A single session of resistance exercise does not reduce postprandial lipaemia

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ABSTRACT

This study investigated the effect of a single session of resistance exercise on postprandial lipaemia. Eleven healthy, normolipidaemic men aged 23 (SE 1.4) years performed two trials at least one-week apart in a counterbalanced randomized design. In each trial participants consumed a test meal (1.2g fat, 1.1g carbohydrate, 0.2 g protein and 68 kJ per kg body mass) between 08.00 and 09.00 following a 12 hour fast. The afternoon before one trial participants performed an 88 minute bout of resistance exercise. Prior to the other trial participants were inactive (control trial). Resistance exercise was performed using free weights and included 4 sets of 10-repetitions of each of 11 exercises. Sets were performed at 80% of 10 repetition maximum with a 2 minute work and rest interval. Venous blood samples were obtained in the fasted state and at intervals for 6 h postprandially. Fasting plasma triacylglycerol (TAG) concentration did not differ significantly between control and exercise trials (mean ± SE: 1.03 ± 0.13 $mmol \cdot L^{-1}$ versus 0.94 ± 0.09 $mmol \cdot L^{-1}$; respectively). Similarly the 6 h total area under the plasma TAG concentration versus time curve did not differ significantly between control and exercise trials (9.84 ± 1.40 $mmol \cdot L^{-1} \cdot 6$ h versus 9.38 ± 1.12 $mmol \cdot L^{-1} \cdot 6$ h; respectively). These findings suggest that a single session of resistance exercise does not reduce postprandial lipaemia.

INTRODUCTION

Elevated postprandial triacylglycerol (TAG) concentrations are independently associated with coronary artery disease (Patsch *et al.*, 1992). Several mechanisms have been proposed to explain this association. Increased postprandial TAG concentrations may promote atherosclerosis due to multiple disturbances in lipoprotein metabolism which encourage: a) an accumulation of triglyceride rich lipoprotein remnants in the plasma; b) catabolism of high density lipoproteins; c) formation of small, dense low density lipoproteins which have an increased susceptibility to oxidation (Cohn, 1998; Karpe and Hamsten, 1995). Interventions that reduce postprandial TAG concentrations may therefore reduce the development of atherosclerosis.

Many studies have shown that a single bout of aerobic exercise reduces postprandial lipaemia (Aldred *et al.*, 1994; Tsetsonis and Hardman, 1996a;Tsetsonis and Hardman, 1996b; Tsetsonis *et al.*, 1997; Gill *et al.*, 2003). This reduction appears to be transient, however, since detraining leads to a prompt elevation in postprandial lipaemia (Hardman *et al.*, 1998; Herd *et al.*, 1998). Thus, exercise must be performed frequently for continued benefit. Two mechanisms have been proposed to explain the TAG-lowering effects of aerobic exercise. One is an increase in lipoprotein lipase (LPL) activity which may enhance the uptake of TAG into previously exercised muscle (Seip and Semenkovich, 1998). The other is a reduced rate of secretion of hepatic very low density lipoproteins (Gill *et al.*, 2001; Malkova *et al.*, 2000). Furthermore, energy expenditure during exercise appears to be the

primary determinant of the exercise-induced reduction in postprandial lipaemia (Gill *et al.*, 2002; Petitt and Cureton, 2003; Tsetsonis and Hardman, 1996b).

Although many studies have addressed the effects of aerobic exercise on postprandial lipaemia, to our knowledge only one study has examined the influence of resistance exercise on postprandial lipaemia (Petitt *et al.*, 2003). This study found that postprandial lipaemia was decreased following resistance exercise in comparison to a control trial. This is surprising because the estimated energy expenditure during the resistance exercise was only 1.7 MJ. This is at the lower end of the range of values reported in studies of aerobic exercise and postprandial lipaemia (1.5 to 7.2 MJ) (Petitt and Cureton, 2003).

Given that most longitudinal studies have not found changes in TAG concentration following resistance training (Durstine and Haskell, 1994), this recent finding of lowered postprandial lipaemia following a single session of resistance exercise requires confirmation. Therefore, the present study sought to examine the influence of resistance exercise on postprandial lipaemia in a group of young adult males. Specifically, we sought to evaluate the hypothesis that a single session of resistance exercise would lower postprandial lipaemia providing that the energy expenditure was sufficient.

METHODS

Participants.

Eleven male volunteers aged 18-40 participated in the study which was approved by the University's Ethical Advisory Committee. The participants gave written informed consent after receiving an explanation of the procedures and risks involved. Participants were recruited only if they met the following criteria: non-smoking, no known history of cardiovascular disease, resting arterial blood pressure <160/95 mm Hg, not taking any medication known to affect lipid or carbohydrate metabolism. Some physical characteristics of the participants are shown in Table 1. Most participants reported that they were involved in some form of recreational physical activity but none of them performed resistance exercise on a regular basis.

TABLE 1 NEAR HERE

Study design.

Prior to the main trials participants visited the laboratory twice. During the first visit anthropometric data were collected and participants performed 10-repetition maximum tests for each of the 11 resistance exercises employed in the study. The 10-repetition maximum values were determined by trial and error by adding/removing weights after each attempt as required (this applied to all exercises including sit ups). Participants were allowed to take as long as they felt necessary to recover from each attempt. The order in which the 10-repetition maximum tests were performed was the same for each participant i.e. dead lift, bench press, upright row, squat, shoulder press, bent-over row, lunges, barbell pullover, bicep curls,

triceps press and sit ups. On the second visit participants completed a practice weight-lifting session. The purpose of this practice session was to ensure that each participant was able to complete the entire exercise session and also to confirm that the weights lifted were producing fatigue from overload by the end of the session. This was confirmed by visual inspection and by verbal feedback from participants.

Following the preliminary visits participants undertook two fat tolerance tests. The first of these tests was performed a minimum of one-week after the practice weight lifting-session. The interval between the two fat tolerance tests was at least one-week. The afternoon prior to one of the fat tolerance tests participants performed an 88 minute bout of resistance exercise starting at approximately 16.00 h (exercise trial). The afternoon prior to the other fat tolerance test participants rested (control trial). Trials were performed in a counterbalanced randomized design.

For two days preceding the main trials participants were asked to refrain from physical activity other than the resistance exercise. Only gentle walking for personal transportation over short distances was permitted. Participants weighed and recorded all food and drink consumed during the 48 h immediately preceding their first trial and they undertook to replicate this intake during the 48 h prior to their second trial. Participants also refrained from alcohol during these periods.

Anthropometry.

Height and weight were determined using standard methods. Skinfold thicknesses were measured at three sites (chest, triceps, subscapular) using calipers. Body density was calculated using a three sites formula as outlined by Jackson and Pollock and body fat percentage then estimated using the Siri equation (Jackson and Pollock, 1985).

Weight lifting protocol.

Each participant performed four sets of 10 repetitions of 11 different weight-lifting exercises at 80% of 10 repetition maximum. Four sets of each exercise were employed in order to maximise the total energy expenditure of the session and hence the likelihood of reducing postprandial lipaemia. A training intensity of 80% of 10 repetition maximum was chosen as a compromise between muscular strength and endurance. Pilot work revealed that a higher training intensity would have prevented most participants from completing four sets of each exercise and thus would have lowered the total energy expenditure. Participants were given 2 min in which to complete each set. On completion of the 10 repetitions participants rested for the remainder of the 2 min. Therefore, the whole exercise session lasted 88 min (11 exercises × 4 sets × 2 min). The order of the exercises was uniform for all participants: dead lift, bench press, upright row, squat, shoulder press, bent-over row, lunges, barbell pullover, bicep curls, triceps press and sit ups. All sets for one exercise were completed before moving on to the next exercise. The session was structured to avoid local muscular fatigue from exercising similar muscle groups

131 consecutively. It was also designed so that large muscle groups were exercised first.
132 Standard free weights were used, including dumbbells, bent bar, an Olympic bar and
133 a mobile bench. Water intake was permitted ad libitum throughout the resistance
134 exercise session.
135
136 *Estimation of energy expenditure during weight lifting.*
137 Expired air samples were collected into Douglas bags (Plysu Protection Systems,
138 Milton Keynes, U.K.) during the third set of each weight lifting exercise (pilot work
139 revealed that there was no systematic variation in oxygen consumption values
140 measured during the first, second, third and fourth set of each exercise). Samples
141 were collected for the full duration of the lifting and recovery period i.e. 2 min.
142 Oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were
143 determined from expired air samples using a paramagnetic oxygen analyzer and an
144 infra-red carbon dioxide analyzer respectively (Series 1400; Servomex,
145 Crowborough, East Sussex, U.K.). Expired air volumes were measured using a dry
146 gas meter (Harvard Apparatus, Edenbridge, Kent, U.K.) and corrected to standard
147 temperature and pressure (dry).
148
149 The short duration, intermittent nature of weight lifting invalidates the typical
150 assumptions of indirect calorimetry because the respiratory exchange ratio (RER) is
151 consistently equal to or greater than 1.0. Therefore, energy expenditure was
152 calculated as being 5.047 kcal (21.1 kJ) per litre of oxygen (McArdle *et al.*, 2001).
153 This reflects the assumption that energy was derived from carbohydrate rather than

fat oxidation and assumes no protein contribution to energy provision during the exercise. This assumption may not be entirely valid, in which case our energy expenditure estimations may be slightly on the high side. No attempt was made to quantify the energy contribution from anaerobic sources.

Fat tolerance tests.

The protocol for the fat tolerance tests is shown in Figure 1. Participants reported to the laboratory at 08.00 h after a 12-h fast. A cannula was inserted into a forearm or antecubital vein and the participant rested quietly for 10 min before a baseline blood sample was obtained. The participant then lay quietly whilst resting metabolic rate (RMR) and RER were measured. The test meal was then consumed. This comprised whipping cream, fruit, cereal, nuts, chocolate and sugar and was given according to body mass (1.2 g fat, 1.1 g carbohydrate, 0.2 g protein, 68 kJ, 0.09 g fiber, per kg body mass). Further blood samples were obtained at 0.5, 0.75 and 1 h after the start of the meal and then hourly for a total of 6 h. The cannula was kept patent by flushing with nonheparinised saline (9 $g \cdot L^{-1}$, B.Braun Medical Ltd, Buckinghamshire, UK). The first 2 mL of blood withdrawn was always discarded to avoid dilution of the sample. Every participant consumed all of the prescribed meal. Only water was consumed during the 6-h postprandial observation period. Water was available ad libitum during the first trial; the volume ingested was recorded and replicated in the second trial. Participants rested (reading, working quietly, watching television) throughout each observation period and were always lying in a supine position for at least 5 min before blood samples were taken.

178 FIGURE 1 NEAR HERE

179

180 *Energy expenditure at rest and during the postprandial period.*

181 Resting metabolic rate and RER were measured using a ventilated hood attached to
182 an automated metabolic cart (GEM Europa Scientific, NutrEn Technology Ltd.,
183 Manchester, U.K.). Participants were asked not to shower on the morning of the tests
184 and to travel to the laboratory by car. This was to ensure that they were in a rested
185 state. On arrival at the laboratory they were comfortably positioned on a bed and
186 RMR was measured after placement of a cannula and withdrawal of a baseline blood
187 sample. Participants spent 15 min under the hood. The first 5 min served as a
188 habituation and calibration period. $\dot{V}O_2$ and $\dot{V}CO_2$ values were calculated every 30
189 seconds for the remaining 10 min. The mean values for $\dot{V}O_2$ and $\dot{V}CO_2$ were used
190 to estimate the amount of fat and carbohydrate oxidized during the measurement
191 period assuming no protein oxidation (Frayn, 1983). Postprandial metabolic rate was
192 measured at 2.5 and 5.5 hours following food consumption using the same procedure
193 employed to measure RMR.

194

195 *Analytical Methods.*

196 At each sampling point, blood samples were collected into pre-cooled 9-mL
197 potassium-EDTA monovettes (Sarstedt, Leicester, UK) and were kept on ice until
198 centrifugation. Plasma was separated within 15 min of collection, divided into
199 aliquots, and stored at -20°C. Plasma samples were analysed (within six months of
200 collection) for TAG, glucose, cholesterol, HDL cholesterol (Randox Laboratories

Ltd. U.K.) and non-esterified fatty acids (NEFA) (Wako Chemicals GmbH, Germany) by enzymatic, colorimetric methods with the use of a centrifugal analyzer (Cobas-Bio; Roche, Basel, Switzerland). Plasma insulin concentration was determined using a solid-phase ^{125}I radioimmunoassay available in a commercial kit (ICN Pharamaceuticals, Inc., Costa Mesa, CA). The within batch coefficients of variation for these assays were as follows: TAG 1.8%, glucose, 1.3%, cholesterol 1.0%, HDL cholesterol 1.4%, NEFA 0.8%, insulin 5.7%. To eliminate inter-assay variation, samples from both trials for each participant were always analysed in the same batch. Haemoglobin concentration and haematocrit were determined in samples collected at baseline and 6-h so that changes in plasma volume could be estimated (Dill and Costill, 1974).

Data Analysis.

Postprandial responses for TAG, NEFA, glucose and insulin were calculated using the six hour areas under the plasma concentration versus time curves using the trapezium rule. Incremental area under curve values were calculated using the same method after correcting for baseline concentrations. Insulin sensitivity was calculated as the ratio of the total area under the insulin concentration versus time curve, to the total area under the glucose concentration versus time curve (Lamarche *et al.*, 1993). Fasting and area under the curve values were compared between trials using Student's t-tests for correlated means. Two-way ANOVA (repeated measures) was used to determine differences between trials and over time for fasting and postprandial plasma concentrations of TAG, NEFA, glucose and insulin as well as

224 for RER, energy expenditure, carbohydrate and fat oxidation. When there was a
225 main effect of time, differences between individual means were assessed using
226 Student's t-tests with a Bonferroni adjustment. Relationships between variables were
227 evaluated using Pearson's product-moment correlation coefficient. A 5% level of
228 significance was adopted throughout, and data are expressed as means ± SE.
229

RESULTS

Responses during weight lifting.

The mean weight lifted during the 88 minute resistance exercise session was 14,214 ± 613 kg. Mean $\dot{V}O_2$ was 1.22 ± 0.03 L· min^{-1} with a mean RER value of 1.05 ± 0.02 over this period. The gross energy expenditure from the exercise was estimated to be 2.3 ± 0.3 MJ and the net (gross minus resting) energy expenditure was estimated at 1.8 ± 0.06 MJ.

Plasma concentrations in the fasted state.

Plasma concentrations in the fasted state are shown in Table 2. No significant differences were seen in fasting plasma TAG, NEFA, total cholesterol, glucose or insulin concentrations between control and exercise trials, although plasma insulin showed a tendency to be higher in the control trial ($P < 0.10$). Plasma HDL-C concentrations were lower in the exercise trial than the control trial ($P < 0.05$).

TABLE 2 NEAR HERE

Postprandial plasma responses to the fat tolerance tests.

Changes in plasma volume over the period of observation were small and did not differ significantly between control (0.2 ± 2.7 %) and exercise (–2.5 ± 2.0 %) trials. No adjustments were made, therefore, to measured concentrations of plasma constituents.

Total and incremental plasma TAG concentrations following the fat tolerance tests are shown in Figure 2, with summary measures (areas under the curve) of these responses in Table 3. No difference was seen in either the total or the incremental area under the curve values between the control and exercise conditions. No difference was observed in the peak TAG concentration (control 2.15 ± 0.26 $mmol \cdot L^{-1}$, exercise 2.16 ± 0.26 $mmol \cdot L^{-1}$) or time-to-peak TAG concentration (control 3.45 ± 0.43 h, exercise 3.45 ± 0.49 h). Positive relationships were seen in both trials between the total area under the curve and the fasting TAG concentrations (control $r = 0.95$, $P<0.01$; exercise $r = 0.93$, $P<0.01$).

FIGURE 2 NEAR HERE

TABLE 3 NEAR HERE

Postprandial plasma NEFA, glucose and insulin responses are shown in Figure 3 and area under the curve values are presented in Table 3. There were no significant differences in area under the curve values between trials for any of these variables. Neither were there any significant differences between trials for peak insulin concentration (control 119.3 ± 15.4 $\mu U \cdot mL^{-1}$, exercise 109.8 ± 29.1 $\mu U \cdot mL^{-1}$), time-to-peak insulin concentration (control 0.64 ± 0.05 h, exercise 0.70 ± 0.07 h) or insulin sensitivity (control 9.42 ± 1.18, exercise 8.59 ± 1.45).

FIGURE 3 NEAR HERE

Substrate utilization and energy expenditure.

The test meal provided 104.0 ± 3.3 g fat, 94.3 ± 3.0 g carbohydrate, 16.2 ± 0.5 g protein and 5.68 ± 0.2 MJ energy, 69% of which was derived from fat. The time taken to consume the test meal did not differ significantly between the two trials (control 15.8 ± 1.6 min, exercise 14.1 ± 1.5 min).

Fasting and postprandial values for RER, energy expenditure, carbohydrate and fat oxidation are presented in Table 4. No interaction effects (trial x time) were found for any of these variables. A main effect of trial was found for RER indicating lower values in the exercise compared to the control trial. A main effect of time was seen for energy expenditure indicating a higher rate of expenditure at 2.5 hours compared to fasting and 5.5 hour values. A main effect of time was also observed for the rate of fat oxidation which was elevated at 2.5 and 5.5 hours compared to fasting values.

TABLE 4 NEAR HERE

DISCUSSION

The main finding in the present study is that a single session of resistance exercise, performed 16 hours prior to an oral fat tolerance test, did not influence postprandial lipaemia. Whatever way the postprandial TAG response was compared (peak TAG concentration, time to peak TAG concentration, total area under the TAG concentration versus time curve, incremental area under the TAG concentration versus time curve) there was no significant difference between trials. This is in contrast to the findings from studies of aerobic exercise, most of which demonstrate a lowering of postprandial lipaemia the day after a single session of exercise (Aldred *et al.*, 1994; Tsetsonis and Hardman, 1996a; Tsetsonis and Hardman, 1996b; Tsetsonis *et al.*, 1997; Malkova *et al.*, 2000; Herd *et al.*, 2001; Gill *et al.*, 2002; Gill *et al.*, 2003). The findings of the present study are also in conflict with those of Petitt and colleagues (2003) who found a significant reduction in postprandial lipaemia 16 hours after a bout of resistance exercise.

A possible explanation for the failure of resistance exercise to influence postprandial lipaemia in the present study is insufficient energy expenditure. The estimated gross energy expended during the resistance exercise in our study was 2.3 MJ (range = 2.0-2.6 MJ). In previous studies examining the acute effects of aerobic exercise on postprandial lipaemia, the average gross energy expenditure found to attenuate the lipemic response has ranged from 1.5 to 7.2 MJ (Petitt and Cureton, 2003). The energy expenditure elicited in the present study is at the lower end of this spectrum. Although Tsetsonis and colleagues (1997) found an expenditure of 2.3 MJ to be

sufficient to attenuate postprandial lipaemia in untrained participants, their participants were females who were on average 20 years older (43.8 y) and 20 kg lighter (62.2 kg) than the males in the present study. Thus, relative to body mass, 2.3 MJ represents a greater level of expenditure for the participants of Tsetsonis et al. (1997) than for the participants in the present study.

Another possible explanation for the conflict between the findings of the present study and those concerning studies of aerobic exercise relates to the enzyme LPL. This enzyme is located on the capillary endothelium and is particularly abundant in the heart, adipose tissue and skeletal muscle (Seip and Semenkovich, 1998). LPL is responsible for hydrolyzing TAG rich lipoproteins and directing the liberated fatty acids into the surrounding tissues. Several studies have demonstrated increases in skeletal muscle LPL activity following a single bout of prolonged aerobic exercise (Lithell *et al.*, 1979; Lithell *et al.*, 1981) and this is thought to be the major mechanism by which such exercise reduces postprandial lipaemia.

To our knowledge no studies have examined the effects of resistance exercise on LPL activity but an acute bout of knee extension exercise appears to have a less dramatic effect on LPL activity than the changes found following aerobic exercise (Kiens and Lithell, 1989; Kiens *et al.*, 1989). This is possibly because knee-extension exercise, unlike whole-body exercise, evokes little catecholamine response and catecholamines are one of the factors leading to activation of LPL (Newsholme and Leech, 1994). Thus, it may be that the small muscle mass utilized in some of the

337 exercises in the present study had minimal influence on catecholamines and
338 therefore LPL activity. However, some of the exercises employed in our study did
339 involve major muscle groups. Moreover, the plasma catecholamine response has
340 been elevated in other studies examining acute hormonal responses to heavy
341 resistance exercise in both trained and untrained men (Kraemer *et al.*, 1993;
342 Kraemer *et al.*, 1999). It seems unlikely, therefore, that a reduced catecholamine
343 response would, on its own, explain our findings.
344
345 Neither of the previous explanations (insufficient energy expenditure, insufficient
346 activation of LPL) can account for the differences between our findings and those of
347 the one previous study which has examined resistance exercise and postprandial
348 lipaemia. Petitt and colleagues (2003) found a 14% reduction in postprandial
349 lipaemia following a bout of resistance exercise involving 3 sets of 10 repetitions of
350 10 exercises performed at 10 repetition maximum. In the present study a greater
351 volume of exercise was completed (4 sets of 10 repetitions of 11 exercises) although
352 the intensity was lower (80% of 10 repetition maximum). The length of the
353 resistance exercise session was identical in the two studies (88 min) but estimated
354 energy expenditure was 35% higher in our study (2.3 versus 1.7 MJ). This was
355 probably due to a greater volume of work being completed in our study although
356 inconsistency between the methods used to estimate energy expenditure may also
357 have contributed to the difference (a precise description of how energy expenditure
358 was estimated is not given by Petitt et al. 2003). The gap between the resistance
359 exercise and the test meal was also identical between studies (16 hours) and the test

meals employed in each study provided very similar energy and macronutrient content. Thus, none of these factors would seem to explain the discrepancy between studies.

One factor which may explain the differences in findings between these studies is the nature of the participants involved. The study of Petitt and colleagues (2003) involved males (*n*=10) and females (*n*=4) who had six-years experience of weight lifting. In contrast, none of the participants in the present study were regularly involved in resistance training. Thus, it is possible that the physiological stress associated with this unfamiliar form of exercise may have caused skeletal muscle damage in our participants which may not have occurred in the relatively well-trained participants of Petitt and colleagues (2003). Skeletal muscle damage may elevate concentrations of the cytokine tumor necrosis factor-α which has been associated with transient insulin resistance due to a down regulation of insulin-receptor-signalling in adipocytes, hepatocytes and skeletal muscle (Kirwan and Jing, 2002). This may have impaired the uptake of TAG into adipose tissue/skeletal muscle (via a reduced stimulation of LPL). It may also have reduced the efficiency of insulin in suppressing fat mobilisation from adipose tissue and the liver. However, we do not have firm evidence to support these ideas.

It is feasible that a reduction in postprandial lipaemia is dependent on the relative substrate contribution to energy metabolism during exercise. If this were the case and if fat was not a major source of fuel during resistance exercise then perhaps

there would be limited impact on postprandial lipaemia. However, there is evidence to show that fat does provide a significant source of fuel during resistance exercise since muscle biopsy samples taken from the vastus lateralis muscles of bodybuilders immediately after a 30 minute bout of heavy resistance exercise indicated a 30% reduction in intramuscular TAG in comparison to pre-exercise values (Essen-Gustavsson and Tesch, 1990). Moreover, Malkova and colleagues (1999) found no difference in the postprandial TAG response following 90 min of treadmill running when fat metabolism was inhibited by acipimox during running on one occasion. They concluded that the mechanisms by which prior exercise attenuates postprandial lipaemia are not influenced by the relative contributions of fat and carbohydrate to energy metabolism during exercise.

Resting fat oxidation is usually increased the morning after an aerobic exercise bout and fasting TAG concentration is often reduced (Tsetsonis *et al.*, 1997; Tsetsonis and Hardman, 1996b). In their study of resistance exercise and postprandial lipaemia Petitt and colleagues (2003) also observed that resting fat oxidation was higher and fasting TAG concentration lower, the morning after exercise compared to the morning of the control trial. These findings may reflect an acute state of negative energy balance that requires greater mobilization and oxidation of fatty acids (Melby *et al.*, 1993). Such changes may be required prior to any lowering of postprandial lipaemia. In the present study fasting TAG concentrations were not reduced the morning after resistance exercise and fat oxidation was not enhanced. This is consistent with the findings of another recent study of resistance exercise (Melanson

406 *et al.*, 2002) and may be an explanation for the lack of change in postprandial
407 lipaemia. However, a reduction in postprandial lipaemia has been observed in the
408 absence of changes to fasting TAG concentration and resting fat oxidation (Herd *et*
409 *al.*, 2001), so this is unlikely to fully explain the lack of change in our study.
410
411 One finding which we are unable to explain is the significantly lower HDL
412 cholesterol concentration observed on the morning after the resistance exercise bout
413 compared with the control trial. Previous studies of acute exercise and postprandial
414 lipaemia have not detected any difference in fasting HDL cholesterol concentrations
415 between exercise and control trials. Moreover, most evidence indicates that
416 resistance exercise does not influence fasting HDL cholesterol concentration
417 (Durstine and Haskell, 1994).
418
419 In conclusion, our findings indicate that a single session of resistance exercise does
420 not influence postprandial lipaemia. These findings conflict with those of the only
421 other study which we are aware of that has examined the relationship between
422 resistance exercise and postprandial lipaemia (Petitt *et al.*, 2003). Further research is
423 required to clarify this relationship, given the emphasis on resistance exercise in
424 physical activity recommendations (U.S. Department of Health and Human Services,
425 1996; American College of Sports Medicine, 1998). In particular, research is
426 required to evaluate whether or not muscle damage, such as that which can occur
427 during resistance exercise, influences postprandial lipaemia.

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578

579 **Figure Legends**

580

581 Figure 1: Study protocol.

582

583 Figure 2: Total (a) and incremental (b) plasma triacylglycerol (TAG) concentrations
584 in the fasted state (0 h) and for 6 h following consumption of a high-fat mixed meal
585 in the control and resistance exercise trials. Values are mean ± SE, n = 11.

586

587 Figure 3: Plasma NEFA (a), glucose (b) and insulin (c) concentrations in the fasted
588 state (0 h) and for 6 h following consumption of a high-fat mixed meal in the control
589 and resistance exercise trials. Values are mean ± SE, n = 11.

Table 1. Physical characteristics of the participants.

Age (y)	23.45 ± 1.39
Height (m)	1.80 ± 0.02
Body Mass (kg)	84.26 ± 2.73
BMI ($kg \cdot m^{-2}$)	25.87 ± 0.71
Body fat (%)	14.4 ± 1.6

Values are mean ± SE, $n = 11$.

Table 2. Plasma concentrations of lipids, lipoproteins, glucose and insulin in the fasted state (prior to consumption of a test meal) in the control and resistance exercise trials.

	Control	Exercise	*P*
Plasma TAG ($mmol \cdot L^{-1}$)	1.03 ± 0.13	0.94 ± 0.09	0.34
Total cholesterol ($mmol \cdot L^{-1}$)	4.50 ± 0.24	4.18 ± 0.31	0.21
HDL cholesterol ($mmol \cdot L^{-1}$)	1.16 ± 0.10	1.02 ± 0.08	0.01
NEFA ($mmol \cdot L^{-1}$)	0.51 ± 0.05	0.48 ± 0.05	0.57
Glucose ($mmol \cdot L^{-1}$)	5.11 ± 0.27	4.86 ± 0.44	0.46
Insulin ($\mu U \cdot mL^{-1}$)	29.37 ± 2.04	24.24 ± 3.23	0.08

Values are mean ± SE, $n = 11$.

Table 3. Six-hour areas under the plasma concentration versus time curves after consumption of a high-fat mixed meal in control and resistance exercise trials.

	Control	Exercise	*P*
Total TAG ($mmol \cdot L^{-1} \cdot 6\ h$)	9.84 ± 1.40	9.38 ± 1.12	0.47
Incremental TAG ($mmol \cdot L^{-1} \cdot 6\ h$)	3.66 ± 0.67	3.81 ± 0.64	0.63
NEFA ($mmol \cdot L^{-1} \cdot 6\ h$)	2.75 ± 0.20	2.95 ± 0.20	0.32
Glucose ($mmol \cdot L^{-1} \cdot 6\ h$)	30.21 ± 0.92	28.93 ± 0.83	0.30
Insulin ($\mu U \cdot mL^{-1} \cdot 6\ h$)	295.4 ± 39.1	273.8 ± 52.4	0.36

Values are mean ± SE, $n = 11$.

Table 4. Substrate utilization and energy expenditure in the fasted and postprandial states.

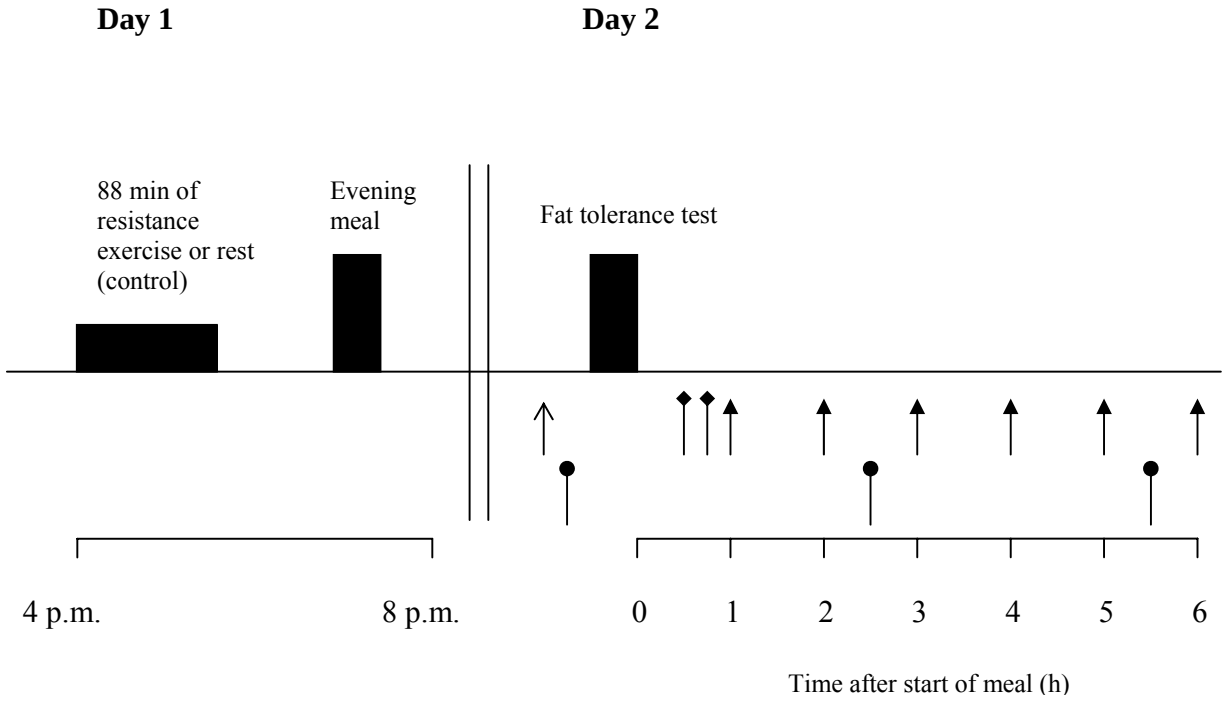
	Fasting	2.5 Hours	5.5 Hours	Mean
RER				
Control	0.82 ± 0.02	0.82 ± 0.02	0.79 ± 0.02	0.81 ± 0.01*
Exercise	0.81 ± 0.02	0.78 ± 0.01	0.78 ± 0.01	0.79 ± 0.01
Mean	0.82 ± 0.01	0.80 ± 0.01	0.79 ± 0.01	
Energy expenditure ($kJ \cdot h^{-1}$)				
Control	338 ± 23	371 ± 23	345 ± 19	351 ± 12
Exercise	331 ± 9	365 ± 19	345 ± 16	346 ± 9
Mean	334 ± 12	368 ± 15†	344 ± 12‡	
Carbohydrate oxidation ($g \cdot h^{-1}$)				
Control	9.01 ± 1.46	9.94 ± 1.79	6.80 ± 1.41	8.58 ± 0.90
Exercise	7.60 ± 1.17	6.01 ± 1.09	5.62 ± 0.94	6.45 ± 0.62
Mean	8.31 ± 0.92	8.07 ± 1.12	6.24 ± 0.85	
Fat oxidation ($g \cdot h^{-1}$)				
Control	4.71 ± 0.49	5.15 ± 0.39	5.86 ± 0.64	5.24 ± 0.30
Exercise	5.16 ± 0.50	6.73 ± 0.51	6.37 ± 0.39	6.06 ± 0.29
Mean	4.93 ± 0.34	5.90 ± 0.36†	6.11 ± 0.38†	

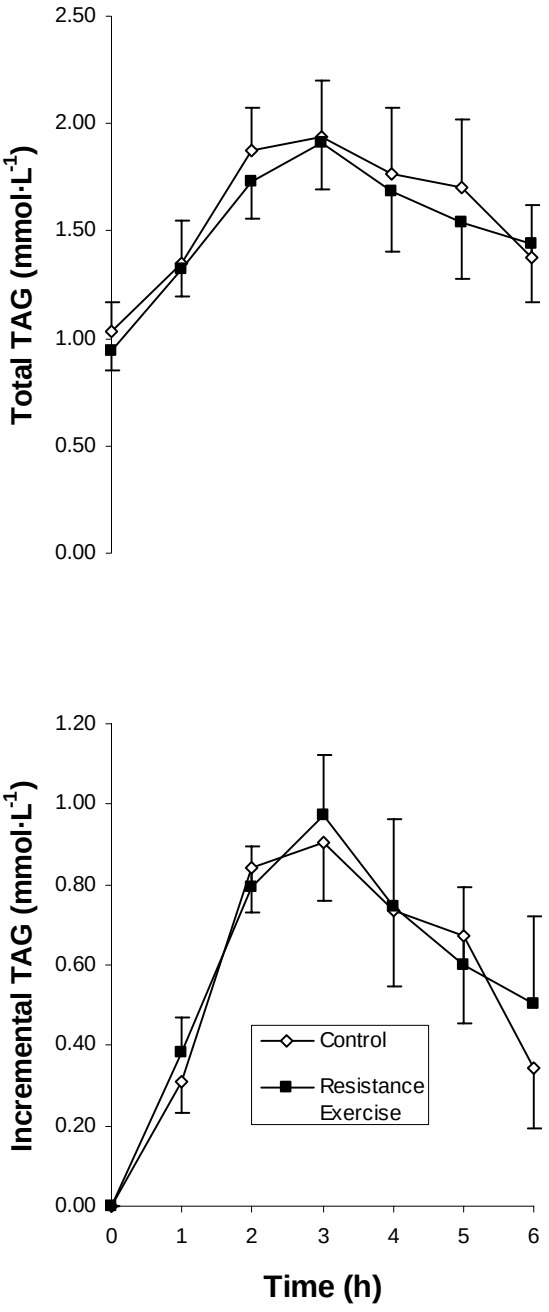
Values are mean ± SE, $n = 11$.

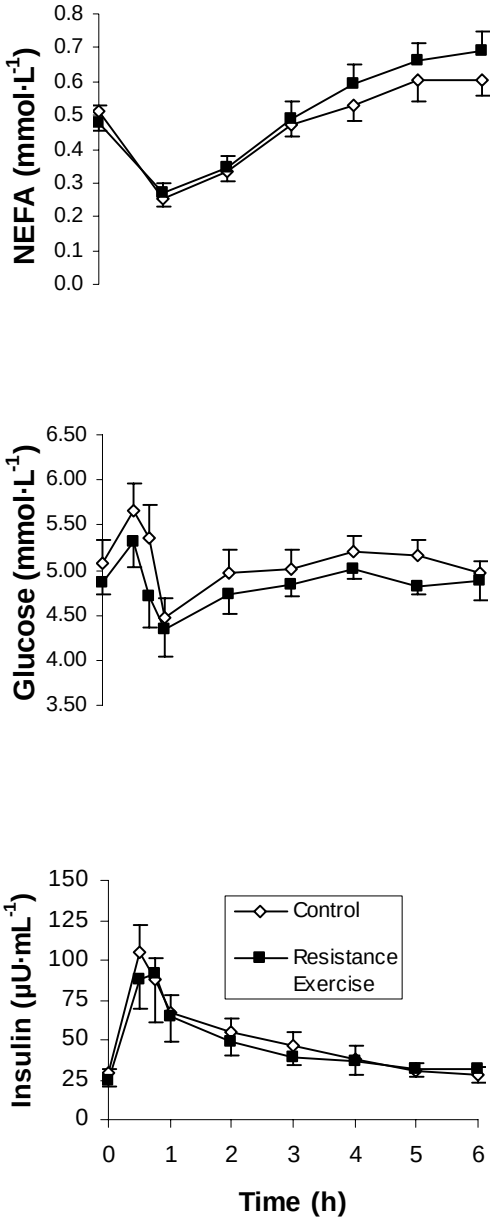
*Main effect of trial by ANOVA $P<0.05$

†Significantly different from fasting $P<0.05$

‡Significantly different from 2.5 hours $P<0.05$


Day 1
Day 2
88 min of resistance exercise or rest (control)
Evening meal
Fat tolerance test
4 p.m.
8 p.m.
0
1
2
3
4
5
6
Time after start of meal (h)
Baseline blood sample (TAG, NEFA, glucose, insulin, cholesterol, HDL cholesterol)
Blood sample (glucose, insulin)
Blood sample (TAG, NEFA, glucose, insulin)
Ventilated hood measurements


Total TAG (mmol·L-1)
2.50
2.00
1.50
1.00
0.50
0.00
Incremental TAG (mmol·L-1)
1.20
1.00
0.80
0.60
0.40
0.20
0.00
Control
Resistance Exercise
0
1
2
3
4
5
6
Time (h)


NEFA (mmol·L-1)
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Glucose (mmol·L-1)
6.50
6.00
5.50
5.00
4.50
4.00
3.50
Insulin (µU·mL-1)
150
125
100
75
50
25
0
Control
Resistance Exercise
0
1
2
3
4
5
6
Time (h)