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LOW INTENSITY EXERCISE, FUNCTIONAL CAPACITY AND LIPOPROTEIN METABOLISM IN WOMEN.

.....

by

Annette Hudson.

A Doctoral Thesis

Submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy of the Loughborough University of Technology.

March 1991.

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ABSTRACT

This thesis examines the potential of a socially-acceptable form of low intensity exercise, brisk walking, to improve functional capacity and modify lipoprotein metabolism in women.

The results of the first study show that the thresholds prescribed by ACSM (1986) for the increase of functional capacity, indicated by maximal oxygen uptake ($\dot{V}O_2$max), might be attained during brisk walking. These thresholds for heart rate and oxygen uptake were exceeded by 94% and 75% respectively of the group of middle-aged sedentary women (mean age 44.2 years, range 30 to 62 years).

A cross-sectional study showed higher total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) concentrations for young females than males but a comparison of sprint, endurance and untrained individuals revealed no differences between groups in this regard. A comparison of middle-aged women however revealed higher ($P<0.05$) HDL-C concentrations for runners (1.33±0.10 $mmol.l^{-1}$) and walkers (1.27±0.07 $mmol.l^{-1}$) than for less active individuals (1.10±0.03 $mmol.l^{-1}$).

The results of a one year training study of brisk walking showed a 16 percent increase in $\dot{V}O_2$max and 11 percent increase in the volume of oxygen consumed at a reference blood lactate concentration of 2 $mmol.l^{-1}$. Changes were also seen for the plasma concentration of TC (6.5% decrease) and HDL-C (27% increase), which occurred in the absence of changes of diet or body composition. Trained subjects also reported an increased sense of well-being.

The effect of walking was confirmed with a detraining study. Three months of training was associated with increases in $\dot{V}O_2$max (7.7%), concentration of HDL_2-C subfraction (19%) and sense of well-being. With three months detraining the changes reverted towards baseline

values whilst not reaching them. Throughout, the controls remained largely unchanged.

The results of these studies suggest that low intensity training, in the form of brisk walking, can favourably alter some aspects of lipoprotein metabolism and indices of functional capacity in middle-aged women.

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Finally, but by no means least, I would like to thank the subjects. Without whose tremendous efforts, understanding and generosity, none of this work would have been possible.

PUBLICATIONS

Unless otherwise indicated by either references to published literature or acknowledgements, the work contained in this thesis is that of the author. Some of the findings which are included in the thesis have reported in the following publications:-

HUDSON, A. & HARDMAN, A.E. (1989). Brisk-walking as a means of improving aerobic fitness in middle-aged and older women. Journal of Sports Sciences. 7: 66.

HUDSON, A., HARDMAN, A.E., GRIMSHAW, K. & WARD, R. (1988). Plasma cholesterol and lipoprotein levels in untrained, sprint-trained and endurance-trained young adults. Journal of Sports Sciences. 6: 166.

HUDSON, A., HARDMAN, A.E., JONES, P.R.M. & NORGAN, N.G. (1988). Influence of a 3-month programme of brisk walking on walking performance, plasma cholesterol and body fatness in middle-aged women. Proceedings of the Nutrition Society. 47: 174A.

HARDMAN, A.E., HUDSON, A., JONES, P.R.M. & NORGAN, N.G. (1989). Brisk-walking influences the physiological responses to a submaximal step test in women. Journal of Physiology. 49: 22P.

HUDSON, A., HARDMAN, A.E., JONES, P.R.M. & NORGAN, N.G. (1988). Physical fitness and body composition in women enrolling on a one year programme of brisk-walking. Annals of Human Biology. Journal of the Society for the Study of Human Biology. 15: 6: 463.

HARDMAN, A.E., HUDSON, A., JONES, P.R.M. & NORGAN, N.G. (1989). Brisk walking and plasma high density lipoprotein cholesterol concentration in previously sedentary women. British Medical Journal. 299: 1204-5.

HUDSON, A., JONES, J.G. & HARDMAN, A.E. (1989). Brisk-walking and psychological well-being in middle-aged and older women. Journal of Sports Sciences. 7: 77-78.

HARDMAN, A.E. & HUDSON, A. (1989). Walking for health-a closer look at exercise. Health Trends. Department of Health. 21: 3: 91-92.

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1. INTRODUCTION.

Up to 70 percent of women aged between the late teens to early forties fail to take regular physical activity (General Household Survey 1983). Furthermore, for older women the proportion of sedentary individuals is even larger. This low rate of exercise involvement is a particular cause for concern since inactivity has been shown to have important consequences for health and quality of life.

Regular physical activity is associated with a number of health benefits. Of particular importance is the maintenance or improvement of functional capacity. Research has shown that maximal oxygen uptake ($\dot{V}O_2max$), the most frequently used index of functional capacity, declines as a natural consequence of the ageing process (Robinson et al. 1973), thus making routine tasks increasingly demanding. However, it has also become apparent that with an appropriate form of physical training this process of decline can be slowed and possibly reversed (Bruce 1984; Robinson et al. 1973; Seals et al. 1984; Dehn & Bruce 1972).

In addition to the maintenance of functional capacity, habitual exercise is also associated with a reduced risk of a number of diseases. Thus, the incidence of obesity, osteoporosis, hyperinsulinaemia and coronary heart disease (CHD) has been reported to be less for active, as compared with less active men (Schultz 1980).

Between 1968 and 1980, the number of female deaths from CHD declined significantly in countries such as Switzerland (66.5%), United States (53.1%) and Australia (37.6%) (Marmot 1984). In Britain however, the decrease achieved was just 10 percent (Coronary Prevention Group 1986; Heller et al. 1983). Since 24 percent of all female

premature deaths (i.e. less than 65 years) in England and Wales are attributed to CHD, making this the second most important cause of death to cancer (OPCS 1986), there would appear to be a need for a more complete investigation of the potential risk factors important for women.

Results derived from The Framingham Study, have led to the suggestion that the major risks for the development of CHD in men and women are similar (Kannel & McGee 1979). Of particular importance are factors such as hypercholesterolaemia, obesity, hypertension, a high fat diet, hyperinsulinaemia, cigarette smoking and physical inactivity (Kannel & McGee 1979). Of additional importance for women is the use of oral contraceptives. The reduction of oestrogen production provoked by this use is associated with unfavourable changes in lipid and lipoprotein metabolism, in particular a reduced concentration of high density lipoprotein cholesterol (HDL-C) (Fotherby 1989). Evidence for this is derived from the observation of an increase in female mortality concomitant with the menopause, the onset of which is associated with a natural decline of oestrogen production (Matthews et al. 1989) (Table 1:1).

In order to offset the effects of the menopause, it is suggested (Harting et al. 1984) that other modifiable risks might be reduced, for example, the level of physical activity. This proposal is based on the results of several studies which have identified a relationship between CHD development and habitual activity. For example Paffenbarger and coworkers (1970) showed that dock workers who expended in excess of 8,000 kcal/week in occupational activity, were at considerably lower risk than their less active contemporaries. Similar trends were also found by Morris (1975), for bus conductors or

Table 1:1. The rate of female mortality from coronary heart disease for different age groups in England and Wales (OPCS 1986).

AGE (yr)	35-44	45-54	55-64
Deaths/100,000 (Women)	6	41	201

postal deliverers compared with less active bus drivers or postal sorters. Together, these observations suggest a potential role for high intensity activity. However, with increased mechanisation in industry and therefore reduced heavy manual labour, there is an increased dependency on leisure-time activities for physical exercise. In the light of this trend, Morris and coworkers (1973) extended their observations to include relatively sedentary occupations (civil servants) and involvement in leisure-time activities. The results led to the recognition that involvement in vigorous activities (i.e. provoking peaks of energy expenditure of 7.5 $kcal.min^{-1}$ or 65% of maximal functional capacity) was associated with a considerably lower incidence of CHD. This observation was also consistent with that reported by Paffenbarger and coworkers (1978), who suggested that an energy expenditure of just 2000 kcal/week was sufficient to provide a protective effect against CHD.

The aforementioned investigations led to the realisation that for relatively inactive men, a considerable reduction in the risk for CHD development could be achieved with exercise participation incurring only a small energy cost. Subsequently, investigations have also identified a number of advantages for low intensity activity programmes. For example, when compared with more intense activities, there is a lower incidence of musculoskeletal injury (Pollock et al. 1977; Schultz 1980). Furthermore, the rate of attrition from low intensity programmes has been shown to be relatively lower than that from high intensity exercise (Wilmore et al. 1970; Massie & Shephard 1971).

However, despite its potential advantages fewer studies of low intensity exercise have been completed. From those investigations which have involved women, $\dot{V}O_2$ max has consistently been reported to improve as a consequence of

brisk walking (Adams & de Vries 1973; Jetté et al. 1988; Santiago et al. 1987; Flint et al. 1974; Johannessen et al. 1986). The extent of this increase, as much as 21% from pre-training levels (Santiago et al. 1987), is of a similar magnitude to that reported for high intensity exercise training (Saltin et al. 1968). The similarity between these results therefore suggests that in the appropriate population, brisk walking might be as effective as higher intensity exercise in increasing functional capacity.

In addition to the aforementioned reports of increased functional capacity, some evidence is also available to support the suggestion that for middle-aged women lipid and lipoprotein metabolism can be altered favourably with a programme of brisk walking. For example Santiago and coworkers (1987) found no change in plasma lipid and lipoprotein concentrations after 20 weeks of training with a group of 30 year old, non obese women. However, Lewis and coworkers (1976) were able to discern an increase in the high:low density lipoprotein cholesterol ratio with a group of obese, 44 year old women, after a 17 week programme. This latter observation is consistent with the results derived from the larger number of walking studies completed with middle-aged men, some of which have also shown favourable changes in lipoprotein metabolism (Williams et al. 1982; Streja & Mymin 1979; Erkelens et al. 1979; Altekruse & Wilmore 1973).

The investigation completed by Lewis and coworkers (1976) emphasizes the effect of additional influences on lipid and lipoprotein metabolism, such as changes in body mass and/or body fatness and diet. A number of studies have reported an inverse relationship between obesity and HDL-C concentrations (Carlson & Ericsson 1975; Glueck et al. 1980; Taylor et al. 1981). Furthermore, both Wilson and Lees (1972) and Contaldo and coworkers (1980), have

reported an inverse relationship between weight loss and change in HDL-C concentration. Dietary manipulation has also been reported to affect lipid and lipoprotein metabolism, particularly of triacylglycerol (Tg) rich lipoproteins and HDL-C (Katan 1984; Gurr et al. 1989). On the basis of this evidence, it is therefore considered necessary to monitor these potential influences when investigating the effects of other possible factors, such as exercise.

The purpose of the studies presented in this thesis was therefore to examine the influence of a socially-acceptable form of low intensity exercise, brisk walking, on functional capacity and lipoprotein metabolism in middle-aged women.

2. REVIEW OF LITERATURE.

2.1. Lipoprotein metabolism.

In a recently published report it was suggested that the typical U.K. diet provided over 40% of the daily energy intake as fat (MAFF 1987). Furthermore, it has been estimated that such an intake would provide a daily absorption of approximately 100 gm of triacylglycerols (Tgs) and 250 mg of cholesterol (Brown & Goldstein 1984).

In order for absorption to occur, the ingested fats undergo partial hydrolysis in the small intestine. This process is facilitated by the action of lipases, which in the presence of bile acids, cholic and chenodeoxycholic acid and some phospholipids, result in the formation of micelles. Monoacylglycerols and fatty acids so formed are absorbed in the duodenum and proximal jejunum, and are re-esterified in the endothelial cells to form Tgs. The transport of the resultant lipid molecules out of the intestine is facilitated by the formation of a number of molecular aggregates of lipid and protein (lipoproteins). These lipoproteins have been classified according to density (Havel et al. 1955; Nelson 1972), which itself is dictated by the composition of the particles (Table 2:1. Figure 2:1). The protein moiety (apoplipoprotein) has several roles; it confers structural stability to the particle, increases the solubility of the otherwise hydrophobic lipids and acts as a marker by which the lipoproteins can be recognized and their metabolism subsequently modified (Goldstein & Brown 1977; Albers & Segrest 1986; Havel 1970, 1986) (Table 2:2).

The lipid transport system in which the lipoproteins and apolipoproteins are involved, can be considered as two separate but integrated pathways (Figure 2:2). Firstly, the exogenous route which carries dietary cholesterol and

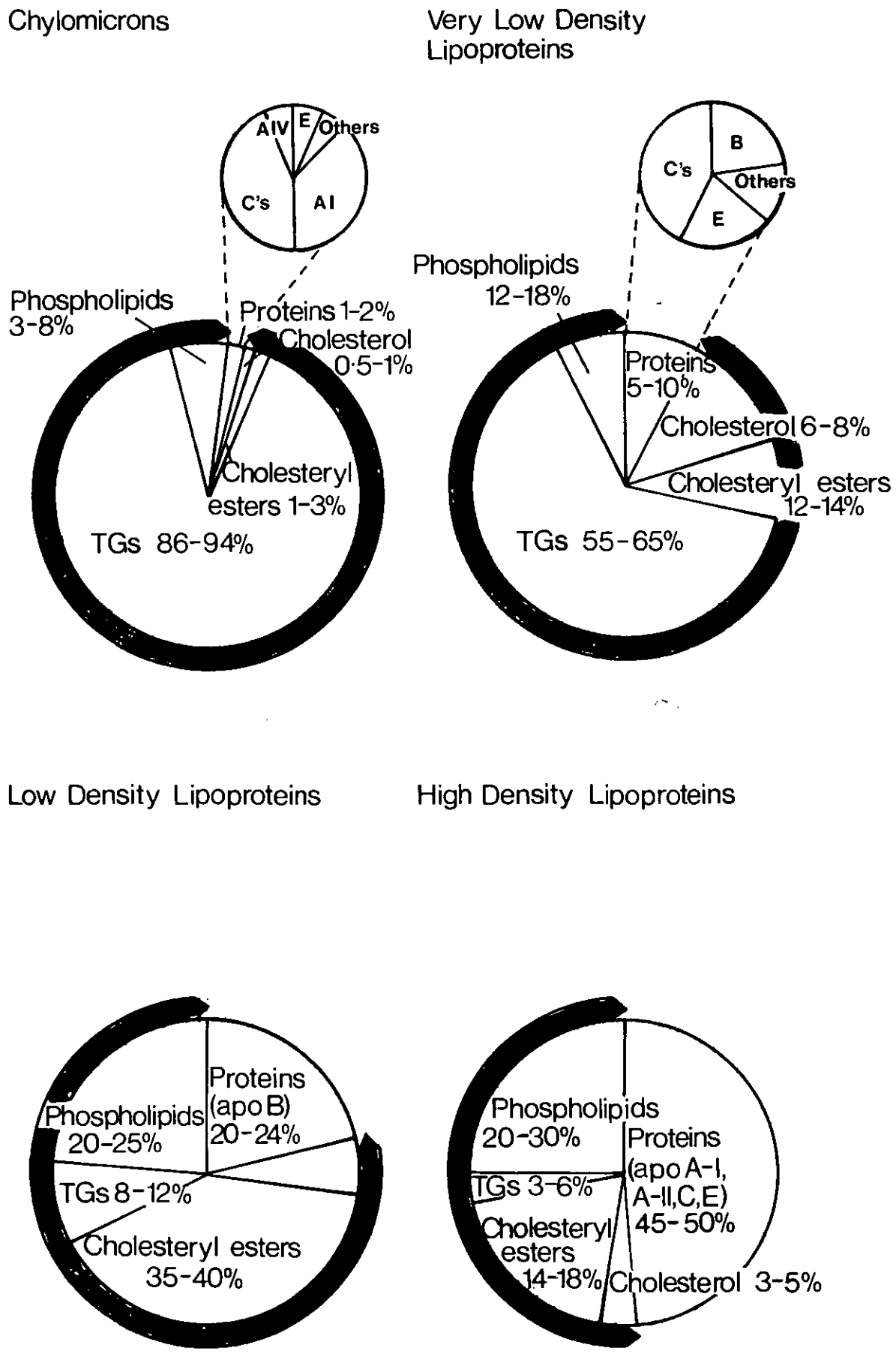


FIGURE 2·1 Composition of serum lipoproteins as derived by ultracentrifugation (Havel et al. 1955).

Table 2:1. The major lipoprotein groups.

Chylomicrons:

Density: <0.95 $g.ml^{-1}$

Source: intestine

Function: transport exogenous Tg

Composition: 98-99.5% lipid, 0.5-2% protein

Apolipoproteins: A-I (33%), A-IV (14%), C (32%)

Very low density lipoprotein (VLDL):

Density: 0.95-1.005 $g.ml^{-1}$

Source: liver

Function: transport endogenous Tg

Composition: 85-90% lipid, 10-15% protein

Apolipoproteins: B (25%), C (55%), E (15%)

Low density lipoprotein (LDL):

Density: 1.019-1.063 $g.ml^{-1}$.

Source: VLDL, apo B from jejunum.

Function: transport cholesterol in the blood.

Composition: 75% lipid, 25% protein.

Apolipoproteins: B (95%).

High density lipoprotein (HDL):

Density: 1.063-1.21 $g.ml^{-1}$.

Source: intestine & liver, mature in the plasma.

Function: reverse transportation of cholesterol from peripheral tissues to the liver.

Composition: 50% lipid, 50% protein.

Apolipoproteins: A-I (65%), A-II (10%), C (13%).

(Havel et al. 1955).

Table 2:2. The major apolipoproteins and their functions.

1. Transcellular lipid transport.	
-B:	Triacylglycerol transport from mucosal cells of small intestine (chylomicrons) and from hepatocytes (VLDL).
-B, E:	Receptor-dependent catabolism of lipoproteins and their remnants.
-A-I:	Cholesterol transport from cell membranes
-other:	?
2. Structure of lipoproteins.	
-interact with lipids and change conformation of lipids and proteins	
-stabilize aqueous micellar suspensions of polar and nonpolar lipids	
3. Cofactors of lipolytic enzymes.	
-A-I,	Lecithin:cholesterol acyltransferase
-C-I:	
-C-II:	Lipoprotein lipase

(Morrisett et al. 1975).

Tgs absorbed from the intestine, and secondly, the endogenous route which carries cholesterol and Tgs entering the bloodstream from the liver and other non intestinal tissues. Together, the two routes provide a means by which cholesterol can be continuously moved between intestinal, hepatic and extra-hepatic tissues.

A more detailed examination of the pathways now follows (Figure 2:2). Firstly, the exogenous route: in the mucosal cells of the duodenum and jejunum, dietary fats are formed into the largest of the lipoprotein particles: chylomicrons. Because of their size, these molecules are precluded from traversing the endothelial barrier. The chylomicrons therefore remain in the lymph and enter the circulation via the thoracic duct. After entering the general circulation, the chylomicrons have a half-life of only a few minutes, being degraded to core remnants (rich in cholesteryl esters and apolipoprotein E) and surface remnants (rich in phospholipids, cholesterol and apolipoprotein C). The enzyme responsible for this process, lipoprotein lipase, is located on the endothelium of capillaries situated in the adipose, mammary and muscle tissue.

The fatty acids which result from this rapid breakdown of the Tgs are either oxidised for energy release, or are stored, depending on whether formation occurs in the muscle or adipose tissue (Goldstein & Brown 1977). However, the remnant particles are subsequently taken up by receptors located on liver cells. The cholesteryl esters are then used in the formation of cell membranes, in the biosynthesis of new lipoproteins and in the production of bile salts. The excess surface material which results, predominantly composed of phospholipids and free cholesterol, is used in the formation of high density lipoprotein (Figure 2:3).

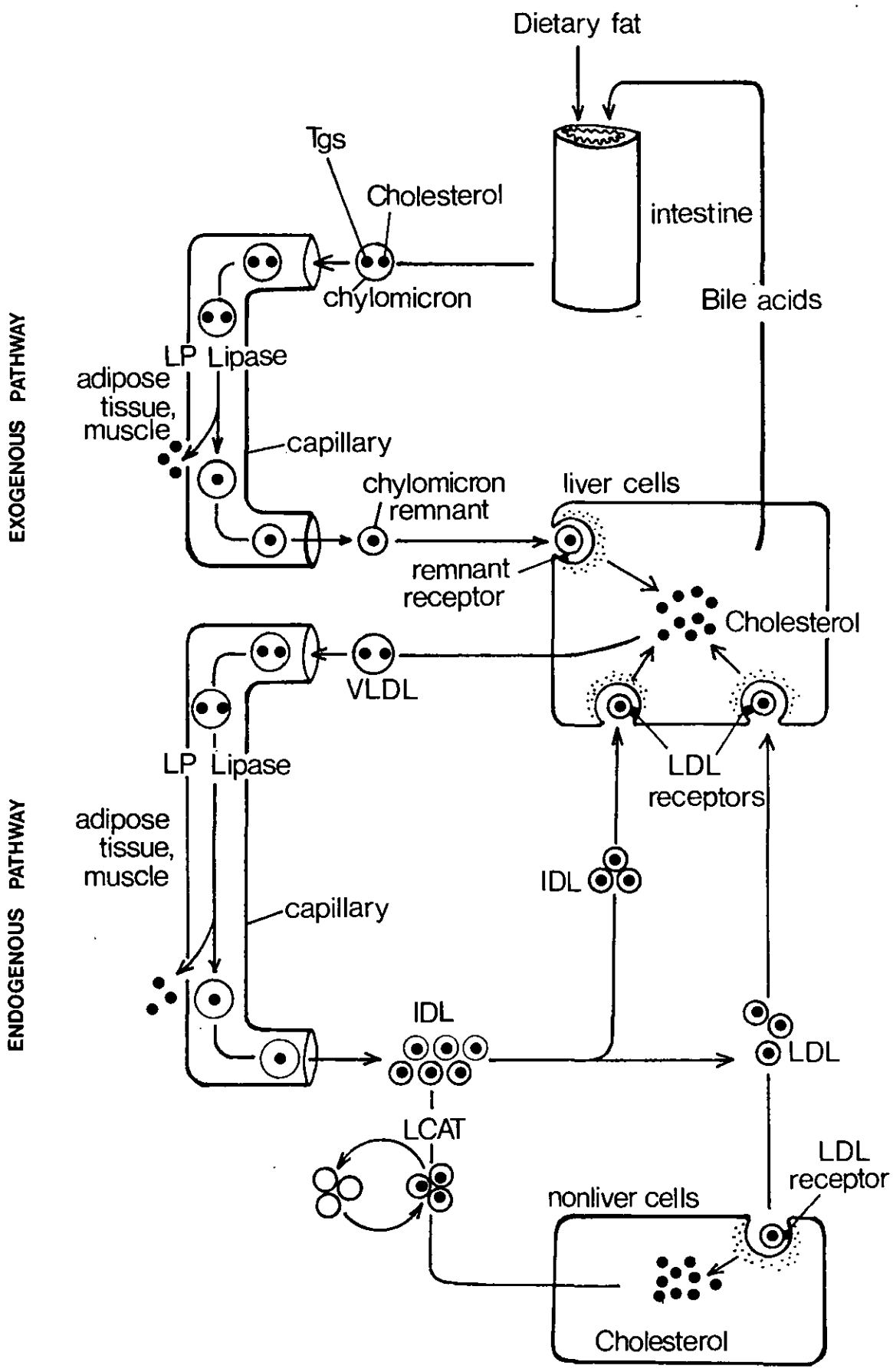


FIGURE 2:2 Exogenous and endogenous fat-transport pathways (Brown & Goldstein 1984).

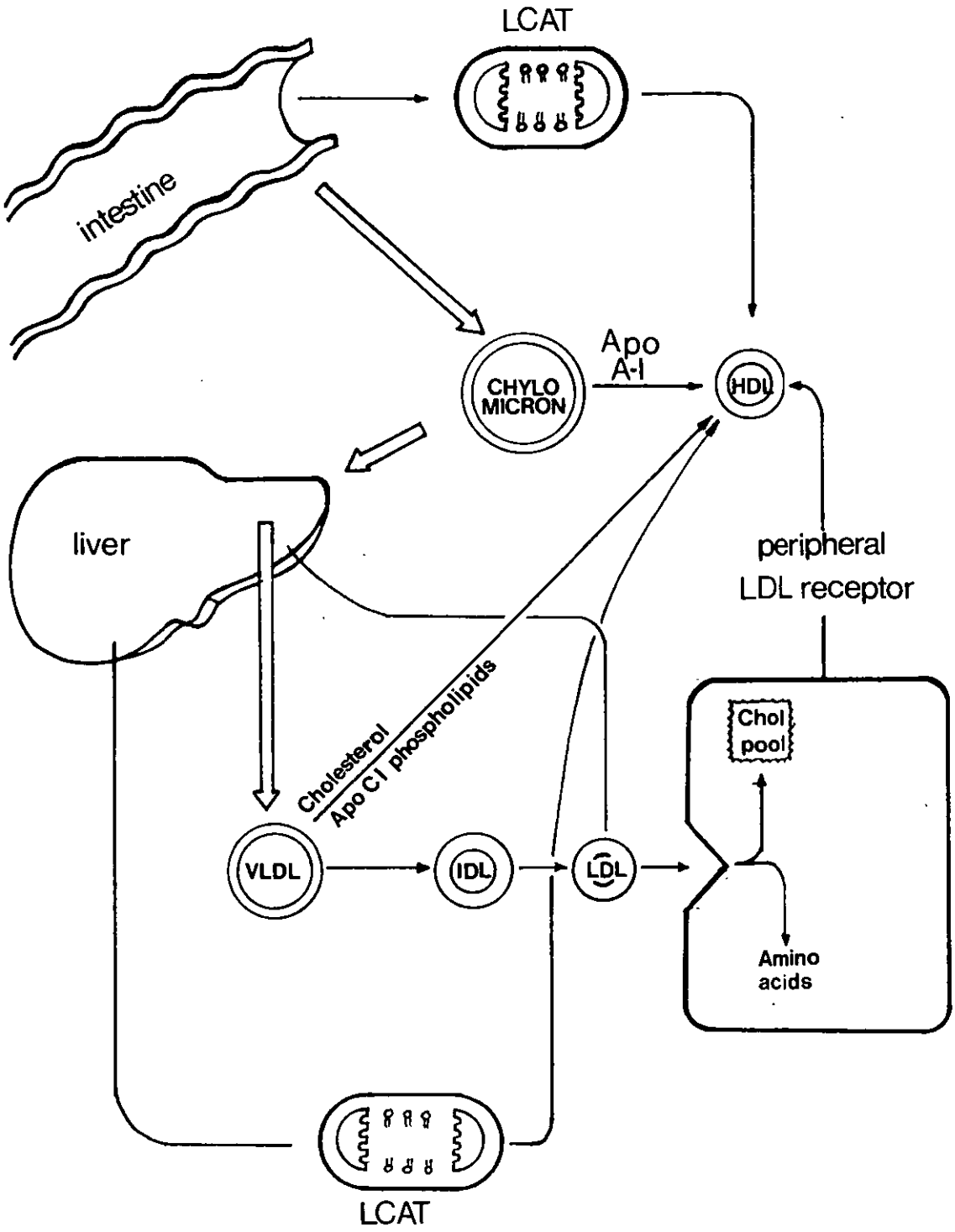


FIGURE 2:3 Metabolism of HDL (Assmann 1982).

The endogenous route: when carbohydrate is the predominant source of dietary energy the metabolism of lipids and lipoproteins from the exogenous route is reduced. It is this relative decrease which evokes an increase in the activity of the controlling enzyme, 3-hydroxy-3-methylglutaryl coenzyme A reductase (E.C. 1.1.1.34; HMG Co A reductase) which acts to catalyse the formation of Tgs from carbohydrate and fatty acids. The resulting Tgs are subsequently packaged with cholesteryl esters and apolipoproteins B-100 and E, to form very low density lipoproteins (VLDL). The VLDLs are subsequently secreted by the liver and transported to adipose and muscle tissue, where the Tg is extracted. The resultant intermediate density lipoprotein (IDL) is rich in cholesteryl esters which are removed in the conversion of IDL to low density lipoprotein (LDL). It is this latter particle which acts as the main carrier of cholesterol in the plasma.

On reaching the tissues the cholesterol associated with LDL is accepted in one of two ways. Either by a process of passive endocytosis, or more effectively, by a specific receptor-mediated uptake mechanism first described by Goldstein and Brown (1977). The latter route involves the recognition of apolipoprotein B-100, the predominate surface protein of LDL, by receptors located in 'coated pits' on the plasma membrane. Once accepted into the pit, the membrane undergoes invagination and the cholesterol is drawn into the centre of the cell (Figure 2:4) (Goldstein & Brown 1977, 1984; Havel 1986). As a consequence of this uptake, the rate of cholesterol synthesis in extrahepatic cells is reduced, and the system therefore is seen to act as a self-regulating type of feed-back loop.

As was observed for the exogenous route, any excess surface material is used in the formation of HDL. Reichl

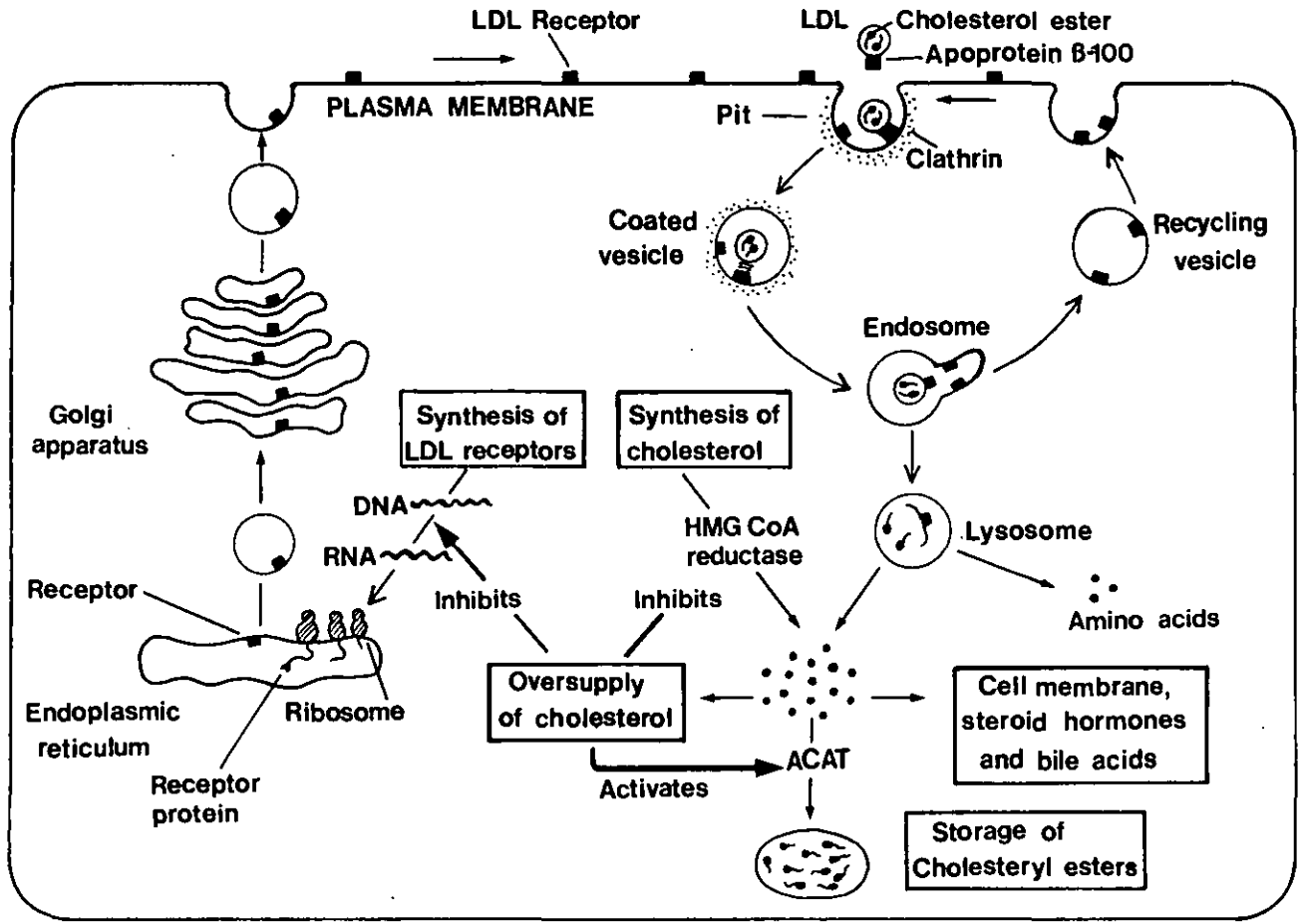


FIGURE 2:4 LDL metabolism (Brown & Goldstein 1984).

and Miller (1986) have revealed HDL to be central to the process of reverse cholesterol transport, in which HDL 'scavenges' unesterified cholesterol from sites of accumulation, for example, in cell membranes. In this way cholesterol is then transported back to the liver in HDL, where lecithin cholesterol acyltransferase (LCAT) acts as a catalyst in the transfer of fatty acids from phosphatidylcholine in HDL to cholesterol and then to cholesteryl esters. The cholesteryl esters so formed are then transferred to VLDL and IDL and are eventually converted to LDL to complete the reverse transport cycle (Glomset & Norum 1973; Roheim 1986).

Although the endogenous and exogenous routes have been considered as functionally distinct, the pathways do utilize three common mechanisms. As a consequence, the dietary fat involved in the exogenous route can influence the endogenous metabolism of lipoproteins. The first point of crossover occurs with the enzyme lipoprotein lipase, which is responsible for the hydrolysis of Tgs carried both exogenously in chylomicrons and endogenously in VLDL. The second point occurs when phospholipids and cholesterol are accepted by HDL. The molecules involved in this process are supplied from both exogenous chylomicron remnants and also from endogenous IDL. Finally, overlap occurs at hepatic and extra-hepatic lipoprotein receptors. The LDL receptor on the extra-hepatic parenchymal cells take up LDL, while lipoprotein receptors in the liver accept chylomicron remnants. However, it is possible that both processes are performed by a single type of receptor. It is therefore possible that, whilst the pathways may be considered as distinct entities, they are also inseparably integrated.

From this brief overview it can be seen that with the continuous cycling of cholesterol into and out of the bloodstream, the concentration observed at any one time

is not a simple additive function of dietary cholesterol intake and endogenous synthesis. Rather, the concentration in the blood reflects the rate of synthesis of the cholesterol-carrying lipoproteins and the efficiency of the receptor mechanisms which determine their catabolism. The balance between these two processes and the concentration of cholesterol which ultimately results, have a number of subsequent repercussions for disease development, particularly for the risk of CHD.

2.1.1. Lipid risk factors for coronary heart disease.

Two major groups of factors associated with an increased risk for CHD development have been identified (Assmann 1982). Firstly, those such as exercise and diet which can be influenced by changes in lifestyle and secondly, those which cannot be altered, such as age and sex (Table 2:3). It has therefore been proposed that CHD development has a multifactorial basis, occurring primarily as an acquired disease determined by genetically-defined factors, upon which are superimposed the effects of the more modifiable factors (Pooling Project Research Group 1978; Blackburn 1980; Hopkins & Williams 1981; European Atherosclerosis Society 1987; Assmann 1982). This proposal is consistent with evidence showing that both genetic (e.g. dyslipoproteinaemias) and environmental factors (e.g. excessive amounts of dietary fat) are causally related to pathological changes in lipoprotein metabolism (Carlson & Bottiger 1972; Miller et al. 1977).

These changes are manifest by increased concentrations of Tg-rich lipoproteins, particularly LDL, and a concomitant decrease in the concentration of HDL. As a consequence of the latter, therefore the ability to 'scavenge' cholesterol from peripheral tissues is potentially reduced. Thus leading to its accumulation and ultimately to the development of atheroma. It has therefore been

Table 2:3. The major factors associated with an increased risk of CHD (Schettler et al. 1978).

Non-influenceable	Influenceable
Age	Serum cholesterol
Sex (male)	Blood pressure
Family history	Body weight
	Physical Inactivity
	Nicotine abuse
	Diabetes mellitus

shown (Goldstein & Brown 1977; Zilversmit 1979) that chylomicron remnants, IDL and LDL, are all associated with the development of fulminant atherosclerosis when their levels are elevated in man. High density lipoprotein however, has been associated with an actual decrease in symptomatic atherosclerosis (Brunner et al. 1987; Miller et al. 1977). However, whilst continuing to support these observations Gordon and Rifkind (1989) offer a note of caution against the designation of lipoproteins as being either 'good' or 'bad':

> "... the popular designation of HDL as "good cholesterol" is misleading, because the antiatherogenic role that has been designated for it pertains not to any unique property of its cholesterol but to the direction in which it transports that cholesterol."
>
> (Gordon and Rifkind 1989).

Thus it is not the actual lipoproteins which are important in the risk of disease development but the direction in which the cholesterol they are transporting is moved.

2.1.2. Cholesterol metabolism and development of atherosclerosis.

In the previous section the importance of cholesterol in the development of atheroma was identified. Cholesterol is a naturally occurring sterol. As such, it is characterized by a basic four ring carbon structure, to which is attached an hydroxyl group and an hydrocarbon side-chain. This configuration provides the molecule with a degree of rigidity. One of the main functions of cholesterol therefore is to modulate the fluidity (flexibility) of cell membranes. A second role is in the formation of bile salts. These substances are essential for the emulsification and solubilisation of dietary

fats, processes which facilitate the digestion and absorption of lipid molecules from the small intestine. Finally, cholesterol acts as a precursor for the formation of steroid hormones including progesterone, testosterone, oestradiol and cortisol.

As a consequence of its many and varied roles, cholesterol is considered an essential metabolite. However, as has been shown, the molecule can also be synthesized in sufficient quantities from endogenous processes. Thus cholesterol is not considered an essential nutrient.

2.1.3. Sex differences in lipoprotein metabolism.

The results of epidemiological studies have shown that for adults below 50 years of age, concentrations of Tg-rich lipoproteins (LDL & VLDL) are higher in males. The opposite is true however, for the concentration of HDL which is lower in males than in females (Carlson & Ericsson 1975; Deshaies & Allard 1982; Brownell et al. 1982).

These sex-linked differences in lipoprotein concentrations have been explained as a consequence of differences in the concentration of sex-hormones. The basis of this proposal is the observation that the onset of the menopause is associated with a natural decline in oestrogen levels. In addition, after the menopause, HDL-C concentration is reduced and the rate of CHD mortality is increased (Table 1:1) (Matthews et al. 1989; Fotherby 1989: Bush & Barrett-Connor 1985). Furthermore, the use of oral contraceptives, which results in similar changes in the oestrogen cycle, is also associated with lowered HDL-C and increased risk of CHD development (Fotherby 1989; Srinivasan et al. 1985; Wahl et al. 1983; Bradley et al. 1978; Hill et al. 1980).

The proposal of a role for sex hormones is also consistent with the results derived from investigations of the relationships between blood lipid and lipoprotein metabolism, age and sex-hormones. The studies showed that the risk for the development of CHD was greater for post-menopausal or oophorectomized women than for pre-menopausal women of a similar age (Oliver & Boyd 1959; Gordon et al. 1978; Kannel et al. 1976). Furthermore, it was reported by Matthews and coworkers (1989) that natural menopause and speculatively, oophorectomy were associated with a decline in the levels of circulating oestrogen. It was therefore suggested that such a decline might adversely affect lipid metabolism. That is, would provoke an increase in the concentration of LDL and decrease in HDL. It was also identified that the changes consequential to modification of the oestrogen cycle exceeded those changes resulting from the ageing process alone.

Further support for these suggestions has been derived from the work completed by Brown and Daudiss (1973). The results of the study showed plasma HDL-C concentrations of females aged between 18 and 55 years to be significantly higher than those of similarly aged males. In addition, the comparison of older groups (55 to 64 year old) revealed no difference in the concentration of HDL-C between males and females. That is, after the menopause, factors affecting lipid and lipoprotein metabolism in women more closely resembled those of their male counterparts.

The results of these studies are also consistent with those derived from investigations of hormone replacement therapy. It was shown that administration of this therapy to post-menopausal women resulted in more favourable lipid and lipoprotein concentrations. There was also a lower incidence of CHD than in age-matched controls

(Brown & Daudiss 1973; Carlson & Ericsson 1975; Kannel 1987). This is supported by evidence derived from epidemiological studies, many of which have also found an increased severity of CHD in post-menopausal women (Wuest et al. 1953; Parrish et al. 1967; Rosenberg et al. 1981; Gordon et al. 1978).

However, despite the apparent abundance of supportive evidence Harlan (1987) attempts to underplay the role of sex-hormones in the development of CHD. In a review, he suggests that age, rather than sex hormones *per se*, is the most important risk factor amongst women. This view therefore contradicts much of the evidence already presented from the observations of the menopause, oral contraceptive use and hormone replacement therapy.

Thus, despite the doubts expressed by Harlan (1987), the majority of studies appear supportive of the relationship between sex-hormones, blood lipid and lipoprotein metabolism and the incidence of CHD.

2.1.4. Influence of diet on lipoprotein metabolism.

In a previous section it was explained that the processes involved in lipoprotein metabolism were determined by a form of feedback mechanism dependent upon the relationship between two intricately linked transport pathways. The exogenous route is responsible for the distribution of dietary fats and it is therefore likely that the composition of diet is a potentially important determinant of the metabolic processes.

The classic works of Keys and coworkers (1965) and of Hegsted and coworkers (1965) demonstrated that in man, the concentration of total and LDL-C can be altered in predictable ways by dietary manipulation. Since these early observations, more detail of the relationship

between diet and its effects on lipid and lipoprotein metabolism has emerged.

The results of investigations into the effects of dietary fat have shown dietary cholesterol to be a particularly important influence on serum cholesterol concentration in animals (Moore & Williams 1966; Beynen et al. 1987). However, similar studies conducted with man have provided somewhat less consistent results. For example, the results of their early study led Keys and coworkers to reject initially any role for dietary cholesterol in the regulation of plasma cholesterol concentration (Keys et al. 1957). Subsequently, investigations have been completed in which eggs have been used in order to manipulate dietary cholesterol intake. The results of these studies have provided evidence both for and against a regulatory role. Gurr and coworkers (1989), however, have criticized this approach recognising that, whilst egg yolk is rich in cholesterol, it also contains an abundant supply of phospholipids. It is suggested therefore (Gurr et al. 1989) that the latter might also have an influence on the changes in lipid and lipoprotein metabolism observed. The same authors also re-emphasize the existence of inter-individual differences in response to dietary cholesterol which would also be expected to be influential on plasma cholesterol concentration.

Furthermore, Appelbaum-Bowden and coworkers (1984) have shown that the effects of dietary cholesterol appeared to vary between the different plasma lipoprotein classes. For example, it was reported by Sacks and coworkers (1984) that plasma LDL-C concentration was raised, whilst TC concentration was unaffected by increasing dietary cholesterol. Beynen and Katan (1985) however reported that increased dietary cholesterol resulted in both an increase in the concentration of LDL-C and also of HDL-C. Therefore, as yet the effect of dietary cholesterol on

the metabolism of lipids and lipoproteins remains unclear.

In contrast, less ambiguity exists with regard to the results of investigations in to the effects of saturated (SFA), monounsaturated (MFA) and polyunsaturated (PUFA) fatty acids. The results from animal studies have repeatedly shown that diets rich in SFAs provoke a greater elevation of plasma cholesterol concentration than that observed for a PUFA-rich diet (Richard et al. 1982; Moore & Williams 1966).

In addition, human studies have shown that the lipoprotein fraction most greatly affected by the cholesterolaemic influence of SFA or PUFA is LDL. The extent to which the other lipoproteins are affected by dietary fatty acids, however, appears dependent upon the quality of the fatty acid consumed. For example, the primary effect of fish oils, which predominantly contain n-3 fatty acid, is to reduce the concentration of plasma cholesterol and Tg associated with VLDL. In contrast, however, vegetable oils which are rich in n-6 fatty acids are associated with low concentrations of LDL-C (Goodnight et al. 1982; Harris et al. 1983). Finally, a high ratio of PUFA to SFA (P:S) has been shown to be associated with reduced plasma HDL-C concentration (Kwiterovitch et al. 1985; Jackson et al. 1984; Kohlmeier et al. 1985). This latter observation is also supported by the results of a study completed by Ehnholm and coworkers (1982). It was identified that a diet low in fat, but containing a high P:S ratio, acted to reduce the concentrations of TC, LDL-C, apolipoprotein B, HDL-C, apolipoprotein A-I, whilst simultaneously leading to an increase in the concentration of apolipoprotein A-II. These changes occurred in the absence of weight loss and were confirmed by the reversal of the changes observed when the habitual diet was re-applied (Ehnholm et al.

1982).

Whilst the evidence for the effect of PUFA and SFA on HDL-C concentration is quite substantial, there is a paucity of information available for MFAs. In general it appears that a diet in which MFAs predominate neither increases low concentrations of plasma TC, VLDL-C, LDL-C and Tg, nor provokes any decrease in the plasma concentration of HDL-C. It has therefore suggested (Mattson & Grundy 1985) that MFAs are relatively inert in relation to lipid and lipoprotein metabolism.

Since most populations derive approximately 80-90 percent of energy from fats and carbohydrates investigations into the influence of these foodstuffs are often completed simultaneously. The results of trials of high carbohydrate:low fat diets have shown an increase in Tg-rich lipoproteins (Ahrens et al. 1961) and a decrease in HDL-C concentration (Wilson & Lees 1972; Brussard et al. 1982). Furthermore, with a reversal of this situation, that is, a high fat:low carbohydrate diet, HDL-C concentration is again increased (Wilson & Lees 1972; Brussard et al. 1982).

Brunner and coworkers (1987) attempted to quantify the relationship between dietary manipulation and changes in HDL-C concentration. From their results, it was estimated that the replacement of 10% of energy usually provided by fat by the equivalent amount of carbohydrate would provoke a decrease in HDL-C of approximately 0.10 $mmol.l^{-1}$. This observation is consistent with epidemiological studies the results of which suggest that, within populations, HDL-C concentration is inversely correlated with carbohydrate consumption (Ernst et al. 1980; Arab et al. 1982). Also arising from these studies is the identification of a positive relationship between alcohol consumption and serum HDL-C

concentrations (Castelli et al. 1977; Hulley & Gordon 1981). Johansson and Mehdus (1974) have quantified this relationship, showing that for each 10 gm of alcohol consumed daily, the concentration of serum HDL-C is increased by 0.03-0.05 $mmol.l^{-1}$. Alcoholics therefore have been shown to have very high concentrations of HDL-C (Johansson & Medhus 1974).

2.1.5. Influences of changes in body mass or body fatness on lipoprotein metabolism.

In addition to the reported effects of diet, an abundance of evidence exists to suggest that body mass and body composition also affect lipid and lipoprotein metabolism (Carlson & Ericsson 1975; Rhoads et al. 1976; Albrink et al. 1980; Glueck et al. 1980). From their investigations Wilson and Lees (1972) suggested that a reduction of body weight provoked an increase in HDL-C and decrease in VLDL-C concentrations. The results of subsequent studies however, contradicted these early findings. For example, Thompson and coworkers (1979) reported that for obese females who experienced an average weight reduction of 8.6 kg in 10 weeks, there was also a reduction of 0.10 $mmol.l^{-1}$ in HDL-C concentration. This observation was substantiated by similar results derived from a number of subsequent studies (LaRosa et al. 1980; Weltman et al. 1980; Wolf & Grundy 1980; Oster et al. 1981). However, for a number of reasons, for example, failure to employ a control group, small number of subjects or short duration of the study period, Gurr and coworkers (1989) suggest that the evidence derived from these studies is open to question. Furthermore, the same authors (Gurr et al. 1989) identify several more stringently controlled investigations, the results of which are consistent with those derived by Wilson and Lees (1972). For example, Contaldo and coworkers (1980), observed the effects of weight reduction for very obese individuals enrolled on a long-term programme. After 15 months, the average weight

loss attained was 22 kg. This was accompanied by a 36% increase in HDL-C concentration. Similar findings were also reported by Streja and coworkers (1980) and by Sorbris and coworkers (1981). In addition, Caggiula and coworkers (1981) found that with a small decrease of body mass, even non-obese men could be shown to have a small but significant increase in HDL-C which is subsequently reversed with weight gain. This latter observation therefore suggests that the relationship between the two parameters is not restricted to the obese or to situations of profound loss of body mass.

The relationship between changes in body mass and HDL-C concentration have been explained through a mechanism involving lipoprotein lipase activity. Nikkilä (1978) observed a positive correlation between the rate of lipoprotein lipase activity and HDL-C concentration. Furthermore, with a decrease in body mass lipoprotein lipase activity declines, therefore reducing the concentration of HDL-C. This finding is consistent with those of LaRosa and coworkers (1980), Weltman and colleagues (1980), Wolf and Grundy 1980, and also Oster and coworkers (1981). However, on the attainment of a steady-state mass, the rate of lipoprotein lipase activity is again seen to rise, as does the concentration of HDL-C, therefore supporting the observations reported by Wilson and Lees (1972), Contaldo and coworkers (1980), and others.

A role for lipoprotein lipase in modifying HDL-C on weight loss is supported by the observation that obese individuals tend to have a lower energy intake than their lean contemporaries (Baecke et al. 1983). With a lower energy intake there would be expected to be less surface material available from the breakdown of VLDL, therefore due to a feedback loop type mechanism, the activity of lipoprotein lipase activity would be reduced. As a

consequence HDL-C concentration would decline. However, with the stabilization of energy intake, lipoprotein lipase activity would be expected to recover, resulting in an increase in HDL-C concentration (Wilson & Lees 1972; Contaldo et al. 1980).

Wood and coworkers (1988) investigated the relationship between dietary composition, body mass and changes in lipoprotein metabolism to a greater extent. The aims of the investigation were to discern whether the loss of body mass through reduced energy intake or through increased exercise produced differences in the resultant adaptations of lipid and lipoprotein metabolism. The results showed similar changes in HDL-C, HDL_2-C and TC:HDL-C in exercisers and dieters. Neither group however, showed any change in TC or LDL-C. It was therefore concluded that decrease in body mass (5.9 kg for dieters and 4.1 kg for exercisers) was responsible for the effects on HDL-C. This conclusion was substantiated by a positive correlation for dieters between the change in total energy intake and change in body fat mass, and also a negative correlation for exercisers between distance completed per week and change in body fat mass. Finally, further confirmation was derived from the negative correlation derived between the change in body fat mass and change in plasma HDL-C concentrations found for both groups. It was therefore concluded that exercise influenced the concentration of HDL-C via fat loss, since energy deficit produced by dieting or exercise had similar effects the concentration of HDL-C.

Tran and Weltman (1985) conducted a meta-analysis of the results of 95 studies concerned with the effects of exercise and changes in body mass on lipoprotein metabolism. The analysis showed that in the absence of a change of body weight, exercise was associated with a

decrease in the concentration of total cholesterol and LDL-C. When body weight was decreased, the decrease in total cholesterol and LDL-C was significantly greater than the decrease derived from exercise alone. Finally, with an increase in body weight during an exercise programme, total cholesterol and LDL-C concentrations were increased. These observations therefore confirm those of Wood and colleagues (1988), suggesting the existence of a combined effect of exercise and changes in body weight/fatness on lipoprotein metabolism.

Further evidence which is supportive of a role for body mass and more particularly for body composition, might be derived from the observation of alternative indices of body fat distribution. The ratio of waist to hip circumference (W:H) has been considered indicative of the degree of truncal fat. Ostlund and coworkers (1990) identified an inverse relationship between truncal fat and HDL_2-C concentrations in older men and women. This observation therefore confirming the importance of body fatness as a determinant of lipoprotein metabolism. Furthermore, the association between body mass index (BMI) and HDL-C concentration has been shown to remain significant even after a correction has been made for other known determinants of HDL-C concentration, such as smoking, age, etc (Heiss et al. 1980). These observations appear consistent with the conclusions made by Wood and coworkers (1988), for an independent effect of changes in body mass and body composition on lipid and lipoprotein metabolism.

Since both diet and body mass/fatness have been shown to influence lipoprotein metabolism, it is important that these factors are monitored when attempting to investigate the influence of exercise training.

2.2. Lipoprotein metabolism and training.

Early investigations revealed habitual exercisers to have higher plasma HDL-C concentrations than their less active contemporaries (Carlson & Mossfeldt 1964; Hoffman et al. 1967; Altekruse & Wilmore 1973). Subsequently, using more sophisticated methodology and more stringent experimental designs, further details of the relationship between habitual exercise and plasma lipid and lipoprotein metabolism have been gained.

2.2.1. Triacylglycerols.

Typically, endurance trained athletes have lower plasma Tg concentrations than those reported for the general population (Martin et al. 1977; Wood et al. 1976; Lehtonen & Viikari 1978; Vodak et al. 1980). This inverse relationship between exercise and plasma Tg concentration has consistently been identified for males. However, similar training studies involving females (Allison et al. 1981; Brownell et al. 1982; Franklin et al. 1979; Lewis et al. 1976) have yielded rather more ambiguous results.

An explanation of the differences in these results has involved the enzyme lipoprotein lipase, the role of which has already been discussed. Additional evidence has also been derived from training studies. For example, Nikkilä and colleagues (1978) showed that prior to training the rate of lipoprotein lipase activity was higher and the concentration of Tgs lower, for females than males. As a consequence, the potential for exercise to provoke further favourable changes in women would be expected to be lower. This is therefore consistent with the observation that the potential for exercise-induced change relates to pre-training status (Goode et al. 1966; Holloszy et al. 1964; Huttunen et al. 1979). Thus individuals with high pre-training Tg concentration would be expected to derive a greater reduction than would a

similar individual with an initially lower Tg concentration.

Further evidence in support of the role of lipoprotein lipase has been derived from observations of athletes trained in power or speed-type exercise. When compared with inactive individuals, no differences were discerned between the groups for plasma Tg concentrations, nor was there a difference in the rate of lipoprotein lipase activity (Goldberg et al. 1984). This is also consistent with the evidence suggesting that endurance, but not power training, results in increased muscle capilliarisation (Ingjer 1979). Since lipoprotein lipase is located on the endothelial tissue of muscle capillaries, any increase in vascularisation might be expected to promote lipoprotein lipase activity and thereby reduce plasma Tg concentration (Saltin & Gollnick 1983; Tesch et al. 1984).

2.2.2. Total cholesterol.

Plasma TC concentration appears resistant to change as a consequence of endurance training in either men or women (Allison et al. 1981; Franklin et al. 1979; Rotkis et al. 1981; Wynne et al. 1980). However, Haskell (1984) has suggested that any modification which does occur might be obscured by 'differential changes in the concentration of cholesterol transported as part of the various lipoproteins'. Where the concentration has been reported to decrease, this may (Brownell et al. 1982) or may not (Moll et al. 1979) have been accompanied by a reduction of body mass.

2.2.3. High density lipoprotein cholesterol and subfractions.

Cross-sectional studies which have compared endurance-trained athletes and relatively sedentary individuals have repeatedly shown a higher concentration of plasma

HDL-C in groups of trained individuals. This has been reported for both endurance-trained men (Hartung et al. 1980; Schnabel & Kindermann 1982; LaPorte et al. 1983) and women (Deshaies & Allard, 1982; Moore et al. 1983; Nikkilä et al. 1978; Vodak et al. 1980; Wood et al. 1977) when compared with age-matched sedentary controls. Evidence to further strengthen the association between activity and plasma HDL-C concentration has been derived from observations of patients suffering spinal injuries. For totally immobilized individuals, plasma HDL-C concentrations were significantly lower than for a second group of more mobile controls, matched for age, sex and relative weight (Nikkilä et al. 1980). This therefore suggests that even a minimal amount of activity is sufficient to provoke a favourable increase in the concentration of plasma HDL-C.

The results of cross-sectional studies support a favourable effect of endurance training on HDL-C concentration. However, the results of training studies are less consistent, particularly for females. Many of the studies of men have shown an increase in the concentration of plasma HDL-C after endurance training (Huttunen et al. 1979; Keins et al. 1980). A smaller number have reported either no change, or a decrease in HDL-C concentration (Brownell et al. 1982; Nye et al. 1981). The results for women are similarly inconclusive, with some studies reporting an increase in plasma HDL-C (Rotkis et al. 1981; Gilliam & Burke 1978) but others failing to show any change (Moll et al. 1979; Lewis et al. 1976) as a consequence of endurance training.

The variability of the results derived for both males and females, has at least partly been explained in relation to the duration of the training programme (Williams et al. 1982). This suggestion is supported by the studies of both men and women in which a non-linear dose-response

relationship has been determined between exercise and HDL-C. For example Wood and colleagues (1983) were unable to establish an overall difference for the concentration of HDL-C for formerly sedentary middle-aged men after the completion of a one-year exercise programme. However, the change in plasma HDL-C concentration was found to correlate strongly with the amount of exercise completed, thus suggesting a dose-response relationship between the two parameters. Furthermore, the relationship was found to be non-linear, with increases in HDL-C concentration becoming apparent only after the completion of 10 or more miles of exercise per week (Wood et al. 1983; Williams et al. 1982; Hartung et al. 1983; Lehtonen & Viikari 1979; Hartung & Squires 1979). It was therefore concluded

> "The existence of a threshold effect suggests the possibility of some physiological process being turned on at this point; for example, increased tissue lipoprotein lipase."
>
> (Wood and coworkers 1983).

The primary hypothesis proposed for the apparent "trigger" effect, an increase in lipoprotein lipase activity, will be considered in greater depth later in this review. However, the existence of a dose-response and trigger mechanism might help to explain why investigations which are of short duration and frequency sometimes fail to show alterations in HDL-C concentration. In the past such results have been considered as evidence against an exercise-related effect.

The relationship between changes in plasma lipoprotein metabolism and duration of training programme has been further explored. Both Ballantyne and colleagues (1981) and Brownwell and coworkers (1982), completed studies in which male and female subjects were enrolled in the same

training programme. The results led to the identification of a sex-related effect of exercise on HDL-C metabolism. For example, Ballantyne and coworkers (1981) identified a significant increase in HDL-C concentration for men (0.18 $mmol.l^{-1}$), but not for women. Similarly, Brownell and coworkers (1982) found a 8% increase in plasma HDL-C concentration for men, as compared with a 1% decrease for women. In addition, it was also shown that the number of sessions attended during the 10-week programme was positively correlated positively with HDL-C change in men but the same comparison produced a negative correlation for women.

On the basis of such evidence Williams and coworkers (1982) have suggested that a much longer period of time is required to elevate the plasma HDL-C concentrations of women relative to that required by men. Such a requirement might therefore explain the results of some studies, particularly with women, which have failed to find any evidence of increased HDL-C with training.

The mechanism considered basic to these sex-based differences in HDL-C response to endurance training, is again the activity of lipoprotein lipase:

> "It has been postulated that sedentary women are more resistant to the effects of exercise on HDL cholesterol than men, due to higher initial HDL cholesterol concentrations, as well as to related levels of lipoprotein lipase activity. These differences in lipid metabolism may be due to the effect of oestrogen on the lipase activity in women."
>
> (Goldberg and Elliot 1985).

As noted above, lipoprotein lipase activity is significantly higher in women than in men. This

observation even holds true for the comparison between sedentary women and endurance trained males (Nikkilä et al. 1982). As a consequence of this and the effect of oestrogen, baseline concentrations of HDL-C tend to be higher in females relative to males. This might constrain the effectiveness of short duration training programmes in women.

The effectiveness of such programmes is also likely to be limited since endurance training is associated with a reduction of the level of circulating oestrogen. As a consequence therefore, the metabolism of HDL-C in women would be expected to be reduced (Frey et al. 1982; Loucks & Horvarth 1984; Bullen et al. 1985; Dale et al. 1979; Bonen et al. 1981). In contrast, endurance training of male subjects is associated with an increase in plasma testosterone. As a consequence of this increase the breakdown of Tg is enhanced and HDL-C concentrations are elevated (Mendoza et al. 1981; Hill et al. 1980; Nordoy et al. 1979). It has therefore been suggested that endurance training acts to provoke an increase in the concentration of both testosterone and androstenedione in men (but not in women), thereby producing a testosterone-mediated increase in HDL-C (Remes & coworkers 1979).

Together, this evidence suggests that endurance training is likely to be less effective in increasing the HDL-C concentration in women than men. To summarize, pre-training oestrogen concentrations result in high levels of HDL-C, the effect of which is added to by the relatively high lipoprotein lipase activity. Secondly, training has the effect of reducing oestrogen therefore depressing HDL-C metabolism (Fotherby 1989). Finally, in the absence of any influence on testosterone, the potential for an exercise-induced increase in HDL-C concentration is reduced for women.

However, this is not to say that women cannot benefit from endurance training. A number of studies have shown favourable changes in lipid and lipoprotein metabolism in women (Rotkis et al. 1981; Wood et al. 1977; Nikkilä et al. 1978). It is therefore suggested:

> ".... that sedentary women may need more frequent, more intensive (Wood et al. 1977), and longer training programs than men to realize increases in C-HDL levels. Since pre-training mean C-HDL in women is 10-12 mg/dl higher than in men (LRC, 1980) it is possible, speculatively, that a greater exercise stimulus may be required in women than in men to elicit further increases in C-HDL."
> (Frey et al. 1982).

It is probable that both the sex-hormone differences and the dose-response relationship contribute in some way to the sex-linked differences observed for changes in HDL-C concentration with exercise training. Wood and coworkers (1983) have suggested that whether or not there is a different time course for the initial training effect in men and women. Cross-sectional data does exist to provide strong evidence of an association between elevated HDL-C and long term aerobic training in both sexes.

In support of this, a number of tightly controlled studies have provided evidence of a positive association between plasma HDL-C concentration and long-term aerobic training for women, similar to that discerned for men (Moore et al. 1983; Wood et al. 1977; Smith et al. 1982; Vodak et al. 1980). Furthermore, the comparison between sedentary and aerobically trained women for plasma HDL-C concentration is essentially the same as for men. It is possible therefore, that this training adaptation does occur in women. However, in order for the response to develop programmes of greater duration maybe required.

High density lipoprotein cholesterol is not a single molecular entity, but a class of particles containing almost equal amounts of lipid and protein. With the development of more sophisticated assay techniques HDL particles have been subclassified according to their size and density. Two of these subfractions, the larger, more lipid-rich HDL_2 and the smaller, denser HDL_3, predominate in human plasma (Anderson et al. 1978). It is currently postulated that HDL_2 is formed from HDL_3 by the incorporation of apolipoprotein C, phospholipids and cholesterol released during lipolysis by lipoprotein lipase and LCAT (Schmitz et al. 1982; Nichols et al. 1981) (Figure 2:5).

Of particular importance in this conversion of one subfraction to another is hepatic lipase (Figure 2:5). Hepatic lipase has been shown to hydrolze HDL, phospholipids and Tg (Ehnholm et al. 1975; Shinomiya et al. 1982). Furthermore, Groot and coworkers (1983) showed that the fractionation of HDL with hepatic lipase resulted in a marked decrease in HDL_2 and increase in HDL_3. Thus with an exercise related inhibition of hepatic lipase activity, the concentration of HDL_2 would be expected to increase. Evidence supporting this proposal has been derived from a number of cross-sectional and longitudinal studies.

Comparisons of endurance trained athletes with sedentary controls have revealed a higher HDL_2 concentration for the more active individuals. However, the same comparisons were unable to discern any difference between the groups for HDL_3 (Dufaux et al. 1982). Further support for this relationship has been derived by Kuusi and coworkers (1982), who discerned a positive correlation (r=0.52) between the concentration of HDL_2-C and exercise test performance in physically active military academy students. A number of cross-sectional studies (Wood &

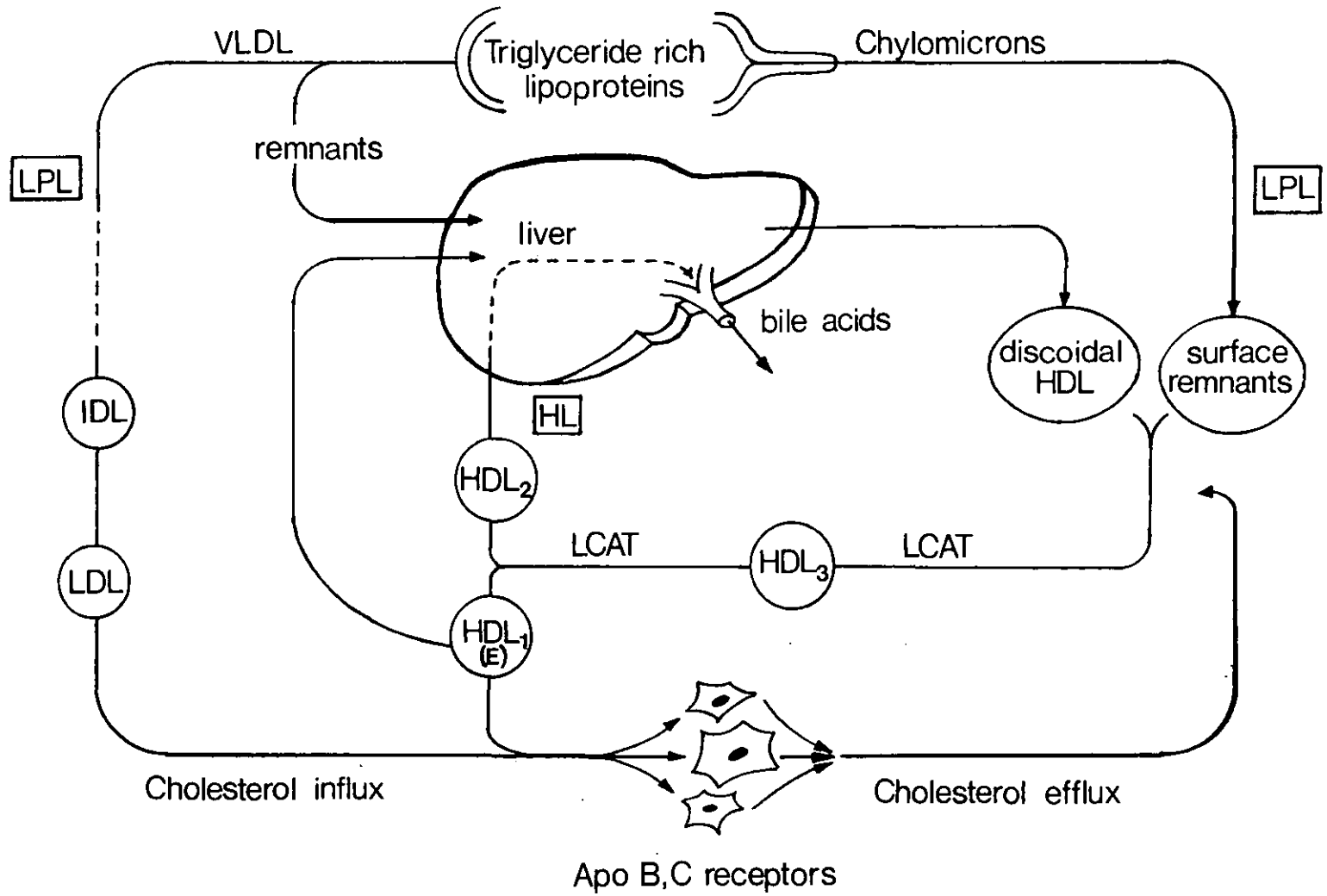


FIGURE 2:5 Metabolism of HDL subfractions (Assmann 1982).

Haskell 1979; Krauss et al. 1979; LaPorte et al. 1983) and longitudinal training studies (Wood et al. 1983; Williams et al. 1983; Ballantyne et al. 1982) have also reported a similar relationship.

The results of studies of HDL_3 are less consistent. In general investigations have shown no significant association between HDL_3 levels and exercise (Wood & Haskell 1979; Krauss et al. 1979; Ballantyne et al. 1982), or a negative correlation (Kuusi et al. 1982; Wood & coworkers 1983). LaPorte and coworkers (1983), however, reported elevations of both HDL_2 and HDL_3 in runners when compared with sedentary controls. The effects of exercise training on HDL_3 therefore, remain inconclusive.

2.2.4. Apolipoproteins.

The functions of the individual apolipoproteins remain only partly understood. They are, however, considered vital in the transport of the various lipids, in the regulation of the enzymes responsible for Tg catabolism and also in the exchange of lipids among the lipoproteins (Table 2:3). Cross-sectional comparisons of male and female endurance athletes have shown trained groups to have an apolipoprotein A-I concentration approximately 30 percent higher than inactive individuals. No difference however, was found for apolipoprotein A-II between the two groups (Wood & Haskell 1979; Krauss et al. 1977; Thompson et al. 1983; Lehtonen et al. 1979). Furthermore, evidence derived from longitudinal studies of the effects of enforced bed rest or detraining have shown a reduction of the concentration of apolipoprotein A-I. This effect has been reported for both men (23% lower) and women (34% lower) when compared with sedentary, but mobile controls (Nikkilä et al. 1980).

Several training studies have also been completed in

which changes in apolipoproteins have been observed. Keins and coworkers (1980) reported a 10% increase in apolipoprotein A-I concentration for men after a 12 week programme of endurance training. Wood and coworkers (1983), however found that neither apolipoprotein A-I or apolipoprotein A-II concentration changed significantly in sedentary men who trained for up to one year.

With the exception of the observations made by Wood and coworkers (1983), much of the available evidence appears supportive of a positive relationship between training and apolipoprotein A-I. This is consistent with the observation that apolipoprotein A-I represents 65% of the apolipoprotein content of HDL_2 (Assmann 1982). Therefore, if there is an exercise-induced increase in HDL_2 it would be expected that apolipoprotein A-I would also increase.

2.2.5. Mechanisms by which training may influence lipoprotein metabolism.

Until the full detail of the processes involved in the synthesis, transport and catabolism of specific lipoproteins is determined, it is unlikely that exercise-induced changes in lipid and lipoprotein metabolism will be completely understood. Some evidence for possible mechanisms does, however, already exist and will be presented in the remainder of this section..

For example, the inverse relationship between endurance training and plasma Tg concentration has been explained by both Krauss and coworkers (1979) and Nikkilä and colleagues (1978) as a consequence of an exercise-related enhancement of lipoprotein lipase activity. The increase in this activity would in turn be expected to provoke an increase in the rate of catabolism of Tg. Support for this proposal has been derived from the results of a number of investigations which have reported an increase in lipoprotein lipase activity after endurance training

(Kantor et al. 1984; Lithell et al. 1984; Sady et al. 1986; Taskinen et al. 1980). Peltonen and coworkers (1981), for example, reported a 56% increase in the activity of lipoprotein lipase in the adipose tissue of previously sedentary men after 15 weeks of moderate-intensity endurance-training.

It was previously reported that the concentration of lipids and lipoproteins observed in the plasma, are dependent on both the rate of synthesis and the rate of catabolism. It is therefore likely that the chronically low plasma Tg concentrations associated with physically active individuals is the consequence not only of increased Tg catabolism (due to enhanced lipoprotein lipase activity), but is also contributed to by a lower rate of Tg synthesis in the liver. Support for this suggestion is derived from the results of Zavaroni and coworkers (1981), whose preliminary studies with rats demonstrated a (38%) decrease in Tg synthesis after 16 weeks of treadmill training. However, whilst there appears to be no reports of complementary studies with humans, confirmatory evidence does exist for the suggestion that lipoprotein lipase is involved in the catabolism of Tg rich VLDL particles. This process results in the transfer of cholesteryl esters, protein and phospholipid remnants to newly formed (nascent) HDL particles which are subsequently secreted by the liver into the bloodstream (Eisenberg 1984). Thus it would be expected that an increase in lipoprotein lipase activity would provide additional substrate for HDL synthesis, contributing to the elevated plasma HDL-C concentrations previously reported in with endurance trained individuals.

Furthermore, endurance training is also associated with increased vascularisation of the capillary bed of the trained muscle. Since lipoprotein lipase is located on

the endothelium of these tissues, increased capilliarisation would be expected to result in a greater lipoprotein lipase activity. This proposal is consistent with the observations made of power or sprint trained athletes. For example, Berg and coworkers (1980) compared weight-lifters, shot-putters and discus throwers; Nikkilä and coworkers (1978) compared sprinters and distance runners, and Farrell and colleagues (1982) compared weight-lifters and speed-skaters. In each of these comparisons the sprint trained athletes could not be distinguished from non-exercisers with regard to plasma HDL-C concentration. In contrast, endurance trained athletes were found to have a significantly higher concentration of HDL-C when compared with non-exercisers. This therefore suggests that duration rather than intensity of training might be a more potent stimulus in the determination of HDL-C concentration (Moore et al. 1983; Hartung et al. 1983). Furthermore, this observation is also supportive of a role for lipoprotein lipase, since endurance, rather than sprint training, results in increased capilliarization of the muscle bed (Tesch et al. 1984; Saltin & Gollnick 1983).

The final line of evidence in support of the proposal of the effects of lipoprotein lipase, has been derived from clinical studies of lipoprotein lipase deficiency disease: hyperchylomicronaemia or Type I hyperlipoproteinaemia (Fredrickson et al. 1978; Nikkilä et al. 1982; Augustin & Greten 1979). The characteristics of the disease are extremely high concentrations of chylomicrons, a normal or mildly increased concentration of VLDL and a decrease in the concentrations of both LDL-C and HDL-C. When untreated, Tg concentration rises to 11.3-45.2 $mmol.l^{-1}$ and in rare cases to 170 $mmol.l^{-1}$. This occurs as a consequence of the retarded degradation of chylomicrons due to the unavailability of lipoprotein lipase to hydrolyse Tg in the chylomicrons (Figure 2:6).

Thus, when lipoprotein lipase activity is reduced, possibly due to insufficient apolipoprotein C-II (an essential co-factor for the hydrolysis process) then chylomicrons accumulate. This observation is consistent with the reported existence of an inverse correlation between lipoprotein lipase activity and plasma Tg concentration (Huttunen et al. 1976). An increase in lipoprotein lipase activity has also been found to be associated with increased HDL concentration (Nikkilä et al. 1978), and with exercise intensity (Budohoski 1985). Finally, low levels of lipoprotein lipase activity have also been correlated with an increased incidence of CHD (Brier et al. 1985). A substantial amount of evidence therefore appears to exist in support of a lipoprotein lipase-mediated effect.

In addition to an exercise-related enhancement of lipoprotein lipase activity with training, evidence also exists to suggest a decrease in the activity of hepatic lipase (Krauss et al. 1979). Hepatic lipase is actively involved in the catabolism of IDL and VLDL, the products of which are used in the synthesis of LDL. Thus any suppression of the activity of this enzyme would be associated with a decrease in the rate of HDL-C catabolism (Grosser et al. 1981). Thus Eisenberg (1984) reported an inverse correlation between HDL-C and hepatic lipase concentration in highly trained runners. A similar relationship was also reported for active military academy students (Kuusi et al. 1982) and for middle-aged men before and after exercise training (Peltonen et al. 1981). Furthermore, Taskinen and Nikkilä (1981) reported a negative correlation between hepatic lipase activity and the concentration of HDL_2, whilst the opposite relationship was found for HDL_3. Nikkilä and coworkers (1982) explained these relationships by proposing that hepatic lipase acts to catalyse the conversion of HDL_2 in to HDL_3. Thus an exercise-induced decrease of hepatic

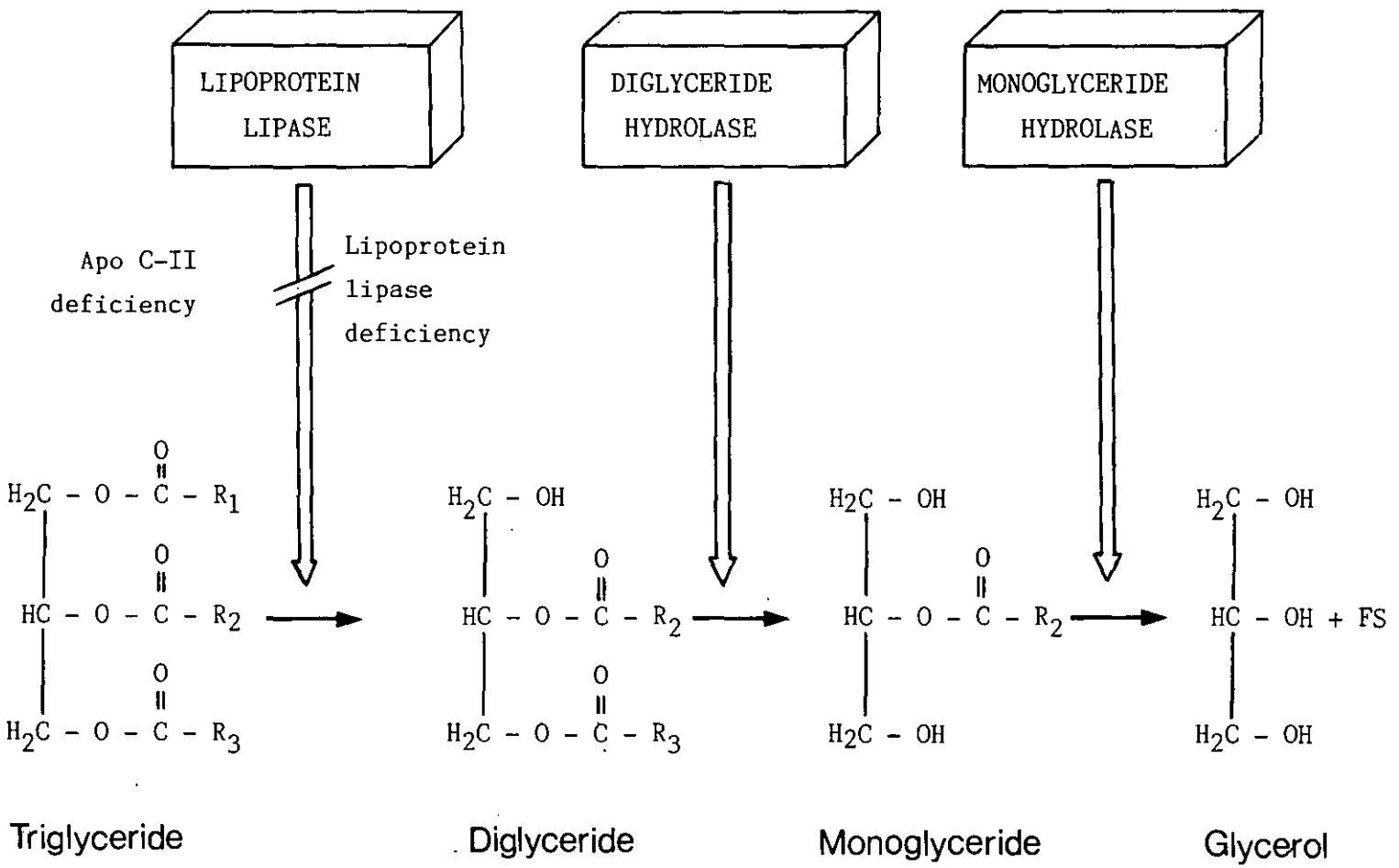


FIGURE 2:6 Reaction scheme of lipolytic enzymes (Havel et al. 1970).

lipase would be expected to provoke an increase in the synthesis of HDL_2 from HDL_3, the concentration of the latter which would thereby decline. This proposal is supported by the results of exercise studies (Dufaux et al. 1982; Kuusi et al. 1982; Krauss et al. 1979) which have shown an increase in HDL_2 with endurance training.

Further evidence to support the role played by catabolic processes in the promotion of HDL concentration with endurance training has been derived from studies in which radioiodinated autologous HDL has been employed. Using this technique, Herbert and coworkers (1984) showed that the mean biologic half life of HDL was 6.2 days for trained endurance runners, as compared with 3.8 days for sedentary men ($P<0.05$). It was also revealed that the fraction of the total HDL pool cleared each day was 27% lower in the more active group. It is therefore probable that the positive relationship between plasma HDL-C concentration and endurance training result from a combined effect of increased lipoprotein lipase activity and decreased hepatic lipase activity, the former encouraging a higher rate of synthesis, the second slowing the rate of catabolism.

Also contributing to the increased concentration of HDL-C associated with endurance training is the enzyme LCAT. The function of this enzyme is to catalyse the transfer of fatty acids located in plasma from lecithin to cholesterol, an important step in the production of HDL. As with lipoprotein lipase, the activity of LCAT has been shown to be enhanced with endurance training (Lithell et al. 1975; Marniemi et al. 1982). It has also been shown that moderate intensity exercise training of young men produces an increase in LCAT activity which is correlated with an increase in both HDL-C ($r=0.49$; $P<0.01$) and TC ($r=0.68$; $P<0.001$) (Marniemi et al. 1982).

Summary.

Endurance exercise appears to produce favourable changes in lipid and lipoprotein metabolism in both males and females. It is probable that these adaptations occur as a consequence of changes in enzyme activity in both muscle and liver tissue. The adaptations derived by women are of a relatively smaller magnitude and may require a longer duration of training to develop. The difference from the response in men is likely to result from sex-linked differences in hormone and enzyme concentrations. However, with the appropriate duration of training, the changes observed for men can also be discerned in women. The resultant changes produce an increase in HDL-C concentration and a decrease in the concentration of Tg-rich lipoproteins. Since such particles have been associated with an increase in the risk of CHD, this could be beneficial.

2.3. Adaptations to low intensity training.

An abundance of evidence exists to illustrate that one traditional measure of functional capacity ($\dot{V}O_2max$) attains a maximum at approximately 20 years of age, after which there is a steady decline with increasing age (Robinson 1938; Astrand 1956; 1960; Simonson 1971; Barry et al. 1966). Dehn and Bruce (1972) were able to quantify these changes and suggested a reduction of approximately 9.9% per decade. Observations derived from this and from subsequent studies, have led Smith and Gillian to conclude:

> "Disuse accounts for about half of the functional decline that occurs between the ages of 30 and 70, and aging causes the other half."
>
> (Smith and Gillian 1983).

Thus functional capacity, ageing and inactivity or disuse

appear inexorably linked. An age-linked decline in functional capacity provokes a reduction of activity, which in turn results in a further decrease in functional capacity. It is likely therefore that inactivity simply acts to exacerbate the natural decline in $\dot{V}O_2$ max associated with ageing. However, evidence exists to suggest that the progression of this downward spiral can be interrupted by aerobic training (Hartley et al. 1969; Robinson et al. 1973).

2.3.1. Brisk walking and functional capacity.

When considering the effects of endurance exercise, $\dot{V}O_2$ max is regarded as the standard international measure of cardiorespiratory fitness and index of functional capacity (Shephard et al. 1968; Taylor et al. 1955). Maximal oxygen uptake is the product of maximal cardiac output and maximal arteriovenous oxygen difference (Mitchell et al. 1958) and therefore defines the functional limits of circulation for aerobic metabolism (Dehn & Bruce 1972). Several factors including activity and inactivity or detraining, have been shown to influence $\dot{V}O_2$ max (Saltin et al. 1968; Fringer & Stull 1974; Saltin & Rowell 1980). Furthermore, $\dot{V}O_2$ max has been found to increase with increased lean body tissue (Buskirk & Taylor 1967). This observation might partly explain the age-related decline in functional capacity, as ageing is also associated with a decrease in lean body mass thereby reducing the amount of 'active' tissue available for oxidative metabolism (Dill et al. 1967).

Having recognised the positive relationship between exercise and increased functional capacity, the American College of Sports Medicine (ACSM) has made a number of recommendations with regard to the quality and quantity of exercise required to develop and maintain endurance fitness in adults (ACSM 1986). The characteristics of any training programme are determined by frequency, duration

and intensity. The ACSM suggest that in order to increase $\dot{V}O_2$max, the intensity at which the exercise is performed must exceed the minimal threshold of approximately 60% of the maximal heart rate reserve, or 50% $\dot{V}O_2$max (Karvonen et al. 1957). The actual intensity of exercise required to meet these thresholds is quite variable, depending to some extent on age and training status of the individual. Therefore an individual with a relatively low endurance capacity would be expected to experience a training effect with a sustained heart rate as low as 110-120 $bt.min^{-1}$. In contrast, a person of higher fitness would require a higher threshold of exercise intensity to stimulate adaptation (Gledhill & Eynon 1972; Hagberg 1984).

On the basis of such observations Pocari and coworkers (1987) suggested that brisk walking might prove to be an adequate stimulus for the improvement of functional capacity in sedentary or older individuals. This conclusion was based on the observation that the mean heart rate evoked during a one-mile brisk walk ranged from 133-150 $bt.min^{-1}$, equivalent to 73 to 86% of maximal heart rate. Thus the minimal threshold level prescribed by ACSM (1986) was exceeded by 91% of all women and by 83% of men aged 50 and older during brisk walking.

The study by Pocari and colleagues (1987) identified the potential of brisk walking to elicit a change in functional capacity. Other studies however have attempted to demonstrate this potential by comparing the results derived from walking with those observed with programmes of relatively high intensity exercise. For example, Santiago and coworkers (1987), investigated the effects of a 20 week programme of either walking or jogging with formerly sedentary women. Both modes of exercise produced a significant increase in $\dot{V}O_2$max from baseline (21% for walking and 31% for jogging). However, the difference in

the extent of these improvements (10%) was not statistically significant.

Seals and coworkers (1984) adopted a different experimental protocol for their examination of the effectiveness of walking. Subjects firstly completed six months of low intensity (walking) training, which was then followed by six months of training at a higher intensity. The results showed $\dot{V}O_2$max to increase by an average of 12% (range from 2 to 49%) with walking, whilst the higher intensity training period produced a further increment of 18%.

Additional supportive evidence for the effectiveness of brisk walking has been derived from studies including middle-aged individuals. For example, Jetté and coworkers (1988) completed a 12 week study of middle-aged (35-53 yrs) men and women. After training $\dot{V}O_2$ max was increased by 13.3% and 16.5% for males and females respectively, whilst the average increase for the whole group was 14.9%.

Pollock and coworkers (1971; 1975) conducted two, twenty week programmes of walking. The first of these involved men with an average age of 49 years. For this group, $\dot{V}O_2$ max was found to increase by 28%. The second study was also conducted with men, however, the mean age of this group was 38 years and the increase in $\dot{V}O_2$ max attained was 11%. The difference reported in the effectiveness of walking in provoking a favourable change in these two groups of different ages, may be indicative of the influence of pre-training status on the potential for change (Saltin 1969). Thus, as $\dot{V}O_2$ max has been shown to decline as a function of increasing age, the pre-training capacity of the older group would be expected to be less than for their younger counterparts. As a consequence, the potential for change would be greater for the older

group.

The results derived from studies conducted by Pollock and coworkers (1971; 1975) appear to support this latter proposal. In addition, support has also been derived from the results of other studies which have shown an inverse relationship been initial $\dot{V}O_2$max and the magnitude of increase experienced in response to training (Astrand & Rodahl 1986; Pollock 1973; Saltin 1969).

The adaptations which occur in response to endurance training and which have been associated with increased functional capacity are largely those which determine $\dot{V}O_2$max, that is, cardiovascular and metabolic parameters. Together these adaptations result in an increased ability to accept oxygen at the lungs, to transport oxygen to the working muscles and to use oxygen in the muscles for the formation of ATP energy. These changes are identifiable both at rest and during exercise, and include amongst others reduced heart rate, an increase in stroke volume and reduced systemic arterial blood pressure.

Whilst much of the data available has been derived from studies employing young adults, some studies have been conducted with older adults. Seals and coworkers (1984) for example, identified a reduction of resting heart rate in a group of older men consequential to endurance training. This observation is consistent with that derived for other studies (Barry et al. 1966; Robinson et al. 1973) which have also employed older adults. Furthermore, as with younger individuals (Saltin et al. 1968), the heart rate evoked during submaximal exercise at the same absolute work rate was also decreased after training (Barry et al. 1966; Robinson et al. 1973; Seals et al. 1984).

Early evidence (Robinson et al. 1966) resulted in the

recognition that normal heart rate response to exercise is the consequence of an inter-related effect of vagal withdrawal and β-adrenergic stimulation. Consequential to endurance training, parasympathetic activity is increased at rest, thereby accounting for the aforementioned bradycardia (Scheuer & Tipton 1977). This proposal is consistent with the evidence provided by Christensen and colleagues (1979) and Cousineau and coworkers (1977) of no change in myocardial tissue concentrations or plasma levels of adrenaline or nor-adrenaline after training. Together this evidence therefore suggests that the observed bradycardia is mediated through neural rather than hormonal means. Similarly, Winder and coworkers (1978) also suggested that exercise was associated with an increase in parasympathetic activity of the vagus nerves, again suggesting a more dominant neural influence.

In addition to a reduction at rest, heart rate is also reduced at any absolute level of oxygen uptake after training. However, unlike resting bradycardia, the available evidence suggests that the effect observed during exercise is more likely to be hormonally mediated. Cousineau and colleagues (1977) for example, reported that whilst no change in the concentrations of adrenaline or nor-adrenaline were apparent at rest, plasma concentrations are reduced at any absolute submaximal workload after training. This observation therefore supports the suggestion of a hormonal basis for both the bradycardia and reduced vasoconstrictor tone observed during exercise performance after training. This relative bradycardia may also be further promoted by other nonneural mechanisms, for example, by the alteration of the baroreceptor response due to chronic stress and relaxation in exercise.

This latter idea for a baroreceptor response was first

proposed by Barcroft and Swan (1953). It was suggested that as a consequence of the increased force developed during the contraction of larger muscle groups employed in the activity, as well as in those located in the thorax, there was a greater volume of blood returning to the heart after training. As a consequence of this increase in venous return and filling, stretch receptors located in the atria would be stimulated and stroke volume would therefore be increased by reflex.

In addition to bradycardia at rest and during submaximal exercise, a number of other myocardial adaptations to endurance training have been recognised. For example, an increase in stroke volume. The enhancement of this capacity might result from increased myocardial dimensions or by the enhancement of the contractile properties of the myocardium, that is, through increased ventricular filling, possibly resulting from the aforementioned bradycardia, or due to a reduction in myocardial work.

Evidence for a change of cardiac dimensions was first derived from early cross-sectional studies. Using bi-plane radiographs, total heart size was found to be closely correlated with $\dot{V}O_2$ max, with cardiac output and with stroke volume (Saltin et al. 1968). Studies completed recently have tended to employ an echocardiographic technique in the cross-sectional comparisons of trained and sedentary individuals.

Echocardiography utilizes high frequency sound waves to determine the depth of cardiac structures within the chest and thus image the heart in real time. This allows for a non-invasive, quantitive assessment of heart size and hypertrophy. The results of one such study (Morganroth et al. 1975) led to the identification of a relationship between endurance training and increased

left ventricular end diastolic volume. In contrast to isometric exercise, this increase in volume occurred in the absence of a change in wall thickness (See Shapiro 1987, for a review of hypertrophy in endurance and power athletes). The observations made by Morganroth and colleagues (1975) are consistent with those of a number of other studies, the results of which have been reviewed by Péronnet and colleagues (1981). The authors of this review concluded that endurance trained athletes have an internal left ventricular end diastolic diameter which is approximately 10% larger than that observed for sedentary individuals. This difference equates to a ventricular volume difference of approximately 33%.

In addition to the results of the review of cross-sectional studies, Péronnet and colleagues (1981) also considered the effects of 8 short term longitudinal training studies (<20 weeks). From this latter review it was concluded that as a consequence of endurance training, the $\dot{V}O_2$ max of formerly sedentary individuals was increased by approximately 17%. In addition, the average increase in ventricular end diastolic diameter was 1.3mm or 2.5%. This translates in to an increase in stroke volume of 16%, which is consistent with the increase reported for $\dot{V}O_2$ max. These results therefore indicate a close relationship between changes in $\dot{V}O_2$ max and myocardial adaptation.

In addition to changes in cardiac dimension, increased stroke volume with endurance training might result from an enhancement of the contractile properties of the myocardium itself. Whilst this effect remains a possibility, there is little evidence from either animal or human studies to support the proposal. For example, the echocardiographic evidence reviewed by Péronnet and coworkers (1981) was unable to identify any evidence for such an effect from either cross-sectional or

longitudinal studies. Thus the overall effects of training on intrinsic myocardial function remains unclear. Furthermore, both human and animal experiments have failed to identify any significant training effects attributable to enhanced contractile state. In addition, data derived from studies of the effects of left ventricular performance during maximal effort (Polinèr et al. 1980) suggests that improved contractile performance would confer little benefit since both the ejection fraction and end systolic volume appear to be little different in trained or untrained individuals.

From the evidence presented above it appears that the increase in stroke volume associated with endurance training and improved $\dot{V}O_2$max, is likely to be the consequence of changes in cardiac size, particularly in left ventricular diastolic diameter. The involvement of changes in the contractile properties of the myocardium remain uncertain. However, also important to the cardiovascular responses to endurance training is a reduction of systemic peripheral resistance. An inverse curvilinear relationship has been identified between $\dot{V}O_2$ max and systemic peripheral resistance from both longitudinal and cross-sectional studies (Clausen 1976). The advantage conferred by this training-induced reduction has been shown to allow the athlete to produce a cardiac output approximately double that produced by a sedentary individual at a similar arterial pressure during maximal exercise.

Experimental evidence also suggests that endurance training provokes an increase in the maximal conductance of the working skeletal muscles due to an increase in the size of the capillary bed and decrease in the arteriolar resistance. This effect appears to be limited to those muscle groups which have undergone training. This latter observation suggests a local rather than systemically

mediated effect. Nevertheless, it appears that close interdependency is required between the two, that is, central and local, systems. This co-ordination is required since the increase in $\dot{V}O_2$ max resulting from peripheral vasodilation is modified by an opposing vasoconstrictor influence, the strength of which is determined by the relationship between systemic oxygen demand and the capacity to transport oxygen.

Evidence exists to suggest that a key factor in the control of the flow of blood through the muscle is the metabolic state of the muscle itself. Thus with exercise and the development of acidosis, hyperosmolarity, or due to the release of adenosine, adrenergic transmission is inhibited and vasodilation occurs (Vanhoutte et al. 1981). As the flow of blood to the working muscle is increased, the potential to deliver oxygen for glycolysis is enhanced. Thus the acidosis, resulting from a build up of metabolites and increased dependence upon anaerobic glycolysis, is reversed and the possibility for continued exercise is increased.

In addition to reduced peripheral resistance, the delivery of oxygen to the working muscles is also enhanced by the proliferation of the skeletal capillary bed. With this increase the distance over which oxygen must diffuse from the haemoglobin into the mitochondria of the muscle cells is reduced. Furthermore, despite the increased rate of blood flow consequential to the aforementioned reduction of peripheral resistance the capillary transit time (time required for the diffusion of oxygen into the muscle cells) remains high due to the increased number of capillaries (Ingjer 1979; Tesch et al. 1984).

Together these adaptations allow for an improvement in the uptake, transport and delivery of oxygen with

training, thus increasing the possibility for the continuance of oxidative metabolism. However, in the absence of adaptations within the muscles themselves, the oxidative potential is restricted. Seals and coworkers (1984), for example noted that improved performance was associated with increased cardiorespiratory fitness ($\dot{V}O_2max$). In addition, however, adaptations in the skeletal muscle resulted in an increased oxidative potential. Together, the improvements resulted in the increased ability to perform tasks more easily and with the incursion of less fatigue.

The following section of the review now turns to the metabolic adaptations associated with endurance training.

2.3.2. Metabolic adaptations.

In the previous section, it was shown that the functional capacity of middle-aged sedentary individuals can be markedly increased with the appropriate form of training. For many years, this increase was considered to be the result of cardiovascular adaptation leading to an improved delivery of oxygen to the working muscles. However, evidence derived from both cross-sectional and longitudinal investigations have shown that muscle and blood lactate concentrations, measured at any given absolute or relative exercise intensity are reduced with training (Bang 1936; Ekblom et al. 1968; Karlsson et al. 1972). These observations therefore suggest that, in addition to cardiorespiratory changes which provide an increase in the availability of oxygen, peripheral metabolic adaptations also result.

It was previously considered that lactate production was the immediate 'energy donor' for muscular contraction. This concept was rejected with the demonstration by Lundsgaard (1930), that muscular contraction is possible

in the absence of lactate production. Further studies elucidated the role for lactate production as the end product of the Embden-Meyerhof pathway whereby some energy can be captured as adenosine triphosphate (ATP) when glucosyl units are degraded by muscle phosphorylase. It is now known that lactate production occurs as the result of the rapid breakdown of glycogen and that this can occur in the presence as well as absence of oxygen (Bang 1936; Connett et al. 1984; Koybashi & Neely 1979).

Normally there is a low concentration of lactate in muscle and blood at rest (approximately 1 $mmol.kg^{-1}$ wet muscle or litre of blood). This lactate originates from the low resting metabolic rate of muscle consequent to a low blood flow and also to the low rate of metabolism of red blood cells. At low exercise intensities (less than 40% $\dot{V}O_2$ max) little increase in lactate concentration occurs. As exercise intensity is increased, a point is attained at which an increase in the concentration of lactate in the blood and muscle is apparent. With increasing exercise intensities, the rise in both blood and muscle lactate concentrations becomes exponential.

The primary substrate for lactate production is the glucosyl unit, derived from muscle glycogen stores. The role of lactate production is to release some of the energy contained in the glucose molecule and to transfer it to ADP for the production of ATP. In this way lactate production acts as a important method for ATP production and, in the situation where oxygen uptake is high, this acts as a supplement to the normal oxidative resynthesis of ATP.

The net production of 3 ATP from the anaerobic degradation of 1 glucosyl unit from glycogen to lactate releases only about 10% of the total energy stored in the glucose molecule (Figure 2:7). Though small, this

capacity can be important when oxygen availability is limited, or more frequently, to supplement the energy produced through oxidative phosphorylation. Thus as recognised by Gollnick and Hermansen (1973), the relative importance of the anaerobic and aerobic components of metabolism to ATP production is related to the intensity and duration of the exercise.

An important influence on this relationship therefore is the training status of the individual. The increase in the concentration of lactate in muscle and blood is lower at the same absolute submaximal exercise intensity in endurance trained individuals than in nontrained individuals (Asmussen et al. 1974; Hermansen 1971). This observation has been explained as a result of improved oxygen delivery, a suggestion based on the belief that working muscles become hypoxic during exercise of relatively moderate intensity. An improved oxygen delivery at the same absolute workload would therefore be expected to induce less hypoxia and thus increase endurance capacity.

However, contrary to this suggestion, as previously disclosed even under resting conditions, when oxygen is abundant, the concentration of lactate in the blood is about 1 $mmol.l^{-1}$. Lactate production therefore has been shown to occur as the result of the rapid breakdown of glycogen, either with or without oxygen, thus questioning the suggestion that improved oxygen transportation can totally account for the improvements of endurance seen with training (Connett et al. 1984; Koybashi & Neely 1979).

Holloszy and Coyle (1984) also suggest that if untrained muscles are hypoxic during submaximal exercise (50-70% $\dot{V}O_2$ max), then improved oxygen delivery would provoke lower lactate production, and would therefore also be expected

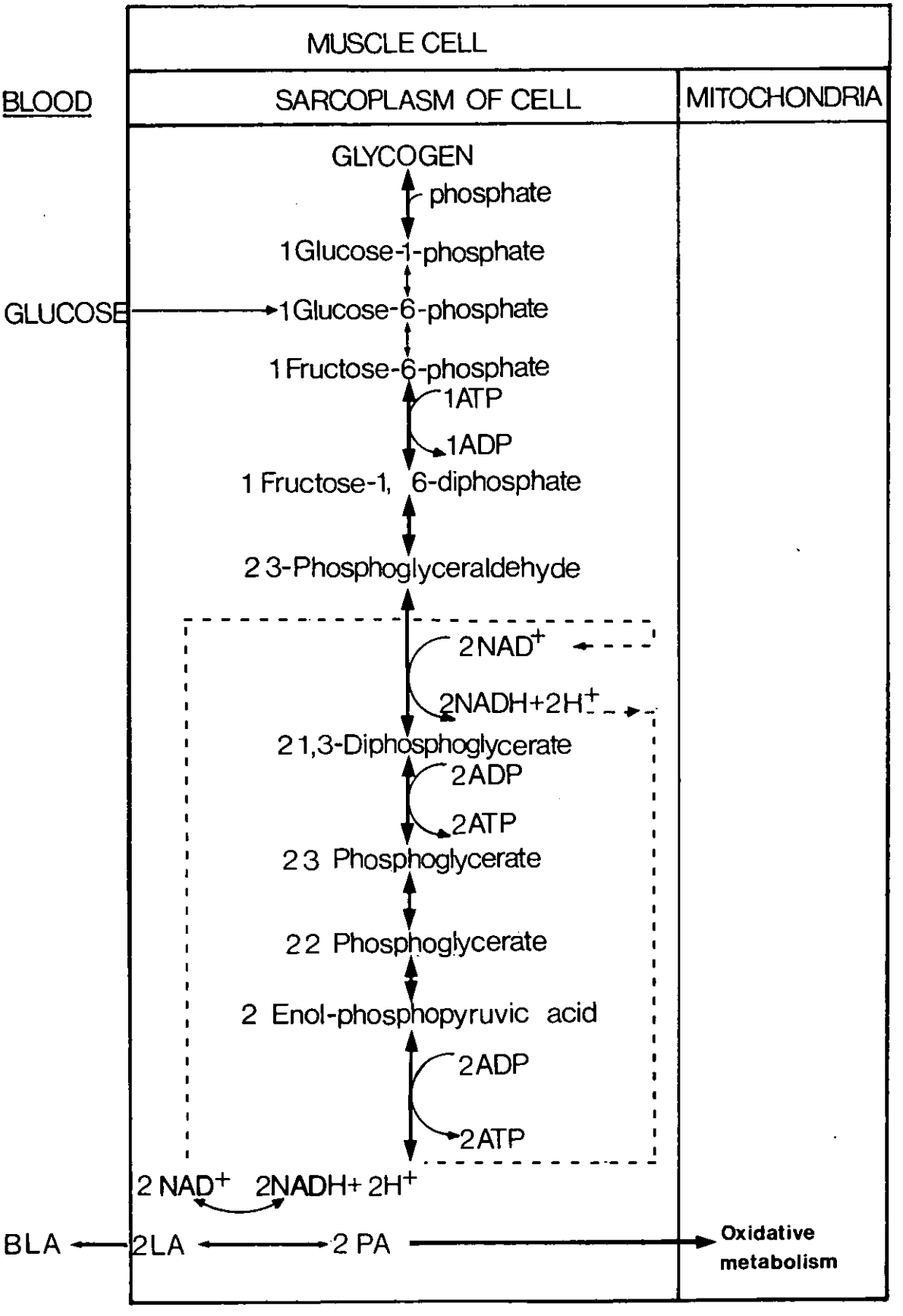


FIGURE 2·7 Anaerobic glycolysis

to be accompanied by an increase in oxygen uptake at the same absolute work rate. As this has not been shown to be the case (Hagberg et al. 1980; Hickson et al. 1978) Holloszy and Coyle (1984) have therefore suggested that lower lactate concentration and increased endurance in the trained state might be due to the metabolic consequences of biochemical adaptations in muscles rather than to improved delivery of oxygen.

In support of this, a number of peripheral adaptations have been shown to accompany endurance training. These include an increased capilliarization of the vascular bed serving the working muscles. The effect of this increased proliferation is twofold. Firstly, it would produce a reduction of the distance between the blood and cell interior so improving the transfer of substances into and out of the muscle. Secondly, the surface area available for this exchange would also be increased. Whilst these adaptations would be expected to have little consequence at rest, during exercise the increased rate of exchange of oxygen and metabolites might increase oxidative potential and thus reduce the accumulation of lactate in the muscle and blood (Saltin 1985).

In addition to vascular changes, both the number and the size of mitochondria has been shown to be greater in trained as compared with untrained muscles (Howald 1982; Saltin & Gollnick 1983). This is of particular benefit since mitochondria contain the enzymes responsible for oxidative phosphorylation. Evidence for an improved capacity for oxidative metabolism has been derived from both animal and human studies, which have shown that endurance training results in a significant increase in the amount of mitochondrial protein in the muscle. For example, Holloszy (1967) reported a twofold increase in the ability to oxidize pyruvate in muscle mitochondria after training, an increase accompanied by a doubling of

succinate dehydrogenase, NADH hydrogenase, NADH-cytochrome c reductase, and cytochrome oxidase activity. The result of these changes therefore is an increased capacity to resynthesize ATP via oxidative phosphorylation and a maintenance of a higher ATP/ADP ratio, the effect of which is to suppress glycogenolysis, thus reducing lactate accumulation (Morgan & coworkers 1971; Barnard et al. 1970; Gollnick & Ianuzzo 1972).

With an increased ability to use oxidative processes for ATP resynthesis, the rate of formation of lactate is reduced. This occurrence is subsequently reflected in the ability to exercise at a greater percentage of $\dot{V}O_2max$ or at the same intensity for longer, prior to the attainment of a reference blood lactate concentration of 2 or 4 $mmol.l^{-1}$ (Kindermann et al. 1979; LaFontaine et al. 1981; Pollock et al. 1977; Rusko et al. 1980; Skinner & McLellan 1980; Davis et al. 1979; Sjodin & Jacobs 1981). However, it has also been recognised (Donovan & Brooks 1983) that the lower blood lactate concentration observed with training is at least partially the consequence of an enhanced removal and utilization of lactate. Thus with an increase in the blood flow through the working muscle the rate of lactate removal is increased. Some of this is taken up by the liver for oxidation or glyconeogenesis. Secondly, evidence exists to suggest that both active and inactive muscle can take up lactate during exercise (Bang 1936; Karlsson et al. 1974). Furthermore, the rate of that uptake is increased with training (Gollnick et al. 1981; Saltin et al. 1976). Hubbard (1973) has suggested that lactate production probably occurs at all intensities of exercise and that the difference between its production and clearance determines whether or not there is an accumulation in the blood.

Whilst many of the training studies completed in this area of lactate metabolism have been conducted with young

adults, some workers have studied older individuals (e.g. Seals et al. 1984). Their study was conducted with a group of men averaging 63 years old. It was observed that after the completion of a 6 month programme of walking, blood lactate concentration measured at the same absolute exercise intensity was reduced by 25-30% from pre-training values. That is, with training, the intensity of exercise necessary to attain the same lactate concentration was increased by 17 percent. These observations are consistent with those reported by Barry and coworkers (1966). This latter group also considered the effects of a programme of walking with older adults. Together, the two studies show that the lactate response in older adults is similar to that described for young individuals (Astrand & Rodahl 1986; Holloszy 1973; Hurley et al. 1984; Robinson & Harmon 1941).

Evidence presented in this brief overview suggests that as a consequence of endurance training a number of central and peripheral physiological and metabolic adaptations occur. As a consequence of these adaptations the ability to deliver and to utilize oxygen at the working muscles is enhanced. Thus the concentration of blood lactate measured at the same absolute submaximal work rate is reduced (Asmussen et al. 1974; Hermansen 1971). Together the adaptations become manifest in an increased ability to maintain the same exercise intensity for longer or to attain a higher work intensity, making the attainment of previously difficult tasks more likely.

2.4. Psychological benefits of exercise.

Many health professionals (Byrd 1963) and advocates of physical activity (Cooper 1983) believe that exercise can confer a number of psychological benefits. In order to gain a more objective view of these often anecdotal claims, an increasing number of scientific investigations

have been completed (Bahrke & Morgan 1978; Folkins & Sime 1981). Whilst many of these have been concerned with clinical populations (Morgan 1981; Hartz et al. 1982), perhaps of greater relevance and interest to habitual exercisers, are those studies which have involved 'normal' subjects in programmes of endurance training.

A plethora of psychological benefits have been proposed for exercise, of which reduced anxiety, as measured by the state-trait anxiety inventory (STAI) (Speilberger et al. 1970), has gained particular attention. Pauly and coworkers (1982), for example, after 14 weeks of training formerly sedentary office workers demonstrated a significant reduction on this measure. This observation is consistent with that made by Blumenthal and coworkers (1982), who found that a 10 week programme of walking was sufficient to reduce anxiety ratings significantly in a group of non clinical middle-aged males. Confirmation of this response to endurance training is derived from a number of other studies from which similar results have been reported (McGlynn et al. 1983; Wood 1977; Bahrke & Morgan 1978).

Having accepted the inverse relationship between exercise and anxiety, Morgan and Hortsman (1976) attempted to discern the level of physical activity required to provoke these effects. In order to do this, all subjects exercised at 80% $\dot{V}O_2$ max on a motorized treadmill to volitional exhaustion. Throughout the performance of the exercise, a shortened form of the STAI was presented. The results of the study revealed an immediate rise in state anxiety concomitant with the onset of exercise. Anxiety then stabilized at this elevated level throughout the remainder of the exercise period. At the cessation of the activity, anxiety was shown to decrease and within 10 minutes of recovery was at a level below that measured prior to the exercise.

The observations of Morgan and Hortsman (1976) therefore lead to the suggestion that exercise itself is a stressor. This view is consistent with the proposal that the stress encountered during exercise acts to condition the individual, thus enabling future stressful situations to be more easily coped with subsequent to exercise training. This is supportive of the earlier suggestion (Michael 1957) that stress might be a learned phenomenon. Thus, after repeated couplings of exercise-induced stress and a non threatening situation, anxiety scores might be expected to be decreased (Lion 1978; Folkins 1976; Young 1979). However, some studies have reported no changes in anxiety status after endurance training (Massie & Shephard 1971; Young & Ismail 1976; Prosser et al. 1981).

A number of possibilities exist to explain these contradictory findings. Firstly, prior to the onset of the exercise programme, the experimental group might have regularly participated in exercise, thus reducing the influence of the prescribed programme. Secondly, poor compliance to exercise may have reduced the effectiveness of the "treatment". Thirdly, the exercise may have been of insufficient intensity. Finally, the measuring devices may have been insufficiently sensitive. Any combination of these would be sufficient to influence the results.

In addition to the anti-anxiety effect of exercise, its influence as an antidepressant is another widely proposed psychological benefit (Byrd 1963). Griest and coworkers (1978) compared the effects of running with psychotherapy for mild-to-moderate depression and suggested that the relief in depression due to exercise was of a similar magnitude as that of psychotherapy. Similar results were also derived by Blumenthal and colleagues (1982). This group used the Perception of Mood State questionnaire (POMS) (McNair et al. 1971), and found a significant decrease in depression, tension and fatigue. Furthermore,

the perception of vigour reported by the exercise group was increased. It was therefore concluded that the increase in physical health provoked by training, was also associated with an improved 'psychological' health. This view is also supported by Young and Ismail (1976).

Hughes and coworkers (1986), considered that the protocol adopted in many of the studies completed in this area of investigation were insufficiently rigorous. Thus, these workers applied a cross-over procedure, in which non-clinical individuals were randomly assigned to either an exercise (walking) or control group. After 12 weeks, the roles were then reversed, such that each individual acted as his own control. Using POMS it was shown that neither mood disturbance nor any of the POMS sub-scales were improved to any greater extent with the exercise period as compared with the control period. This is consistent with other similar randomized trials (Prosser et al. 1981; Stern & Cleary 1982). The results however, do contradict those derived from studies using a different methodological approach (Bahrke 1979; Folkins & Sime 1981; Mobily 1982). Hughes and colleagues (1986) suggest that many of the studies showing improvements of mood states have provided situations more conducive to change. For example, it is suggested that such studies have involved subjects suffering a psychopathological problem, have employed a more intensive training regimen or was influenced by other factors accompanying exercise such as socialization or counselling. The cross-over design, however, was considered by the authors to provide a more realistic situation, that is, the conditions of the study were more representative of those under which most people would begin an exercise programme. The results were interpreted by the investigators as suggesting that men free of psychopathology, beginning an exercise programme which is not social and of moderate intensity would be unlikely to experience any change in mood. This is not to

say however, that after prolonged training that mood changes are not possible.

The effect of exercise on self-concept has also been investigated. Using the Tennessee Self Concept Scale (TSCS), Pauly and colleagues (1982) reported an increase in all areas of self-concept, that is, physical, personal and social self-concept. This result is consistent with other studies which have also considered self-concept and exercise training in a non clinical population. Dowell and coworkers (1968) found a positive correlation between physical self-concept and fitness ($\dot{V}O_2$ max). However, both White (1974) and Henderson (1974), showed all aspects of self-concept to increase as a consequence of exercise. Finally, Hughes (1984) also using TSCS, showed that whilst total self-concept was increased for the group which underwent supervised training, the exercise group who were unsupervised only improved for social self-concept. Hughes therefore suggested that this observation might be indicative of the importance of additional unaccounted for influences (e.g. the effect of supervision) in the beneficial changes accredited by others wholly to exercise.

Not all the evidence is supportive of a positive relationship between exercise and self-concept. A number of studies have been unable to find any change with exercise. For examples, neither Leonardson and Garguilo (1978) with college students, nor Gary and Guthrie (1972) with hospitalized alcoholics, were able to find any change in self-concept despite an apparent increase in physical fitness.

Blumenthal and coworkers (1982), however, conclude that the bulk of evidence suggests that "... basically healthy, well-adjusted people can increase their sense of well-being compared to healthy people who do not

exercise..." a statement consistent with much of the evidence previously presented.

2.4.1. Mechanisms of psychological benefits.

Behavioral, cognitive and physiological processes have been proposed to mediate the psychological benefits of exercise. For example, the antianxiety and antidepressant effects of exercise have both been suggested to be mediated by behavioral and cognitive processes. The suggestion is that cognitive diversion results in a reduction of anxiety and depression in exercise, as subjects are unable to concentrate on their problems when exercising. In support of this proposal evidence exists to suggest that single episodes of exercise, meditation, and rest produce similar reduction in anxiety, perhaps because all provide a cognitive diversion (Bahrke 1979).

Social reinforcement is also consequential to exercise and is considered a major factor in the success of many forms of psychotherapy, and may also be a factor involved in exercise therapy (Chasey et al. 1974; Greist et al. 1979).

Mastery (control) is also developed with exercise and is considered to occur because exercisers readily perceive their improved physical state. Mastery experiences improve the individuals self-confidence or self-efficacy, which in turn improves their ability to cope with their problems (Bandura 1977). Although exercise does improve self-concept, whether this improvement influences mood, personality or cognition is still to be disclosed.

The proposed anti-anxiety effect of exercise has also been suggested to be a consequence of its effects on the somatic symptoms of anxiety. In support of this idea, Schwartz and coworkers (1978) found that with exercise somatic anxiety is decreased, whilst the level of

cognitive anxiety remained unchanged. Hollandsworth (1979) suggested that exercise might act as a form of biofeedback resulting in an improved perception of somatic signals. Such an improvement in the perception of bodily states appears to both decrease anxiety and also to increase the ability to discriminate stress-producing situations (Michael 1957). Alternative ways in which exercise may interact with somatic signals have been shown. For example, on exercising symptoms normally associated with anxiety are experienced, that is, sweating, hyperventilation, fatigue, palpitation and dyspnaea. Because these symptoms occur in the absence of the subjective state of anxiety, after recurrent experience of this situation it is suggested that processes such as the reattribution of symptoms (Schachter & Singer 1962), cognitive dissonance (Festinger 1957), counter conditioning (Wolpe 1958) or extinction (Wilson & Davidson 1971) cause a reduction in the reported state of anxiety.

Other suggestions adopted to explain the antianxiety properties of exercise have related to improvements in physiological and biochemical responses to stress (Ledwidge 1980; Michael 1957). Stressors have been shown to increase physiological responses such as muscle tension, heart rate and skin conductance (Lang et al. 1972), in addition to biochemical responses such as the release or concentration of catecholamines (Frankenhauser 1975; Ransford 1982; Starzec et al. 1983), lactate (Pitts 1969; Walsh & Davidson 1980) and glucocorticoids (Selye 1976). It has therefore been proposed that exercise might act to quicken the onset, to decrease the magnitude or to reduce the recovery time of these responses (Ledwidge 1980; Michael 1957), thereby decreasing stress-induced emotions such as anxiety and hostility (Selye 1976; 1975).

It has been suggested that since habitual endurance exercise has been associated with an increase in the concentration of endogenous opiates (Carr et al. 1981), then improved mood state might also be related to the elevation of these substances (Moore et al. 1982). However, other evidence exists to dissociate increased endogenous opiate concentration from any exercise-related improvement in mood (Farrell et al. 1982). In addition, the opiate antagonist naloxone does not block the psychological benefits derived from exercise (Markoff et al. 1982).

The limited amount of evidence therefore appears to contradict the suggestion that endogenous opioids are responsible for the improved mood during exercise. In contrast high opiate levels are related to low levels of fatigue (Farrell et al. 1982) and opioid antagonists do block exercise-induced analgesia (Haier et al. 1981). Thus exercise-induced increases in endogenous opiates may actually control negative mood more than positive mood, but, this is merely speculation.

Summary.

Evidence exists to suggest a favourable role for endurance exercise in the improvement of indices associated with physiological, psychological and health parameters. The aim of the following chapters is to examine the effectiveness of walking, a socially acceptable form of exercise to improve functional capacity, endurance, lipid and lipoprotein metabolism, indices of body fatness and aspects of psychological well-being.

3. GENERAL METHODS.

The specific procedures followed in the studies reported in this thesis are described briefly in the relevant methods section of each experimental chapter. However, much of the methodology is common to several studies and is reported here. These General Methods are detailed in the present chapter.

3.1. Informed consent.

Prior to the commencement of any form of testing, all subjects were made fully aware of the equipment and procedures to be employed and of any potential risks which they might incur. They were each then asked to sign a statement of informed consent (Appendix 1).

3.2. Oxygen uptake and carbon dioxide production.

In order to collect expired gas samples, subjects wore a nose-clip (Harvard Equipment Ltd.) and a snorkel-type mouthpiece (Harvard Equipment Ltd.). The mouthpiece was attached to a lightweight two-way respiratory valve (Jakeman and Davies 1979), which in turn was fitted to a 1.5 metre section of wide bore (30 mm diameter) lightweight tubing (Falconia; Baxter, Woodhouse and Taylor). The tubing terminated in a two-way tap (Harvard Equipment Ltd.), which was used to open or close a 150 litre capacity Douglas bag (Harvard Equipment Ltd.). Expired samples were collected at rest for six minutes and during exercise for one or two minutes depending upon the nature of the exercise test performed.

The collected samples were analysed as follows:-

i. A paramagnetic oxygen analyser (Taylor; Servomex Model 570A) was used in all studies to measure the oxygen content of expired air samples. The digital readout of the instrument was accurate to 0.1%.

ii. An infra-red carbon dioxide analyser (Lira; Mines Safety Appliances Ltd., Model 3030), was used to determine the carbon dioxide content of the expired air. As the output from the measuring cell was non-linear, the scale reading was converted to a percentage value using a calibration chart supplied by the manufacturer.

The zero value of both analysers was established using 100% nitrogen. A reference gas with a certified composition (approximately 5% carbon dioxide, 15% oxygen, balance of nitrogen: supplied by CryoService Ltd., Worcester, U.K.) was used to establish the span values. During the studies the analysers were re-calibrated between each test. The volume of expired air withdrawn for analysis was measured using a flow meter.

The volume remaining in each Douglas bag after analysis was determined by evacuating the expired air (Moulinex vacuum pump 237) through a Harvard dry gas meter, which had been calibrated against a 600 litre Tissot spirometer (Collins Ltd.). Gas temperature was measured during the evacuation by a thermistor positioned within the air outlet pipe of the dry gas meter. The thermistor was linked to a thermometer (Edale, type 2984, Model C).

All gas volumes were corrected to standard temperature and pressure for a dry gas (STDP). The Haldane transformation was then employed to derive the volume of inspired gas ($\dot{V}I$), thus enabling the calculation of the volumes of carbon dioxide produced ($\dot{V}CO_2$) and of oxygen consumed ($\dot{V}O_2$) (Appendix 2).

3.3. Exercise Test Protocols.

In order to investigate the effects of training on the physiological response to exercise, the following tests

were employed:

3.3.1. Submaximal tests for the prediction of maximal oxygen uptake.

The characteristics of some of the subjects involved in this series of studies (sedentary and/or middle-aged) dictated that $\dot{V}O_2$ max should be predicted rather than measured directly. The predicted values were derived using one of two exercise protocols, either a graded treadmill test or an incremental step-test.

i. Treadmill assessment: subjects walked at a constant speed on an inclined motorised treadmill, the gradient of which was increased by 2.5% every four minutes. The treadmill speed was selected to elicit approximately 50, 60, 70 and 80% $\dot{V}O_2$ max.

ii. Step-test assessment: the height of the step was adjusted to 42% of each individual's leg length, as measured from the greater trochanter (Shahnawaz 1978). Cadences were dictated by a battery operated metronome (Melina) and were selected on the basis of the preliminary trials, to elicit approximately 50, 60, 70 and 80% of each individual's predicted $\dot{V}O_2$max.

At least two preliminary trials were completed on the treadmill or the steps. Based upon a conservative judgement of each subject's $\dot{V}O_2$ max, four submaximal work rates were selected. Each work rate was performed for four minutes. During the final minute of each stage heart rate and oxygen consumption were recorded. These two parameters were then plotted at each workrate and a regression line established. In accordance with the procedure described by Maritz and coworkers (1961), the line was then extrapolated to a heart rate of 180 bt.min^{-1} and the the oxygen consumption corresponding to that level was considered the maximum. Based on this

prediction of $\dot{V}O_2$ max, an accurate judgement was made of the work rates corresponding to 50, 60, 70 and 80% of each individual's maximal capacity.

Once the appropriate work rates had been established, the tests were conducted. Thumb prick blood samples were obtained at rest and at the end of each of the four stages of the test. These were subsequently analysed for lactate. Expired air samples were obtained during the final minute of each stage and analysed for O_2 and CO_2, whilst heart rate was monitored throughout.

For each individual blood lactate concentration was plotted against oxygen uptake, the $\dot{V}O_2$ at 2 $mmol.l^{-1}$ was then interpolated and used as an indicator of changes in aerobic capacity. The adoption of this index is based on the evidence derived a number of studies which have been completed to investigate the relationship between blood lactate concentration and oxygen uptake. The 2 $mmol.l^{-1}$ level on the accumulation curve (the plot of blood lactate concentration oxygen consumption) is a point at which lactate begins to rise above resting level (Wasserman et al. 1973). Recent studies have suggested a close relationship between this concentration and $\dot{V}O_2$max (Davis et al. 1976) and/or endurance performance (Sjödin & Jacobs 1981) therefore being indicative of changes in aerobic capacity.

In the light of observations of the provocative nature of methods for the direct measurement of $\dot{V}O_2$ max (Yoshida 1986), in addition to the characteristics of the subjects involved in the present studies, cardiorespiratory function was assessed submaximally.

3.3.2. Track walks.

Two outdoor walking tests were performed on an athletics track, one was completed at maximal pace, whilst the

other was at a self-selected brisk pace. The distance completed in both was one mile (1609 m).

i. Maximal walk. A maximal walk required that subjects complete the one mile as quickly as possible, whilst still maintaining a walk. This test therefore allowed a direct measurement of performance improvement and functional capacity (Kline et al. 1987). This relationship was also verified by Cunningham and colleagues (1982), and in the study reported in Chapter 5, in which a positive correlation was derived for $\dot{V}O_2max$ and maximal pace ($r=0.376$, $P<0.01$).

ii. Submaximal walk. Submaximal walks were completed at a self-selected, brisk pace, the instructions for which were: "Walk at a brisk pace which you could sustain for half an hour". The aim was to maintain a constant pace throughout. The speed adopted during this walk was then reproduced in the laboratory on the treadmill.

3.3.3. One mile treadmill walk.

This test was completed to evaluate the physiological responses elicited at the 'training pace', that is, at the pace adopted as brisk. The pace of the submaximal track walk was reproduced in the laboratory for each individual. Subjects then walked for the same duration and speed on the treadmill. Heart rate was recorded throughout, as previously detailed. Thumb prick blood samples were obtained at rest and immediately on completing the walk. These duplicate samples were subsequently assayed for the concentration of lactate (Appendix 5). Expired air samples were obtained at rest (six minute sample), over the half mile, and at the end of the walk.

Submaximal treadmill walks were performed on a motorized treadmill. The Woodway model (ELG2) was linked to a Commodore CBM PET computer. A programme written in the Sports Science Research Laboratory by H.K.A. Lakomy was used for the continuous monitoring of treadmill speed, heart rate, exercise time, and distance remaining, this data was updated every few seconds.

3.4. Heart rate monitoring.

Heart rates were monitored throughout all exercise tests. In the laboratory, a standard electrocardiographic technique was used, on the track however, a system of short range telemetry was employed.

3.4.1. Electrocardiography. Heart rates were recorded on an electrocardiograph (Rigel Cardiac Monitor 302, Rigel Research. U.K., Ltd.) from three chest electrodes (3M U.K., Ltd. Type 2255) arranged in a modified Lead I configuration. The first electrode was placed at the sterno-clavicular junction, the second and third electrodes were placed on the left and right sides at approximately 80 mm below the tenth rib. The final electrode, placed on the right shoulder was earthed in order to remove static electricity generated when walking and therefore to improve signal strength. Prior to placement, the four electrode sites were thoroughly cleansed using iso propyl and abraded to decrease skin resistance.

The cardiac monitor was interfaced with a micro-computer and heart rates were automatically recorded at intervals of 15 seconds.

3.4.2. Short range telemetry. Throughout the track walks heart rate was recorded using a short range telemetry system (PE 3000 Sports-tester, Sweden) which comprised an elasticated belt electrode worn about the lower ribcage

and a wrist-watch type micro-computer. The micro-computer was programmed to sample at 15 second intervals. This information was stored and retrieved later.

3.5. Body composition and anthropometry.

These procedures were employed in order to identify the amount and distribution of body fat.

3.5.1. Subject body mass.

When body mass values were required for the computation of body composition it was obtained whilst subjects wore only light underwear. For the exercise tests, however, body mass was determined with subjects wearing the clothes to be worn during the test. A beam balance (Avery Ltd., Model 3306 ABV) with a maximum capacity of 120 kg and accuracy to 50 g was used throughout.

3.5.2. Height.

Subjects were required to stand with their heels together against a metal back plate. Pressure was applied to the mastoid process in order to align the lower left orbit with the upper margin of the external auditory meatus (the Frankfort plane). The horizontal arm of the stadiometer was then brought gently down on the subject's head and the measurement recorded.

3.5.3. Circumferences.

Circumference was measured at a number of pre-specified and marked sites (Weiner & Lourie 1981) using a flexible, steel rule (Stanley, England).

a. Waist. The waist was defined as the mid-point between the lower rib and superior anterior iliac crest.

b. Hip. This measurement was determined on a horizontal line extending around the greater trochanter of each leg.

c. Upper arm. With the arm held relaxed, the measurement was made horizontally at a site mid-way between the tip of the acromion and olecranon processes.

d. Thigh. The subject stood with feet slightly apart and weight evenly distributed on both feet; the tape was placed around the thigh horizontally with the top edge just under the gluteal fold.

e. Calf. The maximum circumference was taken horizontally at a point around the largest part of the calf.

3.5.4. Skinfold thickness.

Skinfold thickness was evaluated using rectangular-jawed calipers with a dial graduated in 0.2 mm divisions (Holtain Ltd., Crymych, U.K.). The surface area of the jaws was 54.9 mm^2, with an applied pressure of 10 g/mm^2. Calibration of the instrument was accomplished by suspending known weights from the jaws in order to establish the resistance of the holding spring. The latter could then be tightened or slackened as appropriate.

At each pre-determined site a standard technique was applied:- the skinfold was isolated between thumb and forefinger, and the jaws of the calipers applied at exactly the marked level. The measurement was read two seconds after the full pressure of the caliper was applied, thus minimizing inaccuracies due to "skin creep". The measurement was then repeated and an average of the two recorded. All measurements were made on the non-dominant side.

The sites measured were:-

a. Biceps. With the arm hanging vertically and relaxed, a point was marked mid-way between the tip of the acromion and olecranon processes. The fold was measured over the

belly of the muscle above the centre of the cubital fossa.

b. Triceps. This site was determined at the same level as the site determined for biceps, but in direct line with the olecranon process.

c. Subscapular. The subscapular skinfold was picked up at a site under the inferior angle of the left scapula at an angle slightly inclined downward and laterally, and in the natural cleavage of the skin.

d. Supra-iliac. This measurement was recorded at two points, firstly, at a site approximately 1 cm above and 2 cms medial to the anterior superior iliac crest and secondly, approximately 3 cms above and 1 cm medial to the same point.

e. Thorax. The thoracic measurement was made on the mid-axillary line, on a level with the xiphoid process.

f. Abdomen. This site was determined at a site 5 cms to the left of, but on the same horizontal level as the umbilicus.

g. Anterior thigh. This was measured on the anterior aspect of the thigh, half-way between the mid-inguinal point and upper border of the patella (with the knee flexed at 90^0).

h. Lateral calf. This measurement was made at the level of the maximal circumference and on the lateral border.

i. Medial calf. This site was measured at the same level as above, but on the medial border.

The skinfold data was used to provide information on

discrete changes at each site, and was also used in the estimation of body density (Womersley & Durnin 1973) and percentage body fat (Siri 1956) (Appendix 3).

3.5.5. Hydrostatic weighing.

The technique used in the estimation of body density by hydrostatic weighing was that developed by Jones and Norgan (1974) and is broadly based upon the principles initially observed by Behnke and coworkers (1942), that is, body density is the product of body mass, divided by body volume:-

$$\text{Density } (kg.m^{-3}) = \text{Mass } (kg) / \text{Volume } (m^3)$$

Mass was determined by weighing the subject in air and body volume by calculating the volume of water displaced. Lung residual volume was measured simultaneously by the nitrogen dilution technique and body volume was corrected accordingly.

As water density is affected by temperature, a correction factor was incorporated in to the calculation of body density. However, in order to minimize this correction and therefore maximize accuracy, as well as for the comfort of subjects, the bath temperature was maintained at 35-36^0 C.

In more detail:-

a. The system. The apparatus used in the measurement of body density consisted of a cylindrical heated water tank containing a light-weight plastic chair suspended in the water from a ceiling hoist. The hoist, in turn was connected to a strain-gauge dynamometer and transducer, through which the underwater body weight was displayed on a digital voltameter (Figure 3:1).

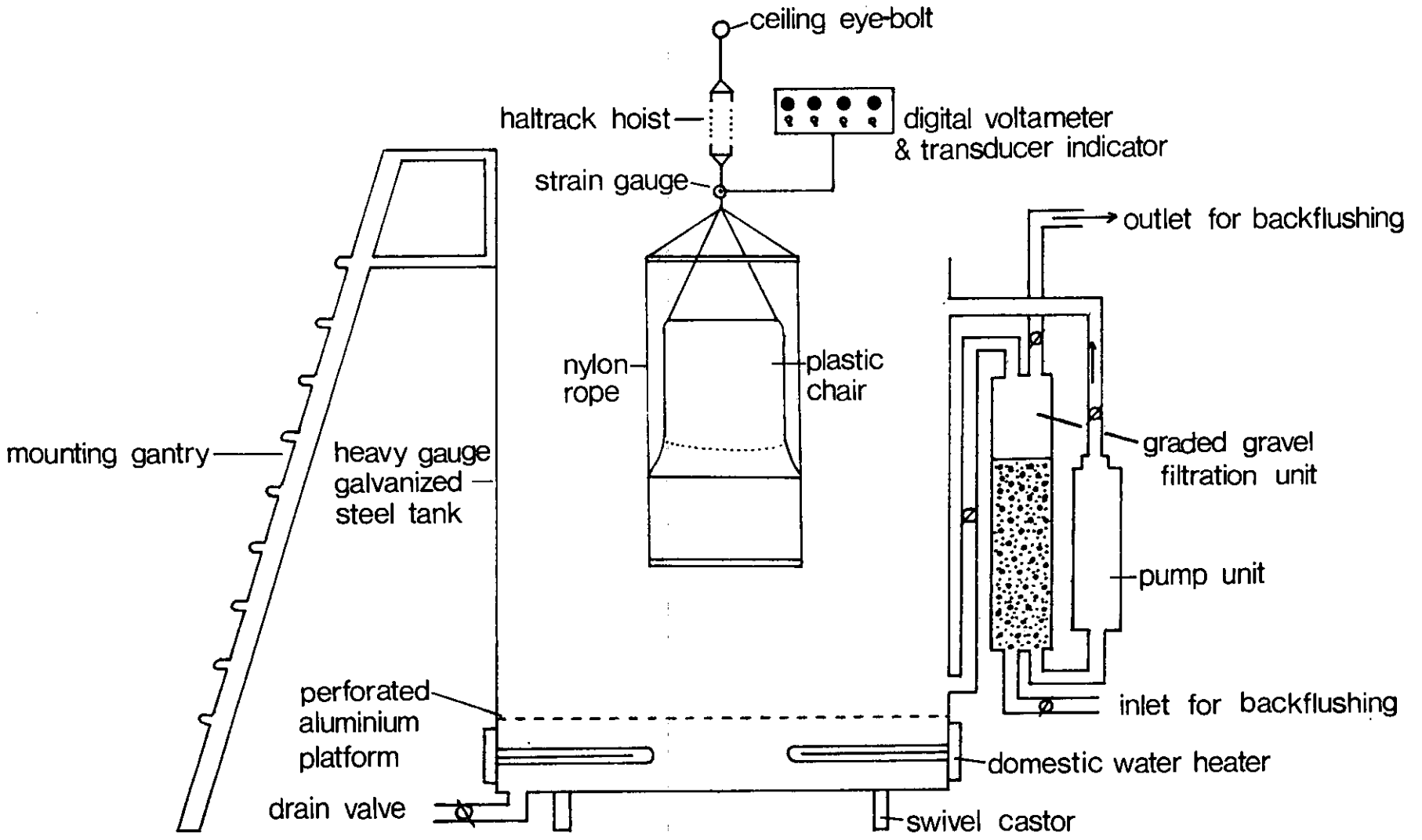


FIGURE 3. Apparatus for hydrostatic weighing.

b. Subject preparation. Subjects were asked to change into a swimming costume and then to shower (in order to minimize body surface fats and hairs). Once completed, body mass was recorded and the subject was then helped in to the tank. When comfortably seated with the lap seat-belt fastened and feet positioned on the footrest the subject's depth in the water was corrected. This adjustment was achieved by raising or lowering the hoist until the subject's chin lay just above the water line.

c. Procedure. Subjects were familiarized with the procedures and completed several practice trials in advance of the actual measurements. When comfortable and confident, a nose-clip and snorkel-tube were provided. Subjects were instructed to bend forwards at the waist and to submerge completely, whilst at the same time making a gentle but maximal expiration. At the point of complete exhalation, the breath was held for approximately 5 seconds and the weight displayed on the voltameter was recorded. A three-way valve was then attached to the top of the snorkel-tube and opened to a four litre anaesthetic bag containing three litres of 100% oxygen. Subjects were instructed to inhale and exhale completely three times over the subsequent nine seconds, the valve was then closed.

The contents of the bag were analysed for carbon dioxide and oxygen (Section 3.2), and the percentage of nitrogen was determined by subtraction. This "three-breath nitrogen dilution" technique (Rahn et al. 1949; Durnin and Rahaman 1967) (Appendix 4) was employed to measure the volume of air remaining in the lungs despite maximal exhalation (the residual volume). The replicability of this nitrogen dilution technique has been tested by a number of authors: Wilmore (1969) r=0.979; Girondola et al. (1977) r=0.95.

Allowing an interval of ten minutes for the re-equilibration of lung gases, the whole procedure was repeated twice more and the mean of the three calculated results for density was accepted as the final value for each subject.

From the derived value for body density it is possible to estimate the percentage of body fat. In order to do so, the density of fat and lean tissue are assumed to be 0.90 and 1.10 $g.cm^3$, respectively (Siri 1956). Percentage body fat was then derived according to the equation of Siri (1956):-

$$\text{Fat \%} = [(4.95/\text{density}) - 4.5] \times 100$$

This assumption has been criticised by a number of authors (MacDougal et al. 1983; Wilmore 1983), since athletes have been shown to have denser bones and muscles than their age-related inactive contemporaries (MacDougal et al. 1983), which could lead to an overestimation of body fat. Similarly, a lower than expected bone density may lead to an underestimation of body fat. These observations have important implications for measurements made before and after a long-term programme of exercise training.

3.6. Blood sampling.

In order to arterialise capillary blood the whole hand was immersed in warm water. Using an Auto-clix automatic lancet (Boehringer Mannheim, U.K., Ltd.) the skin was punctured and two samples of blood were collected using calibrated micro-pipettes (either 20μl or 25μl) (Acupette Pipettes; Scientific Industries Ltd.). The samples were immediately deproteinised in 10 times the sample volume of 2.5% (w/v) perchloric acid (either 200μl or 250μl) (Appendix 5). The same procedure was also employed whilst

the subjects were exercising. Samples were then centrifuged (Eppendorf centrifuge: model 5414) at 12,000 rpm for 3-4 minutes and stored at -20^0 C prior to analysis.

Five or 10 ml volumes of blood were obtained by venepuncture from a vein in the cubital fossa after subjects had fasted overnight. The fasting state was stipulated since evidence exists to suggest that whilst the concentration of TC is largely unaffected (Demacker et al. 1982; Costongs et al. 1985; Rotterdam et al. 1987), triacylglycols increase in the non-fasting state (Olsson & Carlson 1975). Thus in an attempt to minimize this latter effect and that of biological variation (Appendix 6) all samples were collected between 7 and 10 a.m.

A number of investigations have been completed into the intra-person fluctuations of serum TC. Biological variation is the day-to-day variation in an individual even though diet, drug therapy and other recognised influences are kept constant (Von Schenck & Olsson 1990). The results of studies conducted by Hegsted and Nicolosi (1987), Jacobs and Barrett-Connor (1982), Natelson and colleagues (1988) and Bachorik (1985) have shown biological variation to account for a fluctuation of 5-10% in the concentration serum TC. A similar degree of change was also derived from the observations reported in Appendix 6. As a consequence, where possible the mean of two samples collected less than 5 days apart are reported in an attempt to provide a more representative estimate than might be expected from a single sample.

In order to minimize variation resulting from the collection procedure, all samples were obtained with subjects in the supine position. As well as reducing the possibility of subjects fainting, evidence suggests that postural changes cause a redistribution of water between

the blood and tissues, resulting in fluctuations of haematocrit, cholesterol and triacylglycols (Tan et al. 1973), and also of all blood-borne proteins or substances bound to protein (Dixon & Paterson 1978).

Once obtained, the samples were dispensed into plastic tubes (Sarstedt Z/10) and allowed to clot for 30 minutes prior to centrifugation (6,000 rpm for 20 minutes) for the separation of serum. The serum was then filtered (Sarstedt serum/plasma filter No. 53-421). Aliquots were immediately stored at -70^0C for subsequent analysis. The remaining aliquots were stored at -4^0 C for a maximum of three days (Appendix 7). Within this period HDL_2 was precipitated out using sodium heparin (Gaez et al. 1982), and VLDL and LDL with manganese chloride (Appendix 11). The supernatant derived from this procedure was then removed and also stored at -70^0 C.

3.7. Blood biochemistry.

Full details of the methods of analysis and assays employed are included in the appropriate appendices, however, a brief outline is included below:-

3.7.1. Blood lactate concentration.

Blood lactate concentration was determined by the fluorimetric analysis of supernatant after the precipitation of protein by 10 x volume of 2.5% perchloric acid, a modification of the enzymatic method described by Maughan (1982). A Perkin-Elmer fluorimeter (Model 1000M) was used in early studies, but was later replaced by a Locarte fluorimeter (Locarte, London. Model 8-9). The coefficient of variation for this assay was less than 2%. The standards used were made from 1.0 M sodium L-lactate stock solution. To ensure quality control, old and new standards were run against one another, prior to new standards being used in an analysis (Appendix 5).

3.7.2. Blood lipids.

Blood lipids (total cholesterol, lipoproteins and triacylglycerol) were determined using commercially available enzymatic kits, standards and reference solutions (Boehringer Mannheim, U.K., Ltd.).

The stored samples of sera were analysed for total cholesterol (Siedel et al. 1981; Stahler et al. 1977; Trinder et al. 1969) (Appendix 8) and for triacyglycerol (Trinder et al. 1969) (Appendix 9). The samples of supernatant resulting from the precipitation of LDL and VLDL were used in the determination of HDL_3 (Appendix 11). This procedure was employed in the investigations reported in Chapters 5 and 7.

In the remaining studies, plasma rather than serum samples were analysed. Cholesterol was determined in whole plasma and HDL in the supernatant after the precipitation of LDL and VLDL using magnesium chloride and phosphotungstate (Appendices 8 & 12).

Whilst the analysis for HDL should ideally be completed in fresh samples, within hours of collection, this is not realistic. Results of investigations in to the effects of storage have revealed that little deterioration of samples occurs in the first 1-2 months. The reports consistently report an inverse relationship with time, that is, samples high in HDL decrease, samples low in HDL increase, therefore "since the changes were in opposite directions, the group means tended to be preserved" (Bachorik et al. 1980; Bachorik et al. 1982; Demacker & Jansen 1983).

3.8. Dietary analysis.

The major experimental studies reported in this thesis make considerable use of the dietary record technique of analysis which enabled the nutritional content of diets

to be quantified.

The weight of each food item was determined to the nearest gramme using digital scales (Soehnle, model no.8000). These had a maximum capacity of 1000 g. Between 0-64 g the accuracy was to one gramme, above this level the accuracy was two grammes.

Individuals were provided with sheets on which to record their daily food intake, a sheet of concise instructions and an example sheet (Appendix 13). Subjects were then instructed in the use of the scales and the method of completing the forms. Each subject then had a day on which to practise the procedure and identify any problems prior to starting the seven consecutive days of weighing.

The weight, along with a full description of items consumed over the period was recorded, along with the weight of leftovers, this is in accordance with the technique suggested by Marr (1971). For meals eaten out of the home, subjects were asked to describe in full the items consumed and to provide approximate weights/sizes of portions. With pre-prepared meals, subjects were asked to include the nutritional information supplied by the manufacturer.

Each food item was coded in accordance with the index of foods in the MAFF/MRC Food Composition tables (Paul & Southgate 1978) as outlined in McCance and Widdowson (4th edition). The code and weight of each food item was then used to provide an analysis of energy, nutrients, vitamins and minerals. One of two computerized food composition programmes were used. In early studies the University main-frame machine (ICL Prime 1900S) was used with the "OSIRIS" programme developed by the Queen Elizabeth College, University of London. In later studies the analysis was completed using a desk-top PC (Viglen II

HDM) with the "Microdiet" programme developed by Salford University.

3.9. Psychological measurements.

Evidence exists to suggest a positive association between habitual physical activity and positive changes in psychological well-being (Byrd 1963; Pauly et al. 1982; Wood 1977). In order to assess this relationship within the context of the present series of studies, a battery of self-report measures were completed (Appendix 14).

a. Self-esteem. A 10 item scale of self-esteem was employed (Pearlin et al. 1981) which included questions relating to self-concept and confidence, for examples: "I feel that I have a number of good qualities" and "I feel that I do not have much to be proud of."

The possible scores range from a minimum of 10 to a maximum of 40, with a higher total considered to reflect a more positive and self-confident indvidual.

b. General Health. The 12 or 28 item versions of the General Health Questionnaire (GHQ) were employed to discern alterations in general well-being. The GHQ was compiled for the assessment of non psychotic psychiatric disorder in groups of non-clinical populations (Goldberg & Hillier 1979).

The GHQ includes questions relating to the individuals recent ability to concentrate, to sleep, to cope with daily stress and decision making. The possible scores range from 0 to 36, with lower scores considered reflective of a lesser degree of psychological stress.

c. State-trait anxiety inventory (STAI). The 20 item trait scale of the STAI was used in the assessment of trait anxiety (Spielberger et al. 1970). Trait anxiety

refers to the general level of anxiety (i.e., how the subject usually feels), in contrast to state anxiety which refers to the experience of anxiety and tension at the moment the questionnaire is completed (Blumenthal et al. 1982). Higher scores are related to higher levels of anxiety.

d. Profile of mood states (POMS). The 65 item inventory allows an assessment of the moods experienced by the individual during the week prior to completing the questionnaire. Six different aspects of mood and well-being are represented by a series of adjectives which the individual must rate accordingly. The parameters identified included: tension, depression/ dejection, anger/hostility, vigour, fatigue and confusion (McNair et al. 1971).

3.10. Physical activity assessment.

In order to assess the degree of exercise participation, a Physical Activity Questionnare (PAQ) (Appendix 15). This included questions relating to recreation and leisure activities, to type of training (applicable to Chapter 4), to occupation and to home and travel. Higher scores were associated with a more active lifestyle.

3.11. Statistical analysis.

Specific descriptions of the statistics employed are presented in the relevant experimental chapters. All the methods used are described by Cohen and Holliday (1982). Student's t-test for independent means was used for testing the significance of difference between two means. Two-way analysis of variance with repeated measures (ANOVA) was used to test for differences between the means of several groups of scores. Tukey post hoc tests were used to identify significant differences between means which had been identified by ANOVA. The Pearsons Product Moment correlation was used to examine the

relationships between variables. In tables and figures, values shown are means, along with the standard deviation from the mean, or standard error of the mean as appropriate. Throughout, the level of confidence adopted was 5 percent.

4. THE POTENTIAL OF BRISK WALKING FOR IMPROVING ENDURANCE FITNESS IN MIDDLE-AGED WOMEN.

4.1. Introduction.

Evidence was previously presented for the existence of a positive relationship between habitual endurance activity and the maintenance, or improvement, of functional capacity (Hagberg 1987; Hagberg et al. 1989). Furthermore, it has been observed that as a consequence of these favourable physiological adaptations the performance of routine physical tasks is possible with less fatigue (Bouchard et al. 1975; Cureton 1969; Davies & Knibbs 1971; Wenger & MacNab 1975). In addition, a number of possible health benefits are associated with habitual exercise participation. Of particular importance is a reduction of the risk for the development of diseases such as CHD, obesity and adult onset diabetes (Schultz 1980).

The potential advantages associated with exercise have however, largely been identified from studies of male subjects and from the observation of programmes of relatively high intensity activities (Saltin et al. 1968; Gaesser & Rich 1984; Wood et al. 1976). There is therefore a need to investigate the potential of low intensity exercise to confer the health benefits associated with high intensity exercise.

The aim of the present study, therefore, was to investigate the potential of brisk walking to improve the functional capacity of a group of sedentary, middle-aged women. This procedure was completed as a preliminary to the development of a long-term, low intensity training programme.

4.2. Methods.

(i) Subjects.

Fifty two women from the town and university communities volunteered to take part in the study. The women, aged between 30 and 62 years. had not participated in any regular, or structured form of exercise for the year prior to the onset of the investigation. Thus for the purpose of the study the group was considered to be sedentary.

(ii) Protocol.

Each subject completed two. one mile walks around an athletics track. During the first walk, a maximal pace was adopted. The second walk, which was completed on a separate occasion, was performed at a self-selected submaximal. but brisk pace. Subsequently, the pace adopted by each individual during the submaximal track performance was replicated in the laboratory. Subjects then walked for a further mile on the treadmill at the pace which she had previously selected as brisk (Chapter 3).

Heart rate was routinely measured throughout the walks (Chapter 3:4). During the treadmill walk, expired gas samples were collected at half a mile and immediately prior to the completion of the walk. The samples were subsequently analyzed for the content of oxygen and carbon dioxide (Chapter 3:2). Capillary blood samples were obtained at rest and at the end of the walk. and subsequently analysed for blood lactate (Appendix 5).

In addition to the walking tasks, all subjects completed an incremental step-test. Using the oxygen consumption and heart rate values evoked during this test, maximal oxygen uptake was predicted (Chapter 3:3).

Measurements were also made of body mass, skinfold thickness, waist and hip circumferences, and body density measurements, in accordance with the previously described methodologies (Chapter 3:5).

All results are presented as the mean±standard deviation (SD). The relationship between parameters was tested using the Pearson Product Moment Correlation coefficient.

4.3. Results.

Details of the physical characteristics of the subjects, including age, body mass and height are reported in Table 4:1. Table 4:2 includes measurements describing the amount and distribution of body fat, that is body mass index, body density, sum of four skinfold thicknesses and the ratio of the circumference of the waist and hip.

Information relating to the track and treadmill walking tasks is included in Table 4:3. The average heart rate elicited during the submaximal track and treadmill walks (127±15 $bt.min^{-1}$ and 131±18 $bt.min^{-1}$ respectively) was not found to be significantly different. Furthermore, application of the Pearson Product Moment Correlation technique revealed a close relationship between the two observations ($r=0.779$, $P<0.01$).

A low but significant correlation was derived for the relationship between predicted $\dot{V}O_2$ max and maximal pace elicited on the track ($r=0.376$, $P<0.01$), and between $\dot{V}O_2$ max and submaximal walking pace ($r=0.290$, $P<0.05$).

Furthermore, a relationship was also identified between age and $\dot{V}O_2$max ($r=-0.312$, $P<0.05$) and between submaximal pace and measurements of body fatness ($r=0.485$, $P<0.01$; $r=-0.528$, $P<0.01$ for body density and

Table 4:1. Physical characteristics of 52 women enrolled in the present study.

	$\bar{x}$	SD	Range
Age (yr)	44.2	8.6	30 - 62
Body mass (kg)	62.5	8.8	48.8 - 83.7
Height (m)	1.62	0.05	1.46 - 1.73

Table 4:2. Indices of fatness and fat distribution: body mass index (BMI), body density, sum of 4 skinfold thicknesses and the ratio of waist and hip circumferences (W:H). (n=52, †n=49).

	$\bar{x}$	SD	Range
BMI ($kg.m^{-2}$)	23.8	3.3	18.0 - 32.3
†Body density ($kg.m^{-3}$)	1023	16	985 - 1063
Sum 4 skinfolds (mm)	59	19	32 - 101
W:H	0.79	0.06	0.68 - 0.95

Table 4:3. Responses to a one mile track (n=52) and treadmill walk (n=51).

Parameter	$\bar{x}$	SD	Range
Maximal track walk			
Time (mins)	14.20	1.10	12.43 - 18.05
Speed ($m.s^{-1}$)	1.90	0.14	1.49 - 2.16
Heart rate ($bt.min^{-1}$)	143	18	102 - 179
Submaximal track walk			
Time (mins)	15.78	1.33	12.46 - 19.00
Speed ($m.s^{-1}$)	1.71	0.24	1.39 - 2.21
Heart rate ($bt.min^{-1}$)	127	15	100 - 172
Submaximal treadmill walk			
Heart rate ($bt.min^{-1}$)	131	18	96 - 164
Blood lactate concentration at end of the walk ($mmol.l^{-1}$)	2.08	1.06	0.69 - 4.39

for the sum of four skinfolds respectively).

4.4. Discussion.

The physical characteristics of the group, that is, age, body mass and height are included in Table 4:1. Additional information concerning the results of measurements made to indicate the amount and distribution of body fat are presented in Table 4:2. For example, BMI or Quetelet index, was calculated using the equation:

$$BMI = \text{body mass (kg)} / \text{height}^2 \text{ (m)}.$$

Body mass index is therefore a description of the relationship between body mass and height. The index is graded and for simplicity the same grades apply to both men and to women. Thus an index of less than 20 $kg.m^{-2}$ is associated with leanness, 20-24.9 $kg.m^{-2}$ is considered 'ideal', 25-29.9 $kg.m^{-2}$ represents overweight, 30-40 $kg.m^{-2}$ is obese, and over 40 $kg.m^{-2}$ is considered severely obese (Pacy et al. 1986).

The average BMI derived for the present group of women was 23.8 $kg.m^{-2}$. In accordance with the previously outlined classification structured by Pacy and colleagues (1986), it is proposed that the group as a whole lies within the grade designated as 'ideal'. This observation is consistent with the recently published report of a survey of similarly aged British women, for whom a figure of 23 $kg.m^{-2}$ was derived (Rosenbaum et al. 1985). It is therefore suggested that the subjects involved in this study are representative of the typical British female population for the relationship between height and weight. Furthermore, an inspection of the range of BMI derived for the group (18.0 to 32.3 $kg.m^{-2}$) indicates that the group also includes individuals at both extremes of

the normal distribution for this index.

In addition to the distribution of body fatness, functional capacity was also assessed. Maximal oxygen uptake was predicted from the steady-state heart rate and oxygen uptake during an incremental step test. Application of this technique resulted in a predicted average for the group of 27.3±4.6 $ml.kg.^{-1}min^{-1}$. Jetté and coworkers (1988) reported a similar capacity, that is 26.6±3.6 $ml.kg^{-1}min^{-1}$, for Canadian women of the same age. It would therefore appear that the group of women employed in the present study are typical of similar populations of the same age. Furthermore, the range of predicted $\dot{V}O_2$ max values, from 18.7 $ml.kg.^{-1}min^{-1}$ to 44.2 $ml.kg.^{-1}min^{-1}$, confirms that this group included women at both ends of the normal distribution for this parameter and as such the group appears to be a representative sample of the population.

In order to ascertain the potential of brisk walking to increase the functional capacity of these subjects, a one mile submaximal track walk was completed. The speed and duration of each individual's performance was recorded and subsequently replicated in the laboratory, where the same distance was completed on a motorized treadmill. No significant difference was determined between the average heart rate elicited during the track and treadmill walks (Table 4:3). In addition, a relationship was found to exist between the two rates ($r=0.779$, $P<0.01$). As a consequence, the treadmill performance was considered to be an appropriate means by which to investigate the physiological responses to brisk walking.

The major question surrounding the effectiveness of brisk walking to increase $\dot{V}O_2$ max relates to the intensity of the activity. The American College of Sports Medicine (ACSM) has drawn up guidelines with regard to intensity for the

attainment of improved functional capacity. On the basis of experimental evidence (Hollman & Venrath 1962; Crews & Roberts 1976; Gledhill & Eynon 1972), it was suggested that the exercise should be sufficient to attain a minimal threshold level for both heart rate and for the percentage of $\dot{V}O_2$ max:

> "The minimal threshold level for improvement in maximal oxygen uptake is approximately 60% of the maximal heart rate reserve (50% of $\dot{V}O_2$max)".
> (ACSM 1986).

Thus, using the observations made by Hollman and Venrath (1962), and Karvonen and coworkers (1957), ACSM suggested an average heart rate target of 110-120 $bt.min^{-1}$ or in excess of 60 percent of the maximal heart rate, for older or sedentary individuals.

When considered in relation to the present study, it was observed that the intensity of the submaximal walk was sufficient to provoke an average heart rate of 131±15 $bt.min^{-1}$. Furthermore, using the equation:

$$(210-(age \times 0.65))$$

the average age-related maximal heart rate was estimated as 181±6 $bt.min^{-1}$ (range 170 to 191 $bt.min^{-1}$). Thus the average percentage of the maximal heart rate elicited during the submaximal walk was calculated as 70±8% (range 59 to 92%). These results therefore suggest that the intensity adopted on the track during the submaximal walk was sufficient to attain the minimal thresholds identified by ACSM for heart rate. This observation remained whether heart rate was reported as an absolute term or when considered relative to age.

The results of the submaximal walk therefore indicate

that 94 percent of the present group were able to adopt a pace sufficient to elicit a heart rate accounting for 60 percent, or more, of the predicted age-related maximum (Figure 4:1). This observation is supported by the results reported for similar investigations. For example, Pocari and coworkers (1987) reported that 91 percent of all women who completed a one mile brisk but submaximal walk, did so with a heart rate which exceeded that stipulated by ACSM (1986). Together these results therefore suggest that brisk walking might be a sufficient stimulus to provoke an increase in $\dot{V}O_2$ max for over 90 percent of this sedentary, middle-aged female population.

However, in addition to the minimal heart rate threshold, the ACSM also suggested that the form of exercise adopted during training should exceed a minimal relative exercise intensity i.e. percentage of maximal oxygen uptake. In accordance with this, gas samples expired during the treadmill walk were analyzed for the content of carbon dioxide and oxygen. The results of this analysis revealed an average oxygen cost for the walk of 16.2±3.2 $ml.kg^{-1}$ min^{-1}. When expressed as a percentage of the predicted $\dot{V}O_2$ max (27.3±4.6 $ml.kg^{-1}.min^{-1}$) it was shown that an average of 59±14% of this capacity was utilized. Once again therefore the minimal threshold prescribed by the ACSM was exceeded. Furthermore, observation of the group range (35 to 92% $\dot{V}O_2$ max) suggests that 75 percent of this group were able to adopt an intensity of walking sufficient to attain the threshold for improvement (Figure 4:2). Together the observations made for heart rate and oxygen consumption indicate that this form of exercise is of sufficient intensity to increase the functional capacity of between 75 and 90 percent of these individuals dependent upon the index adopted.

However for a smaller number of individuals (25% when

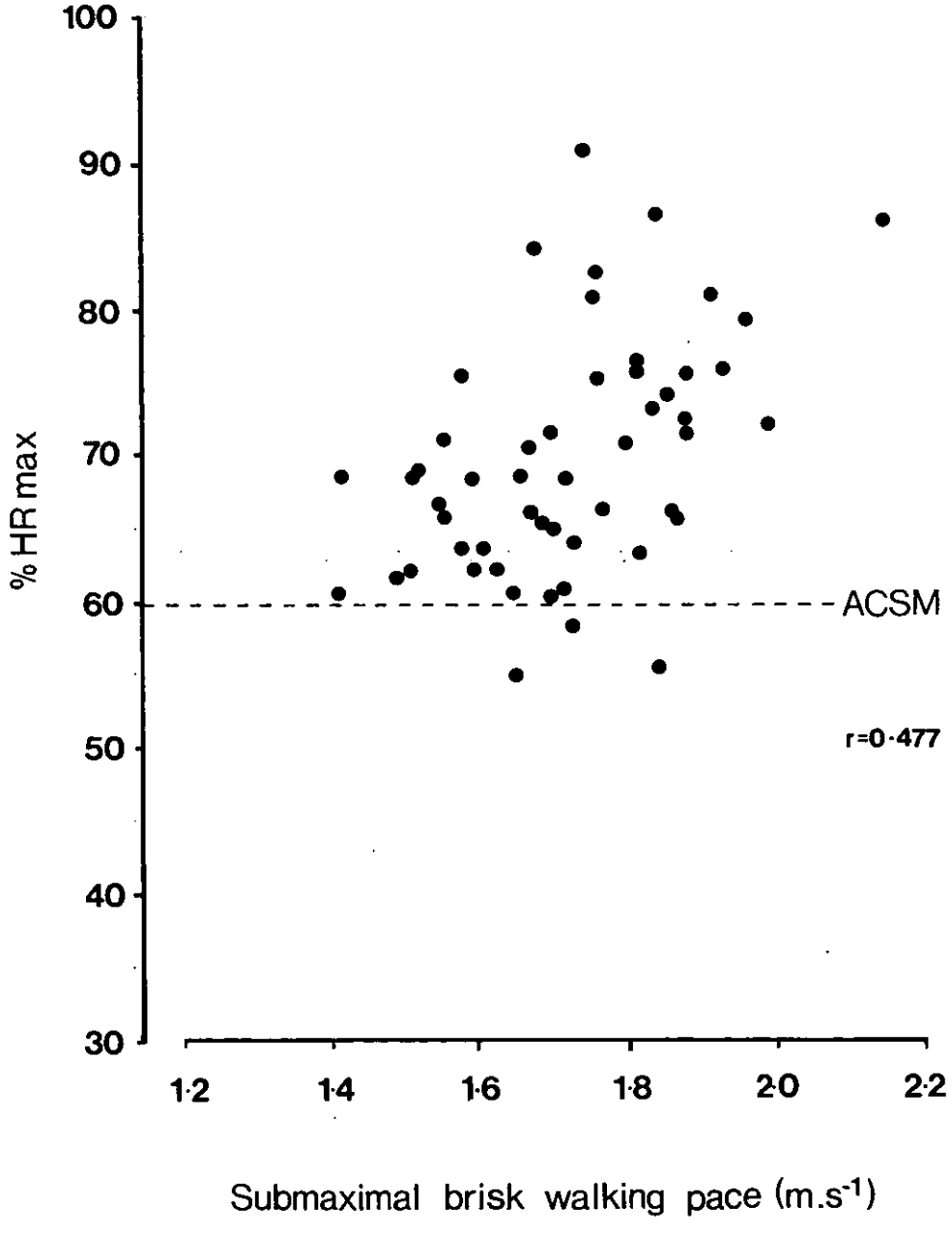


FIGURE 4·1 The relationship between submaximal pace and %HRmax

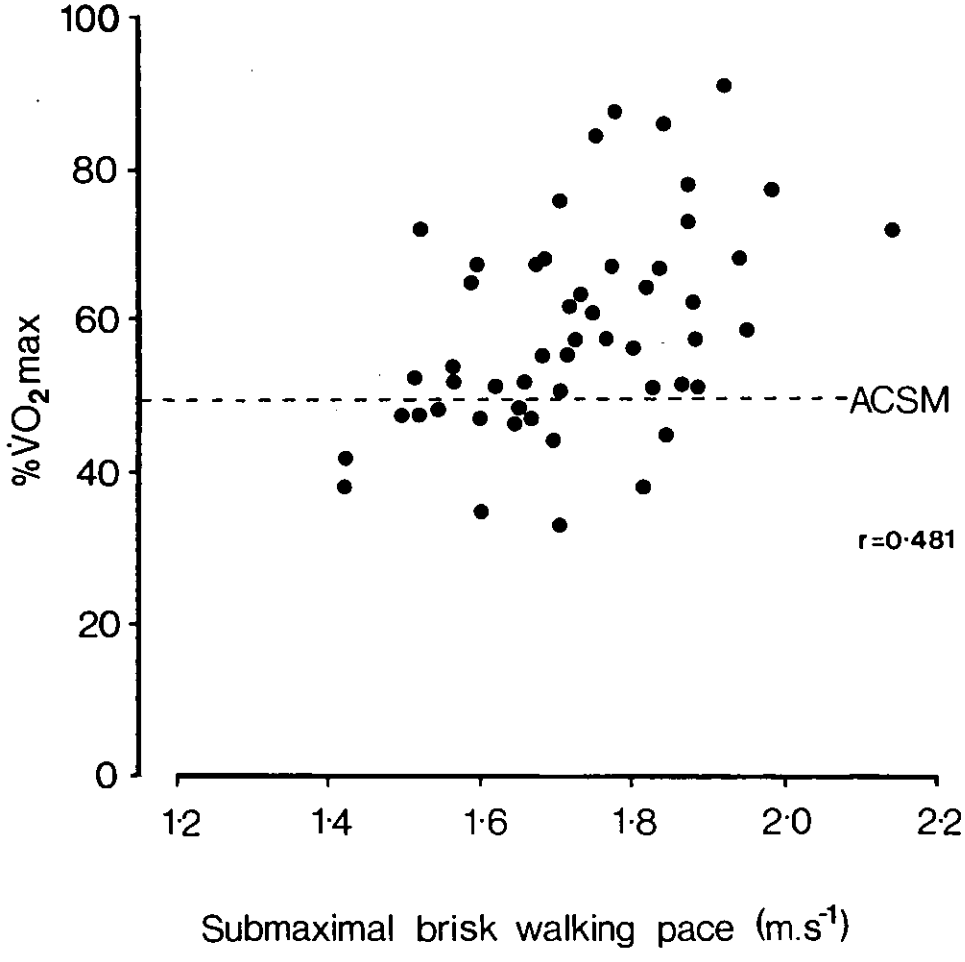


FIGURE 4·2 The relationship between submaximal pace and $\%\dot{V}O_2$max utilized

considered relative to oxygen consumption) the intensity at which the walk was completed was insufficient to attain the assumed threshold. Similar inconsistencies between individuals were also observed by Cunningham and coworkers (1982) and were the basis of an investigation into the possible determinants of self-selected walking speed. The results of the study which involved men aged between 19 and 66 years, indicated that walking speed was associated with $\dot{V}O_2$max, independent of age. This observation is contrary to that reported by Bassey (1978), who favoured the effects of ageing, rather than $\dot{V}O_2$ max, as the most important determinants.

Evidence derived from the present study however, appears to be supportive of a more dominant role for $\dot{V}O_2$ max, and as such, appears more consistent with the observations reported by Cunningham and colleagues (1982). For example, a small but significant inverse relationship was identified between age and functional capacity ($r=-0.312$, $P<0.05$) (Figure 4:3). A similar finding to which was also made by Robinson (1973), Dehn and Bruce (1972), and by Seals and coworkers (1984). Secondly, predicted $\dot{V}O_2$ max was positively related to both the self-selected brisk pace ($r=0.290$, $P<0.05$) (Figure 4:4) and to the maximal pace ($r=0.376$, $P<0.01$) adopted during the one mile walks. However, no significant relationship was determined for the correlation between pace and age.

From these results, it would therefore appear that the submaximal brisk pace adopted was determined at least in part by functional capacity. Since $\dot{V}O_2$ max has been seen to be inversely related to body fatness (Hagberg 1987), the observation of a positive relationship between submaximal pace and body density ($r=0.485$, $P<0.01$) would appear to be further evidence in support of functional capacity. Furthermore, a negative relationship was also identified between submaximal pace and the sum of four skinfold

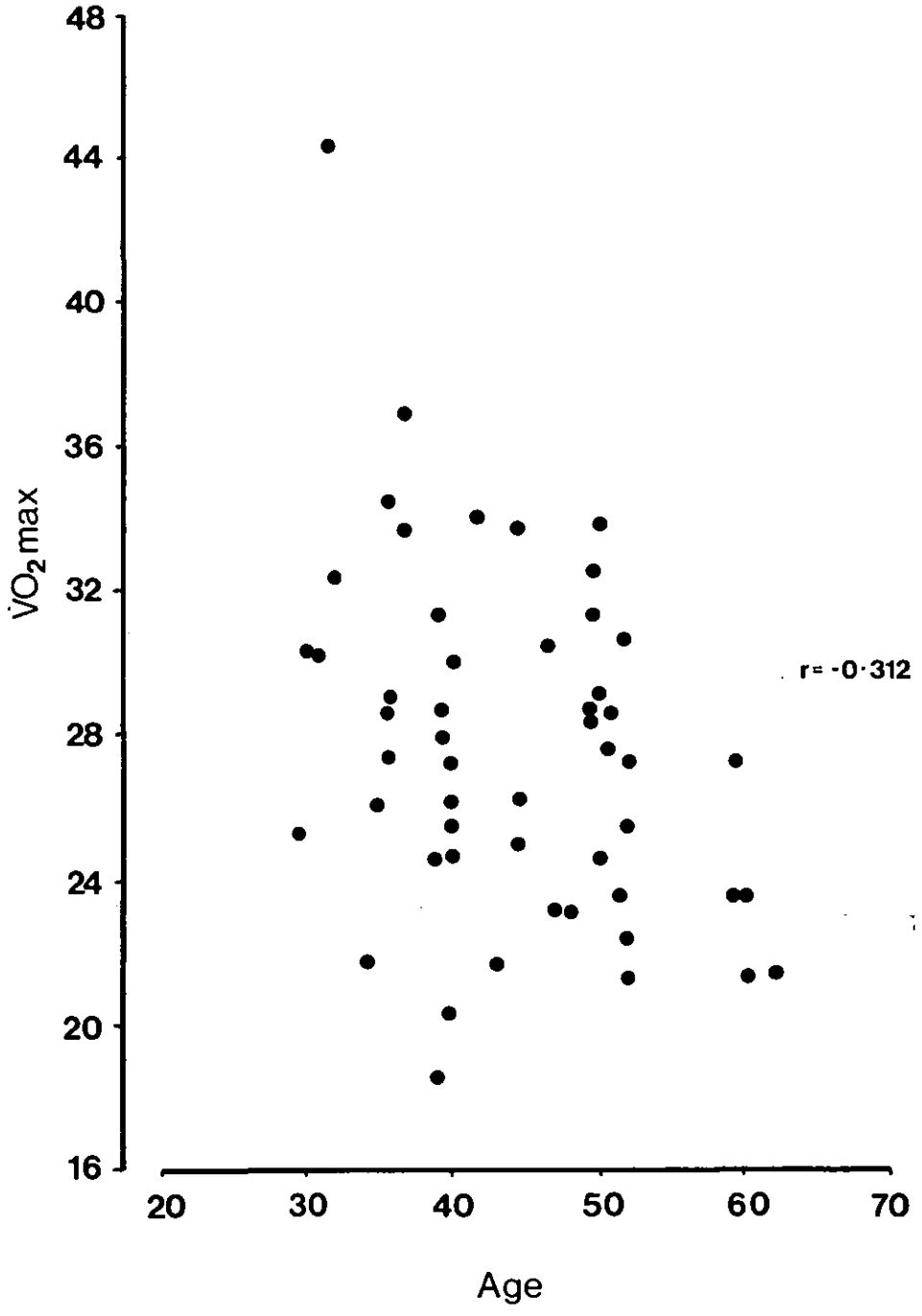


FIGURE 4:3 The relationship between maximal oxygen uptake ($\dot{V}O_2$max) and age.

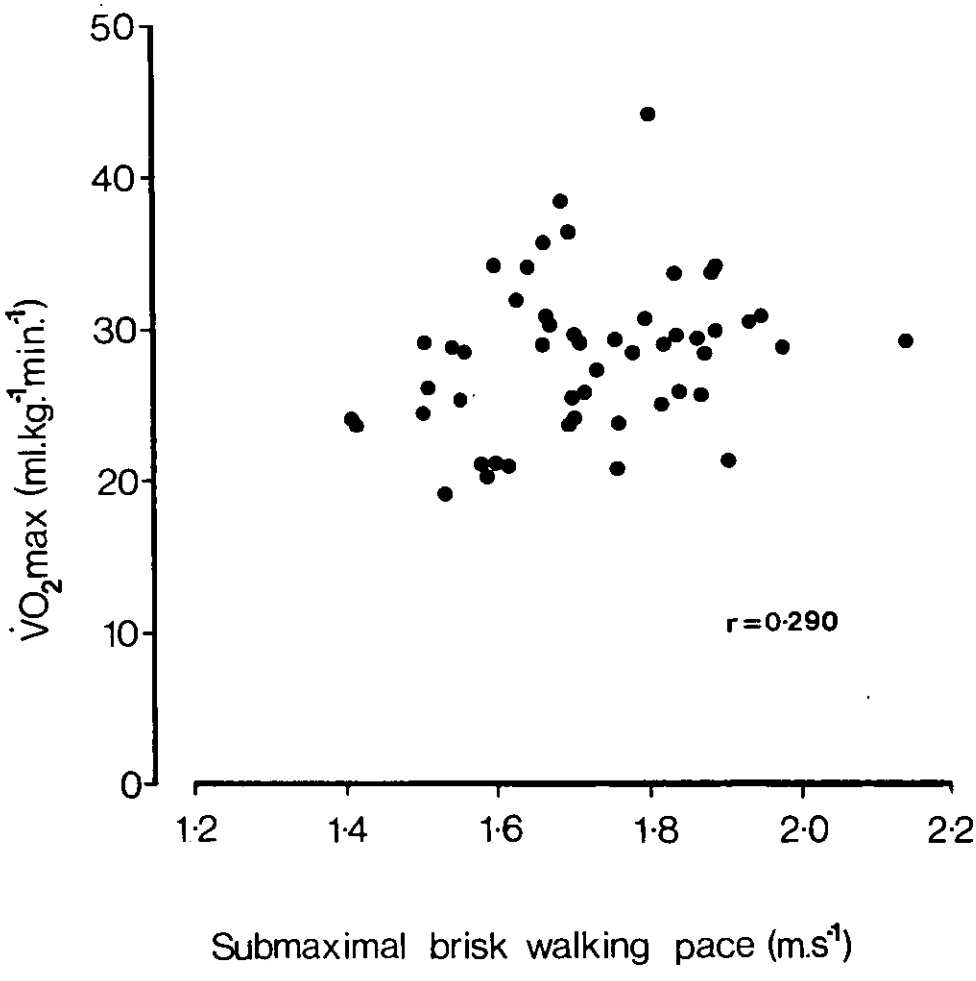


FIGURE 4·4 The relationship between submaximal pace and predicted $\dot{V}O_2$max

thicknesses (r-0.528, P<0.01). Similar correlations between these parameters and maximal pace were also derived. However, these indices of body fatness did not appear to be significantly related to age.

In addition to heart rate, oxygen consumption and functional capacity, blood lactate concentration at the end of the one mile treadmill walk was also assessed. Recent studies have led to the identification of a relationship between $\dot{V}O_2$ at a reference blood lactate concentration and endurance (Davis et al. 1976, 1979; Farrell et al. 1979; Ivy et al. 1980; Reinhard et al. 1979; Wasserman et al. 1973; Yoshida 1984, 1986). The results suggest that with endurance training, exercise requiring the same $\dot{V}O_2$ can be performed with a lower concentration of blood lactate. Alternatively, a higher work intensity is required to attain the same reference blood lactate concentration, for example 2 $mmol.l^{-1}$ (Jacobs et al. 1981; Kindermann et al. 1979; LaFontaine et al. 1981; Londeree & Ames 1975; Mader & Hollmann 1977; Rusko et al. 1980). Capillary blood samples obtained at the end of the walk were found to have an average lactate concentration of 2.08 $mmol.l^{-1}$ (Table 4:3). Evidence from previous studies (Kindermann et al. 1979; Yoshida 1984) suggests that exercise performed at an intensity sufficient to provoke such a low concentration of blood lactate might be maintained over a relatively longer duration than an activity resulting in higher lactate concentrations. The present results therefore indicate that brisk walking might be maintained for a prolonged period prior to the onset of fatigue. However, as was reported for both heart rate and for oxygen consumption, the range of the results (0.69 to 4.39 $mmol.l^{-1}$) reveals differences between individuals within the group. Thus for a small number with relatively high lactate concentrations the onset of fatigue might be expected to occur relatively rapidly and as such would therefore

limit exercise duration.

4.5. Conclusions.

The self-selected pace adopted during a one mile brisk walk, was probably sufficient to provoke adaptations which suggest that walking is a realistic training activity for the majority of these sedentary women. The minimal thresholds for both oxygen consumption and heart rate, as proposed by the ACSM (1986), were exceeded by the mean values of the group. However; it is recognised that for a some of these individuals, the adopted pace was insufficiently provocative.

Furthermore, the average lactate concentration measured in capillary blood samples obtained at the end of the walk also suggest that the exercise might be maintained for a relatively long duration prior to fatigue.

Brisk walking would therefore appear to be an appropriate form of exercise to bring about favourable changes in physiological response to exercise for the majority of this group of sedentary middle-aged women.

5. A COMPARISON OF BLOOD LIPIDS IN YOUNG ADULTS AND MIDDLE-AGED WOMEN WITH DIFFERENT HABITUAL EXERCISE BACKGROUNDS.

5.1 Introduction.

A large body of evidence exists to support the suggestion that blood lipid profiles differ between active and sedentary individuals. Individuals who regularly exercise have been shown to have higher HDL-C concentrations when compared with their less active contemporaries (Williams et al. 1986; Thompson et al. 1983; Hartung et al. 1980; 1983). This is an important observation, since the concentration of HDL-C has also been found to be inversely correlated with coronary risk (Miller & Miller 1979; Miller et al. 1977). The possibility might therefore exist for an indirect link between regular exercise and reduced risk for CHD development (Morris et al. 1976; Brownell et al. 1982; Paffenbarger et al. 1978; McCunney 1987).

Much of the evidence in support of these proposals, however, has been derived from studies of middle-aged males (Wood et al. 1977; Adner & Castelli 1980; Martin et al. 1977) involved in high intensity activities, such as running, skiing or skating (Hartung et al. 1980; Tsopanakis et al. 1986; Enger et al. 1977).

Whilst these investigations have provided invaluable information, it is necessary to distinguish the consequences of types of exercise more attainable and more appropriate to a larger proportion of the population. It was the aim of the present study therefore, to investigate the effects of different types of habitual exercise on the metabolism of lipids and lipoproteins in groups of young and middle-aged adults.

The study consists of two parts. The first includes a comparison of young adults differing with regard to training, that is, sprint, endurance and untrained individuals. The second part is a comparison of three groups of middle-aged women, that is, trained endurance runners, members of a local rambling club and individuals who took part in no regular form of exercise (sedentary).

5.2. Methodology.

All subjects arrived at the laboratory having fasted overnight; a 5 or 10 ml blood sample was obtained by venopuncture, in accordance with the procedure previously described (Chapter 3:6).

Subjects also completed food diaries for seven consecutive days. Analysis of these records provided accurate information with regard to average daily energy intake and the consumption of macro-nutrients. The methodology adopted is described in Chapter 3:8.

Statistical analysis of the differences between group means was evaluated using analysis of variance and Student's t-test for independent means.

5.2.1. A comparison of young adults.

(i) Subjects.

Thirty six subjects (18 males and 18 females) volunteered to participate in the first part of the study. Of these, 12 were sprint-trained (S-T), 12 endurance-trained (E-T) and 12 completed no regular or structured exercise and were termed untrained (U-T). Each of these training-based groups was comprised of six males and six females (Table 5:1). All the trained individuals competed in their particular sport to University club level or higher. The remaining group was comprised of individuals who took no part in regular exercise or training, and for the purpose of the present study were termed untrained.

(ii) Protocol.

The venous blood sample was dispensed into a tube containing diaminoethanetetra-acetic acid (EDTA), centrifuged and the plasma removed. One aliquot was immediately stored at $-20^{0}C$ and subsequently analysed for plasma TC and after tungsten-magnesium precipitation, for

plasma HDL-C (Appendix 8 & 12 respectively).

The second aliquot was assayed within three days of collection using an electrophoretic method for the fractionation of α-, pre-ß and ß-lipoproteins, which are closely associated with HDL, VLDL and LDL respectively.

5.2.2. A comparison of middle-aged women.

(i) Subjects.

Thirty-six women from the town and university communities volunteered to participate in the second part of the study. Of these, twelve were trained endurance runners (R), all being members of local running clubs; twelve pursued walking and rambling as their major leisure activity (W) and the remaining twelve volunteers were involved in no regular or structured form of exercise and for the purpose of this study were described as sedentary controls (S).

(ii) Protocol.

The venous blood sample was dispensed into a heparinised tube, left for 30 minutes to coagulate and then centrifuged. The serum was removed, dispensed into aliquots and treated in accordance with the previously described methodology. The samples were subsequently analysed for TC, HDL-C, HDL_3 and Tgs (Appendices 8-11).

A second venopuncture was obtained within five days of the first and was treated similarly. The results of the analyses on the two samples were averaged in order to derive the end result.

A physical activity questionnaire was completed by each individual in order to determine the amount and frequency of exercise performed (Appendix 15).

5.3. Results.

5.3.1. Results of the comparison of young adult males and females.

The results of the investigation are presented in three sections:

a. differences between males and females

b. differences between sprint-trained, endurance-trained and untrained groups, and

c. differences between sprint-trained, endurance-trained and untrained groups of males and females.

a. Differences between males and females.

A description of the physical characteristics of the groups (i.e. all males and all females) is included in Table 5:1. Differences for body mass were found between the two groups (73.33±2.08 kg cf. 59.23±1.62 kg, for male and female groups respectively, $P<0.01$) and also for height (1.78±0.01 m cf. 1.67±0.01 m, for male and female groups respectively, $P<0.01$), but not for age or for BMI.

Table 5:2 includes the results of the analysis of plasma samples for lipid and lipoprotein concentrations. The results of this analysis are also illustrated in Figure 5:1. Average TC concentration was found to be 11.5% higher in females than males. Furthermore, the average concentration of HDL-C was 0.48 $mmol.l^{-1}$ or 28.6% higher in females. Thus when presented as a ratio (TC:HDL-C) a 19.8% difference was found between the two groups.

Table 5:1. Physical characteristics of males and females within each group: sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T). (n=6 per group) (mean±SEM).

	Age (yr)		Body mass (kg)		Height (m)		BMI $kg.m^{-2}$	
	♂	♀	♂	♀	♂	♀	♂	♀
S-T	23.7 ±1.9	22.3 ±1.2	73.1 ±3.9	62.3 ±2.0 **	1.80 ±0.03	1.68 ±0.02	22.7 ±1.2	22.2 ±0.6
E-T	24.8 ±2.4	21.7 ±0.6	69.1 ±1.3	57.1 ±1.8	1.76 ±0.02	1.69 ±0.01	22.4 ±0.7	20.0 ±0.5
U-T	22.2 ±0.5	21.4 ±0.3	77.8 ±4.5 *	58.2 ±4.1	1.79 ±0.03	1.65 ±1.4	24.6 ±1.2	21.5 ±1.8

Significantly different from E-T **$P<0.01$, *$P<0.05$.

Table 5:2. Plasma total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), TC:HDL-C, percentage of HDL-C and percentage of α-lipoprotein for young males and females. (n=18 males, n=18 females) (mean±SEM).

	TC ($mmol.l^{-1}$)	HDL-C ($mmol.l^{-1}$)	TC:HDL-C	% HDL-C	% α-lipo
♂	4.16 ±0.14	1.20 ±0.06	3.53 ±0.12	29.3 ±1.1	33.2 ±0.9
♀	4.70 ±0.19*	1.68 ±0.08**	2.83 ±0.09**	35.3 ±1.2**	37.2 ±1.3**

Significantly different from male group **P<0.01, *P<0.05.

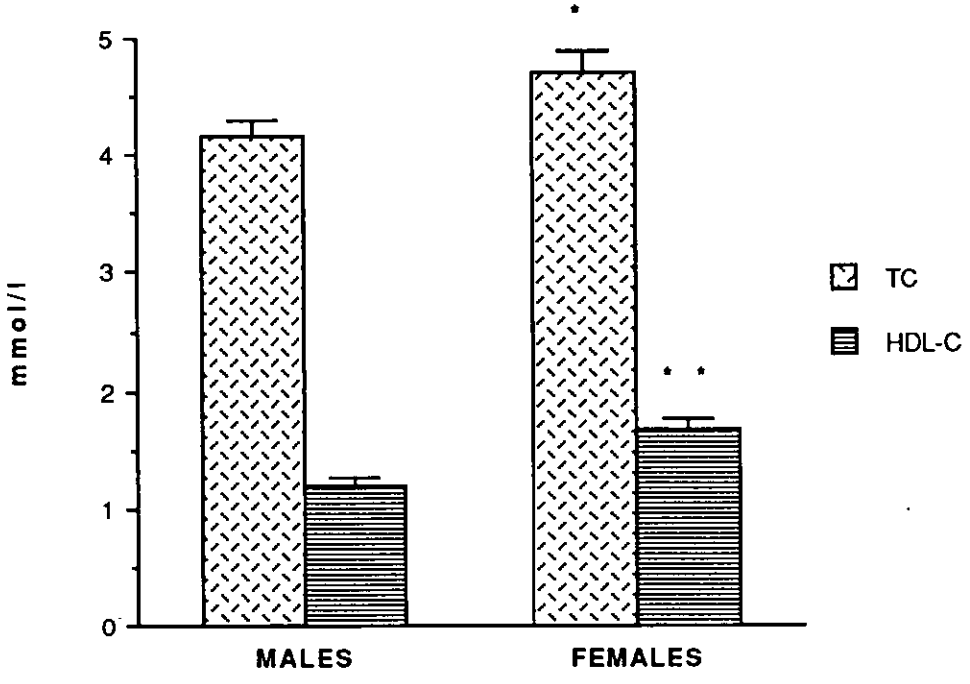


FIGURE 5:1. Concentrations of plasma total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) for young adult males and females (n=18 per group).

Significantly different from males *P<0.05, **P<0.01.

Table 5:3. Average daily energy intake, percentage of average daily energy as fat, carbohydrate and alcohol for young males (n=18) and young females (n=18) (mean±SEM).

	Total daily energy (MJ)	Fat energy (%)	CHO energy (%)	Alcohol energy (%)
♂	12.6±0.7	28.5±1.4	52.4±2.1	5.6±1.5
♀	8.5±0.5**	29.7±1.5	54.4±1.6	1.7±0.9*

Significantly different from males, *$P<0.05$, **$P<0.01$.

In addition to the differences for TC and HDL-C concentrations Table 5:2 also reveals the existence of other differences resulting from the analysis of the plasma samples. The female group was found to have a higher (that is more favourable) % HDL-C (5.3%) and % α-lipoprotein (4.0%) than the male group. Furthermore, application of the Pearson Product Moment Correlation technique resulted in the identification of a positive relationship between % HDL-C and % α-lipoprotein ($r=0.86$, $P<0.01$).

Finally, analysis of the seven day dietary records (Table 5:3) revealed a difference between the sexes for average daily energy intake, the female group consuming only 67.5% of the energy intake of the males. A difference was also observed for the percentage of daily energy consumed as alcohol. However, no difference was observed for the percentage of average daily energy consumed as either fat or as carbohydrate.

b. A comparison of sprint-trained, endurance-trained and untrained groups.

When considered on the basis of training, no differences were found between the three groups for age, body mass, BMI or for height (Table 5:1).

Information relating to the analysis of plasma for lipids and lipoproteins is included in Table 5:4. No differences were found between the groups for any of the parameters (Figure 5:2). In addition, in a comparison of all trained individuals (n=24) with all untrained individuals (n=12), the similarities between the groups remained.

Included in Table 5:5 are the results of the analysis of dietary records. The untrained group consumed only 68% of the average daily energy intake of the sprint-trained

Table 5:4. Plasma total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), TC:HDL-C, percentage of HDL-C and percentage of α-lipoprotein for sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T) young adults (n=12 per group) (mean±SEM).

	TC ($mmol.l^{-1}$)	HDL-C ($mmol.l^{-1}$)	TC:HDL-C	% HDL-C	% α-lipop
S-T	4.64±0.28	1.53±0.13	3.11±0.11	32.3±1.1	35.6±1.4
E-T	4.35±0.23	1.42±0.11	3.21±0.19	32.5±2.2	33.9±1.1
U-T	4.30±0.13	1.38±0.08	3.22±0.20	32.1±1.7	36.1±1.4

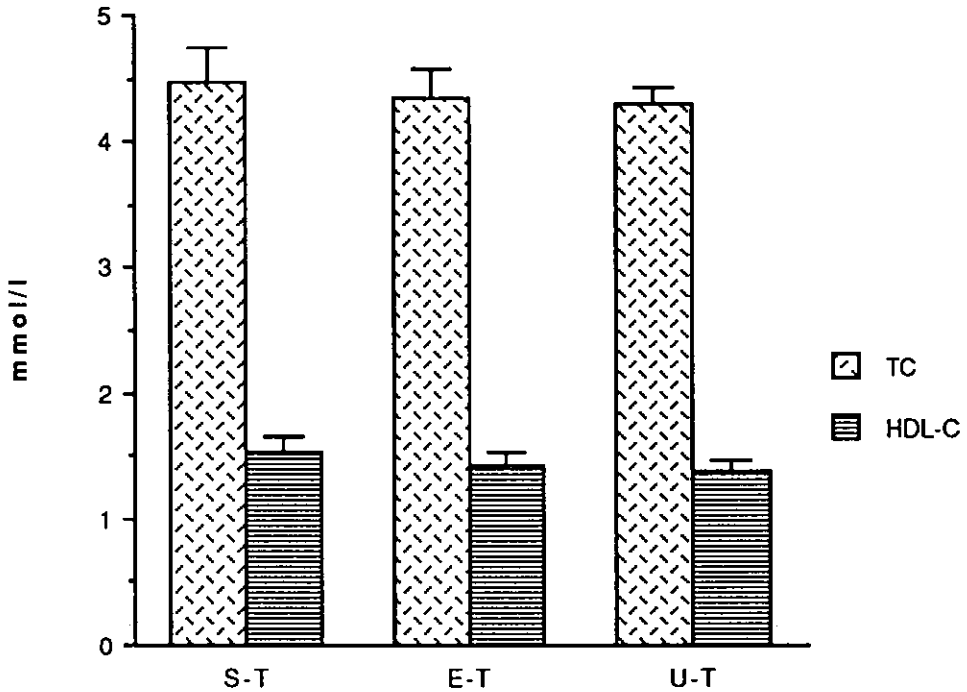


FIGURE 5:2. Concentrations of plasma total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) for groups of sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T) males and females (n=12 per group).

Table 5:5. Average daily energy intake, percentage of average daily energy as fat, carbohydrate and alcohol for sprint-trained (S-T), endurance-trained (E-T) and untrained young adults (n=12 per group) (mean±SEM).

	Total daily energy (MJ)	Fat energy (%)	CHO energy (%)	Alcohol energy (%)
S-T	12.3±1.0††	31.0±0.8**	53.8±0.9*	1.5±0.1††
E-T	11.1±0.8	24.6±2.1	60.4±2.1	1.0±0.2††
U-T	8.4±0.7	31.7±1.3**	46.1±1.5**	8.5±0.6

Significantly different from E-T, $^{**}P<0.01$, $^{*}P<0.05$
Significantly different from U-T, $^{\dagger\dagger}P<0.01$

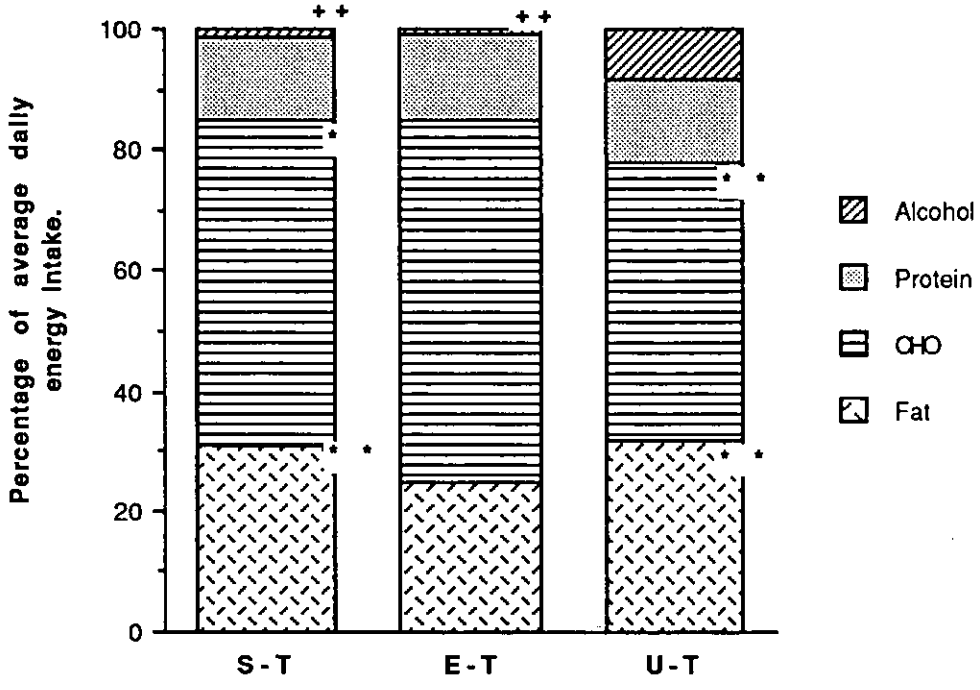


FIGURE 5:3. Percentage of average daily energy intake as fat, carbohydrate, protein and alcohol for groups of sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T) young males and females (n=12 per group).

Significantly different from E-T *P<0.05, **P<0.01.
Significantly different from U-T ++P<0.01.

group. Whilst the comparison between untrained and endurance-trained showed a 24% difference, this however was not statistically significant. Differences were also found for the percentage of average daily energy intake consumed as fat and as carbohydrate. The sprint-trained and untrained groups consumed 6.4% and 7.1% more of their daily intake as fat than did the endurance-trained group. A difference also existed between all trained and untrained individuals for the percentage of average daily energy consumed as carbohydrate.

Finally, the untrained group was shown to consume 7.1% and 7.6% more of their average daily energy intake as alcohol, than did the sprint- and endurance-trained groups respectively (Figure 5:3).

c. A comparison of sprint-trained, endurance-trained and untrained groups of males and females.

This final series of observations enabled the comparison of results derived for groups of young and middle-aged women. In addition, the comparison of the training groups was completed towards the identification of possible sex-linked differences.

The physical characteristics of the sprint-trained, endurance-trained and untrained males and females (n=6 per group) are presented in Table 5:1. (Overall differences between males and females are evident in each subgroup). Endurance-trained males were 9.5% lighter than the untrained males, similarly, endurance-trained females were 9.2% lighter than sprint-trained females.

Table 5:6a includes information for male groups resulting from the analysis of plasma for lipid and lipoproteins. A difference of 7.7% was revealed for TC concentration between sprint-trained males and others. Differences of 8.7% and 5.2% were found between sprint- and endurance-

trained males for TC:HDL-C ratio and % HDL-C respectively. The groups did not differ with regard to either HDL-C concentration or % α-lipoprotein.

Table 5:6b includes data relating to the same parameters for sprint-trained, endurance-trained and untrained females. Total cholesterol concentration was 17% higher for sprint-trained females than for endurance-trained or untrained females. The percentage of α-lipoprotein was 5% lower for the endurance-trained females than for untrained females (Figure 5:4).

In each of the groups, males consumed significantly more energy than did the females (Table 5:3): the latter consumed 67.4%, 68.1% and 66.3% of the average daily energy intake for the respective S-T, E-T and U-T male groups.

A comparison of the percentage of average daily energy intake consumed as carbohydrate showed that both male and female endurance-trained athletes consumed more than their untrained contemporaries. Female endurance athletes (but not male) also had a higher percentage carbohydrate intake than the sprinters. Both male and female sprinters consumed a higher percentage of their energy intake as carbohydrate than the untrained groups.

The untrained individuals (males and females) consumed a significantly higher percentage of their average daily energy intake as alcohol than did the athletes.

Table 5:6a. Plasma total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), TC:HDL-C, percentage of HDL-C, percentage of α-lipoprotein for sprint-trained (S-T), endurance-trained (E-T) and untrained males (U-T). (n=6 per group) (mean±SEM).

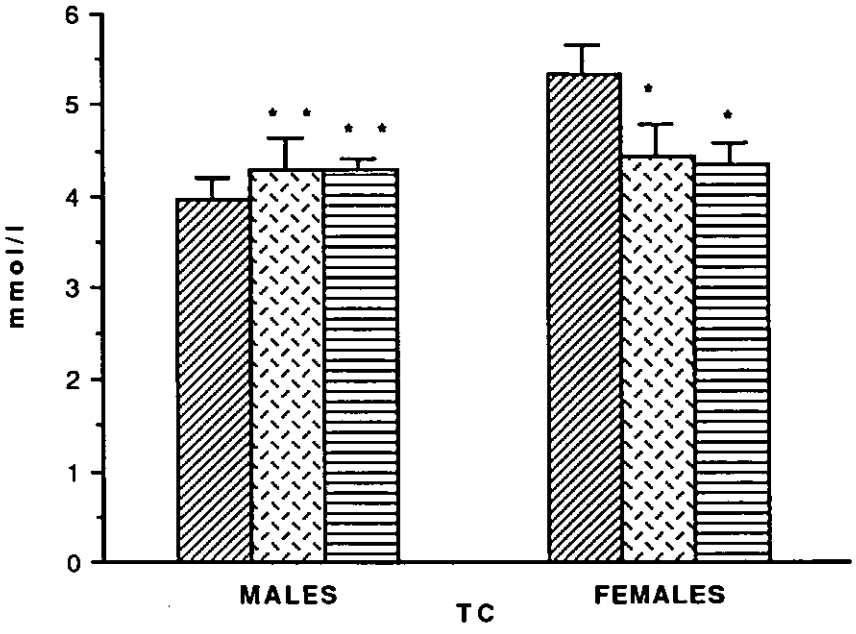
	TC ($mmol.l^{-1}$)	HDL-C ($mmol.l^{-1}$)	TC:HDL-C	% HDL-C	% α-lipop
S-T	3.95 ±0.24	1.21 ±0.07	3.29* ±0.14	31.8** ±1.3	34.4 ±1.8
E-T	4.28†† ±0.34	1.14 ±0.10	3.77 ±0.11	26.6 ±0.9	33.0 ±1.7
U-T	4.27†† ±0.12	1.26 ±0.12	3.54 ±0.33	29.5 ±2.5	32.4 ±1.3

Significantly different from E-T, **$P<0.01$, *$P<0.05$.
Significantly different from S-T, ††$P<0.01$

Table 5:6b. Plasma total cholesterol (TC) high density lipoprotein cholesterol (HDL-C), TC:HDL-C, percentage HDL-C and percentage α-lipoprotein for sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T) females. (n=6 per group) (mean±SEM).

	TC ($mmol.l^{-1}$)	HDL-C ($mmol.l^{-1}$)	TC:HDL-C	% HDL-C	% α-lipop
S-T	5.34 ±0.30	1.85 ±0.16	2.94 ±0.16	32.8 ±1.8	36.8 ±2.2
E-T	4.43* ±0.34	1.69 ±0.13	2.64 ±0.14	38.3 ±2.3	34.9† ±1.5
U-T	4.34* ±0.24	1.50 ±0.09	2.91 ±0.10	34.8 ±1.7	39.9 ±1.2

Significantly different from S-T, *$P<0.05$.
Significantly different from U-T, †$P<0.05$.



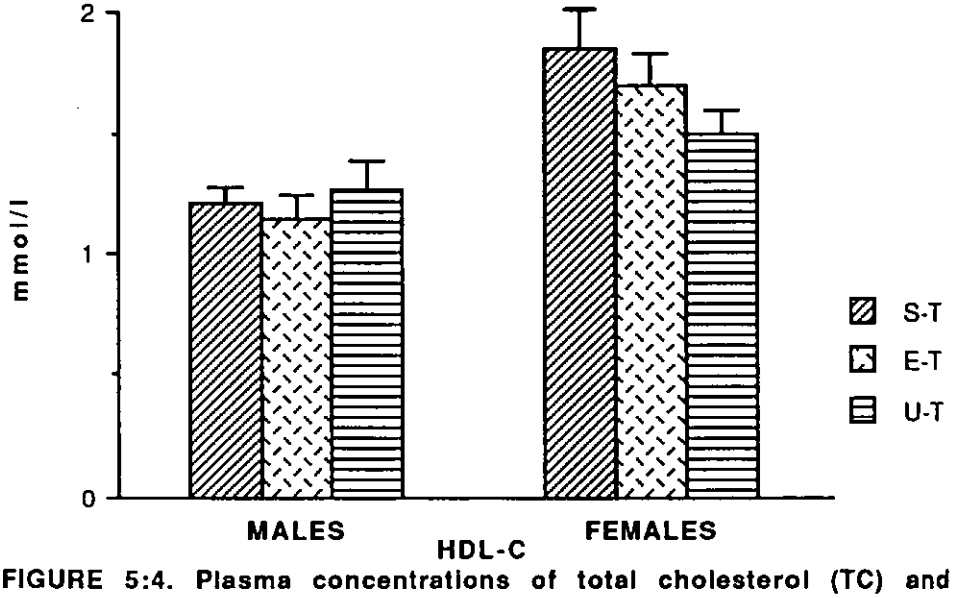


FIGURE 5:4. Plasma concentrations of total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) for sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T) groups of males and females (n=6 per group).

Significantly different from S-T *P<0.05, **P<0.01.

Table 5:7. Average daily energy intake and percentage of total energy derived from fat, carbohydrate and alcohol for sprint-trained (S-T), endurance-trained (E-T) and untrained (U-T) males and females. (n=6 per group) (mean±SEM).

	Total daily energy (MJ)		Fat energy (%)		CHO energy (%)		Alcohol energy (%)	
	♂	♀	♂	♀	♂	♀	♂	♀
S-T	14.7†† ±1.4	9.9** ±0.7	30.6† ±1.1	31.4† ±1.3	53.9* ±1.3	53.6†† ±1.3	2.0** ±1.0	1.1** ±0.5
E-T	13.2** ±0.7	9.0** ±0.9	25.2 ±3.7	24.0 ±2.5	59.2 ±3.0	61.7 ±1.8	1.8** ±1.3	0.2†† ±0.2
U-T	10.1 ±0.8	6.7 ±0.7	29.7† ±1.1	33.7† ±2.1	44.2†† ±2.4	48.0†† ±1.8	13.1 ±2.1	4.0 ±2.4

Significantly different from U-T, $^{**}P<0.01$, $^{*}P<0.05$
Significantly different from E-T, $^{\dagger\dagger}P<0.01$, $^{\dagger}P<0.05$

5.3.2. Results of the comparison of groups of middle-aged women with different exercise habits.

No differences were found to exist between the groups for body mass, height or BMI. Walkers were, however, significantly older than runners (Table 5:8).

Included in Table 5:9 is information concerning the comparison of groups for blood lipids and lipoproteins (Figure 5:5). A difference of 0.66 $mmol.l^{-1}$ (or 12.1%) was discerned for the concentration of TC between runners and sedentary individuals. However, an inverse relationship was determined between TC concentration and exercise training intensity as depicted by PAQ score (higher scores related to higher intensity exercise, that is, running) ($r=-0.336$, $P<0.05$).

No difference was observed between runners and walkers for the concentration of HDL-C. Differences were discerned however between the runners and sedentary groups (0.23 $mmol.l^{-1}$ or 16.3%) and between the walkers and sedentary groups (0.17 $mmol.l^{-1}$ or 13.4%). In both comparisons the more active groups had the higher concentration of HDL-C. In addition a positive, but non significant, trend was determined between HDL-C concentration and PAQ score (opposite to that reported for TC concentration).

No differences were found between the groups for the concentration of HDL_2-C, however, a similar trend to that observed for HDL-C was apparent (that is, a positive relationship between HDL_2-C concentration and activity level). Whilst the comparison for the concentration of HDL_2-C failed to show any difference between the groups, when HDL_2-C was expressed as a percentage of TC, runners were found to have a higher value than that derived for

Table 5:8. Physical characteristics for middle-aged female runners (R), walkers (W) and sedentary (S). (n=12 per group)(mean±SEM).

	Age (yrs)	Mass (kg)	Height (m)	BMI ($kg.m^{-2}$)
R	39.60±1.06*	59.0±2.2	1.64±1.8	21.87±0.53
W	43.06±1.14	62.0±1.3	1.64±1.7	22.92±0.43
S	41.52±1.31	61.9±2.1	1.64±1.7	22.92±0.49

Significantly different from walkers * $P<0.05$.

Table 5:9. Serum total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), high density subfraction (HDL_2-C), TC:HDL-C ratio and triacylglycerol (Tg) concentrations for middle-aged female runners (R), walkers (W) and sedentary (S) groups (n=12 per group)(mean±SEM).

	TC ($mmol.l^{-1}$)	HDL-C ($mmol.l^{-1}$)	HDL_2-C ($mmol.l^{-1}$)	TC:HDL-C	Trigs ($mmol.l^{-1}$)
R	4.81±0.23*	1.33±0.10*	0.73±0.08	3.91±0.39*	0.60±0.50
W	5.30±0.14	1.27±0.07*	0.67±0.07	4.33±0.28*	0.82±0.11
S	5.47±0.20	1.10±0.03	0.54±0.04	5.02±0.13	0.74±0.09

Significantly different from sedentary group *$P<0.05$.

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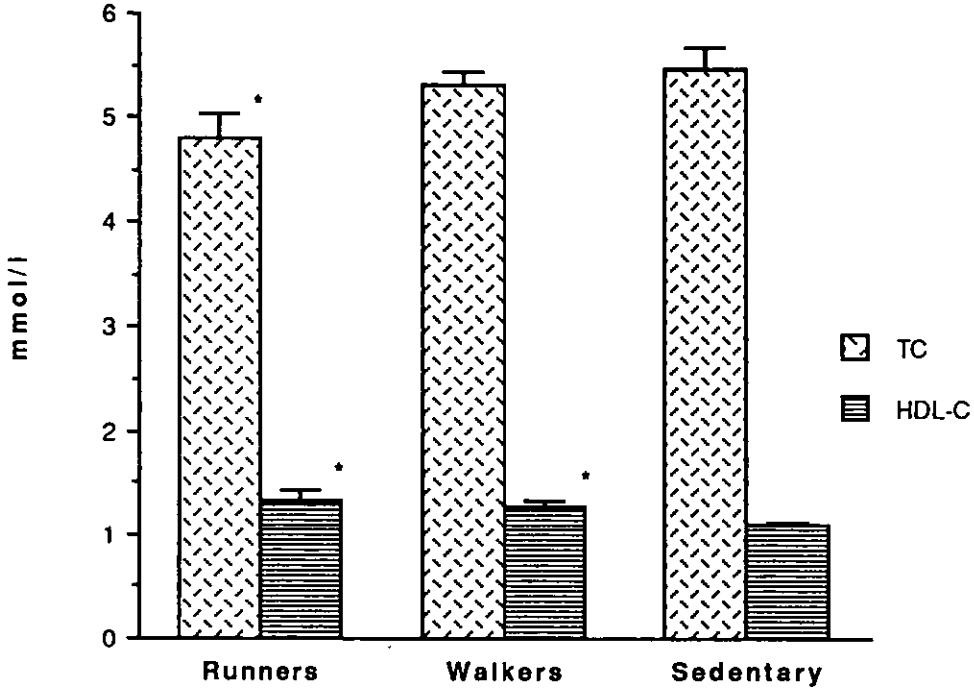


FIGURE 5:5. Serum concentrations of total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) for groups of middle-aged women (N=12 per group).

Significantly different from sedentary group *P<0.05.

the sedentary group (15.8±7.7% cf. 9.8±2.1%, $P<0.05$ for runners and sedentary groups respectively).

When TC and HDL-C concentrations were expressed as a ratio (TC:HDL-C), both trained groups were shown to have a lower value (that is, more favourable) than the sedentary group. No difference was discerned between the two trained groups. Furthermore, as with TC concentration, an inverse relationship was observed between PAQ score and the ratio value for TC:HDL-C ($r=-0.453$, $P<0.01$). No differences were found for any of the comparisons for the concentration of Tgs.

Data derived from the analysis of dietary food records is presented in Table 5:10. A difference was determined for average daily energy intake between runners and sedentary individuals. The sedentary group consumed on average 2.4 MJ or 25.5% less energy than the runners. When average daily energy intake for all subjects (n=36) was correlated with PAQ a significant positive relationship was observed ($r=0.515$, $P<0.01$). Thus the more active individuals were found to have a higher average daily energy consumption than did the sedentary individuals.

No differences were found between the groups for the percentage of average daily energy intake consumed as fat, carbohydrate or alcohol. However, a non significant trend was seen between the percentage carbohydrate consumed and training status (that is, percentage of energy consumed as carbohydrate was highest in runners, then walkers, with lowest percentages for the sedentary group; inevitably, the opposite trend was observed for fat consumption) (Figure 5:6).

Table 5:10. Results of dietary analysis: average daily energy intake, percentage of average daily energy as fat, as carbohydrate and as alcohol for middle-aged women runners (R), walkers (W) and sedentary groups (S). (n=12 per group)(mean±SEM).

	Total daily energy (MJ)	Fat energy (%)	CHO energy (%)	Alcohol energy (%)
R	9.4±0.4**	34.7±1.3	48.0±1.0	2.7±1.4
W	8.2±0.5	35.6±1.5	46.0±1.6	2.8±1.4
S	7.0±0.6	35.2±0.9	44.4±1.9	4.1±1.0

Significantly different from sedentary group **$P<0.01$.

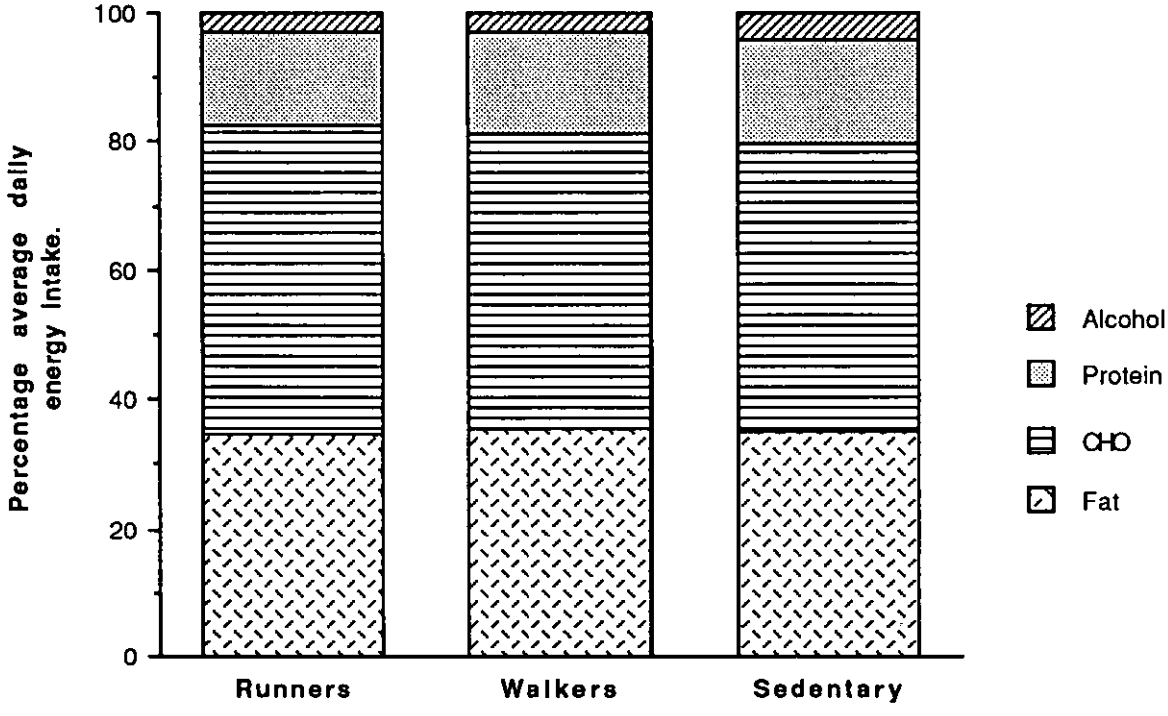


FIGURE 5:6. Percentage of average daily energy intake as fat, carbohydrate, protein and alcohol for groups of middle-aged women (n=12 per group).

5.4 Discussion.

Evidence exists to show that females, particularly in their pre-menopausal years, have an inherently higher concentration of HDL-C and a more favourable (lower) TC:HDL-C ratio than their age-matched male counterparts (Oliver & Boyd 1959; Gordon et al. 1977; Kannel et al. 1971). Much of this information has been derived from studies completed in the United States, however, a recent report (Mann et al. 1988) has identified a similar pattern amongst British adults. The results of the present study of young adults are therefore consistent with these general trends, and provide further evidence for a sex-linked difference in lipid and lipoprotein metabolism (Tables 5:2, 5:6a & 5:6b).

In contrast, a comparison of young adults differing with regard to training status (Table 5:4) revealed no differences for TC, HDL-C, TC:HDL-C, % HDL-C or for % α-lipoprotein. This observation therefore is contradictory to the evidence derived from previously completed cross-sectional studies, many of which have reported higher HDL-C concentrations for endurance athletes when compared with less active controls (Adner & Castelli 1980; Hartung et al. 1983; Haskell 1984; Wood et al. 1983; Lehtonen & Viikari 1978).

However, whilst the results gained for endurance training are relatively consistent, observations derived for the effectiveness of sprint or power training is rather less conclusive. Some studies have reported elevated HDL-C concentrations (Enger et al. 1977; Schnabel & Kindermann 1982; Vodak et al. 1980), whilst others found no difference for the comparison with untrained individuals (Berg et al. 1980; Farrell et al. 1982; Nikkilä et al. 1978). Thus whilst a difference between endurance and untrained groups might have been expected, the absence of

such a difference between the sprinters and untrained groups for HDL-C is not too surprising.

The observed inconsistency in the results for HDL-C concentration and endurance training between the present study and those previously completed, might to some extent be explained by differences in the subjects employed. For example, many of the previous studies were conducted with groups of older (that is, middle-aged) males. The present training groups however involved younger individuals.

As already mentioned most of the existing evidence is concerned with middle-aged individuals, however, some evidence does exist for groups of individuals of a similar age to those presently observed. Differences in lipid and lipoprotein metabolism have been reported where subjects involved have been very highly trained males. For example, elite Olympic athletes were employed in the studies completed by Tsopanakis and coworkers (1986) and also by Deshaies and Allard (1982).

It is therefore possible that the inability to differentiate between the groups of sprint, endurance and untrained individuals involved in the present study was due to insufficient training (that is, the athletes involved were of University club standard and as such could not be considered to be in the 'elite' category considered by Tsopanakis et al. 1986 or by Deshaies and Allard 1982). A further possibility lies with the fact that both males and females were considered together within the training groups, a situation which contrasts with the previous studies which tended to employ only males.

In the absence of any differences derived from the above comparison and with the possibility that males and

females might respond differently to the effects of training an observation of training based differences between male and female groups was also completed.

The results of the analysis of plasma for lipids and lipoproteins (Tables 5:6a & 5:6b) is consistent with the suggestion of a possible sex-linked response to exercise training (Frey et al. 1982; Brownell et al. 1982). Evidence for this proposal has been derived from the observation that endurance training is associated with increased HDL-C concentration in men (Altekruse & Wilmore 1973; Brownell et al. 1982; Lehtonen & Viikari 1980; Lopez et al. 1974), whilst similar observations completed with women are rather less consistent (Wynne et al. 1980; Frey et al. 1982; Moll et al. 1979; Brownell et al. 1982).

Further evidence in support of a sex-linked difference in response is derived from the results of studies in which a direct comparison has been made between males and females undergoing the same training programme. The results indicate that significant post-training differences in lipid and lipoprotein metabolism experienced by male subjects are not necessarily discernible for women (Brownell et al. 1982; Ballantyne et al. 1981). Based on such observations Haskell (1984) notes:

> "...it appears more difficult to change plasma lipoprotein concentration in women by exercise training than in men".
>
> (Haskell 1984).

However, whilst the results of training studies completed with women are less consistent than those involving men, it cannot be concluded that exercise-induced changes in lipid and lipoprotein metabolism do not occur with women.

In fact, cross-sectional studies with women have produced similar results to those conducted with men. That is, women endurance athletes have also been found to have more favourable lipid and lipoprotein concentrations than less active or sedentary individuals (Wood et al. 1977; Moore et al. 1983). The results of the present study, conducted with middle-aged women are also consistent with these latter reports.

This consistency is exemplified with the results of the comparison of exercise groups for the concentration of TC (Table 5:9). Total cholesterol concentration in middle-aged runners was lower than for their sedentary counterparts, an observation which is supported by that reported by Brownell and coworkers (1982).

In addition to the evidence for TC, cross-sectional studies of men typically report higher HDL-C concentrations associated with endurance training: Adner and Castelli 1980; Christie and coworkers 1980; Hartung and colleagues 1980 and 1983. Similar observations have also been reported by the few cross-sectional studies conducted with women (Moore et al. 1983; Wood et al. 1977). These observations therefore support the higher HDL-C concentrations found in the present study for middle-aged walkers and runners when compared with the sedentary group.

Regular endurance exercise has also been associated with relatively lower Tg concentrations in male athletes when compared with sedentary individuals (Martin et al. 1977; Wood et al. 1976; Vodak et al. 1980). The results derived for the present study however were unable to differentiate between the groups on the basis of training. Whilst contrary to the evidence reported for male subjects, this observation is consistent with the results of similar studies conducted with females

(Allison et al. 1981; Brownell et al. 1982; Franklin et al. 1979; Lewis et al. 1976). Nikkilä and coworkers (1978) have suggested that since women have a lower baseline concentration of Tg (relative to men), the potential for further reduction consequential to exercise is limited. These proposals appear to be supported by the results of the above studies which show exercise-induced changes for men, but not for women.

How do the patterns of differences observed with older women relate to those observed with the younger age group?

The comparison of older women revealed a lower TC concentration for the runners when compared with the sedentary group. No difference was found however, for a similar comparison of the equivalent groups of younger women. For HDL-C concentration, although no significant differences were found for the younger groups of women, there was a trend towards an increased concentration of HDL-C for the endurance-trained when compared with the untrained individuals. A similar occurrence was found for the older women, here both walkers and runners had significantly higher concentrations of HDL-C relative to the sedentary group.

In both sets of comparisons (that is, for older and younger women), the resultant ratio values (TC:HDL-C) were lower (that is, more favourable) for the endurance trained individuals. However, as with HDL-C, only a non-significant trend existed for the younger group, whilst the differences between the older groups attained statistical significance.

It might be suggested that at least some of the difference observed for the concentrations of TC and HDL-C between the groups of middle-aged women might be the

result of age: the group of runners were shown to be younger than the walkers. According to a recent survey of British adults (Mann et al. 1988) TC concentration in women increases from 5.1±1.0 $mmol.l^{-1}$ at 25-29 years to 6.7±1.2 $mmol.l^{-1}$ at 55-59 years. Furthermore for the cohorts representing the ages of the present groups of runners and walkers a 0.3 $mmol.l^{-1}$ was reported. This difference however is insufficient to account for the 0.49 $mmol.l^{-1}$ difference in TC concentration existing between the present groups. It is therefore possible that age might be important determinant of the concentration of TC observed. However, it is unlikely to account for the difference between the groups. Furthermore, in population studies age has little effect on the concentration of HDL-C (Mann et al. 1988). It is therefore likely that some alternative influence might be of greater importance.

The mechanisms responsible for mediating the changes identified with exercise have until now been given only cursory consideration. It is known however, that blood lipid and lipoprotein concentrations are the result of the processes of synthesis and catabolism, the rates of which are governed by the enzymes lipoprotein lipase, hepatic lipase and LCAT. Furthermore, the activity of these enzymes has been shown to be indirectly influenced by sex-hormone concentration, by dietary fat intake, by body composition and by training status.

The sex-related differences in lipid and lipoprotein concentrations reported in the first part of the present study, might be explained by differences in endogenous sex hormone production. Evidence for this proposal has been derived from observations of the effects of oestrogen. Oestrogen has been shown to increase the activity of lipolytic enzymes (lipoprotein lipase, hepatic lipase and LCAT) and thus result in an increase

in the synthesis of HDL-C. Furthermore, at the cessation of the oestrogen cycle (either after the menopause or through oral contraceptive use) and therefore the reduction of circulating oestrogen, there is an associated lowering of HDL-C concentration (Assmann et al. 1982; Kannel 1987; Matthews et al. 1989; Fotherby 1989). This proposal is further substantiated by the results of hormone replacement therapy, in which oestrogen concentration is artificially elevated, and which is associated with increases in HDL-C concentration (Krauss 1987).

On the basis of this evidence it is suggested that the higher concentration of HDL-C observed for females compared with males (Table 5:2) was the result of differences in endogenous sex hormones. The magnitude of this influence is emphasised with the comparison of the two untrained groups (Table 5:6a and b). This comparison eliminates the potentially confounding effect of exercise and reveals a 16% higher concentration of HDL-C and a 18% lower TC:HDL-C ratio for females compared with males.

Whilst this situation is beneficial in the untrained state, it has been suggested (Nikkilä et al. 1978) that the potential for further increases in HDL-C concentration for women, is limited as a consequence of these relatively high levels of lipolytic enzyme activity even in sedentary females. In addition, exercise, particularly endurance exercise, provokes a suppression of oestrogen synthesis in women (and thus a lower concentration of HDL-C). In contrast, however, the concentration of testosterone in men is decreased (Remes et al. 1979) resulting in an increase in lipoprotein lipase, hepatic lipase and LCAT activity, and therefore an increase in HDL-C (Mendoza et al. 1981; Hill et al. 1980; Nordoy et al. 1979). Altogether, this evidence suggests that it might be more difficult to provoke

exercise-induced changes of lipid and lipoprotein metabolism in women than in men, as suggested by the results of the training studies described earlier (Brownell et al. 1982; Ballantyne et al. 1981).

Although not measured in the present study, exercise training is associated with cardiorespiratory (Bruce 1984; Saltin 1969) and peripheral (Saltin & Gollnick 1983) adaptations which increase the capacity for aerobic metabolism. One of these changes is an increased capilliarization of the trained skeletal muscle (Holloszy & Coyle 1984). Since lipoprotein lipase is located on the endothelium of this tissue, lipolytic activity and therefore HDL-C synthesis is potentially greater in endurance trained rather than sedentary individuals (Nikkilä 1978). Consistent with this is the result of studies involving sprint and power-trained athletes who rely more heavily on anaerobic forms of energy production. These athletes cannot be differentiated from sedentary individuals on the basis of blood lipids (Enger et al. 1977; Schnabel & Kindermann 1982; Vodak et al. 1980) or lipoprotein lipase activity (Nikkilä 1978).

In the present study, the results for young adults are rather inconsistent. Female sprinters were found to have higher TC concentrations than the other groups, and no differences were observed for HDL-C concentration or for TC:HDL-C ratio. Sprint trained males had lower TC concentration and also lower TC:HDL-C ratio, but again no differences were found for HDL-C concentration.

In contrast, the observations of older women provided evidence of training-related changes more consistent with the results of previously reported studies: the runners had a lower TC concentration and both runners and walkers had higher HDL-C concentrations and lower TC:HDL-C ratios than were found for the controls.

The question therefore arises: what is the basis of the differences in response to training between the age groups, that is, why should a difference exist in lipid and lipoprotein metabolism for the older group of women but not in the younger adults, particularly the younger females?

Additional factors have also been shown to be important in determining lipid and lipoprotein concentrations, which as yet have not been considered in this present discussion. Obesity is associated with low blood HDL-C concentrations. This is evidenced by the results of weight reduction programmes which have shown increased HDL-C concentration with weight loss. Furthermore, weight gain has been associated with the reversal of this effect (Wilson & Lees 1972; Contaldo et al. 1980; Streja et al. 1980). However, in the present study, there were no differences between the respective groups for body fatness, as indicated by BMI (Tables 5:1 & 5:8). It is therefore reasonable to suggest that body fatness was not a significant influence on the lipid and lipoprotein concentrations observed for the individuals involved in the present study.

An additional influence on lipid and lipoprotein metabolism is diet. Diet is known to have an intrinsic role to play in the synthesis of blood lipids and lipoproteins through the exogenous metabolic pathway (Brown & Goldstein 1976; Goldstein & Brown 1977). Thus a diet rich in carbohydrate has been shown to increase the synthesis of Tg-rich lipoproteins and decrease the concentration of HDL-C. A fat-rich diet, however, has the opposite effect, as does alcohol consumption (Gurr et al. 1989; Katan 1985). For these reasons it was necessary for subjects to complete a dietary record as part of the study.

The results of the subsequent analysis of food records completed by the younger groups showed sex-linked differences in average daily energy intake (Tables 5:3 & 5:7). However, when expressed as the percentage of average daily intake consumed as either fat or as carbohydrate no differences were observed. These findings suggest that the differences in blood lipids observed between the sexes (Table 5:2, 5:6a & b) were the consequence of sex-related influences (e.g. the proposed hormone theory) rather than as a result of differences in dietary composition.

When dietary factors are considered in relation to male and female training groups, differences were observed. Endurance trained males consumed a smaller percentage of their average daily energy as fat than did the other male groups. Furthermore, untrained males consumed significantly more alcohol than did the trained groups. As with male endurance athletes, endurance-trained females also consumed a lower percentage of daily energy as fat when compared with the sprint-trained and untrained groups. These dietary differences between the training groups, might go someway to explaining the inability to differentiate between the groups for HDL-C concentration (Table 5:6a & 5:6b).

When diet is considered in relation to training (that is, when males and females of the same training are combined in one group), significant differences emerged. Endurance-trained individuals consumed significantly less fat and more carbohydrate than either sprint-trained or untrained groups (Table 5:5 & 5:7). Furthermore, untrained individuals consumed significantly more alcohol than the trained groups (8.5±3.1%, 1.0±2.3% and 1.5±1.9% for U-T, E-T and S-T respectively $P<0.01$). As previously mentioned recent investigations have shown plasma HDL-C concentration to decrease with an increase in dietary

carbohydrate. This occurs as a result of an increase in the endogenous synthesis of Tg and a concomitant decrease in HDL-C concentration (Haskell 1984; Hartung 1984). As a consequence, any increase induced by exercise would be opposed (Hartung 1984). Also shown to affect plasma lipid and lipoprotein levels are alterations both in total fat intake and changes in the type of fat consumed (Mattson & Grundy 1985). As a result, diets providing a high percentage of energy as fat have been found to provoke an increase in the concentration of plasma TC and HDL-C (Ehnholm et al. 1982). However, diets containing less than 30% of energy as fat may produce a decrease in plasma TC and HDL-C, whilst not necessarily affecting the TC:HDL ratio (Ernst et al. 1980; Hartung et al. 1983).

It might therefore be that differences in sex hormone production along with differences in dietary composition help to explain the similarities in plasma lipids and lipoproteins found between the three groups of young adults with different exercise habits (Table 5:4).

No differences were found, however, between the groups of older women for the percentage of average daily energy intake consumed as fat, as carbohydrate or as protein. On this basis therefore, dietary differences are unlikely to account for the differences in lipids and lipoproteins between walkers, runners and controls (Table 5:9).

For older females therefore, endurance exercise was associated with higher concentrations of HDL-C, a trend towards higher HDL_2-C and a lower TC:HDL-C ratio. However, for young females no differences in HDL-C or TC:HDL-C ratio existed (although a trend did exist for higher HDL-C for endurance-trained compared with untrained group and a lower ratio of TC:HDL-C). Therefore it is possible that the previously quoted statement of Haskell (1984) correctly suggested that exercise-related changes which

do occur for females, might be expected to take a considerable time to establish. This might therefore explain the present findings of discernable differences for older but not younger groups of women, relative to lipid and lipoproteins. Therefore with a longer period of training trends discerned for the young females become observable differences between the groups of older women.

Conclusions.

No differences in lipid and lipoprotein metabolism were discerned between young adults grouped on the basis of habitual exercise training. However, differences were derived from similar comparisons of middle-aged women who were habitual runners, walkers or who were sedentary controls. When young women were grouped on the basis of training, differences in lipid metabolism similar to those of older women were observed, but these were not statistically significant. It is therefore possible that the trends seen with younger women, with time and more prolonged training, are expressed as physiologically important differences, that is, if the training stimulus is maintained.

6. THE INFLUENCE OF A ONE YEAR PROGRAMME OF BRISK-WALKING IN FORMERLY SEDENTARY MIDDLE-AGED WOMEN.

6.1. Introduction.

The results of the exploratory study reported in Chapter 4 provided evidence to suggest that brisk-walking might be an appropriate form of exercise for sedentary, older individuals. The results indicated that this activity might be sufficient to provoke the thresholds of heart rate and $\%\dot{V}O_2$ max thought necessary for the improvement of functional capacity (ACSM 1986). In addition, the results of the cross-sectional study (Chapter 5) indicated the existence of a relationship between long-term endurance training and lower TC concentration and higher HDL-C concentration, in middle-aged women.

It was the aim of the present study therefore to examine the effects of a one year period of brisk walking on endurance capacity, TC and HDL-C concentration, body composition, diet and aspects of psychological well-being with a group of middle-aged, sedentary women. The results of these observations are presented as four separate, inter-related sections:-

6.3.1. Walking performance, functional capacity and endurance fitness. Studies of relatively high intensity activity indicate that regular exercise provokes physiological and metabolic changes which result in a more effective system of oxygen transport and utilization (Wenger & MacNab 1975; Saltin & Karlsson 1971; Astrand & Rodahl 1986). As a consequence, the oxidative potential of the trained muscles is increased, enabling standardised submaximal exercise to be completed with less fatigue (Wood et al. 1983; Wilmore et al. 1987).

6.3.2. Total cholesterol and HDL-C. Cross-sectional studies have identified a favourable relationship between endurance training and the concentration of TC and HDL-C (Lehtonen & Viikari 1978; Hartung et al. 1980; Wood et al. 1976; Nikkilä et al. 1978; Schnabel & Kindermann 1982). In addition, some longitudinal studies have shown a decrease of TC concentration and an increase of HDL-C with endurance training (Brownell et al. 1982; Ballantyne et al. 1982; Leon et al. 1979; Huttenen et al. 1979; Keins et al. 1980; Peltonen et al. 1981). However, the amount and intensity of exercise required to provoke these changes remains uncertain.

6.3.3. Body composition and diet. Body composition (Wilson & Lees 1972; Contaldo et al. 1980) and dietary intake (Gurr et al. 1989) have been shown to be important in determining the concentrations of plasma lipids and lipoproteins. It was therefore necessary to monitor these factors throughout the programme.

6.3.4. Psychological aspects of well-being. Evidence exists to suggest that endurance exercise provokes an improvement of self-concept (Dowell et al. 1968; Hughes 1974), a reduction of anxiety (Morgan 1979; Pauly et al. 1982) and a possible lessening of tension, depression and fatigue (Blumenthal et al. 1982). Together these changes constitute an overall improvement in psychological well-being.

6.2. Methodology.

(i). Subjects.

Forty four women (aged 30 to 62 years) from the town and university communities took part in the study. None of the volunteers had participated in any form of regular or structured exercise in the year prior to the onset of the programme and were for the purpose of the investigation considered as sedentary.

The experimental groups were determined on a convenience basis: that is, the degree of commitment and time required to complete the programme was discussed with the individuals. After consideration each subject made an informed decision. Twenty eight of the women volunteered to join the brisk walking group, whilst the remaining 16 acted as controls. The physical characteristics of the subjects are presented in Table 6:1.

After the completion of the baseline tests by all individuals, the controls maintained their habitual lifestyle, whilst the women in the training group undertook a programme of brisk walking.

(ii). Protocol.

The submaximal track test was performed prior to and at the end of the training programme (Chapter 3:3), as was the measurement of body density (Chapter 3:5). The latter was determined by hydrostatic weighing with the simultaneous determination of residual volume by the three breath nitrogen wash out technique (Appendix 4).

The maximal track walk, submaximal treadmill walk, incremental step-test for the prediction of VO_2max and anthropometrical measurements were all completed at baseline, 3 months, 6 months and at 12 months of the study.

At the same intervals, a 5ml sample of blood was obtained by venopuncture. this was then centrifuged and the plasma was removed. After storage at -20^{0} C the sample was assayed for TC by the colorimetric enzymatic method (Appendix 8). In addition, HDL-C was assayed in the supernatant remaining after the precipitation of low and very low density lipoproteins by phosphotungstic acid and magnesium chloride (Appendix 12). All samples from the same individual were assayed in the same batch. Between batch quality control was established using standard sera (Precinorm, Boehringer-Mannheim) during each assay (Chapter 3.7).

A seven day weighed food intake was completed at baseline, at 6 months and at 12 months (Appendix 13). However, the physical activity questionnaire (Appendix 15) and battery of psychological questionnaires, which included GHQ (12), STAI and self-esteem measures were completed at baseline and were then repeated at 3, 6 and 12 months of the programme (Appendix 14).

All procedures were performed in accordance with the previously described methodology (Chapter 3).

Table 6:1. Physical characteristics of walkers (W) and controls (C) at baseline. (W n=28, C n=16). (Mean±SEM).

	AGE (yr)	BODY MASS (kg)	HEIGHT (m)	BMI ($kg.m^{-2}$)
W	44.9	64.0	1.64	23.9
	±1.5	±1.7	±0.01	±0.6
	30.6-62.8	48.8-83.7	1.52-1.73	19.1-32.3
C	44.4	61.2	1.59	24.0
	±2.3	±2.3	±0.01	±0.1
	29.0-59.0	50.0-81.1	1.48-1.67	20.5-32.1

(iii). Training.

Initially, subjects were required to complete a minimum of 100 minutes of walking per week, in three training sessions. If it is assumed that the pace adopted was similar to that attained during the submaximal track walk (1.72 $m.s^{-1}$ or 3.8 mph), then this would equate to approximately 7 miles per week.

The minimum prescription was gradually increased over the first three months of the study, reaching a maximum of 175 minutes or 12 miles per week (5 sessions). This level was then maintained as the minimum target for the remainder of the programme (Figure 6:1). Throughout the programme, the minimum duration of any training session was 20 minutes, whilst the maximum period was 50 minutes.

Compliance with the programme was assessed using a diary type record, which was checked fortnightly. Adherence was also confirmed by monitoring the physiological response to standardised exercise in the laboratory, that is, the submaximal treadmill walk and incremental step-test.

The significance of difference between means was evaluated using analysis of variance (ANOVA) with appropriate post-hoc test and Student's t-test, adopting a 5% level of significance. Relationships between variables were examined using Pearson's product moment correlation coefficient. Throughout, the results are presented as the mean ± standard error of the mean (mean±SEM).

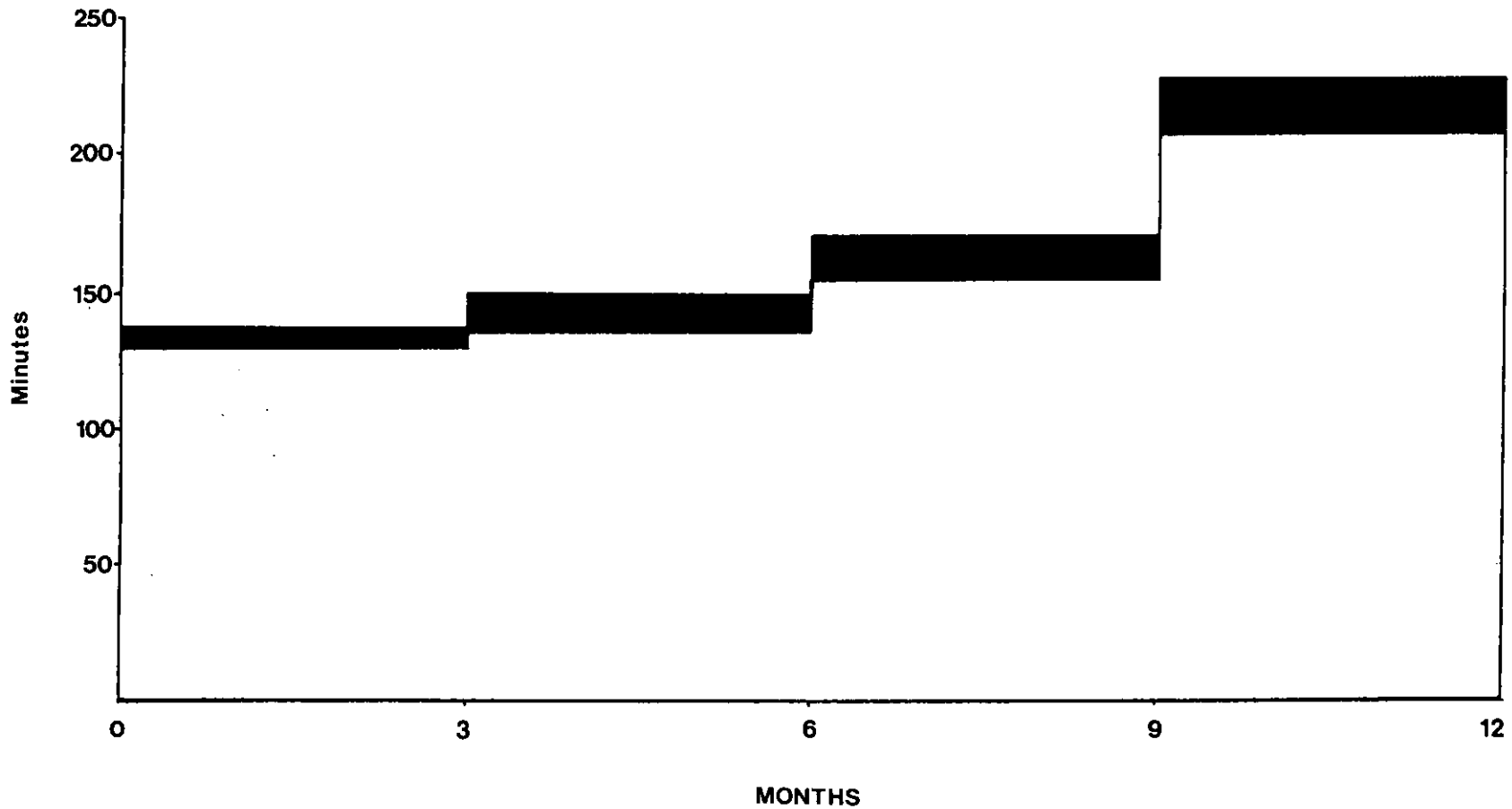


FIGURE 6:1. Average number of minutes walking completed at intervals throughout the one year training programme (n=28).

6.3. Results.

The diary-records revealed that the walkers had completed an average of 8,087±454 minutes (range 3,878 to 14,920 mins) of brisk walking over the year of the programme, approximating to 155 minutes per week (Figure 6:1). A conservative estimate of the distance completed (9 miles or 16.1 km per week) was derived with the adoption of the pre-training brisk pace (1.72 $m.s^{-1}$).

6.3.1. Walking performance, functional capacity and endurance.

The time and pace required for the completion of the one mile maximal-paced walk was the same at baseline for both groups (Table 6:2). The table also reveals that with training, the pace attained by walkers progressively increased. At the end of the study, the overall increase from baseline was 8.4%, which equated with a 1.06 minute reduction in the time required for the completion of the mile. This was also significantly less than the time required by the control group. Concomitant with the observed increase in maximal pace attained by walkers was a significant increase in the heart rate elicited.

Also included in Table 6:2 is data pertaining to the submaximal walk. As was observed for the maximal walk at baseline, there was no difference in the time or pace required by the two groups for the completion of the submaximal test. Similarly, over the duration of the study the pace for walkers increased by 0.14 $m.s^{-1}$ or 7.8% from baseline, and at 12 months differed from the pace attained by controls, which remained unchanged throughout the programme.

No difference existed between the average heart rate derived from the submaximal track or treadmill walks, suggesting that the latter might be an appropriate means

Table 6:2. Pace and mean heart rate of the maximal and submaximal track walks, for walkers (W n=28) and controls (C n=16) (Mean±SEM).

	Baseline	3 Months	6 Months	12 Months
Maximal track walk.				
Speed ($m.s^{-1}$)				
W	1.91±0.03	1.98±0.03**	2.01±0.03**	2.07±0.03** ⁰⁰
C	1.87±0.04	1.84±0.04†	1.86±0.04†	1.89±0.04†
Heart rate ($bt.min^{-1}$)				
W	150±4	151±4	157±3	162±3
C	144±5	138±5	141±4†	147±5†
One mile brisk walk.				
Speed ($m.s^{-1}$)				
W	1.73±0.03			1.87±0.03**
C	1.71±0.05			1.70±0.05†
Heart rate ($bt.min^{-1}$)				
W	129±2			119±3**
C	124±6			127±5†

Significantly different from baseline * $P<0.05$, ** $P<0.01$.
Significantly different from walkers † $P<0.05$.
Response of walkers significantly different from controls, interaction ⁰⁰ $P<0.01$.

by which to monitor the physiological adaptations to endurance training. Consistent with this proposal similar patterns were observed for the average heart rate elicited during the submaximal track and treadmill walks. For example at baseline and throughout the study no difference existed between the groups for the average heart rate elicited during the submaximal treadmill walk (Table 6:3). However, at 3 months heart rate for walkers was decreased by 4 $bt.min^{-1}$. As the programme progressed further decreases were also incurred. The overall reduction at 12 months of 7 $bt.min^{-1}$ or 6.9% from baseline. No change was found for controls. Furthermore the difference between the groups was emphasized with the observation of a significant interaction effect.

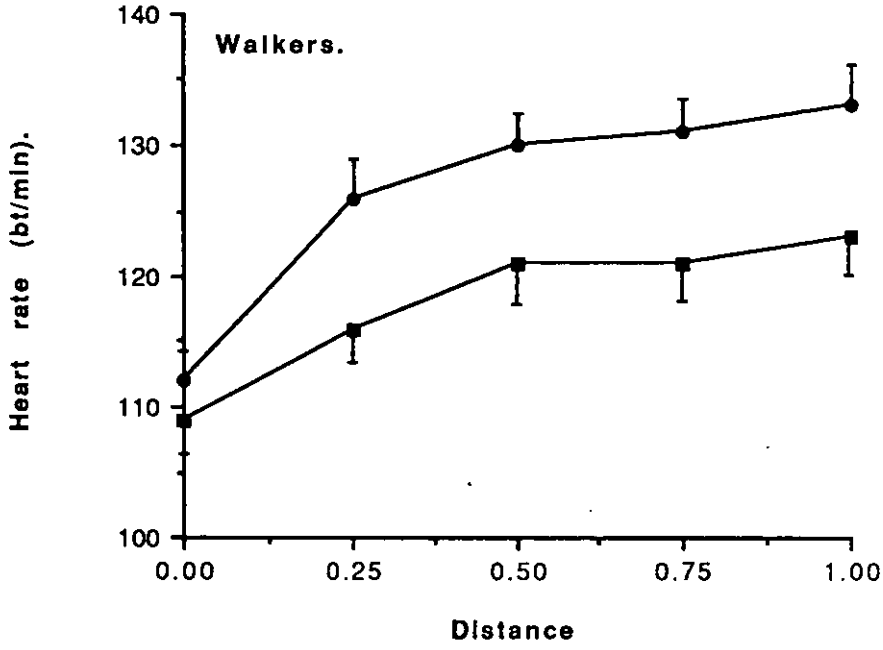
A similar pattern was also observed for the average heart rate elicited during the track walk, that is, a decline for walkers in the absence of any change for the control group. Furthermore the lower heart rate observed for walkers at 12 months occurred despite the selection of a faster pace.

In addition to presenting submaximal treadmill heart rate data as an average for the whole walk, the average heart rate at the start of the walk and at quarter mile intervals was also considered (Figure 6:2). This approach showed that with training the average heart rate at each distance was reduced from baseline to three months. A similar pattern was also identified between six and twelve months. However, between three and six months no differences emerged at any of the distance markers. The analysis of the data in this way therefore re-emphasizes the previous observation that heart rate on this standard exercise task is reduced with training, whilst in addition provides more information regarding temporal changes which occured.

Table 6:3. Mean heart rate, R value and rate of perceived exertion (RPE) during the treadmill walk, for walkers (W n=28) and controls (C n=16) (Mean±SEM).

	Baseline	3 Months	6 Months	12 Months
Heart rate (treadmill) ($bt.min^{-1}$)				
W	131±3	126±3**	127±3*	122±3**°
C	127±5	128±5	125±4	125±5
R				
W	0.91±0.01	0.90±0.01	0.91±0.01	0.91±0.01
C	0.88±0.02	0.88±0.01	0.91±0.01	0.91±0.02
RPE				
W	11.6±0.04	10.8±0.05	10.2±0.05	9.8±0.05**
C	11.4±0.08	11.7±0.07	11.1±0.08	11.4±0.08

Significantly different from baseline *$P<0.05$, **$P<0.01$.
Response of walkers significantly different from controls, interaction °$P<0.05$.



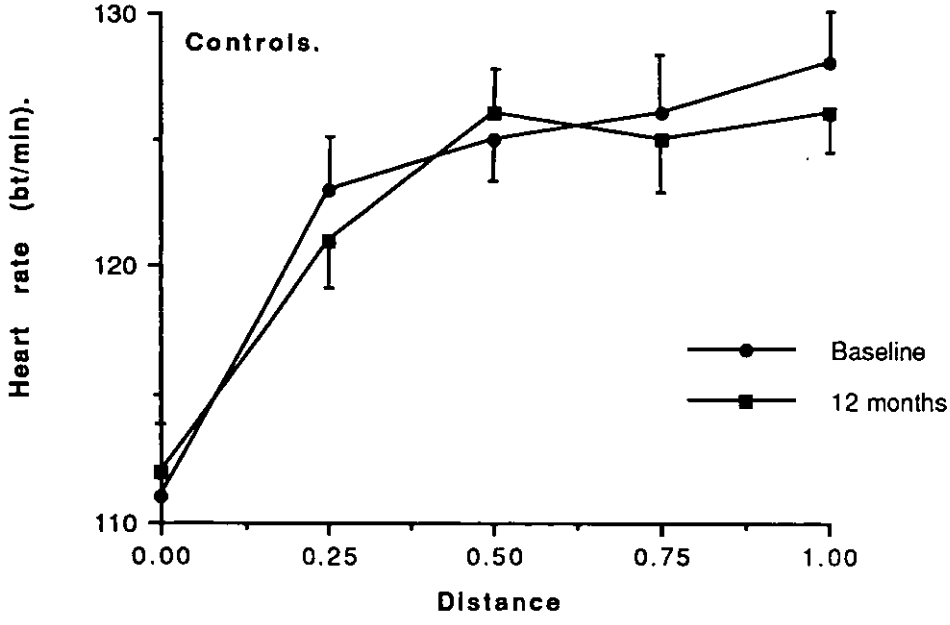


FIGURE 6:2. Average heart rate at intervals during a one mile submaximal treadmill walk at baseline and 12 months for middle-aged female walkers (n=28) and controls (n=16).

No differences were seen between the groups for R value, either at baseline or at any successive point during the programme. However, differences were observed for the rate of perceived exertion (RPE), which decreased for walkers at 12 months as compared with baseline values ($P<0.01$). No differences were observed for the control group on either measure.

No difference was observed between the groups at baseline for $\dot{V}O_2$ consumed during the submaximal treadmill walk (Table 6:4). However both groups there was shown to be a trend towards increase from the baseline measurement (1.0 $ml.kg.^{-1}min^{-1}$ and 2.6 $ml.kg.^{-1}$ min^{-1} for walkers and controls respectively). However, at 12 months the difference between the groups remained statistically non significance.

When expressed as a percentage of predicted $\dot{V}O_2max$ the rate of oxygen consumed during the submaximal walk at 12 months was lower than at baseline for walkers (4.1%). This change is considered indicative of a decrease in the relative intensity of the standardised exercise test consequential to training. For controls, however there was a 3.8% increase for this measure (Table 6:4).

Table 6:4 also contains information regarding blood lactate concentration measured at the completion of the treadmill walk (Figure 6:3). No difference was observed at baseline or throughout the programme between the two groups. Furthermore no significant interaction effect was discerned. However, blood lactate concentration for walkers at 12 months was 0.4 $mmol.l^{-1}$ (or 18%) less than at baseline and was significantly correlated with the reduction of heart rate elicited during the submaximal walk ($r=0.47$, $P<0.05$). In contrast blood lactate concentration for the control group was increased by a similar magnitude 0.3 $mmol.l^{-1}$ (or 20%) over the same

Table 6:4. Average oxygen consumption, oxygen consumption as a percentage of predicted maximal oxygen consumption, blood lactate concentration at rest and at the end of the submaximal treadmill walk for walkers (W n=28) and controls (C n=16) (Mean±SEM).

	Baseline	3 Months	6 Months	12 Months
$\dot{V}O_2$ ($ml.kg^{-1}min^{-1}$)				
W	16.5±0.6	16.1±0.6	17.7±0.5*	17.5±0.5*
C	15.4±0.8	18.3±0.9**	17.9±1.0*	17.6±0.9*
$\%\dot{V}O_2max$				
W	62.1±2.9	57.9±2.7*	60.6±1.9*	58.0±2.2*
C	58.0±2.1	63.2±2.3**	61.9±2.6*	61.8±2.9*
La at rest ($mmol.l^{-1}$)				
W	0.71±0.06	0.72±0.06	0.71±0.06	0.69±0.04
C	0.65±0.10	0.69±0.08	0.74±0.10	0.68±0.07
La end ($mmol.l^{-1}$)				
W	2.27±0.20	2.37±0.17	2.09±0.17*	1.86±0.15** ⁰⁰
C	1.67±0.23	2.28±0.25*	2.30±0.21*	2.00±0.19*

Significantly different from baseline *P<0.05, **P<0.01.
Response of walkers significantly different from controls, interaction ⁰⁰P<0.01.

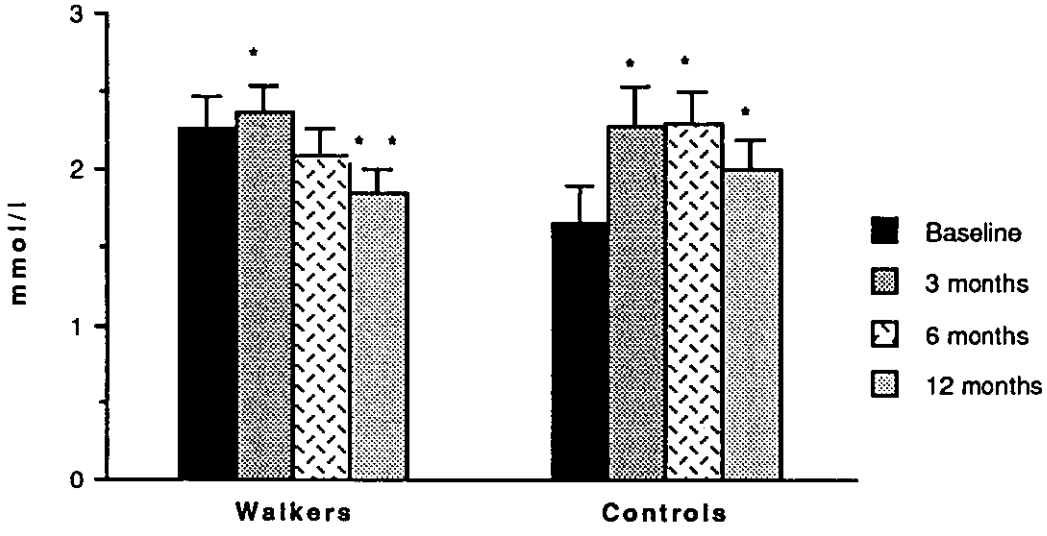


FIGURE 6:3. Concentration of blood lactate at the end of a one mile submaximal treadmill walk at baseline and intervals throughout the one year programme for middle-aged female walkers (n=28) and controls (n=16).

Significantly different from baseline *P<0.05, **P<0.01.

period.

Maximal oxygen uptake, predicted from heart rate and $\dot{V}O_2$ during the step-test, did not differ between groups at the commencement of training (Table 6:5). Furthermore, no change was seen for controls over the duration of the study and no interaction effect was discerned. The $\dot{V}O_2max$ for walkers however, was found to be 3 $ml.kg^{-1}.min^{-1}$ (or 11%) higher at the completion of the training programme. Sixty percent of this increment was achieved within the first three months of the programme, after which the rate of increase was reduced. As with the other indices of physiological adaptation to training, oxygen uptake measured at a blood lactate concentration of 2 $mmol.l^{-1}$ was similar for both groups prior to the commencement of training. Furthermore, no change was observed throughout the study for controls (Table 6:5). However, at 12 months the volume of oxygen consumed by walkers at this reference lactate concentration was increased from baseline by 2.7 $ml.kg.^{-1}$ min^{-1} (or 15.9%). Furthermore, at the end of the study, the difference between the two groups was 2.1 $ml.kg.^{-1}min^{-1}$ ($P<0.05$) (Figure 6:3). However, when expressed as a percentage of $\dot{V}O_2max$ utilized prior to the attainment of a reference blood lactate concentration of 2 $mmol.l^{-1}$ no difference was seen as a consequence of training.

Table 6:5. Predicted maximal oxygen uptake ($ml.kg.^{-1}min^{-1}$), oxygen uptake at a reference blood lactate concentration of $2mmol.l^{-1}$ and oxygen uptake at $2\ mmol.l^{-1}$ as a percentage of the predicted maximal oxygen uptake, from the step-test procedure for walkers (W n=28) and controls (C n=16). (Mean±SEM).

	Baseline	3 Months	6 Months	12 Months
$\dot{V}O_2$ max				
W	26.7±0.7	28.5±0.8*	29.2±0.7**	29.7±0.8**°
C	27.6±1.0	27.6±0.9	27.5±1.0	27.7±1.2
$\dot{V}O_2$ at $2mmol.l^{-1}$				
W	17.0±0.5	17.9±0.6	19.0±0.6**	19.7±0.6**°
C	17.8±0.9	17.8±1.0	17.8±0.9	17.6±0.7
%$\dot{V}O_2$ max at $2mmol.l^{-1}$				
W	65.5±1.5	62.8±1.2	65.3±1.3	66.2±1.2
C	64.1±1.7	63.7±1.8	64.4±1.9	64.1±2.0

Significantly different from baseline *$P<0.05$, **$P<0.01$.

Response of walkers significantly different from controls, interaction °$P<0.05$.

6.3.2. Plasma TC and HDL-C concentration.

Analysis of the plasma samples revealed no difference between the groups for TC concentration at baseline or at any subsequent point throughout the duration of the programme. However, a small (0.35 $mmol.l^{-1}$ or 6.5%) but significant decrease in TC concentration was measured at 12 months for walkers. No change was found for controls (Table 6:6) (Figure 6:4).

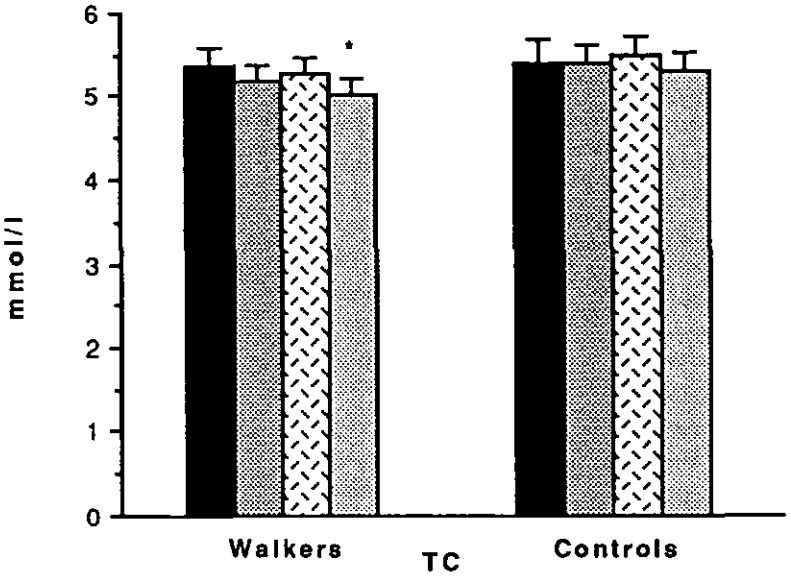
Similarly, no differences were found between the groups at baseline for the concentration of HDL-C. However, over the 12 months of the study, HDL-C concentration increased for walkers: 0.32 $mmol.l^{-1}$ or 27%. Of the overall increase 0.23 $mmol.l^{-1}$ (or 72% of the increase) occurred within the first three months of the programme. This was followed by successive, but smaller increases which occurred over the remaining period.

Data relating to the ratio of TC:HDL-C, sometimes termed the coronary risk ratio (Shaper 1987), is also included in Table 6:6. At baseline, the controls had a more favourable (that is, lower) ratio than walkers. However, at the end of the study this situation was reversed. The largest change in TC:HDL-C for walkers was measured at 3 months, when a decrease of 1.4 or 26.9% was observed from the baseline value. Over the remainder of the programme, the ratio was further decreased, amounting at 12 months to a total reduction of 1.7 (32.7%). Furthermore, as at baseline, there was a difference between the groups at the end of the training period.

Table 6:6. Plasma total cholesterol (TC), high density lipoprotein cholesterol (HDL-C) concentrations and TC:HDL-C ratio, at baseline and intervals throughout the programme for walkers (W n=28) and controls (C n=16) (Mean±SEM).

	Baseline	3 Months	6 Months	12 Months
TC ($mmol.l^{-1}$)				
W	5.35±0.23	5.18±0.20	5.28±0.18	5.00±0.22*
C	5.41±0.26	5.41±0.21	5.50±0.20	5.29±0.24
HDL-C ($mmol.l^{-1}$)				
W	1.17±0.08	1.40±0.06**	1.42±0.06**	1.49±0.06**
C	1.32±0.07	1.35±0.06	1.33±0.06	1.35±0.05
TC:HDL-C				
W	5.2±0.4	3.8±0.2**	3.9±0.2**	3.5±0.2**
C	4.2±0.2+	3.9±0.2	4.4±0.2	4.4±0.3+

Significantly different from baseline *$P<0.05$, **$P<0.01$.
Significantly different from Walkers +$P<0.05$.
Response of walkers significantly different from controls, interaction $^{00}P<0.01$.



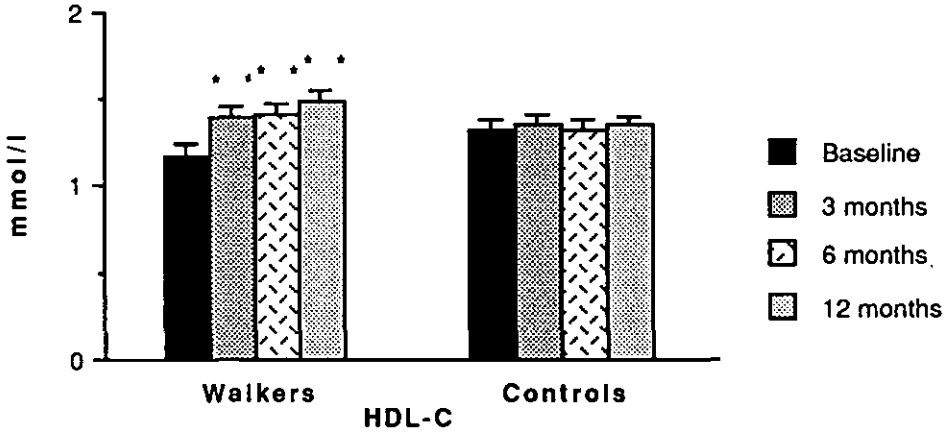


FIGURE 6:4. Concentrations of plasma total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) at baseline and intervals throughout the one year programme for middle-aged female walkers (n=28) and controls (n=16).

Significantly different from baseline *P<0.05, **P<0.01.

6:3:3. Body composition and diet.

No significant differences were discerned between the groups either prior to, or after the commencement of training, for the measures of body composition and subcutaneous fat. It was found however, that over the duration of the programme, there were trends for both the sum of four skinfolds and the ratio of waist to hip circumference to be lower for walkers (Table 6:7).

Information from the analysis of the dietary records is contained in Table 6:8. As illustrated in Figure 6:5 no differences were found between the groups at baseline or throughout the duration of the study for average daily energy intake, percentage of average daily energy as fat, or as carbohydrate. Furthermore, no differences were observed within the groups throughout the programme. However, a difference was observed for the percentage of average daily energy consumed as alcohol. At baseline the amount consumed by the control group was greater than that of the walkers, this trend was maintained throughout the study.

6.3.4. Psychological well-being.

The results of the analysis of the self-report measures are included in Table 6:9. At baseline there were no differences between the groups. The control group was unchanged throughout the programme on all three indices of psychological well-being.

However, favourable changes were identified for the walkers. Scores derived from the self-esteem questionnaire increased throughout the programme, and achieved significance at 12 months. No differences were observed for the comparisons for the GHQ and STAI, however favourable trends were observed for both measures with training.

Table 6:7. Body mass, BMI, sum of four skinfolds, W:H ratio, body density and percentage of body fat derived from body density measurements, at baseline and intervals during the study for walkers (W) and controls (C) (n=28 W, n=16 C; +n=22 W, n=13 C) (mean±SEM).

	Baseline	3 Months	6 Months	12 Months
Body mass (kg)				
W	64.0±1.7	63.4±1.7	64.4±1.7	64.3±1.6
C	61.2±2.3	61.1±2.2	61.6±2.3	61.7±2.5
BMI ($kg.m^{-2}$)				
W	23.9±0.6	23.7±0.6	24.0±0.6	24.0±0.7
C	24.0±0.7	24.0±0.9	24.2±1.0	24.2±1.0
Sum of four skinfolds (mm)				
W	63±4	62±4	62±3	61±4
C	63±5	63±5	64±5	64±6
W:H ratio				
W	0.79±0.01	0.77±0.01	0.76±0.01	0.75±0.01
C	0.79±0.01	0.79±0.02	0.76±0.02	0.77±0.02
+ Body density ($kg.m^{-3}$)				
W	1019±3			1017±3
C	1021±4			1021±4
+ % Body fat				
W	36±1			37±1
C	35±2			35±2

Table 6:8. Average daily energy intake, percentage of daily energy intake as fat, carbohydrate and alcohol at baseline and at intervals during the study for walkers (W n=28) and controls (C n=16) (mean±SEM).

	Baseline	6 Months	12 Months
Average daily energy intake (MJ)			
W	7.7±0.4	7.6±0.4	7.4±0.4
C	6.9±0.3	7.0±0.3	7.0±0.3
% Energy as fat			
W	33.8±0.9	34.5±0.9	34.0±0.7
C	33.5±1.5	33.1±1.6	34.6±1.3
% Energy as CHO			
W	48.2±1.2	48.1±1.2	47.7±1.1
C	45.0±1.8	47.1±1.4	45.3±1.8
% Energy as alcohol			
W	1.9±0.4	1.3±0.5	1.5±0.4
C	5.5±1.4†	3.1±0.9	3.4±1.0

Significantly different from Walkers † $P<0.05$.

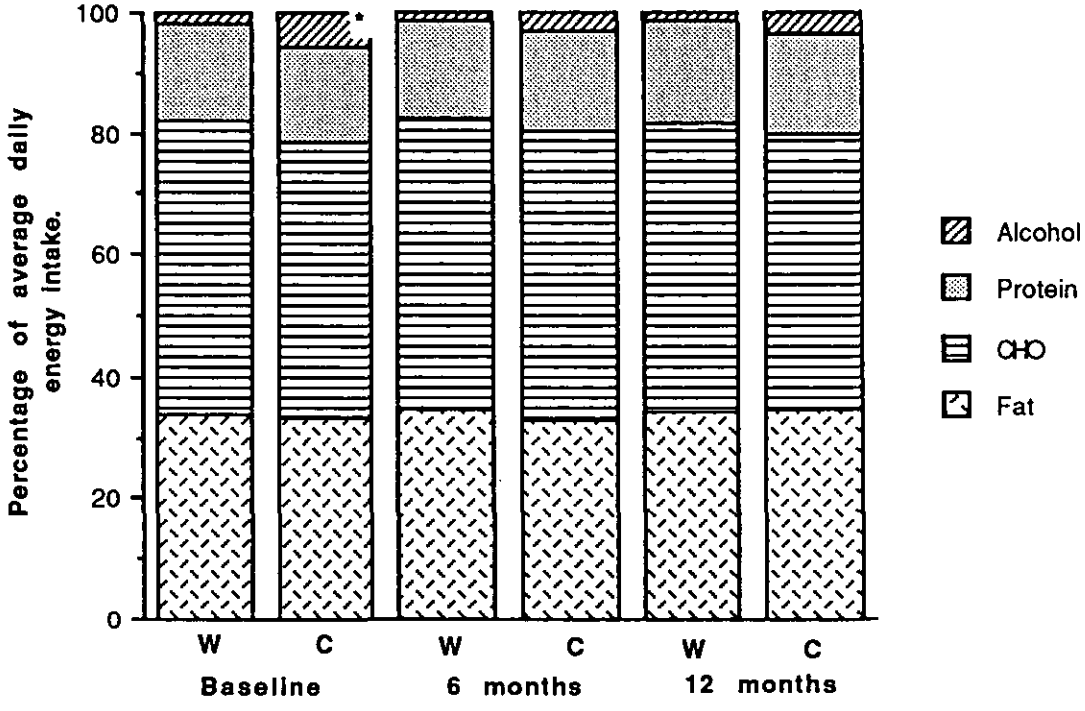


FIGURE 6.5. Percentage of average daily energy intake as fat, carbohydrate, protein and alcohol at baseline, 6 and 12 months of the one year programme for middle-aged female walkers (n=28) and controls (n=16).

Significantly different from Walkers *P<0.05.

Table 6:9. Results of the analyses of the self-esteem questionnaire, the general health questionnaire (GHQ) and the state-trait anxiety inventory (STAI) for walkers (W n=28) and controls (C n=16) (mean±SEM).

	Baseline	3 Months	6 Months	12 Months
Self-esteem				
W	32.9±0.9	33.0±0.9	33.3±0.9	34.2±0.9*
C	30.9±1.6	30.7±1.5	31.2±1.6	31.0±1.6
GHQ				
W	10.3±0.8	10.4±1.2	10.0±1.0	9.3±0.8
C	11.8±1.1	11.6±1.1	11.8±1.1	11.9±1.7
STAI				
W	38.5±2.0	36.5±1.6	36.4±1.7	36.7±1.9
C	41.1±2.4	40.1±2.5	41.3±2.4	40.6±2.9

Significantly different from baseline * $P<0.05$.

6.4. Discussion.

The results of the present study provide further evidence for the benefits of habitual exercise. More specifically, the observations suggest that brisk walking can have a favourable influence on functional capacity, TC and HDL-C concentration, as well as on aspects of psychological well-being.

A particular benefit derived from the training programme was the ability of walkers (but not controls) to perform at a higher intensity of self-paced exercise. This was shown by successive increments in pace attained during the maximal track walk associated with increases of $\dot{V}O_2$ max. Furthermore, the walkers were able to exercise at a heart rate which was 12 $bt.min^{-1}$ or 6.9% higher than that elicited during the pre-training walk. These results are consistent with those reported by Pollock and coworkers (1971) for a study of the effects of walking with middle-aged men.

From these and similar observations it would appear that within a range of $\dot{V}O_2$ max values, walking is an appropriate form of exercise to increase functional capacity. That is, as the functional capacity of the sedentary individual improves, the pace (intensity) at which the training is completed can also be increased thereby achieving the new threshold level required for further improvement. This characteristic was exemplified in the present study by the successive reductions in the time required to complete the maximal walk. Schultz (1980) therefore concludes:

> "Walking can be as beneficial as any regular activity over the long term, or it can serve as gradual training for more strenuous exercise, particularly for sedentary, out-of-shape adults just

starting a conditioning programme."

(Schultz 1980).

In addition to the maximal track walk, a one mile submaximal walk was completed. The purpose of the latter was to identify the pace adopted during training. Studies of self-selected walking pace have revealed the important contributions made by age and $\dot{V}O_2$max (Cunningham et al. 1982). As a result of such observations some authors, for example Bassey (1978), have attributed the decline in self-selected pace associated with older individuals to the ageing process *per se*. However others, for example Cunningham and colleagues (1968), consider the pace selected to be "associated with maximal aerobic power independent of age". This latter proposal is consistent with the observation that appropriate exercise training can provoke at least a partial recovery of $\dot{V}O_2$max with older adults, thereby providing evidence of a dissociation between age and $\dot{V}O_2$max (Astrand et al. 1973; Cunningham et al. 1968; Dehn & Bruce 1972; Moore 1975; Robinson et al. 1973, 1975).

Consistent with the aforementioned proposal for the role of $\dot{V}O_2$ max as a determinant of self-selected pace the results of the present study showed the existence of a positive correlation between self-selected walking pace and predicted $\dot{V}O_2$ max for the walkers ($r=0.62$, $P<0.01$). Furthermore a similar relationship between submaximal pace and age could not be identified. It might therefore be concluded that for this group of women submaximal walking pace was determined by functional capacity rather than age. Therefore, improved functional capacity as indicated by $\dot{V}O_2$ max, is associated with an increased ability to tolerate a higher submaximal pace.

In addition to changes of pace, it was also observed that, after exercise training, individuals were able to

perform at the same absolute intensity with less physiological stress. This adaptation was reflected in changes of heart rate and oxygen consumption when expressed as a percentage of $\dot{V}O_2max$ and is consistent with the results of other studies in which walking was also adopted as the mode of training (Santiago et al. 1987; Flint et al. 1974; Pollock et al. 1971; Jetté et al. 1988). In each of the aforementioned investigations heart rate elicited during submaximal exercise was shown to decline from the pre-training level. The magnitude of these decrements ranged from 4 to 17 $bt.min^{-1}$ (8 to 11%). These observations are therefore supportive of the 9 $bt.min^{-1}$ (7%) decrease reported for the present study.

The effect of training on average heart rate elicited during the submaximal treadmill walk however was not homogenous. Evidence for this non-uniformity is revealed by the range of change measured from +12 to -29 $bt.min^{-1}$. It is therefore suggested that the effectiveness of the brisk walking programme varied considerably. A more pronounced increase being attained by individuals with a low functional capacity at baseline. It is therefore possible that additional factors such as differences in age, baseline heart rate or training pace might have influenced the adaptive response. Thus, as previously discussed (Chapter 4) brisk walking appears to be an adequate stimulus for many sedentary individuals, but not for others. This variable effect would therefore be expected to limit the average decrease observed and might account for the relatively small reduction in heart rate measured during the present study when compared with some of the previous investigations.

In addition to heart rate, blood lactate concentration is also considered indicative of an adaptation to exercise training. As a consequence changes in blood lactate concentration were monitored throughout the present

study. The analysis of capillary blood samples obtained at rest revealed no difference for the concentration of lactate with training. This observation is consistent with suggestion made by Ekblom and colleagues (1968) that resting blood lactate concentration is largely unaffected by exercise training. In contrast, at the same absolute exercise intensity blood lactate concentration is generally reduced after training (Bang 1936; Ekblom et al. 1968; Karlsson et al. 1972) a suggestion which is again consistent with the results of the present study. In addition to resting and post-exercise values, the volume of oxygen consumed at a reference blood lactate concentration of 2 $mmol.l^{-1}$ was also employed to indicate changes in aerobic capacity (Sjödin & Svendenhag 1985; Faria 1984; Denis et al. 1982; Hurley et al. 1984).

Changes in lactate related indices are commonly attributed to training-induced increments in the oxidative capacity of skeletal muscle bed (Holloszy & Coyle 1984; Henriksson & Reitman 1977). They are considered indicative of an improved ability of aerobic processes to meet the energy demands of the exercise (Wenger & MacNab 1975). A reduction of the concentration of lactate in blood, or increase in the volume of oxygen consumed at a reference blood lactate concentration at the same absolute exercise intensity therefore signify a decreased reliance on anaerobic glycolysis (Saltin & Karlsson 1971; Astrand & Rodahl 1986). The 16% increase in $\dot{V}O_2$ at 2$mmol.l^{-1}$ lactate concentration and the 18% decrease in blood lactate concentration measured at the end of the treadmill walk reported for the present study, are therefore consistent with previous results.

Functional capacity, as indicated by $\dot{V}O_2max$, was also found to improve with training. The increase of 11.2% for walkers is consistent with the results of other studies in which low intensity training has been adopted. For

example, Santiago and coworkers (1987) compared the effects of walking and jogging and found no difference between the groups for the increase in $\dot{V}O_2$ max incurred (21% and 31% for walkers and joggers respectively). Gaesser and Rich (1984) reached a similar conclusion from their comparison of the effects of high and low intensity training in young men.

The extent of the $\dot{V}O_2$ max increase observed in the present study (11.2%) was less than that reported for the above investigations. However, several other studies have discerned increases in $\dot{V}O_2$max more comparable with the present data (Brownell et al. 1982; Franklin et al. 1976; Kilbom 1971; Sidney & Shephard 1978). Saltin and colleagues (1968) suggested that differences in the effectiveness of exercise training might be a consequence of differences at baseline. It was therefore proposed that baseline and post-training levels have an inverse relationship. Thus a low pre-training $\dot{V}O_2$ max might confer a greater potential for change (Sharkey 1970; Pollock et al. 1971). Furthermore, differences in training protocol might also be an important determining factor.

This latter point is exemplified by the comparison of the present study with that of Jetté and coworkers (1988), who reported a 16.5% increase in $\dot{V}O_2$max after 12 weeks of walking. The observation of this larger increase derived after a shorter training period might be explained by differences in protocol. For example, the training completed in the present study was conducted in the absence of any experimenter supervision. In contrast, in the study by Jetté all the training was completed on a treadmill at 60% $\dot{V}O_2$ max. Furthermore, after 6 weeks $\dot{V}O_2$max was re-assessed and a pace equivalent to 60% of the new $\dot{V}O_2$ max was adopted. This intervention therefore ensured that subjects always trained at, or above, the threshold intensity for adaptation (ACMS 1986), resulting in a

greater likelihood of favourable physiological adaptation occurring. However, the advantage of the present protocol was its resemblance to 'real-life' changes in exercise habits.

When the pattern of the improvements of $\dot{V}O_2max$ for the present study is considered, 60% of the total increase occurred within the first three months of training. After this initial period, the rate of improvement declined, leading to a plateau effect. This is consistent with the observations of higher intensity training (Daniels et al. 1978; Pollock 1973, 1978; Saltin et al. 1968).

> "...$\dot{V}O_2max$ improves with training but reaches a plateau after several months, whereas performance continues to improve."
>
> (Londeree 1986).

This statement suggests that changes in $\dot{V}O_2max$ provoked by high intensity training decelerate, whilst performance continues to improve. In the present study however, the rate of increase in both parameters declined as training progressed. It is possible therefore that the potential for improvement with low intensity exercise is quickly attained. Thus further increases of $\dot{V}O_2max$ might be limited by the inability to increase walking pace. in this situation the adoption of a walk/jog pattern might be suggested. Alternatively, walking might be adopted as a maintenance rather than improvement activity

Increases of $\dot{V}O_2$ at the reference blood lactate concentration of 2 $mmol.l^{-1}$ and of predicted $\dot{V}O_2max$ together suggest that after exercise training subjects were able to work at the same absolute exercise intensity with less physiological stress and fatigue. In addition to these physiological benefits, favourable changes were also identified for the concentration of TC and HDL-C.

For example, a comparison of the analysis of plasma samples for TC revealed a small, but significant decrease for the walkers over the duration of the study. This observation is supported by the results of a number of previously completed studies, but the literature is rather inconsistent. Thus Altekruse and Wilmore (1973) and Lopez and coworkers (1974) both reported a reduction in the concentration of TC for young healthy males after endurance training. However, other studies with young males, for example Allison and coworkers (1981) and Leon and colleagues (1979) failed to identify any change in TC concentration. Furthermore, similar reports of the ineffectiveness of endurance exercise to reduce TC concentration have been presented for middle-aged men (Freyman et al. 1982; Holloszy et al. 1964; Lewis et al. 1976). Whilst the evidence for the effects of endurance exercise on TC concentration remains inconclusive, less ambiguity surrounds the relationship between endurance training and changes in the concentration of HDL-C.

The concentration of HDL-C increased during the present study for walkers, 72% of the overall increase occurring within the first three months of the training programme. This increase is consistent with that reported for a number of other training studies (Lopez et al. 1974; Streja & Mymin 1979; Keins et al. 1980; Ballantyne et al. 1981; Leon et al. 1979; Rotkis et al. 1981). A smaller number of investigations however, have been unable to distinguish any change in HDL-C concentration (Allison et al. 1981; Savage et al. 1986; Lipson et al. 1980; Moore et al. 1979; Weltman et al. 1980).

In order to clarify the position, Wood and colleagues (1984) compiled a review of available studies. Of the 14 training studies which reported no change in HDL-C concentration, 11 were found to have a duration of less than 10 weeks. In addition, where an increase in HDL-C

concentration was identified, the training tended to be maintained over a period of months rather than weeks. It has therefore been proposed that a possible threshold for the duration of a training programme might exist, the attainment of which is necessary for any increase in the concentration of HDL-C (Wood et al. 1983). Further evidence exists to support this for example, Wood and coworkers (1983) completed a study of middle-aged men involved in a one year programme of running. No overall change in the concentration of HDL-C was determined for the 48 subjects who completed the programme. However, for the 25 men who attained an average of 8 (12.9 km) or more miles of training per week, an increase of HDL-C concentration was indentified. Furthermore, a significant correlation was found to exist between the distance run per week and change in HDL-C (r=0.48).

The threshold for HDL-C increase suggested by Haskell (1984) was related to a minimum energy expenditure of 1,000 kcal per week on moderate intensity exercise. Similarly Hartung and Squires (1980) observed that individuals who completed 11 or more miles of running per week experienced increases in HDL-C concentration. The increases being positively correlated with the distance completed. Together these observations provide evidence in support of the proposal that the relationship between HDL-C concentration and exercise training is not a simple linear function. The identification of a more complex relationship between these two parameters might therefore at least partially explain the inconsistency of results derived from different studies.

The results of the present study also support the suggestion of the possible existence of a threshold effect. For example, of the 28 women who completed the year long training programme, 23 attained an average of 125 minutes (9 miles) or more per week. Furthermore, the

increase in HDL-C concentration for this group was positively associated with time spent walking ($r=0.44$, $P<0.05$). This observation therefore closely corresponds with that reported by Wood and colleagues (1983).

The 27% increase in HDL-C concentration observed during the present study is somewhat larger than that reported for previous training studies (Streja & Mymin 1979; Erkelens et al. 1979; Huttunen & Froberg 1979). This disparity might be explained by differences in the duration of the various studies and/or as a consequence of differences in pre-training HDL-C concentrations. For example, Sutherland and Woodhouse (1980) recognised that changes in HDL-C were often dependent on the baseline concentration. Thus an initially low concentration might be expected to confer a greater potential for change.

The concentration of TC at baseline in the present study was 5.35±1.2mmol.l^{-1} and 5.41±1.38 mmol.l^{-1} for walkers and controls respectively. These figures appear consistent with (i.e. within 90th centile) the data reported by Mann and colleagues (1988) in a recently published survey of similarly aged British women. Whilst the concentration of TC was seen to change with age (Table 6:10), no age-related change was reported for the concentration of HDL-C. The average concentration of 1.6±0.4 mmol.l^{-1} reported by Mann and coworkers (1988) appears higher than those determined for the present groups (1.17±0.42 mmol.l^{-1} and 1.32±0.37 mmol.l^{-1} for walkers and controls respectively). However, there were differences in the methodology used for the precipitation of VLDL and LDL. Mann and colleagues (1988) used the heparin and manganese chloride technique whilst the precipitation procedure used in the present study involved phosphotungstic acid and magnesium chloride. However, irrespective of these differences, a relatively low pre-training concentration might enable greater change than would be expected with higher

Table 6:10. The relationship between plasma TC concentration and age in British women.

Age (yrs)	TC ($mmol.l^{-1}$) $\bar{x}$	sd
30-34	5.2	1.0
35-39	5.3	1.0
40-44	5.6	1.1
45-49	5.9	1.2
50-54	6.4	1.1
55-59	6.7	1.2

Mann and cowokers 1988.

baseline concentrations. This suggestion is substantiated by the results of the present study which revealed an inverse relationship between the concentration of HDL-C at baseline and HDL-C increase found at the end of the study ($r=-0.72$; $P<0.01$). A similar inverse relationship, but of smaller magnitude was identified for TC ($r=-0.42$ $P<0.05$).

Whilst evidence for this effect does exist it is perhaps pertinent to recall a discussion presented in Chapter 2 relating to the influence of diet on HDL-C metabolism. With reference to Table 6:8 it can be seen that the groups involved in the present study were similar in the percentage of average daily energy intake consumed as fat and as carbohydrate. However, at baseline the control group had a higher alcohol consumption than the walkers. Furthermore, this trend was maintained throughout the programme. The results of studies conducted by Johannson and Mehdus (1974) have led to the suggestion that for each 10 gm of alcohol consumed daily the concentration of HDL-C is elevated by 0.03-0.05 $mmol.l^{-1}$. On this basis the effect of alcohol consumption on the concentrations of HDL-C measured for the present groups has been estimated (Table 6:11). Although rather speculative the results of these estimates reveal that the baseline HDL-C concentrations for the two groups were very similar when corrected for alcohol intake. In addition, at 12 months the difference between the groups appears greater than when the influence of alcohol was ignored. Finally, when considered over the duration of the programme, HDL-C concentration for walkers was increased by an estimated 36%, as compared with 27% when the effect of alcohol was not considered.

It was previously suggested that a lower baseline HDL-C concentration might be associated with a greater chance of achieving a subsequent increase with endurance

Table 6:11. Plasma HDL-C concentration before and after estimating the effect of alcohol consumption for walkers (W n=28) and controls (C n=16).

	Baseline	6 Months	12 Months
Before			
W	1.17	1.42	1.49
C	1.32	1.33	1.35
After			
W	1.03	1.33	1.39
C	0.96	1.12	1.13

training. Therefore since the concentration of HDL-C for walkers was 0.15 $mmol.l^{-1}$ lower than that measured for controls it is possible that an increase was more easily attainable for the former group. However, when the effect of alcohol was estimated the baseline difference between the groups no longer existed (i.e. the baseline differences might be attributable to differences in alcohol intake). Thus the original results might be seen to under- rather than over-estimate the effectiveness of walking to increase HDL-C concentration.

A mechanism which might account for the exercise-induced changes in lipid metabolism are the adaptations which occur in the muscles. For example, it has been recognised that endurance training provokes an increase in muscle capilliarization (Saltin 1985; Saltin & Gollnick 1983). As a consequence, lipoprotein lipase located on the endothelium of this tissue is also increased (Nikkilä et al. 1978) thereby promoting the rate of lipolysis of Tgs contained in chylomicrons, VLDL and LDL. This enhanced rate of breakdown therefore provides an increase in the availability of remnant particles for the formation of HDL-C (Grosser et al. 1981; Nicholl & Lewis 1980). Increased muscle capillary density therefore has two major influences on HDL-C formation. Firstly, there is an increase in the provision of sites for the lipolytic activity of lipoprotein lipase, and secondly, a more efficient transportation and supply of chylomicrons and other substrates necessary for HDL-C formation (Ruys et al. 1989).

Ruys and coworkers therefore concluded:

> "The effect of exercise in raising HDL cholesterol, and the inverse relationship between exercise and CHD, may be partly the result of this process".
>
> (Ruys and coworkers 1989).

The above statement introduces the need to consider the implications of such changes relative to CHD risk. Trends relating high plasma concentrations of HDL-C and a decreased incidence of CHD have been observed in a number of prospective studies conducted in several countries (Gordon et al. 1977; Goldbourt & Medalie 1979; Keys et al. 1984; Pocock et al. 1986).

In the Coronary Primary Prevention Trial (CPPT) and Multiple Risk Factor Intervention Trial (MRFIT) a 1.0 $mg.dl^{-1}$ (0.026 $mmol.l^{-1}$) increase in the concentration of HDL-C was associated with a reduction in CHD risk of 2% in males and 3% in females. In the Lipid Research Clinics Prevalence mortality follow-up, a similar (1.0 $mg.dl^{-1}$ or 0.026 $mmol.l^{-1}$) increment was associated with a 3.7% decrease of CHD risk in men and 4.7% decrease in women. Furthermore the 95% confidence intervals for the decrease in CHD risk observed in the above studies overlapped extensively and all contained the range 1.9-2.9% (Gordon et al. 1989). Based on the above epidemiological evidence the 0.32 $mmol.l^{-1}$ increase of HDL-C incurred in the present study would be expected to produce a 54-64% decrease in CHD risk. This decrease is further emphasized by the observation of a favourable decrease of the ratio of TC:HDL-C, a ratio which Shaper (1987) has suggested is the only significant correlate of interpopulation variation in CHD mortality for women ($r=0.56$, $P<0.04$).

The recognition of the relationship between blood lipid and lipoprotein metabolism and the potentially favourable effect of endurance exercise, necessitated the close monitoring of those factors also suspected of affecting the concentration of TC and HDL-C, for example body composition.

Reduced body mass and percentage body fat has been reported to be associated with increases in the

concentration of HDL-C (Wilson & Lees 1972; Contaldo et al. 1980; Streja et al. 1980; Sorbris et al. 1981; Caggiula et al. 1981). In addition, increases in the same parameters of body composition have also been linked with decreases in HDL-C concentration (Wilson & Lees 1972; Contaldo et al. 1980). Body composition and anthropometrical measurements were therefore made at intervals throughout the present study.

No differences were discerned over the period of the study for either of the groups, an observation consistent with those derived from several studies which have also investigated the effect of low intensity training in women. A number of the studies have reported body mass to remain unchanged with training (Flint et al. 1974; Johannessen et al. 1986; Santiago et al. 1987; Atomi & Miyashita 1980; Farrell & Barboriak 1980; Girandola 1976). Similar reports have also been made for percentage body fat (Vaccaro & Clinton 1981; Zwiren et al. 1973; Smith & Stransky 1976; Santiago et al. 1987; Johannessen et al. 1986) and also for skinfold thickness (Johannessen et al. 1986; Adams & de Vries 1973; Santiago et al. 1987).

Other investigations however, for example those completed by Lewis and coworkers (1976); Zuti and Golding (1973); Wynne and colleagues (1980); Massicotte and coworkers (1979); Frey and colleagues (1982) and Gwinup (1987) have reported a decrease in some, or all, of these parameters after a programme of walking.

The inconsistency between these observations makes any conclusion of the effectiveness of low intensity exercise to alter body composition particularly difficult. The interpretation of results is also made more complicated by intervening variables such as the relationship between the degree of change and the pre-training measurements

(Lewis et al. 1976; Zuti & Golding 1973). As was previously recognised for changes in $\dot{V}O_2max$ and for TC and HDL-C concentrations, an inverse relationship exists between the degree of change and pre-training level. A similar relationship is also apparent for measurements of body composition. For example, many studies which have reported changes in anthropometrical parameters have been completed with obese individuals (BMI >35) (Leon et al. 1979; Lewis et al. 1976; Zuti & Golding 1973). However, no change was apparent for the present group in which BMI was within the normal range for British females as reported by Mann and coworkers (1988) and Rosenbaum and colleagues (1985). It would therefore appear that subjects of relatively normal body mass are less likely to experience any decrease.

Inspection of the analysis of food records further complicates the interpretation of the results. As exercise participation is associated with increased energy expenditure it might be expected that in the absence of weight loss a compensatory increase in energy intake may have occurred. However, such an increase was not apparent from the analysis of the food diaries (Table 6:8). As Tremblay and coworkers describe:

> "It is generally accepted that the regulation of body energy reserves functions in accordance with the first law of thermodynamics, i.e. that the changes in body energy are determined by changes in energy intake and energy expenditure. Thus to maintain a constant level of body energy reserves, food energy available for metabolic needs (metabolisable energy) must be equal to the energy expended for the various physiological processes of the organism."
>
> (Tremblay & coworkers 1985).

During the present study energy expenditure was estimated using an indirect calorimetry approach. The volumes of carbon dioxide produced and oxygen consumed were measured at rest and during the submaximal treadmill walk. This information was then used to calculate the net cost of walking for each individual. Inspection of the exercise diaries revealed the total number of minutes of training completed by each individual throughout the study. It was therefore possible to estimate the energy cost incurred with training along with the approximate decrease in body mass which might be expected (5.0 to 5.2 kg) in the absence of a compensatory increase in energy intake. However the data contained in Table 6:7 suggests that the expected decrease in body mass failed to occur. Thus in the apparent absence of an increase in energy intake to balance the energy cost incurred during training some additional factor must exist to explain the stability observed for body mass.

It is possible for example, that an increase in energy intake did occur, but went undetected due to inadequacies in the dietary recording technique. For example, Block (1990) observed that as the period over which the diet was recorded progressed, subjects tended to increasingly under-report. It has also been proposed that subjects become less accurate in measuring and recording their dietary intake (Gersovitz et al. 1978; Livingstone et al. 1990) and that even highly motivated subjects alter their dietary behaviour as a consequence of record keeping (Bingham 1987; Southgate 1986). If one, or all, of these actions were adopted during the present study any increase in energy intake for the walkers might be disguised. Furthermore these might account for the actual decline in energy intake observed over the series of measurements for the walkers. However, in an attempt to minimize the effect of these factors both walkers and controls were made fully aware of the accuracy needed

during the recording period and the importance of the results was emphasized throughout the investigation. Furthermore, the occurrence of such factors fail to explain why the energy intake for the control group remained unchanged. To be the basis of the observed effect, it appears that the walkers were more sensitive to the weaknesses of dietary recording than their sedentary counterparts, the controls, which would seem unlikely.

A second possibility is the existence of a threshold of energy expenditure, the attainment of which is necessary prior to any change in body composition. Franklin and Rubenfire (1980) proposed a minimal threshold level for weight loss and reduction of body fat, which required the expenditure of 300 kcal. or more, per session of continuous exercise.

Using the estimated values for energy expenditure derived from the method of indirect calorimetry it is suggested that in order to attain this threshold level of expenditure, individuals involved in the present study would have to walk 3.8 miles or 58 minutes per session. However, to maintain exercise intensity, as well as to minimize the incidence of orthopaedic injury (Johannessen et al. 1986; Rippe et al. 1986; Schultz 1980) training sessions were limited to approximately 50 minutes. It is therefore unlikely that the threshold for decrease was achieved.

It has also been proposed that exercise-induced increases in resting metabolic rate (RMR) might be of greater importance for weight loss than the level of energy expenditure directly caused by exercise *per se*:

> "The energy cost of exercise is not simply the energy expended during the exercise, since resting

> metabolic rate is substantially increased for many hours after exercise has ceased, thus greatly increasing the true cost of exercise".
>
> (Garrow 1988).

Studies reporting a sustained increase in RMR have tended to involve relatively high intensity exercise, that is 70-75% $\dot{V}O_2$ max (Edwards et al. 1935; de Vries & Gray 1963; Hermansen et al. 1984). However, for lower intensity or short duration exercise, a similar effect was not observed (Knehr et al. 1942; Swindells 1972; Blaza & Garrow 1983). It is unlikely therefore that the present training, which it is assumed was completed at an approximate intensity of 60% $\dot{V}O_2$ max and for less than 60 minutes, would be sufficient to provoke weight loss, irrespective of whether the proposed threshold was related to energy expenditure, exercise duration, exercise intensity or elevations of RMR.

Prentice and coworkers (1985) used the doubly-labelled water technique to investigate the energy expenditure of normal, healthy women. From the results it was concluded:

> "that it is possible to maintain energy balance at much lower levels of intake than has hitherto been acknowledged...and leave considerable energy available for physical activity."
>
> (Prentice & colleagues 1985).

If proved correct, this might indicate the existence of a degree of 'surplus capacity' in the energy balance system (Prentice et al. 1985). Therefore, in the likely absence of any prolonged elevation of RMR, the 'buffer capacity' might be sufficient to protect against weight loss and therefore to maintain homeostasis, possibly therefore accounting for the absence of change in the present study.

Finally, it remains a possibility that the women involved in the study did not develop an energy deficit as walking was used to replace other activities requiring a similar energy cost. For example it is possible that individuals might have opted to walk to work rather than cycling as they might have done prior to the training period. However, this suggestion does not appear to be supported by the results of the physical activity questionnaire. The scores for the walkers increased with training, over and above the baseline level. Thus suggesting that the walking was completed in addition to, rather than instead of, alternative activities.

To conclude a number of possibilities exist to explain the observed effects on energy balance. Firstly, an increase in energy expenditure may have occurred, but was not identified from the analysis of food records due to the influence of under-reporting and/or to inaccuracies in measurement and recording (Block 1988). Secondly, the intensity of the exercise might have been insufficient to attain the proposed threshold of energy expenditure (Franklin & Rubenfire 1980) or increased resting metabolic rate (Blaza & Garrow 1983) to provoke a decrease in body mass. Finally, an 'energy surplus' or 'buffer capacity' (Prentice et al. 1985) might exist to maintain the balance in the energy system and thus protect the individual from loss of body mass.

It has been suggested (Kannel 1987) that the control of body mass might be of major importance for the improvement of metabolic factors involved in the promotion of atherogenesis. Furthermore, that central weight gain may give a greater predisposition towards hypertension, diabetes and CHD, than would result from more generalized obesity. The same author also suggests that since many epidemiological studies only employ BMI to indicate body fatness, their ability to discriminate

the distribution of body fat is therefore limited. Of greater advantage therefore might be the use of skinfold thickness and the ratio of waist to hip circumference.

For the present study, whilst BMI remained unchanged throughout the programme, small (non significant) decreases were seen for both skinfold thickness and W:H with training. This finding is consistent with previous observations. The sum of skinfolds at four standard sites for example, has been associated with heart disease risk factors (Krotkiewski et al. 1983; Hartz et al. 1984). Donahue and coworkers (1987) considered central adiposity (measured by skinfold thickness) in relation to the risk of CHD. From their observations they concluded that "centrally obese individuals are at increased risk of CHD independent of BMI."

However, Van Gaal and coworkers (1987) suggest that skinfold thickness represents only one aspect of central obesity (subcutaneous fat) and therefore the term central adiposity as adopted by Donahue and colleagues (1987) is misleading. Van Gaal and coworkers consider that central obesity can be more appropriately assessed by using the complementary measures of W:H and skinfold thickness.

Several potential mechanisms exist by which central body fat distribution may predispose to increased heart disease (Stern & Haffner 1986) these include lower concentrations of HDL-C (Orchard et al. 1983). However, in the absence of any significant change in the measurements of body composition made during the present study, it is unlikely that this was a major contributor to the favourable changes in TC and HDL-C metabolism previously described.

The absence of a change body composition during the present study achieved further confirmation from the

results of the hydrostatic weighing. Using this technique for the assessment of body fat no differences were observed either within or between the groups. However, the calculation of body fatness from body density is derived from the equation developed by Siri (1956), which itself is based on a number of assumptions. It is assumed that fat and lean tissue have known densities (0.90 and 1.10 $g.cm^{-3}$ respectively) and that bone density remains constant. However, more recent work has shown that these assumptions are not always fulfilled. For example, with training muscle and bone density is increased (MacDougal et al. 1983), whilst bed rest or inactivity has the opposite effect. On this basis Brodie (1988) therefore suggested that the potential exists to overestimate body fatness in trained individuals and to underestimate percentage body fat in inactive individuals. For the present study, this might have important consequences.

In the present study broadband ultrasonic attenuation (BUA) measurements at the calcaneus were made for most of the subjects in this study. Data shown in Table 6:12 was collected by Prof. P.R.M. Jones and Mrs. M. Tomlinson in the Department of Human Sciences. The results showed a significant increase in BUA values for walkers relative to controls after the programme. One might therefore speculate that, if percentage body fat was underestimated at baseline and then due to increased bone density it might be overestimated at the end of the programme. This might suggest that a change in body composition had occurred but it is impossible to estimate the magnitude of any such change. However, this suggestion is of course speculative.

In addition to the favourable gains derived from the present 12 month training programme for endurance capacity, as well as for the concentration of TC and HDL-C, trends towards favourable change were also determined

Table 6:12. Broadband ultrasonic attenuation (BUA) values in the calcaneus at baseline and after 12 months for walkers (n=25) and controls (n=15) (mean±SEM).

	BUA (dB/MHz)	
	Baseline	12 Months
Walkers	103.3±4.9	116.1±3.8*
Controls	103.6±5.5	99.6±5.4

Significantly different from baseline and from controls
*$P<0.05$.

for some aspects of psychological well-being. These observations are consistent with a number of studies which have also reported beneficial changes as a consequence of endurance training.

The results of the analysis of the self-report questionnaire compiled to identify changes in self-esteem indicated an increase in this parameter after training. This finding is consistent with those reported for the effects of similar programmes of low intensity exercise (Folkins et al. 1972; Blumenthal et al. 1982; Pauly et al. 1982). The same authors were also able to report a decrease in the level of anxiety for the subjects at the end of the training programme. However, whilst employing the same tool of assessment (STAI) anxiety was not found to be reduced for the present group of subjects, although a trend towards favourable decrease was identified.

A similar pattern was also identified for the results of the analysis of scores on the GHQ. Favourable changes on this measure have previously been reported by Carter (1977); Snyder and Spreitzer (1974) and by Dowell and coworkers (1988) at the conclusion of a programme of low intensity training with a non clinical population. The results of the present study whilst failing to attain statistical significance were again indicative of a trend towards favourable change.

The difference in results reported for the present and previously conducted studies might be explained by differences in the training programme, for example, supervised or unsupervised, or to differences in the subjects involved in the studies, that is males rather than females. Irrespective of these differences, however, the women involved in the present training programme generally reported "feeling better".

It is therefore possible to suggest that these results are indicative of beneficial change to aspects of psychological well-being consequential to this period of low intensity training.

The mechanisms responsible for this improvement remain open to speculation, however, it is possible that the regular performance of endurance exercise provides the individual with an improved ability to cope with 'stress' or 'anxiety'. Dickman (1988) has suggested that physiological and psychological stressors act similarly and produce similar responses in the nervous-endocrine system. Thus the response to either stress situation is a stimulation of the secretion of the hormones cortisol, adrenaline and noradrenaline.

Noradrenaline is released by the sympathetic nerves which lie in close proximity to smooth, involuntary muscles such as those of the gut. As a consequence of the release of norepinephrine the hydrolysis of glycogen to glucose and fats to fatty acids to provide energy for muscle is stimulated. Adrenaline release acts at the liver and adipose tissue to stimulate glucose and fatty acid release for contraction in the skeletal muscles.

As with adrenaline and noradrenaline, cortisol is involved in the provision of substrates for energy release and also stimulates an increase in the concentration of blood glucose, along with a redistribution of fats from adipose to peripheral tissues, including the coronary arteries (Wahren 1977). This latter effect partly explains how long-term stress may increase the likelihood of stroke and heart attack (Dickman 1988).

The effect of psychological stress therefore is to prepare the body for a state of 'fight of flight',

producing an increase in heart rate, blood pressure, muscular tension, respiration and also increasing the availability of substrates for the release of energy. A situation very similar to that produced as a consequence of physiogical stress therefore develops. It is possibly because of the similarity between these two situations that physical exercise can act as a means of stress management (Charlesworth & Nathan 1984).

Through exercise the individual becomes accustomed to the 'discomforts' (Schwartz et al. 1978) of physiological stress, and as such can reattribute these feelings to gain positive reinforcement (Schachter & Singer 1962). Thus with exercise training there is an increase in the frequency with which the effects of stress are encountered and as such the anxiety produced is lessened. As a possible consequence the reduction of tension induced by psychological stress is likely to result in a lowering of physical stress symptoms such as muscle tension and insomnia thereby increasing the perception of general health (Griest et al. 1979). Furthermore, in being able to cope more easily in a formerly stress-inducing situation an individual is thought to derive an increase in self-esteem (De Vries 1981).

6.5 Conclusion.

At the conclusion of the training programme, functional capacity and aerobic capacity were significantly increased. Improvements were also observed for walking performance and for the physiological responses to a standard exercise test. Together these indicated an increase in the ability to perform at a standard exercise intensity when compared with pre-training.

In addition, this group of formerly sedentary, middle-

aged women were shown to have a marked and progressive increase in HDL-C, along with a small decrease in TC concentration. These changes were apparent in the absence of any significant changes in body mass, body fatness or dietary composition, which therefore acts to confirm the effectiveness of exercise in its own right as a means to alter favourably lipid metabolism.

Finally, the results suggest that brisk walking can also improve psychological well-being.

This study has therefore gone some way to resolving the uncertainty surrounding the effectiveness of low intensity exercise-to bring about favourable changes in (i) aerobic capacity and exercise performance and (ii) lipid metabolism. Finally, there is some evidence to suggest that psychological benefits might also be conferred by this form of low intensity exercise.

7. THE INFLUENCE OF THREE MONTHS OF REGULAR BRISK WALKING, FOLLOWED BY THREE MONTHS OF DETRAINING IN A GROUP OF MIDDLE-AGED, FORMERLY SEDENTARY WOMEN.

7.1. Introduction.

The results of the study presented in Chapter 6 indicate that brisk walking provoked a small decrease in TC and a substantial increase in HDL-C concentrations. In addition, favourable changes were also discerned for indices of functional capacity and endurance: increased predicted $\dot{V}O_2$ max and $\dot{V}O_2$ at a reference blood lactate concentration of 2 $mmol.l^{-1}$, and decreased heart rate and concentration of blood lactate at the same absolute exercise intensity. Finally analysis of the response to a series of self-report questionnaires revealed a trend towards favourable change in aspects of psychological well-being.

Confirmation of the nature and extent of similar adaptations to high intensity exercise training has been derived from the observation of responses to detraining (Saltin et al. 1968; Fringer & Stull 1974; Smith & Stransky 1976; Otto et al. 1978; Saltin & Rowell 1980). A similar approach was therefore adopted for the present study in relation to low intensity exercise training. Since many of the adaptations cited in Chapter 6 became apparent after the first 3 months of the one year programme it was considered appropriate to select a training period of similar duration for the present study. This period of training was then followed by a period of equal length during which the exercise stimulus was withdrawn. In addition, so as to minimize the influence of the menopause on lipid and lipoprotein metabolism an age limit of 55 years was adopted.

7.2. Methodology.

(i). Subjects.

Twenty sedentary, middle-aged females from the town and university communities volunteered to take part in the study. None of the individuals had participated in any form of regular or structured exercise in the year prior to the onset of the programme and were, for the purpose of the study, considered as sedentary.

The experimental groups were determined on a convenience basis. That is, the degree of commitment and time required to complete the programme was discussed with the individuals. On this basis, each subject made an informed decision. In accordance with this procedure, 10 women volunteered to join the training group, whilst the remaining 10 acted as controls. Table 7:1 includes the physical characteristics of the subjects.

(ii). Protocol.

Prior to the onset of training, all subjects completed a one mile self-paced track walk. The submaximal pace adopted by each individual was subsequently reproduced in the laboratory and the same distance was then completed on the treadmill. A submaximal incremental treadmill test was also completed for the prediction of $\dot{V}O_2$ max (Chapter 3:3).

A 10 ml venous blood sample was obtained from each subject whilst in the fasted state (Chapter 3:6). The sample was subsequently assayed for the concentration of serum TC, HDL-C, HDL_3-cholesterol, apolipoprotein AI and B, and Tgs (Appendices 7-11). Body mass, circumferences at the waist and hip and skinfold thicknesses at the biceps, triceps, subscapular and suprailiac were determined (Chapter 3:5). Subjects also completed a seven-day dietary record and a battery of psychological

self-report questionnaires (Appendices 13 & 14). All procedures, were completed at baseline, after training (3 months) and after the 3 month period of detraining. All techniques were employed in accordance with the procedures previously described (Chapter 3).

Analysis of variance was employed to test for differences in response of walkers and controls. A Pearson Product Moment correlation coefficient was used to examine the relationship between variables. Results are presented as mean±SEM.

(iii). Training.

All subjects involved in the study were working mothers and/or were in full time employment. Consequently, it was necessary for the training regimen to be flexible. It was considered appropriate to make a weekly minimum exercise prescription. For example:-

(4 x 40 minutes) + (1 x 30 minutes)
+ 60 (self-determined) = a total of 250 minutes.

The volume of exercise prescribed ranged from 180 minutes during the first two weeks, to 315 minutes during the last four weeks of the programme. The duration of each session was no less than 20 minutes and no more than 50 minutes (Figure 7:1). Individuals were instructed to train at their own brisk submaximal pace. If it is assumed that the pace was similar to that adopted during the one-mile submaximal track walk (1.76±0.03 $m.s^{-1}$), then this would correspond to approximately 12 miles (19 km) in the first weeks, rising to 21 miles (33 km) at the end of the training programme.

A diary-type record of the duration and frequency of sessions was maintained throughout the training period, and was checked every two weeks. Further subject-

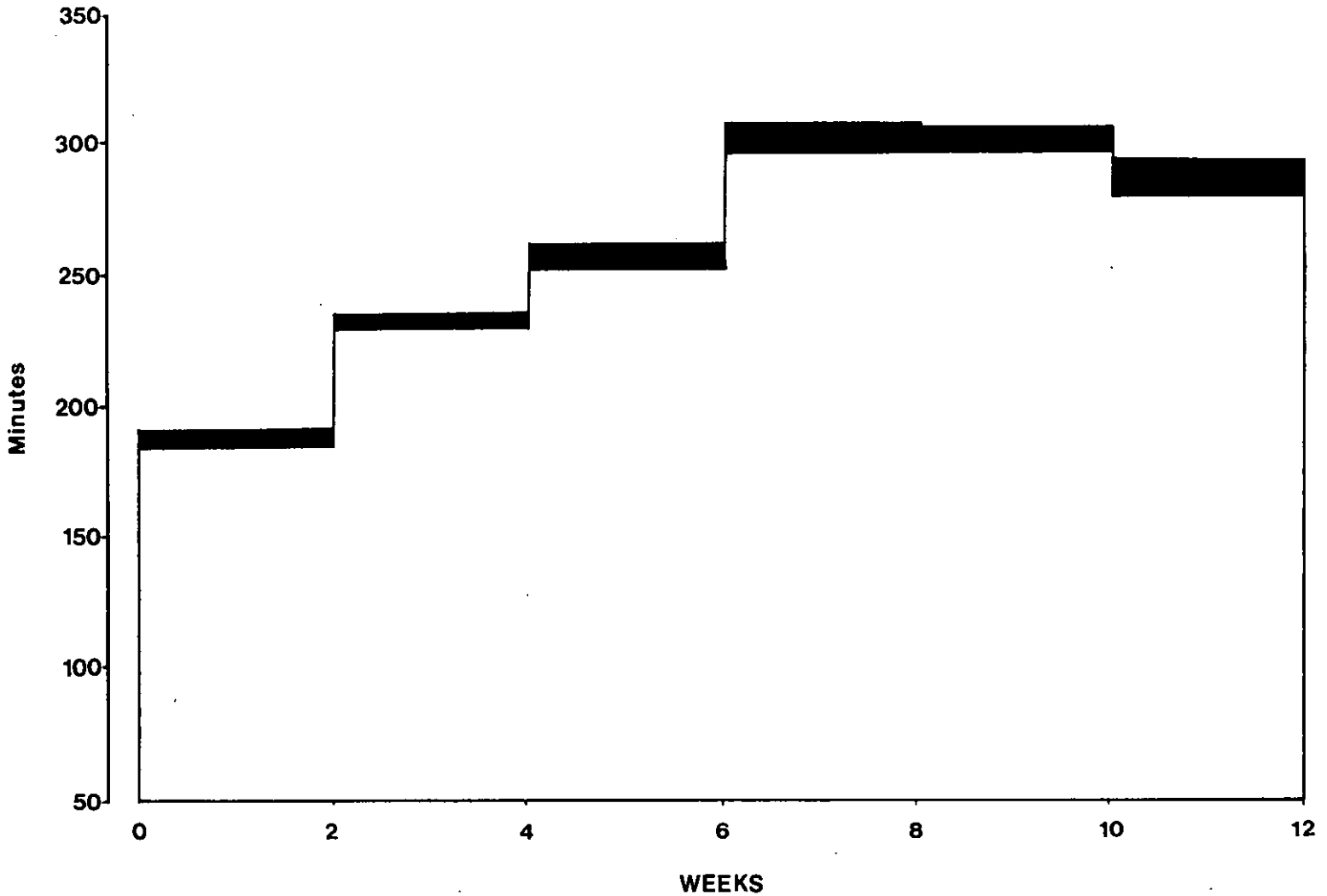


FIGURE 7:1. Average number of minutes walking completed at intervals throughout the training programme (n=10).

experimenter contact was achieved by regular visits to the home or work-place and by telephone. Throughout the training period, the control subjects maintained their habitual lifestyle.

After the completion of the 12 week training period and re-tests, the experimental subjects were encouraged to re-adopt their previous less active lifestyle. Both groups were tested for a final time after a further 12 weeks.

7.3. Results.

Data concerning the physical characteristics of subjects at baseline is contained in Table 7:1. Walkers and controls did not differ with regard to body mass, BMI or height. However, walkers were significantly older than controls (47.3±2.0 yrs cf. 41.6±1.2 yrs for walkers and controls respectively, $P<0.05$).

There were no significant differences between the groups for the percentage of body fat or W:H ratio at baseline (Table 7:2). A difference was discerned for the sum of four skinfolds but at the completion of the training period this difference no longer existed. Furthermore for the walkers there was a trend for body mass to decrease with training and to return towards baseline levels after the period of detraining. Similar trends were also seen for W:H ratio, sum of skinfolds and BMI. Similar trends for controls were not discerned.

Analysis of the food records (Table 7:3) revealed no differences between the groups for average daily energy intake, for the percentage of daily energy as fat or carbohydrate, or alcohol or the ratio of polyunsaturated to saturated fatty acids consumed (P:S). The groups could not be differentiated therefore at baseline, after training, or after the period of detraining on the basis of diet (Figure 7:2).

At baseline there was no difference in the time (or pace) required by the groups to complete the one-mile submaximal track walk (15.27±0.24 mins cf. 15.24±0.34 mins; 1.76±0.03 $m.s^{-1}$ cf. 1.77±0.04 $m.s^{-1}$ for walkers and controls respectively). In the laboratory, no differences were discerned for resting heart rate or for average heart rate elicited when the submaximal pace adopted on the track was reproduced for each individual on the

Table 7:1. Physical characteristics at baseline for walkers (W n=10) and for controls (C n=10) (mean±SEM).

	AGE (yr)	BODY MASS (kg)	HEIGHT (m)	BMI ($kg.m^{-2}$)
W $\bar{x}$	47.3	65.7	1.63	24.8
SEM	±2.0	±2.6	±0.02	±1.4
Range	39.3-54.8	51.1-80.7	1.52-1.75	19.7-34.8
C $\bar{x}$	41.6*	64.4	1.59	23.4
SEM	±1.2	±2.8	±0.02	±1.1
Range	35.7-48.4	53.1-82.9	1.59-1.70	20.2-31.7

Significantly different from walkers: * $P<0.05$.

Table 7:2. Body mass, body mass index (BMI), percentage body fat estimated from the sum of skinfolds (%BF), waist:hip ratio (W:H) and sum of 4 skinfolds (SSF) for walkers (W n=10) and controls (C n=10) (mean±SEM).

	Baseline	3 Months	6 Months
Body mass (kg)			
W	65.7±2.6	64.7±2.7	66.1±2.7
C	64.4±2.8	64.4±2.8	65.6±2.8
BMI ($kg.m^{-2}$)			
W	24.8±1.4	24.5±1.5	25.0±1.5
C	23.4±1.1	23.3±1.0	23.8±1.1
%Body fat			
W	35±2	36±2	36±2
C	33±2	33±2	33±2
W:H ratio			
W	0.81±0.03	0.79±0.03	0.83±0.03
C	0.78±0.03	0.77±0.03	0.79±0.03
SSF (mm)			
W	77±8	71±8*	73±8
C	64±9†	64±8	66±8

Significantly different from baseline: *$P<0.05$.
Significantly different from walkers: †$P<0.05$.

Table 7:3. Average daily intake of energy (MJ), percentage of energy as fat (% Fat) and as carbohydrate (% CHO) and alcohol and in the ratio of polyunsaturated :saturated fatty acids consumed (P:S) for walkers (W n=10) and controls (C n=10) (mean±SEM).

	Baseline	3 Months	6 Months
Energy (MJ)			
W	7.9±0.7	7.3±0.6	7.2±0.6
C	7.3±0.4	7.5±0.3	7.4±0.3
% Fat			
W	35.8±1.9	34.4±1.1	35.1±1.3
C	36.0±2.2	35.8±1.8	36.3±2.1
% CHO			
W	45.2±1.6	44.8±2.0	45.0±2.0
C	46.7±2.0	46.3±2.0	45.5±1.9
% Alcohol			
W	2.6±0.8	4.0±1.2	3.7±0.8
C	2.8±0.8	3.1±0.8	2.0±0.8
P:S ratio			
W	0.31±0.03	0.33±0.03	0.31±0.03
C	0.30±0.03	0.31±0.06	0.29±0.03

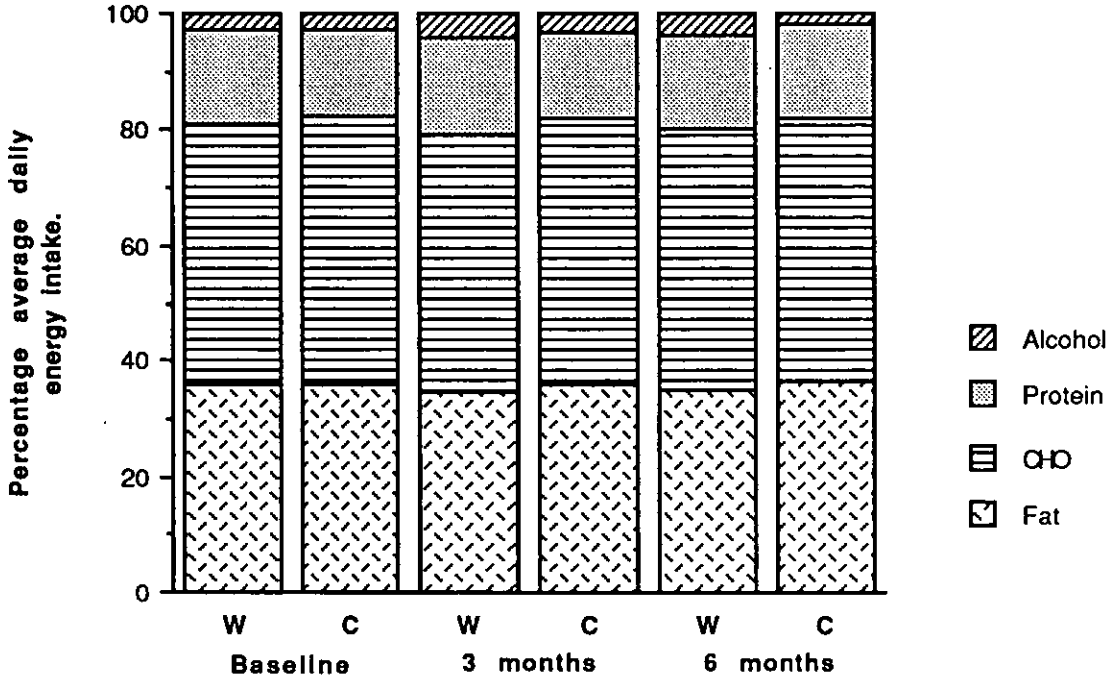


FIGURE 7:2. Average daily energy intake as fat, carbohydrate, protein and alcohol at baseline, 3 months and 6 months of a detraining study for middle-aged female walkers (W) and controls (C), (n=10 per group).

treadmill (Table 7:4). At the completion of the training period however, for walkers a trend towards a decrease in both indices was discerned. Similar changes were not found for the controls.

At baseline there was no distinction between the groups for blood lactate concentration measured at the end of the treadmill walk. At the completion of the training period however the concentration of blood lactate measured for walkers was reduced by 1.11 $mmol.l^{-1}$ (38%) from baseline. With the completion of the detraining period a slight increase from the 3 month value (0.51 $mmol.l^{-1}$) was observed. However, at 6 months blood lactate concentration remained 20.8% below that measured prior to training. No statistically significant differences were observed for controls and there was no difference between the groups at 6 months (Figure 7:3).

Also included in Table 7:4 are the respiratory exchange ratio values (R). No differences were discerned between the groups throughout the investigation. However, there was a decrease in R value for walkers after training. This decrease was followed by a recovery to baseline values at the end of the detraining period.

The heart rate and $\dot{V}O_2$ elicited during the submaximal graded treadmill walk both decreased with training. However, the adaptations for neither parameter attained significance. As a consequence, whilst predicted $\dot{V}O_2$ max for walkers showed a 7.7% increase with training, this was not significant. With the removal of the training stimulus, predicted $\dot{V}O_2$ max for walkers returned towards baseline level. Similar trends were not observed for controls (Figure 7:4).

Adaptations in aerobic capacity were indicated by changes in $\dot{V}O_2$ at a reference blood lactate concentration of

Table 7:4. Response to a one-mile treadmill walk: mean heart rate, end blood lactate concentration (La ($mmol.l^{-1}$)) and respiratory exchange ratio (R) for walkers (W n=10) and controls (C n=10) (mean±SEM).

	Baseline	3 Months	6 Months
Mean heart rate during the walk.			
W	130±6	123±6	124±6
C	128±5	129±4	124±4
Blood La ($mmol.l^{-1}$).			
W	2.89±0.47	1.78±0.20†	2.29±0.34$^{\circ\circ}$
C	2.50±0.24†	2.67±0.27††	2.13±0.28
R value.			
W	0.91±0.01	0.86±0.01†	0.90±0.01
C	0.90±0.01	0.88±0.01	0.87±0.01

Significantly different from baseline: $^{\dagger}P<0.05$.
Significantly different from walkers: $^{*}P<0.05$, $^{**}P<0.01$.

Response of walkers significantly different from controls, interaction $^{\circ\circ}P<0.01$.

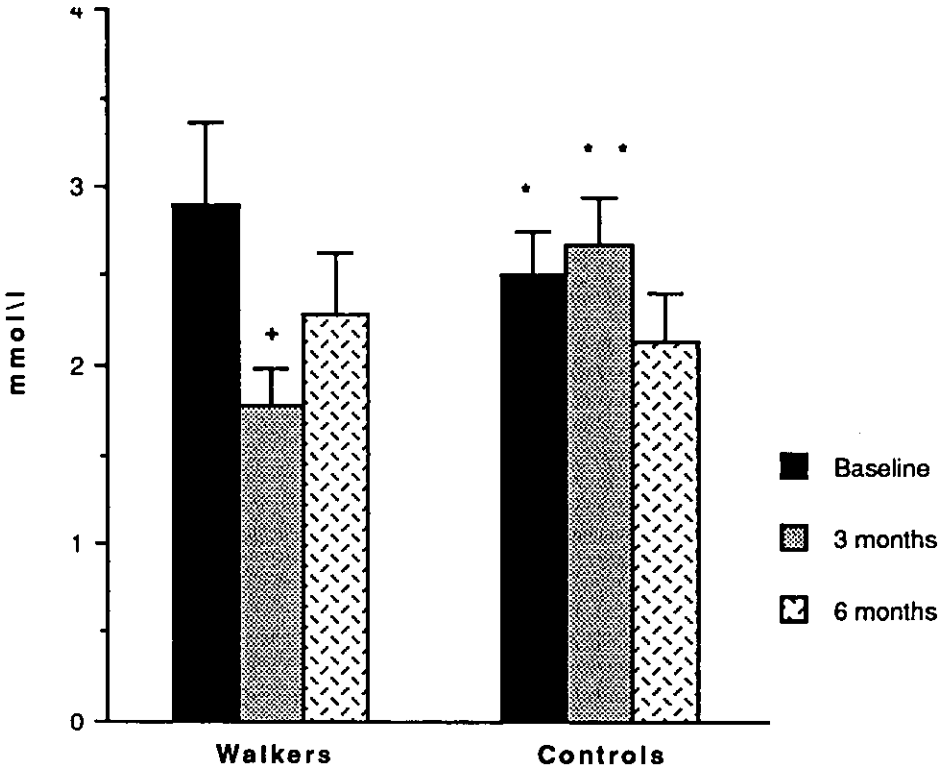


FIGURE 7:3. Concentration of blood lactate at the end of the one mile submaximal treadmill walk at baseline and intervals throughout the detraining study, for middle-aged females (n=10 per group).

Significantly different from baseline +P<0.05.

Significantly different from Walkers *P<0.05, **P<0.01.

Table 7:5. Predicted $\dot{V}O_2$ max ($ml.kg^{-1}min^{-1}$) and ($l.min^{-1}$), oxygen uptake at a blood lactate concentration of 2 $mmol.l^{-1}$ ($ml.kg^{-1}min^{-1}$) and the percentage of $\dot{V}O_2$max utilized at 2 $mmol.l^{-1}$ for walkers (W n=10) and controls (C n=10) (mean±SEM).

	Baseline	3 Months	6 Months
$\dot{V}O_2$ max ($ml.kg.^{-1}min^{-1}$)			
W	31.5 ± 1.9	34.0 ± 2.0	31.0 ± 1.7
C	31.3 ± 1.9	31.2 ± 1.7	31.7 ± 1.6
$\dot{V}O_2$ max ($l.min^{-1}$).			
W	2.05 ± 0.11	2.18 ± 0.12	2.03 ± 0.10
C	2.08 ± 0.14	1.99 ± 0.11	2.07 ± 0.11
$\dot{V}O_2$ at 2$mmol.l^{-1}$ ($ml.kg.^{-1}min^{-1}$).			
W	17.8 ± 1.4	21.7 ± 0.7^{+}	19.3 ± 0.9°
C	18.4 ± 1.3	17.9 ± 1.1**	18.2 ± 1.3
% $\dot{V}O_2$ max at 2$mmol.l^{-1}$			
W	57.2 ± 3.7	64.1 ± 2.9^{+}	62.9 ± 3.1$^{\circ\circ}$
C	56.8 ± 2.7	58.0 ± 3.1**	57.6 ± 3.2

Significantly different from Baseline: $^{+}P<0.05$.
Significantly different from walkers: $^{*}P<0.05$, $^{**}P<0.01$.

Response of walkers significantly different from controls, interaction $^{\circ}P<0.05$, $^{\circ\circ}P<0.01$.

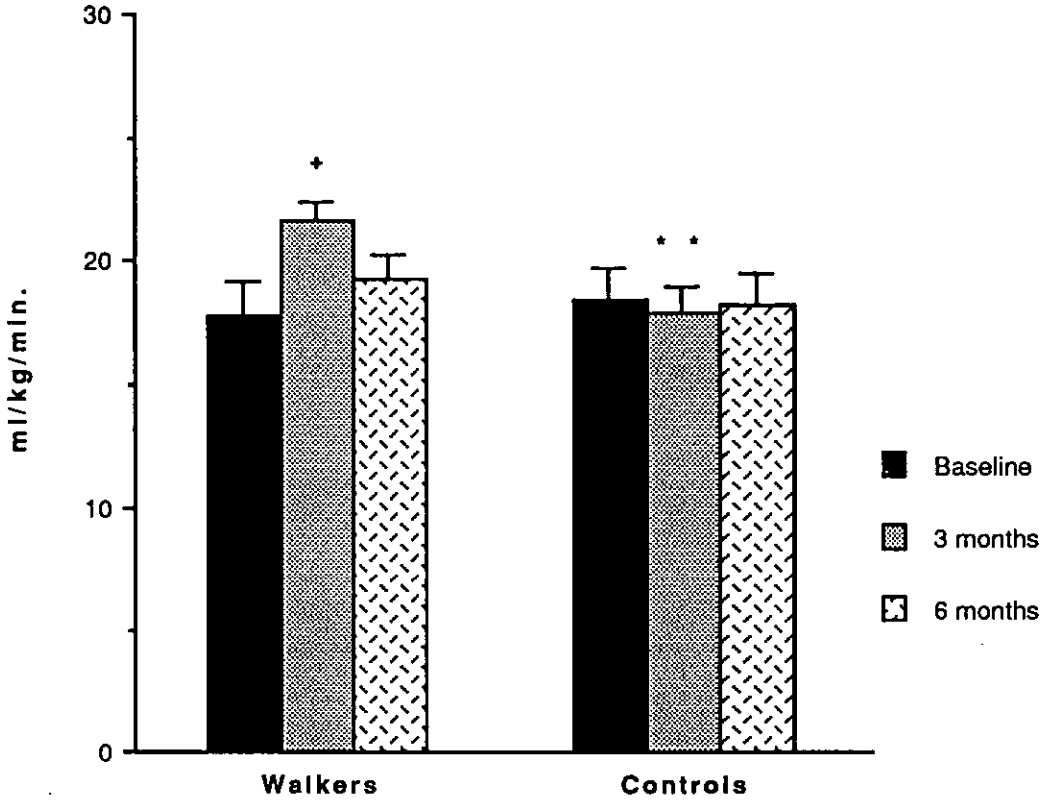


FIGURE 7:4. Oxygen uptake at a reference blood lactate concentration of 2 mmol/l at baseline and intervals throughout the detraining study, for middle-aged females (n=10 per group).

Significantly different from baseline +P<0.05.

Significantly different from Walkers *P<0.05, **P<0.01.

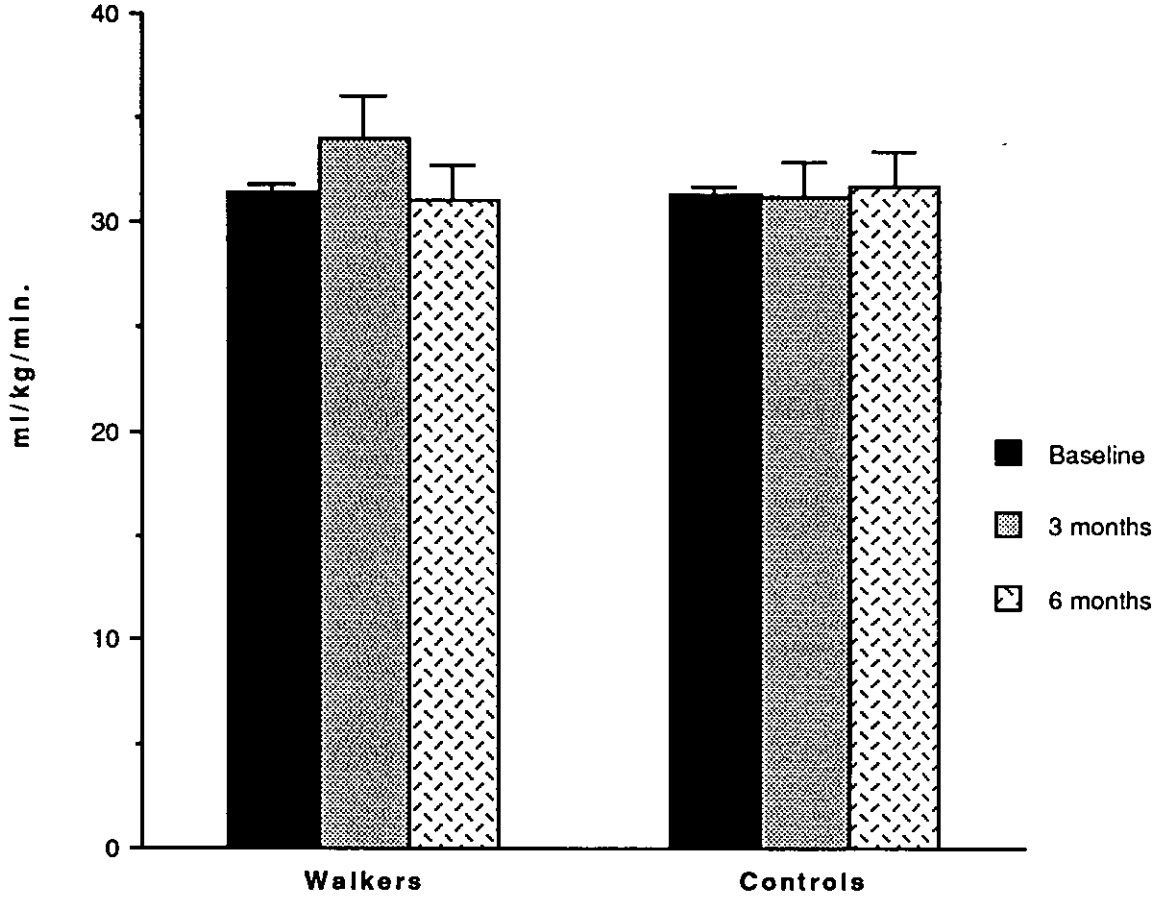


FIGURE 7:5. Predicted maximal oxygen uptake at baseline and intervals throughout the detraining study, for middle-aged females (n=10 per group).

2 mmol.l^{-1}. At baseline, there was no difference between the groups for this parameter (Table 7:5). However, at the conclusion of the training period $\dot{V}O_2$ at 2 mmol.l^{-1} blood lactate concentration was increased above baseline by 3.9 ml.kg.$^{-1}$min^{-1} (21.7%) and remained elevated (1.5 ml.kg^{-1}min^{-1} or 8.4%) after the period of detraining. Similarly when $\dot{V}O_2$ at 2 mmol.l^{1} was expressed as a percentage of the predicted $\dot{V}O_2$max a significant increase (6.9%) was observed at 3 months. Furthermore at the end of the study this parameter remained 5.7% higher than baseline. No changes were observed for controls (Figure 7:5).

The results of the analysis of samples of serum for lipids, lipoproteins and apolipoproteins is contained in Table 7:6. No differences were apparent at any stage of the investigation, either between or within the groups for the concentrations of TC, HDL-C, or for the ratio TC:HDL-C. Furthermore, there were no differences for %HDL-C, for apolipoprotein A-I and B, or for Tgs. A tendency did exist however, for TC and TC:HDL-C to decrease and for HDL-C to increase with training (Figure 7:6 & 7:7). The opposite trends appeared after detraining. A significant increase (0.08 mmol.l^{-1} or 19%) was found however, for the concentration of HDL_2 - cholesterol. With detraining, there was a subsequent decrease of a similar magnitude for walkers.

It is fully appreciated that the interpretation of the results of the lipid analyses is made more difficult by the difference in ages between the two groups. Use of the analysis of covariance technique would allow this weakness in the study design to be overcome to some extent. However, this procedure was not undertaken since the controls were not found to have significantly different cholesterol values from the walkers at any stage of the study.

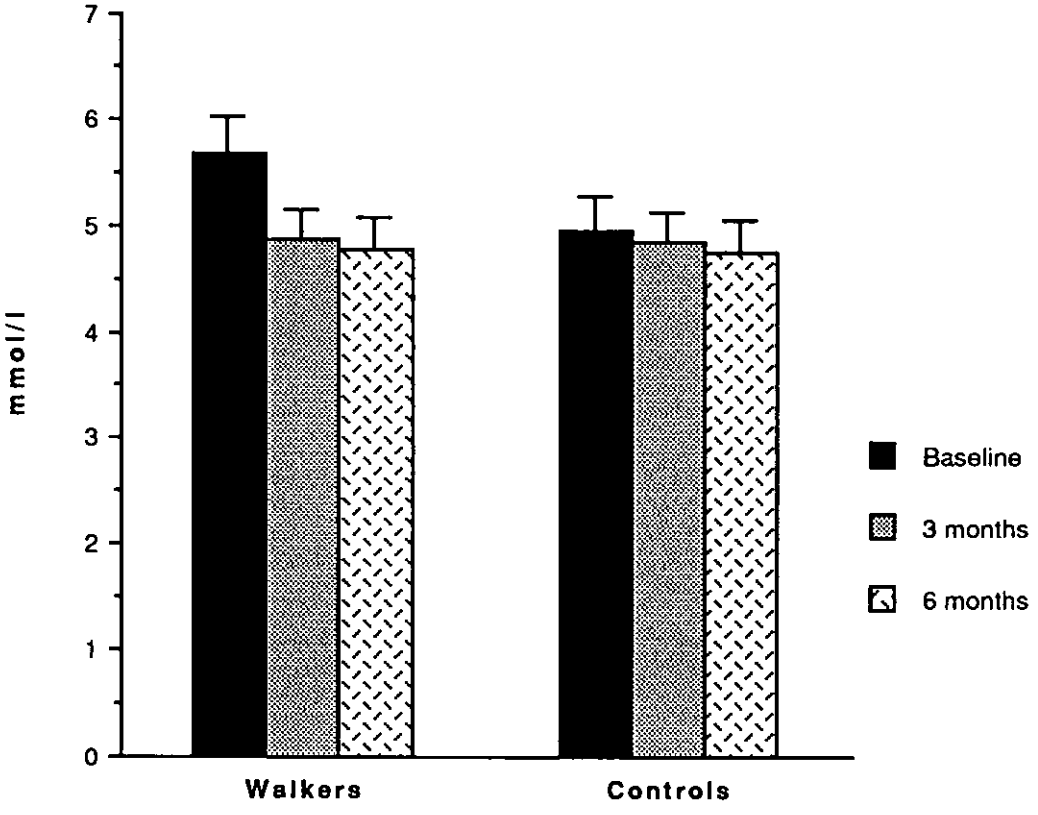


FIGURE 7:6. Concentration of total cholesterol at baseline, 3 and 6 months of a detraining study for middle-aged females (n=10 per group).

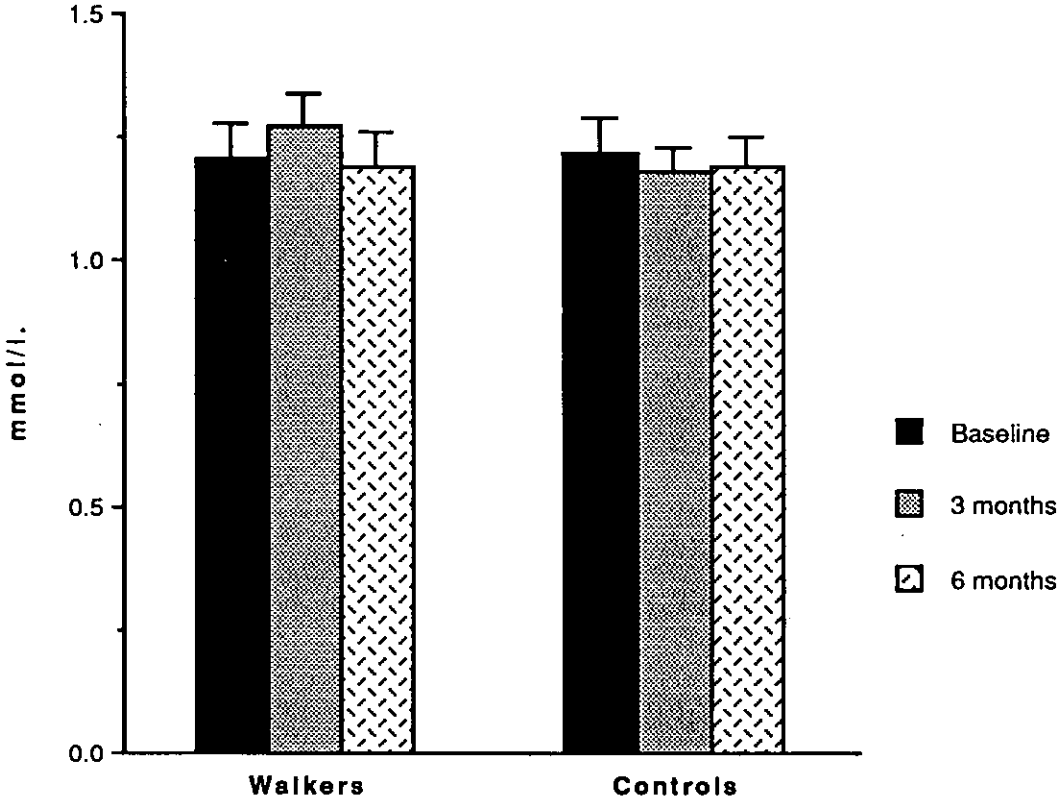


FIGURE 7:7. Concentration of high density lipoprotein cholesterol at baseline and intervals throughout the detraining study for middle-aged females (n=10 per group).

Table 7:6. Total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), ratio of TC:HDL-C, HDL_2-C, triacylglycerols (Tgs), and apo AI and B, for walkers (W n=10) and controls (C n=10) (mean±SEM).

	Baseline	Trained	Detrained
TC ($mmol.l^{-1}$).			
W	5.67±0.35	4.87±0.28	4.77±0.29
C	4.94±0.34	4.84±0.29	4.74±0.30
HDL-C ($mmol.l^{-1}$).			
W	1.21±0.07	1.27±0.07	1.19±0.07
C	1.22±0.07	1.18±0.05	1.19±0.06
TC:HDL-C ratio.			
W	4.75±0.33	3.92±0.29	4.08±0.29
C	4.14±0.32	4.16±0.28	4.11±0.36
HDL_2-C ($mmol.l^{-1}$).			
W	0.42±0.02	0.50±0.02^{++}	0.48±0.03°
C	0.46±0.03	0.44±0.04	0.46±0.04
Tgs ($mmol.l^{-1}$).			
W	0.97±0.13	0.97±0.16	0.95±0.17
C	0.93±0.14	0.96±0.11	0.97±0.15
Apo AI ($mmol.l^{-1}$).			
W	1.48±0.06	1.44±0.09	1.47±0.11
C	1.41±0.04	1.45±0.07	1.40±0.04
Apo B ($mmol.l^{-1}$).			
W	0.75±0.05	0.73±0.05	0.79±0.06
C	0.64±0.05	0.63±0.04	0.67±0.05

Significantly different from Baseline: $^{++}P<0.01$

Response of walkers significantly different from controls, interaction $^{0}P<0.05$.

Table 7:7. Self-esteem, general health (GHQ) and state-trait anxiety inventory (STAI), for walkers (W n=10) and controls (C n=10) (mean±SEM).

	Baseline	Trained	Detrained
Self-esteem			
W	31.2±0.9	33.4±1.1	31.6±1.1
C	30.9±0.8	31.2±0.8	31.0±1.0
GHQ			
W	10.0±0.8	9.5±0.8	10.1±0.8
C	10.3±0.9	10.5±0.8	10.2±0.9
STAI			
W	41.0±1.3	38.2±1.6	38.3±1.5
C	42.3±1.0	42.2±1.0	44.1±1.3

Table 7:7 contains data concerning the analysis of the self-report psychological questionnaires. No significant differences were observed either within or between the groups at any stage of the investigation. However, favourable changes were observed for walkers after training on all aspects of psychological well-being. This tendency was reversed with detraining. No differences were observed for controls.

7.4. Discussion.

Throughout the study no differences were observed either between or within the groups for body mass, BMI, percentage body fat or W:H ratio. However for walkers a trend did exist for these measures to decrease with training and a significant decrease was found for the sum of four skinfolds. With the removal of the training stimulus however there a reversal of these favourable trends. At the conclusion of the detraining period therefore the measurement of all indices, with the exception of the sum of 4 skinfolds, were shown to be at, or above, the levels reported at baseline (Table 7:2).

At the end of the first 3 months of the study controls, unlike walkers, were not shown to decrease on any of the measures of body mass or body fatness. However, at the conclusion of the latter 3 month period the pattern of change observed for controls was consistent with that reported for walkers, that is, there was a tendency for all these indices to increase.

The similarity between the trends observed for walkers and controls in the latter half of the study suggest the possibility of a common influence. For example, the increases in body mass and indices of body fatness might be the consequence of seasonal adjustment. Support for this proposal is derived from the observation that baseline measurements were made in May/June, mid-study measurements were made in August/September and the final assessments were completed in December. It might therefore be that the latter measurements were influenced by the onset of winter.

The absence of a significant change in body mass observed after the present 3 month training period is consistent with the results reported in the previous chapter.

Furthermore reports from previously completed training studies also support this observation (Johannessen et al. 1986; Pollock et al. 1972; Santiago et al. 1987; Moll et al. 1979; Atomi & Miyashita 1980; Farrell & Barboriak 1980; Flint et al. 1974; Girandola 1976; Shire et al. 1977; Zwiren et al. 1973).

In addition to body mass, the significant decrease reported for the sum of 4 skinfolds was consistent with the observation of a trend towards a decrease reported in Chapter 6. No difference however was determined for other indices of body composition (W:H ratio, % body fat). Support for this latter finding has been derived from the results of previous studies which have also reported an absence of change from baseline with training (Smith & Stransky 1976; Vaccaro & Clinton 1981; Zwiren et al. 1973).

This inability to provoke a substantial change in the indices of body mass and body composition with training might be explained in part as a consequence of factors already examined in Chapter 6. For example, assuming energy expenditure is increased as a consequence of exercise training, in the absence of an associated increase in energy intake, body mass and body fatness would be expected to be reduced (Tremblay et al. 1985).

The results from the exercise tests reveal favourable adaptations for blood lactate, heart rate and $\dot{V}O_2$max parameters which therefore support the proposal that the women did complete the training. However, the results of the analysis of dietary records showed no sign of an increase in energy intake. Since body mass and indices of body fatness (except sum of 4 skinfolds) remained unchanged it might be suggested that an increase in energy intake did occur but went unrecognised. Block (1990) suggested that whilst the diary-type record is

possibly the most convenient and useful technique for obtaining dietary information outside the laboratory or clinic, it is subject to a number of disadvantages. Of particular relevance repetition of the procedure is associated with an increased tendency for subjects to under-report their food intake (Livingstone et al. 1990; Bingham 1987; Southgate 1986). If inaccuracy and under-reporting were involved in the present series of measurements these artifacts might act to disguise an actual increase in energy intake.

Whilst remaining a possibility, it is unlikely that these failings of the self-report method are wholly responsible for the absence of an increase in energy intake. This is emphasized by the observation that the energy intake of the control group also remained unchanged throughout the programme which would seem to indicate that the walkers were more prone to the under-report, or to present inaccurate data. Evidence for this is not readily apparent.

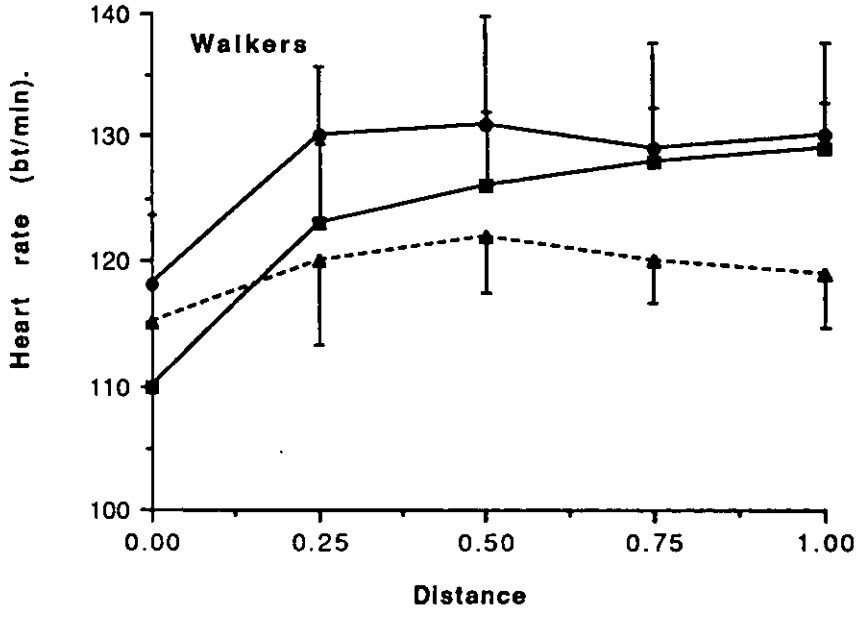
Heart rate was monitored at rest and throughout the submaximal treadmill walk. After exercise training both indices were found to have decreased, only to subsequently increase again after the period of detraining (Figure 7:8). This pattern of change is consistent with reports from previously conducted detraining studies, for example Saltin and coworkers (1968); Saltin and Rowell (1980); Fringer and Stull (1974) and Smith and Stransky (1976).

In general exercise training is associated with a decrease in submaximal heart rate. This occurrence has been reported for young adults (Astrand & Rodahl 1986; Ekblom et al. 1968; Hartley et al. 1969) and for older adults (Seals et al. 1984; Robinson et al. 1973). Similarly, the effect has also been observed for a range

of different exercise intensities, including running (Wilmore et al. 1980) and walking (Jetté et al. 1988). The findings of the present study therefore appear consistent with previously reported data and are supportive of the observations made in Chapter 6.

Figure 7:8 illustrates the pattern of heart rate elicited throughout the one mile submaximal treadmill walk. After training a decrease was observed at each of the distance intervals, the overall decrease being 6.1%. This response was slightly less than the 8% decrease reported by Flint and coworkers (1974) for women, and the 10% decrease found for men by Pollock and coworkers (1971), after a period of walking training. However others have reported a similar magnitude of decrease. Santiago and coworkers (1987) for example, noted a decrease of 5% for women after the completion of a 12 week programme of walking. The findings of the present study therefore appear well supported.

Observations made at the end of the detraining period indicated an increase in the average heart rate elicited during the treadmill walk. However, the rate remained 3.4% below that recorded at baseline. Evidence exists to suggest that central circulatory adaptations to exercise training require a relatively longer period to reverse when compared with peripheral adaptations such as mitochondrial enzyme activity. Support for this proposal comes from the work of Henriksson and Reitman (1977) who observed 32% and 35% increases in the activity of succinate dehydrogenase and cytochrome oxidase with training. However within 2 weeks of detraining cytochrome oxidase was back to baseline, as was succinate dehydrogenase within 6 weeks. In contrast, $\dot{V}O_2$max and heart rate were only slightly changed after the same period. Some evidence does therefore exist to suggest the possibility of a residual training effect. However since



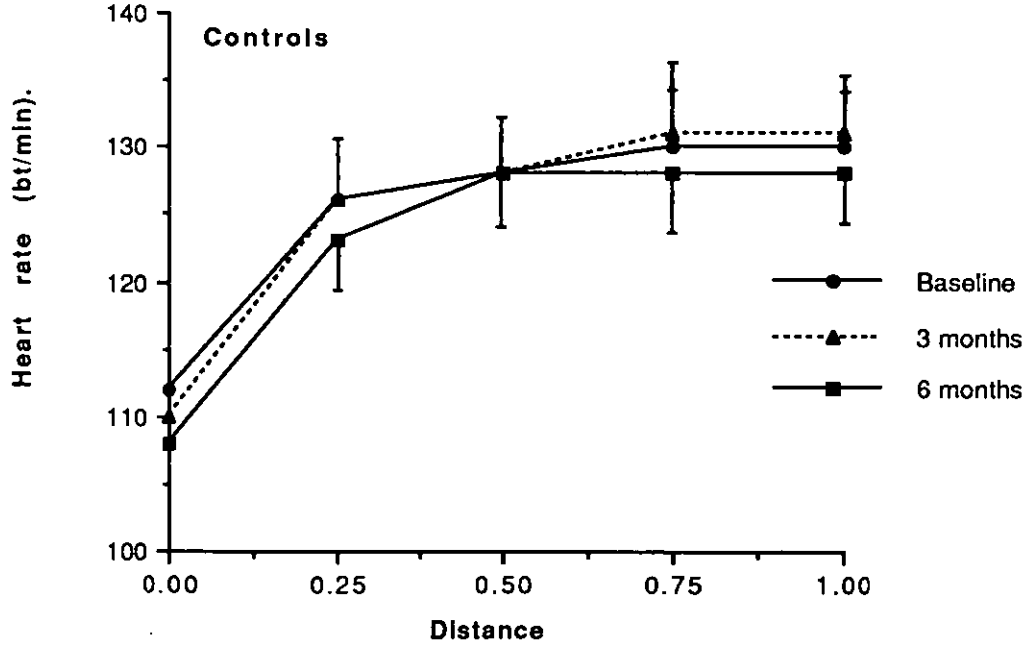


FIGURE 7:8. Average heart rate at intervals during a one mile submaximal treadmill walk at baseline, 3 and 6 months of a detraining study with middle-age females (n=10 per group).

the average heart rate elicited by controls during the treadmill walk also showed a trend towards decrease between 3 and 6 months it is possible that familiarization with the test resulted in lower heart rate response. Whilst this latter suggestion is only speculation it remains quite possible.

In addition to submaximal heart rate, the observations made for oxygen uptake were also consistent with previous reports. For example, no difference was discerned for the oxygen cost of the treadmill walk at baseline, after training, or after detraining. This therefore suggests that endurance training has little or no effect on $\dot{V}O_2$ at a standardized submaximal exercise intensity (Byrd et al. 1974; Fox et al. 1977; Girandola & Katch 1973, 1976; Kilbom et al. 1969). This would be expected in the light of the results of investigations which have revealed a fixed relationship between oxygen uptake and the rate of ATP resynthesis (Holloszy & Coyle 1984).

Whilst $\dot{V}O_2$ at a standard, submaximal exercise intensity remained unchanged with training a significant reduction was identified for the respiratory exchange ratio (R). Together, these two observations (no change in $\dot{V}O_2$ and reduced R value) suggest an increase in the contribution of fat to energy metabolism and thereby a conservation of muscle glycogen stores (Astrand & Rodahl 1986; Holloszy 1973). A number of mechanisms have been considered to be involved in this process of adaptation. For example, an increase in muscle capilliarization would enhance the delivery of oxygen and removal of metabolites. Secondly, an increase in both the number and size of mitochondria, would allow an increase in the concentration and activity of lipolytic enzymes involved in the breakdown of fatty acids (Holloszy & Coyle 1984).

The proliferation of capillaries in the trained muscles

has also been identified as an important determinant of blood lactate concentration (Saltin 1985). Blood lactate concentration has been adopted as an index of changes in aerobic capacity, thus at the same relative or absolute work rate the concentration is lower in the trained compared with the untrained state. This occurrence has been reported for young adults after the completion of relatively high intensity endurance training (Astrand & Rodahl 1986; Holloszy 1973; Hurley et al. 1984; Robinson & Harmon 1941) and also for older adults after a programme of brisk walking (Barry et al. 1966; Seals et al. 1984). These results appear consistent with those derived from the present study in which the concentration of blood lactate measured at the end of the submaximal treadmill walk was reduced by 38% from the pre-training value.

It was also shown that after the period of detraining the concentration of blood lactate measured at the end of the walk was again increased. This pattern of change is consistent with that reported for other detraining studies (Saltin et al. 1969). It has been suggested that the cessation of training is associated with a rapid reduction of the activity of the enzymes involved in aerobic metabolism. The work of Henriksson and Reitman (1977) for example, showed a rapid reversal of the activity of the oxidative enzymes succinate dehydrogenase and cytochrome oxidase whilst $\dot{V}O_2max$ remained 16% above the level observed at baseline. This observation therefore suggests as dissociation between these two measures.

Furthermore, capillary proliferation which occurs with training has also been shown to remain higher than baseline despite detraining (Holloszy & Coyle 1984). The effect of this would be to allow an increase in the capacity to transport oxygen to and to remove metabolites

from the working muscles, to provide a smaller distance over which oxygen must diffuse from the capillary to the mitochondrion and to provide an increase of the capillary transit time in the muscles. As a consequence the lactate formed in the exercising muscles could be transported to non-working muscles, to the heart tissue and to the liver for further degradation and energy release. Thus despite a rapid decline in enzyme activity and therefore oxidative potential of the muscles, it is possible that the capacity to remove lactate remains higher than at baseline. As a consequence therefore blood lactate concentration might remain lower after a period of detraining than when measured prior to training, a proposal consistent with the results of the present detraining study which identified at the end of the study the concentration of blood lactate measured for walkers remained 21% below the baseline value.

This observation is also consistent with the proposal made by Donovan and Brooks (1983), that blood lactate concentration is the result of factors influencing both production and removal. Thus whilst mitochondrial enzyme activity might be quickly reduced, thereby increasing lactate formation, the rate at which removal occurs might be maintained by the continued presence of a large number of capillaries. Such an occurrence would therefore explain the present observation of only a slight increase of blood lactate concentration with detraining.

Maximum oxygen uptake was predicted from oxygen uptake and heart rate elicited during the performance of a graded submaximal treadmill walk. With training a trend was observed for $\dot{V}O_2$ max to increase (7.7%) this adaptation was subsequently reversed with detraining. This observation is consistent with the reports derived from other detraining studies and is reflective of the changes reported by Henriksson and Reitman (1977). Thus, for

example, Saltin and coworkers (1968) monitored the fluctuations of $\dot{V}O_2$max associated with a period of bed rest and training. A decline of 28% in $\dot{V}O_2$max occurred with bed rest, as compared with increments ranging from 2 to 52% as a consequence of training. Other studies have reported similar changes for young males (Ekblom et al. 1968; Rowell 1975) and women (Otto et al. 1978; Fringer & Stull 1974; Johannessen et al. 1986).

In addition to $\dot{V}O_2$ and heart rate, the concentration of blood lactate was also measured during the incremental treadmill test. It was observed that at the reference blood lactate concentration of 2 $mmol.l^{-1}$, oxygen consumption was increased with training by 21.7%. Furthermore, after the period of detraining there remained an 8.4% increase above baseline. Support for the observations is derived from a number of training studies, for example Jacobs and colleagues (1981); Kindermann and coworkers (1979); LaFontaine and colleagues (1981) and Londeree & Ames (1975). This index is considered a sensitive and appropriate method by which to evaluate changes in the oxidative potential of skeletal muscle. It is considered particularly useful where, as in the present investigation, the direct measurement of $\dot{V}O_2$max might be inadvisable for reasons of subject safety (Daniels et al. 1978; Katch et al. 1978; Mader et al. 1976). The changes observed for this index are indicative of an improvement of oxidative metabolism which was not apparent with the more traditionally used index of $\dot{V}O_2$max. This therefore appears supportive of the use of this submaximal index of change in aerobic capacity.

The physiological adaptations indicated by heart rate, $\dot{V}O_2$max and blood lactate concentration are evidence that the women involved in the present study did complete the prescribed training and thus that energy was expended.

However, as discussed previously no significant change in body mass was detected. This observation is consistent with the suggestion that a reciprocal increase in energy intake might have gone unidentified or that the intensity at which the exercise was completed was insufficient to attain a threshold for weight loss as identified by Franklin and Rubenfire (1980). However, since body mass and body fatness are associated with changes in the metabolism of lipids and lipoproteins, particularly of HDL-C (Gurr et al. 1989; Katan 1989) the absence of change therefore suggests that these factors had relatively little effect on the observations of lipid and lipoprotein metabolism made in the present study.

The analysis of serum for the concentration of TC was unable to detect any change either within, or between groups. This finding is supported by the observations made during a number of previously completed studies, which have also reported no significant change in TC concentration. These results were obtained with young and middle-aged men after 11-24 weeks of endurance training completed at various exercise intensities (Holloszy et al. 1964; Milesis 1974; Ballantyne et al. 1981, 1982). However, other studies completed with young men (Campbell 1965) and also with women (Moll et al. 1979) have shown reductions of TC concentration with the completion of 10 or 6 weeks of training respectively. The literature is therefore rather inconsistent with regards to the effect of endurance training on TC concentration.

The occurrence of this inconsistency, as noted for HDL-C previously, might in some part be due to the differences in training duration. For example, the data presented in the previous chapter identified a small but significant decrease in TC concentration after one year of training. However, after 3 months of the same training programme the average decrease was a non significant 0.17 $mmol.l^{-1}$.

After the 3 months training involved in the present study the decrease was 0.80 $mmol.l^{-1}$, but remained statistically non significant. Despite the trend towards a larger change in the latter study, the increase remained non significant possibly as a consequence of the small number of subjects involved in the latter programme.

Cross-sectional studies have consistently reported higher concentrations of HDL-C and Tgs for more active individuals. Again though, as was reported for TC, the results of longitudinal studies have proved rather more ambiguous. Frey and coworkers (1982), observed no change in the concentration of HDL-C or Tgs for young women after a 10 week training programme. Support for this observation has been derived from similar findings made by Moll and coworkers (1979), Gaesser and Rich (1984) and Weltman and coworkers (1980). However, with the completion of a longer training period (6 months), Ballantyne and coworkers (1978) were able to distinguish an increase in HDL-C concentration in middle-aged men, but not for women. Similar sex-linked differences were also reported by Brownell and colleagues (1982) and by Wood and coworkers (1988; 1983).

Brownell and coworkers (1982) have proposed that the effects of training might be specific, suggesting that middle-aged women in particular might be less susceptible to exercise-induced changes in lipid and lipoprotein metabolism (Goldberg & Elliot 1985; Sutherland & Woodhouse 1980). Whilst the results of the present study tend to support this view, the observation of trends for favourable changes for HDL-C, TC and TC:HDL-C might also suggest that with a more prolonged training period, changes might be derived, as exemplified by the results of the one year study presented in the previous chapter.

Further evidence to support the proposal that women can

obtain favourable changes in lipoprotein metabolism through exercise training is obtained from the observation of a significant increase in the concentration of the HDL_2-C subfraction. This increase occurred in the absence of a marked increase in the concentration of HDL. It might therefore be possible to influence the conversion of HDL subfractions, in the absence of a change for total HDL. Evidence to support this suggestion is derived from Eisenberg (1984), who has proposed that HDL particles have the capacity to become larger or smaller in the appropriate physiologic situations. For example with an increase in the availability of cholesteryl esters, apolipoprotein A-I, phospholipids and free cholesterol from the breakdown of VLDL, HDL_3 is converted to HDL_2 (Taskinen et al. 1982) in the presence of LCAT. It therefore appears that an increase in the formation of HDL_2-C might be possible in the absence of any increase in HDL-C. However, this remains only speculative.

Apolipoprotein A-I, along with A-II is the major protein moiety of HDL (Osborne & Brewer 1977; Scanu & Landsberger 1980; Brewer et al. 1988), whilst apolipoprotein B is found in association with the Tg rich lipoproteins (Higuchi et al. 1988; Osborne & Brewer 1977; Scanu & Landsberger 1980). Brewer and coworkers (1983) have recognised apolipoprotein A-I to be an important structural protein for the biosynthesis of HDL. Furthermore, the results of investigations completed by Brewer and coworkers (1988) and by Rajput-Williams and colleagues (1988) suggest that defects in the apolipoprotein A-I gene produce a situation in which HDL is almost absent in the plasma. A situation which has been shown to be significantly related to an increased risk of the development of premature cardiovascular disease (Norum et al. 1982; Schaefer et al. 1985). Further confirmation of this relationship has been

derived from the finding of a positive link between LDL and apolipoprotein B with increased CHD, and a negative association between HDL and apolipoprotein A-I with CHD (Noma et al. 1983; Gofman et al. 1966; Rhoads et al. 1976).

As with reports for HDL, cross-sectional studies consistently report a higher concentration of apolipoprotein A-I with endurance trained athletes when compared with their less active contemporaries (Thompson et al. 1983; Nagao et al. 1988; Krauss et al. 1977; Wood & Haskell 1979; Macek et al. 1989). However, the results of training studies are similarly inconsistent. Some reports exist of a favourable increase in the concentration of apolipoprotein A-I and decrease in apolipoprotein B with training (Kiens et al. 1981; Lehtonen et al. 1979; Magnus et al. 1984). Studies also exist however, whose results are consistent with the present study, that is, no difference was observed as a consequence of exercise training (Wood et al. 1983; Freyman et al. 1982; Iltis et al. 1983, 1984; Huttenen et al. 1979). Wood and colleagues (1983) suggest that the inability to discern an effect for exercise on apolipoprotein A-I might be due to the redistribution of HDL subclasses, that is, increased HDL_2 and decreased HDL_3. If proved correct this might also explain the absence of an increase in apolipoprotein A-I despite an increase in HDL_2 -C subfraction.

The evidence presented so far is generally supportive of a favourable exercise-related effect on physiological indices and on the metabolism of lipids and lipoproteins. These effects have been confirmed through the observation of the consequences of the withdrawal of that stimulus, leading to a reversal back towards the levels measured at baseline. In addition, indices of psychological well-being were also monitored.

The analysis of the self-report questionnaires is presented in Table 7:7. With exercise training there was seen to be a trend towards favourable change on each of the aspects of psychological well-being considered. Thus the scores for self-esteem increased, whilst those for GHQ and STAI decreased relative to the levels observed at baseline. These trends are consistent with those reported in the previous chapter. In addition, Blumenthal and colleagues (1982) and Pauly and coworkers (1982) both presented evidence of similar trends as a consequence of low intensity training programmes in which walking was involved either as the only form of exercise or as one of a number of training activities.

Assessment of the scores derived from the Profile of Mood States (POMS) questionnaire also provided evidence of favourable changes in aspects of psychological well-being. A decrease was measured on the indices for tension, fatigue, vigour and for depression, whilst those of confusion and anger remained unchanged. This pattern of response to a period of training closely corresponds with that reported by Blumenthal and coworkers (1982) for middle-aged men after completing a 10 week programme of walk/jogging.

Since similar trends were not observed for the group of controls it is therefore suggested that the walking regimen adopted in the present study was sufficient to confer some psychological as well as physiological benefit. Furthermore, as was discussed in relation to the latter adaptations, a longer duration of training programme might have resulted in significant changes as reported by Hughes and coworkers (1984).

The potential of this form of exercise to confer such adaptations is emphasized by the observation that a reversal of the trends was apparent with detraining. This

return of scores towards baseline level was observed for all indices except STAI. This observation might be evidence of a residual effect on anxiety. It was previously suggested (Dickman 1988) that the influence of exercise training for the management of anxiety might be due to the similarity between psychological and physiological responses to stress. Thus Schwartz and colleagues (1978) proposed that muscle tension is a common physiological result of both psychological and exercise-induced stress. With training therefore the individual would be expected to become accustomed to the 'discomforts of exercise' and as such be less sensitive to their occurrence. As a consequence the level of anxiety would be expected to be lessened with training.

Consistent with the proposal of Schwartz and coworkers (1978) and Schachter and Singer (1962) both suggested the existence of a process of 'learned association'. These authors suggested that the common responses of increased heart rate, increased respiration and muscle tension when associated with a pleasurable experience, for example a brisk walk in the open air, results in a reattribution of the interpretation of the effect of stress. As a consequence the same responses stimulated by psychological stressors would be expected to provoke less anxiety after a period of exercise training.

Finally, an early suggestion (Wolpe 1958) was that the muscle tension developed during physical exercise provided an increase in the awareness of the subsequent feelings of relaxation. Again therefore with exercise training an the increase in the experience of muscle tension might lead to a reduction of the response to muscular tension resulting from psychological stress.

On the basis of similar observations Griest and coworkers (1979) have suggested that exercise training results in

an overall reduction of anxiety leading to a lower level of stress. With less stress there would be expected to be a reduced occurrence of sleep disorders, tension headaches, backaches and so forth. With the reduction of such physical symptoms there is an associated increase of measures of general health.

It is therefore possible that as a result of the programme of exercise training the level of anxiety encountered was reduced. Furthermore, as a possible learned process, that is, re-attribution, this might explain why the level of anxiety encountered at the end of the study remained lower than that at baseline.

7:5. Conclusion.

The aim of the present study was to confirm the efficacy of brisk walking as a means to modify lipid metabolism, physiological response to exercise and aspects of psychological well-being. In each of these areas the adopted indices tended to move in the desired direction with exercise, a tendency which was reversed when the previously less active lifestyle was re-adopted. This pattern of adaptation and reversal is considered indicative of the potential benefits associated with low intensity exercise first introduced in the cross-sectional studies presented in Chapters 4 and 5.

In addition, the adaptations observed during the period of training acted to confirm those observed in the year long study, the results of which were presented in Chapter 6. Further emphasis of the role of exercise was derived with the removal of the training stimulus, the result of which was a general reversal of the previously observed adaptations. The present detraining study therefore confirmed the potential benefits associated with brisk walking for this particular population of middle-aged, formerly sedentary women.

8. GENERAL DISCUSSION.

The series of investigations presented in this thesis was conducted in order to examine the possible benefits which might be derived from a programme of relatively low intensity exercise training. The conceptual background for this approach was presented in the Review of Literature (Chapter 2), included in which was evidence for the existence of a number of favourable adaptations associated with endurance training. For example, when performed on an habitual basis endurance exercise has been shown to be important for the maintenance and/or improvement of functional capacity. In addition, the results of cross-sectional studies have consistently indicated higher, that is, more favourable concentrations of HDL-C for endurance trained athletes when compared with their less active contemporaries.

Much of the available evidence in support of the benefits of regular endurance exercise participation has however been derived from studies of relatively high intensity activities, mainly involving male subjects. Whilst this approach does have its advantages it has become increasingly apparent that such activities are both inappropriate and largely unappealing to women, or to sedentary or older individuals. The aim of the studies included in this thesis therefore was to examine the possibilities for low intensity exercise, in the form of brisk walking, to provoke adaptations similar to those previously recognised for high intensity endurance activities.

8.1. Recognising the potential.

The results of the first two studies (Chapters 4 and 5) were conducted with the aim of establishing a basis for the adoption of brisk walking as a means of training. In general, training programmes designed for the development

and/or maintenance of functional capacity are founded on the inter-relationship between exercise duration (Shephard 1969), frequency (Pollock et al. 1969) and intensity (Gledhill & Eynon 1972). Whilst duration and frequency are aspects of training which can be easily monitored, intensity is rather more problematic. Furthermore, in the guidelines issued by the American College of Sports Medicine (1986) based on the early investigations completed by Hollmann and Venrath (1962) and Karvonen and colleagues (1957), it was suggested that in order to increase functional capacity, exercise intensity must exceed 50% of $\dot{V}O_2$max (equivalent to 60% of the maximal heart rate reserve).

When considered in relation to the guidelines outlined above (ACSM 1986) the results presented in Chapter 4 suggest that brisk walking might be an adequate stimulus to achieve these minimal thresholds, and as a consequence if adopted as the basis of a training regimen might increase functional capacity. Thus of the women completing the submaximal one mile track walk, 75% achieved the threshold prescribed for $\dot{V}O_2$ max. Furthermore, 94% of the group attained the threshold proposed for heart rate. These observations are consistent with those reported by Pocari and coworkers (1987) which indicated that during a similar exercise task 91% of all women and 83% of men aged 50 or above, attained the pre-specified heart rate training threshold denoted as 70% of maximal heart rate reserve.

The results of the present study also appeared to support the proposal made by Cunningham and coworkers (1982) that $\dot{V}O_2$max is the major determinant of self-selected walking pace. For example a significant positive correlation was determined between self-selected pace adopted during the one mile walk and predicted $\dot{V}O_2$max. Furthermore, this finding contradicts that of Bassey (1978) who suggested a

more dominant role for age, a proposal which is further undermined by the failure of the correlation between age and pace to achieve statistical significance.

It was previously suggested that for the majority of women involved in the study the self-selected brisk walking pace was of a sufficient intensity to potentially increase functional capacity. However, for the much smaller group for whom walking appeared insufficiently provocative, a walk/jog programme might be more appropriate to increase functional capacity. Walking might though remain a useful activity for this latter group if the aim of the individual was to simply maintain, rather than increase functional capacity further.

The observations outlined above have widespread implications. For example, in a society in which the demographic trend is increasingly towards an older population the identification of a form of appropriate exercise might be important. Thus by encouraging older individuals to exercise and thereby maintain functional capacity the period over which quality of life and independence might be maintained would be expected to increase.

In summary therefore:

> "..... it seems that brisk walking may indeed offer an adequate aerobic training stimulus for most adults".
>
> (Pocari & coworkers 1987).

Having obtained evidence to support the proposal that brisk walking might provoke improvements of functional capacity, attention was subsequently directed towards obtaining similar evidence of favourable changes in lipid

and lipoprotein metabolism.

Observations derived from cross-sectional studies have revealed higher concentrations of HDL-C, HDL_2-C (Adner & Castelli 1980; Christie et al. 1980; Lehtonen & Viikari 1978; Thompson et al. 1983) and apolipoprotein A (Cook et al. 1986; Thompson et al. 1983; Nagao et al. 1988) for endurance trained athletes when compared with their less active or sedentary, age-matched counterparts.

Furthermore, the results of training studies have shown habitual endurance exercise to be associated with lower concentrations of Tgs and the Tg-rich lipoproteins, particularly LDL-C (Martin et al. 1977; Wood et al. 1976; 1977). Thus Wood and colleagues (1984) found endurance trained athletes to have a relatively lower TC:HDL-C ratio, alternatively termed the risk ratio (Shaper 1987).

As previously indicated much of the information for the relationship between exercise training and changes of lipid and lipoprotein concentrations has been derived from studies of male endurance trained athletes. However, when groups of young males and females were compared no differences in TC and HDL-C emerged on the basis of training-status (Chapter 5). When males and females were considered as separate groups, differences of TC and HDL-C metabolism did emerge both for the comparison of all males with all females (regardless of training) and between males and females who were endurance-, sprint- or untrained.

Thus, for example, a non significant trend towards higher HDL-C concentration was observed for endurance-trained females when compared with sprint- and untrained females. This observation was substantiated by a similar observation derived from groups of middle-aged women. The results of this latter comparison showed significantly

different TC and HDL-C concentrations for runners and walkers when compared with a less active group.

Evidence derived from other studies of the effects of training on the metabolism of HDL-C in women is somewhat inconsistent. For example Rotkis and coworkers (1981) and Gilliam and Burke (1978) were both able to report an increase with endurance training. Moll and colleagues (1979) and Lewis and coworkers (1976) however were unable to identify any change. Furthermore studies in which the same exercise programme has been adopted by both males and females (Ballantyne et al. 1981; Brownell et al. 1982; Frey et al. 1982) have tended to show favourable changes for men but not for women. Together these observations have resulted in the suggestion that exercise-related changes in lipid and lipoprotein metabolism are possibly more difficult to provoke in women than in men (Williams et al. 1982; Goldberg & Elliot 1985). Nonetheless Wood and coworkers (1984) have suggested that adaptations are possible for women, and that the changes which do occur are essentially the same irrespective of sex. The same authors also suggest that the major difference between males and females is that women require a more prolonged period of training prior to change.

The aforementioned sex-linked differences were previously discussed in Chapter 2. Nikkilä and coworkers (1978) have suggested that the differences in training requirements might relate to baseline differences between males and females for lipid and lipoprotein metabolism. Nikkilä and colleagues (1978, 1980) for example, reported a higher concentration of HDL-C and HDL_2-C, along with a higher rate of lipoprotein lipase activity for sedentary women than for endurance trained men. A possible basis for these differences is the effect of oestrogen, the effect of which is to promote HDL metabolism (Fotherby 1989).

This observation has two possible consequences for women, firstly, at baseline the concentration of HDL-C would be higher than that of male subjects. Secondly, the exercise related depression of the oestrogen cycle might reduce the possibility of increasing HDL-C concentration with exercise training. An additional factor to be considered is that with exercise the concentrations of testosterone and androsterone in males are increased (Remes et al. 1979; Mendoza et al. 1981) thus potentially increasing the formation of HDL-C in men.

Based on the above evidence it seems plausible to suggest that the potential to derive further increases of HDL-C, HDL_2-C or lipoprotein lipase activity with endurance training might be less for women when compared with men.

This suggestion appears to be supported by the results presented in Chapter 5, in which differences in lipid and lipoprotein metabolism were identified between the young males and females. However, training-based differences were generally limited to the middle-aged women, with the younger females showing only favourable trends towards higher HDL-C with endurance training.

Together the evidence presented in Chapters 4 and 5 is supportive of the suggestion that brisk walking might be a successful exercise training activity to provoke favourable changes in both functional capacity and lipid and lipoprotein metabolism. On this basis brisk walking was adopted during a year long training programme.

8.2. Realisation of the potential.

The potential previously recognised for brisk walking was subsequently confirmed by the results of the one year training study reported in Chapter 6. Consistent with relatively high intensity endurance training (Saltin et

al. 1968; Wilmore et al. 1970; Seals et al. 1984) functional capacity was increased after the programme of brisk walking. Furthermore, the magnitude of the increase (3 $ml.kg.^{-1}min^{-1}$ or 11.2%) closely corresponded to that reported for other walking studies (Jetté et al. 1988), as well as for programmes of higher intensity training (Saltin et al. 1968).

Whilst $\dot{V}O_2$ max remains the traditional index of functional capacity, adaptation to training was also monitored using indices related to blood lactate concentration. With training the concentration of blood lactate measured in capillary samples obtained at the completion of the one mile submaximal walk was significantly reduced. This observation is consistent with the report by Seals and coworkers (1984) for the effects of a programme of brisk walking with a group of similarly aged males. The rate of oxygen uptake associated with a reference blood lactate concentration of 2 $mmol.l^{-1}$ was also increased at the end of the present training programme, an effect which is typical of endurance training (Yoshida 1984; Kindermann et al. 1979). Both observations, that is, a decrease in blood lactate concentration and an increase in the rate of oxygen consumption at a reference blood lactate concentration of 2 $mmol.l^{-1}$, are considered indicative of an increase in the oxidative potential of the trained muscles (Holloszy & Coyle 1984).

The results are therefore evidence for the effectiveness of brisk walking to provoke changes in functional capacity and skeletal muscle oxidative metabolism. In addition, at the same absolute submaximal exercise intensity, heart rate was reduced. This observation is again consistent with that reported after high intensity endurance training (Barry et al. 1966; Robinson et al. 1973; Seals et al. 1984).

In summary, the results of the programme of brisk walking suggest an increased ability to perform at the same exercise intensity than was possible prior to training, as was exemplified by the submaximal treadmill walk. Secondly, the adaptations also suggest an improved ability to achieve exercise intensities unattainable prior to training. This latter adaptation was exemplified by the significantly improved pace adopted during the maximal track walk.

The effectiveness of brisk walking to provoke favourable physiological adaptations was further substantiated by the observation of the consequences of the removal of the training stimulus (Chapter 7). The resultant changes were consistent with those reported for the effects of detraining associated with relatively high intensity endurance exercise (Saltin et al. 1968; Smith & Stransky 1976; Fringer & Stull 1974). Thus, the return to relative inactivity after a period of low intensity training, resulted in a reversal towards baseline status of the previously reported adaptations. However, the rate at which the indices reverted was not uniform, as observed by Henriksson and Reitman (1977).

The results of the present studies therefore firstly identified a role for brisk walking as a means to affect favourably the physiological responses to exercise in a population of sedentary middle-aged women. Secondly, the potential of brisk walking was realized when the activity was adopted as the means of training. Finally, the effectiveness of the activity was confirmed through the observation of the effects of the removal of the stimulus, the consequence of which was a reversal of the previously observed adaptations associated with training. It is therefore concluded that for this particular population, brisk walking might be an appropriate means to influence positively indices of functional capacity,

aerobic metabolism and variables affecting performance (for example, reduced heart rate). The exercise related changes in these indices therefore suggest an increased ability to work at the same submaximal intensity with less physiological stress and less fatigue than was incurred in the untrained state.

The physiological mechanisms responsible for the observed improvements in performance were not directly investigated in the present study. However, the results of previous studies suggest that regular endurance exercise acts to provoke both central cardiorespiratory (Seals et al. 1984; Barcroft & Swan 1953) and peripheral (metabolic) adaptations (Holloszy & Coyle 1984). Together these changes facilitate the transportation, uptake and utilization of oxygen, which together enhance the oxidative potential of the trained muscles (Morgan et al. 1971). This improved capacity for oxidative metabolism is reflected in a reduction of the rate of lactate accumulation (Holloszy & Coyle 1984). This adaptation is therefore reflected in the lower blood lactate concentration associated with the performance of a standard exercise task after training (LaFontaine et al. 1981; Pollock et al. 1977). Furthermore, the same adaptations result in an increase in $\dot{V}O_2$ required to attain a reference blood lactate concentration (Kindermann et al. 1979). On the basis of these observations, it would seem possible to speculate that the physiological adaptations confirmed for high intensity activity are also involved in the present adaptations to low intensity exercise.

In addition to the benefits derived for endurance performance, the ability to provoke favourable changes in lipid and lipoprotein metabolism was also explored. The suggestion of Wood and coworkers (1984) that such changes were possible for women was confirmed by the results of

the one year training study (Chapter 6).

The results showed a significant increase (0.23 $mmol.l^{-1}$) in the concentration of HDL-C after 3 months of the year long study. This initial increase was followed by further smaller increments over the subsequent months. The overall increase recorded at the conclusion of the study therefore amounted to 0.32 $mmol.l^{-1}$ or 27%. In addition, there was also found to be a small (0.35 $mmol.l^{-1}$ or 6.5%) but significant decrease for the concentration of TC over the duration of the study. Together, the changes resulted in a 32.7% reduction of the TC:HDL-C ratio. On the basis of epidemiological studies, one could speculate that as a consequence of these exercise related effects on the metabolism of TC and HDL-C there might be a 54-64% decrease in the risk of CHD for this group of women (Gordon et al. 1989). These changes therefore appear supportive of a potential role for low intensity exercise as a possible means by which to favourably alter blood lipid and lipoprotein metabolism.

Further support for this proposal was derived from the results of the effects observed with detraining (Chapter 7). Previous studies have shown inactivity or low activity levels to be associated with concentrations of blood lipids and lipoproteins giving a predisposition towards the development of CHD (Saltin et al. 1968; LaPorte et al. 1983). A similar trend was also observed as a consequence of the detraining and removal of the exercise stimulus (Chapter 7). Associated with this period of reduced activity there was seen to be a general reversal of the favourable adaptations associated with exercise performance back towards baseline values. Again this observation suggests brisk walking might be an appropriate form of exercise for this group of middle-aged formerly sedentary women.

Furthermore, in the studies reported in this thesis, the differences observed between the groups for TC, HDL-C, TC:HDL-C and HDL_2-C concentrations occurred in the absence of differences in the indices of dietary composition, body mass and composition. This is an important observation, since these have also been found to be effective in altering the concentrations of blood lipids and lipoproteins. Reviews by Gurr and coworkers (1989) and by Katan (1989) identify the importance of changes in dietary composition for lipid and lipoprotein metabolism. It has been recognised for example, that diets containing a large percentage of carbohydrate relative to fat are associated with increased metabolism of Tg-rich lipoproteins (LDL-C and VLDL). In contrast, low carbohydrate consumption is associated with high HDL-C metabolism (Ahrens et al. 1961; Wilson & Lees 1972; Brussard et al. 1982). Furthermore, investigations completed by Johansson and Mehdus (1974) identified a positive correlation between alcohol intake and the concentration of HDL-C. These observations therefore illustrate the important effect of dietary modification and the relevance to the present observation for lipid and lipoprotein metabolism.

Also to be considered is the influence of body mass and body fatness. A negative association has been determined between obesity and HDL-C concentration, thus decreases in body mass and body fatness have been found to be associated with increased HDL-C concentrations (Wilson & Lees 1972; Contaldo et al. 1980). Therefore, in the absence of any differences between experimental and control groups, it is suggested that the effects of body fatness and dietary composition had minimal influence on the differences in lipid and lipoprotein metabolism observed in association with increased activity. An alternative explanation of the observed differences must therefore be sought.

In 1982 Assmann made the following statement concerning the knowledge then available for the effects of exercise on the metabolism of HDL-C:

> "The biochemical mechanism producing an increase in HDL cholesterol during physical exercise is unexplained at present. In studies by Nikkilä and colleagues (1978, 1980) athletes were shown to have higher levels of LPL* in muscle and adipose tissue. However, details of the influence of hormones on lipolytic activity in athletes are not presently available".
>
> (Assmann 1982).

(*LPL=lipoprotein lipase)

Whilst this statement remains relevant information is available to suggest a possible basis for the observed effect of endurance training on the metabolism of HDL-C and TC. According to recent studies HDL is essentially formed in the plasma as a result of the lipolysis of chylomicrons (Schaefer et al. 1978). It is proposed that during the hydrolysis of chylomicrons discoidal HDL particles are formed. These precursor particles consist of a lipid bilayer membrane associated with apolipoprotein A-I (Tall & Small 1980). The discoidal particles consist almost totally of free cholesterol and almost no esterified cholesterol. The enzyme lecithin: cholesterol acyltransferase (LCAT) is active in the conversion of these particles to the circulating form of HDL, whereby the discoidal particles are transformed into pseudomicellar globular HDL. This transformation is consequent to the esterification of the cholesterol which the particles contain (Hamilton 1978).

The association between chylomicron metabolism and HDL formation has been supported by evidence that individuals with a high rate of intravascular lipolysis are more

likely to have a high rather than low HDL-C concentration (Nikkilä et al. 1980). It has also been suggested that LCAT is involved in the regulation of the HDL subfractions, whereby HDL_2 is formed from HDL_3 by the incorporation of apolipoprotein C, phospholipids and cholesterol (Patsch et al. 1978, 1980) released in the course of the lipolytic activity of LCAT (Schmitz et al. 1982) and lipoprotein lipase (Eisenberg 1984). The involvement of these two enzymes in the formation of HDL is depicted in Figure 8:1.

Evidence from previous exercise training studies suggest a possible effect on the lipolytic enzymes lipoprotein lipase, hepatic lipase and LCAT (Ruys et al. 1989; Nikkilä et al. 1978). It has been proposed (Nikkilä et al. 1978) that the activity of lipoprotein lipase, found on the endothelial tissue of the muscle capillaries is increased as a consequence of capillary proliferation resulting from training (Keins & Lithell 1985; Saltin & Gollnick 1983). As a consequence of an increase in the activity of lipoprotein lipase there would be expected to be an acceleration of the rate of catabolism of Tg-rich lipoproteins thereby reducing the plasma concentration of Tg. Furthermore, an increase in the availability of cholesterol, phospholipids and apolipoprotein resulting from the breakdown of VLDL would increase the potential for the formation of discoidal ('nascent') HDL particles.

This proposal is consistent with the observation that highly trained endurance athletes have higher adipose tissue (Nikkilä et al. 1978), skeletal muscle (Nikkilä et al. 1978) and heparin-releasable (Krauss et al. 1979) lipoprotein lipase activity when compared with sedentary controls. An increase in lipoprotein lipase activity associated with endurance training was also reported by Lithell and colleagues (1979).

ORIGIN OF HIGH DENSITY LIPOPROTEIN

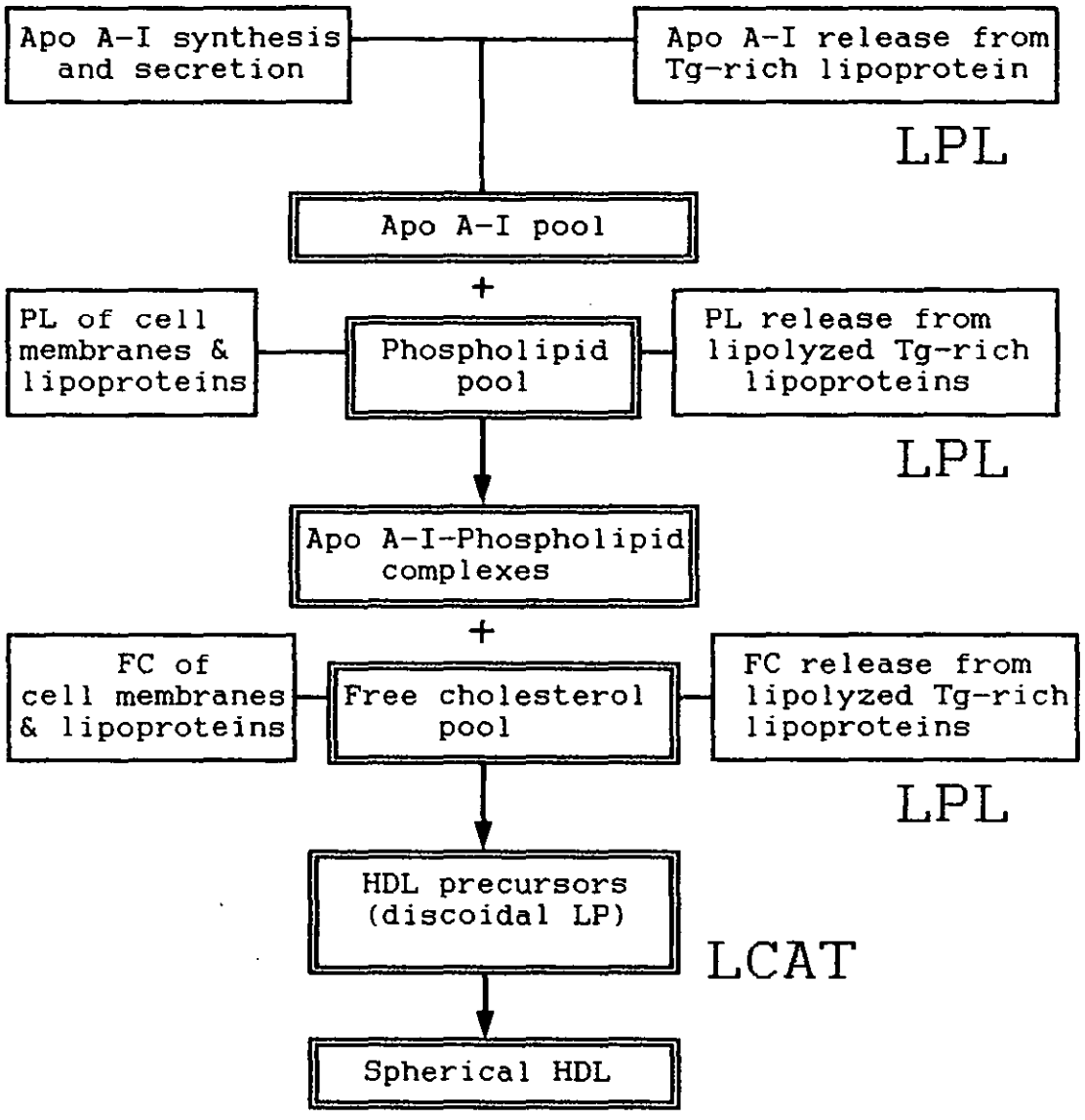


FIGURE 8:1 Schematic representation of HDL formation (Eisenberg 1984).

In addition to the effects of endurance training on the lipolytic activity of lipoprotein lipase, observations of hepatic lipase have also been made. Krauss and coworkers (1979) suggested that as a consequence of endurance training the activity of hepatic lipase is depressed. Evidence to support this proposal has been derived by Kuusi and coworkers (1982) who reported the existence of a significant negative correlation between the concentration of HDL-C and hepatic lipase activity in highly trained runners. Similarly, Peltonen and coworkers (1981) reported the activity of hepatic lipase to be reduced and the concentration of HDL-C to increase in a group of middle-aged men after a 15 week exercise training programme. In addition, Taskinen and Nikkilä (1981) also reported a negative correlation between hepatic lipase activity and HDL_2 concentration. On the basis of their observations the same authors have proposed that hepatic lipase might act to convert HDL_2-C to HDL_3-C, thus an exercise related decrease in the activity of this enzyme would be expected to be associated with an increased concentration of HDL_2-C. This suggestion is consistent with the report by Kuusi and coworkers (1979) and also with the evidence presented in Chapter 7.

In addition to the information on lipoprotein lipase and hepatic lipase evidence is also available for the effects of endurance training on the activity of the enzyme LCAT. As has been shown (Figure 8:1) LCAT is involved in the final steps in the formation of HDL. Lecithin:cholesterol acyltransferase acts to catalyze the transfer of fatty acids in the plasma from lecithin to cholesterol. Thus an increase in LCAT activity might be associated with an increased concentration of HDL-C. Evidence to support this proposal has been derived from a number of training studies with young (Marniemi et al. 1982) and middle-aged (Marniemi & Hietanen 1982) men.

On the basis of this evidence it might be possible to suggest that the lower concentrations of TC and higher concentrations of HDL-C reported in the present series of studies in association with a programme of brisk walking might also be due to similar effects as those reported for training of a higher intensity (e.g. running). Thus the activity of lipoprotein lipase might be increased in the trained skeletal tissue (possibly along with a proliferation of muscle capillaries the endothelium of which is the site for this activity). As a consequence of this increase the rate of catabolism of Tg-rich lipoproteins might be enhanced. Furthermore the resulting products of this breakdown act as the basis for the formation of the 'immature' or discoidal HDL particle. This process would be further encouraged with the increased activity of LCAT which is important in the development of spherical or mature HDL (Eisenberg 1984). Finally, hepatic lipase activity is likely to depress the rate of HDL-C and HDL_2-C catabolism thereby also increasing the concentrations of these particles observed after training.

The results reported in Chapter 6 revealed a significant increase in the concentration of HDL-C (27%) and a small but significant decrease for TC concentration (6.5%). Furthermore, much of the increase of HDL-C was observed within the first three months of the one year study. As a consequence the subsequent study was designed on the basis of three months of training followed by a further three months during which the exercise stimulus was withdrawn. However, whilst the results of the latter study did show a trend towards favourable changes for indices of both fitness and lipid and lipoprotein metabolism, statistical significance was achieved only for the increase in apolipoprotein A. At this point it seems pertinent to discuss the differences in the effectiveness of the training adopted in the studies

presented in Chapters 6 and 7. Table 8:1 includes baseline information derived for the experimental groups of each study.

Age is a factor recognised to influence lipid and lipoprotein metabolism (Mann et al. 1988). The women involved in the second study were found to be on average 2.4 years older than those partaking in the one year programme. Furthermore, the range of the ages was only 15.5 years compared with 32.2 years for the 6 and 12 month studies respectively. On the basis of this evidence it might therefore be suggested that the older group would be more likely to include women approaching or attaining menopausal status.

The influence of the menopause, or more particularly the oestrogen cycle, on the metabolism of HDL-C has already been addressed elsewhere. However, it might be plausible to suggest that for the group of slightly older subjects a depression of the oestrogen cycle, firstly due to the menopause, and secondly due to the effect of exercise training might limit the potential for an exercise related increase in HDL-C metabolism to a greater extent than was observed for the former group.

An additional factor to be identified are differences in body composition between the two groups. At baseline the women involved in the latter study were shown to have higher body mass, body mass index, sum of 4 skinfolds and ratio of the circumference about the waist and hips, however, the actual figures for the percentage of body fat appeared no different between the groups. It should be recognised though that the figures presented were derived using different techniques. Body fatness was assessed using the hydrostatic measuring technique on the women involved in the one year study, however, with the latter study, body fatness was estimated using the sum of

Table 8:1. Characteristics of walkers involved in the one year training programme (Chapter 6) and the training-detraining study (Chapter 7) (Mean±sem).

		One year study	6 month study
Physical characteristics			
AGE (yr)	$\bar{x}$	44.9±1.5	47.3±2.0
	range	30.6 to 62.8	39.3 to 54.8
MASS (kg)	$\bar{x}$	64.0±1.7	65.7±2.6
	range	48.8 to 83.7	51.1 to 80.7
BMI	$\bar{x}$	23.9±0.6	24.8±1.4
($kg.m^{-2}$)	range	19.1 to 32.3	19.7 to 34.8
Body fat (%)	$\bar{x}$	36±1	35±2
Indices of exercise performance			
Submax pace ($m.s^{-1}$)	$\bar{x}$	1.73±0.03	1.76±0.03
$\dot{V}O_2$ max ($ml.^{-1}kg.^{-1}min$)	$\bar{x}$	26.7±0.7	31.5±1.9
$\dot{V}O_2$ at 2 $mmol.l^{-1}$	$\bar{x}$	17.0±0.5	17.8±1.4
Blood lipids			
TC ($mmol.l^{-1}$)	$\bar{x}$	5.35±0.23	5.67±0.35
HDL-C ($mmol.l^{-1}$)	$\bar{x}$	1.17±0.08	1.21±0.07
TC:HDL-C	$\bar{x}$	5.2±0.4	4.7±0.3
Dietary analysis			
Energy (MJ)	$\bar{x}$	7.7±0.4	7.9±0.7
%Fat	$\bar{x}$	33.8±0.9	35.8±1.9
%Carb	$\bar{x}$	48.2±1.2	45.2±1.6
%Alc	$\bar{x}$	1.9±0.4	2.6±0.8

skinfolds approach (Wolmersley & Durnin 1973).

Since body fatness and HDL-C concentration have been found to be inversely related (Wilson & Lees 1972; Contaldo et al. 1980) it is possible that the differences in body composition between the two groups was also a factor which might be influential on the effectiveness of the training programme.

However, whilst the results of the latter study might initially appear disappointing, further inspection of the results reveal that in spite of the disadvantages of age and body composition, the training adopted was sufficient to provoke favourable trends for lipid and lipoprotein metabolism in this group of subjects. Two conclusions might be drawn from these observations, firstly, in support of the suggestion made by Wood and colleagues (1984) women apparently require a prolonged period of training. Furthermore, it might be that menopausal women also require a longer period of training than younger women. Secondly, perhaps an over-riding influence of the absence of any statistically significant change in the observed variables was the small numbers of subjects involved in the second study. With only 10 experimental subjects as compared with 28 in the former study, the attainment of a statistically acceptable level of difference is increasingly difficult to achieve.

8.3. Brisk walking and psychological well-being.
In addition to the benefits already described, endurance exercise is also considered a potentially positive influence on a number of different aspects of psychological well-being. Reports have been made of improved self-esteem (Dowell et al. 1968; Hughes 1984), of general health, and of anxiety and depression (Blumenthal et al. 1982; Pauly et al. 1982) consequential to endurance training. A tendency towards similar changes

was also indicated with brisk walking. Both training studies were able to report a tendency for subjects to report "feeling better", an anecdotal report, which was confirmed by the trends observed from the completed battery of psychological questionnaires. Scores for anxiety were generally decreased, response to the POMS questionnaires revealed a more positive mood as a consequence of training and scores on the GHQ indicated an improvement in indices of general health. Together these tendencies were considered to be indicative of a beneficial effect consequential to brisk walking.

The mechanisms underlying these favourable changes were not investigated, however evidence exists to suggest that habitual endurance exercise brings about a reattribution of psychological stress symptoms and thereby a reduction of anxiety (Griest et al. 1978).

8.4. Conclusion.

The series of studies included in this thesis recognizes the effectiveness of brisk walking to increase functional capacity. Furthermore, with training favourable adaptations of lipid and lipoprotein metabolism were indicated, that is, an increase in the concentration of HDL-C and a decrease in the concentration of TC. Together these changes might reflect a reduced risk for the development of CHD. Finally, the results of the investigations indicate a trend towards the favourable change of aspects of psychological well-being.

It is therefore suggested that low intensity exercise, in the form of brisk walking, might be an appropriate and attractive form of activity for this population of middle-aged, formerly sedentary women. Being of sufficient intensity to stimulate favourable changes in

physiological response to exercise, in lipid and lipoprotein metabolism and in aspects of psychological well-being.

REFERENCES.

ADAMS, G.M. & de VRIES, H.A. (1973). Physiological effects of an exercise training regimen upon women aged 52 to 79. Journal of Gerontology. 28: 50-55.

ADNER, M.M. & CASTELLI, W.P. (1980). Elevated high-density lipoprotein levels in marathon runners. Journal of American Medical Association. 243: 534-536.

AHRENS, E.H., HIRSCH, J., OETTE, K., FARQUHAR, J.W. & STEIN, Y. (1961). Carbohydrate-induced and fat-induced hyperlipidemia. Trans Association of American Physicians. 74: 134-146.

ALBERS, J.J. & SEGREST, J.P. (1986). Eds. The Plasma Lipoproteins. Vol. A. preparation, Structure and Molecular Biology. Vol. B. Characterization, Cell Biology and Metabolism. Methods in Enzymology. 128, 129.

ALBRINK, M.J., KRAUSS, R.M., LINDGREN, F.T., VON DER GROEBEN, J., PAN, S. & WOOD, P.D. (1980). Intercorrelations among plasma high density lipoprotein, obesity and triglycerides in a normal population. Lipids. 14: 668-676.

ALLISON, T.G., IAMMARINO, R.M., METZ, K.F., KULLER, L.H. & ROBERTSON, R.J. (1981). Failure of exercise to increase high density lipoprotein cholesterol. Journal of Cardiac Rehabilitation. 1: 257-265.

ALTEKRUSE, E.B. & WILMORE, J.H. (1973). Changes in blood chemistries following a controlled exercise program. Journal of Occupational Medicine. 15: 110-113.

AMERICAN COLLEGE OF SPORTS MEDICINE (1986). Guidelines for exercise testing and prescription. 3rd Edition. Philadelphia. Lea and Febiger. 34.

ANDERSON, D.W., NICHOLS, A.V., PAN, S.S. & LINDGREN, F.T. (1978). High density lipoprotein distribution: resolution and determination of three major components in a normal population sample. Atherosclerosis. 29: 161-179.

APPELBAUM-BOWDEN, D., HAFFNER, S.M., HARTSOOK, E., LUK, K.H., ALBERS, J.J. & HAZZARD, W.R. (1984). Down regulation of the low-density lipoprotein receptor by dietary cholesterol. American Journal of Clinical Nutrition. 39: 360-367.

ARAB, L., SCHELLENBERG, B. & SCHLIERF, G. (1982). Nutrition and health. A survey of young men and women in Heidelberg. Annals of Nutrition and Metabolism. 26 (Suppl 26): 1-244.

ASMUSSEN, E., KLAUSEN, K., NIELSEN, L.E., TECHOW, O.S.A. & TONDER, P.J. (1974). Lactate production and aerobic work capacity after prolonged exercise. Acta Physiologica Scandinavica. 90: 731.

ASSMANN, G. (1982). Lipid Metabolism and Atherosclerosis. Schattauer Verlag GmbH, Stuttgart, Germany.

ASTRAND, I. (1960). Aerobic work capacity in men and women with special reference to age. Acta Physiologica Scandinavica. 49 (Suppl. 169).

ASTRAND, I., ASTRAND, P-O., HALLBACK, I. & KILBOM, A. (1973). Reduction in maximal oxygen intake with age. Journal of Applied Physiology. 35: 649-654.

ASTRAND, P.-O. (1956). Human physical fitness with special reference to sex and age. Physiological Review. 36: 307.

ASTRAND, P.-O. & RODAHL, K. (1986). Textbook of Work Physiology. Third Edition. New York: Mcgraw-Hill.

ATOMI. Y. & MIYASHITA, M. (1980). Effect of training intensity in adult females on aerobic power, related to lean body mass. European Journal of Applied Physiology. 44: 109-116.

AUGUSTIN, J. & GRETEN, H. (1979). The role of lipoprotein lipase: molecular properties and clinical relevance. In: Atherosclerosis Reviews. Paoletti, R. & Gotto, A.M, (Eds). Raven Press. New York.

BACHORIK, P.S. (1985). Interpretation of plasma lipid and lipoprotein measurement. Clinical Chemistry. 31: 7: 1107-1108.

BACHORIK, P.S., WALKER, R., BROWNELL, K.D., STUNKARD, A.J. & KWITEROVICH, P.O. (1980). Determination of high density lipoprotein-cholesterol in stored human plasma. Journal of Lipid Research. 21: 608-616.

BACHORIK, P.S., WALKER, R.E. & KWITEROVICH, P.O. (1982). Determination of high density lipoprotein-cholesterol in human plasma stored at -70^{0} C. Clinica Chemica. 23: 1236-1242.

BAECKE, J.A.H., VAN STAVEREN, W.A. & BUREMA, J. (1983). Food consumption, habitual physical activity and body fatness in young Dutch adults. American Journal of Clinical Nutrition. 37: 278-286.

BAHRKE, M.S. (1979). Exercise, meditation and anxiety reduction: A review. American Corrective Therapy Journal. 33: 41-44.

BAHRKE, M.S. & MORGAN, W.P. (1981). Anxiety reduction following exercise and meditation. In: Psychology of Running. Sacks, M.H. & Sachs, M.L. (Eds). Champaign, Illinois: Kinetics Publishers Inc. 57-66.

BALLANTYNE, D., CLARK, R.S. & BALLANTYNE, F.C. (1981). The effect of physical training on plasma lipids and lipoproteins. Clinical Cardiology. 4: 1-4.

BALLANTYNE, F.C., CLARK, R.S., SIMPSON, H.S. & BALLANTYNE, D. (1982a). High density and low density lipoprotein subfractions in survivors of myocardial infarction and in control subjects. Metabolism 31: 433-437.

BALLANTYNE, F.C., CLARK, R.S., SIMPSON, H.S. & BALLANTYNE, D. (1982b). The effect of moderate physical exercise on the plasma lipoprotein subfractions of male survivors of myocardial infarction. Circulation 65: 913-918.

BANDURA, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. Psychological Review. 84: 191-215.

BANG, O. (1936). The lactate content of the blood during and after muscular exercise in man. Skandinavisk Archiv Fur Physiologie (Suppl.) 10: 51-82.

BARCROFT, H. & SWAN, H.J.C. (1953). Sympathetic control of human blood vessels. Edward Arnold (Ltd). London.

BARNARD, R.J., EDGERTON, V.R. & PETER, J.B. (1970). Effect of exercise on skeletal muscle I. Biochemical and histochemical properties. Journal of Applied Physiology. 28: 762-766.

BARRY, A.J., DALY, J.W., PRUETT, E.D., STEINMETZ, J.R., BIRKHEAD, N.C. & RODAHL, K. (1966). The effects of physical conditioning on older individuals. I. Work capacity, circulatory-respiratory functions and work electro-cardiograms. Journal of Gerontology. 21: 182-191.

BASSEY, E.J. (1978). Age, inactivity and some physiological responses to exercise. Gerontology. 24: 66-77.

BEHNKE, A.R., FEEN, B.G. & WELHAM, W.C. (1942). The specific gravity of healthy men. Journal of the American Medical Association. 118: 495-498.

BERG, A., RINGWALD, G. & KEUL, J. (1980). Lipoprotein-cholesterol in well-trained athletes. A preliminary communication: reduced HDL-cholesterol in power athletes. International Journal of Sportsmedicine. 1: 137-138.

BESSARD, T., SCHUTZ, Y. & JÉQUIER, E. (1983). Energy expenditure and postprandial thermogenesis in obese women before and after weight loss. Journal of Clinical Nutrition. 38: 690-693.

BEYNEN, A.C. & KATAN, M.B. (1985). Effects of egg yolk feeding on the concentration and composition of serum lipoproteins in man. Atherosclerosis. 54: 157-166.

BEYNEN, A.C., KATAN, M.B. & VAN ZUTPHEN, L.F.M. (1987). Hypo- and hyper-responders: individual differences in response of serum cholesterol concentration to changes in diet. Advances in Lipid Research. 22: 115-171.

BINGHAM, S.A. (1987). The dietary assessment of individuals; methods, accuracy, new techniques and recommendations. Nutrition Abstracts and Review. 57: 705-42.

BLACKBURN, H. (1980). Risk factors and cardiovascular disease. In: The American Heart Association Heart Book. Dutton, E.P (Ed.). New York.

BLAZA, S. & GARROW, J.S. (1983). Thermogenic response to temperature, exercise and food stimuli in lean and obese women, studied by 24h direct calorimetry. British Journal of Nutrition. 49: 171-180.

BLOCK. G. (1990). Human dietary assessment: methods and issues. National Cancer Institute. 653-660.

BLUMENTHAL, J.A., WILLIAMS, R.S., NEEDLES, T.L. & WALLACE, A.G. (1982). Psychological changes accompany aerobic exercise in healthy middle-aged adults. Psychosomatic Medicine. 44: 6: 529-536.

BONEN, A., BELCASTRO, A.N. & LING, W.Y. (1981). Profiles of selected hormones during menstrual cycles of teenage athletes. Journal of Applied Physiology. 50: 545-551.

BOOYENS, J. & KEATINGE, W.R. (1957). The expenditure of men and women walking. Journal of Physiology. 138: 165-171.

BOUCHARD, C., CARRIER, R., BOULAY, M., THIBAULT-POIRIER, MC. & DULAC, S. (1975). Le development du systeme de transport de l'oxygene chez les jeunes adultes. Quebec: Editions du Pelican.

BOYDEN, T.W., PAMENTER, R.W., ROTKIS, T.C., MORRISON, D.A., FREUND, B.J., STANFORTH, P.R. & WILMORE, J.H. (1987). Effects of exercise training on plasma cholesterol, high-density lipoprotein cholesterol and sex steroid concentrations in women. Coronary Heart Disease in Women: proceedings of an Annual National Instute of

Health workshop. Haymarket Doyma Inc. New York. 158-163.

BRADLEY, D.D., WINGERD, J., PETITTI, D.B., KRAUSS, R.M. & RAMCHARAN, S. (1978). Serum high-density-lipoprotein cholesterol in women using oral contraceptives, estrogens and progestins. The New England Journal of Medicine. 299: 17-20.

BREWER, H.B., GREGG, R.E., HOEG, J.M. & FOJO, S.S. (1988). Apolipoproteins and lipoproteins in human plasma: an overview. Clinical Chemistry. 34: B4-B8.

BRIER, C., DREXEL, H., LISCH, H.J., MULHBERGER, V. & HEROLD, M. (1985). Effects of therapy with cholestryamine on progression of coronary arteriosclerosis: result of the NHLBI Type II coronary intervention study. Circulation. 69: 313-324.

BRODIE, D.A. (1988). Techniques for measurement of body composition. Sports Medicine. 5: 11-40.

BROWN, D.F. & DAUDISS, K. (1973). Hyperlipoproteinemia. Prevalence in a free-living population in Albany, New York. Circulation. 47: 558-566.

BROWN, M.S. & GOLDSTEIN, J.L. (1976). Receptor-mediated control of cholesterol metabolism. Science. 191: 150-154.

BROWN, M.S. & GOLDSTEIN, J.L. (1987). Teaching old dogmas new tricks. Nature. 330: 113-114.

BROWN, M.S. & GOLDSTEIN, J.L. (1984). How LDL receptors influence cholesterol and atherosclerosis. Scientific American. 251: 52-60.

BROWNELL, K.D., BACHORIK, P.S., & AYERLE, R.S. (1982). Changes in plasma lipid and lipoprotein levels in men and women after a program of moderate exercise. Circulation. 65: 3: 477-484.

BRUCE, R.A. (1984). Exercise, functional aerobic capacity, and ageing-another viewpoint. Medicine and Science in Sports and Exercise. 16: 1: 8-13.

BRUNNER, D., WEISBORT, J., MESHULAM, N., SCHWARTZ, S., GROSS, J., SALTZ-RENNERT, H., ALTMAN, S. & LOEBL, K. (1987). Relation of serum total cholesterol and high density lipoprotein cholesterol percentage to the incidence of definite coronary events: twenty-year follow-up of the Donolo-Tel Aviv prospective coronary artery disease study. American Journal of Cardiology. 59: 1271-1276.

BRUSSARD, J.H., KATAN, M.B., GROOT, P.H.E., HAVEKES, L.M. & HAUVAST, J.G.A.J. (1982). Serum lipoproteins of healthy persons fed a low fat diet or a polyunsaturated fat diet for three months: a comparison of two cholesterol lowering diets. Atherosclerosis. 42: 205-219.

BUDOHOSKI, L. (1985). Exercise-induced changes in lipoprotein lipase activity (LPLA) in skeletal muscles of the dog. European Journal of Physiology. 405: 188-192.

BULLEN, B.A., SKRINAR, G.S. & BEITINS, I.Z. (1985). Induction of menstrual disorders by strenuous exercise in untrained women. The New England Journal of Medicine. 312: 1349-1353.

BUSH, T.L. & BARRETT-CONNOR, E. (1985). Non contraceptive estrogen use and cardiovascular disease. Epidemiological Review. 7: 80-104.

BUSH, T.L., FRIED, L.P. & BARRETT-CONNOR, E. (1988). Cholesterol, lipoproteins and coronary heart disease in women. Clinical Chemistry. 34(B), B60-B70.

BUSKIRK, E.R. & TAYLOR, H. (1967). Maximal oxygen uptake and its relation to body composition, with special reference to chronic physical activity and obesity. Journal of Applied Physiology. 11: 72-78.

BYRD, O.E. (1963). A survey of beliefs and practices of psychiatrists on the relief of tension by moderate exercise. Journal of Health. 33: 426-427.

BYRD, R.J., SMITH, D.P. & SHACKLEFORD, C.B. (1974). Jogging in middle-aged men: effects on cardiovascular dynamics. Archives of Physical Medical Rehabilitation. 55: 301-305.

CAGGUILA, A.W., CHRISTAKIS, G., FARRAND, M., HULLEY, S.B., JOHNSON, R., LASSER, N.L., STAMLER, J. & WIDDOWSON, G. (1981). The multiple risk factor intervention trial (MRFIT)-IV. Intervention on blood lipids. Preventative Medicine. 10: 443-475.

CAMPBELL, D.E. (1965). Influence of several physical activities on serum cholesterol concentrations in young men. Journal of Lipid Research. 6: 478.

CARLSON, L.A. & BÖTTIGER, L.E. (1972). Ischaemic heart disease in relation to fasting values of plasma triglycerides and cholesterol. Stockholm Prospective Study. Lancet. 865-868.

CARLSON, L.A. & ERICSSON, M. (1975). Quantitive and qualitative lipoprotein analysis. Part I. Studies in healthy men and women. Atherosclerosis. 21: 417-433.

CARLSON, L.A. & MÖSSFELDT, F. (1964). Acute effects of prolonged, heavy exercise on the concentration of plasma lipids and lipoproteins in man. Acta Physiologica Scandinavica 62: 51-59.

CARR, D.B., BULLEN, B.A., SKINAR, G.S., ARNOLD, M.A., ROSENBLATT, M., BEITINS, I.Z., MARTIN, J.B. & McARTHUR, J.W. (1981). Physical conditioning facilitates the exercise-induced secretion of beta-endorphin and beta-lipoprotein in women. The New England Journal of Medicine. 305: 560-562.

CARTER, R. (1977). Exercise and Happiness. Journal of Sports Medicine and Physical Fitness. 17: 307-313.

CASTELLI, W.P., DOYLE, J.T., GORDON, T., HAMES, C.G., HJÔRTLAND, M.C., HULLEY, S.B., KAGAN, A. & ZUKEL, W.J. (1977). Alcohol and blood lipids-the cooperative lipoprotein phenotyping study. Lancet. 2: 153-155.

CHARLESWORTH, E.A. & NATHAN, R.G. (1984). Stress management-A comprehensive guide to wellness. New York Atheneum.

CHASEY, W.C., SWARTZ, J.D. & CHASEY, C.G. (1974). Effect of motor development on body image scores for institutionalized mentally retarded children. American Journal of Mental Deficiency. 78: 440-445.

CHRISTENSEN, N.J., GALBO, H., HANSEN, J.F., HESSE, B., RICHTER, E.A. & TRAP-JENSEN, J. (1979). Catecholamines and exercise. Diabetes. 28. (Suppl. I). 58-62.

CHRISTIE, R.J., BLOORE, H.G. & LOGAN, R.L. (1980). High-density lipoprotein (HDL) cholesterol in middle-aged joggers. New Zealand Medical Journal. 91: 39-40.

CLAUSEN, J.P. (1976). Circulatory adjustments to dynamic exercise and effect of physical training in normal subjects and in patients with coronary disease. Cardiovascular Disease. 18: 459.

COHEN, L. & HOLLIDAY, M. (1983). Statistics for social scientists: an introductory text with computer programs in basic. Harper & Row. London.

CONTALDO, F., STRAZZULO, P., POSTIGLIONE, A., RICCARDI, G., PATTI, L., DI BIASE, G. & MANCINI, M. (1980). Plasma high density lipoprotein in severe obesity after stable weight loss. Atherosclerosis. 37: 163-167.

CONNETT, R.J., GAYESKI, T.E.J. & HONIG, C.R. (1984). Lactate accumulation in fully aerobic working dog gracilis muscle. American Journal of Physiology. 246: H120.

COOK, T.C., LaPORTE, R.E., WASHBURN, R.A., TRAVEN, N.D., SLEMENDA, C.W. & METZ, K.F. (1986). Chronic low level physical activity as a determinant of high density lipoprotein cholesterol and subfractions. Medicine and Science in Sports and Exercise. 18: 6: 653-657.

COOPER, G.R., MYERS, G.L., SMITH, S.J. & SAMPSON, E.J. (1988). Standardization of lipid, lipoprotein and apoplipoprotein measurements. Clinical Chemistry. 34/8(B), B95-B105.

COOPER, K.H. (1983). Aerobic update. Runners World. 49: 79-80.

CORONARY PREVENTION GROUP. (1986) Briefing papers 1-10.

COSTONGS, G.M.P.J., JANSON, P.C.W., BAS, B.M., HERMANS, J., VAN WERSCH, J.W.J. & BROMBACHER, P.J. (1985). Short-term and long-term intraindividual variations and critical differences of clinical chemical laboratory parameters. Journal of Clinical Chemistry and Clinical Biochemistry. 23: 7-16.

COUSINEAU, D., FERGUSON, R.J., DeCHAMPLAIN, J., GAUTHIER, P., CÔTÉ, P. & BOURASSA, M. (1977). Catecholamines in coronary sinus during exercise in man before and after training. Journal of Applied Physiology. 43: 801-806.

CREWS, T.R. & ROBERTS, J.A. (1976). Effects of interaction of frequency and intensity of training. Research Quarterly. 47: 48-55.

CUNNINGHAM, D.A., MONTOYNE, H.J., METZNER, H.L. & KELLER, J.B. (1968). Active and leisure time activities as related to age among males in a total population. Journal of Gerontology. 23: 551-556.

CUNNINGHAM, D.A., RECHNITZER, P.A., PEARCE, M.E. & DONNER, A.P. (1982). Determinants of self-selected walking pace across ages 19 to 66. Journal of Gerontology. 37: 5: 560-564.

CURETON, T.K. (1969). The Physiological Effects of Exercise Programs Upon Adults. Springfield. C. Thomas Company.

DALE, E., GERLACH, D.H. & WILHITE, A.L. (1979). Menstrual dysfunction in distance runners. Obstetrics and Gynaecology. 54: 47-49.

DANIELS, J.T., YARBOROUGH, R.A. & FOSTER, C. (1978). Changes in maximal oxygen uptake and running performance with training. European Journal of Applied Physiology. 39: 249-254.

DAVIES, C.T.M. & KNIBBS, A.V. (1971). The training stimulus, the effects of intensity, duration and frequency of effort on maximum aerobic power output. International Zoological Angew. Physiology. 29: 299-305.

DAVIS, J.A., FRANK, M.H., WHIPP, B.J. & WASSERMAN, K. (1979). Anaerobic threshold alterations caused by endurance training in middle-aged men. Journal of Applied Physiology. 46: 1039-1046.

DAVIS, J.A., VODAK, P., WILMORE, J.H., VODAK, J. & KURTZ, P.K. (1976). Anaerobic threshold and maximal aerobic power for the three modes of exercise. Journal of Applied Physiology. 41: 544-550.

DEHN, M.M. & BRUCE, R.A. (1972). Longitudinal variations in maximal oxygen intake with age and activity. Journal of Applied Physiology. 33: 805-807.

DEMACKER, P.N. & JANSEN, A.P. (1983). Stability of high-density-lipoprotein cholesterol in frozen sera. Clinical Chemistry. 29: 3: 567-568.

DEMACKER, P.N., SCHADE, R.N., JANSEN, R.T. & LAAR, A. (1982). Intraindividual variation of serum cholesterol, triglycerides and high-density lipoprotein cholesterol in normal humans. Atherosclerosis. 45: 259-66.

DENIS, C., FOUQUET, R., POLY, P., GEYSSANT, A. & LACOUR, J.R. (1982). Effect of 40 weeks of endurance training on the anaerobic threshold. International Journal of Sports Medicine. 3: 208.

DESHAIES, Y. & ALLARD, C. (1982). Serum high density lipoprotein cholesterol in male and female Olympic athletes. Medicine in Science, Sports and Exercise. 14: 207-211.

DESPRÉS, J.P., ALLARD, C. & TREMBLAY, A. (1985). Evidence for a regional component of body fatness in the association with serum lipids in men and women. Metabolism. 34: 967-973.

De VRIES, H.A. (1981). Tranquilizer effect of exercise: A critical review. The Physician and Sports Medicine. 9: 47-55.

De VRIES, H.A. & GRAY, D.E. (1963). After effects of exercise upon resting metabolic rate. Research Quarterly. 34: 314-321.

DICKMAN, S.R. (1988). Pathways to wellness. Human Kinetics. Champaign, Illinois.

DILL, D.B., ROBINSON, S. & ROSS, J.C. (1967). A longitudinal study of 16 champion runners. Journal of Sports Medicine. 7: 4-32.

DIXON, M. & PATTERSON, C.R. (1978). Posture and the composition of plasma. Clinical Chemistry. 24: 5: 824-826.

DONAHUE, R.P., BLOOM, E., ABBOTT, R.D., REED, D.M. & YANO, K. (1987). Central obesity and coronary heart disease in men. Lancet. i: 821-824.

DONOVAN, C.M. & BROOKS, G.A. (1983). Endurance training affects lactate clearance, not lactate production. American Journal of Physiology. 244: E83-E92.

DOWELL, L.J., BADGETT, J.L. & LANDIS, C.W. (1968). A study of the relationship between selected physical attributes and the self-concept. In: Contemporary Psychology of Sport. Kenyon, G. (Ed). Washington, D.C.: International Society of Sport Psychology.

DRINKWATER, B.L. & HORVATH, S.M. (1972). Detraining effects on young women. Medicine and Science in Sports. 4: 91-95.

DUFAUX, D.B., ASSMANN. G. & HOLLMAN, W. (1982). Plasma lipoproteins and physical activity: a review. International Journal of Sportsmedicine. 3: 123-136.

DURNIN, J.V.G.A. & RAHAMAN, M.M. (1967). The assessment of the amount of fat in the human body from measurements of skinfold thickness. British Journal of Medicine. 21: 681-689.

DURNIN, J.V.G.A. & WOLMERSLY, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurement on 481 men and women aged from 16 to 72 years. British Journal of Nutrition. 32: 77-97.

EDWARDS, H.T., THORNDIKE, A. & DILL, D.B. (1935). The energy requirement in strenuous muscular exercise. The New England Journal of Medicine. 213: 532-535.

EHNHOLM, C., HUTTANEN, J.K., PIETINEN, P., LEINO, U., MUTANEN, M., KOSTIAINEN, E., PIKKARAINEN, J., DOUGHERTY, R., IACONO, J. & PUSKA, P. (1982). Effect of diet on serum lipoproteins in a population with a high risk of coronary heart disease. The New England Journal of Medicine. 307:850-855.

EHNHOLM, C., SHAW, W., GRETEN, H. & BROWN, W.V. (1975). Purification from human plasma of a heparin-released lipase with activity against triglyceride and phospholipids. Journal of Biological Chemistry. 250: 6756-6761.

EISENBERG, S. (1984). High density lipoprotein metabolism. Journal of Lipid Research. 25: 1017-1058.

EKBLOM, B., ASTRAND, P.-O., SALTIN, B., STENBERG, J. & WALLSTROM, B. (1968). Effect of training on circulatory response to exercise. Journal of Applied Physiology. 24: 518-528.

ENGER, S.C., HERBJORNSEN, ERIKSSON, J. & FRETLAND, A. (1977). High density lipoproteins (HDL) and physical activity: the influence of physical exercise, age and smoking on HDL-cholesterol and the HDL-total cholesterol ratio. Scandinavian Journal of Clinical Laboratory Investigation. 37: 251-255.

ERKELENS, D.W., ALBERS, J.J., HAZZARD, W.R. (1979). High-density lipoprotein-cholesterol in survivors of myocardial infarction. Journal of the American Medical Association. 242: 2185-2189.

ERNST, N., FISHER, M., BOWEN, P., SCHAEFER, E.J. & LEVY, R.I. (1980). Changes in plasma lipids and lipoproteins after a modified fat diet. Lancet. 2: 111-113.

EUROPEAN ATHEROSCLEROSIS SOCIETY (1987). Strategies for the prevention of coronary heart disease a policy statement of the European Atherosclerosis Society. European Heart Journal. 8: 77-88.

FARIA, I.E. (1984). Applied physiology of cycling. Sports Medicine. 1: 187-204.

FARRELL, P.A. & BARBORIAK, J. (1980). The time course of alterations in plasma lipid and lipoprotein concentrations during eight weeks of endurance training. Atherosclerosis. 37: 231-238.

FARRELL, P.A., GATES, W.K. MAKSUD, M.G. & MORGAN, W.P. (1982). Increases in plasma beta-endorphin/beta-lipotrophin immunoreactivity after treadmill running in humans. Journal of Applied Physiology. 52: 1245-1249.

FARRELL, P.A., MAKSUD, M.G., POLLOCK, M.L., FOSTER, C., ANHOLM, J., HARE, J. & LEON, A.S. (1982). A comparison of plasma cholesterol, triglycerides and high density lipoprotein-cholesterol in speed-skaters, weight-lifters and non-athletes. European Journal of Applied Physiology. 48: 77-82.

FARRELL, P.A., WILMORE, J.H., COYLE, E.F., BILLINGS, J.E. & COSTILL, D.L. (1979). Plasma lactate accumulation and distance running performance. Medicine and Science in Sports. 11: 338-344.

FESTINGER, L. (1957). A theory of cognitive dissonance. Stanford University Press. Stanford, California.

FLINT, M.M., DRINKWATER, B.L. & HORVARTH, S.M. (1974). Effects of training on women's response to submaximal exercise. Medicine and Science in Sports. 6: 89-94.

FOLKINS, C.H. (1976). Effects of physical training on mood. Journal of Clinical Psychology. 32: 385-388.

FOLKINS, C.H., LYNCH, S. & GARDNER, M.M. (1972). Psychological fitness as a function of physical fitness. Archives of Physical Medicine and Rehabilitation. 503-508.

FOLKINS, C.H. & SIME, W.E. (1981). Physical fitness training and mental health. American Psychologist. 36: 373-389.

FOTHERBY, K. (1989). Oral contraceptives and lipids. British Medical Journal. 298: 1049-1050.

FOX, E.L., BARTELLS, R.L., KLINZIG, J. & RAAG, K. (1977). Metabolic responses to interval training programs of high and low power output. Medicine and Science in Sports. 9: 3: 191-196.

FRANKENHAUSER, M. (1975). Experimental approach to the study of catecholamines and emotion. In: Emotions, Their Parameters and Measurement. Raven Press. New York.

FRANKLIN, B., BUSKIRK, E., HODGSON, J., GAHAGAN, H., KOLLIAS, J. & MENDEZ, J. (1979). Effects of physical conditioning on cardiorespiratory function, body composition and serum lipids in relatively normal weight and obese middle-aged women. International Journal of Obesity. 3: 97-109.

FRANKLIN, B.A., HODGSON, J. & BUSKIRK, E.R. (1980). Relationship between percent maximal O_2 uptake and percent maximal heart rate in women. Research Quarterly for Exercise and Sport. 51: 616-624.

FRANKLIN, B.A. & RUBENFIRE, M. (1980). Losing weight through exercise. Journal of American Medical Association. 244: 4: 377-379.

FREDRICKSON, D.S., GOLDSTEIN, J.L. & BROWN, M.S. (1978). The familial hyperlipoproteinemias. In: The Metabolic Basis of Inherited Disease. Stanbury, J.B., Wyngaarden, J.B. & Fredrickson, D.S. (Eds). McGraw-Hill, New York.

FREY, M.A.B., DOERR, B.M., LAUBACH, L.L., MANN, B.L. & GLUECK, C.J. (1982). Exercise does not change high-density lipoprotein cholesterol in women after 10 weeks of training. Metabolism. 31: 1142-1146.

FREYMAN, J.F., McNEIL, D.J., ALANPOVIC, P. & McCONATHY. (1982). Effects of 12 weeks of exercise training on plasma lipids and apolipoproteins in middle-aged men. Medicine and Science in Sports and Exercise. 14: 103.

FRINGER, M.N. & STULL, G.A. (1974). Changes in cardiorespiratory parameters during periods of training and detraining in young adult females. Medicine and Science in Sports. 6: 1: 20-25.

GAESSER, G.A. & RICH, R.G. (1984). Effects of high and low intensity exercise training on aerobic capacity and blood lipids. Medicine and Science in Sports and Exercise. 16: 3: 269-274.

GAEZ (1982). Journal of Lipid Research. 23: 1206-1223.

GARROW, J.S. (1988). Effect of exercise on obesity. Acta Medica Scandinavica. Suppl 711: 67-73.

GARY, V. & GUTHRIE, D. (1972). The effect of jogging on physical fitness and self-concept in hospitalized alcoholics. Quarterly Journal of Studies in Alcoholism. 33: 1073-1078.

GERSOVITZ, M., MADDEN, J.P. & SMICIKLAS-WRIGHT, H. (1978). Validity of the 24-hour dietary recall and seven-day record for group comparisons. Journal of the American Diet Association. 73: 48-55.

GILLIAM, T.B. & BURKE, M.B. (1978). Effects of exercise on serum lipids and lipoproteins in girls, ages 8 to 10 years. Artery. 4: 203-213.

GIRANDOLA, R.N. (1976). Body composition changes in women: Effects of high and low exercise intensity. Archives of Physical and Medical Rehabilitation. 57: 297-300.

GIRANDOLA, R.N. & KATCH, F.I. (1973). Effects of physical conditioning on changes in exercise and recovery oxygen uptake and efficiency during constant-load ergometer exercise. Medicine in Science and Sports. 5: 242-247.

GIRANDOLA, R.N. & KATCH, F.I. (1976). Effects of physical training on ventilatory equivalent and respiratory exchange ratio during weight supported steady-state exercise. European Journal of Applied Physiology. 35: 119-125.

GIRANDOLA, R.N., WISWELL, R.A., MOHLER, J.G., ROMERS, G.T. & BARNES, W.S. (1977). Effect of water immersion on lung volumes: implications for body composition analysis. Journal of Applied Physiology. 43: 376-379.

GLEDHILL, N. & EYNON, R.B. (1972). The intensity of training. In: Training Scientific Basis and Application. A.W. Taylor & M.L. Howell (Eds). Springfield: C. Thomas Company. pp.97-102.

GLOMSET, J.A. & NORUM, K.R. (1973). The metabolic role of lecithin-cholesterol:acyltransferase: perspectives from pathology. Advances in Lipid Research. 11: 1-65.

GLUECK, C.J., TAYLOR, H.L., JACOBS, D., MORRISON, J.A., BEAGLEHOLE, R. & WILLIAMS, O.D. (1980). Plasma high-density lipoprotein cholesterol: association with measurements of body mass. The Lipid Research Clinics Program Prevalence Study. Circulation. 62 (Suppl IV): 62-69.

GOFMAN, J.W., YOUNG, W. & TANDY, R. (1966). Ischemic heart disease, atherosclerosis and longevity. Circulation. 34: 679-685.

GOLDBERG, L. & ELLIOTT, D.L. (1985). The effect of physical activity on lipid and lipoprotein levels. Medical Clinics of North America. 69: 41-55.

GOLDBERG, L., ELLIOT, D.L., SCHULTZ, R.W. & KLOSTER, F.E. (1984). Changes in lipid and lipoprotein levels after weight training. Journal of the American Medical Association. 252: 504-506.

GOLDBOURT, T. & MEDALIE, J.H. (1979). High density lipoprotein cholesterol and incidence of CHD: The Israeli Heart Disease Study. American Journal of Epidemiology. 109: 296-308.

GOLDSTEIN, J.L. & BROWM, M.S. (1977). Atherosclerosis: The low-density lipoprotein receptor hypothesis. Metabolism. 26: 1257.

GOLDSTEIN, J.L. & BROWN, M.S. (1977). Annual Review of Biochemistry. 46: 897.

GOLLNICK, P.D. & HERMANSEN, L. (1973). Biochemical adaptations to exercise: anaerobic metabolism. Exercise Sports Science Reviews. 1: 1-43.

GOLLNICK, P.D. & IANUZZO, C.D. (1972). Hormonal deficiencies and the metabolic adaptations of rats to training. American Journal of Physiology. 223: 278-282.

GOLLNICK, P.D., TIMSON, B.F., MOORE, R.L. & REIDY, M. (1981). Muscular enlargement and number of fibers in skeletal muscles of rat. Journal of Applied Physiology. 50: 936.

GOODE, R., FIRSTBROOK, J. & SHEPHARD, R. (1966). Effects of exercise and cholesterol free diet on human serum lipids. Canadian Journal of Physiology and Pharmacology. 44: 575-580.

GOODNIGHT, S.H., HARRIS, W.S., CONNOR, W.E. & ILLINGWORTH, D.R. (1982). Polyunsaturated fatty acids, hyperlipidaemia and thrombosis. Arteriosclerosis. 2: 87-113.

GORDON, D.J., PROBSTFIELD, J.L., GARRISON, R.J., NEATON, J.D., CASTELLI, W.P., KNOKE, J.D., JACOBS, D.R., BANGDIWALA, S. & TYROLER, H.A. (1989). High-density lipoprotein cholesterol and cardiovascular disease. Circulation. 79: 8-15.

GORDON, D.J. & RIFKIND, B.M. (1989). High density lipoprotein-The clinical implications of recent studies. The New England Journal of Medicine. 321: 19: 1311-1316.

GORDON, T., CASTELLI, W.P., HJORTLAND, M.C., KANNEL, W.B. & DAWBER, T.R. (1977). High-density lipoprotein as a protective factor against coronary heart disease. The Framingham study. American Journal of Medicine. 62: 707-714.

GORDON, T., KANNEL, W.B. & HJÔRTLAND, M.C. (1978). Menopause and coronary heart disease. The Framingham Study. Annals of International Medicine. 89: 157-161.

GREIST, J.H., KLEIN, M.H., EISCHENS, R.R. & FARIS, J. (1978). Running out of depression. Physician and Sportsmedicine. 6: 49-56.

GREIST, J.H., KLEIN, M.H., EISCHENS, R.R., FARIS, J., GURMAN, A.S. & MORGAN, W.P. (1979). Running as a treatment for depression. Comprehensive Psychiatry. 20: 41-54.

GROOT, P.H.E., SCHEEK, L.M. & JANSEN, H. (1983). Liver lipase and high density lipoprotein. Lipoprotein changes after incubation of human serum with rat liver lipase. Biochemica Biophysiologica Acta. 751: 393-400.

GROSSER, J., SCHRECKER, O. & GRETEN, H. (1981). Function of hepatic triglyceride lipase in lipoprotein metabolism. Journal of Lipid Research. 22: 437.

GURR, M.I., BORLAK, N. & GANATRA, S. (1989). Dietary fat and plasma lipids. Nutrition Research Reviews. 2: 63-86.

GWINUP, G. (1975). The effect of exercise alone on the weight of obese women. Archives of International Medicine. 135: 676-679.

GWINUP, G. (1987). Weight loss without dietary restriction: efficacy of different forms of aerobic exercise. American Journal of Sports Medicine. 15: 3: 275-279.

HAGBERG, J.M. (1984). Physiological implications of the lactate threshold. International Journal of Sports Medicine. 5: 106-109.

HAGBERG, J.M. (1987). Effect of training on the decline of VO_2 max with aging. Federation Proceedings. 46: 1830-1837.

HAGBERG, J.M., GRAVES, J.E., LIMACHER, M., WOODS, D.R., LEGGATT, S.H., CONONIE, C., GRUBER, J.J. & POLLOCK, M.L. (1989). Cardiovascular responses of 70- to 79-yr-old men and women to exercise. Journal of Applied Physiology. 66: 6: 2589-2594.

HAGBERG, J.M., HICKSON, R.C., EHSANI, A.A. & HOLLOSZY, J.O. (1980). Faster adjustment to and recovery from submaximal exercise in the trained state. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology. 48: 218-224.

HAIER, R.J., QUAID, K. & MILLS, J.S.C. (1981). Naloxone alters pain perception after jogging. Psychiatry Research. 5: 231-232.

HAMILTON, R.L. (1978). Hepatic secretion and metabolism of high density lipoproteins. In: Disturbances in lipid and lipoprotein metabolism. Dietschy, J.M., Gotto, A.M. & Ontko, J.A. (Eds). American Physiological Society, Bethesda, Maryland.

HARLAN, W.R. (1987). Cardiovascular disease care for women: service utilization, disability and costs from the National Medical care utilization service. In: Coronary Heart Disease in Women. Eaker, E.D., Packard, B., Wenger, N.K., Clarkson, T.B. & Tryoler, H.A. (Eds). Proceedings on an NIH Workshop. New York: Haymarket Doyma. pp 55-61.

HARRIS, W.S., CONNOR, W.E. & McMURRAY, M.P. (1983). The comparative reductions of the plasma lipids and lipoproteins by dietary salmon oil versus vegetable oils. Metabolism. 32: 179-184.

HARTING, G.H., MOORE, C.E., MITCHELL, R. & KAPPUS, C.M. (1984). Relationship of menopausal status and exercise level to HDL cholesterol in women. Experimental Aging Research. 10: 13-18.

HARTLEY, L.H., GRIMBY, G., KILBOM, A., NILSSON, N.J., ASTRAND, I., BJURE, J., EKBLOM, B. & SALTIN, B. (1969). Physical training in sedentary middle-aged and older men. Scandinavian Journal of Clinical Laboratory Investigation. 24: 335-344.

HARTUNG, G.H. (1984). Diet and exercise in the regulation of plasma lipids and lipoproteins in patients at risk of coronary disease. Sports Medicine. 1: 413-418.

HARTUNG, G.H., FOREYT, J.P., MITCHELL, R.E., MITCHELL, J., REEVES, R.S. & GOTTO, A.M. (1983). Effect of alcohol intake on high-density lipoprotein cholesterol levels in runners and active men. Journal of the American Medical Association. 249: 747-750.

HARTUNG, G.H., FOREYT, J.P., MITCHELL, R.E., VLASEK, I. & GOTTO, A.M. (1980). Relationship of diet to high-density lipoprotein cholesterol in middle-aged marathon runners, joggers, and inactive men. The New England Journal of Medicine. 302: 357-361.

HARTUNG, G.H., REEVES, R.S., SIGURDSON, A., TRAWEEK, M.S., FOREYT,J.P. & BLOCKER, W.P. (1983). Effect of a low-fat diet and exercise on plasma lipoproteins and cardiac dysrhythmia in middle-aged men. Circulation. 68: 2: 111-226.

HARTUNG, G.H. & SQUIRES, W.G. (1980). Exercise and HDL-Cholesterol in middle-aged men. The Physician and Sportsmedicine. 8: 1: 74-79.

HARTZ, A.J., RUPLEY, D.C. & RIMM, A.A. (1984). The association of girth measurements with disease in 32,856 women. American Journal of Epidemiology. 119: 71-80.

HARTZ, G.W., WALLACE, W.L. & CLAYTON, T.G. (1982). Effects of aerobic conditioning upon mood in clinically depressed men and women: a preliminary investigation. Perceptual and Motor Skills. 3: 1217-1218.

HASKELL, W.L. (1984). Exercise-induced changes in plasma lipids and lipoproteins. Preventive Medicine. 13: 23-36.

HASKELL, W.L. (1984). The influence of exercise on the concentrations of triglyceride and cholesterol in human plasma. Exercise and Sports Science Reviews. 12: 205-244.

HASKELL, W.L., TAYLOR, H.L., WOOD, P.D., SCHROTT, H. & HEISS, G. (1980). Strenuous physical activity, treadmill exercise test performance and plasma high-density lipoprotein cholesterol: The Lipid Research-Clinic Programme. Prevalence study. Circulation. 62: IV-53-IV-61.

HAVEL, R.J. (1986). Functional activities of hepatic lipoprotein receptors. Annual Review of Physiology. 48: 119-134.

HAVEL, R.J., EDER, M.A. & BRAGDON, J.H. (1955). The distribution and chemical composition of ultracentrifugally separated lipoproteins in human serum. Journal of Clinical Investigation. 34: 1345-53.

HAVEL, R.J., SHORE, V.G., SHORE, B. & BIOU, D.M. (1970). Role of specific glycopeptides of human serum lipoproteins in the activation of lipoprotein lipase. Circulation Research. 27: 595.

HEGSTED, D.M., McGANDY, R.B., MYERS, M.L. & STARE, F.J. (1965). Quantitative effects of dietary fats on serum cholesterol in man. The American Journal of Clinical Nutrition. 17: 281-295.

HEGSTED, D.M. & NICOLOSI, R.J. (1987). Individual variation in serum cholesterol levels. Proceedings of the National Academy of Science. USA. 84: 6259-61.

HEISS, G., TAMIR, I. & DAVIS, C.E. (1980). Lipoprotein-cholesterol distributions in selected North American populations: The Lipid Research Clinic Program Prevalence Study. Circulation. 61: 302-315.

HELLER, R.F., HAYWARD, D. & HOBBS, M.S.T. (1983). Decline in rate of death from ischaemic heart disease in the United Kingdom. British Medical Journal. 286: 260-262.

HENDERSON, J.M. (1974). The effect of physical conditioning on self-concept in college females. Dissertation Abstracts International. 35 (6-B).

HENRIKSSON, J. (1977). Training induced adaptations of skeletal muscle and metabolism during submaximal exercise. Journal of Physiology. 270: 661-666.

HENRIKSSON, J. & REITMAN, J.S. (1977). Time course of changes in human skeletal muscle succinate dehydrogenase and cytochrome oxidase activities and maximal oxygen uptake with physical activity and inactivity. Acta Physiologica Scandinavica. 99: 91.

HERBERT, P.N., BERNIER, D.N., CULLIANE, E., EDELSTEIN, L., KANTOR, M.A. & THOMPSON, P.D. (1984). High density lipoprotein metabolism in runners and sedentary men. Journal of American Medical Association. 252: 1034-1037.

HERMANSEN, L. (1971). Lactate production during exercise. In: Muscle Metabolism During Exercise. Pernow, B. & Saltin, B. (Eds). Plenum Press. New York. pp 401-408.

HERMANSEN, L., GRANDMONTAGNE, M., MOEHLUM, M. & INGNER, I. (1984). Post exercise elevation of resting oxygen uptake: possible mechanisms and physiological significance. Medicine and Science in Sports and Exercise. 17: 119-129.

HICKSON, R.C., BOMZE, H.A. & HOLLOSZY, J.O. (1978). Faster adjustment to and recovery from submaximal exercise in the trained state. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology. 44: 877-881.

HIGUCHI, K., HOSPATTANKAS, A.V., LAW, S.W., MEGLIN, N., CORTRIGHT, J. & BREWER, H.B. (1988). Human apolipoprotein B (apoB) mRNA: identification of two distinct apoB mRNAs, an mRNA with the apoB-100 sequence and an apoB mRNA containing a premature frame transitional stop codon in both liver and intestine. Proceedings of the National Academy of Science. USA. 85: 1772-1776.

HILL, P., WYNDER, E., GARBACZEWSKI, L., GARNES, A., WALKER, R. & HELMAN, P. (1980). Plasma hormones and lipids in men at different risk for coronary heart disease. American Journal Clinical Nutrition. 33: 1010-1018.

HOFFMAN, A.A., NELSON, W. & GOSS, F.A. (1967). Effects of an exercise program on plasma lipids of senior Air Force Officers. American Journal of Cardiology. 20: 516-524.

HOLLANDSWORTH, D.H. (1979). Some thoughts on distance running as training in biofeedback. Journal of Sports Behaviour. 2: 71-82.

HOLLMANN, W. & VENRATH, H. (1962). Experimentelle unterssuchungen zur bedentung aines trainings unterhalb and oberhalb der dauerbeltz stungsgranze. In: Carl Diern Festschrift. Korbs (Eds). W. u. a. Frankfort/Wein.

HOLLOSZY, J. (1967). Biochemical adaptations in muscle. Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. Journal of Biological Chemistry. 242: 2278-2282.

HOLLOSZY, J.O. (1973). Biochemical adaptations to exercise: aerobic metabolism. In: Exercise and Sports Science Reviews. Wilmore, J. (Ed). Academic Press, New York. 45-71.

HOLLOSZY, J.O. & COYLE, E.F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology. 56: 4: 831-838.

HOLLOSZY, J.O., SKINNER, J.S., TORO, G. & CURETON, T.K. (1964) Effects of a six-month programme for endurance exercise on serum lipids of middle-aged men. American Journal of Cardiology. 14: 753-760.

HOPKINS, P.N. & WILLIAMS, R.R. (1981). A survey of 246 suggested coronary risk factors. Atherosclerosis. 40: 1.

HOWALD, H. (1982). Training-induced morphological and functional changes in skeletal muscle. International Journal of Sportsmedicine. 3: 1.

HUBBARD, J.L. (1973). The effect of exercise on lactate metabolism. Journal of Physiology. 231: 1-18.

HUGHES, J.R. (1984). Psychological effects of habitual aerobic exercise: a critical review. Preventive Medicine. 13: 66-78.

HUGHES, J.R., CASAL, D.C. & LEON, A.S. (1986). Psychological effects of exercise: a randomized cross-over trial. Journal of Psychosomatic Research. 30: 3: 355-360.

HULLEY, S.B. & GORDON, S. (1981). Alcohol and high-density lipoprotein cholesterol-causal inference from diverse study designs. Circulation. 64 (Suppl III): 57-63.

HURLEY, B.F., HAGBERG, J.M., ALLEN, W.K., SEALS, D.R., YOUNG, J.C., CUDDIHEE, R.T. & HOLLOSZY, J.O. (1984). Effects of training on blood lactate levels during submaximal exercise. Journal of Applied Physiology: Respiratory Environmental Exercise Physiology. 56: 1260.

HUTTENEN, J.K., LANSIMIES, E., VOUTILAINEN, E., EHNHOLM, C., HIETANEN, E., PENTTILA, I., SIITONEN, O. & RAURAMAA, R. (1979). Effect of moderate physical activity on serum lipids. Circulation. 60: 1220-1229.

ILTIS, P.W., THOMAS, T.R., ADENIRON, S.B., AGUIAR, C.A. & ALBERS, J.J. (1984). Different running programs: plasma lipids, apolipoproteins and lecithin:cholesterol acyltransferase in middle-aged men. Annals of Sports Medicine. 2: 16-21.

ILTIS, P.W., THOMAS, T.R., AGUIAR, C. & ADENIRON, S.B. (1983). Effects of different running programs on plasma lipids, apolipoproteins and lecithin:cholesterol acyltransferase activity. Abstract. Medicine and Science in Sports and Exercise. 15: 100.

INGJER, F. (1979). Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in man. Journal of Physiology. 294: 419.

INNIS, F.J. & EDHOLM, O.G. (1981). Studies of gait and mobility in the elderly. Age and Aging. 10: 147-156.

IVY, J.L., WITHERS, R.T., VAN HANDEL, P.J., ELGER, D.H. & COSTILL, D.L. (1980). Muscle respiratory capacity and fiber type as determinants of the lactate threshold. Journal of Applied Physiology: Respiration, Environmental and Exercise Physiology. 48: 523-527.

JACKSON, R.L., KASHYAP, M.L., BARNHART, R.L., ALLEN, C., HOGG, E. & GLUEK, C.J. (1984). Influence of polyunsaturated and saturated fats on plasma lipids and lipoproteins in man. American Journal of Clinical Nutrition. 39: 589-597.

JACOBS, D.A. & BARRETT-CONNOR, E. (1982). Retest reliability of plasma cholesterol and triglyceride. The Lipid Research Clinics Prevalence Study. American Journal of Epidemiology. 116: 6: 878-85.

JACOBS, I., SJÖDIN, B., KAISER, P. & KARLSSON, J. (1981). Onset of blood lactate accumulation after prolonged exercise. Acta Physiologica Scandinavica. 112: 215-217.

JAKEMAN, P. & DAVIES, B. (1979). The characteristics of a low resistance breathing valve designed for the measurement of aerobic capacity. British Journal of Sports Medicine. 13: 81-83.

JETTÉ, M., SIDNEY, K. & CAMPBELL, J. (1988). Effects of a twelve-week walking programme on maximal and submaximal work output indices in sedentary middle-aged men and women. The Journal of Sports Medicine and Physical Fitness. 28: 59-66.

JOHANNESSEN, S., HOLLY, R.G., LUI, H. & AMSTERDAM, E.A. (1986). High-frequency, moderate intensity training in sedentary middle-aged women. Physician and Sportsmedicine. 14: 5: 99-102.

JOHANSSON, B.G. & MEDHUS, A. (1974). Increase in plasma α-lipoproteins in chronic alcoholics after acute abuse. Acta Medica Scandinavica. 195: 273-277.

JONES, P.R.M. & NORGAN, N.G. (1974). Body density by underwater weighing. Journal of Physiology. 204: 63-66.

KANNEL, W.B. (1987). Metabolic risk factors for coronary heart disease in women: perspective from The Framingham Study. American Heart Journal. 114: 413-419.

KANNEL, W.B. & McGEE, D.L. (1979). Diabetes and cardiovascular risk factors: The Framingham study. Circulation. 59: 8-13.

KANNEL, W.B., CASTELLI, W.P., GORDON, T., McNAMARA, P.M. (1971). Serum cholesterol, lipoproteins, and the risk of coronary heart disease. The Framingham Study. Annals of International Medicine. 74: 1.

KANNEL, W.B., HJÔRTLAND, M.C. & McNAMARA, P.M. (1976). Menopause and risk of cardiovascular disease. The Framingham Study. Annals of International Medicine. 85: 447-452.

KANTOR, M.A., CULLINANE, E.M., HERBERT, P.N. & THOMPSON, P.D. (1984). Acute increase in lipoprotein lipase following prolonged exercise. Metabolism. 33: 454-457.

KARLSSON, J., NORDESJO, L.O., JORFELDT, L. & SALTIN, B. (1972). Muscle lactate, ATP, and CP levels during exercise after physical training in man. Journal of Applied Physiology. 33: 199-203.

KARVONEN, M., KENTALA, K. & MUSTALA, O. (1957). The effects of training heart rate: a longitudinal study. Annual Medical Experimental Biology Federation. 35: 307-315.

KATAN, M.B. (1984). Diet and HDL. In: Clinical and Metabolic Aspects of High-density Lipoproteins. Miller, N.E. & Miller, G.J. (Eds). Elsevier Science Publishers.

KATCH, V., WELTMAN, A., SADY, S. & FREEDSON, P. (1978). Validity of the relative percent concept for equating training intensities. European Journal of Applied Physiology. 39: 219-227.

KEINS, B., GAD, P., LITHELL, H. & VESSBY, B. (1981). Minor dietary effects on HDL in physically active men. European Journal of Clinical Investigation. 11: 265-271.

KEINS, B., JORGENSON, I., LEWIS, S., JENSEN, G., LITHELL, H., VESSBY, B., HOE, S. & SCHNOHR, P. (1980). Increased plasma HDL-cholesterol and Apo A-I in sedentary middle-aged men after physical conditioning. European Journal of Clinical Investigation. 10: 203-209.

KEYS, A., ANDERSON, J.T. & GRANDE, F. (1957). Prediction of serum-cholesterol responses of man to changes in fats in the diet. Lancet. ii: 959-966.

KEYS, A., ANDERSON, J.T. & GRANDE, F. (1965). Serum cholesterol response to changes in diet. IV. Particular saturated fatty acids in the diet. Metabolism. 14: 776-787.

KEYS, A., KARVONEN, M.H., PUNSAR, S., MENOTTI, A., FIDANZA, F. & FARCHI, G. (1984). High density lipoprotein serum cholesterol and 24 year mortality of men in Finland. International Journal of Epidemiology. 13: 428-435.

KILBOM, A. (1971). Physical training in women. Scandinavian Journal of Clinical Laboratory Investigation. 28 (Suppl. 119).

KILBOM, A., HARTLEY, L., SALTIN, B., BJURE, J., GRIMBY, G. & ASTRAND, I. (1969). Physical training in sedentary middle-aged and older men. Scandinavian Journal of Clinical and Laboratory Investigation. 24: 315-322.

KINDERMANN, W., SIMON, G. & KEUL, J. (1979). The significance of the aerobic-anaerobic transition for the determination of work load intensities during endurance training. European Journal of Applied Physiology and Occupational Physiology. 42: 25-34.

KLINE, G.M., POCARI, J.P., HINTERMEISTER, R., FREEDSON, P.S., WARD, A., McCARRON, R.F., ROSS, J. & RIPPE, J.M. (1987). Estimation of $\dot{V}O_2$max from a one-mile track walk, gender, age, and body weight. Medicine and Science in Sports and Exercise. 19: 253-259.

KNEHR, C.A., DILL, D.B. & NEUFELD, W. (1942). Training and its effects on man at rest and at work. American Journal of Physiology. 136: 148-156.

KOHLMEIER, M., STRICKER, G. & SCHLIERF, G. (1985). Influences of 'normal' and 'prudent' diets on biliary and serum lipids in healthy women. American Journal of Clinical Nutrition. 42: 1201-1205.

KOYBASHI, K. & NEELY, J.R. (1979). Control of maximum rates of glycolysis in rat cardiac muscle. Circulation Research. 44: 166-175.

KRAUSS, R.M., LINDGREN, F.T., WOOD, P.D., HASKELL, W.L., ALBERS, J.J. & CHEUNG, M.C. (1977). Differential increases in plasma high density lipoprotein subfractions and apolipoprotein subfractions and apolipoproteins (APO-LP) in runners. Circulation. 56: 4.

KRAUSS, R.M., WOOD, P.D., GIOTAS, C., WATERMAN, D. & LINDGREN, F.T. (1979). Heparin-released plasma lipase activities and lipoprotein levels in distance runners. Circulation. 60: 73.

KROTKIEWSKI, M., BJORNTROP, P., SJOSTROM, L. & SMITH, U. (1983). Impact of obesity on metabolism in men and women. Journal of Clinical Investigation. 72: 1150-1162.

KUUSI, N., NIKKILÄ, E.A, SAARINEN, P., VARJO, P. & LAITINEN, L.A. (1982). Plasma high-density lipoproteins HDL_2, HDL_3 and isoheparin plasma lipases in relation to parameters of physical fitness. Atherosclerosis 41: 209-219.

KUUSI, T., SAARINEN, P. & NIKKILÄ, E.A. (1979). Evidence for the role of hepatic endothelial lipase in the metabolism of plasma high density $lipoprotein_2$ in man. Atherosclerosis. 36: 589-593.

KWITEROVITCH, P.O., BACHORIK, P.S., FRANKLIN, F.A., MARGOLIS, S., GEORGOPOULOS, L., TENG, B. & SNIDERMAN, A.D. (1985). Effect of dietary treatment on the plasma levels of lipids, lipoprotein cholesterol and LDL B protein in children with type II hyperlipoproteinemia. In: Detection and Treatment of Lipid and Lipoprotein Disorders of Childhood. New York. Alan R. Liss (Pub) pp 123-137.

LaFONTAINE, T.P., LONDEREE, B.R. & SPATH, W.K. (1981). The maximal steady-state versus selected running events. Medicine and Science in Sports and Exercise. 13: 190-192.

LAMB, D.R. (1984). Physiology of Exercise: responses and adaptations. Second edition. MacMillan. New York.

LANG, P.J., RICE, D.G. & STERNBACH, R.A. (1972). The psychophysiology of emotion. In: Handbook of Psychophysiology. Greenfield, N.S. & Sternbach, R.A. (Eds). Holt, Reinhart & Winston. New York.

LaPORTE, R.E., BRENES,G., DEARWATER, S., MURPHY, M.A., CAULEY, J.A., DIETRICK, R. & ROBERTSON, R. (1983). HDL-cholesterol across a spectrum of physical activity from quadriplegia to marathon running. Lancet. i: 1212-1213.

LaROSA, J.C., FRY, G., MUESING, R. & ROSING, D.R. (1980). Effect of high-protein, low carbohydrate diet on plasma lipoprotein and body weight. Journal of American Diet Association. 77: 264-270.

LEDWIDGE, B. (1980). Run for your mind: Aerobic exercise as a means of alleviating anxiety and depression. Canadian Journal of Behavioral Science Review. 12: 127.

LEHTONEN, A. & VIIKARI, J. (1978). Serum triglycerides and cholesterol and serum high density lipoprotein cholesterol in highly physically active men. Acta Medica Scandinavica. 204: 111-114.

LEHTONEN, A. & VIIKARI, J. (1978). The effect of vigorous physical activity at work on serum lipids with special reference to serum high-density lipoprotein cholesterol. Acta Physiologica Scandinavica. 104: 117-121.

LEHTONEN, A. & VIIKARI, J. (1980). Serum lipids in soccer and ice-hockey players. Metabolism. 29: 1: 36-39.

LEHTONEN, A., VIIKARI, J. & EHNHOLM, C. (1979). The effect of exercise on high density (HDL) lipoprotein apoproteins. Acta Physiologica Scandinavica. 106: 487-488.

LEON, A.S., CONRAD, J., HUNNINGHAKE, D.B. & SERFASS, R. (1979). Effects of vigorous walking program on body composition, and carbohydrate and lipid metabolism of obese young men. Journal of Clinical Nutrition. 33: 1776-1787.

LEONARDSON, G. & GARGRUILO, R.M. (1978). Self-perception and physical fitness. Perception and Motor Skills. 46: 338.

LEWIS, B. (1976). The Hyperlipidaemias. Clinical and Laboratory Practice. Blackwell. London.

LEWIS, S., HASKELL, W.L., WOOD, P.D., MANOOGIAN, N., BAILEY, J. & PEREIRA, M. (1976). Effect of physical activity on weight reduction in obese middle-aged women. American Journal of Nutrition. 29: 151-156.

LION, L.S. (1978). Psychological effects of jogging: a preliminary study. Perceptual and Motor Skills. 47: 1215-1218.

LIPSON, L.C., BONOW, R.O., SCHAEFER, E.J., BREWER, H.B. & LINGREN, F.T. (1980). Effect of exercise conditioning on plasma high density lipoproteins and other lipoproteins. Atherosclerosis. 37: 529-538.

LITHELL, H., ORLANDER, J. & SHELE, R. (1979). Changes in lipoprotein lipase activity and lipid stress in human skeletal muscle with prolonged heavy exercise. Acta Physiologica Scandinavica. 107: 257-261.

LITHELL, H., SHELE, R., VESSBY, B. & JACOBS, I. (1984). Lipoproteins, lipoprotein lipase and glycogen after prolonged physical activity. Journal of Applied Physical Exercise. 57: 698-702.

LIVINGSTONE, M.B.E., PRENTICE, A.A., STRAIN, J.J., COWARD, W.A., BLACK, A.E., BARKER, M.E., McKENNA, P.G. & WHITEHEAD, R.G. (1990). Accuracy of weighed dietary records in studies of diet and health. British Medical Journal. 300: 708-712.

LONDEREE, B.R. (1986). The use of laboratory test results with long distance runners. Sports Medicine. 3: 201-213.

LONDEREE, B.R. & AMES, S.A. (1975). Maximal steady-state versus state of conditioning. European Journal of Applied Physiology and Occupational Physiology. 34: 269-278.

LOPEZ, S.A., VIAL, R., BALART, L. & ARROYAVE, G. (1974). Effect of exercise and physical fitness on serum lipoproteins. Atherosclerosis. 20: 1-9.

LOUCKS, A.B. & HORVATH, S.M. (1984). Exercise-induced stress responses of amenorrheic and eumenorrheic runners. Journal of Clinical Endocrinological Metabolism. 59: 1109-1120.

MAČEK, M., BELL, D., RUTENFRANZ, J., VAVRA, J., MASOPUST, J., NEIDHART, B. & SCHMIDT, K-H. (1989). A comparison of coronary risk factors in groups of trained and untrained adolescents. European Journal of Applied Physiology. 58: 577-582.

MADER, V.A. & HOLLMAN, W. (1977). Zur bedeutung der stoffwechselleistungs-fahigkeit des eliteruderes im training und wettkampf. Leistungssport. 9: 8-62.

MADER, V.A., LEISEN, H., PHILIPPI, H. & ROST, R. (1976). Beurteilung der sportartspezifischen aud dauerleistungsfahigkeist in labor. Sportarzt und Sportmedizin. 29: 80-88: 109-112.

MAGNUS, G. & BERG, K. (1984). Increase in the ratio of serum levels of apoproteins A-I and A-II during prolonged physical strain and calorie deficiency. European Journal of Applied Physiology. 53: 21-24.

MANN, J.I., LEWIS, B., SHEPHERD, J., WINDER, A.F.; FENSTER, S., ROSE, L. & MORGAN, B. (1988). Blood lipid concentrations and other cardiovascular risk factors: distribution, prevalence, and detection in Britain. British Medical Journal. 296: 1702-1706.

MARITZ, J.S., MORRISON, J.F., PETER, J., STRYDOM, N.B. & WYNDHAM, C.H. (1961). A practical method of estimating an individual's maximal oxygen uptake. Ergonomics. 97-122.

MARKOFF, R.A., RYAN, P. & YOUNG, T. (1982). Endorphins and mood changes in long distance running. Medicine and Science in Sports. 14: 11-15.

MARMOT, M.G. (1984). Lifestyle and national and international trends in coronary heart disease mortality. Postgraduate Medical Journal. 60: 3-8.

MARNIEMI, J., DAHLSTROM, S., KVIST, M., SEPPANEN, A. & KIETANEN, E. (1982). Dependence of serum lipid and lecithin: Cholesterol acyltransferase levels on physical training of young men. European Journal of Applied Physiology. 49: 25-35.

MARNIEMI, J. & HIETANEN, E. (1982). Exercise mediated metabolic changes in lipoprotein metabolism. In: Regulation of Serum Lipids by Physical Exercise. Hietanen, E. (Ed). CRC Press, Boca Raton, Florida.

MARNIEMI, J., PELTONEN, P. VUORI, I. & HIETANEN, E. (1980). Lipoprotein lipase of human postheparin plasma and adipose tissue in relation to physical training. Acta Physiologica Scandinavica. 1110: 131-135.

MARR, J.W. (1971). Individual dietary surveys: purposes and methods. World Review of Nutrition and Diet. 13: 105-164.

MARTIN, R.P., HASKELL, W.L. & WOOD, P.D. (1977). Blood chemistry and lipid profiles of elite distance runners. American New York Academy of Science. 301: 346-360.

MASSICOTTE, D.R., AVON, G. & CORRIVEAU, G. (1979). Comparative effects of aerobic training on men and women. Journal of Sports Medicine and Physical Fitness. 19: 23-32.

MASSIE, J. & SHEPHARD, R.J. (1971). Physiological and psychological effects of training. Medicine and Science in Sports. 3: 110-117.

MATTHEWS, K.A., MEILAHN, E., KULLER, L.H., KELSEY, S.F., CAGGIULA, A.W. & WING, R.R. (1989). Menopause and risk factors for coronary heart disease. The New England Journal of Medicine. 321: 641-6.

MATTSON, F.H. & GRUNDY, S.M. (1985). Comparison of effects of dietary saturated, monosaturated, mono-unsaturated, and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. Journal of Lipid Research. 26: 194-202.

MAUGHAN, R.J. (1982). A simple, rapid method for the determination of glucose, lactate, pyruvate, alanine, 3-hydroxybutyrate and aceto-acetate on a single 20µl blood sample. Clinical Chemica Acta. 122: 231-240.

McCUNNEY, R.J. (1987). Fitness, heart disease and high density lipoproteins: A look at the relationships. The Physician and Sportsmedicine. 15: 2: 67-79.

McGLYNN, G.H., FRANKLIN, B., LAURO, G. & McGLYNN, I.K. (1983). The effect of aerobic conditioning and induced stress on state-trait anxiety, blood pressure and muscle tension. Journal of Sports Medicine. 23: 341-351.

McNAIR, D.M., LORR, M. & DOPPLEMAN, L.F. (1971). Manual for the Profile of Mood States. San Diego; Educational and Industrial Testing Service.

MENDOZA, S.G., OSUNA, A., ZERPA, A. GARTSIDE, P.S. & GLUECK, C.J. (1981). Hypertriglyceridemia and hypoalphalipoproteinemia in azospermic and oligospermic young men: relationships of endogenous testosterone to triglyceride and high density lipoprotein cholesterol metabolism. Metabolism. 30: 481-486.

MICHAEL, E.D. (1957). Stress adaptation through exercise. Research Quarterly. 28: 50-54.

MICHAEL, E., EVERT, J. & JEFFERS, K. (1972). Physiological changes of teenage girls during five months of detraining. Medicine and Science in Sports. 4: 214-218.

MICHAEL, E. & GALLON, A. (1959). Periodic changes in the circulation during athletic training as reflected by the step test. Research Quarterly. 30: 300-311.

MILESIS, C.A. (1974). Effects of metered physical training on serum lipids of adult men. Journal of Sports Medicine. 14: 8.

MILLER, G.J. & MILLER, N.E. (1979). Plasma high-density lipoprotein concentration and development of ischaemic heart disease. Lancet. ii: 16-19.

MILLER, N.E., THELE, D.S., FÖRDE, O.H. & MJÖS, O.D. (1977). The Tromso Heart Study. High-density lipoprotein and coronary heart disease: a prospective study. Lancet. i: 965-968.

MINISTRY OF AGRICULTURE, FOOD & FISHERIES (1987). Food consumption and expenditure. HMS Stationery. London.

MITCHELL, J.H., SPROULE, B.J. & CHAPMAN, C.V. (1958). The physiology of the maximal oxygen intake tests. Journal of Clinical Investigation. 37: 538-547.

MOBILY, K. (1982). Using physical activity and recreation to cope with stress and anxiety: A Review. American Corrective Journal. 36: 77-81.

MOLL, M.E., WILLIAMS, R.S., LESTER, R.M., QUARFORDT, S.H. & WALLACE, A.G. (1979). Cholesterol metabolism in non-obese women. Atherosclerosis. 34: 159-166.

MOORE, C.E., HARTUNG, G.H., MITCHELL, R.E., KAPPUS, C.M. & HINDERLITTER, J. (1983). The relationship of exercise and diet on high-density lipoprotein cholesterol levels in women. Metabolism. 32: 2: 189-196.

MOORE, D.H. (1975). A study of age group track and field records to relate age and running speed. Nature. 253: 264-265.

MOORE, J.H. & WILLIAMS, D.L. (1966). The effect of an atherogenic diet on plasma lipid composition and aortic atherosclerosis in two strains of New Zealand white rabbit. British Journal of Nutrition. 20: 571-580.

MOORE, R.L., REIDY, M. & GOLLNICK, P.D. (1982). Effect of training on β-adrenergic receptor numbers in rat heart. Journal of Applied Physiology. 52: 1133-1137.

MORGAN, T.E., COBB, L.A., SHORT, F.A., ROSS, R. & GUNN, D.R. (1971). Effect of long-term exercise on human muscle mitochondria. In: Muscle Metabolism During Exercise. Pernow, B. & Saltin, B. (Eds). New York. Plenum. pp 87-95.

MORGAN, W.P. (1979). Anxiety reduction following acute physical activity. Psychiatry Annals. 9: 141-147.

MORGAN, W.P. (1981). Psychological benefits of physical activity. In: Exercise in Health and Disease. Nagle, F.J. & Montoyne, H.J. (Eds). Springfield, Illinois. Charles C. Thomas.

MORGAN, W.P. & COSTILL, D.L. (1972). Psychological characteristics of the marathon runner. Journal of Sports Medicine and Physical Fitness. 12: 42-46.

MORGAN, W.P. & HORSTMAN, D.H. (1976). Anxiety reduction following acute physical activity. Medicine in Science and Sport. 8: 62.

MORGANROTH, J., MARON, B.J., HENRY, W.L. & EPSTEIN, S.E. (1975). Comparative left ventricular dimensions in trained athletes. Annual International Medicine. 82: 521-524.

MORRIS, J.N. (1959). Occupation and coronary heart disease. Archives of International Medicine. 104: 903.

MORRIS, J.N. (1975). Uses of epidemiology. Churchill Livingstone. London & New York.

MORRIS, J.N., CHAVE, S.P.W., ADAM, C., SIREY, C. & EPSTEIN, L. (1973). Vigorous exercise in leisure-time and the incidence of coronary heart-disease. Lancet. i: 333-339.

MORRIS, J.N., POLLARD, R., EVERITT, M.G. & CHAVE, S.P.W. (1976). Vigorous exercise in leisure-time and the incidence of coronary heart disease. Lancet. i: 33-339.

MORRISETT, J.D., JACKSON, R.L. & GOTTO, A.M. (1975). Lipoproteins: structure and function. Annual Review of Biochemistry. 44: 183.

NAGAO, N., IMAI, Y., ARIE, J., SAWADA, Y. & KARATSU, K. (1988). Comparison of serum apolipoproteins and lipoproteins in active and inactive males. Journal of Sports Medicine and Physical Fitness. 28: 67-73.

NAITO, H.K. (1988). Reliability of lipid, lipoprotein and apolipoprotein measurements. Clinical Chemistry. 34: B84-B94.

NATELSON, B.H., TAPP, W.N., MUNSIF, A. & BURNS, W. (1988). Fluctuating serum cholesterol: implications for coronary prevention. The Lancet. 404-405.

NELSON, G. (1972). Blood lipids and lipoproteins: quantilation, composition and metabolism. Wiley-Interscience. New York.

NICHOLL, A. & LEWIS, B. (1980). Evaluation of the roles of lipoprotein lipase and hepatic lipase in lipoprotein metabolism: In vivo and in vitro studies in man. European Journal of Clinical Investigation. 10: 487.

NICHOLS, A.V., GONG, E.L. & BLANCHE, P.J. (1981). Interconversion of high density lipoproteins during incubation of human plasma. Biochemical and Biophysiological Research Communications. 100: 391-399.

NIKKILÄ, E.A. (1978). Metabolic regulation of high-density lipoprotein concentrations. European Journal of Clinical Investigation. 8: 111.

NIKKILÄ, E.A., KUUSI, T. & MYLLYNEN, P. (1980). High density lipoprotein and apolipoprotein A-I during physical inactivity. Atherosclerosis. 37: 457-462.

NIKKILÄ, E.A., KUUSI, T. & TÄSKINEN, M.R. (1982). Role of lipoprotein lipase and heparin endothelial lipase in the metabolism of high-density lipoproteins: a novel concept on cholesterol transport in HDL cycle. In: Metabolic Risk Factors in Ischaemic Cardiovascular Disease. Carlson, L.A. & Pernow, B. (Eds). Raven Press. New York. 205-215.

NIKKILÄ, E.A., TÄSKINEN, M.R., REHUNEN, S. & HARKONEN, M. (1978). Lipoprotein lipase activity in adipose tissue and skeletal muscle of runners: relation to serum lipoprotein. Metabolism. 27: 1661-1671.

NOMA, A., YOKASUKA, T. & KITAMURA, K. (1983). Plasma lipids and apolipoproteins as discriminators for the presence and severity of angiographically defined coronary artery disease. Atherosclerosis. 49: 1-7.

NORDOY, A., AAKVAAG, A. & THËLLE, D. (1979). Sex hormones and high density lipoproteins in healthy males. Atherosclerosis. 34: 431-436.

NORUM, R.A., LAKIER, J.B. & GOLDSTEIN, S. (1982). Familial deficiency of apolipoproteins A-I and C-III and precocious coronary-artery disease. The New England Journal of Medicine. 306: 1515-1519.

NYE, E.R., CARLSON, K., KIRSTEIN, P. & ROSNER, S. (1981). Changes in high density lipoprotein subfraction and other lipoproteins induced by exercise. Clinical Chimica Acta. 113: 51.

OFFICE of POPULATION CENSUSES & SURVEYS (OPCS) (1986). Monitor DH2 86/2.

OFFICE of POPULATION CENSUSES & SURVEYS (1986). The General Household Survey 1983. London. HMSO.

OLIVER, M.F. (1987). Diet and coronary heart disease. Health Trends. Department of Health and Social Security. 2: 19: 8-10.

OLIVER, M.F. & BOYD, G.S. (1959). Effect of bilateral ovariectomy on coronary artery disease and serum lipid levels. Lancet. ii: 690-694.

OLSEN, C. (1971). An enzymatic fluorimetric micromethod for the determination of acetoacetate, beta-hydroxybutyrate, pyruvate and lactate. Clinical Chimica Acta. 33: 293-300.

ORCHARD, T.J., BECKER, D.J., BATES, M., KULLER, L.H. & DRASH A.L. (1983). Plasma insulin and lipoprotein concentrations: An atherogenic association? American Journal of Epidemiology. 11: 326-337.

OSBORNE, J.C. & BREWER, H.B. (1977). The plasma lipoproteins (A Review). Advances in Protein Chemistry. 31: 253-337.

OSTER, P., SCHLIERF, G., HEIJCK, C.C, HAHN, S., SZYMANSKI, H. & SCHELLENBERG, B. (1981). Diet and high density lipoproteins. Lipids. 16: 93-97.

OSTLUND, R.E., STATEN, M., KOHRT, W.M., SCHULTZ, J. & MALLEY, M. (1990). The ratio of waist-to-height circumference, plasma insulin level, and glucose tolerance as independent predictors of the HDL, cholesterol level in older adults. The New England Journal of Medicine. 322: 4: 229-234.

OTTO, R.M., FOX, E.L. & STEVENS, C.J. (1978). Metabolic responses of young women to training and maintenance/ detraining. Medicine and Science in Sports. 10: 1: 52.

PACY, P.J., WEBSTER, J. & GARROW, J.S. (1986). Exercise and obesity. Sports Medicine. 3: 89-113.

PAFFENBARGER, R.S., HYDE, R.T., HSIEH, C-C. & WING, A.L. (1988). Physical activity, other lifestyle patterns, cardiovascular disease and longevity. Acta Medica Scandinavica. Suppl. 711: 85-91.

PAFFENBARGER, R.S., LAUGHLIN, M.E. & GIMA, A.S. (1970). Work activity of longshoremen as related to death from coronary heart disease and stroke. The New England Journal of Medicine. 282: 1109-1114.

PAFFENBARGER, R.S., WING, A.L., HYDE, R.T. & ASELI, C.C. (1978). Physical activity as an index of heart attack risk in college alumni. American Journal of Epidemiology. 108: 3: 161-175.

PARRISH, H.M., CARR, C.A. & HALL, D.G. (1967). Time interval from castration in premenopausal women to development of excessive coronary atherosclerosis. American Journal of Obstetrics and Gynaecology. 99: 155-162.

PASSMORE, R. & DURNIN, J.V.G.A. (1955). Human energy expenditure. Physiological Reviews. 35: 801-870.

PATSCH, J.R., GOTTO, A.M., OLIVECRONA, T. & EISENBERG, S. (1978). Formation of high density lipoprotein 2-like particles during lipolysis of very low density lipoproteins in vitro. Proceedings of the National Academy of Science USA. 75: 4519.

PATSCH, W., SCHONFIELD, G., GOTTO, A.M. & PATSCH, J.R. (1980). Characterizations of human high density lipoproteins by zonal centrifugation. Journal of Biological Chemistry. 255: 3178-3185.

PAUL, A.A. & SOUTHGATE, D.A.T. (1985). McCance and Widdowson's: The Composition of Foods. 4th revised, edition of MRC special report No.297. London. Elsevier.

PAULY, J.T., PALMER, J.A., WRIGHT, C.C. & PFEIFFER, G.J. (1982). The effect of a 14 week employee fitness program on selected physiological and psychological parameters. Journal of Occupational Medicine. 24: 6: 457-463.

PEARLIN, L.I., LIEBERMAN, M.A., MENAGHAN, E.G. & MULLAN, J.T. (1981). The stress process. Journal of Health and Social Behaviour. 22: 337-356.

PELTONEN, P., MARNIEMI, J., HIETANEN, E., VUORI, I. & EHNHOLM, C. (1981). Changes in serum lipids, lipoproteins and heparin releasable lipolytic enzymes during moderate physical training in man: A longitudinal study. Metabolism. 30: 518-526.

PERONNÉT, F., FERGUSON, R.J., PERRAULT, H., RICCI, G. & LAJOIE, D. (1981). Echocardiography and the athlete's heart. Physician and Sportsmedicine. 9: 102.

PITTS, F.N. (1969). The biochemistry of anxiety. Scientific American. 220: 69-75.

POCARI, M.S., McCARRON, R., KLINE, G., FREEDSON, P., WARD, A., ROSS, J.A. & RIPPE, J.M. (1987). Is fast walking an adequate aerobic training stimulus for 30- to 69- year-old men and women? The Physician and Sportsmedicine. 15: 2: 119-129.

POCOCK, S.J., SHAPER, A.G., PHILIPS, A.N., WALKER, M. & WHITEHEAD, T.P. (1986). High density lipoprotein is not a major risk factor for IHD in British men. British Medical Journal. 292: 515-519.

POLINER, L.R., DEHMER, G.J., LEWIS, S.E., PARKEY, R.W., BLOMQVIST, G.G. & WILLERSON, J.T. (1980). Left ventricular performance in normal subjects. A comparison of the responses to exercise in the upright and supine position. Circulation. 62: 528-534.

POLLOCK, M.L. (1973). The quantification of endurance training programs. Exercise and Sports Sciences Reviews. 1: 155-188.

POLLOCK, M.L. (1978). How much exercise is enough? Physician and Sportsmedicine. 6: 50-64.

POLLOCK, M.L., BROIDA, J., KENDRICK, Z., MILLER, H., JANEWAY, R. & LINNERUD, A.C. (1972). Effects of training two days per week at different intensities on middle-aged men. Medicine and Science in Sports. 4: 4: 192-197.

POLLOCK, M.L., CURETON, T.K. & GRENINGER, L. (1969). Effects of frequency of training on working capacity, cardiovascular function, and body composition of adult men. Medicine and Science in Sports. 1: 70-74.

POLLOCK, M.L., DIMMICK, J., MILLER, H.S., KENDRICK, Z. & LINNERUD, A.C. (1975). Effects of mode of training on cardiovascular function and body composition of adult men. Medicine and Science in Sports. 7: 2: 139-145.

POLLOCK, M.L., GETTMAN, L.R. & MILESIS, C.A. (1977). Effects of frequency and duration of training on attrition and incidence of injury. Medicine and Science in Sports. 9 (i): 31-36.

POLLOCK, M.L., MILLER, H.S., JANEWAY, R., LINNERUD, A.C., ROBERTSON, B. & VALENTINO, R. (1971). Effects of walking on body composition and cardiovascular function of middle-aged men. Journal of Applied Physiology. 30: 1: 126-130.

POOLING PROJECT RESEARCH GROUP (1978). Relationship of blood pressure, serum cholesterol, smoking habits, relative weight and ECG abnormalities to incidence of major coronary events: Final report of the Pooling Project. Journal of Chronic Disease. 31: 201.

PRENTICE, A.M., DAVIES, H.L., BLACK, A.E., ASHFORD, J., COWARD, W.A., MURGATROYD, P.R., GOLDBERG, G.R., SAWYER, M. & WHITEHEAD, R.G. (1985). Unexpectedly low levels of energy expenditure in healthy women. Lancet. June 22: 1419-1422.

PROSSER, G., CARSON, P., PHILLIPS, R., GELSON, A., BUCH, N., TUCKER, H., NEOPHYTOU, M., LLOYD, M. & SIMPSON, T. (1981). Morale in coronary patients following an exercise programme. Journal of Psychosomatic Research. 25: 587-593.

RAHN, H., FENN, W.O. & OTIS, A.B. (1949). Daily variations in vital capacity, residual air and expiratory reserve including a study of the residual air method. Journal of Applied Physiology. 1: 725-736.

RAJPUT-WILLIAMS, J., KNOTT, T.J., WALLIS, S.C., SWEETNAM, P., YARNELL, J., COX, N., BELL, G.I., MILLER, N.E. & SCOTT, J. (1988). Variation of apolipoprotein -B gene is associated with obesity, high blood cholesterol levels and increased risk of coronary heart disease. The Lancet. 1442-1446.

RANSFORD, C.P. (1982). A role for amines in the anti-depressant effect of exercise: A review. Medicine and Science in Sports. 14: 1-10.

REICHL, D. & MILLER, N.E. (1986). The anatomy and physiology of reverse cholesterol transport. Clinical Science. 70: 221-231.

REINHARD, U., MULLER, P.H. & SCHMULLING, R-M. (1979). Determination of anaerobic threshold by the ventilation equivalent in normal individuals. Respiration. 38: 36-42.

REMES, K., KUOPPASALMI, K. & ALDERCREUTZ, H. (1979). Effect of long-term physical training on plasma testosterone and sex-hormone-binding globulin capacity. Scandinavian Journal of Clinical Laboratory Investigation. 39: 743-749.

RHOADS, G.C., GULBRANDSEN, C.L. & KAGAN, A. (1976). Serum lipoproteins and coronary heart disease in a population study of Hawaii Japanese men. The New England Journal of Medicine. 294: 293-298.

RICHARD, M.J., SERBUS, D.C., BEITZ, D.C. & JACOBSEN, N.L. (1982). Effect of type and amount of dietary fat on the concentration of cholesterol in blood plasma and tissues of rabbits. Nutrition Research. 2: 175-183.

RIPPE, J.M., WARD, A., HASKELL, W.L., FREEDSON, P., FRANKLIN, B.A. & CAMPBELL, K.R. (1986). Walking for fitness. The Physician and Sportsmedicine. 14: 10: 145-159.

ROBERTS, L. (1987). Measuring cholesterol is as tricky as lowering it. Science. 28: 482-483.

ROBINSON, B.F., EPSTEIN, S.F., BEISER, G.D. & BRAUNWALD, E. (1966). Control of heart rate by the autonomic nervous system. Studies in man on the inter-relations between baroreceptor mechanisms and exercise. Circulation Research. 19: 400-411.

ROBINSON, S. (1938). Experimental studies of physical fitness in relation to age. Arbeitsphysiologie. 10: 251-323.

ROBINSON, S., DILL, D.B., ROSS, J.C., ROBINSON, R.D., WAGNER, J.A. & TZANKOFF, S.P. (1973). Training and physiological aging in man. Federation Proceedings. 32: 5: 1628-1634.

ROBINSON, S., DILL, D.B., ROSS, J.C., WAGNER, J.A. TZANKOFF & ROBINSON, R.D. (1975). Longitudinal studies of aging in men. Journal of Applied Physiology. 38: 263-267.

ROBINSON, S. & HARMON, P.M. (1941). The lactic acid mechanism and certain properties of the blood in relation to training. American Journal of Physiology. 132: 757-769.

ROHEIM, P.S. (1986). Atherosclerosis and lipoprotein metabolism: role of reverse cholesterol transport. American Journal of Cardiology. 57: 3C-10C.

ROSENBAUM, S.P., SKINNER, R.K., KNIGHT, I.B. & GARROW, J.S. (1985). A survey of heights and weights of adults of Great Britain, 1980. Annals of Human Biology. 12: 115-127.

ROSENBERG, L., HENNEKENS, C.H. & ROSNER, B. (1981). Early menopause and the risk of myocardial infarction. American Journal of Obstetrics and Gynaecology. 139: 47-51.

ROTKIS, T., BOYDEN, T.W., PAMENTER, R.W., STANFORTH, P. & WILMORE, J. (1981). High density lipoprotein cholesterol and body composition of female runners. Metabolism. 30: 994-995.

ROTTERDAM, E.P., KATAN, M.B. & KNUIMAN, J.T. (1987). Importance of time interval between repeated measurements of total or high-density lipoprotein cholesterol when estimating an individuals baseline concentrations. Clinical Chemistry. 33: 1913-5.

ROWELL, L.B. (1975). Human cardio-vascular adjustments to exercise and thermal stress. Physiological Reviews. 54: 75-159.

RUSKO, H., RAHKILA, P. & KARVINEN, E. (1980). Anaerobic threshold, skeletal muscle enzymes and fiber composition in young female cross-country skiers. Acta Physiologica Scandinavica. 108: 263-268.

RUYS, T., SHAIKH, M., NORDESTGAAD, B.G., STURGESS, I., WATTS, G.F. & LEWIS, B. (1989). Effects of exercise and fat ingestion on high density lipoprotein production by peripheral tissues. Lancet. 1119-1122.

SACKS, F.M., SALAZAR, J., MILLER, L., FOSTER, J.M., SUTHERLAND, M., SAMONDS, K.W., ALBERS, J.J. & KASS, E.H. (1984). Ingestion of egg raises plasma low density lipoproteins in free-living subjects. Lancet. i: 647-649.

SADY, S.P., THOMPSON, P.D., CULLINANE, E.M., KANTOR, M.A. & DOMAGALA, E. (1986). Prolonged exercise augments plasma triglyceride clearance. Journal of the American Medical Association. 256: 2551-2555.

SALTIN, B. (1969). Physiological effects of physical conditioning. Medicine and Science in Sports. 1: 50-56.

SALTIN, B. (1985). Hemodynamic adaptations to exercise. American Journal of Cardiology. 88: 10: 420.

SALTIN, B., BLOMQVIST, B., MITCHELL, J.H., JOHNSON, R.L., WILDENTHAL, K. & CHAPMAN, C.B. (1968). Response to submaximal and maximal exercise after bed rest and training. Circulation. 38 (Suppl 7).

SALTIN, B, ESSÉN, B. & PEDERSEN, P.K. (1976). Intermittent exercise: its physiology and some practical applications. In: Advances in Exercise Physiology. Jokl, E., Anand, R.L. & Stoboy, H. S. (Eds). Karger, Basel.

SALTIN, B. & GOLLNICK, P.D. (1983). Skeletal muscle adaptability: significance for metabolism and performance. In: Handbook of Physiology. Skeletal Muscle. Annals of the Physiological Society.

SALTIN, B. & KARLSSON, J. (1971). Muscle ATP, CP and lactate during exercise after physical conditioning. In: Muscle Metabolism During Exercise. Pernow, B. & Saltin, B. (Eds). New York. Plenum Press.

SALTIN, B. & ROWELL, R.B. (1980). Functional adaptations to physical activity and inactivity. Federation Proceedings. 39: 1506-1513.

SANTIAGO, M.C., ALEXANDER, J.F., STULL,G.A., SERFASS, R.C., HAYDAY, A.M. & LEON, A.S. (1987). Physiological responses of sedentary women to a 20-week conditioning program of walking or jogging. Scandinavian Journal of Sports Science. 9: 2: 33-39.

SAVAGE, M.P., PETRATIS, M.M., THOMPSON, W.H., BERG, K. & SMITH, J.L. (1986). Exercise training effects on serum lipids of pre-pubescent boys and adult men. Medicine and Science in Exercise and Sports. 18: 197-204.

SCANU, A.M. & LANDSBERGER, F.R. (1980). Lipoprotein structure. Annual New York Academy of Science. 348: 1-436.

SCHACHTER, S, & SINGER, J.E. (1962). Cognitive, social and physiological determinants of emotional state. Psychology Review. 69: 379-399.

SCHAEFER, E.J, EISENBERG, S. & LEVY, R.I. (1978). Lipoprotein apolipoprotein metabolism. Journal of Lipid Research. 19: 667.

SCHAEFER, E.J., ORDOVAS, J.M. & LAW, S.W. (1985). Familial apolipoprotein A-I and C-III deficiency, variant II. Journal of Lipid Research. 26: 1089-1101.

SCHETTER, G., NUSSEL, E. & BUCHHOLZ, L. (1978). Epidemiological research in Western Europe. Atherosclerosis Review. 3: 201.

SCHEUER, J. & TIPTON, C.M. (1977). Cardiovascular adaptations to physical training. Annual Review of Physiology. 39: 221-251.

SCHMITZ, G., HOLLOWAY, C. & ASSMANN, G. (1982). Analytical isotachophoresis of human serum lipoproteins. Journal of Chromatography.

SCHNABEL, A. & KINDERMANN, W. (1982). Effects of maximal oxygen uptake and different forms of physical training on serum lipoproteins. European Journal of Applied Physiology. 48: 263-277.

SCHULTZ, P. (1980). Walking for fitness: slow but sure. The Physician and Sports Medicine. 8: 9: 24-27.

SCHWARTZ, G.E., DAVIDSON, R.J. & GOLEMAN, D.J. (1978). Patterning of cognitive and somatic processes in the self-regulation of anxiety: effects of meditation versus exercise. Psychosomatic Medicine. 40: 4: 321-329.

SEALS, D.R., HAGBERG, J.M., HURLEY, B.F., EHSANI, A.A. & HOLLOSZY, J.O. (1984a). Endurance training in older men and women. I. Cardiovascular responses to exercise. Journal of Applied Physiology. 57: 4: 1024-1029.

SEALS, D.R., HURLEY, B.F., SCHULTZ, J. & HAGBERG, J.M. (1984b). Endurance training in older men and women. II. Blood lactate response to submaximal exercise. Journal of Applied Physiology. 57: 4: 1030-1033.

SELYE, H. (1975). Stress without distress. Signet. New York.

SEYLE, H. (1976). Stress and physical activity. McGill Journal of Education. 11: 3-4.

SIRI, W.E. (1956). Gross composition of the body. In: Advances in Biological and Medicinal Physics. Vol.IV. J.H.Laurence & C.A.Tobias (Eds). New York Academic Press. 239-280.

SJöDIN, B. & JACOBS, I. (1981). Onset of blood lactate accumulation and marathon running performance. International Journal of Sports Medicine. 2: 23-26.

SJöDIN, B. & SVEDENHAG, J. (1985). Applied physiology of marathon running. Sports Medicine. 2: 83-99.

SKINNER, J.S. & McLELLAN, T.H. (1980). The transition from aerobic and anaerobic metabolism. Research Quarterly for Exercise and Sport. 51: 234-238.

SMITH, E.L. & GILLIAN, C. (1983). Physical activity prescription for the older adult. The Physician and Sportsmedicine. 11: 8: 91-101.

SMITH, M.P., MENDEZ, J., DRUCKENMILLER, M. & KRIS-ETHERTON, P.M. (1982). Exercise intensity, dietary intake, and high density lipoprotein cholesterol in young female competitive swimmers. American Journal of Clinical Nutrition. 36: 251-255.

SMITH, D.P. & STRANSKY, F.W. (1976). The effect of training and detraining on the body composition and cardiovascular response of young women to exercise. Journal of Sports Medicine. 16: 112-120.

SNYDER, E.E. & SPREITZER, E.A. (1974). Involvement in sport and psychological well-being. International Journal of Sport Psychology. 5: 28-39.

SORBIS, R., PETERSSON, B.G. & NILSSON-EHLE, P. (1981). Effect of weight reduction on plasma lipoproteins and adipose tissue metabolism in obese subjects. European Journal of Clinical Investigation. 11: 491-498.

SOUTHGATE, D.A.T. (1986). Obese deceivers? British Medical Journal. 292: 1692-1693.

SPEILBERGER, C.D. (1970). Manual for the state-trait anxiety inventory (STAI). Palo Alto: Consulting Psychologists Press.

SRINIVASAN, S.R., SUNDARAM, G.S. & WILLIAMSON, G.D. (1985). Serum lipoprotein and endogenous sex hormones in early life: observations in children with different lipoprotein profiles. Metabolism. 34: 861-867.

STAHLER et al., (1977). Medical Laboratory Techniques. 30:29.

STARZEC, J.J., BERGER, D.F. & HESSE, R. (1983). Effects of stress and exercise on plasma corticosterone, plasma cholesterol, and aortic cholesterol levels in rats. Psychosomatic Medicine. 45: 219-226.

STATLAND, B.E., WINKEL, P. & BOKELAND, H. (1973) Factors contributing to intra-individual variation of serum constituents. I. Within-day variation of constituents in healthy subjects. II. Effects of exercise and diet on variation of serum constituents in healthy subjects. Clinical Chemistry. 19: 1374-82.

STERN, M.J. & CLEARY, P. (1982). The National Exercise and Heart Disease Project: Long term psychosocial outcome. Archives of International Medicine. 142: 1093-1097.

STERN, M. & HAFFNER, S.M. (1986). Body fat distribution and hyperinsulinaemia as risk factors in diabetes and cardiovascular disease. Arteriosclerosis. 6: 123-130.

STOKER, D.J., WYNN, V. & ROBERTSON, G. (1966). Effect of posture on the plasma cholesterol level. British Medical Journal. 1:336-338.

STREJA, D.A., BOYKO, E. & RABKIN, S.W. (1980). Changes in plasma high-density lipoprotein cholesterol concentration after weight reduction in grossly obese subjects. British Medical Journal. 281: 770-772.

STREJA, A. & MYMIN, D. (1979). Moderate exercise and light density lipoprotein cholesterol: observations during cardiac rehabilitation programme. Journal of American Medical Association. 242; 2190-2192.

SUTHERLAND, W. & WOODHOUSE, S. (1980). Physical activity and plasma lipoprotein lipid concentrations in men. Atherosclerosis. 37: 285-292.

SWINDELLS, Y.E. (1972). The influence of activity and size of meals on caloric response in women. British Journal of Nutrition. 27: 65-73.

TALL, A.R. & SMALL, D.M. (1980). Body cholesterol removal-Role of plasma HDL. Advances in Lipid Research. 17: 2.

TAN, M.H., WILMSHURST, E.G., GLEASON, R.E. & SOELDNER, J.S. (1973). Effect of posture on serum lipids. The New England Journal of Medicine. 416-419.

TÄSKINEN, M.R., GLUECK, C.J., KASHYAP, M.L., SRIVASTAVA, M.L., HYND, B.A., PERISUTTI, G., ROBINSON, K., KINNUNEN, P.J. & KUUSI, T. (1980). Post-heparin plasma lipoprotein and hepatic lipases. Atherosclerosis. 37: 247-256.

SHAHNAWAZ, H. (1978). Influence of limb length on a stepping exercise. Journal of Applied Physiology: Respiratory, Environmental Exercise Physiology. 44: 3: 346-349.

SHAPER, A.G. (1987). Epidemiology and prevention of ischaemic heart disease. Current Opinion in Cardiology. 2: 571-585.

SHAPIRO, L.M. (1987). Left ventricular hypertrophy in athletes in relation to the type of sport. Sportsmedicine. 4: 4: 239-244.

SHARKEY, B.J. (1970). Intensity and duration of training and the development of cardiorespiratory endurance. Medicine in Science and Sports. 2: 197-202.

SHEPHARD, R.J. (1969). Intensity, duration, and frequency of exercise as determinants of the response to a training regime. International Zoological Angew. Physiology. 26: 272-278.

SHEPHARD, R.J., ALEEN, C., BENADE, A.V.S., DAVIES, C.T.M., DI PRAMPERO, P.E., HEDMAN, R., MERRIMAN, J.E., MYHRE, K. & SIMMONS, R. (1968). The maximal oxygen intake: an international reference standard of cardiorespiratory fitness. Bulletin. WHO. 38: 757-764.

SHEPHARD, R.J. & SIDNEY, K.H. (1975). Effects of physical exercise on plasma growth hormone and cortisol levels in human subjects. Exercise Sports Science Review. 3: 1-29.

SHINOMIYA, M., SASAKI, N., BARNHART, R.L., SHIRA, K. & JACKSON, R.L. (1982). Effect of apolipoproteins on the hepatic lipase-catalyzed hydrolysis of human plasma high density lipoprotein$_2$-triacylglycerols. Biochemica Biophysiologica Acta. 713: 292-299.

SHIRE, T.L., AVALLONE, J.P., BOILEAU, R.A., LOHMAN, T.G. & WIRTH, J.C. (1977). Effect high resistance and low resistance bicycle ergometer training in college women on cardiorespiratory function and body composition. Research Quarterly. 48: 391-400.

SIDNEY, K.H. & SHEPHARD, R.J. (1978). Frequency and intensity of exercise training for elderly subjects. Medicine and Science in Sports. 10: 2: 125-131.

SIEDEL et al. (1981) Journal of Clinical Chemistry and Clinical Biochemistry. 19: 838.

SIMONSON, E. (1971). Physiology of work capacity and fatigue. Charles C. Thomas, Springfield, Illinois.

TÄSKINEN, M.R., KASHYAP, M.L., SRIVASTAVA, L.S., ASHRAF, M., JOHNSON, J.D., PERISUTTI, G., BRADY, D., GLUECK, C.J. & JACKSON, R.L. (1982). In vitro catabolism of human plasma very low density lipoproteins. Effects of VLDL concentration on the interconversion of high density lipoprotein subfractions. Atherosclerosis. 41: 381-394.

TÄSKINEN, M.R. & NIKKILÄ, E.A. (1981). High density lipoprotein subfractions in relation to lipoprotein lipase activity of tissues in man-evidence for reciprocal regulation of HDL_2 and HDL_3 levels by lipoprotein lipase. Clinical Chimica Acta. 112: 325-332.

TAYLOR, H.L., BUSKIRK, E. & HENSCHELL, E. (1955). Maximal oxygen intake as an objective measure of cardio-respiratory performance. Journal of Applied Physiology. 8: 73-80.

TAYLOR, K.G., CARTER, T.J., VALENTE, A.J., WRIGHT, A.D., SMITH, J.H. & MATTHEWS, K.A. (1981). Sex differences in the relationships between obesity, alcohol consumption and cigarette smoking and lipid and apolipoprotein concentrations in a normal population. Atherosclerosis. 38: 11-18.

TESCH, P.A., THORSSON, A. & KAISER, P. (1984). Muscle capillary supply and fiber type characteristics in weight and power lifters. Journal of Applied Physiology. 56: 35-39.

THOMPSON, P.D., JEFFERY, R.W., WING, R.R. & WOOD, P.D. (1979). Unexpected decrease in plasma high density lipoprotein cholesterol with weight loss. American Journal of Clinical Nutrition. 32: 2016-2021.

THOMPSON, P.D., LAZARUS, B., CULLINANE, E., HENDERSON,L.O., MUSLINER, T., ESHLEMAN, R. & HERBERT, P.N. (1983). Exercise, diet or physical characteristics as determinants of HDL-levels in endurance athletes. Atherosclerosis. 46: 333-339.

TRAN, Z.V. & WELTMAN, A. (1985). Differential effects of exercise on serum lipid and lipoprotein levels seen with changes in body weight. A meta-analysis. Journal of American Medical Association. 254: 919-924.

TREMBLAY, A., DESPRÉS, J-P. & BOUCHARD, C. (1985). The effects of exercise-training on energy balance and adipose tissue morphology and metabolism. Sportsmedicine. 2: 223-233.

TRINDER (1969). Annals of Clinical Biochemistry. 6:24.

TSOPANAKIS, C., KOTSARELLIS, D. & TSOPANAKIS, A.D. (1986). Lipoprotein and lipid profiles of elite athletes in Olympic sports. International Journal of Sports Medicine. 7: 316-321.

VACCARO, P. & CLINTON, M. (1981). The effects of aerobic dance on the body composition and maximal oxygen uptake of college women. Journal of Sports Medicine and Physical Fitness. 21: 291-294.

VAN GAAL, L., VANSANT, G., MOERKERKE, F. & DE LEEUW, I. (1987). "Central obesity" and coronary heart disease. Lancet. Sept 5th. 579.

VANHOUTTE, P.M., VERBEUREN, T.J. & WEBB, R.C. (1981). Local modulation of adrenergic neuroeffector interaction in the blood vessel wall. Physiological Review. 61: 151-247.

VODAK, P.A., WOOD, P.D., HASKELL, W.L. & WILLIAMS, P.T. (1980). HDL-cholesterol and other plasma lipid and lipoprotein concentrations in middle-aged male and female tennis players. Metabolism. 29: 745-752.

VON SCHENCK, H. & OLSSON, A. (1990). Serum cholesterol determinations: How reliable can they be? Lipid Review. 4: 2: 9-13.

WAHL, P., WALDEN, C. & KNOPP, R. (1983). Effect of estrogen/progestin potency on lipid/lipoprotein cholesterol. The New England Journal of Medicine. 308: 862-7.

WAHLEFELD, A.W. (1974). Triglyceride determination after enzymatic hydrolysis. In: Methods of enzymatic analysis. BERGMEYER, H.U. (Ed). Verlag Chemie Weinheim & Academic Press Inc. New York & London. pp 1831-1835.

WAHREN, J. (1977). Glucose turnover during exercise in man. New York Academy of Science. 301: 45-54.

WALLACE, R.B., HOOVER, J., BARRETT-CONNOR, E., RIFKIND, B.M., HUNNINGHAKE, D.B., MACKENTHUM, G. & HEISS, G. (1979). Altered plasma lipid and lipoprotein levels associated with oral contraceptive and oestrogen use. Lancet. ii: 112-115.

WALSH, R. & DAVIDSON,G.P. (1980). Desensitization to lactic acid as a possible mechanism mediating the therapeutic effect of physical exercise on anxiety neurosis. Journal of Sports Medicine. 20: 158-160.

WASSERMAN, K., WHIPP, B.J., KOYAL, S.N. & BEAVER, W.L. (1973). Anaerobic threshold and respiratory gas exchange during exercise. Journal of Applied Physiology. 35: 236-243.

WEBB, P., SARIS, W.H.M., SCHOFFELEN, P.F.M., SCHENAU, G.J. & HOOR, F.T. (1988). The work of walking. Medicine and Science in Sports and Exercise. 20: 4: 331-337.

WEINER, J.S. & LOURIE, J.A. (1981). Practical Human Biology. Academic Press, London.

WELTMAN, A., MATTER, S. & STAMFORD, B.A. (1980). Caloric restriction and/or mild exercise effects on serum lipids and body composition. American Journal of Clinical Nutrition. 33: 1002-1009.

WENGER, H.A. & MacNAB, R.B.J. (1975). Endurance training: the effects of intensity, total work, duration and initial fitness. Journal of Sports Medicine and Physical Fitness. 15: 199-211.

WHITE, A.J. (1974). The interrelationships between measures of physical fitness of self-concept of selected Mississippi State University male students. Dissertation Abstracts International. 34(8-A): 4849.

WILLIAMS, G.Z., WIDDOWSON, G.M. & PENTON, J. (1978) Individual character of variation in time-series studies of healthy people. Differences in values for clinical chemical analyses in serum among demographic groups, by age and sex. Clinical Chemistry. 24: 313-20.

WILLIAMS, P.T., WOOD, P.D. & KRAUSS, R.M. (1983). Does weight loss cause the exercise-induced increase in plasma high-density lipoproteins? Atherosclerosis 47, 173-185.

WILLIAMS, P.T., KRAUSS, R.M., WOOD, P.D., LINDGREN, F.T., GIOTAS, C. & VRANIZAN, K.M. (1986). Lipoprotein subfractions of runners and sedentary men. Metabolism. 35: 45-52.

WILLIAMS, P.T., WOOD, P.D., HASKELL, W.L. & VRANIZAN, K. (1982). The effects of running mileage and duration on plasma lipoprotein levels. Journal of the American Medical Association. 247: 2674-2679.

WILMORE, J.H. (1969). The use of actual, predicted and constant residual volume in the assessment of body composition by underwater weighing. Journal of Medicine Science and Sports. 1: 87-90.

WILMORE, J.H., DAVIES, J.A., O'BRIEN, A., VODAK, P.A., WLADER, G.R. & AMSTERDAM, E.A. (1980). Physiological alterations consequent to twenty week conditioning programs of bicycling, tennis and jogging. Medicine and Science in Sports. 12:1-8.

WILMORE, J.H., ROYCE, J., GIRANDOLA, R.N., KATCH, F.I. & KATCH, V.L. (1970). Physiological alterations resulting from a 10-week program of jogging. Medicine and Science in Sports. 1: 7-14.

WILSON, D.E. & LEES, R.S. (1972). Metabolic relationships among the plasma lipoproteins. Journal of Clinical Investigation. 51: 1051-1057.

WILSON, G.T. & DAVIDSON, G.C. (1971). Processes of fear reduction in systematic desensitization: Animal studies. Psychological Bulletin. 76: 1-4.

WINDER, W.W., HAGBERG, J.M., HICKSON, R.C., ESHANI, A.A. & McLANE, J.A. (1978). Time course of sympatho-adrenal adaptation to endurance exercise training in man. Journal of Applied Physiology. 45: 370.

WOLF, R.N. & GRUNDY, S.C. (1980). Effects of caloric restriction on plasma lipids and lipoproteins. Clinical Research. 28: 55A.

WOLMERSLEY, J. & DURNIN, J.V.G.A. (1973). An experimental study on variability of measurements of skinfold thickness on young adults. Human Biology. 45: 281-292.

WOLPE, J. (1958). Psychotherapy by Reciprocal Inhibition. Stanford University Press. Stanford, California.

WOOD, D.T. (1977). The relationship between state anxiety and acute physical activity. American Corrective Therapy Journal. 31: 67-69.

WOOD, P.D. & HASKELL, L.W. (1979). The effect of exercise on plasma high-density lipoproteins. Lipids. 14: 417-427.

WOOD, P.D., HASKELL, W.L., BLAIR, S.N., WILLIAMS, P.T., KRAUSS, R.M., LINDGREN, F.T., ALBERS, J.J., HO, P.H. & FARQUHAR, J.W. (1983). Increased exercise level and plasma lipoprotein concentrations: A one-year, randomized controlled study in sedentary, middle-aged men. Metabolism. 32: 31-39.

WOOD, P.D., HASKELL, W.L., KLEIN, H., LEWIS, S., STERN, M.P. & FARQUHAR, J.W. (1976). The distribution of plasma lipoproteins in middle-aged male runners. Metabolism. 25: 1249-1257.

WOOD, P.D., HASKELL, L.W., STERN, M.P., LEWIS, S. & PERRY, C. (1977). Plasma lipoprotein distributions in male and female runners. Annals of New York Academy of Science. USA. 301: 748-763.

WOOD, P.D., STEFANICK, M.L., DREON, D.M., FREY-HEWITT, B., GARAY, S.C., WILLIAMS, P.T., SUPERKO, H.R., FORTMANN, S.P., ALBERS, J.J., VRANIZAN, K.M., ELLSWORTH, N.M., TERRY, R.B. & HASKELL, W.L. (1988). Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. The New England Journal of Medicine. 319: 18: 1173-1179.

WOOD, P.D., WILLIAMS, P.T. & HASKELL, W.L. (1984). Physical activity and high-density lipoproteins. In: Clinical and Metabolic Aspects of High-density Lipoproteins. Miller, N.E. & Miller, G.J. (Eds). New York, Elsevier Science Publishers. pp 133-165.

WUEST, J.H., DAY, T.J. & EDWARD, J.E. (1953). The degree of coronary atherosclerosis in bilaterally oophorectomized women. Circulation. 7: 801-809.

WYNNE, T.P., FREY, M.A.B., LAUBACH, L.L. & GLUECK, C.J. (1980). Effect of a controlled exercise program on serum lipoprotein levels in women on oral contraceptives. Metabolism. 29 1267-1271.

YOSHIDA, T. (1984). Effect of exercise duration during incremental exercise on the determination of anaerobic threshold and the onset of blood lactate accumulation. European Journal of Applied Physiology and Occupational Physiology. 53: 96-199.

YOSHIDA, T. (1986). Effect of dietary modifications on anaerobic threshold. Sports Medicine. 3: 4-9.

YOUNG, J.R. & ISMAIL, A.H. (1976). Personality differences of adult men before and after a physical fitness program. Research Quarterly of Exercise and Sport. 57: 513-519.

YOUNG, R.J. (1979). The effect of regular exercise on cognitive function and personality. British Journal of Sports Medicine. 13: 110-117.

ZAVARONI, I., CHEN, Y.D.I., MONDON, C.E. & REAVEN, G.M. (1981). Ability of exercise to inhibit carbohydrate-induced hypertriglyceridemia in rats. Metabolism. 30: 476-480.

ZILVERSMIT, D.B. (1979). Atherogenesis: A postprandial phenomenon. Circulation. 60: 473.

ZILVERSMIT, D.B. (1979). A cogent argument for the ability of chylomicron remnants and IDL to produce atherosclerosis in humans and experimental animals. Circulation. 60: 473.

ZUTI, W.B. & GOLDING, L.A. (1973). Effects of diet and exercise on body composition of adult women during weight-reduction. Medicine and Science in Sport. 5: 2: 62-67.

ZWIREN, L., SKINNER, J.S. & BUSKIRK, E.R. (1973). Use of body density and various skinfold equations for estimating small reductions in body fatness. Journal of Sports Medicine and Physical Fitness. 13: 213-218.

Appendix 1.

Example of information supplied to subjects and statement of informed consent.

Title: A comparison of endurance trained and sedentary middle-aged women with respect to blood lipids and habitual diet.

Summary of the project.

Subjects will be asked to complete a questionnaire concerned with current and past exercise habits. For a period of 7 consecutive days, each subject will be asked to weigh all items of food and drink consumed. In addition two, 10 ml samples of blood will be obtained by venopuncture from the subjects after a 12-14 hour fast. Serum will be separated, stored and subsequently assayed for total cholesterol (T-C), high density lipoprotein (HDL) cholesterol and its major subfractions.

Background to the project:

Although there is little evidence that physical activity has an independent effect on plasma total cholesterol, plasma high density lipoprotein cholesterol is invariably higher (i.e. more favourable) in middle-aged individuals who engage in high intensity endurance training than their inactive peers (McCunney 1985). We have recently shown that HDL can be increased by regular, low intensity exercise, brisk walking (Hudson et al. 1988). The purpose of the proposed study therefore is to compare blood lipid profiles in three groups of middle-aged women, namely (i) endurance runners, (ii) walkers/ramblers and (iii) sedentary controls. Diet exerts an important influence on blood lipids and may differ between groups. Consequently, diet will be evaluated by a 7-day weighed food intake.

Investigators:

Dr. A.E.Hardman, Lecturer; Miss. A.Hudson, postgraduate

research student and Miss. A.Hollington, postgraduate student.

Location:
This work will be undertaken in the Sports Science laboratories of the Department of Physical Education and Sports Science, Loughborough University of Technology.

Procedures:
1. Completion of the activity questionnaire. This will take place at the individual's own convenience.
2. The seven-day weighed food intake: this consists of the subject weighing and describing all food and drink consumed over a typical period of seven consecutive days.
3a. Blood sampling: a 10 ml sample will be obtained from an antecubital vein, whilst subjects are in a fasted state and in the supine position.
3b. Risks and discomforts: sampling of venous blood may cause minor bruising.

Experience of the investigators:
Dr.Hardman has over 10 years of experience in conducting this type of investigation. Members of the Sports Science Research Group laboratory have conducted numerous studies involving venous blood sampling, all without major mishap, complications or complaints. On this basis, we therefore have every reason to expect a successful outcome to the proposed study.

Statement of informed consent.

I have read the above outline of the procedures which are involved in this experiment and I understand what will be required of me. I have had the opportunity to ask for further information and for clarification of the demands of each of the procedures. I am aware that I have the right to withdraw from the study at any time with no obligation to give reasons for my decision.

I agree to take part in the study of blood lipids and diet in women.

Signed:_____.

Date:_____.

Witnessed by: _____.

Appendix 2.

The calculation of oxygen uptake and carbon dioxide production.

$$\dot{V}E_{STPD} = \dot{V}E_{ATPS} \times \frac{(BP - SWVP_t)}{760} \times \frac{273}{273 + t}$$

Where BP = barometric pressure in mmHg.

SWVP = saturated water vapour pressure in mmHg at ambient temperature.

t = ambient temperature in degrees Celsius.

ATPS = ambient temperature and pressure, saturated with water vapour.

VO_2 = volume of oxygen inspired - volume of oxygen expired

$$VO_2\ ins = \dot{V}I \times \frac{F_I O_2\ \%}{100} \qquad VO_2\ exp = \dot{V}E \times \frac{F_E O_2\ \%}{100}$$

Similarly: $VCO_2 = \dot{V}CO_2 exp - \dot{V}CO_2 ins$

$$\dot{V}CO_2 = \left[\dot{V}E \times \frac{F_E CO_2\ \%}{100}\right] - \left[\dot{V}I \times \frac{F_I CO_2\ \%}{100}\right]$$

As atmospheric air is relatively stable $F_I O_2$ % is assumed to be 20.93% and $F_I CO_2$ is assumed to be 0.03%. $F_E O_2$ %, $F_E CO_2$ % and VE can be measured directly in the collected sample. Whilst VI is derived from the Haldane transformation.

The haldane transformation uses the concentration of nitrogen in inspired and expired air to derive the volume of air inspired from direct measurements of the volume of air expired.

Therefore, as concentration $= \frac{\text{mass}}{\text{volume}}$

$$\frac{\text{mass nitrogen inspired}}{\dot{V}I} = F_I N_2 \% \quad \text{and}$$

$$\frac{\text{mass nitrogen expired}}{\dot{V}E} = F_E N_2 \%$$

But the mass of nitrogen inspired = the mass of nitrogen expired, therefore $F_I N_2 \% \times \dot{V}I = F_E N_2 \% \times \dot{V}E$

and $\dot{V}I = \frac{F_E N_2 \%}{F_I N_2 \%} \times \dot{V}E$

$\dot{V}I$ can therefore be derived if the expired volume is measured and $F_E N_2 \%$ and $F_I N_2 \%$ are known (found by subtracting the carbon dioxide and oxygen concentrations measured from 100%.

Appendix 3.

Estimation of body fat and lean body mass from skinfold measurements.

Calculation.

y = density x = log sum of 4 skinfolds

Age 17 - 19 yrs Males	:	y = 1.1620 - 0.0630x
Age 16 - 19 yrs Females	:	y = 1.1549 - 0.0678x
Age 20 - 29 yrs Males	:	y = 1.1631 - 0.0623x
Age 20 - 29 yrs Females	:	y = 1.1599 - 0.0717x
Age 30 - 39 yrs Males	:	y = 1.1422 - 0.0544x
Age 30 - 39 yrs Females	:	y = 1.1423 - 0.0632x
Age 40 - 49 yrs Males	:	y = 1.1620 - 0.0700x
Age 40 - 49 yrs Females	:	y = 1.1333 - 0.0612x
Over 50 yrs Males	:	y = 1.1715 - 0.0779x
Over 50 yrs Females	:	y = 1.1339 - 0.0645x

(Wolmersley & Durnin 1973).

% Body fat = ([4.950 / Density] - 4.5) x 100 (Siri 1956)

Absolute body fat = (Body mass x % Body fat) / 100

Lean body mass = Body mass - Absolute body fat

Appendix 4.

Calculation of residual lung volume (RV).

$$RV\ (ml) = V \times \frac{F^{1}N - n}{FN - F^{1}N} \times BTPS - D.S.$$

Volume of gas in bladder (V) = 3000 ml

Alveolar nitrogen (FN) = 80.00 %

Dead space (D.S.) = ml.

Initial nitrogen in bladder (n) = 0.05 %

Appendix 5.

Blood Lactate.

The method used was an adaptation of that described by Olsen (1970). It is dependent upon the release of NADH by the following reaction, which is measured by its native fluorescence:

$$\text{Lactate} + \text{NAD}^+ \xrightarrow{\text{LDH}} \text{Pyruvate} + \text{NADH}$$

DEPROTEINIZATION.

Twenty microlitres (or 25 μl) of blood was deproteinized by adding it to 200 μl (or 250 μl) of perchloric acid. It was then mixed thoroughly, centrifuged and stored at -20^0C prior to analysis.

SOLUTIONS.

Perchloric acid: 2.5% w/v.

Hydrazine buffer (1.1 M, pH 9.0): 1.3 g hydrazine sulphate, 5.0 g hydrazine hydrate and 0.2 g disodium ethylenediaminetetra-acetic acid (EDTA) in 100 ml distilled water.

Reaction mixture: prepared immediately before use: 2 mg NAD^+ and 10 μl LDH per ml of hydrazine buffer.

STANDARDS.

These were made from 1.0 M Sodium L-Lactate stock solution.

PROCEDURE.

1. Samples were removed from the freezer and allowed to thaw at room temperature.
2. Samples were then mixed thoroughly and centrifuged.
3. 25 μl of either supernatant or standard was then pipetted in to a nitric acid washed test-tube, whereupon 250 μl of reaction mixture was added.
4. Tubes were mixed and left to incubate for 30 minutes.
5. 1 ml of diluent was then added to the tubes. The samples were then read against the standards and the blank with a Locarte fluorimeter.
6. The value of the blank was then subtracted from that of the sample and standard readings, and the lactate concentration of each sample was calculated from the sample curve.

Appendix 6.

Biological Variation in Serum Lipids

One non-obese female subject, aged 44 years volunteered to undergo a series of eight venopunctures obtained on consecutive mornings. Each sample was obtained under the same conditions, since previous research has shown that posture (Tan et al. 1973) and nutritive state (Statland et al. 1973) are important determinants of serum lipid levels. The samples were dealt with in an identical manner, each being centrifuged for 15 minutes at 6,000 rpm at -4^0 C. The plasma was then removed, four aliquots were immediately stored at -70^0 C for subsequent analysis, whilst 2mls were reserved and within four days of collection underwent precipitation of non-HDL using manganese chloride and sodium heparin. The HDL-containing supernatant from this procedure was removed and also stored at -70^0 C. The assay techniques are detailed elsewhere.

RESULTS

	Hcrt	Hb	TC	HDL-C	HDL_2-C	Tgs
$\bar{x}$	34	11.2	4.15	1.45	0.76	0.98
sd	0.8	0.2	0.51	0.07	0.10	0.12
5-95% C.L.	32 - 36	10.8 - 11.6	3.15 - 5.15	1.31 - 1.59	0.56 - 0.96	0.75- 1.22
C V(%)	2.3	1.8	12.29	4.83	13.16	12.12

The coefficients of variation although large, are comparable to those reported by Natelson and coworkers (1988), Hegsted and Nicolosi (1987), Roberts (1987) and

Cooper and coworkers (1988). All of whom suggest that despite relatively low coefficient of assay variation (<2%), intra individual differences are large (independent of the sampling period: every six hours, daily (Rotterdam et al. 1987), weekly (Williams et al. 1978) or monthly (Costongs et al. 1985). Together this evidence suggests that care should be taken particularly in assessing the relation of this biological variation to training induced changes.

Appendix 7.

Precipitation and Separation of Venous Blood Sample.

Blood samples were obtained from an antecubital vein and dispensed in to tubes containing EDTA. After being allowed to stand at room temperature for 30 minutes the samples were centrifuged for 15 minutes at 6,000 rpm at 4^0 C. The serum was then filtered off and divided in to a number of aliquots, one volume (2.5 ml) was stored under refrigeration for further precipitation, whilst the remainder were stored at -70^0C for use in subsequent assays.

Selective Precipitation Technique.

The samples of sera stored under refrigeration were within 4 days taken through a further precipitation procedure.

SOLUTIONS.

Sodium heparin: Sigma grade I, sodium salt, from porcine intestinal mucosa.

The working solution was made up daily, and the weight of sodium heparin used was dependent upon the number of USP units per mg.

Therefore:

181 USP units- 31.5 mg

178 USP units- 32.0 mg

176 USP units- 32.4 mg

This weight was dissolved in 5 ml of 9mg/L saline.

Manganese chloride: 4.95g $MnCl_2 .4H_2O$ was dissolved in 25 ml of distilled water.

Ethylenediaminetetra-acetic acid (EDTA): 14.88 g of EDTA,

disodium salt, was dissolved in 100 ml of distilled water.

Dextran sulphate: 47.9 gm dissolved in 5 ml normal saline.

PROCEDURE.

1. Each sample was divided in to two 1 ml aliquots of sera.
2. 40 μl of sodium heparin solution was then added to each sample.
3. These were immediately vortexed for 10 seconds.
4. 100 μl of manganese chloride solution was then added and samples again vortexed for 10 seconds.
5. The samples were left to incubate in an ice bath placed in the refrigerator for 30 minutes.
6. After incubation, the samples were centrifuged at 4^0 C for 25 minutes at 3,000 rpm.
7. The supernatant was then removed and stored at -70^0C until required.

This procedure was applied in the studies reported in Chapter 5 (part 2 with middle-aged women) and Chapter 7.

Appendix 8.

Total Cholesterol Assay Technique.

TEST PRINCIPLES.

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{chol.esterase}} \text{Cholesterol} + RCOOH$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{chol.oxidase}} \Delta^4\text{-Cholesterone} + H_2O_2$$

$$2H_2O_2 + \text{4-Aminophenazone} + \text{phenol} \xrightarrow{\text{POD}}$$

$$\text{4-(}\beta\text{-benzoquinone-mono-imino)-phenazone} + 4H_2O$$

SOLUTIONS.

STANDARDS.

1.29 $mmol.l^{-1}$, 2.59 $mmol.l^{-1}$, 3.88 $mmol.l^{-1}$, 5.17 $mmol.l^{-1}$ and 7.76 $mmol.l^{-1}$.

PROCEDURE.

1. Samples of whole sera (or plasma) were removed from the freezer and allowed to thaw at room temperature until required.
2. 10 μl of sera (or plasma) was transferred into nitric acid washed tubes (in duplicate).
3. To each sample was added 1 ml of reaction mixture and mixed immediately.
4. The samples were left to incubate for 30 minutes at room temperature and then mixed again.
5. These were then read against the standards on Hg 546 nm.

Appendix 9.

Triacylglycerol Assay.

TEST PRINCIPLE.

$$\text{Triacylglycerols} + 3H_2O \xrightarrow{\text{lipase}} \text{glycerol} + 3RCOOH$$

$$\text{Glycerol} + ATP \xrightarrow{GK} \text{Glycerol-3-phosphate} + ADP$$

$$\text{Glycerol-3-phosphate} + O_2 \xrightarrow{GPO} \text{Dihydroxyacetone phosphate} + H_2O_2$$

$$H_2O_2 + \text{4-Aminophenazone} + \text{4-Chlorophenol} \xrightarrow{\text{peroxidase}} \text{4-(β-benzoquinone-mono-imino)-phenazone} + 2H_2O + HCl$$

SOLUTIONS.

Buffer: 0.15 $mmol.l^{-1}$, pH 7.6; magnesium sulphate: 17.5 $mmol.l^{-1}$; EDTA, disodium salt: 10 $mmol.l^{-1}$; 4-chlorophenol:3.5 $mmol.l^{-1}$; sodium cholate: 0.15%; potassium hexacyano-ferrate (ii): 6 $\mu mol.l^{-1}$; hydroxypolyethoxy-n-alkanes: 0.12%

Reagent strips: ATP$^{)}$ 0.5 $mmol.l^{-1}$; 4-aminophenazone: 0.35 $mmol.l^{-1}$; lipase $^{)}$ 3 $U.ml^{-1}$; glycerol phosphate oxidase $^{)}$ 2.5 $U.ml^{-1}$; glycerol kinase $^{)}$ 0.2 $U.ml^{-1}$; periodoxase $^{)}$ 0.15 $U.ml^{-1}$.

PROCEDURE.

1. One reagent strip was immersed in each bottle of buffer solution, stirred for approximately 10 seconds and left for 5 minutes.
2. The buffer was again stirred with the reagent strip for 10 seconds and then the strip was discarded.
3. The samples of whole serum were removed from the freezer and allowed to thaw at room temperature prior to use.
4. 10 μl of each sample was pipetted in to nitric acid washed tubes, to which was added 1 ml of the prepared reaction mixture.

5. The samples were mixed and left to incubate at 20-25^{0}C for 10 minutes.
6. The samples were then read against a reagent blank within 60 minutes on Hg 546 nm.

The concentration of triacylglycerol was calculated using the following:

$$11.9 \times A_{sample} = mmol.l^{1}$$

Appendix 10.

Total high density lipoprotein assay for use with sera samples.

STANDARDS.

0.323 $mmol.l^{-1}$, 0.645$mmol.l^{-1}$, 0.968 $mmol.l^{-1}$, 1.29 $mmol.l^{-1}$ and 2.59 $mmol.l^{-1}$.

PROCEDURE.

1. The precipitated samples were removed from the freezer and left at room temperature for at least one hour prior to use.
2. 10 µl of EDTA was added to individual tubes.
3. 250 µl of the thawed sample was dispensed in the tubes containing EDTA, mixed well and left to stand for 5 minutes.
4. The procedure for total cholesterol was then followed, using 20 µl samples in duplicate.

This technique was applied in the study reported in Chapter 5 (part 2) and Chapter 7.

Appendix 11.

High density lipoprotein subfraction assay for use with sera samples.

STANDARDS.

0.323 $mmol.l^{-1}$, 0.645 $mmol.l^{-1}$ and 0.968 $mmol.l^{-1}$.

PROCEDURE.

1. The precipitated samples were removed from the freezer and allowed to thaw at room temperature for at least one hour prior to use.
2. 500 µl of sample was pipetted in to a clean tube, to which was added 50 µl of dextran sulphate.
3. The samples were immediately mixed for 5 seconds and then left for 15 minutes at room temperature.
4. After this period the samples were centrifuged at 4^0 C for 30 minutes at 4,000 rpm.
5. 20 µl of EDTA was added to nitric acid washed tubes.
6. 500 µl of the supernatant was removed and added to the EDTA, and mixed.
7. After 5 minutes standing time, 20 µl of this supernatant was transferred in duplicate to tubes and the procedure for total cholesterol was followed.

Appendix 12.

Total high density lipoprotein assay for use with plasma samples.

Standards:

0.323 $mmol.l^{-1}$, 0.645 $mmol.l^{-1}$, 0.968 $mmol.l^{-1}$, 1.29 $mmol.l^{-1}$ and 2.59 $mmol.l^{-1}$.

Procedure:

1. Samples of whole plasma were removed from the freezer and left at room temperature for a minimum of one hor prior to use.
2. Each sample was thoroughly mixed.
3. 100 μl of the thawed sample was then dispensed into plastic tubes.
4. To each sample was added 10 μl of the magnesium chloride and phosphotungstate precipitant (5:1 ratio).
5. Each tube was then capped, mixed for 10 seconds and then spun at 12,000 revs per minute for 2 minutes.
6. Duplicate 10 μl volumes of the resultant supernatant were then withdrawn and dispensed into nitric acid washed tubes.
7. The procedure for the analysis of total cholesterol was then followed.

This analysis technique was applied in the studies reported in Chapter 5 (part 1 with young adults) and Chapter 6.

Appendix 13.

Instructions for the use of the food diary.

It is important that you weigh and record everything that you eat and drink during the period of the survey, following the instructions below.

Please (a) start a separate page for each day,
(b) start a separate line for each food item.

Column 1.
Record meal, along with the time and place of eating.

Column 2.
Describe each item as accurately as possible, stating where relevant:
(a) the type and brand (clean food wrappers may be included)
(b) whether the food is fresh, dried, canned, etc.
(c) if the food is cooked, give cooking method: fried, etc.

Column 3.
Record the weight of each item, after cooking:
1. Place the scales on a level surface
2. Place plate or container on top of scales.
3. Press ON button. The scale should read 0g.
4. Add the first item of food and record the weight.
5. Press ON to re-zero the scales.
6. Add second food item.
7. Continue until all items are weighed.

Wherever possible, record weights in grammes. If this is not possible (e.g. if eating out), estimate weights in a convenient measure (e.g. teaspoonful, heaped dessertspoon, etc).

Column 4.
Record the weight of any leftovers, such as food remaining on the plate, weight of the container in which food has been weighed, apple cores, etc.

Columns 5 and 6.
Please leave blank.

If a food consists of several ingredients, please list each on a separate line i.e. rather than 'one cheese sandwich', record weight of bread, margarine, cheese, etc.

Please remember to record all drinks, as well as food, giving weights where possible, or volumes. Record separately weights of added milk and sugar.

Items which are used several times each day (e.g. butter, milk, etc) maybe kept in a container which need only be

weighed at the beginning and end of each day, and not each time the item is used.

1. Fill the container with the food item.
2. At the beginning of the day weigh the container with the food, describe the food in column 2 and record the weight in column 3.
3. Do not record the food on subsequent occasions on which it is used from the container.
4. At the end of the day, record the total weight of the container and food remaining in the container in the 'leftovers' column.

Please ensure that you are the only person to use the food from the container.

An example is given overleaf.

FOOD DIARY

Name: A. Walker
Number:

Date: 4/4/90
Day: Monday

1. Meal time	2. Description	. Weight onsumed (g)	4. Waste (g)
8.00 am	Cornflakes (Kelloggs)	28	
	Milk (Fat free. S'bury)	48	
	Bread (white, toasted)	76	
	Marmalade (orange)	12	
	Coffee (instant powder)	2	
	Milk (whole pasteurised)	10	
1.00 Pub	Cheese (Cheddar)	55	
	Bread (crusty white cob)	76	
	Butter	4	
	Chutney (Branston?)	2 tsp	
	Kronenburg lager	1 pt	
3.30 Snack	Coffee (instant)	2	
	Coffee mate	6	
	Mars bar	35	
	Apple (Coxes)	76	8
6.30 Dinner	Beefburger (Asda, grilled)	102	
	Potatoes, old, roast	322	74
	Peas (Birdseye, frozen, boiled)	50	
	Tomato ketchup (Heinz)	14	
	Yoghurt (Ski low fat)	162	10
	Flora margarine (in container)	215	194
	Sugar (white) (in container)	106	98

Appendix 14.

Psychological questionnaires.

a. State-trait anxiety questionnaire.

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate response to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

1= Almost never 3 = Often
2= Sometimes 4 = Almost always

	1	2	3	4
1. I feel pleasant.....				
2. I feel nervous and restless.....				
3. I feel satisfied with myself.....				
4. I wish I could be as happy as others seem to be.....				
5. I feel like a failure.....				
6. I feel rested.....				
7. I am 'calm, cool and collected'....				
8. I feel that difficulties are piling up so that I cannot overcome them..				
9. I worry too much over something that really doesn't matter.....				
10. I am happy.....				
11. I have disturbing thoughts.....				
12. I lack self-confidence.....				
13. I feel secure.....				
14. I make decisions easily.....				
15. I feel inadequate.....				
16. I am content.....				
17. Some unimportant thought runs through my mind and bothers me.....				
18. I take disappoinments so keenly that I can't put them out of my mind.....				
19. I am a steady person.....				
20. I get in a state of tension or turmoil as I think over my recent concerns and interests.....				

b. Self-esteem questionnaire.

How strongly do you agree or disagree with these statements?

1 = Not at all 3 = Moderately
2 = Somewhat 4 = Very much

	1	2	3	4
1. I feel that I'm a person of worth, at least on an equal with others.				
2. I feel that I have a number of good qualities.				
3. All in all, I am inclined to feel that I'm a failure.				
4. I am able to do things as well as most other people.				
5. I feel I do not have much to be proud of.				
6. I take a positive attitude towards myself.				
7. On the whole, I am satisfied with myself.				
8. I certainly feel useless at times.				
9. I wish I could have more respect for myself.				
10. At times I think that I am no good at all.				

c. General health questionnaire (12 item).

Directions: the following questions are about you health. All have been used with other groups. We would like to know if you have had any medical complaints, and how your has been in general over the past few weeks.
Please answer all the questions below.
Tick the box under the answer which most nearly applies to you.
Remember that we want to know about present and recent complaints, not those which you have had in the past.
Compare yourself with how you have usually felt in the past few years.

1. Been able to concentrate on whatever you're doing?	Better than usual	Same as usual	Less than usual	Much less usual
2. Lost much sleep over worry?	Not at all	As usual	Slight more	Much more
3. Had difficulty in staying asleep once you are off?	Not at all	As usual	Slight more	Much more
4. Felt that you're playing a useful part in things?	More than usual	Same as usual	Less useful	Much less useful
5. Felt capable of making decisions about things?	More than usual	Same as usual	Less than usual	Much less able
6. Felt constantly under strain?	Not at all	As usual	Slight more	Much more
7. Felt you couldn't overcome your difficulties?	Not at all	As usual	Slight more	Much more
8. Been able to enjoy your day-to-day activities?	More than usual	Same as usual	Less than usual	Much less usual

9. Been getting edgy and bad tempered?	Not at all	As usual	Slight more	Much more
10. Been getting scared or panicky for no good reason?	Not at all	As usual	Slight more	Much more
11. Been able to face up to your problems?	More than usual	Same as usual	Less able	Much less able
12. Found everything getting on top of you?	Not at all	As usual	More than usual	Much more

d. General health questionnaire (28 item)

Please read this carefully.

We should like to know if you have had any medical complaints and how your health has been in general, *over the past few weeks*. Please answer **ALL** the questions on the following pages simply by underlining the answer which you think most nearly applies to you. Remember that we want to know about present and recent complaints, not those that you had in the past.

It is important that you try to answer **ALL** the questions.

Thank you for your co-operation.

Have you recently

A1 Been feeling perfectly well and in good health?	Better than usual	Same as usual	Worse than usual	Much worse
A2 Been feeling in need of a good tonic?	Not at all	Same as usual	More than usual	Much more
A3 Been feeling run down and out of sorts?	Not at all	Same as usual	More than usual	Much more
A4 Felt that you are ill?	Not at all	Same as usual	More than usual	Much more
A5 Been getting any pains in your head?	Not at all	Same as usual	More than usual	Much more
A6 Been getting a feeling of tightness or pressure in your head?	Not at all	Same as usual	More than usual	Much more
A7 Been having hot or cold spells?	Not at all	Same as usual	More than usual	Much more
B1 Lost much sleep over worry?	Not at all	Same as usual	More than usual	Much more
B2 Had difficulty in staying asleep once you are off?	Not at all	Same as usual	More than usual	Much more

B3 Felt constantly under strain?	Not at all	Same as usual	More than usual	Much more
B4 Been getting edgy and bad-tempered?	Not at all	Same as usual	More than usual	Much more
B5 Been getting scared or panicky for no good reason?	Not at all	Same as usual	More than usual	Much more
B6 Found everything getting on top of you?	Not at all	Same as usual	More than usual	Much more
B7 Been feeling nervous and strung-up all the time?	Not at all	Same as usual	More than usual	Much more
C1 Been managing to keep yourself busy and occupied?	More than usual	Same as usual	Less than usual	Much less
C2 Been taking longer over the things you do?	Faster than usual	Same as usual	Longer than usual	Much longer
C3 Felt on the whole you were doing things well?	Better than usual	About the same	Less well	Much less well
C4 Been satisfied with the way you've carried out your task?	More sat'd	About the same	Less sat'd	Much less sat'd
C5 Felt that you are playing a useful part in things?	More than usual	Same as usual	Less useful	Much less useful
C6 Felt capable of making decisions about things?	More than usual	Same as usual	Less than usual	Much less capable
C7 Been able to enjoy your normal day-to-day activities?	More than usual	Same as usual	Less than usual	Much less
D1 Been thinking of yourself as a worthless person?	Not at all	Same as usual	More than usual	Much more
D2 Felt that life is entirely hopeless?	Not at all	Same as usual	More than usual	Much more

D3 Felt that life isn't worth living?	Not at all	Same as usual	More than usual	Much more
D4 Thought of the possibility that you might make away with yourself	Defin-itely not	I dont think so	crossed my mind	Defin-itely has
D5 Found at times you couldn't do anything because your nerves were too bad?	Not at all	Same as usual	More than usual	Much more
D6 Found yourself wishing you were dead and away from it all?	Not at all	Same as usual	More than usual	Much more
D7 Found that the idea of taking your own life kept coming into your mind?	Defin-itely not	I dont think so	crossed my mind	Defin-itel has

d. Profile of mood state.

Directions: below is a list of words that describe feelings people have. Please read each one carefully. Then circle a number under the answer to the right which best describes HOW YOU HAVE BEEN FEELING DURING THE PAST WEEK INCLUDING TODAY.

The numbers refer to these phrases:

0 = Not at all
1 = A little
2 = Moderately
3 = Quite a bit
4 = Extremely

	Not at all	A little	Moderately	Quite	Extremely
1. Friendly	0	1	2	3	4
2. Tense	0	1	2	3	4
3. Angry	0	1	2	3	4
4. Worn out	0	1	2	3	4
5. Unhappy	0	1	2	3	4
6. Clear-headed	0	1	2	3	4
7. Lively	0	1	2	3	4
8. Confused	0	1	2	3	4
9. Sorry for things done	0	1	2	3	4
10. Shaky	0	1	2	3	4
11. Listless	0	1	2	3	4
12. Peeved	0	1	2	3	4
13. Considerate	0	1	2	3	4
14. Sad	0	1	2	3	4
15. Active	0	1	2	3	4
16. On edge	0	1	2	3	4
17. Grouchy	0	1	2	3	4
18. Blue	0	1	2	3	4
19. Energetic	0	1	2	3	4
20. Panicky	0	1	2	3	4
21. Hopeless	0	1	2	3	4
22. Relaxed	0	1	2	3	4
23. Unworthy	0	1	2	3	4
24. Spiteful	0	1	2	3	4
25. Sympathetic	0	1	2	3	4
26. Uneasy	0	1	2	3	4
27. Restless	0	1	2	3	4
28. Unable to concentrate	0	1	2	3	4
29. Fatigued	0	1	2	3	4
30. Helpful	0	1	2	3	4
31. Annoyed	0	1	2	3	4
32. Discouraged	0	1	2	3	4
33. Resentful	0	1	2	3	4
34. Nervous	0	1	2	3	4
35. Lonely	0	1	2	3	4
36. Miserable	0	1	2	3	4

37. Muddled	0	1	2	3	4
38. Cheerful	0	1	2	3	4
39. Bitter	0	1	2	3	4
40. Exhausted	0	1	2	3	4
41. Anxious	0	1	2	3	4
42. Ready to fight	0	1	2	3	4
43. Good natured	0	1	2	3	4
44. Gloomy	0	1	2	3	4
45. Desperate	0	1	2	3	4
46. Sluggish	0	1	2	3	4
47. Rebellious	0	1	2	3	4
48. Helpless	0	1	2	3	4
49. Weary	0	1	2	3	4
50. Bewildered	0	1	2	3	4
51. Alert	0	1	2	3	4
52. Deceived	0	1	2	3	4
53. Furious	0	1	2	3	4
54. Efficient	0	1	2	3	4
55. Trusting	0	1	2	3	4
56. Full of pep	0	1	2	3	4
57. Bad-tempered	0	1	2	3	4
58. Worthless	0	1	2	3	4
59. Forgetful	0	1	2	3	4
60. Carefree	0	1	2	3	4
61. Terrified	0	1	2	3	4
62. Guilty	0	1	2	3	4
63. Vigorous	0	1	2	3	4
64. Uncertain about things	0	1	2	3	4
65. Bushed	0	1	2	3	4

PLEASE MAKE SURE THAT YOU HAVE ANSWERED EVER ITEM.

Appendix 15.
Physical activity questionnaire.

The following series of questions are intended to help us assess your habitual daily exercise practices, both at present and during the past ten years; please include as much detail as possible and if there is insufficient space, please use the reverse side of the page.

Accuracy in your answers is essential as we intend to inveatigate the consequencies of prolonged habitual exercise of various intensities on plasma cholesterol concentrations. This questionnaire will help us to group individuals based on their habitual activity pattern and then to compare the derived groups for blood lipids. The assessments which we make are not judgemetal. All results will, of course, remain strictly confidential. If any of the questions pose a problem, or indeed, if you yourself have any questions for us, please do not hesitate to ask.

Thank you for your co-operation.

Surname:_____ Forename:_____

Date:_____

Date of birth:_____ Age:_____

Telephone No. Day:_____

Evening:_____

Recreation/Leisure Activities.

1. Please study the following list of activities. Tick that/those which you have pursued continuously (for at least 3 months) on a regular basis (i.e. once or more every two weeks) during the last ten years and indicate how often you participated.
E.g. Hockey ✓ 1-2 week during winter for last 8 years.

Activity	Tick	How often?
1. Golf	____	_____
2. Bowls	____	_____
3. Rambling	____	_____
4. Jogging	____	_____
5. Keep fit	____	_____
6. Aerobics	____	_____
7. Social-dance	____	_____
8. Tennis	____	_____
9. Squash	____	_____
10. Badminton	____	_____
11. Hockey	____	_____
12. Table tennis	____	_____
13. Swimming (Leisure)	____	_____
14. Swimming (Training)	____	_____
15. Athletics (Track)	____	_____
16. Athletics (Field)	____	_____
17. Road running	____	_____
18. Cross country	____	_____
19. Orienteering	____	_____
20. Weight-training	____	_____
21. Rowing	____	_____
22. Cycling	____	_____
23. Sailing	____	_____

If you have pursued any additional recreational/leisure activities continuously on a regular basis that are not in the above list, please name them below.

_____

_____

(Please continue to Q.3 if you are not currently participating regularly in any recreational/leisure activity).

2. Please identify those recreational/leisure activities which you currently participate in regularly (at least once every two weeks) by naming the activity and writing down the frequency (number of times per fortnight) of your participation.
E.g. Swimming twice a fortnight for past year.

_____

_____

3. Please underline what you consider to be your main recreational/leisure activity.

a. Running b. Walking c. Neither

RUNNING

4. If you are a runner please answer the following questions. If you are not a runner, please go on to Q.5.

a. For how long have you been running regularly/ seriously? (If you are affiliated to a club, please also state which and for how long you have been a member).

_____

b. How often do you run? No. times per week _____
Please include an outline of your typical weekly programme.
E.g. Mon a.m. 3 miles warm up jog, 8 x 200m with 40s recovery, 3 miles cool down jog.
Tues a.m. 3 miles easy (8 min pace).

_____
_____
_____
_____
_____

c. What is your present average weekly milage?_____

d. Does your training involve interval sessions?_____
If yes, how many per week?_____

e. If you compete, please tick as appropriate.
Cross country_____
Road races_____ What distance?_____
Track_____ What event?_____

f. Has your training been interrupted through injury/ies? If yes, please state when the injury occurred and for how long (weeks) your training was interrupted.

_____
_____

g. Did you complete any alternative forms of training to maintain your fitness whilst injured? (E.g. swimming, cycling). If yes, please name the type of training and how often you trained?

_____

_____

_____

h. Do you presently supplement your running with additional forms of training? (E.g. weights, aerobics). If yes, please name the alternative training and the frequency of that training.

_____

_____

WALKING.

5. If your activity is rambling, walking or hill walking, please answer the following questions. If not please continue to Q.6.

a. How often do you go out walking? (No. of times per month)_____

b. Are a member of a walking/rambling club?_____
Which?_____

c. Do you go out walking in a group?_____
If yes, do you tend to adopt the pace of the slowest member of the group, or do you maintain your own pace?

_____

d. Approximately how long does it take you to walk one mile?_____

e. How many miles do you cover during a typical walk and how long does this take? (approximately)_____

_____

f. Do you combine your walks with other activities? E.g. Dog walking._____

OCCUPATION

6. Do you maintain a house?_____
Do you have an additional job to maintaining a house?

If yes, please complete the questions below. If no please continue to Q.8.

a. Please describe your job (title, hours per day/week)

_____

_____

b. Does your job entail much physical work?
None/little_____
Moderate_____
Much_____

c. At work are you mainly
Seated_____
Stood_____
Walking_____

d. Does your job entail much stair climbing?_____
If yes about how often per day do you climb stairs?
_____

e. How do you usually travel to work? (Bus, car, etc)
_____

f. If you usually walk, run or cycle, what is the distance and how long does it take you to complete it?
_____

7. Do you have any children?
If yes, what are their ages?_____
To what extent (if any) do they help with household chores?_____
_____

8. On local journeys, such as going to the shops or to friends, how do you travel?_____
_____

9. Do you participate regularly in any hobbies or pastimes, other than the activities you have previously mentioned?_____
_____

10. Is there any additional information which you think might be useful to us?_____
_____
_____

Thank you very much for completing this questionnaire, your assistance is very much appreciated.