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EFFECTS OF INTENSITY AND DURATION OF TRAINING ON  
HIGH DENSITY LIPOPROTEIN CHOLESTEROL

By

Richard A. Washburn

B.S., Cortland State University, 1976

Presented in partial fulfillment of the requirements  
for the degree of

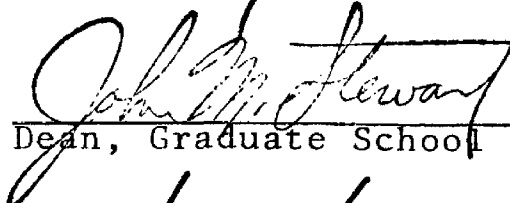
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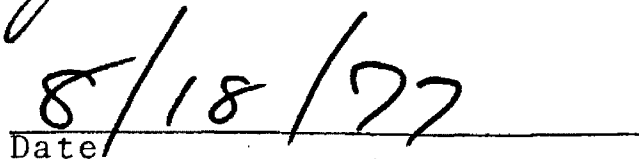
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Effects of Intensity and Duration of Training on High Density Lipoprotein Cholesterol

Director: Dr. Brian J. Sharkey



This study investigated the effects of intensity and duration of training on high density lipoprotein (HDL) cholesterol. Male volunteers from physical education classes at the University of Montana were screened for participation in the study on criteria of age, maximum oxygen uptake, percent body fat, blood pressure, and total serum cholesterol. Fifteen subjects were selected and randomly assigned to 3 groups: Group A (low intensity, long duration exercise), Group B (high intensity, short duration exercise) and Group C (control). Exercise prescriptions were individualized for each training subject. Group A exercised at an intensity of 70 percent of maximum heart rate and a duration of 200 calories during the initial week of training and progressively increased to an intensity of 80 percent of maximum heart rate and a duration of 400 calories during the final 2 weeks of training. Group B initially exercised at an intensity of 80 percent of maximum heart rate and 100 calories duration and progressively increased to an intensity of 90 percent of maximum heart rate and 200 calories in duration during the last 3 weeks of the training program. Exercising subjects were tested for total cholesterol, HDL cholesterol, maximum oxygen uptake, weight, percent body fat, and blood pressure before and after a period of treadmill training, 3 days per week for 7 weeks. The control group was tested once on these variables and served as a comparison group. Total and HDL cholesterol values were the average of 2 blood samples drawn on 2 nonconsecutive days.

Analysis of the data indicated that there were no significant changes within either exercise group in HDL cholesterol, total cholesterol, weight, percent body fat, or blood pressure. Group B experienced a significant increase in maximum oxygen uptake. There were no significant differences between Groups A, B, or C on any of the parameters measured either pre or post training. Negative correlations were found both between the initial level of HDL cholesterol and the change in HDL cholesterol and the initial level of maximum oxygen uptake and the change in maximum oxygen uptake.

It was concluded that neither low intensity, long duration or high intensity, short duration exercise had a significant effect on the HDL or total cholesterol levels of moderately fit and active subjects following a 7-week training period. A 7-week program of high intensity, short duration training produced a significant increase in maximum oxygen uptake. The initial levels of HDL cholesterol and maximum oxygen uptake are inversely related to the amount of change that can be expected as a result of exercise training.

## ACKNOWLEDGEMENTS

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## TABLE OF CONTENTS

	Page
ABSTRACT . . . . .	ii
ACKNOWLEDGEMENTS . . . . .	iii
LIST OF TABLES . . . . .	vii
LIST OF FIGURES . . . . .	viii
 Chapter	
1. INTRODUCTION . . . . .	1
PURPOSE OF THE STUDY . . . . .	4
DEFINITIONS . . . . .	4
2. SURVEY OF RELATED LITERATURE . . . . .	7
THEORIES OF ATHEROSCLEROTIC DEVELOPMENT . . . . .	7
ORIGIN AND TRANSPORT OF SERUM CHOLESTEROL . . . . .	9
HDL AND ATHEROSCLEROTIC DEVELOPMENT . . . . .	11
EXERCISE EFFECTS ON TOTAL CHOLESTEROL . . . . .	14
EXERCISE EFFECTS ON LIPOPROTEIN LEVELS . . . . .	20
SUMMARY . . . . .	21
3. METHODS AND PROCEDURES . . . . .	24
SUBJECTS . . . . .	24
INITIAL SCREENING . . . . .	24
GROUP ASSIGNMENTS . . . . .	27
PRE TRAINING DATA . . . . .	27

Chapter	Page
GENERAL INSTRUCTIONS TO SUBJECTS . . . . .	29
TRAINING . . . . .	29
CONTROLS . . . . .	31
POST TESTING . . . . .	31
STATISTICAL TREATMENTS . . . . .	32
4. ANALYSIS AND DISCUSSION OF RESULTS . . . . .	33
COMPARISON OF CHANGES WITHIN TRAINING GROUPS . . . . .	33
HDL Cholesterol . . . . .	37
Total Cholesterol . . . . .	44
Oxygen Uptake . . . . .	46
Weight . . . . .	49
Percentage Body Fat . . . . .	49
Resting Systolic Blood Pressure . . . . .	49
Age . . . . .	49
COMPARISON OF TRAINING GROUPS A AND B WITH CONTROL GROUP C . . . . .	50
DISCUSSION . . . . .	50
5. SUMMARY, CONCLUSIONS AND RECOMMENDATIONS . . . . .	57
SUMMARY . . . . .	57
CONCLUSIONS . . . . .	59
RECOMMENDATIONS . . . . .	59
REFERENCES . . . . .	61

APPENDIXES

A.	INFORMED CONSENT FORM . . . . .	67
B.	SKINFOLD NOMOGRAM . . . . .	69
C.	MODIFIED BALKE TREADMILL PROTOCOL . . . . .	70
D.	DIET REPORT FORM . . . . .	71
E.	PROCEDURE FOR TOTAL CHOLESTEROL DETERMINATIONS .	72
F.	PROCEDURE FOR SEPARATION OF HIGH DENSITY LIPOPROTEIN FROM SERUM . . . . .	74
G.	PHYSICAL ACTIVITY REPORT FORM . . . . .	75
H.	PREDICTING CALORIES BURNED DURING PHYSICAL ACTIVITY FROM PULSE RATE . . . . .	76
I.	SAMPLE TRAINING PRESCRIPTION . . . . .	77



## LIST OF TABLES

Table	Page
1. Physical Characteristics of Subjects . . . . .	28
2. Effects of Training on All Exercise Groups (A + B) . . . . .	34
3. Effects of Training on Group A (Low Intensity, Long Duration) . . . . .	35
4. Effects of Training on Group B (High Intensity, Short Duration) . . . . .	36
5. Individual Data for Group A (Low Intensity, Long Duration) . . . . .	39
6. Individual Data for Group B (High Intensity, Short Duration) . . . . .	40
7. Individual Data for Group C (Control) . . . . .	41
8. Group C (Control) Data . . . . .	51
9. F- Ratios, Groups A, B, and C . . . . .	51

## LIST OF FIGURES

Figure	Page
1. The Approximate Chemical Composition of the Major Lipoproteins . . . . .	6

## Chapter 1

### INTRODUCTION

Exercise has been proposed as a means of decreasing the risk of coronary heart disease. Several large-scale studies (8, 38, 56, 47) have been conducted comparing the incidence of coronary heart disease among sedentary and active subjects. While the results of these studies seem to support the hypothesis that exercise does offer some degree of cardioprotection, the evidence is not definitive.

The exact mechanisms by which exercise may provide cardioprotection remain unknown; however, several factors have been reported to be favorably influenced by exercise. These factors include blood clotting and fibrinolysis, blood pressure, serum lipids, and myocardial function (20).

More specifically, the literature regarding the effects of exercise on serum cholesterol is conflicting and confusing. Golding (28) reported a significant reduction in serum cholesterol in men following a twenty-five-week period of intense exercise. This decrease in cholesterol paralleled losses in body fat. Campbell (10) found significant decreases in serum cholesterol following a ten-week

training program. These decreases were independent of weight changes and dietary fluctuations. Milesis (44) reported no significant change in serum cholesterol levels in men following an eleven-week training program. Skinner (53) reported no mean change in serum cholesterol levels of men age 35 to 55 following a six-month training program. He also noted that individual fluctuations in cholesterol levels were related to changes in body weight or diet.

Lopez (41) offered a possible explanation for the discrepancies in this research. He suggested that since the total cholesterol measurement is representative of the cholesterol in all lipoprotein fractions, simultaneous changes in cholesterol among these fractions may conceal the effects of exercise. It may be possible that any decrease in the very low density lipoprotein (VLDL) and low density lipoprotein (LDL) cholesterol could be overridden by an increase in the high density lipoprotein (HDL) cholesterol.

A review of the literature produced little information regarding the effects of exercise on HDL cholesterol. Carlson and Mossfeldt (14) reported no significant change in the cholesterol content of any of the lipoprotein classes following eight to nine hours of cross-country ski racing. Vial (58) found a decrease in VLDL and LDL and an increase in HDL following a seven-week exercise program. Miller and Miller (45) have reported higher levels of HDL among skiers

with high levels of activity than among the general male population in Sweden. Wood (65) has made a similar observation with regard to the HDL cholesterol levels of male joggers and non-joggers in the United States.

The importance of studying the effects of exercise on HDL cholesterol has been more clearly pointed out by recent research. A study by Rhoads (48) indicated a stepwise increase in the risk of coronary heart disease associated with increased levels of LDL cholesterol, and that at any fixed level of LDL cholesterol the probability of coronary heart disease increased as the level of HDL cholesterol fell. Most recently, Dr. William Castelli (33), the Director of Laboratories for the Framingham Heart Study, reported that high density lipoproteins are "the single most powerful lipid predictor of inverse cardiovascular risk."

This information points to the need for further investigation into the effects of exercise on HDL cholesterol. While some of the research is suggestive of a positive effect of exercise on this lipoprotein fraction, the type of exercise required to elicit these possible effects remains unclear. Most studies that have reported positive effects (14, 65) have involved habitual levels of low intensity, long duration exercise such as cross-country skiing and jogging. It is unclear what effects exercise over a shorter period of time and of varying intensities

and durations may have on HDL cholesterol. Those studies that have reported no change in total serum cholesterol following training might be explained by increases in HDL cholesterol which have overridden the decreases in some of the other lipoprotein fractions. In an attempt to gain further insight into the effects of exercise on HDL cholesterol, this study individually quantified the exercise as to intensity and duration.

#### PURPOSE OF THE STUDY

The purpose of this investigation was to determine the effects of controlled exercise on HDL cholesterol. A second purpose was to determine if this parameter is affected differentially by exercise of varying intensities and duration.

#### DEFINITIONS

To facilitate understanding of the remainder of this paper, terms that may be confusing or unfamiliar are defined.

1. Atherosclerosis is a form of arteriosclerosis in which there are localized accumulations of lipid-containing materials within or beneath the intimal surface of blood vessels. It is thought to be due to a metabolic defect in the smooth muscle cell involving lipids and lipoproteins and is one of the most common causes of arterial occlusion.

2. Cholesterol is a sterol widely distributed in animal tissues. It occurs in the yolk of eggs, various oils, fats, and nervous tissue. It can be synthesized by the liver and is a normal constituent of bile. It is important in metabolism, serving as a precursor of various steroid hormones like sex hormones and adrenal corticoids.

3. Very Low Density Lipoproteins (VLDL or Pre beta) are small, light, glyceride-rich (60 to 80 percent) lipoproteins that carry endogenous triglycerides, originating predominately in the liver, from the liver to sites in muscle and adipose tissue. Composition of the major lipoproteins is shown in Figure 1.

4. Low Density Lipoproteins (LDL or Beta) are a lipoprotein remnant partially or completely resulting from the metabolism of VLDL and carrying some of the protein, cholesterol, and phospholipid of VLDL.

5. High Density Lipoproteins (HDL or Alpha) are the smallest of lipoproteins. Their function is not completely understood.

6. Chylomicrons are the largest and lightest of the lipoproteins consisting primarily of exogenous triglyceride.

7. Total cholesterol measurement is the sum total of the cholesterol contained in VLDL, LDL, HDL, and chylomicrons.

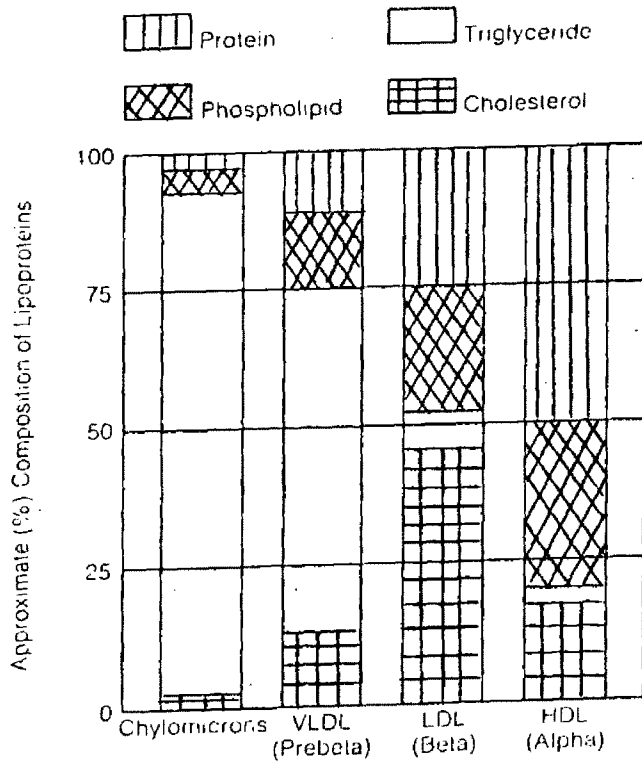


Figure 1

The Approximate Composition of the Major Lipoproteins (54).



## Chapter 2

### SURVEY OF RELATED LITERATURE

For the purposes of continuity and ease of understanding, this review will be divided into the following categories:

Theories of atherosclerotic development.

Origin and transport of serum cholesterol.

HDL and atherosclerotic development.

Exercise effects on total cholesterol levels.

Exercise effects on lipoprotein levels.

Summary.

#### THEORIES OF ATHEROSCLEROTIC DEVELOPMENT

The exact mechanism of atherogenesis remains a mystery; however, several theories have been put forth in attempts at explanation.

The infiltration theory holds that fatty substances from the blood, particularly VLDL and LDL, infiltrate the arterial wall giving rise to deposits of cholesterol. These cholesterol deposits act as irritants causing the inflammation and proliferation of the smooth muscle cell (4, 27). This theory is supported by the fact that no animal species

is immune to the development of atherosclerotic lesions if subjected to sustained levels of cholesterol-rich LDL for several months (63). The mechanism by which the lipid enters the smooth muscle cell is unexplained; however, possible mechanisms are postulated:

1. LDL is trapped from the blood due to an increased permeability of the arterial endothelium. Permeability may be increased by increased blood pressure (54), decreased oxygen tension, or platelets which agglutinate over areas of irritation (27).

2. Lipid may be synthesized by the arterial wall itself (25, 62, 22).

3. Migration of lipid-filled non-nuclear cells, lipophages, through the endothelium after these cells have phagocytized lipoproteins either in the bloodstream or in the organs of the reticuloendothelial system (62).

Recent evidence from Benditt (4) stated that in the initial stages of atherosclerotic development the atherosclerotic plaque contains very little lipid. This would lead one to postulate that lipid infiltration is not the initiating factor in atherogenesis.

The encrustation theory suggests that atherosclerotic plaque begins with a small clot in the arterial wall that is converted to a mass of tissue in the intima as the arterial wall cells migrate to it, multiply, and secrete the characteristic extracellular substances. This theory is in

line with the normal reparative mechanism of the body. The lesions could become large enough to occlude an artery as a result of repeated episodes of injury and repair (4).

The most recent theory of atherogenesis is known as the monoclonal theory (4). This theory postulates that a single smooth muscle cell in the intima of the arterial wall begins multiplying, presumably as the result of exposure to a virus, chemical, or some other stress factor, in a reaction along the lines of the way certain types of normal cells change into cancer cells. This theory seems to be upheld when considered in the light of several well-known coronary risk factors. Factors such as cigarette smoking, high blood pressure, and high levels of serum cholesterol may provide the impetus for arterial cell proliferation.

These theories of atherosclerotic development share the common belief that the lesions begin as a local area of excessive accumulation of smooth muscle cells in the intima of the artery; however, there is no agreement as to what initiates these accumulations and causes them to grow.

#### ORIGIN AND TRANSPORT OF SERUM CHOLESTEROL

Present knowledge is not yet far enough advanced to ascribe to cholesterol a primary role in the development of the atherosclerotic lesion; however, it is well established that cholesterol is the main constituent in quantity in

atherosclerotic plaque (22) and that the risk of developing atherosclerosis increases as the level of serum cholesterol increases (35, 37, 27, 57, 48).

Cholesterol does not circulate freely in the blood, but is present in the form of macromolecules: chylomicrons, VLDL, LDL, and HDL. The composition of these macromolecules is shown in Figure 1. Forty percent of the total serum cholesterol is derived from the diet, while the remainder is synthesized by the body, primarily in the liver and intestinal mucosa. Homeostatic mechanisms are responsible for the control of the serum cholesterol level (7). The liver plays the most important role in the control of the serum cholesterol level through its ability to synthesize cholesterol and form bile acids which serve to catabolize cholesterol (17). The bile acids are reused, but are eventually modified by bacterial action in the intestine and excreted in the feces. Mechanisms are also available to control the absorption of dietary cholesterol. On a high calorie, high saturated fat diet, the serum LDL cholesterol concentration is increased, while substitution of unsaturated fat for the saturated fat in the diet reduces the serum LDL cholesterol concentration (3, 55). It is possible that this reduction is somehow related to the effect of unsaturated fat on the intestinal absorption of cholesterol (16). Cholesterol in the diet is known to inhibit cholesterol synthesis by the liver, but has no effect on the cholesterol synthesis by the small intestine (55).

## HDL AND ATHEROSCLEROTIC DEVELOPMENT

When discussing atherosclerotic development, most attention has been focused on the cholesterol-rich VLDL and LDL fractions. However, evidence is beginning to accumulate that points to an important function of the HDL fraction and indicates that a reduction in the level of HDL may accelerate the development of atherosclerosis.

Bang et al. (3) compared the plasma lipid patterns of 130 Eskimos (69 females and 61 males) living in the northern part of the west coast of Greenland with Danish citizens and Eskimos living in Denmark as controls. He found that the HDL level in the Eskimo males living in Greenland, where the incidence of ischemic heart disease was very low, was significantly higher than that in Danish males. He found no such differences between Eskimo and Danish females.

Castelli et al. (15) investigated the relationship between HDL cholesterol and the prevalence of coronary heart disease in five different populations. In this study, coronary heart disease was defined as a definite myocardial infarction or angina pectoris. They concluded that HDL cholesterol level was inversely related to coronary heart disease prevalence, and that this relationship is essentially independent of the total and LDL cholesterol levels.

Berg et al. (5) determined the serum HDL concentrations in 49 men who have had a myocardial infarction and in

102 healthy middle-age men from northern Sweden. They found the mean HDL concentration to be significantly lower in men with a history of coronary heart disease than among controls.

Miller and Miller (45) reported that plasma high density lipoproteins are reduced in a number of conditions that are associated with an increased risk of future ischemic heart disease. These include hypercholesterolemia, hypertriglyceridemia, male sex, obesity, and diabetes mellitus. They also noted that subjects with clinical ischemic heart disease have lower levels of HDL than do healthy subjects from the same community.

Rhoads et al. (48), in a study of serum lipoproteins and coronary heart disease prevalence in a population of Hawaiian Japanese men, found that the rates for coronary heart disease decreased with an increase in the levels of HDL cholesterol. They found that the HDL cholesterol level made a highly significant contribution to the discrimination of cases of coronary heart disease over and above that made by LDL cholesterol and total cholesterol alone.

The findings of Rhoads et al. have been substantiated by the work of the Framingham Heart Study. Dr. W. P. Castelli (32) indicates that high density lipoproteins are "the single most powerful lipid predictor of inverse cardiovascular risk."

Several mechanisms for the protective factor of increased levels of HDL have been offered. Miller and

Miller (45) proposed that HDL facilitates the uptake of cholesterol from peripheral tissues and its transport to the liver for catabolism and excretion. They also noted an inverse relationship between the plasma HDL concentration and the size of the body cholesterol pools.

Carew et al. (13) and Koschinsky et al. (40) both reported that HDL binds to the surface of porcine smooth muscle cells as effectively as LDL but is internalized and degraded much more slowly. They incubated cells with LDL and found an increase in intercellular cholesterol content; however, when the cells were incubated with the same amount of LDL plus an equal or higher concentration of HDL under comparable conditions, they showed no cholesterol accumulation. This interaction of LDL and HDL could be a second mechanism contributing to the protective effect of high plasma HDL concentrations.

Hisa et al. (32) offered another possible mechanism. They discussed a serum cholesterol binding reserve (SCBR), which is the capacity of serum to solubilize additional amounts of cholesterol above the levels normally carried. They attributed this reserve capacity to two serum lipoprotein subfractions. SFV is a subfraction separated from VLDL and SFH is a subfraction separated from HDL. SFH could be the fraction of HDL that enables it to bind cholesterol and remove it from the tissues. It may be possible that as the subfraction of lipoprotein that enters

the intima by phagocytosis SFH binds cholesterol which has been liberated from other species of lipoprotein after degradation of the apolipoprotein and then returns to the circulation via a reverse process. A second possibility is that SFV and SFH could compete with the extracellular components of the arterial wall for binding of intracellular cholesterol and then transfer it from the intracellular spaces to the circulation.

In the same study Hisa et al. (32) reported that when the SCBR of patients with premature myocardial infarction was compared to that of controls a trend of increasing SCBR with increasing levels of serum cholesterol and triglycerides was noted among controls but not among the patients.

Some degree of caution is advised in the interpretation of the studies by Carew (13), Koschinsky (40), and Hisa (32), in view of the fact that much of the work was done in an artificial environment. Further in vivo studies are needed to confirm these results.

#### EXERCISE EFFECTS ON TOTAL CHOLESTEROL LEVELS

In light of the evidence that the distribution of cholesterol among the various lipoprotein classes may have effects on the atherogenic properties of cholesterol, and that the total serum cholesterol may play a role in the initiation or growth of the atherosclerotic lesion, it is important to consider any factor, such as exercise, which may have favorable effects on these parameters.



The majority of studies concerning the effects of exercise on serum cholesterol have dealt with its effects on the total cholesterol measurement. Those studies that support the hypothesis that exercise lowers the total serum cholesterol level will be reported first.

Golding (28) found a reduction in total serum cholesterol following a 25-week period of hard exercise emphasizing strength and endurance. This reduction in cholesterol was found to parallel losses in body fat.

Garrett (26) put 13 men, mean age 27.9 years, with an apparent predisposition to coronary heart disease based on levels of blood pressure, cholesterol, and obesity, on a supervised program of exercise. The exercise sessions lasted 90 to 120 minutes and were conducted 6 days per week for a period of 6 weeks. Following this program he found that blood pressure, obesity, and serum cholesterol had been significantly reduced.

Mann et al. (43) conducted a study in which 105 volunteer men, mean age 37 years, were randomly assigned to supervised training for 60 minutes per day, 5 days per week, for a period of 18 weeks. During these exercise periods the subjects raised their pulse rate episodically to 130-190 beats per minute, which led to energy expenditures of 400-900 calories per hour. Following this training period, the serum cholesterol concentrations were reduced from 214 to 204 mg/dl.

In a survey of coronary risk factors in the U.S. Army

Second Infantry Division, Dennis (19) found that 1) jogging was more common among officers than among enlisted men, and 2) among officers who jogged (distances of 17.6-32km per week) there were fewer instances of elevated serum cholesterol.

Rochelle (48) studied 12 normal adults with an age range from 20 to 26 years. They participated in an exercise program which involved running 2 miles per day for time. This procedure was repeated 5 days per week for 5 weeks. Times for the 2-mile run ranged from 12 to 15 minutes. Following this period of training the serum cholesterol levels were significantly reduced; however, they returned to the pre-training levels within 4 weeks after termination of the exercise program.

Hernsberg (30) found that persons in the age range 30 to 49 years and who had high physical working capacities as measured by the bicycle ergometer test had lower serum cholesterol levels. He cautioned that factors other than exercise could be responsible for the lower serum cholesterol levels in this group. Genetic factors, differing diets, or differing levels of stress may have been responsible to some degree for the differences noted.

Johnson et al. (36) and Cany et al. (12) looked at the effect of exercise in offsetting the expected hypercholesteremic effects of a high calorie, high saturated fat diet. Johnson had 10 swimmers on a diet consisting of

40 percent or more dietary fat. They swam 1 to 2 miles per day in addition to their regular warm-up and land drills. Following several months of this program there was no significant change in the serum cholesterol levels, indicating that the exercise was having a positive effect. Cany looked at Marine trainees on a high calorie, high saturated fat diet over a 22-week period. Again, the expected increases in serum cholesterol did not occur. No significant change in the total serum cholesterol level was noted.

Campbell (11) found that the type of physical activity undertaken had varying effects on the total serum cholesterol concentrations. He randomly assigned subjects to different physical education classes including cross-country skiing, golf, tennis, tumbling, apparatus, wrestling, and weight training. He found that subjects who participated in vigorous dynamic types of activity showed a significant decrease in serum cholesterol, while those participating in vigorous but static activity did not experience any significant reductions.

In later studies Campbell (9) divided subjects into groups depending on their body type: lean, muscular, or obese. He found that following a 10-week period of treadmill exercise the serum cholesterol level was significantly reduced in the obese active subjects, the reduction occurring independently of variations in weight and diet. These results may be explained by the fact that Campbell used the

same treadmill speed and grade for all subjects in the study. It could be that while this speed and grade were providing the obese subject with exercise of enough intensity to elicit a training effect, they were not of sufficient intensity to elicit a training effect in the lean and muscular subjects.

Various animal studies have also shown exercise to be effective in lowering total cholesterol levels. Carlson (25) and Watt et al. (59) have both reported lowered total serum cholesterol levels in rats following exercise programs. Wong et al. (64) and Warnock (25) studied birds, and Kobernick and Myasnikow (25) studied rabbits; both reported lowered total cholesterol levels following exercise programs.

Several studies in both human beings and animals provide us with results contrary to those previously reported. Skinner et al. (53) placed 15 sedentary middle-aged men who had not engaged in physical activity for at least 3 years on an exercise program. This program was conducted for a 6-month period, each session lasting 30 to 45 minutes. Attendance averaged 3.4 times per week. The exercise emphasized cardiovascular, respiratory, and muscular endurance. The work was increased in amount and intensity as the exercise program progressed. At the conclusion of the 6-month program, no mean change in total serum cholesterol independent of changes in body weight was noted.

Hurter et al. (34) looked at the cholesterol levels

of 14 trained athletes at rest and following a marathon race and noted no significant differences. The resting cholesterol levels of these 14 athletes were compared to 14 sedentary controls matched for age and body type. Again, no significant differences in total cholesterol levels were noted. Hirsch (31) reported similar results when comparing the total cholesterol levels of trained and untrained individuals.

Kasari (39) reported no significant effect on total cholesterol among college women following a moderate training program. The exercise program consisted of jogging 2 miles per day 3 times per week.

Milesis (44) conducted a physical training program with men ages 28 to 54. The program consisted of continuous and interval running, jogging, and rhythmic calisthenics performed 3.7 days per week for 11 weeks. Weekly energy cost calculations showed that the training was progressive in caloric expenditure. The changes observed in serum cholesterol levels suggested a lowering effect; however, they were not statistically significant.

Animal studies have also failed to show a decrease in serum cholesterol following training. McAllister (25) reported that in dogs fed a high cholesterol diet, the highest serum cholesterol levels were among those dogs that ran 5 miles per day on a treadmill. This result may be partially explained by Mahley (42) who found that when fed

a high fat diet dogs seem to be either hyper or hypo responders. The hyperresponders showed a marked increase in serum cholesterol when on a high fat diet, while the others did not. It was not known if this difference was genetic or metabolic.

#### EXERCISE EFFECTS ON LIPOPROTEIN LEVELS

Recently, in light of the evidence regarding the distribution of cholesterol among the lipoprotein classes and its relation to the atherogenic potential, some researchers have begun to look at the effects of exercise on lipoprotein levels. Balart et al. (2) studied 13 medical students who participated in a program of jogging, running, and calisthenics. The program was conducted for a total of 6 weeks, with the sessions lasting 40 minutes 4 times per week. There was a trend toward lower LDL and VLDL values without changes in body weight.

Vial et al. (58) conducted a 7-week exercise program with 4 adult males. This program involved 30 minutes of daily exercise including running, biking, jogging, and calisthenics. They found a decrease in LDL and VLDL with an increase in HDL.

Miller and Miller (45) reported that the levels of HDL are lower among the general male population of Sweden than in male cross-country skiers with high levels of physical activity.

Lopez et al. (41) exercised 13 young medical students 4 times per week, 30 minutes per session, for 7 weeks. A significant decrease was seen in VLDL with a more moderate decrease in total cholesterol, LDL plus VLDL cholesterol, and LDL. An increase in HDL values was also noted.

Wood (65) indicated that long distance male joggers, ages 35-59 years, have lower LDL and VLDL levels and significantly higher HDL levels than sedentary controls. It was also noted that the HDL/LDL ratio was higher in runners and the total cholesterol was moderately lower when compared to the controls.

Carlson and Mossfeldt (14) indicated that there was no significant change in the cholesterol content of any of the lipoprotein classes between skiers during a cross-country race and during normal activities.

#### SUMMARY

The findings indicate that total serum cholesterol is an important factor in the development of atherosclerosis, although our present knowledge is not yet far enough advanced to ascribe to it the primary role in initiation. The manner in which cholesterol is distributed among the lipoprotein classes may be more indicative of its atherogenic potential than is the total cholesterol measurement.

Studies using exercise as a possible means for reducing serum cholesterol, or changing its distribution

among the lipoprotein classes, provide us with confusing and sometimes conflicting results. Some of these discrepancies may be due to the lack of adequate control groups or in the failure to note dietary or weight fluctuations during the study. A major cause for conflict seems to be in the lack of control over the intensity and duration of the exercise used. In many studies the exercise stimulus was highly variable, and in most instances no serious attempts were made to quantify the exercise for each subject involved. An attempt to control this factor was made in a study by Campbell (9), but as previously discussed he failed to consider the differences in energy expenditures between individuals subjected to the same work load.

Exactly what intensity and duration of exercise are required to elicit a possible lowering effect on serum cholesterol remains unclear. Alan Molde (46), in studying the effect of exercise on serum cholesterol, body weight, and food intake, found that an exercise intensity of 90 percent of maximum heart rate produced similar results with respect to serum cholesterol as did a 75 percent of maximum heart rate intensity. Cureton (18) indicated that exercise should be at least 5 or 6 times the resting metabolic rate to be effective in lowering cholesterol levels. The Heart Disease Control Program of the United States Public Health Service (21) recommends a 400 to 500 kilocalorie



expenditure in 45 minutes 3 times per week to effectively lower cholesterol levels.

In the studies that have been reported in this review, the word "effective" is taken to mean that the total serum cholesterol level was lowered as a result of the various exercise programs. However, considering the recent information regarding the protective factor elicited by high levels of HDL cholesterol, the word "effective" could take on new meaning. It is possible that even if exercise increases, or has no effect on the total serum cholesterol level, changes could be taking place in the distribution of cholesterol among the lipoprotein classes that would have beneficial effects in terms of reducing the potential for atherosclerotic development. This study focused on the possible changes in HDL cholesterol.

## Chapter 3

### METHODS AND PROCEDURES

#### SUBJECTS

Male volunteers were selected from undergraduate classes in exercise physiology and health education at the University of Montana during the Spring Quarter 1977. Fifteen of the volunteers, who met the criteria for participation in the study as determined by the results of initial screening tests, were selected as subjects. The criteria for participation were:

1. Ages 20 to 30 years.
2. Maximum oxygen uptake less than 50 ml/kg/min.
3. Blood pressure within the normal range.
4. Percentage body fat less than 20 percent.
5. Total serum cholesterol less than 250 mg/dl.

#### INITIAL SCREENING

Initial screening tests were conducted in the Human Performance Laboratory at the University of Montana from April 7 to April 21, 1977. Potential subjects were scheduled to report to the lab by appointment and were instructed not

to eat or exercise for at least two hours prior to the testing session. The potential subject was seated and informed as to the testing and experimental procedures and asked to read and sign an informed consent form (Appendix A). Initial screening tests were then conducted with the following protocol:

1. Resting blood pressure was measured using a Baumanometer Standby Model mercury sphygmomanometer and a standard stethoscope with a modified head for blood pressure readings.

2. Height and weight were measured on a Detecto Medic balance scale.

3. Three skinfold sites, the tricep, chest, and abdomen, were measured using a Lange skinfold caliper. Repeated measures were taken to obtain the highest degree of accuracy possible. Percentage body fat was estimated from these values using the nomogram of Consolazio (Appendix B).

4. Maximum oxygen uptake was measured directly by a Technology Inc. oxygen consumption computer during a Modified Balke treadmill Test (Appendix C). The test was conducted on a Quinton Model 18-60 treadmill accompanied by a Quinton Model 643 automatic program control. ECG was continuously monitored by an Avionics Stress Test Monitor. The following protocol was used for maximum oxygen uptake determinations:

- a. The subject was instructed as to the testing procedure.
- b. The subject was prepared with Avionics disposable ECG electrodes placed in a modified V5 configuration.
- c. The subject was instructed as to the use of the treadmill and allowed a period of familiarization followed by a five-minute warm-up period.
- d. The subject was fitted with the headgear and a one-way valve breathing system.
- e. All equipment was engaged and tested.
- f. The test protocol was initiated and continued automatically as programmed.
- g. Oxygen uptake and heart rate were measured and recorded during the second minute of each two-minute work load.
- h. The test was terminated when the oxygen uptake leveled off with increasing work loads, or the subject was unable to continue due to other factors.

Upon completion of the initial screening test the potential subject was given an appointment to report to the Student Health Service at the University of Montana for the drawing of a blood sample for the determination of the total serum cholesterol level. All blood drawing was performed

by the Health Service laboratory staff. The potential subject was instructed to report for the blood drawing following a twelve-hour fast, and to complete a dietary report form (Appendix D) for the two days preceding the test. Blood samples were centrifuged and the serum was extracted and placed in the refrigerator until testing. The cholesterol determinations were performed by the author in accordance with the standard Hycel laboratory bench procedure using Hycel reagents (Appendix E).

Physical characteristics of the subjects chosen for this study as a result of the initial screening can be seen in Table 1.

#### GROUP ASSIGNMENTS

The 15 subjects selected as a result of the initial screening were randomly assigned to one of three equal size groups: Group A (low intensity, long duration training); Group B (high intensity, short duration training); and Group C (control).

#### PRE TRAINING DATA

Initial screening data was incorporated into the pre training data. In addition to the initial screening data, the subjects were instructed to report to the Student Health Service, as outlined previously, for the drawing of a second blood sample. This second sample, as well as the

Table 1  
Physical Characteristics of Subjects

Subject	Age (years)	Ht. (cm)	Wt. (kg)
M.P.	23.5	180.34	78.63
R.F.	26.3	171.5	78.65
R.M.	27.0	182.88	76.4
R.B.	25.0	171.5	72.72
T.D.	23.25	176.53	68.64
K.M.	25.5	205.75	113.64
W.D.	23.8	182.84	84.54
B.A.	22.8	189.23	76.36
R.P.	21.6	172.72	78.18
J.D.	27.7	171.53	74.55
B.N.	21.8	170.82	74.55
D.H.	23.17	172.72	75.0
C.V.	21.33	175.26	74.55
M.M.	21.33	172.72	69.0
V.B.	21.75	167.64	79.09
Means	22.15	177.59	78.30

initial screening sample, was tested by the author for HDL cholesterol in addition to total cholesterol. The high density lipoprotein was separated from the serum in accordance with the procedure described by Hatch and Lees (28) (Appendix F). The average of these two values was used as the pre training value for HDL and total cholesterol respectively.

#### GENERAL INSTRUCTIONS TO SUBJECTS

All training subjects in the study were asked not to make any changes in dietary habits during the period of the study. Subjects were also asked not to engage in any type of aerobic activity other than that prescribed by the study and to restrict all physical activity outside the study. A physical activity report form was provided to record any physical activity outside the study (Appendix G).

#### TRAINING

Training was conducted from April 11 through June 7, 1977, in the Human Performance Laboratory at the University of Montana. The exercise was carried out on the Quinton Model 18-60 motor driven treadmill. Subjects exercised three times per week for a period of seven weeks, resulting in a total of 21 exercise sessions completed. The exercise prescriptions were individualized for each subject in the following manner:

Exercise Prescription

Group A (low intensity, long duration)

Week of program	1	2	3	4	5	6	7
Intensity (% maximum heart rate)	70	70	75	75	80	80	80
Duration (calories)	200	225	250	300	350	400	400

Group B (high intensity, short duration)

Week of program	1	2	3	4	5	6	7
Intensity (% maximum heart rate)	80	80	85	85	90	90	90
Duration (calories)	100	100	125	150	200	200	200

The maximum heart rate for each subject was taken to be the highest heart rate obtained during the maximum oxygen uptake test. The number of calories burned per minute at a particular heart rate was estimated from the nomogram of Sharkey (50) (Appendix H). An example of a completed exercise prescription is found in Appendix I.

Each training session was preceded by a five-minute warm-up period. The treadmill speed was then increased to a level anticipated to elicit the desired target heart rate response. The heart rate was checked by carotid palpation after three minutes to determine if it was at the desired level. If the target heart rate had been reached the timer was started and the training period had begun. If not, the appropriate adjustments were made in the treadmill speed and



the heart rate was rechecked following a two- to three-minute period. The heart rate was also checked periodically during the training session to ensure that it was being maintained at the proper level. Necessary adjustments were again made in treadmill speed. All training sessions were followed by an adequate cool-down period. In an attempt to maintain moderate treadmill speeds, the grade for the treadmill was set at four percent for Group A and six percent for Group B.

#### CONTROLS

The same data were gathered on the control group to provide a means of comparing the effects of the training program on Groups A and B. Only one set of data was obtained on the control group due to the difficulty in locating subjects and time limitations.

#### POST TESTING

Within one week of the completion of the seven-week training program all subjects reported by appointment to the Human Performance Lab for post testing. The same tests were conducted and in the same manner as described under initial screening. The subjects were also scheduled to report to the Student Health Service for the drawing of two blood samples, one each week for the two weeks immediately following the completion of training. The total and HDL

cholesterol values were again determined, with the average of the two being used as the post training value for total and HDL cholesterol respectively.

#### STATISTICAL TREATMENTS

A t test for the difference between two dependent means (61) was used to compare the pre and post training values within the exercising groups.

A one-way analysis of variance (60) was used to compare pre and post training values between the three groups: A, B, and C.

A Pearson Product Moment Correlation Coefficient (61) was computed between parameters of interest.

## Chapter 4

### ANALYSIS AND DISCUSSION OF RESULTS

This chapter presents the data obtained, statistical analysis of the data, and a discussion of the results and their relationship to other studies reported in the literature.

#### COMPARISON OF CHANGES WITHIN TRAINING GROUPS

To determine the significance of the effect of the seven-week training period on the parameters measured, the pre and post training results of all exercising subjects (Groups A and B) were compared using a two-tailed t test for dependent means at the .05 level of significance. The value of t needed for significance was 2.62. The differential changes within Groups A and B were also compared using the same statistical procedure. In this case a t value of 2.78 was needed for significance. Table 2 presents the means, standard deviations, percentage change, and the t values for all exercising subjects. Tables 3 and 4 present the same information for Groups A and B. The results of these analyses follow.

Table 2  
Effects of Training on All Exercise Groups  
(A + B), N = 10

	Pre mean (SD) <sup>a</sup>	Post mean (SD)	Percent change	t
HDL Cholesterol (mg/dl)	50.1 (7.53)	52.28 (9.42)	4.35	0.643
Total Cholesterol (mg/dl)	203.25 (33.08)	205.75 (33.42)	1.23	0.686
Oxygen Uptake (ml/kg/min)	44.78 (5.28)	47.18 (4.49)	5.36	2.84 <sup>b</sup>
Weight (kg)	80.04 (12.53)	79.27 (13.53)	-.96	0.90
Percent Body Fat	13.8	12.35	-10.5	-1.83
Resting Systolic Blood Pressure (mm Hg)	128.5 (8.46)	129.6 (10.13)	0.86	0.479

<sup>a</sup>Standard deviation

<sup>b</sup>.05 level of significance

Table 3  
 Effects of Training on Group A  
 (Low Intensity, Long Duration)  
 N = 5

	Pre mean (SD) <sup>a</sup>	Post mean (SD)	Percent change	t
HDL Cholesterol (mg/dl)	53.92 (3.97)	55.62 (8.13)	3.15	0.496
Total Cholesterol (mg/dl)	200.5 (37.27)	199.4 (32.07)	-.549	0.767
Oxygen Uptake (ml/kg/min)	46.53 (5.06)	48.19 (4.20)	3.55	1.08
Weight (kg)	75.36 (4.04)	73.64 (4.32)	-2.28	-1.75
Percent Body Fat	13.9 (3.88)	12.2 (2.4)	-12.0	-1.19
Resting Systolic Blood Pressure (mm/Hg)	128.2 (10.06)	127.4 (9.74)	-0.6	-1.37

<sup>a</sup>Standard deviation

Table 4  
Effects of Training on Group B (High Intensity,  
Short Duration), N = 5

	Pre mean (SD) <sup>a</sup>	Post mean (SD)	Percent change	t
HDL Cholesterol (mg/dl)	46.27 (7.85)	48.94 (8.43)	5.77	0.354
Total Cholesterol (mg/dl)	206.0 (26.59)	212.2 (29.57)	2.92	0.939
Oxygen Uptake (ml/kg/min)	43.04 (4.27)	46.17 (3.07)	7.27	4.18 <sup>b</sup>
Weight (kg)	84.73 (14.92)	84.91 (15.7)	0.21	0.131
Percent Body Fat	13.7 (3.19)	12.5 (3.19)	-8.76	-2.14
Resting Systolic Blood Pressure (mm/Hg)	128.8 (11.03)	131.8 (8.76)	2.33	0.913

<sup>a</sup>Standard deviation

<sup>b</sup>.05 level of significance

## HDL Cholesterol

All of the mean values for HDL cholesterol were within one standard deviation of the normal population mean for HDL cholesterol, 53 (13) mg/dl (Mean and Standard Deviation) reported by Friedwald (24).

There was no significant change in HDL cholesterol levels among all exercising subjects pre to post training or within Groups A or B. The mean difference pre to post training for all exercisers was 2.18 mg/dl. This difference was not statistically significant.

The mean difference in HDL cholesterol for Group A was 1.7 mg/dl and for Group B 2.67 mg/dl. Neither of these differences was statistically significant.

Group B experienced a greater percentage increase in HDL cholesterol, 5.77 percent versus 3.15 percent for Group A. This result is expected due to the lower pre training value (46.27) for Group B compared with the pre training value (53.92) for Group A. A correlation coefficient of  $-.532$ , significant at the .1 level, was found to exist between the pre training level of HDL cholesterol and the change in HDL cholesterol.

Among the total of 10 training subjects, 6 increased and 4 decreased in HDL cholesterol levels. The largest increase, 17.63 mg/dl or 43.3 percent, was observed in subject B.A. The largest decrease in HDL cholesterol was 12.94 mg/dl or 23.57 percent in subject J.D. Tables 5

through 7 present the pre and post training data for individual subjects. The increase in subject B.A. was due to one relatively high post training measurement. Examination of the dietary report form completed during the two days prior to the drawing of this blood sample did not indicate any dietary reason for this elevation. Although much higher than the other post training values for this subject, it is still within one standard deviation of the mean HDL cholesterol value for active joggers as reported by Wood (65). This value is also within two standard deviations of the population mean values as reported by Friedwald (24). There was also some wide variation noted in the HDL cholesterol levels of other subjects. It is not known if this type of wide variation from day to day is typical. The variations might very well be due to the subjects' failure to observe the twelve-hour fast as instructed. Although they were reminded and given written instructions, it is still possible that true fasting samples were not always obtained. Another possible explanation for this variability might be the effect of stress on the HDL cholesterol levels. Since this study was conducted during the school year, the stress of examinations and other anxieties associated with school attendance could have affected the HDL cholesterol values. This is especially true of the post training samples which were drawn during the week of final examinations.



Table 5  
Individual Data for Group A (Low Intensity, Long Duration), N = 5

Subject	Age (years)	HDL <sup>a</sup> cholesterol (mg/dl)		Total <sup>a</sup> cholesterol (mg/dl)		Maximum oxygen uptake (ml/kg/min)		Weight (kg)		Percent body fat		Systolic blood pressure (mm/Hg)	
		Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
R.F.	26.3	51.2	51.54	160.5	160.5	54.54	51.27	78.63	74.09	10	12	120	119
R.B.	25.0	54.3	47.04	239.0	236.5	47.99	49.43	72.72	71.82	20.5	15	140	138
T.D.	23.25	56.2	69.72	168.5	174.5	47.99	52.91	68.64	66.82	12	8	124	122
R.P.	21.6	59.65	50.46	251	238	41.44	46.36	78.18	75.45	11	12	138	138
M.P.	23.5	48.2	59.35	183.5	187.5	40.69	41.00	78.63	80.00	16	14	119	120
Mean	23.94	53.92	55.62	200.5	199.4	46.55	48.19	75.36	73.64	13.9	12.2	128.2	127.4
Mean difference			1.7 2.05 <sup>b</sup>		-1.1		1.64 2.82 <sup>b</sup>		-1.72		-1.7		-.8

<sup>a</sup>Mean of two samples.

<sup>b</sup>Omitting subject R.F.

Table 6  
Individual Data for Group B (High Intensity, Short Duration), N = 5

Subject	Age (years)	HDL <sup>a</sup> cholesterol (mg/dl)		Total <sup>a</sup> cholesterol (mg/dl)		Maximum oxygen uptake (ml/kg/min)		Weight (kg)		Percent body fat		Systolic blood pressure (mm/Hg)	
		Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
J.D.	27.7	54.9	41.96	217	219.5	49.84	52.91	74.44	73.18	8	6.5	123	120
K.M.	25.5	48.3	42.61	239	241.5	39.09	42.05	113.64	115.68	13	14	121	127
B.A.	22.8	40.69	8.32	193	188	39.94	45.86	76.36	80.68	15	13	136	130
W.D.	23.8	53.35	60.19	212.5	244.5	39.96	42.02	84.54	80.91	17.5	16.0	126	132
B.N.	21.8	34.1	41.6	168.5	167.5	46.36	47.99	74.55	74.09	15	13	126	150
Mean	24.32	46.27	48.94	206.0	212.2	43.04	46.17	84.73	84.91	13.7	12.5	128.8	131.8
Mean difference		2.67		6.2		3.13		.18		-1.2		3.0	

<sup>a</sup>Mean of two samples.

Table 7  
Individual Data for Group C (Control), N = 5

Subject	Age (years)	HDL <sup>a</sup> cholesterol (mg/dl)	Total <sup>a</sup> cholesterol (mg/dl)	Maximum oxygen uptake (ml/kg/min)	Weight (kg)	Percent body fat	Systolic blood pressure (mm/Hg)
R.M.	27.0	62.1	225	43	76.4	15	128
D.H.	21.75	43.79	246	43	75.0	13	130
C.V.	21.33	60.85	184.5	39	74.55	11	125
M.M.	23.17	50.22	211.5	46	69.0	10	128
V.B.	21.33	42.61	225.0	46	79.09	9	120
Mean	22.91	51.91	220.4	43.4	74.81	11.6	126.2

<sup>a</sup>Mean of two samples.

The HDL cholesterol level showed a slight negative correlation with percentage body fat ( $r = -.082$ ). This value is in agreement with that reported by Wood (65) ( $r = -.056$ ).

Although the change in HDL cholesterol was not statistically significant, the trend towards increasing levels following training is consistent with the results reported by several other investigators. Vial (58) noted an increase in the HDL fraction on serum electrophoresis in four adult males following a seven-week program of daily aerobic exercise. Lopez (41) reported a significant increase (16%) in the HDL levels of 13 medical students, mean age 22 years, who participated in an exercise program four times per week, 30 minutes per session for a period of seven weeks. Wood (65) reported a higher level of HDL cholesterol in male joggers ages 35-59 years than in sedentary controls. Both Vial and Lopez showed significant training effects by decreased basal pulse rates following training, while Wood showed the group of joggers to have significantly higher exercise capacities than controls as evidenced by longer Bruce Test treadmill times.

This study, while showing a significant fitness training effect on Group B and on the combined training groups as indicated by the significant increase in maximum oxygen uptake, fails to show a significant increase in HDL cholesterol. As previously indicated, Group B showed a

larger percentage increase in HDL cholesterol than did Group A due to the lower pre training value in Group B. Group B also showed an increase in total cholesterol of 1.23 percent while Group A experienced a slight decrease in total cholesterol of -.549 percent. In view of the inverse relationship between the initial level of HDL cholesterol and the change in HDL cholesterol, this lack of significant change is expected. To further examine the possibility that the initial levels of HDL cholesterol were responsible for the lack of significant change following training, an analysis of covariance was calculated equating the initial levels of HDL cholesterol. This analysis revealed no significant differences in the groups following training. The calculated F was 2.42 with a value of 3.88 needed for significance at the .05 level. Other possible explanations for the lack of change in HDL cholesterol lie in the small sample size and the degree of variability observed in the HDL cholesterol measurement.

The trend towards increased levels of HDL cholesterol with simultaneous decreases in total cholesterol, as seen in Group A, is consistent with that reported in the literature. Since the total cholesterol measurement is the sum of the cholesterol contained in VLDL, LDL, HDL, and chylomicrons, a simultaneous decrease in total cholesterol with increasing HDL cholesterol must be accompanied by decreases in VLDL and LDL cholesterol. In his studies on serum lipids in

medical students cited earlier, Lopez (41) suggested that it is the VLDL fraction that is most affected by exercise. Carlson and Mossfeldt (14) have also observed similar decreases in the VLDL and LDL fractions following an eight-to-nine-hour cross-country ski race. These simultaneous changes among the various lipoprotein classes offer a possible explanation for the confusing results reported in the literature concerning the effect of exercise on serum cholesterol.

### Total Cholesterol

The mean values for total cholesterol were all within the normal mean value ( $189 \pm 33$  mg/dl) reported by Friedwald (24) and less than the 225 mg/dl level which is considered normal for the adult population (55).

The mean change in total cholesterol, 2.5 mg/dl, observed among all training subjects was not statistically significant.

Group A experienced a slight decrease in total cholesterol, while Group B showed a slight increase. Neither of these changes was statistically significant.

Four of the training subjects decreased, five increased, and one showed no mean change in total cholesterol level. Subject W.D. showed the largest increase, 32 mg/dl or 15 percent, while subject R.P. showed the greatest decrease, 13 mg/dl or 5 percent. Subject R.P. had the highest mean pre training value of 251 mg/dl and subject

W.D. had the highest mean post training value of 244.5 mg/dl. Neither of these changes is related to changes in weight or percentage body fat.

No significant correlation was found between the total cholesterol level and percentage body fat ( $r = -.055$ ). This is consistent with the correlation reported by Kasari (39) in a sample of college women ( $r = -.02$ ). Studies on the effects of exercise on total cholesterol by Golding (28), Garrett (26), and Mann (43), however, have indicated that losses in body fat parallel losses in total cholesterol. Failure to note this relationship in this study may be due to the relative leanness and the relatively low initial cholesterol levels of the subject.

The lack of significant change in total cholesterol following a training period as observed in this study is consistent with other studies reported in the literature (53, 34, 14, 39, 44). Those studies which have shown significant change in total cholesterol (43, 49, 28, 26, 36, 9) have either used subjects with elevated cholesterol levels (26), shown significant decreases in body fat (28, 26, 43), or employed more frequent and more intense training programs over longer periods of time. To effectively lower total cholesterol, the Heart Disease Control Program of the United States Public Health Service recommends a 400-500 Kcal expenditure in 45 minutes, three times per week. This degree of energy expenditure was only experienced by members

of Training Group A and only for the last two weeks of the training period, or six training sessions. Although Group A did show a slight decrease in total cholesterol, this change cannot be attributed to the training program.

### Oxygen Uptake

There was a significant increase in mean maximum oxygen uptake (2.4 ml/kg/min) among all exercising subjects. Group A showed a slight but insignificant increase in maximum oxygen uptake (1.66 ml/kg/min), while Group B showed a significant increase (3.13 ml/kg/min) following training.

Possible explanations for the differences in changes in maximum oxygen uptake noted between the two training groups lie in the pre training levels and the differences in the training stimuli. The pre training value was lower in Group B (43.04) than in Group A (46.53). A negative correlation ( $r = -.526$ ), significant at the .1 level, was calculated between the pre training level of maximum oxygen uptake and the change in maximum oxygen uptake. This correlation coefficient is in agreement with that reported by Sharkey (50) ( $r = -.539$ ). It would then be expected that Group B, with the lower pre training value, would be more likely to show significant change.

The training intensity for Group A ranged from 70 to 80 percent of maximum heart rate from the early to the later training sessions. This intensity was probably not



sufficient to elicit a training effect in a group with a pre training value of 46.53 ml/kg/min. Sharkey (52) recommends a training intensity of between 80 and 90 percent of maximum heart rate for individuals with maximum oxygen uptakes greater than 45 ml/kg/min to achieve significant training effects.

Individual subject data (Tables 4 and 5) serve to substantiate these observations. All subjects but one showed some degree of increase in maximum oxygen uptake. The subject who showed a decrease, R.F., had the highest pre training level (54.54 ml/kg/min) and was a member of training Group A. Following training he showed a decrease of 3.27 ml/kg/min. The greatest increase in maximum oxygen uptake (5.92 ml/kg/min) was observed in subject B.A., who had one of the lowest pre training levels (39.94 ml/kg/min) and was randomly placed in training Group B.

Slight positive correlations were noted between maximum oxygen uptake and HDL cholesterol ( $r = .086$ ) and the change in maximum oxygen uptake and the change in HDL cholesterol ( $r = .133$ ). In view of the evidence provided by Wood (65) and others (1, 14) relating higher levels of HDL cholesterol among more highly fit individuals, it seems logical to speculate that there would be a stronger relationship between these variables. It is interesting to note that in the data reported by Wood (65) there is virtually no change in HDL cholesterol levels among a sedentary group

ages 35-59 years. This indicates that the decrease in maximum oxygen uptake, which would be expected with increasing age in a sedentary population, is not accompanied by any changes in HDL cholesterol. It may be that changes in HDL cholesterol observed in the active group are more a function of the level of habitual physical activity than the absolute level of maximum oxygen uptake. Other studies seem to point in this direction. A study by Carlson and Mossfeldt (14) indicated that there were no significant changes in the cholesterol content of any of the lipoprotein classes between skiers during a cross-country ski race or during normal activity. Miller and Miller (45), on the other hand, reported the HDL level among Swedish male cross-country skiers with high levels of activity are higher than those of males from the general population.

Maximum oxygen uptake was found to correlate slightly with percentage body fat ( $r = -.376$ ). This is consistent with a correlation reported by Lopez ( $r = -.3$ ) (41) between a physical fitness index derived from the results of the Harvard Step Test and percentage body fat.

The correlation between maximum oxygen uptake and total cholesterol ( $r = -.375$ ) is significant at the .1 level. In a study of college women, Kasari (39) reported a significant correlation ( $r = -.49$ ) between these variables. Golding (28) and HERNBERG (30) have also reported moderate inverse relationships between physical fitness and total cholesterol.

### Weight

The change in body weight for all exercising subjects was not statistically significant. Group A decreased slightly while Group B increased slightly. Again, neither of these changes was statistically significant. The higher mean weights of Group B were due to the inclusion of subject K.M. with pre and post training weights of 113.64 kg and 115.68 kg respectively.

### Percentage Body Fat

There was no significant change in body fat pre to post training among all exercising subjects or within Groups A or B.

A slight but insignificant correlation ( $r = -.082$ ) was calculated between percentage body fat and HDL cholesterol. This correlation is consistent with that reported by Wood (65) ( $r = -.056$ ). Other correlations with percentage body fat have been discussed previously.

### Resting Systolic Blood Pressure

There was no significant change in resting systolic blood pressure among all exercisers or within either training group.

### Age

Group B, mean age 24.32 years, was slightly but not significantly older than Group A with a mean age of 23.94 years.

COMPARISON OF TRAINING GROUPS A AND B  
WITH CONTROL GROUP C

To determine if either of the training groups was significantly different from the control group, either pre or post training, a one-way analysis of variance was computed using both pre and post training values. An F value of 3.88 was needed for significance at the .05 level. The means and standard deviations for Group C are shown in Table 8. This information for Groups A and B is shown in Tables 3 and 4.

The analysis of variance comparing both the pre and post training values of Groups A and B with Group C yielded no significant differences on any of the parameters measured. The calculated F ratios for these differences are shown in Table 9.

DISCUSSION

Very little literature is available regarding the effects of different types of exercise on HDL cholesterol levels. Indications point to the duration of the exercise as the key factor in initiating significant changes. In this instance duration is thought of not only in terms of the length of each exercise session, but also the period of time over which the activity has been undertaken. Many studies that have shown exercise to be associated with higher levels of HDL cholesterol (45, 65, 14) have studied

Table 8  
Group C (Control) Data, N = 5

	Mean (standard deviation)
HDL Cholesterol (mg/dl)	51.91 (8.26)
Total Cholesterol (mg/dl)	220.4 (21.25)
Oxygen Uptake (ml/kg/min)	43.4 (16.25)
Weight (kg)	74.81 (3.26)
Percentage Body Fat	11.6 (2.15)
Resting Systolic Blood Pressure (mm/Hg)	126.2 (3.89)

Table 9  
F- Ratios, Groups A, B, and C

	Pre training	Post training
HDL Cholesterol	1.279	0.685
Total Cholesterol	0.526	0.563
Maximum Oxygen Uptake	0.873	1.71
Weight	1.49	1.67
Percentage Body Fat	0.653	0.126
Systolic Blood Pressure	0.157	0.557

exercise that was not only of the long duration type, but which had been carried out over an extended period of time. Wood (65) reported that joggers who shown higher levels of HDL cholesterol than sedentary controls had been jogging 15 miles per week for the previous year. A similar observation was reported by Carlson and Mossfeldt (14) who indicated that male cross-country skiers in Sweden, with high levels of activity, had higher HDL cholesterol levels than the general male population. Although not specifically indicated in this study, it might be assumed that these skiers had also been active over an extended period of time.

In this study it was hypothesized that exercise Group A (low intensity, long duration), due to the duration of the exercise sessions, would be more likely to experience beneficial changes in both HDL cholesterol and maximum oxygen uptake than would exercise Group B (high intensity, short duration). This, however, was not the case. Only Group B showed a significant increase in maximum oxygen uptake, and neither group showed significant increases in HDL cholesterol.

The negative correlation ( $r = -.53$ ) calculated between the initial level of maximum oxygen uptake and the change in maximum oxygen uptake offers a partial explanation for the lack of significant change in exercise Group A. The pre training level of maximum oxygen uptake was higher in Group A (46.55 ml/kg/min) than in Group B (43.04 ml/kg/min).

Group A would therefore be expected to experience less change as a result of a training program. Group A also contained subject R.F. who had the highest pre training value for maximum oxygen uptake (54.54) and showed a decrease of 3.27 ml/kg/min following the training period. Omitting this subject from the calculations, the mean change in maximum oxygen uptake in Group A is increased from 1.64 to 2.28 ml/kg/min. This change is much closer to the mean change shown in Group B (3.13). The relatively low intensity of the training stimulus, as discussed previously, also serves to explain the lack of significant change in maximum oxygen uptake in Group A.

The failure to note significant change in HDL cholesterol within either exercise group is partially explained by the pre training level of HDL cholesterol in these subjects (50.1) and the negative correlation ( $r = -.532$ ) that was calculated between the initial level of HDL cholesterol and the change in HDL cholesterol. This initial value (50.1 mg/dl) lies approximately half way between the mean values of HDL cholesterol reported by Wood (65) for joggers (64 mg/dl) and controls (43 mg/dl) ages 35-59 years. It is slightly less than the population mean (53 mg/dl) reported by Friedwald (24). This initial level of HDL cholesterol could have been too high to expect significant changes from the training program.

It is also possible that training three days per week

for a period of seven weeks is not of sufficient frequency or length of time to elicit significant change in HDL cholesterol in moderately fit subjects with relatively high initial HDL cholesterol levels. Studies that have reported significant increases in HDL cholesterol following seven-week training programs have used 30-minute exercise sessions either daily or four times per week. Unfortunately, these studies provide no information as to the initial fitness levels or the levels of habitual physical fitness in these subjects. It is possible that more highly fit subjects require higher intensities and durations of exercise to gain benefits in HDL cholesterol than do subjects of lower fitness.

An important observation in this study was the increase in HDL cholesterol level as the total cholesterol level decreased slightly, as seen in exercise Group A. In the past it has been assumed that reductions in total cholesterol were necessary to reduce the risk of coronary heart disease. The exercise required to achieve these significant reductions was thought to be too difficult in terms of intensity and duration to be viewed as a practical approach. However, in light of the evidence of the cardioprotective factor associated with higher levels of HDL cholesterol (3, 15, 33, 45, 48) the role of exercise in providing cardioprotection via its action on serum cholesterol needs re-evaluation. If the observations in this



study and others hold true, it is possible that exercise may be effective in reducing the risk of coronary heart disease without lowering total cholesterol levels. Exercise may be a practical method of increasing HDL cholesterol levels, especially if these changes can be elicited by low intensity activity undertaken over an extended period of time. It is possible that an increased level of HDL cholesterol is an important factor in the lower incidence of coronary heart disease among those engaged in extensive amounts of activity, either leisure time or occupational, as noted in several large-scale studies (8, 38, 47, 56).

The effect of psychological stress in increasing total cholesterol levels as reported by other investigators (23, 39) was also noted in this study. The last blood sample obtained from the training subjects was drawn during the week of final exams. It is assumed that this is a period of increased stress and anxiety for most students. Considering all four of the blood samples drawn for these subjects, seven of ten recorded their highest total cholesterol level on this last sample. Since there was no significant change in other factors which might account for this increase, such as increases in weight or percentage body fat or changes in diet, it is assumed to be the result of stress.

A marked decrease in HDL cholesterol levels was also noted in this last blood sample. Five of the ten training subjects recorded their lowest HDL cholesterol level on this

sample. These low values served to reduce the average of the post training values and therefore could have possibly negated some of the positive effects of the training program. If stress does in fact result in increased levels of total cholesterol and decreased levels of HDL cholesterol, this would have to be accompanied by increases in the VLDL and LDL fractions which are known to be high in atherogenic potential. This might further add credence to the notion of increased levels of coronary heart disease among those with the "Type A" personality. These individuals are characterized by excessive drive and aggressiveness and tend to be restless, time conscious, and perfectionistic (23).

Although not statistically significant, the trend toward increasing levels of HDL cholesterol following a seven-week period of training is encouraging. It seems that the subjects in this study were too fit and too active to benefit significantly from the length and type of training program used. In view of the evidence that is accumulating indicating the cardioprotective advantages of increased levels of HDL cholesterol, further investigation into the relationship between these variables is warranted.

## Chapter 5

### SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

#### SUMMARY

This study investigated the effects of intensity and duration of training on the HDL cholesterol levels in a sample of college men.

Fifteen subjects who met the criteria for participation were randomly assigned to three groups: Group A (low intensity, long duration exercise), Group B (high intensity, short duration exercise), and Group C (control). The subjects in the training groups engaged in seven weeks of treadmill exercise three days per week in accordance with individually calculated training prescriptions. Group C was used as a comparison group and did not engage in training. Following the seven-week training period the pre and post training measurements were compared within each training group and between the combined training groups. Both pre and post training measurements of the training groups were compared with the control group.

There was no significant change in HDL cholesterol within either training group or the combined training group

pre to post training. The initial level of HDL cholesterol was significantly correlated with the change in HDL cholesterol and slightly correlated with percentage body fat. A trend toward increasing HDL cholesterol with simultaneous decreases in total cholesterol was observed in training Group A. No significant differences were noted between either training group and the control group pre or post training.

Total cholesterol levels showed no significant change within either training group or the combined training groups pre to post training. A slight negative correlation was noted between total cholesterol and percentage body fat.

A significant increase in maximum oxygen uptake was seen in training Group B and in the combined training groups pre to post training. Significant negative correlations were noted between the initial level of maximum oxygen uptake and the change in maximum oxygen uptake and between maximum oxygen uptake and total cholesterol. A slight positive correlation was shown between maximum oxygen uptake and HDL cholesterol. A slight negative correlation was found between maximum oxygen uptake and percentage body fat.

No significant changes within either training group or the combined training groups pre to post training were noted in weight, percentage body fat, or resting systolic blood pressure.

There were no significant differences between training

Groups A or B and the control Group C on any of the parameters measured either pre or post training.

### CONCLUSIONS

The results of this study indicate the following conclusions:

A. Neither low intensity, long duration or high intensity, short duration exercise had a significant effect on the HDL or total cholesterol levels of moderately fit and active subjects following a seven-week training period.

B. The initial level of HDL cholesterol is inversely related to the amount of change that can be expected as a result of exercise training.

C. A seven-week program of high intensity, short duration exercise produced significant increases in maximum oxygen uptake.

D. The initial level of maximum oxygen uptake is inversely related to the amount of change that can be expected as a result of training.

### RECOMMENDATIONS

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

A. Large-scale, controlled training studies should be conducted over longer period of time utilizing measures of all the lipoprotein classes.

B. Participants in further studies of the effect of exercise on HDL cholesterol should have low initial levels of HDL cholesterol and maximum oxygen uptake.

C. The HDL cholesterol levels of individuals who have engaged in different types of physical activity over extended periods of time should be compared.

D. The effects of stress on HDL cholesterol should be further investigated.

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## APPENDIX A

### INFORMED CONSENT FORM

#### TO SUBJECTS:

The objective of this project is to determine the effects of differing types of exercise on the level of high density lipoprotein cholesterol. Participants will be evaluated prior to the beginning of training on maximum oxygen uptake utilizing a treadmill test, height, weight, percentage body fat, and total serum cholesterol. Blood for the cholesterol determinations will be drawn at the University Health Service. The training period will involve treadmill running three times per week for a period of seven weeks. During this time four blood samples will be drawn. Periodic dietary reports, as well as a record of physical activity, will be requested. At the conclusion of training the same variables will be measured as described above.

Subjects in the training groups can expect gains in aerobic capacity as a result of participation. Subjects will also become more aware of measures of aerobic capacity and cholesterol. The experimenters will be glad to try and answer any question that may arise during the course of the

study. As a subject you are free to discontinue participation at any time.

I have read and understand the above statement and hereby agree to participate.

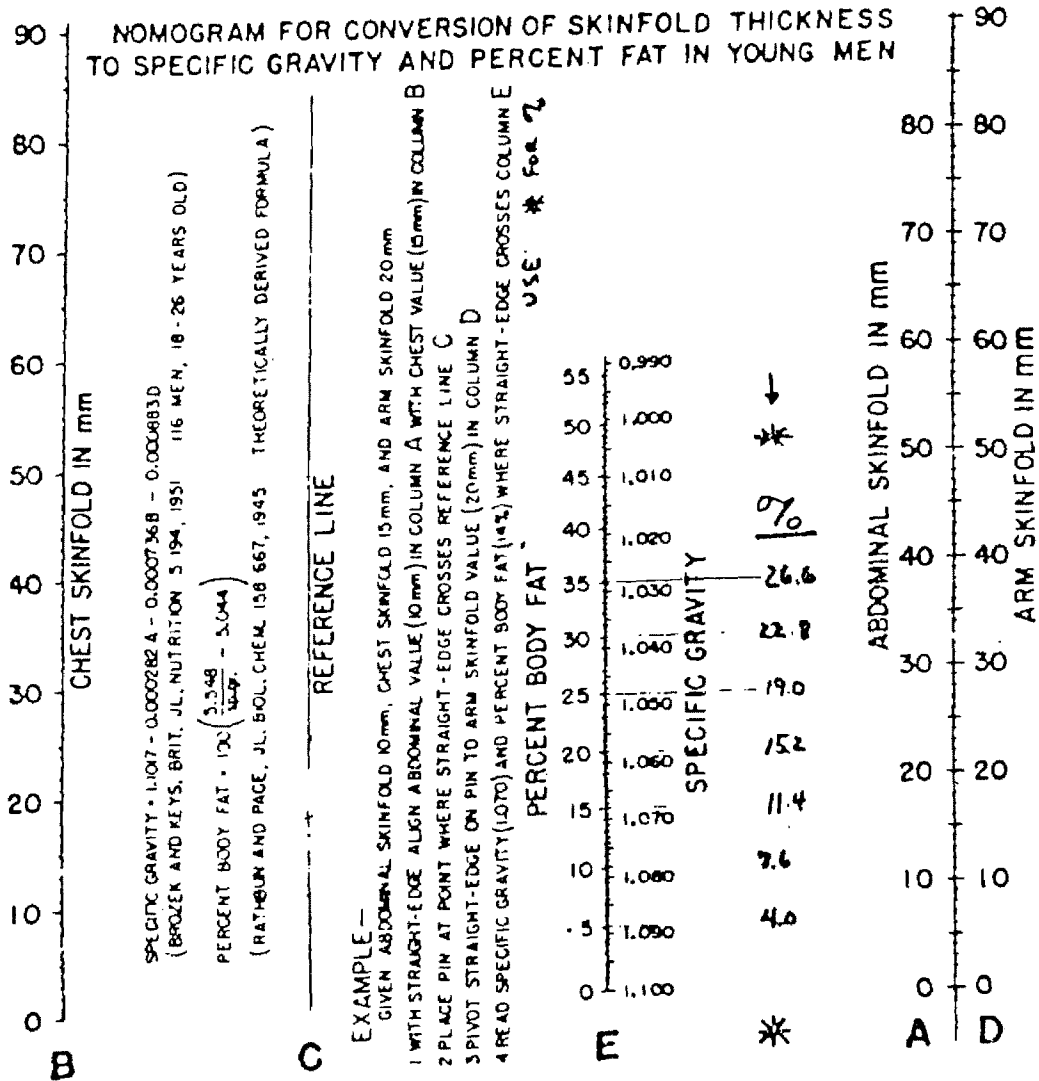
Name \_\_\_\_\_

Date \_\_\_\_\_

# APPENDIX B

## SKINFOLD NOMOGRAM

NAME \_\_\_\_\_



Nomogram taken from Consolazio, C.F., et al. *Physical Measurements of Metabolic Functions in Man*, New York, McGraw-Hill, 1965 by permission of authors.

APPENDIX C

MODIFIED BALKE TREADMILL PROTOCOL

Stage	Speed (miles per hour)	Duration (minutes)	Percent grade
1	3.4	2	4
2	3.4	2	6
3	3.4	2	8
4	3.4	2	10
5	3.4	2	12
6	3.4	2	14
7	3.4	2	16
8	3.4	2	18
9	3.4	2	20
10	3.4	2	22



APPENDIX D  
DIET REPORT FORM

Date _____	Date _____	Date _____
Breakfast	Breakfast	Breakfast
Between meals	Between meals	Between meals
Lunch	Lunch	Lunch
Between meals	Between meals	Between meals
Dinner	Dinner	Dinner
Snacks	Snacks	Snacks

## APPENDIX E

### PROCEDURE FOR TOTAL CHOLESTEROL DETERMINATIONS

The Hycel method for the determination of total cholesterol makes use of the Liebermann-Buchard reaction first described in the nineteenth century. The addition of concentrated sulfuric acid to a mixture of cholesterol and acetic acid produces a green color. The Hycel Stable Cholesterol Reagent, in commercial use since 1954, combines the sulfuric acid and acetic acid with acetic anhydride to form a single reagent.

The Hycel cholesterol test is calibrated with Hycel Cholesterol Standard, a primary standard containing 200 mg/dl of cholesterol in glacial acetic acid. The test requires only one calibration point. Hycel Normal Lipid Reference serum was used as the control.

#### BENCH PROCEDURE: TOTAL CHOLESTEROL, DIRECT SERUM METHOD

1. Mark 19 x 150 mm cuvettes B (blank), S (standard), and C (controls). Mark numbers for unknowns. Record each number assigned to the unknowns.
2. Pipette 0.1 ml of Hycel Cholesterol Standard and

of each control and unknown sample directly into the bottom of the correspondingly marked cuvette. Leave the cuvette marked B empty.

3. At timed intervals and beginning with cuvette B, add 6.0 ml of Stable Cholesterol Reagent to each cuvette. (Use a pipette with a bulb or reagent dispenser.) Mix well and place immediately in a 37 degree centigrade water bath.

4. Exactly 20 minutes later and in the same timed sequence, remove each cuvette from the water bath, wipe dry, mix by swirling, and measure the absorbance against the blank in a spectrophotometer set at 625 nm. A Johnson Junior Spectrophotometer was used.

#### BENCH PROCEDURE: RESULTS

The concentration of cholesterol in the unknowns is calculated according to the formula

$$\frac{A \times B}{C}$$

Where: A = absorbance of unknown

B = concentration of standard in mg/dl

C = absorbance of standard

## APPENDIX F

### PROCEDURE FOR SEPARATION OF HIGH DENSITY LIPOPROTEIN FROM SERUM

1. To 3 milliliter aliquots of the serum samples in 12 milliliter conical centrifuge tubes are added .12 milliliters of 5 percent aqueous dextran sulfate and .15 milliliters of 22.2 percent aqueous calcium chloride.

2. The tubes are thoroughly shaken on a vortex mixer, stoppered, and stored overnight at 2-4 degrees centigrade.

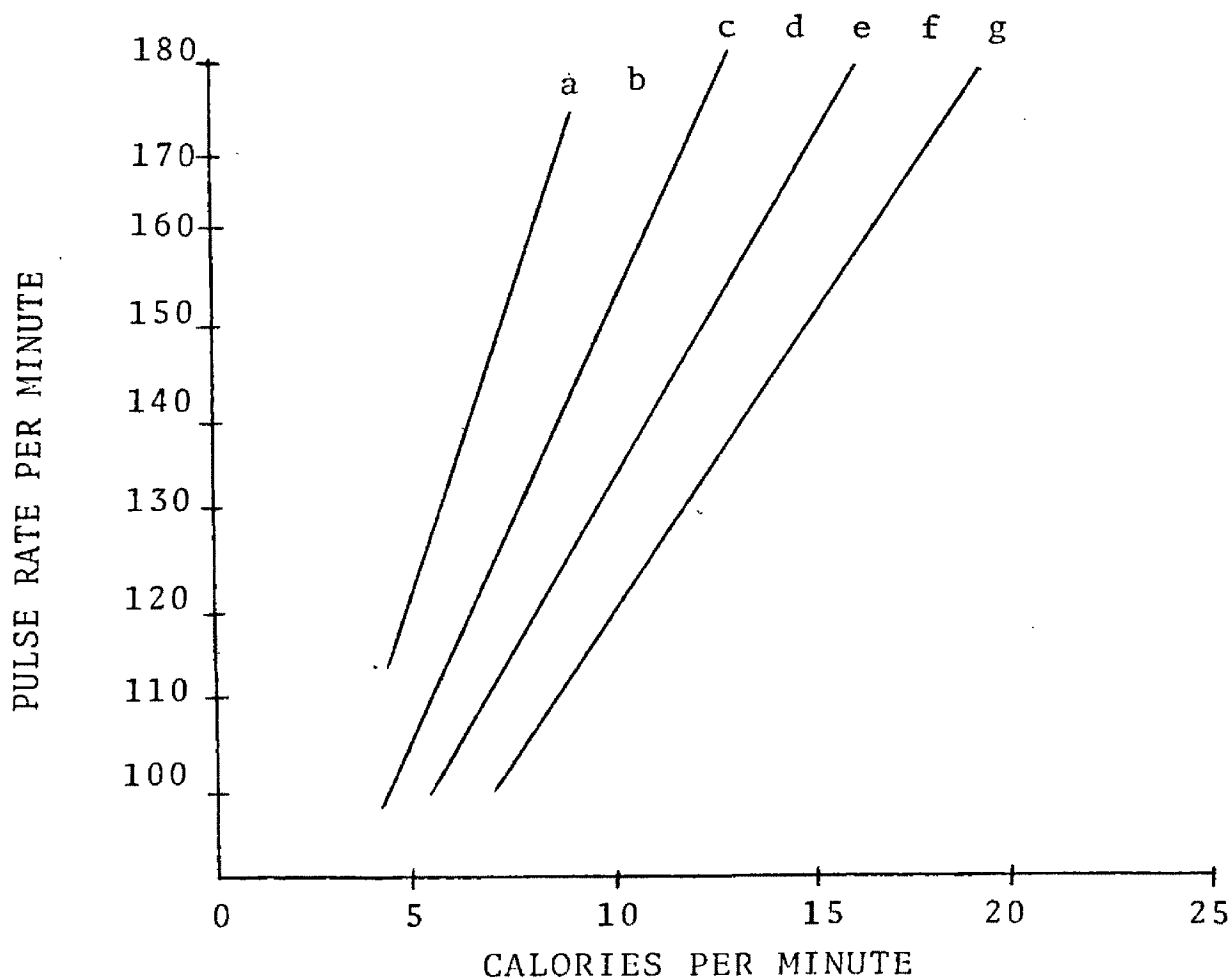
3. Centrifuge at 2 degrees centigrade for 20 minutes at 2000 revolutions per minute.

4. The clear supernatant is extracted for cholesterol analysis of HDL. The values are multiplied by 1.09 to correct for dilution.



APPENDIX H

PREDICTING CALORIES BURNED DURING PHYSICAL  
ACTIVITY FROM PULSE RATE



Fitness levels: a - Very Poor  
b - Poor  
c - Fair  
d - Good  
e - Very Good  
f - Excellent  
g Superior

APPENDIX I

SAMPLE TRAINING PRESCRIPTION

NAME  R.F.  AGE  26.3  GROUP  A

MAXIMUM HEART RATE (MHR)  192 Beats Per Minute (BPM)

TARGET HEART RATE @ 70 Percent MHR  135 BPM

@ 75 Percent MHR  144 BPM

@ 80 Percent MHR  155 BPM

CALORIES PER MINUTE  
EXPENDED

@ 70 Percent MHR  8

@ 75 Percent MHR  11

@ 80 Percent MHR  12

Week	Percent MHR	Target Heart Rate (BPM)	Duration (Calories)	Duration (Minutes)
1	70	135	200	25
2	70	135	225	28
3	75	144	250	33
4	75	144	300	27
5	80	155	350	29
6	80	155	400	33
7	80	155	400	33