Brisk walking and serum lipid and lipoprotein variables in previously sedentary women – effect of 12 weeks of regular brisk walking followed by 12 weeks of detraining

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The purpose of this study was to examine the effectiveness of brisk walking as a means of improving endurance fitness and influencing serum lipid and lipoprotein variables in previously sedentary women. Walkers ($n = 10$, mean(s.e.m.) age 47.3(2.0) years) followed a programme of brisk walking (mean(s.e.m.) speed $1.76(0.03) \text{ m s}^{-1}$) for 12 weeks, after which the training stimulus was withdrawn. Controls ($n = 10$, mean(s.e.m.) age 41.6(1.2) years) maintained their habitual sedentary lifestyle throughout. Endurance fitness was determined using laboratory measures of responses to treadmill walking. Serum lipid and lipoprotein variables were determined in venous blood (12-h fasted). Body fatness was assessed by anthropometry and dietary practice using the 7-day weighed food intake technique. Measurements were repeated after 12 and 24 weeks. Brisk walking resulted in a decrease in heart rate and blood lactate concentration during exercise, while detraining was accompanied by a reversal of these changes. Changes in body mass and the ratio of circumferences at the waist and hip did not differ between groups but the sum of four skinfolds decreased with brisk walking and increased with detraining. High density lipoprotein (HDL) and HDL₂ cholesterol increased with walking and decreased with detraining but no between group changes (analysis of variance, $P < 0.05$) were found in other lipid and lipoprotein variables. These findings suggest that regular brisk walking can improve endurance fitness and increase HDL cholesterol concentration in sedentary women.

Keywords: Walking, women, HDL cholesterol, endurance fitness

Coronary heart disease (CHD) is an important cause of morbidity and mortality in women as well as in men and yet knowledge about means of modifying cardiovascular risk factors in women is surprisingly limited. Arguably, the most important lipid risk factor in women is the blood concentration of high density lipoprotein (HDL) cholesterol¹. Several

epidemiological studies have found it to be a strong, negative, independent predictor of CHD in women, an increase of 0.26 mmol l^{-1} being associated with a 42–50% decrease in risk^{2,3}. One positive influence on HDL cholesterol may be physical activity as endurance trained women are reported to have concentrations which are nearly 30% higher than in comparable sedentary women⁴. Despite the consistency of cross-sectional studies there is conflicting evidence about whether or not increased levels of physical activity result in an elevated HDL concentration in women^{5–8} and, if this seems to be the case, about what type and amount of exercise is effective.

In a previous study we reported that regular brisk walking resulted in a marked increase in plasma HDL cholesterol⁹. The purpose of the present study was to examine more rigorously the hypothesis that brisk walking – performed in a flexible, self-governed programme – is a sufficient exercise stimulus to influence serum lipid and lipoprotein variables in formerly sedentary women. The approach adopted was to monitor these variables during a 12-week exercise programme and a subsequent 12-week period during which the training stimulus was withdrawn.

Subjects and methods

Twenty women volunteered to take part in this study, which had the approval of the University's Ethical Advisory Committee. Each subject completed a physical activity questionnaire (maximum score 200) concerned with their exercise habits over the past 10 years. Responses showed that no individual had engaged in regular exercise during the 2 years before the study (mean(s.e.m.) scores = walkers 42 (3), controls 46(4); analysis of variance, $P < 0.05$, not significantly different). Subjects were allocated into groups (walkers and controls) on a convenience basis – walkers followed a progressive training programme of brisk walking for the first 12 weeks of the study, and returned to their previous sedentary lifestyle for the second 12 weeks. Controls were asked to maintain their habitual lifestyle throughout the whole period of the study. Walkers and controls were of mean(s.e.m.) age 47.3(2.0) years and 41.6(1.2) years,

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respectively, with mean(s.e.m.) body mass indices of $24.8(1.4) \text{ kg m}^{-2}$ and $23.4(1.1) \text{ kg m}^{-2}$. All measurements were made at base line and repeated after 12 and 24 weeks.

Exercise tests

At the start of the study only, each subject completed a 1.6-km walk around an athletics track at a pace she judged to be 'brisk'. Two laboratory exercise tests were conducted at each observation point: (1) a 1.6-km walk on a motorized treadmill at the pace selected by each individual outdoors; and (2) an incremental, uphill treadmill walking test. The latter involved walking at a standard speed (1.34 m s^{-1}) for four consecutive, 4-min periods. At the end of each 4 min the gradient was increased, these being selected to elicit 50, 60, 70 and 80% of each individual's predicted maximal oxygen uptake – as determined from steady state measurements of oxygen uptake and heart rate during a preliminary test¹⁰. During both treadmill tests expired air samples were collected using standard Douglas bag techniques and analysed to determine oxygen uptake and carbon dioxide production. Heart rate was measured from the electrocardiogram (modified Lead I). Capillary blood samples ($20 \mu\text{l}$ in duplicate) were obtained before each test, at the end of the 1.6-km walk and at the end of each stage of the incremental test. These were immediately deproteinized and stored at -20°C before analysis for lactate¹¹. Data from the incremental test were used to interpolate the oxygen uptake ($\dot{V}\text{O}_2$) at a reference blood lactate concentration of 2 mmol l^{-1} , a sensitive index of endurance fitness which could be measured during submaximal exercise without extrapolation.

Blood biochemistry

Subjects attended the laboratory in the morning, after a 12-h fast and a 10-ml blood sample was obtained by venipuncture without stasis, after the subject had been seated for 10 min. Blood was separated and HDL cholesterol and the cholesterol in the HDL₃ subfraction were determined in fresh serum (stored at 5°C for ≤ 3 days) after selective precipitation procedures using heparin–manganese and dextran sulphate^{12,13}. HDL₂ values were determined by subtraction of HDL₃ from total HDL. Serum was stored at -70°C before analysis for total cholesterol by a cholesterol oxidase method (Boehringer Mannheim, Mannheim, Germany), triacylglycerol by hydrolysis (Boehringer Mannheim) and apolipoproteins A-1 and B (Apo A-1, Apo B) by immunoprecipitin analysis (Atlantic Antibodies, Winnersh, UK). Imprecision was evaluated using standard sera (Precinorm, Boehringer Mannheim) and day-to-day coefficients of variation were: $< 3.2\%$ and $< 3.6\%$ for total and HDL cholesterol respectively; 4.5% for triglycerides; and $< 2.4\%$ and $< 1.8\%$ for Apo A-1 and Apo B, respectively.

Anthropometry

Height and body mass were determined and the amount and distribution of body fat was evaluated by

measuring skinfold thicknesses at four standard sites, namely biceps, triceps, subscapular and suprailiac, as well as the ratio of circumferences at the waist and hip¹⁴. All skinfold measurements were made by the same experienced observer at each stage of the study, using previously calibrated calipers (Holtain).

Weighed food intake

Average daily intakes of energy, fat and alcohol were assessed, together with the ratio of polyunsaturated to saturated fat, by the 7-day weighed food intake method with subsequent analysis using a computerized version (Salford Microdiet, Department of Mathematics and Computer Science, University of Salford, Salford, UK) of food composition tables¹⁵.

Exercise programme

After completion of the base line tests the walkers undertook a progressive, unsupervised 12-week programme of brisk walking. A minimum exercise prescription was provided which increased from 180 min in each of the first 2 weeks to 315 min week⁻¹ during weeks 9–12. After initial advice given on an individual basis, subjects trained at their own brisk pace, the only constraints imposed being: (1) no training walk was to be shorter than 20 min; and (2) the total training time had to be achieved by completing at least three sessions each week. A training diary describing the duration and frequency of the sessions was submitted by each walker every 2 weeks.

Statistical analysis

The response over time was compared between walkers and controls using two way analysis of variance for repeated measures, adopting a 5% level of confidence. Walkers were older than controls and therefore age was used as a fixed covariate. Multivariate criteria were employed (Statistica Mac, Statsoft, Tulsa, Oklahoma, USA; Release 3) to test the significance of differences between cell means when the response over time differed between groups.

Results

Mean(s.e.m.) self-selected brisk walking speed was $1.76(0.03) \text{ m s}^{-1}$ and $1.77(0.04) \text{ m s}^{-1}$ for walkers and controls, respectively. When the outdoor walking test was replicated on the treadmill, the same brisk walking speed elicited mean(s.e.m.) oxygen uptake values of $18.3(0.8) \text{ ml kg}^{-1} \text{ min}^{-1}$ and $18.6(0.6) \text{ ml kg}^{-1} \text{ min}^{-1}$ and mean(s.e.m.) heart rates of $130(6) \text{ beat min}^{-1}$ and $128(5) \text{ beat min}^{-1}$, for walkers and controls respectively. The mean(s.e.m.) amount of brisk walking accomplished increased progressively from 187(6) min during each of the first 2 weeks to 292(7) min per week for the last 6 weeks.

Changes in the blood lactate and heart rate responses to exercise differed significantly over time between walkers and controls. Brisk walking resulted in decreases in the heart rate and blood lactate response to uphill treadmill walking (Figures 1 and 2)

Figure 1. Changes in heart rate during uphill treadmill walking for walkers and controls. Values are mean(s.e.m.); *significantly different from baseline, $P < 0.05$

Figure 2. Changes in blood lactate concentration during uphill treadmill walking for walkers and controls. Values are mean(s.e.m.); *significantly different from baseline, $P < 0.05$; + significantly different from 12 weeks, $P < 0.05$

and an increase in the oxygen uptake at a reference blood lactate concentration, our measure of endurance fitness (Figure 3). When brisk walking training was discontinued these adaptations were reversed, the metabolic effect of detraining, as assessed by blood lactate concentration, being more marked than the effect on heart rate.

There were no between group differences in the changes in body mass or the ratio of circumferences at the waist and hip but changes in the sum of four skinfolds differed between walkers and controls, decreasing with brisk walking and increasing with detraining (Table 1). Values for serum lipoprotein variables are shown in Table 2. Total cholesterol (TC) concentration decreased over time but this response did not differ between groups. Both HDL and HDL₂ cholesterol changed in a different way in walkers and controls – for both variables, an increase was apparent with brisk walking and a decrease with subsequent detraining. Controlling for accompanying changes in fatness, by including the sum of

Figure 3. Changes in the oxygen uptake at a reference blood lactate concentration of 2 mmol l^{-1} for walkers and controls. Values are mean(s.e.m.); *significantly different from baseline, $P < 0.05$; + significantly different from 12 weeks, $P < 0.05$

skinfolts as a changing covariate, removed the between group difference in the changes in HDL₂ cholesterol but not in HDL cholesterol. Despite the changes in HDL and HDL₂ cholesterol, there was no influence of brisk walking on serum triacylglycerol concentration or apolipoproteins A-1 or B.

There were no between group differences in total energy intake over time, nor were there differences in the proportion of energy from dietary fat or the ratio of polyunsaturated to saturated fat in the diet (Table 3).

Discussion

The main finding of this study is that, in this group of formerly sedentary women, serum concentrations of HDL and HDL₂ cholesterol increased with brisk

walking and decreased when the training stimulus was removed. Moreover, these changes and the associated improvements in endurance fitness resulted from a flexible, home-based programme of moderate intensity exercise. An estimate of the distance walked can be made from the training records of the walkers; assuming that brisk walking pace approximated that determined during the track walk (1.76 m s^{-1} or about 15 min 38 s per mile), the distance covered was about 19 km in the first week, rising to about 30 km per week over each of the last 6 weeks. No injuries were reported, probably because of the nature of the exercise. Mean(s.e.m.) heart rates during brisk walking ($130(6) \text{ beat min}^{-1}$) represented some 72(3)% of predicted maximal heart rates, values which would be expected to elicit a training effect¹⁶.

The effectiveness of the training programme was confirmed by the marked changes in the heart rate and blood lactate response to standardized exercise.

Table 1. Body mass, sum of four skinfolts and the ratio of circumferences at the waist and hip for walkers and controls

	Walkers (n = 10)			Controls (n = 10)		
	Base line	12 weeks	24 weeks	Base line	12 weeks	24 weeks
Body mass (kg)	65.73(2.52)	64.68(2.67)	66.10(2.74)	64.43(2.78)	64.42(2.74)	65.59(2.76)
Σ four skinfolts (mm)	76.5(7.6)	71.4(7.6)*	73.4(7.4)**	64.4(8.5)	64.3(8.4)	65.7(8.4)
Waist: hip ratio	0.81(0.03)	0.79(0.03)	0.83(0.04)	0.78(0.01)	0.77(0.01)	0.79(0.01)

Values are mean(s.e.m.); *significantly different from base line; †significantly different from 3 months; ‡response over time significantly different from controls, $P < 0.05$

Table 2. Serum lipid and lipoprotein variables for walkers and controls

	Walkers (n = 10)			Controls (n = 10)		
	Base line	12 weeks	24 weeks	Base line	12 weeks	24 weeks
Total cholesterol (mmol l^{-1})	5.32(0.25)	4.87(0.28)	4.77(0.28)	4.94(0.34)	4.85(0.29)	4.74(0.29)
HDL cholesterol (mmol l^{-1})	1.21(0.06)	1.27(0.07)*	1.19(0.06)**	1.22(0.06)	1.18(0.05)	1.19(0.06)
HDL ₂ cholesterol (mmol l^{-1})	0.42(0.02)	0.50(0.02)*	0.48(0.03)**	0.46(0.03)	0.44(0.04)	0.46(0.04)
Triacylglycerol (mmol l^{-1})	0.97(0.13)	0.97(0.16)	0.95(0.17)	0.77(0.08)	0.77(0.06)	0.89(0.12)
Apo A-1 (g l^{-1})	1.48(0.06)	1.44(0.09)	1.47(0.11)	1.41(0.04)	1.45(0.07)	1.40(0.04)
Apo B (g l^{-1})	0.75(0.05)	0.73(0.05)	0.79(0.06)	0.64(0.05)	0.63(0.04)	0.67(0.05)

Values are mean(s.e.m.); *significantly different from base line; †significantly different from 3 months; ‡response over time significantly different from controls, $P < 0.05$

Table 3. Average daily intake of energy, percentages of energy from fat and carbohydrate (CHO), ratio of intake of polyunsaturated to saturated fat (P: S) and alcohol for walkers and controls

	Walkers (n = 10)			Controls (n = 10)		
	Base line	12 weeks	24 weeks	Base line	12 weeks	24 weeks
Energy (MJ)	7.89(0.68)	7.27(0.61)	7.16(0.57)	7.28(0.41)	7.49(0.32)	7.38(0.33)
Energy from fat (%)	36.2(1.8)	33.5(1.1)	34.9(1.0)	35.9(1.8)	35.1(1.8)	36.6(1.8)
Energy from CHO (%)	45.2(1.6)	44.8(2.0)	45.0(2.0)	46.7(2.0)	46.3(2.0)	45.5(1.9)
Energy from alcohol (%)	2.6(0.8)	4.0(1.2)	3.7(0.8)	0.30(0.03)	0.37(0.05)	0.29(0.04)
P: S ratio	0.31(0.03)	0.33(0.03)	0.31(0.03)	0.30(0.03)	0.37(0.05)	0.29(0.04)

Values are mean(s.e.m.)

Thus our findings confirm, using laboratory measures of endurance fitness, earlier reports that, given appropriate initial counselling and instruction, formerly sedentary women will adhere to a flexible, home-based, moderate intensity exercise programme¹⁷. It is noteworthy that the total time invested in brisk walking (maximum of under 5 h per week), although at first glance a lot, was modest in relation to that needed to travel to, and take part in, an organized exercise class on the often recommended thrice-weekly basis.

Oxygen uptake at a reference blood lactate concentration proved the most sensitive index of the effects of varying physical activity levels, increasing by 30% with training and decreasing by 11% when physical levels dropped. This is consistent with rapid changes in the activities of enzymes which are markers for the oxidative capacity of skeletal muscle as a training stimulus is applied and then withdrawn¹⁸. Brisk walking did provoke a decrease in heart rate but this was not entirely reversed with detraining, possibly because of some residual increase in maximal oxygen uptake; training-induced increases in maximal oxygen uptake can persist for up to 12 weeks after training ceases¹⁸ and a given bout of exercise may therefore have represented a somewhat lower relative exercise intensity than at base line, even after detraining. Another reason for the difficulty of identifying a detraining effect using heart rate is that baseline values are inevitably somewhat elevated due to an arousal effect. These observations should be borne in mind when using heart rate as an index of a training effect in the absence of a control group.

At least one observational study suggests that women who are physically active, but not endurance trained, possess more favourable blood lipid profiles than sedentary women¹⁹. Information specifically about walking is more limited but women who walk regularly have been reported to have a markedly lower likelihood of having a TC/HDL-C ratio greater than 5.0 (Reference 20). Furthermore, older women (70 years of age) who walk for at least 30 min daily are reported to show lower plasma triacylglycerol concentrations than women who do less walking²¹.

Despite these cross-sectional observations, the effectiveness of exercise programmes based on walking in modifying serum lipids and lipoproteins in formerly sedentary women is unclear. Two studies involving walking/slow jogging have found no changes in middle-aged women^{8,22} and two explanations have been advanced: (1) that hypoestrogenic women may be insensitive to the stimulating effects of exercise on HDL cholesterol⁸; and (2) that such moderate exercise does not reach the 'threshold' level of exercise needed to be effective in this regard^{8,23}. However, the first explanation is not supported by the increase in HDL cholesterol reported for 68-year-old women following an 8-week walking programme⁵ and the second is not supported by the findings of a 6-month randomized trial involving three different intensities of walking, i.e. 4.8 km h⁻¹, 6.4 km h⁻¹ and 8.0 km h⁻¹ (Reference 24). HDL cholesterol increased by a similar amount, relative to controls, in each group, despite the greater improvements in fitness with faster walking speeds. Our findings agree with

those of Duncan and co-workers²⁴, for women walking briskly at 1.76 ms⁻¹ (6.34 km h⁻¹) and confirm our earlier findings⁹.

Most of the increase in HDL with walking appeared to be attributable to changes in the HDL₂ subfraction, that which differs most markedly between groups in case-control studies²⁵, suggesting that brisk walking is associated with improvements in reverse cholesterol transport in these women. The lack of a complementary decrease in triacylglycerol is surprising because the concentration of HDL cholesterol is dependent on the metabolism of triacylglycerol-rich lipoproteins^{26,27}. However, Duncan and co-workers also found an increase in HDL cholesterol without a corresponding change in triacylglycerol²⁴ and the reason, exacerbated when only a small number of subjects are studied, may be that fasting concentrations may be an insensitive method for examining the metabolic capacity for triacylglycerol²⁸.

There is known to be an inverse relationship between body fatness and HDL cholesterol concentrations in women²⁹. In the present study, significant changes in subcutaneous fat occurred with changing levels of brisk walking. Controlling for these – by analysis of covariance – suggested that they might be related to the changes in HDL metabolism which were observed. However, local adaptations in skeletal muscle³⁰ probably also play a part as HDL cholesterol has been reported to increase with increased physical activity in women even in the absence of changes in adiposity^{9,25}.

The magnitude of the changes in HDL cholesterol in these women are modest but the finding still has important implications for public health policy. Data from the Helsinki Heart Study indicate that every 1% increase may decrease coronary heart disease risk by as much as 3%³¹. Furthermore, in women, HDL cholesterol concentration has been shown to be a stronger risk factor than LDL cholesterol¹. In this country, about eight of ten women are physically inactive³² and consequently the potential to decrease morbidity and mortality by persuading women to take more exercise may be considerable. Brisk walking carries a low risk of injury and is more likely to be adopted as a long-term exercise habit than exercise of a higher intensity³³. Furthermore, exercise may act synergistically with other intervention strategies. Wood and co-workers³⁴ examined the effects of adding exercise to dietary changes which included a decreased intake of saturated fat and cholesterol. In women, only the group who exercised as well as dieting avoided the decrease in HDL cholesterol which accrues from increasing the proportion of dietary energy obtained from carbohydrate.

Finally, it should be noted that, in women as well as in men, higher levels of physical fitness are, independent of other major risk factors, associated with delayed all-cause mortality and in particular with lower death rates from cardiovascular disease and cancer³⁵. Thus the improvements in physical fitness which resulted from brisk walking may be as important in the context of the risk of CHD as the increases in HDL cholesterol concentrations which accompanied them.

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