

Influence of 12 Weeks of Training by Brisk Walking on Postprandial Lipemia and Insulinemia in Sedentary Middle-Aged Women

H.E. Aldred, A.E. Hardman, and S. Taylor

The purpose of this study was to examine the influence of brisk walking on postprandial lipemia in 26 sedentary women aged 41 to 55 years. The lipemic response to a high-fat meal (mean \pm SEM: 73.8 \pm 1.3 g fat, 66% energy; 81.8 \pm 1.4 g carbohydrate) was determined pretraining and posttraining. Blood samples were obtained in the fasted state and hourly for 6 hours after the meal. Serum was analyzed for triacylglycerol (TAG), total cholesterol, high-density lipoprotein (HDL) and HDL₂ cholesterol, apolipoproteins (apop) A-I and B, nonesterified fatty acids (NEFA), glucose, and insulin. Subjects were randomly assigned to one of two groups: walkers (n = 13) followed a program of brisk walking (average of 21 \pm 1 [range, 17 to 27] min \cdot d⁻¹ at 1.76 \pm 0.02 m \cdot s⁻¹), whereas controls (n = 13) maintained their habitual life-style. Procedures were repeated 12 weeks later, with 48 hours between the last training session and determination of postprandial lipemia. Eleven walkers and 13 controls completed the study. Responses over time were compared between groups (Mann-Whitney U, *P* < .05). Brisk walking improved endurance fitness and decreased body fatness, but had no influence on peak TAG concentration (walkers, 1.6 \pm 0.2 v 1.6 \pm 0.2 mmol \cdot L⁻¹; controls, 1.9 \pm 0.3 v 2.1 \pm 0.3) or the area under the TAG/time curve after the test meal. The area under the insulin/time curve decreased in walkers relative to controls. These results suggest that in sedentary women aged 41 to 55, brisk walking attenuates the serum insulin response, but not the lipemic response, to consumption of a high-fat mixed meal when these responses are determined 48 hours after the last exercise bout.

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ENDURANCE-TRAINED men and women exhibit high plasma levels of high-density lipoprotein (HDL) cholesterol.¹ These may reflect a high metabolic capacity for triacylglycerol (TAG) if high TAG levels are indeed the driving force for low HDL cholesterol concentrations.² Consistent with this view, the magnitude and duration of postprandial lipemia after challenge with a fat load, an integrative marker of TAG metabolic capacity, appears to be lower in endurance-trained athletes than in less active individuals.³

A rationale for an influence of exercise training on postprandial lipemia can be advanced because endurance training increases the activity of lipoprotein lipase (LPL),⁴ the enzyme that hydrolyzes TAG-rich lipoproteins. This is probably a consequence of training-induced adaptations of the microcirculation in skeletal muscles,⁵ since LPL is found on the luminal surface of capillaries.

These observations may be important because atherogenesis has been described as a postprandial phenomenon.⁶ Animal studies show that cholesteryl ester-rich remnants of TAG-rich lipoproteins may contribute to the deposition of lipid in the arterial wall,⁷ and cultured human arterial smooth muscle cells take up these remnants, which markedly increases cell cholesterol content.⁸ These observations are concordant with case-control studies reporting that postprandial lipemia is elevated in men with known coronary artery disease.⁹

Man spends most of his life in the postprandial state, and the low lipemic response to ingestion of dietary fat in physically active men may therefore contribute to their reduced risk of coronary heart disease.¹⁰ However, whether moderate-intensity training that might be acceptable to a majority of adults is sufficient to provoke these adaptations in individuals without dyslipidemia is unknown.

Few longitudinal studies have examined the influence of exercise training on postprandial lipemia,¹¹⁻¹⁵ and three of these were in patient groups. Exercise programs have been rather intense, the total number of subjects studied has been small, and findings have been conflicting. All such studies have been conducted on men, despite the paucity of information concerning the influence of exercise on risk factors for coronary heart disease in women. The case for a study in women is strengthened by a recent report that increased nonfasting TAG concentrations are an independent risk factor for mortality from this disease in women.¹⁶

The purpose of the present study was therefore to examine the influence of training by brisk walking on postprandial lipemia in middle-aged women, a group with very low levels of physical activity¹⁷ and in whom this form of exercise is known to improve endurance fitness.¹⁸ The lipemic response was determined 48 hours after the last training session to eliminate possible acute effects of brisk walking previously shown to persist for more than 12 hours in young adults.¹⁹ We chose to study the lipemic response to a test meal that contained a mixture of dietary fat and carbohydrate because this was both more palatable and more life-like than fat alone.

SUBJECTS AND METHODS

Study Design

The study was a randomly controlled, exercise-intervention trial, conducted with the approval of the University Ethical Advisory Committee. All measurements were made at baseline and, with the exception of preliminary tests, were repeated after 12 weeks. Randomization to either the brisk-walking group (n = 13) or control group (n = 13) took place after baseline tests were com-

From the Department of Physical Education, Sports Science and Recreation Management, Loughborough University; and Department of Anesthesia, Glenfield General Hospital, Leicestershire, UK.

Submitted March 7, 1994; accepted June 18, 1994.

Supported by the British Heart Foundation.

Address reprint requests to A.E. Hardman, PhD, Department of Physical Education, Sports Science and Recreation Management, Loughborough University, Loughborough, Leicestershire, LE11 3TU United Kingdom.

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0026-0495/95/4403-0018\$03.00/0

pleted. Groups were stratified by age because the magnitude of postprandial lipemia after ingestion of a high-fat meal is greater in older people.²⁰ Women allocated to the control group were offered the opportunity to take part in a similar exercise program after completion of the study.

Subjects

Subjects were recruited by advertising locally. Volunteers aged 40 to 59 years were accepted provided they met the following criteria: (1) nonsmoker; (2) not involved in a program of regular exercise, including sustained brisk or fast walking; (3) systemic arterial blood pressure less than 160/95 mm Hg; (4) total cholesterol less than $7.8 \text{ mmol} \cdot \text{L}^{-1}$ and fasting TAG less than $2.3 \text{ mmol} \cdot \text{L}^{-1}$; (5) nondiabetic; and (6) free from a clinical history of bleeding or coagulation disorders or physician-diagnosed cardiovascular disease. Twenty-six women (mean \pm SEM: walkers, 49.6 ± 1.3 years; control, 49.1 ± 1.3 years) agreed to take part and gave their informed consent. Confidential interviews before the study revealed that 11 were premenopausal (five walkers, six controls), five perimenopausal (three walkers, two controls), and 10 postmenopausal (five walkers, five controls). None were taking drugs known to influence lipid or lipoprotein metabolism. Fourteen were overweight (body mass index [BMI], 25 to $30 \text{ kg} \cdot \text{m}^{-2}$), and one was obese (BMI $> 30 \text{ kg} \cdot \text{m}^{-2}$) (mean \pm SEM BMI: walkers, 24.8 ± 1.0 ; controls, 26.8 ± 0.8). Assessment of physical activity status was made using a questionnaire (maximum score, 100); at baseline, activity scores were 24 ± 2 for walkers and 22 ± 2 for controls. The questionnaire recorded walking behavior and assessment of pace and duration, and participation in sports and heavy work around the home and garden.

Exercise Tests

Changes in endurance fitness were monitored using a laboratory exercise test. After subjects had become familiar with walking on a motorized treadmill, a preliminary incremental test was conducted at a submaximal level to predict maximal oxygen uptake.²¹ The actual test then consisted of 16 minutes of walking at a constant speed, ie, 4 minutes at each of four gradients selected to elicit 50%, 60%, 70%, and 80% of individual predicted maximal oxygen uptake. At the end of each stage, finger-prick samples of capillary blood were obtained to determine blood lactate concentration. Oxygen uptake and carbon dioxide production were determined throughout using Douglas-bag techniques, and heart rate was measured from an ECG (modified lead I). Oxygen uptake at a reference blood lactate concentration ($3 \text{ mmol} \cdot \text{L}^{-1}$) was interpolated for each subject and adopted as a measure of endurance fitness that could be directly determined without the need for maximal exercise.

Anthropometry

Standard anthropometric techniques were used to measure body mass, height, skinfold thickness (biceps, triceps, subscapular, and suprailiac), and circumferences at the waist and hip.

Oral Fat Tolerance Test

Subjects refrained from exercise during the 2 days immediately preceding this test both pretraining and posttraining. During the pretraining period, food intake was weighed and recorded for these 2 days and then replicated for the corresponding period before the posttraining test. Subjects abstained from alcohol for 24 hours before each test. They reported to the laboratory in the morning, after a 12-hour fast. A cannula was inserted into a vein in the forearm or cubital fossa, and the subject rested quietly for 10 minutes before a baseline blood sample was obtained. The meal

was then ingested within a 15-minute period (walkers, 13.0 ± 0.7 v 11.1 ± 0.7 minutes, pretraining and posttraining, respectively; controls, 13.9 ± 0.6 v 11.9 ± 0.5). This consisted of cereal, fruit, chocolate, nuts, and whipping cream and was given according to each subject's fat-free mass, ie, 1.8 g dietary fat/kg fat-free mass. It provided $73.0 \pm 1 \text{ g}$ fat (contributing 66% of energy), $14.3 \pm 0.3 \text{ g}$ protein, and $81.8 \pm 1.4 \text{ g}$ carbohydrate—a total energy content of $4.3 \pm 0.8 \text{ MJ}$, approximately 50% of the estimated daily energy intake of a typical sedentary, middle-aged woman.²² Further 10-mL blood samples were obtained at hourly intervals for 6 hours after completion of the meal. Only water was consumed during this time. Posture was standardized before each blood sample, and hematocrit and hemoglobin levels were determined to allow changes in plasma volume to be monitored. The total lipemic response was determined as the area under the serum total TAG concentration/time curve normalized to the zero hour. We have previously reported the reproducibility of this procedure.²³

Blood Biochemistry

Within-batch coefficients of variation are shown in parentheses for each of the following assays used. Capillary blood samples obtained during exercise tests were immediately deproteinized and stored at -20°C until assayed for lactate (3.9%) by a fluorimetric micromethod.²⁴ Serum was separated, and an aliquot was removed and stored at 4°C for ≤ 3 days before analysis for HDL (2.0%) and HDL₃ (3.8%) cholesterol by manganese-heparin precipitation.²⁵ HDL₂ cholesterol level was calculated as the difference between HDL and HDL₃ cholesterol. Remaining serum was stored -70°C . Cholesterol (1.2%), glucose (1.0%), TAG (0.6%), and nonesterified fatty acids ([NEFA] 1.9%) levels were measured by enzymatic colorimetric methods (Boehringer, Mannheim, Germany, and Wako, Neuss, Germany). Insulin (4.5%) level was determined by radioimmunoassay (Diagnostic Products, Los Angeles, CA). Accuracy was maintained using quality-control sera (Roche, Basel, Switzerland), and for subfractions of HDL, a pooled serum sample. Apolipoproteins (apos) A-I (0.8%) and B (0.8%) were determined using an immunoturbidimetric method (Roche) in serum from the baseline, 2-, 4-, and 6-hour samples only. With the exception of HDL and HDL₂, which were assayed in fresh sera, all assays were performed after storage, with pretraining and posttraining samples from an individual subject assayed in a single assay or analyzer run. Phenotypes of apo E were determined by isoelectric focusing using Western blot techniques.

Brisk-Walking Program

Subjects allocated to the control group maintained their habitual sedentary life-style, and subjects in the walking group followed the exercise program. All agreed not to make any planned dietary changes during the study. The walking program increased from a target of 60 minutes of brisk walking in the first week to 180 minutes during weeks 11 and 12. Walkers were asked to attend up to two supervised training sessions per week, with the remaining walking being unsupervised but recorded in diaries that were returned every 2 weeks. The minimum length of each walking session was 20 minutes, and the maximum, 50 minutes. A timed "brisk" walk around an athletics track was undertaken at the start of the training program and again after 12 weeks to provide an estimate of training pace.

Data Analysis

Changes over the 12 weeks were compared between walkers and controls using the Mann-Whitney *U* test. Relationships between variables were examined using the Spearman rank-order correla-

tion coefficient. Data are shown as the mean \pm SEM throughout, and a .05 level of significance was adopted.

RESULTS

Of the 13 walkers, 11 completed the study, with one dropping out because of illness and another because of work commitments. All controls returned for testing at the end of the study. At baseline, there were no significant differences between walkers and controls in any of the parameters measured.

Compliance With the Walking Program

The average number of minutes of walking for exercise performed over the 12-week period was 21 ± 1 (range, 17 to 27) per day, with the number of sessions per week increasing progressively from 3.5 ± 0.3 (range, 2 to 6) to 5.6 ± 0.3 (range, 4 to 7). The average duration of each training

session also increased, from 24 ± 1 minutes (range, 20 to 30) to 33 ± 1 (range, 25 to 40). The 1-mile timed walk showed that the brisk-walking speed was $1.76 \pm 0.02 \text{ m} \cdot \text{s}^{-1}$ at baseline and 1.86 ± 0.03 at the end of the study. By taking an average of these two speeds, distance walked in training was estimated as $16.3 \pm 0.8 \text{ km} \cdot \text{wk}^{-1}$ (range, 12.7 to 21.7). Attendance at supervised training sessions was 1.5 ± 0.2 times per week. During these sessions, a mean heart rate of 132 ± 3 (range, 115 to 152) $\text{beats} \cdot \text{min}^{-1}$ was recorded, equivalent to $74\% \pm 2\%$ (range, 63% to 86%) of age-related maximal heart rate.

Apo E Phenotypes

Nine walkers possessed the 3-3 phenotype, one the 4-2, and one the 4-3. Of the controls, 12 were 3-3 and one was 4-3.

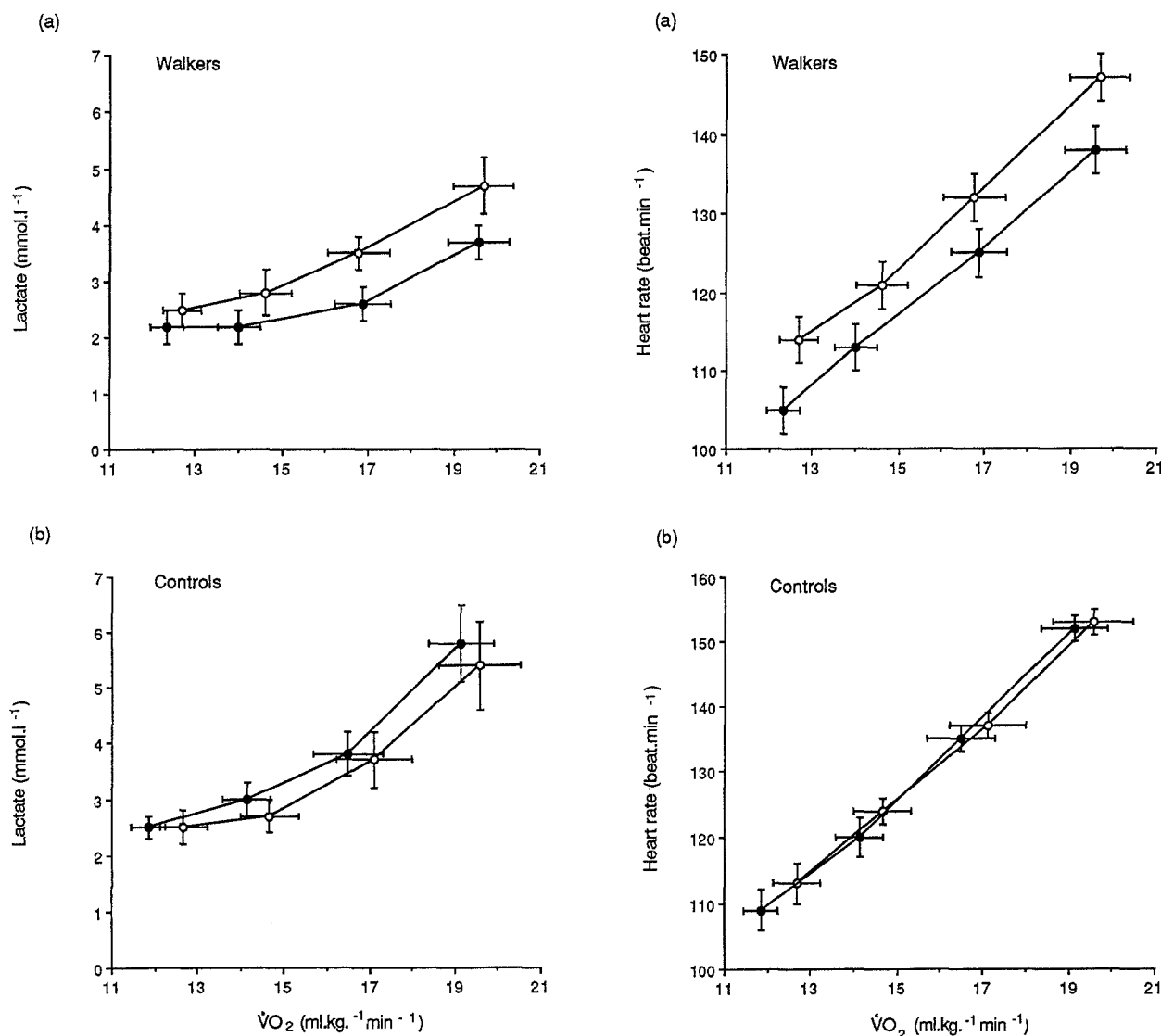


Fig 1. Heart rate and blood lactate concentration during submaximal treadmill walking pretraining (○) and posttraining (●) for (a) walkers ($n = 11$) and (b) controls ($n = 13$). Mean \pm SEM. $\dot{V}O_2$, oxygen consumption.

Body Mass and Fatness

Body mass changed in a different way between groups ($P < .05$), decreasing in walkers (64.4 ± 3.0 v 63.4 ± 3.0 kg, pretraining and posttraining, respectively) and increasing in controls (69.8 ± 2.1 v 70.8 ± 2.4 kg), with considerable interindividual variation. Similarly, the sum of skinfolds decreased in walkers (83.2 ± 9.7 v 76.4 ± 9.0 mm) but increased in controls (100.3 ± 6.4 v 104.6 ± 6.7 mm) ($P < .05$). No significant differences in changes in the ratio of circumferences at the waist and hip were found (walkers, 0.77 ± 0.01 v 0.79 ± 0.01 ; controls, 0.77 ± 0.01 v 0.78 ± 0.01).

Cardiovascular and Metabolic Adaptations to Brisk Walking

Brisk walking decreased both the heart rate and blood lactate concentration during submaximal treadmill walking at a given oxygen uptake (Fig 1). In controls, heart rate at a given oxygen uptake remained unchanged, but blood lactate concentration increased. Consequently, endurance fitness, as measured by oxygen uptake at a reference blood lactate concentration of $3 \text{ mmol} \cdot \text{L}^{-1}$, changed in a different way ($P < .05$) in walkers (16.4 ± 1.1 v $18.8 \pm 1.0 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and controls (16.7 ± 1.0 v 14.9 ± 0.6).

Lipid and Lipoprotein Parameters, Insulin, and Glucose in the Fasted State

Low-density lipoprotein cholesterol (calculated by subtraction and using the Friedwald formula²⁶) changed in a different way between groups (walkers, 3.25 ± 0.25 v $3.07 \pm 0.26 \text{ mmol} \cdot \text{L}^{-1}$; controls, 3.40 ± 0.13 v 3.49 ± 0.14 ; $P < .05$). Similarly, a difference was found in changes in the ratio of total to HDL cholesterol over the study (walkers, 3.49 ± 0.34 v 3.32 ± 0.29 ; controls, 3.52 ± 0.19 v 3.73 ± 0.20 ; $P < .05$). Brisk walking did not influence fasting concentrations of either insulin (walkers, 7.2 ± 0.7 v $6.2 \pm 0.6 \mu\text{U} \cdot \text{mL}^{-1}$; controls, 6.4 ± 0.8 v 5.6 ± 0.4) or glucose (walkers, 5.6 ± 0.1 v $5.4 \pm 0.1 \text{ mmol} \cdot \text{L}^{-1}$; controls, 5.5 ± 0.1 v 5.4 ± 0.1).

Responses to Oral Fat Tolerance Test

No subjects experienced symptoms of malabsorption, but one control did not complete the second test because she could not tolerate the meal. Another subject, also a control, was ill during the days scheduled for posttraining fat tolerance tests. Therefore, these data are presented for 11 walkers and 11 controls. Postprandial TAG concentrations over the 6-hour observation period are shown in Fig 2. Considerable interindividual variation was evident. No differences in changes over the study between walkers and controls were observed for either peak TAG concentration (walkers, 1.6 ± 0.2 v $1.6 \pm 0.2 \text{ mmol} \cdot \text{L}^{-1}$; controls, 1.9 ± 0.3 v 2.1 ± 0.3) or total lipemic response (walkers, 2.4 ± 0.5 v $2.6 \pm 0.4 \text{ mmol} \cdot \text{L}^{-1} \cdot \text{h}$; controls, 3.5 ± 0.6 v 3.6 ± 0.6). Changes in the lipemic response over the study were unrelated to changes in either endurance fitness or body fatness. Fasting TAG concentration was related to total lipemic response (pretraining $\rho = 0.53$, posttraining $\rho = 0.84$, both $P < .01$). There was also a modest inverse

relationship between the magnitude of postprandial lipemia and fasting HDL₂ cholesterol concentration (pretraining $\rho = -0.39$, posttraining $\rho = -0.37$, both $P < .05$).

Concentrations of serum total cholesterol and HDL₂ cholesterol at some observation points during the oral fat tolerance test changed in a different way in walkers and controls (Table 1). Total cholesterol decreased in walkers as compared with controls (4 to 6 hours), whereas HDL₂ increased (2 to 4 hours). Values for 1, 3, and 5 hours are not presented, since they add little further information. Of the apoprotein concentrations, only apo B at 6 hours changed differently ($P < .05$) in walkers (0.94 ± 0.05 v $0.87 \pm 0.06 \text{ g} \cdot \text{L}^{-1}$) and controls (0.92 ± 0.05 v 0.96 ± 0.03) (other values not shown).

Peak postprandial insulin concentration changed differently ($P < .05$) between groups (walkers, 43.0 ± 8.6 v $34.8 \pm 6.6 \mu\text{U} \cdot \text{mL}^{-1}$; controls, 33.4 ± 3.1 v 42.6 ± 4.7).

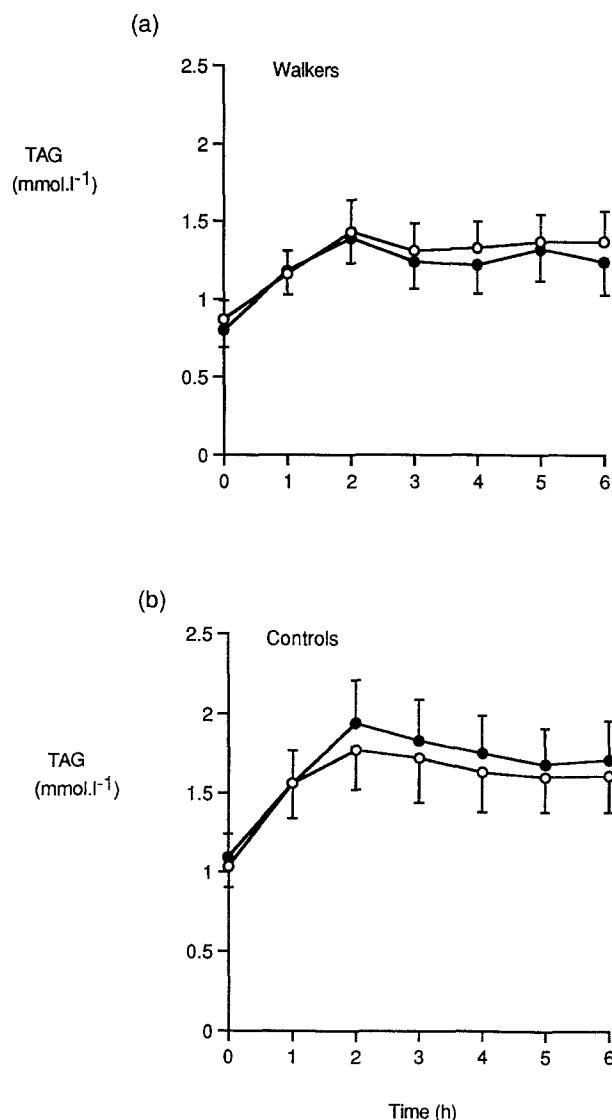


Fig 2. Serum TAG concentration before and after consumption of the test meal pretraining (○) and posttraining (●) for (a) walkers ($n = 11$) and (b) controls ($n = 13$). Mean \pm SEM.

Table 1. Lipid and Lipoprotein Responses (mean \pm SEM) After Consumption of the Test Meal for Walkers (n = 11) and Controls (n = 11)

	Time After Ingestion			
	0 h	2 h	4 h	6 h
NEFA (mmol \cdot L ⁻¹)				
Walkers				
Baseline	0.71 \pm 0.10	0.43 \pm 0.08	0.71 \pm 0.08	0.97 \pm 0.08
Posttraining	0.61 \pm 0.07	0.42 \pm 0.05	0.59 \pm 0.04	0.87 \pm 0.07
Controls				
Baseline	0.75 \pm 0.07	0.49 \pm 0.07	0.71 \pm 0.06	1.10 \pm 0.08
Posttraining	0.78 \pm 0.06	0.44 \pm 0.06	0.63 \pm 0.07	1.09 \pm 0.08
TC (mmol \cdot L ⁻¹)				
Walkers				
Baseline	5.25 \pm 0.25	5.27 \pm 0.25	5.30 \pm 0.25	5.50 \pm 0.25
Posttraining	4.99 \pm 0.25	5.06 \pm 0.25	4.99 \pm 0.26*	5.07 \pm 0.26*
Controls				
Baseline	5.46 \pm 0.16	5.41 \pm 0.13	5.41 \pm 0.14	5.44 \pm 0.15
Posttraining	5.49 \pm 0.11	5.49 \pm 0.11	5.50 \pm 0.11	5.61 \pm 0.11
HDL-C (mmol \cdot L ⁻¹)				
Walkers				
Baseline	1.59 \pm 0.10	1.58 \pm 0.10	1.58 \pm 0.10	1.60 \pm 0.10
Posttraining	1.56 \pm 0.07	1.57 \pm 0.07	1.56 \pm 0.08	1.57 \pm 0.08
Controls				
Baseline	1.59 \pm 0.08	1.58 \pm 0.08	1.54 \pm 0.09	1.55 \pm 0.08
Posttraining	1.53 \pm 0.08	1.54 \pm 0.08	1.53 \pm 0.08	1.57 \pm 0.08
HDL ₂ -C (mmol \cdot L ⁻¹)				
Walkers				
Baseline	0.54 \pm 0.08	0.58 \pm 0.08	0.54 \pm 0.09	0.56 \pm 0.08
Posttraining	0.51 \pm 0.06	0.57 \pm 0.06*	0.57 \pm 0.07*	0.54 \pm 0.06
Controls				
Baseline	0.55 \pm 0.04	0.60 \pm 0.04	0.59 \pm 0.05	0.55 \pm 0.08
Posttraining	0.47 \pm 0.06	0.51 \pm 0.06	0.59 \pm 0.05	0.45 \pm 0.07

Abbreviation: TC, total cholesterol.

*Change pretraining to posttraining significantly different between walkers and controls, $P < .05$.

Similarly, the area under the insulin concentration/time curve (normalized to the fasting concentration) changed in a different way ($P < .05$) in walkers (80.0 ± 23.5 v 56.0 ± 13.0 $\mu\text{U} \cdot \text{mL}^{-1} \cdot \text{h}$) and controls (57.7 ± 8.3 v 80.9 ± 12.7) (Fig 3). The change in the area under the curve was related to the change in body mass ($\rho = 0.48$, $P < .05$) but not to the change in the sum of skinfolds. Therefore, the relationship with change in body mass was not weakened by controlling for change in skinfolds ($\rho = 0.47$). Change in insulin area was inversely related to changes in endurance fitness ($\rho = -0.48$, $P < .05$). Peak postprandial glucose concentration (walkers, 6.7 ± 0.4 v 6.2 ± 0.3 mmol \cdot L⁻¹; controls, 6.3 ± 0.2 v 6.3 ± 0.3) decreased in walkers relative to controls ($P < .05$), but changes in the area under the glucose/time curve did not differ.

DISCUSSION

Most of our volunteers were at the upper end of the desirable range of weight for height and were typical of the 58% of middle-aged English women whose level of physical activity is less than the threshold necessary to achieve health gains.¹⁷ The main finding was that in these sedentary women there was no change in the lipemic response to a high-fat test meal as a result of training by brisk walking, when this response was determined 48 hours after the last training session. In contrast, postprandial serum insulin

response to the meal was clearly blunted, even after 2 days of inactivity.

Random allocation of subjects resulted in two groups similar at baseline with regard to the variables of interest, including the distribution of apo E phenotypes—that expected for a general white population.²⁷ Compliance with the exercise program was good: walkers completed an average of more than 20 minutes of brisk walking per day at a pace of approximately 15 minutes per mile, more than the target time and equivalent to approximately 2.4 km (1.5 mi). No exercise-related injuries were sustained, and a low dropout rate was experienced (15%). Average heart rate during supervised training sessions was $74\% \pm 2\%$ of predicted maximal values, within guidelines proposed by the American College of Sports Medicine²⁸ for maintaining endurance fitness in healthy adults, which suggests that brisk walking elicited more than 60% of maximal oxygen uptake in these women.

Endurance fitness was clearly improved by this exercise regimen, in line with earlier reports in women^{18,29}; heart rate and blood lactate concentration were both reduced at a given oxygen uptake during treadmill walking (Fig 1). Changes in blood lactate concentration were particularly marked and showed that whereas endurance fitness improved over the study in walkers, it actually decreased in controls.

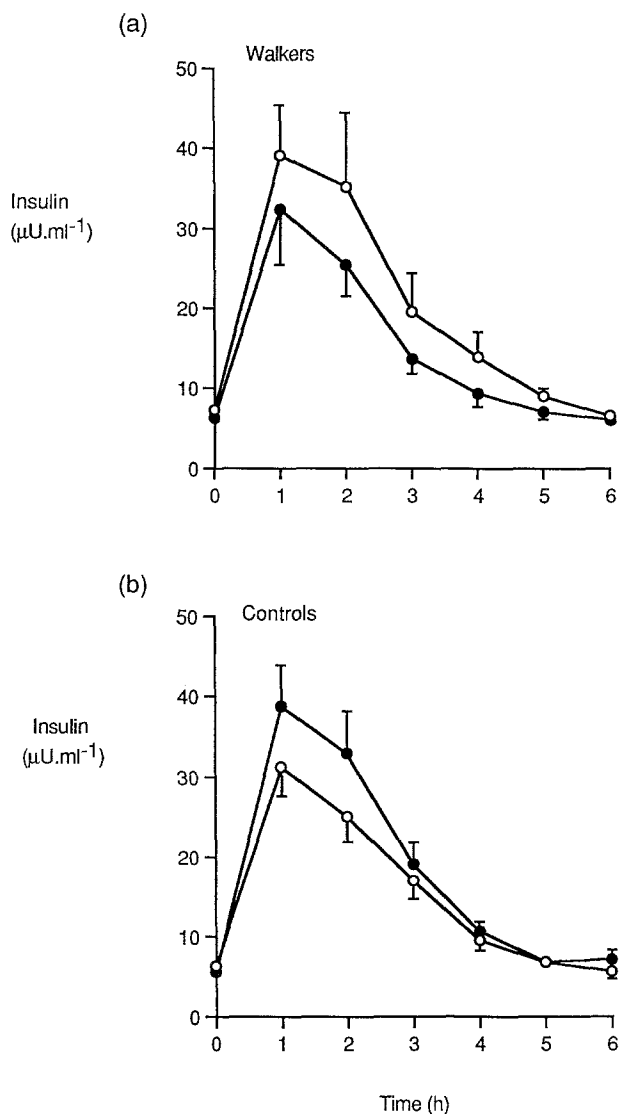


Fig 3. Serum insulin concentration before and after consumption of the test meal pretraining (○) and posttraining (●) for (a) walkers ($n = 11$) and (b) controls ($n = 13$). Mean \pm SEM.

Brisk walking resulted in only subtle changes in serum lipids and lipoproteins determined in the fasted state. The lack of change in total cholesterol is not surprising, since this does not differ consistently between trained and sedentary individuals.¹ However, the lack of change in HDL cholesterol contrasts with our earlier study where HDL cholesterol increased markedly in women of mean age 45 years who followed a similar walking program but possessed lower HDL cholesterol levels at the outset.³⁰ The effectiveness of walking programs in modifying HDL cholesterol in sedentary women is therefore still unclear, with some studies reporting no change^{31,32} and others reporting an increase.^{29,30,33,34} In the present study, although there was no increase in HDL cholesterol, the ratio of total to HDL cholesterol was reduced relative to control levels. This change is suggestive of some alterations in the dynamic exchange of lipid and cholesterol between lipoproteins as a

result of brisk walking, especially since it was accompanied by a decrease in low-density lipoprotein cholesterol.

Arguably, such alterations might be more clearly seen in the postprandial state. Chylomicrons and endogenous TAG-rich lipoproteins are degraded by a common saturable pathway,³⁵ so that the lipemic response can be regarded as an integrated marker for total metabolic capacity for TAG. Preferential clearance of chylomicron TAG by LPL leads to an accumulation of very-low-density lipoproteins,³⁶ which suggests that the size of the endogenous pool will influence postprandial lipemia. Our data support this view with the total lipemic response being positively related to fasting TAG concentration ($p = 0.53$ and 0.84 pretraining and posttraining, respectively), as previously reported.³⁷

However, brisk walking had no long-lasting effect on the total lipemic response to our mixed meal, which suggests that improved endurance fitness does not necessarily result in changes in the lipemic response above those attributable to the residual effects of the last bout of exercise. This finding is not distorted by individual atypical responses, and there was no relationship between changes in endurance fitness and changes in our indices of lipemia. Some differences between walkers and controls were evident during the later postprandial phase, with the most marked being a lower total cholesterol response in the walkers (Table 1), but they do not represent a clearly less "atherogenic" scenario in the trained state. However, an important limitation to this conclusion, is that our methodology does not distinguish between endogenous and exogenous TAGs: up to 25% of the postprandial increase in serum total TAG may be attributable to endogenous TAG-rich lipoproteins,³⁸ which probably contribute to the potentially atherogenic stimulus during this period. Measurements of cholesterol ester-rich remnants of TAG-rich lipoproteins (from both sources) might conceivably reveal alterations in TAG metabolism masked by measurements of total TAG.

Previous studies are not all inconsistent with our results. To our knowledge, five other studies have examined the influence of exercise training on postprandial lipemia, two of which found no change,^{12,15} and three, a decrease.^{11,13,14} All these investigators attempted to remove the effect of the last exercise session. Intervals of 36 hours to 3 days separated training from measurements of lipemia. In two studies, subjects were trained by running an average of more than 50 miles per week for 10 weeks¹¹ or running 15 miles per week at an "intense" pace that elicited 80% maximal heart rate.¹⁴ The third trained patients with primary hypertriglyceridemia using three hour-long sessions per week of "jogging, ball games, callisthenics and short periods of relaxation."¹³ High levels of endogenous TAG in these patients were probably associated with profound postprandial lipemia,²⁰ perhaps leading to a greater potential to demonstrate an influence of exercise training. Thus, where postprandial lipemia has been attenuated by training, the exercise has been more intense than our program, or postprandial lipemia would have been high at baseline.

Intriguingly, although it had no influence on postprandial lipemia, brisk walking was associated with a 35% decrease

in the serum insulin response to the test meal (Fig 3), as compared with a 23% increase in controls. Since this was not accompanied by an increase in blood glucose concentrations, it is suggestive of improved insulin sensitivity.

A contributory factor may have been the modest decreases in body mass (just over 1 kg on average) and subcutaneous fat in these women. Changes in insulin response were related to changes in body mass ($\rho = 0.48$) but not to changes in the sum of skinfolds ($\rho = 0.19$), which suggests that alterations in internal, rather than subcutaneous fat were the more important determinants of the altered insulin response. This would tie in with the proposal that insulin resistance is particularly associated with intraabdominal fat.³⁹ Changes in the waist to hip ratio, our index of central adiposity, were unrelated to changes in the insulin response, but this measure may not reflect visceral fat, which is arguably the more important indicator of diabetic risk. (Haskell WL, personal communication, June 2, 1993).

The decrement in the insulin response with walking, 11% at 2 hours after the meal, appears somewhat greater than expected from the small weight loss⁴⁰ per se, and adaptive changes in skeletal muscle may have been an additional factor. Changes in endurance fitness were related to changes in the insulin response ($\rho = -0.48$), in support of this view. Moreover, other investigators have confirmed that training can influence insulin sensitivity without changes in body composition.⁴¹

Since none of the walking training sessions were longer than 40 minutes or exhaustive, our data suggest that exercise does not have to be glycogen-depleting to stimulate improvement in glycemic control. Moreover, since the effect on the insulin response was clearly evident so long after the last training session, brisk walking three to four times per week might be sufficient to oppose the development of insulin resistance. Fifty-five percent of women aged 45 to 54 in England carry excess weight⁴² ($\text{BMI} > 25 \text{ kg} \cdot \text{m}^{-2}$), and 58% do not achieve suggested target thresholds for physical activity.¹⁷ Plasma insulin levels are known

to be positively associated with both body weight and physical inactivity in women.⁴⁰ The finding that brisk walking can mitigate the postprandial increase in serum insulin concentration therefore strengthens the case for promoting this activity in middle-aged women. It also goes some way toward allaying fears that exercise may have to be intense to improve glycemic control and therefore is contraindicated in patients with non-insulin-dependent diabetes, many of whom exhibit a constellation of cardiovascular risk factors.

In our model, with a meal that contained both fat and carbohydrate, serum insulin concentrations are increased for much of the 6-hour observation period. Changes in this response are of interest not only because of implications for the development of insulin resistance, but also because of the central role of this hormone in the regulation and coordination of postprandial lipid metabolism.⁴³ In adipose tissue, insulin suppresses release of NEFA (concentrations decrease for 2 to 3 hours after our meal, Table 1) and stimulates LPL activity, which enhances hydrolysis of circulating TAG. In contrast, it inhibits LPL in skeletal muscle, which decreases uptake of NEFA from TAG. It has even been suggested that basal muscle LPL activity could be regarded as an indicator of muscle insulin sensitivity.⁴⁴ One might therefore expect increased sensitivity to insulin to be accompanied by a decrease in postprandial lipemia. In this regard, the lack of any indication of a change in lipemia in walkers is particularly surprising.

In conclusion, we have shown that training by brisk walking improves endurance fitness in sedentary middle-aged women and decreases postprandial insulinemia, but not postprandial lipemia, when these responses are determined 48 hours after the last exercise session.

ACKNOWLEDGMENT

We thank Dr S. Mastana, Department of Human Sciences, Loughborough University, for measurement of apo E phenotypes.

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