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THE EFFECTS OF EXERCISE AND DIET ON SELECTED PHYSIOLOGICAL AND BIOCHEMICAL PARAMETERS IN A SEDENTARY INDIAN MALE COHORT

by

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DEDICATION

To my valued critic, friend and **wife,** ***Premla Coopoo.***

In memory of my late **parents,** ***Nadesan and Govindamal,*** who planted the seeds of knowledge and in memory of my late **daughter,** ***Sarushka Coopoo.***

And finally, to my **daughter,** ***Kevanya Coopoo*** who provides the bliss and reduces the pain.

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DECLARATION

It is hereby declared that this is my own work, both in conception and in execution and any opinions and conclusions contained herein do not reflect the view of any person or institution mentioned.

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Y. COOPOO

January, 1995

ABSTRACT

In common with other expatriate Indian Populations, the Indian community of South Africa has a high incidence of coronary heart disease (CHD). Little information is available on the effects of exercise and diet on risk factors in this group. The present study is directed at the functional changes occurring as a result of a moderate aerobic physical activity programme, comprising 30 minutes of supervised exercise, three times per week for 15 weeks. Healthy male volunteers were recruited from the staff of the University of Durban-Westville, who were not on any lipid lowering medication and were not involved in any programme of physical activity for at least 12 weeks before the start of the project. The 41 subjects were assigned into one of three groups: exercise only (E) (15 subjects); exercise and diet (ED) (14 subjects) and a control (C) (12 subjects) group on no intervention. Besides laboratory investigations all participants were subjected to standardized fitness and anthropomorphological evaluation, a brief family history for coronary artery disease and a detailed dietary history was compiled.

Baseline lipid results indicate that only 7 of the 41 subjects had normal lipid profiles using as cut-off points 5.2 mmol/l for cholesterol, 1.5 mml/l for triglyceride and 0.9 mmol/l for HDL-C. Obesity was moderately prevalent before intervention, with a mean decrease of 25% in body fat in both E and ED groups($P \leq 0.05$). The experimental subjects became leaner.

After the intervention programme an average 20 percent increase was evident in physical working capacity as measured by peak VO_2 in both experimental groups ($P \leq 0.01$). The controls showed little variation over the 15 weeks. The indices of muscular endurance and

flexibility showed statistically significant changes ($P \leq 0.05$) in both experimental groups after intervention. This certainly indicates elevated levels of fitness after the intervention. The lipid profiles show little alteration in total cholesterol, with a 7.3% decrease in triglyceride levels in the E group (which was not statistically significant) compared with a 14.7% increase in the controls. HDL-C showed an increase in both experimental groups ($P \leq 0.01$). The total cholesterol to HDL-C ratio had an average fall of 11.9% in the experimental groups ($P \leq 0.05$) compared with a 5.6% decrease in the control group.

These data support the claim that regular, moderate exercise reduces the risk of heart disease through its effects on coronary risk factors in a high risk South African population.

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1. INTRODUCTION

Coronary heart disease (CHD) has reached "epidemic" proportions in the 20th century. It represents the clinical outcome of a chronic process initiated and sustained by a variety of factors, collectively termed risk factors. Risk factors may be classified into those which are modifiable such as diet, exercise, cigarette smoking, hypertension, obesity and plasma lipid profile and those which are non-modifiable such as age, gender and family history. Lifestyle habits and circumstances may influence many of the other modifiable risk factors, which also have a genetic component and thus reduce the risk of CHD. These complex relationships are illustrated in Figure 1.1 and form the conceptual framework for this thesis.

In common with other expatriate Indian populations, the Indian community of South Africa has a high incidence of coronary heart disease (CHD) (Sharper and Jones, 1959; Ashcroft et al., 1970; Pedoe et al., 1975; Miller et al., 1982; Miller et al., 1984; Lowry et al., 1984; Beckles et al., 1986; Seedat et al., 1990). Seedat (1993) reported that in 1985, the relative risk of CHD deaths among South African Indians when compared with Whites, was 1.3 in males and 1.7 in females.

Epidemiological studies have isolated various risk factors which may be implicated in the increased risk of cardiovascular disease. The major known risk factors are hypertension, certain pathogenic plasma lipid profiles, cigarette smoking, obesity, diabetes, decreased HDL-C, lack of physical activity and personality type. (Hietanen, 1982; Byrne, 1991; Gordon and Gibbons, 1991). More controversial risk factors are chronic stress, personality type, life events and social support (Byrne, 1991, Tenebaum and Singer, 1992). The

difficulties in determining causal relationships between risk factors and CHD are numerous, and this also applies to physical activity and its effects on cardiovascular disease.

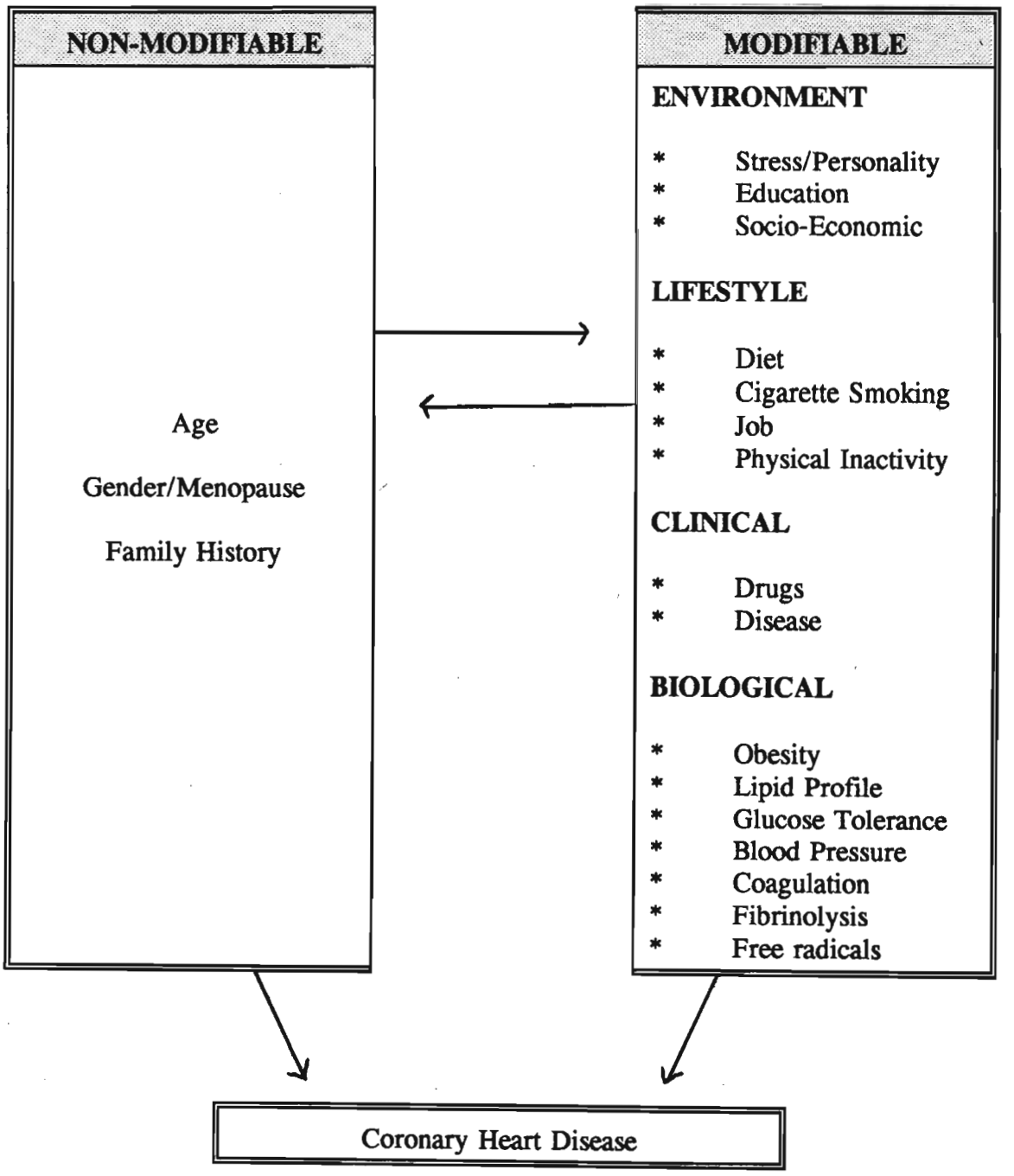

Figure 1.1 **Risk Factor Interaction and Coronary Heart Disease.**

Support for the value of physical activity in reducing the risk of coronary artery disease (CAD) come from the American Heart Association (AHA) in a position statement published in 1992 (American Heart Association,1992). They gave recognition to the relationship between aerobic exercise and cardiovascular health and named inactivity as the fourth modifiable risk factor, the other three being cigarette smoking, hypertension and high serum cholesterol levels. Changes in lifestyle promote optimal health and longevity (Paffenbarger et al., 1993). Seedat (1993) reported that the modifiable risk factors, such as cessation of cigarette smoking, the control of hypertension and reduction of dietary saturated fats are important components in the control of CHD. In addition he stated, that the control of obesity and regular exercise throughout life may be equally important in preventing diabetes and CHD in the Indian population of South Africa.

Walker (1973) indicated that the South African Indian population enjoys better economic conditions than do their rural counterparts in India. This enhanced economic advantage did not improve life expectancy however, as the cause of death simply changed from infectious diseases in India to degenerative diseases in South Africa. South African Indians beyond middle age were worse off in terms of life expectancy than Indians in India.

The high incidence of CHD in the metropolitan hospitals of Durban has been reported by Mayet (1982) Sewdarsen et al.(1987), Sewdarsen et al. (1988) and Seedat et al.(1982 and 1990). In a longitudinal study on Indian hospital admissions to the R.K. Khan Hospital in Durban, records were analysed on 31 101 adult patients over a ten year period (1970-1980). In this study Mayet (1982) established that 11% of all medical admissions involved CHD and

24% of all deaths were CHD related. A noteworthy observation was that 22% of the patients admitted to the coronary care unit of the hospital were under the age of 45 years.

In a risk factor classification study, Sewdarsen et al.(1987), analysed the data obtained from 108 Indian male patients (aged 21-40 years) admitted to the R.K. Khan hospital between 1981-1983 for the treatment of myocardial infarction. The frequency of the risk factors in this particular sample of men are summarized:

1.1 Cigarette smoking was the most common risk factor with 79% of the patients being smokers.

1.2 50% of the patients had serum cholesterol levels above 6.5 mmol/l.

1.3 53% of the patients had triglyceride levels greater than 2,0 mmol/l.

1.4 52% of the sample had HDL-C levels below 0.83 mmol/l.

1.5 37% of the patients had impaired glucose tolerance.

1.6 32% of the patients were hypertensive.

1.7 10% of the patients were both hypertensive and had impaired glucose tolerance.

The study clearly showed that smoking, abnormal plasma lipid levels, hypertension and impaired glucose tolerance were among the major risk factors associated with this sample of patients afflicted with myocardial infarction. The physical activity patterns of these patients were not assessed.

There is good biochemical as well as epidemiologic evidence that these risk factors are causally related to CHD and thus modification through lifestyle changes should reduce such risk. (Noakes, 1986; Ornish et al., 1990; Wood et al., 1991; American Heart Association, 1992).

In cross-sectional studies, data indicate that regular aerobic exercise improves lipid profiles, reduces stress and lowers blood pressure (Hietanen, 1982; Oberman, 1983; Noakes, 1986). Migration studies show that there is a significant increase in CHD events when people migrate from their homeland to their adopted country (Lowry et al.,1984; Beckles et al.,1986). High saturated fat diets, a more affluent lifestyle, smoking and lack of exercise are also implicated in this change (Ashcroft et al., 1970; Pedoe, et al., 1975; Miller, et al.,1984).

This study seeks to contribute further to the growing body of evidence supporting the contention that regular physical activity may reduce the development of CHD among the migrant Indian population of South Africa. It also seeks to provide data indicating that regular physical activity may positively influence a range of preventable medical conditions.

1.2 STATEMENT OF THE PROBLEM

The Indian population of South Africa is at high risk in the areas of CHD and diabetes. These risk factors include pathogenic plasma lipid profiles, obesity, impaired glucose tolerance and hypertension. Daily physical activity levels are low, with little or no cardioprotective effect derived from enhanced activity levels. It would appear that a

significant part of the problem relating to the high incidence of CHD in this population revolves around poor lifestyle habits. These include a high saturated fat intake, cigarette smoking, lack of physical activity, excess caloric intake and a stressful lifestyle. (Sewdarsen et al., 1987 and Seedat, 1993)

This study sought to monitor basal (steady state) changes in coronary risk factors occurring as a result of an intervention programme consisting of diet plus exercise and exercise alone, in a group of sedentary Indian male adults. A secondary objective was to establish whether a moderate exercise programme would bring about an improvement in selected indices of physical fitness and body composition.

1.3 HYPOTHESIS

1.3.1 It was hypothesised that selected indices of physical fitness and body composition would be enhanced by a regime of aerobic exercise, or one of aerobic exercise and dietary modification, in a cohort of sedentary Indian males.

1.3.2. It was further hypothesised that such intervention would reduce the CHD risk factors associated with lipid and lipoprotein levels in the same cohort.

1.4 DELIMITATIONS

This study was delimited to the data obtained from 41 selected Indian males who participated in the research programme conducted between February and June, 1992.

It was further delimited to selected physiological and biochemical evaluations.

1.5 LIMITATIONS

1.5.1. Resources and practical considerations limited the sample size. Totally random selection or perfect matching between the control group and the two experimental groups was also not attainable.

1.5.2 Although every effort was made to ensure the cooperation of the subjects concerning diet and exercise, absolute conformity in this respect was not possible. In particular it was evident that some subjects in the control and exercise groups may have spontaneously introduced improved dietary practices despite instructions to the contrary.

1.6 ASSUMPTIONS

1.6.1 It was assumed that the information pertaining to family history and dietary habits was reliably reported by all subjects.

1.6.2 It was also assumed that the method of placement of subjects in the different experimental groups would not influence the conclusions drawn from the study.

1.6.3 It was further assumed that any minor degrees of non-compliance, among the subjects, would not be sufficient to reduce the significance of the findings.

1.7 ABBREVIATIONS AND SYMBOLS USED

BMI	:	Body Mass Index
CHD	:	Coronary heart disease
ECG	:	Electrocardiogram
HDL-C	:	High density lipoprotein (cholesterol)
HR	:	Heart rate
IHD	:	Ischaemic heart disease
Kcal	:	Calorie
Kg	:	Kilogram
g	:	Gram
LBM	:	Lean body mass
LDL-C	:	Low density lipoprotein (cholesterol)
MI	:	Myocardial infarction
% fat	:	Percentage body fat
VO_2 Max	:	Maximal oxygen uptake
P:S ratio	:	Polyunsaturated to saturated fatty acid ratio
V'e	:	Ventilation

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2. REVIEW OF LITERATURE

Regular exercise is known to increase physiological fitness by improving cardiorespiratory efficiency and muscular capacity. Large scale longitudinal studies have suggested that physical activity decreases cardiovascular mortality (MRFIT, 1982). The exact mechanisms by which exercise may be implicated in the reduction of CHD are not known, although several explanations have been proposed. Increased physical activity is associated with decreased concentrations of plasma triglycerides, VLDL-C and LDL-C and increased levels of HDL-C.

These alterations in the blood lipid profile are associated with a lower risk of CHD (Wood and Haskell, 1979). Another proposal is that exercise increases fibrinolysis induced by venous occlusion and changes in platelet stickiness and thrombosis formation hence inhibiting the progression of thrombosis (Williams et al.,1980; Paffenbarger and Hyde, 1980 and de Geus et al.,1992). There is also evidence to suggest that physical activity increases insulin sensitivity and may prevent the development of insulin-resistant states such as obesity and adult-onset diabetes, both of which are implicated as risk factors in coronary heart disease.(Vranie and Berger, 1979 and Pederson et al., 1980).

Increased fitness has a variety of other beneficial effects. These include increased oxygen uptake, reduced heart rate, lowered blood pressure, increased cardiac output and increased physical working capacity. (Astrand and Rodahl, 1970; Wilmore, 1977). All these may contribute to the reduction of CHD.

This chapter reviews research concerning the nature of coronary heart disease and the influence of exercise on serum lipids and other coronary risk factors.

2.1 CORONARY HEART DISEASE (CHD)

CHD is caused by obstruction in the coronary arteries. Cardiac tissue requires a continuous supply of both oxygen and nutrients in order to function. The mechanism of a thrombosis has been well described (Jackson, 1990). When a blood clot (thrombus) is formed in the coronary artery, it obstructs the blood flow to the cardiac muscle. If this interruption of oxygen is prolonged, death of the segment of cardiac muscle around the affected artery may occur. This is known as myocardial infarction. Clinically the patient experiences severe chest pain, radiating to the left arm and neck. The pathophysiology of CHD is multifactorial and lipids and lipoproteins are implicated. (Schneider et al., 1986).

Williams et al. (1980) has described arteriosclerosis as a term applied to a number of different pathological conditions in which arterial walls thicken and lose their elasticity. Atherosclerosis, is a form of arteriosclerosis and is characterized by deposits of fat, cholesterol, cellular debris, calcium and fibrin on the inner linings of the arterial wall. These deposits are known as plaque, which narrow the lumen and promote the formation of thrombi, reducing the supply of oxygen and nutrients to the cardiac muscle (Williams, et al. 1980).

The precise mechanism at the cellular level of the development of atherosclerosis of the coronary arteries is unclear (Hietanen,1982). Further research is required in order to ascertain why plaque build-up in the coronary arteries occurs at a specific site in one artery and not in another which is seemingly exposed to the same conditions (Haskell,1987). Another point of contention concerns the formation of blockages in the same coronary artery at differing rates as well as in different arteries in the same person (Haskell, 1987).

The most popular and plausible of the current theories is the arterial wall damage theory which suggests a number of stages (Fuster, et al.,1992).

2.1.1 **Stages of Atherosclerosis**

In the development of atherosclerosis, the lumen of the artery gradually narrow in parts of the arterial tree, at bending points and areas near branching vessels. This narrowing of the lumen is due to lipid deposits, smooth muscle cell proliferation, enhanced collagen formation and calcification. The atherosclerotic process appears to involve primarily the intima of the artery, consisting of a single layer of endothelial cells as well as smooth muscle cells (Schaefer,1990 and Fuster et al.,1992). These stages have been described as follows:

2.1.1.1 **Fatty streak stage**

The fatty streak is the first stage in the development of atherosclerosis. It is postulated that fatty streaks arise when excess LDL-C infiltrates underneath the endothelium and is then modified or oxidized. Infiltration of LDL-C into the arterial wall appears to be accelerated

in the presence of arterial hypertension, in smokers and when LDL-C levels are high (Fuster, et al.,1992). This modified LDL-C attracts monocytes, which migrate from the lumen into areas below the endothelium, where they take up the modified LDL-C and become lipid-laden macrophages or foam cells. Cholesterol is the major lipid component within the foam cell. The fatty streak is composed mainly of foam cells beneath the endothelial cell layer (Schaefer, 1990).

2.1.1.2 **Fibrous plaque stage**

The second stage in the development of atherosclerosis is the formation of the fibrous plaque. When the lipid laden macrophages within the fatty streak exert pressure on the endothelial cells, the rupture of this endothelial layer occurs, exposing smooth muscle cells to the luminal blood. Platelets rapidly stick to the smooth muscle cells, aggregate and release platelet derived growth factor. This stimulates smooth muscle cell proliferation and also causes the cells to produce excess amounts of fibrous connective tissue, including collagen fibres and reticulum fibril within the ground substance of the plaque. The fibrous plaque consists of a central necrotic core containing intracellular lipids within macrophages as well as some extracellular lipids with a fibrous cap above it. A fibrous plaque may remain stable for many years. The extent to which such plaques decrease the lumen of the artery appears to depend on the amount of lipid deposited in the necrotic core of the plaque, as well as the amount of associated smooth muscle proliferation and connective tissue formation (Schaefer, 1990).

2.1.1.3. Complicated lesion stage

Ultimately, the areas of fibrous plaque develop into more complicated lesions involving further lipid deposition, thrombosis formation, calcification and ulceration. When the fibrous plaque exerts force against the arterial wall, a rupture of the small arterioles occurs, causing a haemorrhage. Frequently, the hardening of the plaque within the arterial wall occurs, which may cause this fibrous plaque to crack, resulting in fissures or ulcers where the smooth muscle cells are exposed to the perfusing blood in the lumen. At these damaged sites, platelet aggregration occurs, presenting large thrombogenic surfaces. This thrombus formation is usually the terminal event leading to arterial occlusion (Schaefer, 1990).

In conclusion, the process of atherogenesis begins with the formation of fatty streaks in childhood and adolescence. However, diet and lifestyle changes may arrest or even reverse this arterial pollution. Ulbricht and Southgate (1991) suggest that antioxidants in the diet may be much more important than was thought and this may be a direction for further research. The earlier the lifestyle changes occur, the better, because plaque is more resistant once calcification and scarring develop. The key is prevention (Byrne, 1991).

2.1.2 Lipids and Lipoproteins

There is mounting evidence indicating that LDL-C, HDL-C and total cholesterol are strong predictors of CHD, with triglycerides not being regarded as an independent risk factor (Castelli et al.,1977; Gordon et al., 1977; Dufaux et al.,1982 and NIH Consensus Conference, 1993).

Selected studies will be discussed showing the relationship between lipoproteins and CHD, with specific reference to triglycerides, cholesterol, LDL and HDL.

2.1.2.1 Triglycerides (TG)

Clinical trials specifically directed at the lowering of triglycerides with a view to reducing CHD have not been reported. Measurement of triglycerides in other studies where the focus was on LDL-C and total cholesterol have been undertaken. These however showed little association between triglycerides and CHD (NIH Consensus Conference, 1993). However, the Procam data (The New European and United States Guidelines) indicate that elevated triglyceride levels in association with an increased total cholesterol to HDL-C ratio (>5) and low HDL-C (<0.9 mmol/l) is a significant determinant of CHD (Assmann, 1993).

Recent research has associated TG levels with alterations of the coagulation system and elevated TG levels have been associated with an increase in several coagulation factors and altered fibrinolytic factors (increased plasminogen activator inhibitor).

It has been suggested that the effects of elevated TG on CHD may be mediated through its effects on the clotting and fibrinolytic mechanisms (NIH Consensus Conference, 1993).

2.1.2.2 Cholesterol

Numerous studies have shown a relationship between total cholesterol levels and the incidence of CHD (Kannel et al.,1974 and 1977; Thelle et al.,1976; MRFIT, 1982 and Lewis et al.,1986). Serum cholesterol levels show a wide distribution within populations,

geographical areas and ethnic groupings. In a study of four European communities (Lewis et al.,1978), serum lipid and lipoprotein levels were measured in 985 men and women from London, Naples, Uppsala and Geneva. Cholesterol and triglyceride concentrations differed substantially between the four populations. The results indicated that for both sexes, the total cholesterol levels were higher in London and Uppsala than in Naples and Geneva. Higher TC levels also indicated greater incidences of CHD.

Dietary patterns in seven countries were compared to total cholesterol levels and to CHD rates. In populations where the average serum cholesterol level was 6.2 to 7.2 mmol/l, the incidence of CHD was 10 times higher than in countries where the average serum cholesterol level was 4.1 mmol/l. A correlation of .89 was noted between saturated fat intake and serum cholesterol levels (Keys, 1970).

The Lipid Research Clinic Program (LRCCPPT), in a controlled study of asymptomatic middle aged men with elevated cholesterol, indicated that through the drug management of cholesterol, significant reductions in CHD and non-fatal heart attacks could be achieved (LRCCPPT,1984). It would appear that optimal levels of cholesterol ($\leq$5.2 mmol/l) are advisable for the general population, and that lowering the concentrations below normal may reduce the risk factors for CHD.

2.1.2.3 **Low density lipoproteins (LDL-C)**

Studies have indicated clearly that the principal carrier of cholesterol in the plasma is LDL-C, and prospective studies have shown that raised LDL-C is positively associated with

the risk of CHD (LRCCPPT, 1984). Gofman et al.(1950) first identified elevated LDL-C in a group of patients suffering from myocardial infarction. The Helsinki Heart Trial (Frick et al., 1987) supported evidence that reduced LDL-C levels leads to lower risks of CHD. Wood and Stefanick (1990) reported that accumulation of cholesterol within the artery occurs when LDL-C blood levels are high and when the mechanisms involved with intracellular degradation are impaired. These increased LDL-C levels may damage the arterial endothelium, causing platelet aggregation, and trapping cholesterol in the matrix.

Research studies have shown that medication may increase the number of LDL-C receptors which may lead to decreased plasma cholesterol levels and retarded CHD risk (MRFIT,1982).

Thus with the reduction of LDL-C, either through medication or lifestyle changes, the atherosclerotic plaque may well be reduced or reversed resulting in a reduction of CHD risk.

2.1.2.4 **High density lipoprotein (HDL-C)**

Until very recently the major emphasis concerning lipoproteins and risk of CHD focussed on total cholesterol, LDL-C and VLDL fractions. In an important study Barr et al.(1951) found that healthy men had higher HDL-C levels than men with CHD. Miller and Miller (1975) showed that low levels of HDL-C were directly related to the development of ischemic heart disease. Most subsequent studies [Tromso Heart Study (Thelle et al., 1976); Cooperative Phenotyping Study (Castelli et al., 1977) and Israeli Heart Disease Study (Goldbourt and Medalie, 1979)] supported the view that the incidence of CHD increases with

decreased HDL-C levels. In the Framingham study (Kannel et al., 1977) of an entire community, HDL-C was singled out as the best indicator of heart disease risk. In two major drug intervention studies, the increase in HDL-C through medication resulted in decreases in the risk of CHD [LRCCPPT, 1984 and Helsinki Heart Study (Frick et al., 1987)]. The results of the Helsinki Heart Study (1987), showed that in drug-treated men a mean increase in HDL-C of 10% resulted and a decrease in LDL-C of 10%, which reflected an overall decrease in incidence of CHD of 34% ($P<0.02$).

It has been well demonstrated that low concentrations of HDL-C increases the possibility of the clinical manifestation of CHD. Raising the HDL-C in patients with elevated LDL-C levels will have a beneficial effect on reducing the risk for CHD (Schaefer, 1990).

2.1.3 **Physical Activity and CHD**

A number of risk factors are clinically recognized in the pathogenesis of CAD, and many other factors interact in increasing the possibility of the development of CHD. The risk of CHD may be doubled or tripled when more than one risk factor is present (Jackson, 1990). Table 2.1 summarizes the more important known risk factors implicated in the development of CHD.

Table 2.1 **Known Risk factors for CHD**

(Hietanen, 1982; Schneider, et al.,1986; Schaefer, 1990 and Ulbricht and Southgate, 1991)

BIOLOGICAL RISK FACTORS	BIOCHEMICAL RISK FACTORS
* Sex, Age	* Lipoprotein abnormalities
* Family history of premature CHD before age 55	* Hyperlipidaemia
* History of cerebrovascular or occlusive peripheral vascular disease	* Hypertension
* Severe obesity (>30% above ideal body weight)	* Glucose intolerance (diabetes)
* Cigarette smoking	* Coagulation and fibrinolysis factors
* Ethnic and psychosocial factors (stress and personality)	* Environmental Compounds (e.g chorinated hydro-carbons)
* Lack of physical activity	* Estrogens
* Geographic variation	* Alcohol intake
	* Minor factors (Trace elements, hyper-calcemia, vasectomy, coffee, Cardiac transplants, hyper-uricemia).

However, it appears that in many instances, the elimination of these risk factors has not resulted in a retardation or reversal of CHD. Physical activity on the other hand, improves a number of these risk factors, and it's apparent beneficial effect on CHD could reduce or retard the severity of this disease (Byrne, 1991).

Progressive aerobic activity, engaged in on a regular basis plays a role in the primary and secondary prevention of cardiovascular disease (Chandrashekhar and Anand, 1991). Very recently the American Heart Association (AHA) in a position statement on exercise indicated that inactivity is a recognised risk factor for CHD (AHA: 1992).

Various organic systems in the body can alter their function as part of an adaptive process in response to physical activity. These alterations may in some way assist in the retardation of CHD or may protect from the effects of the disease (Chandrashekhar and Anand, 1991).

A number of physiological changes in the cardiorespiratory system may occur as a result of aerobic training, which may indirectly or directly reduce the development of CHD. Only exercise-induced cardiorespiratory changes will be discussed.

There is evidence indicating that aerobic training engaged in by apparently healthy people induces ventricular hypertrophy which occurs rapidly and is reversed during detraining (Saltin et al., 1969; Maron, 1986). Exercise-induced ventricular hypertrophy is unlike pathologic hypertrophy, in that it is not associated with reduced myocardial contractility (Chandrashekhar and Anand, 1991). In healthy persons aerobic training increases left ventricular end-diastolic volume and these changes are lost with deconditioning (Chandrashekhar and Anand, 1991). These changes increase cardiovascular functional capacity and decrease the myocardial oxygen demand for the same level of work performed. This is achieved by a decrease in the product of heart rate and systolic arterial blood pressure (an index of myocardial oxygen consumption). These adaptations also benefit coronary

patients, who after exercise may attain a higher level of physical work before reaching the level of myocardial oxygen requirement that results in myocardial ischemia (AHA, 1992).

Another well documented response to training is the increase in total work load during the exercise stress test. This is measured by assessing maximal oxygen uptake, (VO_2 Max) and increases of between 15% to 20% have been recorded after aerobic training programmes (Astrand and Rodahl,1970 and Fox et al.,1993).

The increase in VO_2 Max is brought about by two main physiological changes, i.e.

(a) An increased oxygen delivery to the working muscles through increased cardiac output.

(b) An increased oxygen extraction from the blood by the skeletal muscle (Wilmore, 1977; Fox et al.,1993).

With the increase in VO_2 Max a decrease in resting, exercise and intrinsic heart rate occurs as well as an increase in stroke volume which would improve the functional capacity of the heart (Chandrashekhar and Anand, 1991).

It is postulated that training triggers the formation of collateral coronary circulation.

There are a number of studies concerning the increase in coronary collateral vascularization in animals after exercise; (Eckstein, 1957; Sanders et al.,1978 and Spear et al.,1978), and

the most important of these was undertaken by Eckstein (1957), using dogs as subjects. This study concluded that coronary collateral vascularization was developed around artificially induced coronary artery constriction in direct proportion to the amount of exercise performed. In sedentary dogs with the same degree of arterial constriction, the vascularization was not as great. It would appear that exercise caused the development of increased collateral circulation in the active dogs.

Studies by Rose et al.(1967) and Curren and White (1961) showed increases in coronary artery size with increased physical activity in humans. In an autopsy study of a famous marathon runner, Clarence De Mar, Curren and White (1961) showed that the runner's coronary arteries were two to three times the size of a normal artery. These large coronary arteries were attributed to the runner's regular physical activity, although genetics could not be discounted as a factor in the development of his large coronary arteries. The development of collateral circulation through exercise still remains controversial however. Haskell et al. (1993) published data on the response of nitroglyceride administration on the dilating capacity and coronary artery size in ultra-distance runners compared to sedentary subjects, using computer assisted arteriographical measurements. The results demonstrated for the first time that the coronary arteries of highly trained middle-aged men have a greater dilating capacity in response to nitroglyceride than those of men who are generally sedentary. Given the cross-sectional nature of the study, it is not possible to know if this difference was caused by running or was due to some other acquired trait or to genetic selection. Further research is needed in order to determine the causes of the greater dilating capacity of epicardial coronary

arteries in highly trained runners and if any clinical benefits is provided by this increased capacity.

Regular exercise appears to decrease the possibility of developing ventricular fibrillation. Noakes et al.(1983) showed that the hearts of rats undergoing regular exercise were more resistant to the development of ventricular fibrillation.

Gordon and Gibbons (1990) summarized the postive impact that regular exercise can have on other CHD risk factors. These are:

* Increased level of HDL-C
* Reduced ratio of total cholesterol to HDL-C
* Lowered triglycerides
* Reduced risk of developing hypertension
* Lowered both systolic and diastolic pressure in hypertensive persons.
* Prevention of obesity by reducing the amount of excess fat
* Relief of stress and a modifying influence on Type A behaviour.
* Prevention or control of diabetes mellitus by aiding carbohydrate metabolism.

In conclusion, evidence indicates that physical activity and the resulting physical fitness have a positive effect on the rate and development of coronary artery disease (Chandrashekhar and Anand, 1991).

2.2 EXERCISE, LIPIDS AND LIPOPROTEINS

2.2.1 Prospective Studies

There is increasing evidence supporting the independent role of increased physical activity in the primary prevention of CHD, with most major reviews concluding that physically active people are at lower risk from CHD than their inactive counterparts (Froelicher et al.,1980; Oberman, 1983; Berlin and Colditz, 1990; Chandrashekhar and Anand, 1991 and American Heart Association, 1992). The role played by exercise in favourably altering lipoprotein profiles, has been most noticeable in the reduction of CHD (Wood and Haskell, 1979; and Dufaux, et al.,1982). This critique of literature comprises a historical review of prospective occupational and leisure time physical activity and it's relationship to CHD, followed by studies relating to lipoprotein studies, where exercise was used as an intervention strategy.

Studies in this area have used various methods of investigation. These include autopsy studies, population studies, cross-sectional and prospective cohort studies. Apart from variations in experimental design, reported research in this area exhibits many features which make uniform conclusions difficult. A brief discussion of the general dissimilarities of these studies follows:

a) The majority of studies have used only males as subjects.

b) The methodology in many trials has been longitudinal, lasting for decades. This may have altered the natural history of the disease, namely through a better understanding of coronary risk factors, increased population awareness and higher standards of medical care.

c) The contentious problem of self-selection is often evident. Whether fit and healthy persons choose to be more active than the unfit, obese and infirm, is open to question.

d) Different methods of assessment have been used (e.g. questionnaires, observational studies, recall questionnaires etc).

e) In a number of studies occupational activity was examined whereas, in other studies leisure activity was assessed.

f) Unknown variables may influence the outcome in non-randomized trials (e.g. changes in smoking patterns, exercise behaviour, or calorie restriction).

Notwithstanding these study deficiencies, a recent meta-analysis, (Berlin and Colditz, 1990) took the study designs of research into account and concluded that the relative risk of death from CHD was 1.9 times greater among sedentary subjects compared with those having active occupations. The authors also found that methodologically stronger studies tended to show a larger benefit of physical activity than the less well designed studies.

The highlights of some of the prospective historical landmark studies concerning occupation or leisure time activities require special mention. The association between sedentary work and CHD was first reported in the early 1950's. Morris and co-workers, in a classic study (1953), first established a relationship between the incidence of heart disease and job related activity. They found that bus drivers were more prone to heart disease than their more active counterparts, the bus conductors. A similar study in the United States compared postmen, who delivered letters door to door, with desk bound postal clerks. A similar trend was noted

in that the active postmen recorded less coronary heart disease than the sedentary postal clerks (Morris and Crawford, 1958). A major limitation of these studies concerned self-selection where the obese or hypertensive or generally less fit person was more likely to apply for a sedentary occupation. These studies however initiated investigation into the association between activity and heart disease.

In a follow-up study of 16,882 men aged between 40-64 years of age selected from a population of civil servants, Morris et al. (1973) obtained data regarding health history and physical activity over weekends, by means of a questionnaire survey. The survey indicated that there were twice as many "sedentary" as "vigorous exercise" men among the coronary patients. Although the distribution of smoking habits was not reported in this study, the researcher subjectively observed that smoking trends were similar in both active and inactive groups.

This study further concluded that vigorous exercise offered protection against heart disease whereas light exercise did not. In essence these results supported the findings of earlier studies by Morris et al.(1953) and Morris and Crawford (1958) which indicated that the more active bus conductors and postmen were protected against heart disease.

Morris and co-workers (1953) and Morris and Crawford (1958), did not account for confounding variables such as self-selection, cigarette smoking, body composition, familial aspects and psychological stress. Their studies did however stimulate further investigation into the probable relationship between exercise and the reduction of CHD.

In the United States, a prospective study of work activity and fatal heart attacks among San Francisco longshoremen was conducted by Paffenbarger et al. (1977). This included nearly 4000 men aged 35 to 74 years who were monitored for 22 years. These workers ranged from high energy expenditure cargo handlers and dockworkers to low energy expenditure supervisors and light machine workers.

These participants were given multiphasic screening examinations on entering the study in 1951 to assess five personal characteristics, namely cigarette smoking, systolic blood pressure, diagnosed heart disease, weight, height and glucose metabolism. Serum cholesterol levels were measured in 1961.

Energy expenditures were calculated by measuring oxygen consumption of longshoremen during actual work-related tasks. Low energy work was categorized as those jobs demanding fewer than 8500 kilocalories per week on the job, while the high energy workers were classified as those who expended 8500 or more kilocalories per week. Leisure activities were not considered. The longshoremen were then monitored over a 22 year period to determine the rate of fatal heart attacks among low energy and high energy workers.

The results may be summarized as follows:

(1) The high-energy and low energy groups differed little in personal characteristics assessed at multiphasic screening at the outset of the study in 1951. High energy and low energy groups were alike in sharing recognised coronary risk factors.

(2) About 10 percent of the longshoremen died of heart attacks during the 22 year period.

(3) Men who had expended 8500 or more kilocalories per week had significantly less risk of fatal heart attacks, particularly sudden death from heart attacks, than did men whose jobs required less energy output.

The results of this study provided the strongest evidence yet of a protective effect of exercise against heart attacks. In drawing conclusions from epidemiological surveys however, it is important to keep in mind that the analyses are relative. Office workers would not expend the same amount of energy as the longshoremen doing work on the docks.

Epidemiological studies of exercise and CHD risk have to consider the problem of self-selection. The question arises of whether the lower incidence of CHD among physically active individuals is due to their physical activity or other genetic factors.

The low rate of sudden death among high energy workers does however suggest that exercise may indeed have a further protective effect because it appears that highly active men may be better able to withstand initial heart attacks and may be less likely to die suddenly (Thomas et al.,1981).

Paffenbarger et al.(1978), in his study of longshoremen, also supported the view that continuing exercise was important and that high energy work output was of great value in prevention of heart disease. The study also noted that the advantage of exercise faded if the

activity was not maintained. This was demonstrated when workers were transferred from heavy-energy work to light activity jobs.

In an extensive study, Paffenbarger et al.(1978) recorded physical activity as an index of heart attack risk among Harvard alumni. This study helped clarify the role of past and contemporary exercise habits in alumni and it's relationship to CHD.

The study documented experiences and characteristics of alumni who had entered college as students between 1916 to 1950 and related this data to the incidence of death from heart attacks. Investigators gathered information from college archives, alumni records, questionnaires, official death certificates, student health and athletic records. Further information about a number of student characteristics such as cigarette smoking, systolic and diastolic blood pressure, stature, mass, parental death and disease were also obtained.

Some 17 000 alumni replied to a questionnaire concerning their personal characteristics, exercise habits and physician-diagnosed disease, including CHD. In 1972 a second questionnaire was mailed to alumni who had entered Harvard between 1916 and 1950 to determine the incidence of physician diagnosed diseases, including CHD.

Weekly updating of death lists by the alumni office provided a means of obtaining death certificates to identify fatal heart attacks. Results of this provided a 6-10 year follow-up of risk of first heart attack, fatal and non-fatal, in relation to physical activity.

The more important results may be summarized as follows:

(1) Nearly 17 000 Harvard alumni aged 35-74 years returned questionnaires and reported themselves free of coronary heart disease in 1962 or 1966.

(2) By 1972, 572 men had experienced first attacks, 357 non-fatal and 215 fatal. Fifty-two of these attacks occurred between ages 35-44 years, 137 between 45-55 years, 213 between age 55-64 years and 170 between ages 65-74 years.

(3) Alumni who reported more than 2000 Kcal expended per week had a 50 % lower risk of heart attack than did their less energetic classmates.

(4) Reduced risk of heart attack with increased physical activity was observed in each age group studied, and patterns were similar in relation to fatal and non-fatal attacks.

(5) Fifty six percent of former varsity athletes reported maintaining their active status in vigorous sports or high-energy exercise and a total of 38 % of non-varsity athletes reported activity sufficient to place themselves in the high energy category. Both these groups experienced substantially lower heart attack rates than their currently less active counterparts. Therefore, only a physically active adulthood was associated with lower heart attack rates, regardless of student athletic status.

(6) Reduced risk of heart attack was observed with increasing energy output in each category of physical activity, especially in vigorous sports activity, but also in such activities as climbing stairs and walking. However, at any given level of energy output, risk of heart attack was markedly lower for vigorous sports than for other activities.

(7) It was estimated that if all alumni had expended 2000 or more Kcal per week, the number of heart attacks would have been reduced by about 26 %. If none had smoked, heart attacks would have been reduced by 25 % and if none were hypertensive rates would have been reduced by about 16 % (Thomas et al.,1981).

The findings from the studies on dockworkers and alumni strengthen the case supporting a protective effect of exercise against heart attack. Possible selective or hereditary influences in the Harvard alumni are partially discounted by the findings that varsity athletes had no special advantage over their non-varsity classmates unless they continued to be vigorously active in their alumni years.

On the other hand students who had not been varsity athletes or physically active in college but who adopted vigorous exercise habits in adulthood stood to achieve a lower heart attack risk.

Morris et al. (1980) reported further data on the relationship between coronary heart disease and physical activity. In this study 17 944 middle aged male civil servants were monitored from 1969 to 1978. The focus of the research was on lifestyle and health.

In a sample of 1400 office workers aged 40-65 years who were engaged in vigorous exercise and who did not smoke, 12 fatal first "coronaries" were registered in 12 000 man years of observation. They represented about one fifth of the total of their colleagues who recorded no vigorous exercise and who did smoke. This study did not take into account other risk

factors predisposing persons to coronary heart disease, such as family history, physique, smoking, hypertension and diabetes. The researchers postulated that increased levels of HDL, resulting from vigorous exercise may have had an effect on coronary arteries.

In the most recent study by Morris et al. (1990) in which 9376 men aged between 45-64 years volunteered in 1976, it was reported that exercise must be vigorous and on-going to protect against heart disease. Persons participating in "non-vigorous" activity such as golf and dancing did not enjoy a protective effect. Among men who played vigorous sports, and those who had performed such activity 25-45 years previously, CHD rates were similar to those who have never engaged in vigorous sports. The number of hours spent walking on a weekly basis was not compared with CHD rates, but the walking speed tended to show an inverse relationship to increased CHD rates.

The CHD rates were not associated with performing calisthenics or with the number of steps climbed daily.Recreational work such as gardening had no effect on CHD rates, regardless of it's quantity or intensity.

A multivariate analysis adjusting for risk factors such as family history and smoking did not change the overall finding that vigorous, aerobic exercise protected all men while lesser degrees of aerobic exercise protected older men.

In summary the initial contention of Morris et al.(1953) that there was an association between physical activity and the reduction in coronary artery disease was born out by

research continued over the next 30 years. Particular attention was paid in later studies to the possible confounding effects of selection bias and other variables (diet, cigarette smoking, genetic factors) which could associate with different levels of physical activity.

2.2.2 **Aerobic Exercise Studies**

Numerous studies have been published, recording the effects of exercise programmes on serum lipids and lipoproteins. Many of these studies used small samples while others did not use control groups. Further compounding factors included seasonal variations, relative body weight and dietary composition, all of which could influence lipoprotein changes, independent of exercise effects (Dufaux et al.,1982).

In semi-longitudinal and longitudinal studies concerning serum lipids and exercise, results have been both varied and inconsistent.

Factors such as duration, frequency, intensity of exercise and mode of training may interact and influence results. There have been wide variations with respect to the magnitude of lipid changes over various time frames. The majority of studies involving exercise training reported differences in lipoprotein levels after 13 weeks (Murray et al.,1990).

There have been exceptions to this general trend however. Lopez-S et al. (1974) detected decreased serum triglycerides and VLDL cholesterol levels with increased HDL values in young men training 4 days per week for 30 minutes per day after 7 weeks of exercise.

Studies by Golding (1961) Brumbach (1961) and Rochelle (1961), were among the first to investigate the effects of exercise on cholesterol levels. The duration of the exercise programmes differed, with periods of 25, 10 and 5 weeks being used respectively.

Both Golding (1961) and Rochelle (1961) used training frequencies of 5 days per week, with aerobic activity being emphasized in training. In both of these studies significant reductions in cholesterol levels were noted following the exercise period. Brumbach (1961) used 35 minutes physical education lessons three days per week as the intervention programme. No predictable effect upon serum cholesterol levels of the young men who participated in the study was discernable. Changes in cholesterol levels in both these studies were not associated with any specific duration although Rochelle (1961) reported that 12-15 minutes of exercise, five days per week (25 sessions) appeared to be the minimum time period for lipid improvements to occur.

Table 2.2 **Serum Lipid Changes Following Aerobic Exercise Programmes**

Researcher and year of study	Purpose of study	Age (yrs)	Sex and No. of Subjects	Term of study (weeks)	Amount of exercise (min.)	How often per week (No.)	Results	Comments
Lopez-S, et al. (1974)	Effects of exercise on serum lipids.	22	13 M	7	30	4	Significant decrease in TG; moderate effect on TC.	No control group; dietary intake to maintain caloric balance.
Lipson, et al. (1980)	Changes in plasma lipoproteins with exercise and a iso-weight diet.	19 to 22	5 M 5 F	6	30	5	O_2 increase; TC decreased significantly, NS change in TG and HDL-C.	ISO weight diet; aerobic - treadmill walking; no drop in body weight; thus no HDL increase.
Farell and Barboriak (1980)	Effects of endurance training on HDL-C, TC, TG.	22.3 M & 23.6 F	7 M 9 F	8	30	3 to 4	TC did not change significantly; HDL increased significantly; VO_2 increased significantly; TG decreased significantly.	No control group; % O_2 uptake increased; no dietary control; 2 kg weight loss in females.
Nye, et al. (1981)	Monitor changes in HDL and other lipoproteins	30 to 45	17 M	10	30 to 45	2	HDL did not change; significant change in LDL; TG rose slightly, 4.7% drop in TC.	Continuous activity to music; body weight did not change. Moderate exercise little effect on HDL.
Brownell, et al. (1982)	To evaluate lipid and lipoprotein variations in men and women	35 F & 41.8 M	37 M 24 F	10	15 to 20	3	Men: TC - 44% decrease; HDL-C 115% increase; LDL - 6% decrease; TG - 0.05% decrease.	Aerobic activity - 70% of max heart rate; good decreases in lipids; also a 3 kg drop in body weight.

Campbell (1965) researched the effects of a number of sports activities on serum cholesterol. Freshman enrolled for cross-country running, golf, tennis, gymnastics, weight training and wrestling. Each activity was engaged in three times per week for a 10 week period.

After a retest, the cross-country and tennis groups showed a 11% and 4.2% decrease in cholesterol respectively, whereas the weight training group showed no difference from pre-training levels. The aerobic nature of cross-country running and tennis was reported to be associated with the decreases in cholesterol levels.

Table 2.2 reflects studies concerning lipoprotein changes after a period of aerobic exercise training lasting between 6 to 10 weeks.

In the studies noted in Table 2.2, the mean number of training sessions was 28. In each of these studies an increase in physical working capacity was recorded, reflecting increased oxygen consumption following the exercise programme. It would appear that improvements in oxygen uptake may occur as soon as six weeks of aerobic training. (Lipson et al.,1980).

The lipid changes reported were not however as consistent, with Farrell et al.(1980) and Linder et al. (1983) being the only researchers to report significant increases in HDL levels (12.3% and 9.7% respectively). Triglyceride levels decreased in all studies, with the greatest decrease (27%) reported by Lopez-S et al. (1974). Some changes in cholesterol levels were reported with a significant decrease being documented by Lipson et al.(1980). The changes achieved by Farrell and Barboriak (1980) and Lipson, et al.(1980) were similar, although the

durations of the studies were eight and six weeks respectively. The subjects in the study by Lipson et al. (1980) trained five days per week compared to four by those in the study by Farrell and Barboriak (1980).

In the Nye et al.(1981) and Brownell et al.(1982) studies no significant changes in lipid profiles were reported, although overall percentage improvements were noted. Brownell et al.(1982) and Nye et al.(1981), used older subjects (30-48 years) a factor which may have influenced the findings.

It would seem that the duration of these studies was not as important as the frequency of training per week (5 days). Several other factors may have been implicated in these various changes, including individual differences, different dietary intakes, age and differences in initial lipoprotein levels.

Evidence indicates that regular aerobic exercise has a positive influence on serum lipid levels. The relationship between the intensity of exercise and changes in serum lipids and lipoproteins is not however clear. Tran et al. (1983), in a meta-analysis of the effects of aerobic exercise on blood lipids reported that lower training intensities ($<$ to 60% of maximum heart rate) were associated with more beneficial changes in lipid and lipoproteins. Total triglycerides, LDL, total cholesterol and LDL-C showed larger decreases with training intensities $\geq$ to 60% of maximum heart rate.

Table 2.3 **Effects of Exercise Intensity on Lipids and Lipoprotein Changes**

COMPONENTS		MYHRE, et al. (1981)	MICHIELLI, et al. (1982)	GAESSER AND RICH (1984)
1.	Purpose	Effect of endurance training on varying amounts and intensities of exercise	To assess lipoprotein changes at different exercise intensities.	Effect of low and high intensity aerobic exercise on blood lipids.
2.	Age, Number and Gender of Subjects	X = 26.3 Years 11 men	X = 44 Years 40 men	20 - 30 Years 16 men
3.	Exercise Intensity	70 to 80% of VO_2 max = low intensity, 80% to 90% of VO_2 max = high intensity.	65%, 75% and 85% of predicted maximal HR.	80% - 85% and 45% of VO_2 max.
4.	Duration	32 weeks	12 weeks	18 weeks
5.	No. of exercise and control groups	2 training groups and a control group	3 Exercise groups and control group.	2 Training groups and no control group.
6.	Subjects Fitness	Fit and sedentary subjects.	Sedentary subjects.	Sedentary subjects.
7.	Fitness Changes	Not stated.	VO_2 max increased significantly ($P \leq 0.01$) in all experimental groups.	VO_2 max increased in both groups ($P \leq 0.05$); 1.5 kg fat loss.
8.	Lipid Changes	HDL-C increased significantly during low intensity as well as shorter duration, greater intensity.	Statistical significance in HDL-C ($P \leq 0.01$) in the 75% and 85%, but not in 65% group. However, 65% showed significant decreased TC and VLDL ($P \leq 0.05$).	No statistical significance found in TC, TG, HDL-C or LDL-C in high and low intensity, but overall positive changes.
9.	Conclusion	That elevated HDL-C levels are associated with high levels of physical activity and are related to both intensity and duration.	Aerobic training favourably alters blood lipids to a greater extent at higher training intensities (75% of max HR).	Min. exercise threshold of 45% needed for increased aerobic capacity. Pre-training levels are of importance in young men before changes HDL-C occur.

A review of three studies relating to intensity of exercise is presented in Table 2.3, indicating the structure and results of these studies. When examining the studies cited in Table 2.3, there is no clarity regarding the intensity of exercise required to reduce cardiovascular risk. Michielli et al.(1982) however reported significant positive changes in HDL-C levels at intensities of 75% and 85% of maximum heart rate. Gaesser and Rich (1984) found no statistically significant improvement at the same work intensity, but indicated that the subjects in the study had normal pre-training lipid levels, which appeared to have affected the overall lipid and lipoprotein changes.

In the Myhre et al.(1981) study six conditioned cross-country skiers were compared to five sedentary men. The results indicated that exercise of long duration and intensity (70-80% of maximum heart rate) increased HDL-C significantly. Myhre et al. (1981) classified 70-80% of maximum heart rate as low intensity which could be regarded as medium to high intensity in other studies (Michielli et al.,1982, and Gaesser and Rich, 1984).

It would appear that initial pre-training levels of serum lipids and lipoproteins, rather than exercise intensity, may be responsible for any changes that may result from an exercise programme. The design limitations of the Myhre's et al.(1981) study indicated that the intensity of the training programme was not well controlled. There were also considerable individual variations in serum cholesterol levels, and the sample size was small.

In a well designed study, Whaley et al.(1992) investigated the changes in total cholesterol concentration following aerobic exercise. The subjects comprised 215 women and 327 men

aged 20 to 75, the intervention programme consisted of individualized aerobic exercise at approximately 75% to 85% of maximal heart rate for 30 to 50 minutes four times per week for 16 weeks. After adjustments for pre-training, total cholesterol, age, changes in body weight, body fat and VO_2 max were significant first order predictors of change in total cholesterol for the total cohort. Gender-specific regression analysis revealed differences between men and women regarding predictors of change in total cholesterol. After adjustments for the pretraining total cholesterol, change in body weight and body fat were significant predictors of total cholesterol change for men, but only age ($r = 0.135$) served as a predictor of change in total cholesterol among women.(Whaley, et al.,1992). The level of total cholesterol prior to training appeared therefore to have an influence on the degree to which total cholesterol changed during training.

These results confirm the importance of the pre-training total cholesterol levels in assessing the potential for a favourable change in total cholesterol subsequent to aerobic training. They also may explain the equivocal results found in previous studies (Tran et al.,1983). This may have been due in part, to the use of samples with normal pretraining total cholesterol or of samples exhibiting a wide range of pretraining total cholesterol levels.

Tran et al. (1983) concluded that one of the most important findings of their meta-analysis study was that the pre-training levels of total cholesterol, total triglycerides, HDL-C and total cholesterol/HDL-C were strongly associated with any changes which occurred as a result of training.

Further, it was reported that lower initial levels of HDL-C resulted in greater increases following exercise. Only LDL-C deviated from this pattern of strong correlations.

Many prospective and longitudinal studies analysed in reviews (Dufaux et al.,1982 and Tran et al., 1983), have reflected positive changes in serum lipid and lipoprotein levels following exercise. Other studies have shown no improvements. When examining these studies, many design limitations were apparent which could account for contradictary results. Such design limitations included small sample groups of less than 10 subjects, the absence of sedentary control groups and the lack of random sampling. Seasonal variations may have effected results, giving rise to wide variations in lipid and lipoprotein concentrations. Research is needed to determine the extent to which changes resulting from training may be due to cumulative delayed reactions after single bouts of exercise (Dufaux et al.,1982). Coupled with the design limitations there are many other variables which may confound the effects of exercise.

These include alcohol intake, smoking, body weight changes, pretraining lipid and lipoprotein levels, dietary changes, gender and hormones (Dufaux et al.,1982 and Tran et al.,1983). Control of these variables is difficult and at times even impossible. Randomized studies do however take a number of these factors into account.

Wood et al. (1983) employed randomized group selection and observed changes in lipoprotein levels during and following aerobic exercise. Eighty one healthy, sedentary men aged between 30 to 55 years were randomly assigned to a supervised running group (N =

48) and to a sedentary control group (N = 33). They participated in a one-year trial with measurements of plasma lipoproteins, fitness and percent body fat being made at three monthly intervals.

Food records and running diaries were kept and verified by trained staff. The subjects exercised progressively on 3-5 days per week, until they could run without needing to stop. The exercise intensity was maintained between 70% to 85% of maximum heart rate.

The results indicated that, after one year, the experimental subjects became leaner and fitter than those in the control group. Lipoprotein concentration changes were greater among the runners. The 25 men who averaged at least 12,9 kilometres per week of running increased their plasma HDL-C significantly.

Significant correlations were established between the distance run per week and HDL-C and LDL-C . The study suggested a threshold of 12.9 kilometres per week in a one year running programme in order to experience beneficial lipoprotein changes (Wood et al.,1983). By increasing the number of kilometres run per week, increases in plasma HDL-C and fitness, and decreases in the LDL-C levels and percent body fat could be effected. Decreases in triglycerides and VLDL-C were not however statistically significant. The author reported that a large proportion of the exercise group (75%) failed to achieve the level of exercise apparently required to promote changes in HDL-C. This may account for the failure of the study to establish significant differences between exercise and control groups concerning lipoprotein changes over a one year period. Elimination of those subjects who failed to run

12.9 kilometres per week, produced significant differences in HDL-C levels over a one year period. This study, although it attempted to control most of the confounding factors, may be limited by the fact that the subjects with higher initial HDL-C and lower triglyceride levels were more easily persuaded to run further (self-selection). It also did not control other confounding factors, such as smoking, alcohol intake and dietary changes.

2.2.3 **Meta-Analysis Studies**

In recent years, several attempts have been made to address the problems which arise in literature reviews with respect to the many confounding variables which may have influenced the results of studies in this area of research. A technique called meta-analysis was developed, which essentially is the statistical analysis of the findings of a number of empirical studies. Meta-analysis is quantitative, using numbers and statistical methods for organizing and extracting information from large masses of data. This technique also applies formal research methods to the characteristics and findings of research studies. This includes problem identification, hypothesis formulation, definition and measurement of constructs, variables, sampling and data analysis (Tran et al.,1983).

Two meta-analysis studies related to the effects of exercise on blood lipids, lipoproteins and body weight have been reported to date. (Tran et al.,1983 and Tran and Weltman, 1985).

In the first meta-analysis study, Tran et al.(1983) compared the effects of exercise on blood lipids and lipoproteins. The results of 66 training studies, spanning a 26-year period and representing 2925 subjects, were analyzed. The authors concluded that overall physical

training seemed to produce beneficial changes in blood lipids and lipoproteins. The lower the initial levels of lipids the less change was likely to occur following exercise.

The study provided no clear indication of the time required to achieve this end. Lower training intensities (60% of maximum heart rate) were associated with more beneficial changes in lipids and lipoproteins. This observed relationship however was valid only at intensities equal to and greater than 60% of maximum heart rate. Changes in total cholesterol were most closely associated with high training intensity while total triglycerides and LDL-C exhibited similar trends with larger decreases being associated with lower exercise intensities.

Larger increases in HDL-C were associated with lower training intensities (Tran et al.,1983). Initial levels of VO_2 max seemed to influence how levels of serum cholesterol changed with training.

Subjects with higher VO_2 max values also had lower total serum cholesterol levels. This relationship was not observed for other lipids and lipoproteins. In addition, a lower initial VO_2 max was associated with larger decreases in the lipids and lipoproteins and increases in HDL-C. This correlation was strongest for total cholesterol.

When overall changes were analysed using meta-analysis, as indicated by Tran et al.(1983), the following mean changes were found to occur in lipoproteins and serum lipids as a result

of the exercise intervention programmes. These changes were:

a) Total cholesterol decreased by .259 mmol/l ($p < 0,01$).

b) Total triglycerides decreased by .169 mmol/l ($P < 0.01$).

c) HDL-C increased by .031 mmol/l (NS).

d) LDL-C decreased by .132 mmol/l ($P < 0.05$).

e) Total cholesterol/HDL-C ratio decreased by 0.48 ($P < 0.01$).

None of the changes for the control groups was significant.

Taken as a whole, this statistical meta-analysis indicated that exercise resulted in positive mean changes in lipid and lipoprotein levels for all experimental groups in comparison to the control groups. This result is consistent with the impressions of authors in previous reviews of literature (Wood and Haskell,1979 and Dufaux et al.,1982).

In the second meta-analysis study by Tran and Weltman (1985), the effects of exercise on serum lipids and lipoprotein levels associated with changes in body weight were analysed. Ninety five studies conducted between September 1955 and October 1983 were analyzed. These studies all measured changes in serum lipid and lipoprotein levels in response to exercise and changes in body weight.

The data obtained from these studies were divided into:

those subjects who gained weight,

those whose weight was maintained and

those who lost weight during the exercise programme.

The results showed significant relationships between body weight changes which occurred during exercise and lipid and lipoprotein level changes. Decreases in cholesterol, LDL-C, triglycerides and cholesterol/HDL-C levels correlated significantly ($P \leq 0.05$) with decreases in body weight. Tran and Weltman (1985) reported further that since changes in body weight effected serum lipid and lipoprotein levels, body weight changes that occurred during an exercise programme may obscure the actual effects of exercise on changes in lipid and lipoprotein levels. Their findings were as follows:

Where body weight did not change, cholesterol and LDL-C levels decreased significantly, (0.189 mmol/l and 0.085 mmol/l respectively).

Where body weight decreased, cholesterol and LDL-C levels also decreased significantly, (0.341 mmol/l and 0.287 mmol/l respectively).

Where body weight increased, cholesterol and LDL-C levels increased by 0.075 mmol/l and 0.078 mmol/l respectively.

In conclusion these results suggest that reductions in cholesterol and LDL-C levels were greatest when exercise and training were combined with body weight reductions.

In summary, the two meta-analysis studies reviewed (Tran et al., 1983 and Tran and Weltman, 1985) demonstrated that aerobic exercise does have a general beneficial effect on serum lipid and lipoprotein levels. However, when conclusions are drawn from such studies many other factors may effect the results. Some of these interceding factors are: initial levels of serum lipid and lipoprotein levels; duration and intensity of exercise programmes; body weight changes; maximum oxygen uptake; and individual differences.

2.2.4 **Summary**

Several studies support the contention that increased duration of exercise affects lipoproteins positively (Lehtonen and Viikari, 1978; Tran et al.,1983 and Wood et al.,1983). Wood et al.(1983) concluded that men who ran 12.9 Kms per week for a year increased their HDL-C levels significantly ($P \leq 0.045$). It would appear that a threshold in duration of exercise is required before beneficial lipoprotein changes can occur.

Initial levels of lipoproteins may also effect the outcome of results concerning exercise as an intervention strategy. Higher initial values of total cholesterol triglycerides and total cholesterol/HDL-C ratios resulted in greater decreases after exercise. Lower HDL-C levels resulted in greater increases after exercise (Tran et al.,1983 and Whaley et al.,1992). It may be postulated that in studies where no significant changes were noted in lipoprotein concentrations after exercise, initial lipoprotein levels were normal or close to normal

(Whaley et al.,1992). Increased body weight correlated significantly ($P \leq 0.01$) with higher total cholesterol, total triglycerides and total cholesterol/HDL-C ratio $r = 0.61$, $r = 0.32$ and $r = 0.44$ respectively) (Tran et al.1983 and Tran and Weltman, 1985). The results suggest when using exercise as an intervention strategy, a decrease in body weight results in a greater decrease in both total cholesterol and LDL-C (Tran and Weltman, 1985). It would seem that the most effective way of altering cholesterol, triglycerides and LDL-C would be to combine exercise with reductions in body weight.

In almost all studies of the effects of exercise on lipoproteins, significant increases in oxygen uptake were recorded after a minimum period of eight weeks of training (Farrel and Barboriak, 1980 and Lipson et al.,1980). In many studies however, no relationship was established between improved fitness, as reflected by increased oxygen uptake and positive changes in lipoprotein profiles. Tran and co-workers (1983), reported that initial VO_2 max levels seemed to influence training. Individuals with larger VO_2 max levels also had lower total serum cholesterol levels ($r=-0.43$, $P \leq 0.01$). This however, was not true for the other lipids and lipoproteins.

It would appear that there are many confounding factors that may influence the final effect of exercise intervention on lipoprotein changes. Some of these confounding variables may be the duration of exercise, initial pre-training lipoprotein levels, increased body weight and initial level of fitness. These variables may interact singly or in concert, with either positive or negative effects.

2.3 DIET AND CHD

Data establishing the link between diet and heart disease emerged in publications which appeared in the early nineteen thirties. Byrne (1991) reports that Nikoloi Anitsckow (1933) was the first to trace the link between cholesterol and dietary factors using rabbits. It was only after World War II, when the industrialized nations became aware of the epidemic increase in CHD, that a surge in research occurred concerning dietary factors and the relationship to CHD (Byrne, 1991). Keys (1970), an epidemiologist at the University of Minnesota, and co-workers in several other countries documented fat intakes, serum cholesterol and CHD statistics of various populations during the early nineteen sixties. This was the first concerted effort to establish a relationship between diet and CHD through population studies. This study was known as the "Seven Country Study" and included Finland, Greece, Japan, Italy, Holland, United States and Yugoslavia. Dietary intakes, blood cholesterol levels and death rates from CHD were recorded in all these populations. This pioneering study showed that in populations where serum cholesterol levels were 6.2 to 7.2 mmol/l the incidence of CHD was 10 times higher than the average of 4.1 mmol/l. Hegsted et al.(1965), Keys (1970), Stamler (1982), Kushi et al.(1985) and Skekelle and Stamler (1989) also identified relationships between elevated cholesterol levels, high intakes of saturated fats and the increased incidence of CHD. Some investigators assumed that hereditary factors were responsible for the explained differences between various nation's rates of CHD. When diets differed within a genetically homogenous population however, atherosclerosis varied as well (Byrne, 1991).

It was also reported that vegetarians in the United States consumed much less saturated fat and had significantly lower levels of LDL and triglycerides (Sacks et al., 1975; Burslem et al.,1978; Zetts et al.,1985). Heredity therefore could not explain why the heart attack rates of vegetarians were lower than those of meat eaters.

In a review article, Rossouw (1983) tabulated the evidence relating diet to heart disease. The contents of this evidence appear in Table 2.4

Table 2.4 Diet and Heart Disease - The Evidence

(Rossouw , 1983)

FOR	AGAINST
1. Interpopulation associations between consumption of cholesterol, total and saturated fat and serum cholesterol	1. Association do not prove causality
2. Interpopulation association between dietary lipids and CHD.	2. Within population groups the associations between diet and serum lipids are frequently unimpressive.
3. Inter and Intrapopulation association of serum cholesterol and certain lipoprotein with CHD.	3. Dietary intervention trials have generally been inconclusive.
4. Metabolic studies in animals and humans confirm effect of dietary effect on serum cholesterol.	4. Results in animal experiments cannot be extrapolated to humans.
5. In animals atherosclerosis can be induced by high lipid diets and regressed by low lipid diets.	
6. Arterial lesions in animals and humans with certain hyperlipaemias contain excessive cholesterol.	
7. Certain genetic hyperlipaemias established in early life slow premature CHD.	

In summary it would appear that Rossouw's (1983) presentation of the evidence may reflect inherent limitations in many of the study designs.

Mensink and Katan (1989) investigated the effects of a diet enriched with monounsaturated or polyunsaturated fatty acids on levels of low and high density lipoprotein cholesterol in healthy women and men. Thirty one women and 27 men were placed on a normal mixed saturated fat diet (19.3% of daily intake from saturated fat, 11,5% from monounsaturated fat and 4,6% from polyunsaturated fat) for 17 days. For the next 36 days, they received a mixed diet with the same total fat content, but enriched with olive or sunflower oil. The results indicated that a mixed diet, rich in monounsaturated fat, was as effective as a diet rich in polyunsaturated fat in lowering LDL cholesterol. Both diets lowered the level of the HDL cholesterol slightly in men but not in women. The results of this study, however, did not agree with those of earlier studies by Hegsted et al.(1965) and Keys et al.(1966), in that polyunsaturated fats are superior to monounsaturated fats in lowering total cholesterol. More research in this respect needs to be embarked upon before definite conclusions can be drawn.

In 1990 Mensink and Katan reported the effects of dietary trans fatty acids on high-density and low density lipoprotein cholesterol levels in healthy subjects. They concluded that the effect of trans fatty acids on the serum lipoprotein profile is at least as unfavourable as that of saturated fatty acids, as they not only raise LDL cholesterol levels but also lower HDL cholesterol levels. It would appear that persons at high risk of CHD should avoid a high intake of trans fatty acids.

More recently McMurray et al.(1991) investigated the effects of an affluent western diet on Tarahumara Indians and the effect on lipid, lipoprotein levels and body weight. The risk of coronary heart disease increased dramatically following the hypercaloric high saturated fat

diet in the population studied. There were large increases in plasma lipid, lipoprotein levels and body weight mass. The subjects gained 3.7 kg during the 21 to 36 days of the study.

In response to the study by McMurray et al.(1991), Mann (1992) reported that if weight loss occurred, the serum lipid levels would fall. The critical factor was sufficient activity to burn off excess calories and to avoid weight gain. Besides the excess calories and weight gain of the experimental population, the most important aspect was the composition of the diet. A diet high in saturated fats,with good weight control via exercise may still not improve the lipid profile, because of the increased cholesterol intake through diet.

Wood et al.(1991) reported on the effects on plasma lipoproteins of a prudent weight reducing diet, with or without exercise. The results indicated that regular exercise, in overweight men and women, further reduced the plasma lipoprotein levels resulting from the adoption of a low saturated, low cholesterol diet. The study used three groups, namely a control group, a low calorie diet group and a low calorie and exercise group. Weight loss from the low calorie diet alone did not significantly change HDL cholesterol levels in either men or women. Plasma HDL increased significantly in men who exercised and dieted. Men who dieted without exercise and men who acted as controls did not register such increases. HDL cholesterol levels remained similar in the women who exercised and dieted, but were higher than the controls. From this it appeared that moderate exercise coupled with a low calorie diet was most effective in reducing selected risk factors in CHD.

Research supports the contention that dietary manipulation modifies plasma lipoprotein composition. The decreased cholesterol intake from saturated fats, and the increased percentage of polyunsaturated fats seem to be the prudent dietary advice for the prevention or retardation of CHD. (Hegsted et al., 1965; Keys et al., 1966; Rossouw, 1983; Hartung, et al., 1983; Mensink and Katan, 1989 and Ulbricht and Southgate, 1991). Hartung et al.(1983) and Rossouw (1983) concurred after reviewing dietary interactions and plasma lipids.

In summary, research indicates the following:

(a) High cholesterol diet tends to increase total cholesterol, HDL-C and LDL-C (Barnard et al.,1992).

(b) Diets containing less than 30% fat and /or those high in polyunsaturated fat may decrease total cholesterol, HDL-C and LDL-C, but do not seem to affect the total cholesterol/HDL-C or HDL-C/LDL-C ratios (Keys, 1970).

(c) Vegetarian diets decrease HDL-C and LDL-C,but it is not known if the results are due to carbohydrate alterations or reductions in fat intake (Ulbricht and Southgate, 1991 and Ornish, et al.,1990 and Ornish et al.,1993).

(d) Aerobic exercise coupled with a prudent low fat diet increases HDL-C and reduces LDL-C (Wood et al.,1991).

(e) A reduction in cholesterol intake below 300 mgs per day, an increase in fibre intake and a reduction in salt consumption to less than 5g/day, may retard or prevent CHD. (Mensink and Katan, 1989).

2.4 MIGRANT POPULATION STUDIES AND CHD

Persons who migrate from their homeland to western countries, in particular, tend to adopt the lifestyle and eating patterns of their new country.

Migrant population studies cast further doubt on a purely genetic explanation for CHD among particular populations. Classic studies by Kato et al. (1973) on Japanese who emigrated from Japan to Hawaii and California indicated that these people quickly attained the same rates of CHD as the locals in their adopted homelands. Similar results were reported on Indians from India who emigrated to Trinidad (Miller et al.,1982 and 1984), Indians who moved to Birmingham England (Lowry et al.,1984) and indentured Indian labourers who moved from India to South Africa (Seedat, 1982). Local populations demonstrated high cholesterol levels coupled with high saturated fat intake. This increase in CHD in migrant populations took only one or two generations to develop, much too short a time for genetics to play a part. However, it is important to note that these studies do not prove direct cause and effect relationships between CHD and dietary fats.

Landmark studies by Robertson et al.(1977) and Kagan et al. (1974) compared men in Japan to those who had emigrated to Honolulu and San-Francisco, with respect to their diets, blood cholesterol and subsequent mortality rates over many years. The traditional Japanese diet contained more unsaturated fats and more fish compared to high saturated fat content of the American diet.

Blood cholesterol levels in Japan averaged 4.7 mmol/l compared to the migrant Japanese men, (Honolulu and San Francisco) whose levels averaged 5,6 and 5,9 mmol/l respectively. Japanese men aged 55-59 years living in California recorded heart attack rates almost three and half times those of men living in Japan (Worth et al., 1975). Japanese men registered saturated fat intake and cholesterol levels of 7% and 4.68 mmol/l respectively compared to Hawaiian migrants who registered 23% and 5.64 mmol/l respectively, and Californian migrants who recorded 26% and 5.89 mmol/l (Marmot et al.,1975). The results of this study further emphasize the importance of dietary habits in the development of CHD.

It would appear from studies by Kato (1973), Kagan et al., (1974) and Robertson et al.(1977) that lifestyle alterations, including changes in dietary habits, may be implicated in the development of CHD in the migrant populations studied.

2.5 GENERAL CONCLUSIONS

Substantial progress has been made during the past thirty years in the accumulation of scientific evidence indicating the relationship between exercise and health. Most recently the American Heart Association reported that ongoing, regular aerobic exercise increases physical working capacity and plays a role in both primary and secondary prevention of cardiovascular disease (American Heart Foundation, Position Statement : 1992). This was a landmark consensus statement as it listed "physical inactivity" as the fourth major risk factor in the development of CHD. More importantly, it was the first definitive statement on the effects of exercise on CHD, to be published by a medical association.

After reviewing the literature it appears that regular, progressive aerobic exercise influences human health, directly or indirectly. Such exercise controls blood lipid and lipoprotein concentrations (Lehtonen and Viikari, 1978; Huttunen et al.,1979 and Hartung et al.,1983) and improves the physiological functioning of the human body (Astrand and Radahl, 1970; Gordon and Gibbons, 1990).

There is ample evidence that aerobic exercise may positively effect selected CHD risk factors, which may delay or retard the development of heart disease.

This study is concerned with the effects of such aerobic exercise on a selected group of sedentary male subjects from the Indian community of Durban, South Africa. With sufficient evidence to indicate that this community is at risk from CHD, the effects of an exercise programme is of particular significance. Such data will contribute to the understanding of the role of physical activity in modern life, with particular reference to the prevention of CHD.

CHAPTER THREE

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3. METHODOLOGY

This chapter describes the selection of subjects, the experimental design, and includes a discussion of the variables affecting the study.It further describes the testing procedures and instruments used in the data collection process. A part of this chapter focuses on the methods used in the blood analysis, which was performed under the auspices of the Department of Chemical Pathology at the University of Natal. The study design was accepted by the Ethics committee of the University of Durban-Westville.

3.1 EXPERIMENTAL DESIGN

3.1.1 Recruitment and Selection of Subjects

The initial recruitment of subjects was from Indian staff members at the University of Durban-Westville. The nature of the employee force at UDW was predominately Indian, and involved in work which was mainly sedentary in nature, and fell within the age-range required in this study. The staff represented a wide socio-economic spectrum of the Indian population in Natal, including maintenance, and administrative staff as well as academics.

The university provided a convenient location for the implementation of the project. Facilities for evaluation and exercise were available using the facilities of the Department of Human Movement Studies and the Sport Bureau.

The recruitment and selection of subjects was undertaken in three stages:

a) Letters of invitation were mailed to all Indian staff members soliciting their participation in the project. (Appendix 1)

b) A preliminary selection meeting of all respondents followed, at which time a medical history questionnaire and lifestyle evaluation inventory was completed by each subject. Smoking, drinking and exercise habits and a brief family history of hypokinetic disease were also recorded. (Appendix 2)

c) A final meeting was held with those subjects selected through the preliminary screening procedure, at which time details of the experimental design were provided.

All subjects read and indicated their understanding of a document explaining the research project. This document outlined the testing programme, the length and duration of the exercise programme, the possible risks and discomforts that might be experienced during testing and the possible benefits of the programme. The freedom of a subject to withdraw consent and to discontinue participation was also emphasized. The subjects were assured that all the data gathered would be treated as confidential. Thereafter all subjects provided their written consent to participate in the programme.

Forty eight subjects responded to the call for volunteers. After the preliminary analysis of the subjects' health and history of physical activity, 44 were selected, each of whom

conformed to the following criteria:

a) They were between 25 and 55 years of age;

b) They were sedentary, healthy and normolipidaemic, and were free of overt chronic disease, especially coronary heart disease;

c) None had participated in regular exercise for at least 12 weeks prior to the study;

d) They all agreed to attend three supervised 30 minutes aerobic exercise sessions per week for 15 consecutive weeks. After the briefing, three subjects withdrew on realising that they would encounter time constraints. Each of the remaining 41 subjects was assigned to one of three groups, matched to body mass index.

These groups were:

Exercise only (E)	- 15 subjects
Exercise and diet (ED)	- 14 subjects
Control (C)	- 12 subjects

The control subjects were selected using the same criteria, but on the understanding that they would not change their living habits during the course of the experimental period and would not participate in any supervised or personal exercise for the period of the study.

3.1.2 **Dietary History of Subjects**

Each subject was personally interviewed by a registered dietician in order to compile a seven day diet history and to assess the average daily nutrient intake. These interviews were

repeated at the end of the 15 week period.

The exercise group and the control group were asked not to alter their pattern of eating, during the intervention period. The exercise and diet group was prescribed a prudent low fat diet as recommended by the South African Heart Foundation. Each subject in the exercise and diet group was counselled regarding dietary principles (see Appendix 3).

The dietician communicated with each subject in this group at least three times during the programme in order to check on the subject's diet. Those in the exercise and diet group were free to telephone the dietician, in order to discuss any aspect of their diet. A recommended meal plan was also suggested and given to each subject (see Appendix 3). The total energy intake of the diet was approximately 1500 Kcal, with the content of the macronutrients being as follows:

240g Carbohydrate	(59,8% of total energy)
68g Protein	(19,9% of total energy)
31g Fat	(20,3% of total energy)

More than 50% of the fats in the meal plan were polyunsaturated and monosaturated fats. For the analysis of data, the nutrients were computer coded and entered into the "The Floro Diet Data Program", which enabled the researcher to analyse the nutrient content of each subject's diet.

3.1.3 Intervention Design

The design of the intervention programmes is indicated by means of Figure 3.1.

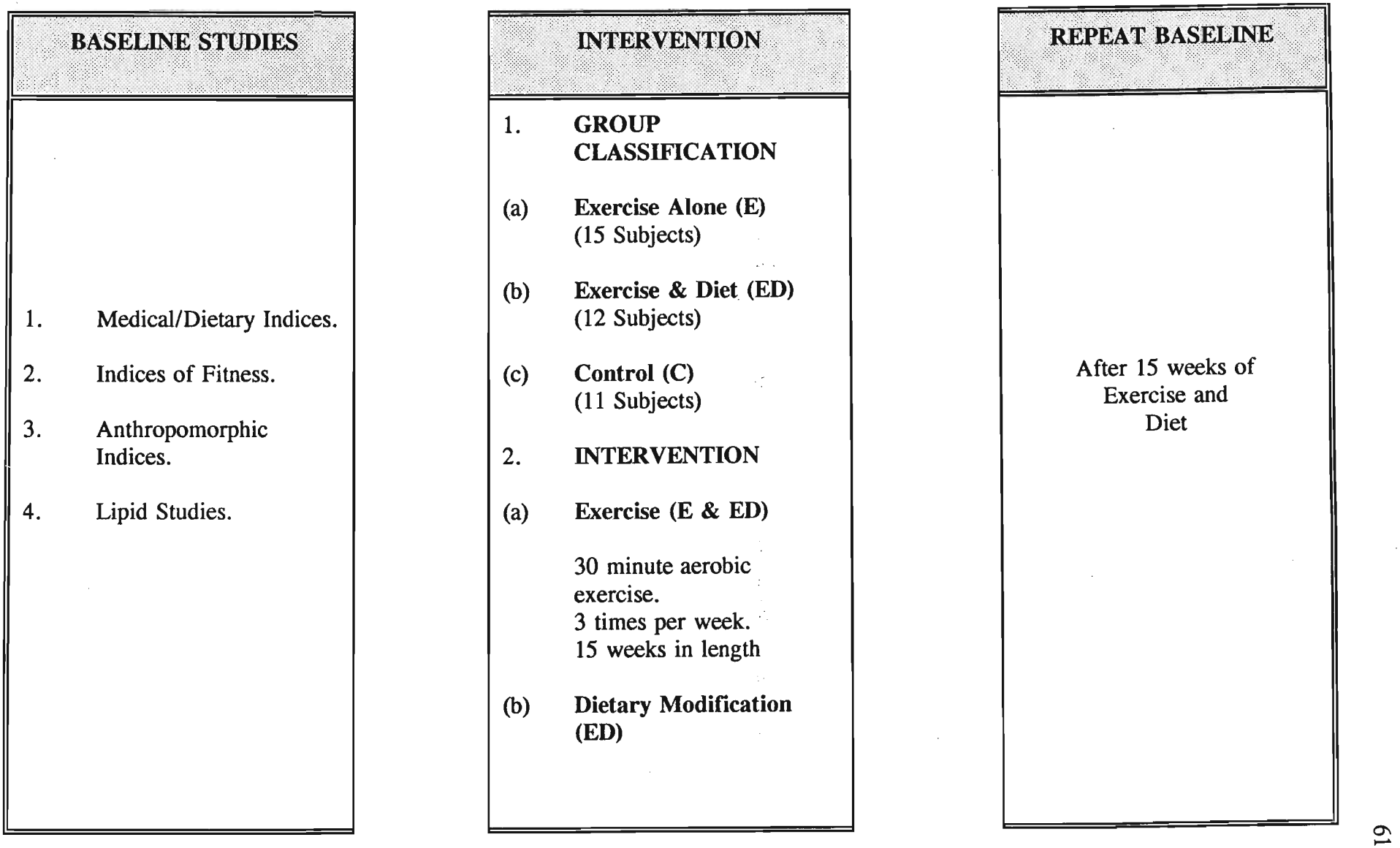
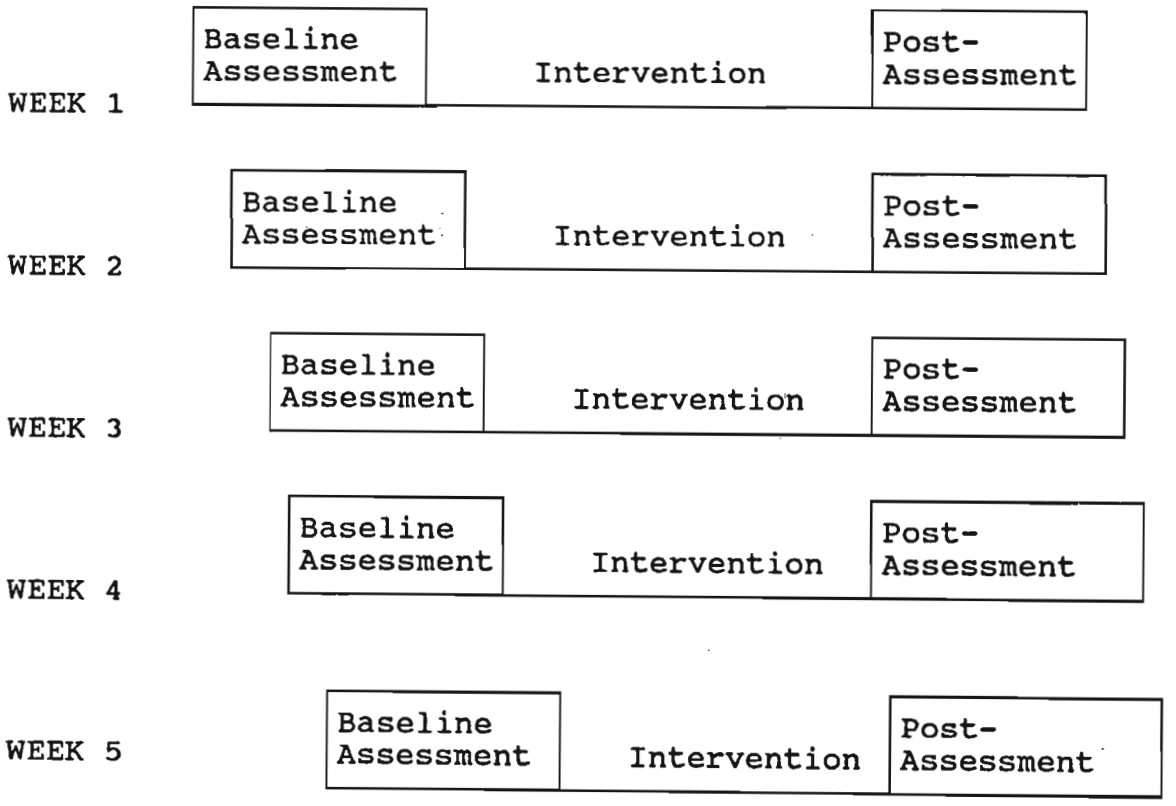


Figure 3.1 **Flow Chart Showing the Intervention Design**

3.1.4 Evaluation Procedures

Baseline and post evaluation investigations were staggered for practical reasons with approximately eight subjects being evaluated per week. The intervention programme for each subject was started immediately following the baseline study. The staggered method is illustrated below :



The programme for each group of eight subjects was as follows:

Monday and Tuesday : Fitness assessment

Wednesday and Saturday : Blood collection

Monday following : Exercise Programme

The entire procedure was repeated five times in both the pre-intervention and post-intervention phases in order to accommodate all the experimental and control subjects. This design was used in order to accommodate the fitness and blood testing sessions.

Three trained post-graduate assistants and the researcher supervised each of these groups as they entered the exercise programme. The exercise programme and intensity was identical for each group. By the eighth week all subjects exercised together.

3.1.5 **Exercise Implementation**

3.1.5.1 **Principles of exercise**

The exercise programme was based on the principles of exercise prescription published by the American College of Sports Medicine (1990). The intensity of the exercise was determined for each subject using maximum heart rate as the criterion. Maximum heart rate was determined through treadmill testing. Training was maintained at 45-70% of maximal heart rate, using a heart rate monitor to assess the intensity of each subject's working heart rate.

Subjects exercised during their lunch times on Mondays, Wednesdays and Fridays between 13h00 and 13h30, for 15 consecutive weeks.

Each exercise session was of 30 minutes duration and was continuous. Increases in exercise intensity for individual participants were prescribed to accomodate the conditioning effect which was evident after 6 weeks of exercise. The conditioning effect permitted certain individuals to cover more distance in 30 minutes, at the same intensity (45% to 70% of maximum heart rate).

3.1.5.2 **Aerobic exercise intervention**

During the first seven weeks the programme was conducted in the gymnasium of the Department of Human Movement Studies.Thereafter, the subjects ran on the University's 400 metre athletic track. From the first to the third week, the participants walked briskly around the gymnasium for 20 minutes, followed by sit-ups, press-ups, burpees and flexibility exercises for five minutes. A final cool down phase of five minutes concluded each session.

During weeks four to six, a three minute warm-up of brisk walking around the gymnasium was followed by 20 minutes of walking and jogging at a moderate pace of between 45%-70% of maximum heart rate. The exercise session was concluded with eight minutes of muscular endurance and flexibility exercises, and followed by a cool down phase.

From the seventh to the tenth week, after a warm-up period, the subjects jogged for 25 minutes non-stop on the track followed by muscular endurance and flexibility exercises,

ending with a cool-down period.

During the final five weeks, the subjects jogged at their own individual pace at the required intensity of between 45-70% of maximum heart rate for 30 minutes around the University track. At this point in the programme, muscular endurance exercises became optional, although almost all the subjects ended their training sessions with sit-ups, press-ups and flexibility exercises. The subjects were able to cover between 4000 and 5200 metres in 30 minutes of running by the end of the fifteenth week.

3.2 THE MEASUREMENT OF INDICES OF FITNESS

The selected fitness tests were reliable and valid for purposes of this study. Standardized methods of testing were employed conforming to criteria set by the American College of Sports Medicine (1990). Table 3.1 indicates the tests selected for the evaluation of the fitness indices. The subjects were briefed before they reported to the laboratory. They were informed of the objective of the testing programme and the procedures to be followed while being tested. All subjects were familarized with the equipment used for testing.

Table 3.1 **The Fitness Indices Selected for Evaluation**

COMPONENT	TEST-SELECTED
1. **Anthropomorphic Indices**	* Height * Mass * Percent Body Fat * Lean Body Mass * Body Mass Index
2. **Muscular Endurance tests:** 2.1 Abdominal Endurance 2.2 Upper Body Endurance	* 1 minute sit-up * 1 minute push-ups
3. **Cardio-Respiratory measures:**	* Maximum oxygen uptake * Ventilation * Heart Rates
4. **Flexibility of lower back and hamstring muscles**	* Sit and reach test

3.2.1 Anthropomorphic Indices

A brief description of the methods used in the evaluation of the anthropomorphic characteristics of the subjects, is presented.

3.2.1.1 Measurement of height and weight

Each subject was weighed in his shorts without wearing shoes, using a Detecto Digital Scale and Stadiometer. Weight was recorded to the nearest 0.2 kg while height was measured to the nearest 0.5 cm.

3.2.1.2 Determination of percent body fat

The rationale for the measurement of subcutaneous skinfold fat was based on the assumption that approximately half of the body's total fat content is located in fat deposits directly beneath the skin, this being closely related to total fat. (McArdle et al.,1981).

A Lange skinfold caliper was used, with a constant pressure of approximately 10 gm/sq.mm and with an accuracy of $\pm$ 1 mm. The designated body sites as outlined by the Jackson and Pollock (1978) protocol was chosen to measure skinfold thicknesses. The fold of subcutaneous fat and skin was lifted away from the underlying muscular tissue, using a firm grasp of the fat fold between the thumb and forefinger. A constant tension was exerted by the pincer arms of the calipers at the point of contact with the fat fold.

The thickness of the skin and subcutaneous tissue was then read directly from the caliper dial. All measures were taken on the right hand side of the body. The calipers were held at

the base of fat fold at about 1-2 cm below the fingers.

The skinfold sites chosen for this study included the chest, abdomen, and front thigh. The anatomical landmarks used for the skinfold measurements were those developed by Jackson and Pollock (1978) and are reflected in Figure 3.2.

The chest skinfold was measured over the lateral border of the pectoralis muscle, halfway between the nipple and the armpit.

The vertical fold at least 2.5 cm to the right of the umbilicus was used for the abdominal skinfold.

The thigh skinfold measurement was taken at the vertical fold, at the midline of the thigh, two-thirds of the distance between the knee-cap and the hip.

The skinfolds were measured in millimetres and the average score of three readings was taken. The generalized equations for predicting body density and fat were obtained using the Jackson and Pollock (1978) formula, yielding percent body fat.

3.2.1.3 **Calculation of lean body mass**

Lean body mass was calculated by subtracting the weight of fat from the body weight. To

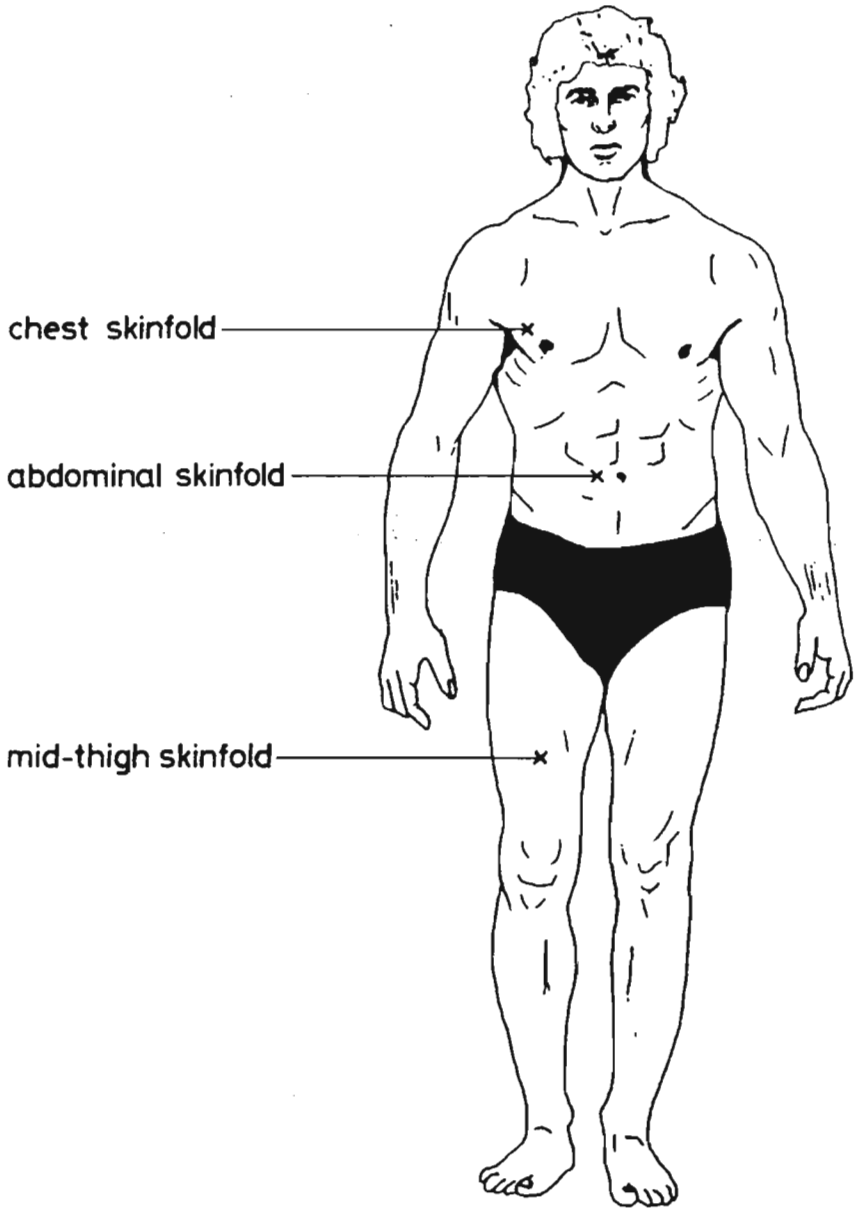


Figure 3.2 : LOCATION OF THE SKINFOLD SITES

calculate fat weight the following formula of McArdle et al.(1981) was used:

Fat weight = (percent fat ÷ 100) x body weight

To compute lean body mass the following equation was used:

Lean Body weight = Body weight - fat weight

3.2.1.4 **The calculation of body mass index**

Body Mass Index (BMI) was computed using the following formula:

$$\text{BMI } (Kg/m^2) = \frac{\text{Weight Kg}}{\text{Height } (m^2)} \quad \text{(Bray, 1985)}$$

3.2.2. **Muscular Endurance Tests**

Muscular endurance of the upper body and arms as well as the abdominal muscles were the components measured. Muscular endurance may be defined as the ability of muscle groups to exert submaximal force repeatedly, for extended periods of time. The push-up and sit-up tests were selected to measure this component.

3.2.2.1 **Push-ups (Coopoo, 1989)**

The hands were placed on a gymnastics mat, shoulder width apart and with the arms and back straight. The feet were slightly apart, with the body weight being supported on the hands and toes. The subject then bent his elbows with the back remaining straight, moving in a downward motion, until the chin and chest touched the floor. Without resting, the subject then straightened his elbows to return to the starting position. The back was not permitted to bend throughout the movement (Figure 3.3). The maximum number of push-

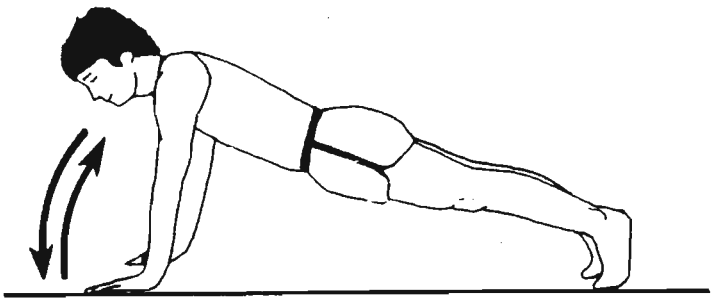


Figure 3.3 : FULL-LENGTH PUSH-UP

ups completed in one minute determined the score obtained.

3.2.2.2 **Sit-ups (Coopoo, 1978)**

The subject started in a supine position, with knees flexed as close to the buttocks as possible, with both feet flat on the ground. The hands were clasped behind the neck as shown in Figure 3.4. A partner held the ankles of the subject firmly on the ground. The subject then moved into a sitting position by raising his head, shoulders and back progressively until his lower back was perpendicular to the floor, and then returned to the starting position. This was counted as one complete sit-up (Figure 3.4). The subject repeated this procedure as many times as possible within a minute. The number of completed sit-ups was recorded as the score.

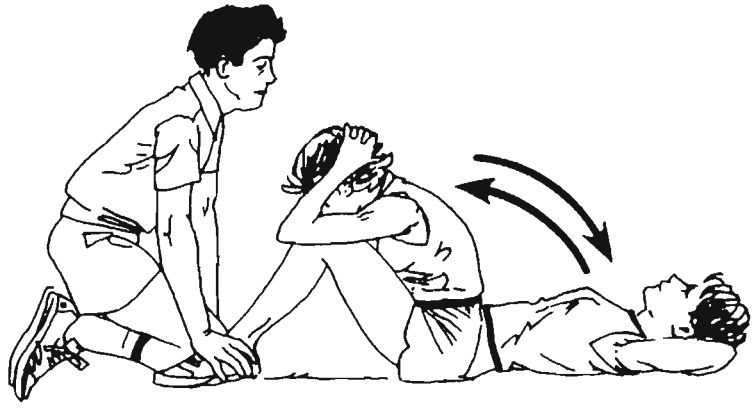


Figure 3.4 : SIT-UP

3.2.3 **Cardio-Respiratory Measures**

Aerobic capacity was ascertained through measurement of maximum oxygen uptake and in the process of measuring aerobic capacity, ventilation, and heart rates were also recorded.

3.2.3.1 **Maximal oxygen uptake**

Maximal Oxygen Uptake is defined as the amount of oxygen that can be consumed per minute during maximal exercise. In most muscular activities, oxygen uptake shows a roughly linear increase as the work load increases, until a steady state is reached which corresponds to the exercise demands (Astrand and Rodahl 1970). The fitter the subject, the greater is the possibility of reaching a sustained plateau in terms of oxygen uptake. In less fit subjects, peak oxygen uptake values are usually achieved, rather than maximum oxygen uptake.

A Woodway motor driven treadmill was used in order to establish consistent workload increases for the determination of oxygen uptake. The Oxycon Sigma was used in order to monitor oxygen uptake and ventilation during the testing procedure. Heart rates were recorded through the use of Hewlett Packard telemetary equipment, linked to the Fukuda Devishi electrocardiogram machine.

The standard electrode locations used were:

RA (right arm electrode, placed near right midclavicular line, directly below clavicle).

LA (left arm electrode placed near 6th and 7th intercostal space on midclavicular line).

RL (right leg electrode, place near left midclavicular line, directly below clavicle).

The metabolic measurements were determined by using a continuous treadmill protocol, with incremental speed changes. After the electrodes were attached, the subject was asked to remain still for three minutes in order to record baseline values. All subjects started walking at six km/hr for three minutes at the first workload, which also served as a warm-up period. Thereafter, the workloads were increased every one minute until exhaustion.

The gradient of the treadmill remained at zero percent throughout the testing session. At the end of the test the subject was required to keep walking on the treadmill for at least five minutes in order to cool down.

The highest oxygen consumption achieved during the last minute of the achieved workload at maximum exercise intensity was regarded as the peak value.(Astrand and Rodahl, 1970).

Peak oxygen consumption was recorded as a definite plateau was not reached when relating oxygen uptake to workload (Astrand and Rodahl, 1970). Peak oxygen uptake was recorded in l/min as an absolute value and in ml/kg/min when corrected for body weight. Other metabolic measures included ventilation and heart rates which were automatically calculated by the Oxycon Sigma.

3.2.4 **Flexibility**

Flexibility is the capacity of the joint to move smoothly through it's full range of motion (Heyward,1991). This ability is important in the performance of sport skills as well as everyday activities such as bending to pick up a shoe or getting out of a car. Most lower back problems occur because of poor flexibility of the back and hamstring muscles (Baumgartner and Jackson, 1982) The modified Wells and Dillon sit and reach test was used to measure flexibility (Figure 3.5).

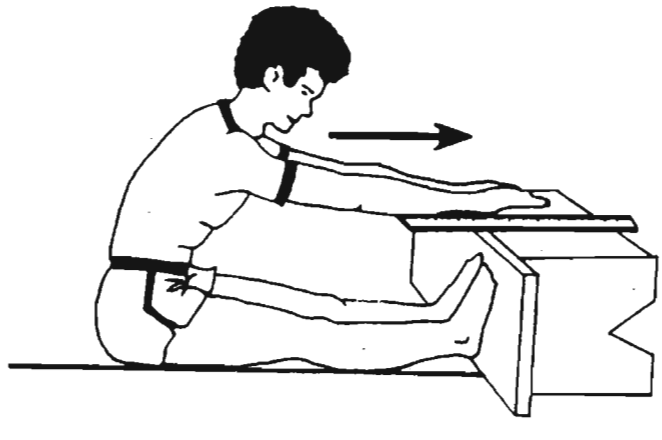


Figure 3.5 : SIT AND REACH

The subject sat on the floor with bare feet flat against the side of the box. The knees were kept straight throughout the test, with the tester holding the knees down to the floor.

To perform the test the subject extended his arms forward with his hands placed on top of each other, palms facing down. He slid his hands along a measuring scale on top of the box. The furthest position that was reached by the subject and held for at least three seconds, was recorded (Figure 3.5). The score was the furthest point reached by the finger-tips of both hands, measured in centimeters. The best of three trials was recorded.

3.3 LABORATORY ASSAYS

3.3.1 Blood Collection

All subjects were tested after a 12 hour overnight fast. There were two blood sampling days, namely Wednesday 7h00-9h30 and Saturday 8h00-9h30. On Wednesday mornings subjects reported to the Human Performance Research Laboratory at the University of Durban-Westville and had 45 ml of venous blood withdrawn from the antecubital vein while in a seated position. The samples were collected in appropriate evacuated glass tubes (Vacutainers), without venistasis.

On Saturday mornings the subjects reported to the Department of Chemical Pathology at the University of Natal for baseline blood measurements. The subjects rested for 15 minutes before blood collection began. Each subject had a 20 ml blood sample drawn from the antecubital vein while in a seated position. The mean value of the two blood samples were recorded.

3.3.2 Blood Analysis and Storage

The blood samples were centrifuged at 3000 rpms for 15 minutes in a standard laboratory centrifuge. The plasma or serum was aliquoted, to avoid repeated freezing and thawing, and analyzed fresh or frozen at -70° C until further analyses. Cells were also collected and stored for possible further DNA analysis. Regular internal quality control measurements were performed during each series of analyses

3.3.3 The Determination of Total Serum Cholesterol

Total serum cholesterol was determined by an enzymatic triglyceride kit (Boehringer Mannheim) and an automated procedure using the Technicon RA 1000 Random Access Analyser.

3.3.4 The Determination of Triglycerides

Serum triglycerides were determined with a fully enzymatic triglyceride kit (Boehringer Mannheim) and an automated procedure using the Technicon RA 1000 Random Access Analyser.

3.3.5 The Measurement of HDL-C

This was achieved using the test kit supplied by Boehringer Mannheim and the Technicon RA - 1000 Random Access Analyser. HDL-C was measured following the precipitation of VLDL and LDL by means of a precipitant containing a polyanion plus a divalent cation. A dextran sulfate-magnesium chloride precipitant was used in this study, according to an established procedure (Warnick et al.,1982). This was calibrated against samples analyzed

by the Centre for Disease Control (Atlanta, USA). The HDL-C in the supernatant was determined by the same method utilized for the total cholesterol assay.

3.3.6 **Calculation of Low Density Lipoprotein (LDL-C)**

LDL levels were calculated using the parameters obtained from the assays. The Friedewald et al., (1972) formula was used:

$$\text{LDL-C} = \frac{\text{TC-(Plasma triglycerides)-HDL-C (mmol/l)}}{2.2}$$

3.3.7. **The Calculation of the Ratio of Total Cholesterol to HDL-C**

The ratio was calculated using the parameters obtained from the assays described previously.

3.4 **STATISTICAL PROCEDURES**

The data collected in this study were subjected to different statistical treatments.

All the data were analysed by a computerized statistical programme known as Systat.

The Students paired t-test was used to establish whether any significant changes occurred following the intervention programmes. Differences between groups was established through the use of a Students unpaired t-test. Inter-variable correlations were calculated using the Pearson Correlation Coefficient technique for baseline variables (Chase, 1967; Phillips, 1978; Behr, 1983 and Brink, 1987).

CHAPTER FOUR

INDEX

4. RESULTS AND DISCUSSSION

The results of this study are presented in two broad categories, namely the pre-intervention baseline data and the data recorded following the intervention programmes of exercise and diet.

In the first category of results, the baseline characteristics of the cohort are sub-divided into those of a physiological, biochemical and dietary nature. This is followed by an analysis of the statistical associations which were recorded between these characteristics.

In the second category, the results recorded after the completion of the 15 week exercise and diet intervention programme are presented for each parameter, namely physiological, biochemical and dietary.

A statistical comparison of the results recorded by each of the experimental gorups (E, ED and C) is then presented. Discussion follows the presentation of the data for each of these parameters.

4.1 PRE-INTERVENTION CHARACTERISTICS OF COHORT

4.1.1 Physiological Parameters

4.1.1.1 Anthropomorphic and demographic characteristics

The demographic and anthropomorphic characteristics of the cohort are presented in Table 4.1.

Table 4.1 **Means and SD of Anthropomorphic Characteristics.**

Parameter	Exercise (N = 15)	Exercise & Diet (N = 14)	Control (N = 12)	Total Cohort (N = 41)
Age (yrs)	33.9 (7.3)	36.7 (7.1)	37.2 (6.4)	35 (6.9)
Height (cm)	171.7 (6.39)	170.5 (4.75)	169 (6.0)	170.6 (5.74)
Weight (kg)	68.3 (10.4)	71.3 (11.4)	76.0 (16)	71.6 (12.7)
Body Mass Index (kg/m^2)	23.3 (3.9)	24.5 (3.5)	26.4 (4.4)	24.5 (4.05)
% Body fat	21.2 (5.5)	23.2 (6.2)	24.5 (5.3)	22.9 (5.8)
Lean body mass (kg)	53.5 (5.4)	54.3 (6.0)	56.7 (9.5)	54.6 (7.28)

Ages ranged from 25 to 55 years, with the mean age for the different groups being 33.9 years for the Exercise (E) group, 36.7 years for the Exercise and Diet (ED) group and 37.2 years for the Control (C) group, respectively. The age range of the participants in this study was similar to the ages in a number of studies relating to lipoproteins and exercise (Baker et al., 1986; Wood et al., 1991). No statistically significant differences existed between the mean ages of the three groups.

Similarly, no statistically significant differences were observed between the mean baseline weights of the E and ED groups, although the mean weight of the control group was higher

than both experimental groups. The means of body mass index and percent body fat measurements also revealed no statistical differences among the groups prior to the commencement of the exercise programme. The subjects were classified using the body mass index method (Bray, 1985). As indicated in Table 4.2 of the total sample, five (12.2%) were underweight, 18 (43.9%) were within the healthy weight range, 15 (36.6%) were overweight and only 3 (7.3%) subjects were classified as obese. The healthy and overweight categories were fairly evenly distributed within each of the three groups. Eight percent of the underweight subjects fell in the exercise group.

Table 4.2 **Body Mass Index Classification.**

Categories for BM1	**Exercise (N = 15)**	**Exercise & Diet (N = 14)**	**Control (N = 12)**	**Total Cohort (N = 41)**
Underweight (No.) (18 - 20 kg/m^2)	4 (26.7%)	1 (7.1%)	-	5 (12.2%)
Healthy weight range (20.1 - 25 kg/m^2)	6 (40%)	7 (50%)	5 (41.7%)	18 (43.9%)
Overweight (25.1 - 30 kg/m^2)	5 (33.3%)	5 (35.8%)	5 (41.7%)	15 (36.6%)
Obese (30.17 kg/m^2 and greater)	-	1 (7.1%)	2 (16.7%)	3 (7.3%)

Demographically the cohort reflected a number of life style characteristics common to a high risk population for CHD. In reviewing the family history of lifestyle disease, the results indicated that the parents (mother or father) of 32 of the 41 subjects (78%) had experienced diabetes, CHD, hypertension, stroke or a combination of these diseases. There was however a high prevalence of hypertension among the mothers (39%). Only nine subjects (22%) of the study population had no family history of hypokinetic diseases (Figure 4.1).

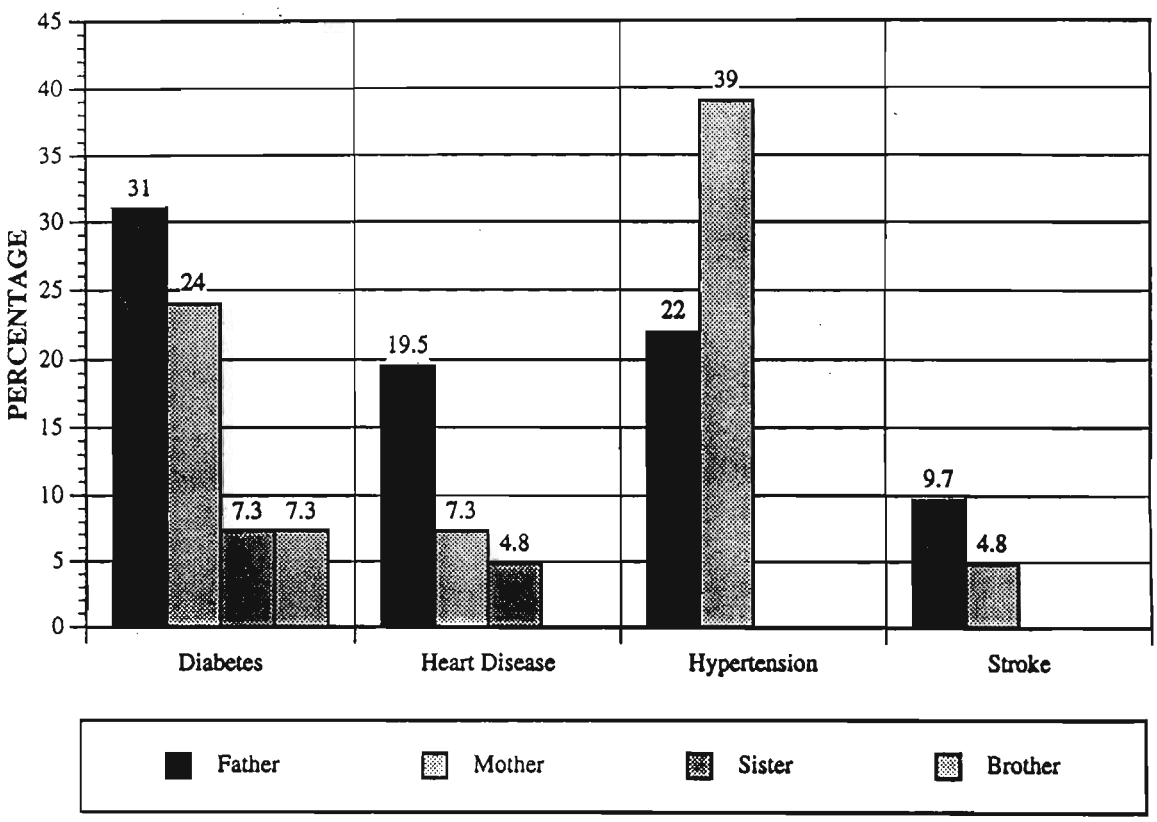


Figure 4.1 : Family History of Cohort
(Percentage)

The mean alcohol intake for groups E, ED and C were 12.7 grams, 14.5 grams and 12 grams respectively. In the E group 60% of the subjects consumed alcohol regularly while in the remaining two groups there was an equal distribution of drinkers and non-drinkers (Figure 4.2).

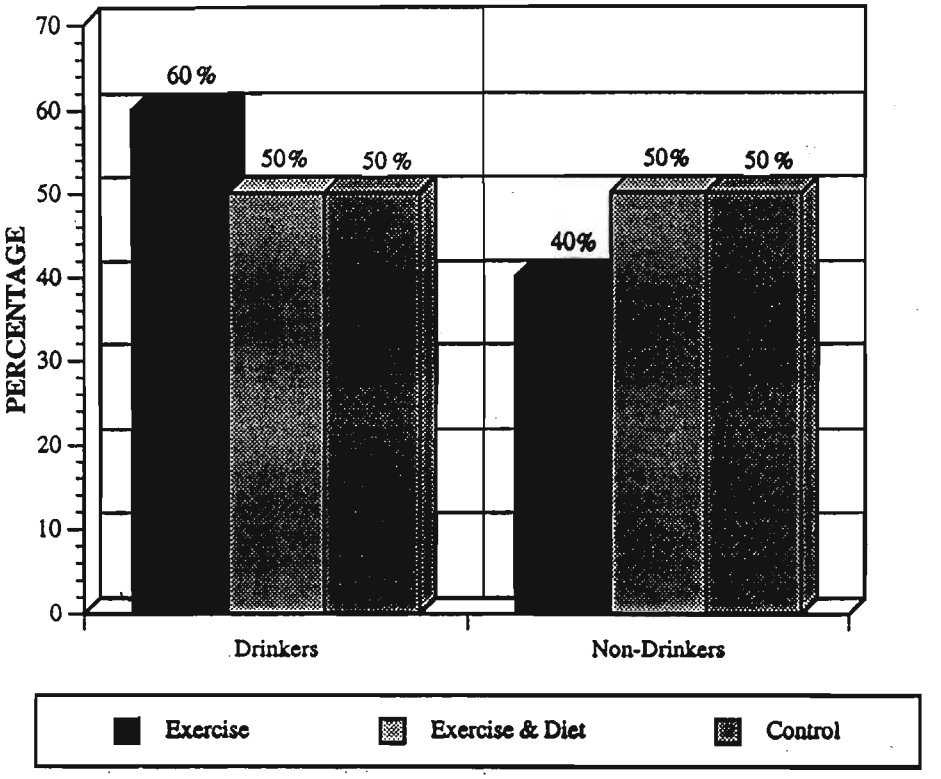


Figure 4.2 : Alcohol Intake of Cohort
(Percentage)

The smoking pattern for the total cohort showed that 46% of the subjects smoked an average of 12.6 cigarettes per day. These subjects started smoking at a mean age of 17 years and were still smoking at the time of the study, an average period of 13.2 years (Figure 4.3).

Among the three groups, the most smokers (67%) fell in the E group with 42% in the C group and 29% in the ED group.

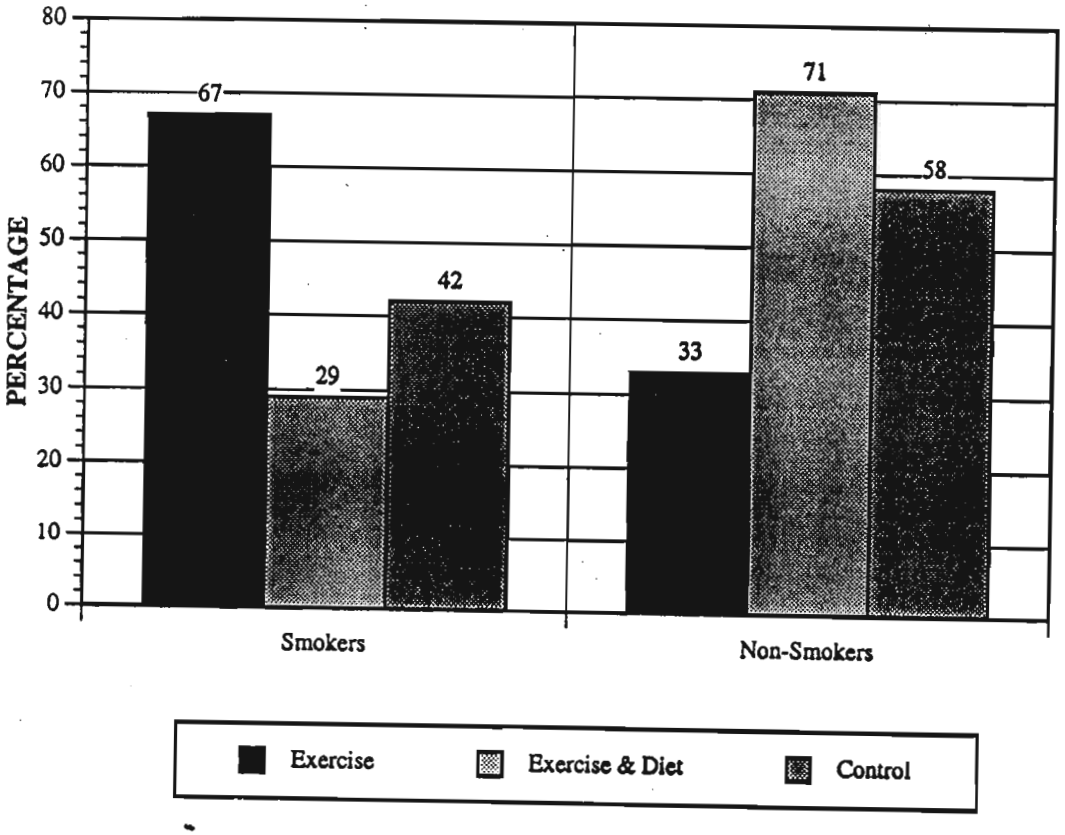


Figure 4.3 : Smoking Patterns of Cohort (Percentage)

4.1.1.2 **Physical Fitness Indices**

Table 4.3 displays the baseline indices of fitness including cardiorespiratory capacity, ventilation, maximum heart rate, muscular endurance and flexibility.

Table 4.3 **Means and SD of Indices of Fitness.**

Parameter	Exercise (N = 15)	Exercise & Diet (N = 14)	Control (N = 12)	Total Cohort (N = 41)
Peak VO_2 (l/m)	2.48 (.35)	2.55 (.33)	2.43 (.53)	2.49 (4.0)
Peak VO_2 (ml/kg/m)	37.86 (7.6)	36.36 (4.39)	32.58 (6.2)	35.8 (6.3)
Ventilation (l/m)	91.2 (17.6)	95.8 (13.3)	93.1 (22.9)	93.35 (17.78)
Maximum heart rate (No.)	181.2 (12.2)	180.71 (12.3)	180.25 (15.1)	180.8 (12.8)
Sit-ups (No.)	31 b (8.79)	26 a (6.7)	18 ab (7.1)	25.6 (9.1)
Push-ups (No.)	25.1 b (11.3)	20.4 (8.5)	15.7 b (6.5)	20.7 (9.1)
Flexibility (cm)	23.5 b (6.7)	17.6 (10.2)	17.00 b (8.7)	19.6 (8.9)

a = $P \leq 0.05$ between ED vs C
b = $P \leq 0.05$ between E vs C

There were no significant differences among the means of the three groups in the absolute values of oxygen uptake (l/m). Slightly lower values were reflected in the control group. When adjustments were made for body weight (ml/kg/min.), these results compared

favourably with other studies where sedentary subjects were tested (Zuti and Corbin, 1977; Wood et al., 1991). These values of oxygen consumption were similar to the norms for the South African adult population (Andrews, 1991) and also compared favourably to international norms (Zuti and Corbin, 1977). The baseline values reflecting maximum peak ventilation and heart rates indicated no statistically significant differences amongst groups. These values reflected what is considered to be normal for the group tested. In describing the results, the t-value is included along with the level of significance in each case.

The exercise group registered superior levels of fitness compared to the control group with respect to muscular endurance reflected by sit-ups ($P \leq 0.05$, $t = 4.037$) and push-ups ($P \leq 0.05$, $t = 2.56$). This group also demonstrated superior flexibility ($P \leq 0.05$, $t = 2.2$). The ED group showed similar muscular endurance superiority over the control group with respect to sit-ups ($P \leq 0.05$, $t = 2.819$). Thus the control group fared relatively poorly on tests of muscular endurance and flexibility compared to the experimental groups but was similar in aerobic capacity. There were no statistical differences among the means of the three groups for ventilation and maximum heart rate measures.

4.1.2 **Biochemical Parameters**

4.1.2.1 **Lipid and lipoprotein profiles**

Table 4.4 reflects the baseline results for lipid and lipoprotein levels of the cohort.

Table 4.4 **Means and SD of Lipid and Lipoprotein Profiles.**

Parameter	Exercise Only (N = 15)	Exercise and Diet (N = 14)	Control (N = 12)	Total Cohort (N = 41)
Total cholesterol (mmol/l)	5.72 (.97)	5.38 (.92)	5.82 (.89)	5.63 (.93)
Triglycerides (mmol/l)	1.5 a (.79)	1.2 b (.46)	2.26 ab (1.08)	1.6 (.88)
HDL-C (mmol/l)	.97 (.23)	1.04 b (.26)	.86 b (.18)	.96 (.23)
LDL-C (mmol/l)	4.14 (.96)	3.81 (1.00)	3.96 (.79)	3.97 (.92)
Total cholesterol to HDL-C Ratio	6.4 (2.14)	5.41 b (1.66)	6.92 b (1.23)	6.23 (1.83)

a = $P \leq 0.05$ between E vs C
b = $P \leq 0.05$ between ED vs C

No differences were observed between the means of the three groups for total cholesterol and LDL-C levels. When the mean cholesterol values of the total cohort were classified in order to determine risk for CHD (Table 4.5) it was found that 66% of subjects had total cholesterol levels above 5.2 mmol/l, while 42% of the total cohort were classed as borderline, with 10% being placed in the high risk category for CHD. It should be noted that all groups had baseline mean total cholesterol levels of above 5.2 mmol/l, which supports the view that the present subject population is at borderline risk for CHD.

Table 4.5 **Lipid and Lipoprotein Classification**

Classifications	Total Cohort (N = 41)	Exercise (N = 15)	Exercise & Diet (N = 14)	Control (N = 12)
		***CHOLESTEROL**		
Desirable = < 5.2 mmol/l	14(34%)	3(20%)	8(57%)	3(25%)
Borderline = 5.2-6.2 mmol/l	17(42%)	9(60%)	4(29%)	4(33%)
High = > 6.2 mmol/l	10(24%)	3(20%)	2(14%)	5(42%)
		***LDL-C**		
Desirable = < 3.4 mmol/l	11(27%)	4(27%)	5(36%)	2(17%)
Borderline = 3.4-4.1 mmol/l	10(24%)	4(27%)	3(21%)	3(25%)
High = > 4.1 mmol/l	20(49%)	7(46%)	6(43%)	7(58%)
		***TRIGLYCERIDES**		
Desirable = < 1.5 mmol/l	25(60%)	11(67%)	12(86%)	2(17%)
		***HDL-C**		
Desirable $\geq$ .9 mmol/l	23(56%)	8(67%)	10(71%)	5(42%)
		***HYPERCHOLESTEROLAEMIA**		
Total cholesterol = > 5.2 mmol/l Triglycerides = < 2.3 mmol/l	25(61%)	11(73%)	6(43%)	8(67%)
		***MIXED HYPERLIPIDAEMIA**		
Total cholesterol = > 5.2 mmol/l Triglycerides = > 2.3 mmol/l	3(7%)	1(7%)	-	2(17%)
		***HYPERTRIGLYCERIDAEMIA**		
Total cholesterol = < 5.2 mmol/l Triglycerides > 2.3 mmol/l	3(7%)	1(7%)	1(7%)	1(8%)
		***RISK RATIO**		
Risk = > 5	31 (76%)	12(80%)	7(50%)	12(100%)

*Assman (1993)

Table 4.4 clearly indicates that the mean LDL-C levels of all three groups were above 3.4 mmol/l which categorizes the cohort as borderline risk for CHD.

If the cut-off limit of 1.5 mmol/l is used, then 40% (16 subjects) of the total cohort may be classified as having undesirable triglyceride levels (Table 4.5).

When examining the three groups separately, it may be noted that the control group included 75% (9 subjects) with elevated triglyceride levels as compared to 17% (2 subjects) in the ED group and 40% (6 subjects) in the E group. A significant difference was noted between the control and both the E ($P \leq 0.05$, $t = 2.09$) and ED groups ($P \leq 0.05$, $t = -3.33$) (Table 4.4).

When 0.9 mmol/l or less of HDL-C was taken as an indicator of CHD risk (Assmann, 1993), then 18 of 41 subjects (44%) had undesirable HDL-C values. An intergroup comparison indicates that both the E and C groups (Table 4.5) included 7 subjects with undesirable HDL-C levels, while 4 subjects (22%) in the ED group had undesirable levels (0.9 mmol/l or less). The ED group recorded significant differences between the means for HDL-C compared to the control groups ($P \leq 0.05$, $t = 1.99$). When the ratio of total cholesterol to HDL/C is used an an indicator of risk for CHD (i.e. < 5) then 76% of the total cohort was at risk for CHD. The ED group's mean scores for total cholesterol to HDL-C ratio were significantly different from those of the controls ($P \leq 0.01$, $t = 2.59$). This compares with the 66% of subjects who had baseline cholesterol levels above the desirable levels.

When examining the hyperlipidaemia classification of the baseline measurements of the cohort (Table 4.5), 61% of the subjects were hypercholesterolaemic, while 7% had mixed hyperlipidaemia and 7% of the subjects were hypertriglyceridaemic.

The analysis of the baseline lipid and lipoprotein profiles of this cohort shows an increased risk for premature CHD.

4.1.2.2 **Dietary aspects**

An analysis of the nutritional intake of the total cohort is presented in Table 4.6. A statistically significant difference was noted between the E group and the ED group for total energy ($P \leq 0.05$, $t = 3.07$) and carbohydrate intake ($P \leq 0.05$, $t = 3.07$). Further to this, statistical differences were also established between the means of ED and C groups with respect to the total energy ($P \leq 0.05$, $t = 2.25$), total fat ($P \leq 0.05$, $t = 2.234$) and polyunsaturated fatty acid intake ($P \leq 0.05$, $t = 3.12$). It would appear from the above data that the food intake of the control group varied somewhat from that of the other two groups. It should be noted that the dietary recall method has inherent limitations for the assessment of dietary intake.

Table 4.6 **Means and SD of Dietary Intake.**

Nutrients	Exercise Only (N = 15)	Exercise and Diet (N = 14)	Control (N = 12)	Total Cohort (N = 41)
Energy (Kcal)	2880 (638) c	2388 (525) bc	2864 (555) b	2707 (609.2)
Protein (gms)	80.4 (18.2)	74.5 (20.6)	82.7 (22.6)	79.0 (20.2)
Total fat (gms)	121.9 (36.2)	107.7 (32.4) b	143.9 (47.2) b	123.5 (40.3)
Monounsaturated (gms)	38 (11.3)	34 (13.1)	45.5 (17.4)	38.8 (14.34)
Polyunsaturated (gms)	41.1 (17.3)	33.4 (10.9) b	51.1 (17.7) b	41.5 (16.7)
Saturated (gms)	34.1 (11.1)	29.7 (9.1)	35.6 (15.1)	33.0 (11.8)
Carbohydrates (gms)	347.8 (79.1) c	263.4 (68.1) c	295.3 (68.1)	303.6 (79.9)
Dietary Fibre (gms)	20.8 (6.4)	24.1 (6.6)	19.5 (6.3)	21.5 (6.57)
Cholesterol (mg)	276.9 (91.2)	230.4 (124)	311.1 (141.2)	271 (120.4)
P:S ratio	1.24 (.41)	1.19 (.42)	1.56 (.68)	1.31 (.52)
Alcohol (gms)	11.3 (12.7)	14.6 (8.9)	12.00 (4.6)	12.54 (9.52)

c = $P \leq 0.05$ between E and ED groups
b = $P \leq 0.05$ between ED and C groups

4.1.3 Statistical Associations between Characteristics of Cohort

Baseline statistical associations between elements of lipids, lipoprotein profiles, fitness indices, morphological characteristics and dietary components were correlated.

Tables 4.7 and 4.8 reflect univariate correlation coefficient analysis of the total cohort with respect to baseline characteristics. Only the computed correlations which were found to be significant at the 1% level are reflected in the tables presented.

Table 4.7 **Baseline Statistical Associations Between Lipids and Lipoproteins, Fitness Indices, Anthropomorphic Characteristics and Other Indices.**

(i) Lipids and Lipoproteins		
Components	**Correlation**	**r***
Triglycerides	Body mass index	.470
	% Body fat	.523
	HDL-C	-.640
Cholesterol	LDL-C	.946
HDL-C	Risk ratio	-.793
	Triglycerides	-.640
	Alcohol	.577
LDL-C	Risk ratio	.730
	Cholesterol	.946
Risk Ratio	Cholesterol	.651
	Triglycerides	.493
(ii) Fitness Indices		
Oxygen uptake (1/m)	Weight	.462
	Ventilation	.609
Push-ups	Sit-ups	.603
(iii) Anthropomorphic Characteristics		
Body mass	% Fat	.829
	Lean body mass	.816
	P:S ratio	.396
	Triglyceride	.470
	Weight	.926
% Body fat	Triglycerides	.523
	Weight	.732
	Lean body mass	.492
Lean body mass	Oxygen uptake (1/min)	.505
	Weights	.951

* Only r values > 0.393 ($P \leq 0.01$) are reported.

Table 4.8 **Baseline Statistical Associations Between Dietary Components and Other Indices**

Dietary Components		
Components	**Correlation**	**r***
Dietary Cholesterol	Kcal	.523
	Monounsaturated fats	.440
	Protein	.695
	Saturated fats	.625
	Total fat	.625
Alcohol	HDL-C	.577
Monounsaturated fats	Polyunsaturated fats	.595
	Protein	.708
	Saturated fats	.801
	Total fat	.930
	Kcal	.736
Polyunsaturated fats	Protein	.486
	P:S ratio	.606
	Saturated fats	.801
	Total fat	.930
	Kcal	.595
Saturated fats	Total fat	.809
	Cholesterol	.415
	Kcal	.595
Protein	Total fat	.729
	Saturated fats	.673
	Kcal	.724
P:S Ratio	Body mass index	.396
Kcal	Total carbohydrate	.741
	Total fat	.798

* Only r values > 0.393 ($P \leq 0.01$) are reported.

The most striking examples in the lipid and lipoprotein category were between triglycerides and HDL-C (r = -0.64), between cholesterol and LDL-C (r = 0.95), and between LDL-C and risk ratio (r = 0.73) (Table 4.7). However, these were expected trends (Tran et al., 1983). Percent body fat correlated with triglycerides (r = 0.52) while related morphological components (Table 4.7) such as body mass index, percent body fat and lean body weight correlated well, supporting previously reported data (Barnard et al., 1992). It thus appears that with body weight reduction, there will be a concomitant decrease in CHD risk factors.

Among the nutritional correlations, a relationship was noted between alcohol and HDL-C (r = 0.58). This correlation may be attributed to the small homogenous number of subjects who consumed alcohol. Other researchers (Ernst, et al., 1980; Frazer, et al., 1983), found similar associations using multivariate correlation analysis. The other correlations indicated in Table 4.8, reflected expected relationships with respect to nutritional components.

4.1.4 **Summary of Baseline Data**

In summary an analysis of the data gathered prior to intervention indicated that:

(a) Thirty six percent of the subjects were overweight, of which three were probably obese.

(b) One third of the subjects had high plasma triglyceride levels.

(c) Sixty six percent of the subjects had undesirable cholesterol levels.

(d) More than 40 % of the subjects had HDL-C levels below desirable values.

(e) When assessing lipid abnormalities, three subjects (7%) had mixed hyperlipidaemia and three (7%) of the cohort had hypertriglyceridaemia. These findings suggest that

substantially more than two thirds of the subjects had an increased risk of CHD, due to abnormal lipid values. It cannot be assumed that this is true of the Indian population as a whole, but other studies with larger numbers of subjects, have also demonstrated the high prevalence of risk factors in this community.

(f) The baseline cardiorespiratory, muscular endurance and flexibility measures reflect normal values for the sedentary South African male.

(g) Total energy intake for the E and C groups was similar. The ED group however reflected a lower intake of total fat and polyunsaturated fats than the C group and a lower intake of carbohydrates when compared to the E group.

(h) Expected univariate correlation coefficients were registered between the baseline parameters.

The Indian population of this country is particularly prone to CHD and this susceptibility is due to a variety of factors, including genetic endowment, raised serum lipids and lipoproteins, moderate obesity, lack of exercise and the high intake of saturated fats (Seedat, 1993). This cohort demonstrated the general characteristics of practically all of the above risk factors, thus reflecting high risk for CHD. Preventive measures such as a prudent diet and regular exercise may therefore serve to retard the development of CHD. These two listed preventive measures are supported by substantial prospective and epidemiological studies.

4.2 POST INTERVENTION CHARACTERISTICS OF EXPERIMENTAL GROUPS

The results in this section represent the results of the evaluation process which followed the aerobic exercise and diet intervention programmes.

These intervention programmes comprised exercise for the E group and exercise and diet for the ED group. The control group (C) maintained a sedentary status and an unchanged diet throughout the 15 weeks of the intervention programme.

Comparisons between pre-intervention and post-intervention data are reflected and further comparisons of the responses of the different groups to the intervention programmes are also presented.

4.2.1 **Physiological Parameters**

4.2.1.1 **Anthropomorphic characteristics**

Table 4.9 indicates the changes which occurred in anthropomorphic characteristics of the different groups following the intervention programme. The ED group effected the greatest changes in Body Mass Index (BMI) (-5.3%) which was statistically significant ($P \leq 0.01$, $t = 9.15$). The E group recorded a 2.5% decrease in BMI which was not statistically significant. Percent body fat and lean body mass in the E group both showed significant mean changes ($P \leq 0.01$, $t = 8.4$ and $P \leq 0.01$, $t = 7.88$). The ED group registered similar significant mean changes for these two components ($P \leq 0.01$, $t = 12.03$ and $P \leq 0.05$, $t = 2.11$).

In Table 4.9 it may be seen that body mass decreased significantly in the ED group ($P \leq 0.01$, $t = 9.89$). The mean weight loss following intervention for the ED group was 3.7 kgs, compared to .9kg in E group and .8 kg in the C group. It would seem that this greater weight loss was due to the dietary changes which occurred in the ED group. The ED group recorded changes in all four components (BMI, % fat, L.B.M. and Body Mass), whereas the E group registered significant change in % fat, LBM and Body Mass ($P \leq 0.05$, $t = 2.09$). This may have been due to the added dietary intervention strategy. The Control group reflected no significant changes. Furthermore, the one obese subject in the ED group lost weight and moved to a lighter category while two subjects from the healthy weight range shifted to the underweight category (Table 4.2). In the other two groups weight shifts were negligible.

Table 4.9 **Post Intervention Changes in Anthropomorphic Characteristics (mean, SD and % change)**

Parameter	Exercise Only			Exercise and Diet			Control		
	Pre	Post	%	Pre	Post	%	Pre	Post	%
B.M.I. (kg/m^2)	23.3 (3.9)	22.7 (3.3)	-2.5	24.5 (3.5)	23.2 * (3.7)	-5.3	26.4 (4.4)	26.1 (4.6)	-1.0
Fat (percentage)	21.3 (5.5)	15.8 * (4.5)	-25.8	23.3 (6.2)	17.0 * (5.88)	-27.0	24.6 (5.3)	23.7 (5.8)	-3.7
L.B.M. (kg)	53.5 (5.4)	56.9 * (6.8)	+6.4	54.3 (6.0)	55.6 * (6.3)	+2.4	56.7 (9.5)	56.7 (9.2)	0
Body mass (kg)	68.3 (10.4)	67.4 * (9.2)	-1.3	71.3 (11.4)	67.6 * (11.8)	-5.2	76.0 (16)	75.2 (16.2)	-1.1

* = significantly different ($P \leq 0.05$).

At the commencement of the study, of the 29 subjects who participated as experimental subjects, ten were in the overweight category and one was obese. At the conclusion of the study eight subjects were still overweight and the single obese subject lost weight and was re-classified as overweight. The mean weight loss for the E group was 0.9kg compared to the 3.7 kgs for the ED group (Table 4.9). The control group remained the same in terms of body weight. This substantial weight loss within the ED group was statistically significant ($P \leq 0.01$), with this group also achieving statistical significance in BMI. There were substantial losses in percent body fat in both intervention groups, (-25,8% for E and -27% for the ED group) which may be attributed to the intervention programme.

Many studies have reported that exercise, accompanied by losses in body weight, are associated with increases in HDL-C levels (Wilson and Lees, 1972; Bradley et al.,1978; Garrison et al.,1980 and Hallak and Nomani, 1988). This association is addressed in detail under lipid and lipoprotein changes.

4.2.1.2 **Physical Fitness Indices**

The results in this section will be discussed under two headings, namely:

(a) muscular endurance and flexibility and

(b) cardiorespiratory and metabolic parameters

(a) **Muscular endurance and flexibility**

The effects of exercise are reflected in the increases which occurred in muscualr endurance and flexibility for both experimental groups. The control group recorded only slight variations on the same fitness parameters (Table 4.10). These data further indicate that the exercise programme was effective in raising the fitness levels of a group of sedentary individuals to standards that reflect the fitness of active persons. The greatest improvement occurred in the press-up scores for the two intervention groups (24.7% for the E group and 28.4% for the ED group), with these improvements being statistically significant ($P \leq 0.05$, $t = -3.3$ for E group and $P \leq 0.01$, $t = -5.28$ for the ED group). Similar improvements were registered for sit-ups and these were also significant ($P \leq 0.05$, $t = -3.54$) for the E group and ($P \leq 0.05$, $t = -3.91$) for the ED group. The ED group also registered the greater percentage improvement (+15.4%) when compared to the E group (+9%). The mean flexibility scores for both experimental groups increased significantly ($P \leq 0.05$, $t = 3.93$ for E group and $P \leq 0.05$, $t = -3.68$ for the ED group). No statistical differences were recorded in the control group. These positive changes in muscular endurance and flexibility for both experimental groups, indicates improved fitness levels for these indices.

Table 4.10 **Post Intervention Changes in Muscular Endurance and Flexibility (mean, SD and % change).**

Parameter	Exercise Only			Exercise and Diet			Control		
	Pre	Post	%	Pre	Post	%	Pre	Post	%
Sit-ups (No)	31 (8.79)	33.8 * (7.0)	+9	26 (6.7)	30* (5.9)	+15.4	18.3 (7.1)	19.8 (7.1)	+7.7
Press-ups (No)	25.1 (11.3)	31.3 * (9.1)	+24.7	20.4 (8.5)	26.3* (8.2)	+28.4	15.7 (6.5)	16.6 (6.0)	+5.8
Flexibility (cm)	23.5 (6.7)	26.8 * (5.2)	+14	17.6 (10.2)	21.4* (9.1)	+21	17 (8.7)	18 (9.5)	+5.9

* = significantly different ($P \leq 0.05$).

The pre-intervention mean values for sit-ups and flexibility for the two experimental groups fell within the average range for the South African population (Andrews, 1991). These mean values improved significantly following the intervention. The ED group recorded higher percentage changes from pre to post intervention on sit-ups, press-ups and flexibility as compared to the E group.

The capacity for improvement among those beginning at a lower baseline level of fitness was therefore greater than those commencing at higher levels. The E group should not be regarded as having shown lesser progress than the ED group, but rather both groups should be viewed as having shown good progress.

The significant increases observed in muscular endurance and flexibility for the experimental subjects demonstrated a definite training effect, while the control subjects showed no change (Table 4.10). These changes in fitness indices among the experimental subjects supports the

view that improvements in initial fitness do not require high levels of intensity or vigorous exercise sessions in a sedentary population. The precise mechanisms whereby increased physical activity may lead to higher plasma HDL-C and other changes in lipoproteins is currently poorly understood, but it seems quite probable that increased physical activity leads to increased muscle and adipose tissue lipoprotein lipase activity which in turn leads to lowered plasma triglycerides, and ultimately to increased HDL-C (Wood and Haskell, 1979).

(b) **Cardiorespiratory and metabolic measures**

Similar mean improvements were observed among both experimental groups following 15 weeks of aerobic exercise (Table 4.11). This was true for cardiorespiratory and metabolic parameters. The improvements in mean peak absolute oxygen uptake (1/m) values were statistically significant for both E and ED groups ($P \leq 0.01$, $t = -6.49$, for the E group and $P \leq 0.01$, $t = -5.66$ for the ED group). For peak oxygen uptake values, after correction for body weight (ml/kg/min), the improvements were also significant for both groups, ($P \leq 0.01$, $t = 9.38$ for the E group and $P \leq 0.01$, $t = -6.39$ for the ED group).

No change was recorded in the mean of the control group. The mean oxygen uptake for an adult male aged 35 years is 38.8 ml/kg/min (Zuti and Corbin 1977). Using this as the population mean the experimental subjects in this study improved their oxygen consumption by 12.4% indicating that the aerobic fitness programme had an impact on these sedentary subjects. The zero percent change in the control group may also confirm that they were not aerobically active during this intervention period, as required in the research protocol. Peak ventilation increased in both intervention groups and these changes were significant in both

experimental groups ($P \leq 0.01$, $t = 6.23$ for E group and $P \leq 0.01$, $t = 3.82$ for ED group). These increased ventilation values correspond to the increased oxygen consumption values. Maximal heart rate did not change significantly among groups, which was to be expected (Astrand and Rodahl, 1970).

Table 4.11 **Post Intervention Changes in Cardiorespiratory and Metabolic Parameters (mean, SD and % change)**

Parameter	Exercise Only			Exercise and Diet			Control		
	Pre	Post	%	Pre	Post	%	Pre	Post	%
Peak VO_2 (l/m)	2.48 (.35)	2.94 * (.47)	+19	2.55 (.33)	2.91 * (.29)	+14.6	2.43 (.53)	2.43 (.57)	0
Peak VO_2 (ml/kg/m)	37.86 (7.6)	44.47 * (8.04)	+18.7	36.36 (4.39)	44.2 * (6.6)	+21.8	32.58 (6.2)	32.45 (6.6)	-.3
Peak Ventilation (l/m)	91.2 (17.6)	113.8 * (19.3)	+24.8	95.8 (13.3)	108.3 * (12.1)	+13	93.1 (22.9)	96.0 (22.8)	+3.1
Peak Heart rate (No)	181 (12.2)	183 (8.3)	+7.6	180 (12.3)	184 (8.9)	+2.2	180 (15.1)	184 (14.8)	+2.2

* = significantly different ($P \leq 0.05$).

The improvements in peak oxygen uptake observed in this study were comparable to improvements found in other studies, using subjects of similar initial fitness levels (Pollock, 1973 and Baker et al.,1986). The experimental subjects in this study improved their peak maximum oxygen consumption by 12.4%, which was an improvement on those established by Zuti and Corbin (1977) for 35 year old males (38 ml/kg/min).

In this study, a mean peak maximum oxygen uptake value of 44 ml/kg/min was recorded for the experimental subjects (Table 4.11). These improvements were obtained using a moderate aerobic exercise programme, three days per week, lasting approximately 30 minutes per session, for 15 weeks. Several cross-sectional studies have indicated that vigorous aerobic exercise may be associated with elevated HDL-C levels when compared to sedentary individuals. Earlier studies showing this relationship between vigorous exercise and HDL-C were distance runners (Martin et al.,1977; Wood et al.,1977), soccer and ice hockey players (Lehtonen and Viikari, 1980), Olympic athletes (Deshaies and Allard, 1981) and cross-country skiers (Enger et al.,1977). However, Haskell (1984) reiterated that low to moderate exercise was also associated with decreased CHD rates and that this may positively influence in plasma lipoprotein metabolism. The present study concurs with the Haskell (1984) finding, that moderate aerobic exercise may achieve lipid changes and that vigorous aerobic exercise is not the only exercise protocol for lipid modification to occur.

4.2.2 **Biochemical Parameters**

4.2.2.1 **Lipid and Lipoprotein Profiles**

In Table 4.12 the mean percentage changes and the results of tests for statistical significance for serum lipids and lipoproteins are indicated. HDL-C and total cholesterol to HDL-C ratio were the only two serum lipid components to register statistically significant mean changes after intervention. The t-test result for HDL-C in the E group was $P \leq 0.01$, $t = -4.1$ and for the ED group $P \leq 0.01$, $t = -4.09$, with the risk ratio registering a mean statistical significance of $P \leq 0.05$, $t = 4.11$ for the E group and $P \leq 0.05$, $t = 3.39$ for the ED group.

Table 4.12 **Post Intervention Changes in Serum Lipids and Lipoproteins (mean, SD and % change)**

Parameter	Exercise Only			Exercise and Diet			Control		
	Pre	Post	%	Pre	Post	%	Pre	Post	%
Cholesterol (mmol/1)	5.72 (.97)	5.86 (1.01)	+2.45	5.38 (.92)	5.21 (.35)	-3.16	5.82 (.89)	6.07 (1.03)	+4.2
Triglycerides (mmol/1)	1.5 (.79)	1.39 (.82)	-7.3	1.2 (.46)	1.195 (.45)	-.42	2.26 (1.08)	2.58 (1.21)	+14.2
HDL (mmol/l)	.97 (.23)	1.08 * (.28)	+11.3	1.04 (.26)	1.18 * (.29)	+13.4	.86 (.18)	.91 (.21)	+5.8
LDL (mmol/1)	4.14 (.96)	4.13 (.99)	-.2	3.81 (1.00)	3.47 (.87)	-8.9	3.96 (.79)	3.95 (.88)	-.25
Total cholesterol to HDL-C Ratio	6.4 (2.14)	5.8 * (2.02)	-9.4	5.41 (1.66)	4.65 * (1.25)	-14	6.92 (1.23)	6.93 (1.32)	-.15

* = significantly different ($P \leq 0.05$).

There were small beneficial percentage changes between pre and post intervention values with respect to cholesterol and LDL-C in the ED group. In the E group a drop of 7.3% in triglycerides was established while an increase of 14.2% was recorded for the Control group, indicating greater risk for CHD, following the intervention programme (Table 4.12).

When the changes in HDL-C and risk ratio (Total cholesterol to HDL-C ratio) are examined (Table 4.13) it may be noted that three more subjects in the E group achieved desirable levels ($\geq$ .9 mmol/l) while in the ED group two more subjects registered acceptable values after intervention. The mean percentage increase for pre- and post-intervention values showed a 13.4% change in the ED group's favour, compared to the 11.3% improvement in the E group's HDL-C levels. Six more subjects achieved desirable levels ($\leq$ 5) which shows

improvement with respect to risk ratio after the intervention for the experimental groups, as compared to no changes in the control group.

The greater improvement in the mean values for the ED group may be due to the large individual changes in HDL-C, while the E group had smaller individual changes, but in more subjects. The same reasoning could be applied to the risk ratio value.

Table 4.13 classifies the subjects into desirable lipid and abnormal lipid categories. No substantial changes occurred following the intervention programme with respect to lipid abnormalities. However, one subject from the ED group who was classified as hypertriglyceridaemic before the intervention programme, was later placed in the hypercholesterolaemic category.

Table 4.13 **Post Intervention Changes in Desirable Lipid and Abnormal Lipid Categories (No. of subjects and %).**

	Total Intervention (N = 29)		Exercise (N = 15)		Exercise & Diet (N = 14)		Control (N = 12)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
HDL-C								
Desirable = ≥ 0.9 mmol/l	18 62%	23 79%	8 53%	11 73%	10 71.4%	12 85.7%	5 41.7%	5 41.7%
Total cholesterol to HDL-C Ratio (Risk Ratio)								
Desirable = < 5	10 34.5%	16 55.3%	3 20%	7 46.7%	7 50%	9 64.3%	0 100%	0 100%
Hypercholesterolaemia								
Total cholesterol = > 5.2 mmol/l Triglycerides = < 2.3 mmol/l	17 58.6%	18 62%	11 73%	11 73%	6 42.9%	7 50%	6 50%	5 41.7%
Mixed hyperlipidaemia								
Total cholesterol = > 5.2 mmol/l Triglycerides = > 2.3 mmol/l	1 3.5%	1 3.5%	1 6.7%	1 6.7%	0 100%	0 100%	3 25%	5 41.7%
Hypertriglyceridaemia								
Total cholesterol = < 5.2 mmol/l Triglycerides = > 2.3 mmol/l	2 6.9%	1 3.5%	1 6.7%	1 6.7%	1 7.1%	0 100%	1 8.3%	2 16.7%
Normal values								
Total cholesterol = < 5.2 mmol/l Triglycerides = < 2.3 mmol/l	9 31%	9 31%	2 13.3%	2 13.3%	7 50%	7 50%	2 16.7%	0 100%

The large majority of the subjects in the experimental groups either improved their lipid profiles or remained unchanged after intervention. It should be noted however that two subjects in the control group, who were included in the normal lipid (cholesterol and triglyceride) profiles at the commencement of the programme deteriorated at the end of the study. Further, an additional control subject deteriorated to hypertriglyceridaemia category and another two control subjects moved to the mixed hyperlipidaemia group. These lipid

profiles of the control group deteriorated after 15 weeks of non-intervention. The experimental groups, on the other hand, either improved or remained the same.

In the present study 15 weeks of moderate aerobic exercise did not result in a statistically significant change in serum cholesterol levels, although a 3.2% decrease occurred in the ED group. The subjects who volunteered for the study were well motivated and co-operative as evidenced in the significant increases in their exercise capacity. Since the men exercised under relatively ideal conditions, and demonstrated no significant mean reductions in total cholesterol, it seems reasonabe to conclude that neither the amount of exercise nor the duration of exercise was effective in lowering serum cholesterol levels. Montoye et al.(1978) and Holloszy et al.(1964), in similar studies with middle-aged subjects, reported no changes in total cholesterol levels after a study period of six months. However, in the present study, when gross changes in body weight were taken for each subject in the ED group and correlated with their net total cholesterol difference, a correlation ($r=0.81$, $P \leq 0.01$) was obtained. It should be noted that when these correlations were calculated, only subjects who lost three kilograms or more over the 15 week period were included in the analysis. A total of 11 of the 14 subjects lost 3 kilograms or more, with the range being 3 to 5.6. Kgs. This correlation ($r=.81$, $P \leq 0.05$) is in keeping with a study by Tran and Weltman (1985) which showed a similar relationship between decreases in body weight and reductions in total cholesterol levels. The greater body weight changes in the ED group may be related to the additional dietary intervention, as compared to the exercise group.

In a meta-analysis study on the differential effects of exercise on lipids and lipoprotein levels seen with changes in body weight, it was concluded that regulating body weight change during exercise is important for effecting favourable changes in the lipid and lipoprotein profiles (Tran and Weltman, 1985).

The present study revealed that although the weight loss in the E group was lower than the ED group, both registered similar significant increases in HDL-C levels ($P \leq 0.01$). The greater mean weight loss (3.7 Kg) of the ED group ($P \leq 0.05$) when compared to the mean weight loss (0.9 Kg) of the E group do not support the findings of Tran and Weltman (1985) who reported significant increases in HDL-C levels with loss of weight. The lean body mass measures recorded statistical significance for both E ($P \leq 0.05$) and ED group ($P \leq 0.05$) which indicates the positive effects of exercise (Table 4.9).

Excess calorie intake and obesity are frequently associated with low HDL-C levels. Very often weight loss alone can significantly decrease plasma triglycerides. It has been shown that weight loss alone can normalize plasma triglyceride levels, and combined with a regular aerobic exercise programme, may increases HDL-C levels by 10% to 20% (NIH Consensus Conference, 1993). The exercise intervention in this study significantly increased HDL-C levels, although unequal weight losses in the experimental groups were recorded.

In this study the HDL-C level was greatly modified in both experimental groups, with a 11.3% and 13.4% increase in the E and ED groups respectively. Both E and ED groups achieved statistical significance ($P \leq 0.01$).

Five subjects attained desirable ($\geq$ .9 mmol/l) HDL-C levels following intervention, and 13 out of the 14 subjects in the ED groups and 11 out of the 15 in the E group increased their HDL-C levels to a lesser extent following the exercise and diet programme. The Helsinki Heart study showed that a mean 12% increase in HDL-C levels and 11% drop in LDL-C levels were both associated with a 34% decline in CHD events (Frick et al.,1987). A number of subjects in this study achieved these changes in both the E and ED groups (Table 4.13). The present results indicate that aerobic training of moderate intensity and frequency can influence the levels of HDL-C positively. The lowered risk ratio further supports the HDL-C results, showing a reduced risk of CHD following intervention.

The Framingham Heart Study, the Lipid Research Clinics's Mortality Follow-up Study, the Coronary Primary Prevention Trial and the Multiple Risk Factor Intervention Trial, indicated a trend that the risk of CHD decreases with 2% to 3% for every 0.03 mmol/l increase in HDL-C levels after adjustment to control for other risk factors (NIH Consensus Statement, 1993). The present study's HDL-C results show that 11 out of 15 subjects in the E group achieved $\geq$ 0.03 mmol/l increases in HDL-C levels pre to post intervention, while the ED group had 13 out of the 14 subjects achieving the $\geq$ 0.03 mmol/l level. It is however, not possible to indicate exactly what percentage decrease in CHD risk was achieved because adjustments to other risk factors were not made in this study.

The possible mechanism by which HDL-C may reduce the risk of CHD, is through the process of reverse cholesterol transport and this theory is supported by animal studies (Berger, 1984). This hypothesis states that HDL-C may prevent the entry of cholesterol into

the process of atherogenesis or even remove cholesterol from the atherosclerotic lesions.

The concentrations of HDL-C are known to be modifed by exercise or by accompanying changes in lifestyle such as consumption of alcohol, which can raise HDL-C, while cigarette smoking may have the opposite effect (Johansson and Medhus, 1974 and Garrison et al.,1978). The alcohol consumption during this study decreased in the experimental groups (12.9g to 10.5g) although such alcohol reductions were not found to be statistically significant in either of the experimental groups. Smoking, on the other hand remained constant throughout the study period, while an increase in HDL-C levels still occurred.

The present study's results indicate a significantly decreased risk ratio (total cholesterol to HDL-C ratio) in the ED group as compared to the other two groups of subjects.

These results are associated with increased HDL-C levels and possibly decreased body weight with small changes in total cholesterol. This further indicates a reduced risk for CHD in this subject population.

4.2.2.2 **Dietary aspects**

Substantial changes in diet were recorded in the experimental groups with fewer significant changes in the control group. The greatest changes occurred in the ED group, which was to be expected (Table 4.14). These changes may be attributed to the general sensitization of all groups as a result of their interaction with the dietician, although the E and control groups were asked not to alter their diets in any way.

All three groups showed significant decreases in energy intake. However, the ED group registered the greatest percentage decrease (-19%) when compared to the other two groups (-14.86%). This larger decrease in caloric intake in the ED group may have contributed to their significant body weight loss over the study period. This can be explained by the lower saturated fat intake values of the ED group, as it was part of the dietary modification. The dietary records of the different sub-groups indicated that although all groups showed changes in their food intake the ED group's pattern of alteration reflected consistently higher percentage changes following intervention than the E or control group. This was due to the fact that the ED group underwent definite dietary modification when compared to the other two groups. The E group registered significant differences between the means of pre to post evaluations for protein ($P \leq 0.05$, $t = 3.01$), total fat ($P \leq 0.05$, $t = 3.78$), monosaturated fats ($P \leq 0.05$, $t = 2.47$), polyunsaturated fats ($P \leq 0.05$, $t = 2.54$), saturated fats ($P \leq 0.05$, $t = 3.85$), carbohydrates ($P \leq 0.01$, $t = 2.68$), dietary fat ($P \leq 0.05$, $t = 2.53$) and dietary cholesterol ($P \leq 0.05$, $t = 3.3$). The ED group recorded significant differences between the means of pre to post evaluations for dietary intake in the following nutrients: protein ($P \leq 0.05$, $t = 4.37$), total fat ($P \leq 0.01$, $t = 4.69$), monosaturated fats ($P \leq 0.01$, $t = 4.81$), polyunsaturated fats ($P \leq 0.05$, $t = 1.71$), saturated fats ($P \leq 0.01$, $t = 5.76$), carbohydrates ($P \leq 0.01$, $t = 2.81$), dietary fibre ($P = 0.59$, $t = 2.07$), dietary cholesterol ($P \leq 0.01$, $t = 5.13$) and P:S ratio ($P \leq 0.05$, $t = 3.12$).

Table 4.14 **Post Intervention Changes in Nutritional Components (mean, SD and % change)**

Parameter	Exercise Only			Exercise and Diet			Control		
	Pre	Post	%	Pre	Post	%	Pre	Post	%
Energy (kcal)	2880 (638)	2456* (463.7)	-14.7	2388 (525)	1920* (500.7)	-19.6	2864 (555)	2435* (633)	-14.9
Protein (g)	80.4 (18.2)	68.8* (15.9)	-14.4	74.5 (20.6)	60* (18.2)	-19.5	82.7 (22.6)	74.8 (21)	-9.6
Total fat (g)	121.9 (36.2)	101.7* (23.7)	-16.6	107.7 (32.4)	81.7* (30.1)	-24.1	143.9 (47.2)	112.0 * (37.4)	-22.2
Monounsaturated fats (g)	38 (11.3)	32.8* (8.9)	-13.7	34 (13.1)	22.1* (9.1)	-35.0	45.5 (17.4)	33.4* (15.5)	-26.6
Polyunsaturated fats (g)	41.1 (17.3)	33.0* (10.4)	-19.7	33.4 (10.9)	28.7* (11.4)	-14.0	51.1 (17.7)	35.8* (12.9)	-29.9
Saturated fats (g)	34.1 (11.1)	27.6* (8.7)	-19	29.7 (9.1)	19.6* (8.8)	-33.8	35.6 (15.1)	29.1* (12.9)	-18.1
Total carbohydrates (g)	347.8 (79.1)	296.2* (51.1)	-14.8	263.4 (68.1)	216.2* (53.6)	-17.9	295.3 (68.1)	262.2 * (68.2)	-11.2
Dietary fibre (g)	20.8 (6.4)	20.4* (5.7)	-1.9	24.1 (6.6)	28.7* (8.0)	+19	19.5 (6.3)	20.0 (5.6)	+2.6
Dietary cholesterol (mg)	276.9 (91.2)	226.3* (92.3)	-18.2	230.4 (124)	135.7* (98.9)	-41.0	311.1 (141. 2)	261.5 (89.9)	-15.9
P:S ratio	1.24 (0.41)	1.3 (0.58)	+4.8	1.19 (0.42)	1.6* (0.42)	+34	1.56 (0.68)	1.38 (0.69)	-11.5
Alcohol intake (g)	11.33 (12.7)	10.8 (10.3)	-4.7	14.6 (8.9)	10.3 (2.17)	-29.5	12.00 (4.6)	8.7 (5.02)	-27.5

* = significantly different ($P \leq 0.05$).

The control group registered mean significant differences between pre to post-evaluations in dietary components for the following: total fat ($P \leq 0.05$, $t = -1.46$), monounsaturated fats ($P \leq 0.05$, $t = 2.56$), polyunsaturated fats ($P \leq 0.05$, $t = 3.69$), saturated fats ($P \leq 0.05$, $t = 2.29$) and total carbohydrates ($P \leq 0.05$, $t = 1.51$). The ED group, however, showed greater percentage profile changes in several dietary components. These were protein, energy, total fat, saturated fats, total carbohydrate, dietary fibre, dietary cholesterol and P:S ratio, which indicates definite changes in dietary intake compared to the other two sub-groups.

There is widespread agreement that the habitual dietary pattern of a population exerts a major influence on the prevalence and incidence of CHD (Keys, 1970; Masironi, 1970; Stamler, 1982; Brown, 1990). Increased intake of saturated fats, cholesterol and calories are uniformly associated with higher blood cholesterol levels, obesity and higher frequency of myocardial infarction and death from CHD (Frank et al.,1986; Stephen and Wald, 1990).

The effect of dietary fat on HDL-C has been intensively studied, primarily because of the empirical association between low HDL-C and increased risk of CHD. In populations with low intake of fat, both plasma LDL-C and HDL-C concentrations are low (Sacks et al.1975, Ornish et al., 1990) whereas in societies where the intake of saturated fat is high, both LDL and HDL levels tend to be high (Mensink and Katan, 1989 and 1990; McMurray et al., 1991). Polyunsaturated fat, but not monounsaturated fat tends to lower HDL-C (Ornish et al., 1990).

Weight loss is also linked to good dietary habits and exercise. Quantifying and characterizing a person's eating patterns is however a difficult task, as the various dietary techniques used to assess long term dietary intake all have inherent limitations (Pekkarinen, 1970; Sorenson, 1982). This study used the seven-day dietary history method which provided the researcher with a method of evaluating the dietary components at the commencement of the study, and for the re-examination of eating behaviour following the intervention programme. This method was also useful in identifying the changes that were required among the ED group for purposes of dietary modification. In this study the addition of a prudent low fat diet, together with the moderate aerobic exercise programme of the ED group, resulted in major reductions in energy intake protein, saturated fatty acids, dietary cholesterol and alcohol consumption, with increases in dietary fibre and P:S ratio.

Although there were no stipulated dietary changes in both the E and C groups, the changes were not as consistent as those in the ED group. The changes experienced by the E and C groups may have been due to the sensitization effect resulting from the personal dietary interviews with the dietician, even though they were requested not to effect any changes to their dietary patterns.

The substantial weight losses within the ED group were accompanied by a 3.16% mean cholesterol decrease. This study further showed that large individual body weight decreases are likely to be accompanied by similar reductions in cholesterol. A longer period of exercise ($\pm$ 12 months) may have produced greater serum cholesterol reductions (Wood et al.,1991). The mean dietary changes that occurred in the ED group following the intervention were

statistically significant ($P \leq 0.05$) compared to the E group (Table 4.12). This was true for caloric intake, total fat, monounsaturated fats, saturated fats, total carbohydrate and dietary fibre, and appears to be associated with the greater weight loss achieved within the ED groups. Even though the lipid results were similar in both experimental groups after intervention, it may be concluded that the greater reduction in total body weight (mean = 3.17 Kg) among the ED group had a positive impact on the reduction of cholesterol as compared to the E group, with it's lower total weight loss (mean = .9 Kg). The dietary modification in the ED group was also effective in the total decrease in body weight, impacting positively on morphological characteristics.

Large population studies shows the link in changes in dietary habits with elevations in plasma cholesterol levels and with the incidence of CHD. One such study correlates the incidence of CHD with increased dietary lipid intake and elevated serum cholesterol levels in Japanese men who migrated from Japan to Hawaii and California (Kato et al., 1973 and Kagan, et al.,1974). This study began in 1965, and indicates that as Japanese men are assimulated into the western society of the USA, they increase their intake of animal products, including total fat, saturated fat and dietary cholesterol. Associated with the dietary changes there is an increase in serum cholesterol levels and an increase in CHD (Kagan, et al.,1974). This increase in CHD risk is also seen in another classical study by McMurray et al. (1991), who fed the Tarahumara Indians of Mexico, with a high fat, high cholesterol western diet for a period of time in order to mimic the changes that would occur when a traditional low fat diet is replaced by a high fat hypercaloric diet. The results were rapid, and the subjects gained 3.8 Kg over a 5 week period. In particular, the level of LDL-C rose dramatically, but there

were also increases in total plasma cholesterol, HDL-C, triglycerides and VLDL triglycerides (McMurray et al., 1991). This study reflects the trend that may occur in dramatic changes in eating patterns, and this may produce overt hyperlipidaemia over many decades.

Similarly, the Indian population of South Africa who were basically vegetarian when arriving in this country in 1860, gradually changed their eating patterns to that of a more western diet (more saturated fats and dietary cholesterol) which may be implicated as one of the risk factors in the increase in the incidence of CHD. Now over 134 years later, the South African Indian highly westernized eating and lifestyle habits, have escalated the incidence of CHD to almost the highest in South Africa.

Very recently studies by Ornish, et al.(1990;1993) and Watts, et al.(1992) demonstrated that a very low fat vegetarian diet and modest exercise can reduce the severity of lesions within the coronary arteries, in patients who had significant CHD and evidence of exertional ischaemia. These findings support the hypothesis that atherosclerotic lesions are responsive to change with correct lifestyle management. This further emphasises the effects of a low fat diet on the reversal of CHD, which were the eating patterns of the early Indian settlers in South Africa, due to religious reasons.

The effects of alcohol intake in the retardation of CHD has been a focus for a number of studies (Johansson and Medhus, 1974; Garrison et al.,1978; and Renaud and de Lorgeril, 1992).

In France a paradox occurs, in that there is a high intake of saturated fat but a low mortality from CHD. This paradox may be attributed to the high wine consumption. Alcohol is believed to reduce the risk for CHD by the action of HDL-C, but the levels of this factor is no higher in France than in other countries. Renaud and co-workers, critically evaluated data from Wales, Scotland and France and concluded that moderate intake of alcohol does not prevent CHD by affecting HDL-C levels, but rather through the inhibition of platelet aggregation and reactivity (Renaud et al., 1985; Renaud et al.,1992 and Renaud and de Lorgeril, 1992).

The authors confirmed that the platelet reactivity was lower in France than in Scotland (Renaud et al.,1985). This hypothesis may explain the "French Paradox" which is a high saturated fat diet and a moderate intake of alcohol with reduced incidence of CHD. In the studies discussed the mean alcohol intake was 20g - 30g per day as compared to the mean of 12.6g per day in the present study. It may be postulated that the impact of the alcohol intake in this study may have had minimal effects on the overall results, if compared to the French intake (20g - 30g per day) (Renaud and de Lorgeril, 1992).

In summary it appears that the amount and type of dietary fat have profound effects on plasma lipids and lipoproteins and these effects may explain many of the influences lipids have on risk factors for several major diseases in affluent societies (Norum, 1992). In prescribing a prudent and safe diet for populations, less than 30% of the food energy should come from fat and no more than 10% of the energy from saturated fat (Heart Foundation of South Africa). The combined effects of a prudent diet, maintenance of ideal body weight and

appropriate exercise can favourably influence CHD risk in normal individuals. Dietary modification is a major part for lifestyle management in order to reduce the risk for CHD.

4.2.3 **Statistical Comparison of Experimental Groups**

Having examined the effects of the intervention programme on each of the experimental groups, this section now compares the t-values of each group for each of various physiological and biochemical evaluations which were conducted before and after the intervention programme.

Table 4.15 **Pre-post Intervention Comparison of t-test Results for Exercise (E) group and Control (C) group.**

PRE-INTERVENTION		POST-INTERVENTION	
Flexibility	$P \leq 0.05$*	BMI	$P \leq 0.05$
Push-ups	$P \leq 0.05$	% Fat	$P \leq 0.001$
Sit-ups	$P \leq 0.001$	Flexibility	$P \leq 0.05$
		VO_2 (ml/kg/min)	$P \leq 0.001$
		Push-ups	$P \leq 0.001$
		Sit-ups	$P \leq 0.001$
		Ventilation	$P \leq 0.05$
		VO_2 (1/min)	$P \leq 0.01$

*All values statistically significant in favour of E group.

When examining the results of the comparison of the scores obtained by the E and control groups prior to the intervention programme, statistically significant differences occurred with regard to flexibility, push-ups and sit-ups (Table 4.15). However, after the intervention, further significant changes occurred in BMI, % fat, flexibility, peak VO_2 max, push-ups, sit-ups and ventilation. These mean significant differences indicate the training effect of the exercise programme on the E group, when compared to the control group. This was however, expected as the control group maintained their sedentary status.

Table 4.16 **Pre-post Intervention Comparison of t-test Results for Exercise and Diet (ED) Group and Control (C) Group.**

PRE-INTERVENTION		POST-INTERVENTION	
HDL-C	$P \leq 0.05$*	% Fat	$P \leq 0.05$
Risk ratio	$P \leq 0.05$	HDL-C	$P \leq 0.01$
Sit-ups	$P \leq 0.05$	VO_2 (ml/kg/min)	$P \leq 0.001$
Triglycerides	$P \leq 0.05$	Pull-ups	$P \leq 0.05$
Kcal	$P \leq 0.05$	Risk ratio	$P \leq 0.001$
Total fat	$P \leq 0.05$	Cholesterol	$P \leq 0.001$
Polyunsaturated	$P \leq 0.05$	Sit-ups	$P \leq 0.001$
		Triglycerides	$P \leq 0.001$
		VO_2 (l/m)	$P \leq 0.05$
		Kcal	$P \leq 0.05$
		Total fat	$P \leq 0.05$
		Monounsaturated	$P \leq 0.05$
		Saturated fat	$P \leq 0.05$
		Dietary fibre	$P \leq 0.05$
		Dietary cholesterol	$P \leq 0.05$

*All values statistically significant in favour of ED group.

When the t-test results of the ED and C groups were compared (pre-intervention), significant differences were recorded in favour of the ED group in seven indices, only one of which, was fitness related (sit-ups) (Table 4.16).

Following the intervention programme, the ED group achieved significantly better results on 15 indices, 4 of which were fitness related. These results were not unexpected, as the ED group included both exercise and diet in their intervention programme.

The larger number of statistically significant differences favouring the ED group, included three new fitness parameters (VO_2ml/kg/min, VO_2 l/min, and pull ups). Among the biochemical parameters six were not previously significantly better. These were % fat, cholesterol, monosaturated fat, saturated fat, dietary fibre and dietary cholesterol. The level of significance also improved for HDL-C, risk ratio and triglycerides.

Table 4.17 **Pre-post Intervention Comparison of t-test Results for Exercise (E) Group and Exercise and Diet (ED) Group.**

PRE-INTERVENTION			POST-INTERVENTION		
Kcal	$P \leq 0.05$	(ED)	Flexibility	$P \leq 0.05$	(E)
Total carbohydrate	$P \leq 0.05$	(ED)	Kcal	$P \leq 0.05$	(ED)
			Total fat	$P \leq 0.05$	(ED)
			Monounsaturated	$P \leq 0.05$	(ED)
			Saturated fat	$P \leq 0.05$	(ED)
			Total carbohydrate	$P \leq 0.01$	(ED)
			Dietary fibre	$P \leq 0.05$	(ED)

( ) statistically significant in favour of the group.

Few pre-intervention statistical differences between the baseline values of the E and ED groups were recorded when compared to the post intervention phase. Predictably, the ED group demonstrated a more positive response to the intervention programme with respect to nutritional components. The E group however demonstrated higher flexibility scores. The exercise intervention programme induced similar increases in fitness for both E and ED groups, demonstrating its effectiveness.

Despite the fact that the ED group was given the additional intervention strategy of dietary change, it appears that the post intervention results were similar in both E and ED groups in many respects, but significantly different from those of the control group (Tables, 4.15 and 4.16). The statistically significant changes ($P \leq 0.05$) between the means of the E and

ED group (Table 4.17) were mainly restricted to dietary changes, following the intervention programme, which was to be expected as a conscious effort was made by the ED group to modify their eating habits.

In concluding this chapter, it may be important to note that the combined effects of a healthy diet, maintenance of ideal body mass and moderate exercise can positively alter lipid and lipoprotein profiles and decrease the risk of CHD in normal sedentary individuals. The mechanism by which diet and exercise may influence lipids and lipoproteins is still not precisely understood. Further investigation is required for precise dietary and exercise guidelines which will alter lipids and lipoproteins positively.

CHAPTER FIVE

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5. CONCLUSIONS AND RECOMMENDATIONS

5.1 CONCLUSIONS

Following the analysis of the results the following conclusions may be drawn:

5.1.1 Although the sample selected for this study was limited to males, the data supports the available evidence indicating that within the Indian community (as in other South African communities) a large number of people have an increased risk of premature CHD.

5.1.2 Fifteen weeks of intervention induced significant changes in anthropomorphic indices, which are compatible with the protective effect of moderate physical activity. An increased level of fitness, was accompanied by a decrease in obesity.

5.1.3 Significant changes in peak oxygen uptake values and percent body fat occurred in both the E and ED groups, following the intervention programme. The subjects thus became leaner and fitter after the intervention programme.

5.1.4 Significant mean increases in HDL-C levels were observed in both the experimental groups, contributing to a lowered CHD risk.

5.1.5 The ratio of TC/HDL-C improved in both intervention groups, also impacting on reduced risk for CHD.

5.1.6 Finally, these data suggest that a regular moderate aerobic exercise programme, supplemented by dietary changes, may be effective in reducing the risk of heart disease in the Indian South African population.

5.2 RECOMMENDATIONS

Based on the results and conclusions derived from this study, the following recommendations appear to be warranted:

5.2.1 The value of exercise as a preventive measure in the management of lifestyle disease should be formally recognized in the development of a primary health care strategy in South Africa.

5.2.2 The following guidelines are recommended when prescribing exercise, with a view to enhancing serum lipid and lipoprotein profiles:

Mode of Exercise	:	aerobic in nature
Intensity of Exercise	:	45% to 75% of VO_2 max
Frequency of Exercise	:	a minimum of three days per week
Duration of Exercise	:	30 minutes per session

Length of Programme : 15 weeks or more

Additional Intervention : dietary modification with a prudent low fat diet

5.2.3 In that the quantity and nature of dietary fat consumed by an individual has been shown to effect serum lipid and lipoprotein profiles, this study supports a prudent, safe diet comprising a daily intake of 30% or less fat, of which 10% should be saturated fat, 10% monounsaturated fat and 10% polyunsaturated fat.

5.2.4 The importance of health education should be emphasized as a primary health care mechanism. The acceptance of responsibility for those aspects of personal health which can be influenced by health behaviour should be included in primary and high school curricula.

5.2.5 Screening programmes for heart disease should be more readily available to the general public, hence encouraging preventive health behaviour among adult South Africans.

5.2.6 Further research, exploring the role of exercise in the prevention of heart disease appears justified.

5.2.7 The effects of exercise programmes of longer duration and of varying intensity merit further study.

5.2.8 Similar research on the female population of South Africa would contribute to a more complete understanding of the role of exercise in the prevention of heart disease.

5.2.9 Further research should endeavour to match the samples according to age, body weight, fitness level and pre-training lipid and lipoprotein profiles.

5.2.10 A more stringently controlled dietary programme should be introduced in order to determine the relative contribution of the dietary variables in the reduction of CHD risk.

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APPENDICES

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7. APPENDICES

Appendix 1

CALL FOR RESEARCH PARTICIPANTS

Dear Member of Staff

I am conducting a study on the effects of an aerobic exercise programme on the serum-lipid levels (cholesterol, triglycerides and others) of **sedentary Indian Males**. This group has been identified as being particularly susceptible to coronary heart disease (Seedat et al., 1990).

This incidence of coronary heart disease (CHD) is cause for serious concern and there is reason to believe that primary intervention is urgently required. As the lack of exercise has been identified as one of the major causes of CHD (Wyndham, 1981; Paffenbarger, 1984; Morris et al., 1990), this study will introduce this variable into the lifestyle of the selected sample.

Subjects will be required to join an exercise group 3 times per week during lunch breaks for a period of 15 weeks.

The exercise programme will be tailored to individual needs and will be carefully supervised. It will include circuit training, walking, jogging and some games.

The study is being conducted by the Department of Human Movement Studies in collaboration with the Department of Chemical Pathology at the University of natal.

May I consequently invite any interested members of staff who fall into this sedentary category, to please contact **Alershnee Reddy at 2394 before Friday, 29 November.**

Should further details be required you may also phone me at 2393. As this programme holds positive benefits for those who join I would encourage participation.

Enjoy the festive season before the hard work begins!

Yours sincerely

YOGA COOPOO
(Department of Human Movement Studies)

Appendix 2

SERUM LIPID RESEARCH PROJECT

PERSONAL DETAILS

DATE OF FIRST VISIT : _____

1. SURNAME: _____ 2. FIRST NAME : _____

3. DATE OF BIRTH : _____ 4. SEX : _____

5. HOME PHONE : _____ WORK PHONE : _____

6. ADDRESS : _____

7. CITY : _____ CODE : _____

8. FAMILY PHYSICIAN : _____

9. PHYSICIAN'S PHONE HOME : _____

SURGERY : _____

10. PROFESSION : _____

11. NAME OF NEXT OF KIN : _____ PHONE : _____

12. ARE YOU ON ANY FORM OF MEDICATION PRESENTLY, IF SO, NAME THE MEDICATION AND THE REASON FOR USE : _____

_____

13. WHEN LAST DID YOU SEE YOUR DOCTOR, AND WHY ? _____

_____

14. Answer the following questions about yourself

YES	NO		
____	____	1.	Has your doctor ever said you have heart trouble?
____	____	2.	Do you frequently have pains in your heart and chest?
____	____	3.	Do you often faint or have spells of severe dizziness?
____	____	4.	Has a doctor ever said your blood pressure was too high?
____	____	5.	Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
____	____	6.	Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
____	____	7.	Are you over age 65 and not accustomed to vigorous exercise?

INFORMED CONSENT

1. PURPOSE OF RESEARCH STUDY

Physical activity is an important aspect of health promotion and disease prevention. It has a role to play int he primary prevention of coronary heart disease. Exercise also appears to improve some aspects of mental health and emotional well-being as well as delaying the more general processes of ageing. Scientific data have supported the legitimacy of developing orchestrated efforts to promote regular physical activity among the broad spectrum of the population as an aid to chronic disease prevention and control.

Sedentary Indian males were selected for this study, because of the existence of many risk factors associated with CHD in this community. It will be determined whether changes in the levels of serum lipids, such as cholesterol, triglycerides, will result following an exercise programme. Exercise fitness parameters will be evaluated and finally the response to fat absorption and digestion will be measured before and after the exercise programme.

2. TESTING

In order to assist in the assessment of cardiovascular function, body composition and other physical fitness components, the undersigned hereby voluntarily consents to engage in one or more of the following tests;

- Graded exercise test to determine aerobic capacity
- Skinfold measurements
- Muscle fitness tests
- Flexibility tests
- Blood tests
- Exercise Prescription

3. EXPLANATION OF THE TESTS

The graded exercise test is performed on a bicycle ergometer or a treadmill. The work load is increased every few minutes until exhaustion or until other symptoms dictate termination of the test. The test may be terminated at any time if fatigue or discomfort require it.

For muscle fitness testing, weights are lifted for a number of repetitions using barbells or exercise machines. These tests assess the strength and endurance of the major muscle groups in the body. For evaluation of flexibility, a number of calithenic exercises are performed. During these exercises, the range of motion in various joints will be measured.

Blood samples will be taken by qualified personnel in order to determine the various biochemical parameters.

4. RISKS AND DISCOMFORTS

During the graded exercise test, certain changes may occur. These changes may include abnormal blood pressure responses, fainting and irregularities in heartbeat. Every effort will be mae to minimize these occurrences. Trained personal will be alerted to deal with unusual situations which may arise during the testing and the exercise programme.

There is a slight possibility of damage to muscles or ligaments during the muscle fitness and flexibility testing. In addition, you may experience muscle soreness 24-48 hours after testing. These risks and discomforts may also be experienced during the prescribed exercise programme.

*The Post heparin test } See attached appendix
*The Standard fat meal test } See attached appendix

5. EXPECTED BENEFITS FROM TESTING

These tests permit the scientific assessment of physical working capacity and the clinical appraisal of physical fitness status. The results will be used to prescribe safe, sound exercise programmes. Records will be kept strictly confidential unless you consent to the release of this information. No assurance can be given that the exercise programme will increase your functional capacity, although evidence indicates that improvement usually results.

6. RESPONSIBILITY OF THE PARTICIPANT

To gain the expected benefit, priority must be given to regular attendance and adherence to prescribed intensity, duration, frequency, progression and type of activity.

To achieve the best possible preventive health care:

DO NOT:

A. Withhold any information pertinent to symptoms from the exercise specialist, exercise programme director, or other professional personnel.
B. Exceed your target heart rate.
C. Exercise when you do not feel well.
D. Exercise within 2 hours after eating.
E. Exercise after drinking alcoholic beverages.
F. Use extremely hot water during showering after exercise (stay out of sauna, steam bath and similar extreme temperatures).

DO:

A. Report any unusual symptoms which you experience before, during or after exercise, or you notice in an exercising colleague.
B. If you plan to use other facilities at the site, please inform the exercise specialist. At that time you must accept responsibility for yourself and exercise at your own risk.

7. DURATION OF THE PROGRAMME

The exercise programme will continue for 15 weeks, with expert exercise supervision 3 days per week for approximately 35-45 mins per exercise session.

8. ENQUIRIES

Questions about the procedures used int he physical fitness tests are encouraged. If you have any questions or need additional information, please ask for further explanation.

9. FREEDOM OF CONSENT

Your permission to perform these tests and participate in the programme is strictly voluntary. You are free to deny consent if you so desire.

I have read this information carefully and fully understand the testing and exercise programme procedures. I consent to participate in the testing the Exercise Programme.

....
SIGNATURE OF PARTICIPANT DATE

.....
WITNESS

QUESTIONS:

.....

.....

RESPONSE:

.....

HEALTH AND FITNESS APPRAISAL

MEDICAL HISTORY QUESTIONNAIRE

Date : .....

SECTION A

1. When was the last time you had a physical examination?.....

2. Are you allergic to any medications, foods, or other substances, name them.....

   .....

3. Do you have any chronic or serious illnesses, name them. eg Heart disease, diabetes : .....

4. Give the following information pertaining to the last three times you have been hospitalized :

	Hospitalization Number 1	Hospitalization Number 2	Hospitalization Number 3
Type of operation or illness			
Month and year hospitalized			
Name of hospital			
City			
Surgeon			
Physician			

SECTION B

During the past twelve months .....

		YES	NO
1.	Has a physician prescribed any form of medication for you.	___	___
2.	Has your weight fluctuated more than a few kilograms?	___	___
3.	Did you attempt to bring about this weight change through diet and/or exercise?	___	___
4.	Have you experienced any faintness, lightheadedness, blackouts?	___	___
5.	Have you occassionally had trouble sleeping?	___	___
6.	Have you experienced any blurred vision?	___	___
7.	Have you any severe headaches?	___	___
8.	Have you experienced chronic morning cough?	___	___
9.	Have you experienced any temporary change in your speech pattern such as slurring or loss of speech?	___	___
10.	Have you felt unsually nervous or anxious for no apparent reason?	___	___
11.	Have you experienced unusual heartbeats such as skipped beats or palpitations?	___	___
12.	Have you experienced periods in which your heart felt as though it were racing for no apparent reason?	___	___

At present .....

		YES	NO
1.	Do you experience shortness of breath or loss of breath while walking with others of your own age?	___	___
2.	Do you experience sudden tingling, numbness, or loss of feeling in your arms, hands, legs, feet, or face?	___	___
3.	Have you ever noticed that your hands or feet sometimes feel cooler than other parts of your body?	___	___
4.	Do you experience swelling of your feet and ankles?	___	___
5.	Do you get pains or cramps in your legs?	___	___
6.	Do you experience any pain or discomfort in your chest?	___	___
7.	Do you experience any pressure or heaviness in your chest?	___	___
8.	Have you ever been told that your blood pressure was abnormal?	___	___
9.	Have you ever been told that your serum cholesterol or triglyceride level was high?	___	___

10. How often would you characterize your stress level as being high? _____ Occassionally _____ Frequently _____ Constantly

11. Have you ever been told that you have any of the following illnesses?

_____ Myocardial infarction
_____ Heart attack
_____ Coronary thrombosis
_____ Heart disease
_____ Coronary occlusion
_____ Heart murmur
_____ Asthma
_____ lower back pain
_____ Arthritis
_____ Arteriosclerosis
_____ Heart block
_____ Rheumatic heart
_____ Aneurysm
_____ Angina
_____ Heart failure
_____ Diabetes
_____ High blood pressure
_____ High cholesterol levels

SECTION C

1. Has any member of your immediate family been treated for or suspected having had any of these conditions? Please identify their relationship to you (father, mother, sister, brother, etc)

	Parents		Siblings		Grandparents	
	Father	Mother	Brother	Sister	Grand-mother	Grand-father
A. Diabetes						
B. Heart disease						
C. Stroke						
D. High blood pressure						
E. Raised cholesterol levels						

2. If yes, how long ago did this condition start? .....

.....

After : American College of Sports Medicine

LIFE STYLE EVALUATION

(after Corbin & Lindsey 1985)

SMOKING HABITS

1. Have you ever smoked cigarettes, cigars or a pipe? Yes _____ No _____

2. Do you smoke presently? Yes _____ No _____

   Cigarettes_____per day.

   Cigars _____ per day.

   Pipefuls _____ per day.

3. At what age did you start smoking? _____ years.

4. If you have quit smoking, when did you quit? _____

DRINKING HABITS

1. During the past month, how many days did you drink alcoholic beverages?

   _____ days

2. During the past month, how many times did you have five or more drinks per occassion?

   _____ times

3. On the average, how many glasses of beer, wine, spirits do you consume per week?

   Beer _____ glasses or cans

   Wine _____ glasses

   Spirits _____ glasses

   Other _____ glasses

EXERCISE HABITS

1. Do you exercise vigorously on a regular basis? **Yes** _____ **No** _____

2. What activities do you engage in on a regular basis?

_____

3. How many minutes on the average is each of your exercise workouts?

_____ minutes

4. How many workouts per week do you participate in on the average?

_____ workouts

5. Is your occupation :

_____ Inactive (e.g., desk job)

_____ Light work (e.g. housework, light carpentry)

_____ Heavy work (e.g. heavy carpentry, lifting)

6. Check those activities that you would prefer in a regular exercise programme for yourself :

_____	**Walking/running/jogging**	_____	**Tennis/badminton/squash**
_____	**Stationary running**	_____	**Soccer/Cricket**
_____	**Skipping**	_____	**Hiking/golf**
_____	**Road Cycling**	_____	**Aerobic dance**
_____	**Stationary cycling**	_____	**Others (Specify)**

DIETARY HABITS

1. What is your current weight? _____ Height? _____

2. What would you like to weigh? _____

3. What is the most you ever weighed as an adult? _____

4. What is the least you ever weighed as an adult? _____

5. What weight loss methods have you tried? _____

6. Which do you eat regularly?

_____ Breakfast	_____ Midafternoon snack
_____ Midmorning snack	_____ Dinner
_____ Lunch	_____ After-dinner snack

7. How often do you eat out per month? _____ times

8. What size portions do you normally have?

_____ Small _____ Moderate _____ Large

_____ Extra large _____ Uncertain

9. How often do you eat more than one serving ?

_____Always _____ Usually _____ Sometimes _____ Never

10. How long does it usually take you to eat a meal?

_____ minutes

11. Do you eat while doing other activities (e.g.,watching TV, reading, working)? .....

12. When you snack, how many times per week do you eat the following?

Cookies,cake,pie	_____	Candy	_____
Diet soda	_____	Soft drinks	_____
Doughnuts	_____	Fruit	_____
Milk or milk beverage	_____	Potato chips, pretzels, etc	_____
Peanuts or other nuts	_____	Cheese & crackers	_____
Ice cream	_____		
Other	_____		

13. How often do you eat dessert? _____ times per day

_____ times per week

14. How often do you eat fried foods? _____ per week

15. Do you salt your food at the table? **Yes** _____ **No** _____

_____ **Before tasting it** _____ **After tasting it**

Appendix 3

GUIDELINES FOR HEALTHY EATING

1.1 Eat less fat, particularly saturated fat.

1.2 Eat fibre rich foods.

1.3 Avoid too much salt.

1.4 Avoid too much sugar and sweetened food.

1.5 Drink sufficient water and/or liquids.

1.6 Use alcohol in moderation.

1.7 Eat a variety of foods.

1.1 Eat less fat, particularly saturated fat and cholesterol.

It is important to keep the total fat intake low, since a high intake may cause overweight and increase the blood cholesterol level.

Animal fats (excluding the fat in fish) contain a high percentage of saturated fatty acids, while plant oils (with the exception of coconut and palm kernel oil) contain more mono-unsaturated and polyunsaturated fatty acids, which have a beneficial effect on blood cholesterol.

When making food choices it is therefore necessary to bear in mind not only the fat content, but also the type of fat a food contains.

General Rules

- Cut off all visible fat before cooking.
- Use only lean cuts of meat.
- Keep portions small.
- Use chicken (without skin) and fish regularly.
- Limit the use of foods with 'hidden' fat, such as fried foods, processed sausages, polony, baked goods and pastries.
- Use skim milk or low fat milk and dairy products.
- Avoid coffee creamers and blends which contain coconut oil or palm kernel oil (high in saturated fat).
- Use polyunsaturated margarine (tub margarine) as a spread for bread, but spread thinly.
- Use sunflower oil in the preparation of food. Use very little oil when frying food.
- Skim fats off sauces, soups, stews and curries, refrigerating after cooking will allow you to remove hardened saturated fats.

1.2 Eat fibre rich foods

Fibre is the part of plant foods that cannot be completely digested. The fibre in whole-grain products helps to combat constipation, while the fibre in fruits, vegetables, dry legumes and oats has a beneficial effect on blood cholesterol levels.

1.3 Avoid too much salt (sodium)

The sodium in table salt may have a detrimental effect on high blood pressure (hypertension). The body needs very little sodium in addition to the sodium occurring naturally in milk, eggs, meat, fish and vegetables.

- Use less salt in the preparation of food
- Do not add salt at the table.
- Limit the use of commercial products (vienna sausages, biltong, salted fish, canned meat) salty biscuits, salted nuts, crisps, cheese, packet soups, Worcester sauce, tomato sauce, stock powder and cubes).
- Avoid flavoured salts eg. onion, garlic, celery salts and monosodium glutamate as they all contain sodium.
- Use herbs and spices to make meals tasty.

1.4 **Avoid too much sugar and sweetened foods**

The balance of the eating pattern is disturbed when too many products with a high sugar content, such as cake, biscuits, puddings, cold drinks and sweets are eaten.

White and brown sugar, honey and molasses are simple sugars that provide energy but very few additional nutrients.

1.5 **Drink sufficient water and/or other liquids.**

A human being is able to survive for a longer period without food than without water. Continuous replenishment of water loss is essential.

In South Africa's relatively hot climate it is important to drink enough water and other liquids (6 - 8 glasses) daily.

1.6 **Use alcohol in moderation**

A moderate intake of alcohol is allowed. The nutritional value of alcohol is insignificant, whereas the energy value is high. The use of alcohol therefore increases the total energy intake which may in turn contribute to overweight.

1.7 **Eat a variety of foods**

Food can be divided into 5 groups in order to simplify the compilation of a balanced diet :

1. Milk and milk products.
2. Meat and meat substitutes.

3. Fruit and vegetables.
4. Grains and grain products.
5. Fats and oils.

Each group will include a variety of foods. The composition of the foods within a group is sufficiently similar for them to be used a s substitutes. For a balanced diet the correct quantities (portion/exchanges) should be chosen from each group daily.

EXCHANGES/PORTIONS

The number of portions or exchanges allowed from each food group are given below. You may choose any combination of exchanges to work out your daily meal plans, but don't exceed the number allowed according to your category. The sample menu below shows how much variety you can achieve by choosing creatively from each food group.

Exchanges allowed from each group daily

	Food Group	**Exchanges/Portions**
List 1	Milk and milk products	2
List 2	Meat and meat substitutes	4
List 3	Fruit and Vegetables	4 3+
List 4	Grains and Cereals	6
List 5	Fats and oils	4

MENU PLAN	SAMPLE MENU
Breakfast	
1 fruit exchanges	125 ml unsweetened orange juice,
2 grain exchanges	2 slices wholewheat bread
1 fat exchange	5 ml PU margarine
Lunch	
1 meat exchanges	30 g tuna
2 grain exchanges	2 slices wholewheat bread
1 fat exchanges	5 ml PU margarine
1 vegetable exchanges	125 ml lettuce/tomato/cucumber
1 fruit exchange	1 apple
Supper	
3 meat exchanges	90 g chicken curry (no skin)
2 fat exchange	10 ml oil for cooking
2 grain exchanges	250 ml rice
2 vegetable exchanges	125 ml green beans
	125 ml carrots
2 fruit exchanges	250 ml fruit salad
½ milk exchange	125 ml FF yogurt
1½ milk exchanges	milk in tea and coffee during the day.

List 1 MILK AND MILK PRODUCTS — 2 PORTIONS/DAY

Skim milk, fresh	250 ml
Skim milk, powdered	25 g
* Fat free yogurt	250 ml
* Fat free cottage cheese	45 g

* May be used as meat alternatives (in the same amounts).

List 2 MEAT AND MEAT SUBSTITUTES — 4 PORTIONS/DAY

* Beef, mutton, pork, all visible fat removed, only lean cuts	30 g (1 thin slice)
* Egg (limit to 2 week)	1
Fish, fresh, frozen or canned (drained)	30 g
Legumes - dried beans, dried peas, lentils	80 ml (1/3 cup)
- dahl	125 ml (1/2 cup)
- Toppers (cooked)	60 g (1/4 cup)
- Soya chunks (cooked)	60 ml (1/4 cup)
* Peanut butter	15 g (3 t)
Poultry (without skin)	30 g (1 drumstick/thigh)
Veal	30 g
Venison	30 g

* NB. Restrict

- Red meat — maximum 3 times per week (do not exceed a 90 g portion)
- Egg yolk — maximum 2 per week
- Peanut butter — maximum 2 times per week

List 3 FRUIT AND VEGETABLES

- **VEGETABLES** — **4+ PORTIONS/DAY**

Vegetables, no added fat	125 ml (½ cup)
Vegetables, high in carbohydrate eg. Potatoes, mealies etc.	see grains and cereal list

Apple, fresh	1 small
Apricots, fresh	3 medium
Banana, fresh	1 small / ½ large
Fruit salad, unsweetened	125 ml (½ cup)
Granadillas	6 medium
Grapefruit	1 small / ½ large
Grapes	10 large / 15 small
Guavas, fresh	2 medium
Kiwifruit	1 medium
Lychees	8
Mango	½ medium
Melon - cantaloupe (spanspek)	1/3 medium(1 c)
- honeydew (sweet melon)	½ medium (1 c)
Naartjie (mandarin)	2 medium
Nectarine	1 medium
Orange	1 small
Pawpaw	1/3 medium (1 c)
Peach, fresh	1 medium
Pear, fresh	1 small
Pineapple, slices (1 cm)	3 slices
Plums	2 medium
Strawberries	300 ml (1 ½ c)
Watermelon	1 medium slice

• DRIED FRUIT

Apple	5 rings
Apricots	8 halves
Currants, raisins, sultanas	15 ml (1½ T)
Dates	4
Peach	2 halves
Pear	1 half
Prunes	2 - 3

• FRUIT JUICE, UNSWEETENED

Apple, apricot, grapefruit guava, orange	125 ml (½ c)
Grape, pear, peach	80 ml (1/3 c)

List 4 GRAINS AND CEREALS	6 PORTIONS/DAY
Bread, brown/wholewheat	1 slice
Bread roll, brown/wholewheat	1 small
Crackers, wholewheat eg Provita	3
Cereals : dry, unsweetened eg.	
- All Bran Flakes	125 ml (½ c)
- Cornflakes, unsweetened	125 ml (½ c)
- Pro Nutro	80 ml (1/3 c)
- Weet-Bix	1
Flour, preferable wholewheat	40 ml (3 T)
Pasta, macaroni, noodles, spaghetti (cooked) preferable wholewheat	125 ml (½ c)
Popcorn (unsweetened, cooked)	250 ml (1 c)
Porridge, cooked	125 ml (½ c)
Rice, preferably brown	125 ml (½c)
Roti (cooked with fat from fat allowance)	1 medium
Sago, tapioca (uncooked)	20 ml (1½ T)
Samp, mealie rice (cooked)	125 ml (½ c)
Starchy vegetables	
Mealie, fresh	1 small
Potato	1 medium (90 g)
Sweetcorn	80 ml (1/3 c)
Sweet potato	60 ml (¼ c)

List 5 FATS AND OILS	4 PORTIONS/DAY
Sunflower/cotton/corn/soya oil	5 ml
Margarine, polyunsaturated ('tub')	5 ml

SUMMARY

	RECOMMENDATIONS
DAIRY PRODUCTS	Skim milk or skim milk products. Avoid cream, ice cream, cheese. Substitute polyunsaturated margarine for butter
MEATS	Lean, smaller servings, remove fats. Choose fish, veal and poultry. Avoid organ meats. Avoid canned and processed meat.

EGGS	Limit to 2 yolks per week; egg white as desired.
VEGETABLES AND FRUIT	Include a wide variety.
BREADS, CEREALS (GRAINS)	Unrefined and wholewheat products.
FATS	Mono-unsaturated or polyunsaturated oils.
PROCESSED FOODS	Avoid biscuits, pastries, cakes.
SWEETS AND DESSERTS	Avoid.

	Food Group	Exchange/Portions
List 1	Milk and milk products	2
List 2	Meat and meat substitutes	2
List 3	Fruit and Vegetables	4 5+
List 4	Grains and Cereals	7
List 5	Fat	5

MENU PLAN	SAMPLE MENU
Breakfast	
1 fruit exchange	125 ml unsweetened orange juice,
2 grain exchanges	2 slices wholewheat bread
1 fat exchange	5 ml PU margarine
Lunch	
1 meat exchanges	15 g Peanut butter
2 grain exchanges	2 slices wholewheat bread
2 fat exchanges	5 ml PU margarine
	5 ml French dressing
2 vegetable exchanges	250 ml lettuce/tomato/cucumber
1 fruit exchange	1 apple
Supper	
1 meat exchange	60 ml Soya chunks
2 fat exchanges	10 ml oil for cooking
3 grain exchanges	250 ml rice
	90 g potato
3 vegetable exchanges	variety of vegetables
2 fruit exchanges	250 ml fruit salad
½ milk exchange	125 ml FF yogurt
1½ milk exchanges	milk in tea and coffee during the day.

Starters

- If you have soup, order a thin fat free soup eg. consommé instead of a cream soup.
- Other good starters include fruit cocktail, asparagus spears (no sauce), fruit juice or salad (without dressing).
- Eating a salad before the main course will help to control your appetite and help you to eat smaller portions of other dishes.

Main Course

- Always choose simple items that fit into your diet.
- Order grilled meat, chicken or fish and specify that it must not be basted with fat.
- All visible fat and skin of chicken must be removed.
- Avoid stews, casseroles, pies and food in rich sauce.
- As a rule order fish or chicken rather than meat.
- Request small portions where possible.
- When unsure about ingredients used in a dish, ask the waiter.

Vegetables and Salads

- Have plain boiled vegetables rather than creamed, glazed, buttered or fried vegetables.
- Order baked potato rather than chips. Ask for cottage cheese or plain low fat yogurt for your potato instead of sour cream or butter.
- Ask for (large) salads without salad dressing.

Dessert

- Have fresh fruit salad or fruit salad (no cream or ice cream) as a rule.

- Do not eat cheese and biscuits.

Beverages

- Cold water, mineral water or soda water.
- If desired, have plain tea or coffee.
- Avoid Irish coffee, cappuccino and liqueur coffees.

Appendix 4

Demographic and Anthropomorphic Data
(1 = pre-intervention; 2 = post-intervention)

NAME		GRP		AGE	SEX		WEIGHT1		HEIGHT1		BMI1		% FAT1		LBM1
PATHER	A	A	1	31	M		60.40		176.50		19.00		15.40		51.10
VENKATSEN	B	A	1	37	M		79.00		167.50		28.30		25.00		59.00
NANDLAL	V	A	1	28	M		71.40		161.00		27.50		29.70		50.20
CHETTY	N	A	1	25	M		70.00		172.00		23.80		23.80		53.40
NAIDOO	T	A	1	34	M		92.10		176.50		29.70		27.20		67.40
NAIDOO	M	A	1	34	M		79.00		171.00		27.00		24.60		59.60
GOVENDER	S	A	1	32	M		73.00		176.50		23.00		15.70		62.00
HATIA	M	A	1	56	M		71.20		180.00		22.00		23.60		54.40
NAICKER	C	A	1	28	M		58.60		180.00		18.10		17.60		48.60
GOVENDER	J	A	1	36	M		64.20		163.00		24.20		25.70		47.70
HARILAL	J	A	1	34	M		75.00		168.00		27.20		23.30		57.60
MUTHUSAMY	R	A	1	29	M		61.20		167.00		22.00		16.20		51.30
RAJBANSI	P	A	1	37	M		60.20		166.00		21.80		24.20		45.70
SINGH	D	A	1	39	M		56.00		173.50		18.70		18.10		45.20
THAKERPERSA	R	A	1	29	M	66.34	53.60	171.79	176.30	23.31	17.20	21.27	9.10	53.46	48.70
NAIDOO	D	B	1	37	M		75.60		171.00		25.80		21.90		59.10
PILLAY	P	B	1	46	M		58.60		167.00		21.00		20.20		46.80
HABIB	A	B	1	26	M		67.40		171.00		23.00		21.60		53.30
BAWA	A	B	1	36	M		66.60		172.00		29.20		34.00		57.20
GOVENDER	R	B	1	37	M		63.60		171.50		21.90		21.70		49.80
NAIDOO	H	B	1	34	M		75.20		172.00		25.40		24.60		56.70
BRAMDAW		B	1	27	M		50.60		164.00		18.80		15.00		43.00
CHINNIAN	H	B	1	33	M		78.60		163.50		29.60		34.20		51.70
PILLAY	K	B	1	26	M		79.60		180.00		24.60		20.00		63.60
NAIDOO	A	B	1	39	M		75.20		171.00		25.70		25.30		56.40
KISTAN	C	B	1	44	M		67.80		176.50		21.90		19.30		54.60
LAZARUS	T	B	1	34	M		66.00		171.50		23.30		17.40		56.20
REDDY	P	B	1	49	M		92.60		173.50		30.90		32.60		63.30
GOBIND	D	B	1	44	M	71.26	58.20	170.58	163.60	24.50	21.90	23.29	18.00	54.25	47.80
NAIDOO	S	C	1	46	M		52.20		161.50		20.10		18.00		41.90
HAMID	R	C	1	36	M		99.20		168.00		35.10		35.70		63.80
RAMPATHI	K	C	1	45	M		65.40		162.00		24.90		26.80		47.80
HARRICHAREN	R	C	1	35	M		97.80		182.50		29.50		24.20		74.30
DUKHI	R	C	1	35	M		71.40		167.00		25.60		22.70		55.20
BEHARILAL	R	C	1	33	M		96.00		172.50		32.40		34.20		63.30
FRANCIS	M	C	1	50	M		60.80		166.50		22.00		18.70		49.50
MAHARAJ	P	C	1	33	M		77.60		173.50		25.90		25.40		58.20
BALGOBIND	R	C	1	31	M		66.50		166.50		24.10		21.20		52.50
ALLIE	M	C	1	33	M		80.20		176.00		25.90		24.60		60.50
VEERAN	D	C	1	36	M		59.60		165.00		21.90		22.20		46.50
GOVENDER	T	C	1	30	M	76.03	65.60	169.29	170.50	26.42	29.60	24.64	22.00	56.66	66.70

NAME		GRP		AGE	SEX2		WEIGHT2		HEIGHT2		BMI2		% FAT2		LBM2
PATHER	A	A	2	31	M		61.80		178.50		19.40		12.70		54.20
VENKATSE	B	A	2	37	M		76.20		167.50		22.40		21.00		60.10
NANDLAL	V	A	2	28	M		70.00		161.00		27.00		20.00		56.00
CHETTY	N	A	2	26	M		71.00		172.00		24.06		17.60		58.90
NAIDOO	T	A	2	34	M		87.40		176.50		28.20		16.60		73.50
NAIDOO	M	A	2	34	M		76.80		171.00		26.30		18.30		63.00
GOVENDE	S	A	2	32	M		72.60		178.50		22.90		10.40		65.30
HATIA	M	A	2	57	M		71.20		180.00		22.00		18.60		58.40
NAICKER	C	A	2	28	M		58.60		180.00		18.45		14.20		51.30
GOVENDE	J	A	2	36	M		63.00		163.00		23.70		20.30		50.40
HARILAL	J	A	2	34	M		73.40		168.00		25.60		19.90		58.80
MUTHUSA	R	A	2	29	M		61.30		167.00		22.00		14.50		54.04
RAJBANSI	P	A	2	37	M		57.90		166.00		21.00		16.90		46.04
SINGH	D	A	2	39	M		56.60		173.50		18.90		10.10		50.84
THARKER	R	A	2	29	M	67.44	53.00	171.79	176.30	22.67	17.20	15.75	5.30	56.67	50.20
NAIDOO	D	B	2	37	M		71.20		171.00		24.30		17.80		58.80
PILLAY	P	B	2	46	M		54.20		167.00		19.40		10.80		48.80
HABIB	A	B	2	26	M		64.80		171.00		22.20		15.90		54.50
BAWA	A	B	2	36	M		66.60		172.00		29.30		25.00		64.90
GOVENDE	R	B	2	37	M		58.00		171.50		19.60		16.10		48.70
NAIDOO	H	B	2	34	M		70.80		172.00		23.90		16.60		58.90
BRAMDAW	N	B	2	27	M		48.40		164.00		18.00		7.80		44.70
CHINNIAN	H	B	2	33	M		74.80		163.50		28.20		26.80		54.80
PILLAY	K	B	2	26	M		75.20		180.00		23.20		16.20		63.02
NAIDOO	A	B	2	39	M		71.20		171.00		24.30		17.30		58.90
KISTAN	C	B	2	44	M		63.20		176.50		20.45		14.50		54.04
LAZARUS	T	B	2	34	M		63.50		171.50		21.70		14.00		54.70
REDDY	P	B	2	49	M		89.60		173.50		29.90		27.90		64.70
GOBIND	D	B	2	44	M	67.56	54.80	170.58	163.60	23.23	20.60	17.04	11.80	55.56	48.80
NAIDOO	S	C	2	46	M		51.20		161.50		19.76		14.30		43.86
HAMID	R	C	2	36	M		98.00		168.00		35.10		35.50		64.40
RAMPATHI	K	C	2	45	M		63.00		162.00		24.00		26.00		46.60
HARRICHA	R	C	2	35	M		96.40		182.50		29.10		25.00		72.30
DUKHI	R	C	2	35	M		71.00		167.00		25.50		21.50		55.90
BEHARILA	R	C	2	33	M		95.00		172.50		32.20		31.70		64.80
FRANCIS	M	C	2	50	M		59.20		166.50		21.50		17.60		48.70
MAHARAJ	P	C	2	33	M		76.20		173.50		25.40		25.40		56.60
BALGOBIN	R	C	2	31	M		65.60		166.50		23.60		18.60		53.30
ALLIE	M	C	2	33	M		80.00		176.00		25.90		24.60		60.30
VEERAN	D	C	2	36	M		59.40		165.00		21.40		21.40		46.70

NAME	GRP			VO2			Ml/Kg/Min			Ven.			HR			Sit-Ups		Push-Ups		Flex.
PATHER	A	1	A		2.96			54.90			82.20			194.00			31.00		31.00		25.00
VENKATSEN	B	1	A		2.66			33.80			113.80			186.00			36.00		25.00		27.00
NANDLAL	V	1	A		2.40			33.90			82.90			175.00			26.00		33.00		35.00
NAIDOO	T	1	A		2.29			32.90			84.20			192.00			34.00		9.00		30.00
CHETTY	N	1	A		2.46			35.00			82.80			176.00			24.00		24.00		21.00
NAIDOO	M	1	A		3.01			38.10			121.40			175.00			55.00		34.00		31.00
GOVENDER	S	1	A		2.82			36.80			97.00			198.00			40.00		39.00		29.00
HATIA	M	1	A		1.75			24.70			56.40			150.00			20.00		0.00		25.00
NAICKER	C	1	A		2.58			44.80			70.10			186.00			31.00		16.00		28.00
GOVENDER	J	1	A		2.47			38.70			92.30			182.00			22.00		17.00		19.00
HARILAL	J	1	A		2.19			29.30			78.70			170.00			23.00		22.00		24.00
MUTHUSAMY	R	1	A		2.54			41.80			107.40			188.00			36.00		24.00		15.00
RAJBANSI	P	1	A		2.86			47.70			114.40			193.00			31.00		43.00		14.00
SINGH	D	1	A		2.16			38.70			96.20			181.00			26.00		27.00		11.00
THAKERPERSA	R	1	A	2.48	2.01	37.86	37.40	91.18	87.90	181.20	172.00	31.00	30.00	25.13	33.00	23.53	21.00			
NAIDOO	D	1	B		2.42			31.90			96.60			180.00			22.00		20.00		6.00
PILLAY	P	1	B		2.11			36.40			66.60			157.00			18.00		18.00		12.00
HABIB	A	1	B		2.38			35.70			95.50			170.00			23.00		6.00		9.00
BAWA	A	1	B		3.05			35.60			114.30			170.00			28.00		22.00		28.00
GOVENDER	R	1	B		2.51			39.30			94.60			192.00			31.00		20.00		6.00
NAIDOO	H	1	B		2.58			34.50			97.10			191.00			26.00		33.00		31.00
BRAMDAW	N	1	B		2.20			44.00			105.40			193.00			30.00		32.00		9.00
CHINNIAN	H	1	B		2.65			33.60			97.40			187.00			20.00		20.00		19.00
PILLAY	K	1	B		2.96			37.50			101.70			198.00			34.00		21.00		10.00
NAIDOO	A	1	B		3.22			43.10			121.30			183.00			25.00		16.00		27.00
KISTAN	C	1	B		2.51			36.90			93.10			183.00			24.00		24.00		27.00
LAZARUS	T	1	B		2.15			31.70			80.90			186.00			29.00		15.00		31.00
REDDY	P	1	B		2.60			28.30			86.50			159.00			14.00		6.00		6.00
GOBIND	D	1	B	2.55	2.35	36.36	40.60	95.84	90.70	180.71	181.00	26.00	40.00	20.43	33.00	17.64	26.00			
NAIDOO	B	1	C		2.03			39.20			73.50			174.00			13.00		6.00		17.00
HAMID	R	1	C		2.36			23.90			70.00			153.00			9.00		20.00		11.00
RAMPATHI	K	1	C		1.87			28.80			77.40			186.00			13.00		6.00		17.00
HARRICHAREN	R	1	C		3.06			31.50			110.60			178.00			15.00		19.00		32.00
DUKHI	R	1	C		1.93			27.30			113.60			188.00			17.00		15.00		20.00
BEHARILAL	R	1	C		2.86			29.90			92.10			188.00			15.00		13.00		15.00
FRANCIS	M	1	C		1.77			29.00			61.30			153.00			10.00		15.00		17.00
MAHARAJ	P	1	C		3.11			40.40			119.80			192.00			26.00		12.00		10.00
BALGOBIND	R	1	C		3.03			46.00			107.80			198.00			26.00		25.00		34.00
ALLIE		1	C		2.48			31.10			87.90			172.00			22.00		11.00		12.00
VEERAN	D	1	C		1.78			30.20			72.10			183.00			29.00		20.00		16.00
GOVENDER	T	1	C	2.43	2.86	32.58	33.70	93.15	131.90	180.25	198.00	18.33	21.00	15.67	26.00	17.00	3.00			
PATHER	A	2	A		3.61			59.30			119.50			196.00			39.00		51.00		25.00
VENKATSEN	B	2	A		3.03			39.00			126.10			175.00			36.00		23.00		29.00
NANDLAL	V	2	A		2.73			39.10			122.10			186.00			30.00		29.00		35.00
NAIDOO	T	2	A		2.86			40.30			115.00			196.00			40.00		20.00		34.00
CHETTY	N	2	A		3.68			42.40			125.30			176.00			29.00		25.00		28.00
NAIDOO	M	2	A		3.32			43.80			138.80			181.00			50.00		38.00		33.00
GOVENDER	S	2	A		3.42			47.50			134.00			187.00			42.00		40.00		31.00
HATIA	M	2	A		1.95			27.50			72.70			169.00			26.00		18.00		30.00
NAICKER	C	2	A		3.10			52.60			99.10			185.00			32.00		30.00		28.00
GOVENDER	J	2	A		2.96			46.90			122.80			195.00			27.00		30.00		22.00
HARILAL	J	2	A		2.24			33.10			79.40			175.00			27.00		28.00		24.00
MUTHUSAMY	R	2	A		2.96			47.00			123.00			185.00			36.00		27.00		18.00
RAJBANSI	P	2	A		3.00			52.40			125.00			189.00			33.00		45.00		23.00
SINGH	D	2	A		2.60			45.80			93.20			175.00			26.00		31.00		21.00
THAKERPERSA	R	2	A	2.94	2.67	44.47	50.50	113.80	111.20	183.53	183.00	33.67	31.00	31.33	35.00	26.80	21.00			
NAIDOO	D	2	B		2.52			35.80			92.70			180.00			27.00		28.00		12.00
PILLAY	P	2	B		2.75			51.10			80.80			180.00			26.00		22.00		19.00
HABIB	A	2	B		2.57			40.20			109.80			181.00			35.00		19.00		10.00
BAWA	A	2	B		3.25			37.80			128.10			175.00			30.00		32.00		28.00
GOVENDER	R	2	B		2.98			51.40			116.10			198.00			31.00		24.00		8.00
NAIDOO	H	2	B		2.84			40.80			101.10			198.00			29.00		33.00		31.00
BRAMDAW	N	2	B		2.54			53.00			112.20			189.00			34.00		33.00		12.00
CHINNIAN	H	2	B		3.36			45.50			121.80			190.00			26.00		21.00		24.00
PILLAY	K	2	B		3.10			41.00			105.00			186.00			43.00		30.00		23.00
NAIDOO	A	2	B		3.45			48.80			112.00			188.00			27.00		25.00		32.00
KISTAN	C	2	B		2.87			42.40			100.30			183.00			28.00		27.00		29.00
LAZARUS	T	2	B		3.02			48.00			118.40			185.00			28.00		21.00		35.00
REDDY	P	2	B		2.85			32.10			106.80			163.00			19.00		9.00		12.00
GOBIND	D	2	B	2.91	2.86	44.24	52.00	108.31	111.40	184.43	186.00	30.07	38.00	26.29	44.00	21.36	24.00			
NAIDOO	B	2	C		2.06			40.80			75.10			180.00			17.00		12.00		21.00
HAMID	R	2	C		2.47			25.30			94.30			172.00			5.00		10.00		11.00
RAMPATHI	K	2	C		1.87			29.80			71.90			171.00			17.00		12.00		21.00
HARRICHAREN	R	2	C		3.42			35.70			125.00			186.00			21.00		20.00		34.00
DUKHI	R	2	C		2.27			28.10			106.40			182.00			19.00		16.00		24.00
BEHARILAL	R	2	C		2.78			29.30			110.90			197.00			15.00		18.00		19.00
FRANCIS	M	2	C		1.42			24.00			51.40			155.00			13.00		14.00		13.00
MAHARAJ	P	2	C		3.04			40.00			119.70			205.00			28.00		10.00		10.00
BALGOBIND	R	2	C		2.81			43.20			106.20			200.00			28.00		23.00		34.00
ALLIE	M	2	C		1.93			24.10			83.80			188.00			24.00		12.00		10.00
VEERAN	D	2	C		2.03			34.40			86.80			175.00			30.00		23.00		17.00
GOVENDER	T	2	C	2.43	3.00	32.45	34.90	96.09	121.80	184.33	201.00	19.75	20.00	16.58	29.00	18.08	3.00			

Lipid and Lipoprotein Data
(1 = pre-intervention; 2 = post-intervention)

NAME		GRP	SCHOL		TRIG		HDL		LDL		R-RATIO
PATHER	A 1	A	5.69		1.20		0.87		4.46		6.5
VENKATSEN	B 1	A	5.87		1.03		1.02		3.77		6.75
NANDLAL	V 1	A	5.99		1.29		0.81		4.74		7.39
CHETTY	N 1	A	5.22		1.39		0.93		3.43		5.61
NAIDOO	T 1	A	6.55		1.66		0.82		5.02		7.98
NAIDOO	M 1	A	3.86		3.74		0.69		2.47		5.6
GOVENDER	S 1	A	5.45		0.92		1.27		3.49		4.29
HATIA	M 1	A	6.99		1.98		0.84		5.21		8.3
NAICKER	C 1	A	4.45		0.81		1.37		2.72		3.24
GOVENDER	J 1	A	5.92		0.98		1.40		4.08		4.2
HARILAL	J 1	A	7.81		2.61		0.65		5.95		12.0
MUTHUSAMY	R 1	A	5.51		1.51		1.02		4.67		5.4
RAJBANSI	P 1	A	5.64		1.04		0.93		4.25		7.94
SINGH	D 1	A	4.79		1.54		0.82		3.28		5.9
THAKERPERSA	R 1	A	5.72	6.02	1.50	0.81	0.97	1.11	4.14	4.55	6.4	5.42
NAIDOO	D 1	B		5.91		1.48		1.18		4.11		5.00
PILLAY	P 1	B		4.61		0.68		1.26		3.04		3.66
HABIB	A 1	B		4.30		1.24		0.77		2.98		5.58
BAWA	A 1	B		5.81		0.91		1.18		4.22		4.92
GOVENDER	R 1	B		7.30		1.29		0.81		5.91		9.01
NAIDOO	H 1	B		6.95		1.52		0.92		5.35		7.55
BRAMDAW	N 1	B		4.92		1.61		1.58		2.61		3.11
CHINNIAN	H 1	B		4.97		0.89		0.83		3.74		5.99
PILLAY	K 1	B		4.96		0.96		1.00		3.53		4.96
NAIDOO	A 1	B		4.82		0.85		1.43		3.00		3.37
KISTAN	C 1	B		4.85		0.75		0.96		3.55		3.94
LAZARUS	T 1	B		4.33		2.45		0.68		2.54		6.37
REDDY	P 1	B		5.97		1.15		0.90		4.55		6.63
GOBIND	D 1	B	5.38	5.68	1.20	1.06	1.04	1.00	3.81	4.21	5.41	5.68
NAIDOO	B 1	C		6.23		1.92		0.69		4.67		9.03
HAMID	R 1	C		4.49		2.01		0.73		2.86		6.15
RAMPATHI	K 1	C		6.32		1.94		0.84		4.61		7.52
HARRICHAREN	R 1	C		6.53				0.92				7.10
DUKHI	R 1	C		5.32		1.35		0.90		3.80		5.91
BEHARILAL	R 1	C		5.48		1.96		0.78		3.81		7.03
FRANCIS	M 1	C		7.34		4.21		1.00		4.93		7.34
MAHARAJ	P 1	C		4.65		2.86		0.66		2.69		7.05
BALGOBIND	R 1	C		5.98		1.61		1.00		4.20		5.98
ALLIE	M 1	C		6.15		4.30		0.68				9.04
VEERAN	D 1	C		4.70		1.17		0.83		3.34		5.66
GOVENDER	T 1	C	5.82	6.64	2.26	1.56	0.86	1.28	3.96	4.65	6.92	5.19
PATHER	A 2	A		5.89		1.15		0.95		4.42		6.20
VENKATSEN	B 2	A		5.86		0.67		1.14		4.42		5.14
NANDLAL	V 2	A		6.34		1.31		0.93		4.82		6.82
CHETTY	N 2	A		4.57		1.28		0.95		3.04		4.81
NAIDOO	T 2	A		6.06		1.22		0.87		4.65		6.99
NAIDOO	M 2	A		4.16		2.95		0.63		2.19		6.60
GOVENDER	S 2	A		5.65		0.54		1.55		3.62		3.65
HATIA	M 2	A		6.88		2.23		0.87		5.00		7.90
NAICKER	C 2	A		4.69		0.87		1.53		2.96		3.06
GOVENDER	J 2	A		6.23		0.98		1.49		4.30		4.18
HARILAL	J 2	A		8.37		3.30		0.74		6.10		11.30
MUTHUSAMY	R 2	A		5.90		0.95		1.00		4.48		5.90
RAJBANSI	P 2	A		5.69		0.93		1.16		4.11		4.91
SINGH	D 2	A		5.23		1.62		1.13		3.13		4.63
THAKERPERSA	R 2	A	5.86	6.36	1.39	0.78	1.08	1.30	4.13	4.70	5.80	4.89
NAIDOO	D 2	B		5.87		1.82		1.36		3.68		4.32
PILLAY	P 2	B		3.91		0.63		1.37		2.30		2.85
HABIB	A 2	B		4.49		1.15		0.92		3.05		4.92
BAWA	A 2	B		5.69		1.22		1.38		3.76		4.12
GOVENDER	R 2	B		5.36		1.26		0.93		3.82		5.76
NAIDOO	H 2	B		6.64		1.78		1.05		4.75		6.32
BRAMDAW	N 2	B		3.93		1.69		1.42		1.74		2.77
CHINNIAN	H 2	B		5.38		1.02		0.88		4.04		6.11
PILLAY	K 2	B		4.91		1.21		1.07		3.29		4.59
NAIDOO	A 2	B		4.89		0.47		1.88		2.79		2.60
KISTAN	C 2	B		5.04		0.91		1.14		3.49		4.42
LAZARUS	T 2	B		4.54		1.88		0.77		2.94		5.92
REDDY	P 2	B		6.59		0.86		1.18		4.79		5.59
GOBIND	D 2	B	5.21	5.71	1.20	0.83	1.18	1.20	3.47	4.14	4.65	4.76
NAIDOO	B 2	C		6.36		3.11		0.75		4.20		8.48
HAMID	R 2	C		4.30		2.47		0.71		2.88		6.06
RAMPATHI	K 2	C		5.50		1.63		0.93		3.83		5.91
HARRICHAREN	R 2	C		7.10				0.86				8.26
DUKHI	R 2	C		5.31		1.07		0.99		3.83		5.90
BEHARILAL	R 2	C		5.87		1.87		0.86		4.16		6.83
FRANCIS	M 2	C		7.55		4.62		1.09				6.93
MAHARAJ	P 2	C		5.33		2.96		0.62		3.36		8.59
BALGOBIND	R 2	C		6.80		0.99		1.30		5.05		5.23
ALLIE	M 2	C		6.07		4.45		0.67				9.06
VEERAN	D 2	C		5.10		3.04		0.89		2.83		5.73
GOVENDER	T 2	C	6.07	7.58	2.58	2.13	0.91	1.22	3.95	5.40	6.93	6.21

Dietary Intake Data
(1 = pre-intervention; 2 = post-intervention)

NAME		GRP	Kcal		Protein		T-Fat		Mono-S		Poly-UN		Sat-F		T-CHO		D-Fibre		P:S Ratio		Alcohol		CHOLO	
PATHER	1	A	3442.00		80.40		136.90		45.60		30.30		52.30		483.60		27.00		0.56		1.70		236.00	
VENKATSEN	1	A	1971.00		67.70		75.00		23.80		25.70		20.80		248.10		14.90		1.25				254.00	
NANDLAL	1	A	2094.00		61.50		85.40		18.70		21.70		18.10		309.00		22.00		1.20				191.00	
NAIDOO	1	A	2864.00		97.50		148.70		42.70		55.40		35.90		267.30		24.90		1.54				291.00	
CHETTY	1	A	2542.00		85.00		104.80		31.30		40.40		26.20		280.90		21.10		1.60		5.70		230.00	
NAIDOO	1	A	3187.00		86.20		177.20		48.80		75.20		43.80		300.00		16.50		1.72		3.80		283.00	
GOVENDER	1	A	2882.00		86.30		136.50		44.10		41.20		40.30		306.20		14.71		1.02		10.30		309.00	
HATIA	1	A	3533.00		116.50		152.00		59.10		35.00		48.80		405.20		38.10		0.72				395.00	
NAICKER	1	A	4336.00		115.80		170.70		49.10		67.90		43.10		406.00		26.20		1.56		43.80		429.00	
GOVENDER	1	A	2267.00		56.10		95.80		29.70		37.80		21.80		291.80		15.60		1.76				185.00	
HARILAL	1	A	3014.00		73.90		114.80		36.20		44.70		27.20		421.10		16.50		1.64		13.20		172.00	
MUTHUSAMY	1	A	3039.00		84.10		111.40		35.30		26.80		37.90		431.40		18.10		0.76		8.20		270.00	
RAJBANSI	1	A	2084.00		62.50		75.20		22.40		18.90		26.10		275.50		16.30		0.75				208.00	
SINGH	1	A	2776.00		74.60		101.40		37.90		29.20		26.30		376.30		17.90		1.03		3.80		266.00	
THAKERPERS	1		2880.20	80.41	74.10	121.86	163.40	37.97	45.10	41.09	64.10	34.15	44.40	347.82	340.10	20.83	17.70	1.34	1.44	11.33	12.20	276.67	320.00	
NAIDOO	1	B	1932.00		62.60		102.10		35.10		25.30		32.30		174.20		12.10		0.78		4.60		284.00	
PILLAY	1	B	1947.00		71.00		93.70		30.80		28.50		23.10		193.40		30.80		1.23				62.00	
HABIB	1	B	2920.00		81.00		142.80		38.20		51.00		42.70		323.30		21.70		1.19				307.00	
BAWA	1	B	2096.00		65.20		95.30		31.70		29.00		26.30		232.30		18.50		1.10				188.00	
GOVENDER	1	B	2146.00		77.90		79.00		20.10		23.00		22.70		263.10		29.60		1.01				244.00	
NAIDOO	1	B	2554.00		91.90		122.70		40.90		33.30		36.10		348.00		32.30		0.82		5.30		270.00	
BRAMDAW	1	B	2314.00		59.90		65.10		19.20		21.50		20.40		340.50		11.80		1.06		24.50		136.00	
CHINNAN	1	B	2809.00		71.00		130.40		49.50		37.80		33.80		326.80		21.40		1.12				198.00	
PILLAY	1	B	2504.00		90.00		111.50		29.40		36.70		29.20		271.40		24.30		1.33		5.30		400.00	
NAIDOO	1	B	3068.00		105.90		136.20		45.80		39.20		42.80		313.90		31.20		0.92		21.80		377.00	
KISTAN	1	B	1973.00		41.00		107.50		25.80		44.70		23.20		201.20		26.20		1.85				64.00	
LAZARUS	1	B	2532.00		64.60		84.00		29.60		16.30		27.90		371.00		21.20		0.54				214.00	
REDDY	1	B	3281.00		113.40		176.40		64.80		52.20		42.40		363.80		27.30		1.23		21.80		447.00	
GOBIND	1		2367.84	74.46	47.00	107.66	58.80	34.09	16.50	33.39	26.90	29.85	12.20	263.44	144.20	24.06	26.00	1.19	2.20	14.56	16.80	230.43	91.00	
NAIDOO	1	C	2618.00		86.30		95.70		29.40		30.20		26.60		345.80		18.10		1.14				317.00	
HAMID	1	C	3376.00		78.40		223.20		62.80		67.80		70.40		262.50		20.00		0.96				376.00	
RAMPATHI	1	C	3911.00		99.30		208.80		80.10		63.20		48.40		306.10		32.80		1.39		8.10		208.00	
HARRICHARE	1	C	2163.00		81.70		116.80		28.10		62.30		30.80		213.10		14.50		3.00		12.10		242.00	
DUKHI	1	C	3058.00		84.20		141.80		46.80		52.90		33.30		366.00		15.80		1.59				346.00	
BEHARILAL	1	C	2906.00		71.70		98.90		27.90		39.90		23.80		451.10		18.10		1.66				280.00	
FRANCIS	1	C	2274.00		74.40		108.50		42.40		26.80		28.10		227.10		17.70		0.91		8.30		211.00	
MAHARAJ	1	C	2967.00		86.20		146.80		42.40		64.20		31.40		336.20		17.80		2.04		12.20		268.00	
BALGOBIND	1	C	3442.00		119.20		194.80		66.40		80.80		80.60		270.20		12.90		1.20		16.30		460.00	
ALLIE	1	C	3277.00		122.90		163.00		64.00		48.00		47.90		321.50		14.40		1.00				646.00	
VEERAN	1	C	1821.00		51.20		76.20		25.10		21.90		18.20		216.50		18.20		1.14				186.00	
GOVENDER	1		2863.82	82.67	84.50	143.86	182.50	45.46	40.10	51.11	76.00	35.59	26.90	295.31	234.80	18.46	31.80	1.56	2.63	12.00	12.00	311.06	176.00	
pather	2	A	3083.00		86.60		126.90		45.10		30.20		45.10		348.70		16.00		0.60				344.00	
VENKATSEN	2	A	1767.00		48.70		66.60		22.30		27.20		13.10		234.20		13.30		2.00				142.00	
NANDLAL	2	A	2050.00		61.90		60.40		17.50		16.30		17.50		306.40		21.20		1.06				167.00	
NAIDOO	2	A	2387.00		64.60		97.50		23.40		43.10		18.09		279.80		32.00		2.30				189.00	
CHETTY	2	A	2206.00		62.90		98.90		36.60		24.90		30.70		244.70		16.50		0.81		8.70		187.00	
NAIDOO	2	A	3000.00		95.80		117.10		39.90		34.00		35.20		376.80		24.20		0.97		2.00		364.00	
GOVENDER	2	A	2827.00		87.40		118.50		37.80		40.90		26.80		336.00		16.90		1.43		10.80		470.00	
HATIA	2	A	2518.00		76.70		108.00		38.00		26.20		32.30		297.40		26.00		0.81				207.00	
NAICKER	2	A	3207.00		81.30		130.30		41.80		43.10		31.30		396.90		30.30		1.36		8.70		283.00	
GOVENDER	2	A	2043.00		82.50		86.00		22.40		40.80		17.09		267.00		17.30		2.49				178.00	
HARILAL	2	A	2711.00		80.10		117.10		36.30		43.00		30.80		286.30		16.10		1.41		35.30		136.00	
MUTHUSAMY	2	A	1931.00		46.80		81.40		24.70		16.90		31.00		234.60		12.30		0.56		7.00		230.00	
RAJBANSI	2	A	2114.00		61.80		84.80		31.70		22.40		26.10		262.40		17.80		0.89				183.00	
SINGH	2	A	2088.00		52.10		91.80		29.70		31.90		21.90		247.50		21.80		1.46		4.80		133.00	
THAKERPERS	2		2456.13	64.65	73.60	101.71	141.80	32.76	44.30	32.96	51.80	27.57	36.30	288.23	325.70	20.40	20.10	1.30	1.42	10.84	10.00	226.33	326.00	
NAIDOO	2	B	1334.00		44.40		45.10		11.80		14.90		10.80		180.00		17.20		1.42		7.70		80.00	
PILLAY	2	B	1910.00		57.80		62.90		15.50		27.80		11.70		191.40		39.80		2.36				22.00	
HABIB	2	B	2309.00		64.50		124.70		36.50		45.50		34.40		224.20		23.90		1.32				218.00	
BAWA	2	B	2114.00		66.60		84.20		21.80		32.80		18.00		263.50		26.80		1.72				136.00	
GOVENDER	2	B	2178.00		56.00		82.40		34.10		26.80		16.70		263.20		41.80		1.73				82.00	
NAIDOO	2	B	1847.00		75.50		89.20		30.70		19.70		19.10		196.10		38.90		1.08		11.40		163.00	
BRAMDAW	2	B	1727.00		46.90		61.40		14.10		26.00		12.70		222.90		37.80		2.06		12.00		76.00	
CHINNAN	2	B	3437.00		86.20		96.80		36.80		26.30		24.80		316.10		26.40		1.07				126.00	
PILLAY	2	B	2081.00		66.90		68.50		23.80		27.80		24.30		231.60		26.20		1.14		7.80		186.00	
NAIDOO	2	B	1834.00		62.20		72.20		20.70		22.00		20.80		206.00		26.60		1.06		12.20		208.00	
KISTAN	2	B	1637.00		36.30		79.80		16.20		21.40		16.20		179.00		21.90		2.02				44.00	
LAZARUS	2	B	1918.00		41.50		64.80		17.10		26.80		13.90		174.70		19.20		1.93				121.00	
REDDY	2	B	3100.00		110.00		160.00		42.00		60.00		40.00		273.00		26.20		1.50		10.00		407.00	
GOBIND	2		1920.07	60.65	45.50	81.70	81.20	22.61	14.80	26.71	22.50	19.64	11.00	216.22	112.40	26.72	26.80	1.80	2.06	10.33	12.00	135.71	71.00	
NAIDOO	2	C	2397.00		66.30		111.30		37.20		39.50		27.40		260.60		14.00		1.44		7.00		227.00	
HAMID	2	C	3650.00		96.60		144.10		50.10		39.70		55.10		413.70		22.80		0.72				318.00	
RAOMPATHI	2	C	3284.00		96.30		173.70		65.90		47.00		42.20		275.90		29.50		1.11		18.30		249.00	
HARRICHARE	2	C	3490.00		47.20		121.80		30.00		61.40		20.30		276.60		17.00		3.08		11.20		306.00	
DUKHI	2	C	2547.00		90.90		93.70		28.50		25.00		22.80		307.90		30.40		1.09				215.00	
BEHARILAL	2	C	3466.00		66.70		102.00		27.40		38.30		26.70		306.80		23.40		1.53				367.00	
FRANCIS	2	C	1481.00		50.60		57.10		13.40		24.00		10.80		164.60		19.60		2.30		5.10		125.00	
MAHARAJ	2	C	2870.00		76.60		134.80		46.30		37.10		32.70		266.00		13.30		1.14		12.10		363.00	
BALGOBIND	2	C	2200.00		64.20		93.20		27.80		32.30		21.40		261.10		16.70		1.52		1.80		211.00	
ALLIE	2	C	2647.00		96.70		134.10		39.70		35.70		46.80		262.70		17.70		0.77				416.00	
VEERAN	2	C	1266.00		38.10		50.70		16.90		8.60		17.30		154.70		14.40		0.50				166.00	
GOVENDER	2		2435.25	74.81	93.50	112.03	112.10	35.36	32.20	35.77	39.40	29.13	27.10	262.19	214.70	19.89	19.10	1.36	1.46	8.70	10.70	261.30	286.00	