THE PHYSIOLOGICAL EFFECTS OF A 14 WEEK WALKING PROGRAMME ON MIDDLE AGED SEDENTARY MEN AND WOMEN

BY

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ABSTRACT

Many epidemiological studies show that a moderate amount of regular exercise can help to prevent heart disease. As an exercise, walking is now widely promoted by many health professionals to encourage greater activity levels. Walking is popular as it is simple, inexpensive, and appropriate for the majority of the population.

Two studies were conducted, the aim of the first study was to compare exercise adherence and physiological changes in a group of middle-aged sedentary dog owners and non-dog owners in response to a 14 week walking programme. The aim of the second study was to measure the physiological changes in a group of sedentary middle-aged women in response to the same walking programme, and compare the results to those of the men.

One hundred and five, and sixty eight healthy sedentary middle-aged males (46 non-dog owners (NDO), 39 dog owners (DO), 20 controls(C)) and females (48 walkers (W), 14 controls (C)) aged between 40-60 years respectively, were recruited. The non-dog owners, dog owners and the female walking group were asked to complete a 14 week brisk walking programme, of 4 walks per week for 30 minutes at 70-75% age predicted maximum heart rate. To monitor adherence training diaries were submitted regularly. The adherence rates were similar for all three exercise groups (non-dog owners 52%, dog owners 56%, women 52%).

In Study One the non-dog owners and the dog owners had no significant change in % body fat, triglyceride and HDL levels. The non-dog owners showed significant reductions in body mass (NDO 79.6 to 78.9, DO 80.0 to 79.4, C 78.6 to 79.4 kg), resting systolic blood pressure (NDO 126 to 121, DO 122 to 121, C 121 to 117 mmHg), total serum cholesterol (NDO 5.86 to 5.49, DO 6.14 to 5.98, C 6.14 to 6.18 $mmol.l^{-1}$), ratio of total cholesterol to HDL (NDO 5.44 to 4.49, DO 5.32 to 5.15, C 4.99 to 5.08). Both exercise groups had significant reductions in heart rate and oxygen costs for the same workload of the treadmill test. These parallel reductions reduced the expected

increases in predicted $\dot{V}O_2$ max which was only significant for the non-dog owners (NDO 35.1 to 36.6, DO 38.6 to 38.8, C 38.5 to 38.3 $ml.kg^{-1}.min^{-1}$).

In Study Two (women) there were no significant changes in blood pressure or body mass for both groups, but the control group did have a significant increase in predicted body fat % (W 38.8 to 39.1, C 38.7 to 40.3). There were no significant changes in serum TC or HDL, but the control group did have a significant increase in serum triglyceride levels (W 1.17 to 1.30, C 0.88 to 1.10 $mmol.l^{-1}$). The walking group showed significant reductions in heart rate for the same stages of the treadmill walking test, but the changes in predicted $\dot{V}O_2$ max. failed to reach significance ($p>0.05$).

The estimated energy expenditure of the walking programme was 25% lower for the women compared to the men (men 11,050, women 8,200 kcal).

These results would indicate that regular walking can promote some health improvements, but dog ownership does not seem to improve adherence to or effect of a brisk walking programme. Women would seem to benefit less from this walking programme, possibly due to a lower energy expenditure.

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Most of all I would like to thank the subjects who took part in both studies.

Finally I would like to dedicate this thesis to the memory of my grandmother Elizabeth Jackson.

AUTHOR'S DECLARATION

The rationale of study one was developed as an externally funded research study in conjunction with The Waltham Centre for Pet Nutrition. The author was not involved in the initial outline stages of this, but as research assistant to the study was responsible for developing the detailed procedures for the project.

The rational and procedures for study two was conceived by the author. The author was responsible for the construction and piloting of all the testing procedures used in the study. All recruitment, physiological tests, data input and statistical analysis were carried out by the author.

The following publications have originated from this work.

Davison R, Grant S, Mutrie N, Nash AS, Kelly MPT, Dargie HJ.(1992) Walk for Health ? J. Sports Sciences 10:556

INTRODUCTION

Many epidemiological studies show that a moderate amount of regular exercise can help to prevent heart disease. As an exercise, walking is now widely promoted by many health professionals to encourage greater activity levels. Walking is popular as it is simple, inexpensive, and appropriate for the majority of the population.

The aim of the two studies was to measure the effects of a 14 week brisk walking programme on health related physiological variables (body composition, blood pressure, blood lipids and aerobic fitness) for a group of middle-aged (40-60 years) sedentary men and women from the Glasgow area.

Study One also compared adherence to and effect of the 14 week walking programme between a group of dog owners (who walked with their dog) and a group of non-dog owners (who walked alone).

The null hypothesis for Study One was that the 14 week walking programme would result in no change in body mass, % body fat, resting blood pressure, fasting blood lipids and aerobic fitness.

The null hypothesis for Study Two was that the 14 week walking programme would result no change in body mass, % body fat, resting blood pressure, fasting blood lipids and aerobic fitness.

1. AEROBIC POWER

Maximal Aerobic Power or $\dot{V}O_2$max. is the maximum rate at which oxygen can be taken up, distributed and used by the body in the performance of work (Holly, 1988). It is often incorrectly referred to as aerobic capacity. $\dot{V}O_2$max. describes a power output or rate of work whereas aerobic capacity refers to an amount of work.

The highest recorded values of $\dot{V}O_2$max. have come from cross-country skiers, 7.4 l.min^{-1} for men and 4.5 l.min^{-1} for women (Astrand & Rodahl, 1986). A typical range of $\dot{V}O_2$max values is shown in Table 1.

Population	Males	Females
Cross-country skiers	84	72
Distance runners	83	62
Sedentary - young	45	38
Sedentary - middle aged	34	30
Post myocardial infarction patient	22	18
Severe pulmonary disease patient	13	13

Table 1. $\dot{V}O_2$max. values measured in healthy and diseased populations (ml.kg.$^{-1}$min^{-1}) (Powers & Howley, 1990)

The main sources of variation in $\dot{V}O_2$max. are associated with age, sex, body weight, exercise habits and hereditary. $\dot{V}O_2$max. values peak at age 18-20 years in both sexes, after which they decline with age (Astrand & Rodahl, 1986; Babcock et al., 1992). The $\dot{V}O_2$max. falls at a rate of 9-19% per decade (beginning at age 30 years) in healthy subjects of both sexes. The primary reason for the decline appears to be an age-related reduction in maximal heart rate causing a reduction in cardiac output. With continued hard training the fall in $\dot{V}O_2$max. can be blunted by about one-half (i.e. to 5% per decade)(Rogers et al., 1990). It appears that highly trained ageing subjects are able to maintain stroke volume, peripheral O_2 extraction and body composition at or near levels

they possessed in their 20's and 30's (Joyner, 1993). The recent Allied Dunbar National Fitness Survey (ADNFS) reported that the average aerobic fitness value for the men aged 65-74 was 60% of that of the young men of 16-24 years. The downward trend for women was similar (ADNFS, 1992). Women have $\dot{V}O_2$max. values that are on average 65-75% of men, primarily because of lower cardiac output, haemoglobin concentration and lean body mass (Astrand & Rodahl, 1986; Skinner et al., 1990).

It has long been considered that there is a large genetic influence on the value of $\dot{V}O_2$max., Klissourus (1971) suggested that 93% of the variation in $\dot{V}O_2$max. values was due to genetics, but a more recent detailed study of the aerobic performance in brothers, dizygotic and monozygotic twins (Bouchard et al., 1986) suggests that the influence may not be as large as once thought. After controlling for the confounding factors of age and sex, the size of genetic variance on $\dot{V}O_2$max.kg^{-1} reached 40% ($p<0.05$), but expressed as $\dot{V}O_2$max.kg^{-1}FFW the genetic effect was reduced to 10% (ns, $p>0.05$). The authors commented that the genetic effect in $\dot{V}O_2$max.kg^{-1} is statistically significant but probably inflated by non-genetic causes such as shared environmental conditions and their consequences on concomitant variables such as body fat. A test of endurance performance (total work output in a 90 min maximal test) was shown to have a much higher genetic effect (60%) even when expressed in terms of fat free mass. Bouchard and colleagues (1986) concluded that a significant genetic effect would seem to be present in the population for endurance performance but that a much lower heritability exists for $\dot{V}O_2$max. Apart from the improved methodology employed in the Bouchard et al. (1986) study, the large difference in the results could be attributed to the young age (7-13 years) of the subjects in the Klissourus (1971) study. Full maturation of the aerobic power does not occur until about age 18 years (Astrand & Rodahl, 1986), thus the subjects in the Klissourus (1971) study would be at different stages of maturity, possibly affecting the results.

One of the problems of trying to estimate the genetic component of $\dot{V}O_2$max. is its plasticity, with some studies demonstrating as much as a 44% increase in $\dot{V}O_2$max. in only 10 weeks of training (Hickson et al., 1977). Regardless of the actual value

attributed to genetic influence clearly genetic predisposition for possessing a high $\dot{V}O_2$max. value perhaps offers an advantage if training for highly aerobic sports.

1.1. LIMITATIONS TO $\dot{V}O_2$MAX.

High $\dot{V}O_2$max. values are dependant on the proper function of three important systems within the body: the respiratory system, which takes up oxygen from inspired air and transports it into the blood; the cardiovascular system, which pumps and distributes the oxygen-laden blood throughout body tissues; the musculoskeletal system, which uses the oxygen to convert stored substrates into work and heat during physical activity. The value of $\dot{V}O_2$max. is limited by the ability of the cardiopulmonary and metabolic systems to increases the oxygen uptake. Oxygen uptake is equal to the product of Cardiac Output (CO) and the difference between arterial and venous oxygen content (a-vO_2). The upper limit of $\dot{V}O_2$max. could be determined by limitations in maximal cardiac output; pulmonary gas exchange: or the ability of the working muscle to extract and metabolise the supplied oxygen. It would seem that the limitation of $\dot{V}O_2$max. differs depending on training status (Dempsey, 1986; Inbar et al., 1992). In untrained normal individuals the $\dot{V}O_2$max. seems to be limited by cardiac output. The most compelling evidence that cardiac output limits $\dot{V}O_2$max. comes from the work by Andersen & Saltin (1985) and the one-leg exercise model, which clearly shows that muscle perfusion, during exercise involving a large part of the total muscle mass, is much less than the maximum flow the muscle can accommodate. If this did occur it would exceed the maximum pumping capacity of the heart. Therefore the maximum perfusion rate of muscle cannot be achieved in whole body exercise such as running or cycling, and the capacity of the muscles to extract and use oxygen exceeds that of the cardiovascular system to supply it (Saltin, 1986). Calculated from the maximal perfusion rates, only 10kg of fully perfused muscle needs to be engaged in exercise to tax the pumping capacity of the heart and reach maximal oxygen uptake. To allow activation of more muscle mass the sympathetic

nervous system needs to induce elevated vasoconstrictor activity, over-riding locally elicited vasodilatation, to maintain blood pressure (Saltin & Strange, 1992).

However as one progresses up the fitness continuum the gas exchange capacity of the lung and/or the maximum responsiveness of the chest wall and ventilatory control systems assume a more critical rate limiting step in determining maximal oxygen consumption. This reordering occurs because the trained state is achieved by increasing the capacity of the cardiovascular system and by greatly enhanced metabolic capacities of the locomotor muscles. The pulmonary system changes little with training and thus eventually the point is reached where its capacity for gas transport no longer exceeds that of the requirements of the other adapted organ systems (Dempsey, 1986).

Untrained individuals seem not to be limited by pulmonary gas exchange for two reasons; 1) normal individuals can generally move more air into and out of the lung than is ever required by exercise, the ratio of maximal ventilation during exercise to maximum voluntary ventilation at rest (VE/MVV) is usually less than 0.7 (Myers & Froelicher, 1991) and the ventilatory equivalent for O_2 (VE/VO_2) is relatively high (Inbar et al., 1992), 2) arterial saturation of O_2 (SaO_2) changes little even with maximal exercise. In contrast the much higher $\dot{V}O_2max$'s in some trained individuals may be limited by pulmonary gas exchange (Inbar et al., 1992; Powers et al., 1989). This difference is demonstrated by trained athletes having; 1) significantly lower ventilatory reserve (VEmax - MVV) indicating that the individual is closer to their ventilatory potential and thus a greater physiological burden on the respiratory system (Inbar et al., 1992); 2) significantly lower peak exercise ventilatory equivalent (VE/VO_2) implying inefficient pulmonary function and a mismatch between metabolic demand and respiratory response (relative hypoventilation) (Inbar et al., 1992); 3) significantly lower, O_2 saturation ($\%SaO_2$) which can be reduced to <90% (Dempsey et al., 1982; Powers et al., 1989). Another factor highlighted by Dempsey (1986) is that as cardiac output increases so does pulmonary blood flow, reducing the 'transit time' causing a significant dis-equilibrium for O_2 at the end of the pulmonary capillary.

1.2. MEASUREMENT OF AEROBIC POWER

Very few individuals ever work at an exercise intensity that requires a maximum oxygen consumption, but measurement of maximal aerobic power gives the most accurate measurement of the functioning of the aerobic system. Where direct measurement of $\dot{V}O_2$max. is neither practical nor safe there is a large range of submaximal tests available to predict $\dot{V}O_2$max. with reasonable accuracy.

1.2.1. DIRECT MEASUREMENT OF MAXIMAL OXYGEN UPTAKE

The $\dot{V}O_2$max. is often considered the 'gold' standard, the most accurate and reproducible (Nordrehaug et al., 1991) assessment of aerobic power. Measurement of this value will however be subject to both biological variation and technological error. In trained individuals this error has been measured as $\pm$ 5.6%, biological variability accounting for more than 90% of this variability (Katch et al., 1982).

A maximal $\dot{V}O_2$ test involves an individual undergoing an exercise protocol that takes an individual to a level of intensity where a maximal oxygen consumption is achieved. To ensure a true $\dot{V}O_2$max. it is important that this plateau of oxygen consumption is reached, so that there is no further increase in oxygen uptake despite an increasing workload. This is normally defined as a rise in oxygen consumption of less than 0.15 $l.min^{-1}$ with a further increase in power output (Shephard, 1984). If a plateau is not achieved then the value should be termed the peak $\dot{V}O_2$, which is simply the highest $\dot{V}O_2$ achieved in a given, presumed maximal exercise test. In general the values of peak $\dot{V}O_2$ and $\dot{V}O_2$max. are similar in motivated young healthy subjects. If an incremental test is stopped because of leg or chest pain, shortness of breath or a lack of motivation obviously a plateau in $\dot{V}O_2$ may not occur, and the peak $\dot{V}O_2$ will be less than the actual $\dot{V}O_2$max. (Wasserman et al., 1987).

This criterion for achievement on $\dot{V}O_2$max. may not be appropriate for children in whom as few as 30-50 % of their maximal tests demonstrate a plateau in oxygen uptake (Rowland, 1993). A recent study has shown that the peak $\dot{V}O_2$ measured in a maximal test with children is indicative of the true $\dot{V}O_2$max. Three of the nine subjects

demonstrated a plateau of oxygen uptake but subsequent supra-maximal tests failed to elicit significantly higher values for $\dot{V}O_2$ than those achieved in the progressive test (Rowland, 1993).

Many older individuals also fail to reach a plateau in oxygen consumption. This is principally due to termination of the test because of another limiting factor such as, fatigue, angina, ST segment depression, leg soreness or weakness before a plateau has been reached (Shephard, 1984; Skinner et al., 1990).

There is a large number of different maximal tests; treadmill, cycle ergometer and step tests, with different continuous or discontinuous protocols to increase exercise intensity. Due to the large range of values no single protocol could be expected to satisfactorily measure $\dot{V}O_2$max. in both a cardiac patient and a world class marathon runner, hence the need for population specific protocols. In general the differences in measured $\dot{V}O_2$max. between different protocols have been small (Wasserman et al., 1987), but in some cases significant (Froelicher et al., 1974; McInnis & Balady, 1994). Buchfurer and colleagues (1983) have suggested that the optimum test duration for the measurement of $\dot{V}O_2$max. is 10 ± 2 min. They found from a series of treadmill and cycle ergometer protocols that the highest VO_2max. values were found in tests lasting 8-17 minutes. In contrast Davis et al. (1982) compared the effect of different cycle ergometer ramp protocols (20, 30, 50, and 100 $W.min^{-1}$) on $\dot{V}O_2$max. These different ramp rates obviously gave dramatically different test durations but this did not affect the measured $\dot{V}O_2$max. Also in a comparison of three protocols, constant inclination of 15% and increasing speed, increasing speed and no inclination and the Bruce protocol (both increasing speed and inclination) Nordrehaug et al. (1991) found no differences in measured $\dot{V}O_2$max.

1.2.2. PREDICTION OF MAXIMAL OXYGEN UPTAKE

Although actual $\dot{V}O_2$max. appears to provide the best measure of physical work capacity or of fitness, on many occasions, especially in clinical or epidemiological studies, the exertion required to attain $\dot{V}O_2$max. may be hazardous to the health and well-being of

the subjects. Hence, a large number of sub maximal tests have been developed to predict $\dot{V}O_2$max. from oxygen consumption and/or heart rate at one or more submaximal workloads and these plus a range of other variables like gender, age, weight, time to walk/run a set distance have been included in regression equations. No submaximal test to predict $\dot{V}O_2$max. will do so with complete accuracy for every individual.

The most common approach of many predictive tests is to use the assumed linear relationship between heart rate and the oxygen consumption or the equivalent workrate (Maritz et al, 1961). Where data are extrapolated from one or more measured sub-maximal values to predict a maximal value there are three significant assumptions: 1) prediction of maximal heart rate, 2) linearity of the heart rate / oxygen consumption relationship, 3) if oxygen cost is not measured but calculated from workload this assumes a constant mechanical efficiency.

Prediction of maximal heart rate (usually 220-age) is subject to considerable individual variations within a given age group, at least $\pm$ 10 beats.min^{-1} (Astrand & Rodahl, 1986).

Linearity of the heart rate / oxygen cost relationship is dependant on a constant (or steadily increasing) cardiac output and arteriovenous (a-vO_2) difference. Divergence from linearity has generally been reported to be small and statistically insignificant (Shephard, 1984). It is however not unusual that at the highest workloads, older subjects experience difficulty in sustaining the stroke volume, leading to a disproportionate increase in heart rate (Shephard, 1984). In younger subjects an efficient redistribution of blood flow to the working muscles at the higher workrates can result in a disproportionate increase in oxygen uptake (Astrand & Rodahl, 1986). Compared to maximal tests the individual variability of oxygen consumption at submaximal levels is much greater and increases with decreasing intensity of the workload (Nordrehaug, et al., 1991).

Mechanical efficiency is not constant and is dependant on exercise mode. Typical variations in efficiency are 4-5% for cycle ergometry, 10% for stepping (Thomas et al., 1992) and 10% for treadmill walking (Shephard, 1984).

Coleman (1976) was able to show that extrapolation of a best straight line from 4-6 points on a $\dot{V}O_2$ / HR plot to predict $\dot{V}O_2$max. yielded a more accurate estimate of actual $\dot{V}O_2$max. than the Astrand-Ryhming nomogram, which relies on the values from one workload. The estimated errors were 8% for the extrapolation and 15% for the nomogram.

Predictive tests of $\dot{V}O_2$max. that use the linear relationship between workload and oxygen cost rely on a 'steady state' being achieved. This is where the oxygen consumption has met the demand and there is no further increase in the oxygen consumption at a specific workload. The time required to attain a steady state oxygen consumption is dependant on the fitness of the individual (Powers et al., 1985), age (Babcock, et al., 1992) and the magnitude of the increase in the workload (Montoye, 1975). Stabilisation of the heart rate is difficult to achieve as with prolonged exercise there is a tendency for the heart rate to rise. This gradual increase in heart rate with time has been attributed to increases in body temperature (Montoye, 1975).

It is generally accepted that a steady state will be achieved more quickly with smaller increases in intensity, especially at smaller relative workloads (Montoye, 1975). Montoye found that a three-minute workload was sufficient to reach a plateau in oxygen consumption but not HR which continued to rise during the three minutes. Nagle et al. (1971) has reported that 2 minutes was long enough to reach a steady state. Both of these studies used continuous protocols allowing a faster achievement of steady state, discontinuous protocols generally require longer stages to allow time to warm up for each workload.

Subjects aged 60 or more years had an effective time constant for oxygen consumption that was 40% longer than in 30 to 40 year olds, indicating a slower response of the aerobic system to increased demand at onset of exercise (Babcock et al., 1992). From a group of highly trained subjects with similar training habits those with a higher $\dot{V}O_2$max. achieved a more rapid $\dot{V}O_2$ adjustment at the onset of work (Powers et al., 1985).

Washburn & Montoye (1984) compared the three most popular methods for estimating $\dot{V}O_2$max.; Astrand-Rhyming nomogram, the extrapolation method described by Maritz

et al. (1961) and, the procedure of Margaria et al. (1965). The Astrand-Rhyming nomogram tended to overpredict $\dot{V}O_2$max. whereas the methods of Maritz and Margaria underpredict, except for the 10-14 age group where all methods overpredicted. The method by Maritz gave the smallest standard error of prediction and the highest correlation coefficient with measured $\dot{V}O_2$max., making it the best predictive method. One study compared 5 different prediction tests, two of which individuals had to cover a specified distance as fast as possible (1.5 mile run, one mile walk). Two tests examined the use of HR response to submaximal workloads on a cycle ergometer (Astrand-Rhyming nomogram, extrapolation method), while the last test examined the recovery heart rate from a stepping task (Zwiren et al., 1991). they concluded that for females aged 30-39 years the mile walk test and the 1.5 mile run gave the most accurate assessment of $\dot{V}O_2$max. The relatively poor performance of the extrapolation method in this study may be explained by the fact that only two workloads were used to create the regression and a small error in one point would be magnified to a larger error in prediction. Previous studies have recommended that 4-6 points are used to create the regression equation and give a more accurate extrapolation (Coleman, 1976; Maritz et al., 1961).

Within a longitudinal 6-week study the correlation between the $\dot{V}O_2$max. predicted by the Astrand-Rhyming nomogram and measured ranged from 0.91 to 0.61. The highest correlation values occurred prior to exercise training and again after the third and sixth week , with the lowest during the first two weeks of training (Rogers et al., 1993).

The present consensus is that predictive tests provide a fairly accurate estimate of the mean $\dot{V}O_2$max. for a group. If applied to an individual they are only satisfactory as a rough approximation of $\dot{V}O_2$max. (Shephard, 1984; Washburn & Montoye, 1984). Predictive tests are limited in comparisons of one individual to another, but are a valuable training guide to determine whether or not a training programme is effective (Astrand & Rodahl, 1986).

Gedhill (1990) concluded that , despite the 10-15% error associated with the results of predictive tests these protocols are recommended for the assessment of cardiovascular - respiratory fitness in apparently healthy sedentary individuals.

1.2.3. MEASUREMENT MODALITY

Direct and predictive tests to measure $\dot{V}O_2$max. use a wide range of equipment to achieve the desired workload. Treadmill, cycle ergometer and step are the most commonly used. For very obese or unfit individuals the cycle ergometer is preferred as it supports the body mass, and can be set at lower workloads. Some individuals may find treadmill walking or running difficult and thus require a period of familiarisation. Wall & Charteris (1980, 1981) have shown that there is an initial rapid accommodation to treadmill walking followed by a much longer gradual period of habituation. They recommend that measurements should not be made on subjects during their initial ten minutes walking as they will not have reached a steady state walking pattern.

The treadmill has generally been shown to produce the largest $\dot{V}O_2$max. values (Astrand & Rodahl, 1986).

On average, protocols that use a cycle ergometer produce $\dot{V}O_2$max. values that are 4-8% lower than treadmill values. This is thought to be due to the larger muscle mass involved being able to extract more oxygen, although highly trained cyclists can normally achieve similar values on ergometer and treadmill (Astrand & Rodahl, 1986).

Matching the energy cost of cycling to that achieved in treadmill exercise places a greater stress on the subject evidenced by a higher heart rate , blood pressure and perceived leg fatigue (Grant et al., 1992).

1.3. IMPROVEMENT OF AEROBIC POWER

The magnitude of increase in $\dot{V}O_2$max. during 2-3 months of training, 30 min per session, 3 times per week is in the order of 10-20%, but with large individual variation (Astrand & Rodahl, 1986). The percentage improvement in $\dot{V}O_2$max. is dependant on the initial value (Powers & Howley, 1991; Wenger & Bell, 1986). Individuals with high initial $\dot{V}O_2$max. values can expect only small changes of 2-3% whereas those with low $\dot{V}O_2$max. values can have increases of up to 30-50% (Powers & Howley, 1991). Very unfit individuals may be able to improve $\dot{V}O_2$max. with virtually any exercise even at the lowest intensities.

The American College of Sports Medicine (ACSM) has based on the existing evidence on exercise prescription produced recommendations on the quantity and quality of training for developing and maintaining cardiorespiratory fitness (ACSM, 1990).

1.3.1. INTENSITY

In their position stand the ACSM recommend that exercise intensity should be 60-90% maximum heart rate (HRmax), or 50-85% of $\dot{V}O_2$max. (ACSM, 1991). This relationship between %HRmax and $\dot{V}O_2$max. suggested by ACSM has recently been questioned. Swain et al. (1994) looking at the relationship between percentage of $\dot{V}O_2$ max. and percentage of HRmax in 81 men and 81 women (aged 18-34 years) found that their %HRmax for a given %$\dot{V}O_2$max. was significantly higher than that used by the ACSM. The authors stated that the inaccuracies of the previous estimates were due to the mathematics involved. Previous studies had used %HRmax as the independent variable and combined the data from many individuals to produce a single group regression. Whereas this study used %$\dot{V}O_2$max. as the independent variable and also performed regressions for each subject and used these regressions to determine each subject's %HRmax. At 40%$\dot{V}O_2$max. the young adults of this study averaged 63% HRmax which was 15% higher than the ACSM value. As both regression lines meet at 100%$\dot{V}O_2$max. and 100%HRmax the difference between the values of %HRmax diminishes but there is

still a significant difference at 85%$\dot{V}O_2$max. This study also showed that there was a trend for slightly higher %HRmax values for women but this failed to reach significance ($P<0.10$). Fitness also affected the %HRmax required for a given %$\dot{V}O_2$max., in that the fitter subjects (those with the highest $\dot{V}O_2$max. values) needed to work at a significantly higher %HRmax than the least fit subjects, especially at lower %$\dot{V}O_2$max. The authors concluded that for healthy young adults to achieve 40%, 60%, 80%, and 85% of $\dot{V}O_2$max., target heart rates should be 63%, 76%, 89% and 92% HRmax respectively. Gossard et al. (1986) compared the effect of a 12 week low (42-60 %$\dot{V}O_2$max) and high (63-81 %$\dot{V}O_2$max) intensity training programme on sedentary middle-aged men. Training was performed 5 times per week, duration was adjusted to give both groups the same energy expenditure (about 300 kcal per training session). $\dot{V}O_2$max. was significantly increased by 8% in the low intensity group and by 17% in the high intensity group.

Wenger & Bell (1986) in their review reported that the greatest improvements in aerobic power occur when the greatest challenge to aerobic power occurs i.e. when exercise intensity is in the range 90-100% of $\dot{V}O_2$max. At the other end of the spectrum the 1990 ACSM position stand states that persons with low fitness levels can achieve a significant training effect with a sustained training heart rate as low as 40-50% of HR reserve (ACSM, 1990).

1.3.2. DURATION

When exercise is performed above the minimum intensity threshold the duration of that exercise is an important factor in the development of aerobic power. ACSM recommend that the duration of training should be 20-60 min of continuous aerobic activity. Improvements in aerobic power can be achieved with durations of 15 minutes or greater, however longer durations (> 35 minutes) result in larger improvements. There is a suggestion that longer duration, lower intensity exercise can produce as much benefit as short duration at higher intensity (Wenger & Bell, 1986). Milesis et al. (1976) found that

at the same intensity (85-90% of HRmax) exercise durations of 15, 30 and 45 min, for 20 weeks all improved $\dot{V}O_2$max. However, the 45 min group improved significantly more than the 15 min group and there was no difference between the 15 and 30 min groups.

1.3.3. FREQUENCY

The amount of improvement in aerobic power tends to plateau when the frequency of training is increased above 3 $d.wk^{-1}$. Studies of less than 2 $d.wk^{-1}$ tend not to show significant improvements in $\dot{V}O_2$max. (ACSM, 1991). With very high intensities (90-100 %$\dot{V}O_2$max) fewer days training are required to achieve improvements in VO_2max (Wenger & Bell, 1986).

1.3.4. MODE

The mode of exercise recommended by ACSM includes any exercise that involves large muscle groups in a maintained continuous rhythmical aerobic activity e.g. walking, running, jogging, cycling, dancing, rowing, stair climbing and swimming,

1.4. AEROBIC POWER AND CHD

1.4.1. AEROBIC FITNESS VS PHYSICAL ACTIVITY

The relationship between aerobic fitness, physical activity and health is a complex one. For example, physically fit individuals tend to be more active and active individuals tend to have higher levels of fitness (Bovens et al., 1993; Ekelund et al., 1988: Sidney et al., 1992). Physically fit and active individuals tend to be healthier (Ekelund et al., 1988; Sobolski et al., 1987), but health is required to be physically active and thus develop and maintain fitness.

There are many large epidemiological studies that have shown a significant negative relationship between levels of physical activity and the levels of CHD and all cause mortality (Dannenberg et al., 1989; Magnus et al., 1979; Paffenbarger et al., 1984,

1993). Similarly a negative correlation has been shown to exist between physical fitness and risk of CHD and all cause mortality (Blair et al., 1989; Brill et al., 1992; Ekelund et al., 1988; Hagan et al., 1991; Jette et al., 1992; Peters et al., 1983; Sandvik et al., 1993; Sobolski et al., 1987; Slattery & Jacobs, 1988). Physical activity and physical fitness are clearly related, Leon et al. (1981) reported that there was a significant positive correlation between treadmill performance (Bruce protocol) and leisure time physical activity. Ekelund et al. (1988) has also shown that fitness level, determined by HR at a submaximal workload, was closely related to regular physical activity. Forty nine percent in the most fit quartile reported regular physical activity, whereas 19% did so in the least fit quartile. The authors suggested that this demonstrated that possibly regular physical activity is more important than genetic factors in determining the level of fitness.

Low levels of both physical fitness and physical activity are recognised independent risk factors for CHD. Care should be taken as, although physical fitness and physical activity are clearly related and seem to have a similar effect on health, the two terms are completely different measures and therefore should not be used interchangeably. This review will focus on the relationship between aerobic fitness and CHD only.

The largest study in this area, carried out at the Cooper Clinic in Dallas, looked at the relationship between treadmill endurance time and all-cause mortality of 10,244 and 3120 middle-aged men and women respectively, after an 8 year follow-up. Blair and colleagues (1989) reported that there was a dramatic drop in the all-cause mortality rates across the physical fitness quintiles, there was a 70% reduction in mortality rates from the least to the most fit men and an even larger 80% reduction from the least to the most fit women. This proved to be a strong relationship as these trends remained significant even after adjustment for age, smoking habit, cholesterol level, systolic blood pressure, fasting blood glucose level, paternal history of coronary heart disease and follow-up interval. There was a similar trend when only death from cardiovascular disease was considered. The major reduction in CHD mortality between the first and second quintile of fitness suggests that a moderate improvement in fitness levels would have a marked impact on mortality rates (Blair et al., 1989). On the basis of this study Blair et al.

calculated that if all 'unfit' persons became 'fit', reductions in death rates of 9% in men and 15.3% in women might be expected.

Ekelund et al. (1988) categorised 3106 healthy men into fitness quartiles based on their HR at a sub-maximal workload. After an average follow-up of 8.5 years they were able to show that the cumulative mortality was much higher in the quartile with the lowest level of fitness than in the most fit quartile; the rate of death from CHD was 6.5 times higher in the low fitness quartile. With a longer follow-up time (16 years) in 1960 healthy men 40 to 59 years of age, Sandvik et al. (1993) also demonstrated a graded, inverse association between physical fitness (defined as maximal work capacity) and mortality from cardiovascular causes. Subjects in the highest fitness quartile also seem to be protected from death from all causes. The data on cumulative mortality from both Ekelund et al. (1988) and Sandvik et al. (1993) show a very similar pattern, with a greater diversion in the curves for each fitness quartile with follow-up time. Sandvik et al. (1993) commented that they would have failed to observe the marked difference in mortality rates between subjects in the intermediate fitness levels and those in the highest fitness levels if the follow-up period had been limited to 10 years.

One criticism of all these studies is the large assumption that physical fitness measured only once at the beginning of a study will be representative of the physical fitness of that subject for the duration the follow-up period. Although technically very difficult to achieve there must be a case for a study of a reasonable sample size that would measure both physical fitness and physical activity every 3-4 years during at least a 15 year follow-up. The results from such a study would give a much clearer indication of the relationships between physical fitness, physical activity and subsequent mortality.

A recent cross-sectional study of 2009 men and 899 women over 40 years of age who were active in sports showed that subjects with higher cardiovascular fitness (assessed from maximal power output on a progressive ergometer test) had significantly better CHD risk profiles. The authors concluded that in an active population, the strength of the association between cardiovascular fitness, physical activity and cardiovascular risk

factors was similar to that in studies on less active men and women (Bovens et al., 1993).

Are improvements in $\dot{V}O_2$max. required to reduce risk of CHD?

Changes in $\dot{V}O_2$max. do not always mirror the changes in other health variables (Duncan et al. 1991). The American College of Sports Medicine (ACSM) recognised this fact in their position statement by stating that 'the quantity and quality of exercise needed to attain health-related benefits may differ from what is recommended for fitness benefits' and 'ACSM recognises the potential health benefits of regular exercise performed more frequently and for a longer duration, but at lower intensities than prescribed in this position statement' (ACSM, 1990).

2. BLOOD PRESSURE

The heart continually pumps blood along the arteries creating pressure therein. Because the heart pumps in a pulsatile manner, the arterial pressure fluctuates between a systolic value of about 120 mmHg and a diastolic pressure of about 80 mmHg, in the normal individual (Guyton, 1980). The distribution of blood pressure in the population is roughly 'normal', but tends to be slightly skewed to the right (higher blood pressures)(Dawber, 1980). This skewing tends to increase with age, suggesting that blood pressure does not rise with age in everybody, only in a proportion of the population as they grow older (Hamilton et al., 1954; Epstein, 1983). This increase in blood pressure with age is partly due to the loss of elasticity in the arterial wall. This is reflected in the increased incidence of high blood pressure with age (Hamilton et al., 1954; Paffenbarger et al., 1983)

Mean Arterial blood Pressure (MAP) is a function of Cardiac Output (CO) and Total Peripheral Resistance (TPR). Therefore any change in blood pressure is due to a change in cardiac output, or total peripheral resistance, or both.

There are many complex control systems involved in maintaining a mean arterial pressure so that all the tissues of the body are properly perfused with blood. These control mechanisms can basically be divided into long term and short term control of arterial blood pressure. Short-term control of blood pressure is achieved by a variety of neural and hormonal reflexes that act to provide a rapid stabilisation of arterial pressure. These mechanisms act within seconds to minutes to return blood pressure to normal levels. These reflexes lose their power to control pressure after a few hours because many of the neural pressure receptors adapt and lose their responsiveness.

Therefore there is a separate group of mechanisms that control long-term blood pressure for days, months and years. For an extensive review of the long term control of arterial blood pressure see Cowley (1992).

It should be noted that blood pressure is a continuous variable with no clear demarcations between normal and elevated values, therefore any definition of hypertension is more or less arbitrary. Tables 2, 3 show the classification of blood pressure constructed by the 1988 Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure

Systolic (when diastolic blood pressure is <90 mmHg)	
<140	Normal blood pressure
140-159	Borderline isolated systolic hypertension
>160	Isolated systolic hypertension

Table 2. Classification of Systolic Blood Pressure: 1988 Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure

Blood Pressure range (mmHg)	Category
Diastolic	
<85	Normal blood pressure
85-89	High normal blood pressure
90-104	Mild hypertension
105-114	Moderate hypertension
>115	Severe hypertension

Table 3. Classification of Diastolic Blood Pressure: 1988 Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure

Excluding those already on drug treatment, 6% of men and 3% of women in Britain have a diastolic blood pressure above 95 mmHg (BHF, 1991).

By definition, essential hypertension is an elevation of blood pressure above normal levels by an unknown cause (Epstein, 1983; Hagberg, 1990). A large amount of research, both animal and human has tried to establish the cause of essential hypertension. There could of course be many different causes.

There is thought to be a genetic factor to hypertension, as offspring of hypertensive parents are at greater risk of developing hypertension (Paffenbarger et al., 1983). The correlation between parent / child hypertension is significant but this relationship is not particularly strong, leaving much of the variance 'unexplained' (Epstein, 1983).

One of the key questions in the search for the cause of hypertension is whether the mechanisms responsible for maintaining the hypertension were responsible for the primary development of the condition. Most hypertensives have normal or subnormal blood volume and normal cardiac output, but they have a greatly increased total peripheral resistance, which accounts for almost all of the increase in arterial pressure(Guyton, 1980; Hagberg, 1990).

Guyton (1980), in his extensive review of arterial pressure and hypertension, develops the theory that essential hypertension is the result of an intra-renal abnormality. The most important evidence for this is that in a hypertensive individual the kidneys excrete salt and water at normal rates only when arterial pressure is at a hypertensive level, signifying a shift in the renal function curve to a higher pressure level. Also if the kidney of a hypertensive rat is transplanted to a normal rat, hypertension is quickly established by a large increase in total peripheral resistance. The cause of the dysfunction of the kidney is unknown. Guyton (1980) postulated that this could be caused by an earlier volume loading event, with increased blood volume and cardiac output. Both of these would disappear as hypertension persisted.

2.1. MEASUREMENT OF BLOOD PRESSURE

Measurement of blood pressure is achieved by two main methods, directly by insertion of a needle or catheter into the arterial tree and connected to a pressure transducer or indirectly by the occluding cuff ausculatory technique. It should be pointed out that the assumption that the two are measuring the same parameter is incorrect. The indirect method attempts to measure the lateral forces on the wall of the brachial artery, whereas the direct method measures the forces at the leading edge of the pressure wave moving down the column of blood in the artery. The two measures will be similar and are highly correlated but they could hardly be expected to be the same (Raferty, 1991).

The most common method used in research is the indirect method. This involves applying a cuff, which encloses an inflatable bladder, around the upper arm. This bladder is then inflated until it occludes blood flow in the brachial artery. The cuff is then allowed to deflate while the observer with the aid of a stethoscope listens over the brachial artery below the cuff, while pressure changes are monitored by a mercury column. When pressure in the artery just exceeds the pressure in the cuff and the critical opening pressure of the brachial artery is exceeded, blood will start to spurt along the blood vessel in a turbulent pulsatile flow, making an audible tapping noise as blood forces open the artery at the peak of each systole. The appearance of this sound is known as Korotkoff I and is taken as the systolic blood pressure. As pressure in the cuff is further released the pulsatile sound gains volume until the critical opening pressure is exceeded in diastole and the quality of each sound decreases and becomes muffled, known as Korotkoff IV which can be taken as diastolic blood pressure. It is however more common to take Korotkoff V as the diastolic blood pressure, which is the disappearance of any pulsatile sound and there is smooth laminar flow of blood in the vessel.

Electronic developments have allowed the development of ambulatory measurement devices that use the same occluding cuff principle but can measure blood pressure at

regular intervals for up to 24 hours and store all the measurements. This allows 24 hour measurement of blood pressure without much inconvenience to the subject.

There are however several errors associated with blood pressure measurement by sphygmomanometry. These errors occur as a result of defective equipment, observer error and failure to standardise the measurement technique (AHA, 1988). Equipment that is not properly maintained and the use of inappropriate cuff sizes will lead to erroneous measurements. Observers need to be trained to correctly recognise the Korotkoff sounds and refrain from digit preference. A standardised technique is vital to give an accurate assessment of an individual's blood pressure.

Any study that wishes to measure changes in casually measured blood pressure must employ a very rigid protocol, that includes an adequate number of preliminary measurements to give a stable baseline measurement. It has been shown that resting casual blood pressure tends to drop after repeated measurements on consecutive days or weeks (Kaufman et al., 1987; Kuyonaga et al., 1985; Urata et al., 1987). Also, two studies have shown different results for the same study depending on the measurement conditions. Van Hoof et al. (1989) reported that after training, resting systolic blood pressure was significantly reduced when the measurement was made while the subject sat on the cycle ergometer prior to exercise but there was no significant changes in blood pressure when the measurement was taken after a 10 minute rest in a comfortable chair. Similarly Seals & Reiling (1991) measured casual blood pressure in a number of positions (supine, sitting and standing) and in two different experimental conditions as well as 24 hour ambulatory measurements. In the first experimental condition, blood pressure was measured in a standard clinic setting, the second measurement was incorporated into the experimental protocol that included measurement of heart rate and cardiac output. The measured results from these two settings were markedly different, both measured similar baseline values but the measured changes as a result of the aerobic training were quite different. All subjects including the control group showed significant reductions in blood pressure from measurements in experimental condition one, but for measurements in condition two the magnitude of changes in the intervention group was

greatly reduced and there were no significant changes for the control group. Interestingly, changes in the 24-hour ambulatory blood pressure measurements were smaller and less significant than both casual measurements. Therefore, this study employed three different measurement conditions for the same subjects and arguably had three different results. The authors concluded that changes in casually determined blood pressure at rest are dependant on measurement conditions and, most importantly, do not necessarily reflect the magnitude or even the direction of changes in arterial pressure throughout an entire day. They did report that average casual levels at rest were most closely linked with daytime ambulatory levels.

A further demonstration of the effect of measurement environment can be seen by the acute pressor effect that the appearance of a doctor for a bedside visit creates, termed 'white coat' hypertension (Siegel et al., 1990). Mancia et al. (1983) using an intra-arterial recorder showed a marked rise in systolic (26.7 ± 2.3 mmHg) and diastolic (14.9 ± 1.6 mmHg) blood pressure and heart rate in 47 normotensive and hypertensive patients as soon as the doctor appeared, before an attempt was made to measure blood pressure by normal sphygmomanometry. The peak rise in blood pressure occurred within 1 to 4 minutes and this steadily declined to near pre-visit levels by the end of the visit (15 minutes). A second visit by the same doctor did not reduce the magnitude or duration of the pressor response.

It has also been shown that following moderate aerobic exercise there is a significant post-exercise reduction in resting blood pressure lasting for up to 90 minutes (Bennet et al., 1984; Kaufman et al., 1987; Wilcox et al., 1982). Therefore it is important to consider the conclusion of Kaufman et al. (1987), that studies of blood pressure response to exercise should ensure a stable baseline measurement in order to prevent over estimation of the blood-pressure-lowering effects of exercise.

The available evidence suggests that any study on changes in casually measured blood pressure must employ a very rigid protocol that has an adequate number of preliminary measurements to give a true stable baseline measurement and that identical measurement conditions should be repeated throughout the study.

Cowley (1992) in his review of long term control of arterial blood pressure commented that, given the enormous lability of arterial pressure related to movement and environmental stimulus, brief recording sessions are insufficient to accurately reflect the 24-hour mean arterial pressure. Therefore 24-hour ambulatory measurement may offer some advantage over casual measurement, although most studies report measurement problems for some subjects with this method. Studies that have used 24 hour ambulatory measurement have generally found much smaller or no changes in blood pressure as a result of an aerobic training programme (Blumenthal et al., 1991; Seals & Reiling, 1991; Van Hoof et al., 1989). One possible reason for the smaller changes is that in general, most of the reduction in blood pressure is found when the pressure is the highest, during the day (Van Hoof et al. 1989; Seals & Reiling 1991). Therefore the average 24-hour blood pressure will dilute the larger changes found during the day.

Measurement of blood pressure is not as simple as it may at first seem and there is a need for further well designed studies relating to exercise.

2.2. HYPERTENSION AND MORTALITY

Hypertensive individuals are at increased risk of developing CHD and /or having a stroke (Beevers, 1983). The Framingham study demonstrated that hypertensives had three times as many strokes and twice the occurrence of CHD compared to those not considered hypertensive (Kannel, 1974).

Numerous antihypertensive drugs are now available that can effectively reduce blood pressure but many have undesirable side-effects. In the MRC Trial of treatment of mild hypertension (1985) propranolol and bendrofluazide were able to immediately reduce blood pressure. This drop in blood pressure did reduce the number of strokes in the treatment group. However, the treatment group had the same incidence of coronary events as the control group which suggests that, although the treatment reduced blood pressure, it offered no protection against coronary events. The Multiple Risk Factor Intervention Trial (1982) used pharmacological treatment and counselling for cigarette

smoking and dietary advice for lowering blood cholesterol for a group of hypertensive individuals. They found that the intervention group had a similar all cause and CHD mortality compared to the non-intervention group. In contrast, the Veterans Administration Cooperative Group on Antihypertensive Agents (1967,1972) reported significantly reduced cardiovascular mortality and morbidity after pharmacological treatment of hypertension.

The Harvard Alumni Study (Paffenbarger et al., 1984) showed that hypertension greatly increased the risk of CHD but that increased physical activity reduced this risk. They also reported that conversion from being hypertensive to normotensive could eliminate 57% of the CHD risk. Paffenbarger et al. (1993) in the nine year follow up of the Harvard Alumni showed that hypertension was associated with double the risk of death from CHD. This risk is greatly increased if other risk factors are also present.

In an 8-year follow-up of 10,224 men and 3120 women, Blair et al. (1989) found that subjects in the lowest aerobic fitness category had a marked increase in relative risk of all-cause mortality regardless of resting systolic blood pressure. The relative risk of all-cause mortality increased as systolic blood pressure rose so that the lowest fitness category with the highest systolic blood pressure had a greatly increased relative risk. Interestingly the relative risk for those in the higher fitness categories did not seem to change as much with increasing systolic blood pressure.

Therefore, there is clear evidence that hypertension is a major risk factor for CHD and mortality, and that exercise or high levels of physical activity and fitness seem to offer some protection even when hypertension is present (Blair et al., 1989).

2.3. Exercise and the Prevention of Hypertension

In an attempt to understand the relationship between exercise and hypertension, researchers have frequently used animal models. Hypertension can be created in animal models by genetic influences (i.e. Spontaneously Hypertensive Rats (SHR)) or as the result of interventions such as hormonal injections (deoxycorticosterone acetate,

DOCA), increased salt intake and constriction of renal arteries. It should be noted that the effect on the cardiovascular system is dependant on the model used, and the models may not mimic the processes involved in the development or maintenance of human hypertension.

Some of these studies have shown that SH rats that are exercised, exhibit an elongated time course for the development of hypertension. Despite reductions in rat heart rate and body mass, the exercise did not prevent the development of hypertension in SH rats, it merely slowed the time course (Fregly, 1984; Tipton et al., 1983). Slowing of the development of hypertension is not found in all animal models, but the best results are found in SH rats and Dahl salt sensitive rats. Also, animal studies have shown that the intensity of exercise may be important in that higher intensity (>75%$\dot{V}O_2$max) exercise is associated with increases and not decreases in blood pressure (Tipton et al., 1983).

Human studies would seem to show a clearer relationship than animal studies. Blair et al. (1984), in their study of 4820 men and 1219 women, showed that those with low levels of aerobic fitness had a significantly higher relative risk of developing hypertension compared with those with higher fitness levels. Also the Harvard Alumni study (Paffenbarger et al., 1993) reported that, when data were adjusted for age and BMI, men who took vigorous sport for 1-2 hours per week had an appreciably lower risk of developing hypertension compared to those who took no vigorous sport. Further analysis revealed that previous physical activity, when younger at college, seemed to offer no protection against hypertension. Other key factors that contributed to the development of hypertension were: elevated student systolic blood pressure, BMI, gain in BMI since college and parental history of hypertension. In a study of middle-aged physicians, Darga et al. (1989) were able to show that the physicians who were regular runners (members of the American Medical Joggers Association) had much lower blood pressures, lower incidence of hypertension and lower medication rates for those who were hypertensive, compared to nonrunners (members of the American Medical Association). The runners, who ran 20-49 miles/week, were also leaner, drank less alcohol and had lower total cholesterol.

These human studies give clearer evidence of a relationship between regular exercise and the reduced incidence of hypertension than the animal models.

2.4. Exercise and the Treatment of Hypertension

Several epidemiological studies have shown that regular exercisers generally have lower blood pressure and lower incidence of hypertension (Blair et al., 1984,1989, 1993; Darga et al., 1989; Paffenbarger et al 1984, 1993). This cross-sectional evidence does not provide casual evidence for exercise as an effective treatment for hypertension. Hagberg (1990) in a meta-analysis of 25 studies examining the blood pressure lowering effect of endurance training concluded that on average systolic blood pressure was lowered by 10.8 mmHg and diastolic blood pressure was lowered by 8.2 mmHg as a result of the various training programmes. A more recent meta-analysis (ACSM, 1993) of 40 studies published up to 1992 revealed that, in subjects with systolic BP > 140 mmHg and diastolic > 90 mmHg, systolic blood pressure was reduced by approximately 11 mmHg and diastolic blood pressure by approximately 9 mmHg.

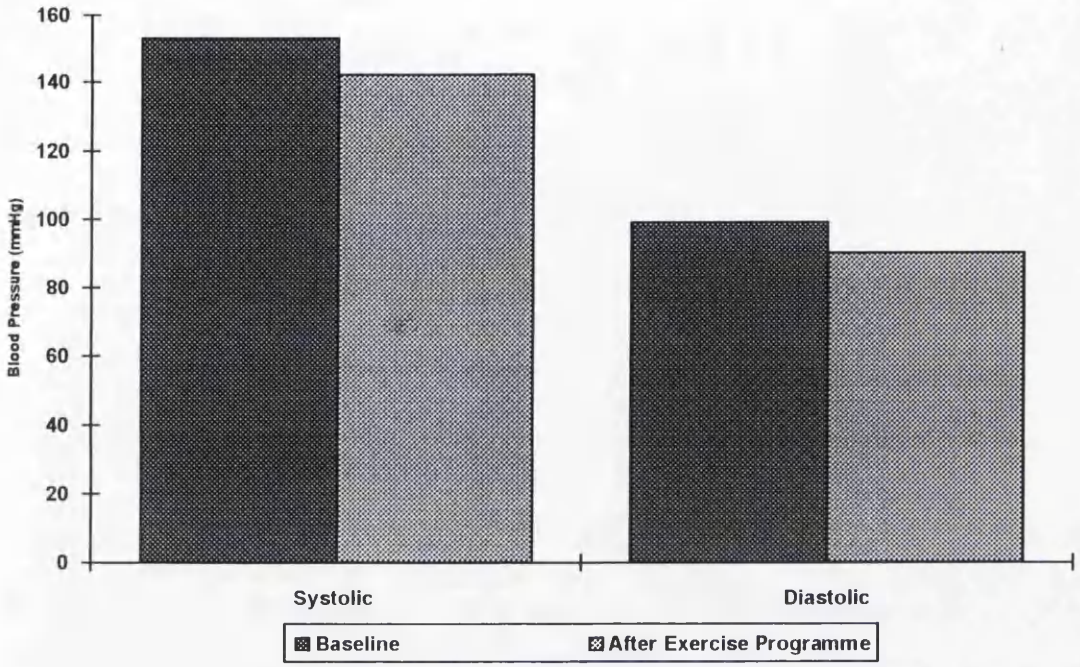

Figure 1. Results of meta-analysis of 40 longitudinal studies measuring the antihypertensive effect of aerobic exercise in subjects with high blood pressure (ACSM, 1993)

Therefore, both of these meta-analyses produced similar conclusions that for individuals with hypertension endurance training reduces systolic and diastolic blood pressure by on average 10 mmHg. For most individuals, reductions of this magnitude would not restore their blood pressure back to normal levels but this a clinically beneficial reduction, especially considering the other benefits of regular exercise.

The smaller numbers of aerobic training studies that have used 24-hour ambulatory blood pressure measurement (Seals & Reiling, 1991; Van Hoof et al., 1989) have generally shown much smaller reductions in blood pressure. They do, show that the largest reductions in blood pressure are found during the day therefore an average 24-hour value would tend to dilute this daytime effect. Although Blumenthal et al. (1991), measuring daytime ambulatory blood pressure, found that after a 4-month aerobic training programme there was no significant reduction in blood pressure. Whereas there was a significant reduction in casually measured blood pressure, with the greatest reductions in

the subjects who had the largest improvements in aerobic fitness. More studies using 24-hour ambulatory measurements need to be carried out to confirm the previous findings. The positive conclusions from the meta-analysis of Hagberg (1990) and ACSM (1993) obviously apply to the majority of the studies of the response of blood pressure to exercise. However there are some studies that have shown very small non-significant changes or no change at all in blood pressure. One reason for this variation could be the subjects used. It is unlikely that, if subjects at baseline are normotensive, there will be any reduction in blood pressure. Also the majority of studies have been carried out on men and only a few have included women. In fact there seem to be no studies that have exclusively used hypertensive women. From the available information, hypertensive women react to aerobic exercise in a similar way to men. Hagberg (1990) reported that women may have greater reductions in blood pressure than men. Further research on the response of hypertensive women to an aerobic exercise programme is required .

Another reason for these variable reductions in blood pressure could be the type, duration and intensity of exercise used in the study. The majority of studies have used exercise protocols that are in agreement with the ACSM (1990) guidelines for the Recommended Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory Fitness in Healthy Adults. However some studies have shown that higher intensity exercise may not be as effective at lowering blood pressure. Jost et al. (1990) showed that when a group of distance runners and swimmers changed to high intensity training they actually had an increase in blood pressure. Also SH rats developed an increased rather than a decreased blood pressure when exercise intensity exceeded 75% VO_2 max (Tipton et al., 1983). The majority of studies that have demonstrated significant reductions in blood pressure have used exercise intensities of 60-80% of maximum heart rate. Hagberg (1990) in his review reported a negative correlation between change in blood pressure and exercise intensity, suggesting that greater reductions in blood pressure were found at lower intensities of exercise.

Most studies have used exercise sessions of 30-60 minutes and there is little evidence that longer exercise sessions are more effective at reducing blood pressure. However

Milesis et al. (1976) showed that, with a relatively high intensity training programme,(85-90% of maximum HR), normotensive subjects who exercised for 45 minutes per session had significantly larger reductions in systolic and diastolic blood pressure compared to the 15 minute group. The 45 minute group also had a significantly larger reduction in body fat % which may account for the larger reduction in blood pressure.

The duration of the training programme can also effect the magnitude of the reduction in blood pressure. The majority of studies have lasted from 4 weeks to 12 months (Duncan et al., 1985; Kiyonaga et al., 1985; Martin et al., 1990; Seals & Reiling, 1991; Van Hoof et al., 1989), with even the shortest (4 weeks) giving significant reductions in blood pressure (Meredith et al., 1991, Urata et al., 1987). In a recent study, older hypertensives showed small further reduction in blood pressure after 12 months compared to 6 months (Seals & Reiling, 1991). Kiyonaga et al. (1985) reported that the blood pressure of 12 hypertensive subjects had stabilised after 5 weeks of aerobic exercise at 50% $\dot{V}O_2$ max. At 10 weeks the exercise intensity was increased to account for the increase in fitness, for a further 10 weeks. A new lower blood pressure level was quickly established and was maintained for the remaining 10 weeks. This indicates a complex relationship between intensity and duration. Hagberg (1990) in his meta-analysis concluded that reductions in systolic blood pressure were not correlated to the length of training, whereas the reduction in diastolic blood pressure was. Therefore the antihypertensive effect of exercise would seem to occur fairly quickly (within 4 weeks) but beyond this there are minimal further reductions without adjustment in exercise intensity.

Studies that have investigated the antihypertensive effect of exercise have concentrated on dynamic endurance-type training like walking, jogging, cycling and swimming. In addition, anaerobic training has been shown to be effective in reducing blood pressure, although the reductions were smaller than that found for an equivalent period of aerobic training (Norris et al., 1990). Hypertensives have normally been advised not to undertake resistive conditioning as the acute increases in blood pressure associated with static muscle contraction were considered dangerous. However Harris & Holly (1987)

demonstrated that circuit weight training, that involves moving a moderate amount of weight in a continuous fashion, was effective at improving $\dot{V}O_2$ max, various strength measures and in lowering diastolic blood pressure.

As well as the long-term changes in resting blood pressure with endurance training, there is evidence of an acute effect of a single bout of endurance exercise. Several studies have reported this finding (Bennett et al., 1984; Floras et al., 1989; Kaufman et al., 1987; Piepoli et al., 1993; Raglin & Morgan, 1987; Urata et al., 1987; Wilcox et al., 1982) where blood pressure has remained depressed for up to 120 minutes after exercise. This reaction is not limited to hypertensives but is also found in borderline hypertensives (Floras et al., 1989) and normotensives (Bennett et al., 1984; Kaufman et al., 1987; Piepoli et al., 1993; Wilcox et al., 1982). Bennett et al. (1984) reported that the time course for this reduction in blood pressure was different in normotensives compared to hypertensives. In a protocol of five, 10 minute exercise bouts separated by 3 minutes of rest, the hypertensive subjects exhibited reductions in blood pressure after the first exercise bout whereas the normotensive subjects did not have a reduction in blood pressure until 5 minutes after the last exercise bout. This effect would suggest that for hypertensives there could be a real benefit of several bouts of aerobic exercise during the day effectively maintaining a reduced blood pressure for most of the day.

The mechanisms responsible for this acute post-exercise reduction in blood pressure is unclear. A study by Floras et al. (1989) suggests that it is due to a reduction in sympathetic activity.

The available evidence indicates that an exercise programme of aerobic exercise may not lower blood pressure to normal values but has the potential to lower blood pressure by about 10mmHg for hypertensives.

3. BODY COMPOSITION

3.1. DEFINITION OF BODY COMPOSITION

One of the most basic methods of describing body composition is from 'average weight for height' tables. These tables constructed by statisticians were based on the average American population. Data, mainly from Insurance Companies were used to produce tables of 'desirable' weights for certain heights. The tables have subsequently been used to describe under-weight, desirable, and over-weight categories, and as such have been used as an aid in health education. By adjusting for height, sex and perhaps age or frame size, a person is theoretically able to evaluate his/her body weight in terms of health risk. Unfortunately many individuals may be over their ideal weight for height and not have a high percentage body fat. Body builders and athletes from power sports for example will have a greatly increased lean body mass (muscle) which will take them above their ideal weight for height, but they will have very low percentage body fat.

The data from which these tables were constructed have been heavily criticised as unreliable (Harrison, 1985; Knapp, 1983). Many of the heights and weights included were self-reported, while others were measured with subjects wearing shoes and fully clothed. In a methodological critique of the 'ideal weight' concept, Knapp (1983) concluded that, perhaps the best that we can hope to do is to study outliers with extreme care, using various indicators of mortality, morbidity and psychological well-being and switch the focus from 'ideal weight' to 'dangerous weight'.

A more detailed description of a typical body composition of young adult men and women is described in Table 4.

Characteristic	Males	Females
Height	1.74 m	1.64 m
Body Mass	70.0 kg	56.8 kg
Muscle	31.4 kg	20.5 kg
	44.8 %	36.0 %
Bone	10.5 kg	6.8 kg
	14.9 %	12.0 %
Essential Fat	2.1 kg	6.8 kg
	3.0 %	12.0 %
Storage Fat	8.4 kg	8.5 kg
	12.0 %	15.0 %
Total Fat	10.5 kg	15.4 kg
	15.0 %	27.0 %
Remainder	17.7 kg	14.2 kg
	25.3 %	25.0 %

Table 4. Body Composition of typical young adult men and women. (adapted from Lamb, 1984)

A much simpler method of describing body composition is the two-compartment model that consists of fat mass and fat free mass.

The fat mass can change dramatically (in comparison with modest changes in fat free mass) and can range from very small values of <5% up to 70 % in very obese individuals (Ward 1984).

Definitions of obesity suffer similar difficulties to those for hypertension. As body weight and body fat are continuous variables that are normally distributed around a mean value (Dawber, 1980), there are no clear dividing lines for different weight categories. Obesity

is generally defined as, an excessive enlargement of the body's total quantity of fat (Katch & McArdle, 1988). In actual measurement this has been interpreted as a Body Mass Index (BMI) of greater than 30 (Royal College of Physicians, 1983), or greater than 20% body fat for young men, 25% for older men and greater than 31% body fat for young women or 37% for older women (Katch & McArdle, 1988). Table 5 shows the weight classification used in the recent Allied Dunbar National Fitness Survey (1992) adopted from the Royal College of Physicians 'Report on Obesity' (1983)'.

Weight Classification	BMI Value Men	BMI Value Women
UnderWeight	20.0 or less	18.6 or less
Acceptable	20.1-25.0	18.7-23.8
Mildly Overweight	25.1-29.9	23.9-28.5
Obese	30.0 or more	28.6 or more

Table 5. Classification of weight categories based on Body Mass Index (BMI) from Allied Dunbar National Fitness Survey (1992)

There is a trend for increased incidence of obesity with age, especially in women (Kluthe & Schubert, 1985). Also post-menopausal women not on Hormone Replacement Therapy (HRT) have been shown to have more total fat and a more 'central' or 'truncal' located fat compared with pre-menopausal women (Greaves et al., 1992).

It is common to use adipose tissue and fat in terms of fat thickness, fatfolds or fat areas interchangeably. Although convenient this is wrong. Fat is a chemical compound, but adipose tissue contains varying amounts of fat, and therefore the densities of the two are different (Martin & Drinkwater 1991).

Changes in body composition generally involve changes in muscle and storage fat as the values of the other components are unlikely to change. With obesity there is an increase

in storage fat by either an increase in the size of fat cells (hypertrophy-obesity) or by an increase in the number of fat cells (hyperplasia-obesity).

The number of fat cells increases in early childhood but, after adolescence there are thought to be only increases in the size of the fat cells (Katch & McArdle, 1988). However, there is considerable controversy due to the technical difficulty of actually counting the number of fat cells (Brodie 1988). There is also considerable debate as to whether adipose tissue cellularity can be altered in childhood. Does over feeding in childhood lead to an excess hyperplasia and obesity in adulthood or is this genetically determined?

The normal pattern of adipose tissue development in animals can be altered by early over feeding. Several studies have shown that early over feeding results in an increase in the number of fat cells in adulthood (Greenwood, 1985). This debate is fuelled by the fact that 80% of obese children remain obese as adults (RCP, 1983). Weight loss does not decrease the number of fat cells but can greatly reduce the size (Katch & McArdle, 1988).

In obesity, there are differences in the distribution of the excess body fat. In the majority of men, adiposity is generally confined to the upper-body and prevalence of metabolic abnormalities rises in proportion with degree of obesity (Kissebah et al., 1982; Pouliot et al., 1994; Slattery et al., 1992). Whereas in women there is a distinct division of upper-body obesity and lower-body obesity. Upper-body obesity is most strongly associated with hyperlipidemia (Kanaley et al., 1993; Pouliot et al., 1994), hypertension, hyperinsulinemia (Kissebah et al., 1982; Pouliot et al., 1994) and diabetes (Kissebah et al., 1980), whereas lower-body obesity is not (Stefanick, 1993). The site of adiposity in the upper-body obese women consists of large fat cells, whereas lower-body obese subjects have fat cells of a normal size (Kissebah et al. 1982). This would suggest that fat cell hypertrophy rather than hyperplasia is responsible for several metabolic aberrations.

3.2. Measurement of Body Composition

Body composition can be measured either directly or indirectly. Direct measurement of body composition can only be made from cadaveric analysis. Dissection of a cadaver can give a two-compartment model of body composition namely fat mass and fat-free mass. Actual fat mass can only be defined as the ether-extractable constituents (Keys & Brozek, 1953). Unfortunately only 8 cadavers have been fully analysed in this way (Martin & Drinkwater, 1991). Most cadaver studies have dissected and weighed all visible adipose tissue. Although the adipose tissue weight is very similar to actual fat mass there will be a slight difference as the major organs and undifferentiated soft tissues, such as major vessels nerves, and muscles will contain fat (Brodie, 1988).
The fat-free compartment has also been described as the lean body mass compartment, although the two terms are not interchangeable. Lean body mass is probably in most cases a more accurate description as it is used to describe a body compartment that contains a small amount of essential fat.

- Indirect measurement of body composition can be divided into reference methods (dual-energy X-ray absorptiometry, deuterium dilution, densitometry and ^{40}K counting), and others may be termed prediction methods (skinfold thickness, bioelectrical impedance, and BMI). Unfortunately none of the indirect methods have been compared to the direct method of actual fat mass measurement (Martin & Drinkwater, 1991).

3.2.1. REFERENCE METHODS

3.2.1.1. DENSITOMETRY

Fat has a lower density than lean tissue, therefore a subject who has a larger amount of adipose tissue will have a lower overall body density. The most common method of assessing body density is by hydrostatic (underwater) weighing. Although this procedure is considered by many as the 'gold standard' method (Brodie, 1988), it does make the assumption that both fat and fat-free density are constant (Martin & Drinkwater, 1991). Fat-free density has been shown to range from 1.0795-1.1110 $kg.l^{-1}$ (Fuller et al., 1992).

3.2.1.2. DUAL-ENERGY X-RAY ABSORPTIOMETRY (DEXA)

Dual-energy X-ray absorptiometry (DEXA) is a relatively new technique and despite its relative high cost has the advantage that it can differentiate between bone mineral, fat and fat-free soft tissues. DEXA has been shown to be an accurate reproducible method of measuring body composition and is highly correlated to measures of fat free mass and % fat from densitometry (Fuller et al., 1992; Hansen et al., 1993).

3.2.1.3. DEUTERIUM DILUTION

About 60 % of the male human body is water, the majority (62.5 %) of which is located in the intracellular compartments (Brodie, 1988). Dilution of a tracer element like deuterium oxide in the body water gives a measure of the fat-free mass. A major limitation of this technique is that it assumes that fat-free mass has a constant fraction of water (Martin & Drinkwater, 1991). The hydration fraction of the fat-free mass has been shown to range from 0.6941-0.7837 (Fuller et al., 1992).

3.2.1.4. TOTAL BODY POTASSIUM

Total body potassium can be used as a measure of lean tissue mass as 90 % of the potassium is found in non-fat cells. Measurement of total body potassium is dependant on the naturally occurring isotope ^{40}K which is a constant fraction of the body

potassium (0.012 %)(Brodie, 1988). As ^{40}K emits a small amount of radiation it can be measured with a sensitive whole body radiation counter. A limitation of this method is that it assumes a constant potassium content of lean tissue (Martin & Drinkwater, 1991). Also accuracy of the measurement can be dependant on the accuracy of the detector, with some having a coefficient of variation greater than 5% (Fuller et al., 1992).

3.2.2. PREDICTION METHODS

3.2.2.1. BODY MASS INDEX (BMI)

Weight for height is one of the simplest way of describing body composition. BMI or weight(kg)/height(m)^2 is commonly measured and used as an indication of obesity, although several indices of weight for height have been evaluated (weight/height, weight/height^2, weight$^{0.33}$/height, height/weight$^{0.33}$) against densitometry, giving correlation coefficients ranging from 0.57 to 0.82, with an average r= 0.76 for the most common measure, BMI or weight(kg)/height(m)^2(Brodie, 1988).

3.2.2.2. BIOELECTRICAL IMPEDANCE

The assessment of body composition by Bioelectrical impedance has gained popularity as an easy reliable field test. The high water and electrolyte content of the non-fat mass give it a much higher electrical conductivity than the fat mass and thus a measure of electrical impedance will give a measure of the fat-free mass. A recent study has shown that bioelectrical impedance measurement was unaffected by a 30-minute exercise session (Liang & Norris, 1993).

3.2.2.3. SKINFOLD MEASUREMENT

As densitometry is considered the 'gold standard' reference method, skinfold measurements are generally considered as the best of the prediction methods (Fuller et al., 1992).

As subcutaneous adipose tissue represents a large fraction of total body fat that it is easily assessable, measurement of this adipose tissue by skinfold thickness at different body sites is a simple method of assessing total body fat (Clarys et al., 1987).
Early research validating this method demonstrated that there was a need for different equations to predict body density depending on age and sex (Durnin & Womersley, 1974). Now there is well in excess of 100 different equations to predict body fat from skinfold measurement. The number of studies using skinfolds and the number of different formulae reflect the extent of sample specificity (Martin et al., 1985). Generalised prediction equations have been produced to reduce the number of different regression equations required, but it is important that any prediction equation takes account of age (Durnin & Womersley, 1974) and the fact that there is a curvilinear relationship between skinfold and percentage fat (Durnin & Womersley, 1974; Jackson & Pollock, 1978).
Recently it has become popular to combine different measurements to give three- and four-compartment models. Combined measurements of densitometry and total body water can in theory give a three-compartment model of fat, water and fat-free dry matter. This model assumes a constant ratio of protein to mineral in the fat-free dry mass but with the addition of Dual-energy X-ray absorptiometry (DEXA) in theory a four-compartment model can be constructed of fat, protein, mineral and water. The advantage of the four-compartment model is that it removes the need for some of the assumptions inherent in the two- and three-compartment models regarding the proportions and average densities of different components of the fat-free mass. One concern of using a four-compartment model is that the errors in the individual measurement techniques add together to give a larger overall error. Fuller et al. (1992) compared a four-compartment model with a three-compartment model and individual measures of body composition by DEXA, deuterium dilution, densitometry, ^{40}K counting, and four prediction methods (skinfold thickness, bioelectric impedance, near-i.r. interactance and BMI), finding that the error with the three- and four-compartment models was as low as or lower than the measurement errors associated with determination of body fat using either densitometry or deuterium dilution in isolation.

Jebb et al. (1993) compared the accuracy of a variety of in-vivo body composition techniques (densitometry, total body water, skinfold thickness, whole body impedance and resistance) with fat balance and concluded that a three-compartment model (Murgatroyd & Coward, 1989) yielded the least bias and greatest precision. It was estimated that the smallest change in fat mass that can be measured with this method in a single subject is 1.54 Kg. Of the prediction techniques, skinfold thickness measurements would appear to be the most accurate, but is associated with a systematic bias such that they underestimate any changes in body composition (Jebb et al., 1993).

It would be true to say that cadaver dissection with ether extraction is the only accurate method of measuring body fat, all other methods use some approximation or assumption that can be criticised. Martin & Drinkwater (1991) have divided the different measurement techniques into three validation levels. Level one is the direct measurement, level two is the indirect method that is based on certain quantitative assumptions and level three is doubly indirect in that the methods are calibrated against a level two method. Table 6 is a modified version of a table from Martin & Drinkwater (1991). Cadaver dissection with no ether extraction has been included as a level II method as there are quantitative assumptions about the amount of essential fat that cannot be dissected out.

Validation Level	Technique
Level I - direct	Cadaver dissection, ether extraction
Level II - indirect, based on quantitative assumptions	Cadaver dissection only ^{40}K counting Densitometry Total body water
Level III - doubly indirect, calibrated against a level II method	Skinfold measurement Bioelectrical impedance

Table 6. Validation levels for different techniques for % fat estimation (adapted from Martin & Drinkwater, 1991)

It would seem that the accuracy of in-vivo measurement of fat mass is limited by various assumptions, however several measurement methods have been shown to be reproducible and have the ability to detect reasonably small changes in fat mass. It should also be remembered, in terms of absolute values, that the amount of fat is a continuous variable and in terms of health there are no clear dividing lines of how much fat is healthy or unhealthy.

3.3. Body Composition and CHD

Obesity is associated with an increased risk of hypertension, heart disease, diabetes, kidney disease, gall bladder disease and joint disease (Black, 1983; Dawber, 1980). The Allied Dunbar NFS has recently shown that an increasing proportion of the English population are over-weight and obese (ADNFS, 1992).

Obesity and weight gain are not considered to be major risk factors for CHD. They clearly influence the risk of CHD events by their rising effect on blood pressure, total and LDL cholesterol, and their lowering effect on HDL cholesterol levels (Leon et al., 1987).

The relationship with CHD also relates to the propensity of obese individuals to metabolic complications such as glucose intolerance, hyperinsulinaemia and hyperlipidaemia (Kissebah et al., 1982).

For women these metabolic complications seem to be related to the body fat distribution. Kissebah et al. (1982) demonstrated that upper-body obese women have significantly higher glucose intolerance and insulin and triglyceride levels compared to lower-body obese women. Measurement of fat cell size from the abdomen and thigh showed that the upper-body obese women had significantly larger abdomen fat cell volumes compared to the lower-body obese women, whereas thigh fat cell volume was not significantly different between the two obese groups and was comparable to a sample of non-obese control women. They concluded that the metabolic complications were as a result of the abdominal fat cell hypertrophy. Hartz et al. (1984) have also reported that waist to hip ratio was significantly associated with diabetes, hypertension and gallbladder disease in women. Post-menopausal women who are not on hormone replacement therapy have been shown to have more total body fat and greater centrally located body fat compared with pre-menopausal women (Greaves et al., 1992), which would seem to put them at higher risk of metabolic complications and disease.

Men tend to exclusively accumulate excess fat in the abdominal region, and therefore for men there is a strong association between overall obesity and hyperlipidemia, hyperinsulinaemia and glucose intolerance (Pouliot et al., 1994).

A recent study has shown that waist circumference and abdominal sagittal diameter are better anthopometric indices of abdominal visceral adipose tissue accumulation, and related cardiovascular risk, in men and women than the commonly used waist to hip ratio (WHR) (Pouliot et al., 1994). In both men and women the waist circumference was more strongly correlated to computed tomography measurements of abdominal visceral and subcutaneous adipose tissue area. Increasing values of waist circumference and abdominal sagittal diameter were more consistently associated with increases in fasting and post glucose insulin levels than increasing values of WHR, especially in women. The authors concluded that the waist circumference or abdominal sagittal diameter should be

used as indices of abdominal visceral adipose tissue deposition in the assessment of cardiovascular risk. Waist circumferences above approximately 100 cm or abdominal sagittal diameter values >25 cm are most likely to be associated with potentially 'atherogenic' metabolic disturbances.

Obese individuals tend to have lower levels of physical activity (Dannenberg et al., 1989; Gardner & Poehlman, 1993; Slattery & Jacobs, 1987). The debate is whether it is the lower levels of physical activity that has caused the obesity, or is it because of obesity that the level of physical activity is lower? It is known however, that the combination of obesity and low levels of physical activity is associated with increased risk of CHD (Blair et al., 1989; Dannenberg et al., 1989; Paffenbarger et al., 1993)

The association of obesity with CHD and all cause mortality is complicated by the fact that most of the large epidemiological studies have only used BMI as a measure of body composition.

Higher BMI and larger gains in BMI since college added to the relative risk of a first coronary heart disease attack for 16,936 Harvard alumni in 1962-1972 (Paffenbarger et al., 1984). In a further follow up of the Harvard alumni 1977- 1985 there was a reversed J-shaped curve for mortality risk seen for the high and low extremes of BMI as compared with intermediate values (Paffenbarger et al., 1993). Intermediate BMI values (24-25) were associated with the lowest mortality risk and the high BMI values (>26) were associated with the highest mortality risk.

Of the studies that have used more detailed measurements of body composition it seems that only those with the longer observation periods have been able to show the association of obesity and CHD (Hubert et al., 1983). Lapidus et al.(1984) hypothesise that the reason for this is that it is only a sub-group of the obese population that is associated with an increased risk, and therefore longer observation periods are needed for this sub-group to influence the risk for all obese subjects. This may point to the sub - opulation of upper-body obesity that has clearly been shown to be at more risk of CHD, stroke, metabolic complications and all-cause mortality (Larsson et al., 1984) especially for women (Lapidus et al., 1984, Kissebah et al., 1982)

3.4. Effect of exercise on Body Composition

Physical fitness and high levels of physical activity are clearly associated with more desirable body composition (Ballor & Keesey, 1991; Blair et al., 1989; Dannenberg et al., 1989; Darga et al., 1989; Jette et al., 1992). Exercise affects three important factors of a weight loss regime : appetite, energy expenditure, and body composition (Elliot & Goldberg, 1985). Moderate treadmill walking has been shown to increase energy expenditure without increasing energy consumption, producing a negative energy balance (Woo et al., 1982).

Some of the studies that have used exercise alone or exercise plus diet as a modality for treating obesity have yielded disappointing results.

Wilmore (1983) in his review of 55 studies on the effects of aerobic exercise on body composition calculated that on average there was only a 1.6% reduction in body fat. Despite training programmes that lasted from 6 to 104 weeks the changes in body composition were surprisingly small. He concluded that future studies needed to have tighter control of energy intake and expenditure to clarify some of the discrepancies in this area.

One explanation for the mixed results could be the fact that all obese individuals tend to be treated as a homogenous group. This is clearly not the case with distinct sub-groups of obesity that physically and metabolically differ (Greaves et al., 1992; Hartz et al., 1984; Katch & McArdle, 1988; Kissebah et al., 1982; Pedersen et al., 1993; Slattery et al., 1992; Zamboni et al., 1993). Bray (1988) in his review developed the theory that there was an important difference in the response to exercise between individuals with hypercellular obesity and those with normocellular obesity, in that aerobic exercise had little or no effect on the body composition of individuals with hyperplastic obesity. It is also possible that the effect of exercise is dependant on body fat distribution. Kanaley et al. (1993) demonstrated that a 16-week programme of exercise and diet was equally effective at reducing weight in a group of upper-body and lower-body obese women.

The upper-body obese women did however have a significantly greater reduction in waist circumference, triglycerides and cholesterol levels. This seems to be the only study that has addressed the possible differential effect of diet and exercise on weight loss in upper and lower-obese individuals. Therefore future studies should consider the possible differential effects of exercise and diet on the different subgroups within obesity.

One of the main problems of dietary programmes to lose weight is that the negative energy balance usually results in a corresponding reduction in basal metabolic rate (Bray, 1990; Hill et al., 1987; Mole, 1990) which in some cases can eventually negate the dietary calorie reduction. For most individuals BMR represents the largest component of daily energy expenditure, therefore reductions in calorie intake that result in reductions in BMR, will not give a proportional reduction in weight. It is thought that this reduction in BMR is initially a result of reduced metabolic intensity followed by a reduction in lean body mass (Mole, 1990). Exercise can help to maintain (Hill et al., 1987) or even increase lean body mass in a period of negative energy balance (Ballor & Keesey, 1991; Broeder et al., 1992). Some exercise-plus-diet studies have shown a lesser reduction in Resting Metabolic Rate (RMR) (Van Dale et al., 1987) or no reduction at all (Bingham et al., 1989), with subjects in negative energy balance. Hill et al. (1987) found a similar (~20%) reduction in RMR in a group of obese women who only dieted or exercised-plus-diet. Both groups had the same diet, thus the exercise group actually had a much greater negative energy balance due to the added energy cost of the exercise. Therefore the larger negative energy balance of the exercise group did not result in a proportionally larger reduction in the RMR. A study by Van Dale et al. (1987) found a significant reduction in RMR in both diet only and diet-plus-exercise groups, although the decrease in the RMR or the exercise group was 10 percent less than for the diet group.

In contrast Lennon et al. (1985) found a significant increase in RMR (~10%) after 12 weeks of diet plus exercise. Also Tremblay et al. (1986) have reported that after an eleven week medium intensity aerobic exercise only programme, there was an increased RMR (~8%)in a group of obese women.

A group of obese women who were given a 16-week exercise and diet programme to give a daily energy deficit of 2.1 MJ had a significant reduction in fat mass with no change in BMR (Kanaley et al., 1993). Poehlman et al. (1989) and Tremblay et al. (1986) have shown that there was a relationship between aerobic fitness and RMR, particularly with highly trained individuals. When adjusted for differences in body weight and fat-free mass, high levels of aerobic fitness were associated with an elevated RMR (~10%)(Poehlman et al., 1989) . Hence it appears that the highly trained male has a high RMR for his metabolic size. The mechanism for this effect has yet to be established.

It would therefore seem that the inclusion of exercise in a weight loss programme has the advantage of, at worst, reducing the fall in RMR associated with dieting.

A study comparing the effect of diet or diet plus exercise on a group of obese women found no difference in the total loss of body mass (~8kg) between the exercising and non-exercising groups but significantly more of the weight loss came from fat and less from fat-free mass in the exercising subjects (Hill et al., 1987). Exercising subjects lost 74% of their weight from fat and only 26% from fat-free mass, whereas corresponding losses for non-exercisers were 57% from fat and 43% from fat-free mass. Hill et al. (1987) calculated that since fat is more calorically dense than fat-free mass the exercising subjects had a much greater loss of body energy than non-exercising subjects. The exercising subjects lost an average of 10,296 kcal more than non exercisers which almost exactly matches the energy cost of the exercise sessions. They concluded that exercise appears to contribute to body weight reduction in two ways; 1) the energy expended in exercise contributes directly to the negative energy balance and 2) exercise produces a more favourable composition of weight loss during food restriction.

A group of 14 sedentary middle aged men who engaged on a two-year running programme showed a significant drop in body fat even though their calorie intake increased. The reduction in weight was slightly less than the loss of body fat indicating a gain in lean body mass (Wood et al., 1985).

After 16 weeks of vigorous walking for 90 minutes, 5 days.week^{-1} at an intensity to expend about 1100 kcal per session, six sedentary obese men had gained 0.2 kg lean

tissue and lost 5.7 kg body fat (Leon et al., 1979). Monitored food intake initially increased, then progressively decreased below pretraining levels. Ohta et al. (1990) demonstrated that a walking programme (10,000 steps.day^{-1}) and diet (1500 kcal.day^{-1}) significantly reduced weight and skinfold thickness in a group of obese, middle-aged subjects.

There are many exercise only studies on non-obese subjects that have failed to show any change in body composition (Duncan et al., 1991, Hamdorf et al., 1992, Hardman et al., 1992, Rowland et al., 1991, Santiago et al., 1987) although there are some that have produced favourable improvements in body composition (Pollock et al., 1971, Whitehurst & Menendez, 1991).

Exercise studies on obese individuals, in particular if accompanied by a diet have been more effective at demonstrating favourable changes in body composition. Ballor & Keesey (1991) in a recent meta-analysis of 53 studies of exercise training reported that 50% of the variance with respect to exercise induced changes in body mass, fat mass and % body fat can be accounted for by pre-intervention body mass and exercise energy expenditure. It is interesting to note that this meta analysis also found that exercise was more effective at reducing body mass for males than females.

The length of exercise session could be an important factor as Milesis et al. (1976) showed that exercising for 45 minutes resulted in significantly greater reductions in body fat than exercising for 15 minutes per session. The ACSM guidelines recommend that exercise programmes that are conducted at least 3d.wk^{-1}, of at least 20 minutes duration, and of sufficient intensity to expend approximately 300 kcal per session are above a threshold for total body and fat mass loss (ACSM 1990). It is generally accepted that aerobic exercise is the most effective mode of exercise for changes in body composition. Broeder et al. (1992) comparing a 12-week resistance training programme (RT) with a 12 week endurance training programme (ET) found that both groups showed significant declines in relative body fat either by reducing total fat and maintaining fat-free weight (ET) or by reducing their total fat weight and increasing fat-free weight (RT). A group of already active older women (> 60 years) who undertook a 24-week weight training

programme also had reductions in percentage body fat and increases in lean-body mass (Omizo et al. 1993).

One of the important factors for weight loss and maintenance of normal body composition is adherence to an exercise regime and programmes should strive to initiate lifestyle changes that include an adequate amount of physical activity that is achievable and sustainable.

4. BLOOD LIPIDS

4.1. CLASSIFICATION AND MEASUREMENT OF PLASMA LIPIDS

Lipoproteins are the transport molecules for cholesterol and triglycerides in the blood stream. They are large globular particles that contain an oily core, non-polar lipid (cholesterol esters or triglycerides) surrounded by a polar coat of free un-esterified cholesterol and apolipoproteins (Brown & Goldstein, 1986; Pronk, 1993). Lipoproteins can be classified on the basis of their gravitational density by ultra-centrifugation. There are six main classes of lipoproteins as can be seen in Table 7.

	Classes of Lipoproteins
1	Chylomicrons
2	Chylomicron remnants
3	Very Low Density Lipoproteins (VLDL)
4	Intermediate Density Lipoproteins (IDL)
5	Low Density Lipoproteins (LDL)
6	High Density Lipoproteins (HDL)

Table 7. Classes of Lipoproteins, from Pronk, (1993)

These lipoproteins can be further classified by the presence of protein constituents called apolipoproteins. Thus far 16 have been characterised, but the biological function of many are still unknown and under investigation (Haskell, 1983). The main apolipoproteins that have been identified and extensively measured are A-I, A-II, (a) and B.

Dietary fats absorbed in the gut are initially transported as chylomicrons and chylomicron remnants. After a meal fat is removed in large quantities from the circulation in the liver by the action of the enzyme Lipoprotein Lipase (LPL). These predominantly triglyceride-containing-particles have a half-life of about one hour (Guyton, 1986).

Very Low Density Lipoprotein (VLDL) is secreted by the liver to transfer triglycerides to muscle and adipose tissue. In the capillaries triglycerides are removed from VLDL by the enzyme LPL. With this reduction in size VLDL returns to the circulation as Intermediate Density Lipoprotein (IDL). The IDL may return to the liver or undergo further hydrolysis to LDL.

The structure of IDL is such that it contains both apolipoprotein (E) and a single copy of apolipoprotein B-100, which allows it to bind to LDL receptors. When converted to LDL the apo (E) leaves the particle and therefore LDL is characterised by the presence of a single copy of apo B-100 (Pronk, 1993).

High Density Lipoproteins are thought to act as cholesterol acceptors in the reverse cholesterol transport system (Campaigne et al., 1993; Davidson, 1992). HDL is thought to acquire unesterified cholesterol from the cell surfaces and other lipoproteins. This is then esterified by the enzyme Lecithin-Cholesterol-Actyltransferase (LCAT) and is subsequently internalised. Apo A-I, one of the major apolipoproteins present in HDL, is known to activate LCAT (Pronk, 1993). As internalisation increases there is an increasing level of HDL_3. Subsequently a large less dense particle HDL_2 evolves. HDL may deposit the cholesterol esters directly into the liver or transfer them to other Apo B containing lipoproteins, LDL and IDL. The hepatic tissues extract and hydrolyse these cholesterol esters forming free cholesterol. The free cholesterol is excreted directly into the bile as bile salts.

Recently a further blood lipid particle Lipoprotein(a)[Lp(a)] has been associated with CHD (Lawn, 1992: Wallace & Anderson, 1987). Lp(a) is very similar in structure to LDL, except that as well as having the apo (E) molecule it also has an additional protein, apolipoprotein (a). The curious feature of Lp(a) is that the blood levels seem to be genetically determined and dietary or drug treatments have little or no effect on levels (Lawn, 1992). Lp(a) has been found to be present in the arterial wall and atherosclerotic plaques (Rath et al. 1989).

4.2. DEFINITION OF HYPERCHOLESTEROLAEMIA

Like many other biological variables the distribution of blood cholesterol values is essentially 'gaussian' , with no clear division of high, normal or low levels. It is common to consider abnormal levels of biological substances as a level which is found in the upper 5% of the population. Although for cholesterol it is considered that a much larger proportion of the population have cholesterol levels that are too high, especially considering the level of CHD. The 1985 Cholesterol Consensus Conference (JAMA, 1985) defined two levels of hypercholesterolaemia for the US population. Their definition of severe hypercholesterolaemia and moderate hypercholesterolaemia can be seen in Table 8

AGE (years)	Moderate Hypercholesterolaemia $mg.dl^{-1}$ ($mmol.l^{-1}$)	Severe Hypercholesterolaemia $mg.dl^{-1}$ ($mmol.l^{-1}$)
20-29	> 200 (5.17)	> 220 (5.69)
30-30	< 220 (5.69)	> 240 (6.21)
$\geq$ 40	> 240 (6.21)	> 260 (6.72)

Table 8. Definition of Hypercholesterolaemia from the 1985 Cholesterol Consensus Conference. (JAMA, 1985)

Their definition of severe hypercholesterolaemia was approximately the 90th percentile of the population cholesterol level. This will include individuals who have hereditary forms of high cholesterol. Moderate hypercholesterolaemia was defined as the values between 75-90th percentile. This would include large numbers of people whose elevated cholesterol level is due in part to their diet. The panel also advised measurement of HDL, as high total cholesterol levels may be due to high concentrations of HDL which are negatively correlated to CHD (JAMA, 1985).

The cholesterol measurements from the Framingham Study (Anderson et al., 1987; Dawber, 1980) revealed a fairly 'normal' distribution curve with only a slight skewing to the higher values. One important finding from their data was that over a 30-year span of adulthood, age did not affect cholesterol level in men. However for women there was a significant rise in cholesterol with age. The shape of the distribution curve for women was unchanged but was shifted to higher values (Dawber 1980). It was also interesting in the comparison of men and women, where in the middle decade (40-49 years) the two distribution curves were almost identical. In the early decade (30-39 years) the women had lower levels compared to the men, but at the later decade (50-59 years) the women were now higher than the men. However, the review of Wallace & Anderson (1987) concluded that longitudinally, cholesterol levels increase in middle-aged men in most Western countries. In agreement the cross-sectional study by Sharlin et al. (1992) reported that men and women with high plasma cholesterol levels were older than those with low plasma cholesterol levels.

Pre-menopausal women have been shown to have lower TC, LDL and higher HDL levels than men of the same age (Clifton & Nestel, 1992; Kim & Kalkoff, 1979). Additionally, unlike men, there are large fluctuations in lipid values in women, that correspond to the phases of the menstrual cycle (Jones et al., 1988; Kim & Kalkoff, 1979). During the luteal phase there is a significant suppression of TC and LDL levels compared with follicular phase. Reductions in triglyceride and increases in HDL during the luteal phase are generally much smaller in magnitude compared with the changes in TC and LDL, and are not consistently significant. These changes in the lipid profile are thought to be a result of the surge of estrogen just prior to ovulation. Because of the duration of the half-life of the lipids involved, the measurable effect is delayed until the luteal phase. This also explains the abrupt change in lipid profile at menopause, to a profile similar to men of the same age. At menopause the reduction in estrogen levels leads to increased TC, LDL and reduced HDL levels (Goldberg & Elliot, 1985; Kim & Kalkhoff, 1979).

Based on the findings of the many epidemiological studies both the 1985 Cholesterol Consensus Conference (JAMA, 1985) and the European Atherosclerosis Society (EAS,

1987) concluded that a total serum cholesterol value below 200 $mg.dl^{-1}$ (5.17 $mmol.l^{-1}$) was desirable. Individuals with levels above this value should attempt to reduce this by diet or drug intervention. Grundy (1986) in his review calculated that if the American cholesterol levels were reduced to the range 130 to 190 $mg.dl^{-1}$ (3.36 to 4.91 $mmol.l^{-1}$) this would result in a 30-50 % reduction in the rate of CHD.

The level of total cholesterol (TC), and in particular LDL in the blood can increase because of a high fat diet (Clifton & Nestel, 1992) or because of defective LDL receptors, resulting in a reduced receptor mediated clearance of LDL (Brown & Goldstein, 1986). The increased plasma levels of LDL leads to an increased infiltration of LDL into the arterial intima. Recent research has suggested that this LDL is then more susceptible to oxidation, altering its structure. This oxidised LDL is thought to be the start to a chain of events leading to fully fledged atherosclerosis (Davidson, 1993).

When measuring plasma lipids levels there is a need for careful controls as there are various short and longer term variations of an individuals lipid levels which include circadian variation, time from last exercise bout, absence of fasting, menstrual cycle phase, dietary composition, alcohol consumption, cigarette smoking, body composition, menopause status and seasonal variations (Clifton & Nestel, 1992; MacRury et al., 1992; Pronk, 1993; Wood & Stefanick, 1990). It should be remembered that the blood level of any substance is a 'still picture' of a complex and dynamic metabolic process (Wallace & Anderson, 1987).

There are clear seasonal variations in total cholesterol levels with the lowest levels in the summer months, rising again in the winter months (MacRury et al., 1992). This variation in TC is though to be primarily due to alterations in diet in the summer months, where more fresh fruit and salads are eaten and this is reflected in the changes in vitamin C consumption. The vitamin C consumption has been shown to exhibit an inverse relationship to the seasonal variation in TC (MacRury et al., 1992).

Goff et al. (1992) found that body fatness modifies the relationship between dietary cholesterol and risk of coronary death. Obese individuals do not benefit as much as leaner individuals by a lower cholesterol diet and leanness does not protect men on a

very high cholesterol diet. Dietary studies where there has been a favourable change in body composition often show improvements in TC and triglyceride levels (Barrett-Connor, 1985; Wood et al., 1991). Although it is not clear whether it is the change in body composition that has affected the improvement in lipid profile or is it solely due to an improved diet.

Distribution of body fat could be an important factor as Kanaley et al. (1993) found that there were different responses for upper-body obese and lower-body obese women to a programme of diet plus exercise. After the 16-week programme the upper-body obese women showed significant reductions in TC, triglycerides and increases in HDL, whereas the lower-body obese women had only reductions in triglyceride levels.

Several cross-sectional studies have shown poorer lipid profiles with increasing age, but this is paralleled by a decrease in fitness level and an increase in obesity, which confuses the relationship (Wood & Stefanick, 1990). Individuals who maintain a high level of fitness (e.g. runners) are able to maintain youthful lipid levels into middle age and beyond (Herbert et al., 1984; Williams et al., 1986).

4.3. Lipid Profile and CHD

Elevation of blood cholesterol level is a major cause of coronary heart disease (CHD)(AHA, 1990; Anderson et al., 1987; Castelli et al., 1986; Dawber, 1980; EAS, 1987; Grundy, 1986; JAMA, 1985; Kannel et al., 1971; LRC-CPPT, 1984; MRFIT, 1982; Stamler et al., 1986; Wallace & Anderson, 1987). There is clear casual evidence supporting the relationship between elevated blood cholesterol levels and CHD from a wealth of experimental pathological, cross-sectional and intervention studies. Wallace & Anderson, (1987) in their review concluded that the relationship between elevated levels of blood cholesterol and CHD was one of the most consistent and well established areas of epidemiological inquiry. It is interesting to note that no population has been reported to have a high incidence of CHD and low cholesterol levels (Wood & Stefanick, 1990).

There is some debate however as to whether there is a threshold for an increase in the incidence of CHD, or whether it is continuous and graded, with increasing cholesterol levels (Grundy, 1986). The Framingham Heart Study reported that up to a serum cholesterol level of 200 $mg.dl^{-1}$ (5.17 $mmol.l^{-1}$) there was little increase in CHD risk (Kannel et al., 1971), indicating that there could be a threshold at this point . However, the MRFIT found a continuous graded increase in CHD risk with rising cholesterol levels from as low as 150 $mg.dl^{-1}$ (3.9 $mmol.l^{-1}$)(Stamler et al., 1986). Nevertheless, both studies clearly show that beyond a cholesterol level of about 200 $mg.dl^{-1}$ there is a dramatic increase in the CHD risk.

Of course it should be noted that hypercholesterolaemia is not the only cause of CHD. Smoking, for example, is considered an independent risk factor for CHD, and in combination with elevated cholesterol levels greatly increases the CHD mortality rate, compared to a non-smoking (Stamler et al., 1986).

Ross (1986) described the pathogenesis of atherosclerosis as a multifactoral cascade of events. The fact that the major constituent of the atheromatous lesion is cholesterol suggests that the blood borne lipid is somehow deposited in the arterial wall. The exact mechanism by which cholesterol is deposited in the arterial wall is unknown. It is clear that, elevated levels of LDL lead to a greater infiltration of LDL into the intimal wall (Davidson, 1993; Davies & Woolf, 1990). Recent research has suggested that this LDL is then more susceptible to oxidation than circulating LDL (Davidson, 1993), and that the oxidants present in cigarette smoke are very effective at oxidising this LDL (Frei et al., 1991). Oxidation modifies the structure of LDL, in particular the apolipoprotein B-100. The oxidised LDL is no longer able to bind to a LDL receptor and instead is engulfed by macrophages (Davidson, 1993), which are transformed into foam cells. Foam cells have greatly reduced migration abilities and accumulate in the arterial wall, thus initiating plaque formation (Davies & Woolf, 1990; Pataki et al., 1992). The formation of a mature plaque in the arterial wall impedes flow and causes ischaemia in the tissue distal to the blockage. After which thrombus formation, possibly associated

with increased platlet adhesiveness and fibrin deposition, frequently leads to complete occlusion, say, of a coronary artery, and myocardial infarction occurs.

Many animal models have been able to demonstrate that diets that result in elevated blood cholesterol levels lead to the development of atherosclerosis and CHD (Kramsch et al., 1981; Wood & Stefanick, 1990)

HDL, which carries approximately 25 % of the total lipids, has received considerable attention because of its potentially protective effect against the development of CHD. Results from the Framingham study have confirmed that the level of HDL is an independent risk factor for CHD, and there is a strong negative correlation for plasma HDL concentration and the incidence of CHD (Castelli et al., 1986). Even after adjusting for cigarette smoking, relative weight, alcohol consumption, casual blood glucose, total cholesterol and blood pressure, study participants at the 80th percentile of HDL were found to have half of the risk of developing CHD compared to the subjects at the 20th percentile. Also in a recent study of 115 men, the presence of CHD determined by coronary angiography, was strongly correlated to HDL level and in particular to HDL_2. The men diagnosed with CHD also had significantly lower HDL_3 and higher triglyceride levels (Drexel et al., 1992).

Studies that have looked at the relationship between blood triglyceride level and CHD have revealed conflicting findings. Possible reasons for the differences are variations in study design, analytic methods, high intra-individual variation in triglyceride levels and fasting state (Wood & Stefanick, 1990).

One of the important conclusions of the 1985 Cholesterol Consensus Conference was that reduction in the blood cholesterol levels will reduce the incidence of CHD (JAMA, 1985). Both dietary and drug manipulations have been effective at lowering blood cholesterol levels, especially in individuals with elevated levels (LRC-CPPT, 1984; MRFIT, 1982).

There have been recent reports linking the concentration of the lipoprotein Lp(a) to the presence and risk of CHD (Lawn, 1992) and elevated levels are clearly an independent risk factor for CHD (Cambillau et al., 1992; Hoefler et al., 1988; Lawn, 1992; Rath et

al., 1989; Rhoads et al., 1986). Diet and drug interventions seem to have no effect on Lp(a) levels. It would therefore seem that elevated levels are responsible for the occurrence of CHD in individuals with no other apparent risk factors (Lawn, 1992).
The majority of studies have been carried out on men, probably because of the larger numbers affected and because women tend to develop CHD at a later age, usually after menopause. There is a need for more study on women, as the epidemiological data on death from CHD show a similar pattern to men, and CHD is the number one cause of premature death of women in America (AHA, 1990), and after cancer is the leading cause of premature death of women in the UK (BHF, 1992).

4.4. THE EFFECT OF EXERCISE ON LIPID PROFILE

The main thrust of treatments for hypercholesterolaemia have been dietary and drug treatment. This should probably be the case for those with extremely high cholesterol levels, as recommended by the 1985 Cholesterol Consensus Conference (JAMA, 1985) and the European Atherosclerosis Society (EAS, 1987), but for the general population, and those with only slightly elevated cholesterol levels, exercise should be considered as an effective additional treatment, especially considering its favourable effect on other CHD risk factors (Froelicher, 1990).
A meta-analysis of 66 aerobic training studies, involving the measurement of blood lipids, concluded that the average exercising subject was found to have a significant reduction in total cholesterol, total triglyceride, low density lipoproteins and increased high density lipoprotein levels (Tran et al., 1983).
However individual studies that have investigated the effect of regular aerobic exercise on the lipid profile have produced mixed results, and several have found no significant changes for some of the lipids (Leon et al., 1979). One of the reasons for this can be termed the 'initial effect'. Many investigators have found a significant relationship between the initial lipid concentration and the magnitude of the change (Leon et al.,

1979; Lockey & Tran, 1989; Tran et al., 1983). For example, the larger the initial value of total cholesterol the larger the reduction as a result of an exercise programme (Hardman et al., 1989; Kanaley et al., 1993). If the majority of the subjects have a baseline level close to normal it is therefore unlikely that there will be any measurable changes. Pre-menopausal women have lower cholesterol and triglyceride and higher HDL levels than men of the same age and this may be one of the reasons why they have generally poorer changes in lipids as a result of exercise (Dawber, 1980). Lockey & Tran (1989) in their review concluded that the initial lipid concentration may be more important than sex in determining the response to exercise training.

Another very important factor when assessing the impact of exercise on lipoprotein concentrations is the change in plasma volume. Generally with exercise there is an increase in plasma volume (Allen et al., 1992; Sullivan et al., 1993; Thompson et al., 1988), therefore any increase in the concentration of HDL with exercise will represent an even greater increase in total circulation HDL. Conversely a small decrease in the concentration of LDL may not represent a reduction in total circulating LDL at all (Wood & Stefanick, 1990). Many of the studies that have not found increases in HDL levels (Brownell et al., 1982; Stein et al., 1990; Stensel et al., 1993) have not considered possible changes in PV. However at present it is not known whether it is plasma concentrations of lipoproteins or total circulating amount that represents the most important variable in relation to CHD. However it would seem prudent that all future studies measure and account for changes in plasma volume.

In his review Haskell (1986) concluded that there was little evidence that physical activity had a significant independent effect on the concentration of total cholesterol circulating in the plasma or serum. Cross-sectional data have shown that regular exercisers tend to have lower TC levels. One study compared TC levels of 1269 runners (members of the American Medical Joggers Association, who had been running at least 10 mlies per week) and 683 non-runners (members of the American Medical Association). This well matched study showed that significantly more of the runners (49% Vs 33%, $p<0.001$) had TC levels below 200mg.dl^{-1}(5.17 mM^{-1})(Darga et al.,

1989). The failure of exercise to influence TC concentration could be obscured by alterations in the concentration of the various lipoproteins making up the total cholesterol. For example an increase in HDL may be nearly matched by a reduction in LDL concentration, with TC remaining constant. Haskell (1986) did conclude that more active persons have lower plasma triglycerides and LDL concentrations and higher concentrations of the subfraction HDL_2 and apoprotein A-1.

The level of LDL is reduced with aerobic exercise, especially in individuals who regularly do very strenuous exercise. Simple increases in the level of activity seem to have little or no effect on LDL levels. Although there is some evidence that the sub-fraction LDL small is lowered in concentration in active groups compared to sedentary groups (Wood & Stefanick, 1990).

Endurance athletes exhibit lower triglyceride levels, probably a reflection on the increased effectiveness of muscles to utilise fat as a fuel, and also the quantity of fat that will be utilised during an endurance type event. Aerobic exercise has been shown to lower triglyceride level in studies where initial levels were elevated (Wood & Stefanick, 1990). There are several studies where there has been no effect where initial levels were in the normal range (Haskell, 1986). Athletes involved in power/speed training generally have similar triglyceride concentrations to the general population. Women and children tend to have lower levels of triglyceride and thus often do not exhibit any change with aerobic exercise (Haskell, 1986).

Many cross-sectional studies have consistently shown that endurance trained individuals have significantly higher concentrations of HDL (Herbert et al., 1984; Williams et al., 1986). This has mainly been a result of expansion of the HDL_2 sub-fraction (Ballantyne et al., 1982; Wood et al., 1983), the effect of exercise on HDL_3 is more variable (Thompson et al., 1988). The levels of the larger less dense sub-fraction HDL_2 appear to be negatively related to the incidence of atherosclerosis (Drexel et al., 1992; Wood & Stefanick, 1990). HDL_2 is the lesser component of total HDL, therefore change in total HDL is a less sensitive measure of changes in HDL_2. Measurement of HDL_2 may be a better predictor of CHD risk.

In a cross-sectional study Williams et al. (1986) found that 12 long distance runners had higher HDL (principally HDL_2) levels. They also had lower concentrations of smaller, denser low density lipoprotein particles than did comparable sedentary men.

In agreement with this clear cross-sectional evidence for increased in HDL levels in physically active individuals, there are many longitudinal aerobic training studies that have reported increases in HDL levels (Ballantyne et al., 1982; Hardman et al., 1989; Kantor et al., 1987; Seip et al., 1993; Stein et al., 1990; Thompson et al., 1988; Whitehurst & Menendez, 1991; Wood et al., 1983; Wood et al., 1991). Only a few have failed to show any change with aerobic exercise (Faber et al., 1992; Ohta et al., 1990; Stensel et al., 1993). One review has suggested that results from studies of less than 10-weeks duration are less convincing, whereas all studies of > 12 weeks there was an increase in HDL averaging 5 $mg.dl^{-1}$, although this was not significant at $p<0.05$ in all studies(Wood & Stefanick, 1990). Much longer studies suggest that there is a dose response e.g. the more miles run the greater the changes. Although a recent study by Stensel et al. (1993) found that after a 12-month daily walking programme that there no changes in the lipid profile of middle-aged men, despite significant improvements in fitness. One criticism of this study is that it did not consider possible changes in plasma volume (PV). Therefore it is possible that there was a small increase in the amount of circulation HDL in this study. The authors suggested that the level of exercise was below the threshold to have an effect. It should be remembered that when comparing longitudinal and cross- sectional studies that sedentary individuals who undertake a training programme are unlikely to reach the fitness levels of dedicated habitual exercisers in a typical training study lasting three months.

Superko (1991) in his review concluded that a threshold of exercise equalling approximately 15 miles per week jogging was required to induce beneficial changes in lipid profiles. This concept of a threshold agrees with the conclusion of Haskell (1986) who suggested that for favourable changes in lipoproteins an increase in moderate intensity endurance type activity requiring an energy expenditure of approximately $4mJ.week^{-1}$(1,500 kcal). Above this level a dose-response relationship exists up to an

energy expenditure of about 19mJ.week^{-1}(4,500 kcal). It could be concluded that energy expenditure, and not exercise intensity, is the important factor for increasing HDL levels. Cook et al. (1986) reported that the number of miles.day^{-1} walked by postal workers was significantly correlated to HDL_2 levels, suggesting that long duration low intensity physical activity may be enough stimulus to increase HDL levels.

Wood et al. (1983) found that after a one-year running programme, the runners were significantly fitter and leaner than the control subjects, but there was no significant alteration in the lipid profile. However there were significant correlations for the distance run.week^{-1} and plasma HDL (HDL_2) and LDL levels or changes. Further analysis of data revealed that there were significant changes in the lipid profile if it only included those runners who completed an average of 8 miles.week^{-1} or more (52% of the sample). The authors concluded that there was a threshold of about 8 miles.week^{-1} above which a one-year running programme leads to beneficial lipoprotein changes. Unfortunately the authors seem not to have considered the effect of initial value on the change.

Stern et al. (1990) in a 12-week study, compared the changes in lipid profile exercising at intensities of 65, 75 and 85% of maximum heart rate for 30 minutes, three times per week. They found that there were significant increases in HDL at 75 and 85% only, and there were no changes in total cholesterol and triglyceride levels. The authors concluded that a minimum exercise intensity of at least 75% of maximum heart rate is required to increase HDL levels. All three exercise groups did show a significant increase in VO_2 max. , therefore the changes in lipids are concurrent but independent of improvements in aerobic fitness. No group had any change in body composition or diet. One factor not commented on by the authors was the fact that the higher intensity groups were actually performing more overall work and this may be a key factor affecting the change in HDL and other lipoproteins.

Dedicated runners clearly have elevated concentrations of apolipoprotein A-I (Wood & Stefanick, 1990), and the total circulating A-I is further increased by the expansion in PV. Bed rest has been shown to greatly decrease the level of A-I (Nikkila et al., 1980). There are relatively few studies of the effects of aerobic exercise on plasma lipids that

have also measured the changes in apolipoproteins. A 12-week study found that A-I increased by 10% (Keins et al., 1980). Conversely a one-year study by Wood et al. (1983) found no changes in A-I and A-II. Little is known about the relationship of A-II to exercise, and there seem to be no studies that have investigated the changes in Lp(a) with exercise. As a predictor of CHD risk measurement of apolipoprotein levels may offer information not available from blood lipid and lipoprotein levels (Wood & Stefanick, 1990).

In agreement with studies on normal subjects Ballantyne et al. (1982) found that a group of male survivors of myocardial infarction who enrolled on a 6-month training programme experienced significant reductions in LDL, triglycerides and increases in HDL and apolipoprotein A-I. Like normal subjects the increase in HDL was principally as a result of and increased HDL_2 sub-fraction.

The combination of regular exercise with a change in diet has been shown to be very successful in favourably altering the lipid profile (Faber et al., 1992; Kanaley et al., 1993; Leon et al., 1979; Ohta et al., 1990; Wood et al., 1991). Faber et al. (1992) found that after a 6-week hiking expedition where the normal dietary habits were altered to have a very much reduced fat and increased carbohydrate intake, there were large reductions in TC and TC:HDL ratio. The subjects also had significant reductions in weight and body fat. There were no changes in HDL levels, but the exercise seemed to have maintained the HDL levels, which would be expected to fall on a very high carbohydrate diet (Mensink & Katan, 1992). This is consistent with the finding of Ohta et al. (1990) who found that 332 obese middle-aged subjects had significant reduced TC, triglycerides and no change in HDL after four months of walking (10,000 steps.day^{-1}) plus a diet (1,500 kcal.day^{-1}).

The majority of longitudinal studies looking at the effect of exercise on lipid profiles have concentrated on men, and only a small number have looked at the effect on women. It is possible, considering the differing initial levels that there would be a different response. Brownell et al. (1982) compared the effects of a 10-week aerobic exercise programme on the lipid profile of a group of men and women. The men showed significant

reductions in TC, triglyceride, LDL and LDL:HDL ratio, with increases in HDL. However the women only had a significant reduction in TC and triglycerides, despite similar changes in $\dot{V}O_2$ max. to the men. However a re-analysis of the results by the authors adjusting for initial values removed all the significant differences between the men and women except for a greater reduction in the LDL:HDL ratio for the men.

A similar finding was reported by Wood et al. (1991) who found that alterations in lipid profile as a result of diet alone were different between men and women. The men had a decrease in TC:HDL ratio, whereas the women had a decrease in HDL_2, LDL and TC. With the addition of an exercise programme to the diet the men had reductions in triglyceride and increases in HDL and HDL_2, the women had significant increases in HDL and HDL_2 compared to the diet only group and a reduction in triglyceride compared to the controls, and a significant reduction in the TC:HDL ratio compared to both the controls and the diet only group. Despite these different responses the authors' calculation of estimated risk of CHD was reduced for both men and women by an identical amount (-35 %) with the exercise plus diet groups.

Whitehurst & Menendez (1991) found that even older women (> 60 years) on an eight-week programme of walking at 70-80% of predicted maximum heart rate, three days per week, lowered total cholesterol and increased HDL levels. The corresponding reduction in the TC:HDL ratio would suggest that there was a reduction in CHD risk. The alterations in lipid profile were paralleled with reductions in BP, body mass, predicted % fat and the time to walk one mile.

In a meta-analysis of 27 studies with women, Lokey & Tran (1989) concluded that exercise was effective at reducing total cholesterol triglycerides and TC:HDL ratio. However there was not a significant increase in HDL or decrease in LDL. These results differ slightly to those found in men. This could be due to the relatively small number of studies or other factors like alcohol consumption, menstrual phases, menopause status or oral contraception use. Like many other reviews the change in lipid profiles were greatest in those who had the greatest reductions in weight and the highest initial levels.

Lower intensity regular aerobic exercise such as walking has been shown to have a favourable effect on the lipid profile. Tucker & Friedman (1990), from a sample of 3,621 adult men and women, reported that individuals that walked for exercise , 2.5-4 hours or more per week had half of the prevalence of elevated TC:HDL ratio (TC:HDL $\geq$ 5.0) than those who did not walk or exercise regularly.

Cook et al. (1986) have also demonstrated that chronic low level physical activity has a favourable effect on the lipid profile. They found that the number of miles walked per day by 35 postal workers was significantly correlated to HDL_2 levels, even after controlling for age, alcohol consumption, body mass index and leisure time activity. The longitudinal study by Hardman et al. (1989) demonstrated that a 12-month walking programme was effective at increasing HDL concentrations and reducing the TC:HDL ratio, in a group of middle-aged sedentary women. There was also a slight ,non-significant drop in TC levels, and those with the largest initial levels had the largest decreases.

As well as clear evidence of regular aerobic exercise having a lipid modifying effect there is also evidence of an acute alteration in lipid profile as a result of a single bout of aerobic exercise, for a recent review see Pronk (1993). The pattern of this acute effect may depend on training status as one study has reported that there were slightly different responses in HDL levels for young normolipidaemic individuals trained (T) and untrained (UT) , after an acute bout of exercise. One hour after a bout of low intensity exercise (2 hours at 30% $\dot{V}O_2$ max.) both groups had significantly increased HDL levels, but after two hours the trained group's HDL level had continued to rise whereas there was no further change for the untrained group (Pay et al., 1992).

Pronk (1993) concluded that a single bout of aerobic exercise had the potential to induce short term, transient increases in HDL and HDL_2 and decreases in triglycerides in men. Further studies are required to clarify if a similar effect exists for women. Exercise bouts of higher intensity and longer duration seem to produce changes of greater magnitude and duration. These short term alterations in lipid profile return to pre-exercise levels after 24 hours.

5. WALKING AS AN EXERCISE MODE

Walking is becoming an increasingly popular mode of exercise for both young and old, with many medical and health professionals regularly prescribing a brisk walk. The Framingham Offspring Study reported that walking for pleasure was the most common leisure activity among the study participants, whether measured by the number of times mentioned or total Kcal expended per year (Dannenberg et al., 1989). The Allied Dunbar National Fitness Survey (ADNFS) of England found that, walking continuously for at least one mile per week was an activity pursued by just over half of the men and women in the sample, and that over 43% of the sample had walked for two miles or more on at least one occasion in the previous four weeks (ADNFS, 1992). The popularity of walking lies in the fact that it is easy to do, requires no special skill, requires no facilities and is achievable by virtually all age groups with little risk of injury. Walking can take many forms - race walking, power walking, aerobic walking, health walking, mall walking, hill walking, dog walking and walking the golf course to name but a few. Several walking studies have shown considerable health benefits for all age groups, with improved aerobic fitness (Duncan et al., 1991; Hardman et al., 1989, 1992; Jette at al., 1988; Leon et al., 1979; Pollock et al., 1971; Rowland et al., 1991; Santiago et al., 1987; Stensel et al., 1993; White et al., 1984; Whitehurst & Menendez, 1991) , reduced body mass (Hudson et al., 1988; Leon et al., 1979; Ohta et al., 1990; Pollock et al., 1971; White et al., 1984; Whitehurst & Menendez, 1991), lower body fat (Leon et al., 1979; Ohta et al., 1990; Pollock et al., 1971; Whitehurst & Menendez, 1991), a fall in blood pressure (Leon et al., 1979; Ohta et al., 1990; Pollock et al., 1971; White et al., 1984; Whitehurst & Menendez, 1991) and improved blood lipid profile (Duncan et al., 1991; Hardman et al., 1989; Hudson et al., 1988; Leon et al., 1979; Ohta et al., 1990; Santiago et al., 1987; Whitehurst & Menendez, 1991). Blair et al. (1992) referred to epidemiological data when they suggested that the most sedentary section of the population "would receive clinically significant health benefits" if this category of adults underwent 30 minutes of walking per day. It is considered that these health benefits

would span a range of chronic diseases leading to a reduced risk of morbidity and mortality.

5.1. Blood Pressure

Regular aerobic exercise is associated with decreases in systolic and diastolic blood pressure in mild and moderate hypertension (ACSM, 1993; Hagberg, 1990). A meta-analysis of 40 longitudinal studies concluded that in subjects with slightly elevated blood pressure (>140/90 mmHg), systolic blood pressure was decreased by approx. 11 mmHg and diastolic blood pressure was reduced by approx. 9 mmHg (ACSM, 1993). Most walking studies have included only normotensives as subjects. Thus a dramatic fall in blood pressure is unlikely as it is not possible to reduce blood pressure in the normal range by a large amount. The majority of walking studies, that have measured blood pressure show a small but significant decrease in blood pressure (Leon et al., 1979; Pollock et al., 1971; Porcari et al., 1988; White et al., 1984; Whitehurst & Menendez, 1991). One study that failed to show any change in blood pressure after 24 weeks of walking (Duncan et al., 1991) had baseline values that were already relatively low (mean 108/73 mmHg). Comparisons with other studies that have employed non-walking exercise modes with normotensive subjects have revealed a similar situation.

5.2. Lipids

Many studies that have investigated the effect of aerobic exercise on lipids have produced conflicting findings. These inconsistencies may be explained by the initial baseline lipid level, variety of training stimulus used in the different studies, the lack of dietary analysis and the confounding factor of changes in body composition during training. There is consensus that training studies of 12 weeks or longer are associated with an increase in HDL cholesterol averaging 5 $mg.dl^{-1}$, although this increase was not significant for all of the studies (Wood & Stefanick, 1990). Several walking studies have resulted in increases in HDL cholesterol in a wide age range of men and women (Duncan

et al., 1991; Hardman et al., 1989; Hudson et al., 1988; Leon et al., 1979; Santiago et al., 1987; Whitehurst & Menendez, 1991).

Duncan et al. (1991) speculated that the exercise prescription needed to increase HDL cholesterol may be different from that required to promote increases in aerobic power. Women who walked at low intensity ("strollers") showed the same increase in HDL cholesterol as those who carried out the same frequency and duration of exercise but completed the training distance faster than the strollers ("aerobic walkers"). In this 24-week study the "aerobic walkers" showed a 16% increase in $\dot{V}O_2$ max. whereas the "strollers" demonstrated only a 4% improvement in $\dot{V}O_2$ max.

The findings of Cook et al. (1986) and Tucker & Friedman (1990) also indicated that low intensity, long duration walking plays an important role in increasing HDL cholesterol or improving the total cholesterol / HDL ratio. The number of miles walked per day by postal workers was significantly correlated to HDL levels (Cook et al., 1986). Even intermittent walking involved in playing golf has been shown to favourably alter the lipid profile. Golfers who played three rounds per week from May to September, walking approximately 14 miles per week, exhibited reductions in LDL (calculated) and TC:HDL ratio compared to a matched control group (Palank & Hargreaves, 1990).

The sub fraction HDL_2,that has been shown to be higher in more active individuals (Cook et al., 1986; Haskell, 1986) and is negatively correlated to the presence of CHD (Drexel et al., 1992), has not been measured in any longitudinal walking studies.

5.3. Body Composition

A considerable number of investigators have examined the effects of aerobic exercise on body composition. Wilmore (1983) in his review of 55 studies on aerobic exercise (training duration 6-104 weeks) and body composition, concluded that mean decreases in body fat of 1.6% were minimal, and he stressed that tighter control of energy intake and energy expenditure must be made to clarify some of the discrepancies in the area.

Several studies have shown that walking can decrease percentage body fat and / or body mass in men and women (Hudson et al., 1988; Leon et al., 1979; Pollock et al., 1971; White et al., 1984; Whitehurst & Menendez, 1991). Combined with a diet, brisk walking has been shown to be very effective at reducing weight and body fat in obese individuals (Ohta et al., 1990). In the detailed study of Leon et al. (1979), 6 obese males walked for 90 minutes per day, 5 days per week on a treadmill. These subjects showed a decrease of 5.7 kg in body mass and a fall of 6% in percentage body fat. This reduction in body mass was due to a fat loss of 5.9 kg and an increase in lean body mass of 0.2 kg. Daily food intake increased initially but later decreased to below pretraining levels. However a number of walking studies have failed to demonstrate any change (Duncan et al., 1991; Hardman et al., 1992; Rowland et al., 1991; Santiago et al., 1987; Stensel et al., 1993).

5.4. OSTEOPOROSIS

Osteoporosis is a common problem faced by older adults, especially post-menopausal women, often resulting in fractures of the vertebrae or femur. Although one study has shown no slowing of bone loss in post-menopausal women with brisk walking (Cavanaugh & Cann, 1988), there are similar studies that have shown positive results with walking (Nelson et al., 1991; Sandler et al., 1987). Additional evidence comes from animal studies that have shown increases in bone density and a slowing of osteoporosis with weight bearing exercise in rats (Myburgh et al., 1989). Cross-sectional studies of athletes and physically active people show that they have greater bone mass than their sedentary counterparts (Smith & Raab, 1986). Thus it is clear from human and animal studies that regular physical exercise, particularly weight bearing exercise like walking, either can increase bone mineral content or at least retard the rate of bone mineral loss (Smith & Raab, 1986). It is estimated that weight bearing exercise can, reduce the risk of fracture by as much as a half, thereby preventing some 20,000 fractures each year in England and Wales (Law et al., 1991).

5.5. AEROBIC POWER

Many studies have shown that regular aerobic exercise carried out 3 times per week for 30 minutes or more at intensities at or above 50% $\dot{V}O_2$ max. will result in increases in aerobic power in previously sedentary individuals (ACSM, 1990). These guidelines hold true for a variety of exercise modes including walking. However, some studies have demonstrated that unfit, middle aged men improved their aerobic power while they exercised at around or under 45% $\dot{V}O_2$ max. (Badenhop et al., 1983; Gossard et al., 1986). A review of recent studies involving walking shows that in terms of intensity they all exceed the American College of Sports Medicine (ACSM) guideline's minimum, but not all the variables show improvements (Table 9).

Generally walking is not perceived as 'physical exercise', but Porcari et al. (1987) were able to demonstrate that for the majority of men and women, fast walking on the flat was able to elicit heart rates high enough to give an adequate training stimulus. The effects of a walking programme on aerobic power have ranged from large to quite modest improvements, over a wide range of age groups.

The middle-aged men and women of the Jette et al. (1988) study improved aerobic power by 17 and 10% respectively after a 12 week programme of 3 times per week for 30 minutes per session at 60% $\dot{V}O_2$ max. Thirty-two women (30-62 years) followed a 3 month programme of 200 minutes per fortnight walking increasing to 350 minutes by the end of 3 months. $\dot{V}O_2$ max. increased from 27.0 ml. kg^{-1}. min^{-1} to 29.1 ml. kg^{-1}. min^{-1} (Hudson et al., 1988). After 11 weeks of 3 days per week walking at 80 % of maximum heart rate Rowland et al. (1991) found a 10 % increase in aerobic power in predominantly obese teenagers.

Top class competitive race walkers have a physiological profile similar to top class marathon runners (Franklin et al., 1981) with an aerobic capacity corresponding to about 85-95% of elite marathon runners (Rippe et al., 1986).

5.6. ORTHOPAEDIC PROBLEMS

Some studies have suggested that walking has an advantage over some other modes of exercise as walking places less stress on bones and joints resulting in a reduced incidence of musculoskeletal injuries. The primary cause of running or jogging injury is the force of impact when the feet hit the ground after the airborne phase. This force is equal to 2.5-3 times body weight and occurs every stride (Stamford, 1986). The highest ground forces observed for walking are about 1.25 times body weight (Rippe et al., 1986). Santiago et al. (1987) in a comparison of walking and jogging programmes for sedentary women found that, for similar physiological improvements, the jogging programme had a 40 % drop out due to muscloskeletal injuries, whereas only 7 % of the walkers dropped out due to injury.

Exercise also has the added benefit of increasing, or slowing the rate of decline in muscular strength (Rogers & Evans, 1993), making the older adult less susceptible to injury and the possibility of falling.

5.7. WALKING SPEED

Self-selected walking speed declines with advancing age, although there would seem to be a critical age of 'slowing down'. A study of 149 females (aged 22 to 95 years) and 289 males (aged 19 to 102 years) revealed that there was only a very small reduction in walking speed (1 to 2% per decade) up to an age of 62 years. After 63 years of age females showed a 12.4% per decade decrease and males showed a 16.1% per decade decrease (Himann et al., 1988). The ADNFS also reported a significant decline in walking pace with increasing age in men, but the final report does not seem to document how this was measured (ADNFS, 1992). Spelman et al. (1993) assessed the self-selected exercise intensity of twenty-nine healthy adults (22 females, 7 males, age 34.9 ± 8.6 years) who were habitual walkers. The mean self-selected walking pace was 1.78 ± 0.19 $m.s^{-1}$. Slower walking speeds have been reported from a similar age group from the

general population. Male and female subjects (aged 19-39 years) asked to walk at a fast pace were measured at 1.71 $m.s^{-1}$ and 1.59 $m.s^{-1}$ respectively (Himann et al., 1988). When 343 subjects were asked to walk as fast as possible 66.7% of the men and 91% of the women attained a training heart rate (THR), which was defined as > 70% of maximum heart rate. The walking pace of the subjects who attained THR was considerably higher than the habitual walkers, ranging from 2.1 ± 0.1 $m.s^{-1}$ for youngest male age group (30- 39 years) to 1.7 ± 0.1 $m.s^{-1}$ for the oldest female age group (60-69 years)(Porcari et al., 1987). This study also demonstrated with a group of ten young men (aged 22-39) that it was possible to walk at an average speed of 2.4 $m.s^{-1}$ for up to 30 minutes. Of course this is much slower than race walkers, who, with their modified walking technique to ensure continual ground contact, can reach speeds of 3.6-4.2 $m.s^{-1}$(Porcari et al., 1989).

5.8. EXERCISE INTENSITY OF WALKING

Generally walking is perceived as a very low intensity activity with limited ability to substantially raise the heart rate. In fact for the majority of men and women brisk walking on the flat is able to elicit heart rates high enough to give an adequate training effect. From a sample of 165 men and 178 women (aged 30-69 years) who were asked to walk one mile as fast as possible, the mean percentage of measured heart rate achieved ranged from 73 to 86%, increasing with age. Overall, 66.7% of men and 91% of women attained THR (defined as $\geq$70% max. HR). Fewer of the younger subjects reached a THR, but Porcari at al. (1987) demonstrated that a second group of young men (aged 22-39 years) in a 30-minute walk were able to maintain a THR for 82.3 ± 19.9 % of the time.
As walking speed decreases there is a near linear reduction in the energy cost, but at speeds lower than 2 mph there is very little further reduction it the energy cost, such that even very slow walking has an energy cost of about 2 Mets (Rippe et al., 1986).
The intensity of walking can be increased by using hand weights (Abadie, 1990; Makalous et al., 1988). During a 30-minute walking session subjects who carried 0.45 kg

in each hand, pumping the arms rhythmically had an increased heart rate (127 Vs 120 beats.min^{-1}), oxygen cost (1.168 Vs 1.086 l.min^{-1}) and greater energy expenditure (171.5 Vs 159.7 kcal) compared to normal walking. One problem of carrying hand weights is that it also increases the blood pressure compared to normal walking. This is believed to be a result of increased peripheral resistance created by the isometric contraction of holding the weights. Abadie (1990) was able to show that if the weight is strapped to the wrist instead of carried that an equivalent increase in energy cost could be achieved without an increase in blood pressure. Simply carrying the weights by the side of the body or on a weight belt results in only minimal increases in the energy cost (Rippe et al., 1986; Stamford, 1986).

5.9. WALKING AS A TESTING MODE

As walking is easy to do, requires no special skill and is a low intensity exercise, it is ideal as a testing modality, especially for very unfit or obese subjects. There are many maximal and sub-maximal treadmill and field tests available to measure or predict aerobic fitness. The treadmill tests typically use a combination of increases in speed and grade to increase workload, many of the tests reaching running speeds for fitter individuals. Klein et al., (1987) developed an equation to predict $\dot{V}O_2$max. using the time to walk one mile and other variables including weight, age, gender and heart rate. The generalised equation showed a correlation of 0.92 between the estimated $\dot{V}O_2$max. from the walking teat and directly measured $\dot{V}O_2$max. (l.min^{-1}). Further gender and age specific equations resulted in similar correlation values as the generalised equation. This equation initially developed with 39-69-year-olds has also been validated with 20-29-year-olds (Coleman et al., 1987). Although one study has shown that these equations do not predict as well and over estimate the cardiorespiratory fitness of adults with mild and moderate mental retardation (Kittredge et al., 1994). Another recent field test to predict $\dot{V}O_2$max. has been developed, based on the time to walk 2km, heart rate at the end of the walk and body weight or BMI (Oja et al., 1991). In two follow-up studies this equation has been

shown to slightly under predict the measured $\dot{V}O_2$max. (Laukkanen et al., 1993 a, b). The underprediction is largest in very fit subjects. Laukkanen et al. (1993, b) calculated that very fit individuals would have to walk at speeds equivalent to professional race walking performance to have an accurate prediction of $\dot{V}O_2$max. Therefore inability of the subjects to walk at that speed would lead to underprediction by the equation.

In theory since both the prediction equations of Klein et al. (1987) and Oja et al. (1991) use several variables to predict $\dot{V}O_2$max. , including time to complete one mile or 2km and the heart rate, a subject could walk at varying speeds without affecting the accuracy of the prediction. Klein et al. (1987) had raised the possibility of this in their discussion but more recently Laukkanen et al. (1993, a) have tested this theory. They found that subjects completing the 2km test (Oja et al., 1991) at maximal and sub-maximal speeds of 60, 70 and 80% of maximum heart rate there were differences in the predicted values. The results revealed that the 80% and maximal walks gave the most accurate prediction of $\dot{V}O_2$max. and the large errors at 60 and 70% resulted in poor predictions of $\dot{V}O_2$max. The authors concluded that when the walking speed is reduced to less than 80% of maximum heart rate the accuracy of the prediction decreases regardless of fitness level and gender.

Many of the treadmill tests commonly used by clinicians and exercise physiologists start with walking but progress to velocities requiring slow jogging or running. The Bruce (Bruce et al., 1973), Modified Bruce (Lerman et al., 1976), Ellestad (Ellsetad, 1980) and STEEP (Northridge et al., 1990) protocols all eventually reach running speeds. Whereas the Naughton (Patterson et al., 1972) and Balke (Balke & Ware, 1959) protocols use increasing gradient at constant walking speeds of 2mph and 3.3mph respectively. All of these protocols can be maximal or submaximal.

5.10. WALKING, HOW MUCH IS ENOUGH FOR HEALTH ?

Research has confirmed that there is an inverse relationship between CHD and physical activity. It is not clear in the literature what intensity of exercise is required to improve health. Wenger & Bell (1986) in their review concluded that for the most effective gains in $\dot{V}O_2$ max. an intensity of 90-100% $\dot{V}O_2$ max. was needed. Some of the large epidemiological studies suggest that there may be a "dose response" to the amount of physical activity (Blair et al., 1989; Duncan et al., 1991; Leon et al., 1987; Morris et al., 1990; Paffenbarger et al., 1993; Sandvik et al., 1993) and that even low levels of exercise can give health benefits. Most epidemiological studies have classified physical activity in terms of energy expenditure and although this figure is useful it may be limited in that intensity of the exercise may well determine some of the possible benefits. Thus in terms of health improvement there is considerable debate to whether there is a threshold intensity or a dose response, and indeed the answer may differ depending on the health variable. Therefore the simple message for the inactive population should be " any walking is better than none at all, but longer faster walks should lead to greater health improvements ".

5.11. SUMMARY

The findings of many walking studies demonstrate that regular walking provides an adequate stimulus for many young, middle aged and elderly people to achieve significant gains in aerobic power. The available evidence also suggests that even a moderate amount of regular walking has the potential to lower blood pressure, improve the lipid profile, reduce body fat, enhance mental well being and reduce the risk of coronary heart disease. At the right pace, walking may be a more appropriate mode of exercise than jogging especially for overweight or extremely unfit individuals. Walking has advantages over other modes of exercise. It is possible to walk almost anywhere, and at any time. The likelihood of injury is lower in walking compared to other exercise modes. Walking

is an activity that can be undertaken by all ages and offers the possibility of a wide range of health benefits.

In general walking affords an excellent opportunity to incorporate some form of regular exercise into a healthier lifestyle.

Author	Subjects			Intensity	Duration	Effect					
	Sex	Age (years)	Number of Subjects			Aerobic Power	Blood Pressure	Body Composi-tion	Total Cholesterol	Triglycerides	HDL Chole stero l
Duncan et al. (1991)	F	20 - 40	16,12,18	86,67,56 % Max HR	24 weeks	I	NC	NC	NC	NC	I
Hardman et al. (1989)	F	44.9	28	60% Predicted $\dot{V}O_2$ Max	12 months	NM	NM	NM	NC	NM	I
Hardman et al. (1992)	F	44.9	28	79.6 % Max HR	12 months	I	NM	NC	NM	NM	NM
Hudson et al. (1988)	F	30 - 62	32	Brisk Walking	3 months	I	NM	I	NC	NM	I
Jette et al. (1988)	F,M	35 - 53	12,14	60 % $\dot{V}O_2$ Max	12 weeks	I	NM	NM	NM	NM	NM
Leon et al. (1979)	M	19 - 31	6	3.2 mph	16 weeks	I	I	I	NC	NC	I
Pollock et al. (1971)	M	40 - 56	16	63 - 76 Max HR	20 weeks	I	I	I	NM	NM	NM
Rowland et al. (1991)	M,F	15.7	1,14	79.6 % Max HR	3 Months	I	NM	NC	NM	NM	NM
Santiago et al (1987)	F	20 - 40	9	71 % Max HR	11 weeks	I	NM	NC	H	H	I
Stensel et al. (1993)	M	42 - 59	72	68 % predicted max. HR	12 months	I	NM	NC	NC	NC	NC
White et al. (1984)	F	50 - 63	27	70 % predicted max. HR	6 months	I	I	I	NM	NM	NM
Whitehurst & Menendez et al. (1991)	F	61 - 81	34	70 - 80 % Predicted Max HR	8 weeks	I	I	I	NC	I	I

Table 9. Table of Walking Studies that specify training intensities, showing their effects on Aerobic Power, Lipid Profile, Body Composition and Blood Pressure

NM-not measured, NC-no change, I-inprovement, H-higher

6. METHODOLOGY

All procedures carried out in both studies were granted ethical permission by the West Ethical Committee, Western Infirmary, Glasgow

6.1. STUDY ONE SUBJECTS

The aim was to recruit 100 sedentary middle-aged (40-60 years) male subjects. It was proposed that this would be made up of ;

1. Forty dog owners recruited to take part in a 14-week walking programme.
2. Forty non-dog owners recruited to take part in a 14-week walking programme.
3. Twenty control subjects who would not participate in the walking programme but maintaining their normal routine and undergo all the physiological testing.

All subjects had to satisfy the following criteria:

a) Male, middle-aged, between 40 and 60 years of age

b) Sedentary - they did not currently walk briskly for more than 60 mins/week.

c) Healthy - no history of heart disease, high blood pressure (not > 150/95 mmHg), bone or joint disorder, which would prevent them participating in the walking programme.

d) Cholesterol - total plasma cholesterol level less than 9 $mmol.l^{-1}$

Subjects satisfying these criteria were not randomly selected but were allocated to the appropriate group. To achieve this, postal invitations were sent to 1250 names randomly selected from the Electoral Register of the 21 Polling Districts within one mile of the University of Glasgow. Names of dog owners close to the University were selected from the University Veterinary School database of dog-owners. Also subjects who were outside the age range for a previous study ' The Health Promotion Project ' (Kelly et al., 1991) were contacted. Unfortunately this initial strategy did not produce enough subjects. Therefore the following new strategies were used to recruit volunteers.

1) An article was placed in the University Newsletter to attract University staff of all categories who might be interested.

2) An article on the project was featured in the national press (Sunday Post) and a request for subjects was made.

3) An interview on a radio programme (BBC Scotland) included a request for subjects.

4) Adverts were placed in the local free press (Glaswegian, Glasgow Gardian).

5) Posters advertising the project were sent to local veterinary surgeries, local hospitals and libraries.

6) Large employers were similarly targeted, namely Glasgow City Chambers, Strathclyde Regional Council.

7) Direct canvassing at supermarket dog food counters.

Although it was not ideal to have such a fragmented recruitment strategy the various methods were necessary to achieve the target number of subjects.

6.2. Study Two Subjects

The aim was to recruit 100 sedentary middle-aged (40-60 years) female subjects, who were willing to take part in a 14-week walking programme. Subjects were randomly selected into the control or exercise group by an age categorised permuted block design in the ratio 1:4. This ratio was chosen in order to match the proportion of exercisers to control subjects in Study One and thus allow comparison between studies. Therefore the subject groups would be made up of;

1. Eighty subjects who would take part in the 14 week walking programme

2. Twenty control subjects who would not participate in the walking programme but would maintain their normal routine, and undergo all the physiological testing.

All subjects had to satisfy the following criteria:

a) Female, middle aged, between 40 and 60 years of age

b) Sedentary - they did not currently walk briskly for more than 60 mins/week.

c) Healthy - no history of heart disease, high blood pressure (not > 150/95 mmHg), bone or joint disorder, which would prevent them participating in the walking programme.

d) Cholesterol - total plasma cholesterol level less than 9 $mmol.l^{-1}$.

Subjects satisfying these criteria were randomly selected to the exercise or control groups. To achieve this two recruitment strategies were employed in study two. Firstly, and the more successful, was an article on the project published in the Glasgow Evening Times. This produced a very large response and provided the majority of subjects for the study. Secondly an article was placed in the Glasgow University Newsletter that again attracted a considerable number of recruits.

6.3. PROCEDURES STUDY ONE AND TWO

The timing of the retest for the women from Study Two was selected to match the phase of the menstrual cycle at the baseline testing. This was to control for the effects of fluctuating hormone levels, known to affect blood lipid levels (Kim & Falkhoff, 1979; Jones et al., 1988).

6.3.1. BLOOD LIPIDS

Following recruitment, subjects attended the Cardiology Department, Glasgow Western Infirmary to give a blood sample for lipid analysis. The subjects fasted for 12 hours prior to 20ml of blood being drawn from an antecubital vein by qualified personnel. If the total cholesterol value was < $9mmol.l^{-1}$ subjects then progressed for physiological tests.

All analysis was carried out by senior biochemists at the Western Infirmary, Glasgow. Serum total cholesterol and triglyceride concentrations were measured on a Cobas-Bio centrifugal analyser (Roche Diagnostics) (see Appendix A), employing enzymatic methods - that is, with cholesterol oxidase and aminoantripyrine for cholesterol and glycerol kinase and pyruvate for triglyceride - Merkotest reagents being used in each case (see Appendix A). High density

lipoprotein cholesterol was separated by using Biomerieux precipitating reagents and the cholesterol concentration estimated as above.

6.3.2. PHYSIOLOGICAL TESTS

These were carried out by the author at the Human Performance Laboratory, Kelvin Hall International Sports Arena. The subjects were given a full explanation of the test procedures and then signed a consent form.

6.3.2.1. BLOOD PRESSURE

After a five minute rest period two Blood Pressure (BP) measurements were made using a standard sphygmomanometer. The sphygmomanometer cuff was placed around the upper arm at the level of the heart. The brachial artery at the elbow was located by palpation and the stethoscope bell placed over it. The cuff was then inflated to 200 mmHg, the pressure was slowly released while listening with the stethoscope for the blood flow through the brachial artery.

Onset of the first tapping sounds, which is the indication of a spurt of blood forcing open the artery during the peak of each systole (Korotkoff I), was taken as the systolic blood pressure. As the pressure was further released the lowering and muffling of the regular cardiac sound (Korotkoff IV) was taken as the diastolic blood pressure.

.Two readings were taken and averaged to give a final result. If the BP was > 150/95 mmHg no further tests were conducted and the subject was referred to his/her Doctor to assess suitability for the project.

6.3.2.2. Body Composition

6.3.2.2.1. Body Mass

Body mass was measured on standard balance scales, with the subject barefooted.

6.3.2.2.2. Height

With the subject again having removed footwear, their height was measured to the nearest cm using a stadiometer.

6.3.2.2.3. Body Fat

All measurements were taken on the right side of the body. Skinfold callipers (see Appendix A) were used to measure skinfold thickness on the biceps, triceps, supra-iliac and sub-scapular sites. The four measurements were totalled and applied to the age-appropriate regression equation (Appendix B) of Durnin and Womersley (1974) to predict body density. The Siri (1956) equation was then used to give a predicted percentage body fat.
The thumb and forefinger were used to elevate a double fold of skin about 1cm proximal to the measurement site. The calliper jaws were placed over the skinfold and slowly released unto the skinfold. After about two seconds a reading to the nearest 2mm was taken. This process was repeated three times at each site and the average score calculated for each site. The triceps measurement was taken midway between the acromium and olecranon process with the arm hanging loosely by the subject's side. The biceps measurement was taken over the middle of the belly of the biceps muscle with the subject's arm hanging loosely by their side. The supra iliac skinfold was measured in the mid-axillary line immediately superior to the iliac crest. Measurement was taken on an oblique skinfold at approximately 45^{o} to the horizontal, following the natural cleavage lines of the skin. The sub scapular measurement

was taken from the site just inferior to the inferior angle of the scapula, approximately at an angle 45^{o} to the horizontal.

6.3.2.3. SUB MAXIMAL TREADMILL TEST

The end point for all tests was at or just before 85% of the subject's age predicted maximum heart rate or for safety reasons when;

1. The monitoring system failed.
2. A subject experienced progressive angina.
3. A subject experienced light headedness, confusion, ataxia, pallor, cyanosis or nausea.
4. A subject experienced discomfort and asked to stop.

6.3.2.3.1. TREADMILL PROTOCOL PILOT

Before the study three different treadmill protocols were piloted in order to design a suitable protocol for the anticipated subject population.

The aim of the pilot study was to construct a sub-maximal protocol that would;

1. Encompass the anticipated range of aerobic fitness of the subjects.
2. Ensure that all subjects completed at least three workloads.
3. Ensure that a steady state had been reached at each workload.
4. Ensure that the total test time was not too long.
5. Ensure that treadmill speeds would not cause the subject to start to run.

For a description of the pilot study see Appendix C.

6.3.2.3.2. TREADMILL PROTOCOL STUDY ONE

Based on the results of the pilot study the protocol in Table 10 was chosen as suitable for this subject group.

WORKLOAD	TIME(mins)	SPEED(km/h)	GRADIENT (%)	PREDICTED $\dot{V}O_2$ (ml kg^{-1} min^{-1})
1	0 - 5	4	0	10.17
2	5 - 10	4.8	2.5	15.9
3	10 - 15	5.3	5	20.25
4	15 - 20	6	6.5	25.2
5	20 - 25	6	9.5	30.6
6	25 - 30	6	12	35.1
7	30 - 35	6	15	40.5

Table 10. Treadmill Walking Protocol Study One. Predicted $\dot{V}O_2$max. is based on the equation from the American College of Sports Medicine (ACSM, 1986)

6.3.2.3.3. TREADMILL PROTOCOL STUDY TWO

It was anticipated that the aerobic power of the women in Study Two would be about 75% of the men in Study One (Astrand & Rodahl, 1986).Therefore to ensure that the majority of the women completed three workloads, to improve the prediction of $\dot{V}O_2$max., it was necessary to create a branch in the original protocol for women with lower aerobic power. This branch was designed to give an oxygen cost that was half way between workload two

and three. If women had reached within 10 beats of their 85% of predicted maximum heart rate by workload 2 they then proceeded to workload 2a as described in Table 11.

WORKLOAD	TIME(mins)	SPEED(km/h)	GRADIENT (%)	PREDICTED $\dot{V}O_2$ (ml kg^{-1} min^{-1})
1	0 - 5	4	0	10.17
2	5 - 10	4.8	2.5	15.1
2a	5 - 10	4.8	5	18.7
3	10 - 15	5.3	5	20.25
4	15 - 20	6	6.5	25.2
5	20 - 25	6	9.5	30.6

Table 11 Treadmill Walking Protocol Study Two. Predicted $\dot{V}O_2$max. is based on the equation from the American College of Sports Medicine (ACSM, 1986)

6.3.2.3.4. Skin Preparation and Electrode Placement

Sites for electrode placement were first shaved, if necessary. Then the site was vigorously rubbed using an abrasive paste (Omniprep) to remove the superficial horny layer of the epidermis (stratum corneum) as this significantly lowers the electrical impedance across the electrode gel / skin interface thus improving the signal / noise ratio (Tam, 1977). Three adhesive electrodes (Blue Sensor, Medicotest) were then placed in the RA, LA and LL positions. RA and LA electrodes were placed three cm below the clavicle three cm medial to the deltoid muscles. The LL was positioned in the axillary line just on the costal margin.

6.3.2.3.5. TREADMILL FAMILIARISATION

At least 5 minutes was allowed as a familiarisation period for the subject to become accustomed to walking on the treadmill. During this time the subject walked at 3.5 $km.h^{-1}$ on a 0% gradient. This time was considered sufficient to familiarise to treadmill walking as the majority of habituation to treadmill walking occurs within five minutes (Charteris & Taves, 1978; Wall & Charteris, 1980). During this familiarisation period the subject was reminded of the exact order of procedures during the test.

6.3.2.3.6. HEART RATE RECORDING

Heart rate was recorded using a three lead ECG recorder (see Appendix A). Recordings were taken during the first and last 15 seconds of the last minute of each workload. From each 15 second recording the heart rate was measured over 10 QRS complexes with a rate ruler. These 20 heart rates were averaged to give an average heart rate during the last minute of each workload.

6.3.2.3.7. GAS SAMPLING

After three minutes of each workload, with the subject still walking, a nose clip was placed over the nose and a mouth-piece attached to a Hans Rudolff valve was placed in the mouth. Via the Hans Rudolff valve and a length of lightweight tubing, exhaled air samples were collected in a 100 L Douglas bag during the last minute of each workload. For comfort the mouthpiece and the nose clip were removed until the next sample period. From the Douglas bag sample pipe 500 ml of exhaled air was drawn through the gas analysers (Appendix A) to measure O_2 and CO_2 concentrations. The Douglas bag was then evacuated through a dry gas meter (Harvard) to determine the volume of exhaled air. The gas meter contained a temperature probe which permitted measurement of the gas temperature.

6.3.2.3.8. CALCULATION OF PREDICTED $\dot{V}O_2$ MAX.

For each workload the oxygen cost was calculated (Appendix E) . Heart rate was plotted against oxygen cost and a regression equation created based on the oxygen cost and heart rate at the three highest workloads. The age predicted-maximum heart rate was then used in the regression equation to give a value of predicted $\dot{V}O_2$max. In Study Two some of the female subjects only achieved two data points, therefore the two points were joined and the equation describing the line was used, with the predicted maximum heart rate, to predict $\dot{V}O_2$max.

6.3.2.4. CALIBRATION

The gas analysis equipment was calibrated before every test to ensure accuracy. Treadmill speed and gradient were accurately checked about every two weeks for the duration of the project. (see Appendix D)

6.3.3. EXERCISE PRESCRIPTION

At the end of the physiological testing the subject was given detailed advice and instruction about the proposed walking programme and on how to measure his/her heart rate by digital palpation of the brachial artery. Accuracy of measurement was checked against the Polar Sports Tester heart rate monitor (see Appendix A). Subjects who had particular difficulty in measuring their heart rate were able to borrow a heart rate monitor for the duration of the project. The exercise prescription was as follows:

WEEK 1	three walks for 20 minutes
WEEK 2	four walks for 20 minutes
WEEK 3	four walks for 30 minutes
WEEK 4	four walks for 30 minutes
.	.
.	.
.	.
WEEK 14	four walks for 30 minutes

The intensity of the walking was set individually at a range of 70 - 75 % of age predicted maximum heart rate. Each subject was given a weekly Training Diary with his individual Training Heart Rate (THR) range (Appendix F). The diary was used to record the number, length and intensity of the walks. Subjects were asked to return training diaries every two weeks to check on progress. If a subject adhered to the programme exactly he/she would complete 55 walks and a total of 1580 minutes of walking.

6.3.4. STATISTICAL ANALYSIS

Analysis of variance was employed to detect any differences among the three subject groups separately at baseline and after the 14 weeks. Any significant result was then followed up by a multiple comparisons test to detect which groups were different.

Paired t-tests were used on all variables to determine if the post value (i.e. 14 weeks) was significantly different from the pre value (i.e. baseline).

These changes (i.e. pre - post) were then compared across the subject groups by an analysis of variance.

Commonly, the observation of interest is related other variables (covariants) that cannot be controlled in the experimental design. To provide a more precise comparison of the

treatments, the effect of all covariants (e.g. age, weight, body fat, total walking time, aerobic power), or indeed the baseline value of any variable on the change (i.e. pre - post), were analysed by means of a General Linear Model. This allowed for tests of homogeneity of the slopes of the subject groups. If homogeneity was found to be reasonable the model was then used to test for differences among the subject groups (as well as any covariate effect).

7. RESULTS

7.1. STUDY ONE (MEN)

7.1.1. RECRUITMENT RESPONSE

From 1250 letters posted to random names generated from the Electoral Register and the Veterinary School database, there were only 115 returns . The additional recruitment methods increased the total number of returns to 211 with the majority of the replies coming from the electoral register (46 %). A breakdown of the response to recruitment strategies can be seen in Fig. 2.

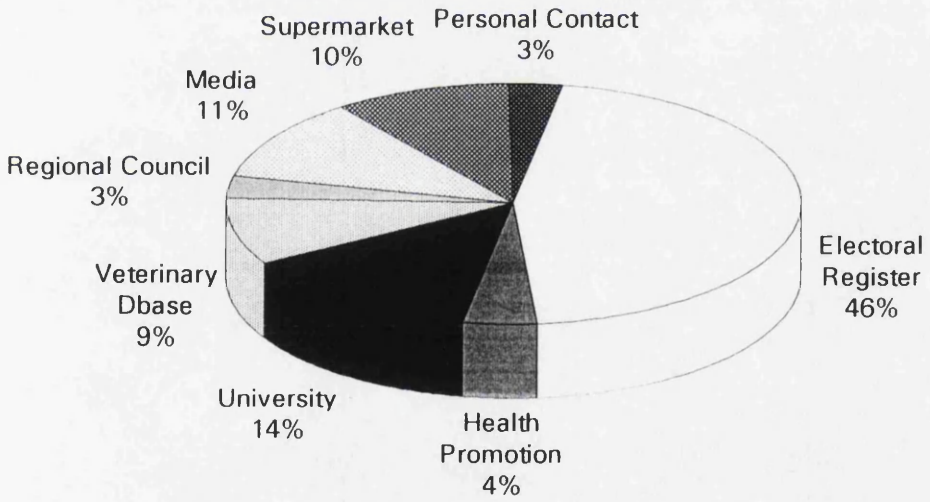

Figure 2. Percentage response from recruitment methods

Of the 211 responses 106 volunteers were rejected because they did not fall into the required subject category . Fig. 3 shows that the major reason for rejecting subjects was that they were "too active" and did not fit our definition of sedentary because they walked briskly more than 60 minutes per week. Interestingly, significantly more dog owners (57.7 %) than non-dog owners (37.7 %) were rejected because they were "too active". The next most common reason for rejecting volunteers was that they were the female. Even though all literature stated that the study was for men only, 18 women still volunteered.

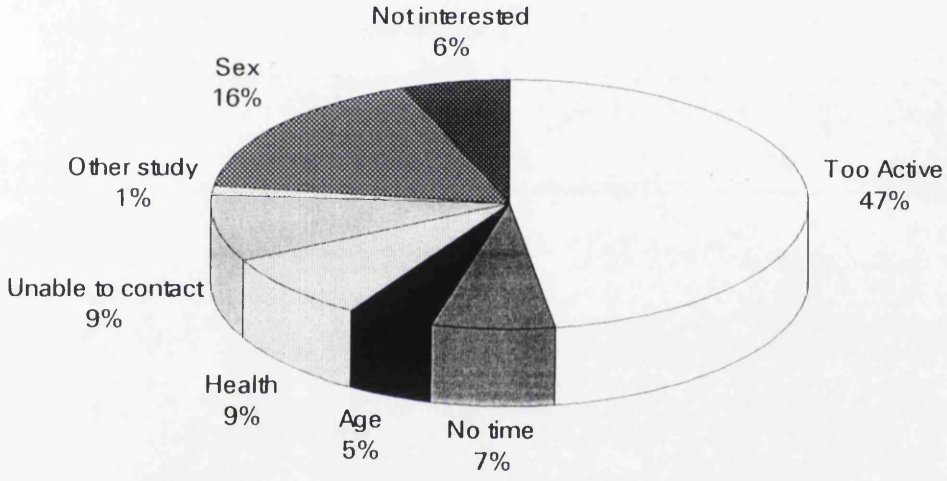

Figure 3. Reasons for rejecting volunteers

7.1.2. DESCRIPTION OF SUBJECTS AT BASELINE

	Non-dog Owners	Dog Owners	Controls
Age	49 ± 6	48 ± 6	48 ± 5
Height (cm)	174.6 ± 12.2	172.6 ± 4.7	174.1 ± 8.1
Weight (kg)	81.2 ± 12.2	82.2 ± 12.4	78.3 ± 10.1
Body Fat (%)	29.2 ± 5.2	30.3 ± 4.6	28.3 ± 4.8
BMI	26.7 ± 4.0	27.6 ± 4.1	25.8 ± 2.3
Predicted $\dot{V}O_2max$ ($l.min^{-1}$)	2.94 ± 0.51	3.15 ± 0.66	3.05 ± 0.67
Predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)	36.4 ± 5.2	38.4 ± 5.8	38.6 ± 6.7
Total Cholesterol ($mmol.l^{-1}$)	5.96 ± 9.6	6.05 ± 7.5	6.12 ± 1.04
Triglycerides ($mmol.l^{-1}$)	1.59 ± 0.95	1.64 ± 1.17	1.28 ± 0.64
High Density Lipoproteins ($mmol.l^{-1}$)	1.19 ± 0.26	1.25 ± 0.31	1.29 ± 0.35
Total Cholesterol / HDL Ratio	5.35 ± 1.60	5.19 ± 1.67	4.93 ± 1.48

Table 12. Study One, Description of physiological variables at baseline (mean ± sd)

Analysis of variance of the values in Table 12 showed that there was no significant difference among the three groups at baseline for any of the variables measured.

7.1.2.1. Body Composition

When subjects were categorised according to their BMI by the classification used in the Allied Dunbar National Fitness Survey (ADNFS, 1990) this revealed that 69% of our sample were classified as being overweight (Table 13).

Weight Classification	BMI Value	%
UnderWeight	20.0 or less	2
Acceptable	20.1-25.0	29
Mildly Overweight	25.1-29.9	52
Obese	30.0 or more	17

Table 13. Study One, Classification of weight categories based on Body Mass Index (BMI) at baseline

7.1.2.2. Blood Lipid Values

Table 12 shows that total cholesterol, triglycerides and HDL values for the three groups were very similar and analysis of variance showed that they were not significantly different. The mean total cholesterol value for all the subjects was 6.03 ± 0.89 mmol.l^{-1}.
If subjects are categorised into risk categories for CHD by total cholesterol values as described by the American Medical Association (1985), it can be seen in Table 14 that

although the majority (57%) are in the normal range for this age group, 18% are considered to be in a high risk category for CHD.

CHD Risk Classification	Total Cholesterol Value ($mmol.l^{-1}$)	%
Normal	< 6.20	57
Moderate Risk	6.21-6.71	25
High Risk	> 6.72	18

Table 14. Study One, CHD risk classification for adults over 40 years (AMA 1985)

7.1.2.3. BLOOD PRESSURE

The mean blood pressure values can be seen in Table 15 . The values were close to normal values, although there were some subjects with values of 160/100 mmHg and thus had to be referred to their General Practitioner for suitability for the project.

	Non-dog Owners	Dog Owners	Controls
Systolic (mmHg)	127 $\pm$ 13	121 $\pm$ 15	121 $\pm$ 10
Diastolic (mmHg)	81 $\pm$ 9	81 $\pm$ 10	79 $\pm$ 9

Table 15. Study One, Blood Pressure measurements at baseline (mean $\pm$ sd)

Figures 4, 5 also illustrate a significant ($p<0.05$) regression between systolic and diastolic blood pressure with age.

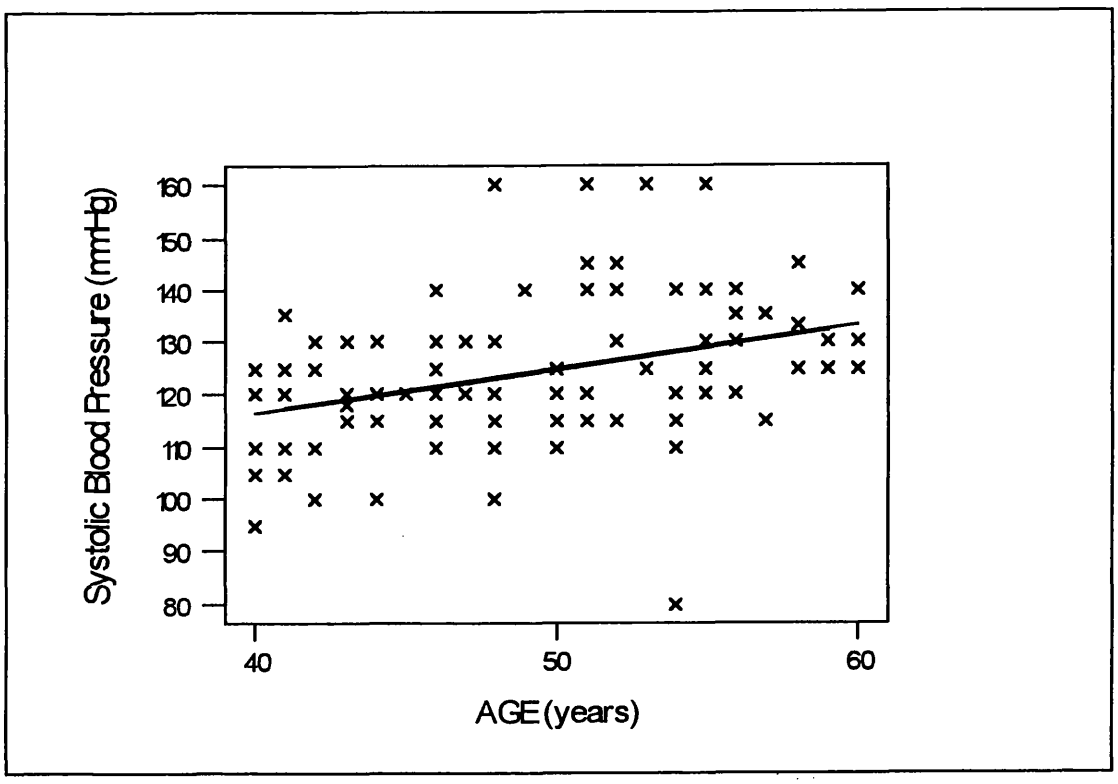

Figure 4. Study One, Regression of systolic blood pressure(mmHg) with age ($p<0.05$)

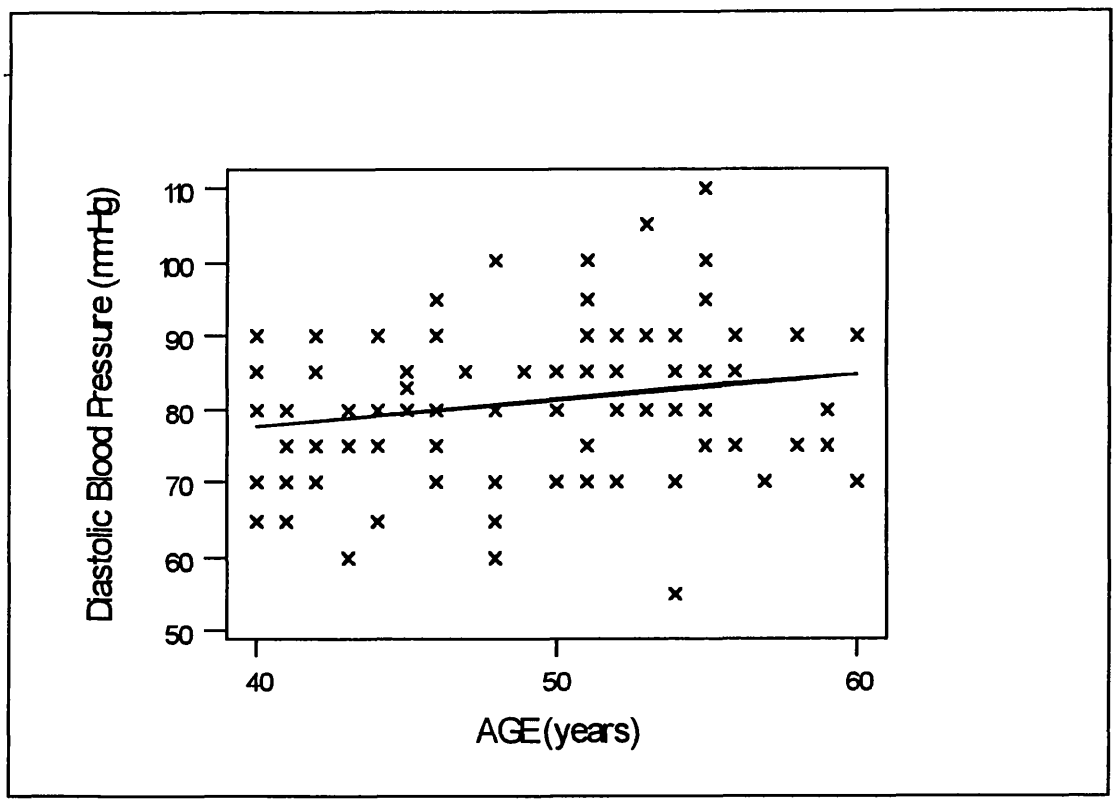

Figure 5. Study One, Regression of diastolic blood pressure(mmHg) with age ($p<0.05$)

7.1.2.4. AEROBIC CAPACITY

Table 16 shows that the majority (65%) of this sample had a predicted $\dot{V}O_2$ max of between 30-39 $ml.kg^{-1}.min^{-1}$.

$\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)	%
Less than 20	0
20-29	7
30-39	65
40-49	25
50-59	3

Table 16. Study One, Predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)

7.1.3. ADHERENCE TO THE WALKING PROGRAMME

Adherence to the walking programme was defined as follows:

Subjects must have completed 66% of the total walk time, at least 1050 minutes of walking. Those achieving less than this were described as drop-outs. One subject was excluded from the analysis as an outlier claiming to have completed more than 3500 minutes walking. Twenty four non-dog owners and 22 dog owners were therefore deemed to have completed the programme. This gave overall adherence figures of 52.2% for the non-dog owners and 56.4% for the dog owners.

Two sample t-tests revealed that there was no difference between the non-dog owners and the dog owners for the number of walks, the total time walked or the average heart rates.

Table 17 shows the actual values. These mean values compare favourably with the exercise prescription of 55 walks with a total walking time of 1580 minutes, and a training heart range for the average aged subject of 120-129 bpm.

	Non-dog Owners (n=24)	Dog Owners (n=22)
Number of Walks	51 ± 6	49 ± 8
Total Walking Time (mins)	1631 ± 211	1674 ± 270
Average Heart Rate (bpm)	122 ± 6	119 ± 12

Table 17. Study One, Description of exercise achievement (mean ± sd)

7.1.4. CHANGES AFTER THE 14 WEEK WALKING PROGRAMME

7.1.4.1. BODY COMPOSITION

7.1.4.1.1. BODY MASS

The non-dog owners showed a significant ($p<0.05$) reduction in body mass (95% CI, 0.75 $\pm$ 0.53 kg) after the 14 weeks (Table 18). The dog owners also tended to have a reduction in body mass but this was not significant. The control group tended to show an increase in body mass which made the absolute change significantly different between the control group and both the exercise groups.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	79.6 $\pm$ 12.5	80.0 $\pm$ 10.5	78.6 $\pm$10.3
Post - Walking Programme	78.9 $\pm$ 12.9	79.4 $\pm$ 10.9	79.4 $\pm$ 10.4
Absolute change	0.8 $\pm$ 1.3 #	0.6 $\pm$ 1.8	-0.5 $\pm$ 1.4 *

Table 18. Study One, Body Mass (kg) (mean $\pm$ sd)

Significant Absolute Change ($p<0.05$)

* Absolute Change Significantly different for control group compared with other two groups ($p<0.05$)

7.1.4.1.2. Body Mass Index (BMI)

The changes in BMI reflect the changes in Body Mass with a significant ($p<0.05$) reduction (95% CI, 0.27 ± 0.18) for the non-dog owners only. In contrast to the exercise groups there was a tendency for the control group to increase BMI illustrated by the change for the controls being significantly different than the other two groups (Table 19).

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	26.1 ± 3.7	27.2 ± 3.6	25.8 ± 2.3
Post - Walking Programme	25.8 ± 3.7	27.0 ± 3.8	26.2 ± 2.0
Absolute change	0.3 ± 0.4#	0.2 ± 0.6	-0.1 ± 0.4*

Table 19. Study One, Body Mass Index (mean $\pm$ sd)

Significant Absolute change ($p<0.05$)

* Absolute Change Significantly different for control group compared with other two groups ($p<0.05$)

7.1.4.1.3. PERCENTAGE BODY FAT

There were no significant within or between group changes in percentage body fat as shown in Table 20.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	28.7 ± 4.4	29.4 ± 5.0	28.2 ± 4.9
Post - Walking Programme	28.5 ± 4.5	28.9 ± 5.4	28.5 ± 5.0
Absolute change	0.2 ± 1.7	0.5 ± 1.9	-0.3 ± 1.9

Table 20. Study One, Percentage Body Fat (mean ± sd)

7.1.4.1.4. SKINFOLD MEASUREMENTS

Analysis of the individual skinfold measurements revealed that there was a significant ($p<0.05$) reduction in the skinfold measurement at the triceps site (95% CI, 0.97 ± 0.87 mm) for the non-dog owners only (Tables 21, 22, 23, 24). An analysis of covariance revealed that the changes in skinfold thickness at the triceps and subscapular measurement sites were significantly ($p<0.05$) dependant on the initial value, for all three groups. The slope of the regression indicated that the larger initial values the greater the reductions in the skinfold thickness.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	14.3 ± 4.9	12.7 ± 3.3	12.7 ± 4.4
Post - Walking Programme	13.4 ± 4.1	12.1 ± 3.6	12.7 ± 4.2
Absolute change	1.0 ± 2.1 #	0.6 ± 2.0	0.0 ± 1.4

Table 21. Study One, Triceps skinfold measurement (mm)(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	7.13 ± 2.99	7.09 ± 2.87	6.37 ± 2.00
Post - Walking Programme	7.24 ± 2.94	6.89 ± 2.92	6.35 ± 1.83
Absolute change	-0.11 ± 0.75	0.20 ± 0.75	0.02 ± 1.18

Table 22. Study One, Biceps skinfold measurement (mm)(mean ± sd)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	24.4 ± 7.5	26.2 ± 8.1	24.7 ± 9.0
Post - Walking Programme	25.3 ± 8.7	25.8 ± 8.5	26.4 ± 9.2
Absolute change	-0.9 ± 3.8	0.4 ± 5.5	-1.8 ± 6.5

Table 23. Study One, Supra iliac skinfold measurement (mm)(mean ± sd)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	19.7 ± 7.4	21.8 ± 6.8	21.5 ± 7.4
Post - Walking Programme	19.1 ± 7.0	21.0 ± 6.8	21.3 ± 7.2
Absolute change	0.7 ± 3.9	0.8 ± 3.5	0.2 ± 1.8

Table 24. Study One, Sub Scapular skinfold measurement (mm)(mean ± sd)

7.1.4.2. BLOOD LIPIDS

7.1.4.2.1. TOTAL CHOLESTEROL

There was a significant ($p<0.05$) reduction in the serum total cholesterol value (95% CI, 0.41 ± 0.22 mmol l^{-1}) for the non-dog owners. Analysis of variance of the post walking programme values revealed that the control group had a significantly higher serum total cholesterol value than the non-dog owners.(Table 25)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	5.86 ± 0.99	6.14 ± 0.72	6.14 ± 1.06
Post - Walking Programme	5.49 ± 0.87 *	5.98 ± 0.85	6.18 ± 0.95 *
Absolute change	0.41 ± 0.55 #	0.09 ± 0.43	0.01 ± 0.55

Table 25. Study One, Serum Total Cholesterol ($mmol.l^{-1}$)(mean ± sd)

Significant Absolute change ($p<0.05$)

* Post Value for Non-Dog Owners significantly different to Controls ($p<0.05$)

The slope of the regression lines in the linear model illustrated in Figure 6 demonstrates the significant relationship between the initial and change in total cholesterol levels. The greater the initial level the larger the reduction in cholesterol. A multiple comparisons test of the separation of the regression lines revealed that the non-dog owners had a significant larger reduction in total cholesterol than the control group, no other of the group comparisons were significant.

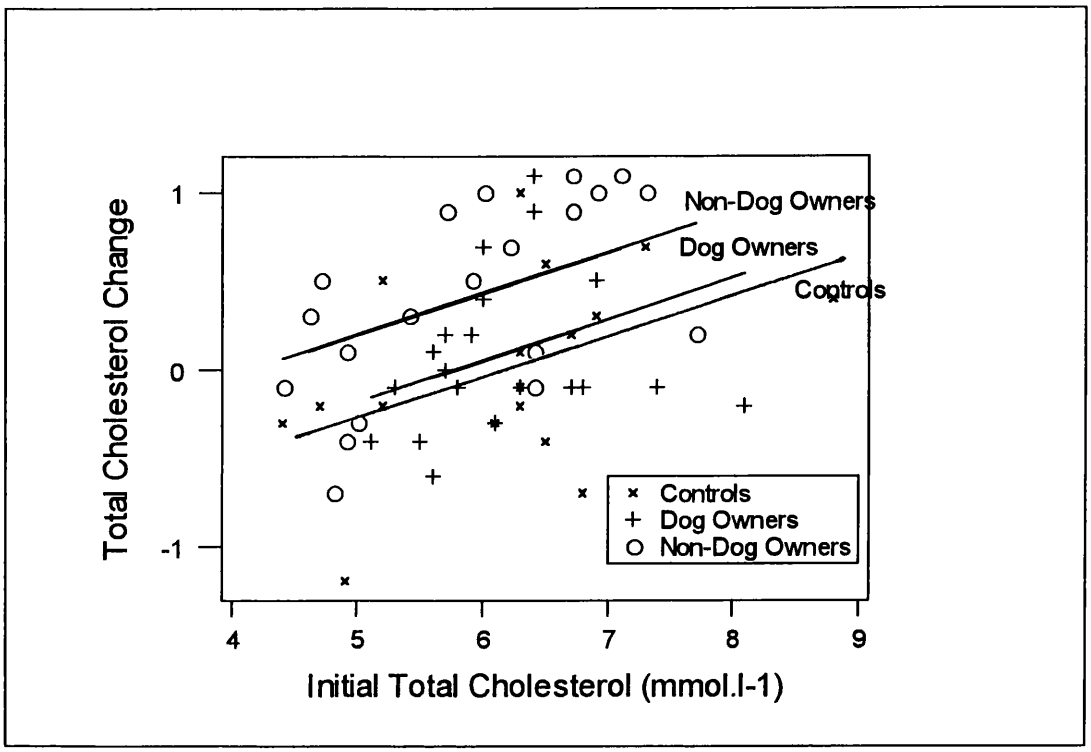

Figure 6. Study One, Change in Serum Total Cholesterol versus initial Serum Total Cholesterol ($mmol.l^{-1}$)

7.1.4.2.2. TRIGLYCERIDES

There were no significant changes in serum triglyceride levels for either of the exercise groups. Whereas the control group had a significant increase (95% CI, 0.21 ± 0.12). As a result the absolute change for the control group was significantly ($p<0.05$) different from the other two groups (Table 26).

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	1.66 ± 1.14	1.78 ± 1.38	1.32 ± 0.64
Post - Walking Programme	1.33 ± 0.79	1.50 ± 0.83	1.58 ± 0.67
Absolute change	0.39 ± 0.86	0.30 ± 0.79	-0.21 ± 0.27 #*

Table 26. Study One, Serum Triglycerides ($mmol.l^{-1}$)(mean ± sd)

Significant Absolute change ($p<0.05$)

* Absolute Change Significantly different for control group compared with other two groups ($p<0.05$)

The slope of the regression lines in the linear model demonstrate the significant relationship between the initial and change in serum triglyceride levels (Figure 7). The greater the initial level the larger the reduction in serum triglyceride. A multiple comparisons test of the separation of the regression lines revealed that the non-dog owners had a significantly larger reduction in triglyceride level than the control group, no other comparisons were significant.

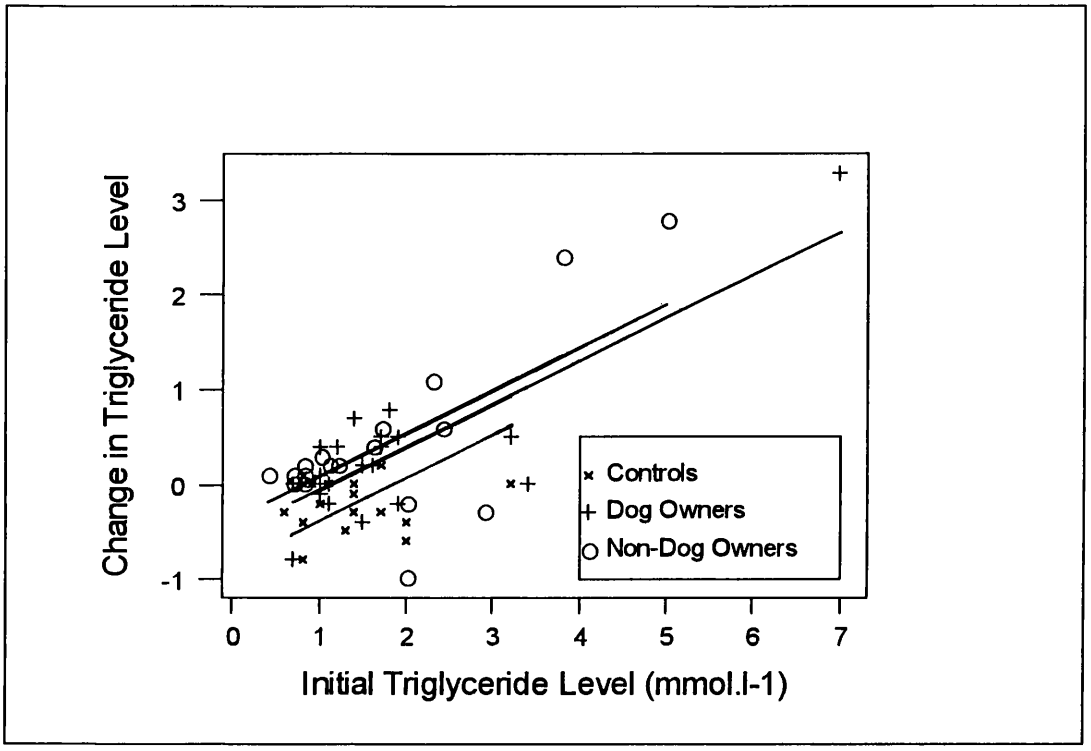

Figure 7. Study One, Change in Serum Triglycerides versus initial Serum Triglyceride level ($mmol.l^{-1}$)

7.1.4.2.3. High Density Lipoproteins (HDL)

As shown in Table 27 there were no significant changes in HDL levels.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	1.18 ± 0.28	1.22 ± 0.26	1.28 ± 0.36
Post - Walking Programme	1.27 ± 0.29	1.22 ± 0.26	1.25 ± 0.28
Absolute change	-0.16 ± 0.35	-0.01 ± 0.13	-0.03 ± 0.23

Table 27. Study One, High Density Lipoproteins ($mmol.l^{-1}$) (mean ± sd)

7.1.4.2.4. TOTAL CHOLESTEROL / HDL RATIO

There was a significant ($p<0.05$) decrease in the total cholesterol / HDL ratio (95% CI, 1.16 $\pm$ 0.66) for the non-dog owners and this change was significantly different than the control group (Table 28).

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	5.44 $\pm$ 1.79	5.32 $\pm$ 1.49	4.99 $\pm$ 1.50
Post - Walking Programme	4.49 $\pm$ 1.18	5.15 $\pm$ 1.38	5.08 $\pm$ 1.27
Absolute change	1.16 $\pm$ 1.61 *#	0.15 $\pm$ 0.70	-0.16 $\pm$ 1.17 *

Table 28. Study One, Total Cholesterol / HDL Ratio (mean $\pm$ sd)

Significant Absolute change ($p<0.05$)

* Absolute Change Significantly different for control group compared with Non-dog Owners ($p<0.05$)

7.1.4.3. BLOOD PRESSURE

Table 29 shows that the systolic blood pressure of the non-dog owners was significantly ($p<0.05$) reduced (95% CI, 5 $\pm$ 3 mmHg) after the walking programme. There were no other changes in either systolic or diastolic blood pressure values. An analysis of covariance revealed that the change in both systolic and diastolic blood pressure was dependant on the initial value for all three groups.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	126 ± 14	122 ± 15	121 ± 10
Post - Walking Programme	121 ± 16	121 ± 13	117 ± 8.
Absolute change	5 ± 8 #	1 ± 10	3 ± 8

Table 29. Study One, Systolic Blood Pressure (mmHg)(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	80 ± 9	82 ± 11	97 ± 9
Post - Walking Programme	79 ± 9	83 ± 11	79 ± 6
Absolute change	1 ± 5	0 ± 8	0 ± 7

Table 30. Study One, Diastolic Blood Pressure (mmHg)(mean ± sd)

7.1.4.4. Sub - Maximal Heart Rates

Analysis of the heart rates for the first three workloads (Tables 31, 32, 33) of the treadmill test revealed that there was a significant ($p<0.05$) decrease at each workload after the

walking programme for the non-dog owners (95% CI, Workload one 6 ± 3, Workload two 3 ± 2, Workload three 5 ± 2 beats . minute^{-1}) and dog owners (95% CI, Workload one 5 ± 3, Workload two 5 ± 3, Workload three 5 ± 3 beats . minute^{-1}) . There were no significant changes at workload four or five, but not all subjects completed these workloads and diminishing numbers reduce the statistical power.

An analysis of covariance revealed that in the first three workloads the change in heart rate was significantly related to the initial value, subjects with higher initial heart rates showing the greatest reductions.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	100 ± 13	94 ± 14	91 ± 11
Post - Walking Programme	94 ± 12	89 ± 12	89 ± 10
Absolute change	6 ± 7 #	5 ± 8 #	3 ± 6

Table 31. Study One, Heart rates for Workload One (beats . minute^{-1})(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	108 ± 12	104 ± 15	101 ± 13
Post - Walking Programme	105 ± 12	99 ± 13	99 ± 11
Absolute change	3 ± 6 #	5 ± 8 #	3 ± 6

Table 32. Study One, Heart Rates for Workload Two (beats . minute^{-1})(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	123 ± 15	118 ± 14	115 ± 13
Post - Walking Programme	119 ± 13	113 ± 14	112 ± 13
Absolute change	5 ± 5 #	5 ± 8 #	3 ± 6

Table 33. Study One, Heart Rates for Workload Three (beats . minute^{-1})(mean ± sd)

Significant Absolute change ($p<0.05$)

Figure 8 shows that there was a tendency for the control group to have a reduction in heart rate of on average just less than three beats at the three workloads but this did not reach significance. The two exercise groups show larger reductions in heart rate but that this was not uniform for the three workloads with an unexplained reduction in the magnitude of change for the non-dog owners at workload two.

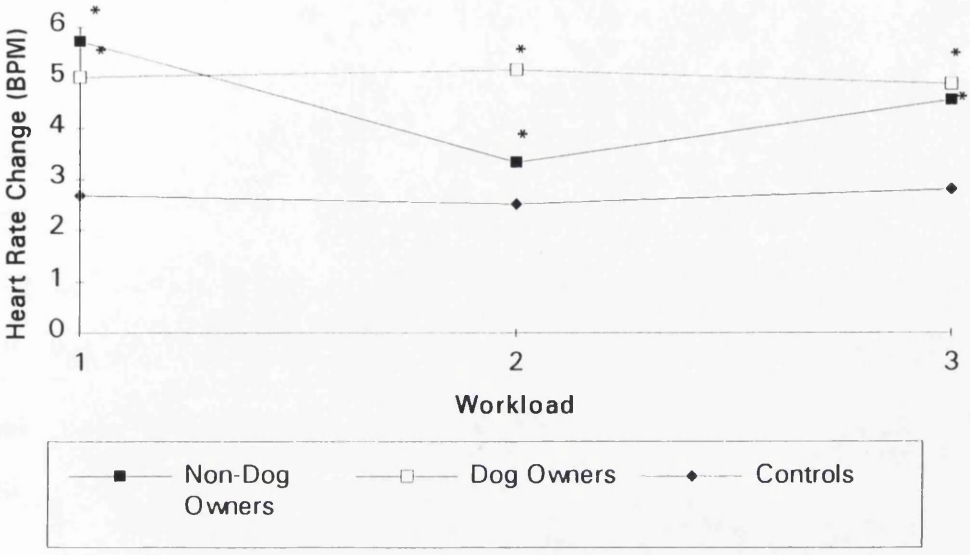

Figure 8. Study One, Mean change in submaximal heart rates for the first three workloads of the treadmill test. * Significant within group change ($p<0.05$)

7.1.4.5. Sub- Maximal Oxygen Costs

Calculation of the oxygen costs at the first three workloads (Tables 34, 35, 36) revealed that there was a significant ($p<0.05$) reductions in oxygen costs for non-dog owners at workload one and three only (95% CI, Workload one 1.2 ± 0.7, Workload three 1.2 ± 0.8 $ml.kg^{-1}.min^{-1}$) whereas there was a significant reduction at workloads one, two, three and four for the dog owners (95% CI, Workload one 0.9 ± 0.7, Workload two 0.8 ± 0.6, Workload three 1.3 ± 0.8, Workload four 1.2 ± 0.8 $ml.kg^{-1}.min^{-1}$).

An analysis of covariance revealed that the change in oxygen cost was significantly ($p<0.05$) related to the initial value for workloads one, three and four in all three groups. Subjects with the highest initial oxygen costs demonstrated the largest reductions.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	13.3 ± 1.8	12.6 ± 1.5	12.5 ± 1.7
Post - Walking Programme	12.2 ± 1.1	11.6 ± 0.8	11.9 ± 1.2
Absolute change	1.2 ± 1.6 #	0.9 ± 1.5 #	0.5 ± 1.7

Table 34. Study One, Oxygen Costs at Workload One ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	16.7 ± 1.3	16.1 ± 1.4	16.0 ± 1.5
Post - Walking Programme	16.3 ± 1.5	15.3 ± 0.7	15.4 ± 1.2
Absolute change	0.5 ± 1.2	0.8 ± 1.5 #	0.6 ± 1.3

Table 35. Study One, Oxygen Costs at Workload Two ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	21.4± 1.5	20.8 ± 1.3	20.3 ± 2.2
Post - Walking Programme	20.3 ± 1.7	19.5 ± 1.9	19.9 ± 2.2
Absolute change	1.2 ± 2.0 #	1.3 ± 2.0 #	0.3 ± 2.5

Table 36. Study One, Oxygen Costs at Workload Three ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	26.8 ± 1.2	26.8 ± 1.6	26.6 ± 1.7
Post - Walking Programme	26.6 ± 1.3	26.0 ± 1.0	26.3 ± 2.1
Absolute change	0.5 ± 1.3	1.2 ± 1.6#	0.4 ± 2.0

Table 37. Study One, Oxygen Costs at Workload Four ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Significant Absolute change ($p<0.05$)

7.1.4.6. VENTILATION

There was a significant ($P<0.05$) reduction in ventilation, at workload one for the non-dog owners (95% CI, 1.9 ± 1.6 $l.min^{-1}$), at workload two for the non-dog owners (95% CI, 2.6 ± 2.3 $l.min^{-1}$) and the dog owners (95% CI, 3.5 ± 2.4 $l.min^{-1}$), at workload three for the dog owners (95% CI, 2.4 ± 2.1 $l.min^{-1}$)(Tables 38, 39, 40). A multiple comparisons test of the data for workload one revealed that the change in ventilation for the control group was significantly ($p<0.05$) different to the change for the other two groups. The change between the controls and the dog owners was also significantly ($p<0.05$) different at workload three.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	24.4 ± 6.0	23.0 ± 5.2	22.1 ± 4.5
Post - Walking Programme	22.5 ± 5.3	21.6 ± 4.2	23.4 ± 9.4
Absolute change	1.9 ± 4.0#	1.4 ± 4.0	-1.8 ± 6.4 *

Table 38. Study One, Ventilation at workload one ($l.min^{-1}$)

Significant Absolute change ($p<0.05$)

* Absolute change for Control group significantly($p<0.05$) different from other two groups

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	30.8 ± 5.6	30.0 ± 6.0	27.2 ± 5.1
Post - Walking Programme	28.2 ± 7.0	26.5 ± 4.8	27.1 ± 6.5
Absolute change	2.6 ± 5.6#	3.5 ± 5.5#	0.2 ± 3.1

Table 39. Study One, Ventilation at workload two ($l.min^{-1}$)

Significant Absolute change ($p<0.05$)

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	36.7 ± 7.6	36.2 ± 6.9	33.3 ± 5.3
Post - Walking Programme	35.5 ± 9.5	33.8 ± 7.2	34.7 ± 7.9
Absolute change	1.2 ± 4.4	2.4 ± 5.0#*	-1.4 ± 4.7*

Table 40. Study One, Ventilation at workload three ($l.min^{-1}$)

Significant Absolute change ($p<0.05$)

* Absolute change significantly ($p<0.05$) different for control and dog owner groups

7.1.4.7. PREDICTED $\dot{V}O_2$ MAX

Significant ($p<0.05$) changes in predicted $\dot{V}O_2$ Max were only found for the non-dog owners (95% CI, 1.5 ± 1.3 ml kg^{-1} min^{-1}) when measured relative to body mass ($ml.kg^{-1}.min^{-1}$)(Table 41). There were no significant changes in absolute predicted $\dot{V}O_2$ Max ($l.min^{-1}$) in any group (Table 42) . An analysis of covariance demonstrated that there was a significant relationship between the initial value and the change in $\dot{V}O_2$ Max ($ml.kg^{-1}.min^{-1}$) for all three groups. This revealed that subjects with the lower initial values had the greatest increases.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	35.1 ± 4.4	38.6 ± 5.8	38.5 ± 6.9
Post - Walking Programme	36.6 ± 4.6	38.8 ± 5.1	38.3 ± 6.9
Absolute change	-1.5 ± 3.1 #	-0.2 ± 4.4	0.2 ± 4.3

Table 41. Study One, Predicted $\dot{V}O_2$ Max ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Significant Absolute change.

	Non-dog Owners	Dog Owners	Controls
Pre - Walking Programme	2.78 ± 0.45	3.07 ± 0.51	3.05 ± 0.69
Post - Walking Programme	2.88 ± 0.56	3.06 ± 0.62	3.06 ± 0.76
Absolute change	-0.11 ± 0.26	0.01 ± 0.41	-0.01 ± 0.35

Table 42. Study One, Predicted $\dot{V}O_2$ max ($l.min^{-1}$)(mean ± sd)

7.2. STUDY TWO (WOMEN)

7.2.1. RECRUITMENT RESPONSE

A total of 88 women volunteered for the study. Twenty subjects were rejected because of being too active (40%), wrong age (25%), health reasons (20%) or decided that they were not interested (15%).

The majority (85%) of the remaining 68 women had been recruited via the newspaper article the remainder were recruited from the University community. High blood pressure and other health problems finally resulted in 62 women entering the study. The group was divided by age categorised random assignment into 48 walkers and 14 controls. Of the 62 starters 38 (61%) were post-menopausal and of these 19 (50%) were receiving Hormone Replacement Therapy (HRT).

7.2.2. DESCRIPTION OF SUBJECTS AT BASELINE

	Walkers	Controls
Age	49 ± 5	49 ± 6
Height (cm)	161 ± 6	161 ± 5
Weight (kg)	69.2 ± 9.0	67.1 ± 7.6
Body Fat (%)	38.6 ± 4.2	38.5 ± 3.1
BMI	26.7 ± 3.5	25.9 ± 2.9
Predicted $\dot{V}O_2max$ ($l\ min^{-1}$)	2.03 ± 0.41	1.82 ± 0.43
Predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)	29.7 ± 6.0	27.3 ± 6.4
Total Cholesterol ($mmol.l^{-1}$)	5.75 ± 0.86	5.24 ± 1.34
Triglycerides ($mmol.l^{-1}$)	1.19 ± 0.43	0.95 ± 0.38
High Density Lipoproteins ($mmol.l^{-1}$)	1.54 ± 0.41	1.65 ± 0.32
Total Cholesterol / HDL Ratio	5.31 ± 1.53	5.98 ± 1.92

Table 43. Study Two, Description of Physiological Variables at Baseline (mean ± sd)

Two sample t-tests revealed that there were no differences between the control and walking group for any variable at baseline.

7.2.2.1. Body Composition

When subjects were categorised according to their BMI by the classification used in the Allied Dunbar National Fitness Survey (ADNFS, 1992) this revealed that 75% of our sample were classified as being overweight or obese (Table 44).

Weight Classification	BMI Value	%
Underweight	18.6 or less	0
Acceptable	18.7-23.8	25
Mildly overweight	23.9-28.5	48
Obese	28.6 or more	27

Table 44. Study Two, Classification of weight categories based on Body Mass Index (BMI) at baseline

7.2.2.2. Blood Lipid Values

Table 44 shows that mean total cholesterol, triglyceride and HDL values at baseline for the two groups were very similar and were not significantly different. The mean total cholesterol value for the total sample was 5.64 $\pm$ 1.0 mmol.l^{-1} . Although the mean value is quite low, classification of the cholesterol levels for CHD risk according to the American Medical Association (1985) reveals that 15% of the sample were in the high risk category (Table 45)

CHD Risk Classification	Total Cholesterol Value ($mmol.l^{-1}$)	%
Normal	< 6.20	76
Moderate Risk	6.21-6.71	9
High Risk	> 6.72	15

Table 45. Study Two, CHD risk classification for adults over 40 years

Splitting the sample by menstrual status (pre/post-menopausal and post-menopausal + HRT) and fitting a linear model revealed that there was a significant ($p<0.05$)difference between the groups in the total serum cholesterol. A multiple comparisons test of the separation of the regression lines revealed that the post-menopausal group had a significantly ($p<0.05$) higher initial total cholesterol level than the pre-menopausal group, even after correction for age.

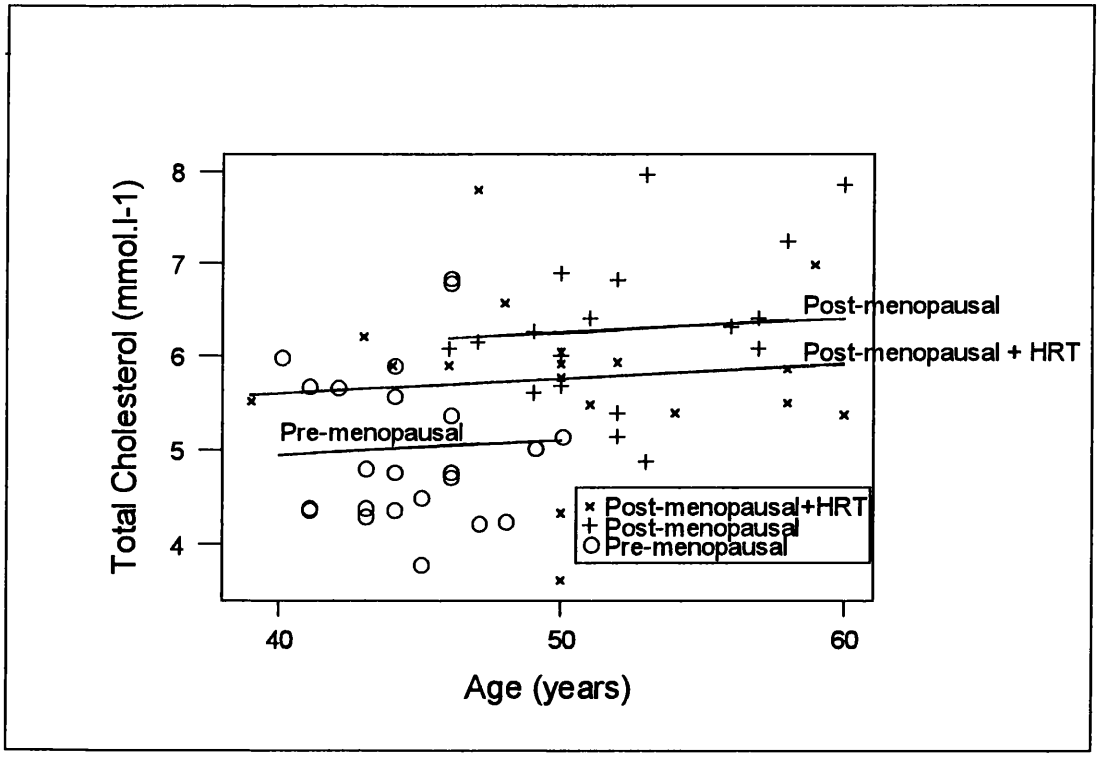

Figure 9. Effect of menstrual status on Total Serum Cholesterol level ($mmol.l^{-1}$)

7.2.2.3. BLOOD PRESSURE

The mean blood pressure values in Table 46 are within the normal range. Figures 10, 11 also show a significant ($p<0.05$) regression between blood pressure and age, so that both systolic and diastolic blood pressure rise with age.

	Walkers	Controls
Systolic Blood Pressure (mmHg)	118 ± 31	121 ± 18
Diastolic Blood Pressure (mmHg)	80 ± 7	80 ± 11

Table 46. Study Two, Baseline blood pressure (mmHg)(mean $\pm$ sd)

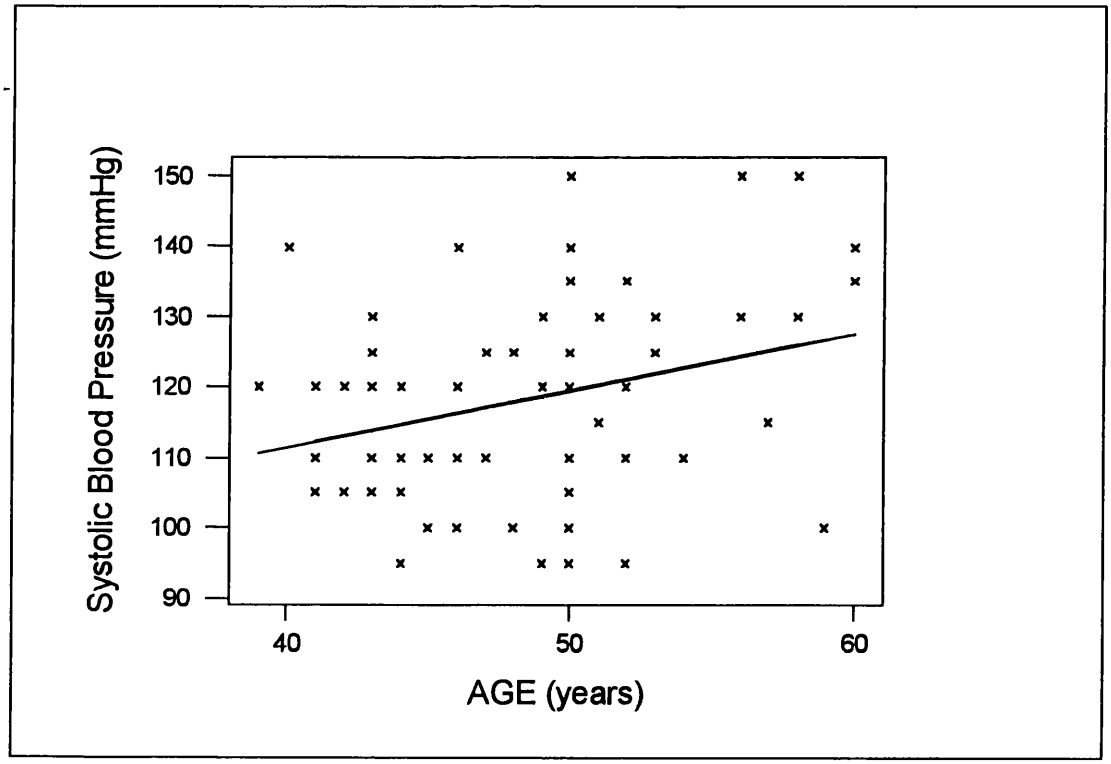

Figure 10. Study Two, Regression of Systolic blood pressure (mmHg) with age ($p<0.05$)

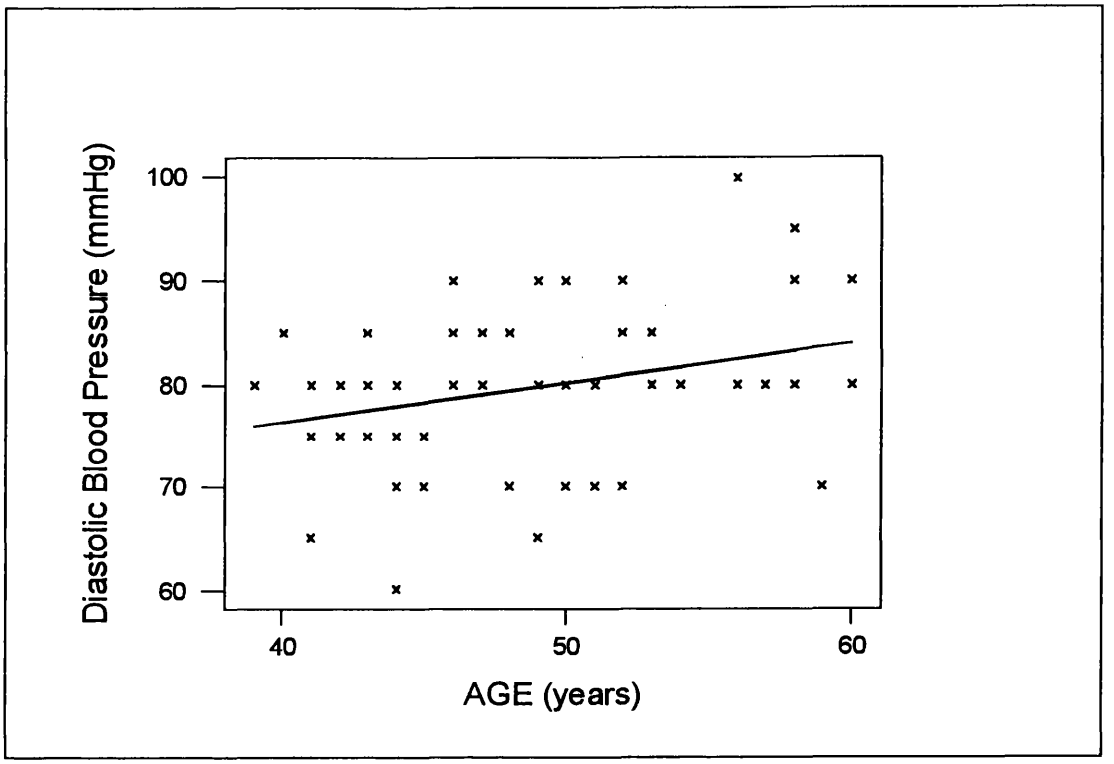

Figure 11. Study Two, Regression of Diastolic blood pressure (mmHg) with age ($p<0.05$)

7.2.2.4. AEROBIC CAPACITY

In general the subjects exhibited a low aerobic capacity with the majority (59%) having a predicted $\dot{V}O_2max$ of less than 30 ($ml.kg^{-1}.min^{-1}$).

$\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)	%
Less than 20	4
20-29	55
30-39	32
40-49	8
50-59	0

Table 47. Study Two, Distribution of Predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)

7.2.3. ADHERENCE TO THE WALKING PROGRAMME

Adherence to the walking programme was defined as follows:

Subjects must have completed 66% of the total walk time, at least 1050 minutes of walking. Those achieving less than this were described as drop-outs.

Twenty five of the women were deemed to have completed the programme. This gave an overall adherence figure of 52%.

Only nine of the original 14 control subjects returned to be retested.

Table 48 shows the mean values for the subjects who were deemed to have adhered to the walking programme. These mean values compare favourably with the initial exercise prescription of 55 walks with a total walking time of 1580 minutes over the 14 weeks.

	Adherers (25)
Number of Walks	49 ± 8
Total Walking Time (mins)	1468 ± 209
Average Heart Rate (bpm)	122 ± 9

Table 48. Study Two, Description of Exercise Achievement (mean ± sd)

7.2.4. CHANGES AFTER THE 14 WEEK WALKING PROGRAMME

7.2.4.1. BODY COMPOSITION

7.2.4.1.1. BODY MASS

There were no significant within- or between-group changes in body mass over the 14 weeks (Table 49).

	Walkers	Controls
Pre - Walking Programme	68.0 ± 8.2	67.9 ± 5.2
Post - Walking Programme	67.2 ± 8.3	68.6 ± 6.3
Absolute change	0.8 ± 2.0	-0.7 ± 1.9

Table 49. Study Two, Body Mass (kg)(mean ± sd)

7.2.4.1.2. BODY MASS INDEX (BMI)

There were no significant within- or between-group changes in Body Mass Index over the 14 weeks (Table 50).

	Walkers	Controls
Pre - Walking Programme	26.3 ± 2.9	26.0 ± 2.6
Post - Walking Programme	26.1 ± 2.9	26.2 ± 3.2
Absolute change	0.3 ± 0.8	-0.2 ± 0.8

Table 50. Study Two, Body Mass Index (BMI)(mean ± sd)

7.2.4.1.3. PREDICTED PERCENTAGE BODY FAT

The control group had a significant ($p<0.05$) increase in % body fat (95% CI, 1.6 ± 1.0 %). There were no significant changes for the walking group.

Analysis of covariance revealed that although the change in % body fat was significantly ($p<0.05$) related to the initial value for both groups there was still no significant difference between the two groups. The larger the initial value the larger the reduction in % body fat.

	Walkers	Controls
Pre - Walking Programme	38.8 ± 4.0	38.7 ± 2.7
Post - Walking Programme	39.1 ± 3.5	40.3 ± 3.3
Absolute change	-0.5 ± 1.5	-1.6 ± 1.5#

Table 51. Study Two, Predicted Percentage Body Fat (mean $\pm$ sd)

Significant Absolute change. ($p<0.05$)

7.2.4.1.4. SKINFOLD MEASUREMENTS

Analysis of individual skinfold measurements revealed a significant ($p<0.05$) increase in skinfold thickness at the triceps (95% CI, 2.7 ± 2.3 mm) for the control group. There was also a significant ($p<0.05$) increase in the biceps skinfold thickness (95% CI, 1.5 ± 1.0 mm) for the walking group. There were no other significant changes either within- or between-groups (Tables 52, 53, 54, 55).

	Walkers	Controls
Pre - Walking Programme	24.2 ± 5.4	23.7 ± 3.1
Post - Walking Programme	24.7 ± 5.1	26.4 ± 5.0
Absolute change	-0.9 ± 3.5	-2.7 ± 3.5#

Table 52. Study Two, Triceps Skinfold measurement(mm)(mean ± sd)

Significant Absolute change. ($p<0.05$)

	Walkers	Controls
Pre - Walking Programme	12.6 ± 4.2	12.5 ± 4.1
Post - Walking Programme	13.9 ± 4.5	14.7 ± 6.0
Absolute change	-1.5 ± 2.6#	-2.3 ± 3.0

Table 53. Study Two, Biceps Skinfold measurement(mm)(mean ± sd)

Significant Absolute change. ($p<0.05$)

	Walkers	Controls
Pre - Walking Programme	25.7 ± 8.0	24.5 ± 6.3
Post - Walking Programme	25.4 ± 7.8	28.3 ± 6.6
Absolute change	-0.3 ± 5.6	-3.8 ± 5.4

Table 54. Study Two, Suprailiac Skinfold measurement(mm)(mean ± sd)

	Walkers	Controls
Pre - Walking Programme	23.4 ± 5.6	24.7 ± 3.1
Post - Walking Programme	23.7 ± 5.3	25.9 ± 5.8
Absolute change	-0.7 ± 4.1	-1.2 ± 4.6

Table 55. Study Two, Subscapular Skinfold measurement(mm)(mean ± sd)

7.2.4.2. BLOOD LIPIDS

7.2.4.2.1. TOTAL CHOLESTEROL

There were no significant within- or between-group changes in the total serum cholesterol values over the 14 weeks (Table 56).

	Walkers (23)	Controls (8)
Pre - Walking Programme	5.74 ± 0.80	5.08 ± 1.47
Post - Walking Programme	5.84 ± 0.94	5.34 ± 1.70
Absolute change	-0.13 ± 0.55	-0.26 ± 0.05

Table 56. Study Two, Total Cholesterol ($mmol.l^{-1}$)(mean ± sd)

7.2.4.2.2. TRIGLYCERIDES

There was a significant ($p<0.05$) increase in serum triglyceride levels (95% CI, 0.23 ± 0.17 $mmol.l^{-1}$) for the control group, there were no significant changes for the walking group.

	Walkers	Controls
Pre - Walking Programme	1.17 ± 0.35	0.88 ± 0.34
Post - Walking Programme	1.30 ± 0.57	1.10 ± 0.50
Absolute change	-0.12 ± 0.42	-0.23 ± 0.26#

Table 57. Study Two, Triglycerides ($mmol.l^{-1}$)(mean ± sd)

Significant Absolute change. ($p<0.05$)

7.2.4.2.3. High Density Lipoproteins (HDL)

Table 58 and appropriate t-tests show that there were no significant within- or between-group changes in the HDL levels over the 14 weeks.

	Walkers	Controls
Pre - Walking Programme	1.52 ± 0.42	1.50 ± 0.21
Post - Walking Programme	1.46 ± 0.35	1.53 ± 0.23
Absolute change	0.06 ± 0.20	-0.03 ± 0.15

Table 58. Study Two, High Density Lipoproteins (HDL) ($mmol.l^{-1}$)(mean ± sd)

7.2.4.2.4. TOTAL CHOLESTEROL / HDL RATIO

There were no significant within- or between-group changes in the total cholesterol / HDL ratio over the 14 weeks.

	Walkers	Controls
Pre - Walking Programme	4.06 ± 1.41	3.38 ± 0.82
Post - Walking Programme	4.24 ± 1.27	3.54 ± 1.10
Absolute change	-0.15 ± 0.54	-0.16 ± 0.39

Table 59. Study Two, Total Cholesterol / HDL Ratio (mean ± sd)

7.2.4.3. BLOOD PRESSURE

There were no significant within- or between-group changes in either systolic or diastolic blood pressure, although the trends were in opposite directions for the two groups. The walking group blood pressure tended to drop as the controls tended to rise but none of the changes was significant.(Tables 60, 61).

	Walkers (25)	Controls (9)
Pre - Walking Programme	118 ± 13	121 ± 18
Post - Walking Programme	111 ± 23	124 ± 13
Absolute change	2 ± 9	-4 ± 12

Table 60. Study Two, Systolic Blood Pressure (mmHg)(mean ± sd)

	Walkers (25)	Controls (9)
Pre - Walking Programme	79 ± 6	79 ± 9
Post - Walking Programme	78 ± 8	82 ± 7
Absolute change	1 ± 7	-3 ± 8

Table 61. Study Two, Diastolic Blood Pressure (mmHg)(mean ± sd)

7.2.4.4. SUB-MAXIMAL HEART RATES

There were significant ($p<0.05$) reductions in sub-maximal heart rates at workloads one, two and three for the walking group (95% CI, Workload one 9 ± 4, Workload two 7 ± 4, Workload three 5 ± 4 beats . minute^{-1}). Analysis of covariance revealed no significant differences between the groups inspite of correcting for a significant ($p<0.05$) relationship between the change in heart rate and the initial value for workloads one and two.

	Walkers	Controls
Pre - Walking Programme	112 ± 14*	111 ± 12
Post - Walking Programme	103 ± 12*	108 ± 12
Absolute change	9 ± 11#	3 ± 8

Table 62. Study Two, Heart Rates Workload One (beats . minute^{-1})(mean ± sd)

Significant Absolute change. ($p<0.05$)

* Post value significantly smaller than pre value for Walkers ($p<0.05$)

	Walkers	Controls
Pre - Walking Programme	126 ± 16	122 ± 10
Post - Walking Programme	119 ± 14	122 ± 12
Absolute change	7 ± 10#	1 ± 9

Table 63. Study Two, Heart Rates Workload Two (beats . minute^{-1})(mean ± sd)

Significant Absolute change. ($p<0.05$)

	Walkers	Controls
Pre - Walking Programme	138 ± 12	140 ± 9
Post - Walking Programme	133 ± 15	143 ± 9
Absolute change	5 ± 8#*	-4 ± 3*

Table 64. Study Two, Heart Rates Workload Three (beats . minute^{-1})(mean ± sd)

Significant Absolute change. ($p<0.05$)

* Absolute change values significantly different ($p<0.05$)

Figure 12 illustrates the change in heart rate after the 14 weeks, showing that the walking group had consistently greater reductions in the heart rate over the three workloads.

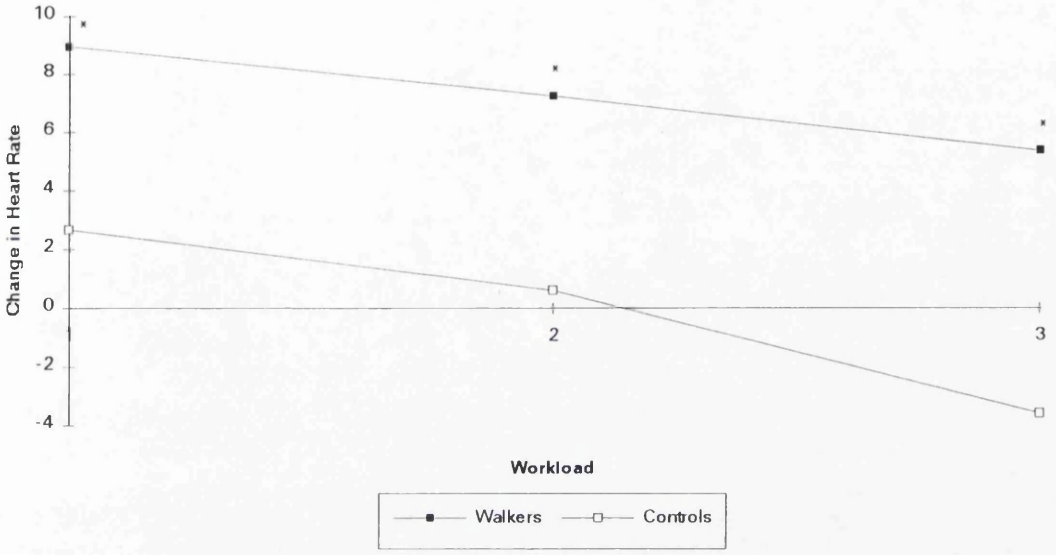

Figure 12. Study Two, Mean changes in Heart Rate (beats . minute^{-1}) at workloads 1,2 and 3

* Significant within-group change ($p<0.05$)

7.2.4.5. Sub-Maximal Oxygen Costs

There were no significant within- or between-group changes in the oxygen costs at any of the workloads (Tables 65, 66, 67).

	Walkers	Controls
Pre - Walking Programme	12.8 ± 1.7	13.3 ± 1.6
Post - Walking Programme	12.4 ± 1.6	13.1 ± 1.3
Absolute change	0.5 ± 1.5	0.2 ± 1.9

Table 65. Study Two, Oxygen Costs Workload One (ml.kg^{-1}.min^{-1})(mean ± sd)

	Walkers	Controls
Pre - Walking Programme	16.8 ± 2.2	16.7 ± 1.1
Post - Walking Programme	16.4 ± 1.9	16.6 ± 1.1
Absolute change	0.4 ± 1.5	0.2 ± 1.3

Table 66. Study Two, Oxygen Costs Workload Two ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

	Walkers	Controls
Pre - Walking Programme	20.8 ± 1.4	21.1 ± 1.0
Post - Walking Programme	21.4 ± 1.8	21.7 ± 1.2
Absolute change	-0.2 ± 1.2	-0.6 ± 1.0

Table 67. Study Two, Oxygen Costs Workload Three ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Figure 13 illustrates the mean change in oxygen costs over workloads one, two and three, both groups showing a similar pattern.

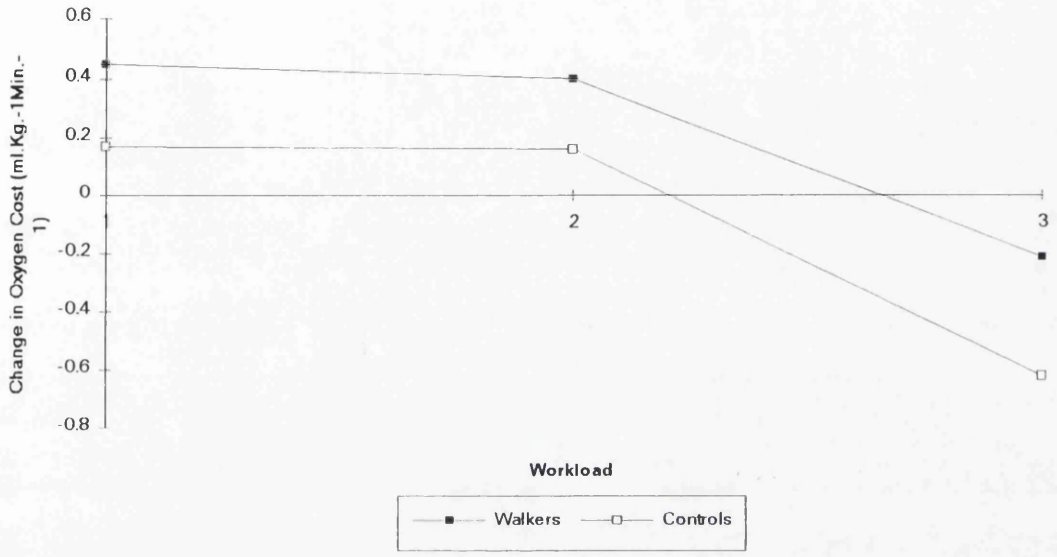

Figure 13. Study Two, Mean change in Oxygen Costs ($ml.kg^{-1}.min^{-1}$) workloads 1,2 and 3

7.2.4.6. PREDICTED $\dot{V}O_2$ MAX

There were no significant within- or between-group changes in predicted $\dot{V}O_2$ Max values after the 14 weeks (Tables 68, 69).

	Walkers	Controls
Pre - Walking Programme	2.01 $\pm$ 0.39	2.03 $\pm$ 0.32
Post - Walking Programme	1.99 $\pm$ 0.31	1.91 $\pm$ 0.19
Absolute change	0.02 $\pm$ 0.34	0.12 $\pm$ 0.32

Table 68. Study Two, Predicted $\dot{V}O_2$ Max($l.min^{-1}$)(mean $\pm$ sd)

	Walkers	Controls
Pre - Walking Programme	29.9 ± 6.5	29.8 ± 5.5
Post - Walking Programme	29.9 ± 5.8	27.8 ± 2.5
Absolute change	-0.1 ± 5.1	2.0 ± 4.4

Table 69. Study Two, Predicted $\dot{V}O_2$ Max ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

7.2.5. Comparison of Men And Women in Baseline Measures

7.2.5.1. Predicted $\dot{V}O_2$ max

A two sample t-test revealed that the male subjects of study one had significantly ($p<0.05$) higher predicted $\dot{V}O_2$max values than the women in study two (95% CI 6.55, 10.37 $ml.kg^{-1}.min^{-1}$).

	Predicted $\dot{V}O_2$ Max ($ml.kg^{-1}.min^{-1}$)
Men	37.6 ± 5.8 #
Women	29.1 ± 6.1 #

Table 70. Comparison of Baseline Predicted $\dot{V}O_2$ Max ($ml.kg^{-1}.min^{-1}$)(mean ± sd)

Significant difference between men and women ($p<0.05$)

7.2.5.2. Total Cholesterol

A two sample t-test revealed that the men had significantly ($p<0.05$) higher total cholesterol levels at baseline than the women (95% CI 0.07, 0.7 $mmol.l^{-1}$).

	Total Cholesterol ($mmol.l^{-1}$)
Men	6.0 ± 0.9 #
Women	5.6 ± 1.0 #

Table 71. Comparison of Baseline Total Cholesterol levels ($mmol.l^{-1}$) Men and Women

Significant difference between men and women ($p<0.05$)

7.2.5.3. TRIGLYCERIDES

A two sample t-test revealed that the men had significantly ($p<0.05$) higher triglyceride levels at baseline than the women (95% CI 0.2, 0.6 $mmol.l^{-1}$).

	Triglycerides ($mmol.l^{-1}$)
Men	1.5 ± 1.0 #
Women	1.1 ± 0.4 #

Table 72. Comparison of Baseline Triglyceride levels ($mmol.l^{-1}$) Men and Women

Significant difference between men and women ($p<0.05$)

7.2.5.4. HIGH DENSITY LIPOPROTEINS

A two sample t-test revealed that the women had significantly ($p<0.05$) higher HDL levels at baseline compared to the men (95% CI 0.5, 0.2 $mmol.l^{-1}$).

	HDL ($mmol.l^{-1}$)
Men	1.2 ± 0.3 #
Women	1.6 ± 0.4 #

Table 73. Comparison of Baseline HDL levels ($mmol.l^{-1}$) Men and Women

Significant difference between men and women ($p<0.05$)

7.2.5.5. TOTAL CHOLESTEROL / HDL RATIO

A two sample t-test revealed that the men had a significantly ($p<0.05$) greater total cholesterol / HDL ratio at baseline compared to the women (95% CI 0.9, 1.9).

	Total Cholesterol / HDL Ratio
Men	5.2 ± 1.6 #
Women	3.8 ± 1.2 #

Table 74. Comparison of Baseline Total Cholesterol / HDL ratio, Men and Women

Significant difference between men and women ($p<0.05$)

7.2.5.6. PREDICTED % BODY FAT

A two sample t-test revealed that the women had a significantly ($p<0.05$) greater % body fat at baseline compared to the men (95% CI 7.7, 10.5)

	Predicted % Body Fat
Men	29.5 ± 5.0 #
Women	38.6 ± 4.0 #

Table 75. Comparison of the Baseline Body Fat % Men and Women

Significant difference between men and women ($p<0.05$)

8. DISCUSSION

8.1. BODY COMPOSITION

8.1.1. BASELINE LEVELS

The majority of subjects in study one and two were classified as overweight (men 69%, women 75%), according to the BMI classification of the Royal College of Physicians (1983). A comparison of the BMI values with a similar age group from the Allied Dunbar National Fitness Survey (ADNFS, 1992) of England (Table 76), reveals that a much higher proportion of both men and women in this study were in the overweight and obese categories (Tables 13, 44).

BMI Classification	% Men (45-54 years)	% Women (45-54 years)
Underweight	1	1
Acceptable	41	40
Mildly overweight	47	44
Obese	11	15

Table 76. Classification of weight categories based on Body Mass Index (BMI), data from the ADNFS (1992)

Considerably less (29% vs 41%) of the men in this study and even fewer of the women (25% vs 40%) were in the 'acceptable' BMI range compared to the equivalent age group of the ADNFS.

An earlier study, The Health and Lifestyle Survey (HPRT, 1987) measured BMI values in 3321 males and 4093 females from several districts in England, Scotland and Wales. Using the same BMI classification as this study, and breaking the data into regions, this survey found that Scotland had the highest percentage of obese men (12%) and the second highest percentage of obese women (20%). Unfortunately these regional data include all age groups

(18+ years), which does not allow a direct age group comparison to this study. These findings suggest that compared to several regions of England, a greater proportion of the Scottish population are obese, which may explain the higher BMI values in this study compared to the ADNFS findings. It should also be remembered that the subjects in this study were specifically selected as sedentary. Thus, considering the strong relationship between obesity and lower levels of physical activity (Dannenberg et al., 1989; Gardner & Poehlman, 1993; Slattery & Jacobs, 1987), it is not surprising that more of the subjects in this study were in the overweight and obese categories than in the ADNFS.

The mean predicted % body fat measures of 29% for the men and 38% for the women in this study are values that would place the subjects in this study in the obese category (Katch & McArdle, 1983) and confirm that the high BMI values are due to excess adipose tissue.

8.1.2. ENERGY EXPENDITURE

An estimation of the Energy Expenditure (EE) can be made based on the oxygen costs during the treadmill test. Inspection of the heart rates during the treadmill test revealed that the heart rate at workload two (Tables 32, 63) matched the mean heart rate reported by the subjects during their walking (Tables 17, 48). Thus it was possible to use the oxygen cost and mean RQ values at workload two to estimate the energy cost of the walking programme, using the following equation:

Energy expenditure = Oxygen cost X Caloric Equivalent (CE)

($kcal.min^{-1}$) ($l.min^{-1}$) $kcal.l(0_2^{-1})$

Where Oxygen cost = is the mean oxygen cost at workload two

CE = is the caloric equivalent of fuel oxidation (estimated as 4.8 $kcal.l^{-1}$ of oxygen, from an exercise RQ = 0.85) (Lamb, 1978)

This value for EE multiplied by the mean number of minutes walked in both studies (Tables 17, 48) gives an estimated overall EE for the whole walking programme. This calculation revealed that the women had an estimated energy expenditure that was about 25% less than the men (Table 77).

	Estimated Energy Expenditure (kcal)
Study One, Men	11,050
Study Two, Women	8,200

Table 77. Estimated energy expenditure of the walking programme

Assuming the subjects maintained a constant Energy Intake (EI) and gained no lean tissue during the training, an energy deficit of 7,700 kcal is required to lose one kilogram of fat (Lamb, 1978). The extra estimated EE from the walking programme should have resulted in a 1.4 kg and a 1.1 kg loss of fat by the men and women respectively. It should be noted that this is an over-estimate as this calculation assumes that all the energy expenditure during the walking was additional energy expenditure. To have a more accurate assessment of the extra energy expenditure it would be necessary to subtract the energy expenditure for the time period if the subject was not taking part in the walking programme.

8.1.3. Experimental Error

It is possible that the inability to measure any changes in body fat in this study was due to some error in the measurement process. This would include experimenter error, equipment error, the use of inappropriate prediction equations and inappropriate measurement site selection. Experimenter and equipment errors were minimised in this study by the same experienced experimenter using the same skinfold callipers throughout the study. The body fat prediction equations used in this study (Durnin & Womersley, 1974) were chosen as the most appropriate as there were developed from measurements made in an equivalent

population (i.e. Glasgow). Thus the apparatus, experimenter and methodological errors were considered to be small in this study.

Although several recent studies that have compared different methods of predicting body fat and changes in body fat have concluded that the skinfold measurement was the most accurate of the bedside methods (Fuller et al., 1993; Jebb et al., 1992) Martin et al. (1985) have outlined several errors that are associated with body fat prediction by skinfold measurement. Firstly there is an assumption that a double layer of skin plus adipose tissue has a constant compressibility. A decline in skinfold measurement after the initial application of the calliper is familiar to all users of skinfold callipers. It has been recommended that skinfold measurement should be taken within a measured time of less than 4 seconds (Becque et al., 1986). In this study measurement of skinfold thickness was taken two seconds after the application of the skinfold callipers, thus it is unlikely that a dynamic change in compressibility has a significant effect on the measurement.

In addition to this dynamic compressibility there is also a static one, in that at certain sites the adipose tissue is more compressible. The Brussels Cadaver Analysis Study measuring skinfold compressibility at 14 sites found a range of 38.3% to 69.3%. As well as demonstrating a wide range of compressibility at different sites this study also reported intraindividual differences (Clarys et al., 1987). This static range of compressibility presumable would only affect absolute measures, as it is unlikely that this would change over time.

The second assumption of skinfold measurement is that the thickness of skin is a constant fraction or negligible compared to total skinfold thickness. Skin thickness does vary according to body site, with the thinnest skin found on the biceps and the thickest found on the subscapula (Clarys et al., 1987). Since skin thickness is in the order of a few millimetres, variability in thickness is likely only to affect measurement of very lean individuals with little adipose tissue.

The third assumption is that there is a fixed pattern of adipose tissue location. In general most of the skinfold methods concentrate on sites on the upper body but Martin et al. (1985), in cadaver dissection, found that there was actually better correlation between adipose tissue mass and calliper reading on lower body sites. This would suggest that some lower body sites should be used in the estimation of body fat from skinfold measurement. This may explain why the non-dog owners in this study had a reduction in weight with no reduction in predicted body fat. Fat loss may have occurred in the lower body (i.e. legs), thus the upper body sites measured in this study would not be able to detect this change.

The fourth assumption is that adipose tissue contains a constant proportion of fat. Information on the fat content of adipose tissue is limited but assuming there is an inverse relationship between water and fat content it is estimated that there is a 20% range in the fat content (Clarys et al., 1987).

The fifth assumption is that there is a constant proportion of internal to subcutaneous fat. The Brussels CAS showed that there was a significant correlation between the majority of skinfold measurements and subcutaneous and total fat mass but the correlation between skinfold measurements and dissectable internal fat was not significant (Martin et al., 1985). Durnin & Womersley (1974) commented that, a proportion of adipose tissue is sited subcutaneously, and whether this proportion changes with body fatness is unclear. To add to this, it is not clear whether exercise or indeed exercise-plus-diet reduces adipose tissue centrally and subcutaneously in equal proportions. Therefore in this study subjects may have reduced centrally located adipose tissue which would not be detected by measuring subcutaneous skinfolds.

8.1.4. Comparison to Similar Studies

This study found a significant reduction in body mass and the skinfold thickness at one site (triceps) for the non-dog owners only. There were no changes in body fat % for either men or women as a result of the walking programme. These findings would agree with similar

walking studies (Duncan et al., 1991; Hardman et al., 1992; Hudson et al., 1988; Rowland et al., 1991; Santiago et al., 1987; Stensel et al., 1993; White et al., 1984) whereas others have found significant reductions in body fat (Leon et al., 1979; Pollock et al., 1971; Whitehurst & Menendez, 1991).

Hudson et al. (1988) found that after three months of brisk walking (eight walks/fortnight for a minimum of 20 minutes on each occasion), a group of middle-aged women had a small (63.5 $\pm$ 8.7 vs 62.7 $\pm$ 8.3 kg) but significant reduction in weight, and a reduction at one skinfold site (anterior thigh) but there was no reduction in the sum of the skinfolds at the other four sites.

This study did not measure energy intake, therefore it is possible that subject increased energy intake to match energy expenditure and thus reduce or eliminate any energy deficit. Similar studies that have measured energy intake have produced mixed results.

A group of middle-aged women who walked briskly on average 157 min.week^{-1} for twelve months demonstrated an improved aerobic fitness but showed no significant reduction in body mass or body fat, despite an unchanged energy intake (assessed from a 7-day weighed food intake) (Hardman et al., 1992). With a much greater volume of brisk walking (90 min.d^{-1}, 5 d.wk^{-1}, for 16 weeks) Leon et al. (1979) found a net loss of 5.7 kg fat mass in six obese men (19-31 years). Three-day dietary records taken at 4, 8, 12, and 16 weeks of training revealed that calorie intake initially increased to a peak at 4 weeks, but this fell progressively to below pre-training levels by week 16.

Similarly a study by Woo et al. (1982) found that when 6 obese women walked on a treadmill to increase daily energy expenditure to 110% (mild) and 125% (moderate) of sedentary expenditure, there was no corresponding increase in energy intake, resulting in an overall negative energy balance (mild -114 and moderate -369 kcal. d^{-1}). Thus it would seem that walking does not increase spontaneous energy intake and should result in a negative energy balance.

8.1.5. SUMMARY

The results of this study indicate that middle-aged men and women who undertake regular 30 minute brisk walks 4 times per week are unlikely to decrease body fat in 14 weeks without also reducing calorie intake.

8.1.6. FUTURE RESEARCH

The assessment of changes in body composition as a result of aerobic training is dependent on the ability to measure three key factors involved, namely energy intake, energy expenditure and the ability to accurately measure changes in body composition. Recommendations for future research should focus on better measurement of the three factors;

1) With skinfold measurement the choice of appropriate measurement sites and population specific prediction equations.

2) An accurate measurement of energy expenditure of the training programme.

3) An assessment of other leisure time activity before and during the training period, to detect if other activity has been substituted for the training programme.

4) The use of 3-7 day weighed food inventories regularly during the training programme to measure energy intake.

8.2. BLOOD LIPIDS

8.2.1. TOTAL CHOLESTEROL

8.2.1.1. BASELINE VALUES

The mean baseline total cholesterol levels for both men and women in this study were slightly high, as the European Arteriosclerosis Society (1987) has recommended that individuals over 30 years of age should strive to attain a total cholesterol level of <200 $mg.dl^{-1}$ (5.17 $mmol.l^{-1}$). This recommendation is based on the findings of the Framingham (Anderson et al., 1987) and the MRFIT studies (Stamler et al., 1986) which found that even total cholesterol levels of 200 $mg.dl^{-1}$ were associated with increased mortality from CHD compared to lower values. In this study there was a large proportion of men (18%) and women (15%) who had total cholesterol values in excess of 6.72 $mmol.l^{-1}$, which would place them in the high risk category for CHD according to the 1985 Consensus Conference (AHA, 1985).

At baseline the total cholesterol level was significantly ($p<0.05$) higher for the men compared to the women (95% CI 0.1, 0.7 $mmol.l^{-1}$). This lower cholesterol level in women is not surprising as a large proportion (39%) of the women were pre-menopausal and it has been shown that pre-menopausal women have a significantly lower total cholesterol level than age matched men (Kim & Kalkhoff, 1979). The baseline data of this study (Figure 9) confirms other cross-sectional data (Notelovitz, 1988) that post-menopausal women have significantly higher levels than pre-menopausal women even after correcting for age. Although not reaching statistical significance at $p<0.05$ in this study there is a suggestion that those post-menopausal women taking Hormone Replacement Therapy (HRT) have lower total cholesterol levels than those not taking HRT. This finding has been confirmed in other studies (Green & Bain, 1993).

8.2.1.2. COMPARISON WITH SIMILAR STUDIES

In this study there was a significant ($p<0.05$) reduction in total cholesterol level from 5.9 ± 1.0 to 5.5 ± 0.9 mmol.l^{-1} for the non-dog owning group only.

A meta-analysis of 66 aerobic training studies revealed a small (10 mg.dl^{-1}) but significant ($p<0.01$) reduction in total cholesterol (Tran et al., 1983). In contrast Haskell (1986) in his review concluded that there was little evidence that exercise had a significant independent lowering effect on total cholesterol levels. In general cross-sectional studies have shown that regular exercisers (e.g. runners) have lower cholesterol levels than sedentary controls (Darga et al., 1989; Williams et al., 1986). In a more recent review Wood & Stefanick (1990) concluded that despite a tendency for training studies to show a reduction in total cholesterol these reductions as a result of increased exercise were seldom significant or independent.

It is surprising that the non-dog owners in this study demonstrated a reduction in total cholesterol especially as a review of similar walking studies (Table 9) reveals that no studies have reported a significant reduction in total cholesterol.

The failure of exercise to reduce total cholesterol levels can be the result of alterations in the concentrations of the various lipoproteins making up the total cholesterol. An increase in HDL concentration may be matched by a decrease in LDL levels, with total cholesterol remaining constant. This does not explain the lack of reduction in total cholesterol levels for the dog owners and the women walkers as neither of these groups demonstrated an increase in HDL levels.

In study one the reduction in total cholesterol was significantly ($p<0.05$) related to the initial value, larger initial values experienced the greatest reductions. This effect was also reported in the reviews by Tran et al. (1983) and Lokey & Tran (1989).

8.2.2. TRIGLYCERIDES

8.2.2.1. BASELINE LEVELS

The baseline triglyceride levels in both studies were in the normal acceptable range outlined by the European Arteriosclerosis Society (1987). At baseline the men had significantly ($p<0.05$) higher triglyceride levels than the women (95% CI 0.2, 0.6 $mmol.l^{-1}$).

8.2.2.2. COMPARISON TO SIMILAR STUDIES

There was a significant ($p<0.05$) increase in triglyceride levels for the control groups in both study one (95% CI, 0.21 ± 0.12 $mmol.l^{-1}$) and study two (95% CI, 0.23 ± 0.17 $mmol.l^{-1}$). There were no significant changes in the exercise groups.

This lack of reduction in the exercise groups is in agreement with the majority of similar studies. Haskell (1986) concluded that, if pre-training values were low (1.35-1.69 $mmol.l^{-1}$), it was unlikely that any further lowering would be achieved with exercise.

Although cross-sectional studies have shown that long distance runners have particularly low levels of triglyceride (Williams et al., 1986) even longitudinal studies of up to one year, running (Wood et al., 1983) or walking (Stensel et al., 1993) have failed to show any significant reduction in triglyceride levels.

There is often a link made between reductions in triglyceride levels and body fat, this in part stems from cross-sectional data that show that the very lowest levels are found in very lean endurance runners (Haskell, 1986) and obese individuals tend to have high triglyceride levels (Kissenbah et al., 1982; Lapidus et al., 1984). Longitudinal studies would seem to support this with two walking studies (Duncan et al., 1991; Stensel et al., 1993) finding no significant reduction in triglyceride levels or body fat. Although in possible contradiction to this, Leon et al. (1979) found that after 16 weeks of brisk walking six obese men had a considerable reduction (5.9 kg) in fat mass and a 12.7% reduction in triglyceride levels, but this reduction was not significant. The relatively small number of subjects in the Leon et al. study and the high intra-individual variability normally found in the measurement of triglyceride levels

(Godsland, 1985) could be responsible for the reduction in triglyceride levels not attaining significance.

One walking study did find a reduction in triglyceride levels in a group of older women (61-81 years) after a 8 week walking programme (Whitehurst & Menendez, 1991). These women also had a significant reduction in % body fat (40.1 $\pm$ 7.7 to 38.8 $\pm$ 7.2 %). In their discussion Whitehurst & Menendez (1991) suggested that it was possible that the reduction in body fat with enhanced mobilisation of free fatty acids, in conjunction with increased lipase activity with endurance training, could result in and increased cellular uptake of triglyceride.

It is therefore possible that the lack of a reduction in triglyceride levels in this study could in part be due to the fact that there was no change in fat mass. This finding of no reduction in triglyceride levels with a walking programme is consistent with the findings of other walking studies (Table 9).

It is difficult to explain why the control groups of both studies had significant increases in triglyceride levels. One reason could be that the control subjects especially at the retest were less motivated than the walking subjects and did not adhere as rigidly to the 12-hour fasting requirement. As triglyceride levels are influenced by fasting status this could have resulted in the increase levels in the control groups. Although, it should be noted that all subjects confirmed fasting status prior to blood sampling.

8.2.3. High Density Lipoprotein (HDL)

8.2.3.1. Baseline Levels

The mean baseline serum HDL levels were above the minimum level (0.9 mmol.l^{-1}) recommended by the European Atherosclerosis Society (1987). The women had significantly higher baseline HDL levels than the men (95%CI 0.21, 0.46 mmol.l^{-1}). This agrees with other studies that have compared HDL levels in men and women of equivalent age (Kim & Kalkhoff, 1979; Lokey & Tran, 1989; Wood & Stefanick, 1990).

8.2.3.2. COMPARISON TO SIMILAR STUDIES

This study found no significant changes in HDL levels for men or women after 14 weeks of brisk walking. This finding is in contrast to several other similar walking studies (Table 9) that have generally found significant increases in HDL levels after 8-52 weeks of brisk walking.

Many cross-sectional studies have shown that endurance trained individuals have significantly higher concentrations of HDL (Herbert et al., 1984; Williams et al., 1986) and in particular increased HDL_2 subfraction (Ballantyne et al., 1982; Wood et al., 1983).

The majority of longitudinal studies would seem to confirm that regular physical activity increases HDL levels, although some studies have failed to show any increase (Faber et al., 1992; Ohta et al., 1990; Stensel et al., 1993).

Three separate reviews (Haskell, 1986; Wood & Stefanick, 1990; Superko, 1991) all suggest that there is a threshold amount of exercise required to see any increase in HDL levels.

Superko (1991) concluded that a threshold of exercise equalling approximately 15 miles.wk^{-1} jogging is required to induce beneficial changes in the lipid profile. The author did not specify whether there were different thresholds for the different lipids.

According to Haskell (1986) the threshold is dependant on baseline status, very sedentary subjects with low HDL levels may only need an energy expenditure of 200-300 kcal per session three times a week to increase HDL levels. For healthy inactive individuals with normal HDL levels the lower threshold was an endurance type exercise requiring an energy expenditure of 1000 kcal.wk^{-1}. Beyond this lower threshold there would seem to be a 'dose response' effect. This was demonstrated by Wood et al. (1983) in a one-year training study of middle-aged men finding that the distance run per week was highly correlated to the change in plasma HDL ($r=0.48$, $p<0.001$). Wood et al. (1983) also concluded that the lower threshold for changes in HDL was approximately 8 miles.wk^{-1} running. Neither of these reviews suggest that there is a time scale for the increase in HDL levels.

In their review Wood & Stefanick (1990) pointed out that only half of studies of less than 10 weeks duration reported increases in HDL, whereas all studies of greater than 12 weeks duration did report an increase in mean HDL concentration, averaging about 5mg.dl^{-1}, although not all were significant at $p<0.05$.

These reviews concentrate on the amount of exercise in terms of increased energy expenditure or study duration. There seems to be limited information on the exercise intensity required for changes in HDL and whether there is an intensity threshold. Duncan et al. (1991) found that there were similar increases in HDL levels after 24 weeks, walking 5days.wk^{-1} at three different intensities (4.8, 6.4 and 8.0 km.h^{-1}). Since improvements in aerobic fitness increased in a linear 'dose response' manner the authors concluded that there would seem to be a difference in the quantity and quality of exercise required to raise HDL levels and what is required for improvements in aerobic fitness. Stein et al. (1990) compared the effect of training at 65, 75 and 85% of maximum heart rate, three times per week (30 minutes) for twelve weeks. They found that there were significant increases in HDL at 75 and 85% only, despite increases in aerobic fitness at all three intensities.

Comparing the results of this study to similar walking studies (Table 9) it is surprising that there were no significant increases in HDL levels, although this result does agree with the recent study of Stensel et al. (1993) who failed to find any change in HDL levels after one year of brisk walking on average 28 min.d^{-1}, despite significant improvements in aerobic fitness. This study however unlike Stensel et al. (1993) failed to reach the minimum threshold of 1000 kcal.wk^{-1} recommended by Haskell (1986).

8.2.4. Total Cholesterol /HDL Ratio

8.2.4.1. Baseline Values

A total cholesterol / HDL ratio of 5.0 or greater is associated with increased CHD risk, therefore ratios of <5.0 are recommended (Pollock et al., 1984). At baseline the men had a mean ratio value just above this (5.2 $\pm$ 1.6 mmol.l^{-1}) and the women were significantly

($p<0.05$) lower (3.8 + 1.2 $mmol.l^{-1}$) than the men (95% CI 0.9, 1.9 $mmol.l^{-1}$), reflecting both their higher HDL and lower total cholesterol.

8.2.4.2. Comparison to Similar Studies

This study found that after 14 weeks of brisk walking the non-dog owners had a significantly ($p<0.05$) reduced total cholesterol / HDL ratio (95% CI, 1.2 + 0.7 $mmol.l^{-1}$), and there were no significant changes in any of the other groups. There are few other studies that report the total cholesterol / HDL ratio, although this could be calculated from the measurements of the two lipids. A meta-analysis of 66 training studies reported a large significant ($p<0.01$) reduction in the total cholesterol / HDL ratio of 0.48 (Tran et al., 1983). Another meta-analysis of 27 studies of the effects of exercise training on serum lipids in women also reported a significant ($p<0.05$) reduction of 0.12 + 0.1 in the total cholesterol / HDL ratio (Lokey & Tran, 1989).

A cross-sectional study of 3,621 adults found that after controlling for age, gender, income, body fat, alcohol use, exercise other than walking and cigarette smoking, the subjects who reported greater than 2.5 hours walking per week had significantly ($p<0.05$) lower total cholesterol / HDL ratios than those who reported no walking (Tucker & Friedman, 1990).

Of the walking studies in Table 9 only two specifically report total cholesterol / HDL values. Duncan et al. (1991) found that the total cholesterol / HDL values were only significantly reduced by 7% in the low intensity exercise group (strollers). The high intensity group (aerobic walkers) failed to show a reduction despite a significant increase in HDL levels as their total cholesterol levels increased slightly.

One study that has compared the responses of men and women to a 10 week programme of moderate exercise (15-20 minutes aerobic activity at 70% maximal heart rate, three times per week) found that the men had a significant 12.4% increase in HDL / LDL ratio whereas there was no significant change for the women (Brownell et al., 1982).

8.3. POSSIBLE MECHANISMS BY WHICH AEROBIC EXERCISE ALTERS LIPID PROFILE

Although this study did not investigate any of the mechanisms by which exercise affects the lipid profile recent studies in this area may indicate possible reasons why this study failed to find more significant changed in the lipid profile.

One of the reasons put forward for increased HDL levels as a result of aerobic exercise is prolonged HDL survival. This improved survival has been found in cross-sectional (Herbert et al., 1984) and prospective studies (Thompson et al., 1988). The cross-sectional study by Herbert et al. (1984) compared the HDL metabolism of five regular runners and five inactive controls. They found that as well as higher concentrations of HDL (primarily due to increased HDL_2 concentrations), the mean biological half-life of HDL proteins was 6.2 days in the runners compared to 3.8 days in the sedentary men. In a small scale study (Thompson et al., 1988), HDL metabolism was studied in eight sedentary men over a maximum of 48 -weeks exercise training. They found that as well as increased HDL levels, the half-life of apolipoproteins A-I and A-II was increased. Both the cross-sectional studies by Herbert et al., (1984) and Williams et al., (1986) found that the runners had significantly increases LPL activity and reduced hepatic lipase activity compared to inactive controls. Also the longitudinal studies by Thompson et al., (1988) found increased LPL activity after a maximum of 48 weeks of exercise and Kantor et al., (1987) found increased LPL activity after one two hour bout of exercise. The conclusion from these studies is that the prolonged survival of HDL in the exercisers was due to augmented lipid transfer to HDL by LPL and/or diminished HDL clearance by hepatic lipase. It is important to note that both Thompson et al. and Kantor et al. used relatively high intensity (80% HR max) exercise lasting from one to two hours, both well in excess of the exercise prescription in this study. One low intensity (30% HRmax) walking study has shown an acute increase in HDL levels, but this was after two hours of walking (Pay et al. 1992).

A very recent study by Houmard et al. (1994) has suggested that the antiatherogenic benefits of a 14-week exercise programme may not be apparent from the calculated LDL concentrations. They found that after the 14 weeks there was no significant change in LDL concentration, but analysis of the LDL chemical composition revealed an increase in LDL free cholesterol, and LDL lipid to protein content. Individuals with CHD tend to have cholesterol-poor, protein-enriched LDL particles. The authors therefore conclude that consideration of only absolute LDL mass may greatly underestimate the cardioprotective effects of long term physical activity.

8.3.1. SUMMARY

A 14-week brisk walking programme was able to improve the lipid profile in one group of middle-aged men (non-dog owners) by reducing total cholesterol and total cholesterol / HDL values. A similar walking programme was unable to improve the lipid profile in a separate group of middle-aged men (dog owners) and a group of middle aged women.

The lack of a reduction in the total cholesterol level for the women could be due to their relatively low baseline values.

A possible reason for the lack of increase in HDL could be that the exercise programme was below the threshold of exercise required.

8.3.2. FUTURE RESEARCH

There are considerably fewer studies on the effects of exercise on the lipid profile of women and in particular post-menopausal women. Considering the poorer lipid profile commonly found after menopause there is a need for further study in this area.

Many studies fail to account for changes in plasma volume that normally accompany aerobic training and it is not clear whether the concentration or amount of blood lipids represent the greatest risk for CHD. If all future studies measure plasma volume, this question may be answered.

The relationship between the amount and intensity of exercise required to improve lipid profile is far from clear. Few studies have considered the intensity of the exercise and there seem to be none that has looked at the effect of different intensities with the same energy expenditure.

8.4. Blood Pressure

8.4.1. Baseline Values

The baseline values for both the men and women are what would be considered to be normotensive. It should be noted that no subjects with a blood pressure in excess of 150/90 mmHg entered the study. There was a significant relationship between the baseline blood pressure and age for both men and women. This agrees with the findings of many cross-sectional studies (Bots et al., 1991).

8.4.2. Comparison to Similar Studies

As concluded by the recent meta-analysis by the American College of Sports Medicine aerobic training reduces both systolic and diastolic blood pressure by on average 10mmHg for individuals with mild hypertension (blood pressures 140-180/90-105 mmHg) (ACSM, 1993). The effects for individuals with blood pressures below this range and considered to be 'normal' are less convincing.

One walking study by White et al. (1984) actually found an increase (5 mmHg) in resting diastolic blood pressure after 6 month walking programme in post-menopausal women. Further analysis of the data by the authors revealed that the increases were found primarily in those individuals who initially had resting diastolic blood pressures under 80 mmHg. More importantly, decreases in blood pressure were found in those individuals having higher (>80mmHg) initial values.

The non-dog owners in this study did show a significant reduction in systolic blood pressure (95% CI, 5 $\pm$ 3 mmHg), although none of the other groups showed any significant changes. In a review of similar walking studies (Table 9) only three show significant reductions in resting blood pressure (Leon et al., 1979; White et al., 1984; Whitehurst & Menendez, 1991). The 20-week study of Pollock et al. (1971) found a significant reduction in diastolic blood pressure and the 8-week study of Whitehurst and Menendez (1991) found reductions in both systolic and diastolic blood pressures. Whereas the 24-week study by Duncan et al. (1991) found no significant changes in blood pressure, but examination of the baseline measurements in these studies reveal that Duncan et al. (1991) had the lowest values (average 108/73 mmHg) and Whitehurst & Menendez (1991) had the highest (135/77 mmHg). Therefore the difference in these results is not surprising, and it would be unlikely considering the initial value that the blood pressure in the Duncan et al. (1991) study would have been lowered further.

The findings from this study would seem to agree with the findings of similar studies in that a programme of regular brisk walking has the ability to reduce blood pressure if initial levels are slightly elevated.

8.4.3. Possible Mechanisms of the Antihypertensive Effect of Exercise

Research has suggested many different mechanisms whereby exercise lowers resting blood pressure. Possible mechanisms include: reduced cardiac output, decreased peripheral resistance, resetting of baroreceptors, changes in blood volume, changes in renin-angeotensin axis and reduction in sympathetic activity.

Guyton (1980) has suggested established hypertension is maintained due to abnormal kidney function and the arterial pressure - sodium excretion curve is shifted to the right to give a new elevated 'set point' for blood pressure.

As for the non-dog owners in this study a reduction in heart rate is commonly accompanied by a reduction in blood pressure, thought to be due to alterations in the sympathetic nervous system. There would seem to be good evidence for this link in the reduction of blood pressure. Stimulation of the renal sympathetic nerves and infusion of norephedrine into renal arteries has the effect to shift the renal function curve similar to that found in essential hypertension (Guyton, 1980). It is generally accepted that aerobic training leads to a reduction in the activity of the sympathetic nervous system, as measured by a reduction in norephedrine spill-over. Jost et al. (1990) demonstrated that long distance swimmers and runners had lowered catecholamine levels at rest. Jennings et al. (1986) were able to show that there were parallel reductions in arterial blood pressure, noradrenalin spill-over and total peripheral resistance after 7 weeks aerobic exercise in normotensive subjects.

The most convincing evidence for the effect of exercise on the sympathetic nervous system, the kidneys and blood pressure comes from Meredith et al. (1991). Using normotensive subjects this cross-over design study was able to show that after one month of cycling 3 times/week for 40 minutes at 60-70% maximum power output, subjects had a significant reduction in blood pressure which was attributed to a 12% reduction in total peripheral resistance. In parallel to this there was a 24% reduction in total norephedrine spill-over and in particular there was a 41% reduction in renal norephedrine spill-over which accounted for two thirds of the reduction in total spill-over. This reduction in renal norephedrine spill-over resulted in a 10% increase in the conductance of the renal vasculature, but this represented only 18% of the total increase in conductance. Therefore, there must have been considerable decreases in vascular resistance elsewhere. There was no reduction in the cardiac norephedrine spill-over thus the reduction in sympathetic activity was largely confined to the kidneys.

To add to this Floras et al. (1989) found that an acute bout of 45 minutes of sub-maximal treadmill exercise in young borderline hypertensives, resulted in a reduction in systolic blood pressure that was paralleled by a reduction in muscle sympathetic nerve activity. It was

interesting to note that two subjects who did not demonstrate post exercise hypotension also did not have any reduction in muscle sympathetic nerve activity. The authors concluded that post-exercise hypotension may in part be mediated by inhibition of sympathetic nerve activity.

It is interesting to compare these findings to those of Duncan et al.(1985) who exercised a group of 56 mildly hypertensive males for 16 weeks, three times per week for 60 minutes at 70-80% maximum heart rate. They found a significant reduction in systolic and diastolic blood pressure but no changes in catecholamine levels. However, if the group was divided into normoadrenergic and hyperadrenergic according to baseline measurements, the hyperadrenergic had a much greater reduction in systolic blood pressure compared to the normoadrenergic and control groups. The hyperadrenergic also had a significant reduction in both total plasma catecholamine and plasma norephedrine levels. Therefore for the hyperadrenergic group reductions in blood pressure were associated with reductions in plasma catecholamines.

Clearly the sympathetic nervous system plays a major role in the response of blood pressure to acute or chronic exercise, with a large number of studies showing a parallel reduction in blood pressure and sympathetic activity (Duncan et al., 1985; Meredith et al., 1991; Urata et al., 1987; Kiyonaga et al., 1985; Floras et al., 1989).

The non-dog owners in this study had both a reduction in blood pressure and body weight, a relationship between the two that has been found in other studies (Ramsay et al., 1978; Tuck et al., 1983). However, in a meta-analysis of 25 studies Hagberg (1980) reported that there was no correlation between changes in weight and changes in systolic or diastolic blood pressure elicited by endurance training. This would agree with the 10 week cross-over study by Martin et al. (1990) who found that the changes in blood pressure were not associated with any changes in weight or body fat. The studies that have found a correlation between weight loss and reductions in blood pressure have generally used very obese subjects, who were placed on very low calorie diets (Ramsay et al., 1978). This type of regime has been

shown to have major effects on the general metabolism with, for example, a marked reduction in Basal Metabolic Rate (BMR) (Mole, 1990) and probably more significantly large reductions in sympathetic activity measured as a reduction in norephedrine levels (Tuck et al., 1983). Therefore as suggested by Hagberg (1990) the reductions in blood pressure are due to the large disruption to the overall metabolism created by the large negative energy balance experienced rather than any loss of body fat directly.

Although there seems to be a link between obesity and hypertension the relationship is complex and further work is required to establish the relationship between reductions in body fat and reductions in blood pressure.

It would therefore seem that, for hypertensive individuals and possibly only a sub-group of those who are hyperadrenergic, the main effect of aerobic exercise is to reduce the sympathetic activity particularly for the kidney which matches the conclusion of Guyton (1980), that the kidney was central to the control of blood pressure and any changes to lower blood pressure that did not involve the kidney would be futile.

8.4.4. SUMMARY

This study found that 14 weeks of brisk walking was able to reduce systolic blood pressure in a group of middle-aged men (non-dog owners). The lack of changes for another group of middle-men (dog owners) and a group of middle-aged women is most likely due to their low initial blood pressures.

8.4.5. FUTURE RESEARCH

There are clearly many issues to be resolved in the measurement of blood pressure. Further studies using 24-hour ambulatory measurements are needed to confirm the findings of the existing studies and assess the validity of the one-off measurement currently widely used. The concept of a post-exercise reduction in blood pressure and the possibility that several short walks per day could maintain a lower blood pressure warrants further investigation. This is particularly relevant considering the new exercise guidelines for inactive individuals proposed by the American College of Sports Medicine (ACSM, 1993) which advocate that individuals should try to accumulate 30 minutes or more of moderate intensity physical activity over the course of most days of the week.

8.5. AEROBIC FITNESS

8.5.1. SUB MAXIMAL HEART RATES

For both men and women the 14-week brisk walking programme resulted in a significant ($p<0.05$) reduction in heart rate during the first three workloads of the sub-maximal treadmill test. The reductions in heart rate were on average 5 and 7 beats.min^{-1} for the men and women respectively.

This a typical cardiovascular response to an aerobic training programme and is normally used as a marker of an improvement in the function of the cardiovascular system.

8.5.2. SUB MAXIMAL OXYGEN COSTS

It is generally accepted that the relative oxygen cost for a fixed workload is constant and would not change over time. Indeed many prediction tests that do not measure oxygen costs make this assumption in their calculation of $\dot{V}O_2max$. Assuming that there would be no large improvement in walking efficiency, similar oxygen costs would be expected at baseline and retest. In study two this was the case and there was no change in oxygen cost from pre- and

post-test. In contrast in study one there was a significant reduction in the oxygen costs at workloads one to four for the two exercise groups, except for the non-dog owners at workload two and workload four.

This is a finding that is not often reported in the literature, but Taylor (1988) found that after a 30-week marathon training programme middle-aged male subjects had significantly reduced oxygen costs in a sub maximal treadmill walking test. Similarly to this study, Taylor (1988) also found that the reductions in oxygen costs were greatest in those individuals with the highest initial values, which could suggest cardiovascular or biomechanical inefficiencies. It is impossible to discount biomechanical improvements, but as walking is a skill that is fully mastered and regularly practised by most individuals, and the fact that the training mode (running) was different than the testing mode (walking) in the Taylor study, this theory would seem to be discounted.

Another possible explanation of the reduction in oxygen costs could be that subjects had become familiar with the laboratory, testing conditions and walking on the treadmill. Treadmill walking is somewhat different to over-ground walking and novice treadmill walkers may take up to 10 minutes to reach a steady state pattern of walking (Charteris & Taves, 1978; Wall & Charteris, 1980). In a kinematic study Wall & Charteris (1981) have shown that there is an initial rapid (30 seconds) accommodation to treadmill walking followed by a longer more gradual habituation lasting up to 10 minutes. However after five 10 min practice sessions this habituation takes place much more rapidly.

In this study all subjects had an initial familiarisation period of at least five minutes that would mean that the first gas sample would be taken after approximately 10 minutes, therefore it is unlikely that the reductions in oxygen costs were due to treadmill walking familiarisation.

Repeat testing on consecutive days has been shown to reduce anxiety associated with the testing environment and lead to reductions in submaximal heart rates, although this effect diminishes with increasing intensity of the exercise (Shephard, 1969). One study where five

subjects completed an average 8-20 repeat $\dot{V}O_2$max tests over a 2-4 week period there was no test-retest effect with no increase in $\dot{V}O_2$max with test number (Katch et al., 1982). Shephard (1969) concluded that this habituation is lost if the test procedure is not repeated during training. Since the subjects in this study were only exposed to the test procedure on two occasions 14 weeks apart it is unlikely that habituation to the test procedures had any effect on the results.

It is possible that the reductions in oxygen cost were a result of cardiovascular adaptations to the training programme. With a reduced blood pressure (non-dog owners) and a reduced heart rate this would indicate a reduction in the rate pressure product, indicating a reduction in cardiac oxygen consumption. Also the significant reductions in ventilation for both exercise groups during the first three workloads would reduce the ventilatory oxygen cost. Although individually these reductions in oxygen cost would be quite small together they may account for the reduction in the oxygen cost.

The fact that the control group showed no reduction in oxygen costs would confirm that the reductions in the exercise group were a result of the training and not some measurement effect.

There was no similar reduction in oxygen cost for the women, possibly because, as for many of the other variables measured, the exercise programme did not achieve the threshold for cardiovascular adaptation.

8.5.3. PREDICTED $\dot{V}O_2$MAX

8.5.3.1. BASELINE VALUES

The subjects in studies one and two were specifically selected as sedentary, therefore it is not surprising that their baseline $\dot{V}O_2$max values were lower than the equivalent age group from the Allied Dunbar National Fitness Survey who were selected at random from the population. The significantly ($p<0.05$) lower $\dot{V}O_2$max values for the women compared to

men of the same age found in this study (95% CI 7.7, 10.5 $ml.kg^{-1}.min^{-1}$ is a commonly reported finding (Astrand & Rodahl, 1986).

8.5.3.2. Comparison to Similar Studies

In this study only the non-dog owners showed an increase in predicted $\dot{V}O_2max$ after the walking programme. Because the $\dot{V}O_2max$ is predicted from the relationship between heart rate and oxygen cost, the reduction in oxygen costs found for the exercising men had the effect of reducing the increases in $\dot{V}O_2max$ that might have been expected from the reductions in heart rate. It is not possible to determine whether all of the reduction in heart rate was due to a reduced oxygen cost, but if this reduction in oxygen cost is a common occurrence this has implications for measuring the changes in prediction maximum oxygen cost where oxygen cost is in fact not measured.

The women in study two failed to show an increase in predicted $\dot{V}O_2$ max despite having significantly lower heart rates at the three sub maximal workloads. It is possible that this reduction of on average 7 beats was not enough to significantly affect the heart rate / oxygen cost regression in order to give a significant increase in predicted $\dot{V}O_2max$.

Of the walking studies reviewed in Table 9, all studies that included some measure of aerobic power reported a significant improvement. Four of the studies measured actual $\dot{V}O_2max$, these included a total of 45 men and 54 women who walked for 12-24 weeks with an average increase in $\dot{V}O_2max$ of 12%. These studies used a similar exercise prescription to this study therefore similar gains in $\dot{V}O_2max$ would have been expected.

8.5.4. Adaptations to Aerobic Training

The reduction in heart rate at submaximal workloads and an increase in $\dot{V}O_2max$ (non-dog owners) in this study is a typical response to aerobic training. These adaptations are normally attributed to an augmented stroke volume (Saltin et al., 1968; Wolfe et al., 1985) and/or an increased capability of the working skeletal muscles to extract and utilise oxygen. This

training induced bradycardia is also evident at rest, with some highly trained athletes having resting heart rates as low as 30 beats.min^{-1} (Astrand & Rodhal, 1986).

The changes from aerobic training involve a complex series of both central and peripheral adaptations. One of the main central adaptations to aerobic training is the improved performance of the heart. The reduction in the rate pressure product for the non-dog owners would suggest that some training adaptation had taken place in the heart. Like skeletal muscle, the heart is subject to a training effect with increase in size and capillary density. After a 20-week endurance exercise programme maximal oxygen uptake increased significantly in 20 sedentary individuals (Landry et al., 1985). Statistically significant increases in left ventricular diameter, posterior wall and septal thickness, as well as left ventricular end-diastolic volume and left ventricular mass were also observed. In addition to the moderate cardiac hypertrophy there is evidence of angiogenesis, increased coronary blood flow capacity and coronary capillary diffusion capacity (Laughlin & McAllister, 1992). A large number of experiments, based on a variety of methods applied to humans and animals, have failed to demonstrate any significant training effects that can be attributed to enhanced intrinsic contractile state - i.e. an improved quality of the myocardium (Blomqvist & Saltin, 1983). Therefore training seems to cause an increase in the quantity but not the quality of the myocardium.

The importance of peripheral adaptation is demonstrated by the results from the one-legged exercise model (Saltin, 1986). By training two legs individually and then inactivating one leg (placing it on plaster) for four weeks, exercising the inactivated leg results in a significantly higher heart rate and lactate concentration compared to the control leg. This increase in heart rate for a similar cardiac output resulted in a 15 ml reduction in stroke volume. In another series of experiments when only one leg was trained a reduction in heart rate was only found when the trained leg was exercised. In this case the lowering of the heart rate is the primary regulating variable and the enlargement of the stroke volume is due to longer diastolic filling and via the Frank Starling mechanism results in an elevated stroke volume. The mechanism

behind this response is not fully understood but it is believed that both lactate and K^+ are thought to play an important role in adjusting cardiovascular response. This has been confirmed with the one-legged model where increases in heart rate were related to levels of lactate and K^+ (Saltin, 1986).

Other peripheral adaptations of the muscle that could lead to improved performance in a sub-maximal test include increased capillary density, increased mitochondrial volume and increased maximal blood flow through the muscle (Astrand & Rodhal, 1986).

A cross-sectional study using light microscopy reported that the capillary density per mm between trained and untrained individuals was not significantly different. However the average size of the muscle cells were significantly larger in the trained subjects resulting in a larger number of capillaries per muscle fibre (Hermansen & Wachtlova, 1971). A more recent cross-sectional study using quantitative electron microscopy has shown increases both in capillary density and number, in trained individuals compared to untrained. They also found that the difference in capillary supply between fibre types was accompanied by similar differences in mitochondrial content (Ingjer, 1979). Longitudinal studies have confirmed this training adaptation (Coggan et al., 1992). The increased capillary density will allow blood in the trained muscle to maintain a longer transit time and thus achieve a higher oxygen extraction. The increased density will also result in a shorter diffusion distance (Maughan, 1992).

8.5.5. SUMMARY

A 14-week brisk walking programme was able to reduce sub-maximal heart rates in both middle-aged men and women. The walking programme also resulted in a reduction of sub-maximal oxygen costs for the men. Despite reductions in sub-maximal heart rates for all walking groups only the dog owners showed a significant increase in predicted $\dot{V}O_2max$.

8.5.6. FUTURE RESEARCH

One of the key questions raised by this study is what was the cause of the reduction in sub-maximal oxygen costs for the men at test two? Further investigation is required to determine whether this effect is unique to walking, or if it is a more general cardiovascular adaptation to aerobic training. It is not clear whether there is a gender difference.

This study did not find any increase in $\dot{V}O_2max$ for the women, was this because the exercise intensity was too low or the study period was too short.

9. CONCLUSIONS

As a result of 14 weeks of brisk walking, four times per week for 30 minutes, a group of middle-aged sedentary men (non-dog owners) had significant reductions in serum total cholesterol levels, body mass ,submaximal heart rates, resting systolic blood pressure and a significant increase in predicted $\dot{V}O_2max$.

A similar group of middle-aged men who completed the walking programme accompanied by their dog had a significant reduction in submaximal heart rates.

A similar group of middle-aged women who completed the walking programme had a significant reduction in submaximal heart rates.

Analysis of the training diaries suggests that all three groups completed a similar amount of training. However, an estimate of energy expenditure showed that the women expended 25% less energy than the men during the programme, possibly explaining the fewer significant improvements for the women.

The exercise bradycardia found as a result of the walking programme in the three groups is a common adaptation to aerobic training, signifying improved cardiovascular function. This reduction in exercise heart rate is most likely mediated by the sympathetic nervous system.

Both groups of men who completed the walking programme also had significant reductions in submaximal oxygen costs for the same workload of the treadmill test. This reduction in oxygen cost as a result of aerobic training is not a commonly reported finding in other studies. Considering the reduced ventilation and rate pressure product, the reduction in oxygen cost may be accounted for by reductions in cardiac and ventilatory oxygen costs.

The lack of change in resting blood pressure for the dog owners and the women was most likely a result of their low baseline values.

The walking programme failed to increase HDL levels in any of the exercise groups, possibly because the walking programme was below the exercise threshold required for significant changes in HDL.

No change in body composition is possibly due to the relatively small increase in energy expenditure and the limitations of prediction methods to measure small changes in body composition. Subjects may have increased their energy intake reducing or eliminating any negative energy balance. Since the non-dog owners had a significant reduction in body mass but not % body fat there may have been reductions in subcutaneous fat at sites not measured in this study.

Dog ownership would seem to offer no advantage in terms of adherence to or adaptation to a walking programme.

Regular brisk walking would seem to promote some health benefits but the intensity and/or amount of walking required to influence the individual health variables still requires further research.

In Study One the null hypothesis has to be rejected for body mass, resting systolic blood pressure, total cholesterol, sub-maximal heart rates and predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$) for the non-dog owners. For the dog owners the null hypothesis has to be rejected for sub-maximal heart rates.

In Study Two the null hypothesis has to be rejected for sub-maximal heart rates.

It is possible that the results from these two studies were affected by a type II statistical error. The logistical limitations of the sample size may have reduced the statistical power, such that the null hypothesis was wrongly accepted and a "real change" did not reach statistical significance.

APPENDIX A DETAILS OF EQUIPMENT USED IN THE STUDY

Treadmill
Powerjog EG30
Sport Engineering Limited
Stirchley Trading Estate
Hazelwell Road
Birmingham

Clinometer
The Watkin Clinometer was made by;

Ptikin
London

Oxygen Analyser
Sevomex 570A
Servomex Limited
Crowborough
-Sussex

Carbon Dioxide Analyser
P.K. Morgan Limited
Chatham
Kent

Gas Meter
Harvard Dry Gas Meter

Mouthpiece Valve
Hans Rudolph
2700 Series
Large 2-way NRVB
Supplied by;

ECG Recorder
Hewlett Packard 43200A
Hewlett Packard Company
McMininnvlle
Oregon, USA

Sphygmomanometer
Manufactured by
Accoson
England

Skin Abrasive
Omniprep
Omniprep is manufactured by;

D.O. Weaver & Co.
565-C Nucla Way
Aurora, CO
USA

ECG Electrodes
The Blue Sensor disposable electrodes are manufactured by;

Medicotest
Rugmarken 10
DK-3650 Olstykke,
Denmark

Skinfold Caliper
The skinfold caliper was made by;
Holtain Limited
Crosswell
Crymuych
Dyfed
Wales
SA 41

Centrifugal Analyser
The Cobias-bio centrifugal analyser was made by;

Roche Diagnostics
Welnyn Garden City

Heart Rate Recorder
The Sports Tester PE 3000 heart rate recorders were made by;

Polar Electric DY and supplied by;
Hampden Sports Limited
Edinburgh

APPENDIX B EQUATIONS TO ESTIMATE BODY FAT %

Body density Equations (Durnin & Womersley, 1974)

Men

40-49 years old

Density = 1.1620 - 0.0700 X Log sum of skinfolds

50+ years old

Density = 1.1715 - 0.0779 X Log sum of skinfolds

Women

40-49 years old

Density = 1.1333 - 0.0612 X Log sum of skinfolds

50 +

Density = 1.1339 - 0.0645 X Log sum of skinfolds

Siri Equation

% Body Fat = (4.95 / density - 4.50) x 100

PILOT STUDY

Treadmill Protocol Pilot

Before the study three different treadmill protocols were piloted in order to design a suitable protocol for the anticipated subject population.

The aim of the pilot study was to construct a sub maximal protocol that would

1. Encompass the anticipated range of aerobic fitness of the subjects
2. Ensure that all subjects completed at least three workloads
3. Ensure that a steady state had been reached at each workload
4. Ensure that the total test time was not too long
5. Ensure that treadmill speeds would not cause the subject to start to run.

Treadmill Protocol Pilot One

Based on the metabloic equations for treadmill walking (ACSM, 1986) the first protocol was devised to encompass the expected fitness levels of the subject group. The protocol was designed to ensure that all subjects would complete three workloads and that the increase in intensity was uniform between workloads. Gas sampling proceedures were identical to those outlined in the methods section.

This protocol was repeated on subsequent days to evaluate test retest reliability.

Subjects

Eight subjects from the University community in an appropriate age group (48 ± 4.69 years) and fitness level were selected for the pilot.

WORKLOAD	TIME(mins)	SPEED(km/h)	GRADIENT (%)	PREDICTED $\dot{V}O_2$ (ml.kg^{-1}.min^{-1})
1	0 - 5	4	0	10.17
2	5 - 10	5.5	2.5	16.8
3	10 - 15	6	5	22.8
4	15 - 20	6	7.5	27.3
5	20 - 25	6	12.5	36.4
6	25 - 30	6	15	41

Table 78.Treadmill Walking Protocol Pilot Study One. Predicted $\dot{V}O_2$max. is based on the equation from the American College of Sports Medicine (ACSM, 1986)

Results

Actual oxygen cost values were slightly different from predicted values, seeming to underpredict the value at lower workloads and over predict at higher workloads. From the measured oxygen costs it was evident that the increases between workloads were not uniform therefore further modifications were deemed necessary.

Workload	Actual Oxygen Cost ($ml.kg^{-1}.min^{-1}$)	Predicted Oxygen Cost ($ml.kg^{-1}.min^{-1}$)	Difference
1	12.36 ± 1.41	10.17	+2.19
2	19.08 ± 1.82	16.8	+2.28
3	24.49 ± 2.12	22.8	+1.99
4	27.61 ± 2.58	27.3	+0.61
5	35.31 ± 2.68	36.4	-0.69
6	38.53 ± 0.64	41	-1.96

Table 79. Oxygen costs pilot treadmill protocol one

A paired t-test of the test retest values for predicted $\dot{V}O_2$ max revealed that there was no significant difference in the two values.

Subject	Predicted $\dot{V}O_2$max ($ml.kg^{-1}.min^{-1}$) Test 1	Predicted $\dot{V}O_2$max ($ml.kg^{-1}.min^{-1}$) Test 2	Difference
1	51.87	47.37	-4.5
2	27.86	32.29	+4.43
3	36.91	39.50	+2.59
4	47.15	42.01	-5.14
5	42.25	43.14	+0.89
6	35.56	37.51	+1.95
7	37.15	42.94	+5.79
8	48.19	45.16	-3.74
Mean $\pm$ SD	40.96 $\pm$ 8.05	41.24 + 4.74	

Table 80. Predicted $\dot{V}O_2$ max values pilot study one.

Treadmill Protocol Pilot Two

The treadmill protocol was modified (Table 81)to make the increases between the workloads more uniform and was piloted on a different younger (32 $\pm$ 15 years) group of volunteers from the University community.

WORKLOAD	TIME(mins)	SPEED(km/h)	GRADIENT (%)	PREDICTED $\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)
1	0 - 5	4	0	10.17
2	5 - 10	5.3	2.5	16.3
3	10 - 15	6	5	22.8
4	15 - 20	6	8.5	28.8
5	20 - 25	6	12.5	35.1
6	25 - 30	6	15.5	41.4

Table 81. Treadmill protocol pilot study two

Results

The measured oxygen cost demonstrated a similar pattern of difference in actual and expected values as was found in pilot study one. The modifications to the protocol had helped to make the differences between workloads more uniform, but it was still felt that the increase in oxygen cost from workload two to workload three was too large. It was also considered that the actual value for workload may be slightly high and that the most unfit subjects would exceed 85% of maximum heart rate at this oxygen cost.

Workload	Actual Oxygen Cost ($ml.kg^{-1}.min^{-1}$)	Expected Oxygen Cost ($ml.kg^{-1}.min^{-1}$)	Difference
1	13.09 ± 1.34	10.17	+2.92
2	18.50 ± 0.92	16.3	+2.2
3	24.62 ± 1.18	22.8	+2.12
4	29.01 ± 1.85	28.8	+0.21
5	34.7 ± 1.79	35.1	-0.4
6	40.25 ± 2.01	41.4	-1.15

Table 82. Oxygen costs pilot treadmill protocol two

Again a paired t-test revealed that there was no significant test-retest difference in predicted $\dot{V}O_2max$.

Subject	Predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$) Test1	Predicted $\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$) Test 2	Difference
1	55.74	48.08	-7.66
2	58.76	52.22	-6.54
3	53.87	51.13	-2.74
4	49.42	49.22	-0.20
5	53.74	56.88	+3.14
6	40.58	42.15	+1.57
Mean ± SD	52.02 ± 6.38	49.95 ± 4.89	

Table 83. Predicted $\dot{V}O_2max$ values pilot study two

Treadmill Protocol Pilot Three

The protocol was further modified to lower the oxygen cost of workload three and to have uniform increases for each workload. A protocol was designed that in theory would have an increase of 5 $ml.kg^{-1}.min^{-1}$. This was piloted on another group of volunteers from the University community (aged 40 $\pm$ 3 years). Only one test was performed as the previous repeat tests had shown good reliability.

WORKLOAD	TIME(mins)	SPEED(km/h)	GRADIENT (%)	PREDICTED $\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)
1	0 - 5	4	0	10.17
2	5 - 10	4.8	2.5	15.9
3	10 - 15	5.3	5	20.25
4	15 - 20	6	6.5	25.2
5	20 - 25	6	9.5	30.6
6	25 - 30	6	12	35.1

Table 84. Treadmill protocol pilot study three

Results

Workload	Actual Oxygen Cost ($ml.kg^{-1}.min^{-1}$)	Expected Oxygen Cost ($ml.kg^{-1}.min^{-1}$)	Difference
1	13.95 ± 1.85	10.17	+3.78
2	16.99 ± 0.43	15.9	+1.09
3	22.47 ± 1.48	20.25	+2.22
4	28.42 ± 0.72	25.2	+3.22
5	32.90 ± 1.11	30.6	+2.3
6	36.44 ± 0.86	35.1	+1.34

Table 85. Oxygen costs pilot treadmill protocol three

Subject	Predicted $\dot{V}O_2$ max
1	49.36
2	55.93
3	64.16
4	48.01

Table 86. Predicted $\dot{V}O_2$ values pilot study three

These measured oxygen costs seemed to give the appropriate oxygen costs for the expected subject group and thus protocol three was used in the main study.

The pilot studies had also shown that the test proceedures were repeatable and reliable.

APPENDIX D CALIBRATION PROCEDURES

Treadmill Calibration

To ensure accuracy of the set workload the treadmill was regularly checked by calibration for both speed and gradient by the following methods

Speed

The speed of the treadmill belt was checked at 4, 5 and 6 $km.h^{-1}$ as this encompassed the range of speeds used in the treadmill protocol. The treadmill was started at the selected speed and upon starting a stopwatch a measuring wheel was dropped on to the treadmill belt and held in position, to allow measurement of the distance covered by the belt. After exactly three minutes the measuring wheel was lifted off the treadmill belt and a note made of the distance travelled. This procedure was repeated at the other two speeds. The distance covered in the time was then used to calculate the treadmill speed. Table 87 shows the measurements taken over the study period.

Treadmill Speedometer reading	4 $km.h^{-1}$	5 $km.h^{-1}$	6 $km.h^{-1}$
Date	Actual Speeds $km.h^{-1}$		
19/02/91	3.93	4.95	5.9
05/03/91	3.9	4,93	5.91
09/04/91	3.97	4.96	5.98
14/05/91	3.99	4.94	5.92
28/05/91	3.98	4.98	5.98
11/06/91	3.99	4.98	5.96
26/06/91	4.02	4.94	5.94
19/07/91	3.98	4.99	5.96
19/08/91	3.99	5.01	5.96
30/01/91	3.97	5.01	5.98
13/09/91	3.99	4.97	5.96
26/09/91	3.99	5.02	5.99
14/10/91	3.98	4.97	6.01
13/11/91	4	5.01	5.97
04/12/91	4.01	5	6
06/09/93	3.99	5	6
09/09/93	3.98	4.98	5.98
29/09/93	4.01	5.01	5.98

Table 87. Treadmill speed calibration values

As can bee seen from Table 87 the treadmill speedometer was accurate and there were no alterations in this accuracy over time.

Treadmill Gradient

The actual gradient of the treadmill was measured using a 'clinometer' (Watkin, London). A one meter piece of flat steel was placed accurately in the middle of the belt of the treadmill to even out small inconsistencies in the rough surface of the treadmill belt. The clinometer was then placed on top of the piece of steel and measurements taken of actual gradient at treadmill gradient settings of 0, 2.5, 5, 6.5, 9.5, 12 and 15%.

Treadmill Gradient (%)	0	2.5	5	6.5	9.5	12	15
Date	Actual	Gradient	(%)				
19/02/91	0.44	2.79	4.54	5.76	8.46	10.45	13.35
05/03/91	0.35	2.41	4.5	5.76	8.23	10.37	12.85
09/04/91	0.31	2.41	4.47	5.76	8.28	10.54	13.19
14/05/91	0.45	2.85	4.55	6.07	8.89	11.18	13.74
28/05/91	0.47	2.48	4.59	5.89	8.49	10.418	13.278
11/06/91	0.47	2.53	4.54	5.72	8.33	10.25	13.19
26/06/91	0.44	2.58	4.59	5.97	8.33	10.37	12.67
19/07/91	0.35	2.57	4.85	6.07	8.8	10.84	13.57
19/08/91	0.4	2.65	4.66	5.81	8.37	10.8	13.02
30/01/91	0.47	2.5	4.62	5.84	8.33	10.42	12.91
13/09/91	0.49	2.67	4.89	6.23	9.06	11.06	13.62
26/09/91	0.35	2.5	4.62	5.79	8.28	10.51	13.26
14/10/91	0.38	2.58	4.76	5.98	8.72	10.75	13.54
13/11/91	0.35	2.67	4.85	6.23	8.8	10.89	13.45
04/12/91	0.49	2.67	4.7	5.9	8.42	10.54	13.19
06/09/93	0.58	2.92	5.01	6.24	8.89	10.99	14
09/09/93	0.58	2.76	5.1	6.5	9.7	11.25	14.11
29/09/93	0.65	2.62	4.49	6.08	8.31	10.42	13.08
07/10/93	0.61	2.71	4.68	6.03	8.54	10.74	13.38

Table 88. Treadmill gradient calibration values

Table 88 shows that the there was an appreciable difference between the treadmill gradient and the actual gradient especially at the higher values, but the actual values were consistent during the period of the study.

Gas Analysis Calibration

Gas analysers were calibrated against known standard test gasses prior to all tests by the following procedure.

Firstly pure nitrogen (zero gas) was passed through both analysers in order to set a zero value.

A test gas (16% O_2, 4.14% CO_2) was then passed through the analysers and the span was adjusted to give correct readings on both analysers.

Pure nitrogen was again sampled and zero adjustments were made if necessary.

This procedure was repeated until both zero and test gas gave correct readings. At this point a second test gas (15.4% O_2, 6.21% CO_2) was measured. If the analysers failed to give the correct readings for this test gas the whole calibration procedure was started from the beginning. When the analysers could correctly measure the two test gases and the zero gas, the calibration procedure was compete.

APPENDIX E CALCULATION OF OXYGEN COSTS

Vapour Pressure for H_2O (P_{H_2O})

Gas temperature * 0.03783 + 6.25012 + 0.0264 * Gas temperature^2

Ventilation STP (Ve STP)

Ventilation (atmospheric pressure - P_{H_2O}) / (760 * (1 + 0.00367 * Gas temperature))

True O_2

(100 - O_2 conc. - CO_2 conc.) * 0.265 - O_2 conc.

True CO_2

CO_2 conc. - 0.03

RQ

True CO_2 / True O_2

Oxygen Cost ($l.min^{-1}$)

(Ve(STP) * True O_2) / 100

APPENDIX F TRAINING DIARY

Week No

Name

Target Heart rate

Walk No.	Date	Time	Resting Heart Rate	Wakling Heart Rate	Duration	Comments
1						
2						
3						
4						
other						
other						

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