Multiple Bouts of Resistance Exercise and Postprandial Triacylglycerol and Serum C-Reactive-Protein Concentrations

Stephen F. Burns, Masashi Miyashita, Chihoko Ueda, and David J. Stensel

The present study examined how multiple bouts of resistance exercise, performed over 1 d, influence 2 risk factors—postprandial triacylglycerol (TAG) and serum C-reactive-protein (CRP) concentrations—associated with coronary heart disease. Twenty-four men age 23.5 (SD 3.4) y completed two 2-d trials, exercise and control, at least 1 wk apart in a counterbalanced randomized design. On day 1 of the exercise trials participants completed 20 sets of 15 repetitions of 5 different resistance exercises divided into five 45-min bouts of exercise—100 sets and 1500 repetitions in total for all exercises. Exercises were performed at 30–40% of 1-repetition maximum. Blood samples were taken before and after exercise. On day 1 of the control trial participants were inactive, with blood samples taken at time points corresponding to the exercise trial. On day 2 of both trials participants consumed a test meal (0.89 g fat, 1.23 g carbohydrate, 0.4 g protein, 60 kJ per kg body mass). Blood samples were obtained fasted and for 6 h postprandially. Total area under the postprandial TAG concentration versus time curve was 12% lower in the exercise than in the control trial (8.76 [3.54] vs. 9.94 [4.31] mmol·L⁻¹·6 h, respectively; \( P = 0.037 \)). Serum CRP concentrations did not change over the 2 d in the control trial but increased in the exercise trial: trial × time interaction (\( P = 0.028 \)). Multiple bouts of resistance exercise reduce postprandial TAG concentrations but increase serum CRP concentrations. The extent to which these findings are clinically relevant requires further study.

Key Words: lipoproteins, fat metabolism, weight lifting, energy expenditure, cardiovascular disease risk

Recent reports (6, 17, 36) highlight the benefits of resistance exercise for health, and evidence for a protective health effect from regular resistance exercise is growing (11, 14, 21, 32). Muscle strength and fitness have been shown to have a protective effect on all-cause mortality and the metabolic syndrome (11, 14). There is also evidence from epidemiological studies for a reduction in coronary heart disease risk (21, 32) with regular resistance exercise. Paffenbarger and Hale (21) investigated the effect of occupational activity on coronary heart disease in
dockworkers. Dockworkers who performed hard physical labor such as lifting, pushing, and carrying heavy cargo had lower coronary heart disease mortality than dockworkers in more sedentary jobs. A more recent study (32) found that those who participated in 30 min or more of weight training per week had a 23% lower risk of coronary heart disease than those who performed no weight training. Resistance training might also improve endurance capacity and performance in older adults (1, 2). Nonetheless, the benefits of resistance exercise on individual risk factors for cardiovascular disease remains to be established.

Cardiovascular diseases share a common pathophysiology involving atherosclerosis and thrombosis. Disturbances in lipoprotein metabolism in the postprandial period might promote atherosclerosis because of an accumulation of triacylglycerol (TAG)-rich lipoprotein remnants in the plasma, catabolism of high-density lipoproteins, and formation of small, dense, low-density lipoproteins, which have an increased susceptibility to oxidation (15). Atherosclerosis is not only a disease of lipid accumulation but also a chronic inflammatory process (27). C-reactive protein (CRP) is a marker of the inflammatory process associated with atherosclerosis (37) that might also play an etiological role in atherogenesis (22).

To date, 5 studies have examined the effect of a single session of resistance exercise on postprandial TAG concentrations (4, 5, 25, 29, 39). Two of these reported a lowering (25, 39), 2 no change (5, 29), and 1 an increase (4) in postprandial TAG concentrations after resistance exercise. Differences in the energy expenditure of exercise among these studies might be one reason for the discrepant results because energy expenditure appears to be the primary determinant of the exercise-induced reduction in postprandial TAG concentrations with aerobic exercise (34). The epidemiological investigations (21) into dockworkers suggest that hard, physical labor is beneficial for health, and it is possible that the energy expenditure of this labor provided some of the cardioprotective effect for the workers. Thus, it remains important to determine how much exercise is beneficial for health. Three studies (20, 23, 30) have examined the effect of a single bout of resistance exercise on serum CRP concentrations, with 2 showing no change (20, 30) and 1 showing an increase (23) in serum CRP concentrations postexercise. The volume of exercise performed, the muscle mass employed, and the extent of muscle injury (31) might explain the differences between the findings of these studies.

The effect of resistance exercise on cardiovascular-disease risk factors is unclear. The aim of the current study, therefore, was to examine the effect of lifting light weights in multiple bouts performed throughout the day on postprandial TAG and CRP concentrations. Multiple bouts were used in an effort to mimic the labor of the dockworkers studied by Paffenbarger and Hale (21) over 30 y ago. The high energy expenditure of such activity might enhance the potential impact on postprandial TAG concentrations.

**Methods**

**Participants**

Twenty-four male volunteers age 20–32 y participated in this study, which was approved by Loughborough University’s ethical advisory committee. Postprandial TAG metabolism was measured in all volunteers. In a subset of 10 of these
volunteers blood samples were also collected for the measurement of CRP. 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: nonsmoking, no known history of cardiovascular disease, body-mass index <30 kg/m², resting arterial blood pressure <140/90 mm Hg, and not taking any medication known to affect lipid or carbohydrate metabolism. All volunteers were regularly active at the time of participation, with some participating in regular resistance training. Some physical characteristics of the participants are shown in Table 1.

### Preliminary Tests

Before the main trials participants visited the laboratory once for collection of anthropometric data and to complete 1-repetition-maximum weight-lifting tests for 5 different resistance exercises.

**Anthropometry.** Height and weight were determined using standard methods. Skinfold thicknesses were measured at 4 sites (biceps, triceps, subscapular, and suprailiac) using calipers. Body density was calculated using a 4-site formula and body-fat percentage then estimated using the Siri equation (8).

**One-Repetition-Maximum Tests.** One-repetition-maximum (1-RM) tests were completed for each of the 5 resistance exercises employed in the study. The 1-RM values were determined by trial and error by adding or removing weights after each attempt as required. Participants were allowed to take as long as they felt necessary to recover from each attempt. The order in which the 1-RM tests were performed was the same for each participant: squat, bench press, lunges, bicep curl, and shoulder press (seated).

**Control of Diet and Exercise.** For 2 days before the main trials participants were asked to refrain from physical activity. Only gentle walking for personal transportation over short distances was permitted. Participants weighed and recorded all food and drink consumed 24 h immediately preceding their first main trial and on day 1 of that trial. They tried to replicate this intake during the same time period on their second trial. Participants also refrained from alcohol during these periods.

### Main Trials

After the preliminary tests participants undertook two 2-d trials—exercise and control. On day 1 of the exercise trial participants lifted multiple bouts of light

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean (standard deviation)</th>
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<tbody>
<tr>
<td>Age, y</td>
<td>23.5 (3.4)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.78 (0.07)</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>75.8 (10.9)</td>
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<tr>
<td>Body mass index, kg/m²</td>
<td>23.8 (2.7)</td>
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<tr>
<td>Body fat, %</td>
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weights over the course of the day. On day 1 of the control trial participants rested. On day 2 of both trials participants came to the lab fasted and consumed a test meal. The order of the trials was randomized and balanced. The interval between the 2 trials was at least 1 wk.

**Resistance-Exercise Protocol: Day 1.** A schematic representation of the exercise protocol is given in Figure 1. In the exercise trial participants reported to the laboratory at 8:30 AM. They sat on a bed in a semisupine position for 10 min after their arrival. A 6-min resting sample of expired air was then collected into a Douglas bag. Oxygen consumption and carbon-dioxide production were determined from the resting air sample using a paramagnetic oxygen analyzer and an infrared carbon-dioxide analyzer (Servomex Analyser Series 1400, Servomex, Crowborough, East Sussex, UK). Expired air volumes were measured using a dry gas meter (Harvard Apparatus, Edenbridge, Kent, UK) and corrected to standard temperature and pressure (dry). Energy expenditure was calculated using equations assuming no protein oxidation (12). A blood sample was then taken by venipuncture into a 9-mL serum monovette for the determination of CRP.

At 9:00 AM participants began the resistance exercise. Over the day, each participant completed 20 sets of 15 repetitions of 5 different resistance exercises—100 sets and 1500 lifts in total for all exercises. Periods of resistance exercise were divided into five 45-min bouts. Each 45-min bout of lifting consisted of 4 sets of each of the 5 different exercises. Participants completed 1 set of each exercise sequentially in the same order as described for the preliminary tests—squat, bench press, lunges, bicep curl, and shoulder press. Upper body exercises were completed at 30% of 1-RM, whereas lower body exercises were completed at 40% of 1-RM. Participants were given 1 min to recover between exercises. After the last exercise (shoulder press) participants were given 5 min to recover. The sequence was then repeated until 4 sets of each exercise had been completed.

On completion of each 45-min bout of lifting (4 sets of each exercise) participants were given a rest interval. There was a 15-min rest interval after the first two 45-min bouts, a 105-min lunch break after the third 45-min bout, and a 15-min rest interval between the fourth and fifth 45-min bouts. The fifth bout of exercise finished at 3:15 PM. The high volume of work over the day was designed to maximize the total energy expenditure of the session and hence increase the likelihood of reducing postprandial TAG concentrations. A second blood sample was taken by venipuncture into a 9-mL serum monovette at the end of exercise (3:30 PM). In the control trial participants reported to the lab at 8:45 AM and at 3:30 PM for a blood sample taken by venipuncture for the determination of CRP.

**Estimation of Energy Expenditure During Resistance Exercise.** Expired air samples were collected into Douglas bags (Plysu Protection Systems, Milton Keynes, UK). Samples were collected during sets 1 and 4 of the first and fifth 45-min bouts of exercise. Separate Douglas bags were used for each exercise. Samples were collected for the full duration of the exercise and recovery period—the exercise time plus 1 min recovery. For the shoulder press the sample was collected for the exercise time plus 5 min recovery. Oxygen consumption, carbon-dioxide production, and expired air volumes were determined using previously described procedures.
Figure 1 — Schematic representation of weight-lifting protocol: day 1.
Oxygen uptake per minute was calculated as the mean of all 5 exercises over the 4 sets collected. The short-duration, intermittent nature of resistance exercise invalidates the typical assumptions of indirect calorimetry because the respiratory-exchange ratio is consistently equal to or greater than 1.0. Energy expenditure was therefore calculated as being 21.1 kJ (5.047 kcal) per liter of oxygen (19). Gross energy expenditure was calculated as mean oxygen uptake per minute multiplied by 21.1 kJ multiplied by the duration of exercise. Net energy expenditure was calculated as gross minus resting energy expenditure over the same duration.

**Main Trial Protocol: Day 2.** Participants reported to the laboratory at 8.00 AM after a 12-h fast. A cannula was inserted into an antecubital or forearm vein and a baseline blood sample taken. Participants then consumed a test meal. This meal comprised white bread, cheddar cheese, butter, potato chips, whole milk, mayonnaise, and milkshake powder and was given according to body mass (0.89 g fat, 1.23 g carbohydrate, 0.4 g protein, 60 kJ/kg body mass). The macronutrient composition of the meal was 33% carbohydrate, 56% fat, and 11% protein. All participants consumed the meal within 20 min. A clock was started with the first bite of this meal (time 0). Further blood samples were obtained 0.5, 0.75, and 1 h after the start of the meal and then hourly for a total of 6 h. Blood samples for the determination of serum CRP were collected at baseline and 3 and 6 h into the trial. The cannula was kept patent by flushing with nonheparinized saline (9 g/L, B. Braun Medical Ltd., Buckinghamshire, UK). The first 2 mL of blood withdrawn were always discarded to avoid dilution of the sample. Water was available ad libitum during the first trial; the volume ingested was recorded and replicated in the second trial. Participants rested (reading, working, watching television) throughout the observation period and were always lying in a supine position for at least 5 min before each blood sample was taken.

**Analytical Methods**

At each sampling point, blood samples were collected into precooled 9-mL potassium-EDTA monovettes (Sarstedt, Leicester, UK) and were kept on ice until centrifugation. Plasma was separated within 15 min of collection, divided into aliquots, and stored at –80 °C. Blood samples for the determination of CRP were collected into 9-mL serum monovettes (Sarstedt, Leicester, UK). Serum blood samples were left to clot for 60 min before centrifugation. Serum was divided into aliquots and stored at –80 °C.

Plasma samples were analyzed (within 6 months of collection) for TAG, glucose, 3-hydroxybutyrate (Randox Laboratories Ltd., UK), and nonesterified fatty acids (NEFA; Wako Chemicals GmbH, Germany) by enzymatic, colorimetric methods with the use of a centrifugal analyzer (Cobas Mira Plus, Roche, Basel, Switzerland). Plasma insulin concentration was determined using a solid-phase $^{125}$I radioimmunoassay available in a commercial kit (MP Biomedicals, Orangeburg, NY). Radioactivity was measured using an automated gamma-counting system (Cobra II, Packard Instruments, Downers Grove, IL). Serum samples were analyzed (within 6 months of collection) for CRP using a high-sensitivity enzyme-linked immunoassay (DRG International Inc., USA) and measured on a plate reader (Opsys microplate reader, Dynex Technologies Inc., USA). The within-batch coefficients
of variation for the assays were as follows: TAG 0.6%, glucose 0.9%, 3-hydroxybutyrate 2.7%, NEFA 0.3%, insulin 5.7%, and CRP 10.5%. To eliminate interassay variation, samples from both trials for each participant were always analyzed in the same batch. Hemoglobin concentration and hematocrit were determined in samples collected at baseline and at the end of the day so that changes in plasma volume could be estimated (7).

Data Analysis

Results were analyzed using statistical software (SPSS 12.0, SPSS Inc., Chicago, IL). Two-way ANOVA (with repeated measures) was used to determine differences between oxygen uptake at each measurement point (i.e., sets 1 and 4 of the first and fifth 45-min bouts of exercise) and the exercise being performed (i.e. squat, bench press, lunges, bicep curl, or shoulder press). Six-hour total area under the plasma TAG-concentration-versus-time curve was calculated using the trapezium rule. The incremental area under the plasma TAG-concentration-versus-time curve was calculated using the same method after we corrected for baseline concentrations. Fasting and area under the curve values were compared between trials using Student’s t-tests for correlated means. Two-way ANOVA (with repeated measures) was used to determine differences between trials and over time for concentrations of TAG, glucose, insulin, NEFA, 3-hydroxybutyrate, and CRP. When appropriate, post hoc pairwise comparisons were made using the Bonferroni method. Relationships between variables were evaluated using Pearson’s product–moment correlation coefficient. A 5% level of significance was adopted throughout, and data are expressed as mean (standard deviation) unless otherwise stated.

Results

Resistance-Exercise Session

The mean weight lifted over the course of the day was 36,997 (6210) kg. The mean weight lifted for each exercise was 25 (4) kg. There was no difference in oxygen uptake between any expired air-sample measurement points, that is, over time ($P = 0.170$). Oxygen uptake differed between exercises ($P < 0.0005$). The average rate of energy expenditure during weight lifting and recovery was estimated to be 22.5 (3.5) kJ/min. The mean gross energy expenditure from the exercise was estimated to be 5.1 (0.8) MJ over the day. Mean net energy expenditure from exercise was estimated to be 3.5 (0.7) MJ.

Plasma Concentrations in the Fasted State

Plasma concentrations in the fasted state on day 2 are shown in Table 2. There were no between-trials differences for TAG, glucose, NEFA, or 3-hydroxybutyrate. Plasma insulin concentrations were significantly higher in the control trial than the exercise trial at baseline.
Postprandial Plasma Responses

Changes in plasma volume over the period of observation did not differ between trials ($P = 0.728$). No adjustments were made, therefore, to measured concentrations of plasma constituents. Plasma TAG concentrations were higher in the control than the exercise trial throughout the postprandial period (Figure 2): main effect of trial, $P = 0.044$, and main effect of time, $P < 0.0005$. There was no difference in the pattern of response between plasma TAG concentrations across trials. The total area under the TAG-concentration-versus-time curve during the postprandial period was 12% lower in the exercise than in the control trial (8.76 [3.54] vs. 9.94 [4.31] mmol·L$^{-1}$·6 h$^{-1}$, respectively, $P = 0.037$). There was large variation in the TAG response to resistance exercise. For some subjects, area under the curve values were higher in the exercise trial than in the control trial; for most, however, lower area under the curve values were recorded after resistance exercise than in the control trial (Figure 3). The incremental area under the TAG-concentration-versus-time curve was 18% lower in the exercise than in the control trial (2.99 [1.84] vs. 3.64 [1.90] mmol·L$^{-1}$·6 h$^{-1}$, respectively, $P = 0.043$). There was no difference in peak TAG concentration between trials or time to peak TAG concentration between trials.

Plasma concentrations for insulin and glucose are shown in Figure 4. Mean insulin concentrations over the postprandial period were significantly lower in the exercise than in the control trial: main effect of trial ($P = 0.043$). Insulin concentrations rose steeply and then fell after the test meal in both the control and exercise trials: main effect of time ($P < 0.0005$). There was no difference in the pattern of response between insulin concentrations across trials. Peak insulin concentrations were significantly higher in the control than the exercise trial ($P = 0.041$). Adjusted peak insulin response (peak minus fasting concentrations) also showed a tendency to be higher in the control than in the exercise trial ($P = 0.052$). Plasma glucose concentrations did not differ between trials. Plasma glucose concentrations in both trials rose steeply in the early postprandial period and then fell back to baseline concentrations within the hour: main effect of time ($P < 0.0005$). There was no difference in the pattern of response between glucose concentrations across trials.

Plasma 3-hydroxybutyrate concentrations (Figure 5A) were higher in the exercise than in the control trial: main effect of trial ($P = 0.001$). Plasma 3-hydroxybutyrate dropped for the first 2 h in the postprandial period on both
Figure 2 — Postprandial total plasma triacylglycerol concentrations for 6 h after consumption of a test meal in the control and resistance-exercise trials. Values are mean ± standard error, \( N = 24 \). Main effect of trial, \( P = 0.044 \); main effect of time, \( P < 0.0005 \).

Figure 3 — Individual changes (exercise minus control) in total area under the triacylglycerol (TAG) concentration versus time curve in response to the resistance-exercise trial.
Figure 4 — Postprandial plasma (A) glucose and (B) insulin concentrations for 6 h after consumption of a test meal in the control and resistance-exercise trials. Values are mean ± standard error, N = 24. Main effect of time for glucose, $P < 0.0005$; main effect of trial, $P = 0.043$; main effect of time for insulin, $P < 0.0005$. 
Figure 5 — Postprandial plasma (A) 3-hydroxybutyrate and (B) nonesterified fatty acid (NEFA) concentrations for 6 h after consumption of a test meal in the control and resistance-exercise trials. Values are mean ± standard error, N = 19. Main effect of trial, P = 0.001; main effect of time, \( P < 0.0005 \); trial × time interaction for 3-hydroxybutyrate, \( P = 0.028 \); main effect of time for NEFA, \( P < 0.0005 \). *Significantly different (\( P < 0.05 \)) between trials using a Bonferroni post hoc test.
Resistance Exercise, Triacylglycerol, and CRP

Thereafter, 3-hydroxybutyrate rose until the end of both trials, with a steeper increase in the exercise than control trial: main effect of time ($P < 0.0005$), trial $\times$ time interaction ($P = 0.028$). Post hoc tests revealed that the difference in 3-hydroxybutyrate concentrations occurred at 6 h into the trial ($P = 0.012$). Plasma NEFA concentrations (Figure 5B) showed a tendency to be higher in the exercise than the control trial: main effect of trial ($P = 0.058$). Plasma NEFA concentrations fell in both trials during the first hour after the test meal. In both trials NEFA concentrations then rose steeply until the end of the trial: main effect of time ($P < 0.0005$).

**Serum CRP Concentrations**

One participant who had serum CRP concentrations >10 mg/L in the control trial was excluded from the CRP analysis. A CRP concentration >10 mg/L might be indicative of infection or trauma (24). Further conversation with the participant revealed that he had been ill with a cold before the trial. Baseline concentrations of serum CRP did not differ significantly between trials on day 1 when the remaining 9 participants were included ($P = 0.149$).

Serum CRP concentrations were not significantly different between trials or over time—no main effect of trial or time. The pattern of serum CRP response was significantly different between trials: trial $\times$ time interaction ($P = 0.028$; Figure 6). There was a steady rise in serum CRP concentrations from baseline on day 1 of the exercise trial to baseline on day 2. Thereafter, a sharp rise was observed in serum CRP concentrations at 3 h into the postprandial period on day 2 of the exercise trial. Serum CRP then dropped slightly between 3 and 6 h into the postprandial period. In the control trial serum CRP fell slightly between the mornings of days 1 and 2. The serum CRP concentration then remained steady throughout day 2 of the control trial. Serum CRP concentrations are not always normally distributed. When serum CRP concentrations in the current study were tested for normality using a Shapiro–Wilks test this was found to be the case. A logarithmic transformation was applied to these data, and a Shapiro–Wilks test confirmed normality with the new data set. Repeated-measures ANOVA applied to the normalized data revealed no difference in the pattern of serum CRP concentrations between trials: trial $\times$ time interaction ($P = 0.101$).

The mean peak serum CRP concentration in the exercise trial was 2.08 mg/L. This was 249% higher than the mean baseline value (0.83 mg/L). Relationships between indices of serum CRP and indices of plasma TAG were examined. No significant correlations were observed between serum CRP and plasma TAG.

**Discussion**

There are 2 main findings in the current study. First, performing multiple bouts of resistance exercise the day before a test meal causes a significant reduction in postprandial TAG concentrations. This finding suggests that energy expenditure is a determinant in reductions in postprandial TAG concentrations with resistance exercise, because previous studies from this laboratory employing a lower energy expenditure failed to detect a reduction in postprandial TAG concentrations (4, 5). Second, a high-volume bout of resistance exercise causes a subsequent rise in serum CRP concentrations.
Different exercise intensities produce a similar reduction in postprandial TAG concentrations with aerobic exercise (34) when the total energy expenditure of the exercise bout is the same. The current study supports the proposal that total energy expenditure is also a determinant of the reduction in postprandial TAG concentrations after resistance exercise. The estimated total energy expenditure in the present study (5.10 MJ), however, is much greater than that seen in previous investigations examining the effect of resistance exercise on postprandial TAG concentrations (4, 5, 25, 29, 39). Two of those studies (25, 39) demonstrated reductions in postprandial TAG concentration with estimated energy expenditures (0.76–1.70 MJ) from resistance exercise much lower than that used in the current study. The other studies (4, 5, 29) examining this topic failed to observe any reduction in postprandial TAG after resistance exercise with estimated energy expenditures ranging from 0.57 to 2.58 MJ. The current evidence is not in agreement, and it appears that energy expenditure is not the only determinant of reduced TAG concentrations after resistance exercise. Further studies need to elucidate the other mechanism(s) involved.

Despite a statistically significant reduction in both the total and incremental areas under the TAG-concentration curves there was considerable interindividual variation in response to the exercise bout. Seven of 24 subjects demonstrated an increase in total TAG concentrations after exercise, whereas there was a reduction in 17 subjects. It could be argued that this was a control issue because subjects left
the laboratory after day 1 of each trial and some subjects could have consumed more food after the exercise trial than the control trial despite being asked to consume standardized meals. Nonetheless, we have previously observed (4) increased TAG concentrations in 1-d trials—resistance exercise and test meal given the same day—when control of diet was not an issue because subjects did not leave the laboratory. In addition to the difficulty of controlling participants’ energy intake, diets high in polyunsaturated fats or carbohydrates can affect postprandial TAG concentrations. We have no reason to suspect that any participants in the present study were following a diet high in either of these dietary constituents, but we did not analyze food diaries, and therefore some variation in subjects’ TAG concentrations might have resulted from a high dietary intake of either constituent. Despite these possible dietary influences it appears that postprandial TAG responses after resistance exercise are highly variable (both between and within studies), and future research needs to investigate the cause of these differences.

The considerable interindividual variation in the TAG response to the exercise bout in the current study is reflective of the variable findings to date on the effect of resistance exercise on postprandial TAG concentrations (4, 5, 25, 29, 39). Differing fitness levels between individuals in these studies might explain some of the variation. In addition, the current study used a long-duration, high-volume, low-intensity resistance-exercise protocol that subjects were not accustomed to. Responses of TAG to the exercise protocol in the current group might therefore be different from that of individuals accustomed to regular hard physical labor on a daily basis, such as the previously highlighted San Francisco dockworkers (21).

Although many individuals would not choose, or have time for, a pattern of exercise similar to that used in the current study the findings might be applicable to individuals involved in heavy manual labor. San Francisco dockworkers (21) who performed heavy work had a greater protective effect against coronary heart disease than their colleagues performing light or moderate work. Although the heavy work in the study on dockworkers did not involve solely lifting or carrying weight, the estimated rate of energy expenditure of the dockworkers (21.8–31.5 kJ/min) was similar to that generated by the exercise in the current study (22.5 kJ/min). The total estimated energy expenditure of the weight lifting (5.1 MJ), however, was much less than that expended by the longshoremen involved in heavy work over an 8-h day (7.9 MJ). Nonetheless, it is possible that one mechanism for protection from coronary heart disease in individuals involved in heavy manual labor is through favorable changes to postprandial TAG concentrations.

Two primary mechanisms have been proposed to explain the reduction in TAG after a bout of exercise. One is an increase in skeletal-muscle lipoprotein lipase activity (28), which might enhance the uptake of TAG into the previously exercised muscle, and the other is a reduced rate of hepatic very-low-density-lipoprotein (VLDL) TAG secretion (18). It is difficult to establish which influenced the reduction in TAG concentrations in the current study. There was a tendency toward increased NEFA concentrations in the exercise trial. Increased delivery of NEFA to the liver in the postprandial period could increase hepatic VLDL-TAG output. The significant increase in the ketone body 3-hydroxybutyrate in the exercise trial in this study, however, suggests that NEFAs were being oxidized rather than esterified in the liver, possibly in response to the energy debt created by the exercise. There is some evidence, therefore, for a decreased VLDL output in the exercise trial.
Increased insulin sensitivity is a well-known effect of aerobic exercise (38). It has also been seen with strength training (26). Reduced insulin concentrations in the current study suggest that resistance exercise in the absence of training also has the potential to increase insulin sensitivity. The reduced insulin concentrations observed after exercise in the current study might also have affected TAG concentrations because the activity of the enzyme skeletal-muscle lipoprotein lipase is decreased in muscle by insulin (16). Thus, lower insulin concentrations after resistance exercise might have lead to increased skeletal-muscle lipoprotein lipase activity and thus enhanced TAG removal from blood. Despite the decreased insulin concentrations with exercise in the current study, however, no indices of insulin and TAG were significantly related. This is consistent with findings from aerobic exercise studies in which no relationship was observed between the exercise-induced changes in fasting and postprandial TAG and insulin concentrations (13).

The finding in the current study that a high volume of resistance exercise causes a rise in serum CRP concentrations the next day has been observed previously (23) when a 168% over-baseline-value increase in serum CRP concentrations was seen 23 h postexercise. Concentrations of serum CRP in the current study peaked at 3 h into the postprandial period (approximately 11 AM) on day 2 of the exercise trial and were 149% higher than baseline. This was approximately 26 h after the start of the resistance exercise (9:00 AM, day 1) and 19.5 h after the end of the resistance exercise (3:30 PM, day 1). These data show a time and magnitude of response similar to those seen previously (23). Nonetheless, some caution needs to be exercised toward the reported finding of a rise in serum CRP concentrations after exercise in the current study. Serum CRP data were not normally distributed, and no rise in CRP concentrations was observed when data were normalized, and others studies have also seen no rise in CRP after exercise (20, 30).

If the current study’s finding that resistance exercise causes a rise in CRP concentrations is valid, the literature on the effect of acute resistance exercise on CRP concentrations is divided (20, 23, 30). Methodological differences between studies could explain the variation in findings. Many muscle contractions were performed by participants (1500 in the present study and 300 in a previous study [23]) in studies in which CRP concentrations increased after exercise in comparison with studies (20, 30) that found no effect of resistance exercise on CRP (no more than 60 contractions). Exercise duration was short—less than 7 min—in studies (20, 30) in which there was no effect of exercise on CRP concentrations. Finally, the muscle mass used by participants in the current study was larger than that in studies (20, 30) in which no difference was seen in CRP concentrations after exercise—multiple muscle groups compared with isolated muscle groups. All of these variables have been related to interleukin-6 response (10). Interleukin-6 is the main stimulant for hepatic CRP production (3) and might explain the discrepant findings between the studies.

It is thought that increases in CRP with acute strenuous aerobic exercise (9, 33) (marathons, ultramarathons, and triathlons) might be a result of the muscle damage induced by these types of exercise (33, 35). Postexercise muscle damage and increases in creatine kinase have been related to increases in interleukin-6 (3). We cannot provide evidence for this, however, because neither creatine kinase nor interleukin-6 was measured in the present study.
In summary, the current study demonstrated that multiple bouts of weight lifting caused a reduction in postprandial TAG concentrations. In combination with previous findings from this laboratory (4, 5), the current finding suggests that, as with aerobic exercise, energy expenditure is a determinant of the exercise-induced reduction in postprandial TAG concentrations. Other mechanisms might also be involved, however. The current finding might be most applicable to individuals involved in heavy manual labor. The current study also demonstrated that a high volume of resistance exercise can cause a significant postexercise increase in serum CRP concentrations. Therefore, in the short term, the benefit of resistance exercise to lipid metabolism might be offset by the concomitant inflammatory response. Further research is required to assess the long-term effects of regular resistance exercise on cardiovascular disease risk.

Acknowledgments

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References


