain the necessary variables needed in the research project. The variables included: TC level, HDL level, LDL level, body weight, percent body fat and VO\textsubscript{max}.
A 5cc blood sample from each subject was analyzed for TC level, HDL level and LDL level. Percent body fat was estimated from the subject's weight underwater and the residual volume in his lungs. Prior to the underwater weigh-in the subject was weighed on dry land. A treadmill graded exercise test to exhaustion was administered to each subject to determine his VO max.

**Analysis of Variance Between the Two Groups**

The analysis of variance showed there was a significant difference in TC, LDL, TC:HDL ratio, LDL:HDL ratio and percent body fat between the two groups. An interesting finding was no significant difference in the HDL levels between the two groups. This is not in agreement with other reports (1,12,20,60).

**Aerobic Capacity vs. Other Variables**

Aerobic capacity was positively correlated to HDL at the 0.02 level of confidence. Aerobic capacity was negatively correlated to LDL:HDL, TC:HDL, percent body fat and body weight at the 0.01 level of confidence. TC showed no correlation to aerobic capacity.

When combined with HDL in the TC:HDL ratio, TC did show a significant correlation to aerobic capacity. This would support the importance of using the lipoprotein ratios in studies involving total serum cholesterol.
Variation in $V_O^{\max}$, HDL and LDL

Percent body fat correlated with the greatest amount of variation in $V_O^{\max}$. The 0.05 level of significance was reached upon addition of LDL to the regression equation. In the sample population, both percent body fat and LDL could be used as predictors of $V_O^{\max}$. Although not significant at $p=0.068$, TC:HDL did account for some variation in $V_O^{\max}$.

Body weight correlated with the greatest amount of variation in HDL. No other variables made a significant contribution to the variation in HDL.

The variable accounting for the greatest amount of variation in LDL was TC. The level of confidence was 0.00 making TC an accurate predictor of LDL levels in the sample population. Although body weight showed a significant correlation with LDL ($p=0.01$), weight did not show a significant contribution to the variation of LDL ($p=0.29$).

As previously mentioned, this research showed a significant amount of variation of selected CHD risk factors between the two test groups. The only factor that did not show a significant variation was HDL. Although correlational studies such as this one do not show a cause and effect relationship, they do lend support to the current hypothesis with regard to CHD. That is, regular exercise and thus an increased aerobic fitness may help reduce the risk of CHD by reducing the magnitude of CHD risk factors.
Conclusions

Summary of Results

A. Significant differences existed between the aerobically fit group and aerobically less fit group in percent body fat, VO max, TC level, LDL level, TC:HDL and LDL:HDL.

B. HDL level was not significantly different between the two groups.

C. VO max was significantly correlated to HDL, percent body fat, body weight, TC:HDL and LDL:HDL.

D. VO max was not significantly correlated to LDL or TC.

E. Body fat was the factor correlating to the greatest amount of variation in VO max. LDL was the next significant factor in VO max variation.

F. Body weight was the only factor to significantly correlate to variation in the HDL level.

G. TC was the only factor to significantly correlate to variation in the LDL level.

Conclusions

The results of this study indicate the following conclusions:

This study showed aerobically fit 16-18 year old boys to be at less risk for CHD than aerobically less fit 16-18 year old boys. The evidence from this study and the literature suggest that it would be beneficial for adolescent boys to increase their aerobic fitness level and thus decrease their risk for CHD.
This study also indicated that individuals with a lower percent body fat were less at risk for CHD as indicated by their blood lipid profile. The study also showed a significant inverse correlation between aerobic fitness and percent body fat. Therefore, decreasing percent body fat would also be beneficial in reducing the risk for CHD.

An important finding of this study is that young males can be identified as being at risk for CHD through analysis of the aerobic fitness level. The subjects who were in the less fit group were more at risk for CHD because of their greater TC level, LDL level, percent body fat, TC:HDL ratio and LDL to HDL ratio. Therefore, a regular exercise program started at an early age and maintained throughout a lifetime would help decrease an individual's risk for CHD.

Recommendations

Based on the results of this study, the following recommendations for further study are proposed.

A complete dietary analysis should be done on each subject so that the researcher may examine the possible role diet has on the presence of CHD risk factors.

Information on family history should be collected on each subject so that the role heredity plays in CHD may be explored.

Investigators might consider the effect exercise intensity has on CHD risk factors with special attention being given to HDL levels and LDL levels.
The comparison of physical activity and vigorous physical activity (>7.5 cal/min) to the presence of CHD risk factors.

A long-term follow-up study to examine the changes in CHD risk factors with time would be valuable.

A longitudinal study starting in early childhood and focusing on the effect physical activity and diet have on CHD risk factors would be beneficial.
REFERENCES


57. Streja, D. & Mymin, D. Moderate Exercise and High-Density Lipoproteins in a Man. Lipids, 1975, 13, 914.


APPENDIX A
INFORMED CONSENT

The experiment in which you are partaking is being used to determine whether there is any correlation in your fitness level, percent body fat and dietary content with the amount of cholesterol and high density lipoproteins (HDLs) in your blood. HDL is a carrier molecule of cholesterol.

A graded exercise test will be used to determine your fitness level. Three electrodes will be applied to your chest so that we may monitor your heart rate during the test. Your expired air will be collected and analyzed by the Beckman Metabolic Cart. Prior to the graded exercise test, a nurse will take a 3.0ml venous sample of your blood so that we may determine your blood HDL level and cholesterol level. You will be underwater weighed and your weight underwater will be used to calculate your percent body fat. I will also ask you to fill out a dietary index. The dietary index will give me an indication of your dietary cholesterol content.

Discomforts you may feel will be experienced during the graded exercise test. Since you will be asked to exercise as hard as possible, you may experience temporary shortness of breath at the end of the test. Also you may feel a slight pinch in your arm when the nurse draws your blood sample.

This study will show you your fitness level, your percent body, dietary cholesterol, blood cholesterol level and blood HDL level. I will use your test results along with the other results to determine whether fitness level, percent body fat and dietary content is correlated to blood cholesterol and HDL levels. If you choose not to participate in the study you could obtain similar information from a clinical physician.

All experimental procedures will again be explained to you prior to testing. If at any time you wish to withdraw from the test you are free to do so.

Please feel free to ask me any questions you may have with regard to the experimental procedures or the purpose of this study.

In the event physical injury results from biomedical or behavioral research the human subject should individually seek appropriate medical treatment and shall be entitled to reimbursement or compensation consistent with the self insurance program for Comprehensive General Liability established by the Department of Administration under authority of MCS Title 2, Chapter (or by satisfaction of the claim or judgment by the means provided by MCA, section 2-9-315). In the event of a claim for such physical injury further information may be obtained from the University Legal Counsel.

I understand the above statement and wish to be a subject in this study.

DATE____________________

PARENT (if under 18)____________________

INVESTIGATOR____________________
APPENDIX B
LETTER TO THE SUBJECT PRIOR TO TESTING

Dear

Thank you for your participation in my research project.

Enclosed is an informed consent form which should be read by you and your parents. I will go over this information again with you before the testing session. Please bring the signed informed consent form with you when you come to the field house.

You will need to bring a swimsuit for the underwater weighing. In order that we get accurate results, it is important that you do not eat 12 hours before the test. If you are scheduled for 8 a.m. then please do not eat after 8 p.m. the preceding night. Again to insure accurate results, it is important that the testing be done on an empty stomach.

For the treadmill test you will need shorts and your running shoes.

The field house is located on the university campus. It is where basketball games and concerts are held. When you enter the lab, I will have someone there to meet you and he will show you where the lab is located.

If you have any concerns or questions, please call me at 549-0568.

You will receive a copy of your results as soon as I have them all compiled.

Thank you for your help.

Sincerely,

Kerstin Stoedefalke
**APPENDIX C**

**BODY COMPOSITION WORKSHEET**

<table>
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<th>Weight Air (Wa)</th>
<th>Residual Vol (RV)</th>
<th>M/P</th>
</tr>
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<tbody>
<tr>
<td>Apparatus WT (Aw)</td>
<td>Kg</td>
<td>Water T</td>
</tr>
<tr>
<td>Weight Water (Ww)</td>
<td>Kg</td>
<td>Density W (Dw)</td>
</tr>
</tbody>
</table>

\[
X Wx \quad Aw \quad =
\]

Calculate Density:

\[
D = \frac{Wa}{Wa - (Ww - Aw) - (RV + .100)}
\]

Then using the Formula developed by Siri (1956):

\[
\% \text{ Fat} = \frac{(495 - 450)}{D} = \%
\]

**Summary**

\[
\% \text{ Fat} =
\]

\[
\text{Fat} = \quad \text{Lbs} \quad \text{Fat} = \quad \text{Kg}
\]

\[
\text{LBW} = \quad \text{Lbs} \quad = \quad \text{Kg}
\]

Comments - (bathing suit, etc.)

56
APPENDIX D

UNIVERSITY OF MONTANA  HUMAN PERFORMANCE LABORATORY

BODY COMPOSITION

To get an accurate measure of percent body fat and lean body weight follow these instructions:

* - Fast 8 hours before test (water allowed)
* - No exercise or sauna before test
* - Take a soap shower before coming to Lab
* - Wear a brief bathing suit (2 piece for women)

You'll enter the water, sit on seat suspended from scale, exhale and hold breath briefly. Several trials will be necessary. Bring a towel to dry off after the test.

A lung function test will determine the air remaining in the lungs after exhale.
APPENDIX E

Data

1. Total serum cholesterol (mg/dl)
2. High density lipoprotein (mg/dl)
3. Low density lipoprotein (mg/dl)
4. Total serum cholesterol : high density lipoprotein ratio.
5. Low density lipoprotein : high density lipoprotein ratio.
6. $\text{VO}_2\text{max}$ (ml/kg.min).
7. $\text{VO}_2\text{max}$ (L/min).
8. Percent body fat.
9. Weight.
10. Age.
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