Acute Effects of Accumulating Exercise on Postprandial Lipemia and C-Reactive Protein Concentrations in Young Men

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

The current study investigated the acute effects of accumulating short bouts of running on circulating concentrations of postprandial triacylglycerol (TAG) and C-reactive protein (CRP). Ten men, age 21–32 yr, completed two 1-d trials. On 1 occasion participants ran at 70% of maximum oxygen uptake in six 5-min bouts (i.e., 8:30, 10, and 11:30 a.m. and 1, 2:30, and 4 p.m.) with 85 min rest between runs. On another occasion participants rested throughout the day. In both trials, participants consumed test meals at 9 a.m. and 12 p.m. In each trial, venous blood samples were collected at 8:30, 10, and 11:30 a.m. and 1, 2:30, 4, and 5:30 p.m. for plasma TAG measurement and at 8:30 a.m. and 5:30 p.m. for serum CRP measurement. Total area under the curve for plasma TAG concentration versus time was 10% lower on the exercise trial than the control trial ($M \pm SEM$: 13.5 ± 1.8 vs. 15.0 ± 1.9 mmol ∙ 9 hr⁻¹ ∙ L⁻¹; $p = .004$). Serum CRP concentrations did not differ between trials or over time. This study demonstrates that accumulating short bouts of running reduces postprandial plasma TAG concentrations (a marker for cardiovascular disease risk) but does not alter serum CRP concentrations.

Keywords: accumulation, triacylglycerol, inflammation, energy deficit, postprandial metabolism

Physical activity guidelines advise that adults engage in moderate-intensity activity for 30 min/day on most, preferably all, days of the week (Department of Health, 2004; Haskell et al., 2007). These reports also specify that activity can be accumulated in 10-min bouts to attain the 30-min total. The concept of accumulation is supported by findings from an observational study showing an association between accumulated physical activity and decreased risk of cardiovascular disease (CVD; Lee, Sesso, & Paffenbarger, 2000). Although the concept of accumulation is supported by epidemiological research, only three randomized controlled trials have examined the acute effects of accumulating short (<10-min) bouts of exercise on individual CVD risk factors (Miyashita, 2008; Miyashita, Burns, & Stensel, 2006a, 2006b).
To date, nine studies have reported that accumulated exercise lowers postprandial triacylglycerol (TAG) concentrations measured on the same day (Murphy, Nevill, & Hardman, 2000) or on the day after exercise (Altena, Michaelson, Ball, & Thomas, 2004; Barrett, Morris, Stensel, & Nevill, 2006, 2007; Burns, Miyashita, Ueda, & Stensel, 2007; Gill, Murphy, & Hardman, 1998; Miyashita, 2008; Miyashita et al., 2006a, 2006b). However, we could find no studies concerning the effect of accumulating short, <10-min, bouts of activity on postprandial lipemia measured on the same day that exercise is performed. This issue is important to examine for at least two reasons. First, partitioning exercise sessions into multiple short bouts throughout the day may be easier to accommodate in everyday life than longer bouts of exercise. Second, people in developed countries often spend most of their day in the postprandial state, and impaired clearance of TAG from the blood is a strong and independent risk predictor for CVD (Bansal et al., 2007; Nordestgaard, Benn, Schnohr, & Tybjærg-Hansen, 2007). Therefore, the current study examined the immediate (same-day) effect of accumulating short bouts of exercise on postprandial lipemia rather than the delayed (next-day) effects that have been examined in three previous studies (Miyashita; Miyashita et al., 2006a, 2006b).

In addition to postprandial lipemia, we examined C-reactive protein (CRP), a marker for inflammation and atherosclerosis (Libby, Ridker, & Maseri, 2002). Epidemiological studies have reported that elevated plasma CRP concentrations are associated with an increased risk of CVD in initially healthy men and women (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1997; Ridker, Hennekens, Buring, & Rifai, 2000). Although longitudinal and cross-sectional studies indicate that CRP is reduced with exercise training (for a review, see Kasapis & Thompson, 2005), several investigations have reported that CRP concentrations are increased 24–48 hr after an acute bout of exercise (Fallon, 2001; Siegel et al., 2001; Taylor et al., 1987). However, these studies measured CRP after prolonged strenuous exercise that may have caused local tissue injury. It is possible that CRP responses after shorter, more moderate exercise bouts in which little muscle injury occurs differ from responses after extended bouts of exercise.

Therefore, the purpose of the current study was to investigate the effects of accumulating short, 5-min, bouts of exercise (30 min in total), performed over the course of a single day, on postprandial plasma TAG and serum CRP concentrations measured on the same day in young men. We hypothesized that accumulating short bouts of moderate-intensity exercise would attenuate elevated postprandial plasma TAG and would not elevate serum CRP concentrations.

Methods

Participants

Ten healthy men (age 21–32 years) volunteered to participate in this study. All participants were recreationally active and had been weight stable (± 2.5 kg) for at least 3 months before the study. To minimize risks, participants were only recruited if they met the following criteria: were nonsmoking, were free of known CVD or abnormalities, were not taking any medication known to influence lipid or carbohydrate metabolism, had resting arterial blood pressure <140/90 mm Hg, and had a body-mass index <35 kg/m². Mean ± SEM values for the participants’ age, height, weight,
body-mass index, waist circumference, percentage body fat, and maximum oxygen uptake were 24.4 ± 1.4 years, 176.8 ± 1.8 cm, 71.2 ± 2.1 kg, 22.8 ± 0.6 kg/m², 78.0 ± 1.1 cm, 8.8% ± 0.7%, and 56.0 ± 4.1 ml · kg⁻¹ · min⁻¹, respectively. Loughborough University’s ethical advisory committee approved the study, and written informed consent was obtained from all participants before they took part in the study.

**Preliminary Exercise Tests**

Participants completed two preliminary exercise tests on a motorized treadmill (RUNRACE, Technogym, Gambettola, Italy): a 16-min submaximal test to establish the relationship between oxygen uptake and treadmill speed and a maximal oxygen-uptake test using an incremental uphill protocol at a constant speed until volitional fatigue (Taylor, Buskirk, & Henschel, 1955). Expired-air samples were collected during both tests using Douglas bags (Plysu Protection Systems, Milton Keynes, UK). Heart rate was monitored throughout both tests using short-range telemetry (Polar A3, Kempele, Finland), and ratings of perceived exertion were periodically assessed (Borg, 1973). Data generated from these two tests were used to determine running speed at 70% of each participant’s maximum oxygen uptake, and this speed was used for the main trials.

**Standardization of Diet and Exercise**

Participants weighed and recorded all food and drink they consumed for 2 days before the first main trial. They then consumed identical amounts of the same food and drink for 2 days before their second trial. Participants refrained from drinking alcohol during this time. In addition, they were asked to remain inactive for 2 days before each main trial and throughout the main trials (other than the exercise performed as part of the experiment). Food diaries were analyzed using computerized software (Comp-EAT Version 5.0. Nutrition Systems, London) to determine caloric intake and macronutrient content.

**Main Trials**

Each participant underwent two 1-day trials: an exercise trial and a control trial. There was a 7-day gap between trials, and the trials were performed in a randomized design. A schematic representation of the study protocol is shown in Figure 1. For each trial, participants reported to the laboratory at 8 a.m. after a 10-hr overnight fast (no food or drink except water). Participants sat in a semisupine position on a bed for 10 min after arriving at the laboratory. A cannula (Venflon, Becton Dickinson, Helsingborg, Sweden) was then inserted into an antecubital vein, and a baseline venous blood sample was collected at 8:30 a.m. Participants then either performed one 5-min bout of treadmill running at a running speed predicted to elicit 70% of maximum oxygen uptake (exercise trial) or rested (control trial) before consuming a test meal for breakfast at 9 a.m. In the exercise trial, participants then performed five additional 5-min runs at 10 and 11:30 a.m. and 1, 2:30, and 4 p.m. (i.e., six 5-min runs in total). Thus, there was an 85-min rest interval after each run. Expired-air samples were collected during the last minute of each exercise bout to determine energy expenditure during exercise. Heart rate was monitored during each bout of exercise using short-range telemetry. Ratings of perceived exertion were assessed
periodically during each bout of exercise using the Borg scale. In the control trial, participants rested throughout the day in the laboratory. During each trial a second test meal (identical to the first) was consumed at 12 p.m. Venous blood samples were collected immediately before each exercise bout or at equivalent time points during the control trial, and one final blood sample was collected at 5:30 p.m. in each trial to measure TAG, glucose, and insulin concentrations. For serum CRP measurement, blood samples were collected at 8:30 a.m. and 5:30 p.m. in each trial.

Test Meals

The test meal consisted of white bread, Cheddar cheese, butter, mayonnaise, potato crisps, whole milk, and milkshake powder. The meal was prescribed according to body mass and provided 0.69 g fat, 0.95 g carbohydrate, 0.31 g protein, and 46 kJ energy per kilogram body mass. The $M \pm SEM$ macronutrient content of each test meal was $49.3 \pm 1.5$ g fat, $64.4 \pm 2.0$ g carbohydrate, and $22.2 \pm 0.7$ g protein, which provided $3.29 \pm 0.10$ MJ (786 ± 24 kcal) energy (56% fat, 33% carbohydrate, and 11% protein). Participants were asked to consume each meal within 20 min. The time taken to consume each meal was recorded and replicated in subsequent trials. The times taken to consume the first meal (breakfast) and the second meal (lunch) were $14.1 \pm 0.9$ min and $12.8 \pm 1.1$ min ($M \pm SEM$), respectively. None of the participants reported nausea or any gastrointestinal discomfort during or after meals. Because the trial order was randomized participants consumed water ad libitum during each main trial, but the volume ingested was recorded in each trial.

Sample Collection and Analysis

Expired-air samples were collected into Douglas bags (Plysu Protection Systems, Milton Keynes, UK) during the final min of each 5-min bout of exercise during the exercise trial. Oxygen consumption and carbon dioxide production were determined from expired-air samples using a paramagnetic oxygen analyzer and an infrared carbon dioxide analyzer, respectively (Series 1400, Servomex, Crowborough, UK). Expired-air volumes were measured using a dry-gas meter (Harvard Apparatus, Edenbridge, UK) and corrected to standard temperature and pressure dry. Oxygen consumption and carbon dioxide production values were used to calculate energy expenditure (Frayn, 1983).

For plasma TAG, glucose, and insulin measurements, venous blood samples were collected into precooled 9-ml potassium EDTA-coated Monovette tubes (1.6 mg/ml; Sarstedt, Leicester, UK) and immediately centrifuged, (GS-15R centrifuge, Beckman Coulter, Fullerton, CA) at 4,000 rpm for 10 min at 4 °C. After separation, plasma was stored at –80 °C for later analysis. For serum CRP measurement, venous blood samples were collected into a precooled Monovette with clot activator. Thereafter samples were allowed to clot for 45 min at room temperature and then centrifuged at 4,000 rpm for 10 min at 4 °C. After separation, serum was dispensed into plain microtubes and stored at –80 °C for later analysis.

Concentrations of plasma TAG and glucose (Randox Laboratories, Crumlin, UK) were determined by enzymatic, colorimetric methods using a centrifugal analyzer (Cobas Mira Plus, Roche Diagnostic Systems, Basel, Switzerland). Plasma insulin concentration was measured by radioimmunoassay (MP Biomedicals, Orangeburg, NY). Serum CRP (high-sensitivity) concentrations were determined by enzyme-linked immunosorbent assay using commercially available kits (DRG
Exercise trial: participants performed six (08:30, 10:00, 11:30, 13:00, 14:30 and 16:00), 5-min runs (at 70% of maximal oxygen uptake) throughout the day with 85 min rest intervals between bouts.

Control trial: participants rested in the laboratory throughout the day.

Note: Blood samples were collected before performing each bout of exercise.

Figure 1 — Schematic representation of the study protocol.
Instruments GmbH, Marburg, Germany). Accuracy and precision were monitored using quality-control sera (Randox Laboratories for TAG and glucose and MP Biomedicals for insulin). Intra-assay coefficients of variation were 0.5% for TAG, 0.8% for glucose, 8.9% for insulin, and 10.7% for CRP. Hemoglobin concentration and hematocrit were measured at baseline and the end of observation period to estimate changes in plasma volume (Dill & Costill, 1974).

Statistical Analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS) software, version 12.0 for Windows (SPSS Inc., Chicago). Area under the curve values for plasma concentrations over time were calculated using the trapezium rule. Student’s t tests for correlated data were used to assess trial differences between fasting plasma/serum concentrations and area under the curve values. Two-factor analysis of variance (ANOVA) with repeated measures was used to examine differences in plasma/serum concentrations between trials over time. Statistical significance was accepted at the 5% level. Results are presented as $M \pm SEM$.

Results

Exercise Responses

Estimated gross energy expenditure during accumulated exercise bouts was $1.76 \pm 0.08$ MJ/30 min ($421 \pm 18$ kcal/30 min). The exercise intensity and heart rate attained during the accumulated exercise bouts were $72.9\% \pm 1.7\%$ of maximum oxygen uptake and $164 \pm 3$ beats/min, respectively. The median rating of perceived exertion during the accumulated exercise bouts was 12 (midway between fairly light and fairly hard). The respiratory-exchange ratio during accumulated exercise bouts was $1.00 \pm 0.01$ (mean of six running measurements).

Dietary Data

Analysis of food diaries revealed that energy and macronutrient intake did not differ significantly for 2 days before the control and exercise trials. The $M \pm SEM$ energy intake 2 days before the main trials was $10.1 \pm 0.9$ MJ ($2,424 \pm 225$ kcal). The $M \pm SEM$ energy intake 1 day before the trials was $8.4 \pm 0.8$ MJ ($2,008 \pm 201$ kcal). The $M \pm SEM$ dietary intakes of fat, carbohydrate, and protein were $90.8 \pm 18.0$, $317.6 \pm 19.8$, and $110.9 \pm 11.4$ g, respectively, 2 days before the trials and $75.2 \pm 10.5$, $255.8 \pm 32.7$, and $101.1 \pm 26.0$ g, respectively, 1 day before the trials.

Plasma and Serum Concentrations in the Fasted State

Plasma and serum concentrations in the fasted state before the first test meal in each trial are shown in Table 1. There was no difference in fasting plasma TAG, glucose, insulin, or serum CRP concentration between the exercise and control trials. Two participants were excluded from the CRP analysis because they had CRP values $>10$ mg/L, indicating that they had infection (Ridker, 2003). Further conversation with these participants revealed that they had been ill with a cold before the trial. CRP data are therefore presented for 8 participants.
Plasma and Serum Concentrations in the Postprandial State

Changes in plasma volume during the observation periods were small (exercise 0.8% ± 2.5%, control 0.6% ± 2.7%) and did not differ significantly between trials. Thus, plasma concentrations were not adjusted for changes in plasma volume. Plasma TAG responses are shown in Figure 2. Plasma TAG concentrations were lower in the exercise trial than the control trial. The total and incremental areas under the curve for plasma TAG concentration versus time are given in Table 2. These were 10% and 22% lower in the exercise trial than the control trial, respectively.

Table 1  Fasting Circulating Concentrations of Triacylglycerol, Glucose, Insulin, and C-Reactive Protein for the Exercise and Control Trials, $M \pm SEM$

<table>
<thead>
<tr>
<th>Trial</th>
<th>Triacylglycerol (mmol/L)</th>
<th>Glucose (mmol/L)</th>
<th>Insulin (pmol/L)</th>
<th>C-reactive protein (mg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise</td>
<td>0.99 ± 0.13</td>
<td>5.11 ± 0.14</td>
<td>140.2 ± 19.3</td>
<td>0.54 ± 0.30</td>
</tr>
<tr>
<td>Control</td>
<td>0.98 ± 0.12</td>
<td>5.05 ± 0.11</td>
<td>129.4 ± 14.5</td>
<td>0.35 ± 0.10</td>
</tr>
<tr>
<td>$p$</td>
<td>.989</td>
<td>.544</td>
<td>.534</td>
<td>.390</td>
</tr>
</tbody>
</table>

Note. Means were compared using Student’s $t$ tests for correlated data. $N = 10$ (C-reactive protein, $n = 8$).

Figure 2 — Mean ($\pm$ SEM) fasting and postprandial plasma triacylglycerol concentrations for the exercise (○) and control (■) trials ($n = 10$). The arrows indicate the treadmill run (5 min for each run). The black rectangles indicate consumption of the test meals. Data were analyzed using two-factor ANOVA with repeated measures. There was a main effect of trial ($p = .009$) and a main effect of time ($p = .001$).
Circulating concentrations of glucose/insulin and CRP throughout the test days are shown in Figures 3 and 4, respectively. On inspection of the raw data, 1 participant was found to have a very high fasting insulin value in the exercise trial. The reason for this is unknown. Fasting glucose and TAG values from this participant were within the expected range, and all other insulin values for this participant followed the expected pattern with feeding. Therefore, the fasting exercise value for this participant was replaced with a fasting value equal to the mean fasting value of the other 9 participants. Plasma glucose and serum CRP concentrations did not differ between trials or over time. Plasma insulin concentrations were lower in the exercise trial than the control trial and changed over time, peaking after each meal. Area under the curve values for glucose and insulin are shown in Table 2. There were no significant differences in the total or incremental area under the curve values for glucose between trials. The total area under the curve value for plasma insulin was significantly lower in the exercise trial than the control trial, but the incremental area under the curve values for insulin did not differ significantly between trials.

### Discussion

The main finding of this study is that postprandial plasma TAG concentrations are reduced in young men when they accumulate 30 min of running in 5-min bouts spaced throughout 1 day. Accumulated exercise did not influence CRP concentrations measured at the end of the trial day.

Although one study has shown that three 10-min bouts of brisk walking lower postprandial TAG concentrations measured on the same day (Murphy et al., 2000), the current study is the first to show that accumulating short, 5-min, bouts of activity lowers postprandial plasma TAG concentrations on the same day that the exercise is performed. The current study has advanced understanding of accumulated exercise and the associated energy deficit by demonstrating that accumulating 30 min of running in short bouts is effective in reducing postprandial plasma TAG concentrations. Our findings have practical importance because some individuals may prefer to accumulate activity in short bouts throughout the day rather than perform all of their exercise in one bout. Our findings are consistent with the data from studies.

<table>
<thead>
<tr>
<th></th>
<th>Exercise</th>
<th>Control</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total triacylglycerol (mmol · 9 hr⁻¹ · L⁻¹)</td>
<td>13.5 ± 1.8</td>
<td>15.0 ± 1.9</td>
<td>.004</td>
</tr>
<tr>
<td>Incremental triacylglycerol (mmol · 9 hr⁻¹ · L⁻¹)</td>
<td>4.8 ± 1.2</td>
<td>6.2 ± 1.0</td>
<td>.027</td>
</tr>
<tr>
<td>Total glucose (mmol · 9 hr⁻¹ · L⁻¹)</td>
<td>44.6 ± 1.5</td>
<td>46.6 ± 1.7</td>
<td>.196</td>
</tr>
<tr>
<td>Incremental glucose (mmol · 9 hr⁻¹ · L⁻¹)</td>
<td>1.6 ± 0.5</td>
<td>2.7 ± 0.5</td>
<td>.143</td>
</tr>
<tr>
<td>Total insulin (pmol · 9 hr⁻¹ · L⁻¹)</td>
<td>2,433 ± 249</td>
<td>2,893 ± 206</td>
<td>.045</td>
</tr>
<tr>
<td>Incremental insulin (pmol · 9 hr⁻¹ · L⁻¹)</td>
<td>1,507 ± 188</td>
<td>1,833 ± 205</td>
<td>.148</td>
</tr>
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</table>

Note. Incremental values indicate the area above fasting concentration. N = 10. Means were compared using Student’s t tests for correlated data.
examining postprandial TAG responses to continuous exercise performed on the same day as the postprandial monitoring (Hardman & Aldred, 1995; Katsanos & Moffatt, 2004; Schlierf, Dinsenbacher, Kather, Kohlmeier, & Haberbosch, 1987; Zhang, Thomas, & Ball, 1998). However, some studies have failed to detect an exercise-induced postprandial TAG-lowering effect when exercise is performed on

**Figure 3** — Mean (± SEM) fasting and postprandial (A) plasma glucose and (B) insulin concentrations for the exercise (○) and control (■) trials (n = 10). The arrows indicate the treadmill run (5 min for each run). The black rectangles indicate consumption of the test meals. Data were analyzed using two-factor ANOVA with repeated measures. There was a main effect of trial (p = .037) and a main effect of time (p < .0005) for insulin.
the same day as postprandial monitoring (Petridou et al., 2004; Pfeiffer, Wenk, & Colombani, 2006). The lack of change in these studies may have been the result of insufficient energy expenditure during exercise (Petridou et al.) or the duration of blood-sample collection (Pfeiffer et al.), which may not have continued for long enough after the cessation of exercise to detect differences.

The mechanisms for the exercise-induced lowering of postprandial plasma TAG observed in the current study may be related to the lowered postprandial plasma insulin concentrations in the exercise trial. Insulin inhibits skeletal-muscle lipoprotein lipase activity (Kiens, Lithell, Mikines, & Richter, 1989), and thus reductions in insulin concentration may lead to increased skeletal-muscle lipoprotein lipase activity and hence enhanced TAG clearance. Indeed, insulin itself is a strong independent risk factor for CVD (Després et al., 1996), suggesting that a regular pattern of accumulated physical activity might reduce the risk of CVD by keeping insulin concentrations low. In addition, it is possible that accumulated exercise increases the exposure of TAG to skeletal-muscle lipoprotein lipase through an increase in muscle blood flow for a prolonged period throughout the day. Alternatively, the exercise-induced reductions in postprandial plasma TAG concentrations observed in this study may be related to lowered hepatic very-low-density-lipoprotein TAG secretion (Gill, Frayn, Wootton, Miller, & Hardman, 2001) or increased adipose-tissue lipoprotein lipase activity (Taskinen & Nikkila, 1987) through the energy deficit created by exercise. In the current study, caloric intake (MJ/day) in the exercise trial was matched to that of the control trial—hence participants were in energy deficit in the exercise trial. Indeed, a previous study demonstrated that the TAG-lowering effect of exercise is only evident with an accompanying negative
energy balance (i.e., exercise-induced energy deficit) and not with energy replacement (i.e., increased energy intake to compensate for the energy expended during exercise; Burton, Malkova, Caslake, & Gill, 2008). Therefore, the energy deficit created by accumulated exercise is likely to be an important mediator of the reduction in postprandial lipemia observed in the current study. In addition, because the participants were running in close proximity to meals, we speculate that a reduction in gastrointestinal blood flow may have occurred in the exercise trial. This could have contributed to the reduction of postprandial lipemia by slowing gut fat absorption and chylomicron secretion.

It is difficult to determine the predominant mechanism for reducing lipemia in the current study. However, skeletal-muscle lipoprotein lipase activity peaks >8 hr after exercise (Seip, Mair, Cole, & Semenkovich, 1997). For this reason and because of the increased blood flow from exercise throughout the day, we speculate that reduced hepatic TAG secretion and increased exposure of TAG to lipoprotein lipase as a result of increased blood flow are likely potential mechanisms for the lowering of postprandial plasma TAG concentrations in the current study.

The magnitude of the reduction in postprandial plasma TAG concentrations was smaller in the current study than we reported in a similar study that involved ten 3-min bouts of running at 70% of maximum oxygen uptake (Miyashita et al., 2006b): 10% lowering of the plasma TAG area under the concentration versus time curve in the current study versus a 22% lowering in our previous study. The major difference between these studies is that postprandial testing was conducted on the day after exercise in our previous study. This suggests that the TAG-lowering effects of exercise begin soon after the initiation of accumulated exercise, but optimal reductions occur the next day, possibly because skeletal-muscle lipoprotein lipase activity peaks >8 hr after exercise (Seip et al., 1997). A useful avenue for future research might be to measure postprandial lipemia on consecutive days—the day of exercise and the day after. This would give a more comprehensive picture of the postprandial responses to exercise.

Previous studies examining CRP responses after an acute bout of exercise have used strenuous and prolonged activity bouts (Fallon, 2001; Siegel et al., 2001; Taylor et al., 1987; Weight, Alexander, & Jacobs, 1991). In the current study accumulating short bouts of running throughout the day did not alter serum CRP concentrations measured 9 hr after the first bout of running. One reason for this may be the short duration of the exercise bouts. It is possible that the acute-phase inflammatory response was minimal compared with investigations in which physical activity was prolonged and strenuous. It is also possible that changes in serum CRP are not seen for at least 24 hr postexercise. Thus, CRP concentrations may have risen the day after exercise in the current study. However, previous studies have shown that a 30-min bout of moderate-intensity walking (Davis et al., 2008) or running (Plaisance et al., 2007) does not alter CRP concentrations measured 24 hr postexercise. Nonetheless, further research on short bouts of moderate- to high-intensity exercise is needed to examine CRP responses over extended postexercise periods.

One issue worthy of mention before concluding this discussion is the nature of the study design. Although the exercise duration was consistent with guidelines for health (Department of Health, 2004; Haskell et al., 2007) in this study, the total energy expenditure was still high (1.76 MJ = 421 kcal) compared with the minimum expenditure (200 kcal) recommended in public health guidelines (Pate et al., 1995).
In addition, the exercise intensity (70% of maximum oxygen uptake) is still not applicable for many middle-aged and older adults. Therefore, further research is required to determine whether the findings reported here apply in middle-aged and older adults performing low-intensity exercise such as walking. It is worth noting, however, that a previous study involving sedentary middle-aged adults demonstrated that 30 min of brisk walking performed in one continuous bout or in three 10-min bouts reduces postprandial plasma TAG concentrations measured over a 12-hr period. The reduction in postprandial TAG concentrations was comparable in both exercise trials (Murphy et al., 2000).

In summary, the findings of the current study demonstrate that accumulating short 5-min bouts of running at 70% of maximum oxygen uptake throughout the day for a total of 30 min is effective in reducing postprandial plasma TAG and insulin concentrations measured on the same day in young men. These findings suggest two possible mechanisms (i.e., reduced postprandial TAG and insulin concentrations) that might explain epidemiological findings indicating that accumulated physical activity lowers the risk of CVD. Moreover, accumulating 30 min of exercise at 70% of maximum oxygen uptake does not increase serum CRP concentrations, indicating that such exercise does not lead to an acute-phase inflammatory response.

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M.M. and D.J.S. conceived the study. M.M. supervised the data collection, performed blood analysis, and wrote the first draft of the manuscript. S.F.B. assisted M.M. with data collection and blood analysis and edited the manuscript. D.J.S. performed the venous cannulations. All authors contributed to the writing of the manuscript. None of the authors had a conflict of interest regarding any aspect of this research.

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