Effect of Prior Exercise on Postprandial Triglycerides in Overweight Young Women After Ingesting a High-Carbohydrate Meal

Joel B. Mitchell, James R. Rowe, Meena Shah, James J. Barbee, Austen M Watkins, Chad Stephens, and Steve Simmons

To examine the effect of prior exercise on the postprandial lipid response to a high-carbohydrate meal in normal-weight (NW = BMI <25) and overweight (OW = BMI ≥25) women (age 18–25), 10 NW and 10 OW participants completed 2 conditions separated by 1 month. In the morning, the day after control (CT = no exercise) or exercise conditions (EX = 60 min cycling at 60% VO\textsubscript{peak}), participants consumed a high-carbohydrate meal (80% CHO, 15% protein, 5% fat; 75 kJ/kg BM) followed by 6 hr of hourly blood sampling. Blood was analyzed for triglycerides (TG), blood glucose (BG), and insulin (IN). TG levels over the 6-hr period were lower in NW than OW (p = .021) and lower in EX than in CT (p = .006). Area under the curve (AUC) for TG was lower in NW than OW (p = .016) and EX than CT (p = .003). There were nonsignificant tendencies for reduced BG over time (p = .053) and AUC (p = .083), and IN AUC was lower in EX than in CT (p = .040) for both groups and lower in NW than in OW (p = .039). Prior exercise improved TG levels after a high-carbohydrate meal in both groups, and OW women demonstrated a greater postprandial lipemic response than NW regardless of condition. There were tendencies for improved glucose removal with prior exercise in NW vs. OW. Acute exercise can improve postprandial TG responses and might also improve postprandial BG and IN after a large meal in NW and OW young women.

Keywords: blood glucose, insulin, lipemia

Elevated fasting blood lipid levels are a risk factor for cardiovascular disease (CVD), and serum triglycerides (TG) are a significant part of the lipid-based CVD-risk profile because they interact with the lipoproteins to contribute to the atherosclerotic process (Gotto, 1998; Hardman, Lawrence, & Herd, 1998). Postprandial lipemia (PPL) is also a component of this risk profile because individuals consuming their meals according to a typical Western dietary pattern might create repeated,
and therefore prolonged, states of elevated lipids, especially TG (Carstensen et al., 2004). High-fat diets typically produce high PPL responses; however, high-carbohydrate (CHO) diets also have been shown to produce an elevated PPL in some individuals, particularly those with elevated fat mass (Dallongeville et al., 2002; Koutsari, Karpe, Humphreys, Frayn, & Hardman, 2001; Sharman, Gomez, Kraemer, & Volek, 2004). A low-fat, high-CHO diet has been recommended for its effect on lowering LDL cholesterol; however, in the sedentary, overweight population, this recommendation might not be effective because an elevated PPL might counteract other possible benefits by increasing TG (Abbasi et al., 2000; Chahoud, Aude, & Mehta, 2004).

Individuals who are overweight (body-mass index [BMI] ≥25) or obese (BMI ≥30) have an increased risk of CVD and other chronic diseases such as Type II diabetes. Relative to PPL, increased fat mass is an important factor, particularly considering that greater PPL has been observed with obesity (Dallongeville et al., 2002). Elevated postprandial insulinemia and fat mass are also associated, and aside from the obvious relationship to Type II diabetes, insulin also affects PPL because of its effect on lipoprotein lipase activity (Zhang, Ji, Fretwell, & Nunez, 2006).

An additional consideration when examining the interaction between excess fat mass, PPL, dietary composition, and the risk of CVD is the fact that a regular regimen of aerobic exercise has been shown to reduce PPL (Hardman et al., 1998; Herd et al., 2000; Koutsari et al., 2001; Tsetsonis, Hardman, & Mastana, 1997). Although trained individuals exhibit a reduced PPL, there is evidence that this response might result, at least partially, from the influence of the most recent bout of exercise (Hardman et al.; Herd et al.; Malkova, Hardman, Bowness, & Macdonald, 1999; Tsetsonis et al.; Zhang, Thomas, & Ball, 1998); thus, a trained individual might benefit from the most recent single bout of exercise’s exerting its effects on PPL for at least the next 24 hr. Furthermore, as a potentially beneficial therapeutic intervention for sedentary, overweight individuals, exercise has been shown to counteract the elevated PPL observed with high-CHO diets (Gill & Hardman, 2003; Koutsari et al.).

Although the relationship between excess fat mass, exercise, and a high-CHO diet has been studied in various combinations, particularly in older individuals, to our knowledge, the influence of a single bout of exercise in a young, overweight population on PPL after the intake of a high-CHO test meal has not been investigated. The examination of a disease-free group of young overweight women is meaningful because these individuals are not typically monitored for responses that are indicative of future risk of diseases associated with the metabolic syndrome. Information derived from this population might provide the basis for clinical dietary and exercise recommendations given as a preventive measure to sedentary, overweight individuals at an early age.

CVD, Type II diabetes, and obesity are associated with an elevated inflammatory state that is indicated by elevations in such markers as C-reactive protein (CRP; Kahn, Zinman, & Haffner, 2006; Matsuda & DeFronzo, 1999; Thorand et al., 2007). There is evidence that those who exercise on a regular basis have reduced levels of inflammatory markers (Milani, Lavie, & Mehra, 2004). Despite evidence that both lipid levels and glucose responses are positively affected by prior exercise, there has been little work describing the responses of CRP after acute exercise.

The primary purpose of this investigation, therefore, was to determine the effects of a single bout of exercise on TG levels after a high-CHO feeding in sedentary
normal-weight and overweight young adult women. We also examined the influence of body composition and exercise on additional metabolic factors, including blood glucose and insulin levels. In addition, resting levels of an inflammatory-based CVD risk factor, CRP, were measured with and without prior exercise. We hypothesized that the increase in postprandial TG, glucose, and insulin responses would be greater in the overweight than in the normal-weight women and that the single bout of exercise would reduce the lipemic and glycemic response in both groups.

Methods

Participants
Ten sedentary, overweight (BMI ≥25, waist circumference >88 cm) women and 10 sedentary, normal-weight (BMI <25) women were selected as participants. All completed a medical-history questionnaire to screen for any contraindications to exercise, and they read and signed an approved consent form before participating in the study. Sedentary participants were defined as individuals who had not exercised on a consistent basis (≥2 days/week) over the preceding 6 months.

Experimental Design
Based on their body-composition measurements, the participants were assigned to either a normal-weight (BMI ≤24.5) or an overweight (BMI ≥25.0) group (Table 1). Each participant underwent two testing sequences: an experimental condition (EX) with an exercise bout and a control condition (CT) with no exercise. The participants were randomly assigned to treatment order using a balanced crossover design. The CT and EX conditions were separated by approximately 1 month and were conducted at the same phase of the menstrual cycle based on self-report.

Preliminary Testing
The initial testing consisted of determining BMI, percent body fat, and hip and waist circumference. BMI was determined using a ratio of mass (kg) to height (meters squared), and percent body fat was measured using a seven-site skinfold formula. After this was completed, each participant was acclimated to exercise on a semirecumbent cycle ergometer for approximately 20 min at a range of low exercise intensities. During this session, a rating of perceived exertion (Borg scale of 6–20) and heart-rate data were collected and used to determine each participant’s starting workload for the maximal test.

On the next visit to the laboratory, each participant completed a test for peak aerobic capacity (VO₂peak) on the ergometer using an incremental graded protocol with increases of 25 W per stage. Expired gases were collected and measured on a computer-based gas-analysis system (Fitco-Max 1, Physiodyne Instruments, Quogue, NY), heart rate was monitored via telemetry (Polar Heart Watch), and blood pressure was determined by sphygmomanometry. Standard termination criteria were used for this test. The results of the VO₂peak test were used to determine the resistance needed to elicit 60% of VO₂peak for the EX trials. After all the preliminary testing was completed, all participants continued to refrain from any structured physical activity for at least 1 week before initiating the subsequent trials.
<table>
<thead>
<tr>
<th></th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Age (years)</th>
<th>Body-mass index (kg/m²)</th>
<th>% Fat</th>
<th>Waist circumference (cm)</th>
<th>Waist:hip ratio</th>
<th>VO₂peak (L/min)</th>
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<tr>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>$M$</td>
<td>63.0</td>
<td>61.10</td>
<td>18.9</td>
<td>22.17</td>
<td>22.71</td>
<td>72.67</td>
<td>0.853</td>
<td>1.97</td>
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<td>1.42</td>
<td>1.52</td>
<td>0.28</td>
<td>0.70</td>
<td>1.37</td>
<td>1.88</td>
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<td></td>
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<td>17.97–24.55</td>
<td>17.1–28.7</td>
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<td></td>
</tr>
<tr>
<td>$M$</td>
<td>66.12</td>
<td>85.08*</td>
<td>21.2</td>
<td>29.94*</td>
<td>36.10*</td>
<td>92.56*</td>
<td>0.900*</td>
<td>2.17</td>
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<tr>
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<td>4.63</td>
<td>0.53</td>
<td>1.16</td>
<td>1.12</td>
<td>2.59</td>
<td>0.02</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>25.2–37.5</td>
<td>29.4–37.5</td>
</tr>
</tbody>
</table>

*Significant difference between groups.
Experimental Testing

Each sequence for both the CT and EX conditions consisted of the 2 days of dietary control followed by 2 days of testing. During the 2 days before the EX and CT conditions, the participants consumed a standard mixed diet (55% CHO, 30% fat, and 15% protein), and they were asked to refrain from nicotine or alcohol use and any structured exercise. If they were accustomed to drinking coffee, their caffeine intake was limited to no more than the equivalent of two cups per day.

On the first test day, participants in the EX condition continued with their standardized diet, and during the afternoon (3–5 p.m.) they completed 60 min of semirecumbent cycling at 60% of their VO\textsubscript{2peak}. During the exercise test, VO\textsubscript{2}, heart rate, and blood pressure were monitored. On the second test day, after an overnight fast, the participants returned to the laboratory (between 8 and 10 a.m.) for the test-meal protocol. In the CT condition participants also ate a standardized diet but refrained from any structured physical activity. After voiding their bladders, the participants assumed a supine position and a flexible Teflon catheter was inserted into an antecubital or forearm vein and maintained patent with a saline flush. A baseline blood sample was obtained at this time. Each participant then ingested a high-CHO meal consisting of an energy load of 72 kJ/kg BM with the following balance of macronutrients: 80% CHO, 15% protein, and 5% fat. The CHO content of the meal was selected based on the work of Dallongeville et al. (2002), and the energy load of the meal was based on previous work examining the effect of prior exercise on PPL (Altena, Michaelson, Ball, & Thomas, 2004; Gill, Murphy, & Hardman, 1998; Hardman et al., 1998). The CHO meals were a blended drink composed of fat-free cottage cheese, strawberry jam, and a dextrin–maltose mixture (Dallongeville et al., 2002). Participants were instructed to finish this meal in no more than 20 min, and the consumption time was duplicated for each condition. After ingestion of the meal, blood samples were taken every 30 min for the first 2 hr and then every 60 min for the next 4 hr. Participants were allowed to drink water during the 6-hr sampling period.

Blood Analyses

All blood samples were collected in 5-mL evacuated collection tubes treated with EDTA. A small aliquot of whole blood was removed from the premeal sample for analysis of hematocrit and hemoglobin (microcapillary tube and cyanomethemoglobin methods, respectively), which was used to determine changes in plasma volume. The remainder of the blood was centrifuged at 2,000 RPM, and the plasma was removed and stored at –80 °C until the samples were analyzed in batches at a later date. All blood samples were assayed for plasma glucose, TG, and insulin levels. The glucose and TG assays used enzymatic, spectrophotometric methods; insulin was analyzed by radioimmunnoassay. All samples for a given participant were conducted in the same batch with an intra-assay coefficient of variation for blood glucose, TG, and insulin of less than 6.0%. The premeal samples were also assayed for CRP, which was measured via an ELISA method.
Calculations

Area under the 6-hr sampling curve (AUC) for the TG, plasma glucose, and plasma insulin results was calculated using the trapezoidal method. The composite insulin-sensitivity index was calculated using the formula described by Matsuda and DeFronzo (1999). This index represents whole-body insulin sensitivity and is calculated as 10,000/square root of [(FPG x FPI) x (mean glucose x mean insulin)], where FPG = fasting plasma glucose and FPI = fasting plasma insulin. The mean glucose and insulin values are from the postfeeding measurements and are expressed as mg/dL and µU/mL, respectively.

Statistical Analysis

The time-course data were analyzed using a three-factor analysis of variance (ANOVA) with repeated measures on the second (condition: exercise and non-exercise) and third (time) factors. The first factor was a between-groups (normal weight and overweight) factor. The AUC and other analyses that did not involve the time-course blood sampling were analyzed using a two-factor, group-by-condition ANOVA with repeated measures on the second factor. The Huyn–Feldt correction was used for repeated-measures analysis. Significant differences detected by the ANOVA were clarified using a Newman–Keuls post hoc test. Significance was accepted at the \( p < .05 \) level.

Results

Dietary Control and Metabolic Exercise Data

Data on heart rate, oxygen uptake, and caloric expenditure for the EX trials are displayed in Table 2. There were no differences between groups in these measures. The dietary control data for the 2 days before each trial are shown in Table 3.

Triglycerides

The TG time-course responses demonstrated significant group (\( p = .021 \)) and exercise (\( p = .006 \)) main effects. In general, the responses in the normal-weight group were lower than in the overweight group, and the responses in the EX condition were lower than those in the CT condition. The prefeeding TG values were 0.72, 0.69, 1.27, and 1.06 mM for normal-weight exercise, normal-weight nonexercise, overweight exercise, and overweight nonexercise, respectively. The postfeeding responses showing the group and condition differences are indicated in Figure 1(a). The AUC analysis for this variable also showed significant group (\( p = .016 \)) and condition (\( p = .003 \)) main effects (Figure 1[b]), with lower areas in the normal-weight than in the overweight group and in the EX than the CT condition. There were no significant changes in plasma volume over the course of the sampling period; thus, for this and all other blood variables, no adjustments for plasma volume changes were necessary.
**Table 2  Metabolic Data During the Exercise Trials**

<table>
<thead>
<tr>
<th>Group</th>
<th>VO&lt;sub&gt;2&lt;/sub&gt; (L/min)</th>
<th>VO&lt;sub&gt;2&lt;/sub&gt; %max</th>
<th>Respiratory-exchange ratio</th>
<th>Total kcal</th>
<th>CHO energy (kcal)</th>
<th>Fat energy (kcal)</th>
<th>kcal/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal weight</td>
<td>1.14 ± 0.18</td>
<td>58.7 ± 2.5</td>
<td>0.82 ± 0.01</td>
<td>298.3 ± 15.3</td>
<td>115.4 ± 30.0</td>
<td>182.8 ± 52.13</td>
<td>4.92 ± 0.30</td>
</tr>
<tr>
<td>Overweight</td>
<td>1.43* ± 0.26</td>
<td>65.7* ± 1.57</td>
<td>0.81 ± 0.01</td>
<td>378.4* ± 21.1</td>
<td>140.7 ± 52.0</td>
<td>237.6 ± 73.8</td>
<td>4.51 ± 0.24</td>
</tr>
</tbody>
</table>

*Note.* Values are the $M ± SE$.

*Significant difference between groups.

**Table 3  Macronutrient Intake Before the Nonexercise and Exercise Trials**

<table>
<thead>
<tr>
<th>Condition</th>
<th>kcal</th>
<th>% CHO</th>
<th>% Fat</th>
<th>% Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal weight no exercise</td>
<td>1,730.59</td>
<td>54.42</td>
<td>30.28</td>
<td>15.42</td>
</tr>
<tr>
<td>Normal weight exercise</td>
<td>1,755.12</td>
<td>54.95</td>
<td>29.73</td>
<td>15.41</td>
</tr>
<tr>
<td>Overweight no exercise</td>
<td>1,725.46</td>
<td>53.39</td>
<td>30.75</td>
<td>15.84</td>
</tr>
<tr>
<td>Overweight exercise</td>
<td>1,690.09</td>
<td>53.95</td>
<td>30.37</td>
<td>15.66</td>
</tr>
</tbody>
</table>

*Note.* Each value is an average of the 2 days before each test day.
There were no main effects for either group ($p = .13$) or exercise ($p = .16$) for the blood glucose time-course analysis (Figure 2[a]). The time-by-group interaction for glucose approached a significant difference ($p = .053$). There was also a tendency ($p = .083$) for a group main effect when the AUC for the glucose responses was analyzed (Figure 2[b]). There was a group-by-condition-by-time interaction for the time-course insulin responses ($p = .029$). The differences identified by the post hoc analysis are indicated in Figure 3(a). For the insulin AUC, there was no group-by-condition interaction; however, there was a group main effect, indicating that the normal-weight responses were lower than the overweight responses ($p = .039$). There was also a significant condition effect, indicating that the responses in the EX condition were lower than in the CT condition ($p = .040$; Figure 3[b]). The insulin-sensitivity indexes calculated for the postfeeding responses were not different from each other: $12.99 \pm 1.71$ and $12.37 \pm 1.98$ for the normal-weight exercise and no-exercise conditions, respectively, and $12.91 \pm 3.72$ and $9.09 \pm 1.92$ for the overweight exercise and no-exercise conditions, respectively, $M \pm SE$. 

**Glucose and Insulin**

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**Figure 1** — (a) Triglyceride responses ($M \pm SE$) plotted for each time point before (0 hr) and after the test meal. (b) Triglyceride responses plotted as the area under the curve (AUC). *Both normal-weight responses are different from both overweight responses (group main effect). †Exercise is lower than nonexercise for both groups (condition main effect).
Figure 2 — (a) Blood glucose responses ($M \pm SE$) plotted for each time point before (0 hr) and after the test meal. (b) Blood glucose responses plotted as the area under the curve (AUC).

**CRP**

The mean ($\pm SE$) values for CRP were 1.19 ± 0.72, 1.03 ± 0.65, 1.27 ± 0.37, and 2.38 ± 1.06 µg/mL for normal-weight exercise, normal-weight nonexercise, overweight exercise, and overweight nonexercise, respectively. These means are based on an analysis of the premeal blood sample only. No significant differences were found among these values.

**Discussion**

The primary finding of this investigation was that a single bout of exercise reduced the postprandial lipemic response, as indicated by circulating TG levels, in normal-weight and overweight young women after they ingested a high-CHO meal. There was no interaction between body composition and exercise; thus, both groups experienced an equal amount of exercise-induced reduction in TG. An additional
finding was that the lipemic response in the normal-weight participants was lower than that of the overweight participants regardless of the presence of prior exercise. Finally, prior exercise elicited tendencies and some significant differences in variables related to insulin resistance. These tendencies and differences were also found when normal-weight and overweight participants were compared.

**Control Data**

Because of the influence of diet on lipemic and glycemic responses, the standardization of diet before the test days was an important control variable in this investigation. The macronutrient intake (Table 3) of the participants was nearly...
identical for the 2 days before they reported to the laboratory for the test meal, and the participants consumed a percentage of calories from CHO, fat, and protein that would be considered a standard mixed diet. The total caloric intake was based on the self-reported customary diets of these women and was lower in the overweight group than would be expected based on the fact that their body composition is indicative of an energy surplus. Regardless of the low values reported, dietary variations likely did not have a significant impact on the postprandial lipemic responses, because the caloric intake and the balance of macronutrients were the same between the CT and EX trials in both groups for the 2 days before testing.

Based on the exercise metabolic data, despite nonsignificant mean differences, the relative intensity (percent of maximal aerobic capacity), the absolute VO\(_2\) (L/min), and the relative caloric expenditure (kcal/kg) were similar between the two groups. The total exercise load, as represented by the metabolic variables, is an important control issue because previous investigators have found that the intensity and total caloric expenditure of the prior exercise can influence subsequent PPL responses (Gill & Hardman, 2003; Petitt & Cureton, 2003). The exercise intensity of 60–65% of VO\(_2\)\(_{\text{peak}}\) is comparable to that reported in a number of studies in which PPL was reduced by prior exercise (Gill et al., 1998; Katsanos, Grandjean, & Moffatt, 2004; Tsetsonis & Hardman, 1996; Zhang et al., 2006); however, the total caloric expenditure was not as high as in some previous studies (1,100 kcal) that showed reduced PPL responses (Gill et al.; Katsanos et al.; Petitt, Arngrimsson, & Cureton, 2003). In fact, it has been suggested that total caloric expenditure is an important factor in stimulating reduced PPL (Petitt & Cureton). Conversely, there is evidence that exercise energy expenditure at a level comparable to that used in the current study (300–400 kcal) can reduce PPL (Altena et al., 2004; Koutsari et al., 2001). Regardless of this discrepancy, the moderate intensity and 60-min duration, which represented a reasonably demanding, yet feasible, level of exertion for this low-fit population of women, were adequate to produce an altered PPL and insulin response. Furthermore, the greater energy contribution from fat observed in the current study was probably not a factor in subsequent PPL responses, because previous work has indicated that the exercise substrate is not an influential factor (Malkova et al., 1999).

**TG Data**

The TG responses over the 6-hr period showed little or no elevation as a result of the meal; in fact, there was not a significant main effect for time. This result occurred despite the use of a relatively high caloric load (72 kJ/kg body mass) compared with previous studies (60–70 kJ/kg; Altena et al., 2004; Gill et al., 1998; Hardman et al., 1998). Most of the group and condition differences in both the time-course response and the AUC are the result of differences that were already present at the prefeeding time point, especially in the overweight group. This tendency for a reduced fasting TG pool after an exercise bout has been observed in previous studies (Gill et al.; Petitt et al., 2003), and it has been suggested that reductions in endogenous TG stores might be partially responsible for the fasting reduction. The acute effects of prior exercise would explain the condition effects; however, the group effect would be a result of the influence of body composition or factors associated with the greater fat mass, which would be a chronic state independent of exercise-induced
changes in TG stores. Previous work has indicated that fat mass might negatively affect lipoprotein lipase activity (LPLA), thereby raising TG levels. Weight loss has been associated with an increase in lipoprotein lipase mRNA that might contribute to lower TG observed with a reduction in fat mass (Patalay, Lofgren, Freake, Koo, & Fernandez, 2005); thus, the higher TG levels in the overweight participants in the current study might be related to reduced LPLA.

Most previous researchers have studied lipemic responses after a high-fat meal, whereas our study was designed to examine the effects of a high-carbohydrate meal. Dallongeville et al. (2002) reported that massively obese participants exhibited a substantially lower lipemic response after a high-CHO meal than after a high-fat meal. It is likely that the magnitude of the lipemic responses would be greater after high-fat test meals; thus, the interaction between the presence of excess adiposity and the effects of exercise might be different than that observed in the current study. Future research should examine the possible interaction between exercise, levels of obesity, and the lipemic response.

The significant main effects for group and condition in the TG data, in the absence of a statistical interaction between these factors, indicate that the overweight participants had a higher lipemic response than the normal-weight participants under both conditions combined; however, the prior bout of exercise produced a lowering effect in both groups. The latter finding is in agreement with a number of previous reports in which prior exercise reduced PPL responses to a high-fat meal in nonobese participants (Kahn et al., 2006; Tsetsonis & Hardman, 1996; Tsetsonis et al., 1997; Zhang et al., 1998). The current study extends this finding to a group of apparently healthy overweight young adult women after the ingestion of a high-CHO meal. The exercise-induced reduction in both groups suggests that if overweight participants engage in 60 min of moderate-intensity, sustained aerobic exercise, they will benefit from this intervention to the same extent as their normal-weight counterparts.

The beneficial effects of exercise are thought to be mediated by an elevation in skeletal-muscle LPLA (Gill et al., 1998; Katsanos et al., 2004; Malkova et al., 2000). The increased LPLA might be associated with processes necessary to restore lowered intramuscular TG stores. Given the relatively low energy expenditure and substrate demands in the current study in combination with evidence from others that the amount and type of substrate used in the preceding exercise does not influence PPL, it is unlikely that the relationship between substrate depletion and LPLA explains the reduced PPL in the exercise condition (Malkova et al., 1999). If elevated LPLA occurred in the exercise condition, it might have been associated with a reduction in insulin-mediated inhibition of LPLA.

The main effect for group in the postprandial TG levels suggests that the overweight participants experienced a greater lipemic response than their normal-weight counterparts regardless of the presence of exercise. Women age 20–39 years in the increased-risk category (BMI >25 kg/m$^2$) have been found to have body-fat contents of 33–38%; thus, the 36% body fat of the overweight participants represents a substantially increased adiposity that is a significant component of their disease-risk profile (Gallagher et al., 2000). Dallongeville et al. (2002) reported that obese women experienced a greater postprandial lipemia than normal-weight controls; however, their participants were older and morbidly obese (age = 38 years, BMI = 50.8 kg/m$^2$). Our overweight group consisted of apparently healthy young
women with considerably less fat mass; thus, the current study extends previous observations of greater PPL in those with above-normal adiposity to a broader spectrum of individuals.

Both of the significant main effects related to the TG responses are clinically relevant. The exercise effect shows that a single bout of exercise can reduce baseline TG levels, which are then sustained near that lower level throughout the postprandial period. The exercise intervention thus provides a therapeutic measure that with a typical meal-consumption pattern can be applied in an effort to reduce a sustained lipemia after each meal in both normal-weight and overweight individuals. The group effect is important because it emphasizes the need for early implementation measures to prevent the development of potential disease-risk factors because even at a relatively young age, and even without severe obesity, these women displayed responses that can be indicative of elevated risk for metabolic disorders. Despite the group differences, it is of practical significance to note that based on the AUC analysis, the overweight participants experienced an exercise-induced reduction in the lipemic response that was comparable to that observed in the normal-weight controls.

**Glucose and Insulin Data**

There were tendencies for and outright differences between exercise and nonexercise conditions and between normal-weight and overweight participants for glucose and insulin responses. The *p* values less than .10 but not less than .05 for main effects by group (normal weight vs. overweight) and by condition (exercise vs. nonexercise) in the glucose time-course and AUC data suggest that both of these factors might be influential in altering glucose disposal. There are numerous reports in the literature to support the idea that prior exercise exerts a beneficial effect on this response (Van Baak & Borghouts, 2000; Wojtaszewski et al., 2003). In the case of insulin, the significant reduction in the AUC produced by exercise would further substantiate the beneficial effect of exercise in improving glucose homeostasis. It is possible that the lack of difference in glucose responses was achieved via the greater insulin responses; however, calculations of insulin sensitivity (Matsuda & DeFronzo, 1999) did not produce differences between conditions. An aspect of the aforementioned tendencies that has not been reported extensively in the literature is the effect of moderate obesity in young, apparently healthy women on glucose disposal. Based on these tendencies, it would appear that the overweight women already show a tendency to exhibit one of the symptoms of the metabolic syndrome. Given the near significance with the glucose and the differences in the insulin responses, it is likely that a larger participant population would provide enough power to allow clarification of these tendencies. As was the case with the TG responses, the tendency for decreased glucose disposal can be ameliorated with exercise. Obesity has been identified as a factor associated with insulin resistance. Studies conducted to determine the independent effects of exercise and weight loss suggest that both factors can improve insulin sensitivity, with the exercise effect likely a result of the carryover from the most recent bout of exercise (Racette, Evans, & Weiss, 2006; Ross et al., 2004; Van Baak & Borghouts). Although they are based on an examination of acute exercise and a cross-sectional analysis of body composition with no change in weight, the data from the current study support the
independent effects of both prior exercise and existing adiposity. Although these data do not provide a basis for examining the mechanisms of either effect, there are practical implications of the exercise response.

The differences in insulin levels might be significant relative to an interaction with the TG responses discussed previously. Because of the inhibitory effect of insulin on muscle LPLA and the tendency for insulin to enhance hepatic TG release, the lower insulin response in the normal-weight group and the exercise condition for both groups might have been associated with the lower TG levels observed. Our data do not allow us to specifically identify the presence of either of the insulin-mediated mechanisms. The only indication of these effects would be a statistical relationship between the AUC for insulin and the AUC for TG; however, the correlation between these variables was low \( (r = .39 \) for all scores combined) and nonsignificant.

**CRP Data**

CVD, Type II diabetes, and obesity are known to be associated with a general elevation in systemic inflammation, particularly in older individuals; thus, CRP levels might be an additional indicator of the presence of a metabolic state that might be influenced by fat mass or prior exercise in a manner similar to blood lipids and glucose-disposal variables (Kahn et al., 2006; Milani et al., 2004; Okita et al., 2004; Thorand et al., 2007). Despite differences in lipid and carbohydrate metabolism as a result of differences in fat mass and prior exercise, the similarity in the prefeeding CRP responses with and without exercise and the similarity between groups suggest that CRP levels do not parallel lipid and glucose responses. Our findings are in contrast to those of Kahn et al., who reported that obesity and inflammatory factors were highly related; however, most of their participants were diagnosed with the metabolic syndrome and had a higher fat mass than those in the current study. Elevations in CRP have been shown to be clinically significant; however, all the values observed in the current study were relatively low and were below those observed in populations with or at risk for CVD or Type II diabetes. The levels found for the young women in the current study were comparable to those reported by Kahn et al. in their older (52.9 years) participants without the metabolic syndrome (1.8–2.7 µg/mL).

Given that TG, glucose, and insulin respond in a favorable manner to recent bouts of acute exercise, we wanted to determine whether CRP as a marker of inflammation would respond in a similar manner. Previous work has suggested that chronic exercise is associated with reduced CRP levels; however, despite the fact that some other markers of inflammation such as IL-6 are known to respond to single bouts of exercise, little is known about how CRP responds to the acute effects of exercise. Based on the lack of difference between the EX and CT conditions, it might be that CRP is a marker more indicative of a chronic systemic state in which factors such as age and body composition along with regular exercise have a greater effect, particularly in populations who are older or have greater fat mass than those in the current study (Milani et al., 2004). With variables where regular exercise produces a beneficial response, there is often an acute effect of exercise or a recency effect that establishes a mechanistic relationship between the acute response and the improved chronic state. The present data do not provide
insight into this relationship; thus, future studies should examine the combination of variables such as age, gender, adiposity, and exercise effects that are associated with an improvement in the inflammatory state.

**Conclusion**

Prior exercise in the form of a single, 60-min aerobic bout reduced the TG response before and after the ingestion of a high-CHO meal. The fact that the differences were present before the ingestion of the meal suggests that the TG-lowering effects of exercise are independent of the postprandial period and might represent a sustained beneficial effect of exercise for those who exercise regularly. Despite the fact that the overweight young women showed a greater TG response before and during the postprandial period, they were able to derive TG-lowering effects from exercise similar to those of the normal-weight controls; thus, their greater adiposity does not interfere with the beneficial effects of exercise. In addition, tendencies for better glucose-disposal responses in the normal-weight than in the overweight participants suggest that even moderate elevations in adiposity negatively affect this aspect of metabolism; however, exercise can improve glucose disposal in overweight young women. Finally, CRP as a marker of inflammation in this population was not influenced by body composition or prior exercise.

**References**


