

The effect of 13 weeks of running training followed by 9 d of detraining on postprandial lipaemia

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(Received 22 May 1997 – Revised 9 January 1998 – Accepted 27 January 1998)

The present study examined the influence of training, followed by a short period of detraining, on postprandial lipaemia. Fourteen normolipidaemic, recreationally active young adults aged 18–31 years participated, in two self-selected groups: three men and five women (BMI 21.7–27.6 kg/m²) completed 13 weeks of running training, after which they refrained from exercise for 9 d; three men and three women (BMI 21.5–25.6 kg/m²) maintained their usual lifestyle. Oral fat tolerance tests were conducted at baseline and again 15 h, 60 h and 9 d after the runners' last training session. Blood samples were drawn after an overnight fast and at intervals for 6 h after consumption of a high-fat meal (1.2 g fat, 1.4 g carbohydrate, 70.6 kJ energy/kg body mass). Heparin was then administered (100 IU/kg) and a further blood sample was drawn for measurement of plasma lipoprotein lipase (EC 3.1.1.34; LPL) activity. Endurance fitness improved in runners, relative to controls (maximal O₂ uptake +3.2 (SE 1.1) ml/kg per min v. -1.3 (SE 1.2) ml/kg per min; *P* < 0.05). In the absence of the acute effect of exercise, i.e. 60 h after the last training session, there was no effect of training on either postprandial lipaemia or on post-heparin LPL activity. However, changes during 9 d of detraining in both these variables differed significantly between groups; after 2 d without exercise (60 h test), the runners' lipaemic response was 37 % higher than it was the morning after their last training session (15 h test; runners v. controls *P* < 0.05), with a reciprocal decrease in post-heparin LPL activity (*P* < 0.01). These findings suggest that improved fitness does not necessarily confer an effect on postprandial lipaemia above that attributable to a single session of exercise.

Exercise: Postprandial metabolism: Triacylglycerols

Endurance-trained individuals have been reported to possess blood lipid and lipoprotein profiles that are consistent with a low risk of developing CHD (Durstine & Haskell, 1994), most noticeably high plasma concentrations of HDL-cholesterol. The principal determinant of the latter characteristic may be an enhanced metabolic capacity for triacylglycerol (TAG), because the degradation in the circulation of TAG rich lipoproteins proceeds with the transfer of surface material to nascent HDL. In line with this view, endurance-trained men (Sady *et al.* 1988; Cohen *et al.* 1989) and women (Podl *et al.* 1994) exhibit enhanced plasma TAG clearance rates compared with sedentary controls.

One explanation (Podl *et al.* 1994) has focused on augmented TAG hydrolysis in skeletal muscle by the action of lipoprotein lipase (EC 3.1.1.34; LPL). Several mechanisms arising from the adaptations of skeletal muscle to training may contribute (Kiens & Lithell, 1989): improvements in the microcirculation would increase the

number of binding sites available to LPL; the longer transit time of blood through more extensive muscle capillary beds would increase the exposure of enzyme to substrate; improvements in muscle oxidative capacity would, theoretically, enhance tissue uptake of fatty acids and reduce product inhibition of LPL activity. Collectively, these adaptations would increase the potential for the hydrolysis of TAG and for the removal and utilization of fatty acids from the site of this reaction.

Doubt exists, however, about the extent to which high TAG removal rates in trained people reflect chronic adaptations to training, because there is also an acute effect of a single session of exercise, which may be mediated by an increase in muscle LPL activity. TAG removal rates have been reported to be elevated 1 d after a prolonged session of exercise (Sady *et al.* 1986), and serum TAG concentrations measured in the fasted state may be reduced for more than 2 d (Annuzzi *et al.* 1987). In the cross-sectional studies

Abbreviations: LPL, lipoprotein lipase; NEFA, non-esterified fatty acids; TAG, triacylglycerol; $\dot{V}O_2$ max, maximal O₂ uptake.

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referred to previously, athletes were asked to refrain from exercise training for 12 h (Cohen *et al.* 1989; Podl *et al.* 1994) or for 24 h (Sady *et al.* 1988) before measurements of TAG clearance rate. As athletes invariably wish to keep any period without exercise to a minimum, individuals are likely to have trained just before these deadlines, and the high clearance rates reported may have been strongly influenced by the effects of a recent training session.

The purpose of the present study was therefore to evaluate the relative importance of chronic adaptations to training and the acute effects of a single exercise session as determinants of a high metabolic capacity for TAG. We studied two groups of subjects, all of whom were untrained at baseline, over a period of 13 weeks; one group followed a progressive programme of running training (runners), whilst the other group maintained their habitual lifestyle (controls). The objectives of our study were twofold, to examine: (1) the chronic adaptive effect of training on postprandial lipaemia in the absence of an acute effect of exercise, i.e. 2 d after the last training session (2) the time scale of changes in lipaemia when trained individuals refrain from training for a short period. A high-fat meal was employed as a challenge to TAG metabolic capacity and lipaemia was determined as the plasma TAG response over a 6 h postprandial period.

Materials and methods

Study design

This was a controlled exercise-intervention study. Postprandial lipaemia was determined for runners and controls on four separate occasions: at baseline and then 15 h, 60 h and 9 d after the intervention group completed the last session of a 13-week programme of running training. Controls maintained their normal lifestyle for the duration of the study.

All subjects refrained from all exercise other than activities of daily living for the 2 d preceding baseline measurements, and controls did the same before each subsequent measurement. For some individuals, activities of daily living included short journeys at slow pace by bicycle or on foot. After their last training session, runners refrained from all exercise other than activities of daily living (defined previously) until the study was completed. Their final training session took place the evening before the first post-training fat-tolerance test. This comprised a 40 min run at a pace described as 'somewhat hard' to 'hard' on the Borg scale of perceived exertion (Borg, 1973).

Habitual diet influences lipaemia (Williams, 1996) and so subjects agreed to maintain their usual dietary habits over the 14-week study period. In addition, they weighed and recorded the food and drink consumed the day before their baseline fat-tolerance test and replicated this intake before each subsequent test. Subjects agreed not to consume alcohol for 24 h before a test. Practical constraints prevented control for the menstrual cycle phase of women subjects. However, the one study of which the authors are aware found no effect of cycle phase on postprandial lipaemia (Wendler *et al.* 1992).

Subjects

Seventeen young adults aged between 18 and 31 years, all of them non-smokers, volunteered to participate. They were physically active, but only at a recreational level (e.g. occasional tennis, badminton, swimming). None reported a history of cardiovascular disease, dyslipidaemia or any condition which meant that heparin administration would be contraindicated. The study was approved by the University's Ethical Advisory Committee, and after being acquainted with the risks and demands, subjects gave their written informed consent. Ten subjects (six women, four men) volunteered to follow the running training programme, while seven (four women, three men) agreed to act as controls, i.e. the groups were self-selected.

Three subjects withdrew over the course of the study: in the running group, one woman and one man sustained knee injuries; in the control group, one woman was unable to attend for post-training measurements due to illness. Some characteristics of the fourteen subjects who completed the study are shown in Table 1.

Four of the runners possessed the apolipoprotein E phenotype 3-3 and four the 4-3 phenotype. Of the controls, three possessed the 3-3 phenotype, two the 4-3 phenotype and one the 3-2 phenotype.

Oral fat tolerance test

Subjects reported to the laboratory after an overnight fast. A cannula was introduced into a forearm or antecubital vein and a blood sample was drawn. On three of the four trials, i.e. at baseline, and at 60 h and 9 d post-training, a muscle sample for determination of LPL activity was obtained under local anaesthesia (Lignocaine) by needle biopsy from the *vastus lateralis*. After the biopsy, the high-fat

Table 1. Age and some physical characteristics of subjects (*n* 14) at baseline

(Values are means and ranges)

<i>n</i> ...	Runners				Controls			
	Men 3		Women 5		Men 3		Women 3	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Age (years)	22	18–29	22	19–29	28	23–31	21	18–24
BMI (kg/m ²)	25.7	23.6–27.4	23.9	21.7–27.6	23.0	21.5–25.6	23.6	21.8–25.0
$\dot{V}O_2$ max (ml/kg per min)	58.6	52.1–65.3	47.1	41.2–54.0	56.9	56.6–57.6	40.0	29.0–46.6

$\dot{V}O_2$ max, Maximal O_2 uptake.

meal was ingested over a maximum period of 15 min. This comprised whipping cream, cereal, fruit, nuts and chocolate (1.2 g fat, 1.4 g carbohydrate, 0.2 g protein, 70.6 kJ energy/kg body mass). The meal was well-tolerated by all subjects and the time taken to consume it was not significantly different within groups (over time) or between groups for any of the four trials (data not shown). The reproducibility of the TAG response to this meal has been shown to be acceptable, with an average difference on a test–retest basis of 1.6 (95 %, CI –16 to +20) % (JMR Gill and AE Hardman, unpublished results). Further blood samples were drawn hourly during a 6 h period of observation, whilst subjects rested quietly. No food or drink, other than water, was consumed during this time. All samples were drawn with the subject seated. The cannula was kept patent by flushing with saline (9 g NaCl/l, non-heparinized).

Immediately after the 6 h blood sample, heparin was administered intravenously (100 IU/kg body mass). A further sample of blood was drawn exactly 15 min later for determination of plasma LPL activity.

Exercise tests

Endurance fitness was assessed at baseline and again during the last week of training by measuring maximal O₂ uptake ($\dot{V}O_{2\max}$) using a modification of the Taylor treadmill test (Taylor *et al.* 1955) and by determination of capillary blood lactate concentrations during treadmill running at speeds selected to elicit 60, 70, 80 and 90 % of baseline $\dot{V}O_{2\max}$. O₂ uptake and CO₂ production were measured using Douglas-bag techniques. Heart rate was monitored continuously using short-range telemetry (PE 3000 Sport-Tester; Polar Electro, Kempele, Finland).

Anthropometry

Height, body mass, skinfold thicknesses (biceps, triceps, subscapular and suprailliac) and waist and hip circumferences were measured by the same observer at baseline and again 13 weeks later.

Analysis of blood samples

Samples of capillary blood were immediately deproteinized and stored at –70° until analysed for lactate by a fluorimetric enzymic micro-method (Maughan, 1982).

Serum and plasma were separated and a portion of serum was stored at 4° until analysed for HDL-cholesterol, within 5 d. The remaining serum and plasma were divided into portions and stored at –70°. HDL-cholesterol was determined in fresh serum after precipitation of apoprotein B-containing lipoproteins with a heparin–Mn complex (Gidez *et al.* 1982). Serum total cholesterol, HDL-cholesterol, TAG, non-esterified fatty acids (NEFA) and plasma glucose concentrations were all determined photometrically by enzymic methods (Boehringer Mannheim UK Ltd., Lewes, Sussex, UK and Wako Chemicals GmbH, Neuss, Germany) on a centrifugal analyser (Cobas Bio; Roche Diagnostic Systems, Welwyn Garden City, Herts., UK). CV were 0.7, 4.0, 1.4, 1.5 and 1.5 % respectively. Values refer to intra-assay variation, except for HDL-cholesterol which is inter-assay. Serum concentrations

of LDL-cholesterol and VLDL-cholesterol in samples obtained in the fasted state were calculated using the Friedewald formula (Friedewald *et al.* 1972). Serum insulin concentrations were determined by solid-phase I¹²⁵ radioimmuno-assay (Diagnostic Products Corporation, Los Angeles, CA, USA) on an automated gamma-counter (Cobra II; Canberra Packard, Pangbourne, Berks., UK) (intra-assay CV 3.7%). Apolipoprotein E phenotypes were determined in a human genetics laboratory by isoelectric focusing using Western blot techniques. Haemoglobin concentrations and packed cell volume were measured before and after each fat-tolerance test so that changes in plasma volume could be monitored (Dill & Costill, 1974).

Analysis of all samples from the same subject was done in a single batch, with the exception of HDL-cholesterol. Accuracy and precision were monitored using quality control sera (Precinorm L; Boehringer Mannheim UK Ltd.; Control Serum N (Human), Roche Diagnostic Systems; Seronorm Lipid, Nycomed Pharma AS, Asker, Norway; CON6, Diagnostic Products Corporation).

Portions of post-heparin plasma were stored at –70° and later assayed for LPL activity by a method based on that described by Nilsson-Ehle & Schotz (1976). Enzyme activity was expressed as nmol oleate released/min per ml plasma at 37° and the intra-assay CV was 3.1 %. LPL activity in skeletal muscle could not be determined because of methodological problems.

Running training

Runners followed a progressive training programme for 13 weeks, divided into three phases: an introduction to running (4 weeks), with three 20 min steady paced runs/week; a build-up phase (4 weeks) with interval training on an athletics track, as well as long easy runs and shorter hard runs, the frequency of training gradually increasing to five sessions/week; and a maintenance phase (5 weeks). At least two training sessions/week were supervised, but all training was recorded. Performance improvements were monitored by 5 km time-trials at the end of weeks 6, 9 and 12. Heart rate was recorded by short-range telemetry (PE 3000 Sport-Tester; Polar Electro) during these trials.

Statistical analysis

Postprandial lipaemia and insulinaemia were quantified as the total areas under the serum TAG concentration and serum insulin concentration *v.* time curves respectively.

Logarithmic transformation of all data was undertaken to overcome heteroscedasticity, and statistical analyses were performed on the transformed data. The significance of differences between runners and controls at baseline was determined using unpaired *t* tests. Differences between groups in important variables were identified at baseline, so that the effects of training and detraining were evaluated on baseline-adjusted data, *i.e.* differences from baseline values using a logarithmic scale. All analyses were performed after logarithmic transformation of data, but changes with training in $\dot{V}O_{2\max}$ and in run times have been reported in absolute units for ease of interpretation.

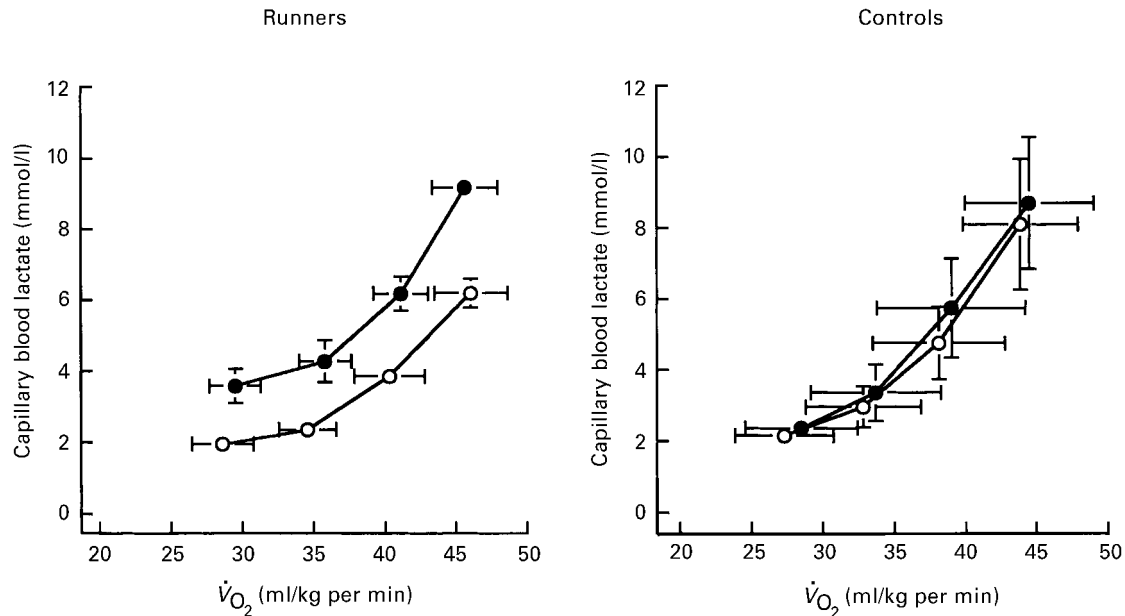


Fig. 1. Capillary blood lactate concentration during submaximal exercise at baseline (●) and after 13 weeks (○) for runners (n 8) and controls (n 6). The change from baseline was significantly different between groups for all four test stages ($P < 0.05$). Values are means with their standard errors represented by horizontal and vertical bars. $\dot{V}O_2$, submaximal oxygen uptake.

The chronic adaptive effect of training was evaluated by comparison between groups of baseline-adjusted 60 h values, using unpaired t tests. The same approach was used to evaluate the significance of differences between groups in the cardiovascular, metabolic and anthropometric variables. The time-course of the effect of a short period of detraining was examined using repeated-measures two-way ANOVA of baseline-adjusted data, at time points 15 h, 60 h and 9 d, with Scheffé *post-hoc* tests. A significance level of $P < 0.05$ was adopted throughout and results are presented as means with their standard errors, unless otherwise stated.

Results

Cardiovascular, metabolic and anthropometric changes with training

There were no differences at baseline between runners and controls. Endurance fitness improved significantly in runners, relative to controls, as evidenced by an increase in $\dot{V}O_2\text{max}$ (+3.2 (SE 1.1) ml/kg per min *v.* -1.3 (SE 1.2) ml/kg per min), and a decrease in blood lactate concentration during submaximal running (Fig. 1). The decrease in blood lactate

concentration was particularly marked, and the response over time differed significantly between groups at each of the four test stages. For runners, 5 km run time decreased from 23.6 (SE 1.3) to 21.7 (SE 1.2) min over the course of the training programme ($P < 0.05$), with no increase in heart rate at the end of the run (baseline 185 (SE 3) beats/min *v.* 183 (SE 1) beats/min after training).

Relative to controls, runners lost subcutaneous fat and showed a decrease in waist:hip circumference (both $P < 0.05$) and possibly a decrease in body mass ($P = 0.06$; Table 2).

Changes in serum variables with training and detraining

No systematic changes in plasma volume were discerned, either over the course of the study or during oral fat-tolerance tests, so that no adjustments were made in this regard. Postprandial lipaemia and serum insulin concentrations in the fasted state were both higher ($P < 0.05$) at baseline in runners and the difference between groups in fasting plasma TAG concentration approached significance ($P = 0.06$). Consequently, all between-group comparisons were baseline-adjusted (see p. 59).

Table 2. Indices of amount and distribution of body fat in runners and controls (Values are means with their standard errors)

	n	Body mass (kg)				Sum of skinfolds (mm)				Waist:hip circumference			
		Baseline		13 weeks		Baseline		13 weeks		Baseline		13 weeks	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Runners	8	71.1	3.4	69.8	3.6	49.4	6.3	41.8*	4.5	0.81	0.01	0.79*	0.01
Controls	6	66.7	5.0	66.6	5.1	51.8	10.0	49.9	9.7	0.80	0.02	0.80	0.02

The differences from mean values at baseline were significantly different from the corresponding control values: * $P < 0.05$.

Table 3. Serum concentrations of triacylglycerol, total and HDL-cholesterol, non-esterified fatty acids, insulin and glucose measured in the fasted state

(Values are means with their standard errors)

		Runners (n 8)		Controls (n 6)	
		Mean	SE	Mean	SE
Triacylglycerol (mmol/l)	Baseline	1.15	0.19	0.70	0.08
	15 h	0.72	0.09	0.71	0.08
	60 h	0.92	0.09	0.62	0.12
	9 d	1.03	0.15	0.79	0.10
Total cholesterol (mmol/l)	Baseline	4.81	0.35	4.17	0.23
	15 h	4.46†	0.20	4.54	0.22
	60 h	4.36†‡	0.26	4.42	0.30
	9 d	4.81	0.29	4.43	0.24
HDL-cholesterol (mmol/l)	Baseline	1.68	0.10	1.73	0.06
	15 h	1.58	0.13	1.66	0.07
	60 h	1.49	0.13	1.59	0.06
	9 h	1.48	0.11	1.54	0.06
Non-esterified fatty acids (μmol/l)	Baseline	714	154	834	131
	15 h	743	133	579	117
	60 h	514	87	776	115
	9 d	524	103	578	131
Insulin (μIU/ml)	Baseline	9.55*	1.65	5.70	0.73
	15 h	7.12	1.38	6.46	1.20
	60 h	8.61	1.40	4.24	0.48
	9 d	7.89	0.84	4.25	0.31
Glucose (mmol/l)	Baseline	4.40	0.10	4.58	0.06
	15 h	4.40	0.40	4.58	0.13
	60 h	4.43	0.25	4.40	0.28
	9 d	4.46	0.14	4.64	0.13

Mean values were significantly different from controls at baseline: * $P < 0.05$.
 The difference from the mean value at baseline was significantly different from that for the controls: † $P < 0.05$.
 The difference from the mean value at 9 d was significantly different from the controls: ‡ $P < 0.05$.

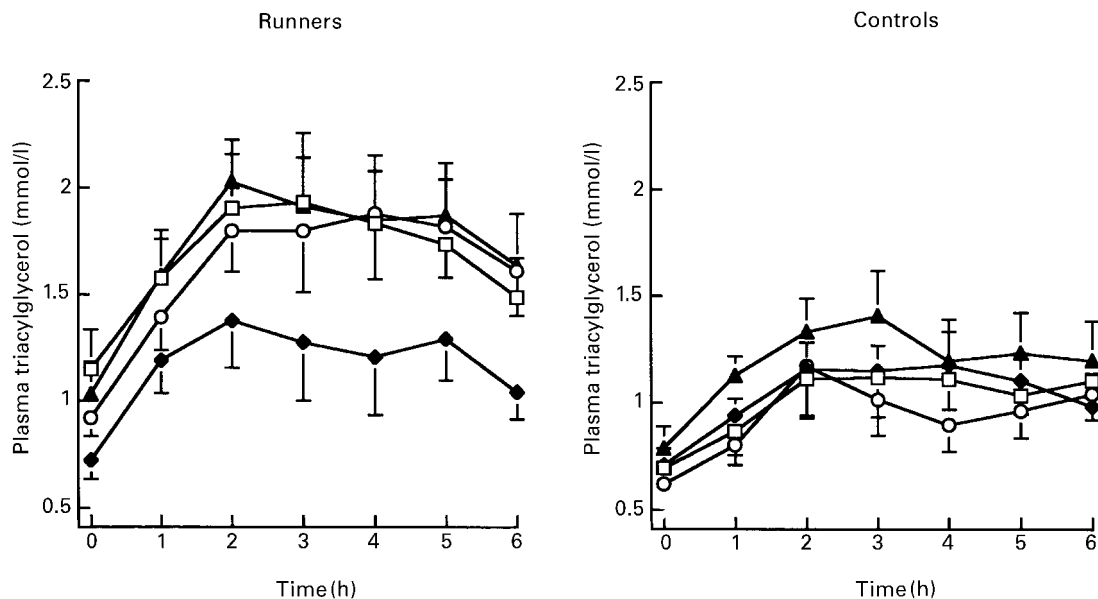


Fig. 2. Serum triacylglycerol concentrations in the fasted and postprandial states at baseline (□) and at 15 h (◆), 60 h (○) and 9 d (▲) after the last training session for runners (n 8) and controls (n 6). Values are means with their standard errors represented by vertical bars.

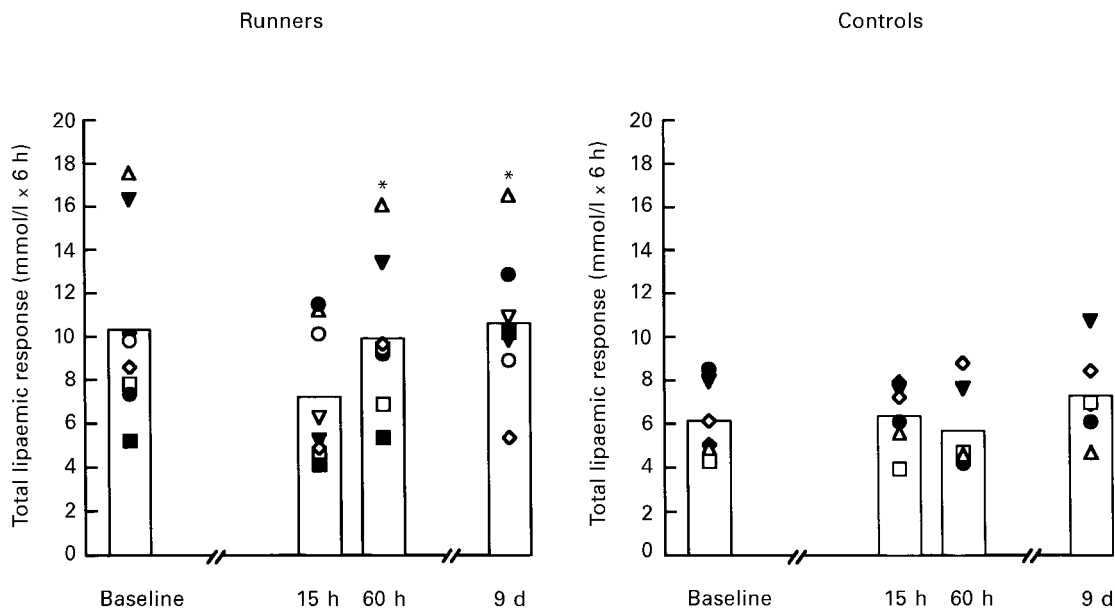


Fig. 3. Individual and mean (\square) values for total lipaemic response (area under the plasma triacylglycerol concentration v. time curve) at baseline and at 15 h, 60 h and 9 d after the last training session for runners (n 8) and controls (n 6). ($\blacktriangledown, \bullet, \blacksquare, \blacklozenge$), Men; ($\triangle, \circ, \square, \diamond$), women. The difference between values at 15 and 60 h and those at 15 h and 9 h for runners was significantly different from the corresponding values for the control group, i.e. group \times time interaction was significant (ANOVA): * $P < 0.05$.

Table 3 presents some of the serum constituents measured in the fasted state. There was considerable inter-individual variation in the lipaemic response, particularly in runners (Fig. 2). Individual values are shown for this response (Fig. 3) and also for LPL activity and the serum insulin response (Figs. 4 and 5). Concentrations of total cholesterol exhibited little fluctuation during the postprandial period so the mean concentration over 6 h was employed as a summary measure (runners 4.77, 4.29, 4.39 and 4.80 mmol/l, controls 4.17, 4.36, 4.33, 4.35 mmol/l at baseline, 15 h, 60 h and 9 d respectively).

The chronic effects of training: comparison of baseline values with those obtained 60 h after the runners' last training session. Fasting and mean postprandial concentrations of total-cholesterol fell with training (by 9 and 7 % respectively), but increased somewhat in controls (difference between groups in response over time $P < 0.05$). However, there were no significant differences between groups for concentrations of TAG, insulin, HDL-cholesterol, NEFA and glucose in either the fasted or postprandial states. Post-heparin plasma LPL activity did not change in a different way in runners and controls.

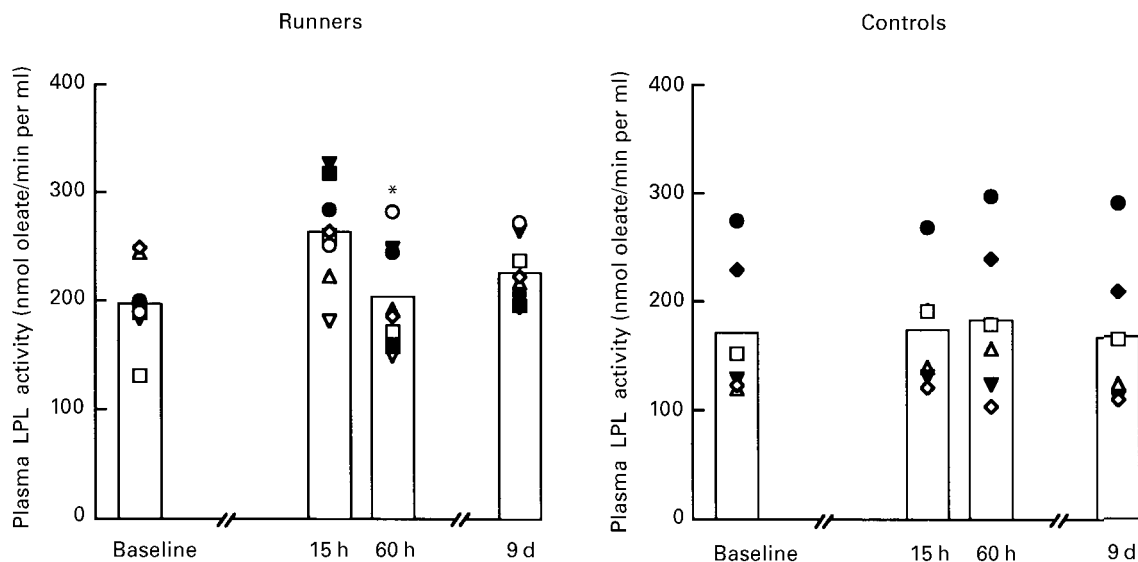


Fig. 4. Individual and mean (\square) values for post-heparin plasma lipoprotein lipase (*EC* 3.1.1.34; LPL) activity at baseline and at 15 h, 60 h and 9 d after the last training session for runners (n 8) and controls (n 6). ($\blacktriangledown, \bullet, \blacksquare, \blacklozenge$), Men; ($\triangle, \circ, \square, \diamond$), women. The difference between values at 15 and 60 h was significantly different from the corresponding values for the control group, i.e. group \times time interaction was significant: * $P < 0.05$.

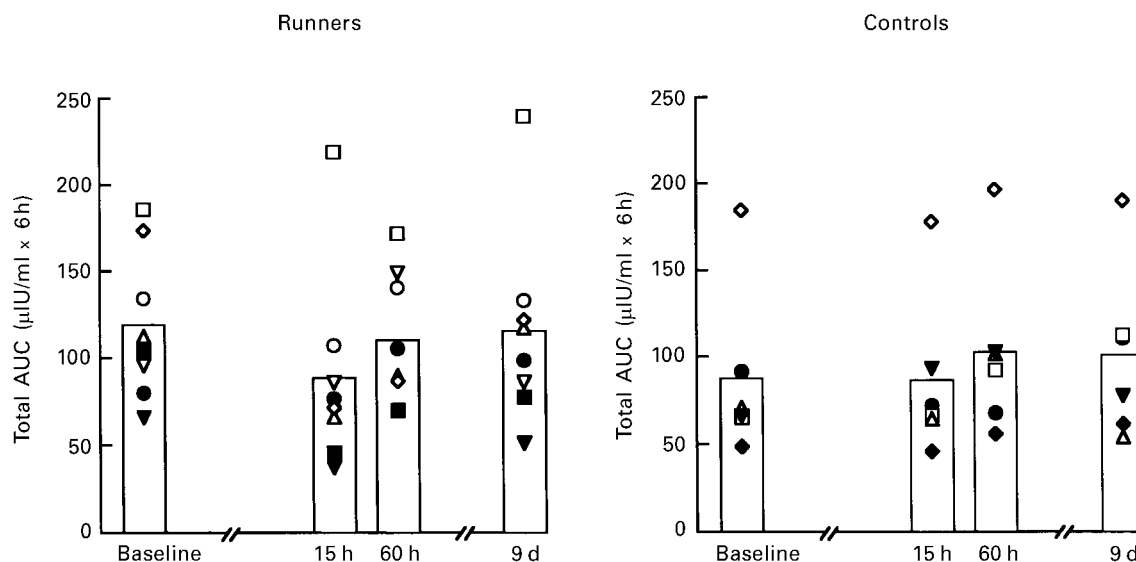


Fig. 5. Individual and mean (\square) values for total area under the serum insulin concentration v. time curves (AUC) at baseline and at 15 h, 60 h and 9 d after the last training session for runners (n 8) and controls (n 6). ($\blacktriangledown, \bullet, \blacksquare, \blacklozenge$), Men; ($\blacktriangledown, \triangle, \circ, \square, \diamond$), women.

The time-course of changes during detraining: comparison of values obtained 15 h, 60 h and 9 d after the last training session. In the fasted state there were no significant differences between runners and controls in changes over time for concentrations of HDL-cholesterol or glucose. The change in TAG approached significance ($P = 0.08$). Fasting concentrations of insulin and NEFA changed in a different way between groups during detraining (group \times time interaction $P < 0.05$).

For both fasting and mean postprandial total-cholesterol concentration, the change between 15 h and 9 d post-exercise differed between runners and controls, as did the change between 60 h and 9 d (both $P < 0.05$).

For runners, postprandial lipaemia was 37 % higher at 60 h than at 15 h and 46 % higher at 9 d than at 15 h; these changes were significantly different from those in controls (Fig. 3).

Changes with detraining in post-heparin plasma LPL activity are shown in Fig. 4. Between 15 h and 60 h LPL activity decreased markedly in runners, with little change in controls (difference in response $P = 0.01$).

There were no significant differences between groups in the changes over time for the insulinaemic response (Fig. 5), for mean postprandial HDL-cholesterol concentration or for postprandial concentrations of glucose or NEFA (data not shown).

Discussion

The present study examined the effects on postprandial lipaemia of 13 weeks of endurance running training followed by 9 d of detraining in normolipidaemic young adults. Its unique design allowed the lipaemic response in the untrained state to be compared with that determined in the trained state, both in the presence and in the absence of the effect of a single session of exercise.

Runners exhibited the classic adaptations to endurance training, i.e. increased $\dot{V}O_2$ max and reduced heart rate and blood lactate responses to submaximal exercise, confirming

their improved fitness and compliance with the training regimen. The 5 km run times fell by an average of 2 min, a considerable improvement in running performance for individuals who, by population standards, were of above average fitness level at baseline (Allied Dunbar National Fitness Survey, 1992). None of these changes was evident in controls, who remained untrained.

Our main findings were: (1) 13 weeks of running training did not influence postprandial lipaemia when this was determined in the absence of the acute effect of a training session, i.e. 60 h later; (2) postprandial lipaemia increased rapidly when the training stimulus was withdrawn; compared with 15 h values, lipaemia was 37 % higher after 60 h without exercise and 46 % higher after 9 d without exercise (Fig. 3). The only clear difference in the response over time between runners and controls was between 15 and 60 h, suggesting that the increase in lipaemia in runners over 9 d was little more than the removal of the acute effects of the last training session. The low level of lipaemia observed the morning after the runners' last training session would have been an acute effect of that exercise. In our earlier studies, with men and women of varying habitual physical activity levels, we have demonstrated that a session of exercise taken on the previous afternoon reduces the lipaemic response to a high-fat meal consumed the following morning by between 16 and 33 % (Aldred *et al.* 1994; Tsetsonis & Hardman, 1996a,b; Tsetsonis *et al.* 1997). The present findings emphasize the important contribution of the acute effect of the last exercise session to low levels of postprandial lipaemia in endurance-trained individuals.

Inter-individual variation in postprandial lipaemia, a characteristic of this response (Cohn *et al.* 1988), was considerable (fourfold across runners and controls). The determinants of this variation are, however, unknown but were not obviously sex-linked (Fig. 3), nor was there a simple relationship with genetic variation in apolipoprotein E, which plays an important role in receptor-mediated clearance of lipoprotein particles from plasma. For example,

neither of the two subjects with a particularly high lipaemic response (Fig. 3) possessed the E2 allele, which binds poorly and delays the clearance of dietary fat (Weintraub *et al.* 1987).

Despite the high intensity of the training programme followed and marked improvements in fitness, we found no evidence of an effect on lipaemia above that attributable to the effects of a recent session of exercise. This contrasts with several reports of training-induced improvements in indices of lipaemia based on measurements made at intervals after the last training session of 3 d, 48–72 h and 36 h respectively (Altekruse & Wilmore, 1973; Wirth *et al.* 1985; Weintraub *et al.* 1989). These workers report improvements in postprandial lipaemia determined by a variety of methodologies (plasma TAG response to a high-fat meal, intravenous clearance rate of a fat emulsion, and vitamin A fat-loading test respectively). In all these studies, however, subjects were sedentary at outset and might therefore have undergone more profound increases in physical activity levels than our runners; in the six subjects studied by Weintraub *et al.* (1989), for example, $\dot{V}O_2$ max increased by 43% with only 7 weeks of training, a level of improvement seldom matched in the literature. In another study, where an increase with training of 24% in the disappearance rate of TAG was reported (Thompson *et al.* 1988), measurements were made when subjects 'had not exercised during the preceding 10 h' and were surely influenced by the residual effects of a training session. Our study, with its thorough examination of the early detraining period, provides new evidence of the relative importance of short- and longer-term effects of exercise in men and women who are physically active but who further improve their fitness level.

The present findings are entirely consistent with an earlier study from our laboratory in sedentary low-fitness, middle-aged women (Aldred *et al.* 1995). In that study, training by brisk walking clearly improved fitness but did not alter postprandial lipaemia determined 2 d after the last training session. One interpretation of those data was that the intensity of the exercise (approximately 60% $\dot{V}O_2$ max) was not sufficient to influence lipaemia. The present findings, where the intensity of training was consistently higher, provide no support for this view. They are concordant, however, with those of Mankowitz *et al.* (1992) who studied seven runners in the trained state (≥ 20 h after exercise) and again after 14–22 d of detraining. These workers found a 'dramatic' decrease in chylomicron and chylomicron remnant metabolism (assessed by retinyl palmitate levels) after 2–3 weeks without exercise. They presented no data, however, to describe the early days of the detraining period.

Hydrolysis at the endothelial surface of capillaries by LPL is thought to be the rate-limiting step in TAG clearance (Eckel, 1989), and changes in the activity of the enzyme with changes in exercise or training status are a likely explanation for our findings. Post-heparin plasma LPL activity was highest when determined just 15 h after the last training session, falling by an average of 23% during the first 2 d without exercise. Thus, we observed rapid and reciprocal changes in postprandial lipaemia, the response to our functional challenge, and post-heparin LPL activity as soon as the training stimulus was withdrawn. By the time

runners had been 60 h without exercise, LPL activity differed little from the value determined at baseline. There were no such changes over this interval in controls, who did not exercise at all during the 2 d preceding any fat-tolerance test. This finding is consistent with previous studies where the rate of LPL activity has been reported to relate to the rate of TAG clearance (Terjung *et al.* 1983) and where increases in LPL activity have persisted for up to 18 h after a session of exercise (Kantor *et al.* 1984).

As we were unable to obtain data for LPL activity in skeletal muscle, we cannot identify the site of the changes in LPL activity. However, muscle has been reported to be an important site of TAG uptake, clearing 50% of a dose of intravenous fat emulsion compared with 13% by adipose tissue (Rossner, 1974). Training-induced increases in muscle LPL activity have been coupled with enhanced capillary density (Svedenhag *et al.* 1983; Kiens & Lithell, 1989), but this is probably not the explanation in the present study, because the higher LPL activity observed immediately after training ceased was not sustained. Our runners trained for 13 weeks, compared with 8 weeks in the studies of both Svedenhag *et al.* (1983) and Kiens & Lithell (1989), at similar relative intensities. Thus, the length of the training period *per se* would not preclude a stimulus to improved capillary density. One might speculate that the exercise models employed by these workers, two-leg cycling and one-leg knee extensions respectively, were more powerful stimuli to structural changes in the microcirculation of muscle than running training.

With our model, the dominant effect of the exercise programme may have been to increase LPL activity rather than to confer microcirculatory changes which provide more sites of enzyme action. Marked increases in LPL mRNA level and LPL protein mass have been reported in sedentary men after a brief period (5–13 d) of daily exercise at relative intensities similar to those we employed (Seip *et al.* 1995). Changes in the opposite direction have been reported after a 2-week period of detraining in trained men (Simsolo *et al.* 1993), a period too short to result in reversal of structural microcirculatory changes.

In our study the difference between LPL activity measured at baseline and 60 h post-training, on average an increase of 6% but with considerable inter-individual variation, provides no evidence of a chronic effect of training. This contrasts with the findings of Weintraub *et al.* (1989) who reported a significant (16%) increase in plasma LPL activity following a 7-week programme of jogging. Post-training measurements were made 36 h after the last training session in the latter study, however, so the last session of exercise may have made a more important contribution to the post-training levels than in our data.

Insulin plays a pivotal role in the regulation of postprandial lipaemia, through its effect on LPL activity (Fraysn, 1993), but an altered insulinaemic response to the test-meal does not appear to have been an important mediator of the change in lipaemia during the early phase of detraining.

Plasma total cholesterol changes differed between groups (Table 3), with a progressive increase in both fasting and postprandial levels in runners when training ceased. These changes followed a time-course which was similar to the changes in lipaemia. Most of the cholesterol is carried in

LDL, the remnant particles from VLDL, and changes in their removal rate as the exercise stimulus was withdrawn could conceivably be responsible. We found no change in concentrations of HDL-cholesterol with either training or detraining. This is unexpected, given the wealth of literature describing increases in HDL-cholesterol following exercise training (Durstine & Haskell, 1994), and especially given the changes in postprandial lipaemia which presumably reflect enhanced metabolism of TAG-rich lipoproteins. However, although some previous studies have found a positive relationship between high concentrations of HDL-cholesterol in the fasted state and the ability to clear dietary fat (Sauar *et al.* 1980; Sady *et al.* 1988) others have not (Weintraub *et al.* 1989; Cohen *et al.* 1991).

Repeated episodes of exaggerated alimentary lipaemia are linked to multiple disturbances of lipoprotein metabolism, which are, in turn, related to premature atherosclerosis (Karpe & Hamsten, 1995). Our findings confirm that a session of exercise decreases postprandial lipaemia but, despite the variability in individual response, provide the strongest evidence to date that a short period of training confers no synergistic benefit. The importance of the repeated effects of frequent exercise sessions as determinants of the health benefits of exercise is embodied in recent recommendations on physical activity and public health (Pate *et al.* 1995). Our data confirm the existence of one particular effect, and emphasize the need for frequent, regular exercise if benefits in metabolic risk factors for cardiovascular disease are to be sustained.

Acknowledgements

This research was supported by the British Heart Foundation. The authors wish to thank Dr A. M. Nevill (Liverpool John Moores University, Liverpool, UK) for statistical advice, Dr S. J. Mastana and Mrs A. Pacynko (Department of Human Sciences, Loughborough University, Loughborough, UK) for phenotyping of apolipoprotein E, Dr N. N. Vaghela and Professor R. Goldsmith (both Loughborough University) for clinical assistance and Dr J. E. M. Knapper of Surrey University, Guildford, Surrey, UK for advice in setting up the assay for lipoprotein lipase activity.

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