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A single session of resistance exercise does not reduce postprandial lipaemia

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A single session of resistance exercise does not reduce postprandial lipaemia

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Abbreviated title: Resistance exercise and postprandial lipaemia

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Key Words: triacylglycerol, weight lifting, energy expenditure, substrate oxidation

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1 **ABSTRACT**

2 This study investigated the effect of a single session of resistance exercise on
3 postprandial lipaemia. Eleven healthy, normolipidaemic men aged 23 (SE 1.4) years
4 performed two trials at least one-week apart in a counterbalanced randomized
5 design. In each trial participants consumed a test meal (1.2g fat, 1.1g carbohydrate,
6 0.2 g protein and 68 kJ per kg body mass) between 08.00 and 09.00 following a 12
7 hour fast. The afternoon before one trial participants performed an 88 minute bout of
8 resistance exercise. Prior to the other trial participants were inactive (control trial).
9 Resistance exercise was performed using free weights and included 4 sets of 10-
10 repetitions of each of 11 exercises. Sets were performed at 80% of 10 repetition
11 maximum with a 2 minute work and rest interval. Venous blood samples were
12 obtained in the fasted state and at intervals for 6 h postprandially. Fasting plasma
13 triacylglycerol (TAG) concentration did not differ significantly between control and
14 exercise trials (mean $\pm$ SE: $1.03 \pm 0.13 \text{ mmol}\cdot\text{L}^{-1}$ versus $0.94 \pm 0.09 \text{ mmol}\cdot\text{L}^{-1}$;
15 respectively). Similarly the 6 h total area under the plasma TAG concentration
16 versus time curve did not differ significantly between control and exercise trials
17 ($9.84 \pm 1.40 \text{ mmol}\cdot\text{L}^{-1}\cdot\text{h}$ versus $9.38 \pm 1.12 \text{ mmol}\cdot\text{L}^{-1}\cdot\text{h}$; respectively). These
18 findings suggest that a single session of resistance exercise does not reduce
19 postprandial lipaemia.

20 INTRODUCTION

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

31
32 Many studies have shown that a single bout of aerobic exercise reduces postprandial
33 lipaemia (Aldred *et al.*, 1994; Tsetsonis and Hardman, 1996a; Tsetsonis and
34 Hardman, 1996b; Tsetsonis *et al.*, 1997; Gill *et al.*, 2003). This reduction appears to
35 be transient, however, since detraining leads to a prompt elevation in postprandial
36 lipaemia (Hardman *et al.*, 1998; Herd *et al.*, 1998). Thus, exercise must be
37 performed frequently for continued benefit. Two mechanisms have been proposed to
38 explain the TAG-lowering effects of aerobic exercise. One is an increase in
39 lipoprotein lipase (LPL) activity which may enhance the uptake of TAG into
40 previously exercised muscle (Seip and Semenkovich, 1998). The other is a reduced
41 rate of secretion of hepatic very low density lipoproteins (Gill *et al.*, 2001; Malkova
42 *et al.*, 2000). Furthermore, energy expenditure during exercise appears to be the

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

45

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

54

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

63 **METHODS**64 *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.

74

75

TABLE 1 NEAR HERE

76

77 *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,

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

92

93 Following the preliminary visits participants undertook two fat tolerance tests. The
94 first of these tests was performed a minimum of one-week after the practice weight
95 lifting-session. The interval between the two fat tolerance tests was at least one-
96 week. The afternoon prior to one of the fat tolerance tests participants performed an
97 88 minute bout of resistance exercise starting at approximately 16.00 h (exercise
98 trial). The afternoon prior to the other fat tolerance test participants rested (control
99 trial). Trials were performed in a counterbalanced randomized design.

100

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

107

108 *Anthropometry.*

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

114

115 *Weight lifting protocol.*

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

consecutively. It was also designed so that large muscle groups were exercised first. Standard free weights were used, including dumbbells, bent bar, an Olympic bar and a mobile bench. Water intake was permitted ad libitum throughout the resistance exercise session.

135

Estimation of energy expenditure during weight lifting.

Expired air samples were collected into Douglas bags (Plysu Protection Systems, Milton Keynes, U.K.) during the third set of each weight lifting exercise (pilot work revealed that there was no systematic variation in oxygen consumption values measured during the first, second, third and fourth set of each exercise). Samples were collected for the full duration of the lifting and recovery period i.e. 2 min.

Oxygen consumption ($\dot{V}\text{O}_2$) and carbon dioxide production ($\dot{V}\text{CO}_2$) were determined from expired air samples using a paramagnetic oxygen analyzer and an infra-red carbon dioxide analyzer respectively (Series 1400; Servomex, Crowborough, East Sussex, U.K.). Expired air volumes were measured using a dry gas meter (Harvard Apparatus, Edenbridge, Kent, U.K.) and corrected to standard temperature and pressure (dry).

148

The short duration, intermittent nature of weight lifting invalidates the typical assumptions of indirect calorimetry because the respiratory exchange ratio (RER) is consistently equal to or greater than 1.0. Therefore, energy expenditure was calculated as being 5.047 kcal (21.1 kJ) per litre of oxygen (McArdle *et al.*, 2001). This reflects the assumption that energy was derived from carbohydrate rather than

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

158

159 *Fat tolerance tests.*

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

177

FIGURE 1 NEAR HERE

178

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

201 Ltd. U.K.) and non-esterified fatty acids (NEFA) (Wako Chemicals GmbH,
202 Germany) by enzymatic, colorimetric methods with the use of a centrifugal analyzer
203 (Cobas-Bio; Roche, Basel, Switzerland). Plasma insulin concentration was
204 determined using a solid-phase ^{125}I radioimmunoassay available in a commercial kit
205 (ICN Pharmaceuticals, Inc., Costa Mesa, CA). The within batch coefficients of
206 variation for these assays were as follows: TAG 1.8%, glucose, 1.3%, cholesterol
207 1.0%, HDL cholesterol 1.4%, NEFA 0.8%, insulin 5.7%. To eliminate inter-assay
208 variation, samples from both trials for each participant were always analysed in the
209 same batch. Haemoglobin concentration and haematocrit were determined in
210 samples collected at baseline and 6-h so that changes in plasma volume could be
211 estimated (Dill and Costill, 1974).

212

213 *Data Analysis.*

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

229

The mean weight lifted during the 88 minute resistance exercise session was 14,214 ± 613 kg. Mean $\dot{V}\text{O}_2$ was $1.22 \pm 0.03 \text{ L} \cdot \text{min}^{-1}$ with a mean RER value of 1.05 ± 0.02 over this period. The gross energy expenditure from the exercise was estimated to be $2.3 \pm 0.3 \text{ MJ}$ and the net (gross minus resting) energy expenditure was estimated at $1.8 \pm 0.06 \text{ MJ}$.

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

248 Changes in plasma volume over the period of observation were small and did not
249 differ significantly between control ($0.2 \pm 2.7 \%$) and exercise ($-2.5 \pm 2.0 \%$) trials.
250 No adjustments were made, therefore, to measured concentrations of plasma
251 constituents.

253 Total and incremental plasma TAG concentrations following the fat tolerance tests
 254 are shown in Figure 2, with summary measures (areas under the curve) of these
 255 responses in Table 3. No difference was seen in either the total or the incremental
 256 area under the curve values between the control and exercise conditions. No
 257 difference was observed in the peak TAG concentration (control 2.15 ± 0.26
 258 $\text{mmol}\cdot\text{L}^{-1}$, exercise $2.16 \pm 0.26 \text{ mmol}\cdot\text{L}^{-1}$) or time-to-peak TAG concentration
 259 (control $3.45 \pm 0.43 \text{ h}$, exercise $3.45 \pm 0.49 \text{ h}$). Positive relationships were seen in
 260 both trials between the total area under the curve and the fasting TAG concentrations
 261 (control $r = 0.95$, $P < 0.01$; exercise $r = 0.93$, $P < 0.01$).

262

263 FIGURE 2 NEAR HERE

264 TABLE 3 NEAR HERE

265

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

273

274 FIGURE 3 NEAR HERE

275

276 *Substrate utilization and energy expenditure.*

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

281

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

289

290 TABLE 4 NEAR HERE

291 **DISCUSSION**

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

305

306 A possible explanation for the failure of resistance exercise to influence postprandial
307 lipaemia in the present study is insufficient energy expenditure. The estimated gross
308 energy expended during the resistance exercise in our study was 2.3 MJ (range =
309 2.0-2.6 MJ). In previous studies examining the acute effects of aerobic exercise on
310 postprandial lipaemia, the average gross energy expenditure found to attenuate the
311 lipemic response has ranged from 1.5 to 7.2 MJ (Petitt and Cureton, 2003). The
312 energy expenditure elicited in the present study is at the lower end of this spectrum.
313 Although Tsetsonis and colleagues (1997) found an expenditure of 2.3 MJ to be

314 sufficient to attenuate postprandial lipaemia in untrained participants, their
315 participants were females who were on average 20 years older (43.8 y) and 20 kg
316 lighter (62.2 kg) than the males in the present study. Thus, relative to body mass, 2.3
317 MJ represents a greater level of expenditure for the participants of Tsetsonis et al.
318 (1997) than for the participants in the present study.

319

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

329

330 To our knowledge no studies have examined the effects of resistance exercise on
331 LPL activity but an acute bout of knee extension exercise appears to have a less
332 dramatic effect on LPL activity than the changes found following aerobic exercise
333 (Kiens and Lithell, 1989; Kiens *et al.*, 1989). This is possibly because knee-
334 extension exercise, unlike whole-body exercise, evokes little catecholamine response
335 and catecholamines are one of the factors leading to activation of LPL (Newsholme
336 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

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

363

364 One factor which may explain the differences in findings between these studies is
365 the nature of the participants involved. The study of Petitt and colleagues (2003)
366 involved males ($n=10$) and females ($n=4$) who had six-years experience of weight
367 lifting. In contrast, none of the participants in the present study were regularly
368 involved in resistance training. Thus, it is possible that the physiological stress
369 associated with this unfamiliar form of exercise may have caused skeletal muscle
370 damage in our participants which may not have occurred in the relatively well-
371 trained participants of Petitt and colleagues (2003). Skeletal muscle damage may
372 elevate concentrations of the cytokine tumor necrosis factor- α which has been
373 associated with transient insulin resistance due to a down regulation of insulin-
374 receptor-signalling in adipocytes, hepatocytes and skeletal muscle (Kirwan and Jing,
375 2002). This may have impaired the uptake of TAG into adipose tissue/skeletal
376 muscle (via a reduced stimulation of LPL). It may also have reduced the efficiency
377 of insulin in suppressing fat mobilisation from adipose tissue and the liver. However,
378 we do not have firm evidence to support these ideas.

379

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

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

394

395 Resting fat oxidation is usually increased the morning after an aerobic exercise bout
396 and fasting TAG concentration is often reduced (Tsetsonis *et al.*, 1997; Tsetsonis
397 and Hardman, 1996b). In their study of resistance exercise and postprandial lipaemia
398 Petitt and colleagues (2003) also observed that resting fat oxidation was higher and
399 fasting TAG concentration lower, the morning after exercise compared to the
400 morning of the control trial. These findings may reflect an acute state of negative
401 energy balance that requires greater mobilization and oxidation of fatty acids (Melby
402 *et al.*, 1993). Such changes may be required prior to any lowering of postprandial
403 lipaemia. In the present study fasting TAG concentrations were not reduced the
404 morning after resistance exercise and fat oxidation was not enhanced. This is
405 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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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 $\pm$ 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 $\pm$ 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 ($\text{kg}\cdot\text{m}^{-2}$)	25.87 ± 0.71
Body fat (%)	14.4 ± 1.6

Values are mean $\pm$ 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	<i>P</i>
Plasma TAG (mmol·L ⁻¹)	1.03 ± 0.13	0.94 ± 0.09	0.34
Total cholesterol (mmol·L ⁻¹)	4.50 ± 0.24	4.18 ± 0.31	0.21
HDL cholesterol (mmol·L ⁻¹)	1.16 ± 0.10	1.02 ± 0.08	0.01
NEFA (mmol·L ⁻¹)	0.51 ± 0.05	0.48 ± 0.05	0.57
Glucose (mmol·L ⁻¹)	5.11 ± 0.27	4.86 ± 0.44	0.46
Insulin (μU·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	<i>P</i>
Total TAG (mmol·L ⁻¹ ·6 h)	9.84 ± 1.40	9.38 ± 1.12	0.47
Incremental TAG (mmol·L ⁻¹ ·6 h)	3.66 ± 0.67	3.81 ± 0.64	0.63
NEFA (mmol·L ⁻¹ ·6 h)	2.75 ± 0.20	2.95 ± 0.20	0.32
Glucose (mmol·L ⁻¹ ·6 h)	30.21 ± 0.92	28.93 ± 0.83	0.30
Insulin (μU·mL ⁻¹ ·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	$0.81 \pm 0.01^*$
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 ($\text{kJ}\cdot\text{h}^{-1}$)				
Control	338 ± 23	371 ± 23	345 ± 19	351 ± 12
Exercise	331 ± 9	365 ± 19	345 ± 16	346 ± 9
Mean	334 ± 12	$368 \pm 15^\dagger$	$344 \pm 12^\ddagger$	
Carbohydrate oxidation ($\text{g}\cdot\text{h}^{-1}$)				
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 ($\text{g}\cdot\text{h}^{-1}$)				
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	$5.90 \pm 0.36^\dagger$	$6.11 \pm 0.38^\ddagger$	

Values are mean $\pm$ 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 $P < 0.05$

- ↑ Baseline blood sample (TAG, NEFA, glucose, insulin, cholesterol, HDL cholesterol)
- ◆ Blood sample (glucose, insulin)
- ↑ Blood sample (TAG, NEFA, glucose, insulin)
- Ventilated hood measurements