The effect of mild aerobic physical activity on serum lipid, lipoprotein, and apolipoprotein concentrations in sedentary middle aged males

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THE EFFECT OF MILD AEROBIC PHYSICAL ACTIVITY ON SERUM LIPID, LIPOPROTEIN, AND APOLIPOPROTEIN CONCENTRATIONS IN SEDENTARY MIDDLE AGED MALES

A thesis submitted in partial fulfilment of the requirements for the award of the degree of

HONOURS MASTER OF SCIENCE

from

THE UNIVERSITY OF WOLLONGONG

by

JEREMY P. SHEARMAN, B.Phed.

DEPARTMENT OF HUMAN MOVEMENT SCIENCE
FACULTY OF HEALTH AND BEHAVIOURAL SCIENCES

1990
This thesis is dedicated to
the memory of
Judith Anne Margaret Shearman
who passed away
April 7, 1987.
The purpose of this study was to determine if mild aerobic physical activity effected specific serum lipid concentrations to a greater extent than other serum lipid concentrations.

Forty sedentary, healthy adult males (mean age 43.4 years) were divided into an untrained and trained groups. The trained members underwent twelve weeks of mild aerobic physical activity to determine the effects of mild aerobic physical activity on serum cholesterol, triglyceride, lipoprotein, and apolipoprotein concentrations.

The trained subjects showed a significant increase in aerobic capacity ($\text{VO}_2 \text{ max}$) from 33.2 ml.kg.min$^{-1}$ to 39.1 ml.kg.min$^{-1}$, while the untrained subjects remained stable at around 32.0 ml.kg.min$^{-1}$.

The serum cholesterol and lipoprotein concentrations showed no overall training effect. Both the untrained and untrained groups experienced a significant decrease in serum cholesterol (0.8 mmol/L & 1.1 mmol/L respectively) and low density lipoprotein (0.67 mmol/L & 0.84 mmol/L respectively) concentrations. Surprisingly, both groups showed a non significant decrease in high density lipoproteins of 0.19 mmol/L and 0.12 mmol/L respectively.
Meanwhile, the trained group showed a significant decrease in serum triglyceride (1.8 to 1.2 mmol/L) and apolipoprotein B (0.94 to 0.78 g/L). Although the untrained group also showed a significant decrease in apolipoprotein B (0.95 g/L to 0.88 g/L), the ratio of apolipoprotein A-1 to apolipoprotein B shown in the trained group was over double that of their untrained colleagues. This was due to the trained group maintaining a constant apolipoprotein A-1 level, which decreased in the untrained group, and the greater decrease observed in apolipoprotein B concentrations in the trained individuals.

From these data it appears that a twelve week mild aerobic physical activity program effects the serum triglyceride and apolipoprotein concentrations with greater consistency and effectiveness, than either serum cholesterol or lipoprotein concentrations.
This thesis dissertation could not have been completed without the help of many individuals, not all of whom can be acknowledged here.

Thank you to my supervisor, Dr. Graham Ward for his advice, guidance and understanding, and to all the staff from the Department of Human Movement Science at the University of Wollongong who contributed in any way.

A special thank you to the Forty dedicated subjects who remained dedicated to the study until the bitter end! Without you guys, there would be no study.

To the staff at the B.H.P. Port Kembla Steel Works Rehabilitation Services who helped in the organisation of subjects and overall support of this project, thank you also. Appreciation is expressed to Val Dukes at the Stress Laboratory, Dr. Gan and the Biochemistry Department, and the blood collection sisters at Wollongong Hospital. Also, thank you to all the volunteers who helped with the testing.

To my friends and flat mates here in Australia, thanks heaps guys for putting up with my financial and academic worries.
Finally, to family and friends at home, the biggest thank you to you all. Your financial and moral support was never more needed, and gained!!
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Abstract</strong></td>
<td>ii</td>
</tr>
<tr>
<td><strong>Acknowledgments</strong></td>
<td>v</td>
</tr>
<tr>
<td><strong>List of Tables</strong></td>
<td>xi</td>
</tr>
<tr>
<td><strong>List of Figures</strong></td>
<td>xiii</td>
</tr>
</tbody>
</table>

**CHAPTER**

## I INTRODUCTION

A. Context of the Problem

B. Statement of the Problem

C. Research Hypothesis

D. Delimitations

E. Assumptions

F. Significance of the Study

## II REVIEW OF THE LITERATURE

A. Physical Activity and Coronary Heart Disease

B. Pre Exercise Screening
   i) Exercise Capacity Evaluation
   ii) The Electrocardiograph

(continued . . . )
TABLE OF CONTENTS
(Continued)

C. Physiological Responses to Physical Activity .................................................. 27
D. Haemodynamic Responses to Physical Activity ............................................... 31
E. Exercise Capacity and the Cardiac Patient ......................................................... 39
F. Cholesterol, Triglycerides, and Lipoproteins ...................................................... 42
G. Serum Lipid Responses to Physical Activity ................................................... 49
H. Serum Apolipoprotein Responses to Physical Activity ...................................... 65
I. Serum Lipid Research - the Conflicting Results ................................................ 71

III METHODOLOGY
A. Selection of Subjects ......................................................................................... 75
B. Height, Body Weight, and Percentage Body Fat Analysis .................................. 77
C. Health and Fitness Questionnaire ..................................................................... 78
D. Dietary Evaluation ......................................................................................... 78

(continued . . . )
TABLE OF CONTENTS
(continued)

E. Serum Lipid Evaluations ....................................................7 8
   3.1. Specimen Preparation ..................................................8 0
   3.1.1. Serum Cholesterol and
           Serum Triglyceride ..................................................8 0
   3.1.2. High Density Lipoprotein ..........................................8 1
   3.2. Serum Cholesterol Analysis ..........................................8 1
   3.3. Serum Triglyceride Analysis ..........................................8 2
   3.4. High Density Lipoprotein
        Analysis .................................................................8 2
   3.5. Low Density lipoprotein and
        Very Low Density Lipoprotein
        Analysis .................................................................8 3
   3.6. Apolipoprotein A-1 and B
        Analysis .................................................................8 4

F. Cardiovascular Analysis ....................................................8 5
   3.7. Electrocardiogram Analysis ..........................................8 6
   3.7.1. Resting Electrocardiogram .........................................8 6
   3.7.2. Exercising Electrocardiogram .....................................8 7
   3.8. Heart Rate Analysis ...................................................8 8
   3.9. Blood Pressure Analysis .................................................8 9

(continued . . . .)
TABLE OF CONTENTS
(continued)

3.10. Aerobic Capacity (VO$_2$ max)
   Analysis ................................................................. 90

3.11. Aerobic Conditioning Program ................................. 91

G. Statistical Analysis .................................................. 93

IV. PROCEDURE
   A. The Testing Session ................................................ 95
      4.1. Equipment Calibration ....................................... 95
      4.2. Physiological Testing ....................................... 96
   B. The Aerobic Conditioning Sessions ............................. 97

V. RESULTS
   A. Subjects .................................................................... 99
   B. Heart Rate .............................................................. 100
   C. Haemodynamic Responses ......................................... 100
   D. Aerobic Capacity ..................................................... 101
   E. Cholesterol and Triglyceride ...................................... 102
   F. Lipoproteins ............................................................ 103
   G. Apolipoproteins ....................................................... 105

(continued . . . )
TABLES OF CONTENTS

(continued)

VI. DISCUSSION AND CONCLUSIONS

A. Weight, Percentage Body Fat, and Aerobic Capacity ........................................... 125
B. Cardiovascular Responses to Conditioning ................................................................. 128
C. The Effect of Mild Aerobic Physical Activity on Serum Cholesterol and Triglyceride ................................................................. 133
D. The Effect of Mild Aerobic Physical Activity on Serum Lipoproteins ......................... 136
E. The Effect of Mild Aerobic Physical Activity on Serum Apolipoproteins .................... 139
F. Summary and Conclusions ....................................................................................... 146
G. Recommendations for Future Study ......................................................................... 147

Bibliography .................................................................................................................. 149
Appendix A .................................................................................................................... 168
Appendix B .................................................................................................................... 171
Appendix C .................................................................................................................... 176
Appendix D .................................................................................................................... 179
Appendix E .................................................................................................................... 182
Appendix F .................................................................................................................... 184
# LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Percent Body Composition of Lipoproteins in Man</td>
<td>48</td>
</tr>
<tr>
<td>2. Means and Standard Errors for Physical Characteristics of Untrained and Trained Subjects (T = 0 Weeks)</td>
<td>107</td>
</tr>
<tr>
<td>3. Changes in Mean Physical Characteristics of Untrained and Trained Subjects from Initial (T = 0 Weeks) to Final (T = 12 Weeks) Testing</td>
<td>108</td>
</tr>
<tr>
<td>4. Pre and Post Conditioning Changes in Heart Rate Values for Untrained and Trained Subjects During Treadmill Testing</td>
<td>110</td>
</tr>
<tr>
<td>5. Mean Haemodynamic Responses During Physiological Testing for Untrained Subjects</td>
<td>111</td>
</tr>
<tr>
<td>6. Mean Haemodynamic responses During Physiological Testing for Trained Subjects</td>
<td>112</td>
</tr>
</tbody>
</table>

(continued . . .)
LIST OF TABLES
(continued)

7. Pre and Post Conditioning Changes in Serum
Cholesterol and Triglyceride Concentrations
for Untrained and Trained Subjects ........................................113

8. Pre and Post Conditioning Changes in Serum
Lipoprotein Concentrations in the
Untrained Subjects ......................................................................116

9. Pre and Post Conditioning Changes in Serum
Lipoprotein Concentrations in the
Trained Subjects ..........................................................................117

10. Pre and Post Conditioning Changes in Serum
Apolipoprotein A-1 and B Concentrations in
Untrained and Trained Subjects ......................................................121

11. Summary of Significant Pearson's Correlations
Between Age, Weight, % Body Fat, And
VO$_2$ max and all Serum Lipid Concentrations .................................124
LIST OF FIGURES

FIGURE

1. Changes in aerobic capacity (VO₂ max) for untrained and trained subjects observed over 12 weeks ................................................................. 109

2. Mean cholesterol levels for untrained and trained subjects expressed over time ....................................................................................... 114

3. Mean triglyceride values for untrained and trained subjects expressed over time ..................................................................................... 115

4. Mean high density lipoprotein (HDL) and low density lipoproteins (LDL) values for untrained and trained subjects expressed over time ........................................................................ 118

5. Percentage of high density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) present in total cholesterol at T = 0 weeks (fig. 5a) and T = 12 weeks (fig. 5b) for untrained subjects ........................................ 119

(continued . . .)
LIST OF FIGURES
(continued)

6. Percentage of high density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) present in total cholesterol at T = 0 weeks (fig. 6a) and T = 12 weeks (fig. 6b) for trained subjects ...........................................120

7. Mean apolipoprotein A-1 and B levels for untrained and trained subjects expressed over time ..........................................................122

8. Changes in the ratio of apolipoprotein A-1 to apolipoprotein B for untrained and trained subjects expressed over time .....................123
CHAPTER I

INTRODUCTION

Context of the Problem

The concept of prevention of disease seems very applicable in the case of coronary heart disease. As public awareness of the complications associated with coronary heart disease increases, people are viewing prevention as a viable alternative to currently used treatments in reducing the risk of this disease. With many sophisticated diagnostic techniques now available, it would be reasonable to assume that the ability to detect and treat coronary heart disease would have greatly improved. However, the increased knowledge of causes of coronary heart disease and the superior pharmaceutical drug therapies and surgical intervention regimes currently employed, have not been globally accepted as being sufficient in reducing the incidence of coronary heart disease.

One alternative to these contingencies in the battle against coronary heart disease is the primary prevention and secondary treatment of the disease with lifestyle modifications. The secondary treatment of coronary heart disease is specifically termed cardiac rehabilitation and involves drug therapy, counselling, dietary and life-style modification as integral components of the rehabilitation process. Brooks and
Fahey (1984) reported that physical activity in the rehabilitation stages of coronary heart disease can help restore an individual's physiological and psychological competence. Despite the amount of research in the area, the overall relationship between non pharmaceutical intervention, including physical activity, and coronary heart disease is still far from clear (Edye, Mandryk, Frommer, Healey, & Ferguson, 1989, Shephard, 1986). It is also evident when examining the recent data relating to the incidence of coronary heart disease in Australia, that there still remains great scope for investigation.

Coronary heart disease is presently the second leading cause of mortality and morbidity in Australia. In the decade up to 1989, the death rate in Australia from coronary heart disease has remained constant at 29 per cent (Hopkins, 1982). In 1982 coronary heart disease accounted for 57,696 deaths in Australia (Grant & Lapsey, 1984).

Heart disease has often been thought of as a disease of old age. This is far from correct. Although the incidence rate increases with age, studies by Vaccaro & Mahon (1989) and Wilmore & McNamara (1974), have shown the beginning stages of coronary heart disease in American children less than 5 years of age. Furthermore, 62 per cent of children between the ages of 7-12 years had at least one coronary heart disease risk factor (Gilliam, Catch, Thorland, & Weltman, 1977) while 14 per cent of children exhibited two or more risk factors (Wilmore & McNamara, 1974).
Heart disease mortality rates are higher in the male population. In 1978, 32.5 per cent of all deaths in the Australian male population were attributed to coronary heart disease, compared to 27.0 per cent of all female deaths (Hopkins, 1982). In 1980, 23 per cent more Australian males died from heart disease per 100,000 population than their female counterparts (Grant & Lapsey, 1984). New Zealand data have a similar pattern to Australia. In 1978, in excess of twice as many 65-74 year old males died from heart disease than New Zealand females (Hyslop et al, 1983).

From these data, it is clear that coronary heart disease and more specifically, prevention and rehabilitation are major present day health issues. In the last twenty years, the general public have developed a greater awareness of the risk factors associated with coronary heart disease (Cummings, Barton, Fahey, Wilson, & Leeder, 1989), and thus heart disease mortality has declined (Jamrozik, & Hockey, 1989). In New Zealand for example, the mortality rate in white males has declined by 17 percent in the decade up until 1979 (Hyslop et al, 1983). This decline has been associated with a multitude of factors, including dietary intervention (especially decrease in dairy products, red meat, saturated fats, and salt intake), an increased awareness of the risks of cigarette smoking, recognition of the importance of hypertension control, and an apparent increase in habitual physical activity in the community (Gill et al, 1989; Hyslop et al, 1983).
According to Brooks & Fahey (1984), most of the deaths are potentially preventable by modifying life-style activities. The genetic risk factors, including age, sex, and an individual's ability to metabolise lipoproteins are perhaps the only non life style related risk factors we cannot control (Schaefer, McNamara, Genest, & Ordovas, 1988). To what extent increased habitual activity can decrease the risk of coronary heart disease is not yet clear. Data do support a causal relationship between a lack of physical activity and coronary heart disease (Goldberg & Elliot, 1987; Joseph & Bena, 1977; McNaughton & Elliot, 1987; Shephard, 1986). This thesis will therefore investigate the effects that physical activity has on both the cardiovascular function and the lipid profile and will attempt to determine if the relationship between the two is causal in nature.

In attempting to establish the extent of the relationship, this thesis will in particular investigate six major serum lipid components in response to aerobic physical activity. These serum lipid components are; (1) total serum cholesterol, (2) high density lipoproteins, (3) low density lipoproteins, (4) very low density lipoproteins, (5) triglycerides and (6) apolipoproteins A-I and B. Cardiovascular functioning in response to physical activity will also be investigated by measuring aerobic capacity and examining recordings of blood pressure, heart rate, and the activity of the electrocardiogram.

To analyse the effect that physical activity has on coronary heart disease, requires an understanding of the mechanisms of heart disease. Simplified, the disease process appears to involve the
build up of atherosclerotic plaques in the coronary arteries, sometimes followed by thrombosis, resulting in decreased myocardial blood flow. This decreased blood flow occurring in the atherosclerotic arteries is a slow, progressive process.

Atherosclerotic plaque development is thought to begin with disruption of the arterial endothelium which is found on the inner coat of the arterial wall. This often allows plasma substances including lipids, and in particular cholesterol, to be deposited into the intima. Along with the increase of plasma borne substances, there is frequently an increase in smooth muscle present in the vessel lumen (Brooks & Fahey, 1984), which in turn causes the arteriovascular space to become restricted. This proliferation of the smooth muscle cells is stimulated by platelets and monocytes which release serotonin, epinephrine and adenosine diphosphate (A.D.P.), at the injury site (Lamb, Ingram, Johnston, & Pitman, 1984).

In many cases, the plaques take over twenty years to cover the entire circumference of the artery. The arterial occlusion process is accelerated by factors such as hyperlipidemia and hypertension. The plaque can often become calcified, and connective tissue may form. This produces a narrow, rigid blood vessel, which is commonly referred to as "hardening of the arteries" (Astor & Roth, 1980; Brooks & Fahey, 1984; Fox & Mathews, 1981; Groer & Shekleton, 1983; Guyton, 1982; Krupp & Chatton, 1984; Miller, 1983; Pelkovic, 1981; Ramsey, 1982).
It is generally accepted that the total lipid profile or the amount and types of lipids in the circulating plasma, appear to contribute to the formation of coronary heart disease (Astor & Roth, 1980; Brooks & Fahey, 1984; Fox & Mathews, 1981; Kannel, Castelli, & Gordon, 1979). Furthermore, the association of serum lipid level reduction with physical activity has frequently been made (Christie, Bloore, & Logan, 1980; Gaesser & Rich, 1984, Goldberg & Elliot, 1987; Hall, Meyer, & Hellerstein, 1984; Shephard, 1989).

Based on the research data in this field, there is little doubt that a lack of physical activity contributes to the risk of coronary heart disease. It has been suggested by numerous authors (Astor & Roth, 1980; Astrand & Rodahl, 1970; Shephard, 1986; Wenger, 1978) that the lack of physical activity causes an overall reduction in body fitness which may contribute towards obesity, a factor strongly associated with coronary heart disease. Despite this, in over two decades of research, the specific role of physical activity with respect to heart disease is yet to be identified. However, many researchers have supported the need for an active lifestyle, which may lead to a reduction in coronary heart disease (Astor & Roth, 1980; Gaesser & Rich, 1984; Golding, 1961; Shephard, 1986, 1989). These include:

* males in sedentary occupations or males undertaking minimal physical activity, seem to have a higher rate of myocardial infarction than those engaging in regular physical activity.

* conditioning via physical activity improves "work efficiency" of the body.
* progressive physical activity or conditioning regimes, seems to improve tolerance to work.
* regular endurance activity produces increased cardiac efficiency.

(Astor & Roth, 1980).

Physical activity is only one factor which has been associated with serum lipid levels, which may also be effected by the nutritional intake of any given individual. Thompson et al. (1984), in an examination of the effect of caloric restriction and cessation of training on lipid concentration, employed a control group which continued to train 112 miles per week and consumed a diet containing 3670 kilocalories per day. Two subject groups also trained 112 miles per week but consumed a diet containing 20 per cent fewer calories. The study found that all three groups reduced their low density lipoproteins levels by 10 to 15 per cent. Both of the exercising groups had similar serum lipid profiles (total cholesterol 163-166 mg/dl, triglycerides 46-62 mg/dl). Even after one of the exercising groups ceased to train, there were only small additional reductions in low density lipoproteins observed in the group which continued to exercise. The subjects who ceased to train showed a 10 per cent increase in low density lipoproteins after only two days. Cook (1958) also found that diets consisting of less than 10 per cent fats showed more dramatic reductions in cholesterol concentration. Unfortunately, the patient condition and health became an issue, because of gastrointestinal disturbances, flatulence, depression, apathy, and poor muscle tone, finally resulting in the studies being concluded.
Studies by Thompson et al. (1984) and Cook (1958) raised questions about the effect of physical activity on serum lipids. Thompson et al. (1984) demonstrated in their study that physical activity may effect the levels of serum lipoproteins. Blood vessels and muscle tissue are thought to play an important role as they "massage" to increase the removal of lipids from vessel walls, via the lymphatic system. In support of this, Cook (1958) previously reported that atheromas appear more often in sites where the vessels are not subject to the "massaging" action of these tissues, such as below the chest wall in the thoracic cavity and the abdomen.

Another study has disputed that a relationship exists between physical activity and the lipid profile. Despres, Bouchard, Savard, Tremblay, & Allard (1985) reported that there is a lack of a causal relationship between physical activity and plasma lipids. The study, lasting 20 weeks, showed although there was a reduction in obesity (mean loss 3 kg) and an overall decrease in total cholesterol (mean decrease 14.5 mg/dl), there were no significant changes in high density lipoproteins, triglycerides, or the ratio of high density lipoproteins to total cholesterol.

Many questions regarding the relationship between physical activity and the lipid profile remain unanswered. Theories are now being forwarded relating to the ratios of the different serum lipid subfractions (high density lipoproteins, low density lipoproteins, very low density lipoproteins, and apolipoproteins) to total cholesterol, and how these ratios affect coronary heart disease (Goldberg & Elliot, 1987; McNaughton & Davis, 1987; Streja
& Mymin, 1979). Triglycerides, the most common form of dietary lipids, have also been investigated and may well hold the key to the dilemma of reduction of coronary heart disease (Gaesser & Rich, 1983).

In conclusion, it may well be very prudent to ask what effect physical activity has on the lipid profile and the lipid subfractions. The following literature review will investigate the relationship between physical activity and the lipid profile. In attempting to establish whether a causal relationship does exist between physical activity and coronary heart disease, it will be necessary to discuss the relationship between physical activity and the lipid subfractions, including cholesterol, triglycerides, lipoproteins, and apolipoproteins.

Reference to the relationship between physical activity and blood pressure (especially hypertension), body weight (especially obesity), the heart profile (heart rate and the electrocardiogram) and other related life-style habits including smoking, and stress, will complete the assessment of overall coronary heart disease risk and physical activity.
Statement of the Problem

Because serum lipids and apolipoproteins, have been identified as cardiac risk markers, this study aimed to determine whether a 12 week program of mild aerobic physical activity changes the serum lipid concentrations in sedentary, low fit, middle aged males.

The aerobic conditioning regime used was defined as mild aerobic stage I aerobic physical activity, as this form of physical activity has been shown to be most effective in improving individual lipid profiles. Two groups were randomly selected, in an attempt to rule out selection bias influencing physiological and biochemical responses to the aerobic conditioning program.

Research Hypothesis

1) Subjects partaking in mild aerobic physical activity for 12 weeks, and maintaining a static diet, will show significant changes in serum lipid concentrations compared to the non active control group.

2) Subjects partaking in mild aerobic physical activity for 12 weeks, and maintaining a static diet, will show significant changes in serum lipid concentrations compared to their initial before training concentrations.
3) Subjects partaking in mild aerobic physical activity for weeks, and maintaining a static diet, will show a significant increase in maximal aerobic capacity at the conclusion of the twelve week training period.

Delimitations

A) All subjects were male employees from B.H.P. Steel, and the Wollongong City Council between 35 and 60 years. They were healthy, sedentary, and unfit. This however reduced the ability of the study to draw conclusions on gender and age of the general population of Australia.

B) Forty subjects were included in the study, following consultation with a university statistician. It was shown that 36 subjects were sufficient to produce a statistically significant result. However, the small selected population of this study limits the ability of the researchers to draw conclusions on the sample's representativeness of the population of Australia.

C) Time constraints necessitated the period of physical activity undertaken to be restricted to 12 weeks. Although this period of physical activity has been shown to induce significant changes in serum lipid concentrations, a longer period may have confirmed the long term effects of physical activity.
Assumptions

The following assumptions were made for this study:

A) All subjects adhered to the physical activity protocols and dietary programs as instructed.

B) All subjects accurately completed the questionnaires on health status and previous physical activity levels.

C) All subjects accurately recorded dietary intake during the 24 recall time periods.

D) The control group did not become contaminated with the treatment variable (partaking in physical activity).

Significance of the Study

This study contributes to existing data pertaining to the effectiveness of physical activity in reducing coronary heart disease risk. Specifically, the relationship between physical activity and serum lipid subfractions, particularly apolipoproteins A-1 and B is important, with the emergence of apolipoproteins in recent years as a more sensitive and valid indicator of coronary heart disease risk than other serum lipid subfractions.
It is hoped that some of the apparent benefits of aerobic physical activity in relation to serum lipid concentrations will be supported, and that this may contribute to the development of a more effective regimen of physical activity with the aim of reducing the incidence of coronary heart disease.
CHAPTER II

REVIEW OF THE LITERATURE

Medical experts and research scientists have for many years lauded the benefits of physical activity with respect to coronary heart disease prevention. Significant changes in kinanthropometric dimensions, cardiovascular functioning, and the blood profile have been repeatedly shown to occur with physical activity. Furthermore, significant changes have been shown to occur in specific, highly sensitive serum lipid subfractions, in response to various aerobic training regimes.

Physical Activity and Coronary Heart Disease

According to Ferguson (cited by Berridge and Ward, 1987), physical activity is important in the prevention of coronary heart disease. Physical activity can be used as a diagnostic technique, enabling the measurement of the individual's level of incapacity and also may be used with medical and surgical interventions. Thirdly, physical activity can be employed to investigate the efficacy of pharmaceutical cardiac therapies, especially antianginal drugs. Ferguson (cited by Berridge and Ward, 1987) also reported that physical activity was now accepted as an integral part of many cardiac rehabilitation programs.
Conversely, physical inactivity is a listed risk factor for coronary heart disease (American College of Sports Medicine, 1986), whilst participation in physical activity serves a protective function against coronary heart disease (Basmajian, 1984). The reduction of risk for active individuals compared to inactive individuals is 3-fold, with the incidence of sudden cardiac death being similarly reduced (Basmajian, 1984). Earlier studies have shown that these findings are not universally accepted. According to Basmajian (1984), the failure of the earlier studies to show a positive relationship between physical activity and coronary heart disease risk was due to bias and pre-selection of the subjects. More recent studies such as those carried out by Pfaffenbarger (1977, 1979), and Morris, Everett, Pollard, & Chave (1980) have used complex statistical analysis and methodology designs to demonstrate that physical activity acts independently of any other known risk factor for coronary heart disease.

In an American College of Sports Medicine publication (Blair, Painter, Pate, Smith, & Taylor, 1988), it is suggested that physical activity intervention with patients in whom several risk factors have manifested poses many practical problems. Two such problems that arise and were reported by Hjermann (1981) are the multitude of ways in which cigarette smoking can effect the functioning of the cardiorespiratory system, and secondly, how consistently do dietary interventions succeed with high risk patients? However, Blair et al. (1988) pointed out that after the subject reaches a stable physical activity pattern, other reductions in risk categories may be more feasible. These include reduction of cigarette smoking and implementation of dietary interventions
(Hjermann, Velve-Byre, Holme, & Leren, 1981). Thus, there may be sufficient justification for the inclusion of physical activity programs for coronary heart disease patients on the basis that exercise may facilitate other life-style modifications.

Sutton (1979a), in a paper presented during a symposium on exercise and the post coronary patient, examined whether physical activity is of benefit in the management of the post coronary patient, and further discussed research shortcomings in the area of cardiac rehabilitation. Several reasons were forwarded for the apparent lack of a definitive relationship between physical activity and the reduction of coronary heart disease not being obtained from previous studies. Most studies were retrospective, which may lead to a selective population being incorporated in any given study. Thus, rehabilitation programs may appear to be beneficial or worthless when, in actual fact, their success has been incorrectly gauged. The logistics alone involved in completing a study considered substantial enough to draw significant conclusions are daunting. Furthermore, quality control during studies warrants closer scrutiny, apparent when one considers the methods of obtaining physiological data such as oxygen uptake measurements. This is especially the case when several centres are involved in the same study and reproduction of the results is often questionable or unobtainable due to equipment differences alone (Sutton, 1979a).

Shephard (1979, 1989) highlighted one of the problems with research relating to cardiac rehabilitation in his study addressing the status and prospects of exercise programs for cardiac
rehabilitation patients. Shephard (1979) reported that the Ontario Multi Centre Trial which included follow-up data over a two year period, may have yielded more substantial data if the study had been continued for several more years. However, attrition, non-compliance with the prescribed exercise programs, and contamination of the control group with interest in physical activity may have affected the data gained. Thus, experimental design, specifically the length of the study and reduction of unwanted contamination of subject and control groups, requires special attention.

The design of the experimental protocol was also addressed by Rechnitzer (1979), who suggested that the population and sample size for studies concerning coronary heart disease are vital. The experimenters must take into account the length of the study, and the length of the period of observation because of the very high attrition rates experienced (Rechnitzer, 1979).

Another problem existing with the data available pertaining to physical activity and coronary heart disease is the lack of studies addressing the relationship between physical activity and increased cardiovascular mortality. Two such studies were completed by Ekelund et al. (1988), and Redwood, Rosing, and Epstein (1972). Ekelund et al. (1988) examined this question in a study incorporating 3106 male subjects in the Lipid Research Clinic Prevalence Study, observed over an average 8.5 year follow up period. The participants were divided into two groups, one consisting of healthy individuals whilst the second group contained patients suffering from cardiovascular disease.
Sub-maximal treadmill exercise tests were carried out on all participants, using a seven step 3-minute protocol commencing at 2.2 kilometres per hour (kph) at 10 per cent inclination, increasing to 5.5 kph, at 15 per cent inclination. Heart rate and blood pressure were regularly monitored with an electrocardiograph trace also taken. Fitness levels were assessed using heart rate in the healthy group, and the duration of the test in the cardiovascular disease group, as these individuals usually terminated the test during stage one.

The results of the experiment performed by Ekelund et al. (1988) were assessed after dividing the healthy group into four sub-groups on a basis of heart rate during the second increment of the test to account for fitness of this group. The first finding of this study was that the "fittest" healthy group members had a significantly lower exercising heart rate at the second increment test protocol of 112 beats per minute (beats/min) as compared to 156 beats/min in the "least fit" healthy group. Thus, an individual with a higher level of fitness, may have been placed under less stress, with respect to cardiovascular functioning. Furthermore systolic blood pressure was 7 mm Hg lower in the "fittest" healthy group, and resting heart rate was 10 beats/min slower compared to the "least fit" healthy group. The relative risk of death from exercise was evaluated using the Proportional Hazards Model (Cox, cited by Ekelund et al, 1988), and the findings suggest that the cardiovascular disease patients had an increased risk of sudden death from coronary heart disease of 2.8 times greater than the healthy individuals. Ekelund et al. (1988) also concluded that physical activity was more important than genetic factors in
determining one's fitness level and may also be an important factor in reducing coronary heart disease. Overall, higher cardiovascular fitness levels were associated with improved coronary heart disease risk factor profiles, and thus, decreased levels of cardiovascular fitness may result in increased mortality during exercise.

Sudden death has also been recorded during sporting events. Noakes (1987), stated that 36 cases of heart attack or sudden death had been recorded in marathon races. Marathon runners and especially those with a family history of heart disease should not consider themselves immune to either sudden death or coronary heart disease. Most likely, according to Noakes (1987), the cases of sudden death or infarctions are under-reported in which case, the frequency of cardiac mishaps during such events is hard to ascertain. It is apparent though that sudden death may be a significant hazard of exercise for coronary heart disease sufferers and that some precautions should be taken to decrease this risk, such as preparing realistic exercising regimes.

It follows that some scepticism may exist as to what role physical activity can play in reducing coronary heart disease. Shephard (1986, 1989), in two major review articles suggested that it may take another century to gather the data proving that physical activity does significantly effect the incidence of coronary heart disease. However, in the interim, it may be advisable to accept that physical activity is of benefit in secondary and tertiary management of coronary heart disease.
Although the research into physical activity and the effect on coronary heart disease has not been conclusive, many exercise responses in the post myocardial infarct patient have been well documented (Cunningham, Ingram, & Rechnitzer, 1979; Gaesser & Rich, 1984; Jelinek, 1988; McNaughton & Davis, 1987; Sutton, 1979a, 1979b).

**Pre Exercise Screening**

i) Exercise Capacity Evaluation

Before the cardiac rehabilitation patient becomes involved in exercise programs, a graded exercise test (GXT) is often carried out, to decrease the likelihood of the individual suffering further trauma. Abbott et al. (1989), in an experiment investigating the association between exercise endurance and cardiovascular risk factor profiles, studied 2606 young and middle aged adults (15-59 years) in the Framingham Offspring Study. The participants were examined in terms of the length of time they were able to walk or run on the treadmill before reaching an age-sex specific target heart rate. Testing was terminated with the onset of any contraindicated symptoms of exercise, such as fatigue, weakness, exercise-induced ST segment abnormalities, chest pain, arrythmia, or a mean blood pressure drop of over 30 mm Hg. A total of 727 subjects were excluded from the study by failing to fulfil one or more of the criteria for continuation. These factors included having any form of heart disease, resting ST segment depression, or currently receiving pharmaceutical medications. Ninety three per cent of the participating subjects terminated exercise upon
reaching their target heart rate or because of fatigue or weakness. The next most common attributions for cessation were ST segment depression (1.6 per cent), bigeminy (1.9 per cent), and premature ventricular complexes (1.1 per cent). These data suggest that an individual's ability to endure exercise may be associated with cardiovascular disease risk. Despite these findings, no cause and effect relationship was found.

It may be possible that risk factors which predict coronary heart disease are responsive to exercise conditioning (Abbott et al, 1989). Ninety three per cent of subjects terminated exercise after reaching their target heart rate, rather than at their maximal heart rate, making the use of the treadmill in testing questionable, especially at sub maximal levels. This is because one can not gain a true indication of maximal aerobic capacity if the subject terminates testing at a target heart rate. Furthermore other factors, such as cigarette smoking and alcohol, may have more short term rather than long term effects on treadmill performance, including alterations in blood pressure, rhythmic defects, and myocardial irritability (Abbott et al, 1989). The results of this study suggest that poor exercise endurance in asymptomatic adults requires further investigation, because it is likely to be associated with a poor risk factor profile.

Another investigation into exercise testing monitored early mobilisation in post myocardial infarction patients (Jelinek et al. 1977). This study examined the practice of rapid mobilisation after myocardial infarction, and exercise testing to determine the patients' capacity for physical activity, and the rate of recovery of
Ten subjects were initially selected from a group of patients admitted to the coronary care unit at St. Vincent's Hospital in Melbourne. All of the subjects selected fulfilled the following criteria. Their age was under 60 years (mean age 51.7 years), and they had no evidence of congestive heart failure. Cardiac abnormalities including intraventricular conduction defects, tachycardia, atrial flutter, or fibrillation were not present during their period in coronary care. Eighteen patients were subsequently selected in a sub-group for the study which investigated the rate of recovery of exercise tolerance post infarct.

Final selection in the study depended upon the subjects having suffered from their first infarct, which was transmural in nature, with no evidence of psychological imbalance. No pharmaceutical interventions were to be used during the study, and all of the subjects agreed to the study protocol. A standard twelve lead electrocardiograph was performed to eliminate any new ischemia, and all but one subject produced abnormal Q waves, ST segments, or T waves at the time of the first post infarct test. The subjects completed a monitored isometric test using a hand dynamometer where they were asked to perform a 50 per cent maximum voluntary contraction and, maintain it for as long as possible. A cycle ergometer test was also completed, with the test being terminated due to exhaustion or leg pain. Blood pressure, heart rate, and electrocardiograph traces were also recorded (Jelinek et al, 1977).
The results showed that all patients ceased exercise due to symptoms of fatigue, with the exception of three subjects who suffered from various anginal chest pains. The duration of maximum cycle ergometer exercise increased by 18 per cent from the first week of testing to the fifth week post infarct. Maximal voluntary contraction increased from 42.7 kilograms to 45.3 kilograms, while a 50 per cent maximum voluntary contraction could be sustained for 195.3 seconds at week five, compared to only 149.3 seconds during testing at week one. Maximum heart rate increased from 100.3 beats/minute to 107.5 beats/minute during exercise over the five week period, but no significant change in blood pressure during isometric exercise was observed. It was suggested by Jelinek et al. (1979), that subjects such as these may benefit from early exercise testing and mobilisation. One suggested reason for the improvement of exercise tolerance was an increase in participant self confidence, which resulted in subjects being prepared to try harder in subsequent testing. Most importantly, the authors believed that exercise tolerance had almost returned to normal eight days post uncomplicated myocardial infarction, relative to the subjects self confidence. These findings indicate that with careful planning and implementation of rehabilitation programs, the subjects can successfully return to most forms of work, leisure, and sexual activity in two to four weeks after the initial infarction.

Jelinek et al. (1979) stated that exercise testing is safe after myocardial infarction provided suitable medical and technical staff are in attendance, to constantly monitor the
electrocardiograph, and that resuscitation equipment is available, and the subjects involved have uncomplicated myocardial infarctions.

It has been shown that when exercise testing high risk groups such as cardiac patients, use of the heart rate response is often questionable (Powles, Sutton, Wicks, Oldridge, & Jones, 1979). In these individuals, this method offers no safety margin. Furthermore an extrapolation of sub maximal data can lead to an over estimation of aerobic capacity (Powles, et al, 1979). With cardiac patients, other monitoring methods, including the electrocardiograph, should be used.

ii) The Electrocardiograph

The electrocardiograph has been fundamentally employed to monitor the progress of patients following cardiac trauma. However, an increasing number of studies have specifically looked at the electrocardiograph responses to physical activity (Ehsani, et al, 1981; Ogirimah, et al, 1974). The electrocardiograph provides an indirect measure of the heart functioning and can be used as an integral part of pre-activity fitness evaluation. Ogirimah et al. (1974) compared the effects of two different exercising programs on the electrocardiogram of patients with previous myocardial infarction. Twenty-two male subjects (mean age 49.7 years) undergoing cardiac rehabilitation, were used for this study. Each participant was randomly assigned to one of two groups. The first group took part in a program of jogging and calisthenics, with the major emphasis being on the jogging phase.
This exercise was considered to be moderately heavy. The second group took part in a series of activities including recreational swimming, volleyball, and bowling. This activity was considered as light exercise. During the twenty-five week study, the resting and warm up heart rates of both groups were monitored. The time period for which a heart rate above 130 beats/minute could be maintained was also recorded (Ogirimah et al, 1974).

A twelve lead electrocardiogram trace was obtained at the beginning of the program and again at the end of the 25 week study. The subjects walked on a treadmill at an initial speed of 3.0 mph at zero gradient for three minutes. The speed of the treadmill was maintained whilst the gradient was raised 3 per cent every three minutes. Thereafter, the subjects continued to walk until a heart rate of 160 beats/minute or exhaustion was reached.

The purpose of this study was to investigate if there were any quantitative electrocardiograph changes and differences in the two groups of cardiac patients. Overall, the differences between the two groups were minimal. The reasons reported for stopping the treadmill test changed from the pre-program test to the post-training test. Nine per cent of the subjects ceased the test due to S-T segment depression during initial testing, while no subjects in either group stopped the test due to S-T segment depression during the post training test. The jogging group showed a significant improvement in time to exhaustion when walking (8.6 to 14.0 minutes) after the training program, whilst the games group showed a non-significant increase (9.0 to 11.2 minutes).
The lack of significant differences in the electrocardiograms between the two types of exercise did not support previous research findings in which modification of the S-T segment following exercise suggested improved myocardial blood flow (Ogirimah et al, 1974).

Ehsani et al. (1981) completed a study which suggested that an intense, prolonged exercise program can result in reduced myocardial ischemia as indicated by a decrease in S-T segment displacement during initial exercise testing. Ten cardiac rehabilitation patients, aged 44-63 years participated in a twelve month exercise program. The program consisted of an initial ten minute period of calisthenics, stretching, and walking, followed by 30-60 minutes of endurance exercise. This was then followed by alternate walking and jogging or alternate jogging and bicycle ergometer sessions. During the first three months, the subjects exercised at 50-70 per cent of their maximum oxygen uptake, thereafter increasing to 70-90 per cent. Patients were instructed to exercise three times per week in the first three months and five times per week over the following nine months. Exercise testing sessions, including an electrocardiogram were completed before, and immediately following the twelve month exercise program.

The results of the exercising electrocardiograph tests showed that the extent of S-T segment depression in the exercising group, expressed as a double product maximum, was decreased after training (24.6 bpm/SBP/10 to 23.7 bpm/SBP/10). The double product maximum is a threshold measure of the heart rate (bpm)
with systolic blood pressure (SBP) required to induce a 0.1 millivolt depression of the S-T segment. This threshold is used to indicate ischemia, by locating at which intensity ST segment displacement first occurs. The untrained group showed a similar small decrease in the double product maximum for S-T segment depression. Overall, the S-T segment depressions decreased in six of the ten exercising patients whilst the segment depressions increased significantly in the untrained group. A resting electrocardiograph showed that the QRS voltage had significantly increased in the training group from 2.7 millivolts to 3.1 millivolts, indicating ventricular enlargement. These findings suggest that prolonged and intense exercise may facilitate cardiac adaptations which result in the increased double product threshold for myocardial ischemia. This response can be readily detected during the exercising electrocardiograph.

Physiological Responses to Physical Activity

Following the initial evaluation of the health status of post myocardial infarct patients, these individuals may then partake in graded physical activity, under the supervision of qualified fitness experts and the cardiologist. The physiological responses of the cardiac patient to physical activity have been widely investigated in studies such as that completed by Sutton (1979b). This study demonstrated that physiological responses may change under differing testing procedures. The subjects were enrolled in the Canadian Exercise-Heart Collaboration study, and performed exercise on both the treadmill and cycle ergometer. Heart rates, electrocardiographs, and ventilation data were recorded for each
subject. From the ventilation data, several variables were calculated including peak oxygen uptake (VO$_2$), expired ventilation (VE), carbon dioxide output (VCO$_2$), respiratory exchange rate, dead space to total volume ratio (Vd/Vt), and cardiac output.

The results showed that all of the 40 male subjects achieved a higher peak oxygen uptake on the treadmill (26.5 ml.kg.min.$^{-1}$) compared to the cycle ergometer (22.6 ml.kg.min.$^{-1}$). Similarly, higher maximum heart rates were obtained during the treadmill phase (166 beats/minute) compared to the cycle ergometer (159 beats/minute). However, maximal systolic blood pressure was lower on the treadmill (180 mm Hg.) whilst the cycle ergometer produced higher recordings (194 mm Hg). Almost half (47.5 percent) of the subjects showed exercising electrocardiograph changes with ST segment depressions greater than 1 millimetre. From these data, both the treadmill and cycle ergometer are comparable in their likelihood of eliciting ST segment changes in coronary heart disease patients. The treadmill however, produced a reduced response in systolic blood pressure, which seems to indicate that it may be a more applicable method of testing cardiac rehabilitation patients than the cycle ergometer (Sutton, 1979b).

Numerous authors have continued to question the relationship existing between physical activity and coronary heart disease risk reduction. One such study by Ruddel, Berg, Todd, McKinney, Buell, and Roberts (1985) examined cardiovascular reactivity during physical activity using six healthy male volunteers from the
Nebraska Medical Centre as the subjects. Each participant completed a sub-maximal exercise test on a cycle ergometer. The protocol included 15 minutes at 75 per cent maximum oxygen capacity, immediately followed by a further 15 minutes at 70 per cent maximum oxygen capacity. Expired air was collected along with measures of heart rate, blood pressure, and an electrocardiograph reading. Blood samples were taken at regular intervals during testing for biochemical analysis. The effect of physical activity on biochemical variables will be discussed in a separate section of the literature review (see page 49).

Heart rate and systolic blood pressure increased immediately at the commencement of exercise, and continued to increase over the remaining period of testing. Heart rate increased from a resting value of approximately 75 beats/minute to a maximal heart rate of 170 beats/minute, whilst systolic blood pressure increased from 125 mm Hg to 170 mm Hg. Conversely, diastolic blood pressure decreased from 80 mm Hg at rest to 45 mm Hg during exercise. All three variables returned to resting levels within fifteen minutes of terminating the exercise. There were no significant pathological changes observed in any of the subjects electrocardiograph traces. Ruddel et al. (1985) suggested that these haemodynamic and pulmonary responses to physical activity were regularly observed occurrences.

While studying patients with coronary heart disease, Redwood, Rosing, and Epstein (1972) investigated the effects of physical training on the circulatory system and symptoms elicited over a six week program of intensive training. Seven subjects (mean age
48 years), exhibiting symptoms such as chest pains, including angina, and who had suffered at least one myocardial infarction, were hospitalised for the period of the study. Each patient exercised on a cycle ergometer, with the initial work load for each subject being determined following several trial periods. The test protocol involved increasing the workload in increments of 20 watts every three minutes, twice daily, for five days a week. Exercise was terminated at the onset of chest pain. An electrocardiograph was monitored constantly, with pre-training, mid-training (3 weeks into the study), and post training tests conducted to examine the changes in circulatory functioning. Time to onset of angina, oxygen consumption, heart rate, and blood pressure were measured.

The results showed that with training, the onset of angina during exercise was delayed by almost seven minutes. Subjects also were able to increase work capacity by 40 watts. Oxygen consumption increased by 9.6 ml.kg.min.\(^{-1}\) to 15.0 ml.mg.min.\(^{-1}\) after the training period while systolic blood pressure dropped from a mean of 153 mm Hg to 132 mm Hg. Exercising heart rate dropped from 119 beats/minute to 96 beats/minute for the same given work load. In a follow up study using a similar protocol, all of the participants reported that they were able to engage in more strenuous physical activity before anginal chest pains occurred (Redwood et al, 1972). One patient suffered a myocardial infarct ten months after the study. However, this was not included as a valid conclusion from this study.
These findings supported the theory that a relatively brief program of physical training caused a significant improvement in the exercising capacity of patients with coronary heart disease and angina pectoris. It seems that progressive and maintained exercise capacity can be induced by the training program followed in the study (Redwood et al, 1972). However, the benefit derived was dependent on the increased efficiency in myocardial oxygen delivery. This study, although not conclusively supporting the increase in myocardial efficiency, suggested that the response does occur.

Haemodynamic Responses to Physical Activity

Hypertension is a common manifestation in patients with coronary heart disease (Astor & Roth, 1980; Guyton, 1982; Shephard, 1986, 1989). The responses of systolic and diastolic blood pressure to physical activity has often been the topic of investigation. In a study reported by Raglan and Morgan (1987), up to 60 million adults in the United States are considered "borderline" hypertensive (a diastolic blood pressure of 90-104 mm Hg). They also suggest that ten year mortality rate for males with borderline hypertension is 60 per cent higher than the non-hypertensive population of the United States. Furthermore, according to Raglan and Morgan (1987), some pharmaceutical treatments which are prescribed to control hypertension, actually promote impotence, electrolyte imbalance, arrythmias, fatigue, depression, and may cause excessive hypertension. It follows then that many patients will seek alternative methods of controlling hypertension, especially in the borderline cases. Not all drug interventions will
be effective for all patients and not all patients will comply with the regimen that they have been prescribed (Raglan & Morgan, 1987).

Raglan and Morgan (1987) completed two specific experiments during this study, with the first of these involving non-hypertensive subjects. Fifteen male subjects with a mean age of 34.2 years volunteered for the first study. Relaxation or "quiet rest" and aerobic training consisting of jogging, racquetball, basketball, swimming, or cycling were prescribed. The quiet rest sessions were 40 minutes in duration, whilst the exercise sessions were based on a predetermined percentage of maximum aerobic power. Each subject completed their chosen activity at a self paced intensity to enhance ecological validity of the study. That is, they were simulating a field response in the laboratory setting. Levels of anxiety were also measured using the State Trait Anxiety Inventory (STAI). The results showed that mean blood pressure decreased significantly by 9 mm Hg during both exercise and rest. Similarly, diastolic blood pressure decreased significantly by 6 mm Hg post exercise, and remained so for two hours.

The second experiment by Raglan and Morgan (1987), involved fifteen hypertensive males, with a mean age of 60.5 years. All subjects were using some form of pharmaceutical therapy to control their hypertension. The protocol used was identical to that of the initial experiment (Raglan & Morgan, 1987). Again, systolic blood pressure was significantly reduced by 8.5 mm Hg after exercise. Diastolic blood pressure decreased by 2.5 mm Hg which
was not significant. However, immediately post treatment, diastolic blood pressure increased significantly by 3.5 mm Hg. Both studies supported the theory that reductions in blood pressure were positively related to aerobic exercise and a reduced level of state anxiety. Exercise in particular, resulted in significant reductions in diastolic blood pressure.

The results of this investigation seem to suggest that the blood pressure responses to physical activity are more stable than with quiet rest. Patients with pharmaceutically controlled hypertension appear to respond positively to an aerobic exercise regimen, as their diastolic blood pressure is markedly reduced (Raglan & Morgan, 1987). The authors concluded that the responses of blood pressure to exercise have been established with these investigations although more research is required to fully develop an understanding of the responses of hypertension to physical activity.

In contrast to Raglan and Morgan (1987), other studies have not supported the use of physical activity instead of anti-hypertensive medications in controlling abnormal blood pressure. Kaufman, Hughson, and Schaman (1987) conducted a study involving 24 subjects at the University of Waterloo which repeated a study protocol that was originally performed by Wilcox, Bennett, Brown, and MacDonald (1982). The original study by Wilcox et al. (1982) suffered from an experimental design problem with respect to evaluation of resting blood pressure. Kaufman et al. (1987) believed that the resting blood pressure in the study by Wilcox et al. (1982) was over-estimated as no
familiarisation session was used. Therefore, resting blood pressure may have been increased by anxiousness and apprehension. A modified protocol which incorporated a familiarisation session was completed in the latter study which reduced fluctuations in blood pressure at rest.

Kaufman et al. (1987) studied three groups, a young normotensive group (aged 19-29 years), an older normotensive group (aged 35-62 years), and an older hypertensive group (aged 44-57 years). The subjects were all exercising, but none were involved in organised training sessions. The hypertensive group consisted of subjects with diastolic blood pressure in excess of 90 mm Hg with no members of this group having taken hypertensive medication for at least three months prior to the study. The study continued for four weeks and involved three testing sessions. The first two sessions were basically familiarisation sessions to decrease the levels of anxiety and apprehension. The first session involved resting for one hour, whilst the second session had a twenty minute rest followed by ten minutes of treadmill exercise at 10 per cent inclination at 67 per cent of age predicted heart rate maximum. The third session involved five bouts of exercise under the same protocol as in session two, but three minutes rest was allowed between each ten minute exercise bout.

The hypertensive group had significantly higher resting blood pressure than both of the normotensive groups. The mean resting blood pressure was 26 mm Hg higher in session one and 21 mm Hg higher in session three. Resting systolic blood pressure decreased significantly in all three groups by 5-10 mm Hg over
the testing period, whilst resting diastolic blood pressure decreased significantly in the younger normotensive and the hypertensive groups. At the end of a 60 minute recovery period, diastolic blood pressure was no longer significantly depressed, decreasing only 5-7 mm Hg in all three groups.

These findings did support the findings of other studies (Wilcox et al, 1982; Seals & Hagberg, 1984) in that systolic and diastolic blood pressure readings decreased during recovery from dynamic exercise rather than in pre-exercise conditions. However, the differences between normotensive and hypertensive patients were not significant and the differences in reduction of blood pressure were not as great as those produced by Wilcox et al. (1982). Therefore, the conclusion that exercise could be beneficial in the treatment of diastolic hypertension made by Wilcox et al. (1982) was not supported by Kaufman et al. (1987). This was due to the rapid return of diastolic blood pressure to pre-exercise levels.

The study by Kaufman et al. (1987) also highlighted the need for care in experimental design protocol, a topic addressed by Seals and Hagberg (1984), in a review of exercise training and the effect on human hypertension. Many variables seem to require increased consistency in their measurement when researchers are investigating the effect of physical activity on hypertension. Such considerations as the classification of hypertension, subject selection, the measurement of blood pressure, and most importantly, the type of exercise training programs prescribed
need to follow carefully determined protocols. The type of exercise training programs that have been used range from large muscle group activities such as jogging and walking to various sports, such as dance and calisthenics. Unfortunately, the amount of time actually spent exercising is often not recorded.

Seals and Hagberg (1984) reported that the effect of exercising on lowering blood pressure had not been extensively studied. A majority of the studies which did investigate blood pressure had shown small, statistically significant decreases in blood pressure with training. These findings must be questioned because there were often inadequacies in study designs and methodology, especially with respect to obtaining resting blood pressure values and consistency in the techniques of the experimenters. Existing data do not conclusively establish that physical activity is a proven alternative to pharmaceutical drug therapies and there seems to be considerable scope for further investigation (Seals & Hagberg, 1987).

Longitudinal studies have recently investigated the changes in prevalence of hypertension in attempting to identify target populations which may benefit from physical activity as an intervention technique. The Framingham Heart Study (1948-1981) is considered a "classical" longitudinal study. During the 30 year follow up period of the Framingham Heart Study, over 5,000 male and female subjects were examined every six months (Dannenberg, Garrison, & Kannel, 1988). Included in this study was measurement of both systolic and diastolic blood pressure, with a percentage of definite hypertensives being calculated.
Participants exhibiting a systolic blood pressure of 160 mm Hg or greater, and/or diastolic blood pressure of 95 mm Hg or greater, were considered hypertensive. Furthermore, all subjects who were using hypotensive drugs were considered hypertensive, regardless of their blood pressure readings.

The results indicated that the percentage of hypertensive subjects increased with age for both male and female subjects. Definitive hypertension was recorded in 3.3 per cent of males aged 30-39 years, and 1.5 per cent of females aged 30-39 years. At age 70-79 years, the hypertensive subjects had increased to 6.2 per cent and 8.6 per cent respectively for males and females. The number of subjects receiving hypertensive medications increased over time (1954 to 1981), with in excess of 80 per cent of all subjects in 1981, aged 60-89 years prescribed hypertensive medications (Dannenberg et al, 1988).

According to Dannenberg et al. (1988), the increase in cases of hypertension may have been due to several factors, which included decreased elasticity in arterial walls, weight gain with age, and the increase in sodium intake in some affluent societies. The reasons for the increased rate of hypertension in males under 50 years as compared to woman under 50 years, followed by a cross over effect are speculative (Dannenberg, et al, 1988). This may have been due to the males being selectively removed from the study by premature death or that younger males were at a greater risk to the genetic and environmental determinants of hypertension. Secondary prevention of hypertension is now the goal of many current public health studies. Dannenberg et al.
(1988) have suggested that anti-hypertensive medications are, in the main, very costly and have many associated side effects, especially with elderly patients, which warrant concern. Furthermore, data presented from this study indicated that target high risk groups such as males under 50 years may benefit from primary prevention of hypertensive disorders, which may include physical activity as part of the overall prevention strategy.

Cunningham, Ingram, and Rechnitzer (1979), continued the investigation into physiological responses to exercise, using selected participants from the Ontario Health Collaborate Study. The 301 post myocardial infarction male subjects were involved in one of two experimental groups. The first group followed a high intensity exercise program of walking and/or jogging at 65-85 per cent of their maximum oxygen uptake. The second group undertook low intensity exercise of recreational activities such as volleyball, swimming, and relaxation exercises. Cardiovascular fitness was evaluated in each subject by examining heart rate changes at specific oxygen consumption levels. During initial testing, the heart rates elicited at 1250 ml.min.$^{-1}$ oxygen consumption were not significantly different for the two groups (139 beats/minute and 140 beats/minute respectively). Over the ensuing 24 months of the study, the high intensity endurance exercise group heart rate changes were greatest. They decreased at rest by 11.0 beats/minute compared to only 2.0 beats/minute in the low intensity exercise group.

The endurance exercise program of walking and jogging therefore resulted in significant changes in heart rate response in the
myocardial infarct patients. This two year follow up study showed that significant haemodynamic changes can occur with a training program, carried out more than twice a week for 30 minutes, over a period of two years (Cunningham et al, 1979).

**Exercise Capacity and the Cardiac Patient**

Obviously, not all post cardiac trauma patient's will be capable of sustaining such a program as suggested by Cunningham et al. (1979), due to the extent of the cardiac muscle damage. The problem of exercise prescriptions for cardiac patients was addressed by Jelinek (1988). The paper concluded that the prescription of exercise depends on three factors, the patient's age, state of recovery, and presence of major complications. The presence of major complications is considered a contraindication to physical activity, by the American College of Sports Medicine (1975). Other factors including marked obesity, angina pectoris, and the use of certain medications such as beta-blockers and digitalis are also considered as contraindications to physical activity. Conduction disturbances require special precautions including individual monitoring in the presence of suitably qualified staff, such as qualified fitness instructors. Cardiac enlargement, uncontrolled dysrhythmia, repeated ventricular ectopic activity, and untreated severe hypertension, are considered relative contraindications to physical activity. That is, the value of exercise testing often exceeds the risk for the patients with these conditions. Finally, disorders such as congestive heart failure, acute myocardial infarction, active myocarditis, and increasing anginal pectoris with effort are absolute contra-
indications for exercise (American College of Sports Medicine, 1975).

Other factors apart from the characteristics of the participant and the program may also contribute to the benefit or risk any individual can gain from physical activity. One such factor is the physical environment in which the exercise is completed. This can be highlighted with exercise induced angina pectoris when subjects exercise in cold conditions (Brown & Oldridge, 1985). To investigate the relationship between physical activity and the temperature of the environment, Brown and Oldridge (1985) tested 10 male cardiac patients in the McMaster Cardiac Rehabilitation Programme who had a history of effort induced angina pectoris in the cold. Testing was conducted under four conditions using a climate chamber, ranging from room temperature (24 degrees Celsius) to a cold environment (-7.5 degrees Celsius). Several dependent variables were tested including heart rate, systolic blood pressure, rate pressure product (measured from heart rate and systolic blood pressure), minute ventilation, oxygen consumption, and measures of expired air and skin temperature. All patients were monitored using the bipolar electrocardiograph technique.

Overall, exercise in the cold environment resulted in the early onset of angina pectoris in all of the subjects, with a 15-24 percent reduction in exercising time. In addition, there was also a tendency towards higher oxygen consumption with the decreased exercise duration in the cold environment. The critical rate pressure product (RPP) was reached earlier in the cold, which was
due primarily to an increase of 12 mm Hg in mean systolic blood pressure during sub-maximal exercise. The RPP figure is an estimate of myocardial oxygen consumption. These results suggest that physical activity in cold environments for cardiac rehabilitation patients also requires special precautions, including suitable clothing and a reduced exercise intensity (Brown & Oldridge, 1985).

Over the past twenty years, many physiological responses with regard to physical activity, have been researched. Some of these examples have included heart rate and function, cardiac output, blood pressure, oxygen uptake, respiratory quotient, and arterial-ventricular differences. In conjunction with the research, various positive effects of physical activity with respect to coronary heart disease risk have appeared (Gaesser & Rich, 1984; Golding, 1961; Morris et al, 1980; Pfaffenbarger, 1979; Shephard, 1979, 1986, 1989). During the past two decades, there has been an increase in research literature concerning the effect and efficiency of physical activity with respect to the lipid profile. Crow et al. (1986) reported that a multiple risk factor association with exercise exists. Their findings seem to show that there is a negative correlation with physical activity and age, body mass index, heart rate at rest, cigarette smoking, percent body fat, and serum cholesterol levels. Similarly, the authors hypothesised that decreased fitness is often associated with all of these factors. Thus, increased fitness levels should not only correlate with an improved physiological adaptation but also to an improved lipid profile, resulting in decreased overall cardiac risk (Crow et al, 1986).
As stated earlier in this review, specific lipids including cholesterol and triglycerides appear to play an important role in the formation of the atherosclerotic plaques which develop during coronary heart disease (Fox & Mathews, 1981; Groer & Shekleton, 1983; Guyton, 1982; McCunney, 1986; Miller, 1983; Ramsey, 1982). Furthermore, physical activity has been cited by various authors as playing a significant role in the reduction of coronary heart disease risk (Ballantyne et al, 1982; Bonetti et al, 1988; Christie et al, 1980; Danner et al, 1984; Goldberg & Elliot, 1987; Hunter et al, 1968; Johnson & Wong, 1961; Joseph & Bena, 1977; McNaughton & Davis, 1987; Shephard, 1986; Wood et al, 1983). It seems logical therefore, that physical activity may contribute in some way in reducing the risk of coronary heart disease associated with a desirable lipid profile.

**Cholesterol, Triglycerides, and Lipoproteins**

Cholesterol and triglycerides seem to be the two most common lipids associated with coronary heart disease risk (McArdle, Katch, & Katch, 1985; Rochelle, 1961; Streja & Mymin, 1979; Thompson, Cullinane, Eshleman, Kantor, & Herbert, 1984; Thompson, Cullinane, Eshleman, Sady, & Herbert, 1984). Mortality from coronary heart disease and sudden death due to coronary heart disease, and non fatal myocardial infarction have all been shown to be significantly related to serum lipid levels, in a comprehensive study by Stamler, Forman, and Krol (1978). This extensive study involved over 2,500 subjects whom had suffered from at least one myocardial infarction and were all undergoing
placebo treatment during a cardiac drug study. Monitoring of the serum cholesterol and triglyceride levels showed that increased levels of both triglyceride and total cholesterol correlated with increased cardiac trauma. The study indicated that recovery from myocardial infarct may be improved if an accompanied reduction in serum cholesterol and triglyceride levels are achieved.

These findings were duplicated during the Lipid Clinics Primary Prevention Trial carried out by the Lipid Research Clinic (1984). A 19 per cent lowering in the incidence of coronary heart disease was observed, following a mean decrease in total cholesterol of 8 per cent and a mean decrease of low density lipoproteins of 12 per cent were reported. This study showed that the lowering of total plasma cholesterol and low density lipoprotein levels was associated with a decrease in coronary heart disease risk.

This relationship was again demonstrated by Pelkonen et al. and Nikkila et al. (1978). Pelkonen et al. (1977) studied the triglyceride and total cholesterol concentrations in 1648 middle aged Finnish males (50-53 years). A triglyceride level in excess of 150 mg/100 ml, was shown to increase the risk of coronary heart disease, regardless of the level of serum cholesterol. Significantly, smokers or obese individuals had 5.7 and 4.1 times greater risk of coronary heart disease respectively, when compared to their non smoking, non obese counterparts. The second study by Nikkila et al. (1978) investigated the activity of enzymes as catalysts for cholesterol metabolism. The study included 41 male and female subjects, aged 20-50 years, and the data obtained showed that the
rate of triglyceride catabolism may have been a factor in determining the concentration of high density lipoproteins.

It is known that cholesterol and triglycerides do not circulate freely in blood plasma, but are combined with various proteins to form lipoproteins. Serum cholesterol is the measure of the total cholesterol contained in the different forms of lipoproteins (McArdle, Katch, & Katch, 1986). There is a relationship between the serum cholesterol level and the risk of coronary heart disease, but the distribution of the total cholesterol amongst the various types of lipoproteins has been shown to be a more powerful predictor of coronary heart disease (Ballantyne, Clarke, Simpson, & Ballantyne, 1982; Christie, Bloore, & Logan, 1980; McArdle et al, 1985; Streja, & Mymin, 1979).

Miller, Mead, Kwiterovich, & Pearson (1990), reported that the National Cholesterol Education Program of America has recommended a plasma cholesterol of less than 200 mg/dl. However, total cholesterol is not the only serum lipid concentration which predicts cardiac risk. An elevated level of high density lipoproteins is thought to be associated with decreased coronary heart disease risk. In contrast, elevated levels of both low density lipoproteins and very low density lipoproteins appear to increase cardiac risk (Ballantyne et al, 1982; Christie et al, 1980; McArdle et al, 1985; Streja, & Mymin, 1979). Recent studies (Cooper et al, 1985; Freedman et al, 1986; Maciejko et al, 1983; Naito, 1987, 1988a) have identified more specific lipid binding proteins, known as apolipoproteins. Apolipoproteins are widely acknowledged as being more probable determinants of the
functioning of lipoproteins than are other serum lipids and therefore, may be correlated to coronary heart disease risk more-so than lipoproteins alone (Alaupovic, McConathy, Fesmire, Tavella, & Bard, 1988; Brewer, Gregg, Hoeg, & Fojo, 1988; Palkovic, 1980; Nikkila, Kuusi, & Myllynen, 1980; Nagao, Imai, Arie, Sawada, & Karatsu, 1988).

The formation of these lipids, especially cholesterol, has been studied for many years, and according to Bloch and Lynen (1964) the association between cholesterol and sterol synthesis was initially discovered in the 1930's. A sterol is a form of steroid which is characterised chemically according to the pattern of methylation. This refers to the pattern in which the methyl group molecules are arranged about the central molecular configuration (Luckner, 1984). Although cholesterol has been regularly associated with coronary heart disease, this sterol seems responsible for many functions of the human system, including stabilisation of cell membranes. Cholesterol also occupies a central position in the metabolism of other steroids in most organisms (Luckner, 1984).

Cholesterol is present in all cells of the body and is derived exogenously (from dietary intake), or endogenously (synthesised within cells). Although the liver is the main site of cholesterol synthesis, other tissues including those in the artery walls can also produce cholesterol (Cook, 1958; McArdle et al, 1985; Rochelle, 1961). Cholesterol molecules are important in the functioning of the body, including the synthesis of vitamin D, and the adrenal gland hormones, estrogen, androgen, progesterone and bile
secretions. Rochelle (1961) also reported that cholesterol was important in the transport of free fatty acids as well as being an integral structural unit of all tissues in the body (Cook, 1958; Gurr & James, 1980; Heftmann, 1970; Luckner, 1984; McArdle et al, 1985; Zubay, 1983).

Triglycerides differ distinctly from cholesterol, both biochemically and in their proliferation. Triglycerides are the most common form of fats in the body. Over 95 per cent of all lipids are in the form of triglycerides, which are formed as an ester of glyceride (Luckner, 1984; McArdle et al, 1985). An ester is formed as a results of a reaction between an activated fatty acid and an alcohol molecule. Glyceride is in turn an ester of glycerol, which is a colourless, odourless alcohol obtained by the saponification (decomposition) of natural fats and oils (Luckner, 1984).

The function of both triglycerides and cholesterol in the formation of coronary heart disease is still undergoing vigorous debate. A general consensus of opinion exists amongst experts in the field that the distribution of these two lipids amongst the various forms of lipoproteins may be the crucial factor in coronary heart disease risk evaluation (Ballantyne et al, 1982; Christie et al, 1980; McArdle et al, 1985; Streja & Mymin, 1979). According to Dietschy, Gotto, & Ontko (1978), lipoproteins have been traditionally divided into four basic groups based on their rate of flotation in salt solutions which determines the density of proteins.
These four categories of lipoproteins include:

a) high density lipoproteins (HDL)
   - density of 1.063-1.210 g/ml
b) low density lipoproteins (LDL)
   - density of 1.006-1.063 g/ml
c) very low density lipoproteins (VLDL)
   - density of 0.95-1.006 g/ml
d) chylomicrons
   - density of < 0.95 g/ml

(Skipski, 1974)

Further, less frequently used classifications have been made, including intermediate density lipoproteins (IDL - density of 1.019-1.063 g/ml) and very high density lipoproteins (VHDL - density of 1.210-1.250 g/ml). These lipoproteins can be differentiated with respect to the amount of cholesterol, phospholipids, and triglycerides present, as summarised in table 1.
TABLE 1

Percent Composition of Lipoproteins in Man

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Cholesterol</th>
<th>Phospholipids</th>
<th>Triglycerides</th>
<th>Proteins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chylomicrons</td>
<td>5</td>
<td>3</td>
<td>90</td>
<td>2</td>
</tr>
<tr>
<td>VLDL</td>
<td>12</td>
<td>18</td>
<td>60</td>
<td>10</td>
</tr>
<tr>
<td>ILDL</td>
<td>30</td>
<td>20</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>LDL</td>
<td>50</td>
<td>15</td>
<td>10</td>
<td>25</td>
</tr>
<tr>
<td>HDL</td>
<td>20</td>
<td>25</td>
<td>5</td>
<td>50</td>
</tr>
</tbody>
</table>

Note: Values Expressed as Percentages.

Of all the plasma lipoproteins, the high density lipoproteins have received the greatest attention in recent years (Dietschy et al, 1978). The level of high density lipoproteins are thought to be inversely related to the development of atherosclerosis and may have a protective effect in the development of coronary heart disease (Hall, Meyer, & Hellerstein, 1984). The levels of serum high density lipoprotein may be increased with weight loss, smoking cessation, and exercise. An increase in high density lipoproteins from normal levels is observed in pre-menopausal females which may help to explain the lower rate of coronary heart disease in women (Hall et al, 1984).
Conversely, low density lipoproteins contain a higher level of cholesterol and are highly correlated with coronary heart disease (Hall et al, 1984). Low density lipoproteins are formed as a result of the metabolism of very low density lipoproteins, and are thought to facilitate the entry of triglycerides into the circulation. The levels of low density lipoproteins are increased with foods that are high in saturated fats and cholesterol. Furthermore, a major component of atherosclerotic plaques is low density lipoprotein (Hall et al, 1984; McArdle et al, 1985).

**Serum Lipid Responses to Physical Activity**

A majority of recent studies investigating the relationship between physical activity and the lipid profile, have concentrated on the presence of high density lipoproteins, especially as a proportion of total cholesterol (Ballantyne et al, 1982; Bonetti et al, 1988; Christie et al, 1980; Danner et al, 1984; Dietschy et al, 1978; Jelinek, 1988; Nye et al, 1984; Wood et al, 1983). However, Superko (1988), in a review of lipid management techniques, concluded that factors other than lipoproteins can contribute to the development of atherosclerosis. Many of these factors have yet to be discovered and current researchers are working in the evolutionary phase of coronary heart disease therapy (Superko, 1988). However, clinical investigations have demonstrated that lipid management incorporating physical activity, pharmaceutical medications, and dietary interventions can significantly benefit cardiovascular health (Superko, 1988).
Several major longitudinal studies, including the Lipid Research Coronary Primary Prevention Trial completed in the early 1980's and the Helsinki Heart Study of 1972, between them involving over 20,000 subjects, demonstrated that elevated total cholesterol was associated with coronary heart disease. Furthermore, fewer coronary heart disease events occur when elevated low density lipoprotein levels are controlled or, high density lipoprotein levels are raised (Superko, 1988). Lipid research has led to the development of recommended target lipid and lipoprotein levels, especially for high risk patients. Superko and Haskell (1987) suggest that high risk patients should maintain a plasma total cholesterol level of less than 180 mg/ml, with a low density lipoprotein level of less than 110 mg/ml and total triglycerides of less than 120 mg/ml. The ratio of total cholesterol to high density lipoproteins should be less than 3.5. These figures were established following an extensive review of data by Superko and Haskell (1987), and were ratified as a guide at the 1985 conference of the National Institute of Health in the United States of America.

There appears little doubt that modification of the concentration of specific lipids and lipoproteins can significantly reduce the risk of coronary heart disease. Several methods of obtaining the desired lipid profile have been investigated. These have included the use of physical activity, dietary intervention, pharmaceutical medications, and body composition modification (Superko, 1988). However, elimination of any secondary causes of hyperlipidemia must occur if the true effect of physical activity on abnormal lipid levels is to be known (Superko, 1988). Disorders such as thyroid
disease, nephrotic syndrome (kidney disease), diabetes mellitus, and alcohol abuse need to be controlled. Of the primary determinants of an abnormal lipid profile, maintaining an appropriate diet is considered a cornerstone of the successful lipid therapy program (Superko, 1988). Both diet and physical activity are important in the determination of body composition. The reduction in total body fat often results in substantial decreases in total cholesterol and triglyceride levels, and may also increase the ratio of high density lipoproteins to total cholesterol (Superko, 1988).

Physical activity is prominent in the prescription for a healthy life-style and is a significant factor in the reduction of total body fat content, although the role of physical activity in lipid management is as yet undecided (Shephard, 1986, 1989; Superko, 1988). Physical activity may be of little consequence unless combined with dietary intervention. Physical activity appears to have a profound effect on the ratio of high density lipoproteins to total cholesterol, but like many other therapies, determining the correct dose is crucial. Although this topic is undergoing vigorous debate, recent evidence suggests that jogging for 10 to 15 miles per week must be exceeded before a significant alteration in lipoprotein levels is evoked (Wood, Haskell, & Kline, 1976; Superko, 1988).

Research into the efficacy of physical activity in the management of coronary heart disease has proliferated since the 1950's. As the studies have evolved, the use of increasingly refined research protocols, and greater care in subject selection has given modern
experts a vastly improved knowledge of this non-pharmaceutical method of coronary heart disease treatment. An early study by Johnson and Wong (1960) demonstrated some of the common faults apparent in the earlier studies. This study involved twelve healthy male swimmers (mean age 19.3 years) who were monitored for 14 months to determine the effect of a "typical" training and competition program on plasma cholesterol and phospholipid levels. The protocol was antiquated, with no objective measure of work output recorded. The authors recorded that the athletes swam twice a day over a distance of 1-2 miles in a series of 440 yard and 220 yard sprints. Weight loss, total plasma cholesterol, and phospholipid levels were regularly monitored over the period of the study (Johnson & Wong, 1960).

The results of the study were not conclusive, with no elevations or depressions in mean cholesterol or mean phospholipid levels recorded. Johnson and Wong (1960) attributed the lack of significant findings to several factors. This included the initial low base line plasma cholesterol levels (169 mg/ml) and the use of non-obese subjects made the detection of subtle changes in cholesterol levels difficult. However, the shortcomings in experimental design included the lack of a control group and no control of training distances. Cessation of training during examination periods and over the summer vacation would also have affected the validity of the study. Regardless of the poor research design, the study did conclude that "adequate" exercise may cause the utilisation of caloric intake and may also cause levels of cholesterol to be affected (Johnson & Wong, 1960). With
better monitoring of the exercise prescription and research protocol, this study may have produced significant results.

Further experimental design shortcomings were evident in a study by Hunter, Nye, and Heslop (1968) during which 55 subjects were observed to determine the effect of a twenty week jogging program on serum cholesterol and triglyceride levels. Thirty-two of the subjects (mean age 42.4 years) were in the experimental group which jogged 25-30 miles per week, whilst the control group of 23 subjects (mean age 39.9 years) undertook no physical activity. Body weight, skin fold measurement, blood pressure, serum cholesterol, and serum triglyceride levels were measured. The weight loss of 1.1 kg and a 5.1 mm decrease in the skin fold measurement observed in the experimental group were both significant results, as were the between control and experimental group difference in skin fold measurement of 2.3 mm. However, there were no significant changes in blood pressure, serum cholesterol levels or serum triglyceride levels in either group. In contrast to these results there were the non-significant elevations in both serum cholesterol and serum triglyceride levels (4.1 mg/100 ml and 9.7 mg/100 ml respectively) exhibited by the control group (Hunter et al., 1968).

Hunter et al. (1968) concede that the results did not support the concept that physical activity significantly reduced cholesterol and triglyceride levels. Several reasons were forwarded for the lack of significant findings. No consideration was given to the interval which had elapsed between the previous jogging period and the testing session. This may have contributed to the failure to detect
the changes in plasma lipid levels. Furthermore, there was no control of caloric intake, which may have counteracted any beneficial effects from the physical activity. Again, there was no objective measure of physical work output, with the authors merely stating that the subjects jogged 25-30 miles per week. Finally, Hunter et al. (1968) suggested that further studies were warranted to determine if physical activity of a different nature or continuing for a longer period would elicit significant changes.

In the same year, Zauner (1968) attempted to account for the changes in caloric intake by examining the response of blood lipids with daily habitual activity after fasting, and during the postprandial (after food ingestion) period. Thirty one healthy males were assigned to one of four groups according to age. Each group had mean ages of 19.1 years, 22.0 years, 25.3 years, and 46.1 years respectively. All of the participants fasted for twelve hours overnight, after which they had a venous blood sample drawn. A diet with 60-65 grams of fat was then consumed. Each participant then carried out a normal day's activity, with no further food or beverage intake, while cigarette smoking was also completely restricted. Further blood samples were taken at three, five, and seven hours after the meal was ingested.

The results showed that age was not associated with elevated lipid levels, but physical activity may be related to increased serum lipid densities. Two conclusions were reported from this investigation. During the fasting phase, no significant differences were observed between the active and sedentary subjects. Furthermore, advancing age alone did not inhibit postprandial
plasma lipid clearance or result in elevated fasting serum lipid concentration. Habitual physical activity appeared to enhance the mechanisms of lipid metabolism, especially in older age groups.

Zauner (1968) suggested that a decrease in habitual physical activity may contribute to elevated serum cholesterol levels. A follow up study by Penny and Wells (1975) investigated the effect of cessation of training on serum cholesterol levels, and used as a comparison six college football players (experimental group) and six non-athlete college students (control group). Immediately at the conclusion of the football season, serum cholesterol and triglycerides, heart rate and blood pressure were taken to determine if detraining affected any of these variables. All of the subjects performed an initial cycle ergometer test at a work load of 1200 kpm with venous blood being drawn five minutes post exercise. An identical testing procedure was carried out at three week intervals for nine weeks.

The results were not conclusive. The athletes had a significantly decreased heart rate of 23 beats/minute compared with the control group members at the initial testing stage. No significant differences in heart rate or blood pressure existed at any other testing session. Serum cholesterol levels were also not significantly altered, even though the control subjects showed double the resting cholesterol concentrations (4 mg/100 ml vs 8 mg/100 ml). The failure of this experiment to provide significant results did not confirm the findings of Rochelle (1961) where significantly decreased total cholesterol levels were observed in trained athletes. With respect to the detraining effect, Penny and
Wells (1975) indicated that increased resting heart rates were a more recognisable detraining affect than was an alteration in serum lipid concentrations.

Many studies appeared to have suffered from a lack of a quantitative assessment of the work intensity (Golding, 1961; Hunter, Nye, & Heslop, 1968; Rochelle, 1961; Johnson & Wong, 1960). However, Joseph and Bena (1977) investigated the effect of a long term exercise program on cholesterol reduction, and reported that their study did in fact quantify physical effort. Fifty seven males ranging in age from 33-62 years were assigned to three groups. An experimental group of 17 new enrollees in a college fitness program were recruited along with two control groups of 20 subjects each. The first control group was an active control group, whose members were highly trained, and had been in the college fitness program for 2-4 years, while the second control group were inactive.

All of the subjects in the active control and experimental groups received a comprehensive fitness examination prior to and during the course of the study. The exercise classes were attended by both the active control and experimental groups for 30 weeks. The exercise programme consisted of a 5 minute warm up, twenty minutes of continuous calisthenics, followed by a choice of 25 minutes of jogging or swimming, and concluding with a 5 minute cool down. The exercise intensity of the calisthenics was monitored using heart rate and extrapolated into a cost in kilocalories per each 30 minute session. The energy cost of the jogging and swimming was calculated using time, distance
covered, and heart rate. A minimum target heart rate was established for each participant and a blood sample was drawn for biochemical analysis.

The results showed that the only significant decreases in cholesterol levels were observed in the experimental group, with their mean total cholesterol being reduced by 18 per cent, or 41.5 mg/100 ml of serum. Based on these results, Joseph and Bena (1977) concluded from their study that an intense exercise program carried out for three days per week would significantly reduce total serum cholesterol levels in previously inactive middle aged men. In addition, in the experimental subjects, a loss in body fat from 11.2 per cent to 9.3 per cent, and a significant decrease in skin fold of almost 26 percent was shown although, a loss of body weight did not necessarily occur in each subject.

In the early 1980's, a majority of studies reported a positive relationship between physical activity and cholesterol reduction. However, what mode and intensity of activity produced the optimal response was still not, and is yet to be conclusively determined (Ballantyne et al, 1982; Despres et al, 1985; Morris et al, 1980; Nakamura, Uzawa, Maeda, & Inomoto, 1983; Nye et al, 1984; Shephard 1986, 1989). Nakamura et al, (1983) completed an extensive study involving 20 male subjects who jogged regularly and 644 male and female subjects who undertook varying degrees of habitual physical activity. The experimental subjects had been jogging for an average of 7.2 years, 3 or more days per week. Each session averaged 53 minutes in duration at an average speed of 175 metres per minute. Two separate
investigations were completed. A comparison of selected physiological variables was made between the regular joggers (mean age 62.2 years) and a control group of 20 subjects (mean age 61.5 years) taken randomly from the large group of irregular exercisers. Blood pressure, body composition, resting and exercising electrocardiographs for the non exercising controls were recorded. A questionnaire was completed to determine dietary and life-style habits. Serum cholesterol, triglyceride, high density lipoprotein, low density lipoprotein, and very low density lipoprotein concentrations were determined from blood samples drawn.

A significant increase was observed in high density lipoprotein levels in the experimental group (71.4 mg/dl) compared to the control group (57.9 mg/dl). Also, a significantly increased ratio of high density lipoprotein to total cholesterol was shown in the experimental group (0.304) when compared to the control group (0.259). According to the authors, these results indicated that subjects who ran for longer durations (50+ minutes) each week had higher anti-atherogenic indices (Nakamura et al, 1983).

A second investigation of the 644 male and female subjects from which the control group was initially drawn was completed to determine if varying amounts of habitual activity affected the lipid concentration (Nakamura et al, 1983). Each subject was assigned to one of four groups on a basis of how much physical activity they completed each week. Each group then completed a 12 minute performance test to determine fitness levels. Cholesterol levels were obtained using an identical protocol as
employed in the earlier investigation. With both males and females, the correlation between the results of the twelve minute performance test and high density lipoprotein were positive (0.075 and 0.078 respectively). The correlation between the performance test and the ratio of high density lipoprotein to total cholesterol was again positive and significant for males and females (0.292 and 0.282 respectively).

Findings from both of these studies suggested that exercise conditioning may be a factor which effects serum cholesterol levels, particularly high density lipoprotein concentrations. It was observed that as fitness levels increased, the level of high density lipoprotein and the ratio of high density lipoprotein to total cholesterol also increased. As there appears to exist a general negative correlation between the morbidity and mortality from coronary heart disease and increased levels of high density lipoproteins in the western world, these results would indicate that physical activity may have a protective function against coronary heart disease (Nakamura et al, 1983).

To test this theory, Rauramaa et al. (1984) studied 31 healthy males, undertaking regular physical activity for two months. Twenty nine males with a mean age of 38 years acted as controls. Each individual was screened by a medical examination and after a one month familiarisation period, base line serum lipid levels were determined. A maximal cycle ergometer exercise test was used to obtain an initial fitness level. The eight week training program consisted of walking or jogging 8-15 miles each week during sessions of 45 minutes per day. A five minute warm up
and cool down period was also used. After one month, the exercise sessions were increased to 60 minutes in duration. Intensity was determined using heart rate responses taken from the initial testing session. In contrast to the exercising subjects, the control group subjects were asked to refrain from all physical activity. Blood sampling and exercise testing were repeated at the end of the two month study.

The initial testing showed no significant differences in maximum oxygen consumption or serum lipid levels between the two groups. After the exercise programme was completed, the experimental group had increased oxygen consumption by 0.24 l.min.\(^{-1}\) and low density lipoproteins had decreased significantly by 15 mg/100 ml of serum. Furthermore, high density lipoprotein levels were significantly elevated from 1.11 to 1.26 mmol/L. These results suggest that mild aerobic activity has a beneficial effect on the serum cholesterol concentration. Rauramaa et al. (1984) concluded that these physiological responses highlight the preventative potential of regular physical activity against coronary heart disease. At the least, mild physical activity may, in the short term, favourably effect the serum lipid concentrations in healthy, middle aged men.

According to Nye, Anderson, and Sutherland (1984), the effects of short term physical activity programs on blood lipoprotein lipids have been frequently reported. It seems logical therefore to investigate the effects of a longer term physical activity prescription. During research involving 40 typically healthy rural New Zealand males, aged 30-45 years, Nye et al. (1984)
investigated the effects of a year long exercise regime on serum lipid levels. Each subject was required to keep a week long dietary record at the beginning and at the end of the study. Aerobic capacity was determined at the beginning of the study, and at six and twelve months, using the Cooper's twelve minute run/walk. The exercise program was devised to increase general mobility, and to increase muscle strength and endurance. Calisthenics were carried out, under the guidance of a qualified physical education instructor, with most subjects also partaking in jogging and squash. Various blood lipid concentrations were determined after overnight fasting. Total cholesterol, triglycerides, very low density lipoprotein, low density lipoprotein, and high density lipoproteins were examined.

Comparisons were made between the initial levels of all of the lipid fractions in order to determine the effect of the exercise program, thus, controls were considered unnecessary. Total mean blood cholesterol fell significantly from 6.57 mmol/L to 5.80 mmol/L after six months, and decreased further at the conclusion of the 12 month study (5.79 mmol/L). Similarly, low density lipoproteins showed a significant decrease after six months from 14.46 mmol/L to 3.99 mmol/L, and once again remained depressed at the twelve month recording. However, the high density lipoproteins showed a surprising non significant decrease over the twelve months of the study, dropping from 1.65 mmol/L to 1.37 mmol/L.

The study showed that the effects of long term exercise programs are difficult to determine. The ratio of high density lipoprotein to
low density lipoprotein did not show a favourable improvement, which suggests that the effects of a long term exercise program as prescribed by Nye et al. (1984) may not alter coronary heart disease risk. However, this study may well have been more conclusive if a more specific determination of work output was completed, and a control group was used.

Wood et al. (1983) attempted to eliminate some of these experimental design flaws in a study involving eighty one male subjects from Stanford University. The subjects were organised into four consecutive groups of twelve exercising and eight non-exercising control participants to determine the effect of a one year running program on serum lipid concentrations. The control group members did not take part in any structured physical activity. After a base line evaluation, the exercising participants joined an exercise class on three days per week for calisthenics, running, and stretching. After the initial three weeks, these sessions were carried out on four days per week. The exercise was conducted at a work intensity of 70-85 per cent of maximum heart rate. At three month intervals, the exercising and control participants were tested for total cholesterol, triglycerides, high density lipoproteins, low density lipoproteins, and apolipoproteins A-I and B.

Overall, the exercising group showed elevated high density lipoprotein levels and lower low density lipoprotein concentrations, and all other lipid fractions changed in a direction considered suitable for reduced coronary heart disease risk although, none were significant. The lipid profile of the exercising
group showed a decrease in total cholesterol of 8.0 mg/dl, an increase in high density lipoprotein of 1.8 mg/dl, a decrease in low density lipoprotein of 5.2 mg/dl, and an increase in apolipoprotein A-I of 4.9 mg/dl. In contrast to the exercise group, the control group displayed an increase of total cholesterol of 1.8 mg/dl, an increase of high density lipoprotein of 0.5 mg/dl, an increase in low density lipoprotein of 1.3 mg/dl, and an increase of apolipoprotein A-I of 3.7 mg/dl. Wood et al. (1983) suggested that if the participants who ran less than eight miles per week were removed from the study, significant changes in some of the lipoprotein concentrations, especially high density lipoprotein subfractions, would be observed. With these findings, the question still remains, as to what effect physical activity has on lipoproteins if caloric intake is held constant. It was shown in Wood et al. (1983) that increased physical activity correlated closely with increased caloric intake. Overall, these results generally support the concept that more vigorous forms of physical activity are associated with reduced risk of coronary heart disease.

The study by Wood et al. (1983) exhibits many characteristics of recent studies into this topical health issue of coronary heart disease. One such characteristic which is almost universal in investigations into physical activity and the lipid profile is the use of an almost exclusive male population. There may be several reasons for this, including that males have a higher overall rate of coronary heart disease than females. Moll, Sanders-Williams, Lester, Quarfordt, and Wallace (1979) recognised the need to study females and investigated the effect of an intense six week
exercise program on the lipid profile. Fourteen non-obese female medical students, aged 22-26 years took part in the study. All subjects underwent a 3 week de-conditioning period, refraining from all forms of physical activity. A six week conditioning period followed, and involved exercising 5 days per week for 45 minutes of continuous aerobic exercise, at 70 per cent of the subjects maximal predicted heart rate.

Serum lipid analyses were performed on two separate occasions during pre-conditioning, with a further couplet of tests performed after the 6 weeks of conditioning. Total cholesterol, and high density lipoprotein cholesterol levels were determined. Three subjects were excluded from the study due to a lack of increase in aerobic capacity during the study, or due to injury. Of the remaining 14 subjects, 10 exhibited a significant decrease in total plasma cholesterol from 170 mg/100 ml of serum during de-conditioning to 160 mg/100 ml of serum after conditioning. However, the level of high density lipoprotein and the decrease in the ratio of total cholesterol to high density cholesterol from 2.9 to 2.85 was not significant. Moll et al. (1979) believe the study was somewhat unique in that it produced a significant decrease in total cholesterol without the associated significant change in either high density lipoprotein or the important ratio of total cholesterol to high density lipoprotein. This may reflect on the disparity between the populations used, namely, males versus females. A gender difference may exist in the ability to metabolise lipoproteins, but this is difficult to ascertain as most other studies have used male subjects. The lack of studies involving females, is
an issue which may require further consideration, especially when studying physical activity and serum lipid concentrations (Moll et al, 1979).

Serum Apolipoprotein Responses to Physical Activity

Regardless of gender difficulties, the use of lipoprotein fractions in determining cardiac risk has become a widely used practice. As suggested earlier, considerable evidence from laboratory and epidemiological studies has shown an especially strong correlation between low levels of high density lipoprotein and increased risk of coronary heart disease (Ballantyne et al, 1982; Christie et al, 1980; Danner et al, 1984; Morris et al, 1980; Penny & Wells, 1975; Wood et al, 1983). However, research over the last 5 years has forwarded strong evidence that the levels of apolipoproteins may be a more specific and sensitive indicator of coronary heart disease risk (Cooper, Smith, Weibe, Kuchmak, & Hannon, 1985; Freedman, Srinivasan, Shear, Franklin, Webber, & Berenson, 1986; Macieko, Holmes, Kottke, Zinsmeister, Dinh, & Mao, 1983; Naito, 1987, 1988a).

The major protein components of lipoproteins are known as apolipoproteins. Two of the most researched apolipoproteins are apolipoproteins A-I and B. Apolipoprotein A-I is the major protein component of high density lipoprotein whilst apolipoprotein B is the major protein component of low density lipoprotein. Naito (1988) has recently found that total cholesterol (sensitivity - 63 per cent) is a less sensitive marker of coronary heart disease risk than measured fractions of high density
lipoprotein and low density lipoprotein (76 per cent), and is an even less accurate indicator of coronary heart disease risk than the apolipoproteins A-I and B markers (87 per cent). Therefore, the ratio of apolipoprotein A-I to B appears to be a more accurate measure of coronary heart disease risk. These markers are the protein components of plasma lipoprotein, and are two of approximately sixteen apoproteins which have been identified in all forms of lipoproteins. It is now recognised that the apolipoproteins have four distinct functions. They are necessary for the synthesis and catabolism of lipoproteins and are necessary structural components of lipoprotein particles for fat transport. Apolipoproteins are also co-factors or activators of enzymes associated with lipid and lipoprotein metabolism, as well as being involved in the transfer of lipids between the lipoprotein particles (Naito, 1988a).

The biochemical significance of apolipoproteins with respect to coronary heart disease risk, accompanied with the ease of detection of these lipoprotein fractions, as described by Naito (1988a) has prompted increased research over the last decade into the apolipoproteins as an indicator of overall coronary heart disease risk. However, comparatively little research into the responses of apolipoproteins in relation to physical activity has been reported. Most of the research so far has been directed towards apolipoprotein A-I, the major protein component of high density lipoprotein.

Ballantyne, Clarke, Simpson, and Ballantyne (1982) completed a study investigating the effect of moderate physical activity on
plasma lipoprotein subfractions, including apolipoprotein A-I. Forty two male post myocardial infarct patients (mean age 52.2 years) were divided into an experimental group of nineteen with a control group of twenty three subjects. The experimental group subjects had previously been involved in a six month exercise program, whilst the control group had not been involved in regular exercise. All participants had similar dietary habits, and were similar in height and weight.

All subjects underwent a pre-study treadmill test, with the experimental group taking longer to reach maximum exercise than did the control group. Overnight fasting took place before blood sample were drawn. Very low density lipoprotein, low density lipoprotein, high density lipoprotein, and apolipoprotein A-I were assessed at 0, 1, 2, 4, and 6 months during the study. The experimental group followed the Canadian Air Force 5BX plan as a protocol for physical activity. This method was used as it was considered easy to monitor, and produced the best results in terms of fitness (Ballantyne et al, 1982).

The results of the initial lipoprotein and apolipoprotein fractions of the control and the experimental groups were well matched. During the period of the study, total cholesterol showed a non significant decrease in the experimental group from 253 mg/dl to 246 mg/dl. The total cholesterol concentrations in the control group remained constant at about 259 mg/dl. In fact, all of the variables measured remained constant in the control group. Several variables showed significant changes in the experimental group. A significant increase in apolipoprotein A-I from 122
mg/dl to 137 mg/dl was shown while a non-significant increase in high density lipoprotein level of 7 mg/dl to 56 mg/dl was recorded. Both serum triglyceride and low density lipoprotein concentrations significantly decreased during the six month study (Ballantyne et al, 1982).

These results suggest that physical activity affects apolipoprotein A-I, even when there is no significant increase in overall high density lipoprotein or decrease in serum cholesterol values. Ballantyne et al. (1982) believed that the results gained following a moderate intensity exercise program were important in that many cardiac rehabilitation patients could not be expected to maintain vigorous physical activity.

Vigorous physical activity has been shown to affect the serum cholesterol levels and apolipoprotein A-I, as reported in a study by Danner, Wieling, Havekes, Leuven, Smit, and Dunning (1984). Fifteen male oarsmen, training for the Netherland National Championships were observed, training for an average of 14 hours per week for seven months. Twenty one medical students matched for age, and life-style habits and whom undertook minimal physical activity (less than one hour per week) acted as the control group. The oarsmen were tested for selected lipid concentrations and various anthropometrical measures were also recorded, at the beginning of the study. This was followed by further testing at two weeks, and then one, four, and seven months. The control group was similarly tested at the beginning of the training, followed by two weeks, one month and seven months. Each testing session included measuring body weight,
skin folds, total cholesterol, high density lipoprotein, and apolipoprotein A-1.

The results were conclusive. There were significant changes in all lipid parameters in the training oarsmen, with the exception of the four month test which followed a two month enforced lay off period from vigorous training due to weather. Total cholesterol, total triglyceride, high density lipoprotein and apolipoprotein A-I all differed significantly when compared to each of the preceding test results with the exception of the four month test. Overall, total cholesterol decreased significantly from 4.32 mmol/L to 3.76 mmol/L, whilst total triglyceride decreased from 1.47 mmol/L to 0.85 mmol/L. High density lipoprotein increased from 1.24 mmol/L to 1.32 mmol/L, with apolipoprotein A-I increasing from 144 mg/100 ml of serum to 174 mg/100 ml. No significant changes in any of the lipid parameters were recorded in the control group (Danner et al., 1984).

Danner et al. (1984) believed that this study restricted the effect of confounding variables by employing control populations matched for diet, alcohol intake, smoking habits, age and weight. The changes in serum lipid concentrations brought about by physical activity only lasted for the duration of the training. Inactivity may reduce the effect of physical activity on the lipid profile to virtually zero (Danner et al., 1984).

Nikkila, Kuusi, and Myllynen (1980) and Nagao, Imai, Arie, Sawada, and Karatsu (1988) further investigated the effect of physical inactivity of the lipid profile with similar results. Nikkila
et al. (1980) showed during a study of twenty three Finnish males and females aged between 17-60 years, completely immobilised due to traumatic fractures, showed significantly reduced apolipoprotein A-I levels when compared to the matched control group. The control group members were representative of the adult Finnish population and were drawn from a study known as the "Mini Finland Study". The immobilised male subjects had an apolipoprotein A-I level of 117.1 mg/dl compared to 150.8 mg/dl in the male control group. Similarly, the immobilised female subjects had an apolipoprotein A-I level of 118.2 mg/dl compared to 178.4 mg/dl in the control subjects. Although the possibility existed that neurological lesions or being constantly horizontal may have effected these results, Nikkila et al. (1980) concluded that this seemed less likely than the effect of physical activity itself.

Nagao et al. (1988) produced similar results when they compared highly active, moderately active, and inactive Japanese males. The highly active group of twenty three subjects had been running or swimming for 60 minutes everyday for two years, whilst the moderately active group had been jogging for 30-60 minutes, four days per week for 2-20 years. The control group was completely inactive. The mean values of serum cholesterol in the moderately active group was significantly higher (209.85 mg/dl) than both the highly active (182.27 mg/dl) and the inactive group (181.94 mg/dl). The high density lipoprotein level was significantly lower in the inactive group (58.7 mg/dl) than in both the moderately active and highly active groups (75.92-76.33 mg/dl). Similarly, the inactive group had a significantly decreased mean
apolipoprotein A-I level (117.00 mg/dl) compared to the moderately active group (127.35 mg/dl) which was significantly less than the highly active group (133.60 mg/dl).

These results suggest that physical activity may have a greater effect on apolipoprotein A-I than other lipid subfractions, with apolipoprotein A-I levels increasing in proportion to the amount of physical activity (Nagao, 1988).

**Serum Lipid Research - the Conflicting Results**

There appears to be a large contingent of experts (Ballantyne et al, 1982; Christie et al, 1980; Danner et al, 1984; Gaesser & Rich, 1984; McNaughton & Davis, 1987; Rauramaa et al, 1984; Shephard, 1979, 1986, 1989, Streja & Mymin, 1979) who believe that moderate aerobic physical activity is important in improving the lipid profile. Despite this, there are numerous studies which provide evidence refuting any causal relationship. One such study was completed by Christie, Bloore, and Logan (1980) where elevated serum high density lipoprotein levels had been regularly displayed in middle aged long distance runners, which is a factors which has been often associated with decreased coronary heart disease risk. In an attempt to provide support for this concept, Christie et al, (1980) studied 25 sedentary Rotarians and active harrier club members over two weeks to determine the differences in high density lipoprotein levels. Each participant completed a questionnaire to ascertain the duration and extent of their physical activity, family history, and life-style habits. Total
cholesterol, high density lipoprotein, and glucose were determined.

The results showed that the harriers spent an average of 4.5 hours jogging between 32-80 kilometres per week. None of the Rotarians undertook regular physical activity. The mean resting heart rate of the harriers (54.5 beats/minute) was significantly lower than that of the Rotarians (63.6 beats/minute). Similarly, the total serum high density lipoprotein concentration of 1.19 mmol/L in the Rotarians was significantly lower than that of the harriers (1.42 mmol/L). No other significant results were obtained. The study did not show that jogging, a form of moderate aerobic physical activity, would significantly alter the lipid profile even though this had been noted in previous studies. The changes in high density lipoprotein concentration appeared to be related to the intensity of exercise. However, Christie et al. (1980) concluded that it was not possible to say whether it was the distance or the duration of jogging which had the greatest metabolic effect on lipoproteins. Therefore, it may well be that those individuals who wish to significantly alter positively their risk factor for coronary heart disease by jogging alone, may have to run for a longer duration. As Christie et al. (1980) have suggested, these individuals will literally have to "run for their lives".

Despres, Bouchard, Savard, Tremblay, and Allard (1984) also demonstrated from their study that the relationship between physical activity and the lipid profile was not conclusive. Thirteen normal healthy male subjects (mean age 25.5 years) trained four
to five days per week, for 45 minutes at 85 per cent of heart rate reserve for a twenty week period. No control group was included in this study. Maximum aerobic power was determined using a bicycle ergometer, and percentage body fat was determined using hydrostatic weighing, before and after the training program. Blood samples were drawn during the two testing sessions for total serum cholesterol, triglycerides, and high density lipoprotein concentrations.

Overall, there were significant changes in all of the non serum lipid characteristics of the subjects. Body weight decreased significantly (2.4 kg) as did percentage body fat (2.7 per cent), while oxygen uptake increased significantly from 41.9 ml.kg.min.$^{-1}$ to 53.6 ml.kg.min.$^{-1}$. Of the lipid fractions, only total cholesterol decreased significantly from 186.6 mg/dl to 172.1 mg/dl. High density lipoprotein remained almost constant at 43-44 mg/dl whilst the ratio of high density lipoprotein to total cholesterol was stable at 0.27. Despres et al. (1984) reported that the study may have involved exercise at an intensity which may have led to the lack of statistically significant changes in serum lipid concentrations. Furthermore, the subjects were healthy young males with normal lipid profiles, which makes alterations in the lipid levels difficult to detect. The nature of the study group was also suggested as a reason for no relationship between total body fat, and the serum lipid levels being shown. It was concluded that non obese subjects often displayed decreased plasma lipid levels compared to obese individuals.
Similar, non-conclusive findings have been found in other studies. Bassett-Frey, Doerr, Laubach, Mann, and Glueck (1982) showed that neither interval or continuous training programs significantly altered the lipid profile, whilst other studies have shown that performance enhancing drugs such as steroids may alter the effect of physical activity on the lipid profile (Cohen, Faber, Benade, & Noakes, 1986; Costill, Pearson, & Fink, 1984). A majority of these studies suffered from poor experimental design techniques including limited pre test screening, not including a control group, poorly selected subjects, a lack of exercise quantitation, contamination of the control group with the treatment (exercise), and not eliminating confounding variables.

Although the debate over what effect physical activity has upon the lipid profile is far from settled, there is a general consensus that physical activity does perform a protective function against coronary heart disease (Ballantyne et al, 1982; Christie et al, 1980; Danner et al, 1984; Gaesser & Rich, 1984; McNaughton & Davis, 1987; Pfaffenbarger, 1979; Rauramaa et al, 1984; Seals & Hagberg, 1984; Shephard, 1979, 1986, 1989; Stewart, 1989; Streja & Mymin, 1979; Superko, 1988; Superko & Haskell, 1987; Sutton, 1979; Wood et al, 1983; Zauner, 1968; Zauner & Benson, 1977).
CHAPTER III

METHODOLOGY

Selection of Subjects

Forty males, aged 33-59 years, were randomly selected into trained (exercise) and untrained (control) groups, from office staff within the central business district of Wollongong, and from B.H.P. Steel, Port Kembla. Each subject was sedentary (partaking in no organised sporting or recreational activities), healthy (no history of major medical problems), and were unfit individuals (an aerobic capacity of less than 40 ml. kg. min\(^{-1}\)).

Demographically, the 35-64 year age group have been shown to have a higher risk of coronary heart disease than the general population, with coronary heart disease risk generally increasing exponentially with age (Farmer & Miller, 1985; Hyslop, Dowland, & Hickling, 1983). However, the incidence of mortality from coronary heart disease in the elderly (75 years and over) is thought to be distorted as other causes of death, especially sudden death, are often reported as coronary heart disease related deaths. Therefore, for this study, 33-59 year old subjects were selected.

Although historically, manual workers have a higher incidence of coronary heart disease, it is recognised that physical inactivity is a likely contributing factor to increased coronary heart disease risk.
(Shephard, 1986). Furthermore, longitudinal studies including the London bus drivers study (1953) and the Longshoreman study (1977) have shown that inactivity in the work place may also be related to increased coronary heart disease risk.

For this reason, office workers were invited to partake in this study. Also, it may have been difficult to allow for differing levels of work energy output with manual workers, a problem that should not present itself with fully sedentary office workers involved in similar office routines.

It has been suggested that the failure of many studies investigating the relationship between physical activity and the lipid profile has been due to the selection of individuals with initially low lipid levels. This seems to be a typical characteristic of non-obese, aerobically fit individuals. For this reason, low fit, slightly obese (over 15 per cent body fat) subjects were studied.

Finally, the risk of coronary heart disease is considerably enhanced in males than in females (Grant & Lapsey, 1984; Hyslop et al, 1983). Also, the use of both males and females during this study may have resulted in some gender bias, especially with such a limited population. Therefore, a protocol involving male subjects only was undertaken.
Height, Body Weight, and Percentage Body Fat Analysis

Subjects height and weight were measured using a Mercury balance scale and somatometre (model 211daPD). Individual height was measured at the beginning of the study, while all subjects were weighed and had body fat percentage calculations completed at \( T = 0 \), \( T = 6 \), and \( T = 12 \) weeks during the study.

Percentage body fat was determined with four selected skin fold measurements using Harpenden callipers, using standard techniques;

a) Biceps:
- over the mid-point of the muscle belly with the arm resting supinated on the subjects thigh.

b) Triceps:
- over the mid-point of the muscle belly, mid-way between the olecranon and the tip of the acromion with the upper arm hanging vertically.

c) Subscapular:
- just below the tip of the inferior angle of the scapula, at an angle of 45 degrees to the vertical.

d) Suprailiac:
- just above the iliac crest in the mid axillary line.

(Durnin & Rahaman, 1967).

At these four sites, the skin fold was pinched up firmly between the thumb and forefinger, and pulled slightly away from the underlying tissues prior to applying the skin fold callipers for the measurement (Durnin & Rahaman, 1967).
The percentage of body fat was calculated using the formula of Durnin and Rahaman (1967) (see appendix).

**Health and Fitness Questionnaire**

All subjects completed a questionnaire to evaluate their level of health and fitness, prior to the start of testing (see appendix). The questionnaire was developed from several questionnaires, and was primarily based on the Health and Fitness Institute of Florida Medical History Questionnaire.

**Dietary evaluation**

A 24 hour recall record was kept by each subject of dietary intake for the 24 hour period immediately prior to each physiological testing session (T= 0, 6, & 12 weeks). This evaluation was undertaken for the purpose of establishing if the subjects maintained a similar diet, prior to each of the three testing sessions.

**Serum Lipid Evaluations**

Seven lipid subfractions were evaluated during the three (3) testing sessions over the duration of the study:
These included:

- total serum cholesterol (CHOL)
- total serum triglyceride (TRIG)
- high density lipoprotein (HDL-C)
- low density lipoprotein (LDL-C)
- very low density lipoprotein (VLDL-C)
- apolipoprotein A-1 (APO-A1)
- apolipoprotein B (APO-B)

All of these components, except for the two apolipoprotein measures, were determined using the Kodak Ektachem DT60 Analyser. The Kodak Ektachem DT60 Analyser is a system for in-vitro diagnosis of serum, plasma, and whole blood specimens. Using specific reagents contained within the appropriate Kodak Ektachem DT slides, a 10 microlitre sample was used to complete a single test. Prior to all testing being carried out, a series of calibrations and control experiments were completed to ensure that the analyser was reading accurately.

The Kodak Ektachem DT slides for testing serum cholesterol, serum triglyceride, and high density lipoproteins, are dry and multi layered, with a self contained analytical element coated on a clear polyester support. Each of these lipid subfractions reacts with specific reagents to accurately determine the concentrations of each component in the blood sample. Using reflective spectrometry, the Kodak DT 60 analyser can determine these three serum lipid levels.
3.1. Specimen Preparation

Three separate slides determining serum cholesterol, serum triglyceride, and high density lipoprotein, were analysed in order to obtain the complete lipid and lipoprotein subfraction concentrations. From these three components, the low density lipoprotein and very low density lipoprotein concentrations were calculated using two simple formulae (see page 83).

3.1.1. Serum Cholesterol and Serum triglyceride

Sample preparation for the total serum cholesterol and the serum triglyceride analysis are identical. A venous blood sample was drawn to complete all of the serum lipid blood evaluations. The sample was centrifuged using the Heraeus Sepatech Biofuge A Centrifuge, at 4500 revolutions, for ten minutes. This ensured the complete separation of the erythrocytes to a condensed pellet, distinct from the resultant containing blood plasma. The Ektachem analysis was completed immediately to avoid any decrease in cholesterol solubility and concentration. Approximately 2 millilitres of the blood plasma was pipetted from the blood sample tube into an Ektachem DTE dual sample cup using the Labysystems Finnpipette adjustable pipette (model 4007 010). Finally, 10 microlitres of the plasma contained in the Ektachem DTE dual sample cup was pipetted using the Kodak Ektachem DT pipette onto the appropriate Ektachem DT slide (cholesterol and triglyceride) for analysis.
3.1.2. High Density Lipoprotein

Evaluation of the high density lipoprotein concentration also required a venous blood sample, with each subject required to undergo 12-14 hours of fasting prior to drawing the specimen. Two millilitres of the venous was pipetted into the Ektachem sample tube, which contains a chemical reagent (see High Density Lipoprotein analysis, page 83), and centrifuged for 5 minutes at 5000 revolutions. Ten microlitres of the resultant was then spotted onto the high density lipoprotein Kodak Ektachem DT slide.

3.2. Serum Cholesterol analysis

Chemicals in the spreading layer assists in dissociating cholesterol and cholesterol esters from lipoprotein complexes in the 10 microlitre specimen. In a reaction catalysed by the cholesterol ester hydrolase, cholesterol esters are hydrolysed to cholesterol. Cholesterol is then oxidised to form cholesatenone and hydrogen peroxide.

Finally, hydrogen peroxide oxidises a triarylimidazole leuco dye in the presence of peroxidase to generate a coloured product. The dye is measured through the transparent polyester support layer using a reflective spectrometer, with the white underside of the spreading layer acting as a diffuse reflector.
3.3. Serum Triglyceride analysis

This analysis is based on a totally enzymatic method to generate hydrogen peroxide. A subsequent reaction between the hydrogen peroxide and a leuco dye produces a coloured compound. Calorimetric measurement by reflectant spectrophotometry again provides the basis for determining the triglyceride concentration.

A surfactant within the spreading layer dissociates triglycerides from lipoprotein complexes in the 10 microlitre specimen. These triglyceride molecules are then hydrolysed by lipids to yield glycerol and fatty acids. The glycerol molecules are small enough to diffuse through the scavenger layer on the slide, to the reagent layer below, where they are phosphorylated by adenosine triphosphate (ATP) in the presence of glycerole kinase and magnesium chloride.

3.4. High Density Lipoprotein analysis

When testing for high density lipoprotein, the sample must be first prepared using the Ektachem HDL Cholesterol Kit. This involves treating the specimen with a reagent, dextran sulphate/magnesium, to remove the very low density and low density lipoproteins, which also contain cholesterol. The amount of high density lipoproteins can then be accurately determined.

During this analysis, cholesterol esters are hydrolysed to cholesterol, which is then oxidised to generate hydrogen peroxide. The hydrogen peroxide oxidises a leuco dye in a peroxidase
catalysed reaction to produce a coloured compound. Calorimetric measurement by reflectance spectrophotometry are then used to determine the concentration of high density lipoproteins.

Again, 10 microlitres of the sample is pipetted onto the slide, with the surfactant in the spreading layer dissociating cholesterol and cholesterol esters from the high density lipoprotein complexes. In a reaction catalysed by cholesterol ester hydrolase, cholesterol esters are hydrolysed to cholesterol, which is then oxidised in the presence of cholesterol oxidase to form cholestenone and hydrogen peroxide. Hydrogen peroxide oxidises a trilimidazole leuco dye, in the presence of peroxidase, to generate a coloured product.

3.5. Low Density Lipoprotein (LDL) and Very Low Density Lipoprotein (VLDL) analysis

The Kodak Ektachem DT slides for obtaining serum cholesterol, serum triglyceride, and high density lipoprotein levels can be used in combination to accurately calculate the low density lipoprotein and very low density lipoprotein levels.

The formulations for the calculations are:
(modified for S.I. units for the Kodak DT60 analyser)

a) Very low density lipoproteins (VLDL) = Triglyceride/2.07
b) Low density lipoproteins (LDL) = Cholesterol - HDLC - VLDL
   = Chol.- HDLC- Trigs./2.07
For the purpose of serum lipid appraisal, the three components of total cholesterol, triglycerides, and high density lipoprotein have been shown to return accurate measures of both low density and very low density lipoprotein concentrations (Friedwald et al, 1972; Kannel et al, 1972).

3.6. Apolipoprotein A-I and B analysis

A sample of 200 microlitres of blood plasma obtained from the venous blood sample drawn for serum lipid evaluation was delivered to the Wollongong Hospital Biochemistry Laboratory, where the apolipoprotein tests were carried out. The apolipoprotein subfractions were analysed using the Beckman Array Protein System.

The Beckman Array Protein System automatically performs the apolipoprotein tests, treating the specimen with the appropriate reagents as required. This system involves an in-vitro diagnostic quantitation of components of biological fluids by rate nephelometry. Rate nephelometry measures the intensity of light as it is scattered by particles in suspension in a flow cell, when a beam of light is passed through the flow cell. These particles are formed by the immunoprecipitin reaction that occurs when a specific anti body is brought in contact with the specific antigen (apolipoprotein A-I or B).
Cardiovascular Analysis

Eight cardiovascular variables were measured during the three (3) testing sessions. The first series of tests (T =0 weeks) indicated the sedentary fitness level of the subjects. Any subjects who recorded an aerobic capacity in excess of 40 ml. kg. min.\(^{-1}\) were excluded from the study. Both the untrained and trained groups participated in the second (T =6 weeks) and third (T = 12 weeks) testing sessions which measured the training effect of the physical activity.

The variables which were monitored included;

a) resting electrocardiogram
b) exercising electrocardiogram
c) resting heart rate
d) exercising heart rate
e) resting blood pressure
f) exercising blood pressure
g) post exercise blood pressure over 30 minutes (to determine return of blood pressure to resting values)
h) aerobic capacity \((\text{VO}_2 \text{ max})\)

All of the variables above were measured at the Stress Laboratory at the Wollongong Hospital.
3.7. Electrocardiogram Analysis.

Electrocardiograms (ECG) were recorded both at rest and during exercise, at each of the three testing sessions (T = 0, T = 6, and T = 12 weeks).

3.7.1. Resting electrocardiogram (ECG)

The resting electrocardiogram was recorded using the Quinton 5000 Exercise Test Monitor. This monitor is a full exercise test system, which acquires, displays and prints exercise test data from each subject. The E.C.G. recording consisted of the conventional 12 lead ECG, immediately prior to commencing the exercise test, with the subject remaining stationary and standing on the treadmill. The subjects were instructed before any testing, to refrain from smoking for at least 1 hour and to fast for 12-16 hours in preparation for the serum lipid analysis.

Standard electrode positions for an exercising electrocardiogram were used in recording the 12 lead ECG as described below:

- **limb leads**
  a) VR - (right arm) - positioned on the right clavicular margin in the mid axillary line.
  b) VL - (left arm) - positioned on the left clavicular margin in the mid axillary line.
c) VF - (left leg) - positioned on the left costal margin at the mid clavicular line chest leads
d) V1 - right internal margin at 4th intercostal space.
e) V2 - left internal margin at 4th intercostal space.
f) V4 - intersection of left mid clavicular line and 5th intercostal space.
g) V3 - midway between V2 and V4.
h) V5 - mid axillary line with a horizontal line through V4.
i) V6 - posterior axillary line with a horizontal line through V4 and V5.

ground lead
j) Gr - positioned on the right costal margin at the mid clavicular line.

(Marriot, 1983)

3.7.2 Exercising electrocardiogram (ECG)

The exercising electrocardiogram was recorded during the maximal graded treadmill exercise tests. All tests were step wise, with a constant treadmill speed and increases in treadmill gradient (see aerobic testing, page 90).

Using the Quinton 5000 Exercise Test Monitor, a conventional 12 lead ECG was recorded, with identical electrode placements as in the resting E.C.G (see pages 86-87).
Interpretation of both the resting and exercising electrocardiographs involved identifying persistent ST segment depression (at least 1 mm that is horizontal or down sloping at 0.08 seconds after the J point). According to the American College of Sports Medicine (1986), an ST segment depression which occurs early in exercise and remains throughout testing is more likely to be due to coronary heart disease than ST depressions that usually occurs at high intensity exercise.

3.8. Heart Rate analysis

Heart rate calculations carried out during the physiological testing sessions were determined directly from the printout obtained from the Quinton 5000 Exercise test Monitor. The monitor automatically displayed and recorded heart rates at 3 second intervals during the entire maximal aerobic capacity test, and these values were recorded on a summary printout at the conclusion of the test.

a) resting heart rate
   - the lowest heart rate recorded during the resting electrocardiograph.

b) maximal heart rate
   - the maximum heart rate recorded during the maximal aerobic treadmill (VO$_2$ max) test.
3.9. Blood Pressure analysis

All blood pressure recordings were determined indirectly, via auscultation. During the pre exercise test and exercise test phases, blood pressures were automatically recorded with a sphygmomanometer cuff placed on the left arm, using the Quinton 5000 Exercise Test Monitor. Again, the blood pressure readings were displayed and recorded on the Quinton 5000 Exercise Test Monitor, at 3 second intervals.

Blood pressure recovery to 30 minutes post testing was recorded by manual mercury sphygmomanometry from the left arm, with the subject seated. According to Seals and Hagberg (1984), when manual auscultation is used, at least three measurements must be made to assess blood pressure levels accurately.

a) resting blood pressure
   - resting blood pressure was recorded immediately before the resting electrocardiograph was performed, and prior to the maximal aerobic treadmill (VO₂ max) test.

b) exercising blood pressure
   - in accordance with the A.C.S.M. guidelines (1986), blood pressure was monitored at each stage of the maximal aerobic treadmill (VO₂ max) test.

c) post exercise (recovery) blood pressure
   - Kaufman et al (1986), have suggested monitoring heart rate up to 60 minutes after the cessation of physical activity. However, in this study, the
haemodynamic responses will be measured at 0, 5, 10, 15, and 30 minutes of recovery, due to time constraints.

3.10. Aerobic Capacity (VO$_2$ max) analysis

The Modified Balke treadmill protocol was followed during the maximal aerobic capacity test during this study (see appendix). This protocol was the protocol of choice and the protocol currently being performed at the Wollongong Hospital Stress Laboratory. The test involved a constant treadmill speed of 3.4 mph (5.4 kph), with a step wise 1 per cent increase in gradient at 1 minute intervals until the subject reached their maximal aerobic capacity. This was indicated by a heart rate plateau at high work intensities, or when the test was terminated due to the presence of any of several contraindications as set out by the A.C.S.M. (1975, 1986).

This protocol which involves dynamic exercise, with a step wise progression in workload, has several advantages for high risk subjects. Most importantly, there is a controllable increase in cardiac output, which means that the subjects can be protected from rapidly increasing myocardial oxygen demand. Furthermore, this protocol has the advantage over other protocols which involve step wise changes in treadmill speed in that the constant speed requires only an initial adaptation of stride. This reduces the amount of technical adjustments required when compared to a running protocol. A constant speed protocol also produces less electrocardiograph and blood pressure artifact than in protocols
involving higher treadmill speeds (Detrano and Froelicher, 1988).

3.11. The Aerobic Conditioning Program

The training program prescribed for the experimental group members focused on aerobic endurance, and was primarily designed to:

a) improve cardiorespiratory fitness

b) see if the subjects undertaking regular aerobic physical activity displayed significant changes in serum lipid concentrations.

The program was designed under the American College of Sports Medicine Guidelines (1986), and was specifically developed for sedentary, low fit, healthy subjects. Stage one aerobic activity (i.e. walking, jogging, cycling, swimming) are recommended for these individuals as the exercise intensity is easily sustained, with little variation in heart rate response. Furthermore, it has been shown in several studies that offering a greater variety of activities in such studies as this, leaves room for great fluctuations between individuals with respect to exercise effort quantitation (Joseph & Bena, 1977; McCunney, 1987; Shephard, 1989). Therefore, the subjects were instructed to attend conditioning sessions during which walking/jogging was carried out to an intensity, predetermined from the initial physiological testing.

The intensity of the aerobic activity was determined using target heart rates, as it was easy for the subjects to self monitor during
physical activity. Also, a general linear relationship has been shown to exists between heart rate and exercise intensity (A.C.S.M., 1986). Using maximum heart rate and resting heart rate data obtained during the initial physiological testing session, a target heart rate range for aerobic conditioning can be set, using the following formula;

\[ \text{Target HR} = .x \times (F_{\text{max}} - F_{\text{rest}}) + F_{\text{rest}} \]

where: \( .x \) = constant at 0.6 (lower target HR limit)

and 0.9 (upper target HR limit)

\( F_{\text{max}} \) = maximum heart rate

\( F_{\text{rest}} \) = resting heart rate

(A.C.S.M. 1986)

The conditioning phase of the exercise was over 10-20 minutes for these healthy, asymptomatic adults for the initial two weeks, increasing to a maximum of 45 minutes after eight weeks, as to accommodate the training effect, as shown below:

a) 1 week x 10-15 min. x 3 days per week
b) 1 week x 15-20 min. x 3 days per week
c) 2 weeks x 20-30 min. x 3 days per week
d) 2 weeks x 30 min. x 4 days per week
e) 2 weeks x 35-40 min. x 4 days per week
f) 4 weeks x 40-45 min. x 5 days per week

These individuals should be able to maintain exercise at 5-8 METS (walking/jogging a twelve minute mile), 3-7 periods per week (A.C.S.M., 1986). To obtain the maximum training effect, and in an attempt not to compromise exercise adherence, the experimental group members attended organised training sessions 1 to 3 times
per week over the study period. Each subject was instructed as to how to obtain a heart rate estimation using radial artery palpation, and were required to maintain a heart rate in the training zone as described earlier (page 92).

**Statistical Analysis**

The data was analysed using Statview 512+, Minitabs, and SPSS-X statistical packages. A one way analysis of variance (ANOVA) and a non parametric Mann Whitney test were completed to determine the between group differences at T = 0, 6, and 12 weeks. A repeated measures factorial ANOVA was then performed to determine the group and time main effects, and the group by time interactions. Finally, Pearson correlation coefficients were completed to determined the degree of relationship between specific criterion and predictor variables. All descriptive data is presented as $X = \text{SEM}$. 
CHAPTER IV

PROCEDURE

Initial contact was made with prospective subjects during an introductory seminar, two months prior to the study. During this session, the purpose and expected outcomes of the study were explained, as well as each individual being made aware of the commitment in terms of time devoted to the study. Furthermore, the benefits available to the individuals from this research project were discussed. Following this hour long meeting, 40 subjects were signed up to partake in the study.

Each subject was randomly assigned to one of two groups:

a) trained group (experimental group)
   - following a twelve week program of mild aerobic activity, on a static diet (i.e. no dietary intervention).

b) untrained group (control group)
   - undertaking no physical activity, on a static diet.

During the week immediately prior to the study commencing, each subject attended a familiarisation session to become aware of all of the procedures which were to be used during subsequent physiological testing sessions. All procedures including blood collection techniques, cardiovascular analysis, and training programs were addressed. This session was an attempt to reduce
pre test anxiety. During this session, the lifestyle, fitness, and health evaluation questionnaire was completed (see appendix).

All subjects completed a 24 hour dietary recall during the 24 hour period immediately preceding each of the three physiological testing sessions. All subjects fasted for 8-12 hours before testing for the serum lipid evaluation. It has been suggested that a fasting period is not necessary for most lipid determinations, including apolipoproteins (Hester, Shephard, Walmsley, & White, 1989). However, fasting was carried out because the Wollongong Hospital Biochemistry Department suggested it.

The Testing Sessions

During each testing session (T =0, T = 6, T = 12 weeks), each subject underwent kinanthropometric, cardiovascular, and serum lipid evaluation, as described in the methodology section (pages 77-91).

4.1. Equipment calibration

Prior to each subject being tested, several calibrations were carried out. The metabolic cart used to analyse oxygen and carbon dioxide fractions in expired air, was calibrated using a sample of known gases. The ventilation meter was calibrated using a 1000 millilitre syringe, expired 10 times, with a value between 9.9 to 10.1 litres being accepted. Between tests, mouth pieces were replaced and sterilised.
4.2. Physiological Testing

Each subject underwent an identical testing procedure. Prior to the initial test, all subjects completed informed consent forms for both the study, and the maximal aerobic test (see appendix).

Subjects had their chests shaved and sterilised with alcohol in preparation for the 12 lead electrocardiograph. Following this, a 10 millilitre sample of venous blood was drawn and centrifuged. The serum plasma was pipetted into a second tube and stored for analysis, with the remaining blood sample being discarded.

Kinanthropometric evaluations were then completed. Skin fold measurements were taken three times and then averaged. Weight and height were also recorded as stated in the methodology section (page 77).

The subjects then had electrocardiograph electrodes positioned and the resting electrocardiograph was recorded, prior to placing the mouth piece from the oxygen analysis apparatus in place.

Following the positioning of the mouth piece, each subject was advised of several hand signals which would be used during the test to communicate with the testers. Subjects then walked up to the 5.5 k.p.h. pace of the treadmill test protocol, and thereafter, the test commenced.
The Modified Balke protocol used during testing terminated after a maximum of 26 minutes. Following the completion of the test, each subject was walked down, and the mouth piece was then removed. The subjects remained seated until 5 minutes recovery had elapsed, at which time they were removed from the exercise test monitor and completed the 30 minute blood pressure recovery period as explained in the methodology section (page 89).

Subject to each individual obtaining a satisfactory return to testing blood pressure levels similar to resting values after 30 minutes, the testing session was then deemed complete.

An identical procedure was followed at each of the three testing sessions at T = 0, 6, and 12 weeks during the study.

The Aerobic Conditioning Sessions

The aerobic conditioning was completed as stated in the methodology section (page 91). All treatment group members were supplied with a dossier containing information about physical conditioning intensity, duration, frequency, and instructions on how to self monitor their individual training programs. Organised training sessions were initially conducted three times per week, tapering off to one organised training session per week, as the time commitment became greater nearing the final month of the study.
Following a 5 minute warm up, the subjects recorded their heart rates, which allowed them to estimate how much harder they would need to exercise to maintain their individual target heart rates. At the conclusion of the conditioning phase, each subject again recorded their heart rate, and proceeded with a slow cool down.

This procedure was to be followed by all treatment group subjects when conditioning was carried out away from the organised treatment sessions.
CHAPTER V

RESULTS

This chapter summarises and presents results and descriptive statistics (i.e. means and standard errors of the means) for the untrained controls and trained groups, for all variables investigated during this study.

Subjects

All of the 40 subjects originally included in the study completed all testing sessions during the 12 week study. However, three subjects in the untrained control group were excluded from the results after reporting that they had become contaminated with the treatment variable (aerobic physical activity), or had markedly modified their diets as indicated by the 24 hour dietary recalls.

No significant differences were found between the untrained and trained groups for age, height, weight, percentage body fat, and aerobic capacity ($\text{VO}_2\text{ max}$) during initial testing at week $T = 0$ ($p > 0.05$) (see Table 2). Both the untrained and untrained groups showed non significant reductions in weight of 0.9 kg and 1.9 kg respectively over the 12 week study ($p > 0.05$). However, the trained group showed a significant decrease from 21.8% to 19.5%
(p < 0.01) in percentage body fat, while the untrained group produced no significant difference (p > 0.05) (see table 3).

Three of the untrained group members and two of the members in the trained group showed significant electrocardiograph abnormalities, defined as S-T segment depressions of in excess of 1 mm, as stated by the A.C.S.M. (1986).

**Heart Rate**

No significant differences in resting heart rates were shown between the untrained and trained groups prior to physical conditioning (p > 0.05). The resting heart rate of the trained group decreased significantly from 72.8 bpm to 67.6 bpm (p < 0.05), whilst the untrained group showed a slight increase from 72.2 bpm to 72.9 bpm, following the 12 week study. Both groups maintained a maximal exercising heart rate during the three treadmill tests of between 169.4 bpm to 174.2 bpm, again with no group displaying significant changes over the 12 week study period (p > 0.05) (see table 4).

**Haemodynamic Responses**

Resting systolic and diastolic blood pressure showed no significantly different results at T = 0 weeks between the untrained and trained groups (132/85 mm Hg and 127/82 mm Hg respectively). There were no significant changes in resting blood pressure for the trained group. However, the untrained group showed a significant reduction in both resting systolic (132.2 mm
Hg to 120.2 mm Hg) and diastolic (85.1 mm Hg to 79.2 mm Hg) blood pressure over the 12 week period (p < 0.01).

During the treadmill test at T = 6 weeks, the trained group showed a significant reduction in maximal systolic blood pressure of 32.6 mm Hg from 232.4 mm Hg to 199.8 mm Hg (p < 0.05). Conversely, the untrained group's mean maximal systolic blood pressure did increase significantly by 17.9 mm Hg from 214.0 mm Hg to 231.9 mm Hg between testing at T = 0 and T = 12 weeks (p < 0.05). The trained group produced a significant increase in the 30 minute post exercise systolic blood pressure from 110.6 mm Hg to 115.9 mm Hg at T = 6 weeks. This was followed by a significant reduction in the 30 minute post exercise diastolic blood pressure from 78.2 mm Hg to 71.8 mm Hg, at T = 12 weeks (p < 0.05).

All haemodynamic responses are summarised in tables 5 and 6.

**Aerobic Capacity**

There were no significant differences between initial aerobic capacities for the untrained and trained groups (31.5 ml.kg.min⁻¹ vs. 33.2 ml.kg.min⁻¹) (p > 0.05). However after 12 weeks of training, a significant increase in aerobic capacity to 39.1 ml.kg.min⁻¹ was shown in the trained group (p <0.01). The untrained group, although showing a significant increase in aerobic capacity of 1.9 ml.kg.min⁻¹ at T = 6 weeks (p < 0.05), decreased there aerobic capacity thereafter to 32.1 ml.kg.min⁻¹ (p > 0.05) (see table 3).
Several significant correlations were shown between aerobic capacity and specific serum lipid subfractions. A significant positive correlation existed at the 5 per cent level for high density lipoproteins \( r = 0.34 \); the ratio of high density lipoproteins to serum cholesterol \( r = 0.35 \); and for the ratio of apolipoprotein A-1 to apolipoprotein B \( r = 0.33 \), for the trained group. Furthermore, a significant negative correlation existed at the 5 per cent level between aerobic capacity and high density lipoproteins \( r = -0.33 \) and the ratio of high density lipoproteins to serum cholesterol \( r = -0.34 \), for the untrained group. Finally, a significant negative correlation was found at the 5 per cent level between aerobic capacity and very low density lipoproteins, for both the untrained and untrained subject (see table 11).

**Cholesterol and Triglycerides**

Again, there were no significant differences in group means for either serum cholesterol or serum triglyceride levels during initial testing \( p > 0.05 \). Significant reductions in serum cholesterol levels were recorded in both the untrained group (6.1 mmol/L to 5.3 mmol/L) and the trained group (6.1 mmol/L to 5.0 mmol/L), over the 12 week study \( p < 0.01 \).

While the mean serum triglyceride levels in the untrained group remained constant at 1.7 mmol/L, the trained group mean declined significantly from 1.8 mmol/L to 1.2 mmol/L \( p < 0.01 \) (see table 7).
A strong positive correlation was shown between serum triglyceride levels and weight in the trained subjects (r = 0.54). This result was significant at the 1 per cent level (see table 11).

**Lipoproteins**

There were no significant differences in group means for any of the three serum lipoprotein subfractions of high density lipoproteins, low density lipoproteins, and very low density lipoproteins, during initial testing at T = 0 weeks (p > 0.05).

Although both the untrained and trained groups showed significant decreases in high density lipoproteins at T = 6 weeks (p < 0.05), there was no significant difference between the untrained and trained subjects at the conclusion of the 12 week study. The untrained subjects showed a reduction in high density lipoproteins from 1.34 mmol/L to 1.15 mmol/L while the trained group showed a decrease from 1.28 mmol/L to 1.16 mmol/L (see tables 8 and 9).

Mean low density lipoprotein levels decreased significantly in both groups by T = 12 weeks. A 17.1 per cent decrease was observed in the trained group from 3.97 mmol/L to 3.29 mmol/L (p < 0.05). A smaller 16.0 per cent decrease from 4.01 mmol/L to 3.34 mmol/L was shown in the untrained group (p < 0.05).

There was also a significant change in the trained group for very low density lipoproteins (p < 0.01). The trained group reduced very low density lipoproteins from 0.88 mmol/L to 0.58 mmol/L,
whilst the untrained group remained stable at 0.80 mmol/L (see tables 8 and 9).

Both low density lipoproteins $(r = 0.51)$ and very low density lipoproteins $(r = 0.54)$ produced significant positive correlations with body weight in the trained group at the 1 per cent level. Very low density lipoproteins also produced a significant, but negative correlation with aerobic capacity $(r = -0.33)$ for both study groups, at the 5 per cent significance level.

The changes in serum lipid and lipoproteins were not reflected in any of three ratios, high density lipoprotein to cholesterol; low density lipoprotein to cholesterol; or low density lipoproteins to high density lipoproteins $(p > 0.05)$. However, several important correlations were made. The ratio of high density lipoproteins to serum cholesterol in the trained group had a highly negative correlation with both body weight $(r = -0.48)$ and body fat $(r = -0.48)$, at the 1 per cent significance level. This ratio produced conflicting results when correlated with aerobic capacity. The trained group showed a positive correlation $(r = 0.35)$ while the untrained group a negative correlation $(r = -0.34)$. Both the trained and untrained results were significant at the 5 per cent level (see table 11).

In the trained group, the ratio of low density lipoproteins to serum cholesterol showed a significant positive correlation when compared to percentage body fat $(r = 0.41)$ at the 5 per cent level. Finally, the trained group also showed significant negative correlations between the ratio of low density lipoproteins to high
density lipoproteins with both body weight ($r = 0.49$) and percentage body fat ($r = 0.49$) at the 1 per cent level.

**Apolipoproteins**

There were no significant differences in apolipoprotein A-1 or Apolipoprotein B levels between groups at $T = 0$ weeks. Both the untrained and trained groups showed non significant changes in apolipoprotein A-1 levels after the 12 week study. The trained group concentration remained at 1.06 g/L, whilst the untrained group concentrations decreased from 1.15 g/L to 1.10 g/L.

Conversely, both the untrained and trained groups showed significant decreases in apolipoprotein B. The trained group apolipoprotein level decreased from 0.94 g/L to 0.78 ($p < 0.01$) following the 12 week conditioning program. The untrained group's apolipoprotein B level however decreased significantly only at $T = 6$ weeks from 0.95 g/L to 0.89 g/L ($p > 0.05$). There was no significant decrease at $T = 12$ weeks for the untrained group. (see table 10).

Both the untrained and trained groups showed significant increases of 10.1 per cent and 19.7 per cent respectively in the ratio of apolipoprotein A-1 to B at the conclusion of the study ($p < 0.01$). The untrained group ratio increased from 1.28 to 1.41, while the trained group increased from 1.17 to 1.40 (see table 10).

Apolipoprotein B showed a positive correlation with body weight ($r = 0.55$), while the ratio of apolipoprotein A-1 to B showed a
negative correlation with body weight ($r = 0.54$) for the exercising group at the 1 per cent significance level. Finally, there was a significant positive correlation with the apolipoprotein A-1 to Apolipoprotein B ratio and aerobic capacity, at the 5 per cent significance level (0.33) for the trained group (see table 11).
### TABLE 2
Means and Standard Errors for Physical Characteristics of Untrained and Trained Subjects (T = 0 Weeks)

<table>
<thead>
<tr>
<th></th>
<th>Untrained Subjects (n = 17)</th>
<th>Trained Subjects (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>44.5 ± 1.5</td>
<td>42.4 ± 1.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175.7 ± 1.7</td>
<td>177.7 ± 1.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>86.7 ± 3.3</td>
<td>80.6 ± 1.9</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>21.5 ± 0.7</td>
<td>21.8 ± 0.5</td>
</tr>
<tr>
<td>Max. VO₂ (ml/kg/min)</td>
<td>31.4 ± 1.6</td>
<td>33.2 ± 1.1</td>
</tr>
</tbody>
</table>

**Note:** Values are Mean ± S.E.M.
TABLE 3

Changes in Mean Physical Characteristics of Untrained and Trained Subjects From Initial (T = 0 Weeks) to Final (T = 12 Weeks) Testing

<table>
<thead>
<tr>
<th></th>
<th>Untrained Subjects</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial Test (T = 0)</td>
<td>Second Test (T = 6)</td>
<td>Final Test (T = 12)</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>86.7 ± 3.3</td>
<td>85.8 ± 3.4</td>
<td>85.8 ± 3.4</td>
<td></td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>21.5 ± 0.7</td>
<td>21.8 ± 0.7</td>
<td>22.4 ± 0.7</td>
<td></td>
</tr>
<tr>
<td>Max. VO₂ (ml/kg/min)</td>
<td>31.4 ± 1.6</td>
<td>34.8 ± 2.1*</td>
<td>32.1 ± 1.2</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Trained Subjects</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial Test (T = 0)</td>
<td>Second Test (T = 6)</td>
<td>Final Test (T = 12)</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>80.6 ± 1.9</td>
<td>80.2 ± 1.8</td>
<td>78.7 ± 1.7</td>
<td></td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>21.8 ± 0.5</td>
<td>20.9 ± 0.5</td>
<td>19.5 ± 0.6**</td>
<td></td>
</tr>
<tr>
<td>Max VO₂ (ml/kg/min)</td>
<td>33.2 ± 1.1</td>
<td>35.9 ± 0.9**</td>
<td>39.1 ± 1.3**</td>
<td></td>
</tr>
</tbody>
</table>

Note: Values are Mean ± S.E.M.
* p < 0.05
** p < 0.01
Figure 1. Changes in aerobic capacity ($VO_2_{max}$) for untrained and trained subjects observed over 12 weeks.
<table>
<thead>
<tr>
<th></th>
<th>Untrained Subjects (N = 17)</th>
<th>Trained Subjects (N = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Resting Heart Rate (beats/min)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T = 0</td>
<td>72.2 ± 3.4</td>
<td>72.8 ± 2.3</td>
</tr>
<tr>
<td>T = 6</td>
<td>73.4 ± 3.5</td>
<td>68.0 ± 2.0*</td>
</tr>
<tr>
<td>T = 12</td>
<td>72.9 ± 3.3</td>
<td>67.6 ± 1.6*</td>
</tr>
<tr>
<td><strong>Maximal Heart Rate (Beats/min)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T = 0</td>
<td>172.8 ± 5.4</td>
<td>169.8 ± 3.9</td>
</tr>
<tr>
<td>T = 6</td>
<td>172.6 ± 5.3</td>
<td>174.2 ± 2.5</td>
</tr>
<tr>
<td>T = 12</td>
<td>169.4 ± 5.7</td>
<td>172.5 ± 3.8</td>
</tr>
</tbody>
</table>

**Note:** Values are Mean ± S.E.M.
* p < 0.05
## TABLE 5

### Means Haemodynamic Responses During Physiological Testing for Untrained Subjects

<table>
<thead>
<tr>
<th></th>
<th>Initial Test (T = 0)</th>
<th>Second Test (T = 6)</th>
<th>Final Test (T = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting Systolic (mmHg)</td>
<td>132.2 ± 3.3</td>
<td>123.0 ± 2.3*</td>
<td>120.2 ± 3.0**</td>
</tr>
<tr>
<td>Resting Diastolic (mmHg)</td>
<td>85.1 ± 2.2</td>
<td>80.2 ± 1.9</td>
<td>79.2 ± 82.0**</td>
</tr>
<tr>
<td>Maximal Systolic (mmHg)</td>
<td>214.0 ± 6.5</td>
<td>211.8 ± 8.0</td>
<td>231.9 ± 8.0*</td>
</tr>
<tr>
<td>Maximal Diastolic (mmHg)</td>
<td>91.7 ± 1.8</td>
<td>89.0 ± 1.6</td>
<td>90.9 ± 1.4</td>
</tr>
<tr>
<td>Minimum Diastolic (mmHg)</td>
<td>55.6 ± 2.3</td>
<td>63.8 ± 2.3**</td>
<td>52.6 ± 2.7</td>
</tr>
<tr>
<td>30 Minute Post (mmHg)</td>
<td>116.2 ± 2.9</td>
<td>115.0 ± 2.8</td>
<td>113.5 ± 2.6</td>
</tr>
<tr>
<td>Exercise Systolic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 Minute Post (mmHg)</td>
<td>78.6 ± 2.0</td>
<td>77.2 ± 1.3</td>
<td>76.5 ± 1.6</td>
</tr>
<tr>
<td>Exercise Diastolic</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: Values are Mean ± S.E.M.*

* \(p < 0.05\)

** \(p < 0.01\)
<table>
<thead>
<tr>
<th></th>
<th>Initial Test (T = 0)</th>
<th>Second Test (T = 6)</th>
<th>Final Test (T = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting Systolic (mmHg)</td>
<td>127.1 ± 2.6</td>
<td>124.5 ± 2.5</td>
<td>121.6 ± 3.3</td>
</tr>
<tr>
<td>Resting Diastolic (mmHg)</td>
<td>82.0 ± 2.5</td>
<td>80.1 ± 1.8</td>
<td>79.7 ± 2.5</td>
</tr>
<tr>
<td>Maximal Systolic (mmHg)</td>
<td>232.4 ± 10.5</td>
<td>199.8 ± 6.4*</td>
<td>222.4 ± 8.7</td>
</tr>
<tr>
<td><strong>Maximal Diastolic (mmHg)</strong></td>
<td><strong>90.2 ± 1.8</strong></td>
<td><strong>86.3 ± 1.9</strong></td>
<td><strong>88.3 ± 2.0</strong></td>
</tr>
<tr>
<td>Minimum Diastolic (mmHg)</td>
<td>51.6 ± 2.2</td>
<td>54.1 ± 2.3</td>
<td>55.4 ± 2.6</td>
</tr>
<tr>
<td>30 Minute Post (mmHg)</td>
<td>110.6 ± 1.9</td>
<td>115.9 ± 1.9*</td>
<td>111.1 ± 2.0</td>
</tr>
<tr>
<td>Exercise Systolic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 Minute Post (mmHg)</td>
<td>78.2 ± 1.7</td>
<td>76.2 ± 2.3</td>
<td>71.8 ± 2.3*</td>
</tr>
<tr>
<td>Exercise Diastolic</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: Values are Mean ± S.E.M.*

* p < 0.05
### TABLE 7

Pre and Post Conditioning Changes in Serum Cholesterol and Triglyceride Concentrations for Untrained and Trained Subjects

<table>
<thead>
<tr>
<th></th>
<th>Untrained Subjects (N = 17)</th>
<th>Trained Subjects (N = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Cholesterol (mmol/L)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>T = 0</strong></td>
<td>6.1 ± 0.3</td>
<td>6.1 ± 0.3</td>
</tr>
<tr>
<td><strong>T = 6</strong></td>
<td>5.8 ± 0.3</td>
<td>5.3 ± 0.3**</td>
</tr>
<tr>
<td><strong>T = 12</strong></td>
<td>5.3 ± 0.2**</td>
<td>5.0 ± 0.2**</td>
</tr>
<tr>
<td><strong>Total Triglyceride (mmol/L)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>T = 0</strong></td>
<td>1.7 ± 0.1</td>
<td>1.8 ± 0.3</td>
</tr>
<tr>
<td><strong>T = 6</strong></td>
<td>1.7 ± 0.2</td>
<td>1.5 ± 0.2</td>
</tr>
<tr>
<td><strong>T = 12</strong></td>
<td>1.7 ± 0.1</td>
<td>1.2 ± 0.1**</td>
</tr>
</tbody>
</table>

*Note: Values are Mean ± S.E.M.
** p < 0.01
Figure 2. Mean cholesterol levels for untrained and trained subjects expressed over time.
Figure 3. Mean triglyceride values for untrained subjects and trained subjects expressed over time.
<table>
<thead>
<tr>
<th></th>
<th>Initial Test (T = 0)</th>
<th>Second Test (T = 6)</th>
<th>Final Test (T = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High Density Lipoproteins</td>
<td>1.34 ± 0.09</td>
<td>1.17 ± 0.09*</td>
<td>1.15 ± 0.12</td>
</tr>
<tr>
<td>Low Density Lipoproteins</td>
<td>4.01 ± 0.29</td>
<td>3.80 ± 0.30</td>
<td>3.34 ± 0.30*</td>
</tr>
<tr>
<td>Very Low Density Lipoproteins</td>
<td>0.80 ± 0.07</td>
<td>0.82 ± 0.08</td>
<td>0.80 ± 0.07</td>
</tr>
<tr>
<td>Ratio of H.D.L. to</td>
<td>0.23 ± 0.02</td>
<td>0.21 ± 0.02</td>
<td>0.23 ± 0.03</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of L.D.L. to</td>
<td>0.64 ± 0.02</td>
<td>0.65 ± 0.02</td>
<td>0.62 ± 0.03</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of L.D.L. to</td>
<td>3.26 ± 0.35</td>
<td>3.54 ± 0.40</td>
<td>3.51 ± 0.51</td>
</tr>
<tr>
<td>H.D.L.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: Values are Mean ± S.E.M.
  * p < 0.05
  ** p < 0.01
TABLE 9

Pre and Post Conditioning Changes in Serum Lipoprotein Concentrations in the Trained Subjects

<table>
<thead>
<tr>
<th></th>
<th>Initial Test (T =0)</th>
<th>Second Test (T =6)</th>
<th>Final Test (T = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High Density (mmol/L) Lipoproteins</strong></td>
<td>1.28 ±0.06</td>
<td>1.08 ±0.04*</td>
<td>1.16 ±0.05</td>
</tr>
<tr>
<td><strong>Low Density (mmol/L) Lipoproteins</strong></td>
<td>3.97 ± 0.25</td>
<td>3.43 ± 0.22*</td>
<td>3.29 ± 0.21*</td>
</tr>
<tr>
<td><strong>Very Low Density (mmol/L) Lipoproteins</strong></td>
<td>0.88 ± 0.13</td>
<td>0.70 ± 0.08*</td>
<td>0.58 ± 0.06**</td>
</tr>
<tr>
<td>Ratio of H.D.L. to Total Cholesterol</td>
<td>0.22 ± 0.02</td>
<td>0.21 ± 0.01</td>
<td>0.24 ± 0.01</td>
</tr>
<tr>
<td>Ratio of L.D.L. to Total Cholesterol</td>
<td>0.64 ± 0.02</td>
<td>0.64 ± 0.02</td>
<td>0.64 ± 0.02</td>
</tr>
<tr>
<td>Ratio of L.D.L. to H.D.L.</td>
<td>3.64 ± 0.24</td>
<td>3.75 ± 0.27</td>
<td>3.19 ± 0.23</td>
</tr>
</tbody>
</table>

**Note:** Values are Mean ± S.E.M.

* p < 0.05
** p < 0.01
Figure 4. Mean high density lipoprotein (HDL) and low density lipoprotein (LDL) values for untrained and trained subjects expressed over time.
Figures 5a. and 5b. Percentage of high density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) present in total cholesterol at T=0 weeks (fig. 5a.) and T=12 weeks (fig. 5b.), for untrained subjects.
Figures 6a. and 6b. Percentage of high density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) present in total cholesterol at T=0 weeks (fig. 6a.) and T=12 weeks (fig. 6b.), for trained subjects.
<table>
<thead>
<tr>
<th></th>
<th>Untrained Subjects (N = 20)</th>
<th>Trained Subjects (N = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Apolipoprotein A-1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(g/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T = 0$</td>
<td>$1.15 \pm 0.06$</td>
<td>$1.06 \pm 0.03$</td>
</tr>
<tr>
<td>$T = 6$</td>
<td>$1.14 \pm 0.05$</td>
<td>$1.04 \pm 0.03$</td>
</tr>
<tr>
<td>$T = 12$</td>
<td>$1.10 \pm 0.06$</td>
<td>$1.06 \pm 0.03$</td>
</tr>
<tr>
<td><strong>Apolipoprotein B</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(g/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T = 0$</td>
<td>$0.95 \pm 0.06$</td>
<td>$0.94 \pm 0.04$</td>
</tr>
<tr>
<td>$T = 6$</td>
<td>$0.89 \pm 0.05^{**}$</td>
<td>$0.81 \pm 0.04^{**}$</td>
</tr>
<tr>
<td>$T = 12$</td>
<td>$0.88 \pm 0.08$</td>
<td>$0.78 \pm 0.04^{**}$</td>
</tr>
<tr>
<td><strong>Ratio of Apolipoprotein A-1 to Apolipoprotein B</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T = 0$</td>
<td>$1.28 \pm 0.11$</td>
<td>$1.17 \pm 0.05$</td>
</tr>
<tr>
<td>$T = 6$</td>
<td>$1.36 \pm 0.11$</td>
<td>$1.33 \pm 0.07^{**}$</td>
</tr>
<tr>
<td>$T = 12$</td>
<td>$1.41 \pm 0.11^{**}$</td>
<td>$1.40 \pm 0.06^{**}$</td>
</tr>
</tbody>
</table>

**Note:** Values are Mean ± S.E.M.

**p < 0.01**
Figure 7. Mean apolipoprotein A-1 and B levels for untrained and trained subjects expressed over time.
Figure 8. Changes in the ratio of apolipoprotein A-1 to apolipoprotein B for untrained and trained subjects expressed over time.
### TABLE 11

Summary of Significant Pearsons Correlations Between Age, Weight, % Body Fat, and VO$_2$ max and All Serum Lipid Concentrations

<table>
<thead>
<tr>
<th>Subjects</th>
<th>All Subjects (N = 37)</th>
<th>Control Subjects (N = 17)</th>
<th>Trained (N = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H.D.L.</td>
<td>N.S.</td>
<td>- 0.48</td>
<td>N.S.</td>
</tr>
<tr>
<td>2. Weight with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.54**</td>
</tr>
<tr>
<td>L.D.L.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.51**</td>
</tr>
<tr>
<td>V.L.D.L.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.54**</td>
</tr>
<tr>
<td>Apo. B</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.55**</td>
</tr>
<tr>
<td>H.D.L./Chol.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>- 0.48**</td>
</tr>
<tr>
<td>L.D.L./H.D.L.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.49**</td>
</tr>
<tr>
<td>Apo A/Apo B</td>
<td>- 0.56**</td>
<td>N.S.</td>
<td>- 0.54**</td>
</tr>
<tr>
<td>3. % Body Fat with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H.D.L.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>- 0.59**</td>
</tr>
<tr>
<td>H.D.L./Chol.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>- 0.48**</td>
</tr>
<tr>
<td>L.D.L./Chol.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.41*</td>
</tr>
<tr>
<td>L.D.L./H.D.L.</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.49**</td>
</tr>
<tr>
<td>4. VO$_2$ max with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H.D.L.</td>
<td>N.S.</td>
<td>0.34*</td>
<td>- 0.33*</td>
</tr>
<tr>
<td>V.L.D.L.</td>
<td>- 0.33*</td>
<td>N.S.</td>
<td>N.S.</td>
</tr>
<tr>
<td>H.D.L./Chol</td>
<td>N.S.</td>
<td>- 0.34*</td>
<td>0.35*</td>
</tr>
<tr>
<td>Apo A/Apo B</td>
<td>N.S.</td>
<td>N.S.</td>
<td>0.33*</td>
</tr>
</tbody>
</table>

Note: Values are Mean ± SD
* p < 0.05
** p < 0.01
N.S. not significant
CHAPTER VI

DISCUSSION AND CONCLUSIONS

The purpose of this study was to determine the responses of serum lipids to aerobic physical conditioning. These responses were examined to test the following hypothesis:

a) That subjects partaking in mild aerobic physical activity for 12 weeks will show significant changes in serum lipid concentrations compared to non-active, untrained control subjects.

b) That subjects partaking in mild aerobic physical activity for weeks will show significant changes in serum lipid concentrations compared to initial concentrations.

c) That subjects partaking in mild aerobic physical activity for 12 weeks will show a significant increase in maximal aerobic capacity.

Weight, Percentage Body Fat, and Aerobic Capacity

The responses of both body weight and percentage body fat experienced by the untrained and trained groups were as expected. Both groups showed a non-significant reduction in body weight. The untrained group mean body weight was reduced by 0.9 kg to 85.8 kg, while the trained group mean reduction was 1.9 kg to 78.7 kg. This reduction in the trained group was
accompanied by a significant decrease in percentage body fat of 2.3 per cent to 19.5 per cent. In contrast, the untrained group showed a 0.9 per cent increase in percentage body fat to 22.4 per cent.

Other studies have documented anthropometric changes of this nature. For example, Bassett-Frey et al. (1982) showed that females had non significant reductions in body weight of 0.2 kg, whilst percentage body fat decreased significantly by nearly 2.0 per cent following a 10 week exercise programme. Additional studies in 1984 displayed similar patterns. Gaesser and Rich (1984) showed that two groups following high and low intensity aerobic exercise both maintained body weight at nearly identical levels to initial values after 18 weeks of conditioning. However, both groups showed significant reductions in percentage body fat of 2.1 per cent and 1.8 per cent respectively.

Similar results were obtained by Danner (1984), and by Despres et al. (1984), although body weight did decline significantly in the earlier study. Danner et al. (1984) showed that over a 21 week training program, exercising oarsman lost only 0.9 kg in weight, but reduced percentage body fat markedly by 8.5 per cent to 22.2 per cent. Untrained subjects in this study showed neither a significant change in either body weight or percentage body fat.

Maximal aerobic capacity again responded as expected in both the untrained and trained groups. Aerobic capacity in the untrained group increased slightly from 31.4 ml.kg.min⁻¹ to 32.4 ml.kg.min⁻¹ over the study period. At T = 6 weeks, aerobic capacity showed a
significant increase to 34.8 ml.kg.min\(^{-1}\), due primarily to one untrained subject obtaining an aerobic capacity in excess of 55.0 ml.kg.min\(^{-1}\). If this score was removed, no significant changes occurred.

The trained groups mean aerobic capacity showed significant increases at both \(T = 6\) and 12 weeks. From an initial mean of 33.2 ml.kg.min\(^{-1}\), the aerobic capacity of the trained group increased to 35.9 ml.kg.min\(^{-1}\) at \(T = 6\) weeks, and further increased to 39.1 ml.kg.min\(^{-1}\) at \(T = 12\) weeks.

These results confirm the predicted responses of the trained and untrained males with respect to aerobic capacity. When the changes observed in the trained individuals are compared to the untrained controls, it appears likely that the aerobic exercise regime followed during this study contributed to the change. Many studies including those by Nagao, Arie, & Sawada (1988), Shephard (1986), and Thompson et al. (1984) have shown that maximal aerobic capacity correlates highly to the level of activity or inactivity in an individual. The effect of aerobic physical activity is thus directly measured using maximal aerobic capacity, with a higher aerobic capacity indicating greater cardiovascular adaptation.

It is worth noting here that many subjects felt that their performance on the treadmill test was inhibited by the mouthpiece and nose clip. Although it is reasonable to suggest that this may have effected each individual in a similar fashion, the overall aerobic capacities could have been understated.
Morrison, Collins, Stovall, and Friefeld (1989) showed that exercise duration at a given workload decreased by approximately 25 per cent, along with an 18 per cent decrease in aerobic capacity when a mouthpiece and noseclip is used, in subjects aged 54-74 years.

Cardiovascular Responses to Conditioning

The responses observed in mean heart rates for the untrained subjects and trained group subjects followed similar patterns to other previous studies. The changes in resting heart rate observed in both groups reflect the results of earlier studies by Wood et al. (1983) and Siscovick et al. (1988), during which a decrease of 5.0 beats/minute in resting heart rate was observed between active and non-active males aged 35 to 59 years. During this current study, both the untrained subjects and trained subjects had similar base line resting heart rates. However, the untrained groups mean resting heart rate increased by 0.7 beats/minute to 72.9 beats/minute after the 12 week study, while the trained group showed a significant decrease in resting heart rate of 5.2 beats/minute to 67.6 beats/minute.

An increase in resting heart rate has been associated with a detraining effect, whilst a decrease in resting heart rate has indicated cardiovascular adaptation (Penny and Wells, 1975). With the associated significant increase in aerobic capacity (33.2 ml.kg.min\(^{-1}\) to 31.9 ml.kg.min\(^{-1}\)) recorded in the trained group, it appears some cardiovascular adaptation to mild aerobic activity occurred during the 12 week study. The constant nature of the untrained group aerobic capacity at around 32.0 ml.kg.min\(^{-1}\)
throughout the study suggests that the change observed in the trained group can be attributed to the aerobic conditioning program.

Maximal exercising heart rates obtained by both groups during treadmill testing contrasted the findings of Penny and Wells (1975) and Siscovick et al. (1988) in that the untrained group showed a non significant decrease in maximal heart rate obtained at $T = 12$ weeks, compared to the initial testing. Furthermore, the trained group showed a non significant increase in maximal heart rate from 169.8 bpm during initial testing, to 172.5 bpm at $T = 12$ weeks. Both Penny and Wells (1975) and Siscovick (1988) indicated that exercising males showed a non significant decrease in maximal exercising heart rate from initial values, during subsequent testing, and that these values were less than the matched controls at nearly all testing sessions.

These changes in maximal heart rate responses may be explained by the effort put in by the subjects during each testing session. The trained group consistently produced a higher respiratory quotient during later testing, which suggested that they may have been prepared to work harder during the second and third treadmill tests. It was apparent that the trained subjects were far more motivated than their untrained counterparts to perform during testing, especially nearing the conclusion of the study as they became interested in determining the effect of the training program on their own health.
The haemodynamic responses to exercise were varied. Both the trained and untrained group members showed a decrease in mean systolic and diastolic blood pressure over the duration of the study. The untrained group showed a significant reduction in both readings (132.2 mm Hg to 120.2 mm Hg and 85.1 mm Hg to 79.2 mm Hg respectively). Mean systolic and diastolic blood pressure in the trained group also decreased (127.1 mm Hg to 121.6 mm Hg and 82.0 mm Hg to 79.7 mm Hg respectively). These responses were not significant.

These findings were not consistent with several previous studies. Nye et al. (1968) reported that male joggers systolic blood pressure remained constant at 132.9 mm Hg after 20 weeks of training, while a matched control group showed a 4 mm Hg increase to 131.4 mm Hg. Similarly, Penny and Wells (1975) showed that male footballers showed an increase in systolic blood pressure of 10 mm Hg, accompanied by a 6 mm Hg increase observed in control group members.

Both the study by Hunter et al. (1968) and a further study by Dickhuth et al. (1989) showed that males whom exercised showed a reduced diastolic blood pressure compared to non active males, and that diastolic blood pressure decreased over time, following exercise.

In this study, although the exercising males in the trained group did present a non significant decrease in diastolic blood pressure following the exercise regime, this was the only resting blood pressure response which followed normal responses to an exercise
regime. It was not expected that the untrained group members would show significant decreases in both diastolic and systolic blood pressure. This may have been due to reduction in state anxiety, as reported during a study by Raglan and Morgan (1987), where decreases in mean blood pressure occurred following regimes aimed at anxiety reduction. Anxiety levels would probably have been greatest during initial testing as all subjects were not totally familiar with the procedure, resulting in abnormally high blood pressure readings. It has also been reported by Kaufman et al. (1987) that over estimation of blood pressures can result from anxiety and apprehension in normotensive young males.

Although a familiarisation session was carried out prior to initial testing, most subjects reported feeling more comfortable with the procedure during the second and final testing sessions. It appears that to reduce the effect of stress and anxiety on base line haemodynamic responses, it may be necessary to run the test protocol as practice for each subject, prior to the commencement of any study.

Blood pressure changes during exercise produced only one significant finding over the three testing sessions. The maximal systolic blood pressure in the untrained group members increased significantly from 214.0 mm Hg to 231.9 mm Hg over the 12 week study. Conversely, the trained group showed a non significant decline in maximal exercising systolic blood pressure from 232.4 mm Hg to 222.4 mm Hg over the 12 week study. Both the untrained and trained groups showed non significant fluctuations
of 3-4 mm Hg in minimal exercising diastolic blood pressure from initial to final testing.

The significant increase in maximal exercising systolic blood pressure is not readily explainable. As reported by Seals and Hagberg (1984), there has been little research into the field of exercise related haemodynamic responses. We know that systolic blood pressure increases during exercise, while diastolic blood pressure declines, a pattern followed by both groups in this study. However, why the untrained group showed a marked increase in maximal exercising systolic blood pressure when the trained group did not is unclear.

Post exercise blood pressure recordings were as expected on all three testing occasions for both groups. At 30 minute post exercise recovery, blood pressures were lower than initial resting blood pressures. This again supports the view that many subjects may have been apprehensive prior to testing, resulting in elevated resting blood pressure. Similarly, both the untrained and trained groups showed constant reductions in recovery blood pressure over the 12 week study in all but one case. The trained subjects 30 minute post exercise diastolic pressure increased sharply at week 6, but declined again to near initial levels at $T = 12$ weeks.

Many of these responses, especially at rest and during exercise, are varied. The untrained group did contain five individuals who were prescribed hypertensive drugs including Tenormin (Atenolol), Betaloc (Metoprolol Tartrate), and Minipress (Prazosin),
while the trained group had only two individuals using Tenormin, which may have contributed to the great fluctuations noted in exercising blood pressures. Overall, the support for exercise as a non pharmaceutical treatment of hypertension is divided, but authors such as Wilcox et al. (1982) and Kaufman et al. (1987) have established basic responses of blood pressure to exercise. It may be that the reduction in maximal exercising systolic blood pressure experienced by the trained group is related to some haemodynamic adaptation to aerobic exercise. Thus, it is hard to explain why the control group experienced such a sharp decline in resting blood pressure levels, which was not seen in the experimental group.

The Effect of Mild Aerobic Physical Activity on Serum Cholesterol and Triglyceride

The mean changes in serum cholesterol and triglyceride concentrations recorded during this study show several clear trends. Both the untrained group and the trained group presented almost identical concentrations of both of these lipid subfractions during initial testing. Serum cholesterol concentrations for both groups were 6.1 mmol/L. Serum triglyceride levels were 1.7 mmol/L and 1.8 mmol/L for the untrained group and trained group respectively.

As expected, the serum cholesterol level for the trained group declined significantly to 5.0 mmol/L at the conclusion of the study. A similar unexpected significant reduction to 5.3 mmol/L occurred in the untrained group. In contrast, the untrained
groups serum triglyceride remained constant at 1.7 mmol/L throughout the study, while the trained group showed a significant reduction of 0.8 mmol/L to 1.2 mmol/L.

These findings support the belief held by many researchers, that aerobic physical activity may reduce serum cholesterol and triglyceride concentrations. However, as the body of knowledge concerning other lipid subfractions has become more prolific in the past 20 years, it appears that the importance placed on serum cholesterol and triglyceride concentrations may be too great.

It is not the purpose of this study to decide on the efficacy of various lipid components in heart disease risk determination. However, failure to address the effect physical activity has on these serum lipid subfractions would make this a somewhat regressive rather than progressive contribution to our current knowledge in this field. It is evident from papers by Nikkila et al, (1980), Kannal (1988); Naito (1977, 1988(a), 1988(b)); Nagao, Arie, & Sawada, 1988; Nagao, Imai, Arie, Sawada, & Karatsu, 1988), and others, that the lipid profile as related to coronary heart disease paints a complex picture. This area will be addressed when discussing serum lipoproteins and apolipoprotein concentrations (see page 139).

The reduction in serum cholesterol observed in the trained group was as expected. Golding (1961) showed that over a 24 week period, serum cholesterol was significantly reduced in exercising males, but not in sedentary controls. Streja and Mymin (1979) also showed a significant reduction in serum cholesterol over a 13
week period in middle aged men, accompanied by a non
significant reduction in serum triglyceride. The significant
reduction in serum cholesterol observed in the control group
suggests that physical activity is only one of many factors relating
to serum cholesterol levels, as suggested by Kannal et al. (1979).
Other factors include diet, sex, and age. For example, it is
considered by Kannal et al. (1979) that cholesterol is not a reliable
predictor of coronary heart disease risk in males beyond 65 years
of age. Furthermore, the baseline testing was completed
immediately after Christmas in this current study, and both
groups may have had abnormally elevated serum cholesterol
levels due to their dietary intake, following the festive season.

Cooper et al. (1988), suggested that the reliability of all serum
lipid concentrations are subject to biological and behavioural
variations. Both serum cholesterol and triglyceride concentrations
have previously shown great intra-person variations in readings
over any given day. Serum cholesterol was shown to fluctuate 2.5
per cent (range 0.7 - 4.3 per cent) when tested at four different
times of the day, whilst serum triglyceride fluctuated by 35 per
cent (range 6.3 - 65.0 per cent). Doubts relating to both of these
variables and analytical sources of variation, including collection,
handling, and shipping of specimens have also been questioned by
Cooper et al. (1988) and Naito (1988). However, during collection
and testing, all care was taken to ensure these variations were
minimised.

The great variations observed by Cooper et al. (1988) in serum
triglyceride concentrations may explain the inconsistency of the
data in regards to this variable. Several papers report no significant changes in serum triglyceride levels following exercise programs of between 13 to 52 weeks (Streja & Mymin, 1979; Bassett-Frey, 1982; Gaesser & Rich, 1983; Wood et al, 1983). Conversely, papers by Ballantyne et al. (1982) and Nye et al. (1984) have shown significant reductions in serum triglyceride with six months of physical activity.

When testing at monthly intervals, Cooper et al. (1988) have shown that serum triglyceride concentration variance was reduced to 24.2 per cent. This suggests that the 33 per cent reduction in serum triglyceride in the trained group during this study period is at least somewhat due to factors other than cyclic variations in serum values. The stability of the untrained groups mean serum triglyceride concentration also suggests that the aerobic physical activity carried out by the trained group had some beneficial effect. It would also be reasonable to conclude that within each group the subjects would show similar intra-individual fluctuations in serum triglyceride concentrations. This again suggested that aerobic physical activity contributed to serum triglyceride reduction in the trained group while this response was not apparent in the untrained group.

The Effect of Mild Aerobic Physical Activity on Serum Lipoproteins

The overall significance of serum cholesterol in determination of coronary heart disease risk has in the past 20 years been highly associated with the presence of specific lipoproteins. Many
studies have suggested that elevated high density lipoprotein in tandem with reduced low and very low density lipoproteins may predict coronary heart disease risk with more certainty than serum cholesterol alone. However, the research into the effect of physical activity on these serum lipoproteins is divided. Previously mentioned studies by Streja & Mymim (1979) and Ballantyne et al. (1982) support exercise as a method of lipoprotein control, while others including Despres et al. (1985), Nye et al. (1984), and McNaughton & Davis ((1987), found no significant alterations when subjects exercised.

The results of this study do suggest that low intensity aerobic physical activity may effect low and very low density lipoproteins more so than high density lipoproteins. Both the untrained and trained groups showed a surprising, non significant reduction in high density lipoproteins of 0.19 mmol/L and 0.12 mmol/L respectively. Gaesser and Rich (1983) in a similar study also found similar results. It was concluded by Gaesser and Rich (1983) that the lack of response may have been related to the multiple factor nature of lipoprotein determination, including reduced base lipoprotein levels, weight, age, diet, and smoking.

The data displayed in this study revealed that the trained group had a significant reduction in both low and very low density lipoproteins. Low density lipoproteins decreased from 3.97 mmol/L to 3.29 mmol/L, with an associated decrease in very low density lipoproteins from 0.88 mmol/L to 0.58 mmol/L. The reduction in low density lipoproteins was mirrored in the untrained group, with a significant 0.67 mmol/L reduction to 3.34
mmol/L recorded. Nye et al. (1984) also found a significant reduction in low density lipoproteins in males aged 30-45 years following a 1 year exercise program. Two other studies by Streja and Mymin (1979) and Gaesser and Rich (1983) found no significant changes in middle aged males over a 13 to 20 week exercise program. Again, the inconsistency of these results poses questions about lipoproteins. Whether these questions relate to lipoproteins as a predictor of coronary heart disease or to physical activity as a factor in lipoprotein concentrations is as yet unanswered.

Naito (1988b) has suggested that lipoproteins are a more sensitive marker of coronary heart disease risk compared to serum cholesterol. The study by Gaesser and Rich (1984) also suggests that lipoproteins may be affected by aerobic physical activity more so than serum cholesterol, but this may also be dependent on the nature and the intensity of exercise.

The results of this study however seem to suggest that there may be doubt with regards to the response to physical activity of both serum cholesterol and lipoproteins. Even though the control group was not undertaking any physical activity, both groups showed significant reductions in total cholesterol. Yet, both groups showed similar trends in serum lipoprotein concentrations. The trained group showed an unexpected decrease in high density lipoproteins, as did the untrained group, and both groups showed significant decreases in low density lipoproteins. The decrease in low density lipoproteins in the untrained group was again totally unexpected.
Did the duration of this study, the nature of the physical activity, the effect of extraneous variables, or the determination of serum lipid concentrations contribute to the inconsistent serum cholesterol and lipoprotein results? These questions will be addressed in the following section of the discussion.

The Effect of Mild Aerobic Physical Activity
on Serum Apolipoproteins

Despite the existence of extensive research material pertaining to serum lipid concentrations, little of this material relates to serum apolipoproteins as a marker of coronary heart disease risk. Subsequently, data examining the effect of mild aerobic physical activity on serum apolipoprotein concentrations is minimal.

Of the research papers included in the literature review and discussion of this study pertaining to physical activity and apolipoproteins, few studied the high risk male. Only three papers, by Ballantyne et al. (1982), Nagao (1988) and Wood et al. (1984) from in excess of 130 reviewed during this current research study, was specifically directed at sedentary males in the high risk age group. Almost all of the remainder discussed the responses in the elite athlete in comparison to untrained controls. The papers here are by no means the sum total in the area of serum lipids, but it is remarkable that less than 2 per cent of these studies addressed serum apolipoproteins and this high risk population.
During this current study, both apolipoprotein A-1 and B concentrations were determined. Apolipoprotein A-1 decreased from 1.15 g/L to 1.10 g/L in the untrained group, whilst the trained group remained at 1.06 g/L throughout the study. Conversely, apolipoprotein B showed a non-significant decrease in the untrained group from 0.95 g/L to 0.88 g/L. A significant reduction in apolipoprotein B from 0.94 g/L to 0.78 g/L in the trained group was shown.

Nagao (1988) reported differing results in a study which only investigated serum lipids, lipoproteins, and apolipoprotein A-1. A group of active males showed a significantly higher apolipoprotein A-1 level than matched controls. The trained subjects had been exercising for a minimum of 2 years. Both groups maintained almost identical serum cholesterol concentrations of 182.27 mg/dl and 181.94 mg/dl respectively. This result was accompanied by a significant reduction in high density lipoproteins, but no change in low density lipoproteins.

Similar data was presented by Nikkila et al. (1980), when studying the effect of inactivity on serum lipid concentrations. Patients who were confined to hospital beds showed highly significant reductions in both apolipoprotein A-1 and high density lipoproteins after 3-6 weeks. Remarkably, serum cholesterol concentrations actually decreased significantly when compared to the active controls. It was concluded that daily activity was accompanied by an increase of both high density lipoproteins and apolipoprotein A-1, but which may not be accompanied by a
decrease in serum cholesterol. These studies emphasise the need for research into the high risk male population, as neither study investigated the relationship between exercise and apolipoproteins in this group.

Data by Wood et al. provides little evidence to support the findings of Nikkila (1980). In a year long study, no significant changes in either apolipoprotein A-1 or B occurred, although apolipoprotein B concentrations declined more so than apolipoprotein A-1 increases in exercising males aged 33-60 years. These findings are reflected in this current study, where the trained group maintain apolipoprotein A-1 levels, while apolipoprotein B levels decrease significantly after 12 weeks of training.

It was unexpected that the untrained group should have significantly reduced apolipoprotein B at T = 6 weeks, but this was not the case at T = 12 weeks. The reduction in apolipoprotein A-1 in the untrained group does follow the findings of Nikkila et al. (1980), and Nagao et al. (1988).

The study completed by Ballantyne et al. (1984) also showed that non significant changes in serum cholesterol can be accompanied by significant changes in other lipid subfractions. With 42 male post infarct males as subjects, a six month exercise regime based on the 5BX plan was completed. The non exercising controls showed no significant changes, while the trained group produced significant increases in apolipoprotein A-1 and high density
lipoproteins. A similar significant reduction in low density lipoproteins and serum triglyceride was also recorded.

Overall, the changes in apolipoprotein were greater than any other serum lipid variable, and were showing greater changes from testing at 4 months to testing at 6 months than either the lipoproteins or serum triglyceride (Ballantyne et al, 1984).

The ratio's of all the serum lipoproteins and apolipoproteins to serum cholesterol confirm these findings. No significant changes were found in any of three ratio's with respect to high density and low density lipoproteins to serum cholesterol. Conversely, both the untrained and trained group showed significant increases in the ratio of apolipoprotein A-1 to apolipoprotein B. Although both groups had a similar ratio at week 12 of 1.40, the trained group showed a 20 per cent increase, twice that of the untrained group.

The discrepancies evident in the serum cholesterol and lipoprotein results, accompanied by significant changes and clear trends in serum apolipoproteins pose several questions.

Firstly, were the untrained controls in fact exercising, which would bias the results? This appears unlikely. Given the opportunities forwarded to the untrained group members to disclose this on many occasions during the study, it is assumed that little or no physical activity was performed by these individuals. This is supported by the maximal aerobic capacity value which remained relatively constant for the untrained group.
Secondly, would the coefficients of variation present in testing for these variables, as reported by Cooper et al. (1988) and Naito (1988) effect the between session results. Again, this appears unlikely. All precautions were taken during specimen collection, handling and testing to reduce any variations. Any samples frozen during initial testing were frozen for subsequent testing sessions, and testing methodologies were identical. The only sample variations which may have effected results would have been those present in intra person variations, within subjects. This was again reduced by testing each subject at the same time of the day at each of the three testing sessions.

Several subjects appeared to have made minor adjustments to their dietary intake, as indicated by the 24 hour dietary recalls. One untrained control subject changed from a normal daily diet to a Pritiken style diet, and also took up training. His data, as reported earlier (page 99), had to be excluded from the study. As pointed out by Gill et al. (1989), Sady et al. (1985), and Superko (1988), an appropriate diet may be the foundation of a well designed lipid therapy programme.

Although this point is not in dispute, the current study was not primarily designed to investigate diet as related to coronary heart disease management. Conversely, the study design attempted to reduce the effect of this variable by trying to get the subjects to maintain a static diet.

Thirdly, was the duration and intensity of exercise not sufficient to induce significant results? It is conceded here that a longer
period of physical activity would be preferred. It has been shown that aerobic exercise programs carried out for a shorter duration than the 12 weeks employed in this study, will produce significant changes in serum lipid concentrations as shown by Shephard, 1988. The findings of Ballantyne et al. (1984) also showed that a six month exercise regime effected serum apolipoprotein A-1 to a greater extent nearing the concluding stages. Hence, a longer period of physical activity may have given clearer results. However, the significant changes exhibited in the serum apolipoprotein concentrations suggest that some effect of aerobic physical activity occurred. Furthermore the intensity of exercise, carried out under A.C.S.M. (1988) guidelines has also been shown to be most likely to produce changes in serum lipid concentrations. The fact that this was not clearly shown with other serum lipid concentrations may then suggest a lack of an effect rather than a non efficable treatment regime.

Finally, are serum cholesterol and serum lipoprotein concentrations less effected by mild aerobic physical activity than other serum lipid concentrations? From the data presented from the current study, and as reported from previous studies, this appears to be possible. This was clearly shown by both Nikkila et al. (1980) and Nagao et al. (1988) with both short term and long term effects being shown.

The responses exhibited by individuals during this study suggested that the changes in lipid concentrations following a 12 week aerobic exercise programme are wide and varied. Individual difference has been the "catch call" of exercise
prescription experts over recent years, and there is strong evidence from this study to suggest the same theory holds true for serum lipid responses to physical activity.

During this study, apolipoprotein A-1 remained stable in the trained group while decreasing in the untrained. Apolipoprotein B decreased significantly in the trained group, but no significant decrease was noted in the untrained group. While the ratio of apolipoprotein increased significantly in both groups, the change observed in the trained group was twice that of the untrained control group. Similarly, serum triglycerides were reduced significantly in the trained group, while remaining unchanged in the untrained group. This pattern was not present with either serum cholesterol concentrations or serum lipoprotein concentrations in response to aerobic physical activity over 12 weeks.

As stated in the literature review (page 66), Naito (1988b) has reported that apolipoproteins are a more sensitive measure of coronary heart disease risk than either cholesterol or lipoproteins. It appears from the data collected during this study that not only are apolipoproteins a more sensitive marker of coronary heart disease, but mild aerobic physical activity may have a greater effect on apolipoprotein concentrations compared to other serum lipids, in healthy adult males aged 33 to 60 years old.
Conclusions

The purpose of this study was to determine the overall effect of mild aerobic physical activity on serum lipid concentrations. On the basis of the significance of the ANOVA statistical analysis and correlations relating to mild aerobic physical activity, the following conclusions were made:

It was concluded that mild aerobic physical activity resulted in an increased aerobic fitness level. Cardiovascular adaptation was shown to occur in the trained group only. This is supported by the significant reduction in resting heart rate and the significant increase in maximal aerobic capacity observed in the trained group.

From anthropometric changes observed during the present study, it was concluded that a 12 week mild aerobic physical activity programme will not necessarily reduce body weight, but will increase lean body mass. This was supported by the trained individuals maintaining weight over the study period, but showing a significant reduction in percentage body fat.

The significance of results derived for the serum lipid concentrations suggest that mild aerobic physical activity may affect serum triglycerides and apolipoproteins (especially apolipoprotein B) more effectively and consistently than either serum cholesterol or lipoproteins. Mild aerobic physical activity produced a significant decrease in apolipoprotein B which was not evident in the untrained control group after 12 weeks. No
significant changes were observed in serum apolipoprotein A-1 concentrations, although this value remained constant in the trained subjects while decreasing by 5 per cent in the untrained group. The ratio of apolipoprotein A-1 to apolipoprotein B increased significantly in both the untrained and trained groups. However, the trained group showed an increase twice that of the untrained group.

Furthermore, serum triglyceride concentrations decreased significantly with training, with neither the responses of serum cholesterol or serum lipoproteins showing a treatment effect. It was concluded that a change in apolipoprotein ratio would be the most likely outcome in individuals following a 12 week programme of mild aerobic physical activity.

Recommendations for Future Study

From the results of this study, evidence was found to suggest that mild aerobic physical activity may effect serum triglyceride and apolipoproteins A-1 and B to a greater extent than either serum cholesterol or lipoproteins. The following recommendations are suggested for further research in the area:

1. A comparative investigation of a longer term physical activity programme on serum lipids, especially apolipoprotein concentrations.
2. Assessment of other exercise regimes including weight training and serum lipid reduction in high risk groups.

3. Further investigation on the effectiveness of physical activity on cardiac risk factors in high risk groups, including post infarct patients, compared with other intervention techniques including hospitalisation and pharmaceutical treatment.

4. Comparison of other methods of life style modification including dietary intervention, and stress management in relation to modification of the apolipoprotein ratio. These areas are of particular importance to high risk, middle aged males.

5. An increase in case study data looking specifically at the effect of mild aerobic physical activity on individual serum apolipoprotein concentrations.


Bassett-Frey, M.A., Doerr, B.M., Laubach, L.L., Mann, B.L., & Glueck, C.J. (1982). Exercise does not change high density lipoprotein cholesterol in women after ten weeks of training. Metabolism, 31(11), 1142-1146.


APPENDIX I

INFORMED CONSENT FORMS FOR
MAXIMAL EXERCISE TEST
AND EXPERIMENTAL
CONSENT
Informed Consent for Maximal Exercise Test

As part of the study investigating the effect of physical activity on the lipid subfractions, you will be required to complete three (3) maximal capacity (VO₂ max) tests on a motorised treadmill. The exercising testing will begin at a level which you can easily accomplish and will be advanced in stages, depending on your fitness level. We may stop the test at any time because of signs of fatigue or you may stop when you wish because of personal feelings of fatigue or discomfort.

It is important that you are aware of certain changes which may occur during the test. They may include abnormal blood pressure, alterations to heart beat patterns, and fainting. Every effort will be made to minimise these through the preliminary examination and by constant observation during testing. Emergency equipment and trained personnel are available to deal with any unusual circumstances which may arise.

The results obtained from this exercise test will assist in determining your level of aerobic fitness, prior to, during, and after the aerobic conditioning programme. Any questions about the procedure used in the exercise test are encouraged. If you have doubts or questions, please ask for further explanations.

I have read this form and I understand the test procedures that I will perform. I consent to participate in the test.

Date ............... Signature of Subject .................

Signature of Witness .................
Experimental Consent Form

My signature on this sheet, by which I volunteer to participate in the experiment;

The Effect of Physical Activity on Serum Apolipoproteins and Other Lipid Subfractions in Sedentary, Low Fit, Healthy, Male Adults.

Conducted by;

Dr. Graham Ward & J.P. Shearman
Faculty of Human Movement
Department of Health Sciences
University of Wollongong
P.O. Box 1144
Wollongong N.S.W. 2500
AUSTRALIA.

Indicates that I understand that all the subjects in the project are volunteers, that I can withdraw at any time from the experiment, that I have been informed as to the nature of the experiment, that the data I supply will be anonymous and my identity will not be revealed without my permission, and that my performance in this experiment may be used for additional approved projects. Finally, I shall be given an opportunity to ask questions prior to the start of the experiment and after my participation is complete.

Date .......... Signature of Subject ..........

Signature of Witness ............
APPENDIX II

GENERAL HEALTH QUESTIONNAIRE
General Information and Health Habits Questionnaire.
(all information will be kept in strict confidence)

Date: ........
Name: ......................
Home Address: ......................
Company Name: ......................
Department: ...................... Position: ......................
Number of years employed in current position: ........
Birth date: ........ (year) ........ (month) ........ (day)
Name of Family Physician: ...................... Phone: ........

General Health

1. Which category in the scale below best describes how healthy you feel in relation to other people of your own age? (circle one only)

very much worse
slightly worse
average
slightly better
very much better

2. Are you currently involved in any organised recreational or sporting physical activity? (circle one only)

yes
no (go to question 5)

3. If you answered yes to question 2., how many days per week do you carry out physical activity? (circle one only)

1 2 3 4 5 6 7

4. Approximately how many minutes would each of these sessions of physical activity last?

............ minutes
5. Taking into account the amount of activity involved with your job, do you feel that the amount of physical activity you are currently doing is adequate (circle one only)

   yes           no

**Exercise Habits**

6. List the 3 main recreational activities you have been involved in during the past year

   ..................................

   ..................................

   ..................................

7. Do you have any problems with partaking in regular physical activity? If so, please list them in the space provided.

   ..............................................................

   ..............................................................

**Benefits of Exercise**

8. List the 3 main reasons for you wanting to join this exercise programme.

   ..............................................................

   ..............................................................

   ..............................................................

**Health Status**

9. How often do you feel stress/tension? (circle one only)

   never    almost never    occasional    frequently    nearly always

10. Do you currently smoke: cigarettes: yes  no
cigars: yes  no
pipe: yes  no
other: yes  no
11. If you answered "yes" to question 10., how many of the above would you smoke per day?

- cigarettes: ....... per day
- cigars: ....... per day
- pipe: ....... per day
- other: ....... per day

12. Do you drink alcohol:
- spirits: yes no
- beer: yes no
- wine: yes no
- other: yes no

13. If you answered "yes" to question 12., what approximate quantity in millilitres of each beverage would you consume each week?

- spirits: ....... mls.
- beer: ....... mls.
- wine: ....... mls.
- other: ....... mls.

14. Do you have or have you ever had, any of the following? (please tick the appropriate choices)

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
<th>Yes</th>
<th>No</th>
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</thead>
<tbody>
<tr>
<td>Heart attack/disease</td>
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<td>Allergies</td>
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<tr>
<td>Rheumatic Fever</td>
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<td>Asthma/hay fever</td>
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<tr>
<td>High blood pressure</td>
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<td>Chronic cough</td>
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<tr>
<td>Varicose veins</td>
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<td>Shortness of breath</td>
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<td>Chest pains</td>
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<td>Ulcer/cancer</td>
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<td>Stroke</td>
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<td>Dizziness/fainting</td>
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<td>Frequent headaches</td>
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<td>Backaches</td>
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<td>Arthritis</td>
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<td>Rupture/hernia</td>
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<td>Kidney/bladder</td>
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<td>Recent Illness</td>
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<td>Nervous disorders</td>
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<td>Fever</td>
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<td>Diabetes mellitus</td>
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<td>surgery/hospitalisation</td>
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</table>
13. If the answer to any of the above was "yes", explain, giving date, duration of treatment, and current status:

........................................................................................................................................

........................................................................................................................................

........................................................................................................................................

14. List any medications which you are currently taking:

........................................................................................................................................

........................................................................................................................................

........................................................................................................................................

THANK YOU FOR YOUR PARTICIPATION
APPENDIX III

EXPERIMENTAL RESULT

SHEETS
UNIVERSITY OF WOLLONGONG
RESEARCH TEST RESULTS

Testing session three (12 weeks) Subject reference number ______

Date _______ Time _________

Temp ______ Humidity ______

a) MAXIMAL AEROBIC CAPACITY TEST. Bar. pres. ______

STPD. ________

<table>
<thead>
<tr>
<th>workload</th>
<th>time</th>
<th>VE</th>
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b) ANTHROPOMETRICAL TEST VARIABLES

- height ________ (cm)
- weight ______ (kg)
- skin fold measurements
  - biceps ______  _____  _____  ave. ______ (mm)
  - triceps ______  _____  _____  ave. ______ (mm)
  - subscapula ______  _____  _____  ave. ______ (mm)
  - suprailiac ______  _____  _____  ave. ______ (mm)
  - abdominal ______  _____  _____  ave. ______ (mm)
- TOTAL PERCENTAGE BODY FAT ______ (%)  

c) CARDIOVASCULAR TEST VARIABLES

1. ELECTROCARDIOGRAPH
- number of T wave changes ______
- ST segment depression ______ (mm)
- other E.C.G. abnormalities present and comments

2. HEART RATE
- resting heart rate ______ (bpm)
- maximal exercising heart rate ______ (bpm)

3. HAEMODYNAMICS
- resting blood pressure ______ (mmhg)
- maximal exercising systolic ______ (mmhg) at ______ (min)
- maximal exercising diastolic ______ (mmhg) at ______ (min)
- minimal exercising diastolic ______ (mmhg) at ______ (min)
- post exercise blood pressure recovery
  - at 0 minutes recovery ______ (mmhg)  at 15 minutes recovery ______ (mmhg)
  - at 5 minutes recovery ______ (mmhg)  at 30 minutes recovery ______ (mmhg)
  - at 10 minutes recovery ______ (mmhg)
d) BLOOD LIPID TEST VARIABLES

- total serum cholesterol (mg/100mls)
- total serum triglyceride (mg/100mls)
- high density lipoprotein (mg/100mls)
- low density lipoprotein (mg/100mls)
- V-low density lipoprotein (mg/100mls)
- apolipoprotein A-1 (mg/100mls)
- apolipoprotein B (mg/100mls)
- ratio HDL/total chol.
- ratio LDL/total chol.
- ratio HDL/LDL
- ratio apo A-1/ apo B
APPENDIX IV

TREADMILL PROTOCOL
MODIFIED BRUCE TREADMILL PROTOCOL

constant treadmill speed of 5.5 kph (3.3 mph)

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<tr>
<th>Stage</th>
<th>Minutes</th>
<th>Grade (%)</th>
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Here are the details for the training regime to be followed for a period of twelve weeks.

All training will be carried out at a predetermined intensity from your initial test data. Train with your heart rate between two figures calculated using your maximum heart rate obtained during initial testing. You will be informed of these two values. You can take your own pulse rate from your arm or neck, with your first two fingers (not your thumb). Count the beats for a ten second period, beginning with zero (i.e. 0, 1, 2, 3, etc, not 1, 2, 3, 4). Multiply this by six to get your minute pulse rate, and endeavour to keep between the two values given to you.

Training

At this time, official training will be carried out on three days per week, Mondays, Wednesdays, and Fridays at 5.30 p.m., at Lysarah's Oval, Figtree. Try to make all of these sessions, but if you cannot attend, please train in your own time. You will find that training in a group is more enjoyable, and will help you to adhere to the training programme.

When you train at home, here are some tips:

1. have a good stretch, especially legs, for least 5 minutes
2. walk/jog to warm up, for another 5 minutes
3. take your pulse rate immediately at the end of the warm up, so as to get an idea of how much harder you will need to work during the conditioning time
4. train for the specified time (the warm up and stretching are not part of this total time!!)
5. take your pulse rate again immediately at the end of conditioning and record both of the heart rates obtained (post warm up and conditioning)
6. take at least five minutes to cool down. Most physical problems occur due to blood pooling in the legs by not cooling down, which often results in fainting.
7. Any queries, phone me at home (293-827) day or night.
APPENDIX VI

PERCENTAGE BODY FAT EQUATIONS
Calculations of the body fat were based on the following formulas:

\[
\text{Fat} \, \% = \left( \frac{4.95}{\text{density}} - 4.5 \right) \times 100
\]

\[
\text{Density for men} = 1.160 - 0.0632 \times X
\]

where \( X \) = sum of skin fold thicknesses at all four sites in mm.

from Durnin and Rahaman (1967).