Effects of acute sprint interval cycling and energy replacement on postprandial lipemia

Eric C. Freese, Ari S. Levine, Donald P. Chapman, Dorothy B. Hausman, and Kirk J. Cureton

Metabolism and Body Composition Laboratory, Department of Kinesiology, University of Georgia, Athens, Georgia

Submitted 7 April 2011; accepted in final form 16 August 2011.

High postprandial serum triglycerides (TG) lead to reduced serum high-density lipoprotein cholesterol and increased low-density lipoprotein cholesterol (12), impaired endothelial function (43), and increased atherosclerotic plaque formation (49), increasing cardiovascular disease risk. Repeated exposure to elevated serum TG increases atherogenic low-density lipoprotein cholesterol and decreases atheroprotective high-density lipoprotein cholesterol, indirectly stimulating atherosclerosis (18). Reducing hypertriglyceridemia is important to reduce the risk of cardiovascular disease risk.

Low-, moderate-, or high-intensity aerobic or resistance exercise has been shown to be an effective intervention for reducing postprandial serum TG. A reduction in fasting and postprandial serum TG of 20–25% occurs 14–18 h following an acute bout of moderately intense [50–70% maximum \( \dot{V}O_2 \text{max} \)] aerobic exercise (15–17, 20, 40) and may or may not occur following moderate- or high-intensity resistance exercise (6, 7, 29, 35). Exercise is thought to reduce postprandial lipemia (PPL) through increased skeletal muscle hydrolysis of TG and reduced hepatic output of very-low-density lipoprotein (VLDL) (25), along with an increase in skeletal muscle and hepatic blood flow (21).

The influence of intensity and duration of exercise on the magnitude of reduced PPL following exercise has been extensively investigated (40, 41, 46). Aerobic walking sessions of different intensities (3 h at 30% \( \dot{V}O_2 \text{max} \) and 1.5 h at 60% \( \dot{V}O_2 \text{max} \)), but constant energy expenditure, elicited a similar reduction in PPL (29, 41), indicating that energy expenditure is more important than the intensity or duration of exercise. Likewise, a single bout of continuous running at 60% \( \dot{V}O_2 \text{max} \) causes the same reduction in PPL as three 10-min bouts of intermittent exercise at the same intensity (1, 17). A moderately strong inverse correlation has been found between aerobic exercise energy expenditure and PPL reduction (\( r = -0.62 \) (30). While the magnitude of the reduction in PPL may partially be due to the energy expended during aerobic exercise, this relationship is not observed when the effects of aerobic and resistance exercise are compared. A decrease in PPL was found using resistance exercise of 3 sets of 10 repetitions of 10 exercises performed at the subjects’ 10 repetitions maximum, whereas aerobic exercise of the same energy expenditure showed no attenuation (29).

It is possible that the reduction in PPL following an acute bout of aerobic or resistance exercise is due to the energy deficit created by exercise. For this reason, replacement of the energy deficit created by exercise by eating after exercise may alter PPL. The reduction in PPL due to an exercise-induced energy deficit was negated when carbohydrate feedings equivalent to 105% of the energy deficit were given at 0, 2, and 4 h after 90 min of cycling at 70% \( \dot{V}O_2 \text{max} \) and ten 1-min sprints (19). Replacing the energy deficit with a postexercise mixed meal 30 min after walking at 50% \( \dot{V}O_2 \text{max} \) for ~90 min also negated the benefit of the exercise (8).

Sprint interval training is a form of low-volume, high-intensity exercise that has effects on skeletal muscle similar to more prolonged, moderate-intensity exercise (5). Because sprint interval exercise involves very high intensity (150–250% \( \dot{V}O_2 \text{max} \)), all muscle fiber types are thought to be recruited. Coyle (9) suggested that sprint interval training may have health benefits similar to moderate-intensity, high-volume exercise training, and studies on insulin sensitivity (2, 38) and endothelial function (37) have confirmed this hypothesis. High-intensity, low-volume exercise also may be an effective treatment in reducing PPL.

The primary purpose of the study was to determine the influence of sprint interval cycling (SIC) on PPL. The secondary purpose was to determine the effect of replacement of the energy deficit created by exercise on PPL. We hypothesized...
that SIC would reduce PPL compared with a nonexercise control, and that attenuation of PPL would be greater following SIC with energy deficit compared with SIC with energy balance.

METHODS

Participants. Twelve recreationally active young healthy adults, six men and six women, participated in this study. In young adults, there is no significant sex difference in fasting (10) or postprandial (45) plasma TG concentrations. Exclusion criteria included subjects who were smokers, had a body mass index >30 kg/m², had a history of cardiovascular disease or diabetes (type 1 or 2), had hypertension, or had any metabolic disease that involved the ingestion of medications that could affect carbohydrate or fat metabolism. Participants’ physical characteristics can be found in Table 1. Using G*Power 2 (http://www.psycho.uni-duesseldorf.de/abteilungen/aap/gpower3) (11), a sample size of 12 was sufficient to detect a moderate main effect (Cohen’s $d = 0.57$) for postprandial TG levels with an alpha level of 0.05 and a power of 0.8, assuming a correlation between repeated trials of 0.9. The study protocol was approved by the institutional review board, and all subjects gave written, informed consent following a description of the study procedures and risks.

Study design. A repeated-measures crossover study design was used in which each subject served as his or her own control. Following familiarization with the protocol, participants were tested under three different conditions in random order: control (Con), SIC with energy deficit (Ex-Def), and SIC with energy balance (Ex-Bal), separated by at least 1 wk. Each testing condition took place over 2 days, in which exercise or the resting control was performed on the day 1. On day 2, a high-fat mixed-meal (HFM) was consumed after a 13-h overnight fast. Serum TG and other responses to the meal were then measured for 3 h following the meal. Participants refrained from planned exercise and alcohol ingestion 48 h before the first day of testing.

Body composition. At the first visit to the laboratory, participants’ body composition was assessed using the dual-energy X-ray absorptiometry (iDXA, GE Healthcare, Fairfield, CT). The fat-free mass (FFM) was used to estimate the cycle resistance used for the SIC exercise session.

SIC treatment. Subjects ate their third meal of the day by 1600, giving at least 3 h to digest consumed food before the exercise session. During the first visit, subjects practiced SIC to familiarize them with the protocol. SIC is a high-intensity, low-volume exercise that involved four 30-s all-out sprints with 4 min of active recovery between each sprint. SIC was administered on a mechanically braked, stationary cycle ergometer (Monarch Ergomedic 874 E, Monarch, GIM, Stockholm, Sweden) with resistance on the pedals set to 0.088 kp/kg FFM (5). Following a 5-min warm-up, the first sprint began. Participants pedaled against the resistance as fast as possible for 30 s, attempting to achieve as many revolutions as possible. The number of revolutions and the total work performed on each sprint were quantified. An optical sensor (Sports Medicine Industries, St. Cloud, MN) was attached to the ergometer to measure flywheel revolutions. The sensor was interfaced to a computer and SMI Power software (version 1.02) recorded flywheel revolutions and calculated power output each second (W/s). To obtain the total work performed during each session, the power output for the 30-s sprint was summed and converted to Joules (J). Following each of the four sprints, participants actively cycled against no resistance for 4 min until the next sprint was started. The time spent during cycling and recovery totaled 18 min.

Energy expended during each SIC session was based on the estimated anaerobic energy contribution to a 30-s all-out effort (25 kcal) (27) and from the measured oxygen uptake. Participants’ oxygen uptake was measured continuously by open-circuit spirometry using a PARVO Medics TrueOne 2400 Metabolic Measurement System (Parvıc Medics, Salt Lake City, UT). Energy expenditure was measured to estimate energy replacement needed during the energy balance exercise testing session.

Treatment protocol. On day 1 between 1900 and 1930 of each testing session, participants performed SIC in the laboratory (for Ex-Def or Ex-Bal) or rested at home without performing exercise. On days in which participants reported to the laboratory, BM was measured to use in energy expenditure estimation.

Between 2000 and 2030 during the Ex-Bal testing session, participants consumed a mixed meal provided by Zone Perfect Nutrition Bars (Abbott Nutrition, Columbus, OH) that replaced 100% of the energy depleted during the SIC session. A Zone Perfect Nutrition bar is composed of 210 calories with 7 g fat, 24 g carbohydrate, and 14 g protein. The bar was measured and cut so participants consumed the same energy as they expended during their exercise session. An energy bar with a mixture of macronutrients was used for energy replacement because it would be more typical of a meal consumed after exercise and because it facilitated glycogen resynthesis in the exercised muscles. Consumption of a carbohydrate and protein mixture following exercise has been shown to increase muscle glycogen levels faster than a carbohydrate alone (22). Muscle glycogen levels replenished through carbohydrate energy replacement attenuated the reduction in PPL after an acute bout of exercise (19).

HFM tolerance test. The HFM tolerance test was administered on day 2 of each testing session (35). It was administered ~14 h after each treatment protocol and after a 13-h overnight fast. On the morning of the HFM tolerance test, participants arrived at the laboratory by 0800 in a fasted state, performing as little physical activity as possible. After measuring BM, an intravenous catheter was inserted into an antecubital vein and kept patent with 0.5 ml of 10 USP units/ml heparin lock flush. The participant rested until a fasting blood sample was obtained at 0900. The subject consumed the test meal between 0930 and 1000. The meal had a macronutrient composition of 1.2 g fat, 0.9 g carbohydrate, and 0.4 g protein/kg BM and provided ~68 kcal/kg BM (Burger King, Miami, FL, http://www bk com/en/us menu nutrition index html), similar to the meal used in the study by Singhal et al. (35). The meal was a commercially available breakfast that consisted of a croissant, an omelet, two slices of cheese, four sausage patties, and hash browns/potatoes. To keep the macronutrient content of the meal consistent across the subjects and trials, each component of the meal was individually weighed and cut to account for the differences in BM among the participants. After the meal was consumed, participants’ neither ate nor drank anything for the following 3 h. PPL was assessed for 3 h to approximate the time between meals for this particular population and to reduce participant burden. Shorter PPL tests of 4 h have been used recently and have been demonstrated to be valid substitutions for the usual 8-h postprandial assessment (44), particularly when assessing total TG area under the curve (AUC), which was the primary outcome of the study. Furthermore, nonfasting TG concentrations acquired 2.4 h after a HFM provide a better estimation of an intervention’s effectiveness than TG measured later in the postprandial period (3).

Blood samples were taken at 0, 30, 60, 120, and 180 min postprandial. Water consumption was held constant across all trials to 4.3 ml/kg BM with the high-fat meal. Participants rested in a sitting position throughout the 3-h postprandial period, getting up only to use the restroom as needed.

---

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>$n$</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Age, yr</td>
<td>22.0 ± 3.2</td>
<td>20.8 ± 0.8</td>
</tr>
<tr>
<td>Height, cm</td>
<td>181.0 ± 5.1</td>
<td>166.6 ± 4.5</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>80.77 ± 9.6</td>
<td>61.5 ± 8.8</td>
</tr>
<tr>
<td>Fat, %</td>
<td>18.7 ± 4.3</td>
<td>27.8 ± 4.7</td>
</tr>
<tr>
<td>Fat-free mass, kg</td>
<td>66.24 ± 8.49</td>
<td>44.9 ± 4.3</td>
</tr>
</tbody>
</table>

Values are means ± SD; $n$, no. of subjects.
Table 2. Fasting serum/plasma concentrations of TG, insulin, glucose, BHB, and NEFA

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Ex-Def</th>
<th>Ex-Bal</th>
<th>Con</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>0.78 ± 0.60</td>
<td>0.85 ± 0.54</td>
<td>0.98 ± 0.69</td>
</tr>
<tr>
<td>Insulin</td>
<td>14.67 ± 4.44</td>
<td>13.90 ± 4.64</td>
<td>14.31 ± 4.76</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.11 ± 0.54</td>
<td>5.20 ± 0.47</td>
<td>5.14 ± 0.43</td>
</tr>
<tr>
<td>BHB</td>
<td>0.15 ± 0.16</td>
<td>0.13 ± 0.1</td>
<td>0.16 ± 0.18</td>
</tr>
<tr>
<td>NEFA</td>
<td>0.71 ± 0.33</td>
<td>0.72 ± 0.25</td>
<td>0.69 ± 0.32</td>
</tr>
</tbody>
</table>

Values are means ± SD in mmol/l. Ex-Def, exercise-deficit; Ex-Bal, exercise-balance; Con, control; TG, triglyceride; BHB, betahydroxybutyrate; NEFA, nonesterified fatty acids.

Analytic methods. At each blood sampling, 8 ml of blood were collected; the first 2 ml of blood were discarded, while the next 6 ml were collected into BD Vacutainer 3.0-ml serum separation tubes and 3.0-ml sodium-heparin tubes (Becton Dickinson, Franklin Lakes, NJ) for preparation of serum and plasma, respectively. The serum separation tubes were allowed to clot for 30 min before they were centrifuged at 5°C. The serum was separated and divided into aliquots and stored at −70°C until it was analyzed for TGs. Plasma was separated within 20 min of collection. The sodium heparin tubes were centrifuged for 10 min, and resulting plasma removed, divided into aliquots, and stored at −70°C until analyzed for insulin, glucose, nonesterified fatty acids (NEFA), and betahydroxybutyrate (BHB). Enzymatic, colorimetric assays were used to measure serum TG (Wako L-Type TG M assay, Wako Chemicals USA, Richmond, VA), plasma glucose (Wako Glucose C2 assay, Wako Chemicals USA), plasma BHB (Autokit 3-HB assay, Wako Chemicals USA), and plasma NEFA [Wako NEFA-HR (2) assay, Wako Chemicals USA]. Insulin was measured using a radioimmunoassay (RIA Kit, Human Insulin Specific, Millipore, Billerica, MA). Intra-assay coefficients of variation were 1.6% for glucose, 2.1% for insulin, 2.4% for TG, 3.0% for BHB, and 4.8% for NEFA.

Dietary analysis. Each participant’s diet was held constant for 2 days before each testing session. On day 1 of each testing session, participants were instructed to consume three meals during the day, eaten for 2 days before the first treatment and then repeated the same diet on subsequent treatments. Dietary records for the day of each testing session. On day 1 of each testing session, participants were instructed to consume three meals during the day, eaten for 2 days before the first treatment and then repeated the same diet on subsequent treatments. Dietary records for the day of each testing session. Dietary records for the day of each treatment were assessed using the United States Department of Agriculture National Nutrient Database for Standard Reference (http://www.nal.usda.gov/fnic/foodcomp/search/) to assess the quantity of food consumed.

Calculations and statistical analysis. Statistical analysis was performed using SPSS for Windows (SPSS 17.0, Chicago, IL). Postprandial responses for TG, insulin, and NEFA were quantified by summing the 3-hour AUC for serum/plasma concentrations vs. time using the trapezoidal rule. With n _ 1 measurements yi at times ti (i = 0, 0.5, 1, 2, and 3 h), the AUC (mmol·l⁻¹·h⁻¹) was calculated as follows: 0.5 × [(y0 + y1)/2] + 0.5 × [(y1 + y2)/2] + 1.0 × [(y2 + y3)/2] + 1.0 × [(y3 + y4)/2]. Incremental AUC was calculated by subtracting the fasting value from the postprandial values before calculating the AUC. A one-way repeated-measures ANOVA was conducted on fasting serum/plasma concentrations of TG, incremental TGs, insulin, glucose, BHB, and NEFA, and on the AUC responses of these variables. If a significant treatment effect was found, follow-up one-tailed modified Bonferroni post hoc tests were performed to test for simple effects among treatment conditions. One-tailed tests were justified because the direction of the expected effects was known. To determine differences among conditions at different points in time after the meal, a two-way (treatment × time) repeated-measures ANOVA was performed on serum/plasma concentrations of TG, insulin, glucose, BHB, and NEFA. If a main effect was detected, a one-way repeated-measures ANOVA was performed at each time point, and then follow-up one-tailed modified Bonferroni post hoc tests were performed. An α-level of 0.05 was used for all tests of significance. Results are expressed as means ± SD.

RESULTS

There was no difference between male and female participants in responses to Ex-Def, Ex-Bal, and Con in fasting TG or the AUC TG response (P > 0.05). Men and women elicited a similar reduction in postprandial AUC TG. Therefore, the data for men and women were analyzed together.

The diet consumed on the day of Ex-Def, Ex-Bal, and Con was not different (P > 0.05) in total energy (4.9 ± 1.1, 5.0 ± 1.0, 4.8 ± 1.0 MJ), fat content (36.5 ± 4.0, 37.7 ± 3.7, 35.3 ± 6.0 g), carbohydrate content (160.6 ± 19.2, 149.8 ± 15.3, 143.5 ± 23.3 g), or protein content (62.2 ± 16.3, 60.3 ± 15.9, 55.8 ± 14.8 g). There was no significant difference (P > 0.05) between the Ex-Def and Ex-Bal session in total work performed (54.6 ± 16.4, 56.6 ± 17.3 kJ) or energy expenditure (1.2 ± 0.5, 1.0 ± 0.1 MJ) during SIC.

There was a trend toward a reduction with treatment in fasting TG (P = 0.068), but no significant treatment effect for fasting insulin, NEFA, or BHB (P > 0.05) (Table 2). The TG response expressed as AUC (mmol·l⁻¹·3 h⁻¹) was significantly different among treatments (P < 0.05) (Fig. 1B). TG AUC was significantly lower following Ex-Def than following CON (21%, P < 0.05) and Ex-Bal (12%, P < 0.05). TG AUC following Ex-Bal also was significantly lower than following Con (10%, P < 0.05). Incremental TG AUC did not differ with treatment (P > 0.05), suggesting the reduction in TG AUC was proportional to a reduction in fasting TG concentration. There was no treatment effect (P > 0.05).
observed for AUC responses of insulin, glucose, NEFA, or BHB (Table 3).

In the two-way ANOVA, there was no significant treatment × time interaction (P > 0.05) for the postprandial response of TG, insulin, glucose, NEFA, or BHB (Figs 1A, 2, and 3). However, there was a significant treatment main effect for postprandial TG (P < 0.05). There was a significantly lower postprandial TG response following Ex-Def (P < 0.05) and a nearly significantly lower TG response following Ex-Bal (P = 0.07) compared with Con (Fig. 1A). Ex-Def significantly reduced serum TG levels at 0, 30, 60, and 120 min postprandial (0 min: 22%; 30 min: 25%; 60 min: 24%; 120 min: 22%, P < 0.05) and tended to reduce TG levels at 180 min postprandial (15%, P = 0.06) compared with Con. Ex-Bal reduced serum TG levels at 0 and 30 min postprandial (0 min: 17%; 30 min: 17%, P < 0.05), but failed to lower TG levels at any other time point (Fig. 1A). There was no statistically significant treatment effect observed for insulin, glucose, NEFA, or BHB (Figs. 2 and 3).

DISCUSSION

The main finding of our study was that SIC reduced the postprandial TG response to a HFM compared with a Con condition. This study is the first to show that persistent effects from an acute bout of high-intensity, low-volume SIC reduce PPL. The results extend findings from previous research establishing that persistent effects from moderate-intensity aerobic exercise (14–17, 19, 20, 23–26, 36, 40–42, 47, 48) and resistance exercise (7, 29, 34, 35, 46) reduce PPL. The results from our study add to the growing literature indicating that the physiological effects and health benefits of high-intensity, low-volume exercise training.

The minimum threshold for intensity and duration of exercise to effectively reduce PPL has been of interest for many years. Previous research has shown aerobic exercise at 30–70% VO2max, performed for 30–90 min, with an energy expenditure of 1.5–4 MJ, reduces PPL. But, within this energy expenditure range, the reduction in PPL is independent of aerobic exercise intensity and duration (41), as long as energy expenditure is constant. These findings lead to the conclusion that the exercise energy expenditure was a more important mediator of the response than either intensity or duration (41). However, resistance exercise reduces PPL with much less energy expenditure, dissociating the apparent link between energy expenditure and the magnitude of reduction in PPL (28, 29, 35, 46). These studies show that the relation of intensity to the reduction in PPL is different in resistance exercise than in aerobic exercise.

In the present study, SIC lasted only 18 min, and the energy expenditure was similar to that in the studies on resistance exercise and lower than that in many other studies investigating the effect of exercise on PPL (15, 19, 28, 35, 40, 41), yet the reduction in PPL was similar. Our study shows that, like resistance exercise, very-high-intensity aerobic exercise can reduce PPL with much less energy expenditure than moderate-intensity aerobic exercise. Apparently, the very high intensity of SIC is more important than the low volume in reducing postprandial TG levels.

The reduced PPL following acute SIC is most likely due, at least in part, to increased skeletal muscle LPL protein expres-

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Ex-Def</th>
<th>Ex-Bal</th>
<th>Con</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incremental TG</td>
<td>1.00 ± 0.66</td>
<td>1.26 ± 0.87</td>
<td>1.28 ± 0.51</td>
</tr>
<tr>
<td>Insulin</td>
<td>93.03 ± 67.94</td>
<td>114.11 ± 48.75</td>
<td>120.13 ± 57.90</td>
</tr>
<tr>
<td>Glucose</td>
<td>15.81 ± 1.38</td>
<td>16.18 ± 1.32</td>
<td>16.08 ± 2.36</td>
</tr>
<tr>
<td>BHB</td>
<td>0.17 ± 0.10</td>
<td>0.19 ± 0.11</td>
<td>0.25 ± 0.26</td>
</tr>
<tr>
<td>NEFA</td>
<td>1.24 ± 0.37</td>
<td>1.44 ± 0.37</td>
<td>1.29 ± 0.54</td>
</tr>
</tbody>
</table>

Values are means ± SD in mmol·L⁻¹·h⁻¹.

Fig. 2. A: postprandial insulin response. B: postprandial glucose response. Values are means ± SD.

Fig. 3. A: postprandial nonesterified fatty acid (NEFA) response. B: postprandial betahydroxybutyrate (BHB) response. Values are means ± SD.
sion, thus increasing hydrolysis of circulating TG. Skeletal muscle contractions cause a transient (33), tissue-specific (32) increase in LPL activity. Increased LPL activity following exercise is, in part, due to elevated LPL mRNA levels, which peak 4 h after exercise with a peak in LPL protein 8 h after exercise, returning to baseline values within 24 h postexercise (33). In addition, reduced hepatic VLDL secretion contributes to increased TG clearance (25). Reduced hepatic VLDL secretion reduces competition between exogenous chylomicron and endogenous VLDL, resulting in greater hydrolysis of exogenous chylomicrons than endogenous VLDLs (31).

A second important finding was that the reduction in PPL is dependent, in part, on the energy deficit created by SIC. When the energy deficit created by SIC was replaced with a mixed meal bar (Ex-Bal), the reduction in PPL was approximately halved compared with that for Ex-Def. Other studies in which the energy deficit created by exercise was totally or partially replaced after exercise also have found the reduction in PPL was attenuated or eliminated (8, 35). The exercise effect appears to involve more than just creation of an energy deficit, however, because creating an energy deficit by reducing caloric intake does not reduce PPL as much as creating the same energy deficit with exercise (14). Nevertheless, maintenance of an energy deficit following exercise is important for maximizing the reduction in PPL.

The mechanism underlying the reduced effect of exercise on PPL after replacing the energy deficit is unknown, but it is likely due to a reduced rise in skeletal muscle LPL protein content (13) or an increased hepatic secretion of VLDL. These changes could be modulated by changes in skeletal muscle and liver glycogen, but there is little direct evidence to support this hypothesis. If this is the case, the mechanism is unknown. It has been reported that carbohydrate feedings following exercise replenished glycogen and attenuated the postprandial TG response, but there was no relation between postprandial TG concentrations and glycogen depletion (19). Reduction of muscle glycogen is not necessary for upregulation of LPL, as increased LPL activity is evident in light (<50% \( V_{O2max} \)) intermittent walking (4). Intense interval cycling reduces skeletal muscle glycogen (19, 13), but whether this change plays a role in reducing PPL is unknown.

Although there was only a trend toward a statistically significant difference in fasting TG levels, they differed enough such that the lower fasting TG levels in Ex-Def were proportional to the postprandial reduction in AUC TG. There was no difference in incremental TG AUC across the three sessions, signifying the reduction in postprandial TG concentrations may be attributable to the reduction in fasting TG. Similar results were found by Singhal et al. (35). Studies investigating the effects of moderate and heavy resistance exercise on PPL have shown reductions in both fasting and postprandial incremental TG concentrations (28, 41, 46). The discrepancy in the effect of prior moderate and intense resistance or sprint exercise on fasting TG concentrations may be due to the difference in exercise protocols and energy expended during the exercise session, or to differences in statistical power.

SIC lowered postprandial TG levels, but failed to change fasting or postprandial insulin, glucose, BHB, and NEFA plasma concentrations. A reduction in insulin and glucose concentrations and an increase in NEFA and BHB ketone production would be expected following an acute exercise session. Our results are similar to those observed by most (29, 34, 35, 46), but different from the fasting results observed by others (35). The lack of changes may be due to the low volume and caloric expenditure of SIC, or due to inadequate statistical power.

The findings of our study have shown that low-volume, high-intensity exercise can cause a significant reduction in PPL and reinforce the importance of creating an energy deficit through exercise to reduce PPL. Further investigation is needed to determine the effect of replacing the energy deficit at different points in time following exercise. Also, the effect of differing macronutrient compositions of postexercise meals should be examined to help understand the effect of postexercise carbohydrate, fat, and protein consumption on PPL.

Although SIC has been shown to be an effective treatment for improving postprandial TG metabolism, increasing skeletal muscle (5) and total body oxidative capacity (39), and increasing insulin sensitivity (2), there are practical implications for introducing SIC into the public health message. SIC is high-intensity exercise, and although it has been shown to be effective in some sedentary, at-risk individuals (39), it may not be safe, well-tolerated, or appealing to some individuals in the general population or in those who are at moderate or high risk of medical complications during exercise. Further investigation into the acceptability to SIC in different populations is necessary.

We conclude that SIC is an effective mode of exercise to reduce PPL when the energy deficit created by exercise is maintained following exercise. The magnitude of the effect of high-intensity, low-volume SIC on PPL is similar to the effects of aerobic and resistance exercise. High intensity can apparently substitute for greater energy expenditure (exercise volume). Inclusion of the option of SIC into the public health message may help people exercise who are worried about time restraints and the need to sustain moderate-to-high intensities of exercise for sustained periods of time. The energy deficit created by exercise plays an important role in the reduction in PPL, suggesting that delaying replacement of the energy used during exercise has important health benefits. Additional research is needed on the extent to which health effects of persistent acute effects of exercise are linked to the creation of an energy deficit and on the timing and macronutrient make-up of the diet used to replace the energy deficit.

**ACKNOWLEDGMENTS**

The authors thank Eugene Fan and Sahir Ahsan for assisting with data collection.

**DISCLOSURES**

No conflicts of interest, financial or otherwise, are declared by the author(s).

**REFERENCES**

10. Demacker PN, Schade RW, Stalenhoef AF, Stuyt PM, van’t Laar A.
