# THE EFFECTS OF DIFFERENT PATTERNS OF BRISK WALKING ON ASPECTS OF FITNESS, CARDIOVASCULAR RISK AND PSYCHOLOGICAL WELL-BEING

by

Marie Harriet Murphy

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## ABSTRACT

The use of a pattern of accumulated bouts of exercise has become enshrined in recent public health recommendations. Although there is epidemiological evidence to support this, the experimental evidence for such recommendations is limited. The four studies described in this thesis investigated the effects of different patterns of exercise on fitness, cardiovascular risk and selected psychological parameters.

Two training studies were undertaken to compare the effects of brisk walking performed in either, one continuous 30 minute bout or three, 10 minute bouts spread throughout the day. In one study 34 sedentary women (aged 31-57 y) were randomly assigned to a programme of brisk walking (totalling 30 $min \cdot d^{-1}$, 5 $d \cdot wk^{-1}$) performed in either one continuous bout (LONG) or three shorter bouts spread throughout the day (SHORT) or a no-exercise (CON) group for 10 weeks. Endurance fitness, (measured by $\dot{V}O_2$ max and heart and blood lactate response to a submaximal treadmill test) improved in response to both programmes with no differences between SHORT and LONG groups. Both training groups exhibited decreases in body mass, sum of 4 skinfolds and waist and hip circumference compared to controls but there were no differences in the magnitude of these changes between the two patterns of walking. Both walking programmes resulted in a decrease in resting systolic blood pressure with no difference in the magnitude of this reduction between programmes. The second training study compared the effects of similar programmes of brisk walking on physiological, physical and psychological variables in a randomly assigned cross-over trial. In addition to confirming changes in endurance fitness, body composition and blood pressure found in the previous study, the results indicated that a six-week programme, of either pattern of brisk walking, was sufficient to increase fasting HDL cholesterol and reduce fasting triacylglycerol and total cholesterol concentrations among 21 previously sedentary men and women (aged 35-55 y). Adherence of 88% and 91% for SHORT and LONG programmes respectively did not differ between programmes. Both patterns of walking resulted in a decrease in tension/anxiety, measured by an abbreviated Profile of Mood States, while the perceived barriers to exercise of 'effort' and 'health' were reduced after participation in SHORT and LONG programmes respectively. Although both programmes tended to increase self-efficacy for both walking and other activity and improve post-exercise feelings, these differences were not significant. The results of these studies suggest that short accumulated bouts of brisk walking are as effective as

long bouts in improving fitness, reducing cardiovascular risk, by decreasing resting blood pressure and body fatness and altering fasting blood lipid profiles, and changing aspects of mood and perceived barriers to activity.

Plasma HDL cholesterol concentrations may be determined, in part, by the rate of degradation of triacylglycerol rich lipoproteins. One of the established benefits of exercise is the reduction in postprandial lipaemia in the period following prolonged exercise. Two studies were undertaken to consider whether splitting continuous moderate intensity exercise (60% of $\dot{V}O_2$ max) into shorter intermittent bouts, taken over the course of the day, alters this effect. In the first study, 9 normolipidaemic and 3 borderline hyperlipidaemic men (aged 18-54) performed three trials each over two days. On day 1 subjects completed either one 90 minute bout (LONG) or three 30 minute bouts (SHORT) of treadmill running spread over the course of the day or no exercise (CON). On day 2 subjects consumed a high-fat breakfast and then rested for 6 hours. Postprandial lipaemic response to the high fat meal was reduced in both SHORT and LONG trials compared to CON trial with no differences between exercise trials. The findings of this study suggest that splitting a continuous exercise bout into three smaller bouts spread throughout the day does not alter its effectiveness in reducing postprandial lipaemia during the extended recovery period, 15-21 hours after the last bout of exercise. The fourth study compared the effect of one continuous bout of 30 minutes of brisk walking (LONG) performed just before breakfast with a three 10-minute bouts, performed before breakfast, lunch and dinner (SHORT) on postprandial lipaemic responses to typical meals among 10 sedentary men and women (aged 34-66). Postprandial lipaemia was attenuated in both SHORT and LONG trials compared to CON with no difference between trials in the extent of this reduction. The findings suggest that 30 minutes of brisk walking, performed in a continuous or discontinuous pattern, reduces postprandial lipaemic response in a similar manner. This finding is consistent with the HDL cholesterol alterations noted in the training study.

The results of the studies described in the thesis suggest that short bouts of brisk walking, accumulated over the course of a day, are as effective as one continuous bout of equal total duration in reducing aspects of cardiovascular risk among previously sedentary individuals.

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## DEDICATION

*-To Juana Lux Carillo and Patricia Tzoc Granados*
*whose selflessness has allowed me to fulfil a lifelong dream.*

## PUBLICATIONS AND PRESENTATIONS

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# CHAPTER 1

## INTRODUCTION

> "All parts of the body which have a function, if used in moderation and exercised in labours in which each is accustomed, become thereby healthy, well-developed, and age more slowly, but if unused and left idle, they become liable to disease, defective in growth and age quickly."
>
> Hippocrates (460-370 BC)
> (cited in MacAuley, 1994 #179)

The association between physical activity and health has been established for over two centuries. In earlier times Man's lifestyle was characterised by high levels of physical activity, with survival depending largely on the ability to hunt and gather food (Blair et al. 1992). During the two thousand years since Hippocrates made this observation, there have been wholesale alterations in the everyday lifestyles of human beings. In the last century in particular, automation, the exploitation of alternate power sources such as steam and electricity and technological advances have resulted in lifestyles which bear little resemblance to our early ancestors. As we reach the end of the second millennium, mankind relies less on physical activity for survival than ever before. The human body, however, has not adapted to these changing needs so readily. It has been argued that our genetic constitution and physical composition is still designed for activity (Eaton et al. 1988). In parallel with this technological development have come advances in medicine which have all but eradicated many of the infectious diseases responsible for death 200 years ago and Man's typical lifespan has increased dramatically (Astrand 1992). However, the decline in physical activity has been accompanied by a steady rise in chronic degenerative diseases such as coronary heart disease, stroke, obesity and hypertension (Blair et al. 1992) and in recent times the associations between physical activity and health first made by Hippocrates have been substantiated.

During the latter half of the 20$^{th}$ century scientific evidence linking regular physical activity to health and longevity has amassed (Haskell 1996). Beginning with the pineering work of Morris in the 1950s, a host of cross-sectional studies have

documented the relationship between the risk of coronary heart disease (CHD) and level of physical. Morris and colleagues (1953) showed that bus conductors were significantly less likely to suffer from a myocardial infarction than their driver colleagues and that differences in their occupational activity levels were a major factor in this reduced risk (Morris et al. 1953). This inverse relationship between physical activity and risk of CHD has subsequently been confirmed in a multitude of epidemiological studies. Powell and colleagues, having reviewed the literature concluded that inactivity carries with it a relative risk of CHD which is equivalent to that associated with hypertension, hypercholesterolaemia and smoking (Powell et al. 1987).

While the association between physical activity and CHD risk heralds back to the 1950s it was over two decades later that the first guidelines on exercise for healthy adults were published by the American College of Sports Medicine (ACSM) (Pollock 1978). These guidelines made recommendations for developing and maintaining cardiorespiratory fitness that became the dictum of physical educationalists and fitness and health professionals for more than two decades. The guidelines recommended that individuals should exercise 3-5 $d \cdot wk^{-1}$ at an intensity of 50-85% of maximum oxygen uptake for 15-60 minutes per session using some type of rhythmical activity employing large muscle groups. These guidelines were the result of a consensus of the findings from a multitude of experimental studies where fitness was measured before and after a variety of exercise interventions (Haskell 1994a). These recommendations remained the cornerstone of exercise prescription from 1978 until 1990. In the 1990s, the ACSM issued further guidelines based on an evolving body of scientific literature and for the first time began to distinguish the type of activity required to improve fitness from a somewhat more modest intensity thought to improve health. In terms of enhancing cardiovascular fitness however, the 1990 position stand did not drastically alter its 1978 predecessor. The statement recommended 20-60 minutes at 50-85% of maximum oxygen uptake on 3-5 $d \cdot wk^{-1}$ in order to improve fitness and promised future guidance on the level required to gain health benefits alone (ACSM 1990).

There is now fairly widespread public acceptance that exercise is beneficial to health (Phillips et al. 1996). Despite this understanding and the existence of such clear physical

activity recommendations from authoritative agencies, several population fitness surveys in the 1990s have illustrated that a significant proportion of individuals in the UK (Allied Dunbar 1992; MacAuley et al. 1994) the USA (Pate et al. 1995) Canada (Stephens and Craig 1990) and probably most of the developed world do not take sufficient physical activity to gain health benefits. One of the major barriers cited by individuals for such inactivity is a 'lack of time'. In a world where many people live by rigid daily time schedules, finding time to incorporate 20-60 minutes of exercise is perceived, by many, as impracticable. Although the validity of such perceptions may be questionable, if greater proportions of the population are to be encouraged to engage in regular physical activity, such perceived barriers need to be overcome.

Having reassessed the ever-increasing body of literature investigating the optimal amount of physical activity required to enhance fitness and health, the ACSM published further position stands in 1993 (ACSM 1993) and more recently in 1998 (Pollock et al. 1998). The recent guidelines go some way towards addressing this 'lack of time' barrier by suggesting that every adult accumulate 30 minutes or more moderate intensity physical activity on most days of the week (ACSM 1993; Pollock et al. 1998). The statements recommend that this 30 minutes be accumulated from bouts which are a minimum of 10 minutes in duration (Pollock et al. 1998). The accumulated approach to physical activity is now enshrined in the recent consensus statements by Centres for Disease Control and Prevention, the National Institute of Health and other expert bodies in the United States (Pate et al. 1995; National Institute of Health and Panel 1996; Prevention 1996). These consensus statements provide the basis upon which the UK and many other countries have modelled their recommendations.

The recommendation for an accumulation of physical activity is based largely on indirect evidence from epidemiological studies which have included activities such as stairclimbing, gardening and walking for personal transport, all likely to have been performed in short sessions rather than continuous bouts (Paffenbarger et al. 1986). While this new feature of the recommendations provides great potential, at least intuitively, for incorporating physical activity into one's lifestyle, the empirical evidence upon which such suggestions are based is far from abundant. When the research which is described in this thesis began, there were only two published studies considering the effect of accumulated bouts of physical activity spread throughout the

day (Ebisu 1985; DeBusk et al. 1990). Since then, a small but significant number of studies have confirmed the potential of such accumulated exercise to improve fitness, decrease fatness and enhance adherence of a variety of sedentary subjects (Jakicic et al 1995; Snyder et al. 1997; Woolf-May et al. 1998). The question which remains is not whether short bouts of exercise can enhance fitness and health, but how do the effects of such exercise compare with the effects of more traditional continuous approaches, in terms of fitness, health and psychological benefit? Two associated questions are also outstanding. Firstly what are the mechanisms underlying the effect of such accumulated activity on the reduction in cardiovascular risk? Secondly, are individuals more or less likely to maintain exercise if performed in such a discontinuous pattern? The comparability of different patterns of exercise on cardiovascular risk and psychological health are the aspects of the new physical activity guidelines that are least well supported by existing scientific evidence. This thesis hopes to make some contribution to this body of evidence.

In the initial exercise guidelines issued by the ACSM, the mode of exercise recommended is continuous rhythmical activity which employs large muscle groups (Pollock 1978; ACSM 1990). Although traditionally, exercise intervention studies used cycling or jogging as the main exercise modes, more recent studies have investigated the effects of brisk walking. The National Fitness Survey (England) and the Northern Ireland Health and Activity Survey revealed that walking was one of the most popular physical activities undertaken. Over 50% of the English population surveyed reported walking continuously for at least one mile (Allied Dunbar 1992) while over 39% of the Northern Irish population walked continuously for at least 2 miles (MacAuley et al. 1994) during the previous week. The physiology of and benefits associated with brisk walking have been recently reviewed by Morris and Hardman (Morris and Hardman 1997). Walking is an eminently suitable form of activity for the sedentary population at whom the current guidelines are aimed. It is low impact and therefore carries lower risk of injury than jogging (Pollock et al. 1991), it requires no special equipment or facilities and no particular skill. Walking is socially acceptable, can be maintained throughout the lifespan and is, to some extent, independent of weather conditions. Perhaps most importantly however, walking for exercise, can be easily incorporated into the daily routine and is therefore ideally suited to an accumulated approach to physical activity. It is somewhat ironic that prior to the technological age referred to earlier, walking was

the primary source of human locomotion. It is perhaps fitting therefore, that in attempting to reinstate physical activity into increasingly sedentary lifestyles, we should return to this versatile mode of physical activity.

The purpose of the studies reported in this thesis is to compare both the short and medium term effects of accumulated bouts of exercise with the effects of continuous exercise of a similar total duration. A series of four studies were carried out in order to make this comparison. Two studies (Chapters 4 and 5) compared the medium to long-term effects of three short (10 min) brisk walks spread over the course of the day with one longer (30 min) walk, on aspects of fitness, health and psychological status, among healthy but sedentary individuals. The two remaining studies (Chapters 6 and 7) compared the short-term effects of accumulated and continuous patterns of physical activity on a specific cardiovascular risk factor, namely postprandial lipaemia.

# CHAPTER 2

# REVIEW OF LITERATURE

## 2.1 Introduction to and scope of the review

The central concern of this thesis is comparing the consequences of accumulating moderate intensity physical activity, performed in short bouts over the course of the day, with the effects of a more traditional approach involving one continuous daily bout of activity. Therefore the overall aim of this chapter is to provide an overview of the scientific literature concerning the effects of accumulated physical activity on aspects fitness, health and psychological function.

The initial recommendations for an accumulated approach to physical (ACSM 1993) cited only two intervention studies to support the notion that short bouts of exercise, dispersed throughout the day, was an effective method for enhancing physiological function (Ebisu 1985; DeBusk et al. 1990). Both of these studies involved short and long bouts of jogging. When selecting an activity that lends itself to short bouts accumulated over the course of a day, jogging is not an obvious choice. Moreover, jogging is also less suitable for the sedentary population at whom the accumulated approach to physical activity is aimed. Cycling, stairclimbing and walking are modes of physical activity that are more suitable for use in short bouts and are more likely to fit easily into a typical daily lifestyle lifestyle. Of these, brisk walking is undoubtedly the most accessible for the largest majority of the sedentary population. Brisk walking does not require special equipment or a change of clothing, it can be performed, by a sedentary population, with minimum injury risk and it is widely socially acceptable. Moreover, in the course of a day, walking is probably the most pervasive mode of personal transport. For these reasons, brisk walking has been chosen as the mode of exercise in most of the studies reported in this thesis. Since the initial recommendations for an accumulated pattern of physical activity were first made, a small number of studies have considered the effect of a pattern of short bouts of accumulated activity (Jakicic et al. 1995; Snyder et al. 1997; Woolf-May 1998) and an even less structured pattern involving opportunistic or 'lifestyle' activity (Dunn et al. 1997; Andersen et al. 1999; Dunn et al. 1999) on fitness and health-related parameters. While these studies

will be a central focus of this review, all of the studies that have examined the effects of brisk walking, performed in any pattern, will be considered.

The first part of the review considers the effects of moderate intensity brisk walking on fitness. The second part of the review considers the effect of brisk walking on selected health-related measures. Given that one of the major health benefits of physical activity, is its effects on lipoprotein metabolism, the third section of the review deals more closely with this particular health benefit. The fourth and final part of the review focuses on the psychological benefits of brisk walking. The review is intended to establish the need for and provide a conceptual background to the four studies presented in the experimental chapters.

## 2.2 Brisk walking as a form of exercise

Walking is the most popular physical activity among the EU population (Lappalainen 1998). Its appeal lies in the fact that is acceptable and accessible to all (Siegel et al. 1995), requires no skill, can be performed alone or with others and has little risk of causing injury (Pollock et al. 1991). As a primary form of human locomotion walking has long phylogenetic history (Jabolinski and Chaplin 1993). When walking purely for locomotion, the pace at which an individual walks has been linked to their gender, age and socio-economic status (Schmitt and Atzwanger 1995) as well as their environment (Bornstein 1979). When walking for exercise, healthy middle-aged men and women typically self-select a speed of approximately 1.8 $m \cdot s^{-1}$. (Spelman et al. 1993). Despite the centrality of walking to everyday life, it is only relatively recently that the physiological benefits of regular walking have been specifically investigated. In recent years comprehensive reviews of the effects of brisk walking have been produced (Davison and Grant 1993; Morris and Hardman 1997)

## 2.3 The effects of brisk walking on endurance fitness and metabolism

### 2.3.1 Overview

The influence of exercise on endurance fitness has been the topic of a considerable volume of literature dating back to the middle of the 19$^{th}$ century. Early research

investigating the effects of exercise on endurance fitness focused primarily on vigorous jogging or cycling activity. Until the 1970's little qualitative evidence on the effects of brisk walking existed and it is only very recently that the effects of an intermittent pattern of brisk walking have been considered. In this section, all of the studies that have considered the effects of moderate intensity (50-70% of $\dot{V}O_2$ max) brisk walking on endurance fitness will be reviewed.

Since 1971, to the author's knowledge, 30 published studies have investigated the effect of brisk walking on the endurance fitness of healthy, adult, men and women. All but three of these studies have reported improvements in endurance fitness following a programme of brisk walking. These studies are summarised in Table 2.1.

The term 'endurance fitness' as it is used in this thesis, refers to the ability of the body to perform vigorous muscular work, which demands a high proportion of $\dot{V}O_2$ max for an extended period. Muscular work requires the body to utilise oxygen in the resynthesis of adenosine triphosphate, which has been hydrolysed to create the energy required for muscular contraction. The higher the endurance fitness of an individual, the greater the degree to which the energy demands of a given level of muscular contraction can be met through oxidative metabolism. As endurance fitness increases two aspects of this ability to use oxygen may increase. Firstly, the absolute maximum amount of oxygen that the individual is capable of utilising ($\dot{V}O_2$ max) may increase. Secondly the relative proportion of this $\dot{V}O_2$ max that an individual is able to sustain while exercising without having to rely to a great extent on anaerobic energy sources may increase. Both the absolute and relative alterations have been used as measures of endurance fitness in the studies of the effects of brisk walking reviewed in this section. Maximal oxygen uptake ($\dot{V}O_2$ max), heart rate response to exercise and blood lactate concentration in response to exercise are the three main methods by which endurance fitness has been quantified in these studies.

| Reference | Subjects | | | Brisk Walking Programme | | | | Change in Fitness |
|---|---|---|---|---|---|---|---|---|
| | Sex | Age | N | Intensity | Duration min | Frequency $d \cdot wk^{-1}$ | Intervention wk | |
| Pollock et al (1971) | M | 48.9 | 24 | 63-76% of max heart rate | 40 | 4 | 20 | 26.1% increase in $\dot{V}O_2$ max |
| Kilbom (1971) | F | 19-45 | 10 | 60% of $\dot{V}O_2$ max | 30 | 3 | 6 | no change in $\dot{V}O_2$ max<br>decrease in heart rate response* |
| Luria and Koepke (1975) | M<br>F | 22-55 | 8<br>5 | >70% of max heart rate | 30 | 5 | 10 | 15.7% increase in $PWC_{150}$ |
| Leon et al (1979) | M | 19-31 | 6 | 1.5-3.2 mph | 15-90 | 5 | 16 | 18.2% increase in predicted $\dot{V}O_2$ max |
| Seals et al (1984) | M<br>F | 61-67 | 21 | 40% of heart rate reserve | 20-30 | 3 | 26 | 11% increase in $\dot{V}O_2$ max |
| Juneau et al (1987) | M<br>F | 49<br>47 | 60<br>60 | 65-77% of peak heart rate | 47<br>54 | 5 | 26 | 15% increase in $\dot{V}O_2$ max<br>9% increase in $\dot{V}O_2$ max |
| Santiago et al (1987) | F | 30 | 17 | 70-75% of max heart rate | 55 | 4 | 20 | 20.6% increase in $\dot{V}O_2$ max |
| Jetté et al (1988) | M<br>F | 35-53 | 14<br>12 | 60% of $\dot{V}O_2$ max | 30 | 3 | 12 | 9.7% increase in $\dot{V}O_2$ max<br>17.3% increase in $\dot{V}O_2$ max |
| Hagberg (1989) | M<br>F | 64 | 33 | 50% of $\dot{V}O_2$ max | 60 | 3 | 36 | no change in $\dot{V}O_2$ max<br>decrease in heart rate response* |
| Pollock et al (1991) | M<br>F | 70-79 | 25<br>32 | 70-85% of heart rate max reserve | 30-45 | 3 | 26 | 20.4% increase in $\dot{V}O_2$ max |
| Bergman and Boyungs (1991) | F | 40-82 | 27 | 75-80% max heart rate | 20-40 | 4 | 10 | 6.3% decrease in heart rate response * |
| Duncan et al (1991) | F | 20-40 | 59 | 4.8, 6.4 or 8.0 $km.h^{-1}$ | 36-60 | 5 | 24 | 4.4, 9.3 and 16.3% increase in $\dot{V}O_2$ max |
| Whitehurst and Menendez (1991) | F | 61-81 | 31 | 70-80% max heart rate | 20-40 | 3 | 8 | 8% decrease in 1 mile walk time |
| Cononie et al (1991) | M<br>F | 70-79 | 25<br>31 | 50% of $\dot{V}O_2$ max | 20-45 | 3 | 26 | 20% increase in $\dot{V}O_2$ max |
| Davison et al (1992) | M | 40-60 | 46 | 70-75% max heart rate | 30 | 4 | 14 | Decrease in heart rate response * |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Hardman et al (1992) | F | 30-61 | 54 | 70 % max heart rate | 20-57 | ≥3 | 52 | 16% increase in oxygen uptake at 2mmol·l$^{-1}$ blood lactate |
| Stensel et al (1993) | M | 42-59 | 66 | 68 % max heart rate | 20-45 | 2-3 | 52 | 14.9% increase in oxygen uptake at 2mmol·l$^{-1}$ blood lactate |
| Hinkleman and Nieman (1993) | F | 25-45 | 32 | 60% of heart rate reserve | 45 | 5 | 15 | No change in $\dot{V}O_2$ max<br>Decrease in heart rate response* |
| Kingwell et al (1993) | M<br>F | 22-58 | 7<br>7 | 50% of maximum heart rate | 60 | 5 | 4 | 8.3% increase in $\dot{V}O_2$ max |
| Hardman and Hudson (1994) | F | 44 | 20 | 72% of predicted max heart rate | 20-105 | ≥3 | 12 | 30 % increase in oxygen uptake at 2mmol·l$^{-1}$ blood lactate |
| Suter et al (1994) | M | 41 | 47 | 50% of $\dot{V}O_2$ max | 30 | 6 | 26 | 7.1% increase in $\dot{V}O_2$ max |
| Davison et al (1995) | M | 40-60 | 107 | 70-75% of predicted max heart rate | 30 | 4 | 14 | Decrease in heart rate response* |
| Jakicic et al (1995) | F | 25-50 | 56 | 70% of heart rate reserve | 20-40 | 5 | 20 | 5-6% increase in predicted $\dot{V}O_2$ peak |
| Santiago et al (1995) | F | 22-40 | 27 | 72% maximal heart rate | 49-56 | 4 | 40 | 22% increase in $\dot{V}O_2$ max by week 20, no further increase |
| Ready et al (1996) | F | 61.3 | 56 | 60% of $\dot{V}O_2$ peak | 60 | 3<br>5 | 24 | 12.1% increase in $\dot{V}O_2$ peak<br>13.9% increase in $\dot{V}O_2$ peak |
| MacRae et al (1996) | M&F | 90 | 31 | Self-selected pace | 11-20 | 5 | 12 | 92% increase in maximum walk distance |
| Snyder et al (1997) | F | 43 | 13 | 50-65% of heart rate reserve | 30 | 5 | 32 | No change in $VO_2$ peak |
| Kukkonen-Harjula et al (1998) | M<br>F | 41.2 | 55<br>61 | 65-75% of $\dot{V}O_2$ max | 50 | 4 | 15 | 14% increase in $\dot{V}O_2$ max |
| Woolf-May et al (1998) | M&F | 40-71 | 49 | 68% of predicted $\dot{V}O_2$ max | 10-45 | 2-5 | 18 | 3-4.5% decrease in heart rate response * |
| Asikainen et al (1998) | F | 48-63 | 133 | 65% of $\dot{V}O_2$ max | 20-60 | 5 | 52 | 8-11.5% increase in $\dot{V}O_2$ max |

* = heart rate response to a submaximal exercise test

**Table 2.1** Summary of studies examining the effect of brisk walking on endurance fitness

### 2.3.2 Effects of brisk walking on $\dot{V}O_2$ max

$\dot{V}O_2$ max is the maximal volume of oxygen which an individual is capable of utilising per unit time. The use of $\dot{V}O_2$ max as a measure of endurance fitness has been widely reported in the literature and has been the main outcome measure of many of the studies which have considered the effects of brisk walking on endurance fitness (Pollock et al. 1971; Johannessen et al. 1986; Juneau et al. 1987; Santiago et al. 1987; Jetté et al. 1988; Pollock et al. 1991; Ready et al. 1996; Asikainen et al. 1998; Kukkonen-Harjula et al. 1998). In addition to direct assessment by the analysis of expired air during an incremental walking test to volitional fatigue, $\dot{V}O_2$ max has been predicted from oxygen uptake at submaximal workloads according to a range of validated models (Leon et al. 1979; Jakicic et al. 1995). The latter method is often used when the risks of performing maximal tests are considered to be too great for the subject population being observed.

Only three of the studies which have used $\dot{V}O_2$ max as an outcome measure have failed to find increases following a brisk walking programme (Kilbom 1971; Hinkleman and Nieman 1993; Snyder et al. 1997).

In the study by Kilblom (1971) the short duration of the intervention (6 wk) may account for the lack of change in $\dot{V}O_2$ max. In two of the studies however while no change in $\dot{V}O_2$ max was detected, a significant decrease in heart rate response to submaximal walking (see 2.3.4. below) was observed (Kilbom 1971; Hinkleman and Nieman 1993). In the study by Snyder et al (1997) although there was no statistically significant alteration in mean $\dot{V}O_2$ max as a result of 32 weeks of an intermittent pattern of brisk walking, 7 of the 13 subjects showed a mean increase in $\dot{V}O_2$ max of 9% following the programme.

To the author's knowledge, no study has compared the effects of accumulated and continuous patterns of brisk walking on $\dot{V}O_2$ max. One of the studies comparing programmes of multiple short bouts of brisk walking spread throughout the day with a more traditional pattern of one continuous daily bout did, however, observe changes in $\dot{V}O_2$ peak. Despite completing more exercise on more days than long bout walkers, the short bout walkers displayed similar increases in $\dot{V}O_2$ peak (5%) to the long bout

walkers (5.6%) (Jakicic et al. 1995). This finding may suggest that more walking is required when using short bouts than when using long bouts to gain equivalent benefits in endurance fitness. The contention that short bouts of exercise accumulated over the course of a day is slightly less effective than a similar amount of continuous exercise has been previously advanced for jogging (DeBusk et al. 1990). Asikainen and coworkers compared the effect on predicted $\dot{V}O_2$ max of walking a prescribed distance (expending 300 kcal per day) in one continuous bout or two bouts per day. (Asikainen et al. 1998b). In contrast to the suggestions by DeBusk et al (1990) that long bouts of exercise may be more effective in increasing endurance fitness, these researchers noted an increase in mean predicted $\dot{V}O_2$ max of 8% and 11.5% in long and short bout walkers respectively with no statistically significant difference between the two patterns.

Using a variety of activities, including walking, two further studies have compared the effects of structured and lifestyle activity interventions on $\dot{V}O_2$ peak (Dunn et al. 1997; Dunn et al. 1999). In the most recent of these, Dunn and colleagues reported an increase in mean $\dot{V}O_2$ peak of 5.9% and 13.7% following 6 months of lifestyle and structured activity respectively (Dunn et al. 1999). In a follow-up of these subjects 18 months later, both groups showed decreases in $\dot{V}O_2$ peak but maintained a mean increase of 2.9% and 5.1% above baseline in lifestyle and structured groups respectively.

In summary, the majority of studies that have considered the effect of brisk walk training on the $\dot{V}O_2$ max of sedentary subjects, have shown increases. When different patterns of brisk walking have been compared the increases from accumulated or lifestyle approaches appear to be similar or slightly less than from more traditional approaches. However the magnitude of the increases in $\dot{V}O_2$ max with any programme of brisk walking vary widely with increase in mean $\dot{V}O_2$ max ranging from 8% (Asikainen et al. 1998b) to 26% (Pollock et al. 1971). It is probable that these differences are due to the frequency, intensity and duration of brisk walks, the length of the intervention, the characteristics of the subjects at baseline (age, fatness, fitness etc.) as well as genetic differences in trainability (Montgomery et al. 1998).

### 2.3.3 Effects of brisk walking on blood lactate concentration during submaximal exercise

Following a period of endurance training $\dot{V}O_2$ max is known to increase. However the magnitude of this alteration varies depending on the pre-training levels. In the study by Snyder et al (1997) the five subjects who showed no improvement in endurance fitness following training were the younger subjects who displayed higher initial $\dot{V}O_2$ max values. The measurement of $\dot{V}O_2$ max in response to training in subjects who are already habitually physically active or have reasonable levels to begin with may therefore be problematic. For this reason, several authors have measured blood lactate concentration at a given workload or oxygen uptake as a more sensitive index of endurance fitness. Traditional forms of exercise training have been shown to decrease the body's reliance on anaerobic metabolism to meet the energy demands of muscle at constant submaximal workloads. Blood lactate concentrations reflect the net result of lactate efflux from the muscle to the blood and its removal from the blood to the liver, kidneys, heart and non-exercising skeletal muscle (Brooks 1991). Increased lactate production by the muscle occurs largely as a result of anaerobic metabolism when the ability of the muscle to derive energy from aerobic sources does not give rise to a sufficient rate of ATP production. Training adaptations that increase the utilisation of oxygen by the muscle delay the point at which blood lactate rises in response to increasing workloads without necessarily altering $\dot{V}O_2$ max. Therefore a lower blood lactate response to a submaximal exercise intensity, or, conversely, an ability to perform exercise of a higher intensity for a given blood lactate concentration is often interpreted as an improvement in endurance fitness. Several of the studies considering the effects of brisk walking on endurance fitness have used blood lactate response to submaximal exercise as a main outcome measure (Hardman et al. 1992; Stensel et al. 1993; Hardman and Hudson 1994).

These 3 studies concur that brisk walking increases the absolute oxygen uptake (and hence exercise intensity) at which a reference concentration of blood lactate (2 $mmol \cdot l^{-1}$) occurs. The mean increase observed in these studies ranged from 6.5% (Stensel et al. 1993) to 30% (Hardman and Hudson 1994). Differences between subjects, in terms of gender and initial fitness levels, and differences in the self-selected walking speed may account for these variations. Interestingly, the largest increase in oxygen uptake at 2

mmol·l$^{-1}$ occurred in the study incorporating the exercise programme of the shortest duration (12 weeks) (Hardman and Hudson 1994). This observation supports the suggestion that many of the adaptations to endurance training occur in the first few weeks of training (Hickson et al. 1981). To the author's knowledge no study that has compared different patterns of brisk walking has used blood lactate responses to monitor alterations in endurance fitness.

### 2.3.4 Effects of brisk walking on heart rate response to submaximal exercise

Heart rate response to a standardised workrate has also been used as a measure of alterations in endurance fitness following brisk walking training. A rise in stroke volume with endurance training accommodates a decline in heart rate without altering cardiac output (Saltin and Rowell 1980). For this reason, a reduced heart rate response to submaximal exercise is often interpreted as an increase in the capacity of the cardiovascular system and thus enhanced endurance fitness. Several of the studies reviewed have used heart rate response to submaximal exercise as the main index of alterations in endurance fitness response (Luria and Koepke 1975; Bergman and Boyungs 1991; Woolf-May et al. 1998). The study by Luria and Koepke (1975) found that the workload required to achieve a heart rate of 150 beat·min$^{-1}$ increased following 10 weeks performing 30 min of brisk walking on 5 d·wk$^{-1}$ (Luria and Koepke 1975). In the other two studies, heart rate response to submaximal treadmill walking (Bergman and Boyungs 1991) and a standardised step test (Woolf-May et al. 1998) was reduced after a 10 and 18 week programme of brisk walking respectively.

Heart rate response to a submaximal workload may, indeed, be a more sensitive indicator of changes in endurance fitness than $\dot{V}O_2$ max. In the studies by Kilblom et al (1971) and Hinkleman and Nieman (1993) decreases in heart rate response to a programme of brisk walking occurred despite no alterations in $\dot{V}O_2$ max.

Two of the studies which have compared different patterns of brisk walking on endurance fitness have used heart rate response data in addition to measures of $\dot{V}O_2$ max and $\dot{V}O_2$ peak (Jakicic et al. 1995; Asikainen et al. 1998b). Jakicic et al (1995) reported increases in mean oxygen uptake at a heart rate of 125 beats·min$^{-1}$ of 7.6% and

14.5% for subjects completing a programme of long and short bouts of brisk walking respectively (Jakicic et al. 1995). Asikainen and colleagues noted similar decreases in heart rate response to submaximal intensity treadmill walking although these were not significantly different from controls (Asikainen et al. 1998b).

The studies reviewed demonstrate that regular brisk walking evokes increases in $\dot{V}O_2$ max and decreases in heart rate and blood lactate response to submaximal workloads associated with more traditional forms of exercise training. A study by Porcari and colleagues (1987) has confirmed that walking is a suitable exercise to elicit 70% of maximal heart rate among sedentary adults (Porcari et al. 1987). In the study of 343 subjects, 83% of men aged 50 and over 91% of all of the women were able to attain 70% of maximal heart rate during a 1-mile track walk. In a subsidiary study, 10 young men with a high $\dot{V}O_2$ max (mean $\pm$ SD, 59.9 $\pm$6.8 $ml \cdot kg^{-1} \cdot min^{-1}$) were asked to walk for 30 min at a pace which elicited this same target heart rate. Feedback on heart rate was provided throughout the walk. At a mean walking speed of 2.3 (0.13) $m \cdot s^{-1}$ all 10 subjects were capable of achieving a mean heart rate at or above 70% of maximum.

Despite inconsistencies in the literature, the available evidence suggests that a programme of brisk walking is sufficient stimulus to cause alterations in endurance fitness in previously sedentary middle-aged or older adults. There is also some evidence that an accumulated approach to brisk walking training may provide similar benefits to more traditional approaches but the extent of this evidence is limited.

Given that walking employs large ambulatory muscles and may impose a moderate stress on the cardiovascular system, it is perhaps not surprising that fitness changes similar to those observed for other forms of exercise such as jogging and cycling. However, although walking has been demonstrated to improve endurance fitness, no study has specifically considered the mechanisms underlying these effects. Given that many of the alterations reported for brisk walking are similar to those associated with more vigorous exercise programmes the underlying mechanisms are likely to be similar. Improvements in fitness are likely to be the result of central and peripheral adaptations in the body's oxygen transport systems (Clausen 1977). Central adaptations include increased maximum cardiac output of the heart while the main peripheral adaptations

involve increased vascularisation of skeletal muscle and an increased activity of oxidative enzymes in that tissue (Saltin and Rowell 1980).

### 2.3.5 Frequency, intensity and duration of brisk walking required to increase endurance fitness

Although the studies reviewed have confirmed the effectiveness of a programme of brisk walking for improving endurance fitness, the wide variation in the characteristics of the programmes used prevents a definitive exercise prescription based on brisk walking from being advanced with any certainty.

Santiago and colleagues (1995) have investigated the length of a brisk walking training programme necessary to achieve measurable improvements in endurance fitness. Using a 40-week intervention of brisk walking at 72% of maximum heart rate reserve on 4 $d \cdot wk^{-1}$, the authors found a 20% increase in $\dot{V}O_2$ max, the majority of which occurred during the first 20 weeks of the programme. Kilbom et al (1971) noted no change in $\dot{V}O_2$ max after just 6 weeks of a brisk walking programme, whereas the majority of studies using interventions of 8 weeks or more appear to have noted considerable increases in $\dot{V}O_2$ max. It appears therefore that an intervention of more than 8 weeks may be required but that in terms of endurance fitness, further gains are unlikely to be made beyond 20 weeks unless exercise intensity (speed or gradient) is increased.

The intensity of brisk walking required to achieve an increase in endurance fitness has been the subject of some debate. Duncan et al (1991) suggests that endurance fitness, as determined by $\dot{V}O_2$ max, increases in response to a programme of brisk walking in a dose-response fashion. Improvements in $\dot{V}O_2$ max of 4%, 9% and 16% after a six month programme depended on whether subjects were assigned to strolling (3 mph) brisk walking (4 mph) or 'aerobic walking' (5 mph) groups respectively, with all three groups walking equal distances. (Duncan et al. 1991). This is in keeping with findings by Gossard and colleagues (1986) who found increases in $\dot{V}O_2$ max (17%) following 12 weeks of moderately high intensity jogging training (63-81% of $VO_2$ max) which were approximately double the increases resulting from a similar amount of lower intensity (42-60% $\dot{V}O_2$ max) walk/jog training (Gossard et al. 1986). In contrast the studies of Hinkleman and Neiman (1993) and Snyder et al (1997) found no increases in

$\dot{V}O_2$ max, following low to moderate intensity exercise 60% and 52% of heart rate reserve respectively (Hinkleman and Nieman 1993; Snyder et al. 1997). The available evidence appears to suggest that in order to improve endurance fitness brisk walking should be performed at a pace that elicits at least 60% of $\dot{V}O_2$ max or 70% of maximum heart rate.

To the author's knowledge only one study has considered the frequency of brisk walking required to improve endurance fitness. Ready and colleagues (1996) found similar increases in $\dot{V}O_2$ peak in subjects who performed 60 minutes of brisk walking on 3 and 5 $d \cdot wk^{-1}$ (12.1% and 13.9% respectively) (Ready et al. 1996). Given that frequency and duration are closely inter-related, the prolonged duration of the walks in this study may reduce the degree to which one could confidently conclude that 3 and 5 days per week of prolonged brisk walking provide equal benefits in endurance fitness. In a similar study, using higher intensity exercise, Pollock et al (1977) showed that when subjects performed 30 minutes of jogging 1, 3 or 5 $d \cdot wk^{-1}$ for 20 weeks there was a mean increase in $\dot{V}O_2$ max of 8.3, 12.9 and 17.4% respectively (Pollock et al. 1977). Despite this apparently graded response, there were no statistically significant differences in the increases between 1 and 3 or between 3 and 5 $d \cdot wk^{-1}$. The brisk walking studies reviewed suggest that frequencies between 3 and 5 $d \cdot wk^{-1}$ are effective for achieving improvements in endurance fitness. However the question of whether more or less frequent brisk walking would alter these improvements remains to be investigated.

The duration of walking necessary to achieve improvements in endurance fitness has not been widely investigated. The majority of studies have used a daily walking duration ranging from 15 to 90 minutes. More recently several investigators have considered the effect of shorter bouts of brisk walking on endurance fitness.

### 2.3.6 Use of different patterns of brisk walking to improve endurance fitness

Several recent studies have sought to investigate whether the duration of brisk walking, necessary to achieve increases in endurance fitness, can be divided into shorter bouts but still retain its effectiveness. Of the walking studies reviewed above, four have

considered whether the effects of brisk walking on endurance fitness can be achieved by accumulating short bouts over the course of the day (Jakicic et al. 1995, Snyder et al. 1997, Asikainen et al. 1998b, Woolf-May et al. 1998). Jakicic at al (1995) were the first to demonstrate that several 10 minute bouts of brisk walking spread throughout the day resulted in the same increases in endurance fitness as a continuous bout of the same overall duration (Jakicic et al. 1995). The findings from the three other studies, reviewed in 2.3.2-2.3.4 above, suggest that it may be possible to divide the 20-60 minute brisk walk into shorter bouts of 10-15 minutes spread throughout the day, and still achieve similar improvements in endurance fitness. Despite these findings, the extent of such experimental support for the fitness benefits of an accumulation of brisk walking over the course of a day remains limited and the initial findings require confirmation.

## 2.4 Health Benefits of brisk walking

### 2.4.1 Overview

The amount (Paffenbarger et al. 1986) and pace (Morris et al. 1990) of regular walking have been associated, respectively, with lower risk of all-cause and coronary mortality. The reduction in coronary risk is probably mediated in part by an alteration in one or more of the established risk factors for CHD risk, including endurance fitness (Blair et al. 1989) as reviewed above. In this section of the review the effects of brisk walking on blood pressure, body composition and resting metabolic rate will be considered. Where the effects of different patterns of brisk walking have been investigated, the findings of these studies will be highlighted.

### 2.4.2 Effects of brisk walking on resting blood pressure

Approximately 6% of all males and 5% of all females in Northern Ireland are hypertensive (systolic blood pressure $\geq$160 mm Hg and/or diastolic blood pressure $\geq$ 95 mm Hg), with the proportion rising with age such that by age 55-56 some 21% of males and 18% of females may be classified as hypertensive (MacAuley et al. 1994). Elevated blood pressure is strongly associated with the incidence of coronary artery disease, congestive heart failure, intermittent claudication and stroke (Kannel et al.

1984). Indeed Paffenbarger and colleagues (1986) found that hypertensive men had nearly twice the risk of death from all causes than their normotensive peers (Paffenbarger et al. 1986). Such epidemiological studies however, do not allow a causal relationship between exercise training and lower blood pressure to be established. A number of longitudinal studies have therefore been carried out in order to consider the effects of endurance training on resting blood pressure. In clinical trials regular physical activity has been shown to lower blood pressure among normotensive and hypertensive subjects. Arroll and Beaglehole (1992) provide a comprehensive review of these studies. In this section those studies which have considered the effects of brisk walking on resting blood pressure will be considered.

Of the brisk walking studies reviewed, only nine have considered the effects on resting blood pressure in previously normotensive subjects (Pollock et al. 1971, Duncan et al. 1991, Whitehurst and Menendez 1991, Davison et al. 1992, Hamdorf et al. 1992, Kingwell and Jennings 1993, Jakicic et al. 1995, Ready et al. 1996, Asikainen et al. 1998a). All but three of these (Duncan et al. 1991, Hamdorf et al. 1992, Ready et al. 1996) have found reductions in systolic and/or diastolic blood pressure ranging from 2 to 14 mmHg following a programme of brisk walking.

Some authors who have noted a reduction in resting blood pressure have suggested that these favourable alterations may be mediated by a change in body mass (Berchtold et al. 1982). For example, in normotensive women (137/80 mm Hg) aged 61-81 years, Whitehurst and Menendez (1991) demonstrated decreases in both systolic and diastolic blood pressure of 3.8 mm Hg following an 8 week programme of brisk walking (20-40mins, 3d·wk$^{-1}$) which were accompanied by a decrease in body mass of 1.1 kg (Whitehurst and Menendez 1991). Variations in the magnitude of the effect of brisk walking on resting blood pressure noted in the literature may also be attributable to the initial blood pressure of subjects. Among hypertensive subjects the potential for reducing blood pressure is of course even greater (Fagard and Tipton 1994). Several authors have considered the effect of brisk walking on hypertensive subjects (Hagberg et al. 1989, Ohta et al. 1990, Seals et al. 1991, Dengel et al. 1998, Cononie et al. 1991). Hagberg and colleagues have demonstrated a formidable reduction (20/12 mm Hg) in resting blood pressure in hypertensive subjects following 9 months of walking training. (Hagberg et al. 1989). This reduction was independent of body weight change.

However they are achieved, even modest reductions in blood pressure have the potential to be clinically important. Blair and colleagues, for example, have suggested that small increases in systolic blood pressure, within the range 120-129 mm Hg are associated with a three-fold increase in developing hypertension (Blair et al. 1989).

The mechanisms underlying a reduction in blood pressure in response to regular brisk walking are probably linked to reduced peripheral resistance caused by vasodilation in active skeletal muscle which occurs during and persists for some time after dynamic exercise (Rowell 1993). This may be mediated by a decrease in resting plasma concentrations of noradrenaline resulting in a decrease in sympathetic nervous system activity (Jennings et al. 1989). There is evidence that these alterations occur very early in response to a programme of physical activity (ACSM 1993) and disappear within 2 weeks after exercise stops (Elrick 1996).

In addition to elevated blood pressure, many subjects in such studies may possess other CHD risk factors such as overweight, obesity or insulin resistance, and therefore other benefits of regular activity may combine with a reduction in blood pressure to reduce risk. However when studies have adjusted for alterations in body mass and other CHD risk factors the anti-hypertensive effect of exercise remains (Blair et al. 1992).

It appears therefore that a programme of brisk walking is capable of altering resting blood pressure and thereby may reduce CHD risk in both normo- and hypertensive subjects.

In light of recent physical activity recommendations, there is a need to clarify whether brisk walking performed in short bouts spread throughout the day has the same effects on resting blood pressure associated with more traditional exercise prescriptions (Haskell 1994b). To the authors knowledge, only two studies that have compared accumulated and continuous patterns of brisk walking have considered the effects of such programmes on resting blood pressure (Jakicic et al. 1995, Asikainen et al. 1998a). Jakicic and colleagues (1995) noted decreases in resting systolic and diastolic blood pressure which did not differ between exercise patterns (Jakicic et al. 1995). Asikainen et al (1998) found mean reductions in resting diastolic blood pressure of 2.7 and 3.2 mm

Hg among subjects performing two short bouts or one long bout respectively on 5 d·wk$^{-1}$ for 1 year, with no significant differences between these decreases (Asikainen et al. 1998a). It appears therefore, that both traditional and accumulated patterns of brisk walking have the potential to reduce resting blood pressure among subjects with initially elevated levels.

### 2.4.3 Effect of brisk walking on body mass and fatness

In Northern Ireland an estimated 58% of men and 56% of women are overweight (defined as BMI>25 kg·m$^{-2}$) with 16% of males and 21% of females being classified as obese (BMI>30 kg·m$^{-2}$) (MacAuley et al. 1994). Overweight and obesity are associated with premature illness and death (N.I.H. 1985).

Regular physical activity, if not accompanied by a compensatory increase in energy intake will tend to reduce the amount of fat stored in adipose tissue, reduce body mass and increase the percentage of the body mass that is occupied by lean tissue. Biologically at least, the role of exercise in weight management seems straightforward. The evidence for the effectiveness of brisk walking in weight reduction is, however, less clear-cut. When body mass or measures of body fatness have been examined in relation to a programme of brisk walking the results have not always been concordant. Several studies have reported decreases in body mass ranging from 0.7 kg to 8.9 kg and decreases in body fat of 1- 21% of body mass (Pollock et al. 1971, Leon et al. 1979, Wood et al. 1983, Juneau et al. 1987, Ohta et al. 1990; Bergman and Boyungs 1991; Whitehurst and Menendez 1991, Davison et al. 1992, Hardman and Hudson 1994; Jakicic et al. 1995, Ready et al. 1996, Snyder et al. 1997, Asikainen et al. 1998b, Dengel et al. 1998, Kukkonen-Harjula et al. 1998) while others have failed to notice any alterations in body composition (deVries 1970,; Kilbom 1971, Adams and deVries 1973, Luria and Koepke 1975, Santiago et al. 1987, Cramer et al. 1991a, Duncan et al. 1991, Hamdorf et al. 1992, Hardman et al. 1992, Hinkleman and Nieman 1993, Stensel et al. 1993). The variations in these findings are probably due, in part, to the initial fatness of subjects and to the difficulty in controlling confounding factors such as energy intake among free-living subjects. In a meta-analysis of 53 studies which have considered the effects of exercise training on body mass and fatness, Ballor and Keesey

(1991) have suggested that the energy expended during exercise and the initial body fat levels were the two factors which most influence the decreases in body fatness observed (Ballor and Keesey 1991). Although some authors have suggested that brisk walking may contribute to weight loss by decreasing appetite (Rippe et al. 1988), it is probably more likely that, if not strictly controlled, a natural adjustment in food intake in response to regular brisk walking and/or a decrease in the physical activity performed outside of the programme, are responsible for the lack of change noted in many studies (Hardman 1996). Not surprisingly therefore those exercise studies which have used obese subjects and attempted to modify eating behaviours have noted the largest decreases in body mass and fatness (Dengel et al. 1998).

Recently, the distribution of adipose tissue, rather than the absolute amount *per se* has been more closely linked with adverse health outcomes (Després et al. 1990b). Individuals with more centrally located, intra-abdominal fat stores have been shown to be at greater risk of premature mortality than individuals with similar amounts of fat distributed on more peripheral sites (Lapidus et al. 1984). For this reason a few studies of brisk walking have attempted to discover whether such activity favourably alters fat distribution. Hip and waist circumferences together have been used as a proxy measure for fat distribution with waist circumference being the best available proxy for deep abdominal or visceral fat (Jones et al. 1986). Dengel and colleagues (1998) noted a 9% decrease in waist circumference and a 3% decrease in waist to hip ratio among obese hypertensive men in response to a 6 month programme of exercise and eating behaviour modification (Dengel et al. 1998). Even when subjects do not alter body weight, a programme of exercise training has been shown to favourably alter body fat distribution by decreasing the waist:hip ratio (Krotkiewski 1990). However in subjects with more typical initial fatness, a programme of brisk walking in the absence of any attempt to alter diet, did not result in an alteration in waist or hip circumference (Hardman et al. 1992; Stensel et al. 1993; Hardman and Hudson 1994)

It appears that a programme of brisk walking has the potential to alter body composition particularly when used with overweight and obese subjects, over a prolonged period and/or in combination with calorie restriction measures. Since the primary mechanism underlying these alterations is the increased energy expenditure resulting from a brisk walking programme, it seems likely, in principle, that short accumulated bouts of brisk

walking would be as effective as a more prolonged bout of similar total duration, in this regard, but the findings of such intermittent exercise studies have been equivocal. Two of the brisk walking studies which have used intermittent bouts accumulated throughout the day have noted a decrease body mass among previously overweight subjects (Jakicic et al. 1995; Snyder et al. 1997). In one of these studies Jakicic and colleagues compared short walks accumulated throughout the day with one long walk of equal total duration. Short and long bout walkers lost 8.9kg and 6.5 kg respectively after 20 weeks of training (Jakicic et al. 1995). Although the decreases in body mass were not significantly different between the two patterns of walking there was a tendency for short bout walkers to lose more weight than long bout walkers. In contrast, Snyder and colleagues (1997) found no alteration in body weight or body composition among 13 overweight women who walked 30 minutes each day (5d.wk-1) in an intermittent pattern for 32 weeks (Snyder et al 1997)

In addition to the energy expended by skeletal muscle contraction, physical activity may alter the rate at which energy is consumed at rest in two ways. Firstly, several researchers have noted that following exercise, energy expenditure remains elevated for a period of minutes (Brehm and Gutin 1986) or hours (Bahr 1992). This transient elevation in energy expenditure above resting levels is often termed the excess post-exercise oxygen consumption (EPOC)(Bahr 1992). In addition to this acute elevation or EPOC some investigators have suggested that regular exercise training may cause a persistent augmentation in resting metabolic rate (Tremblay et al. 1985).

None of the brisk walking studies reviewed have considered the short or long term effects of brisk walking on resting metabolic rate. However it seems theoretically possible that such dynamic exercise, involving large percentage of skeletal muscle mass, has the potential to influence metabolic rate in a similar manner to other modes of exercise. Moreover when an intermittent pattern of walking is considered, the combined EPOC from each session, accumulated over the course of a day, may exceed the elevation caused by a single continuous bout. However, this possibility has not been tested empirically.

## 2.5 Exercise and lipoprotein metabolism

One of the mechanisms thought to underlie the inverse relationship between regular physical activity and CHD is the effect of such activity on lipoprotein metabolism. Regular physical activity has been associated with lower concentrations of triacylglycerol and total cholesterol and higher concentrations of high density lipoprotein cholesterol in human plasma (Goldberg and Elliot 1987). Such changes in lipoprotein profiles have long been associated with a reduced risk of coronary heart disease (Paffenbarger et al. 1984). In this section a brief overview of lipoprotein metabolism is followed by a consideration of the influence of exercise on lipoprotein metabolism in the fasted and postprandial states.

### 2.5.1 Overview of lipoprotein metabolism

Lipoprotein is the generic term that refers to a group of spherical particles that transport both endogenously synthesised lipids and dietary lipids in circulation. As the name suggests lipoproteins are composed of lipids and proteins.

Lipids can be defined as substances that are soluble in organic solvents but not in water (Brooks and Fahey 1988). There are three major subclasses of lipid: triacylglycerol, phospholipids and cholesterol. Triacylglycerol (TAG) is formed by a combination of fatty acids (which may vary in chain length) with glycerol, resulting in three fatty acids being attached to a carbon skeleton. TAG is formed by esterification and broken down by hydrolysis. The synthesis, digestion storage and mobilisation of TAG occurs in a series of esterification and hydrolytic reactions aided by specific enzymes. Phosopholipids are another important category of lipid and are composed of two fatty acids, glycerol and a nitrogen molecule. Phopholipids are amphipathic molecules, which are crucial components of all cell membranes. A third important lipid, cholesterol, is a steroid molecule present in plasma membranes which plays an important role in membrane fluidity and stability.

The protein components of lipoproteins are called apolipoproteins. Apolipoproteins are involved in the structure, cell recognition and metabolism of lipoproteins. Fourteen human plasma apoproteins have been identified. Specific apoproteins are associated

with certain lipids and act as cofactors for enzymes involved in lipoprotein metabolism allowing the exchange of lipids from one lipoprotein type to another (Kostner 1983). Apoprotein classes are labelled alphabetically (A-H) with subclasses within each type being described numerically (Brewer et al. 1988).

Lipoproteins, then, are macromolecular complexes, soluble in plasma and classified according to the amount and type of lipid and protein they contain into four basic groups; chylomicrons, very low density lipoproteins, low density lipoproteins and high density lipoproteins

Due to their high lipid content (up to 90% by volume) chylomicrons are the least dense of the lipoproteins. They are formed in the intestinal mucosa after digestion of a meal in order to transport dietary fat in the blood (see description of digestion and absorption below). Chylomicrons are composed of 98-99.5% lipid with the remainder being apoprotein (primarily A-I A-IV C and E). After entering the circulation chylomicrons are quickly degraded (half-life of approximately 4-6 minutes) to chylomicron remnants (mainly cholesterol and phospholipid) by the action of LPL (Grundy 1976). Chylomicron remnants are taken up by the liver.

Very low density lipoproteins (VLDL) resemble chylomicrons except they are smaller in size. They contain mainly endogenous TAG which has been synthesised from circulating non-esterified fatty acids. They are synthesised in the liver. They contain 85-90% lipid and 10-15% proteins (primarily B, C and E). The catabolism of VLDL is similar to that of chylomicrons with endothelial LPL remaining the key regulatory enzyme. VLDL particles have a half-life of about 2-4 hours (Durstine and Haskell 1994).

Low density lipoproteins (LDL) are a product of the catabolism of VLDL and are the primary carrier of cholesterol in the body. They are composed of 75% lipid (40-60% cholesterol) and 25% protein (primarily Apo B) (Goldstein and Brown 1977). LDL is taken up by peripheral tissue cells for subsequent hydrolysis. LDL particles have a half-life of approximately 3 days (Frayn et al. 1996).

High density lipoproteins are the smallest and most dense lipoprotein fraction containing approximately 50% proteins and 50% lipids. HDL function largely in cholesterol exchange reaction in the plasma, receiving cholesterol from other lipoproteins and other tissues and returning it to the liver. HDL particles have a half-life of 5-6 days (Brown and Roberts 1991).

TAG constitutes 90% of lipid taken into and stored in the body (Gurr and Harwood 1991). When a meal containing TAG is ingested mechanical agitation in the mouth breaks the fat globules into smaller droplets. Once it has passed through the oesophagus and into the stomach it is emulsified and delivered to the duodenum. In the duodenum, it is further emulsified by bile salts and then hydrolysed into monoglycerides and free fatty acids in the small intestine. Together with bile salts and phospholipids these products of hydrolysis form micelles which are absorbed into the epithelial cells lining the small intestine where they are resynthesised into TAG and combined with absorbed cholesterol and phospholipids and coated with protein forming chylomicrons. When chylomicrons have been formed in the epithelial cells they are secreted by the lacteals (lymphatic vessels in each intestinal villus) into the lymph circulation and further via the thoracic duct to the general circulation. Once in the circulation, TAG exists in a state of dynamic equilibrium between the various classes of lipoproteins. Their metabolism is controlled by the action of three enzymes, lipoprotein lipase (LPL) hepatic lipase (HL) and lecithin cholesterol acyltransferase (LCAT). LPL is responsible for the hydrolysis of plasma chylomicron and VLDL TAG (Durstine and Haskell 1994). LPL is contained on the luminal surfaces of capillary endothelial cells of many tissues including adipose tissue and cardiac and skeletal muscle (Eckel 1989). LPL hydrolyses circulating TAG to fatty acids and glycerol. HL hydrolyses phospholipids and TAG in HDL particles. LCAT plays an important role in HDL metabolism by promoting the esterification of cholesterol. A more detailed account of lipoprotein metabolism is beyond the scope of this review. However Brewer and colleagues (1988) provide a complete review (Brewer et al. 1988).

### 2.5.2 Plasma lipids concentrations in the fasted and postprandial state

The concentrations of different lipids and lipoproteins in the plasma are determined by a myriad of pathophysiological and physiological factors including nutritional status and exercise. In the fasted state endogenously synthesised lipids are released into the plasma from adipocytes or circulating lipoproteins, are carried in VLDL produced by the liver (Chappell and Spector 1991). In normolipidaemic individuals therefore this results in plasma lipoprotein concentrations illustrated in table 2.2.

| Lipid | Fasting plasma concentration |
|---|---|
| TAG | 0.7-1.8 $mmol \cdot l^{-1}$ |
| Total Cholesterol | 3.5-7.8 $mmol \cdot l^{-1}$ |
| HDL Cholesterol | 0.8-1.7 $mmol \cdot l^{-1}$ |
| LDL Cholesterol | 2.3-6.1 $mmol \cdot l^{-1}$ |

**Table 2.2** Typical plasma lipid concentrations (range) in fasting adults (male and female) (adapted from Ball and Mann 1988)

The level of lipoproteins in the plasma during the postprandial period depends on the meal content and the length of time from ingestion as well as a host of individual factors including the action of insulin and the size of the endogenous TAG pool. Within one hour of ingestion of a fatty meal, chylomicrons appear in the plasma and these are usually removed within eight hours of ingestion (Patsch 1987).

### 2.5.3 Influence of brisk walking training on fasting lipoprotein metabolism

Superko (1991) and Durstine and Haskell (1994) have produced comprehensive reviews of the studies which have been conducted to examine the influence of exercise on lipoprotein metabolism (Durstine and Haskell 1994; Superko 1991). The findings of such studies provide limited support for the notion that regular endurance training alters blood lipoproteins in a manner which is likely to reduce the progression of

atherosclerotic disease (Superko 1991). Although by no means unequivocal, the literature dealing with the effects of regular endurance exercise on fasting blood lipid and lipoprotein concentrations, has generally found increases in HDL cholesterol concentrations in normolipidaemic individuals, reductions in total cholesterol concentrations in hypercholesterolaemic subjects and reductions in TAG concentrations among hypertriglyceridaemic individuals, with little consensus on the effects on TAG or total cholesterol in normolipidaemic individuals. The variety of exercise modes and intensities employed in these studies are two of the many factors that may contribute to the discrepancies in the findings. The purpose of this section of the review is to consider the findings of those studies which have used brisk walking, in either a continuous or an accumulated pattern, and observed the effect of training on fasting blood lipoprotein concentrations.

Several authors have investigated the effects of brisk walking on fasting lipoprotein concentrations. Approximately half of the brisk walking intervention studies reviewed have demonstrated alterations in fasting blood lipoprotein concentrations (Leon et al. 1979; Duncan et al. 1991; Whitehurst and Menendez 1991; Davison et al. 1992; Hardman and Hudson 1994; Kukkonen-Harjula et al. 1998) with the remaining studies failing to note any alteration (Juneau et al. 1987; Santiago et al. 1987; Hinkleman and Nieman 1993; Stensel et al. 1993; Davison and Grant 1995, Asikainen, 1998 #298; Santiago et al. 1995; Ready et al. 1996; Woolf-May et al. 1998). Even when flaws in study design such as inadequate subject numbers, lack of a control group or inappropriate statistical procedures (Wood et al. 1984) are taken into account there is still only moderate support for an alteration in fasting lipoprotein concentrations in the blood after a period of brisk walk training.

A few well-designed longitudinal studies illustrate this disparity in findings. Kukkonen-Harjula and colleagues (1998) randomly assigned 119 healthy middle aged subjects to a walking ($1h \cdot d^{-1}$ on $4\ d \cdot wk^{-1}$) group or a no exercise control group for 15 weeks (Kukkonen-Harjula et al. 1998). The authors noted a decrease in total and HDL cholesterol, an increase in $HDL_2$ cholesterol and the ratio of HDL cholesterol to total cholesterol in the walking group as well as a decrease in serum TAG among the male subjects who undertook the brisk walking training (n=25). By contrast, Stensel and colleagues assigned healthy but sedentary male subjects to a progressive walking

programme (20-45 min·d$^{-1}$) for 12 months. These authors noted no change in any plasma lipid or lipoprotein variable (Stensel et al. 1993). The differences in findings in these and the other studies reviewed are probably attributable to a variety of factors. The frequency, intensity and duration of walking (Haskell 1994b), the length of the intervention (Wood et al. 1983), the initial lipoprotein levels of subjects (Hardman et al. 1992), alterations in body mass or adiposity (Whitehurst and Menendez 1991), changes in diet (Wood et al. 1991) and methodological issues surrounding blood lipoprotein determination probably combine to affect the observed outcome of these studies. In addition to such confounding factors, the timing of the post-training blood sample may inadvertently have increased the possibility of interpreting 'last bout effects' on lipoprotein concentrations as 'training effects'.

However, when the evidence from these longitudinal intervention studies are combined with that from epidemiological and observational or cross-sectional studies, a role for brisk walking in favourably alterating fasting blood lipoprotein concentrations can be cautiously advanced. Cook and colleagues (1986) noted that regular walking by postmen was positively associated with HDL cholesterol concentration (Cook et al. 1986). Tucker and Friedman assessed weekly frequency and duration of walking and serum cholesterol in over 3,000 adults and concluded that walking more than 2.5 h·wk$^{-1}$ was associated with higher HDL cholesterol and HDL to total cholesterol ratios (Tucker and Friedman 1990). Palank and Hargreaves (1990) observed an increase in the HDL to total cholesterol ratio among male golfers over the playing season.

The intensity frequency and duration of brisk walking required to achieve alterations in blood lipids has been investigated in a series of intervention studies. Duncan and co-workers assigned young women to three different walking intensities described as strolling (4.8 km·h$^{-1}$), brisk walking (6.4 km·h$^{-1}$) and 'aerobic walking' (8 km·h$^{-1}$) for 24 weeks (Duncan et al. 1991). Although fitness improved in a dose-response manner, HDL concentration increased to a similar degree in all walking groups suggesting that the benefits are due to increases in energy expenditure rather than intensity or duration *per se* (Hardman 1999). This suggestion is well supported by epidemiological evidence (Paffenbarger et al. 1986).

All of the intervention studies reviewed in this section so far have considered the effect of a structured programme of exercise on blood lipid profiles. If the alterations in blood lipid profiles are related to energy expenditure then it seems reasonable to suggest that short sessions of brisk walking accumulated throughout the day or other less structured 'lifestyle' activities currently promoted by public health recommendations would be a sufficient stimulus to alter blood lipid profiles. Several studies have recently investigated this proposition (Dunn et al. 1997, Snyder et al. 1997, Asikainen et al. 1998b, Woolf-May 1998, Andersen et al. 1999, Dunn et al. 1999). Snyder and colleagues (1997) found no alterations in blood lipid profiles in middle aged overweight women, following 32 weeks of brisk walking training performed in discontinuous format, with three 10 minute walks spread throughout the day. Even subjects who showed improvements in fitness and reductions in adiposity failed to alter fasting blood lipids (Snyder et al. 1997). A similar lack of effect of a discontinuous brisk walking programme (two short walks of approximately 15 min or one long walk of approximately 30 min on 5 $d \cdot wk^{-1}$ for 15 wk) was noted by Asikainen and colleagues (Asikainen et al. 1998b).

Three recent studies of 'lifestyle activity', provide more encouragement for the notion that such accumulated activity could alter blood lipid profiles (Dunn et al. 1997; Andersen et al. 1999; Dunn et al. 1999). Subjects in all three studies were encouraged to find ways of accumulating 30 minutes of physical activity during their typical day by, for example, choosing to walk short journeys or climbing stairs rather than taking the elevator. In the study by Andersen and co-workers a significant reduction in serum TAG and total cholesterol concentrations was observed after 16 weeks (Andersen et al. 1999). However these subjects were initially obese and the lifestyle intervention included dietary modification so it is difficult to separate the effects of increased physical activity from alterations in food intake as both are known to alter blood lipid profiles. Even in studies where there is no dietary manipulation concomitant changes in weight often occur as a result of an exercise programme and these are associated with alterations in blood lipid profiles. Indeed, the work of Nieman and colleagues suggests that changes in diet and body weight alone account for the decreases in total cholesterol with a 5 week programme of diet and exercise in mildly obese women and that exercise training is only responsible for alterations in HDL cholesterol (Nieman et al. 1990). The

studies by Andrea Dunn and colleagues compared the more traditional structured exercise programme with a lifestyle intervention that did not alter dietary intake. In one study, significant reductions in total cholesterol and total cholesterol to HDL cholesterol ratios noted at six months (Dunn et al. 1997) which did not differ between interventions. Moreover in the lifestyle activity group, these reductions occurred in the absence of a significant decrease in body weight. In the more recent study, only the structured exercise group experienced any alteration in blood lipids but again this effect was independent of an alteration in body weight (Dunn et al. 1999).

Farrell and Barboriak (1980) have investigated the time course of the alterations in plasma lipid and lipoprotein concentrations. Their findings suggest that during 8 weeks of moderately high intensity training (70% $VO_2$ max), HDL cholesterol decreases during the first two weeks and increase during the next 6 weeks of training whereas TAG concentrations remain stable for the first 4 weeks and then decreased from 4 to 8 weeks training (Farrell and Barboriak 1980). Once alterations in blood lipid profiles have been achieved, a regular programme of physical activity is necessary to maintain this favourable profile. Hardman and Hudson (1994) noted a decrease in HDL and $HDL_2$ cholesterol following 12 weeks of detraining from a programme of brisk walking (Hardman and Hudson 1994)

The mechanisms underlying the alterations in fasting lipid profiles are incompletely elucidated but one may involve the rate-limiting enzyme lipoprotein lipase (Katan 1990). LPL facilitates the removal of TAG from chylomicrons and VLDL and allows the cholesterol, protein and phospholipid remnants from these particles to be transferred to HDL. The net effect, an increase in HDL cholesterol and a decrease in TAG are consistent with the findings of cross-sectional studies of physically active individuals (Sady et al. 1984). Whatever the mechanism of change, the alterations in plasma lipid concentrations may have important health implications. An increase of 0.26 $mmol \cdot l^{-1}$ of HDL cholesterol in women has been associated with a 40-50% decrease in CHD risk (Kannel 1983). Manninen et al (1988) in a cross-sectional study observed a powerful negative correlation between fasting HDL cholesterol concentration and coronary disease risk which suggests that a rise in HDL cholesterol of 1% is associated with a 3% lower coronary disease risk by (Manninen et al. 1988).

In its entirety, the literature considering the effects of brisk walking on fasting blood lipids suggests that a programme of brisk walking, of approximately 12 weeks or longer, which results in an energy expenditure of 1200 kcal or more per week (Haskell 1986), may increase HDL cholesterol in sedentary individuals and may reduce total cholesterol and TAG concentrations in subjects with initially elevated levels.

### 2.5.4 Influence of a single bout of brisk walking on postprandial lipaemia

Many of the studies reviewed have, for reasons of control, studied subjects in the fasted condition. Humans however spend the majority of their life in the postprandial state where TAG and cholesterol are circulating in chylomicrons as well as VLDLs. Additionally, most exercise is performed in this postabsorptive state. It seems logical therefore when attempting to elucidate the effect of exercise on lipoprotein metabolism, that some consideration is given to postprandial concentrations.

One of the first studies which described the effect of exercise on postprandial lipaemia was published by Cohen and Goldberg (1960) who noted a reduced plasma turbidity following a high-fat meal in subjects who walked for 9.6 km in the postprandial period (Cohen and Goldberg 1960). Using a more qualitative analysis of blood lipids Nikkila and Konttinen (1962) found a reduced serum TAG concentration following a high fat meal among army recruits who marched 16 km compared to those who rested during the postprandial period (Nikkila and Konttinen 1962). Since these studies were first published, several authors have examined the effect of blood lipid concentrations when exercise was performed during the postprandial period (Welle 1984, Schlierf et al. 1987, Klein et al. 1992, Aldred et al. 1993). Klein and co-workers showed reductions in TAG concentrations after a high fat meal when subjects performed 30 minutes of vigorous activity (75% of $\dot{V}O_2$ peak) an hour after ingestion (Klein et al. 1992). Aldred and Hardman (1993) noted a 24% reduction in postprandial lipaemia when 90 minutes of low intensity activity (40% of $\dot{V}O_2$ max) was performed 1.5 h after consuming a high fat meal (Aldred et al. 1993).

Only two authors have considered the effect of exercise taken immediately before a meal on blood lipid concentrations in the postprandial period (Annuzzi et al. 1987; Burnett et al. 1993). Both studies found a reduction in postprandial lipaemia when a

meal was taken 15 minutes after a 90-minute exercise bout. There is therefore only limited evidence on the effect of exercise typical of current recommendations on postprandial lipaemia. Given the 'last bout effects' (Haskell 1994b) referred to earlier, it seems worthwhile to consider the effects on 'real life' activity over the course of a day and the influence of 'real life' meals on postprandial lipaemia.

Exercise however has been shown to alter intestinal activity (Soffer et al. 1991) and modify blood distribution in the body (Rowell 1993). The rate of blood flow through a muscle has been shown to be major determinant of FFA uptake and utilisation during exercise. (Brooks and Fahey 1988). When exercise is performed immediately before or immediately after a meal this may alter the digestion absorption and uptake of dietary fats and hence cause alterations in blood lipid concentrations that are not due to alterations in lipid metabolism. For this reason several authors have considered the effect of a single bout of exercise performed 12 hours or longer before the ingestion of a meal to gain a clearer insight to the effect of exercise on lipoprotein metabolism. Using brisk walking (Aldred et al. 1993, Aldred et al. 1994, Tsetsonis and Hardman 1996a) running (Sady et al. 1986, Herd 1997, Zhang et al. 1998) and cycling (Herd 1997) at intensities ranging from 30-60% of $\dot{V}O_2$ max and duration's from 90-200 minutes, the studies have found mean reductions in postprandial lipaemia of between 16 and 65%. The variation in the magnitude of reduced postprandial lipaemia is probably attributable to differences in subjects or exercise employed, the amount or timing of the meal ingested or the timing of blood samples. Despite discrepancies in the magnitude of the findings the studies provide unequivocal support for the notion that a prolonged bout of exercise reduces the plasma TAG response to a high fat meal when this meal is ingested during the extended recovery period after exercise.

All of these studies have used prolonged exercise of moderate intensity some considerable hours before the ingestion of an abnormally high fat load. It is not clear whether dividing such prolonged activity into shorter bouts of equal total duration would alter postprandial lipid-lowering properties. Even if an alteration in postprandial lipaemia can be achieved the day after prolonged bout, which has been divided into smaller bouts throughout the day, this still has limited practical application for three reasons. Firstly the sedentary population are unlikely to perform prolonged activity (>90 min) of the type used in the studies reviewed above, even if divided into smaller bouts.

Secondly individuals exercising for health benefit are unlikely to follow activity with an overnight fast and a high fat meal. Finally, it is unlikely that free-living individuals would consume meals as high in fat as many of the test meals used in these studies. It is more likely that in a typical day an individual will exercise at some time point between two meals and that this exercise will be moderate in intensity and relatively short in duration. Whether such circumstances will decrease the postprandial response to the meal and hence reduce the exposure of the arterial wall to atherogenic blood lipids is unclear. Finally, to the author's knowledge no study to date has compared the effects of different patterns of physical activity on postprandial lipaemia.

## 2.6 Effect of exercise on psychological parameters

### 2.6.1 Overview

Although the close association between exercise and psychological function dates back to early civilisation, only relatively recently has the effect of exercise on psychological health become a rapidly expanding research topic. Several authors provide comprehensive critical reviews of the effects of exercise on psychological well-being (Scully et al. 1998). This section of the review will focus specifically on the effect of brisk walking on self-efficacy, mood state, and perceived barriers to exercise.

### 2.6.2 Effect of brisk walk training on self-efficacy

The term self-efficacy, which can be defined as one's confidence of being able to perform a specific activity or behaviour, was first coined by Bandura in the 1970s (Bandura 1977). Self-efficacy or the belief that one can perform a given exercise or physical activity is a social cognitive function based upon past social learning. Self-efficacy, together with an understanding of the outcomes of physical activity, are considered by some authors to be important determinants of physical activity in adults (King et al. 1992). In addition to this role of self-efficacy as an antecedent of exercise behaviour, research attention has also considered how exercise affects self-efficacy. Self-efficacy expectations are affected by reciprocal determinism. In other words an individuals self-efficacy not only determines which activities they will choose but successful participation in an activity is thought to influence the individuals feelings of

self-efficacy towards physical activity (Sonstroem and Morgan 1989). It is this latter use of self-efficacy, as an outcome of a programme of moderate intensity exercise, which will be considered in this section of the review.

Although there is a widespread intuitive belief that exercise and physical activity increase self-efficacy and other psychosocial variables, a host of methodological and conceptual problems have resulted in only a small body of reliable literature. McAuley (1994) in a comprehensive review of the studies which have used self-efficacy as an outcome measure and observed changes in response to an exercise programme noted only 16 studies which fulfilled these criteria (McAuley et al 1994 ). Of these studies 3 considered the effects of a single bout of exercise and 13 considered the effect of an exercise programme on self-efficacy. In general there is a consistent finding that regular exercise positively influences an individuals perception of their physical capabilities. Self-efficacy beliefs are derived from four sources of information available to the individual namely, performance attainment, modelling, verbal and social persuasion and self-assessment of physiological state. In other words, an individual's confidence in performing a particular physical activity, for example, jogging for 30 minutes, will depend on their personal experience of jogging, the degree to which they observe other individuals with whom they identify achieving the activity, the persuasion of others and their own assessment and interpretation of physiological responses to the activity (e.g. laboured breathing or elevated heart rate). Given that the first of these factors, 'performance attainment' is considered to be the most important source of information (Biddle and Mutrie 1991) it is not surprising that a programme of regular exercise has been associated with an increase in self-efficacy.

The majority of studies of the effect of exercise on self-efficacy have been with cardiac rehabilitation patients (Ewart et al. 1983) and subjects with chronic obstructive pulmonary disease (Kaplan et al. 1984). These and other patient population studies have proposed a strong positive affect on self-efficacy of exercise. However, given that expected outcomes play a role in self-efficacy (Schunk 1995) and that the incentive value of exercise is likely to be higher in patient groups (Rejeski 1992) than in asymptomatic individuals, the applicability of this literature to sedentary but otherwise healthy populations is perhaps limited. Five of the studies reviewed have considered the effect of regular exercise on self-efficacy using walking (McAuley et al. 1991), jogging

(Long and Haney 1988) strength training (Holloway et al. 1988) swimming (Hogan and Santomier 1984) and aerobic dancing (Corbin et al. 1984). Although an increased self-efficacy was an almost unanimous finding of these studies a few methodological and conceptual problems are worthy of mention. Firstly several studies have failed to use a random assignment of subjects or a control group (Hogan and Santomier 1984; Holloway et al. 1988; Long and Haney 1988; McAuley et al. 1991). A second gap in the literature is consideration of the effect of changes in physiological function interact with alterations in self-efficacy perceptions (McAuley 1994). It may be that an improvement in fitness, rather than participation in exercise alone, increases the degree to which an individual perceives themselves capable of performing other physical activity (Steptoe 1992). Finally although authors have agreed on definition of the concept of self-efficacy, there is no 'gold standard' tool for its assessment.

A recent study by McAuley, Mihalko and Bane (1997) is illustrative of the exercise and self-efficacy literature. The study considered the effect on self-efficacy of five months participation in a progressive brisk walking programme (15-40 $min \cdot d^{-1}$ on 3 $d \cdot wk^{-1}$). Physical self-efficacy was measured using a scale developed by Ryckmann and colleagues (1982) where subjects were asked to indicate their confidence to successfully execute several activities on an 11-point scale (Ryckmann et al 1982). The results showed an increase in self-efficacy and a concomitant increase in $\dot{V}O_2$ max.

None of the studies examining the effects of short bouts of brisk walking have considered self-efficacy. Only one of the studies, which have compared the effects of lifestyle and structured patterns of activity, has considered the effect on self-efficacy. Dunn and colleagues administered a self-efficacy questionnaire before random assignment to a structured exercise programme or a lifestyle programme that encouraged the accumulation of 30 minutes of physical activity per day. After six months of participation in the programme, self-efficacy increased to a similar degree in both groups. Substantial weekly or fortnightly meetings in which behavioural and cognitive strategies for initiating and maintaining physical activity were learned accompanied both programmes. Whether participation in the physical activity alone would have resulted in such alterations in subjects perceived self-efficacy is unclear.

### 2.6.3 Effect of brisk walk training on mood and affective state

Mood can be defined as transient affective states or an individual's feelings at a specific moment. There has been considerable investigation into the relationship between exercise and mood. A plethora of cross-sectional and quasi-experimental studies have demonstrated associations between level of physical activity and positive mood. Such associations have been made for a single bout of exercise (Steinberg et al. 1997), for a programme of exercise training, for low moderate and high intensity exercise (Moses et al. 1989), with clinically depressed (Griest 1987) and normal populations (Moses et al. 1989). A recent meta-analysis of 26 studies concluded that exercise decreased tension/anxiety, depression fatigue and confusion while increasing vigour (McDonald and Hodgdon 1991). However, well-designed randomised controlled studies, which constituted only 3 of the studies in McDonald and Hogdgon's meta-analysis, have demonstrated only limited benefits (Stanton and Arroll 1996). Several authors provide comprehensive reviews of these studies (Biddle and Mutrie 1991; McDonald and Hodgdon 1991; La Fontaine et al. 1992; Mutrie and Biddle 1995 Byrne and Byrne 1993). No studies to date have compared the effects of different patterns of exercise on mood. In this section, studies, which have observed the effects of a programme of brisk walking on mood and affective state of normal populations, will be considered.

Two measures of mood dominate the literature reviewed. The Profile of Mood State (POMS) was developed by McNair Lorr and Droppleman in the early 1970's for use in clinical populations. It is composed of six scales designed to assess tension, depression, anger, vigour, fatigue and confusion and requires subjects to respond to a list of adjectives using a 5 point Likert scale. The second, less popular mood measure used in the literature, the Multiple Affect Adjective Checklist (MAACL) was developed by Zuckerman and Lubin in the 1960's. Having reviewed the findings which result from the studies of the effects of exercise training on mood, McDonald and Hodgdon conclude that POMS is less influenced by various measures of response set such as social desirability and acquiescence or expectancy than MAACL. In addition they suggest that POMS has the advantage that it includes a vigour scale which has been shown to increase in many exercise related studies (McDonald and Hodgdon 1991).

Despite several methodological concerns, POMS remains the most popular measure of mood state in the literature and it has been utilised in many exercise intervention studies. Cramer, Nieman and Lee (1991) used POMS to compared the effects of brisk walk training at 62% of $\dot{V}O_2$ max (40 min·d$^{-1}$, 5 d·wk$^{-1}$ for 15 weeks) on mood state in mildly obese women. Although general psychological well-being (assessed by another psychometric questionnaire) was improved by participation in the programme, no effect on mood (assessed by POMS) was observed (Cramer et al. 1991b). A recent randomised control trial by Stanton and Arroll (1996) is perhaps typical of the studies of brisk walking which have failed to note any alteration in mood in response to a brisk walking programme. 177 mildly hypertensive men were randomly assigned to a programme of brisk walking at 60% of heart rate reserve (40 min·d$^{-1}$, 3 d·wk$^{-1}$ for 26 weeks) or to a no exercise control group. Despite alterations in blood lipid concentration, systolic blood pressure and body mass, no change in mood state was reported (Stanton and Arroll 1996). This finding echoes earlier studies (Blumenthal et al 1982) which undermine the notion that brisk walk training enhances mood. The failure of such studies to notice an effect on mood may be due in part to the initial mood of subjects at baseline. In studies where subjects reported low initial mood scores (Simons and Birkimer 1988) or high levels of anxiety (Steptoe et al. 1989) the improvements in mood have been most marked. In contrast, a recent study by DiLorenzo and colleagues assigned 111 adults with normal baseline mood to a cycling training programmes of 24 or 48 min.d-1 on 4 d.wk-1 or a sedentary control group, for 12 weeks. Both exercise groups reported improvements in mood, in particular the 'vigour' subscale compared to controls which were still evident 12 months after the structured exercise programme had ceased (DiLorenzo et al. 1999).

The hypothesised mechanisms thought to underlie alterations in mood resulting from exercise have been frequently reviewed (Raglin 1990). These include physiological factors such as alterations in brain neurotransmitters, alpha brain waves, decreases in muscle electrical tension, deep body temperature and increased oxygen transport and psychological factors such as distraction, social reinforcement, positive expectations and mastery. Given that many of the studies noting a relationship between exercise and mood have shown a concomitant improvement in fitness (Goldwater and Collins 1985; Blumenthal et al. 1982; Moses et al. 1989; Steptoe et al, 1989) it is difficult to separate

the effects of involvement in an exercise programme from improvements in endurance fitness as causes of mood alterations.

To the author's knowledge no study has attempted to compare the effects of different patterns of brisk walking on mood. However given that studies in the area have reported an improvement in mood after a single bout of 25 minutes of moderate intensity exercise (Steinberg et al. 1997) and after as little as 5 minutes of walking (Thayer 1987), it seems plausible that several bouts spread throughout the day may have an even greater potential for enhancing mood. This suggestion however has not been tested empirically.

### 2.6.4 Effect of brisk walk training on perceived barriers to exercise

Despite repeated messages about the effects of physical inactivity on health, recent populations surveys show that many individuals throughout the developed world do not take sufficient physical activity to confer health benefits (Allied Dunbar 1992; MacAuley et al. 1994). Psychological theories of behaviour and social psychological theories of decision making have provided much insight into the determinants of physical activity in previously sedentary individuals and indicate the stages through which people pass on their route to adopting an active lifestyle. One important aspect of such theories is the perception by the individual of barriers to exercise. Perceived barriers to exercise are the factors associated with an individual's disinclination to partake in physical activity. Steinhardt and Dishman (1989) have found four major barriers which people perceive as preventing them from taking regular exercise; time, effort, obstacles and limiting health. Recent national population fitness and activity surveys have suggested that perceived barriers to activity fall into five main categories; physical, emotional, motivational, time and availability (Allied Dunbar 1992). In the Northern Ireland population, the perceived barriers to activity are broadly similar to these five with some shifts within each category (MacAuley et al. 1994). In both surveys 'lack of time' was the most frequently cited barrier to physical activity. In the Northern Ireland survey, work and caring for children were cited as the primary reasons for lack of time. In the group of factors considered to be emotional barriers, over one third of males and over one half of females surveyed stated that they were 'not the sporty type'. Among the motivational barriers, the need to rest and relax in their spare

time was the most cited reason for inactivity. Among the physical barriers the presence of an injury or disability was the main reason for lack of activity while those reporting availability barriers referred almost equally to lack of a partner to exercise with, inability to afford exercise and lack of facilities.

Walking overcomes many of these barriers since it is inexpensive, requires no special facilities and no particular physical skill while an accumulated approach to brisk walking allows the time barrier to be addressed as well. Whether participation in a programme of exercise alters an individual's perception of their barriers to exercise is not clear. Given that self-efficacy can be enhanced by regular exercise (see 2.6.2. above) and that self-efficacy is a major determinant of exercise behaviour it is probable, as well as logical, that participation in a pattern of short accumulated brisk walks would reduce the perceived barriers of individuals to physical activity. This notion has not been examined experimentally.

All experimental studies using exercise interventions and hoping to draw inferences about perceived barriers to exercise have one unavoidable, inherent flaw. Volunteers to an exercise training study, even before random allocation, may be somewhat different psychologically than other sedentary individuals in the population. The stages of change model derived from behavioural psychology theory has recently been applied to the adoption of exercise behaviours. The model suggests that individuals move through a series of stages when adopting a new behaviour such as physical activity; pre-contemplation, contemplation, preparation, action, maintenance termination and relapse (Marcus and Owen 1992). In any exercise intervention study that recruits sedentary volunteer subjects are likely to be at the contemplation stage, in other words they have an intention to try physical activity and the study provides them with an opportunity. The lifestyle intervention study by Dunn and colleagues, referred to earlier, illustrates this point. When asked their stage of readiness for physical activity using a psychological questionnaire, 85% of subjects reported 'intending to change but not yet taking action', with only 1% reporting 'no intention to change' (Dunn et al. 1997). The barriers to exercise for individuals intending to begin a programme of physical activity may be qualitatively and quantitatively different to the barriers facing individuals at an earlier pre-contemplation stage. Hence the applicability of such findings on barriers to

exercise to the sedentary majority in the population, for whom such patterns of physical activity are designed, may be limited.

### 2.6.5 Effects of intermittent and lifestyle activity on adherence

Many researchers have shown that 50% in a typical supervised exercise programme. Given that a lack of time is proposed as the main reason why individuals dropout of exercise programmes (Dishman 1990) some authors have suggested that physical activity patterns be developed which fit conveniently with an individuals daily routine (Blair et al. 1992). Although the idea that more flexible patterns of exercise will promote greater adherence has strong intuitive appeal only a limited number of studies have compared adherence to continuous bouts of exercise with shorter bouts accumulated over the course of a day (Jakicic et al. 1995; Jakicic and Wing 1997; Woolf-May 1998; Andersen et al. 1999). Of these studies only the two by Jakicic and colleagues demonstrate increased adherence to accumulated or lifestyle activity. In two randomised, controlled, clinical trials (20 weeks and 24 weeks) with overweight females Jackicic et al (1995, 1997) demonstrated that subjects assigned to the short bout walking reported exercising on more days and for a greater total duration than the long bout walkers (Jakicic et al. 1995; Jakicic and Wing 1997). In the 1995 study, subjects in the short bout walking group were advised to walk for 10 minutes four times each day. Both self-reported exercise diaries and accelerometer data indicated that subjects chose to exercise in durations which were nearer to 15 minutes on three rather than four occasions per day. Indirect evidence for the superiority of lifestyle activity in terms of adherence comes from the study by Dunn et al (1999) described in 2.3.2 above. In a follow-up of subject, 18-months after a 6-month programme of either lifestyle or structured physical activity, the lifestyle group exhibited a much less marked decline in fitness and a smaller increase in weight than the structured physical activity group (Dunn et al. 1999). This suggests that subjects assigned to lifestyle physical activity were able to maintain their physical activity routines more effectively than the structured physical activity group. It seems therefore, that there is some limited evidence to suggest that short bouts may be a more efficacious way in which to achieve adherence to regular physical activity among sedentary individuals. There is however a possibility with random assignment that those allocated to the short bout groups were

more motivated to adhere to an exercise programme while those assigned to the long bout groups were less likely to adhere to any programme to which they were assigned. A comparison of adherence to both patterns of activity, in the same subjects, in a cross-over design trial may provide a clearer comparison of the effects of the two different patterns on adherence.

## 2.7 Summary

The preceding review indicates that the body of literature, which has compared the effects of a pattern of physical activity involving short bouts of exercise accumulated over the course of a day with more traditional approaches to exercise, is limited. The findings of this literature suggest that an accumulated approach, in line with current public health recommendations, may be suitable for enhancing fitness and decreasing fatness among previously sedentary individuals. There is also some evidence that such an accumulated pattern of exercise may favourably alter resting blood pressure and blood lipid profiles although the extent of such evidence is scanty. To date, few comparisons of the effects of these two approaches to exercise on psychological outcomes and no comparisons of the effects on postprandial lipaemia have been published.

The purpose of the research described in this thesis is to add to the limited body of empirical data examining the effectiveness of an accumulated approach to physical activity. More specifically, this thesis aims to compare the short- and medium-term effects of short accumulated bouts and longer single daily bouts, of an acceptable and accessible form of moderate intensity physical activity, on aspects of fitness, and physical and psychological health.

# CHAPTER 3

# GENERAL METHODS

Several of the procedures used in this thesis are common to two or more of the studies. This chapter describes these procedures and indicates where each was utilised.

## 3.1 Ethical approval

All four studies described in this thesis were approved by the Research Ethical Committee of the University of Ulster. Notice of ethical approval for the four studies described in this thesis is contained in Appendix A.

## 3.2 Subject recruitment

All of the subjects who volunteered to participate in the studies described in this thesis were recruited from within the University of Ulster and the surrounding local community by means of advertisement through posters, articles in university newsletters and electronic log-on messages.

## 3.3 Informed consent

Prior to beginning each study, all of the procedures involved and the potential risks and benefits of participation were fully explained to subjects. Each subject was then asked to sign a statement of informed consent (Appendix B).

## 3.4 Health Screening

Prior to the first exercise test in each study, subjects were required to complete a confidential health history questionnaire which requested details of personal and family medical history (Appendix C). Subjects with a family history of premature cardiac episode or with a personal history or symptoms of cardiac or systemic disease, hypertension, blood coagulation disorders, dyslipidemia, musculoseketal injury or acute illness were excluded from participation. In addition regular smokers and female

subjects who were pregnant or anticipated becoming pregnant were also excluded. Full descriptions of current medication was requested and subjects taking medication known to influence any parameters being considered in the study were also excluded. Specific exclusion criteria for each study are detailed in each experimental chapter.

### 3.5 Expired air analysis

In the studies described in Chapters 4, 6 and 7, oxygen uptake and carbon dioxide were determined from the analysis of expired air. During collection of the air, subjects breathed through a mouthpiece attached to a two-way non-rebreathing valve (2700 series, Hans Rudoph Inc. USA), while wearing a nose clip. Two different integrated expired air analysis systems were employed. In the study described in chapter 4, the valve was attached to 90 cm of wide bore lightweight tubing which had an internal diameter of 30 mm. Expired air was sampled every 60 seconds by an Oxycon-4 integrated system (Mijnhardt B.V. Holland). This system used an infra-red carbon dioxide analyser, a paramagnetic oxygen analyser and a dry gas meter to determine the volume and content of expired air. The gas analysis system was zeroed, calibrated and spanned before and after each test using certified reference gases (British Oxygen Co. Belfast). These reference gases were calibrated against a 'gold standard reference gas' (British Oxygen Co. Belfast). The gas meter was calibrated using 10 litres of air inserted using a 1 litre syringe. A full description of the calibration procedures is given in Appendix D. In the experimental studies described in Chapter 6 and Chapter 7, the two-way valve was attached to 100 cm of lightweight tubing with an internal diameter of 34 mm. Oxygen and carbon dioxide content was analysed using a Quinton Metabolic Cart (Quinton Instrument Co., USA), which used an infrared carbon dioxide analyser, a zirconia oxide high temperature furnace to analyse oxygen content and a pneumotachograph to determine the content of the expired air collected every 30 seconds in a mixing chamber. The analysers were calibrated by high, low and zero calibration gases (Vickers Medical , Belfast). The pneumotachograph was calibrated using a 3 litre syringe. A full description the calibration procedures are contained in Appendix D. During all tests, environmental temperature and humidity were determined using a digital thermohydrometer (Whatman International, England), and barometric pressure was determined using a Fortin barometer (F.D. & Co., Watford). The software associated with both gas analysis systems applied the Haldane

transformation to calculate the inspired gas volumes and all gas volumes were corrected to standard temperature and pressure for a dry gas (Consolazio et al. 1963).

### 3.6 Treadmill exercise tests

The exercise tests and all treadmill walking described in this thesis were performed on motorised treadmills. In the study reported in Chapter 4, the tests and some of the training bouts were performed on a Powerjog GM100 (Sport Engineering, Ltd., England). In the studies described in Chapters 6 and 7, a Powerjog M30 (Sport Engineering, Ltd., England) was used. Each subject was familiarised with treadmill walking or running prior to undertaking any exercise tests.

#### 3.6.1 Calibration of treadmills

The treadmills were calibrated for speed and gradient before and after each study. Calibration of the treadmills involved measuring the length of the treadmill belt and the time taken for it to complete 25 complete revolutions. A subject (body mass 60-75 kg) walked or ran on a level treadmill at three speeds that were typical of the speeds used in the corresponding study. The length of the belt (304 cm for Powerjog GM100 389 cm for Powerjog M30), was multiplied by the number of revolutions to calculate the distance travelled. This value was then divided by the time taken to determine the actual speed that the treadmill was travelling. This was compared with the speed indicated on the electronic display. The calibration procedure was then repeated using a 5% gradient to check the effect of altering gradient on treadmill speed. A digital spirit level (R.S. Components Ltd., England) with an adjustable graduated vial was used to calibrate the treadmill gradient. A subject ran at three gradients, 2.5%, 5.0% and 7.5%, while the spirit level was placed on the side of the treadmill close to and parallel with the treadmill belt. The treadmill calibration results for the study described in Chapter 7 of this thesis are presented, as an exemplar, in Table 3.1. The maximum variation noted between actual treadmill speed and indicated treadmill speed on any calibration performed in the series of studies reported in this thesis, was 3%. The maximum variation in treadmill gradient was 1.4%

Treadmill belt length = 3.04 m

Total distance travelled by the belt in 25 revolutions = 76m

| Electronic Display $km \cdot h^{-1}$ | Speed $m \cdot s^{-1}$ | Time (s) (25 revolutions) | | Calculated speed $m \cdot s^{-1}$ | | Difference % | |
|---|---|---|---|---|---|---|---|
| | | Pre | Post | Pre | Post | Pre | Post |
| Gradient = 0% | | | | | | | |
| 5 | 1.39 | 54.9 | 55.2 | 1.38 | 1.38 | 0.7 | 0.7 |
| 6 | 1.67 | 46.0 | 44.9 | 1.65 | 1.69 | 1.2 | 1.2 |
| 7 | 1.94 | 39.4 | 40.1 | 1.93 | 1.90 | 0.5 | 2.0 |
| Gradient = 5% | | | | | | | |
| 5 | 1.39 | 55.4 | 56.2 | 1.37 | 1.35 | 1.4 | 3.0 |
| 6 | 1.67 | 45.5 | 44.6 | 1.67 | 1.70 | 0.0 | 1.8 |
| 7 | 1.94 | 39.7 | 39.9 | 1.91 | 1.90 | 1.5 | 2.0 |

**Table 3.1** Results of treadmill speed and gradient calibration procedure before and after the study described in Chapter 7.

### 3.6.2 Maximal oxygen uptake test

Maximal oxygen uptake ($\dot{V}O_2$ max) was determined using an incremental exercise test which was divided into three minute stages. In the studies presented in Chapter 4 and Chapter 7 this involved subjects walking at a constant speed (range 1.6-1.8 $m \cdot s^{-1}$). Speeds were selected depending on subject comfort and an initial assessment of their ability to walk for a continuous period at the chosen speed. Treadmill incline was set at 0% for the first three minutes and increased by 2.5% every three minutes thereafter until volitional fatigue. Heart rate was monitored continuously throughout the test using a short range telemetry (Sportstester, Polar Electro, Finland). Rate of perceived exertion was measured at the end of each three minute stage using the 15 point Borg Scale (Borg 1973). At the end of the test the treadmill was stopped and the expired air collection apparatus was removed. The treadmill was returned to level and the treadmill was restarted to allow the subject to cool down gradually for a further five minute period at their chosen speed. In the study described in Chapter 6, the $\dot{V}O_2$ max test involved subjects running on the treadmill at a self selected speed (range 2.26 –3.89 $m \cdot s^{-1}$ ) with all other aspects of the protocol being identical to the walking tests. Four criteria were

used to determine attainment of $\dot{V}O_2$ max: a maximum heart rate within 10 beat·min$^{-1}$ of the age-predicted maximum (220 – age); a perceived rate of exertion of 19 or 20; an increase in oxygen uptake of less than 5% between the penultimate and final workloads; and a respiratory exchange ratio >1.11. In the majority of tests all four requirements were achieved, in every test at least three out of the four criteria were met. The degree to which each criterion was met is outlined in the methods section of each experimental chapter.

### 3.6.3 Submaximal incremental exercise test

In the studies described in Chapters 4 and 6 subjects completed a submaximal incremental exercise test to establish a relationship between oxygen uptake and work rate. Subjects were required to walk (Chapter 4) or run (Chapter 6), on a level treadmill, for a total of 16 minutes at a constant, individually selected, treadmill speed. Four speeds designed to elicit approximately 50%, 60%, 70% and 80% of the subject's $\dot{V}O_2$ max were chosen. Heart rate and Rating of Perceived Exertion were measured in the same manner as in the maximal tests described above. In the study described in Chapter 4, capillary blood lactate concentration was determined on duplicate 20 µl samples of capillary blood were obtained from a fingerprick, at the end of each stage of the test. Subjects continued to walk during blood sampling. Blood lactate concentration was plotted against oxygen uptake for each subject. Oxygen uptake, at a blood lactate concentration of 2 mmol·l$^{-1}$ was interpolated as a measure of endurance fitness.

### 3.6.4 2km walk field test

In the study described in Chapter 5, subjects performed the UKK 2 km walk field test (Oja et al. 1991). This involved subjects walking on a flat surface in an indoor sports hall for a distance of 2 km. Subjects were given the instruction to "walk the distance as fast as you can but do not risk your health" according to the recommended procedures. Heart rate was monitored continuously throughout the test using short-range telemetry (Sportstester, Polar Electro, Finland). The time taken to complete the walk, heart rate at the end of the walk, age and BMI were used to predict $VO_2$ max using gender-specific prediction models detailed in the methods section of chapter 5.

## 3.7 Anthropometry

Anthropometric measurements were obtained in all of the studies described in this thesis. These measurements provided a description of subjects and information about their body composition.

### 3.7.1 Height and body mass

Height and body mass was determined using digital scales and stadiometer (Seca delta, Model 707.) Body mass was measured to the nearest 0.1 kg with subjects in light clothing, without shoes. For height measurement, subjects stood with heels together against the metal upright. Subjects were instructed to stand up straight and breathe in. Height was measured using a horizontal ruler which rested on the subject's head, ensuring a perpendicular angle between the ruler and the upright to which it was attached. Height was recorded to the nearest 0.1 cm from the graduated scale on the upright. All measurements of height and body mass were taken in the morning to avoid diurnal variation. In the training studies reported in Chapters 4 and 5, body mass was determined at the same time on the morning after an overnight fast.

The body mass and height measures were used to calculate the commonly used ratio of body mass index (BMI). This was calculated according to the equation:

$$\frac{\text{body mass (kg)}}{\text{height (m)}^2}$$

### 3.7.2 Waist:Hip circumference ratio

Waist and hip circumferences were measured using standard techniques (Jones et al. 1986). Subjects, wearing underclothes only, stood in an upright position with stomach muscles relaxed. The circumference of the waist was measured at the point midway between the iliac crest and the twelfth rib. Hip circumference was determined at the level of the trochanters or the broadest part of the lower body. Both dimensions were measured to the nearest 0.1 cm using a flexible but inelastic fibreglass tape measure.

### 3.7.3 Skinfolds

Skinfold thicknesses were determined at four sites, i.e. biceps, triceps, subscapular and suprailiac, using a skinfold calipers (John Bull, British Indicators Ltd, England). Skinfold calipers were calibrated for thickness and pressure to manufacturer's specifications before and after each study. All measurements were made on the subject's left side with the subject standing in a relaxed upright position with the left arm hanging loosely at their side and the palm facing anteriorly. Skinfolds were located as follows:

Triceps- A vertical skinfold was measured at the midway point between the tip of the acromiom process of the scapula and the inferior portion of the olecranan process of the ulna, above the belly of triceps.

Biceps- A vertical skinfold was measured at the same level as the triceps but on the anterior aspect of the arm over the belly of tricep muscle.

Subscapular- The subscapular skinfold was measured at an inferior angle at the lowest point of the scapula, on a diagonal plane directed from the upper medial position, to the lower lateral position, following the normal cleavage of the skin.

Suprailiac- The suprailiac skinfold was measured at the midpoint between the last rib and the superior iliac crest, on a diagonal plane directed from the iliac crest toward the umbilicus, following the normal cleavage of the skin.

Skinfolds were isolated and maintained between thumb and forefinger and readings to the nearest 0.1 mm were taken two seconds after the pressure of the caliper was applied. Measurements were repeated twice at each site and the two readings were averaged. All four readings were added together to give a sum of four skinfolds which was used as an indicator of superficial adipose tissue thickness and a proxy measure body fatness. One experienced individual was used to determine skinfold thicknesses for all subjects in a given study. To check the precision of the measurement, the individual measuring skinfold thicknesses repeated measurements on three subjects, on five consecutive days, at the same time each day. Mean and standard deviation for the sum of four

skinfolds were calculated for each subject and a co-efficient of variation was calculated by dividing standard deviation by the mean and multiplying by 100. This precision estimation was made before each phase (pre-training and post-training) of the experimental studies described in Chapter 4 and 5. On all occasions the coefficient of variation was below 3.4%.

### 3.8 Blood pressure

In the two training studies described in Chapters 4 and 5, resting arterial blood pressure was measured before and after the brisk walking programmes. In the two studies described in Chapters 6 and 7, resting blood pressure was assessed as part of the health screening of all subjects. Duplicate measurements were made by an observer, who was blinded to the subject's group assignment, using a random zero mercury sphygmomanometer (Accoson MK2, England) calibrated to manufacturer's specifications. Subjects rested for 10 minutes in a supine (Chapter 4) or seated position (Chapters 5, 6 and 7) before the inflatable cuff was wrapped around the upper arm. The cuff was inflated to 180 mm Hg then slowly released. The observer listened for the soft tapping sounds and their subsequent muffling through a stethoscope (Magnatone, Eschmann, England) placed on the antecubital fossa over the brachial artery following the procedures described by Elliot and Stamler (1988).

### 3.9 Blood sampling

Capillary blood samples were collected in the study described in Chapter 4. Venous blood samples were collected in the studies described in Chapters 5, 6 and 7.

#### 3.9.1 Capillary samples

In the study described in Chapter 4, thumb-prick samples of capillary blood were collected during the final minute of each stage of the submaximal incremental exercise test. The skin was punctured using an Autoclix automatic lancet (Boehringer Mannheim U.K. Ltd., U.K.). After wiping the initial blood from the skin, two 20 µl samples were drawn into heparinised capillary tubes containing sodium fluoride and sodium nitrite (Analox, England). Samples were mixed gently by tilting the capillary tube. Blood was

subsequently analysed for lactate concentration between 3 and 4 minutes after collection.

### 3.9.2 Venepunctures

Venous blood samples, in the studies described in Chapters 5 and 6, were obtained by venepuncture of an antecubital or forearm vein. Subjects rested for five minutes in a seated (Chapter 6) or semi-supine position (Chapter 5) with the hand and forearm submerged in a basin of hot water to promote blood flow. A tourniquet was applied to the upper arm. Samples were obtained by an 18 gauge (45mm) needle attached to a 10 ml syringe (BOC Ohmeda, Sweden). Care was taken to minimise stasis.

### 3.9.3 Intravenous cannulation

In the study described in Chapter 7 venous blood samples were obtained from a forearm vein using an intravenous cannula (Venflon 2, 18G/45mm; BOC Ohmeda, Sweden). Prior to cannulation local anesthetic (1% lignocaine hydrochloride: Evans Medical Ltd., England) was injected subdermally at the cannulation site to reduce discomfort for subjects. A 10 cm connector tube and 3-way stopcock (Connecta, BOC Ohmeda, Sweden.) was then attached to the cannula. After cannulation the subject rested quietly for 10 minutes before the first blood sample was drawn. The cannula was kept patent throughout the day using 3-4 ml of 0.9% sodium chloride solution (Antigen Pharmaceuticals Ltd., Ireland) injected using positive pressure into the cannula after sampling . Prior to each sample 3-5 ml of fluid was withdrawn and discarded before a 10ml blood sample was obtained.

All blood samples in the studies described in Chapters 5 and 7 were drawn with subjects in a semi-supine position (seated on a reclined plinth with legs outstretched). In the study described in Chapter 6, samples were drawn from subjects in a seated position. In both cases the subject's position was standardised throughout the studies as changes in body posture are known to affect plasma volume (Rowell 1993).

### 3.9.4 Preparation of venous blood samples

Each venous blood sample obtained was divided between two 5ml plastic tubes (Sarsted, Germany). The first tube contained potassium ethylene diamine tetra acetic acid (EDTA) to prevent the blood from clotting. After mixing by gently inverting the tube, the sample was centrifuged within 15 minutes of collection. The second tube contained beads coated in a clotting activator and blood was left to clot for one hour before being centrifuged. Both samples were centrifuged at 4°C at a speed of 6000 rpm (3985g) for 15 minutes in a refrigerated centrifuge (Harreus Sepatech 17RS, Germany). Plasma or serum samples respectively, were then removed and stored in 1 ml microcentrifuge tubes within 10 minutes of centrifugation. These tubes were stored at –20°C until the end of each the study.

### 3.10 Blood analyses

A brief overview of the blood biochemistry undertaken in the studies described in this thesis is provided below, with an indication of which analyses were performed for each study. Full details of the assays used, the calibration methods and accuracy and precision data for each assay is included in Appendix E. All blood biochemistry (except for serum insulin assay) for the study described in chapter 6 was performed in the laboratory of the Department of Sports Science at Loughborough. The insulin assay for this study was performed in the Human Genetics radiochemistry laboratory at the Department of Human Sciences at Loughborough. With the exception of plasma insulin, blood analyses for the study reported in Chapter 7 was performed in the Northern Ireland Centre for Diet and Health at the University of Ulster at Coleraine. The insulin assay for the study reported in Chapter 7 and all other assays for the study reported in Chapter 5 were carried out at the Wellcome Laboratories in the Department of Medicine at the Queens University, Belfast.

#### 3.10.1 Haematocrit

Haematocrit was determined by centrifugation for each blood sample in the studies reported in Chapters 4, 6 and 7. Small duplicate samples of blood (40-50 µl) from the serum tubes were transferred into a heparinised capillary tubes and centrifuged at 12,000 rpm (14000g) for five minutes using a microcentrifuge. Haematocrit was

determined using a micro-haematocrit reader (Hawksley & Sons Ltd. England). Full details of the procedure are described in Appendix E.

### 3.10.2 Haemoglobin

In the studies reported in chapters 6 and 7 haemoglobin concentration was determined photometrically using a haemoglobinometer (Coulter Electronics Ltd , England). In the study reported in chapter 5, haemoglobin was determined by a dry chemistry assay using reagent strips and a Reflotron analyser (Boehringer Mannheim Ltd., U.K.). Full details of the assay are described in Appendix E. Plasma volume was estimated using haemoglobin concentration and haematocrit values according to the methods described previously (Dill and Costill 1974).

### 3.10.3 Blood lactate

During the incremental test used in the study described in Chapter 4, capillary samples were obtained from the finger. Two 20 µl samples were collected in capillary tubes containing flouride heparin and nitrite and were then mixed gently by tilting the capillary tube. Samples were analysed between 3 and 4 minutes after collection using a whole blood lactate analyser (GM7 Analox, England ). Full details of the assay are described in Appendix E.

### 3.10.4 Blood lipids

In the studies described in Chapters 5, 6 and 7 blood lipid concentrations were used as dependent variables. Plasma total cholesterol , triacylglycerol (TAG) and non-esterified fatty acid concentrations were determined spectrophotometrically by enzymatic methods (Boehringer Mannheim Ltd., U.K.). Spectrophotometric assays were performed on an automated centrifugal analyser (Cobas Bio, Roche Diagnostic Systems, U.K.). The analyser was programmed with the parameters for each assay and the absorbance readings were used to calculate concentrations. Full details of the assays are described in Appendix E.

### 3.10.5 Insulin

In the study described in chapter 6, insulin concentrations were determined on serum samples using by a solid phase $^{125}I$ radioimmunoassay (COAT-A-COUNT Insulin, Diagnostic Products Co, USA). Radioactivity was measured in an automated gamma counting system (Cobra II, Packard Instrument CO. Inc. U.S.A.). In the study described in Chapter 5 the insulin concentration was determined on plasma samples using a liquid phase competitive binding $^{125}I$ radioimmunoassay (Amersham Pharmacia Biotech.). Full details of the assays are described in Appendix E.

### 3.10.6 Glucose

Glucose concentrations were determined spectrophotometrically, by enzymatic methods (Boehringer Mannheim Ltd., U.K.). Spectrophotometric assays were performed on an automated centrifugal analyser (Cobas Bio, Roche Diagnostic Systems, U.K.). Full details of the assay are described in Appendix E.

### 3.10.7 Accuracy and precision

The accuracy of the assays described in this thesis were monitored using quality control sera. With the exception of lactate, haematocrit and haemoglobin which were performed on fresh blood, all samples from one subject were analysed in a single batch. The intra-assay and inter-assay coefficients of variation for each type of analysis is shown in Appendix E.

## 3.11 Statistical analysis

Descriptive statistics were used to summarise data. For variables which describe subjects at baseline measurement, the values reported are mean $\pm$ standard deviation (s.d.). Change over time, summary measures and all other values reported are mean $\pm$ standard error of the mean (s.e.m.) unless otherwise stated. A 5% level of confidence

has been adopted throughout unless otherwise stated. In the training study described in chapter 4 change from baseline was used as a derived measure of alteration in specified parameters. In studies involving postprandial responses (chapters 6 and 7) the area under the plasma TAG concentration versus time curve is used as an index of postprandial lipaemia. The usefulness of this summary measure in interpreting uptake from blood has previously been described (Altman 1991). Area under the curve was determined using the trapezium rule. In some subjects, fasting plasma triacylglycerol concentrations showed a degree of variation. Such differences are known to influence postprandial lipaemia and therefore the incremental area under the curve was used [O'Meara , 1992 #209]. The incremental area under the curve was taken as the area under the curve minus the concentration in the fasted state, extrapolated over the observation period. The statistical techniques employed, are described by Altman (1991). The specific statistical tests used are described in each experimental chapter.

In the study described in Chapter 4 data analysis software (Statview 4.5, Abacus Concepts Inc., U.S.A.) was used to perform statistical tests. In the studies described in Chapters 6 Microsoft Excel (Microsoft Excel 97, Microsoft corporation, U.S.A.) and Graphpad Prism (Version 2.01, Graphpad Software Inc., U.S.A.) were employed. In the studies described in Chapter 6 and 7 Minitab (version 12) and was used.

## CHAPTER 4

# THE EFFECTS OF SHORT AND LONG BOUTS OF BRISK WALKING ON FITNESS, FATNESS AND BLOOD PRESSURE IN PREVIOUSLY SEDENTARY WOMEN

## 4.1 INTRODUCTION

For many years regular physical activity has been positively associated with a reduced risk of contracting many of the diseases most prevalent in Western societies (Blair 1993). Even moderate intensity exercise training can result in an amelioration of many metabolic and physiological parameters and thereby may reduce the risk of diseases such as coronary heart disease, non-insulin dependent diabetes, cardiovascular disease and obesity (Fentem 1988). Despite the associations between physical activity and health, the quantity and quality of physical activity necessary to achieve such benefits is still more a matter of debate than of scientific certainty.

Despite public health initiatives highlighting the effect of physical activity on health, only a small percentage of the UK population currently takes this amount of exercise (Allied Dunbar 1992, MacAuley et al. 1994) Lack of time is the reason most often cited for inadequate levels of physical activity (Dishman 1990). Finding ways of fitting physical activity into lifestyle without having to put aside extended periods of time may be an effective way of increasing participation in and adherence to exercise programmes. Indeed, encouraging participation in shorter but more frequent bouts of physical activity has become a central feature of recent physical activity recommendations (ACSM 1994 and 1998). However such a recommendation was based on indirect evidence from epidemiological studies (Paffenbarger et al. 1983) and the evidence from a limited number of empirical studies (Ebisu 1985). One of these empirical studies, found substantial improvements in $VO_2$ max in men jogging in one long (30 minutes) bout versus three short (10 minutes) bouts per day on five days per week, for eight weeks (mean increase of 7.5% and 13.8% in short and long bouts respectively) (DeBusk et al. 1990). The study did not employ a control group however and it is therefore difficult to attribute the improvements in fitness exclusively to the exercise intervention with confidence. Similar findings have been reported when

running was used as the exercise modality and the training distance was divided into two and three intermittent bouts (Ebisu 1985; DeBusk et al. 1990). The use of men in both studies may limit the extent to which the findings can be generalised to women who in most population studies are a more sedentary group. (Allied Dunbar 1992; MacAuley et al. 1994).

Walking is a popular form of moderate intensity physical activity, which has been widely reported to improve fitness females (Juneau et al. 1987, Aldred et al.1995, Duncan et al. 1991, Hardman and Hudson 1994, Santiago et al. 1987, Jetté et al. 1988) and decrease body mass or fatness (Aldred et al. 1995, Ready et al. 1996). However the duration of bouts in these studies exceeded 20 minutes continuous walking and so cannot contribute to a rationale for the efficacy of the accumulation of physical activity in intermittent short bouts most recently advanced (Pate et al. 1995).

Two studies have, more recently, added support for the notion that accumulated bouts of brisk walking may have the potential to enhance fitness (Jakicic et al. 1995, Snyder et al, 1997). In both studies the subjects were obese females and the duration of the intervention was 20 and 32 weeks respectively. Whether a shorter programme of accumulated bouts of brisk walking would be an effective means of enhancing fitness in a sedentary but otherwise normal female population is not clear.

The purpose of this study, therefore, was to investigate whether similar training effects can be achieved by short intermittent bouts of brisk walking as by long continuous bouts, among previously sedentary women. By using the same total exercise time and same relative exercise intensity the study aimed to determine whether a recommendation for such 'accumulated' physical activity can be advanced for this group on the basis of empirical evidence.

## 4.2 METHODS

### 4.2.1 Study design

This study was a controlled, exercise intervention trial conducted with the approval of the University of Ulster Research Ethical Committee. All measurements were made at baseline and after 10 weeks randomised assignment to a short walks (SHORT), long walks (LONG) or control (CON) group

### 4.2.2 Subjects

Forty-seven women, aged between 31 and 57 years were recruited from the university population and the local community by means of advertisements. All subjects had sedentary occupations and reported no involvement in regular physical activity (< 1 x 20 minute bout per week) during the preceding six-month period. The requirements of the study were explained to all subjects and informed consent was received before testing began (Appendix B). Subjects completed a pre-test screening questionnaire regarding their medical history (Appendix C). Subjects reporting a history of cardiovascular disease, diabetes, musculoskeletal injury, or reliance on any medication, were excluded from participation in the study. After the first visit to the laboratory, subjects with resting systolic blood pressure above 150mmHg, a resting diastolic blood pressure above 95 mmHg or a Body Mass Index above 30 $kg \cdot m^{-2}$ were also excluded from the study. In total, 47 subjects began the study. Thirteen women dropped out during the course of the study (5 CON, 4 SHORT, 4 LONG) due to loss of interest (4 CON, 1 SHORT, 1 LONG), illness (1 CON, 0 SHORT, 1 LONG), and time pressures (0 CON, 3 SHORT 3 LONG). Data are reported for the 34 subjects who completed the study (10 CON, 12 SHORT, 12 LONG).

### 4.2.3 Baseline measures

Height and body mass were determined using a scales and stadiometer according to the method described in chapter 3.6. Duplicate measurements of resting blood pressure, in a lying position, after 5 minutes rest, were made by the same experienced observer using a mercury sphygmomanometer as described in 3.8. Skinfold thicknesses at 4 sites

(biceps, triceps, subscapular and suprailiac) were taken by the same experienced experimenter using the technique described in 3.7.3. Hip and waist circumferences were measured according to established methods described in Chapter 3.7.2.

### 4.2.4 Exercise tests

After habituation to treadmill walking and the expired air collection apparatus all subjects took part in two exercise tests. First, an incremental walking test was performed to determine maximal oxygen uptake. Walking speed was determined during habituation sessions with subjects instructed to "walk as briskly as possible" on the treadmill without having to alter gait from walking to jogging. Once selected, walking speed remained constant for the subject throughout all the exercise tests. In this study treadmill test speeds varied from 1.53 $m \cdot s^{-1}$ to 1.78 $m \cdot s^{-1}$. A full description of the maximal test including the criteria used for determination of $\dot{V}O_2$ max can be found in Chapter 3.6.2. In 62 of the 68 maximal tests all 4 of the criteria were achieved with the remaining 6 tests fulfilling 3 out of the 4 criteria.

A second incremental walking test, used the same individually-selected treadmill speed and four gradients designed to elicit approximately 50%, 60% 70% and 80% of each subject's maximal oxygen uptake. The test is described fully in Chapter 3.6.3. At the end of each stage of the test, capillary blood samples were obtained from a fingerprick and analysed for lactate concentration as described in Chapter 3.9.3. Blood lactate concentration was plotted against oxygen uptake for each subject and oxygen uptake at $2mmol \cdot l^{-1}$ of blood lactate was interpolated as a measure of endurance fitness (Hardman et al. 1992).

### 4.2.5 Training programme

Following the exercise tests, subjects were randomly assigned to SHORT, LONG or CONTROL groups. Based upon the highest heart rate recorded during the maximal treadmill test a heart rate "target zone" corresponding to 70 - 80% of maximum heart rate was established for each walker. Most walkers were given a heart rate monitor (PE4000 Sports Tester, Polar Electro, Finland) and instructed in its use. Six subjects, who were proficient in determining their heart rate by palpation of the carotid or radial

artery for 10 seconds, were asked to monitor their heart rate at the end of each walk. All subjects were asked to walk briskly, keeping their heart rate within the prescribed zone on 5 days per week for 30 minutes duration each day. Women assigned to the LONG group (n=12) completed the 30 minutes in one continuous bout of walking whilst those assigned to the SHORT group (n=12) completed the 30 minutes in three, 10 minute bouts with a minimum of 4 hours between bouts. All subjects trained for a period of ten weeks. Control subjects were asked to maintain their usual lifestyle for the duration of the study. Control subjects were offered the opportunity to take part in a brisk walking programme at the end of the ten week period. All subjects were asked to make no changes to their normal diet.

Training subjects were given weekly activity diaries in which to record the duration of each brisk walk and their heart rate during the walks. Each week subjects performed one of their training bouts under supervision. During this training session, heart rate was recorded but the heart rate monitor display was concealed so that subject's heart rate response to self-paced 'brisk' walking, without feedback, could be checked. Subjects were asked to walk at their usual training speed. After the session, subjects were given feedback on the heart rate achieved and thus the appropriateness of the intensity of the walk. Supervised sessions also allowed confirmation that those subjects who were monitoring their own heart rate, without the use of a monitor, were achieving the required exercise intensity.

### 4.2.6 Blood analysis

Blood lactate concentration during the second treadmill test was determined using fresh samples on a whole blood analyser (Analox GM7). On each day, the analyser was calibrated before use, with two standards (3.0 and 5.0 $mmol \cdot l^{-1}$). Precision was evaluated by analysing ten aliquots of the same venous blood sample. Coefficients of variation for these samples were equal to or below 2.7%. Accuracy was ensured using two quality control sera (1.6 and 2.8 $mmol \cdot l^{-1}$) frozen (at -20° C) in aliquots before the study. One aliquot of each was thawed and assayed on each test occasion. Coefficients of variation on all test occasions were below 3.2% (see Appendix E)

### 4.2.7 Statistical analysis

Changes in each parameter from pre-training to post-training measurement were used as a summary measure of the response over time for each subject (Bland 1995). Mean changes were compared using one-way analysis of variance (factorial) with Tukey post-hoc tests to identify significant differences in response between groups. Tests were considered statistically significant at the 5% level.

## 4.3 RESULTS

### 4.3.1 Subjects

The physical and physiological characteristics at baseline for all three groups and the subjects who did not complete the study are shown in Table 4.1. At baseline measurement, there were no significant differences for any of the measures between groups. In addition, the mean self-selected treadmill test speeds of 1.69, 1.63, 1.67 and 1.66 $m \cdot s^{-1}$ for long walks, short walks, control and drop-out groups respectively, did not differ between groups.

| | CON n=10 | SHORT n=12 | LONG n=12 | Drop-outs n=13 |
|---|---|---|---|---|
| Age (years) | 47.3 (4.1) | 44.8 (8.4) | 48.0 (5.5) | 44.2 (5.6) |
| Height (cm) | 164.2 (2.1) | 164.6 (1.9) | 160.9 (2.2) | 163.4 (2.3) |
| Body Mass (kg) | 72.5 (13.9) | 66.5 (9.7) | 66.7 (8.3) | 65.6 (6.5) |
| Body Mass Index ($kg.m^{-2}$) | 26.5 (4.6) | 25.1 (3.4) | 25.8 (2.9) | 24.7 (4.1) |
| Sum of 4 skinfolds (mm) | 66.9 (6.3) | 72.4 (6.0) | 66.8 (6.5) | 66.5 (7.9) |
| Waist circumference (cm) | 82.3 (8.5) | 78.2 (7.1) | 76.8 (6.9) | 80.1 (7.4) |
| Hips circumference (cm) | 105.5 (12.9) | 98.8 (8.0) | 101.3 (7.3) | 100.9 (10.1) |
| Ratio of circumferences at waist and hip | 0.79 (0.06) | 0.79 (0.02) | 0.76 (0.04) | 0.79 (0.07) |
| Systolic blood pressure (mmHg) | 128.6 (13.3) | 125.5 (10.8) | 124.2 (11.1) | 123.3 (9.9) |
| Diastolic blood pressure (mmHg) | 79.4 (6.1) | 76.5 (7.1) | 73.3 (5.6) | 80.4 (7.2) |
| $VO_2$ max ($ml.kg^{-1}.min^{-1}$) | 25.0 (4.0) | 27.8 (3.2) | 28.1 (3.0) | 26.4 (3.3) |
| $VO_2$ at 2 $mmol \cdot l^{-1}$ blood lactate ($ml \cdot kg^{-1} \cdot min^{-1}$) | 19.1 (3.1) | 18.9 (2.5) | 19.3 (1.8) | 18.4 (3.4) |

**Table 4.1** Mean (s.d.) physical characteristics of CON (n=10) SHORT (n=12) LONG (n=12) groups and subsequent dropouts (n=13) at baseline.

### 4.3.2 Adherence to training programme

Subjects in the SHORT group completed a mean of 128 (range 114-150) out of a possible 150 (3 per day x 5 days per week x 10 weeks) walks, i.e. 85% of sessions. Subjects in the LONG group completed a mean of 44 (range 39-50) out of a possible 50 (1 per day x 5 days x 10 weeks) walks, i.e. 88% of sessions.

### 4.3.3 Training Intensity

Mean heart rate during training sessions was 131 (±2) beat·min$^{-1}$ for the SHORT group and 136 (±3) beat·min$^{-1}$ for the LONG group. This corresponded to 73% and 75% of maximum heart rate respectively and did not differ between groups.

### 4.3.4 Changes in body composition

Body mass decreased with training from 66.5 to 64.8 kg in the SHORT group, increased from baseline measurement of 72.5 to 73.2 kg post training in the CON group, and showed a non-significant change from 66.7 to 65.8 kg in the LONG group. There was a decrease in the sum of skinfold thicknesses in the SHORT group and LONG group, with no significant alteration in the CON group. Both training groups demonstrated a significant decrease in both waist and hip circumference from baseline to post measurement but only the SHORT group had a decrease in the ratio of circumferences at the waist and hip. No changes in mean hip or waist circumference were noted in the CON group. When the anthropometric changes from baseline to post test were compared between groups, despite significantly greater alterations between the training groups and the CON group on all variables, except the ratio of circumferences at waist and hip, there were no differences between the changes induced by the two training regimens. Changes in body composition from baseline to post-training are presented in table 4. 2.

| | CON | SHORT | LONG |
|---|---|---|---|
| Body Mass (kg) | +0.6 (0.2) * | -1.7 (0.5) *^ | -0.9 (0.6) |
| Sum of 4 Skinfolds (mm) | +2.6 (0.8) * | -3.3 (1.0) *^ | -2.8 (1.1) *^ |
| Waist (cm) | +0.6 (0.3) | -3.0 (0.7) *^ | -1.8 (0.7) *^ |
| Hips (cm) | +0.8 (0.4) | -1.7 (0.8) * | -3.3 (1.4) *^ |
| Ratio of circumferences at waist and hip | +0.23 (0.08) | -0.66 (0.2) * | -0.33 (0.2) |

* change from baseline to post-test
^ change different from change in controls

**Table 4. 2.** Mean (s.e.m.) changes in physical characteristics of CON (n=10) SHORT (n=12) LONG (n=12) groups from baseline to post-test.

When the alterations in body composition were considered in terms of individual response it is apparent that in the exercise groups subjects who had the highest pre-training body mass had the greatest reduction in body weight after the walking programme. Individual changes in body mass and sum of four skinfolds are illustrated in Figure 4.1 and 4.2 respectively.

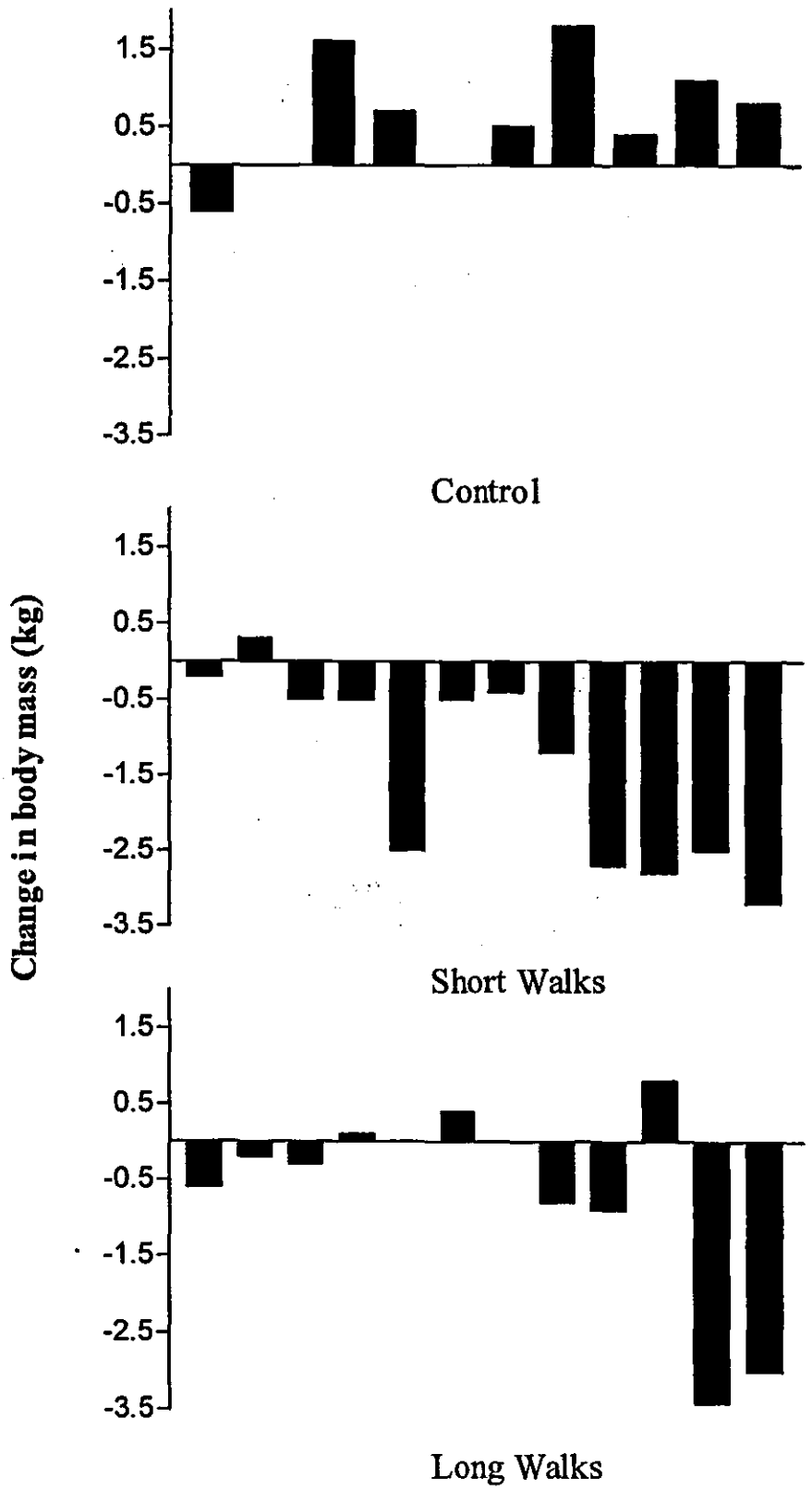


**Figure 4.1** Changes in body mass, after 10 weeks, for subjects assigned to CON (n=10) SHORT (n=12) and LONG (n=12) groups. Subjects are arranged in ascending order of pre-training body mass

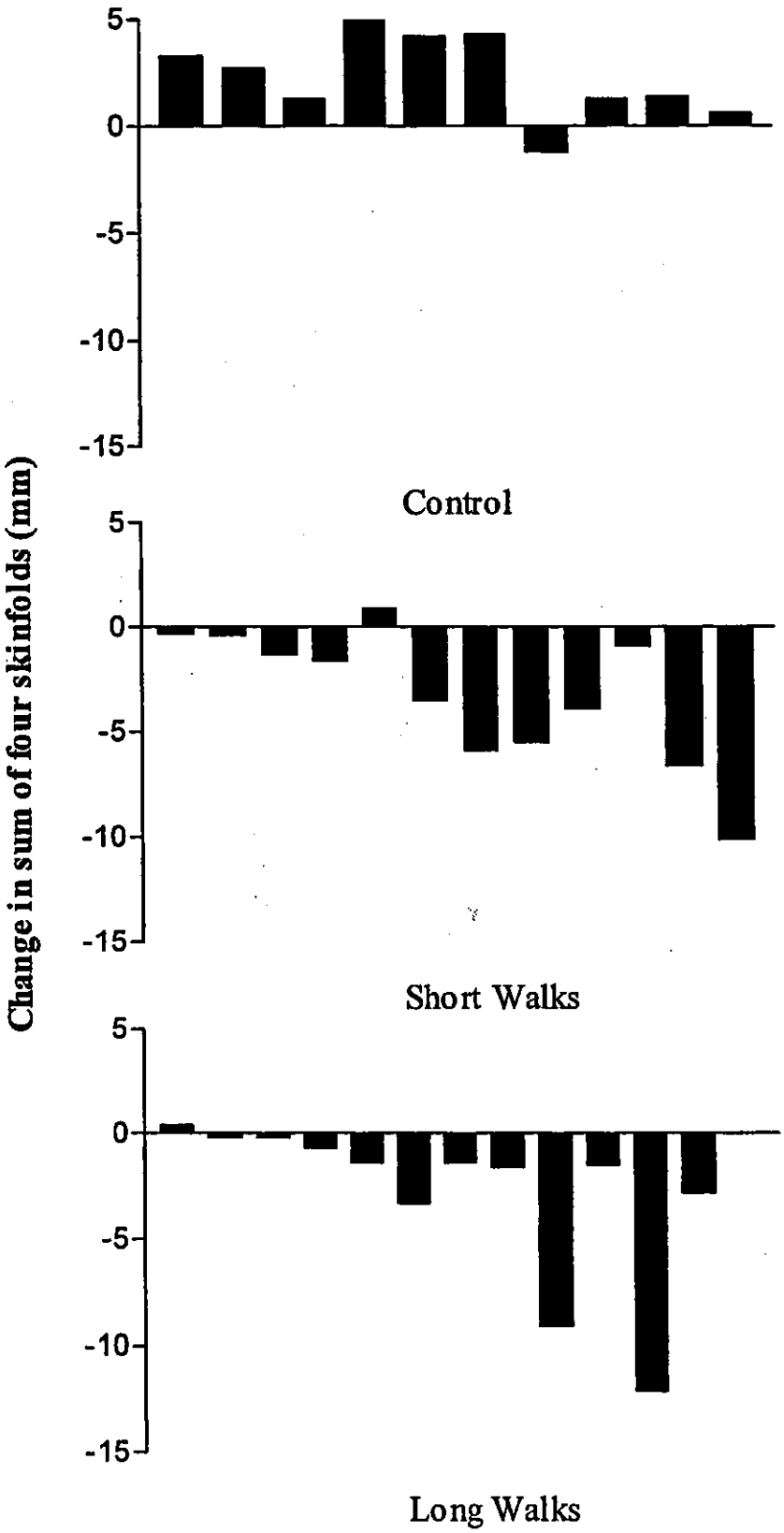


**Figure 4.2** Changes in the sum of four skinfolds, after 10 weeks, for subjects assigned to CON (n=10) SHORT (n=12) and LONG (n=12) groups. Subjects are arranged in ascending order of pre-training skinfolds

### 4.3.5 Changes in blood pressure

Resting systolic blood pressure decreased in both training groups with no alteration in the CON group. The changes in systolic blood pressure of -7.4 (±2.1) -4.6 (±1.7) mm Hg in the SHORT and LONG groups respectively, were not, however, different between training groups. No alterations in diastolic blood pressure were noted in any group. Changes in systolic blood pressure were greatest among those with initially elevated levels. Individual alterations in systolic blood pressure are illustrated in Figure 4.3.

### 4.3.6 Changes in fitness

Maximum oxygen uptake increased in both the SHORT group and the LONG group with no change in the CON group. There was no significant difference in the magnitude of the improvement between the two training groups. Mean oxygen uptake at a blood lactate concentration of 2 $mmol \cdot l^{-1}$ increased from 18.9 (0.72) $ml^{-1} \cdot kg^{-1} \cdot min^{-1}$ to 21.5 (0.65) $ml^{-1} \cdot kg^{-1} \cdot min^{-1}$ in the SHORT group and from 19.3 (0.5) $ml^{-1} \cdot kg^{-1} \cdot min^{-1}$ to 22.8 (0.68 $ml^{-1} \cdot kg^{-1} \cdot min^{-1}$ in the LONG group. The alteration in oxygen uptake (at the two reference points) was significantly greater in the two training groups than in the CON group but there was no difference between training groups. Changes in maximal oxygen uptake and oxygen uptake at a blood lactate of 2 $mmol \cdot l^{-1}$ are illustrated in Figure 4.4.

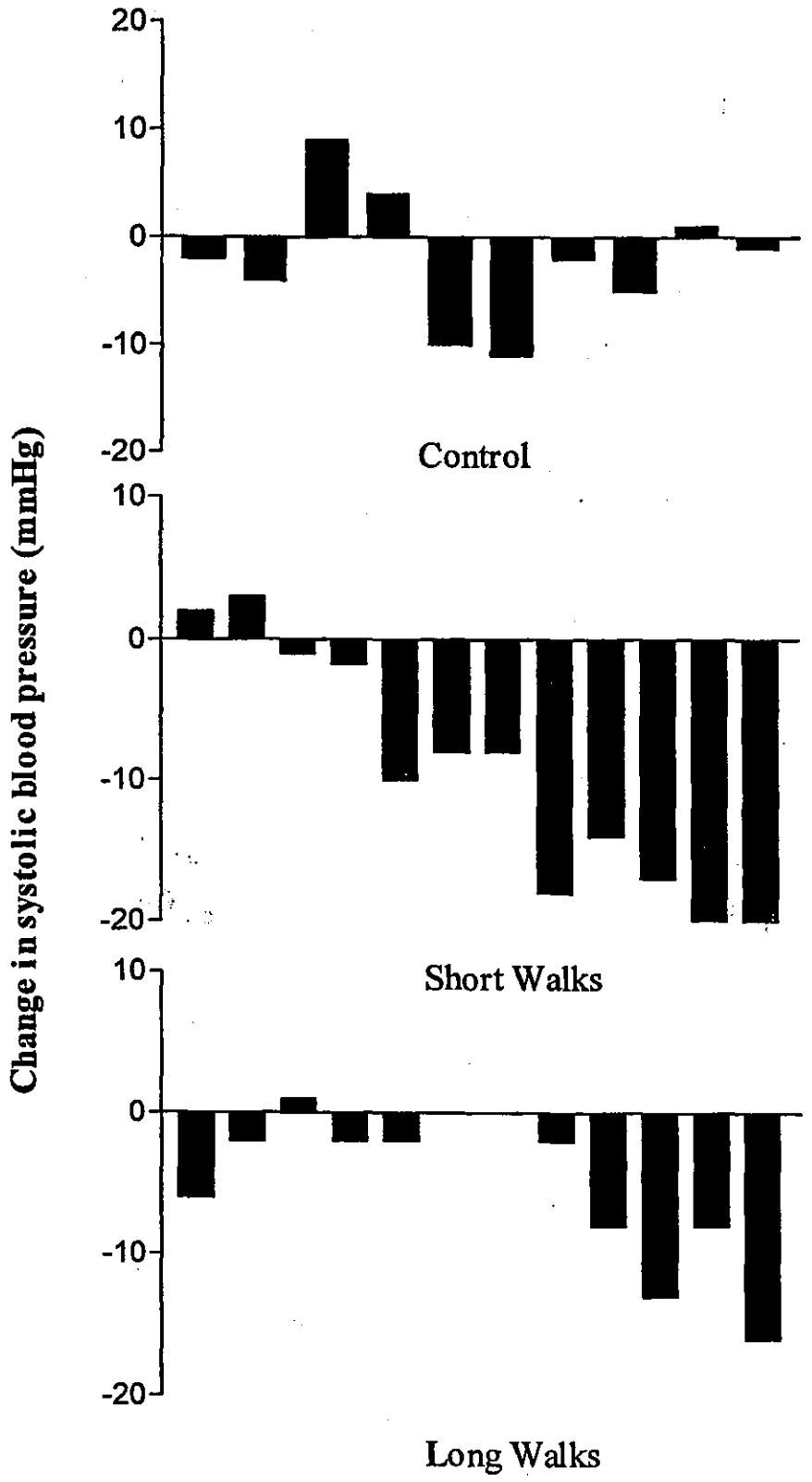


**Figure 4.3** Changes in resting systolic blood pressure, after 10 weeks, for subjects assigned to CON (n=10) SHORT (n=12) and LONG (n=12) groups. Subjects are arranged in ascending order of pre-training systolic blood pressure

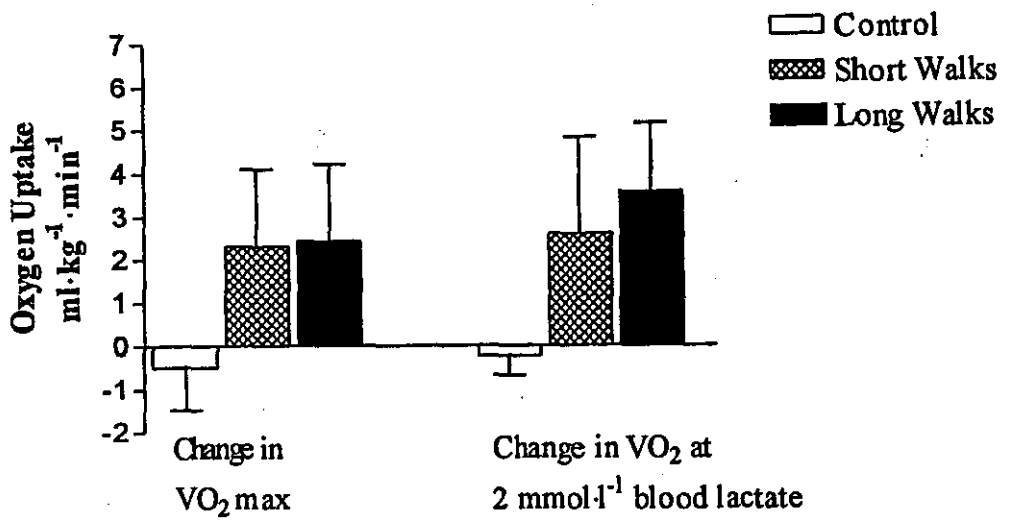


**Figure 4.4** Changes (s.e.m.) in maximal oxygen uptake and oxygen uptake at a blood lactate concentration of 2mmol·l$^{-1}$ in CON (n=10) SHORT (n=12) LONG (n=12) groups after 10 weeks of training. (change in $VO_2$ max main effect treatment p=0.03) (change in blood lactate at 2mmol·l$^{-1}$ main effect treatment p=0.04)

During the standard submaximal treadmill test, both training groups showed attenuated heart rate and blood lactate responses at all four stages but no change in the either variable was observed in the control group. When the change in heart rate and blood lactate response from baseline to post training was considered however, there were no significant differences between the magnitude of these changes between training groups. The blood lactate and heart rate responses to the submaximal test for each group are shown in Figure 4. 5 and 4.6 respectively.

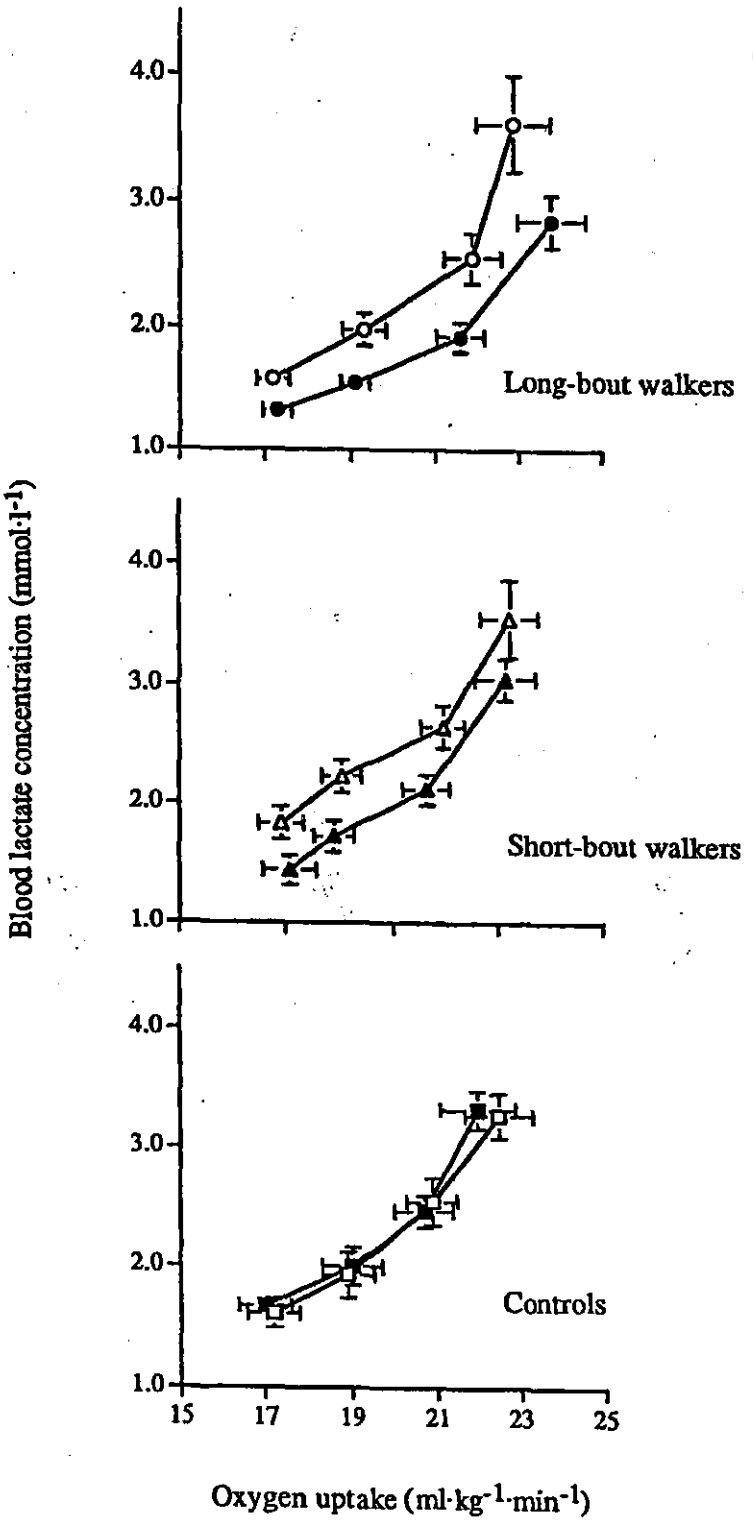


**Figure 4.5** Mean blood lactate concentrations (s.e.m.) during submaximal treadmill walking at baseline (open symbols) and 10 weeks later (solid symbols) in CON (n=10) SHORT (n=12) and LONG (n=12) groups.

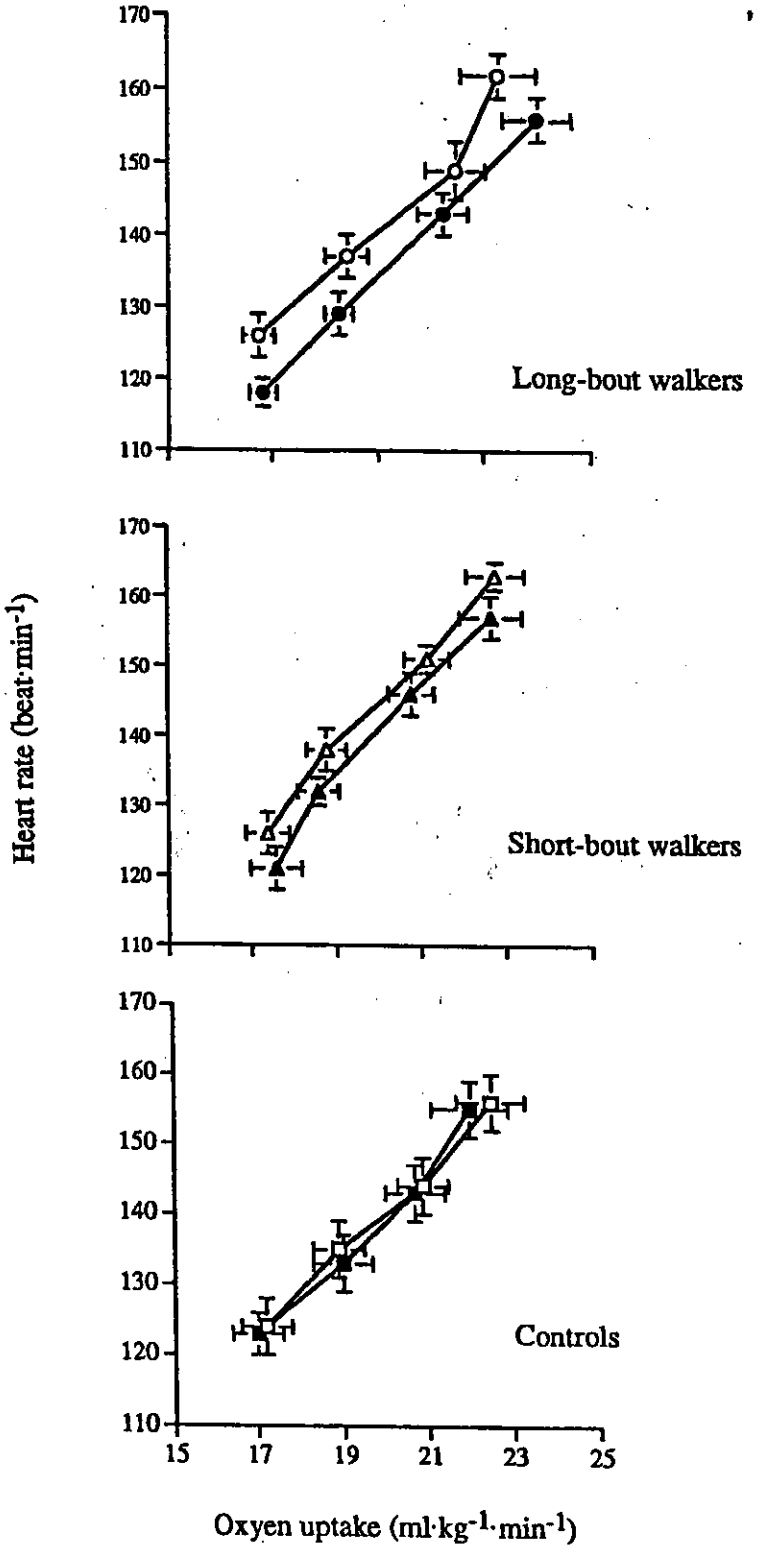


**Figure 4.6** Mean heart rate (s.e.m.) during submaximal treadmill walking at baseline (open symbols) and 10 weeks later (solid symbols) in CON (n=10) SHORT (n=12) and LONG (n=12) groups.

## 4.4 DISCUSSION

The study described in this chapter compared the effects of 10 weeks of training using either short or long bouts of brisk walking on physical fitness, blood pressure and body composition of previously sedentary women.

Random allocation to two training and one control group resulted in groups that did not differ in terms of age, indices of body composition and endurance fitness or resting blood pressure before training. When compared to recent surveys of the Northern Irish population, these subjects were of a similar weight but fatter (total skinfolds 68.7 mm) and lower in $VO_2$ max (27.0 $ml \cdot kg^{-1} \cdot min^{-1}$) than other females of their age (total skinfolds 65.5 mm, $VO_2$ max 29.5 $ml \cdot kg^{-1} \cdot min^{-1}$) (MacAuley et al. 1994). These differences are perhaps not unexpected since only sedentary subjects were included in the study and they probably reflect the differences between the overall population and the large population subgroup of individuals who are habitually inactive.

Adherence to the training programme in this study was good with subjects completing 85% and 88% of short and long walks respectively. Subjects in short and long walk groups exercised at an intensity of 73% and 75% of maximum heart rate respectively. This represents moderate to vigorous activity or an intensity described as 'hard' by the most recent exercise guidelines (Pollock et al. 1998).

The main finding of this study is that if the intensity and total duration of a brisk walking programme remain constant, splitting the duration of traditional exercise prescriptions and performing shorter walks throughout a day does not alter its physiological effects.

Training resulted in similar increases of 0.11 $l \cdot min^{-1}$ and 0.14 $l \cdot min^{-1}$ (8.3 and 8.6%) in maximal oxygen uptake in the SHORT and LONG groups respectively and there was no change in the maximal oxygen uptake of the CON group. Even when the changes in body mass are considered these improvements in functional capacity remain significant (2.3 and 2.4 $ml \cdot kg^{-1} \cdot min^{-1}$). The mechanisms underlying such changes in maximum oxygen uptake have been well established and are probably the result of both an

improvement in cardiovascular function, through an enhanced maximum cardiac output and an increased ability of skeletal muscle to extract and use oxygen to meet energy demands through enhanced oxidative metabolism. (Ekblom 1969). Walking at this intensity appears to be effective in providing an training stimulus among sedentary individuals (Porcari et al. 1987).

The effectiveness of brisk walking to increase maximal oxygen uptake has been established for over 25 years (Pollock et al. 1971). The magnitude of the change in $VO_2$ max in this study is similar to that reported previously for brisk walking programmes with sedentary males (Stensel et al. 1993) and females(Juneau et al. 1987) (Juneau et al. 1987, Duncan et al. 1991, Hardman and Hudson 1994), but somewhat below those reported by others (Santiago et al. 1987, Jetté et al. 1988). The variation in the alteration in $VO_2$ max with a programme of brisk walking reported in the literature is large, ranging from no increase to a 28% improvement (Pollock et al. 1971). Although it tempting to use the length of the intervention as to explain of these variations, the effects of training are likely to occur early in such interventions with sedentary individuals. MacRae and co-workers showed no additional improvements in $VO_2$ max when 12 weeks of brisk walking was followed by a further period of 10 weeks at the same intensity (MacRae et al. 1996). It is perhaps more plausible that the differences in the magnitude of the alterations in endurance fitness which have been observed following brisk walking training, are due to either a difference in the exercise intensity achieved or a difference in the initial condition of the subjects recruited.

In the present study the differential increase in oxygen uptake reported by DeBusk and colleagues (13.9% in 30 min walks 7.6 % in 10 min walks) was not replicated (DeBusk et al. 1990). This may however be explained by the tendency for subjects in the 'long walk' group of the deBusk study to work above target heart rate values for 33% of the time compared to 17% of time spent above target heart rates (65-75%) in the 10 minute group. By contrast, neither group of walkers in the present study spent more than 9% of any session above the target heart rate range. This meant that the training stimulus in the two walking groups was more similar. Previous studies have shown greater increases (17%) in maximal oxygen uptake in subjects training at 75-87% of peak heart rate and more modest increases (8%) in subjects training at a lower intensity (60-72%) (Gossard et al. 1986). In the present study subjects spent no more than 9% of any brisk walking

session (10 min or 30 min walks) above target heart rates (70-80% maximal heart rate). The tendency for long bout joggers in the deBusk study to spend a greater percentage of their time above target heart rates may represent a practical problem. In untrained individuals the progressive rise in heart rate during an exercise bout may only be opposed by a reduction in exercise intensity. In a group with low $VO_2$ max any reduction in jogging speed may necessitate subjects changing their gait to walking. In order to comply with the requirements of jogging, subjects may have been forced to exercise at a higher heart rate than intended by the researchers.

Normative data for UK populations suggest that the subjects in this study had slightly lower mean $VO_2$ max (25.0 -28.1 $ml.kg^{-1}.min^{-1}$) than their counterparts in Northern Ireland and England populations of 29.5 and 31.9 $ml.kg^{-1}.min^{-1}$ respectively (MacAuley et al. 1994, Allied Dunbar, 1992 #178). This indicates a possible increased potential for benefit from a programme of physical activity for this group of sedentary subjects (Haskell 1994a). Indeed, such subjects, with lower maximal oxygen uptakes may be more able to attain target heart rates above 70% of maximal heart rate, while walking, than subjects with a higher functional capacity (Porcari et al. 1987).

Following training, both exercise groups showed an attenuated heart rate response to submaximal exercise. Heart rate response to submaximal work was lower at all 4 stages in both exercise groups after training but remained unchanged among controls. In addition, heart rate was lower for a given oxygen uptake after training in both exercise groups. This bradycardia in response to a submaximal workload is widely reported for many forms of endurance training including brisk walking (DeBusk et al. 1990, Stensel et al. 1993, Hardman and Hudson, 1994), and may be due to increased parasympathetic activity and perhaps a decreased sympathetic discharge (Stromme and Ingjer 1982). The decrease in heart rate during submaximal exercise in the present study is in agreement with that of deBusk and colleagues suggesting functional adaptations of the cardiovascular system associated with increased fitness.

In a similar manner to these alterations in heart rate response, both exercise groups also exhibited a reduced blood lactate response to an incremental exercise test following training.

These alterations are similar to those cited in the literature for running training and are thought to include structural changes such as increased capillarisation, myoglobin concentrations and mitochondrial density, as well as biochemical alterations such as increased activity of oxidative enzymes (Holloszy 1988). These changes are attributed local adaptations of the skeletal musculature in response to a given exercise intensity. Brisk walking employs similar muscles to running and the training intensity employed in this study is similar to that achieved by running training among fitter individuals. It seem reasonable therefore to suggest that the alterations reported in the literature, may also be responsible for the decreased blood lactate response to submaximal exercise found, after training, in the present study. Many of the most rapid changes in response to an exercise training programme are known to take place at a cellular level, namely alterations in the activity of oxidative enzymes (Henriksson and Reitman 1977). These changes increase the degree to which the energy demands of a given workload may be met by aerobic metabolism and may therefore be an indication of improvements in endurance fitness

In addition to the lactate response to each stage of the submaximal exercise test, oxygen uptake at a 2 $mmol \cdot l^{-1}$ of blood lactate was interpolated as a measure of endurance fitness (Hardman et al. 1992). Oxygen uptake at a blood lactate concentration of 2 $mmol \cdot l^{-1}$ increased by 13.8% and 18.4% in the SHORT and LONG groups respectively with no change in the CON group. These increases in oxygen uptake, at a reference blood lactate concentration, are similar to those reported previously for a programme of brisk walking (Hardman and Hudson 1991). There was no change in blood lactate in response to the submaximal test in controls. This decreased accumulation of blood lactate at a given workload or given oxygen uptake is similar to that reported in the literature (Hardman and Hudson 1994) and may be indicative of changes in the oxidative capacity of skeletal muscle associated with training which result in a decreased production and/or increased removal of lactate (Holloszy 1988, Brooks, 1991). There were, however, no significant differences between exercise regimens in the magnitude of these changes, suggesting that this improved oxidative capacity of skeletal muscle, resulting from endurance training, may not be dependent upon the duration or frequency of walks but on the intensity and/or total duration of the exercise.

These findings of equivalent increases in endurance fitness in intermittent and continuous exercise concur, at least in part, with the findings of three of the four other studies which have considered the accumulation of shorter bouts throughout the day (Ebisu 1985; Jakicic et al. 1995; Snyder et al. 1997). Ebisu et al (1985) required some subjects to split daily running distances into three bouts per day while others performed the same distance in one continuous bout. The distance per day rose from 3 to 6 miles during the 10 week programme, which is likely to involve short bouts of between 8 to 15 minutes and training was carried out on 3 days per week. The authors report an increase in mean maximal oxygen uptake of approximately 7% for both groups– an improvement which is similar to the 8% rise noted in the present study.

More recently, the ACSM have refined their prescription to suggest that intermittent exercise should be accumulated in bouts which are at least 10 minutes in duration. In the studies of Jakicic et al (1995) and Snyder et al (1997) the brisk walking accumulated in 10 minute bouts was compared to one continuous 30 minute bout (Jakicic et al. 1995, Snyder et al. 1997). The results of these studies are therefore more comparable in terms of format duration and mode to the present study. Jakicic and colleagues (1995) considered the effects of 20 weeks of intermittent or continuous brisk walking on obese women. Improvements in predicted $VO_2$ max of approximately 5.0% and 56% (101 and 103 $ml \cdot min^{-1}$) for short and long bout groups respectively were not different between the two walking patterns. The results of the present study support the similarity in improvements between groups, however, the magnitude of the changes in this study is larger despite an exercise programme which was shorter in duration (10 compared to 20 weeks). Two explanations for this discrepancy might be advanced. Firstly subjects in Jakicic's study were prescribed exercise at an intensity which would elicit 70% of their heart rate reserve. In the present study subjects were required to walk at an speed which resulted in heart rate between 70 and 80% of their maximum which had been determined in a maximal treadmill test. It is likely therefore that the subjects in the present study were exercising at a higher intensity which may in part explain the larger increase in maximal oxygen uptake. Secondly Jakicic and colleagues do not report how the exercise intensity was monitored during the session. In the present study many subjects wore heart rate monitors. On one session per week the display of these monitors was concealed while the subject walked. Our experience of these observed

sessions suggest that when denied access to the heart rate monitoring, subjects will walk at a slightly lower intensity (no significant difference) than in those sessions when they could alter pace in line with the biofeedback received from the heart rate monitor. A study by Porcari et al (1987) supports this suggestion. Young, male subjects had difficulty attaining a heart rate corresponding to 70% of their target heart rate while brisk walking at a self-selected speed. However, when a subgroup of these subjects were given continuous visual feedback of heart rate, they were able to achieve and maintain the required target heart rate for a 30 minute walking session (Porcari et al. 1987).

In contrast, Snyder and colleagues (1997) found no significant increase in aerobic capacity after 32 weeks of intermittent brisk walking despite an improvement of between 0.5ml and 7.5 ml in 7 of the 13 subjects (Snyder et al. 1997). The authors concluded that these 7 subjects were 'responders', who were significantly older, fatter and less fit than the non-responders. When the subjects in the present study are examined in terms of individual responses, those subjects with the lowest initial maximum oxygen uptake experienced the greatest increased after 10 weeks of brisk walk training. This supports the suggestion by Snyder et al (1997) that those most likely to benefit from a programme of intermittent brisk walking are those who are perhaps most sedentary. Indeed public health recommendations suggest that the health benefits of taking additional physical activity are likely to be greatest in those individuals who are most sedentary (Haskell 1994a)

Woolf-May and co-workers (1998) compared an 18 week programme of intermittent training using brisk walks accumulated in bouts of between ten and fifteen minutes with continuous bouts of a similar total duration. Although the focus of the study was alterations in blood lipids with training, they also showed similar increases in endurance fitness (defined as submaximal heart rate response to a step test) among short and long bout walkers of 4.9 and 4.3% respectively. Subjects in this study also had excess weight (mean BMI 25.3-25.8) and relatively low fitness, supporting the notion that the magnitude of improvements is related to initial fitness levels.

Body mass decreased significantly only among the short bout walkers, while total skinfolds decreased in both exercise groups. A decrease in body fatness has been noted

in several walking studies (Pollock et al. 1971, Porcari et al, 1989, Bergman and Boyungs 1991, Whitehurst and Menendez 1991, Hardman and Hudson 1994) and may be due to the increased total energy expenditure due to the walking programme (Ballor and Keesey 1991). Snyder et al (1997) failed to notice an alteration in body mass among a group of overweight subjects in response to an intermittent exercise programme. However when the authors classified subjects as either responders or non-responders and reanalysed the data, a 1.4kg or 1.6% decrease in body mass was observed (Snyder et al. 1997). The decrease in body mass of 1.7kg (2.5%) and 0.9kg (1.4%) among the SHORT and LONG bout walkers respectively in the present study, is of a similar magnitude to those reported for both intermittent and continuous exercise groups (2.1%) in the De Busk study (DeBusk et al. 1990). Jakicic et al (1995) also reported a trend for greater weight loss in the short bout group (8.9kg) than in the long bout group (6.4kg) although this may be accounted for by a somewhat higher total volume of exercise and a greater decrease in energy intake (Jakicic et al. 1995).

In the present study the additional energy expenditure caused by the walking programme was estimated to be approximately 2.8 MJ per week (Ainsworth 1993). When this rough approximation of additional energy expenditure is extrapolated for the 10 week period, it may account for a weight loss of 3.6 kg, well above the reduction noted in either training groups.

These smaller-than-predicted reductions in body mass may be explained in two ways. Subjects assigned to an exercise study may unintentionally reduce their habitual activity due to the time or psychological effects of taking part in a structured exercise programme. In other words, their non-exercising time may be more sedentary than prior to taking up exercise. A second possibility, is the widely held belief that increases in physical activity increases appetite and thereby food intake in a compensatory manner which may have result in the subjects in the exercise groups increasing energy intake. Despite strong intuitive appeal, the latter explanation does not withstand empirical scrutiny. Research indicates that in both normal weight and obese individuals exercise does not increase food intake (Blundell and King 1998) and the coupling between energy expenditure and energy intake is both weak and short-term (King et al. 1997).

When comparing the actual reductions in body mass between exercise groups in the present study, the greater weight loss in the SHORT group (1.7 kg) compared to the LONG group may be explained by several possibilities. The intermittent pattern of exercising necessitates subjects performing exercise in their lunch hour, which may decrease the time remaining for eating (Hardman 1999). Alternatively, the more frequent boosts to metabolic rate offered by the short bout programme may increase the total daily energy expenditure of this group. An increase in excess post-exercise oxygen consumption (EPOC) has been demonstrated for 20 minutes of exercise at 70% of VO2 max in female subjects (Quinn et al. 1994). The proposed mechanisms underlying such rises involve increases in catecholamine levels. Catecholamine levels are known to increase at the start of exercise (McArdle et al. 1991) and therefore subjects in the SHORT may have benefited from this increased metabolic rate on three occasions each day compared with a once per day elevation in the LONG bout group. Although exercise duration is an important factor in determining the magnitude of excess post-exercise oxygen consumption, Quinn and colleagues have demonstrated that the relationship between EPOC and exercise duration is not linear. The magnitude of elevation of a after a 20 minute bout of moderate intensity exercise is not significantly different than after a forty minute bout. Indeed a recent study by Almuzaini and colleagues demonstrated that when a 30 minute bout of exercise is split into two 15 minute bouts, the combined EPOC from the intermittent bouts is higher than the EPOC caused by the continuous exercise (Almuzaini et al. 1998). This may account for the additional potential for intermittent exercise to reduce body mass noted in the present study. Although both explanations appear theoretically plausible, in the absence of any estimation of metabolic rate or any attempt to quantify energy intake during the present study, such interpretations are speculative.

Although none of the groups were significantly different from each other at baseline measurement the SHORT group had mean total skinfolds above the reported average for this population of 66.5mm (MacAuley et al. 1994) which may, again, indicate an increased potential for adjustments in body composition.

The increase in body mass and skinfolds among control subjects was surprising and cannot be accounted for by seasonal fluctuations sometimes cited in the literature (Ingemann-Hansen and Halkjær-Kristensen 1982) since this study was conducted in the

Spring/Summer. Similar findings however have been reported by other authors (Duncan et al. 1991, Aldred et al 1995) and may be the result of over-zealous control subjects. Despite instructions not to alter food intake or exercise it is possible that a desire "not to be active" may have resulted in habitual activity levels which were even lower than normal without a concomitant reduction in energy intake resulting in an increase in body mass and skinfolds. As no record of activity before joining the study or during the study was made for the control group this explanation remains conjectural.

Waist and hip circumferences decreased in both the SHORT and the LONG groups with no change in the control group. However only the SHORT group displayed a decrease in waist hip ratio. This measure of the distribution of fat has been linked to risk of cardiovascular disease in women (Lapidus et al. 1984). The mean waist hip ratio for females of this age group in the Northern Ireland population is 0.80 (±0.07) (MacAuley et al. 1994)The low mean ratio of 0.76 (±0.04) in the 1x30 group may account for the lack of change noted in this group. There were however no differences between the two training groups in the amplitude of any of these alterations in fat distribution. Snyder et al (1997) noted a decreased waist-hip ratio among 'responders' to an accumulated exercise programme, however subjects in this group had higher initial ratios and were fatter than subjects in the present study (Snyder et al. 1997). The ability to alter abdominal adiposity is an important feature of health-enhancing exercise. From an analysis of the individual responses of subjects in the present study it appears that intermittent exercise is at least as effective as continuous exercise at modifying abdominal girth among those whose proportions represent an increased cardiovascular risk.

Mean resting systolic and diastolic blood pressure of all three groups was similar to that reported for female subjects of this age in the local population of 124 mmHg and 79mm Hg respectively (MacAuley et al. 1994). A decrease in systolic blood pressure was noted in both exercise groups with no alteration in controls. The wide variation in blood pressure alterations with training reported in the literature is undoubtedly a function of initial blood pressure levels with those individuals demonstrating borderline hypertension showing greatest effect (Fagard 1995). In the present study all exercise group subjects with systolic blood pressure above 130 mmHg (n=10) showed a decrease with only 4 of the 9 subjects with blood pressure below 120mmHg

experiencing a decrease from baseline to post-training measurement. To our knowledge none of the brisk walking studies using female subjects have reported the effects on resting blood pressure. However, this decrease in resting systolic blood pressure is similar to that reported for male subjects with a similar mean baseline blood pressure after 12 weeks of aerobic exercise (Vroman 1984). Although the precise mechanisms for a reduction of blood pressure are still not fully understood they may include decreased systemic vascular resistance or a reduction in sympathetic drive (Jennings et al. 1989) Resting diastolic blood pressure did not differ from baseline to post training in any group. Mean diastolic pressure at baseline measurement was below 80mmHg for all three groups. This low initial level may account for the lack of alteration with training.

Epidemiological evidence indicates a reduced risk of coronary heart disease (CHD) with regular physical activity of moderate intensity such as that undertaken by the exercising subjects in this study (Blair et al. 1989). When compared with normative data for similar populations the subjects in this study were in most respects 'typical' in physical attributes with the exception of aerobic power which were in the lower percentiles for females of this age. The brisk walking programme brought about moderate improvements in functional capacity, from this initial level of fitness which , although modest, may have important implications for CHD risk. Several studies indicate that even small changes in physical fitness particularly among subjects with low initial levels of fitness are associated with a significant reduction in risk of mortality from CHD (Blair et al. 1989). Moreover the increased capacity for exercise, post-training, may enhance the acute or 'last walk' effects of future exercise sessions among trained subjects (Haskell 1994a).

In conclusion both the continuous and intermittent training programmes of moderate intensity brisk walking brought about significant improvements in physiological parameters which were not evident in the control group. Brisk walking of this intensity appears to have induced physiological changes linked to enhanced health and fitness. Furthermore it appears that multiple bouts of exercise are as effective as continuous bouts at producing these outcomes among sedentary female subjects. This and previous studies (Ebisu 1985, DeBusk et al. 1990, Jakicic et al., 1995, and Snyder et al. 1997)

suggest that a recommendation for an accumulation of exercise throughout the day is valid for previously sedentary individuals.

Aside from physical and physiological improvements, regular exercise is also associated with an amelioration of a number of psychological outcomes including enhanced mood, positive affective function and self-confidence (McAuley 1994). Whether dividing the traditional exercise regime into the type of shorter bouts employed in this study, alters it's impact on psychological factors is not clear. Moreover, although it is intuitively appealing to suggest that such intermittent exercise is more acceptable to the sedentary population, this suggestion has not been subjected to empirical scrutiny. In order to have an impact on the health of the sedentary majority, exercise regimens must be adhered to in the long-term. Only one of the intermittent studies to date has specifically compared the effect of short and long bouts of exercise on adherence among a sedentary population.

## CHAPTER 5

# A COMPARISON OF TWO DIFFERENT PATTERNS OF BRISK WALKING ON PHYSICAL, PHYSIOLOGICAL AND PSYCHOLOGICAL PARAMETERS IN SEDENTARY ADULTS.

## 5.1 INTRODUCTION

The study described in Chapter 4 of this thesis confirms the potential of short bouts of brisk walking, performed over the course of a day, to increase endurance fitness in previously sedentary individuals. In addition to alterations in endurance fitness, regular brisk walking is thought to enhance both physical and mental health (Morris and Hardman 1997). Whether performing this brisk walking in different patterns alters these beneficial effects is not clear.

Regular brisk walking has been associated with reduced risk of CHD (Paffenbarger et al. 1986; Morris et al. 1990). One of the mechanisms underlying such altered risk is thought to involve an alteration in both the type and concentration of lipids in the blood (Kannel 1983). In particular, a programme of regular brisk walking has been shown to increase fasting HDL cholesterol concentrations (Tucker and Friedman 1990) and decrease fasting TAG concentrations (Frändin et al. 1991). Of the studies which have considered the effects of accumulated bouts of brisk walking, only two have measured blood lipid profiles. Snyder and co-workers failed to notice any change in blood lipid concentrations in overweight females who undertook short bouts of brisk walking for 32 weeks despite alterations in fitness and fatness (Snyder et al. 1997). Similarly, Woolf-May and colleagues noted no alterations in blood lipid profiles in response to 18 weeks of intermittent or continuous brisk walking (Woolf-May et al. 1998). In both studies however, the initial blood lipid profiles of subjects were already quite favourable and thus the scope for an alteration in blood lipids was reduced (Snyder et al. 1997; Woolf-May 1998).

A variety of psychological health benefits have also been associated with regular physical activity including improved mood (McDonald and Hodgdon 1991) decreased depression (North et al. 1990) increased self-confidence (McAuley 1994) and self-efficacy (McAuley et al. 1991) and improved affective state (Tuson et al. 1995).

Traditionally however, these psychological health benefits have been demonstrated in response to participation in vigorous exercise Whether more moderate intensity exercise exerts a similar influence on an individuals psychological state is unclear. The effect of brisk walking in particular, on mental health has received little research attention (Morris et al. 1990). Furthermore, whether differences in psychological effects occur when an accumulated pattern of brisk walking is compared to continuous bouts has yet to be investigated.

Exercising, by using short bouts spread throughout the day, has been proposed as a more palatable form of physical activity prescription which may improve participation rates among sedentary populations (Blair et al. 1992). The notion that this pattern of exercise will encourage greater levels of physical activity however has not been widely investigated. In order to have long-term health consequences, exercise regimes must be maintained. In a typical supervised exercise programme, about 50% of individuals will drop out within six months to a year. (Dishman 1988). Only one study to date has compared the effects of prescribing exercise in multiple short bouts versus one continuous bout on adherence to the programme (Jakicic et al. 1995). The authors report improved adherence to short bouts of brisk walking compared to long bouts of similar total duration. Although promising, the findings of Jakicic et al (1995) must be viewed with some caution. Random assignment to short and long bout groups may have resulted in some in-built bias. For example, many subjects who were assigned to the short bout group may have thereby, received a pattern of exercise which they would have chosen themselves, if given the option, and therefore motivation to participate may have remained high. Conversely, subjects in the long bout group may have preferred the short bout pattern and thus their initial assignment served to decrease adherence. Although this is entirely speculative, it illustrates a point, the random assignment of subjects to different exercise patterns makes it difficult to make direct comparisons between the exercise patterns on adherence. A research design where each subject is required to try both patterns of activity for similar periods of time is likely to provide more reliable comparisons of adherence.

The purpose of the present study was to compare the effects on blood lipids, psychological well-being and adherence of subjects undertaking two different patterns of brisk walking.

## 5.2 METHODS

### 5.2.1 Study design

This was a cross-over design training study with subjects randomly assigned to the two different orders. The study was approved by the Research Ethical Committee of the University of Ulster (Appendix A).

### 5.2.2 Subjects

Subjects were recruited through advertisement by poster and computer log-on messages within the university and in the local community. All subjects were asked to complete a health history questionnaire (Appendix C). Subjects reporting a history of cardiovascular disease, diabetes, musculoskeletal injury or reliance on any medication known to influence fat or carbohydrate metabolism, were excluded from participation. After initial screening, subjects with resting systolic blood pressure above 150 mmHg, resting diastolic blood pressure above 105 mm Hg or a Body Mass Index above 35 $kg \cdot m^{-2}$, were excluded from the study. All subjects were sedentary, having taken no more than 20 minutes of continuous activity in the previous six week period. All details of the study were explained prior to obtaining the consent of each subject (Appendix B).

In all, 32 normolipidaemic adults (12 men 20 women) were recruited for the study. Subjects were assigned to the two intervention orders (LONG/SHORT and SHORT/LONG) in a random balanced design. During the first training period, three subjects (1 SHORT/LONG and 2 LONG/SHORT) dropped out of the study. Two subjects (1 SHORT/LONG and 1 LONG/SHORT) completed the first intervention but dropped out during the two week period between interventions. Four subjects (LONG/SHORT) dropped out of the study during the second intervention. Reasons given for drop-out were lack of time (4) loss of interest (3) and injury or ill-health (2). Two female subjects (SHORT/LONG) were excluded from the analysis because they failed to complete more than 60% of the prescribed walks in both interventions. Data are reported for the 21 (14 female 8 male) subjects who completed the study.

### 5.2.3 Baseline Measures

#### 5.2.3.1 Physical

Prior to assignment to training groups subjects visited the laboratory on two occasions. On the first occasion, height and body mass were determined and hip and waist circumferences were measured using the method described in 3.6. During this visit, subjects rested in a seated position and after 5 minutes rest, duplicate measurements of resting blood pressure were made by the same experienced observer using the procedure described in 3.7. Skinfold thicknesses at 4 sites were measured using the technique described in 3.6.3.

#### 5.2.3.2 Psychological

During the initial visit to the laboratory subjects completed three psychometric inventories. The abbreviated 'Profile of Mood States' (Terry et al. (in press)) required subjects to indicate present feelings by selecting the point on a 5-point Likert scale (0-4 representing 'not at all', 'a little', 'moderately', 'quite a bit' and 'extremely') which best represented the degree to which they were feeling each of 27 adjectives describing mood. The 'Barriers to Exercise' scale (Steinhardt and Dishman 1989) required subjects to indicate their agreement or disagreement to 15 suggested barriers to regular brisk walking by selecting the point on a 5 point Likert scale (1-5 representing 'strongly disagree' to strongly agree') which best represented their opinion. Finally, a 'Perceived Efficacy' scale (adapted from (Treasure and Newbery 1998)) required subjects to indicate how confident they were to perform each of 8 physical activities by selecting the point on a 10 point Likert scale (0-9 representing 'not confident' to 'very confident') which best represented their confidence to perform the task indicated. Instructions for completion of each scale were given both verbally and in written form. Subjects were given time to complete each inventory in privacy. All three psychological inventories are contained in Appendix F.

#### 5.2.3.3 Walking test

After anthropometric measures and psychological inventories were completed all subjects performed the UKK 2 km walk test (Oja et al. 1991). Subjects walked a

distance of 2 km as fast as possible, on a flat level surface in an indoor sports hall as described in 3.5.4. Time taken to complete each 0.5 km segment of the test was recorded. In addition heart rate was recorded throughout the test using methods described in 3.5.4. Time (min) to complete 2 km, mean heart rate (beats·min$^{-1}$) during the last 30 seconds of the walk, age (yr) and Body Mass Index (BMI in kg·m$^{-2}$) were used to predict $\dot{V}O_2$ max according to gender –specific equations:

Males:

$\dot{V}O_2$ max (ml·kg$^{-1}$·min$^{-1}$) =

184.9-(4.65 x Time) – (0.22 x Heart Rate) – (0.26 x Age) – (1.05 x BMI)

Females:

$\dot{V}O_2$ max (ml·kg$^{-1}$·min$^{-1}$) =

116.2-(2.98 x Time) – (0.11 x Heart Rate) – (0.14 x Age) – (0.39 x BMI)

(Oja et al. 1991)

**5.2.3.4 Blood lipids**

On another occasion, subjects reported to the laboratory on the morning after a 10-hour overnight fast. A 10 ml venous blood sample was obtained from subjects in a semi-supine position after 5 minutes rest. Blood samples were obtained from an antecubital vein using the procedure described in 3.8.2. Samples were separated and stored using the procedures described in 3.8.4. Plasma aliquots were stored at –20°C and analysed within 5 months for total cholesterol, HDL cholesterol and triacylglycerol concentrations according to the procedures outlined in 3.9.4. All four samples for a given subject were analysed in the same batch. All assays were carried out in the Institute of Clinical Science of the Queens University of Belfast. All samples for each subject were analysed in the same batch. Accuracy and precision were maintained using quality control sera (Randox Ltd., Antrim, Northern Ireland). Within-batch coefficients of variation were 2.3% for TAG, 2.9% for HDL cholesterol and 2.1% for total cholesterol.

Some authors have suggested that fluctuations in endogenous female sex hormones during the menstrual cycle may influence blood lipid profiles (Woods et al. 1987) while

others have suggested minimal (Bush et al. 1988) or inconsistent (Demacker et al. 1982) alterations in blood lipids with cycle phase. In the present study, of the 14 female subjects who completed the study, 9 subjects reported having menstrual cycles between 23 and 29 days with the remainder being post-menopause. Menstrual cycle phase on the day of the first blood sample was recorded. However, holiday commitments, time available and the need to standardise both the training periods and blood sampling timing meant that it was not possible to control for this cycle phase when arranging subsequent blood samples. Cullinane and colleagues (1995) have suggested that alterations in blood lipids during the menstrual cycle are attributable, at least in part, to plasma volume changes (Cullinane et al. 1995). Changes in plasma volume from pre- to post-training for each intervention and for post training 1 to pre training 2 (i.e. during the wash out period) were calculated according to methods previously described (Dill and Costill 1974). The maximum plasma volume changes did not differ between eumenorrhoeic women (range -2.6% to + 6.1%) and other subjects (-4.2% to + 4.6%). There were no significant differences in mean plasma volume between any of the four time-points.

### 5.2.4 Training programme

Following the exercise test, subjects were randomly assigned to two trial orders (LONG/SHORT or SHORT/LONG). Subjects in both trials were asked to walk for 30 $min \cdot d^{-1}$ on 5 $d \cdot wk^{-1}$. In the LONG trial subjects were required to walk continuously for 30 minutes at any time during the day. In the SHORT trial subjects were required to walk for 10 minutes on three occasions during the day, with a minimum of 3 h between walks.

Subjects were given instruction on how to monitor their heart rate by palpation of the carotid or radial artery for 10 seconds. Target heart rate zones of 70-80% of maximum heart rate were calculated for each subject. Maximum heart rate ($HR_{max}$) was predicted according to the equation $HR_{max}$ =220- age. Target heart rate for a 10 second period was calculated to allow rapid measurement at the end of each walk before significant heart rate recovery occurred. Four subjects, who had difficulty monitoring heart rate accurately by palpation, were given heart rate monitors and instructed in their use.

Subjects trained on 5 $d \cdot wk^{-1}$ for 6 weeks in each of the interventions. Most walking took place outdoors on a level surface on the university campus or near the subjects' homes. Treadmills were made available to subjects wishing to complete their walks indoors when adverse weather conditions prevailed. Subjects were requested to refrain from any other regular physical activity during the trial and not to alter their diet for the duration of the trial.

### 5.2.5 Training diaries and affective state measures

To record each walk subjects completed weekly training diaries. After each walk subjects were required to enter the time of day, heart rate, the length of the walk completed and the circumstances under which the walk occurred. A measure of affective state, the 'Feelings Scale' (Rejeski et al. 1987) was included in the training diary. After each walk subjects were required to circle the number on an 11 point Likert Scale (from –5 to +5 representing 'very bad' to 'very good' with 0 being labelled as 'neutral') which best represented their feelings immediately after the walk. Instructions for completion of the training diary and Feelings Scale were given to each subject. An example of the training diaries used for each intervention is shown in Appendix G.

### 5.2.6 Post-training procedures

On the second day after the last training bout, subjects reported to the laboratory after an overnight fast. Post training blood samples were obtained in the same manner as baseline samples. Exercise is known to alter blood lipids for a period following exercise. Such 'last bout effects' have often been thought to obscure the blood lipid changes following training in exercise intervention studies (Haskell 1994a). In this study, the morning after the second day without training was deliberately chosen for obtaining all post-training blood samples. Although there may be some residual last bout effects, day 2 represents the probable 'worst case scenario' for individuals who are exercising on 5 $d \cdot wk^{-1}$. On the same morning as the blood sample, the anthropometric measures and the three psychological inventories were repeated. Within two days of the blood sample, the 2 km walking test was repeated. This was followed by a minimum period of two weeks (maximum 17 days) where subjects refrained from training. After this 'wash-out period, all measures were repeated before subjects completed a further 6

weeks of training using the alternate brisk walking pattern. At the end of the second training period all post-training procedures were repeated in a similar manner.

### 5.2.7 Statistical Analysis

For the physical, physiological and blood lipid data, a three factor (2 x 2 x 2) repeated measures analysis of variance was carried out with 1 between subject factor, trial order and 2 within subject factors, time (pre v post) and pattern of exercise (short v long) to determine alterations in the response variables from pre to post training and differences between exercise patterns. Repeated measures ANOVA was carried out using a statistical software package (Minitab, version 12).

The abbreviated version of the Profile of Mood States contained 27 items which were grouped into six sub-scales (tension/anxiety, depression, vigour, anger, confusion and fatigue) (Terry et al. (in press)). In addition, a total mood disturbance score was calculated by summing the 6 negative factors from which vigour (the only positive scale) was subtracted (McNair et al. 1981). The Barriers to Exercise scale contained 15 items which were grouped into 4 sub-scales (effort, time, health and obstacles) (Steinhardt and Dishman 1989). The Perceived Efficacy Scale contained 8 items which were analysed individually and then grouped into 2 sub-scales (walking activity, other physical activity) and reanalysed.

Given that the reliability the measures used in the psychometric tests is related to the homogeneity of the items within each sub-scale, all items within a given sub-scale were checked for internal consistency using Cronbach's alpha (Hammond 1995). Alpha coefficients were deemed acceptable based on Nunally's (1978) criterion of 0.70 for such psychometric tests (Nunnally (1978)). Items that reduced the internal consistency below this level were removed before further analysis. Cronbach's alpha values were calculated using a statistical software package (SPSS version 8.0)

Mean, standard deviation and standard error were calculated for each sub-scale of each measure using a statistical software package (Microsoft Excel 97, Microsoft Corporation). Data from the feelings scale completed after each walk were averaged to give one global post-exercise feeling score per week.

All of the psychometric tests (mood, barriers, self-efficacy and feelings) used ordinal scales and therefore non-parametric comparative statistics were utilised (Altman 1991). A Friedman's test was used to determine whether there were differences between pre and post walking programme scores in each of the sub-scales used. Where significant differences were determined Dunn's Multiple Comparison Test was performed to locate these differences. The comparative statistical analyses were carried out using a statistical software package (Graphpad Prism version 2.01, Graphpad Software Inc.)

Adherence was calculated by adding the total number of minutes of walking completed during each six week programme, expressed as a percentage of the maximum prescribed walk time of 900 min (30 $min \cdot d^{-1}$ x 5 $d \cdot wk^{-1}$ x 6 wk).

## 5.3 RESULTS

### 5.3.1 Subjects

The physical and physiological characteristics at baseline for subjects are shown in Table 5.1. There were no differences between subjects assigned to LONG/SHORT or SHORT/LONG orders in any of these characteristics.

### 5.3.2 Adherence to training programmes

Subjects showed good adherence to the exercise programmes completing a mean of 837 min (93%) and 765 min (85%) in SHORT and LONG trials respectively during the first six week intervention period Subjects who finished the second trial completed a mean of 828 min (92%) and 720 min (80%) in SHORT and LONG trials respectively during the second intervention period. Number of minutes of walking per week (maximum possible 150) for each trial and each order are presented in Table 5.2.

| | Male n=7 | Female n=14 | All n=21 | Order | |
|---|---|---|---|---|---|
| | | | | SHORT/LONG n=13 | LONG/SHORT n=8 |
| Age (yr) | 44.7 (35-55) | 44.4 (35-55) | 44.5 (6.1) | 43.8 (6.6) | 45.5 (5.5) |
| Height (cm) | 171.5 (164.0-181.8) | 161.7 (152.0-173.2) | 165.0 (7.7) | 166.0 (7.8) | 163.3 (7.7) |
| Body Mass (kg) | 84.97 (68.5-114.3) | 67.8 (53.1-90.8) | 73.5 (15.2) | 74.7 (17.4) | 71.6 (11.7) |
| Body Mass Index ($kg \cdot m^{-2}$) | 28.7 (25.4-35.0) | 25.8 (20.8-31.0) | 26.8 (3.6) | 26.8 (4.2) | 26.7 (2.7) |
| Sum of 4 skinfolds (mm) | 60.7 (39.7-102.6) | 65.1 (40.8-95.1) | 63.7 (16.4) | 64.6 (18.9) | 62.1 (12.4) |
| Waist circumference (cm) | 96.9 (83-115) | 81.3 (67-99) | 86.5 (12.6) | 88.1 (14.3) | 83.8 (9.6) |
| Hip circumference (cm) | 103.3 (93-117) | 104.6 (89-119) | 104.2 (9.3) | 104.2 (10.2) | 104.2 (8.3) |
| Ratio of circumferences at waist and hip | 0.93 (0.89 -0.99) | 0.76 (0.7-0.8) | 0.82 (0.07) | 0.84 (0.09) | 0.80 (0.08) |
| Systolic blood pressure (mmHg) | 134.9 (126-148) | 120.9 (100-148) | 125.6 (16.2) | 128.5 (14.3) | 120.1 (16.4) |
| Diastolic blood pressure (mmHg) | 91.1 (82-98) | 80.3 (62-102) | 83.9 (10.5) | 87.0 (9.8) | 78.9 (10.1) |
| Predicted $\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$) | 28.1 (25.4-36.0) | 26.2 (20.4-33.5) | 26.8 (4.2) | 26.5 (5.5) | 27.5 (3.5) |
| Fasting plasma triacylglycerol concentration ($mmol \cdot l^{-1}$) | 1.83 (0.94-4.16) | 1.30 (0.54-2.56) | 1.48 (0.75) | 1.45 (0.96) | 1.52 (0.70) |
| Fasting total cholesterol concentration ($mmol \cdot l^{-1}$) | 5.52 (4.11-7.16) | 5.38 (3.78-7.42) | 5.42 (0.95) | 5.5 (1.10) | 5.31 (0.66) |
| Fasting HDL cholesterol concentration ($mmol \cdot l^{-1}$) | 0.96 (0.67-1.21) | 1.44 (0.80-2.08) | 1.28 (0.41) | 1.26 (0.40) | 1.31 (0.46) |

**Table 5.1** Mean (range) characteristics of male (n=7) and female (n=14) subjects and mean (s.d.) characteristics of the whole group (n=21), SHORT/LONG order (n=13) and LONG/SHORT order (n=8) subjects at baseline

| | | Minutes of walking completed | | |
|---|---|---|---|---|
| | Week | Short/Long Order n=13 | Long/Short Order n=8 | Total |
| **Short Walks** | 1 | 136.9 (5.8) | 121.4 (5.9) | 132.4 (4.4) |
| | 2 | 140.8 (5.9) | 115.7 (9.7) | 132.9 (5.5) |
| | 3 | 142.3 (2.6) | 121.4 (8.8) | 135.2 (3.9) |
| | 4 | 140.8 (3.5) | 115.7 (10.9) | 131 (4.9) |
| | 5 | 139.2 (6.1) | 117.1 (10.6) | 131.0 (5.6) |
| | 6 | 133.1 (6.7) | 124.3 (9.5) | 131.0 (5.3) |
| | Mean | 138.8 (2.1) | 121.5 (3.4) | 132.2 (2.0) |
| **Long Walks** | 1 | 132.7 (5.0) | 136.1 (6.6) | 148.6 (4.3) |
| | 2 | 136.2 (3.7) | 134.3 (4.8) | 134.8 (2.8) |
| | 3 | 139.2 (4.0) | 126.4 (6.2) | 134.0 (15.9) |
| | 4 | 142.3 (3.6) | 132.1 (9.1) | 139.3 (17.5) |
| | 5 | 125.8 (4.3) | 124.3 (9.5) | 132.6 (4.2) |
| | 6 | 142.0 (3.8) | 112.9 (8.9) | 132.7 (4.8) |
| | Mean | 138.0 (1.7) | 129.3 (3.0) | 134.7 (1.6) |

**Table 5.2** Mean (s.e.m.) minutes walked per week (max -150) in short and long walks training programme, among subjects assigned to SHORT/LONG order (n=14), LONG /SHORT order (n=8) and for whole group irrespective of order (n=21).

### 5.3.3 Changes in body composition, endurance fitness and blood pressure

There were no changes in body mass from pre to post training in either walking pattern. Sum of four skinfolds, waist circumference and hip circumference all decreased significantly from pre- to post- training in both exercise patterns with no difference between interventions. Significant order effects were found for both waist and hip measures with subjects assigned to the SHORT/LONG walks showing a greater decrease in these parameters than subjects who completed the trials in the LONG/SHORT order. Predicted $\dot{V}O_2$ max increased following training using both long and short walks. Greater increases in predicted $\dot{V}O_2$ max were observed following the short walks than the long walks programme ($p=0.017$). The alterations in predicted $\dot{V}O_2$ max with each intervention are illustrated in figure 5.1. Mean body mass, sum of four

skinfolds, hip and waist circumferences and predicted $VO_2$ max before and after each walking programme are presented in table 5.3.

| | SHORT | | LONG | |
|---|---|---|---|---|
| | PRE | POST | PRE | POST |
| Body Mass (kg) | 73.4 (15.5) | 73.7 (16.0) | 73.7 15.8) | 73.5 (15.7) |
| Sum of four skinfolds (mm) | 62.3 (17.2) | 61.3 (17.0)* | 63.7 (16.4) | 61.6 (17.1)* |
| Waist Circumference (cm) | 86.1 (12.7) | 85.8 (13.0)* | 86.1 (12.9) | 85.1 (12.4)* |
| Hip Circumference (cm) | 103.5 (9.5) | 102.7 (9.2)* | 103.0 (9.6) | 102.4 (9.6)* |
| Systolic blood pressure (mmHg) | 124.5 (15.9) | 123.5 (18.6) | 126.9 (15.0) | 123.5 (14.0) |
| Diastolic blood pressure (mmHg) | 84.2 (9.6) | 83.5 (10.1)* | 83.6 (8.8) | 80.7 (8.9)* |
| Predicted $VO_2$ max $ml \cdot kg^{-1} \cdot min^{-1}$ | 27.3 (5.6) | 31.3 (6.7)* | 28.9 (6.3) | 30.1 (6.1)* |

* significantly different from pre intervention ($p<0.05$)

**Table 5.3** Mean (s.e.m.) anthropometric, blood pressure and predicted maximal oxygen uptake of subjects before and after short and long bout walking programmes (n=21)

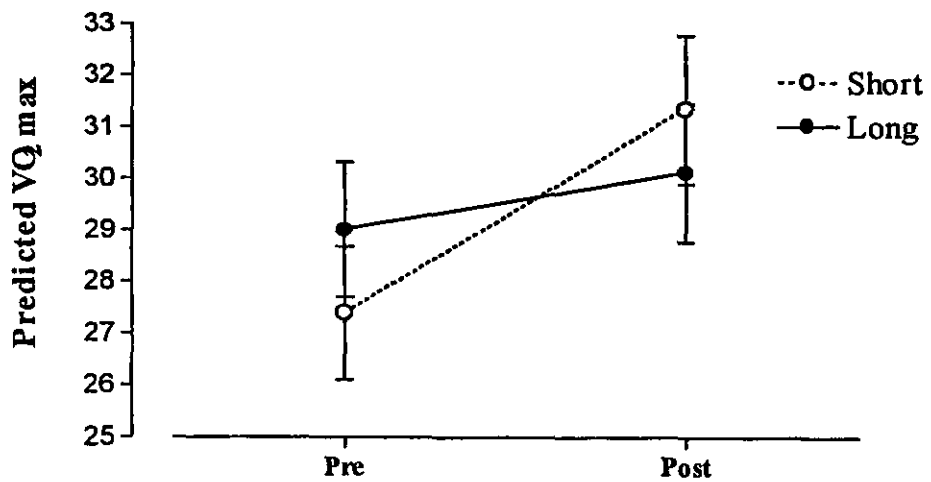


**Figure 5.1** Mean (±s.e.m.) predicted $VO_2$ max before and after short walks and long walks programme (n=21). (main treatment effect p=0.0002, interaction effect order x treatment p=0.04)

For subjects assigned to the LONG/SHORT order the magnitude of the improvement in $VO_2$ max was similar following each intervention. Subjects who were assigned to the SHORT/LONG order showed a greater response to the short walks programme than to

the subsequent long walks programme. This interaction between order (SHORT/LONG v LONG/SHORT) and time (pre v post) is illustrated in figure 5.2.

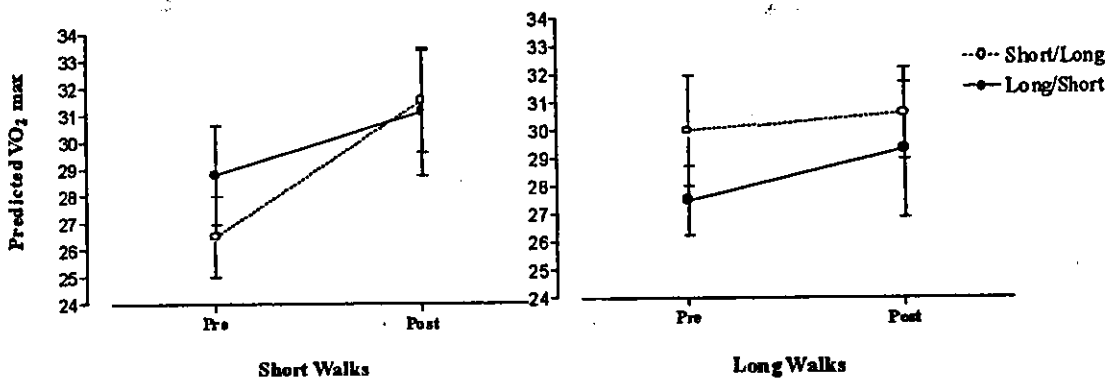


**Figure 5.2** Mean (±s.e.m.) predicted $VO_2$ max before and after short walks and long walks programme for subjects completing programmes in SHORT/ LONG order (n=13) and for subjects completing programmes in LONG/SHORT order (n=7) ( 3-way interaction effect between order treatment and time p=0.045)

### 5.3.4. Changes in blood lipid profiles

Fasting plasma total cholesterol and TAG concentrations decreased from pre- to post-measurement in both long and short trials with no differences in the magnitude of these decreases between trials. Fasting plasma HDL cholesterol concentrations increased from pre- to post- training in both long and short trials with no differences in the magnitude of these decreases between trials. Plasma total cholesterol, HDL cholesterol and triacylglycerol concentrations before and after each intervention are presented in table 5.4.

| | SHORT | | LONG | |
|---|---|---|---|---|
| | **PRE** | **POST** | **PRE** | **POST** |
| Plasma total cholesterol concentration ($mmol \cdot l^{-1}$) | 5.33 (0.21) | 5.06 (0.20)* | 5.48 (0.23) | 5.09 (0.23)* |
| Plasma HDL cholesterol concentration ($mmol \cdot l^{-1}$) | 1.29 (0.09) | 1.36 (0.11)* | 1.30 (0.09) | 1.42 (0.10)* |
| Plasma triacylglycerol concentration ($mmol \cdot l^{-1}$) | 1.40 (0.18) | 1.25 (0.15)* | 1.40 (0.13) | 1.24 (0.14)* |

* significantly different from pre intervention ($p<0.05$)

**Table 5.4** Mean (s.e.m.) fasting plasma concentrations of total cholesterol, HDL cholesterol and triacylglycerol of subjects before and after short and long bout walking programmes (n=21).

### 5.3.4 Changes in mood state

There was a significant reduction in tension/anxiety after both long and short walking programmes with no difference in the magnitude of this alteration between interventions. There were no differences in any of the other subscales or in the total mood disturbance estimate between pre and post measurement in either walking programme. The mean scores on each sub-scale (on a scale of 0-4) and on the estimates of total mood disturbance before and after each walking programme are contained in Table 5.5.

| | Short Walks | | Long Walks | |
|---|---|---|---|---|
| | Pre | Post | Pre | Post |
| Tension/Anxiety | 0.55 (0.19) | 0.26 (0.09)* | 0.48 (0.13) | 0.20 (0.07)* |
| Depression | 0.33 (0.15) | 0.39 (0.17) | 0.35 (0.13) | 0.19 (0.07) |
| Anger | 0.26 (0.16) | 0.12 (0.08) | 0.22 (0.11) | 0.09 (0.05) |
| Confusion | 0.31 (0.15) | 0.10 (0.05) | 0.24 (0.10) | 0.19 (0.09) |
| Fatigue | 0.70 (0.18) | 0.69 (0.22) | 0.78 (0.14) | 0.58 (0.13) |
| Vigour | 1.7 (0.20) | 1.89 (0.20) | 1.89 (0.17) | 1.86 (0.16) |
| Total Mood Disturbance | 3.10 (3.6) | -0.14 (2.93) | 1.76 (2.56) | -3.14 (1.95) |

**Table 5.5** Mean (s.e.m.) scores on tension/anxiety, depression, anger, confusion, fatigue and vigour sub-scales and on total mood disturbance before and after short and long bout walking programmes (n=21).

### 5.3.5 Changes in self-efficacy

At all four time-points, subjects reported greater confidence in performing walking activities than other activities (cycling, jogging and stair climbing). Although there was an increase in self-efficacy for all activities following each walking programme, these increases were not statistically significant. Mean self-efficacy ratings for each item, before and after short and long walks programmes are shown in Table 5.6. Self-efficacy for walking and other activities before and after each intervention are illustrated in Figure 5.3.

| Confidence to perform activity (scale of 0-9) | Short Walks | | Long Walks | |
|---|---|---|---|---|
| | Pre | Post | Pre | Post |
| Walk briskly for 2 miles | 6.1 (0.8) | 7.3 (0.4) | 6.0 (2.6) | 6.8 (0.5) |
| Walk briskly for 3 miles | 5.8 (0.8) | 6.3 (0.5) | 5.2 (0.6) | 6.2 (0.6) |
| Walk briskly for 30 min | 6.0 (0.8) | 7.3 (0.5) | 6.9 (0.5) | 8.1 (0.3) |
| Walk briskly up hills | 4.8 (0.8) | 5.6 (0.5) | 4.9 (0.5) | 6.4 (0.5) |
| Climb 5 flights of stairs | 5.9 (0.8) | 5.9 (0.5) | 5.2 (0.5) | 6.3 (0.4) |
| Jog 1 mile | 3.5 (0.8) | 4.0 (0.5) | 3.1 (0.5) | 4.3 (0.5) |
| Jog 2 miles | 2.9 (0.8) | 3.1 (0.5) | 2.3 (0.5) | 3.4 (0.5) |
| Cycle 5 miles | 4.3 (0.7) | 5.6 (0.6) | 4.4 (0.7) | 5.1 (0.7) |

**Table 5.6** Mean self-efficacy ratings (s.e.m.) for walking (items 1-4) and other activities (items 5-8) before and after short and long bout walking programmes (n=21).

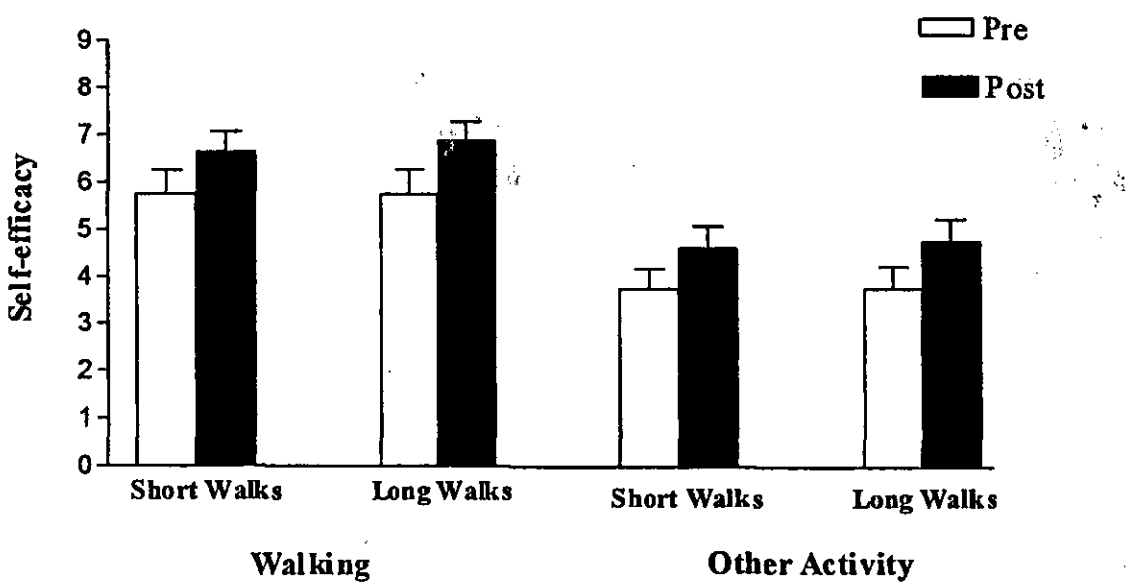


**Figure 5.3** Mean self-efficacy ratings (±s.e.m.) for walking and other activities before and after short and long bout walking programmes (n=21).

### 5.3.6 Changes in perceived barriers

Mean scores for all 4 barriers decreased from pre to post training in both trials. However these reductions were only statistically significant for the effort barrier in short walks trial and for the health barrier in long walks trial. Mean scores for each barrier, before

and after the two walking programmes are presented in Table 5.5.

| Perceived Barriers (scale of 1-5) | Short Walks | | Long Walks | |
|---|---|---|---|---|
| | Pre | Post | Pre | Post |
| Effort | 3.5 (0.2)^ | 2.5 (0.2) * | 2.7 (0.2) | 2.2 (0.1) |
| Time | 3.0 (0.3) | 2.9 (0.2) | 3.2 (0.2) | 2.6 (0.2) |
| Obstacles | 2.4 (0.1) | 2.2 (0.2) | 2.1 (0.2) | 2.1 (0.2) |
| Health | 1.5 (0.2)^ | 1.6 (0.2) | 2.1 (0.1) | 1.4 (0.1)* |

*difference pre to post ($p<0.01$)

^ difference pre short v pre long

**Table 5.7** Mean perceived barrier scores (s.e.m.) for walking (items 1-4) and other activities (items 5-8) before and after short and long bout walking programmes (n=21).

### 5.3.7 Changes in post-exercise feelings

Feelings recorded immediately after each brisk walk, improved as the programmes progressed in each intervention. However these increases were only significant for the long walks trial. No differences in feelings between trials were noted. Mean feelings (on a –5 to +5 scale) for the six weeks of each intervention are illustrated in Figure 5.4

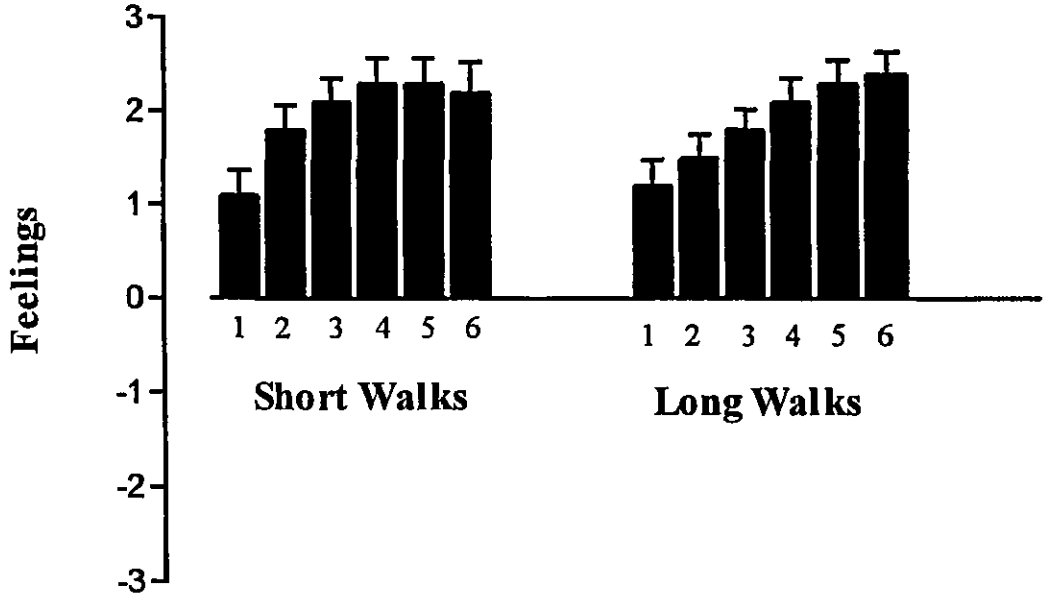


**Figure 5.4** Mean subjective rating of 'feelings' (s.e.m.) for each of 6 weeks during long and short walks programmes (n=21).

## 5.4 DISCUSSION

This study compared the effects of two different patterns of brisk walking on endurance fitness and selected psychological and health-related parameters. The findings suggest that whether 30 minutes of brisk walking is performed in one continuous bout or split into three 10 minutes bouts performed over the course of the day, its effects on fitness and the aspects of physical and psychological well-being measured here were similar.

All subjects completed both trials. Random allocation to trial order resulted in two groups that did not differ in terms of age, body composition, endurance fitness, resting blood pressure, blood lipid profile or response to psychological measures of mood or self-efficacy. Subjects were of comparable weight, body fatness and endurance fitness to sedentary individuals in the Northern Ireland population (MacAuley et al. 1994).

Despite the short duration of the interventions, both patterns of brisk walking resulted in increases in endurance fitness that did not differ between patterns. Two previous studies have considered the effects of such short programmes of walking on endurance fitness. Kilblom (1971) found a decrease in heart rate response to a submaximal cycle ergometer test among middle-aged women who completed just 6 weeks of a brisk walking programme (30 $min \cdot d^{-1}$, 3 $d \cdot wk^{-1}$) (Kilbom 1971). Whitehurst and Menendez found that an 8 week programme of brisk walking (20-40 $min \cdot d^{-1}$ 3 $d \cdot wk^{-1}$) resulted in an 8% decrease in the mean time taken to complete a 1 mile walk among older women (Whitehurst and Menendez 1991). The method used to predict $\dot{V}O_2$ max in the present study relied on the time taken to complete a 2 km walk (Oja et al. 1991). In agreement with Whitehurst and Menendez (1991), time taken to complete the walk decreased by approximately 3% with each of the walking programmes. The total improvements in predicted $\dot{V}O_2$ max, although small in absolute terms, (3.5 $ml \cdot kg^{-1} \cdot min^{-1}$ in SHORT/LONG and 3.6 $ml \cdot kg^{-1} \cdot min^{-1}$ in LONG/SHORT), may allow subjects to walk at a quicker speed after the training programme, while maintaining the same relative exercise intensity as before training. The improvements in endurance fitness after the first six week programme are supported by the suggestion by Hickson and colleagues that many of the adaptations to $\dot{V}O_2$ max occur after 3 weeks when training variables such as frequency intensity and time remain constant (Hickson et al. 1981). Such alterations may be due to improvements in oxygen transport which allow greater

absolute intensities of exercise to be achieved after a period of training. Two other factors however, may contribute to the improvements in walk time and hence the alterations in predicted $\dot{V}O_2$ max . Firstly, although all of the subjects were accustomed to walking, many may not have walked at such a brisk pace before. In keeping to the prescribed target heart rate of 70-80% of predicted maximum during training, subjects may have had to walk at a pace with which they were previously unfamiliar. This may have produced neuromuscular adaptations that resulted in them being able to walk faster on subsequent tests (Rejeski 1994). Secondly, and in a related manner, as subjects walked regularly as part of the exercise programme, their belief in their ability to complete a 2 km walk, may have increased, causing them to walk at a pace which was greater than before the exercise intervention. Some support for this suggestion may be derived from the alterations observed in perceived efficacy before and after the first intervention. Irrespective of the order in which the programmes were completed, mean (s.d.) perceived efficacy for the 4 walking activities (walking briskly for 30 min, walking briskly for 2 miles, walking briskly for 3 miles and walking briskly uphill) increased from 5.4 (1.8) to 6.3 (1.6). Although not significant, this trend suggests that, as perhaps expected, a programme of brisk walking may enhance an individual's self-efficacy for walking activities. There were however no differences in the heart rate during the walk test or in the maximum heart rate reached during the last 30 seconds of the test, despite an increased walking speed, suggesting that at least some of the alteration was due to physiological adaptation.

The difference in the magnitude of the changes in predicted $\dot{V}O_2$ max between the two trial orders is noteworthy. Subjects assigned to complete the two programmes by doing long walks for 6 weeks and then short walks for 6 weeks showed similar improvements in predicted $VO_2$ max in response to both programmes. Subjects who completed the short walks programme first however, showed a greater alteration in predicted $\dot{V}O_2$ max after this programme compared to a more modest increase after the subsequent long walks programme. This is perhaps best explained with reference to the adherence data. Subjects who were randomly assigned to the SHORT/LONG order completed more brisk walking during the first six week programme than subjects assigned to the LONG/SHORT order (93% vs 85% of prescribed walking minutes). Although it is tempting to conclude that a short walks programme achieves greater adherence among sedentary individuals, a finding supported by two recent studies (Jakicic et al. 1995; Jakicic and Wing 1997) the findings of this study do not bear this out. In the present

study, subjects completed 88% and 91% of the walks prescribed in the SHORT and LONG trials respectively. The differences in adherence therefore, are essentially differences between the two orders rather than between the two walking patterns. Subjects assigned to SHORT/LONG walks trials had a tendency to complete more walking, in both trials, than their counterparts who were assigned the LONG/SHORT order. These differences between the two orders are reinforced, anecdotally at least, by the increased dropout rate among subjects assigned to the LONG/SHORT order. Four subjects assigned to the LONG/SHORT order dropped out during the second training period whereas all of the subjects assigned to the SHORT/LONG order, who began the second training period completed the programme. This may suggest that when starting an exercise programme, sedentary individuals find it easier to maintain a programme of continuous bouts of brisk walking, if first introduced to short bouts of brisk walking spread throughout the day. Whereas once a pattern of continuous brisk walking has been accommodated in one's lifestyle, a more intermittent pattern may be less fulfilling. Although there is intuitive logical appeal in this suggestion, the design of the current study and the size of the sample studied do not permit such speculation from being advanced on the basis of scientific evidence.

Both walking programmes resulted in similar decreases in plasma TAG (SHORT –0.13 $\pm$0.08 mmol·l$^{-1}$, LONG –0.16 $\pm$0.07 mmol·l$^{-1}$) and total cholesterol (SHORT –0.27 $\pm$0.17 mmol·l$^{-1}$, LONG –0.39 $\pm$0.08 mmol·l$^{-1}$) concentrations and similar increases in HDL cholesterol concentration (SHORT –0.07 $\pm$0.04 mmol·l$^{-1}$, LONG –0.12 $\pm$0.02 mmol·l$^{-1}$). The direction of these small, but significant alterations, is in keeping with a more favourable blood lipid profile and a reduced CHD risk (Hardman 1996). Although the literature investigating the role of brisk walking in enhancing blood lipid profiles is far from consistent, many cross sectional and some longitudinal studies have noted such favourable associations. In a cross-sectional study of middle-aged subjects, Tucker and Friedman (1990) found an association between regular walking, of more than 150 min per week, and elevated HDL cholesterol concentrations (Tucker and Friedman 1990). In a well designed longitudinal study, Duncan and colleagues noted a mean increase of 6% in HDL cholesterol concentration after a programme of 24 weeks of strolling or aerobic walking (4.8 km·d$^{-1}$, 5d·wk$^{-1}$) (Duncan et al. 1991). Although shorter interventions of only 12 weeks of brisk walking in previously sedentary women (Hardman and Hudson 1994) and 15 weeks of walking in men and women (Kukkonen-

Harjula et al. 1998), have resulted in increases in HDL cholesterol, few studies, using such short duration interventions as the present study (6 weeks), have noted alterations in blood lipid profiles and therefore the improvements in the current study may be somewhat surprising. Indeed both the weekly estimated additional energy expenditure in walking and the length of each intervention in this study are below the 5 MJ (Haskell 1986) and 12 weeks (Wood et al. 1984) respectively generally considered necessary to alter HDL cholesterol. However, a study considering the time course of alterations in plasma lipid and lipoprotein concentrations with endurance training supports the current HDL cholesterol changes. Farrell and Barboriak (1980) suggest that after an initial decline in HDL cholesterol during the first 2 weeks of training at 70% of $\dot{V}O_2$ max (30 $min \cdot d^{-1}$, 3-4 $d \cdot wk^{-1}$) concentrations then rise in a linear fashion at a rate of approximately 0.03 $mmol \cdot l^{-1} \cdot wk^{-1}$ for 8 weeks in both male and female subjects (Farrell and Barboriak 1980). Although this study employed running as the exercise mode, the intensity and total duration of the exercise was roughly similar to that undertaken by subjects in the current study. The increase in mean HDL cholesterol of 0.07 $mmol \cdot l^{-1}$ and 0.12 $mmol \cdot l^{-1}$ in short and long trials in the current study are in keeping with such findings.

Given the increase in HDL cholesterol in the present study the concomitant reduction in plasma TAG concentration is perhaps not surprising. Plasma TAG and HDL cholesterol concentrations are inversely related (Patsch et al. 1992). The decrease in fasting plasma TAG concentration often occurs in tandem with HDL cholesterol concentrations as the degradation in plasma TAG-rich lipoproteins provides additional material for HDL formation (Katan 1990). Despite such associations between plasma TAG and HDL cholesterol, neither the study by Duncan and colleagues (1991) nor that by Hardman and Hudson (1994), who both noted increases in HDL cholesterol concentrations following a brisk walking programme, noted any systematic alteration in plasma triacylglycerol concentrations (Duncan et al. 1991; Hardman and Hudson 1994). In both of these studies however mean TAG concentrations of subjects at baseline (0.85-1.10 $mmol \cdot l^{-1}$) were lower than in the current study (1.40 $mmol \cdot l^{-1}$). Durstine and Haskell (1994) have suggested that changes in fasting TAG concentrations may be related to baseline concentrations of subjects (Durstine and Haskell 1994). In contrast, Kukkonen-Harjula and colleagues (1998) have noted reductions in plasma TAG concentrations, following 15 weeks of brisk walk training, among subjects whose mean intial level

(1.14 mmol·l$^{-1}$) were closer to that of subjects in the present study. The increase in HDL cholesterol and reduction in TAG concentrations may be the result of an increase in the activity of lipoprotein lipase and an increased rate of lipolysis in the exercising muscle (Kiens and Lithell 1989).

The decrease in fasting plasma total cholesterol is again somewhat surprising, since several studies have failed to observe such alterations. Having reviewed the literature, Haskell (1986) suggests that physical activity does not independently influence total cholesterol concentration. (Haskell 1986). Despite this lack of association, several studies have shown reductions in total cholesterol after endurance training using running (Hespel et al. 1988; Despres et al. 1990a) and brisk walking (Kukkonen-Harjula et al. 1998) similar to those noted in the current study. Indeed a meta analysis of 27 exercise intervention studies employing female subjects suggests that exercise training results in a decrease in fasting total cholesterol concentrations (Lokey and Tran 1989)

Only three studies have considered the effects of an accumulated pattern of brisk walking on blood lipoproteins (Snyder et al. 1997; Asikainen et al. 1998a; Woolf-May 1998). None of these studies have noted alterations in fasting blood lipoprotein concentrations following programmes ranging from 15 – 32 weeks. This suggests that for the subjects in these studies, the quantity of brisk walking was insufficient to alter fasting blood lipoproteins concentrations. Given that no alterations were noted in either group in the two studies which have compared the effects of accumulated and continuous patterns of brisk walking, it is difficult to conclude whther or not different patterns of exercise have different effect on blood lipid profiles.

Initial blood lipid concentrations (Tran et al. 1983) and alterations in body fat (Tran and Weltman 1985) may have contributed to the favourable alterations in blood lipid profiles noted in the present study. The mean plasma total cholesterol concentration of the subjects in this study at baseline (5.42 $\pm$0.95 mmol·l$^{-1}$), although within recommended desirable levels (<5.5 mmol·l$^{-1}$) were at the upper end of the desirable range (Ball and Mann 1988). Similarly initial fasting plasma HDL cholesterol concentrations for the men (0.96 $\pm$0.32 mmol·l$^{-1}$) and all subjects (1.28 $\pm$0.42 mmol·l$^{-1}$) were below or only slightly above desirable values (>1.15) respectively (Ball and Mann 1988). Such initial lipoprotein concentrations undoubtedly resulted in an enhanced

potential for change. In the current study an alteration in body fat may also have enhanced the alterations in fasting plasma lipoprotein concentrations. In recent cross-sectional studies, an inverse relationship between body fatness and HDL cholesterol concentrations has been noted for women (Manson et al. 1990) and men (Wood et al. 1991).

The timing of the fasting blood samples in this study may also have influenced the favourable changes observed. Subjects refrained from exercise for just one day before reporting to the laboratory for a fasting blood sample. Lee and co-workers (1991) have shown that when mildly obese women walked for 45 minutes at 60% of $\dot{V}O_2$ max, serum TAG remained lower and serum HDL cholesterol concentrations remained higher some 23 hours after the walk (Lee et al. 1991). Although such acute effects of the last bout of exercise may appear to confound the findings and be the result of poor research design, the timing of the samples in the present study was deliberately chosen. If subjects were to adopt the patterns of activity used in this study on most days of the week, in line with current recommendations, then on any day the longest period since their last brisk walk is likely to be between 36 and 48 hours. Blood samples, drawn after one day of rest, mirror this scenario most closely. Therefore the plasma lipid levels noted in this study may represent those which an individual undertaking regular brisk walking may experience. One final factor which may have lead to an alteration in blood lipid profile is dietary modification. Diet was not controlled in this study. Although subjects were asked not to alter their diet, readiness to take up an exercise programme may coincide with an increased motivation to alter other health behaviours (Marcus et al. 1996).

There were no significant alterations in body mass as result of either pattern of walking. This may reflect a large individual variation in the direction and magnitude of changes in body mass. Alterations in body mass ranged from -1.8 kg to +2.9 kg following the short walks programme and from - 2.0 kg to + 2.1 kg following the long walks programme. The six-week brisk walking programmes are likely to have resulted in an energy expenditure, which when extrapolated would account for a reduction in body mass of approximately 2 kg (Ainsworth 1993). Therefore, any alteration below this is level likely to be accounted for by other factors. As previously suggested, subjects taking part in a structured exercise programme may unintentionally reduce their habitual activity or increase their food intake. Although instructed not to alter their diet or vary

their physical activity, other than by participation in the prescribed brisk walks, no assessment of food intake or daily physical activity was made so such explanations are speculative. Another possible confounding factor in the body mass alterations noted in this study is menstrual status. Although body mass was measured at the same time of day, after an overnight fast, using standardised methods, menstrual cycle phase was not controlled . Nine of the 14 women in the study were eumenorrhic. Body mass, in some women, has been reported to increase up to 1 kg during menstruation due to an increase total body water (Resener et al. 1997).

Despite a lack of change in body mass, both brisk walking patterns resulted in decreases in measures of body fatness. The sum of 4 skinfolds decreased following both trials. Although the long walks intervention appears to have produced greater mean decreases in total skinfolds (-2.1 mm) than short walks (-1.0 mm) these decreases were not statistically different. Such decreases in body fat in the absence of alterations in body mass have been previously reported for brisk walking (Pollock et al. 1971; Pollock et al. 1975 Kukkonen-Harjula et al. 1998) and are likely to represent an increase in lean body mass (Leon et al. 1979). Small but significant changes in waist and hip circumferences occurred with both walking patterns. These alterations (mean ) in hip and waist circumference were comparable for short (waist -0.3 cm; hips –0.9 cm) and long trials (waist -0.9 cm; hips –0.6 cm) It appears that an accumulated pattern of brisk walking is as effective as a continuous approach in achieving favourable alterations in body fatness. The mechanisms underlying such alterations have been discussed in 4.4.

Mean resting systolic and diastolic blood pressure at baseline was similar to that reported for this population (MacAuley et al. 1994). Diastolic blood pressure was reduced following training in long and short programmes, The mean (SEM) reductions in diastolic blood pressure of 1.4 and 1.5 mm Hg for the short and long walk interventions respectively, were not different between the two patterns of exercise. These reductions are somewhat similar to those of Asikainen and co-workers (1998) who found mean decreases in diastolic blood pressure of 2.7 and 3.2 mmHg following one or two bouts of walking per day respectively ($5d \cdot wk^{-1}$, 15 wk) among postmenopausal women (Asikainen et al. 1998a). In concordance with Asikainen and colleagues, there were no alterations in systolic blood pressure following either intervention the present study. The mechanisms underlying such alterations have already been discussed in 4.4.

Both programmes produced reductions in tension/anxiety with no differences between the two patterns of walking in the magnitude of this reduction. A reduction in anxiety is a commonly noted outcome of regular vigorous aerobic exercise (McDonald and Hodgdon 1991). However these findings have not, in general, been replicated for brisk walking programmes [Blumenthal et al 1982, Cramer et al 1991, Stanton and Arroll 1996]. Most of the hypotheses, advanced for the anxiolytic effect of exercise such as, an increased body temperature, reduced tonic muscle activity, increases in catecholamines and elevations in endorphins are transient physiological explanations for reduced tension and anxiety (Murphy 1994). When mood has been measured, two days after an exercise session, these theories may have limited application in explaining alterations. One hypothesis for improvements in mood which may fit with the current study is the 'time out' hypothesis (Bahrke 1978). Subjects taking part in regular exercise thereby take periods of time away from everyday worries and this, in turn, reduces levels of anxiety. If the 'time-out' hypothesis is the mechanism by which decreases in tension/anxiety occur one might reasonably expect greater reductions following the short walks trials where more frequent distraction from one's concerns is likely to result in greater reductions in tension/anxiety (Bahrke 1978). In the present study there were no differences in the reduction in anxiety between patterns of exercise.

Reductions in tension/anxiety with exercise are most often noted among subjects who are intially anxious (La Fontaine et al. 1992). The mean score for tension and anxiety, at baseline, was only 0.62 (0.20) (out of a maximum of 4) suggesting that subjects in this study were not particularly anxious. This significant reduction is therefore somewhat surprising. However if individual scores are examined, those with higher tension/anxiety 1.5-3.75 showed greatest alteration following exercise. This reduction in tension/anxiety may be an important psychological benefit for those individuals who perceive their lives to be stressful and anxiety-causing.

No other alterations in mood were noted in this study. Given that mood is a transient state affected by many factors the use of mood state questionnaire before and after training, although popular in the literature, may be of limited use in identifying medium-and long-term exercise effects. For this reason, the Feelings Scale was used in this study, immediately after each walk to provide a snapshot of the effects of brisk walking. As the weeks progressed the feelings induced by completing the prescribed

brisk walk improved in both trials but this increase was only statistically significant for the long walks programme. During the first week of walking subjects reported a mean (s.d) post-exercise mood of 1.1 (1.4) which on the scale of –5 to =5 was just above neutral (0). By week 5 of their participation in the first walking programme this had risen to 2.1 (1.2). Since these feelings scales were only completed after each walk no conclusions about the acute effect of a single brisk walk can be drawn. However, given that the mean scores during the final week of each walking programme were between 1.7 and 2.9 (on a scale of –5 to +5) it seems reasonable to conclude that walking activity results in at least some degree of positive affect. Several theories may explain this improvement in affective state as the programme progressed. Of these, the theory of optimal stimulation may provide a plausible explanation for the increase in positive affect over the course of the long walk programme. The theory posits that when individuals perceive a balance between their capabilities and the demands of physical activity increases in positive affect will occur (Csikszentmihalyi 1982). As endurance fitness improved and individuals became more experienced at brisk walking, it is likely that they found it easier to attain and maintain the prescribed heart rate and so improved their self-esteem which enhanced their post-exercise feelings.

Prior to taking part in brisk walking, subjects in the present study reported effort and time as the two most significant barriers to physical activity. Specifically, the two most highly rated individual barriers were a lack of motivation (mean (s.d) rating of 3.9 (1.1) out of 5) and a lack of time (mean (s.d) rating of 3.7 (1.4) out of 5). This finding is in keeping with several studies, including a recent cross-sectional study of 2,298 inactive adults (Booth et al. 1997). With training, the mean score for the effort barriers decreased after both short and long walk trials however this decrease was only significant for the short walks trials. The difference between the two patterns of brisk walking is probably best explained by the initial scores, where prior to beginning the short walks programme, subjects perceived greater effort barriers than prior to the long walks programme. Conversely, the long walks trials resulted in a decrease in perceived health barriers, which may be attributable to initial scores on these barriers, which were higher than in the short walk trial. Interpretations of such alterations in perceived barriers to brisk walking, given the current study design are complex. When asked about the barriers to brisk walking at the start of the study, subjects may be able to provide a reliable indication of their reasons for non-participation. However, when asked to rate each barrier, at other times during the study, subjects are likely to reflect on the barriers

which prevented them from taking part in the past few weeks. Given that inactivity during the 'wash-out' period between trials was a prescribed part of the programme, any measure of the perceived barriers to walking at the start of the second walking programme may be erroneous. In all four barriers, subjects reported a decrease (n.s.) in the mean score from the end of the first walking programme to the commencement of the second programme. This is unlikely to be an affect of participation in a programme. Given the decline in mean scores for each barrier following both walking programmes, it seems reasonable to suggest that taking part in regular brisk walking, whatever the pattern, reduces an individuals perceptions of the barriers to such activity. Although simplistic, this suggestion is intuitively logical, since for example, participation in an activity is likely to require a prioritising of time for the activity and once achieved within one's lifestyle the time barrier is largely eliminated (Wankel 1988)

In summary, the findings of the current study suggest that in terms of physical and psychological health benefits, there are no differences between a short accumulated pattern of brisk walking and one which is based on longer, continuous, 'once-a-day' walking.

# CHAPTER 6

# THE EFFECTS OF CONTINUOUS VERSUS INTERMITTENT BOUTS OF EXERCISE ON POSTPRANDIAL LIPAEMIA DURING RECOVERY

## 6.1 INTRODUCTION

One of the benefits of exercise on health status and cardiovascular disease risk appears to be its effect on lipoprotein metabolism (Haskell 1986). By altering the quantity and/or quality of different lipoprotein species circulating in the blood, exercise may reduce the accumulation of plaque on the intima of arteries, a major factor in cardiovascular disease. Amplified postprandial lipaemia is indicative of poor TAG metabolic capacity and is now implicated in the development of atherosclerosis (Karpe and Hamsten 1995, Patsch et al. 1992). In a case-controlled study, Weintraub and colleagues have shown that patients with coronary artery disease show a more elevated plasma triacylglycerol (TAG) response to the ingestion of high fat meal than controls (Weintraub et al. 1997). Any intervention, which could reduce postprandial lipaemia, could, speculatively, slow the rate of progression of atherosclerosis.

Although the acute physiological and metabolic responses to exercise have been widely documented, many of the studies have used subjects in a fasted state, for control reasons. Human beings however, spend a majority of the day in the postprandial state. Since the 1960s, exercise during the postprandial period has been associated with a decrease in plasma turbidity (Cohen and Goldberg 1960) probably through an improved clearance in dietary fat. When exercise is performed after a high fat meal, variations in the clearance of TAG from the blood may be due, in part, to the increased energy expenditure, altered intestinal activity (Soffer et al. 1991) and decreased splanchnic and hepatic blood flow (Rowell 1993). In order to investigate the exercise-induced changes in lipoprotein metabolism therefore, many authors have examined postprandial lipaemia some hours after prolonged exercise, when the effects of these confounding factors have subsided.

There is now considerable evidence that the performance of prolonged exercise, 12 to 18 hours prior to ingesting a meal, alters the lipaemic response to the meal. This reduction in postprandial lipaemia has been demonstrated for walking (Aldred et al. 1993; Aldred et al. 1994; Tsetsonis and Hardman 1996a) running (Sady et al. 1986 ,

Zhang et al. 1998, Herd et al. 1998) and cycling (Herd 1997) exercise. The mechanisms underlying this reduced response have not been fully elucidated, but they may relate to the reduction of muscle TAG brought about by exercise (Annuzzi et al. 1987) and subsequent increase in LPL activity in muscle, during the hours (4-24 h) following exercise (Kantor et al. 1984; Sady et al. 1986; Kiens et al. 1989)

In all of these studies exercise has been low to moderately high in intensity (30-70% of $VO_2$ max) and prolonged (60-180 minutes). The mechanisms underlying this reduced response are not fully elucidated, but they may relate to the reduction of muscle TAG brought about by the exercise (Annuzi et al. 1987) and subsequent increase lipoprotein lipase activity in muscle, during the hours (4-24 h) following exercise (Kantor et al. 1984; Sady et al. 1986; Kiens et al. 1989).

The effectiveness of such prolonged bouts of continuous physical activity in reducing postprandial lipaemia during the recovery period has been established, however, the utility of such prolonged exercise for the sedentary majority is obviously limited. Prolonged, continuous exercise is not compatible with most individuals' lifestyles. In an attempt to entice the sedentary majority to take regular physical activity, recent exercise guidelines have promoted the use of multiple short exercise sessions throughout the day (Pate et al. 1995). 'Lifestyle' exercise programmes which encourage an accumulation of exercise throughout the course of a day are now thought to be effective in overcoming many of the most often-cited barriers to regular physical activity (Dunn et al. 1999). The study reported in Chapter 4 of this thesis confirmed the effectiveness of accumulated exercise in enhancing fitness, but the health benefits of such intermittent patterns of activity have not been widely investigated (Baringa 1997). Whether the effect of exercise on postprandial lipaemia remains when the exercise bout is divided into shorter, intermittent bouts has not been established.

The purpose of this study was to consider whether splitting the duration of an exercise bout has an effect on its capacity to reduce postprandial lipaemia.

## 6.2 METHODS

### 6.2.1 Subjects

Nine normolipidaemic and three borderline hyperlipidaemic men, aged between 18 and 56 years, were recruited from the university population and the local community by means of advertisements. All subjects were physically active non-smokers. The requirements of the study were explained to all subjects and informed consent was received before testing began (Appendix B). Prior to initial tests, subjects completed a health screening questionnaire regarding medical history (Appendix C). Subjects reporting a history of cardiovascular disease, diabetes, musculoskeletal injury or reliance on any medication known to influence fat or carbohydrate metabolism were excluded. After initial screening, subjects with resting systolic blood pressure above 150 mmHg, resting diastolic blood pressure above 95 mmHg or a Body Mass Index above 30 $kg \cdot m^{-2}$, were excluded from the study. Some physical characteristics of the subjects are shown in Table 6.1

| | Age (yr) | Height (cm) | Body Mass (kg) | $VO_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$) |
|---|---|---|---|---|
| Mean ± SD | 32.8 ± 12.7 | 177.6 ± 3.2 | 72.2 ± 4.2 | 56.2 ± 5.8 |
| Range | 18-56 | 172.0-183.5 | 63.0-78.4 | 44.9-68.5 |

**Table 6.1** Mean physical characteristics (± s.d.) of subjects (n=12)

### 6.2.2 Study design

This was a three-trial study conducted with the approval of the University of Ulster Research Ethical Committee (Appendix A). Each subject took part in three separate trials undertaken a minimum of seven days apart, in a random balanced design. Each trial was conducted over two days.

### 6.2.3 Preliminary Measures

Height and body mass were determined using the method described in 3.7.1. After habituation to treadmill running and the expired air collection apparatus, all subjects took part in three treadmill tests. During all three tests, expired air samples were analysed using a integrated gas analysis system (Oxycon, Mijnhardt, Holland) and heart rate was monitored using short range telemetry (Sport Tester, Polar Electro, Finland). Details of the methods used for expired air analysis are described in 3.5.

After habituation to treadmill running, an incremental treadmill test was performed to determine $\dot{V}O_2$ max. The procedures, together with the criteria used for attainment of $\dot{V}O_2$ max, are described in 3.6.2. In all 12 maximal tests, all 4 of the criteria were achieved. On the second visit to the laboratory, subjects took part in a submaximal treadmill test, as described in 3.6.3 to establish the relationship between oxygen uptake and speed. From this test a speed which elicited 60% of the subject's $\dot{V}O_2$ max was identified for the exercise bouts. One final preliminary test involved the subjects running on a treadmill for 15 minutes, at this pre-determined speed to ensure that it elicited the required oxygen uptake.

### 6.2.4 Experimental Protocol.

In the control trial, subjects refrained from exercise on day one. On day one of the exercise trials, subjects completed either three 30-minute bouts of treadmill running) at 60% of $\dot{V}O_2$ max (intermittent) or one 90-minute bout of treadmill running at the same speed (continuous). In the intermittent trial, the three bouts were spaced throughout the day at four hour intervals. In the continuous trial, the exercise was performed in the afternoon. For each subject, the time of completion of exercise was the same in both intermittent and continuous exercise trials in order to minimise diurnal variations and allow equal recovery time before the oral fat tolerance test.

Subjects refrained from exercise and alcohol consumption for two days prior to each trial. On day one of their first trial, subjects recorded their food intake using a portable weighing scale and a food diary. Subjects were then required to replicate this food intake on day one of their next two trials. On day two of each trial, subjects reported to the laboratory in the

morning after a twelve-hour fast and took part in an oral fat tolerance test. The experimental protocol is illustrated in Figure 6.1.

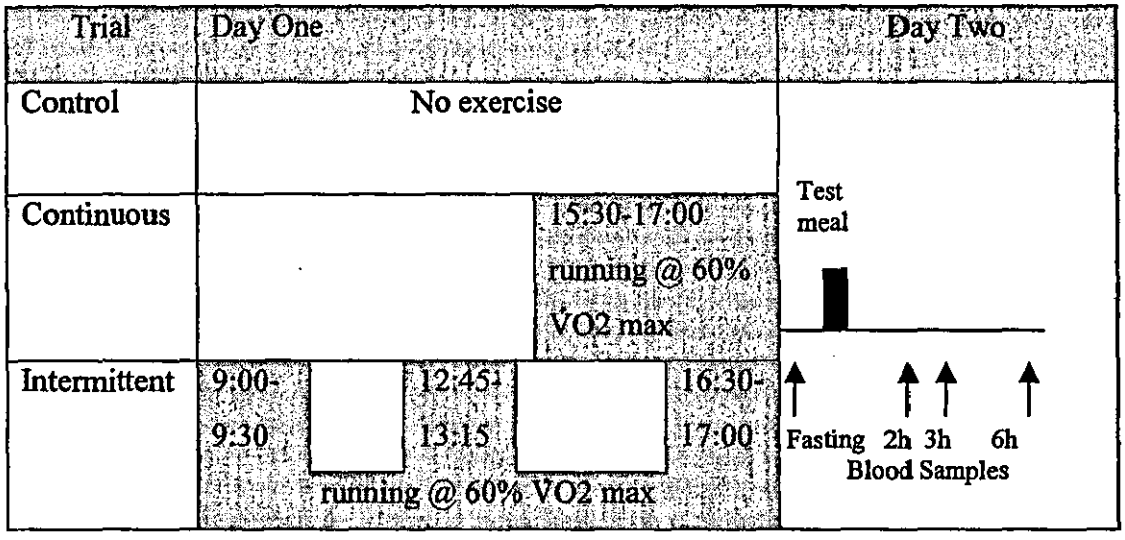


**Figure 6.1** Experimental protocol for the three trial study. Each trial was conducted over two consecutive days

### 6.2.5 Oral Fat Tolerance Tests

After baseline expired air analysis, a 10 ml sample of venous blood was obtained from an antecubital vein while subjects rested in a seated position. Subjects then consumed a high fat test meal. The meal was comprised of cereal, fruit, chocolate, nuts and whipping cream and is described fully in Table 6.2. The content of the meal was calculated according to body mass, with each subject receiving 1.2g fat, 1.2g carbohydrate, 0.2g protein per kg of body mass. This represents an intake of 70 kJ energy per kilogram of body mass. Subjects consumed 86.4 $\pm$ 5.1 g of fat 86.4 $\pm$ 5.1 g of carbohydrate, 14.4 $\pm$ 0.9 g protein and 5038 $\pm$ 299 kJ in the test meal. When the energy density of the different food groups are taken into account, the total energy intake was derived from fat, carbohydrate and protein in a proportion of approximately 67%, 28.5% and 4.5% respectively. The meal was consumed within 15 minutes and was well tolerated by all subjects with no signs of malabsorption being reported.

Further expired air and venous blood samples were obtained at 2, 3 and 6 hours after completion of the test meal. During the day, subjects fasted and performed no physical activity. Water was consumed ad libitum during the first trial and this water consumption was replicated during the two subsequent trials. The reproducibility of this oral fat tolerance test has previously been described (Aldred and Hardman 1993).

| Food | Quantity g per kg body mass | Food | Quantity g per kg body mass |
|---|---|---|---|
| Whipping cream | 2.53 | Chocolate | 0.13 |
| Apple | 0.67 | Brazil nuts | 0.15 |
| Banana | 1.13 | Oats with bran | 0.75 |
| Sultanas | 0.13 | Coconut | 0.07 |

**Table 6.2** Composition of the meal used for the oral fat tolerance test

### 6.2.5 Expired air and blood analysis

During the oral fat tolerance test on day two, expired air samples were analysed for oxygen uptake and carbon dioxide production using an integrated gas analysis system (Oxycon, Mijnhardt, Holland) according to the procedures outlined in 3.5.

Small volumes of sampled blood were used to determine haemoglobin concentration and haematocrit for the baseline and 6 h postprandial samples, as described in 3.10.1 and 3.10.2 respectively. Haemoglobin concentration and haematocrit were used to estimate plasma volume differences between trials (Dill and Costill 1974). The remainder of the blood sample was divided into serum and pre-cooled EDTA tubes as described in 3.9.4. Blood collected in EDTA tubes was separated within 15 minutes of collection. Blood collected in serum tubes was allowed to clot for 60 minutes before separation. Aliquots of plasma and serum were stored at –20°C for later determination of TAG, non-esterified fatty acids, glucose, insulin, total cholesterol and high density lipoprotein cholesterol concentrations as described in Appendix E.

Samples were thawed and analysed within 2 months of collection. All samples for each subject were analysed in the same batch. Accuracy and precision were mantained using quality control sera (Roche and Nycomed Pharma, Oslo, Norway). Within-batch coefficients of variation were 1.5% for TAG, 1.6% for HDL cholesterol, 1.8% for total cholesterol, 1.0% for glucose, 2.3% for NEFA and 2.5% for insulin.

### 6.2.5 Statistical Analysis

Postprandial lipaemia was described by the area under the TAG concentration versus time curve derived by the trapezium rule (Altman 1991). The use of area under the curve as a summary measure to which simple statistical comparisons are then applied, has been recommended as a more valid and relevant method than a series of comparisons at each time-point (Matthews et al. 1990). Since differences in initial plasma TAG concentrations are known to influence postprandial lipaemia (O'Meara et al. 1992), the incremental area under the plasma TAG concentration versus time was also determined. The incremental area under the TAG concentration versus time curve was determined as the area under the plasma TAG concentration vs. time curve, minus the plasma TAG concentration in the fasted state, extrapolated over the observation period.

Area under the curve calculations were also used to evaluate postprandial responses of insulin, glucose and non-esterified fatty acid concentrations. The area under the curve values were checked for normality using a Kolmogorov-Smirnov test using an estimation of the mean and standard deviation from the experimental population (Dallal and Wilkinson 1986). When the data from all three trials (36 trials in total) were considered, the area under the plasma versus TAG time curve was found to be normally distributed.

The ratio of the total area under the serum insulin concentration versus time curve to the total area under the plasma glucose concentration versus time curve was used as an indirect estimate of insulin sensitivity (Lamarche et al. 1993).

These indices of postprandial lipaemia and insulinaemia were compared using a repeated measures ANOVA for correlated means with Tukey post hoc tests. Paired t-tests were used

to compare physiological data from the two exercise trials. A 5% level of confidence was adopted.

Pearson's product-moment correlation was calculated to examine the relationships between variables.

## 6.3 RESULTS

### 6.3.1 Exercise Trials

Subjects ran at a mean speed of 2.8 (± 1.8) m·s-1 (range 2.3-3.9 m·s-1) which elicited 60.7 (±3.4) % of their maximum oxygen uptake. There was no significant difference in average oxygen uptake between trials. Heart rate response to exercise did not differ between the three 30- minute runs but rose significantly during each thirty 30-minute period of the continuous run. Mean heart rate response to the run is shown in figure 6.2

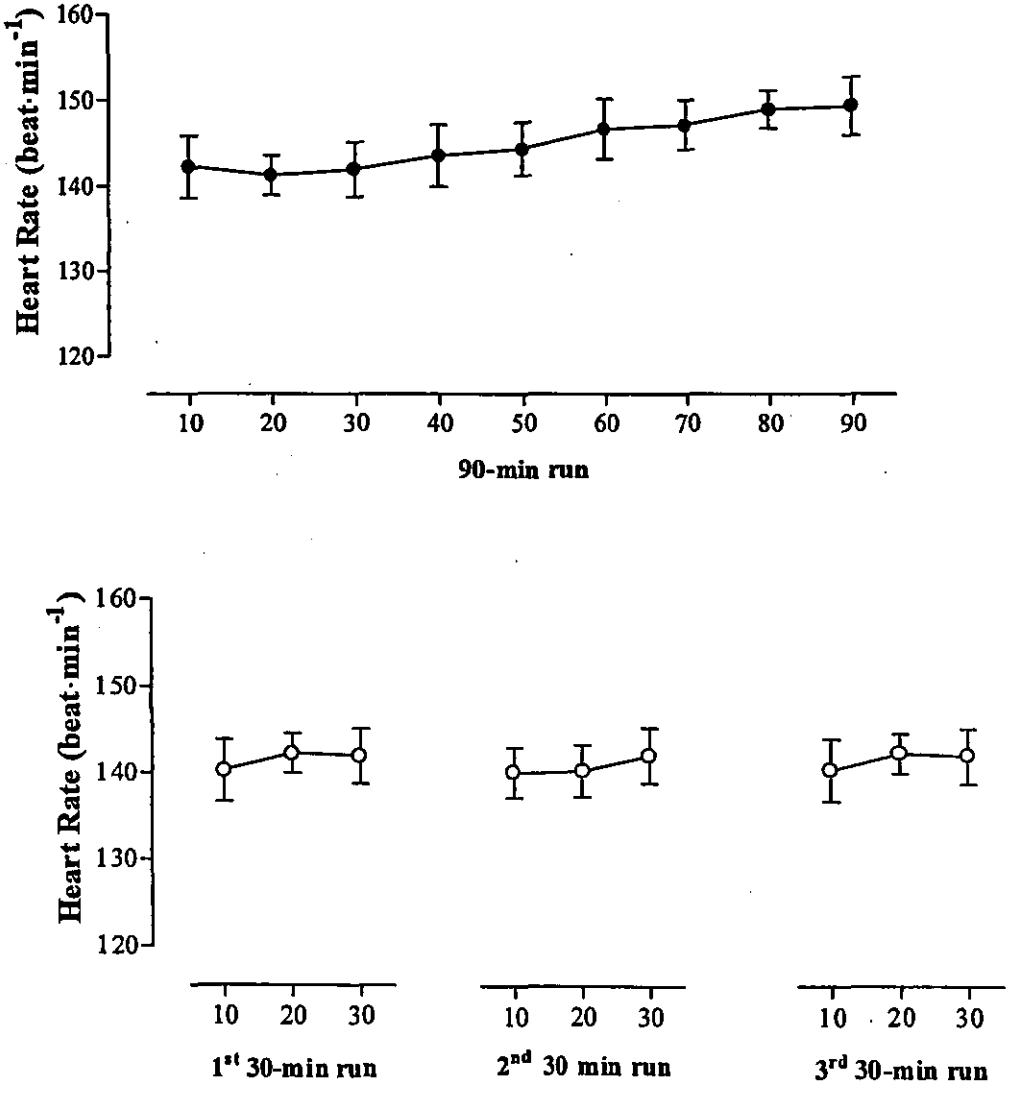


**Figure 6.2** Mean heart rate response (± s.e.m.) to 90 minutes continuous running and three 30-minute bouts of running at 60% VO2 max.

### 6.3.2 Fasting blood lipid concentration

Fasting HDL cholesterol concentrations were higher on the morning after continuous exercise than the control trial but there was no difference between exercise trials or between intermittent and control trials. Fasting plasma TAG concentrations were lower after both exercise trials than after the control trial with no difference between exercise trials. Fasting, non-esterified fatty acid concentrations were higher after continuous exercise than after intermittent exercise with no difference between exercise and control trials. There were no differences in fasting total cholesterol, glucose or insulin concentrations between trials. Fasting blood lipid concentrations for each trial are presented in Table 6.3.

| | Control | Intermittent | Continuous |
|---|---|---|---|
| Total Cholesterol (mmol·l-1) | 4.11 ±1.0 | 4.15 ± 1.13 | 4.29 ± 0.97 |
| HDL Cholesterol (mmol·l-1) | 1.12 ± 0.38 | 1.18 ± 0.30 | 1.20 ± 0.29* |
| Triacylglycerol (mmol·l-1) | 0.89 ± 0.40 | 0.70 ± 0.23* | 0.72 ± 0.18* |
| Non-esterified fatty acids (mmol·l-1) | 0.31 ± 0.13 | 0.30 ± 0.16 | 0.43 ± 0.18# |
| Glucose (mmol·l-1) | 5.16 ± 0.35 | 5.24 ± 0.57 | 5.20 ± 0.13 |
| Insulin (□IU·ml-1) | 8.53 ± 2.94 | 7.30 ± 4.73 | 7.15 ± 2.27 |

*significantly different from control #significantly different from intermittent

**Table 6.3** Fasting blood profiles (mean ± s.d.) during continuous intermittent and control trials (n=12)

### 6.3.3 Blood Lipid Responses to OFTT

All subjects tolerated the test meal, showing no signs of malabsorption. Changes in plasma volume during the oral fat tolerance test were minimal (control -1.4 ±1.0%;

continuous -0.9 ±1.2%; intermittent -1.3 ±2.0 %) and did not differ between trials. No adjustments were made to the concentrations of serum or plasma parameters.

The highest plasma TAG concentrations observed occurred at 2 hours in the intermittent exercise trial and at 3 hours in the continuous exercise and control trials. When the highest TAG concentrations were compared, both exercise trials were lower than the control trial ($p \leq 0.05$). No differences in lipaemic response was noted between exercise trials. The TAG concentration versus time curve for all three trials are shown in figure 6.3.

The area under the TAG versus time curve and the incremental area under the curve were lower in both the continuous and intermittent exercise trials than in the control trial. These indices of postprandial lipaemia are presented in Figure 6.4.

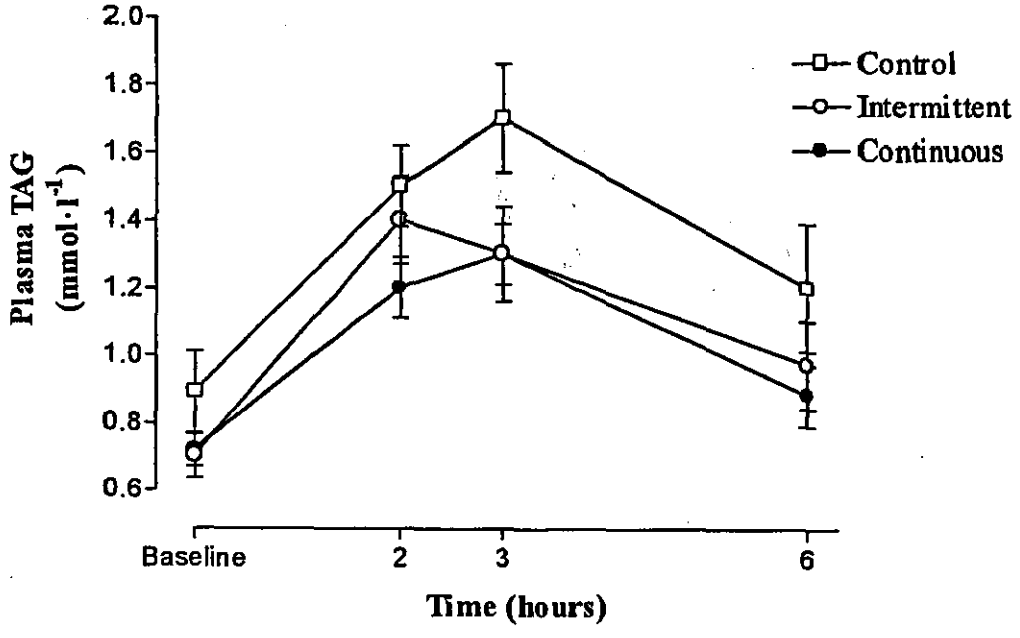


**Figure 6.3** Mean plasma TAG concentrations (± s.e.m.) in the fasted state and for 6 hours after the ingestion of a high fat mixed meal in control, intermittent exercise and continuous exercise trials. (n=12)

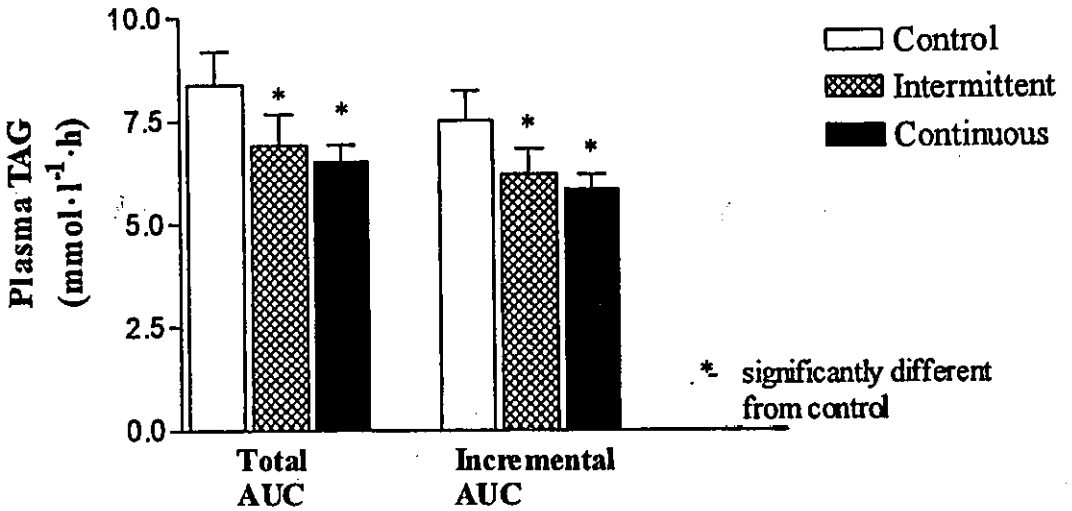


**Figure 6.4** Mean (± s.e.m.) area under the plasma TAG versus time curve and incremental area under the TAG versus time curve following the ingestion of a high fat mixed meal in control, intermittent exercise and continuous exercise trials. (n=12) (main effect treatment p=0.018)

### 6.3.4 Glucose/insulin dynamics

The postprandial concentrations of insulin and glucose are shown in Figure 6.5 and 6.6 respectively. The highest observed serum insulin concentrations were not different between trials, however these values occurred at 3 hours in the control trial and at 2 hours in both exercise trials. Insulin concentration at 3 hours after meal ingestion was higher in the control trial than in the exercise trials with no difference between the two exercise trials. The area under the insulin concentration versus time curve was lower in the continuous trial compared to the control trial. There were, however, no between-trial differences for the incremental area (above baseline) under the insulin concentration versus time curve. Plasma glucose concentrations did not differ between trials.

The differences in insulinaemic response, were mirrored in differences in the insulin to glucose index between continuous and control trials with no differences between exercise trials (control 2.94 (±0.31); intermittent 2.61(±0.25); continuous 2.31 (±0.19)).

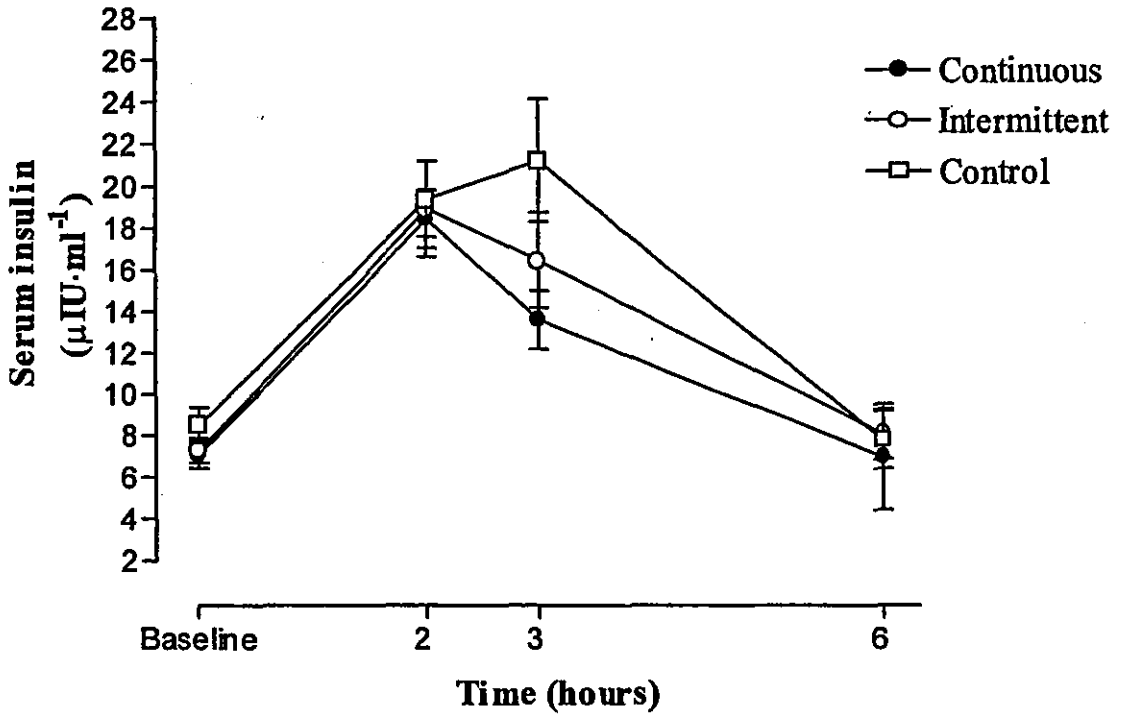


**Figure 6.5** Mean (± s.e.m.) serum insulin concentrations in the fasted state and for 6 hours after the ingestion of a high-fat mixed meal in control, intermittent exercise and continuous exercise trials. (n=12).

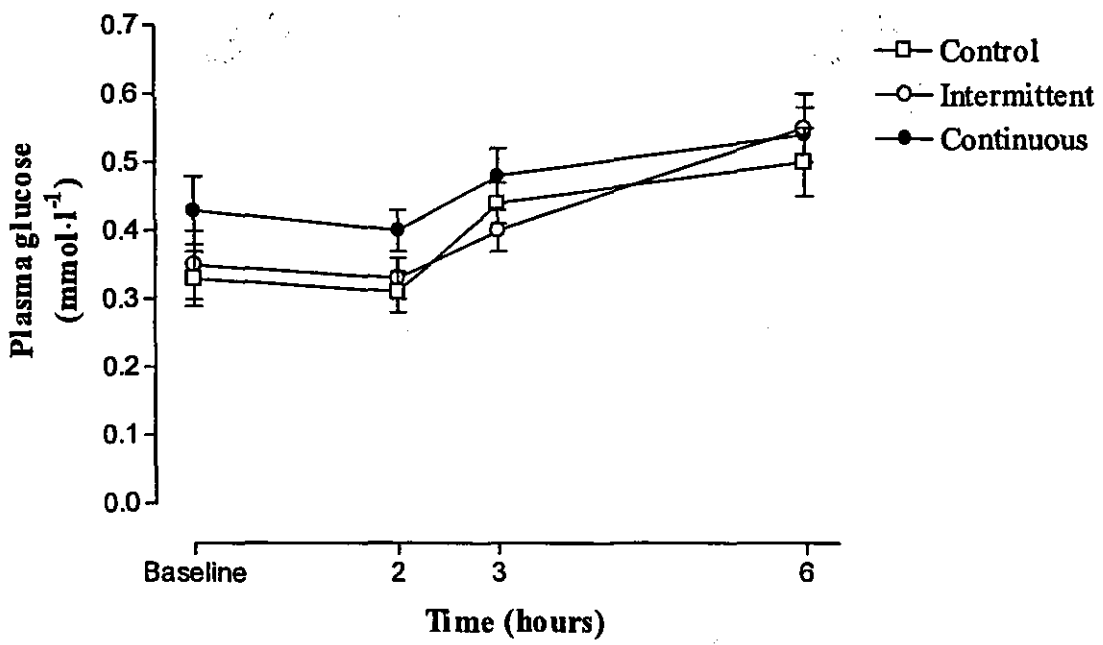


**Figure 6.6** Mean (± s.e.m.) plasma glucose concentrations in the fasted state and for 6 hours after the ingestion of a high-fat mixed meal in control, intermittent exercise and continuous exercise trials. (n=12).

### 6.3.5 Plasma non-esterified fatty acids

Concentrations of non-esterified fatty acids were higher at baseline in the continuous trial compared with the control trial. However there were no differences in plasma non-esterified fatty acid concentrations at any other time point, between trials. The magnitude of supression of non-esterified fatty acid concentration, defined as the area under the curve which was beneath the baseline value was significantly greater in the continuous exercise trial than in the control trial but total area under the non-esterified fatty acid concentration versus time curve did not differ between trials. Plasma non-esterified fatty acid concentrations during each trial are illustrated in Figure 6.7.

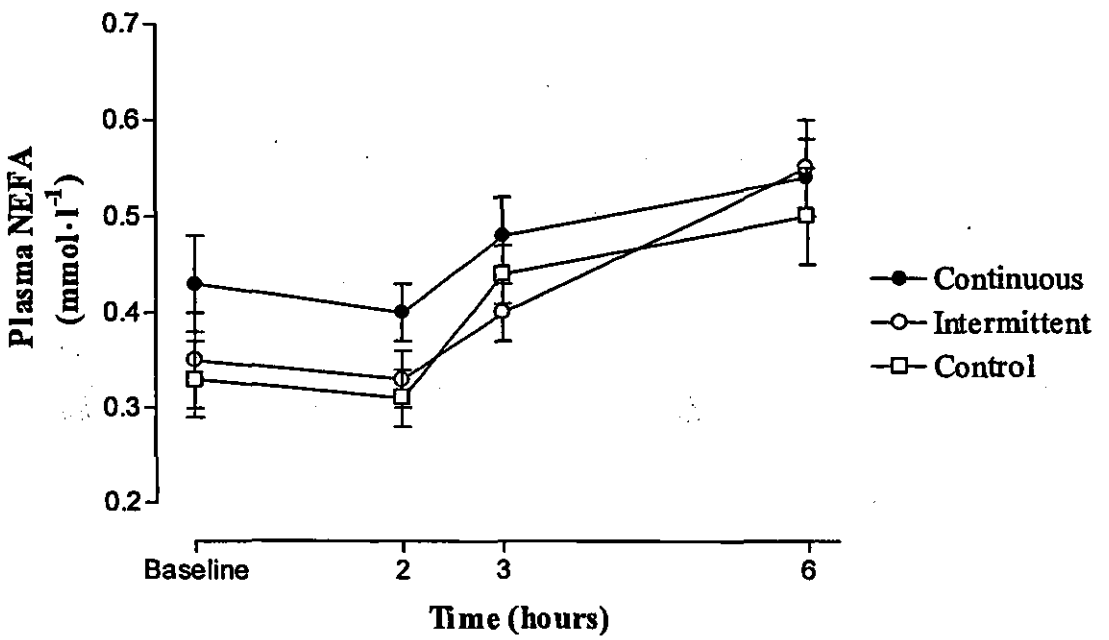


**Figure 6.7** Mean (± s.e.m.) plasma non-esterified fatty acid concentrations in the fasted state and for 6 hours after the ingestion of a high-fat mixed meal in control, intermittent exercise and continuous exercise trials. (n=12).

## 6.4 DISCUSSION

The main findings of this study were that 90 minutes of treadmill running at 60% of $VO_2$ max, performed in one continuous bout or three thirty-minute bouts, reduces postprandial lipaemia during the recovery period and that there is no difference in the magnitude of this reduction between the two exercise patterns.

Subjects ran at a speed which elicited a mean relative oxygen uptake of 61.2 $\pm$ 2.1% and 60.7 ($\pm$1.8)% of their $VO_2$ max for the continuous and intermittent trials respectively. The two exercise patterns resulted in no significant difference in oxygen uptake or respiratory exchange ratio and it is therefore likely that the physiological demands of the exercise were largely similar. The slightly higher heart rate responses (approximately 7 beat·min$^{-1}$) noted in the final 30 minutes of the continuous exercise trial are probably attributable to cardiovascular drift (Brooks and Fahey 1988). The magnitude of the difference between trials was small (4 beat·min$^{-1}$) and is probably of little physiological importance.

Several researchers have demonstrated increased metabolic activity, lasting several hours after aerobic exercise which has been termed EPOC (introduced and explained in Chapter 4.4) (Bahr 1992). Quinn and co-workers (1994) noted that 20 minute of treadmill walking at 70% of $VO_2$ max evoked a similar EPOC as a 40 minute walk but that this was approximately half of the elevation caused by a 60 minute bout (Quinn et al. 1994). If this is so, then it could be argued, that the elevation of metabolic rate, on three occasions in the intermittent exercise trial, compared to once for the continuous trial, may have increased the total energy expenditure and thereby altered the comparability of the effects of exercise. However, given that the magnitude of the elevation in metabolic rate following such exercise, has been shown to be small (1-9% of total) (Gore and Withers 1990), and that both exercise duration and intensity, which were matched in the present study, are important factors in determining the magnitude of this elevation, the argument probably lacks justification (Poehlman et al. 1991). The total energy expended during the two exercise trials is likely, therefore, to have been similar .

The total area under the plasma TAG concentration versus time curve represents a summary measure of the effect of the previous day's activity on the lipaemic response to the high fat meal. The area under this curve was lower (intermittent by 18%; continuous by 22%) for both exercise trials than the control trial, with no difference between the two exercise patterns. The magnitude of this reduction in lipaemic response closely resembles that observed by some authors (Tsetsonis 1995, Tsetsonis et al. 1997, Herd 1997). The postprandial TAG response is somewhat below the reductions reported in other studies (Aldred et al. 1994; Tsetsonis and Hardman 1996a; Tsetsonis and Hardman 1996b; Zhang et al. 1998), despite similarities in subjects, exercise intensities and meals. These discrepancies may be attributable, at least in part, to the large variation in postprandial lipaemia observed between individuals (Brown and Roberts 1991, Cohen et al. 1992). In the present study, the area under the plasma TAG concentration versus time curve ranged from 3.46 mmol·l-1·h to 12.31 mmol·l-1·h

The plasma TAG concentrations, at any time point, reflect the rate of appearance into and disappearance of TAG-rich lipoproteins from the circulation. Exercise causes decreased splanchnic and hepatic blood flow and may therefore alter the absorption, digestion, and transport of intestinally derived TAG (Rowell 1993). In this study, since the exercise sessions were completed 15 hours before the ingestion of the high fat meal, the rates of exogenous TAG appearance in the blood are unlikely to have differed between trials. The replication of both content and timing of food and water intake, on day one of each trial, is likely to have prevented any variation in endogenous TAG production. Alterations in plasma TAG concentrations are therefore likely to be due mainly to a variation in the rate of removal of exogenous TAG from the circulation by the muscle.

Fasting plasma TAG concentration was approximately 11% lower on the day after both exercise trials compared to control trial (control 0.89 $\pm$0.40 mmol·l$^{-1}$ continuous 0.72 $\pm$0.18 mmol·l$^{-1}$ intermittent 0.70 $\pm$0.23 mmol·l$^{-1}$). Although consistent with the findings of similar studies (Sady et al. 1986; Annuzzi et al. 1987; Aldred et al. 1994; Tsetsonis and Hardman 1996a; Herd 1997), these differences in fasting plasma TAG concentrations may serve to exaggerate the differences in total TAG response between exercise and control trials. Endogenous and exogenous TAG are removed by a common saturable pathway and therefore lower plasma TAG concentration at the start of an oral fat tolerance test may accelerate the clearance of exogenous TAG (Chen and Reaven

1991). The observation that a reduction in fasting TAG pool is a contributing factor in reduced postprandial lipaemic response to a high fat meal has been widely reported (Nestel 1964; Brunzell et al. 1973; Chen and Reaven 1991). For this reason, the incremental area under the plasma TAG concentration versus time curve is often used to consider the additional effect of exercise, beyond its effect on fasting TAG concentrations, on the way in which the body deals with an ingested fat load. In the present study, when this incremental area under the curve was considered, the reduced postprandial lipaemia (intermittent-17% continuous-23%), in exercise compared to control trials, remained. This suggests that, at rest, in the period following ingestion of the high fat meal the clearance of TAG from the blood, into other body tissues was greater following exercise than no exercise.

Rossner (1974) has suggested that, at rest, skeletal muscle is the tissue likely to be responsible for most TAG clearance. TAG clearance by skeletal muscle has been linked to the activity of the enzyme, LPL (Rossner 1974). Although not the sole regulator of plasma TAG after a meal, LPL is considered a major determinant of the fate of intestinally derived lipoproteins (Fielding and Frayn 1998). The findings of this study, point indirectly to increased plasma LPL activity during the recovery from prolonged exercise, which does not appear to differ markedly when the exercise is split into three intermittent bouts.

This hypothesised elevation in LPL activity has been documented in the literature (Taskinen et al. 1980, Sady et al 1986, Siep et al 1995) and has been implicated as a mechanism in studies which have noted a decreased postprandial lipaemia in conjunction with an increase in plasma LPL in the recovery period following prolonged bouts of exercise of varying intensities (Kantor et al. 1984; Sady, 1986; Herd 1997). Moderate intensity exercise, of the type undertaken in the two exercise trials in the present study, is known to rely on the degradation of muscle TAG for the resynthesis of ATP . It is estimated that, at an exercise intensity of 60-65% of $VO_2$ max, muscle TAG usage is at its greatest (Romijn et al. 1993). The net utilisation of fat during the exercise trials in the present study, estimated using indirect calorimetry, was approximately 31 g (intermittent, 30.4 g; continuous 32.2 g) with no differences between trials. It is possible, that the enhanced activity of LPL in skeletal muscle, served to hydrolyse fatty acids from chylomicrons, and that these fatty acids were used to re-establish or indeed increase muscle TAG levels (Kantor et al 1984 , Carlson et al

1976, Annuzzi et al 1987, Oscai et al 1990). However, the subjects in this study consumed a typical evening meal after exercise, before beginning an overnight fast. Intuitively, one would assume, therefore, that muscle TAG stores were adequately replenished before the test meal was ingested. Recent evidence refutes such an assumption and provides insight into the time course of muscle TAG depletion and regeneration following prolonged exercise. Kiens and Richter studied healthy young men in the period before and after exhaustive cycle ergometer exercise. They noted that at the end of exercise, muscle TAG concentrations were not altered but that these decreased significantly during the 18 hour period following exercise and were subsequently restored between 18 and 42 hours after exercise. This recovery in muscle TAG stores was accompanied by a 72% increase in the activity of muscle LPL activity compared to pre-exercise levels (Kiens and Richter 1998). When this time course is transposed on the present study, it seems likely that the ingestion of a high fat meal, some 15 hours after exercise, may have been at a time when muscle TAG stores were low and skeletal muscle LPL activity was high. This would account, at least in part, for the reduction in postprandial lipaemia noted between exercise and control trials. It would also support the assertion that the fate of the dietary TAG from the test meal was muscle storage.

It is perhaps also plausible that some of the increase which may have occurred in plasma LPL activity may be a result of its increased activity in adipose tissue (Siep et al. 1995). Although qualitatively lower than the exercise-induced increase in LPL in skeletal muscle (Nikkila 1987) the increase in this enzymes activity in adipose tissue is likely to have had some effect on the uptake of plasma TAG by the adipocytes. During the oral fat tolerance test, therefore, the fate of TAG might have been storage in both muscle and adipose tissue and oxidation in muscle. Serial measurement of heparin releasable LPL activity is not possible (Frayn 1993), and determination was not undertaken in this study, therefore explanations based on the effects LPL activity remain speculative.

The activity of LPL is affected by the action of insulin. In response to a rise in plasma glucose concentrations following a meal, insulin concentration rises, reaching a peak at approximately 30 to 45 min after ingestion. Insulin decreases TAG lipolysis from adipose stores, by inhibiting the action of hormone sensitive lipase, and increases the rate of removal of TAG rich lipoproteins, by stimulating adipose tissue LPL activity

(Frayn 1993). In contrast, insulin is thought to inhibit the activity of muscle LPL activity (Kiens et al. 1989). A reduced insulin response to a high fat meal therefore, may decrease the inhibition of skeletal muscle LPL activity and thereby augment the increased skeletal muscle LPL activity known to occur as a result of exercise (Taskinen et al. 1980; Siep et al. 1995). In the present study, although the magnitude of the insulinaemic response to the meal was lower in the continuous exercise trial than in the control trial, when the baseline levels are taken into account there were no differences in insulin response between the three trials. Despite the lack of a statistically significant difference in insulinaemic response above baseline levels (as determined by the area under the serum insulin concentration versus time curve) postprandial lipaemia was positively correlated with the insuliaemic response ($r=0.45$; $p=0.006$). This suggests that the differences in postprandial lipaemia, between exercise and control trials, may be linked, at least in part, to a blunted insulinaemic response to the test meal after the continuous exercise trial. Associations, between insulin response and postprandial lipaemia, have been reported elsewhere (Herd 1997). In the present study, it is worth noting that, although there were no statistically significant differences in fasting serum insulin concentrations (control 8.53 $\pm$2.94 IU·ml-1 intermittent 7.30 $\pm$4.73 IU·ml-1 continuous 7.15 $\pm$2.27 IU·ml-1) or in the incremental area under the insulin concentration versus time curves (control 40.65 $\pm$30.91 IU·ml-1 x 6 h intermittent 36.95 $\pm$21.01 IU·ml-1 x 6 h continuous 29.61 $\pm$14.91 IU·ml-1 x 6 h) the difference between control and exercise trials in both measures were approaching significance (For fasting insulin concentration; control v continuous $p= 0.065$, control v intermittent $p= 0.069$. For incremental area under curve; control v continuous $p= 0.058$, control v intermittent $p=0.064$)

The primary function of insulin is in the regulation of glucose homeostasis. However, given the number of roles which insulin plays in the postprandial state, alterations in insulin sensitivity can influence glucose TAG and nonesterified fatty acid metabolism. When the insulin response is considered in tandem with glucose response to the test meal, some indication of changes in glucose/insulin dynamics can be gained. In the present study, an index of glucose/insulin dynamics was taken as the ratio of the total area under the serum insulin concentration versus time curve to the total area under the plasma glucose concentration versus time curve (Lamarche et al. 1993). This index was greater in the continuous trial than in the control trial, suggesting that, a prolonged bout

of exercise, results in enhanced insulin sensitivity which persists for many hours after exercise. There were no differences in insulin sensitivity between exercise trials or between the intermittent and control trials. Insulin is secreted from the pancreas in a pulsatile manner and has a relatively short half-life in the blood (approximately 4 minutes) (Cooper et al. 1996). The use of three postprandial measurements in the six hours following a test meal is therefore a very crude indicator of a total insulin response.

Patsch and colleagues have suggested that postprandial lipaemia is inversely related to fasting HDL cholesterol concentrations (Patsch et al. 1992). In the present study fasting levels of HDL cholesterol were higher in the continuous trial compared to the control trial but not different between exercise trials. In general, the literature considering the effects of exercise on fasting HDL cholesterol concentration is not supportive of an acute exercise effect (Hardman 1996). However, acute and transient increases in fasting HDL cholesterol concentrations following exercise, such as those found in the present study, have been demonstrated for a variety of exercise durations and intensities (Lennon 1983; Hicks 1987; Swank et al. 1987; Angelopoulos et al. 1993). When such changes have been found they have been attributed to increased activity of LPL activity or lecithin-cholesterol acyltransferase . Whatever the mechanism, this increase in plasma HDL cholesterol concentration on the morning after continuous exercise in the present study is concordant with the lower postprandial lipaemia between continuous and control trials. There was an inverse correlation between fasting plasma HDL concentration and total area under the plasma TAG concentration versus time curve ($r=-0.42$; $p=0.01$), of a similar magnitude to that observed by other authors (O'Meara et al. 1992). However, postprandial lipaemia was lower in the intermittent trial than in the control trial despite the fact that fasting plasma HDL concentrations did not differ between these two trials.

Fasting TAG, fasting HDL cholesterol and postprandial TAG responses were all lower in the continuous trial than in the control trial in the present study. Given the established role of LPL activity in mediating each of these alteration, it seems reasonable to suggest, even in the absence of LPL determination, that this mechanism is central to the differences observed. In all three of these parameters, continuous exercise had a tendency to produce the greatest alterations from control. It may be that some of the mechanisms underlying such alterations depend on the extent of the depletion of muscle

TAG stores. It is likely that an intermittent exercise session, with normal dietary intake between bouts, will not have resulted in the same magnitude of depletion of muscle TAG stores (Hurley et al. 1986). However given that the two exercise trials did not differ significantly in any of these responses it seems probable that the threshold beyond which exercise alters the metabolic handling of dietary fat appears to be related more to the total energy expenditure of the preceding exercise, than either the intensity or duration of the exercise alone. This assertion is supported by the work of Tsetsonis and Hardman who observed similar reductions in postprandial lipaemia after 180 minutes of exercise at 30% of $\dot{V}O_2$ max after 90 minutes at 60% $\dot{V}O_2$ max, when compared to a control trial with no exercise (Tsetsonis and Hardman 1996a). In a similar study, when LPL activity was measured during the post-prandial period, significant associations were observed between LPL activity and energy expended in exercise (Herd 1997).

The results of the present study, reinforce the suggestion that it is the total energy expended during exercise, rather than the duration of the preceding exercise, which alters lipaemic response. Since the total energy expenditure in continuous and intermittent exercise is likely to be roughly similar, the recommendation for splitting a continuous bout of exercise into smaller bouts distributed throughout the day seems to be justified.

In this study, the measurements of postprandial lipaemia were limited to the day after intermittent and continuous exercise. It is possible that the postprandial responses of these subjects to their normal food intake on day one differed, depending on whether or not they exercised and on the pattern of exercise employed. Indeed, several studies have considered the effect of exercise taken after a meal (Schlierf et al. 1987; Klein et al. 1992; Hardman and Aldred 1995), and just before a meal (Annuzi et al. 1987; Burnett et al. 1993) on postprandial lipaemia. Observing plasma TAG response to a meal on the same day as exercise is taken, presents difficulties when interpreting results, since exercise is known to decrease splanchnic and hepatic blood flow and may, thereby, alter the absorption, digestion and transport of intestinally derived TAG (Soffer et al. 1991). However, in freely living individuals, this undoubtedly represents a very real difference in the handling of dietary fat between individuals who exercise daily, and those who choose inactivity. Some researchers have suggested that the health benefits of exercise may be due, at least in part, to the accumulation of repeated acute

effects during and for some time following each bout of activity (Haskell 1994a). Whether the similarity between intermittent and continuous exercise, in their power to alter postprandial lipaemia, remains, when responses to meals taken soon after exercise are considered, is not clear.

CHAPTER 7

# THE EFFECTS OF DIFFERENT PATTERNS OF BRISK WALKING ON PLASMA TRIACYLGLYCEROL CONCENTRATIONS THROUGHOUT A DAY WITH NORMAL MEALS

## 7.1 INTRODUCTION

The use of accumulated bouts of intermittent exercise has become enshrined in public health recommendations (Pate et al. 1995). In 1990, the American College of Sports Medicine (ACSM) recommended that in order to enhance health, every adult should accumulate 30 minutes of moderate intensity activity on most days of the week. Recent ACSM position stands (ACSM) suggest that for fitness and health benefits, these bouts should be a minimum of 10 minutes duration (Pollock et al. 1998). The studies described in Chapters 4 and 5 of this thesis add to the evidence that such exercise has the potential to alter fitness and body fatness over a period of time (Ebisu 1985; DeBusk et al. 1990; Jakicic et al. 1995; Snyder et al. 1997; Woolf-May et al. 1998). Despite such evidence, the specific health-related outcomes of accumulated exercise have not been fully examined.

Epidemiological evidence has established the link between physical activity and risk of cardiovascular disease (Haskell 1994a). An elevated postprandial TAG response has been recognised as an independent risk factor for atherosclerosis (Patsch et al. 1992). One of the established benefits of acute exercise is the reduction in postprandial lipaemia which occurs in the period following prolonged exercise (Hardman 1998). Most of the studies which have considered the effects of exercise on the postprandial lipaemic response have used high fat meals ingested the morning after a prolonged bout of exercise (Cullinane et al. 1981; Annuzzi et al. 1987; Aldred et al. 1993; Burnett et al. 1993; Aldred et al. 1994; Tsetsonis and Hardman 1996a; Tsetsonis and Hardman 1996b; Herd 1997; Tsetsonis et al. 1997; Hardman 1998). The study described in Chapter 6 of this thesis suggests that splitting such a prolonged bout of exercise into three shorter bouts spread over the course of a day does not alter its effects on postprandial lipaemic response to a meal consumed the following morning. The use of one high fat meal (>1.2 g per kg), provides insight into the handling of dietary fat, by providing a maximum challenge to TAG metabolic capacity (Patsch et al. 1992), however, it is unlikely that

individuals would consume such meals as part of their typical dietary intake. The use of prolonged exercise (60-180 min), also provides maximum physiological perturbation but does not represent the type of activity undertaken by individuals or promoted by current recommendations.

The purpose of the present study was to compare the effects of continuous and intermittent exercise, as promoted by recent health guidelines, on the postprandial response to meals which are typical of the daily nutritional intake of middle-aged adults.

## 7.2 METHODS

### 7.2.1 Subjects

Subjects were recruited through advertisement within the university and in the local community. Volunteers were asked to complete a health history questionnaire (Appendix C). Subjects reporting a history of cardiovascular disease, diabetes, musculoskeletal injury or reliance on any medication known to influence fat or carbohydrate metabolism were excluded from participation. After initial screening, subjects with resting systolic blood pressure above 150 mmHg, resting diastolic blood pressure above 95 mmHg or a Body Mass Index above 35 $kg \cdot m^{-2}$, were excluded from the study. All subjects were sedentary, having taken no more than 20 minutes of continuous activity in the previous six week period. All female subjects were post-menopausal because oestrogen has been reported by some researchers to alter lipid metabolism (de Mendoza et al. 1979) and so excluding pre-menopausal women would decrease between-trial variation. All details of the study were explained prior to obtaining the consent of each subject (Appendix B). Fifteen subjects were initially recruited for the study. Of these; one became ill (female), one sustained an injury (male) during the course of the study, one subject failed to tolerate the test meals (female) and two subjects (female) were excluded from the study because of incomplete blood samples. Nine (7 female 2 male) normolipidaemic and one hyperlipidaemic (male) adults aged 34-66 years completed the study. The physical characteristics of subjects are shown in Table 7.1.

| | Female Subjects n=7 | Male Subjects n=3 | All subjects n=10 |
|---|---|---|---|
| Age (years) | 55.0 (49-66) | 46.0 (34-52) | 52.3 (8.4) |
| Height (cm) | 159.9 (151-164) | 181.7 (178-185) | 166.5 (11.2) |
| Body Mass (kg) | 69.7 (55.5-85.6) | 98.6 (79.5-118.0) | 78.2 (18.7) |
| Body Mass Index ($kg \cdot m^{-2}$) | 27.2 (23.9-33.2) | 29.7 (25.1-34.5) | 27.9 (4.0) |
| Hip circumference (cm) | 105.4 (97-116) | 114.7 (101-136) | 108.2 (11.2) |
| Waist circumference (cm) | 83.8 (72.5-106) | 106.7 (91-131) | 90.7 (17.6) |
| $\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$) | 28.8 (23.4-34.8) | 40.3 (26.3-48.3) | 32.3 (8.5) |

**Table 7. 1** Mean (range or s.d) physical characteristics of female (n=7), male (n=3) and all (n=10) subjects.

### 7.2.2 Study design

This was a three trial study conducted with the approval of the University of Ulster Research Ethical Committee (Appendix A). Trials were conducted over a 12 hour period. Each trial was conducted at least 7 days apart in a random balanced design.

### 7.2.3 Preliminary measures

Prior to the trials, subjects visited the laboratory on three occasions. On the first occasion, height and body mass were determined using the method described in 3.7.1. Hip and waist circumferences were measured using the method previously described (Jones et al. 1986). On this occasion subjects were also habituated to treadmill walking and the expired air collection apparatus.

On the second and third visit to the laboratory, treadmill walking tests were performed. During both tests, expired air samples were analysed using an integrated gas analysis system (Quinton Metabolic Cart, Quinton Instrument Co., U.S.A.) and heart rate was monitored using short range telemetry (Sport Tester, Polar Electro, Finland). Details of the methods used for expired air analysis are described in 3.5 . First, an incremental

walking test was performed to determine $\dot{V}O_2$ max. Walking speed for the maximal treadmill tests was determined during habituation sessions with subjects instructed to "walk as briskly as possible" on the treadmill without having to alter gait from walking to jogging. Once selected, walking speed remained constant for the subject. In this study, treadmill test speeds varied from 1.25 $m \cdot s^{-1}$ to 2.0 $m \cdot s^{-1}$. A full description of the maximal test including the criteria used for determination of $\dot{V}O_2$ max can be found in Chapter 3.6.2. In 6 of the 10 maximal tests all 4 of the criteria were achieved, with the remaining 4 tests fulfilling 3 out of the 4 criteria.

On the final pre-trial visit to the laboratory subjects took part in a submaximal treadmill test, as described in 3.6.3, to establish the relationship between oxygen uptake and speed. From this test, a speed which elicited 60% of the subject's $\dot{V}O_2$ max was determined for the exercise bouts.

### 7.2.4 Experimental protocol

On the day prior to the first trial, subjects recorded their food intake using a portable scale and a food diary. This intake was replicated for the day prior to each of the other trials. Subjects fasted from 22:00 h. on the night before each trial. On the day of each trial subjects reported to the laboratory at 07:30 h. After an expired air sample was obtained, a cannula was inserted into a forearm vein. After five minutes rest a 10 ml baseline blood sample was obtained. Subjects then performed either a 10 minute walk (SHORT) or a 30 minute walk (LONG) on a motorised treadmill at a speed corresponding to 60 % of their $\dot{V}O_2$ max, or rested (CONTROL). They then consumed a breakfast which contained 20% of their daily energy intake (see 7.2.5). After breakfast subjects remained at rest for three hours, during which time expired air and 10ml blood samples were obtained at hourly intervals after the ingestion of the meal. All expired air and blood samples were obtained with subjects in a supine position and after 5 minutes' rest. Three hours after the ingestion of breakfast, subjects either rested (LONG and CONTROL) or performed 10 minutes of treadmill walking at the required intensity (SHORT). Following this, subjects consumed a midday meal which contained 50 % of their daily energy intake and then rested quietly for a further three hours, with blood and expired air samples being obtained hourly. Three hours after the ingestion of lunch, subjects either rested (LONG and CONTROL) or performed 10 minutes of treadmill walking at the required intensity (SHORT). Following this, subjects consumed dinner

which contained 30 % of their daily energy intake and then rested quietly for a further three hours, with blood and expired air samples being obtained hourly. At the end of this three hour period, the cannula was removed and the trial was completed. The protocol for each trial is illustrated in Figure 7.1.

| Time h | *Air and Blood Samples* ▸ | CONTROL ↓ | LONG ↓ | SHORT ↓ |
|---|---|---|---|---|
| 0730 | Baseline | ▸ | ▸ | ▸ |
| 0750 | | | Walk @ 60% $VO_2$ max | |
| 0810 | | | | Walk |
| 0830-0850 | Breakfast (0.37 g·kg$^{-1}$ fat 30.6 kJ·kg$^{-1}$) | | | |
| 0950 | 1 h post | ▸ | ▸ | ▸ |
| 1050 | 2 h post | ▸ | ▸ | ▸ |
| 1150 | 3 h post | ▸ | ▸ | ▸ |
| 1200 | | | | Walk |
| 1220-1240 | Mid-day Meal (0.93 g·kg$^{-1}$ fat 77.4 kJ·kg$^{-1}$) | | | |
| 1340 | 1 h post | ▸ | ▸ | ▸ |
| 1440 | 2 h post | ▸ | ▸ | ▸ |
| 1540 | 3 h post | ▸ | ▸ | ▸ |
| 1550 | | | | Walk |
| 1600-1620 | Evening Meal (0.56 g·kg$^{-1}$ fat 45.5 kJ·kg$^{-1}$) | | | |
| 1720 | 1 h post | ▸ | ▸ | ▸ |
| 1820 | 2 h post | ▸ | ▸ | ▸ |
| 1920 | 3 h post | ▸ | ▸ | ▸ |

**Figure 7.1** Experimental protocol for the three trial study. Each trial was conducted over two consecutive days. Blood and expired air samples are indicated by the symbol ▸.

### 7.2.5 Test meals

During each trial, subjects consumed three meals which contained a total energy intake of 153.5 kJ per kg body mass. Breakfast, midday and evening meals accounted for approximately 20,% 50% and 30% of this energy intake respectively. The largest meal was consumed in the middle of the day, in keeping with common practice in many rural Irish communities and in order to allow for maximum observation of the effects of fat ingestion. The energy content of each meal was derived 35% from fat, 47% from carbohydrate and 18% from protein, representing the typical diet of the Northern Irish population (COMA 1991). The energy content of each meal is described in Table 7.2.

| | Breakfast | | Midday Meal | | Evening Meal | |
|---|---|---|---|---|---|---|
| | $g \cdot kg^{-1}$ | $kJ \cdot kg^{-1}$ | $g \cdot kg^{-1}$ | $kJ \cdot kg^{-1}$ | $g \cdot kg^{-1}$ | $kJ \cdot kg^{-1}$ |
| Fat | 0.29 | 10.7 | 0.73 | 27.1 | 0.43 | 15. 9 |
| Carbohydrate | 0.84 | 14.4 | 2.11 | 36.4 | 1.24 | 21.4 |
| Protein | 0.33 | 5.5 | 0.82 | 13.9 | 0.48 | 8.1 |
| Total | | 30.6 | | 77.4 | | 45.5 |

**Table 7.2** Fat, carbohydrate protein and total energy content of meals ingested during each trial.

Breakfast consisted of cereal with milk, hard-boiled egg and yoghurt (for these subjects 22.8 $\pm$2.7 g of fat, 65.7 $\pm$4.9 g of carbohydrate, 25.6 $\pm$2.3 g protein, 2445 $\pm$223 kJ). Lunch consisted of peanuts, pasta with a meat and tomato sauce, bread and butter, chocolate biscuits and fruit juice (for these subjects, 57.1 $\pm$6.8 g of fat, 165.0 $\pm$12.2 g of carbohydrate, 64.7 $\pm$6.3 g protein, 6184 $\pm$564 kJ). Dinner consisted of chicken sandwiches, potato crisps and fruit juice (for these subjects, 33.6 $\pm$4.1 g of fat, 96.9 $\pm$7.3 g of carbohydrate, 37.3 $\pm$3.5 g protein, 3658 $\pm$151 kJ). The content of each meal is described fully in Table 7.3. Subjects were given 20 min to complete each meal and hourly intervals were designated from the end of the meal for the timing of subsequent expired air and blood samples. Subjects were allowed water *ad libitum* throughout the trials. The volume of water ingested was recorded during the first trial using graduated bottles and subsequently replicated for the other two trials.

| Food | Per kg body mass | Food | Per kg body mass |
|---|---|---|---|
| **Breakfast** | | | |
| Maple & Pecan Cereal | 0.71 g | Wholemeal bread | 0.47 g |
| Milk | 1.88 ml | Boiled Egg | 1.32 g |
| Yoghurt | 1.0 g | | |
| **Mid-day meal** | | | |
| Pasta Twists | 2.44 g | Butter | 0.19 g |
| Creamy Tomato Sauce | 2.26 g | Chocolate biscuits | 0.64 g |
| Minced Beef | 1.69 g | Dry roasted peanuts | 0.55 g |
| Wholemeal Bread | 0.49 g | Orange juice | 1.32 ml |
| **Evening meal** | | | |
| Wholemeal bread | 1.4 g | Tomato | 1.41 g |
| Butter | 0.42 g | Orange drink | 2.73 ml |
| Chicken | 1.41 g | Potato crisps | 0.38 g |

**Table 7.3** Food composition of each test meal. Quantities of each food described are per kilogram of subject's body mass.

### 7.2.6 Expired air analysis

At hourly intervals after the consumption of each meal, subjects rested for five minutes in a semi-supine position. Expired air was collected and analysed according to the method described in 3.5. Oxygen consumption and carbon dioxide production were used to estimate fat oxidation, carbohydrate oxidation and energy expenditure using indirect calorimetry (Consolazio et al. 1963).

### 7.2.7 Blood analysis

Following each expired air sample, subjects remained in a semi-supine position while a 10 ml blood sample was obtained via the cannula. The cannula was kept patent by flushing with 0.9% sodium chloride. For baseline and final (3 h post evening meal ) samples small volumes of blood were used to determine haemoglobin concentration and haematocrit using the method described in 3.10.1 and 3.10.2 respectively. Haemoglobin concentration and haematocrit were used to estimate plasma volume changes (Dill and

Costill 1974). Blood was collected in pre-cooled EDTA tubes and separated within 15 minutes of collection. Aliquots of plasma were stored at –20°C for later determination of TAG, non-esterified fatty acids, glucose, insulin, total cholesterol and HDL cholesterol concentrations as described in Appendix E. Samples were thawed and analysed within 4 months of collection. All samples for each subject were analysed in the same batch. Accuracy and precision were maintained using quality control sera (Randox Laborotories Ltd., Northern Ireland). Within-batch coefficients of variation were below 1.2% for TAG, 0.9% for HDL cholesterol, 1.2 % for total cholesterol, 0.7% for glucose and 1.5% for NEFA and 4.8% for insulin.

### 7.2.8 Statistical analysis

Fasting plasma TAG, non-esterified fatty acids, total cholesterol, HDL cholesterol and insulin concentrations at baseline were compared between trials using a repeated measures analysis of variance. Since there was no difference in fasting concentrations for any variable, the baseline values were omitted to allow plasma TAG, non-esterified fatty acids, total and HDL cholesterol and insulin concentrations at the nine postprandial time points to be compared by a repeated measures analysis of variance using a balanced 3 x 3 x 3 factor design. The 3 factors of treatment (CONTROL, SHORT, LONG) x Meal (breakfast, midday meal, evening meal) x Time ( 1 h, 2 h and 3 h post meal ingestion) were compared within and between trials. To confirm that omitting baseline values from subsequent analysis was justified, data for each parameter at each time point was normalised to baseline level for each subject and the comparative statistical analysis was repeated for the change from baseline values. This did not alter any of the findings. Analysis of variance was performed using a statistical software package (Minitab version 12) which examined all of the residuals from these analyses for normality. In every case, the assumptions regarding the normal probability plot were acceptable.

In parameters where no differences between trials were uncovered by the initial comparative analysis and where the response to both exercise trials were observed to be close, the two data sets were collapsed into a single condition and comparative analysis was repeated to compare CONTROL versus exercise trials. This use of an indicator variable was employed for the indirect calorimetry data, namely, respiratory exchange ratio, estimated fat and carbohydrate oxidation and estimated energy expenditure.

## 7.3 RESULTS

### 7.3.1 Exercise bouts

Subjects walked at a speed of 1.84 ±0.71 $m \cdot s^{-1}$ during brisk walking bouts which elicited an oxygen uptake of 19.4 $ml \cdot kg^{-1} \cdot min^{-1}$ (60.4 ±0.9% of $\dot{V}O_2$ max) and 19.2 $ml \cdot kg^{-1} \cdot min^{-1}$ (59.6 ±1.2% of $\dot{V}O_2$ max) for LONG and SHORT trials respectively. There was no significant difference in oxygen uptake between trials. The heart rate response to exercise did not differ between the three 10-minute bouts in the SHORT trial or between walks performed in SHORT and LONG trials. Estimated fat oxidation during brisk walking was higher ($P<0.05$) during the LONG trial than during the SHORT trial (i.e. sum of 3 walks). Total fat oxidation during 30 minutes of brisk walking was estimated to be 8.8 ± 1.2 g and 6.7 ± 0.9 g for the LONG and SHORT trials respectively.

### 7.3.2 Fasting blood lipids, glucose and insulin

Fasting concentrations of plasma TAG, non-esterified fatty acids, glucose, total and HDL cholesterol and insulin did not differ between trials. Fasting blood profiles are detailed in Table 7.4.

| | CONTROL | SHORT | LONG |
|---|---|---|---|
| Plasma TAG concentration ($mmol \cdot l^{-1}$) | 1.04 (0.30) | 1.09 (0.42) | 1.05 (0.39) |
| Plasma non-esterified fatty acid concentration ($mmol \cdot l^{-1}$) | 0.55 (0.28) | 0.48 (0.41) | 0.43 (0.13) |
| Plasma total cholesterol concentration ($mmol \cdot l^{-1}$) | 6.08 (0.94) | 5.95 (1.18) | 6.18 (0.91) |
| Plasma HDL cholesterol concentration ($mmol \cdot l^{-1}$) | 1. 16 (0.33) | 1.18 (0.32) | 1.19 (0.37) |
| Plasma glucose concentration ($mmol \cdot l^{-1}$) | 5.29 (0.89) | 5.01 (0.52) | 5.10 (0.57) |
| Plasma insulin concentration ($\mu IU \cdot ml^{-1}$) | 9.52 (4.7) | 11.03 (5.6) | 11.06 (4.3) |

**Table 7.4** Mean (s.d.) fasting blood profiles during CONTROL, SHORT and LONG trials.

### 7.3.3 Blood lipid response

All subjects consumed all of the three test meals on each trial. Subjects ingested 1.55 (± 0.63) l of water during each trial. The volume of water ingested did not differ between trials. Changes in plasma volume during the day were small (CONTROL, –1.7 (1.2)%; SHORT, –2.1 (1.5)%; LONG, –1.9 (1.4)%) and did not differ between trials. No adjustments were made to the concentrations of blood parameters. There were very large variations in plasma TAG response ($p<0.001$) between subjects. The area under the plasma TAG concentration versus time curve ranged from 6.8 $mmol \cdot l^{-1} \cdot 9h$ to 31.6 $mmol \cdot l^{-1} \cdot 9h$. The highest average plasma TAG concentrations were observed 2 h after the midday meal in all three trials. There was a significant main effect for treatment ($p=0.009$) with plasma TAG concentrations being significantly lower in the LONG and SHORT trials compared to the CONTROL trial There were no differences in plasma TAG response between LONG and SHORT trials. The area under the plasma TAG concentration versus time curve, in the CONTROL trial, was 10.3% and 11.0% higher than the LONG and SHORT trials respectively. There was a significant treatment x meal effect ($p=0.03$) indicating that the difference in plasma TAG between CONTROL and exercise trials increased as the day progressed. In addition there was a meal x time effect ($p=0.001$) indicating a different pattern of plasma TAG response to dietary fat as the day progressed. Plasma TAG responses during each trial are shown in figure 7.2.

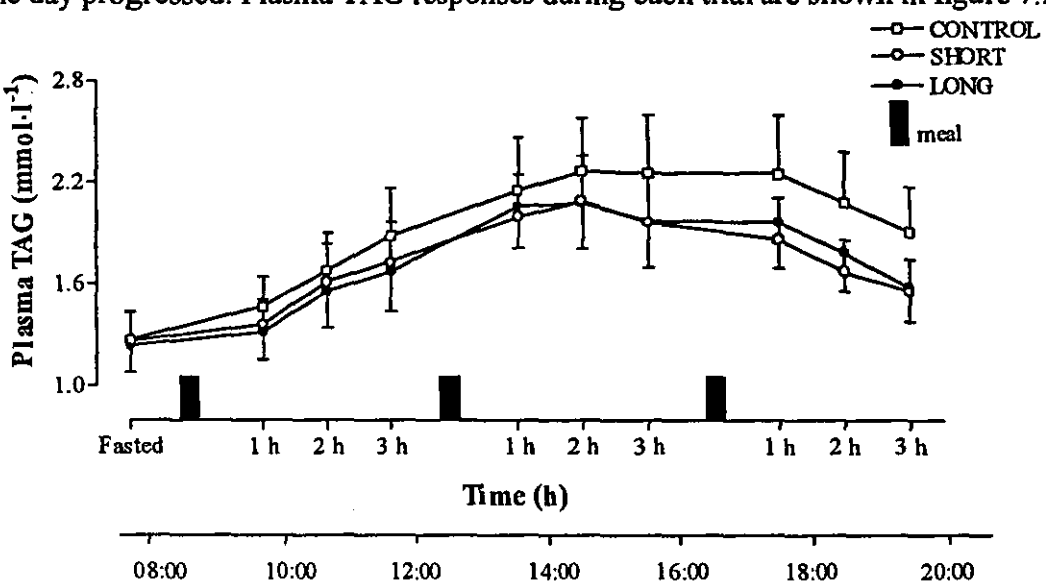


**Figure 7.2** Mean (s.e.m.) plasma TAG responses during CONTROL, SHORT and LONG trials (n=10) (approximate real time is shown on the lower x-axis). (main effect treatment $p=0.009$, interaction effects treatment x meal $p=0.03$, meal x time $p=0.001$)

Plasma non-esterified fatty acid concentration declined in response to each meal. There were no differences in the plasma non-esterified fatty acid response between the three trials. Plasma non-esterified fatty acid responses are shown in figure 7.3

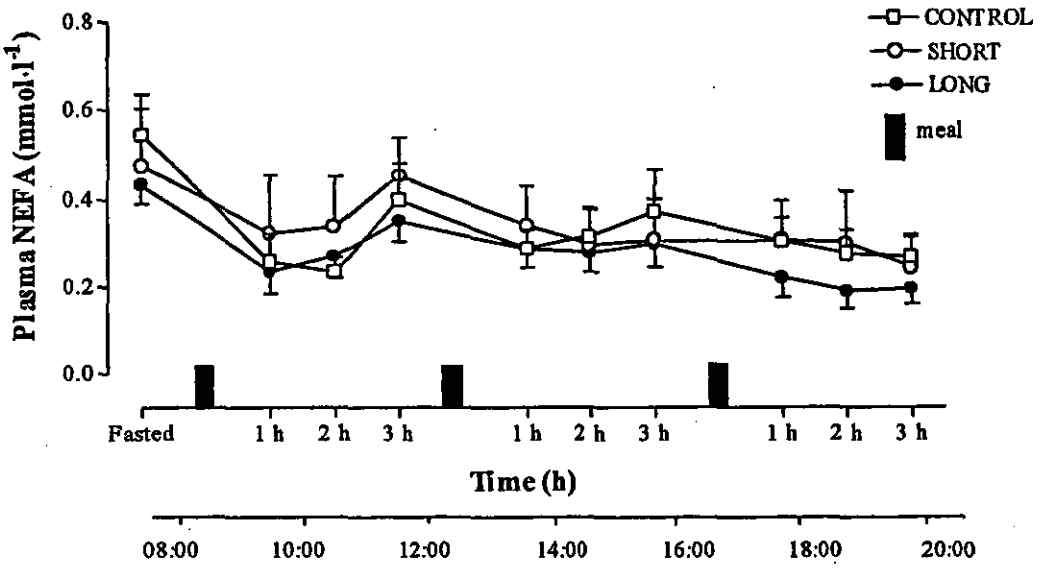


**Figure 7.3** Mean (s.e.m.) plasma non-esterified fatty acid during CONTROL, SHORT and LONG trials (n=10) (approximate real time is shown on the lower x-axis).

### 7.3.4. Plasma insulin and glucose

Plasma insulin concentration increased after each meal and then declined during the postprandial period. The highest mean insulin concentration was reached at 1 h after the evening meal in the CONTROL and LONG trial and at 2 h after the evening meal in the SHORT trial (CONTROL, 71.2 (11.85) $\mu IU \cdot ml^{-1}$; SHORT, 70.8 (17.7) $\mu IU \cdot ml^{-1}$; LONG, 74.9 (16.7) $\mu IU \cdot ml^{-1}$). There were no differences in this plasma insulin response between trials.

The highest mean concentration of plasma glucose occurred at 1 h after the midday meal in the CONTROL and SHORT trials, and at 2 h after the midday meal in the LONG trial (CONTROL, 6.38 (1.46) $mmol \cdot l^{-1}$; SHORT, 6.60 (1.45) $mmol \cdot l^{-1}$; LONG, 6.43 (1.45) $mmol \cdot l^{-1}$). There were no differences in plasma glucose concentrations between trials. Plasma insulin and glucose responses are shown in figures 7.4 and 7.5 respectively.

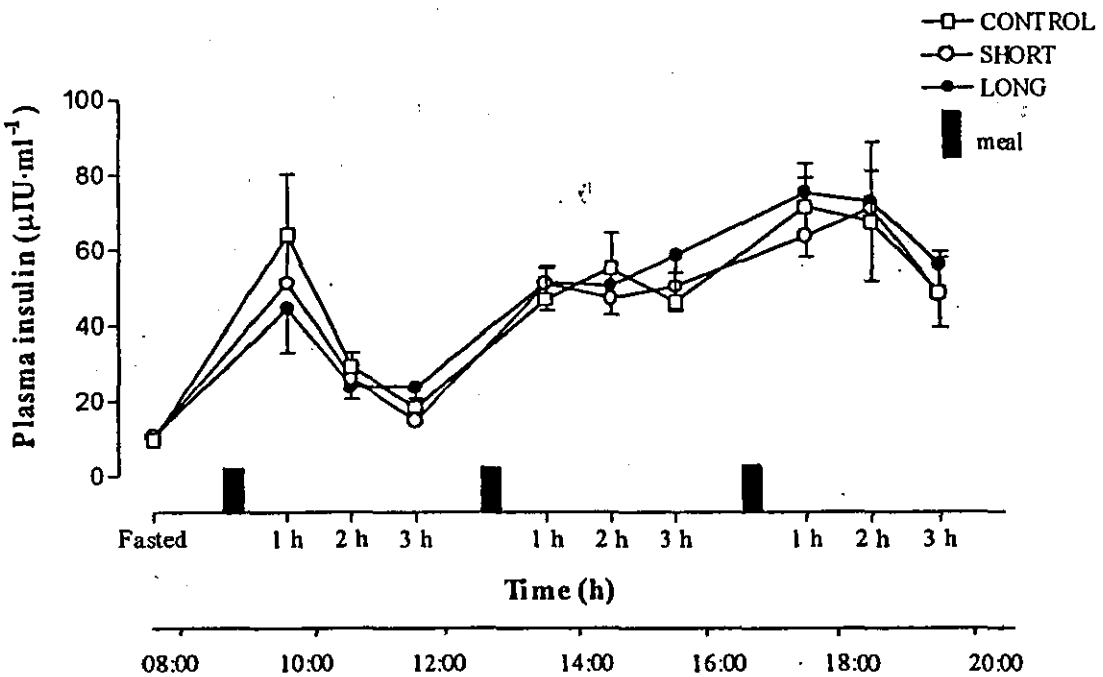


**Figure 7.4** Mean (s.e.m.) plasma insulin responses during CONTROL, SHORT and LONG trials (n=10) (approximate real time is shown on the lower x-axis).

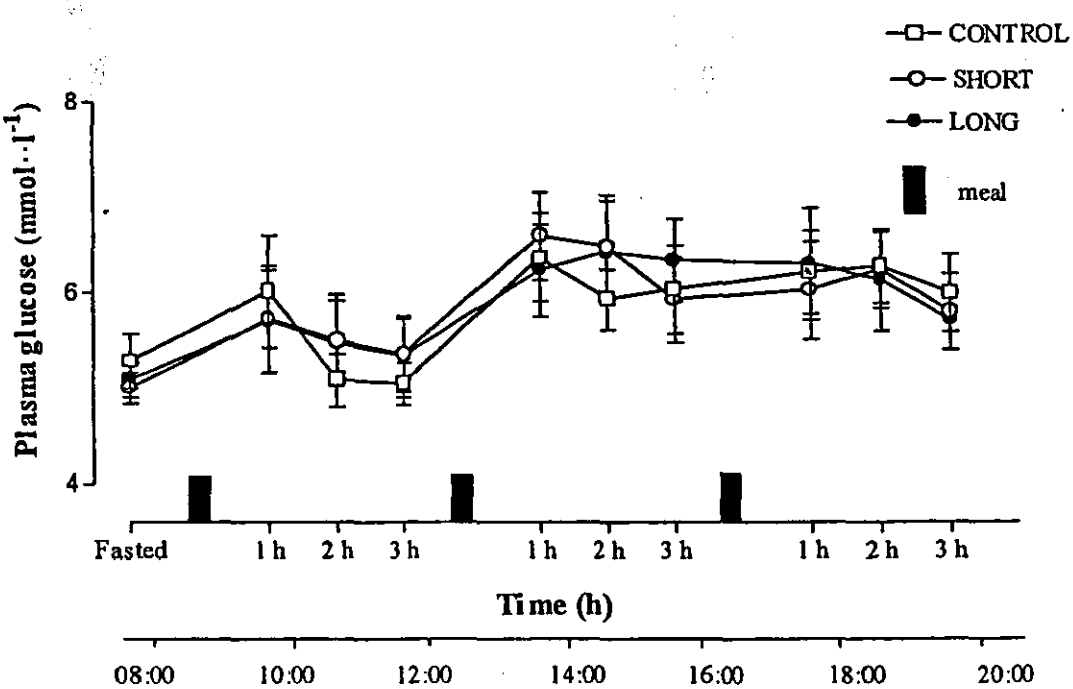


**Figure 7.5** Mean (s.e.m.) plasma glucose responses during CONTROL, SHORT and LONG trials (n=10) (approximate real time is shown on the lower x-axis).

### 7.3.4 Indirect Calorimetry

There were no differences in oxygen uptake, carbon dioxide production or respiratory exchange ratio, at rest, between trials. When data from the SHORT and LONG trials are collapsed, respiratory exchange ratios were significantly lower during exercise compared to CONTROL trials (CONTROL, 0.85 (± 0.02) range 0.82-0.90; exercise, 0.83 (± 0.02) range 0.80-0.87). Respiratory exchange ratios for exercise and CONTROL trials are illustrated in figure 7.6. Estimated fat oxidation at rest did not differ between trials but when the two exercise trials are collapsed there was significantly higher fat oxidation during exercise trials (p=0.009). Fat oxidation for exercise and CONTROL trials are shown in Figure 7.7.

Estimated energy expenditure did not differ between trials. Total resting energy expenditure for the nine hour observation period was CONTROL, 3508 (411) kJ; SHORT, 3428 (608) kJ and LONG, 3486 (550) kJ.

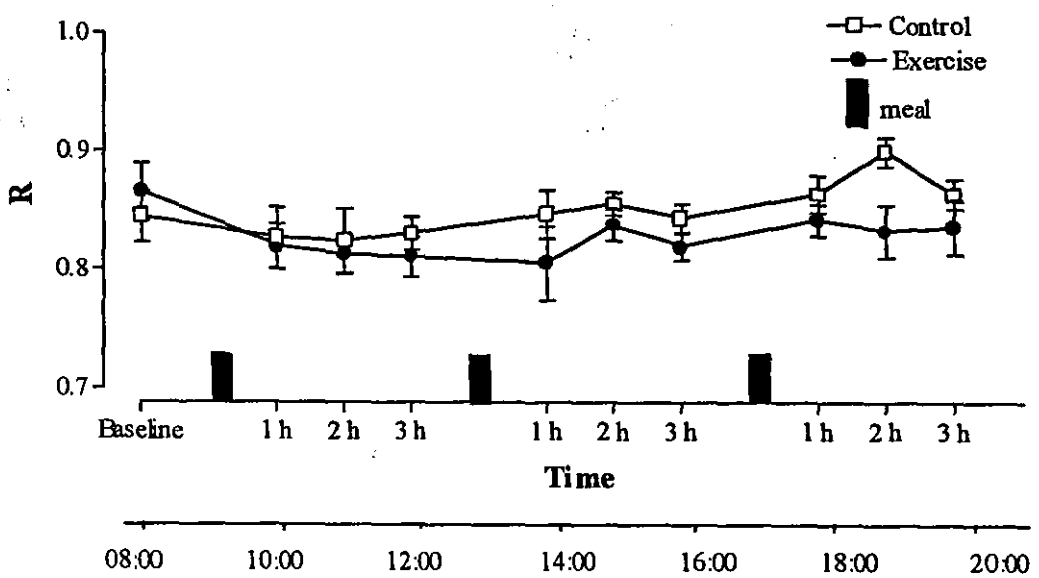


**Figure 7.6** Mean (s.e.m.) respiratory exchange ratio (R) for exercise (n=20) and CONTROL trials (n=10) (approximate real time is shown on the lower x-axis). (main effect treatment p=0.005)

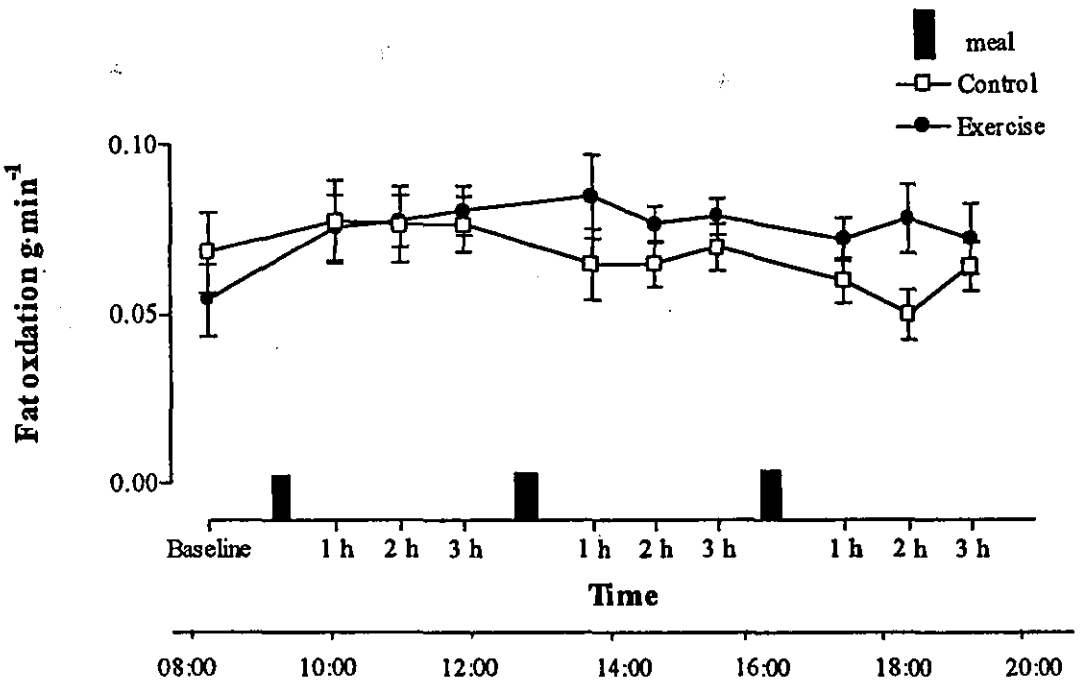


**Figure 7.7** Mean (s.e.m.) fat oxidation for exercise (n=20) and CONTROL trials (n=10) (approximate real time is shown on the lower x-axis). (main effect treatment p=0.009)

## 7.4 DISCUSSION

The main findings of this study were that 30 min of brisk walking diminishes postprandial lipaemia, during a day with normal meals. Dividing the exercise into three 10 min walks, a pattern endorsed by recent public health recommendations, did not alter this effect.

Subjects in the present study were typical in their physical characteristics and fasting lipid profiles of sedentary individuals of a similar age in the Northern Ireland population (MacAuley et al. 1994). The physiological demands of the exercise were similar in the LONG and SHORT trials. There was no difference in oxygen uptake, respiratory exchange ratios or heart rate during brisk walking between exercise trials. The energy expended during the exercise trials was estimated to be approximately 600 to 800 kJ depending on walking speed and body mass (Ainsworth 1993, Ross and Jackson 1986).

In addition to the energy cost of brisk walking, EPOC (discussed in Chapters 4.4 and 6.4) may have enhanced energy expenditure, in a different manner in LONG and SHORT trials, leading to a discrepancy in the total metabolic cost of the two exercise patterns. The effect of exercise on resting energy expenditure has been the subject of considerable research which has yielded inconsistent findings. Since the work of Edwards et al (1935) the observation that the rate of energy expenditure following exercise remains higher than the resting metabolic rate for several hours after exercise has been widely replicated (Edwards et al. 1935, Tremblay et al. 1988, Poehlman et al. 1991, Quinn et al. 1994). An almost equal body of literature suggests that the metabolic rate returns to pre-exercise resting conditions within an hour after exercise and that the net effect on postexercise energy expenditure is negligible (Knuttgen 1970, Pacy et al 1985, Brehm and Gutin 1986, Sedlock et al. 1989). The equivocal nature of the findings in such studies is due, at least in part, to differences in the duration and intensity of exercise as well as the training status of subjects. The earliest study which is similar in method to the current study is that of Bradfield (1968) who found that 45 minutes of brisk walking followed by ingestion of a test meal resulted in an elevation of resting metabolic rate which persisted for a period of 5 hours (Bradfield et al. 1968). The authors estimated the magnitude of this additional energy expenditure to be approximately 30 kcal. Bahr and colleagues (1987) have suggested that the magnitude of the elevation in postexercise energy expenditure is related to the duration of exercise

in a linear manner (Bahr et al. 1987). If this were true for all exercise durations, then the total of the additional energy expenditure caused by the exercise in both LONG and SHORT trials in the present study would be equal. To the authors knowledge no study has considered the effect of bouts of exercise which are as short as 10 min. A study by Quinn and coworkers (1994) noted that 20 min of treadmill walking at 70% of $\dot{V}O_2$ max evoked a similar excess post-exercise oxygen consumption as a 40 minute walk but that this was approximately half of the elevation caused by a 60 minute bout (Quinn et al. 1994). If the findings of Quinn et al (1994) are extended, one might reasonably suggest that performing three 20 minute bouts of exercise would cause an excess post exercise energy expenditure which is above that caused by one 60 minute continuous bout. Whether this would hold true for three 10 min bouts versus one 30 min bout is not clear. No study has identified a duration threshold required to incur an elevation in postexercise energy expenditure.

Only two other studies have considered the effect of splitting an exercise bout into smaller bouts on post-exercise energy expenditure. Kaminsky et al (1987) observed that dividing a 50 min run into two 25 min bouts significantly increases the post-exercise energy expenditure (Kaminsky et al. 1987). Using intensities and durations more akin to the present study, Almuzaini et al (1998) found that two 15 minute periods of cycling at 70% of $\dot{V}O_2$ max, 6 h apart, evoked a combined excess postexercise oxygen consumption which was approximately 40% greater than that induced by the same amount of continuous exercise (Almuzaini et al. 1998). Some authors have suggested that the thermic response to food may also be increased after exercise of 20 mins or longer in duration (Segal and Gutin 1983). This may cause an increased energy demand within resting muscle which is met by the metabolism of TAG-derived fatty acids. In the present study there was no difference in the total energy expenditure at rest between the three trials. However, since measurements were made 1 h after a meal i.e. 1.5 h after walking, any increase in metabolic rate following exercise may not have been detected. Moreover, the magnitude of any increase evoked by exercise, when observed as part of resting energy expenditure estimates over a 9 h period are likely to be negligible.

While the differences in metabolic rate and physiological response to exercise between SHORT and LONG trials may be negligible, metabolic differences in substrate metabolism were apparent. Total fat oxidation during the brisk walks, estimated from indirect calorimetry, was lower in the SHORT trial (6.7 ($\pm$ 0.9) g), compared to the

LONG trial (8.8 (± 1.23) g). At an intensity of 60% of $VO_2$ max, the contribution of fat oxidation to energy production increases as exercise progresses. Therefore the higher total fat oxidation in the LONG trial probably reflects an increased reliance on fat as a fuel with increasing exercise duration (Romijn et al 1993).

Expended endogenous TAG needs to be replaced by exogenous supplies (Annuzi et al. 1987) and therefore it might be reasonable to suggest that the greater deficit in fat stores incurred by exercise in the LONG trial might lead to an increased postprandial clearance. Several factors mitigate against such a straightforward explanation. A recent study has shown that the exercise-induced lowering of postprandial lipaemia is not due to the nature of the fuels metabolised during exercise (Malkova et al. 1999). Secondly, despite differences between exercise trials total fat oxidation during exercise in the present study (6.7-8.8 g) was modest compared to fat ingested in the breakfast meal alone (29.6 ±2.7 g). Even if muscle TAG stores were fully replenished from exogenous TAG-rich lipoproteins this would still not account for the differences in plasma TAG response between control and exercise trials. Finally, the postprandial lipaemic response to breakfast is similar in the SHORT and LONG trials despite the fact that in the SHORT trial the exercise expended only one third of the energy and approximately one quarter of the fat. Zhang and colleagues (1998) have suggested that after exercise, non-esterified fatty acids are taken up from the blood to replenish endogenous TAG stores (Zhang et al. 1998). In the current study there were no differences in the reduction in plasma non-esterified fatty acids from baseline to 1 h after breakfast between trials. It seems unlikely therefore, that the differences observed in plasma TAG response between exercise and CONTROL trials were solely the result of the need to replenish skeletal muscle TAG stores after exercise.

To the authors knowledge only two studies have considered the effect of exercise on postprandial lipaemia when the meal is ingested immediately after exercise. Burnett et al (1993) compared the effect of 90 minutes of low or moderate intensity running (48% and 66% of $VO_2$ max) immediately prior to ingesting a high fat meal. The study found a lower lipaemic response in both exercise conditions compared to controls (Burnett et al. 1993). Annuzi and coworkers (1987) considered the serum TAG response to an intravenous fat load administered immediately after 90 and 180 min of exercise. The decrease in TAG was not noted in the 40 min following intravenous fat tolerance test,

but became apparent 24 h later (Annuzzi et al. 1987). No observation of TAG response was made between in the period between 40 min and 24 h after the lipid injection.

Many of the studies considering the effect of exercise on postprandial lipaemia have used large fat loads. The magnitude of lipaemia is known to be proportional to the fat content of a meal (Cohen et al. 1988). In the current study meals were designed to reflect the energy and fat content of a typical western diet. It appears that such meals have the potential to challenge plasma TAG clearance mechanisms and result in levels of plasma TAG which are thought to be atherogenic (Patsch et al. 1992). In the present study, there were very large inter-individual variations in plasma TAG response. Incremental area under the plasma TAG concentration versus time curve ranged from 1.1 $mol \cdot l^{-1} \cdot 9h$ to 16.4 $mol \cdot l^{-1} \cdot 9h$. Although the variations in postprandial lipaemia between subjects are known to be large, the reproducibility of such measures within a given subject are good (Patsch et al. 1983). The plasma TAG response was lower for both exercise trials than for the CONTROL trial with remarkably similar postprandial responses between SHORT and LONG trials. A rise in plasma TAG during the postprandial period is the net result of the secretion of TAG rich lipoproteins from the intestine and liver and the removal from the plasma by tissues, mainly skeletal muscle and adipose tissue. When exercise is closely followed by the ingestion of a high fat meal it is possible that decreased splanchnic blood flow, caused by the re-distribution of blood during exercise, may decrease the rate at which TAG rich lipoproteins enter the plasma from the digestive system. The concomitant enhanced blood flow to skeletal muscle may also serve to increase the rate of removal of TAG from plasma by increasing exposure of LPL to its substrate. During the LONG trial, exercise was completed before breakfast yet plasma TAG was lower after the midday and evening meal, some 4 and 7 hours after exercise respectively, than during the CONTROL trial. This suggests that altered blood flow alone, cannot be responsible for the difference in plasma TAG response between exercise and CONTROL trials.

Kiens and Richter (1998) have suggested that after exercise skeletal muscle uses glucose to restore its glycogen levels and therefore, to meet the other energy needs of the muscle at rest, it has a greater reliance on TAG (Kiens and Richter 1998). The reduced plasma TAG response in the SHORT and LONG trials and the lower respiratory exchange ratios in the exercise compared to CONTROL trials support this suggestion. However, the energy cost of such short duration activity is low, and

therefore glycogen depletion as a result of exercise is likely to have been minimal. The lack of difference in postprandial TAG response to breakfast between SHORT and LONG trials, despite very different exercise durations (10 min and 30 min), suggests that the depletion and subsequent restoration of muscle energy stores was not a major cause of the differences in plasma TAG response between exercise and CONTROL trials.

Increased LPL activity is the most cited mechanism for the reduced postprandial lipaemia noted after exercise (Kantor et al. 1984; Sady et al. 1986; Herd 1997). The activity of skeletal muscle LPL increases after exercise but this increase is thought to be delayed to approximately 4 h after the exercise bout (Kiens et al. 1989). This delayed increase in LPL activity may account for the increasing meal x time effect found in the present study. In the LONG trial, the post lunch period was, in effect, 4 hours after exercise. At this point there is an increased departure in postprandial TAG response between exercise and CONTROL trials. Whether LPL activity is increased after such small amounts of exercise has not been investigated. It seems particularly unlikely that 10 minutes of brisk walking could increase LPL activity to such an extent that it would reduce postprandial lipaemia after lunch in SHORT versus CONTROL trials. More recently, Kiens and Richter (1998) have suggested that the increase in LPL following exercise may be more immediate, but suggest that this is more likely to be observed after longer, more exhaustive exercise (Kiens and Richter 1998). Annuzzi et al (1987) have suggested that only exercise beyond 90 minutes in duration increases the capacity of TAG removal as only in such exercise is glycogen depleted (Annuzzi et al. 1987). The partial or complete depletion of intramuscular glycogen stores has been suggested as one factor responsible for triggering the increase in LPL activity (Jacobs et al. 1982). The 30 minute brisk walk performed in the LONG trial of the present study would normally be considered insufficient to challenge glycogen stores. However, occurring, as it did, after an overnight fast, such exercise may have induced some degree of glycogen depletion (Dohm et al. 1983).

Studies have suggested that the ingestion of fat causes greater fat oxidation at rest. Respiratory exchange ratios have been shown to decrease following an all fat-meal (108 g) and to rise initially and then fall in the period following a mixed meal containing 121g, 70g and 50g of fat (Whitley 1997). In contrast, some authors have shown that fat taken in a meal does not stimulate greater fat oxidation in the subsequent 9 hour period

(Flatt et al. 1985). In a study by Griffiths (1994) adding 80g of fat resulted in 10g being oxidised and 70 g being stored (Griffiths et al. 1994). In the present study, estimated fat oxidation was 41, 52 and 53 g during CONTROL SHORT and LONG trials respectively. Mean fat intake during the trials was 139 g and therefore estimated fat storage of 98, 87 and 86 g occurred in CONTROL SHORT and LONG trials respectively. The mixed meals in this study resulted in a respiratory exchange ratio which was lower in exercise versus CONTROL trials. This suggests that when exercise is taken, the body preferentially utilises fat to meet the energy demands of skeletal muscle at rest.

The mean plasma TAG concentration over the course of the day follows a characteristically diurnal pattern (Nevill 1999). Indeed some authors have noted a wave-like TAG response with a peak at 14:00 in subjects who consumed meals at 09:00, 13:00 and 17:00 (Pagano Mirani-Oostidijk et al. 1983). These authors suggest that this is due to an increase in adipose tissue LPL activity that occurs over the course of the day and in particular in the late afternoon or early evening. Although this diurnal variation might explain the decrease in plasma TAG despite the ingestion of the evening meal the use of three trials in the present study allows the effects of intermittent and continuous exercise to be separated from this diurnal effect.

Several mechanisms for the reduced postprandial lipaemia observed in the SHORT and LONG trials compared to controls have been advanced in this discussion. It is of course possible that the reductions in postprandial lipaemia are the cumulative result of two or more of these mechanisms acting independently, sequentially or even synergistically. In the SHORT trial it could be a combination of increased blood flow to skeletal muscle after each exercise bout coupled with muscle TAG repletion while in the LONG trial this initial effect declines after 30 min of exercise giving way to a delayed increase in LPL activity which takes over after the midday meal. In the absence of any measure of LPL activity in the present study this explanation of the reduced postprandial TAG observed in LONG and SHORT trials remains speculative. Irrespective of the underlying mechanisms, the reduced postprandial lipaemia noted in SHORT and LONG trials compared to CONTROL, represents a reduction in the extent to which the arterial wall is exposed to high levels of TAG-rich particles.

The findings of the current study confirm that 30 min of brisk walking, performed in one continuous bout, or divided into three shorter bouts spread throughout the day lowers the lipaemic response to typical daily dietary intake among sedentary middle-aged subjects. This finding adds support to the notion that accumulating exercise over the course of a day may evoke a reduction in postprandial lipaemia and thereby contribute to one of the benefits sometimes associated with regular exercise, namely, decreased rate of progression of atherosclerosis.

# CHAPTER 8

## GENERAL DISCUSSION

The studies described in this thesis sought to compare the effects of continuous bouts of exercise with shorter bouts, of equal total duration, spread throughout the day. The findings of the studies suggest that splitting a continuous bout of exercise into smaller bouts does not alter its short- or medium-term physiological, or psychological effects.

The initial recommendations for an accumulated approach to physical activity were founded, to a large extent, upon epidemiological evidence with only a small number of longitudinal studies supporting this departure in physical activity guidelines (Pate et al 1995). Although a discontinuous pattern of activity is unlikely to harm sedentary individuals, advancing such recommendations, in the absence of solid scientific evidence that this type of activity enhances health, was perhaps a decision made in the face of large scale inactivity in the US population (Dunn et al 1999, Baringa 1997). Since they were first published, empirical support for the new recommendations has increased, but is still, by no means compelling. Specifically, there have been only a few well-controlled studies comparing the health benefits of this pattern of activity with the established benefits of a more traditional continuous approach. The studies described in this thesis add to the evidence which supports an accumulated approach to physical activity.

The results of the two training studies (Chapter 4 and Chapter 5) suggest that for sedentary individuals, a programme of short bouts of brisk walking, spread throughout the day, may enhance fitness, decrease selected cardiovascular risk factors and improve psychological well-being. The increases in $VO_2$ max in the two training studies are reasonably consistent, with a mean improvement of 8-9% for 10 weeks of short or long bout walking (Chapter 4) and 12-13% for 12 weeks of a similar programme (Chapter 5). Although the mechanisms underlying these improvements in fitness were not investigated in these studies, indirect evidence from the reduced blood lactate and heart rate response to submaximal tests suggests that both skeletal muscle and the cardiovascular system adapted. The results of these two studies suggest that such adaptations are a function of either exercise intensity or total energy expenditure or, more likely, a combination of these two parameters. The close agreement in the

magnitude of endurance fitness improvements between the two different patterns of brisk walking, in both studies, refute the suggestion that exercise must be continuous in order to have an effect. Some authors have reported greater improvements in endurance fitness with continuous bouts of activity (DeBusk et al 1990) The present studies do not support this conclusion. The recommendation for a pattern of short bouts of accumulated moderate intensity physical activity has been made, with the specific proviso that this type of activity is sufficient to promote health but that for fitness gains the more traditional approach may be required (Pollock et al 1998). The evidence from the two training studies contained in this thesis and several studies recently published (Asikainen et al 1998, Jakicic et al 1995, Snyder et al 1997, Woolf-May et al 1998) suggest that when recommending activity for sedentary middle-aged individuals such a proviso is unnecessary. Of course, once sedentary individuals have improved endurance fitness, further gains will probably necessitate activity that is more vigorous or of longer duration. Although the use of hilly terrain (Sutherland et al 1993), a graded treadmill or the addition of weight to the body (Evans, 1994), may increase the demands of brisk walking, an increase intensity is likely to require an increase in speed. There is a finite limit to the speed at which an individual can walk. At some given point, which may vary slightly between individuals, further increase in speed will require a transition to jogging (Thorstensson and Robertson 1987).

As well as enhancing fitness, regular physical activity also alters other aspects of cardiovascular risk. In the studies described in this thesis, three of these risk factors were considered; fatness, blood pressure and aspects of lipoprotein metabolism.

An established effect of regular exercise is the decrease in fat mass and increase or maintenance of lean body mass, particularly muscle (Ballor and Keesey 1991). These changes in body mass with a programme of brisk walking have been observed in many studies. In the two training studies reported in this thesis, alterations in body mass differed somewhat. In the study reported in Chapter 4 both patterns of brisk walking resulted in decreases in body mass, compared to controls after 10 weeks of training. Although there was no significant difference in mass loss between the two patterns, there was a tendency for short bout walkers to show a greater decrease in body mass. This trend has been previously suggested in similar studies (deBusk et al 1990, Jakicic et al 1995). Participating in short bouts of brisk walking may encourage a greater energy expenditure by increasing resting energy expenditure on several occasions during the

day or by requiring subjects to expend additional energy in preparing for or returning from such short walking sessions. Alternatively, three short bouts may necessitate walking during a lunch break, thereby reducing the time available for food consumption. In contrast, the findings of the training study reported in Chapter 5 suggest that neither pattern of brisk walking is sufficient to alter body mass after 6 weeks. Two explanations might reconcile these differences between the two training studies. Firstly, although the walking pattern and intensity in both studies was identical, the length of interventions was different (10 weeks vs 6 weeks). Secondly, although similar in body mass, subjects in the study described in Chapter 4 had somewhat higher body levels of body fat (sum of 4 skinfolds 68.0) at baseline than the subjects (female) in the study described in Chapter 5 (sum of 4 skinfolds 65.1). In both studies, alterations in body mass were below that which might be expected, given the additional energy expenditure caused by the programme. It is probable that free-living individuals, undertaking a new programme of physical activity, adjust their dietary intake or level of additional daily activity and that this resulted in the less-than-expected shift in body mass.

In both training studies, there were similar reductions in both waist and hip circumference with both patterns of walking. Waist:hip ratio, an important measure of fat distribution which has been linked to cardiovascular disease (Lapidus et al 1984) showed greater alteration in subjects using the short bout pattern than the long bout in the study reported in Chapter 4. However this difference between patterns was not noted in the training study described in Chapter 5. The difference in the alterations in hip and waist circumferences noted in the study described in Chapter 4 may have been due, in part, to initial waist and hip circumferences. For most body fatness measures, in both training studies, there was a tendency for subjects with the highest levels at baseline to experience the greatest decreases in fatness. Taken together however, the two training studies suggest that a short bout pattern of brisk walking is, at least as effective, as a long bout pattern in favourably altering body composition among overweight, sedentary individuals.

The findings of both training studies support the suggestion that regular physical activity can alter body fatness. The notion that a decrease in body fat may be linked to increased fat oxidation, mediated by skeletal muscle LPL, has been suggested in the literature. In a study of respiratory quotient over a 24 hour period, Zurlo and colleagues

have noted that a decreased ratio of fat to carbohydrate oxidation is a predictor of body-weight gain (Zurlo et al 1990). The mechanism underlying the relationship between decreased body mass and the increased ratio fat to carbohydrate oxidation is reported to involve an increase in skeletal muscle LPL activity. Some authors have found a directly proportional relationship between skeletal muscle LPL activity and whole body fat to carbohydrate oxidation ratios (Ferraro et al 1993). An increase in skeletal muscle LPL activity may cause a greater removal of plasma TAG by this tissue while conversely a lower skeletal muscle LPL activity promotes greater fat storage in adipose tissue. This suggestion that brisk walking promotes fat oxidation is supported by the study described in Chapter 7. Subjects completing 30 minutes of brisk walking in either pattern displayed greater fat oxidation (estimated by indirect calorimetry) over the course of the day than inactive controls. If extrapolated for the training programmes described in Chapters 4 and 5, this may account, in part, for the decreases in body fat observed.

In the two training studies reported in this thesis, both patterns of brisk walking resulted in decreases in resting blood pressure. The decreases occurred in the mean systolic and diastolic blood pressure, but were only significant for systolic blood pressure for the study reported in Chapter 4 and for diastolic blood pressure for the study described in Chapter 5. There were however no differences in the magnitude of these changes between the two walking patterns. Decreases in either component of blood pressure are likely to be the result of a reduction in sympathetic tone (Jennings et al 1989). This response does not therefore, appear to be dependent on continuous exercise but may be linked to the relative exercise intensity at which exercise is performed. An examination of the individual baseline resting blood pressures, in both training studies, supports the view that those with initially elevated levels are most likely to benefit. This is because physical activity tends to normalise blood pressure and once normal, exercise is unlikely to have further effects (Hagberg and Brown 1995). Blair and colleagues suggest that even a small increase in systolic blood pressure within the range 120-129 mm Hg is associated with a three-fold increase in developing hypertension (Blair et al 1989). Cross-sectional data from Paffenbarger and colleagues suggest that hypertensive men have twice the risk of all-cause mortality than normotensives (Paffenbarger et al 1986). Reductions in blood pressure in the training studies may have been mediated by alterations in body mass (Berchtold et al 1982) or body fatness. Although the design of the training studies described in Chapters 4 & 5 make it difficult to identify an independent effect of exercise on blood pressure, the decreases in blood pressure

following both patterns of brisk walking in both studies still represent a reduction in cardiovascular risk (Blair et al 1992).

Interventions which alter the circulating levels of different lipoprotein species have the potential to slow the rate of progression of atherosclerosis by reducing the build-up of plaque on the intima of the arteries. Three of the studies described in this thesis considered the effect of exercise on blood lipids. In the study described in Chapter 5, favourable alterations in fasting blood lipid profiles were noted in response to both patterns of brisk walk training, with no differences in the magnitude of these alterations between the two patterns. Although small, the increase in fasting plasma HDL cholesterol and the decreases in fasting TAG and total cholesterol concentration may have important implications for cardiovascular risk. A 1% increase in HDL cholesterol is associated with a 3% reduction in coronary disease risk (Manninen et al 1988). The importance of the reductions in total cholesterol and fasting TAG is less well defined, however both are associated with reduced cardiovascular risk (Patsch et al 1992, Bush et al 1988).

Haskell (1994) has suggested that some of the major health-related changes associated with physical activity may be due to the acute physiological alterations which occur during and persist for some time after a bout of activity (Haskell 1994). How are these 'last bout effects' likely to have influenced the findings of the two training studies? In the study described in Chapter 4 , post-training measurements were taken two or more days after the last bout of exercise. In the study described in chapter 5, post-training tests were carried out on the morning after one day without training. Although a bout of exercise has been shown to reduce blood pressure for a period after exercise, this effect is reported to last up to 12 h in sedentary hypertensive individuals (Brown et al 1994). The post-training blood pressure in both studies, therefore is likely to be unaffected by such last bout effects. Alterations in body fat are dependent upon a negative energy balance that is only likely to be detected over a period of weeks and months rather than days and therefore the timing of body mass and body composition measurements is unlikely to have influenced the alterations. In contrast, alterations in blood lipids are known to occur acutely and persist for up to 48 hours after exercise (Lee et al 1991). Although this may serve to confound the measurement of the long-term effects of exercise, measuring blood lipids within 48 hour after the last session is likely to represent the most realistic estimation of the day-to-day benefits of a physically active

lifestyle. The alterations in blood lipids in the study described in Chapter 5 therefore may reflect both short- and medium-term effects on lipoprotein metabolism, but these alterations best represent the benefits which a sedentary middle-aged person may expect by adopting a moderately active lifestyle.

One of the short-term effects of physical activity is a reduction in the postprandial lipaemic response. Repeated episodes of exaggerated postprandial lipaemia may hasten the progression of atherosclerosis (Patsch et al 1992). The studies described in Chapters 6 and 7 attempted to compare the effects of different patterns of physical activity on this reduction in lipaemic response to a meal. Given that a prolonged bout of both low and moderate intensity exercise is known to reduce the lipaemic response to a high fat meal consumed the following morning (Tsetsonis et al 1996a) the study reported in Chapter 6 considered the effect of splitting this exercise into smaller intermittent bouts. The similar reduction in lipaemic response in both patterns of exercise suggests that many of the benefits, in terms of postprandial lipaemia must be related to the energy expenditure caused by the exercise. The use of prolonged bouts, followed by an overnight fast and an abnormally high fat breakfast, whilst illuminating, has limited applicability to everyday life. The study described in Chapter 7 attempted to compare the effects of the two patterns of brisk walking, using more modest amounts of exercise (30 min), on the postprandial response to meals that are typical of the population. The results suggest that both patterns of brisk walking have the potential to reduce the postprandial triacylglycerol response to typical meals and that there is no difference in the magnitude of this reduction between exercise patterns.

If this modest amount of exercise, taken in either a continuous or an accumulated pattern, is performed daily, it seems plausible that over time, the resultant reduction in postprandial lipaemic responses may reduce the rate of progression of atherosclerosis (Patsch et al 1992). This may be one of the primary mechanisms underlying the inverse relationship between lifestyle activity and coronary heart disease risk often noted in the epidemiological literature (Powell et al. 1987)

One prevailing theme in the explanation of the alterations in endurance fitness, and the other cardiovascular risk factors observed in response to training, has been the importance of the initial physiological state of the individual, prior to embarking on a training programme. It appears the brisk walking, in either pattern, has the greatest

potential to enhance fitness and improve health among those who are least fit and most at risk of cardiovascular disease (Snyder et al 1997). Although perhaps obvious, this is an important concept where an intermittent pattern of brisk walking is concerned. Persuading the most sedentary to become even minimally active is considered to be the most efficient way of reducing the public health burden of cardiovascular disease (Haskell 1994). This intermittent pattern of brisk walking offers a bridge between inactivity and the more traditional exercise prescription and on the basis of an accruing body of scientific literature, can be endorsed, with confidence, for sedentary individuals. There is a possibility that even less activity may be of benefit for the chronically sedentary individual. The work of Dunn and colleagues (1999) show that even those participants who fell well short of the physical activity goal of 30 minutes per day significantly improved fitness and reduced cardiovascular risk factors (Dunn et al 1999). If the dose-response relationship is accepted then the most sedentary portions of the population may initially benefit from even very modest amounts of physical activity, perhaps even less than the level currently recommended..

The notion that energy expenditure is a principal feature of health-enhancing physical activity is reinforced by the findings from all four studies described in this thesis. Indeed, Hambrecht and colleagues (1993) have suggested that an energy expenditure of 6.7MJ per week (1600 kcal) may halt the progression of CAD and an energy expenditure of 9.2 MJ per week (2200 kcal ) may be needed to promote regression (Hambrecht et al. 1993). Although the energy expended by the brisk walking performed in each pattern of brisk walking employed in the studies reported in this thesis is below this level, it is probable that subjects in these studies, and individuals adopting physical activity programmes will also continue performing other occupational or household activity which may raise the total energy expenditure closer to the level required to produce significant cardiovascular benefit.

While the physiological changes derived from regular physical activity appear to rely upon energy expenditure, there is little evidence that this factor affects the alterations in psychological status observed after a programme of physical activity. The mechanisms underlying the reductions in tension and anxiety and increases in positive affect noted in the study reported in Chapter 5 are less clear. However, the role of increased self-efficacy, in both parameters is indicated (Csikszentmihali 1982). Mean self-efficacy scores for walking and other activities were higher after training in both patterns of

brisk walking and although these increases were not significant, they suggest that involvement in either short or long bouts of activity increases an individual's belief in their ability to perform continuous bouts of brisk walking and a range of other challenging activities. In this way, participation in a programme of short bouts of brisk walking may self-reinforcing and may be an entry route to more prolonged and varied physical activities for a sedentary individual.

Long term health benefits require long term behavioural changes (Andersen et al 1999). Despite the intuitive belief that accumulated bouts of brisk walking will fit more easily into lifestyle and thereby address time as a perceived barrier, a lack of time was still the primary reason for drop out from the short walks programme in both training studies. Although these motives were not probed, it seems that any structured exercise programme, may be perceived by sedentary individuals, as another demand in an already busy schedule. In attempting to address this perceived 'time barrier', a more ecologically-based regimen such as the recently described lifestyle activity (Andersen et al. 1999, Dunn et al. 1997, Dunn et al. 1999, Phillips et al. 1996) may be more beneficial, in terms of adherence than any variation in patterns of exercise performed with in an structured programme. For reasons of control and comparability the short bouts of exercise in all four studies described in this thesis were performed at specific minimal intervals apart and were always of a defined duration e.g. 10 minutes in the three walking studies. In real-life, free-living individuals would most likely accumulate exercise in a more sporadic pattern with bouts of varying duration being performed when the opportunity or time allowed. The findings of the studies described in this thesis can only endorse the effectiveness of three 10 minute bouts of brisk walking. Since energy expenditure has been highlighted as the stimulus underlying many of the physiological changes observed, it seems that shorter more frequent bouts and other forms of physical activity are likely to be equally beneficial. However before such patterns are widely publicly promoted they require further investigation in randomised clinical trials.

This notion that improvements in $VO_2$ max and selected health benefits of exercise can be derived, by sedentary middle-aged individuals, from an accumulated patterns of brisk walking, may have implications for public health promotion. Several of the barriers to regular physical activity are overcome by the use of brisk walking and by an accumulated approach. Specifically lack of time, inadequate facilities, expense, social

acceptance and poor self-efficacy for sporting pursuits are barriers which are directly overcome by this somewhat novel exercise prescription. Even barriers such as limiting health, excess weight and inconvenience are partially addressed by accumulated brisk walking.

One important limitation of the training studies reported in this thesis and those upon which current activity guidelines are based is the representativeness of the subjects. Subjects in these studies are, most often, willing volunteers who *a priori* are interested in taking up physical activity. Although in physical and physiological attributes these individuals may be similar to sedentary individuals at whom the current exercise guidelines are aimed, it is probable that their psychological readiness to take part in regular physical activity is somewhat different.

In conclusion, the studies which have been presented in this thesis provide support for an accumulated approach to physical activity. Moreover it seems that for sedentary individuals, who represent a majority of the population, short bouts of brisk walking may bring considerable physiological benefits and may reduce cardiovascular risk.

Such findings are encouraging for public health promotion, where a perceived lack of time and self-perceptions about sporting ability are considered popular reasons for sedentariness. A pattern of short bouts of exercise is easier to incorporate into a busy lifestyle and brisk walking is a widely accessible form of exercise that has little association with more skilful sporting pursuits. It appears that encouraging individuals to perform short brisk walks in the course of their daily routines may be as effective in producing increases endurance fitness and selected health benefits as more traditional approaches to exercise prescription.

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# APPENDIX A

## ETHICAL APPROVAL FROM UNIVERSITY OF ULSTER RESEARCH ETHICAL COMMITTEE

Ms M Murphy

Nick Curry, Research Office

NC:REC

6 November 1995

**RESEARCH ETHICAL COMMITTEE APPROVAL** Project no. 95/28

*The chronic and acute physiological responses to intermittent and continuous brisk walking among sedentary women*

At the meeting of 27 October 1995 the Research Ethical Committee considered your application and gave approval for it to proceed with no amendments.

NICK CURRY

UNIVERSITY of ULSTER
MEMORANDUM

To: Mrs M H Murphy, Leisure & Tourism, JN

From: Nick Curry, Research Office, JN

Ref: NC:KB

Date: 21 September 1998

**Research Ethical Committee**

**Project 98/27:** *The effects of short and long bout brisk walking programmes on selected fitness, health and psychological parameters*

The Chairman, on behalf of the Research Ethical Committee, has considered the above application and has given permission to proceed.

It has been suggested, however, that a shorter consent form might be appropriate. You might like to consider separating the detailed information from the form to use as a leaflet and using a summary for the purposes of obtaining a signature.

NICK CURRY

UNIVERSITY of ULSTER

MEMORANDUM

To: Marie H Murphy, Leisure & Tourism, JN

From: Nick Curry, Research Office, JN

Ref: NC:KB

Date: 29 April 1997

**Research Ethical Committee**

**Project 97/8:** *The acute effects of intermittent v continuous brisk walking on post-prandial lipaemia*

The Chairman, on behalf of the Committee, has considered your response (dated 9 April 1997) to queries raised at the meeting of 25 February 1997 and has given permission for the project to proceed.

NICK CURRY

# APPENDIX B

# INFORMED CONSENT

**WALKING STUDY 1**

**CONSENT FORM**

The aim of the study is to consider the effects of two different walking programmes on fitness among previously sedentary women. The following pages detail what is involved for subjects- please read these carefully before agreeing to participate. If you have any questions do not hesitate to ask.

**PRE-TRAINING PROCEDURES**

Health and Screening questionnaire

Following the return of this form (informed consent) you will be asked to fill out a short questionnaire about current and past health. All answers will be treated in strictest confidence.

LAB - VISIT 1 Habituation

During the first visit to the lab (which is located in Block 11 - behind the fitness suite) you will be given an opportunity to practice walking on the treadmill and using the mouthpiece, in addition blood pressure and body composition will be measured as described below.

Resting Blood Pressure

On the first two visits to the laboratory resting blood pressure will be measured. After ten minute rest lying down blood pressure will be recorded in the normal way using an inflatable cuff wrapped around the upper arm

Body Composition

Height and weight will be measured using appropriate scales. A measure of the thickness of the skinfolds at four sites on the body (front and back of upper arm, under shoulder blade and above hip bone) will be used to estimate the percentage of the body that is made up of fat and fat free weight. Waist and hip measurements will be taken to indicate distribution of body weight.

LAB - VISIT 2 Maximal Treadmill Test

A treadmill walking test will be conducted. This will last between 9 -16 minutes. The treadmill speed will stay the same throughout the test but at regular intervals the slope will get steeper, so that it gets a bit harder. The test is maximal so that you decide when you cannot continue and the treadmill will be stopped. Your heart rate will be monitored continually and the air which you breathe out will be analysed. You will need to wear comfortable trousers and flat supportive shoes.

LAB - VISIT 3 Submaximal Treadmill Test

A 16 minute treadmill walking test will be conducted. You will be asked to walk on the treadmill at 4 different slopes. These will represent 50% 60% 70% and 80% of your own maximum exercise capacity. During this test 4 small thumb prick samples of blood will be taken. Again you will need to wear comfortable trousers and flat supportive shoes.

**TRAINING PROGRAMME**

Having completed the pretest procedures subjects will be randomly assigned to one of three groups

3 x 10 minutes

Individuals in this group are asked to walk briskly for 10 minutes continuously, on three occasions during the day, on five days per week. Walking bouts should be separated by at least 4 hours (e.g. 8:00, 12:30 and 5:00) The walking can be done outside on level ground or in the lab or fitness suite on the treadmill.

1 x 30 minutes

Individuals in this group are asked to walk briskly for 30 minutes per day, five days per week. The walking can be done outside on level ground or in the lab or fitness suite on the treadmill

Supervised Training sessions
Individuals in both of the training groups are asked to attend one supervised session (10 or 30 minutes) per week to ensure that the correct training intensity is being achieved.

No Training
Individuals in this group are asked to take part in no training or additional activity for the 12 week period of the study. At the end of the study individuals in this group will be given an opportunity to take part in a supervised training programme.

All three groups will be asked to keep a weekly activity diary to record any activity above resting levels (including training sessions). All individuals taking part in the study are asked not to change their diet in any way during the study and as far as possible maintain a usual lifestyle.

**POST-TRAINING PROCEDURES**
At the end of the 12 week study subjects will be asked visit the lab on two final occasions to repeat the measurements made before the study began.

| Lab Visit 1 | Lab Visit 2 |
|---|---|
| Submaximal Treadmill Test | Maximal Treadmill Test |
| Resting Blood Pressure | Resting Blood Pressure |
| Body Composition | |
| Submaximal Treadmill Test | |

**RISKS AND DISCOMFORTS**
The possibility exists that very occasionally certain changes may occur during the test. These include abnormal blood pressure, fainting or very rarely, minor disorder of the heartbeat. Sampling of blood may cause minor bruising to thumb. All tests will be conducted by trained staff and closely monitored.

**BENEFITS**
All individuals will receive feedback on their resting blood pressure, body composition and "aerobic" fitness at the end of the study.

Those individuals assigned to one of the two training groups will receive individualised advice on the optimal walking pace and will have any improvements in fitness or changes in body composition monitored during the study.

Those who are assigned to the no-exercise group will be given an opportunity to take part in a similar training programme at the end of the study.

Informed Consent

I have read the above description of the requirements of the study. All tests and procedures have been fully explained to me and I agree to take part in the study. I am aware that I am free to withdraw from the study at any time without the need for explanation

Name (PRINT)_____

Signed_____Date_____

Witnessed by_____

## WALKING STUDY
## CONSENT FORM

The purpose of this study is to assess the effects of two different walking programmes on selected fitness, health, and psychological parameters in men and women who do not at present take exercise. The following pages detail exactly what is involved for subjects - please read these carefully before agreeing to participate. If you have any questions please do not hesitate to ask.

**PRE-TRAINING PROCEDURES:**

**Health and screening questionnaire** - following the return of this form (informed consent) you will be asked to fill out a short questionnaire about current and past health. All answers will be treated in strictest confidence.

**Visit 1** - during the first visit to the lab (which is located in Block 15) a number of baseline measurements will be made, as described below:

- Resting Blood Pressure - after ten minutes rest sitting down, resting blood pressure will be recorded in the normal way using an inflatable cuff wrapped around the upper arm.
- UKK 2km Walk Test - this will involve walking 2 km (just over 1 mile) as briskly as you can on a level surface (sports hall). During this walk, your heart rate will be monitored.
- Psychological Measures - you will be asked to complete 3 short questionnaires on how you feel about exercise. These will involve circling your chosen response to a series of questions.

**Visit 2** - during the second visit to the Lab, a number of blood, fitness, and psychological measures will be recorded.

- Fasting Blood Lipids - this will involve taking a small blood sample from a vein in your arm in order to determine the fat levels in the blood. You will be required to fast from 10pm the night before this blood sample.
- Body Composition - height and weight will be measured using appropriate scales. Waist and hip measurements will be taken to indicate distribution of body weight. A measure of the thickness of the skinfolds at designated sites on the body (arm, hip, and shoulder blade) will be taken to estimate the percentage of the body that is made up of fat and fat free weight

**TRAINING PROGRAMME:**

Having completed these tests, you will be assigned to one of two groups. The first group will walk for 3 x 10 minutes per day for the first six weeks, and for 1 x 30 minutes per day for the second six-week period. The second group will walk for 1 x 30 minutes per day for the first six weeks, and 3 x 10 minutes per day for the second six week period. There will be a two week rest period between these two training programmes.

1. **3 x 10 minutes** - individuals in this group will be asked to walk briskly for 10 minutes continuously, on three occasions during the day, on five days of the week. Walking bouts should be separated by at least 3 hours (e.g. 8.00, 11.30 and 4.00). The walking can be done outside on level ground, or in the lab, or in the fitness suite on the treadmill.

2. **1 x 30 minutes** - individuals in this group will be asked to walk briskly for 30 minutes per day, five days per week. The walking can be done outside on level ground, or in the lab, or in the fitness suite on the treadmill.

**Supervised Training Sessions** - heart rate will be monitored during 2 days of each six week training perio to ensure that the correct training intensity is being achieved.

**Diary** - you will be asked to keep a weekly activity diary to record the walks that you do, your pulse, an how you feel at the end of each walk. For the duration of the study you are asked not to change your diet o lifestyle in any way.

**POST-TRAINING PROCEDURES:**

At the end of the first 6 week training period, you will again visit the Lab, and the measurements outlined i the pre-training section will be repeated (blood pressure, height, weight, waist and hip measurements skinfolds, blood lipids, 2km Walk Test, and the psychological measures).

Following this there will be a 2 week rest period, during which time you will be asked to refrain from an exercise, and not make any changes to your diet. At the end of this 2 week period, you will again visit th Lab, and the measurements outlined above will be repeated.

After these measurements have been taken, the training programme will resume. This will involve anothe six week exercise programme, of either 1x 30 minutes walking, or 3 x 10 minutes. At the end of this period you will return to the Lab for the final time, and the pre-training measurements will be repeated.

**RISKS AND DISCOMFORTS:**

The possibility exists that very occasionally certain changes may occur during the walking test. Thes include abnormal blood pressure, fainting, or very rarely, minor disorder of the heartbeat. Sampling o blood may cause minor bruising to the arm. All tests will be conducted by trained staff and closel monitored.

**BENEFITS:**

You will receive feedback on your resting blood pressure, body composition, blood lipids, and "aerobic fitness at the end of the study.

You will also receive individualised advice on optimal walking pace and will have any improvements i fitness or changes in body composition monitored during the study.

**INFORMED CONSENT:**

I have read the above description of the requirements of the study. All tests and procedures have been full explained to me and I agree to take part in the study. I am aware that I am free to withdraw from the study a any time without the need for explanation.

Name (PRINT): _____

Signed: _____ Date: _____

Witnessed by: _____

## RUNNING STUDY

## CONSENT FORM

The aim of the study is to consider the effects of two different patterns of running on the response to a high-fat meal. The following pages detail what is involved for subjects- please read these carefully before agreeing to participate. If you have any questions do not hesitate to ask.

**PRE-TEST PROCEDURES**

Health and Screening questionnaire

Following the return of this form (informed consent) you will be asked to fill out a short questionnaire about current and past health. All answers will be treated in strictest confidence.

LAB - VISIT 1 Maximal Treadmill Test

A treadmill test will be conducted. This will last between 9 -16 minutes. The treadmill speed will stay the same throughout the test but at regular intervals the slope will get steeper, so that it gets a bit harder. The test is maximal so that you decide when you cannot continue and the treadmill will be stopped. Your heart rate will be monitored continually and the air which you breathe out will be analysed.

LAB - VISIT 2 Submaximal Treadmill Test

A 16 minute treadmill walking test will be conducted. You will be asked to run on the treadmill at 4 different speeds. This will allow us to identify the correct speed for your exercise trials.

**3 ONE-DAY TRIALS**

Having completed the pre-test procedures, you will be asked to undertake three trials in a random order. Each trial will take place over 2 days. The day before your first trial and day 1 of this trial, you will be asked to keep a record of the food and drink that you consume. You will then be asked to match this intake exactly for the two remaining trials.

On Day 1 you will be asked to:

Run on the treadmill for 30 minutes on three occasions, at 9:00 am, 12:30 pm and 4:30 pm. The speed of the treadmill will be set at 60% of your maximum.

or

Run on the treadmill for 90 minutes at 3:30 p.m. The speed of the treadmill will be set at 60% of your maximum.

or

No exercise.

On Day 2:

You will be asked to arrive in the lab (Block 11) after an overnight fast. A small blood sample will be taken from a vein in your arm. You will then be asked to eat a breakfast containing muesli, cream, fruit and nuts. At 2, 3 and 6 hours after the meal another blood sample will be taken. During the trial you will be required to remain at rest in the laboratory or within the university (working quietly).

The three trials will be spaced a minimum of one and a maximum of 2 weeks apart.

The possibility exists that very occasionally certain changes may occur during the maximal treadmill test. These include abnormal blood pressure, fainting or very rarely, minor disorder of the heartbeat. Blood sampling may cause minor bruising to the arm which will normally disappear within a few days.

Informed Consent

I_____

Of_____

Have read the description of the requirements of the study. All procedures have been fully explained to me and I agree t take part in the study. I am aware that I am free to withdraw at any point without the need for explanation.

Signed_____Date_____

Witnessed by_____

# CONSENT FORM

The aim of the study is to consider the effects of two different walking regimes on the blood lipid responses to food intake among men and women. The following pages detail what is involved for the subjects - please read these carefully before agreeing to participate. If you have any questions do not hesitate to ask.

## PRE-TESTING PROCEDURES

LAB - VISIT 1 Habituation and Maximal Treadmill Test

Following the return of this form (informed consent) you will be asked to fill out a short questionnaire about current and past health. All answers will be treated in strictest confidence. During the first visit to the lab (15C14) you will be given an opportunity to practice walking on the treadmill and using the mouthpiece, in addition resting blood pressure will be measured. After ten minutes rest lying down blood pressure will be recorded in the normal way using an inflatable cuff wrapped around the upper arm.

A treadmill walking test will also be conducted. This will last between 9-16 minutes. The treadmill speed will stay the same throughout the test but at regular intervals the slope will get steeper, so that it gets a bit harder. The test is maximal so that you decide when you cannot continue and the treadmill will be stopped. Your heart rate will be monitored continually and the air which you breathe out will be analysed. You will need to wear comfortable trousers and flat supportive shoes or trainers.

LAB - VISIT 2 Verification Test

A verification test will be conducted on the treadmill to ensure that the required walking intensity, calculated from the above procedure, is correct. This will involve approximately 10 - 15 minutes walking on the treadmill. You will need to wear comfortable trousers and flat supportive shoes or trainers.

## 3 ONE-DAY TRIALS

Having completed the pretest procedures you will undertake three trials in a random order. Each trial will last one full day (8 am - 7 pm). On each trial after an overnight fast you will come to the laboratory at 8 am. A very small plastic tube (cannula) will be inserted into a vein in your arm by qualified personnel. This feels similar to having a blood sample taken. This will remain in the vein for the duration of the trial but will be covered by a plaster, and should not cause any discomfort. This allows small blood samples to be taken during the day without the need for further needles. At the end of the day this will be removed.

1. On one trial you will be asked to walk on the treadmill for 10 minutes in the morning, ten minutes at lunch time and ten minutes in the evening. After each walk you will be given a meal (breakfast, lunch or dinner). Every hour thereafter a small blood sample will be taken and the air that you breathe out will be analysed to determine fat and carbohydrate usage. At 7 pm (3 hours after dinner) the last blood sample will be taken, the cannula will be removed and you will be free to go home.

2. On one trial you will repeat all of these procedures but the exercise will be one 30 minute walk on the treadmill at just before breakfast.

3. On one trial you will repeat all of these procedures but there will be no exercise during the day.

Where possible, trials will be spaced a minimum of one week and a maximum of two weeks apart. On all three trials you will be asked to remain at rest, reading or doing desk-based work. Television/ videos/ music and newspapers will be available if you wish. Where possible 2-3 men or women will be tested each day.

## RISKS AND DISCOMFORTS

The possibility exists that very occasionally certain changes may occur during the maximal treadmill test (Visit 1). These include abnormal blood pressure, fainting or very rarely, minor disorder of the heartbeat. The insertion of the cannula (on each trial) may cause bruising to the arm which will normally disappear within a few days. All tests will be conducted by trained staff and closely monitored.

## BENEFITS

All individuals will receive feedback on their resting blood pressure, "aerobic" fitness, fasting blood lipids, metabolic rate and lipid response to food at the end of the study. In addition you will be assisting us in determining which type of exercise, intermittent or continuous is most beneficial for your health.

All meals will be provided on trial days.

## Informed Consent

I (print name) _____

of (print address) _____

_____

_____

have read the above description of the requirements of the study. All tests and procedures have been fully explained to me and I agree to take part in the study. I am aware that I am free to withdraw from the study at any time without the need for explanation.

Signed _____ Date _____

Witnessed by _____ Date _____

Investigator _____ Date _____

# APPENDIX C

# HEALTH HISTORY QUESTIONNAIRE

**HEALTH HISTORY QUESTIONNAIRE**

Name: _____ DOB: _____ Age: ____

Address: _____

Telephone Number - Daytime: _____ Evening: _____

Any information contained herein will be treated as confidential.

*** Please answer all questions. Circle the appropriate answers. ***

1. Have you ever been diagnosed as having any type of heart complaint? YES/NO
   If YES, please give details _____
   _____
   _____

2. Have you had to consult your doctor within the last 6 months? YES/NO
   If YES, please give details _____
   _____
   _____

3. Are you presently taking any form of medication? YES/NO
   If YES, please give details _____
   _____
   _____

4. Do you, or have you ever, suffered from:

| | |
|---|---|
| Asthma? | YES/NO |
| High Blood Pressure? | YES/NO |
| Heart Disease? | YES/NO |
| Lung Disease? | YES/NO |
| Diabetes? | YES/NO |
| Epilepsy? | YES/NO |
| Thyroid Disease? | YES/NO |
| Heart Murmur? | YES/NO |

   If YES, please give details _____
   _____
   _____

5. Have any of your immediate family had:

| | |
|---|---|
| High Blood Pressure? | YES/NO |
| Heart Attack? | YES/NO |
| Stroke? | YES/NO |
| Diabetes? | YES/NO |
| Early or Sudden Death? | YES/NO |
| Clotting Disorders? | YES/NO |

If YES, please give details _____

_____

_____

6. Do you currently have any form of muscle or joint injury? YES/NO

If YES, please give details _____

_____

_____

7. Is there anything to your knowledge that may prevent you from completing the tests that are outlined in the Informed Consent Form. YES/NO

If YES, please give details _____

_____

_____

8. Do you smoke? YES/NO If YES - _____ cigarettes per day.

Signature: _____ Date: _____

# APPENDIX D

# CALIBRATION PROCEDURES FOR GAS ANALYSIS SYSTEMS

## Calibration of Oxycon-4

**Procedure:**

1. The analyser was switched on for 60 minutes prior to calibrating

2. A 3 1 calibration syringe was connected to the patient hose input. Ten volume strokes were pumped into the system. Displayed volume ($V_B$ key) and respiratory frequency ($F_R$) were confirmed or adjusted to read 30 1 and 10 breaths respectively.

3. Zero calibration: A gas bag containing pure nitrogen was connected to the 'reference gas' input. After 15 seconds $O_2$ should read '0'. If not adjustments to the $O_2$ potentiometer were made.

4. Span with atmospheric air: Gas bag was removed from the input pipe and within 15 seconds $O_2$ display should return to 20.9%

5. $CO_2$ calibration: A gas bag containing 6% $CO_2$ was connected to the 'reference gas' input. After 15 seconds $CO_2$ should read '6.0'. If not adjustments to the $CO_2$ potentiometer were made.

6. Steps 3-5 are repeated until no adjustments to the potentiometers are required.

7. Calibration check: A gas bag containing a 'gold standard' gas of a known concentration within the range 4 - 4.2% $CO_2$ and 16-16.4% $O_2$ was connected to the 'reference gas' input to check the calibration.

## Calibration of Quinton Metabolic Cart

Procedure:

1. The QMC was switched on for 30 minutes prior to calibrating
2. Using 'low' calibration gasses supplied by manufacturer (10±0.3% $O_2$), the $O_2$ and $CO_2$ volts were adjusted to within 0.020 volts using the 'low $O_2$' and 'low $CO_2$' knobs if necessary.
3. Using 'high' calibration gasses supplied by manufacturer (25±0.3% $O_2$ and 5±0.3% $CO_2$), the $O_2$ and $CO_2$ volts were adjusted to within 0.020 volts using the 'high $O_2$' and 'high $CO_2$' knobs if necessary.
4. The pneumotachometer was calibrated using a 3 litre syringe (Hans Rudolph Inc. USA). The syringe was attached to the subject hose via the 2-way non-rebreathing valve. Zero flow volts were adjusted to within 0.020 using the 'Pneumotach zero' knob.
5. Steps 2-4 are repeated until no adjustments have to be made to 'high' 'low' or 'pneumotach zero' knobs
6. 30 litres of air were pumped through the syringe, this was repeated 3 times before the calibration factors were updated and saved.

# APPENDIX E

# ASSAY PROCEDURES

## Haematocrit determination

Haematocrit was determined on fresh blood using a Hawksley microcentrifuge (Hawksley & Sons Ltd. England) and micro-haematocrit reader. Plasma volume was estimated from haematocrit and haemoglobin concentration using the method described by Dill and Costill (1974)

Equipment and Materials:
Heparinised capillary tubes
Cristaseal
Micocentrifuge MK5
Micro- maematocrit reader
(All supplied by Hawksley & Sons Ltd. England)

Procedures:

1. 20 µl of fresh whole blood was drawn into a heparinised capillary tube
2. Tube was sealed at both ends with Cristaseal
3. Sample was centrifuged for 5 min at 11,800 rpm
4. Haematocrit read using microhaematocrit reader

## Haemoglobin Assay

In the study described in Chapter 7 Haemoglobin concentration was determined using a photometric cyanmethaemoglobin method using a Coulter Haemoglobinometer (Coulter Electronics Ltd). In the studies described in Chapters 5 and 6 haemoglobin concentration was measured using dry chemistry methods on a Reflotron reflectance photometer (Boehringer Mannheim UK Ltd). The principle underlying both tests is similar and both procedures are described below.

Materials used :

Principle:

Haemoglobin + $K_3$ [Fe(CN) $_6$] ⟶ methmoglobin

Procedure for Coulter Haemoglobinometer

5. 40 µl of whole blood was mixed with 20 ml of diluent (ISOTON-II) in a cuvette
6. Six drops of reagent was added to each cuvette
7. Samples were mixed and left to incubate for 5 mins at 37°C
8. The haemoglobinometer calculated haemoglobin concentration by measuring the optical density of cyanthaemoglobin at 550 nm

## Total Cholesterol Assay

Total cholesterol concentration was determined from plasma samples by an enzymatic colourimetric method using commercially available kits on a Cobas Bio or Cobas Fara automated analyser. Although the total cholesterol assays for the studies reported in Chapters 5, 6 and 7 were performed in laboratories in Belfast, Loughborough and Coleraine respectively using 2 different assay kits, the principle and the procedures were similar to that described below.

Principle (Cholesterol endpoint kit):

$$\text{cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{cholesterol} + \text{Fatty Acids}$$

$$\text{cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{cholestene-3-one} + H_2O_2$$

$$2\,H_2O_2 + \text{4-aminoantipyrine} + \text{phenol} \xrightarrow{\text{POD}} \text{quinoneimine} + H_2O$$

Kits used :

Cholesterol C System, CHOD-PAP method
(Boehringer Mannheim, U.K. Ltd.)
Each kit contained 2 reagents:
Reagent A: precipitant (phosphotunstic acid)
Reagent B: cholesterol reagent containing 4-aminophenazone, magnesium asparate, cholesterol oxidase, cholesterol esterase and peroxidase
Standards: Preciset Cholesterol
Quality Control: Seronorm Lipid L and Lipid Control (Roche UK)

Cholesterol Endpoint
(Randox, Northern Ireland)
Each kit contained 1 reagent and a standard
Reagent contained 4- aminophenazone, cholesterol oxidase, cholesterol esterase and peroxidase phenol
Standard: supplied
Quality Control: Multisera normal and elevated (Randox, N.I.)

Procedure using Cholesterol Endpoint kit and Cobas Fara analyser:

1. 3 µl of sample was diluted in 30 µl of water
2. 300 µl of Reagent was added
3. Samples were mixed and left to incubate for 10 s at 37°C
4. Absorbance of samples was read at a wavelength of 500 nm
5. Concentrations of glucose were calculated by COBAS Fara using a regression equation established from a reagent blank and the standards.

## Blood Lactate Assay

Lactate analysis was carried out on whole blood using an Analox micro-stat GM7 (Analox Instuments, England)

Principle:
L-lactate: oxygen oxidoreductase (LOD) catalyses the oxidation of L-Lactate to pyruvate.

$$\text{L-lactate} + O2 \xrightarrow[\text{pH 6.5}]{\text{LOD}} \text{Pyruvate} + H_2O_2$$

Reagents and Materials- Analox Instruments Ltd. England

Lactate reagent buffer solution + lactate oxidase

Standards: 2.0, 3.0, 5.0 and 8.0 $mmol \cdot l^{-1}$

Capillary tubes which contain heparin flouride and nitrite

Lactate/ Pyruvate Quality Control Serum

Procedure:

1. After 30 minute warm-up time, lactate reagent at room temperature was cycled through analyser.

2. 7 µl of each standard was pippetted into the analyser cuvette with three readings obtained for each standard

3. Two capillary tubes were half filled with blood for each sample and immediately mixed for 3 minutes by gently tilting.

4. 7 µl of the sample was then delivered into the analyser cuvette using a positive displacement pippete (Gilson Medical Electronics, France) which was rinsed twice in distilled water between samples

5. Where readings for duplicate samples differed by more than 0.01 $mmol \cdot l^{-1}$ a third sample was analysed from one of the capillary tubes.

6. Before and after each test in which lactate was analysed, one aliquot of plasma, from a sample taken at the start of the study was defrosted for analysis before and after each test.

## Total Cholesterol Assay

Total cholesterol concentration was determined from plasma samples by an enzymatic colourimetric method using commercially available kits on a Cobas Bio or Cobas Fara automated analyser. Although the total cholesterol assays for the studies reported in Chapters 5, 6 and 7 were performed in laboratories in Belfast, Loughborough and Coleraine respectively using 2 different assay kits, the principle and the procedures were similar to that described below.

Principle (Cholesterol endpoint kit):

$$\text{cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{cholesterol} + \text{Fatty Acids}$$

$$\text{cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{cholestene-3-one} + H_2O_2$$

$$2\,H_2O_2 + \text{4-aminoantipyrine} + \text{phenol} \xrightarrow{\text{POD}} \text{quinoneimine} + H_2O$$

Kits used :

Cholesterol C System, CHOD-PAP method
(Boehringer Mannheim, U.K. Ltd.)
Each kit contained 2 reagents:
Reagent A: precipitant (phosphotunstic acid)
Reagent B: cholesterol reagent containing 4-aminophenazone, magnesium asparate, cholesterol oxidase, cholesterol esterase and peroxidase
Standards: Preciset Cholesterol
Quality Control: Seronorm Lipid L and Lipid Control (Roche UK)

Cholesterol Endpoint
(Randox, Northern Ireland)
Each kit contained 1 reagent and a standard
Reagent contained 4- aminophenazone, cholesterol oxidase, cholesterol esterase and peroxidase phenol
Standard: supplied
Quality Control: Multisera normal and elevated (Randox, N.I.)

Procedure using Cholesterol Endpoint kit and Cobas Fara analyser:

1. 3 µl of sample was diluted in 30 µl of water
2. 300 µl of Reagent was added
3. Samples were mixed and left to incubate for 10 s at 37°C
4. Absorbance of samples was read at a wavelength of 500 nm
5. Concentrations of glucose were calculated by COBAS Fara using a regression equation established from a reagent blank and the standards.

## HDL Cholesterol Assay

HDL cholesterol concentration was determined from plasma samples by an enzymatic colourimetric method using commercially available kits on a Cobas Bio or Cobas Fara automated analyser. Although the HDL cholesterol assays for the studies reported in Chapter s 5, 6 and 7 were performed in laboratories in Belfast, Loughborough and Coleraine respectively, using 2 different assay kits, the principle and the procedures were similar to that described below.

Principle:
Pre incubation with sulfated cyclodextrin buffer causes the formation of water soluble complexes with LDL, VLDL and chylomicrons which are resistant to polyethylene glycol modified enzymes. Therefore when the second enzyme reagent (containing 4-amino-phenazone, polyethylene cholesterol oxidase, polyethylene esterase and peroxidase) is added the concentration of HDL is determined enzymatically.

$$\text{cholesterol} + O_2 \xrightarrow{\text{Peg Cholesterol oxidase}} \Delta^4\text{–cholestenone} + H_2O_2$$

$$2\,H_2O_2 + \text{4-aminophenazone} + \text{phenol} + \text{EMSE} + H+ + H_2O \xrightarrow{\text{POD}} \text{quinone imino dye} + 5\,H_2O$$

Kits used :

Cholesterol C System, CHOD-PAP method
(Boehringer Mannheim, U.K. Ltd.)
Each kit contained 2 reagents:
Reagent A: precipitant (phosphotunstic acid)
Reagent B: cholesterol reagent containing 4- aminophenazone, magnesium asparate, cholesterol oxidase, cholesterol esterase and peroxidase
Standards: Preciset Cholesterol
Quality Control: Seronorm Lipid L and Lipid Control (Roche UK)

Direct HDL-Cholesterol
(Randox, Northern Ireland)
Each kit contained 2 reagents
Reagent 1 (sulfated cyclodextrin buffer)
Reagent 2 Cholesterol reagent containing 4-aminophenazone, cholesterol oxidase, cholesterol esterase and peroxidase
Standards: Randox Direct HDL calibrator
Quality Control: Direct HDL-C control (Randox, N.I.)

Procedure Direct HDL-Cholesterol (Randox kit)on Cobas Fara:

1. 20 µl of plasma was added to 1.5ml of precipitant (Reagent 1) in a microcentrifuge tube
2. Mix well at 37° C and read initial absorbance
3. Add 500 µl Reagent 2,
4. Mix at 37° C and start timer simultaneously
5. Read absorbance after exactly 5 minutes at a wavelength of 600nm
6. Concentrations of total cholesterol were calculated by COBAS Bio using a regression equation established from a reagent blank and three standards of known concentration

## Triacylglycerol Assay

Plasma triacylglycerol concentration was determined from plasma samples by an enzymatic colourimetric method using commercially available kits and a Cobas Bio or Cobas Fara automated analyser. Although the triacylglycerol assays for the studies reported in Chapters 5, 6 and 7 were performed in laboratories in Belfast, Loughborough and Coleraine respectively, and used 2 different assay kits, the procedures were similar to that described below.

Principle:

Principle: Hydrolysis of triacylglycerol with water and lipase results in glycerol and three fatty acids. Glycerol is then determined by calorimetry.

$$\text{Triacylglycerol} + 3H_2O \xrightarrow{\text{lipase}} \text{glycerol} + \text{Fatty Acids}$$

$$\text{glycerol} + \text{ATP} \xrightarrow{\text{Glycerol kinase}} \text{glycerol 3 phosphate} + \text{ADP}$$

$$O_2\ \text{glycerol 3 phosphate} + O_2 \xrightarrow{\text{Glycerol phosphate oxidase}} \text{dihydroxyacetone phosphate} + H_2O_2$$

$$H_2O_2 + 4\text{-aminophenazone} + \text{4-chlorophenol} \longrightarrow \text{4-(p-benzoquinone mono-imino)-phenazone} + 2\,H_2O_2 + \text{HCL}$$

Kits used :

Perodochrom Triacylglycerides GPO-PAP Method
(Boehringer Mannheim, U.K. Ltd.)
Each kit contained buffer and strips to make up 1 reagent containing: chlorophenol, 4-aminophenazone, lipase GPO, GK and POD
Standards: Precimat Glycerol (Boehringer Mannheim, U.K. Ltd.)
Quality Control: Serum N (Roche UK)

Triglycerides Liquid Reagent GPO-PAP Method
(Randox, Northern Ireland)
Each kit contained 1 reagent and a standard
Reagent contained 4-aminoantipyrine, lipase GPO, GK POD, peroxidase, chlorophenol and magnesium ions, sodium azide
Standard: supplied
Quality Control: Multisera normal and elevated (Randox, N.I.)

Procedure using Boehringer kit and Cobas Bio analyser:

1. 5µl of plasma was pipetted into a cuvette
2. 350µl of reagent mixture was added to each cuvette
3. Samples were mixed and left to incubate for 5 mins at 37 °C
4. Absorbance of samples was read at a wavelength of 500nm
5. Concentrations of triacylglycerol were calculated by COBAS Bio using a regression equation established from a reagent blank and three standards

## Non-esterified fatty acid Assay

Non-esterified fatty acid concentration was determined from plasma samples by an enzymatic colourimetric method using commercially available kits on a Cobas Bio or Cobas Fara automated analyser. Although the non-esterified fatty acid assays for the studies reported in Chapters 5, 6 and 7 were performed in laboratories in Belfast, Loughborough and Coleraine respectively using 2 different kits, the procedures were similar to that described below.

2W

Principle:
With the addition of reagent A non-esterified fatty acids in the sample form thiol esters of CoA called Acyl CoA and by-products AMP and pyrophosphate. The addition of reagent B (Acetyl-CoA oxidase and Peroxidase) causes the Acyl CoA to be oxidised producing hydrogen peroxide which with peroxidase forms a purple adduct whose absorbance can be determined.

$$\text{NEFA} + \text{ATP} \xrightarrow{\text{Acyl CoA synthetase}} \text{Acyl CoA} + \text{AMP} + \text{Ppi}$$

$$\text{Acyl Co A} + O_2 \xrightarrow{\text{Acyl CoA Oxidase}} \text{2,3,-trans-Enoyl-CoA} + H_2O_2$$

$$2\,H_2O_2 + \text{TOOS} + \text{4-AAP} \xrightarrow{\text{Peroxidase}} \text{pupple adduct} + 4\,H_2O$$

Kits used :

NEFA C ACS-ACOD Method
(Wako, Germany)
Each kit contained 2 reagents:
Reagent A: acyl-conenzyme A synthetase, ascorbate oxidase, coenzyme A, ATP and 4-aiminoantipyrine
Reagent B: acety CoA oxidase and peroxidase
Standards: Oleic Acid
Quality Control: Seronorm Lipid (Nycomed, Norway)

NEFA
(Randox, Northern Ireland)
Each kit contained 2 reagents
Reagent 1 acyl-conenzyme A synthetase, ascorbate oxidase, coenzyme A, ATP and 4-aiminoantipyrine
Reagent 2 acety CoA oxidase and peroxidase And TOOS (N-ethyl-N-(2hydroxy-3-sulphopropyl) m-toluidine
Standards: provided with kit (Randox )
Quality Control: Mulit-sera normal and elevated (Randox, N.I)

Procedure using NEFA C ACS-ACOD Method (Wako kit) on Cobas Bio:

1. 10µl of plasma was added to 100 µl of Reagent A
2. Samples were mixed and left to incubate for 10 mins at 37 °C
3. 75 µl of Reagent B was added
4. Samples were mixed and left to incubate for 10 mins at 37 °C
5. Absorbance of samples was read at a wavelength of 550 nm
6. Concentrations of non-esterified fatty acids were calculated by COBAS Bio using a regression equation established from a reagent blank and three standard

## Insulin
## Solid Phase Radioimmunoassay

In the study described in Chapter 6 insulin was determined from serum samples by a solid phase radioimmunoassay using a commercially available kit. All samples from the study were analysed in a single batch.

Kit: Coat-A-Count supplied by Diagnostics Products Corporation

Principle

$^{125}$I-labelled insulin competed with insulin in the sample for sites on insulin-specific antibody immobilised to the wall of a polypropylene tube. After incubation, isolation of the antibody-bound fraction is achieved by decanting supernatent. The tube is then counted in a gamma counter, the counts being inversely related to the amount of insulin present in the sample. The quantity of insulin in the sample is then determined by comparing the counts to a standard curve.

Materials

Insulin Ab-coated tubes
Uncoated tubes
Non-specific binding tubes
$^{125}$I-labelled insulin
Insulin calibrators: in order to set up callibration curve, standards are provided in concentrations of 0.5.15.50.100.200 and 400 $\mu U \cdot ml^{-1}$
Control material

Procedures*

1. Pipette 200 µl of the zero callibrator into four uncoated tubes and four non-specific binding tubes, add 200 µl of the remaining calibrators, samples and control to the antibody tubes. Pipette directly to the bottom.
2. Add 1 ml of $^{125}$I-labelled insulin to every tube and vortex.
3. Incubate for 18-24 hours at room temperature
4. Decant thoroughly and allow tubes to drain for 2-3 minutes. Strike tubes sharply on absorbant paper to shake off all residue droplets.
5. Count for 1 minute in a gamma counter

*These details were provided by Dr Sara Herd who carried out this assay at Loughborough University

## Insulin
## Competitive Radioimmunoassay

In the study described in Chapter 7 insulin was determined from plasma samples by a competitive radioimmunoassay routinely performed by the Wellcome Research Laboratories. All samples from the study were analysed in a single batch.

Principle

$^{125}$I-labelled insulin competes with insulin in the sample for sites on insulin-specific antibody immobilised to the wall of a polypropylene tube. The amounts of antibody and labelled hormone are fixed, the only variable being the unlabelled hormone concentration. On removal of the bound from the free hormone the amount of labelled hormone bound to the antibody can be measured by counting radioactivity on a gamma counter and comparison of the count to a standard curve.

Materials

Antibody code GP25 (raised in guinea pigs to natural porcine insulin)
$^{125}$I-labelled insulin (supplied form Amersham Pharmaceuticals product # IM166)
Dextran-coated charcoal suspension (1g of Norit A Charcoal per 100ml phosphate buffer)

General Procedures*

1. Known quantities of antibody and labelled hormone are introduced into the specified volume of sample .
2. Samples are left to incubate for 24 hours. Cooled (4°C)
3. Dextran coated charcoal is added to the assay tubes with a repeating dispenser.
4. Tubes are then centrifuged at 4°C and the supernatant decanted from the carbon pellet.
5. The activity associated with each pellet is counted on a Nuclear Enterprises NE1600 gamma counter.

* These details were supplied by Miss Bronagh McKibben who carried out the assay at the Wellcome Research Laboratories in the Department of Clinical Science at the Queens University of Belfast.

## Glucose Assay

In the study described in chapter 6, glucose concentration was determined from plasma samples by an enzymatic colourimetric method using a commercially available kit on a Cobas Bio automated analyser.

In the study described in chapter 7, glucose concentration was determined from plasma samples by an enzymatic method using a commercially available kit on a Cobas Fara automated analyser.

Principle- enzymatic colourimetric method:

$$\text{Glucose} + O_2 + H_2O \xrightarrow{\text{Glycerol phosphate oxidase}} \text{gluconate} + H_2O_2$$

$$2\,H_2O_2 + + \text{4-sminophenazone} + \text{phenol} \xrightarrow{\text{Peroxidase}} \text{4 -(p-benzoquinone mono-imino) phenazone} + 4\,H_2O$$

Principle- hexokinase method:

$$\text{Glucose} + \text{ATP} \xrightarrow{\text{Hexokinase}} \text{G-6-P} + \text{ADP}$$

$$\text{G-6-P} + \text{NAD} \xrightarrow{\text{G6P dehydrogenase}} \text{gluconate-6-P} + \text{NADH} + H^+$$

Kits used :

Glucose/GOD-PAP Method
Enzymatic colourimetric
(Boehringer Mannheim, Uk. Ltd))
Each kit contained 2 bottles which are mixed to make up 1 reagent containing:
Glycerol phosphate oxidase, peroxidase, and phenol
Standards: supplied
Quality Control: Precinorm L and Control serum (Roche UK Ltd.)

Hexokinase Mehtod
(Randox, Northern Ireland)
Each kit contained 2 bottles which are mixed to make up 1 reagent containing:
Hexokinase, glucose 6 phosohate dehydrogenase, and sodium azide
Standard: supplied (5.55 $mmol \cdot l^{-1}$)
Quality Control: Assayed Multi-sera normal and elevated (Randox N.I.)

Procedure using Glucose/GOD-PAP Method (Boehringer Mannheim kit):

1. 300 µl of Reagent was added to 4µl of plasma
2. Samples were mixed and left to incubate for 1 min at 37 °C
3. Absorbance of samples was read at a wavelength of 500 nm
4. Concentrations of non-esterified fatty acids were calculated by COBAS Bio using

a regression equation established from a reagent blank and three standards of 2, 4, and 6 $mmol \cdot l^{-1}$

Procedure using Hexokinase Method (Boehringer Mannheim kit):

1. 3 µl of sample was diluted in 30 µl of water
2. 300 µl of Reagent was added
3. Absorbance of samples was read at a wavelength of 340 nm
4. Concentrations of glucose were calculated by COBAS Fara using a regression equation established from a reagent blank and the standard.

## Within and between batch coefficients of variation for assays

| Chapter | Assay | Lab | Maximum within-batch variation CV (%) | Between-batch variation CV (%) |
|---|---|---|---|---|
| 4 | Blood Lactate | UUJ | 2.7 | 3.2 |
| 5 | Plasma triacylglcerol | QUB | 2.3 | 3.4 |
| | Plasma HDL cholesterol | | 2.9 | 3.1 |
| | Plasma Total Cholesterol | | 2.1 | 3.2 |
| 6 | Plasma triacylglycerol | Loughborough | 1.5 | 1.9 |
| | Plasma HDL Cholesterol | | 1.6 | 5.3 |
| | Plasma Total Cholesterol | | 1.8 | 2.9 |
| | Plasma Glucose | | 1.0 | 1.7 |
| | Plasma NEFA | | 2.3 | 3.2 |
| | Serum Insulin | | 2.5 | 4.9 |
| 7 | Plasma triacylglycerol | NICDH at UU Coleraine | 1.2 | 2.3 |
| | Plasma HDL Cholesterol | | 0.9 | 3.9 |
| | Plasma Total Cholesterol | | 1.2 | 1.8 |
| | Plasma Glucose | | 0.7 | 1.8 |
| | Plasma NEFA | | 1.5 | 2.4 |
| | Plasma Insulin | QUB | 4.8 | 7.8 |

# APPENDIX F

# PSYCHOMETRIC INVENTORIES

# (Mood Scale)

Name _____ Date_____

INSTRUCTIONS:
Below is a list of words that describe feelings people have.
Please read each one carefully.
Then tick the answer which best describes HOW YOU FEEL RIGHT NOW.
Make sure you answer every question.

| | | Not at all | A little | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|---|---|
| 1 | Panicky | 0 | 1 | 2 | 3 | 4 |
| 2 | Sad | 0 | 1 | 2 | 3 | 4 |
| 3 | Lively | 0 | 1 | 2 | 3 | 4 |
| 4 | Confused | 0 | 1 | 2 | 3 | 4 |
| 5 | Furious | 0 | 1 | 2 | 3 | 4 |
| 6 | Worn out | 0 | 1 | 2 | 3 | 4 |
| 7 | Depressed | 0 | 1 | 2 | 3 | 4 |
| 8 | Downhearted | 0 | 1 | 2 | 3 | 4 |
| 9 | Annoyed | 0 | 1 | 2 | 3 | 4 |
| 10 | Exhausted | 0 | 1 | 2 | 3 | 4 |
| 11 | Mixed-up | 0 | 1 | 2 | 3 | 4 |
| 12 | Sleepy | 0 | 1 | 2 | 3 | 4 |
| 13 | Bitter | 0 | 1 | 2 | 3 | 4 |
| 14 | Unhappy | 0 | 1 | 2 | 3 | 4 |
| 15 | Anxious | 0 | 1 | 2 | 3 | 4 |
| 16 | Worried | 0 | 1 | 2 | 3 | 4 |
| 17 | Energetic | 0 | 1 | 2 | 3 | 4 |
| 18 | Miserable | 0 | 1 | 2 | 3 | 4 |
| 19 | Muddled | 0 | 1 | 2 | 3 | 4 |
| 20 | Nervous | 0 | 1 | 2 | 3 | 4 |
| 21 | Angry | 0 | 1 | 2 | 3 | 4 |
| 22 | Active | 0 | 1 | 2 | 3 | 4 |
| 23 | Tired | 0 | 1 | 2 | 3 | 4 |
| 24 | Bad tempered | 0 | 1 | 2 | 3 | 4 |
| 25 | Bushed | 0 | 1 | 2 | 3 | 4 |
| 26 | Alert | 0 | 1 | 2 | 3 | 4 |
| 27 | Uncertain | 0 | 1 | 2 | 3 | 4 |

## (Perceived Barriers)

Name _____ Date_____

| Read each statement- circle the number which best indicates your agreement. | 1 Strongly Disagree | 2 | 3 | 4 | 5 Strongly Agree |
|---|---|---|---|---|---|
| A major reason when I do not exercise is that I cannot get motivated | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I am too lazy | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I am too busy | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is because of illness | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is because of a physical disability | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I do not have enough time | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I am too tired | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that is interferes with my work | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that it is too inconvenient | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is bad weather | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is the lack of facilities available to me | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I find exercise boring | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that it makes me too fatigued | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is my family obligations | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is because of some aspect of my health | 1 | 2 | 3 | 4 | 5 |

(Efficacy Measure)

Name_____ Date_____

On the scale below- indicate how confident you feel at performing the following exercise:

| | not confident | | | | | | | | | very confident |
|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly for 2 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly for 3 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly for 30 minutes | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly up hills | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Climb 5 flights of stairs | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Jog 1 mile | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Jog 2 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Cycle 5 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |

# APPENDIX G

# TRAINING DIARIES

| Day | Time of Day | Walk duration | Heart rate 10 sec count | Details- How was the walk done; alone or with friend? to a specific destination or out just to walk? | Feelings Scale: At the end of the walk circle the number which best describes how you are feeling at this moment | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | -5 very bad | -4 | -3 bad | -2 | -1 fairly bad | 0 neutral | 1 fairly good | 2 | 3 good | 4 | 5 very good |
| Sun | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Mon | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Tue | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Wed | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Thurs | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Fri | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Sat | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |

| Day | Time of Day | Walk duration minutes | Heart rate 10 sec count | Details- how was the walk done: alone or with friend? to a specific destination or out just to walk? | Feelings Scale: At the end of the walk circle the number which best describes how you are feeling at this moment | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | -5 very bad | -4 | -3 bad | -2 | -1 fairly bad | 0 neutral | 1 fairly good | 2 | 3 good | 4 | 5 very good |
| Sun | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Mon | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Tue | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Wed | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Thurs | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Fri | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Sat | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |

# APPENDIX F

# PSYCHOMETRIC INVENTORIES

# (Mood Scale)

Name _____ Date _____

INSTRUCTIONS:
Below is a list of words that describe feelings people have.
Please read each one carefully.
Then tick the answer which best describes HOW YOU FEEL RIGHT NOW.
Make sure you answer every question.

| | | Not at all | A little | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|---|---|
| 1 | Panicky | 0 | 1 | 2 | 3 | 4 |
| 2 | Sad | 0 | 1 | 2 | 3 | 4 |
| 3 | Lively | 0 | 1 | 2 | 3 | 4 |
| 4 | Confused | 0 | 1 | 2 | 3 | 4 |
| 5 | Furious | 0 | 1 | 2 | 3 | 4 |
| 6 | Worn out | 0 | 1 | 2 | 3 | 4 |
| 7 | Depressed | 0 | 1 | 2 | 3 | 4 |
| 8 | Downhearted | 0 | 1 | 2 | 3 | 4 |
| 9 | Annoyed | 0 | 1 | 2 | 3 | 4 |
| 10 | Exhausted | 0 | 1 | 2 | 3 | 4 |
| 11 | Mixed-up | 0 | 1 | 2 | 3 | 4 |
| 12 | Sleepy | 0 | 1 | 2 | 3 | 4 |
| 13 | Bitter | 0 | 1 | 2 | 3 | 4 |
| 14 | Unhappy | 0 | 1 | 2 | 3 | 4 |
| 15 | Anxious | 0 | 1 | 2 | 3 | 4 |
| 16 | Worried | 0 | 1 | 2 | 3 | 4 |
| 17 | Energetic | 0 | 1 | 2 | 3 | 4 |
| 18 | Miserable | 0 | 1 | 2 | 3 | 4 |
| 19 | Muddled | 0 | 1 | 2 | 3 | 4 |
| 20 | Nervous | 0 | 1 | 2 | 3 | 4 |
| 21 | Angry | 0 | 1 | 2 | 3 | 4 |
| 22 | Active | 0 | 1 | 2 | 3 | 4 |
| 23 | Tired | 0 | 1 | 2 | 3 | 4 |
| 24 | Bad tempered | 0 | 1 | 2 | 3 | 4 |
| 25 | Bushed | 0 | 1 | 2 | 3 | 4 |
| 26 | Alert | 0 | 1 | 2 | 3 | 4 |
| 27 | Uncertain | 0 | 1 | 2 | 3 | 4 |

## (Perceived Barriers)

Name _____ Date_____

| Read each statement- circle the number which best indicates your agreement. | 1 Strongly Disagree | 2 | 3 | 4 | 5 Strongly Agree |
|---|---|---|---|---|---|
| A major reason when I do not exercise is that I cannot get motivated | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I am too lazy | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I am too busy | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is because of illness | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is because of a physical disability | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I do not have enough time | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I am too tired | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that is interferes with my work | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that it is too inconvenient | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is bad weather | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is the lack of facilities available to me | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that I find exercise boring | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is that it makes me too fatigued | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is my family obligations | 1 | 2 | 3 | 4 | 5 |
| A major reason when I do not exercise is because of some aspect of my health | 1 | 2 | 3 | 4 | 5 |

(Efficacy Measure)

Name_____ Date_____

On the scale below- indicate how confident you feel at performing the following exercise:

| | not confident | | | | | | | | | very confident |
|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly for 2 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly for 3 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly for 30 minutes | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Walk briskly up hills | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Climb 5 flights of stairs | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Jog 1 mile | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Jog 2 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Cycle 5 miles | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |

# APPENDIX G

# TRAINING DIARIES

| Day | Time of Day | Walk duration | Heart rate 10 sec count | Details- how was the walk done: alone or with friend? to a specific destination or out just to walk? | Feelings Scale: At the end of the walk circle the number which best describes how you are feeling at this moment | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | -5very bad | -4 | -3 bad | -2 | -1 fairly bad | 0 neutral | 1 fairly good | 2 | 3 good | 4 | 5 very good |
| Sun | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Mon | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Tue | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Wed | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Thurs | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Fri | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| Sat | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |
| | | | | | -5 | -4 | -3 | -2 | -1 | 0 | 1 | 2 | 3 | 4 | 5 |