Acute and chronic effects of sprint interval exercise on postprandial lipemia in women at-risk for the metabolic syndrome

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Abstract

The metabolic syndrome (MetS) is characterized by a clustering of risk factors underlying cardiovascular disease (CVD) and type 2 diabetes. Findings from the National Health and Nutrition Examination Survey (NHANES) indicate that 37% of women over the age of 40 in the United States can be diagnosed with MetS (11). Individuals with MetS exhibit elevated postprandial triglycerides (TG) (26, 27), increasing risk for CVD. Hypertriglyceridemia in the postprandial state is an independent risk factor for CVD (8) and has been suggested as a better predictor of myocardial infarctions than fasting TG concentrations (3). Elevated postprandial TG lead to reduced high-density lipoprotein cholesterol production and increased low-density lipoprotein cholesterol production (13), impaired endothelial function (40), and increased atherosclerotic plaque formation (42). Effective, sustainable exercise interventions that reduce postprandial lipemia (PPL) and limit the onset of MetS may reduce atherogenesis and CVD risk.

High-intensity interval exercise has been shown to be an effective training modality to attenuate fasting and postprandial TG (12, 16, 36, 39) and has been shown to be more potent at reducing the incremental rise in TG compared with aerobic or resistance exercise (14). Sprint interval training (SIT) is a form of low-volume, high-intensity exercise that has been proposed as a potent time-efficient training modality to induce physiological adaptations similar to more prolonged, moderate-intensity aerobic exercise (18). SIT has been shown to improve circulatory function (38), glucose tolerance (28), skeletal muscle oxidative capacity (17), and cycle endurance (5, 6). Although a single session of SIT has been shown to attenuate PPL in young, healthy men and women (16), the effect of a single session of SIT in middle-aged women at-risk for MetS has not been established.

Repeated acute bouts of exercise (i.e., training) are frequently suggested as a successful measure to combat MetS and associated comorbidities. Exercise training attenuates PPL (10, 43), although when participants were asked to abstain from exercise for 60 h prior to the test meal administration, the effect of training was abolished, and trained participants exhibit a similar postprandial TG response to sedentary individuals (23). This suggests the attenuation of PPL is acute, lasting 24–48 h. Although the exercise training has not been shown to reduce PPL at 48 h (1) or 60 h (21) after the last training bout, the effect of chronic SIT and the comparison of the effects of a single session of exercise (i.e., acute) and chronic exercise on PPL have not been investigated.

The overarching goal of this investigation was to determine the effectiveness of 6 wk of SIT to attenuate PPL. The primary aim was to determine if an acute bout of SIT can attenuate PPL in middle-aged women at-risk for MetS. It was hypothesized that an acute bout of SIT would attenuate PPL. The secondary aim of this investigation was to determine if 6 wk of SIT would magnify the attenuation of PPL compared with a single session of SIT prior to the training. It was hypothesized that the attenuation of PPL would be greater following 6 wk of SIT than a single session of SIT.

Methods

Participants. Women (n = 45; 30–65 yr of age) who were at risk for developing MetS, defined as having abdominal obesity (waist circumference > 88 cm) and at least 1 of the following risk factors—hypertensive (defined as >130/85 mmHg), hypertriglyceridemic (defined as fasting TG > 150 mg/dl), hyperglycemic (defined as fasting
glucose > 100 mg/dl), or high-density lipoprotein cholesterol < 50 mg/dl or on medication for any of these risk factors (20)—were recruited to participate in this randomized controlled trial. Women enrolled in the study had to be either premenopausal, defined as having a normal menstrual cycle either naturally or through medication, or postmenopausal, defined as not having cycled within 1 yr. Perimenopausal women were excluded from this trial. Other exclusion criteria included individuals diagnosed with type 2 diabetes, a chronic disease, or condition that would not permit exercise or dietary restriction, cardiopulmonary disease, severe orthopedic problems that would contraindicate exercise, dementia, any medication that would impact primary outcomes (including but not limited to statins), uncontrolled blood pressure, performing ≥150 min of light to moderate physical activity each week or any vigorous physical activity, or body weight greater than 181 kg. The study protocol was approved by the University of Georgia Institutional Review Board, and all participants gave written, informed consent following a description of the study procedures and risks.

**Study design.** This study was part of a larger 2 × 2 parallel arm (diet × exercise) randomized controlled trial to determine acute and chronic postprandial responses to a high-fat meal in response to a 6-wk exercise and macronutrient dietary intervention. The study was powered on the primary aim to compare the effects of diet and exercise on the total area under the curve (AUC) TG response. An a priori power analysis revealed a sample of 20 per group (80% adherence) was sufficient to detect a moderate effect of 0.5 SD with a power of 0.80 and a correlation of repeated measures of 0.90. Women in the study did not significantly alter the macronutrient composition of their diet (P > 0.05) and the dietary intervention did not alter any outcome variables associated with the current investigation (P > 0.05). Two number allocations, one for premenopausal women and one for postmenopausal women, were created using an online random-number generator (www.randomizer.org), with a ratio of 1:1 to each condition. Women were block randomized, based on menopausal status (pre or post), to one of two groups for 6 wk: SIT [n = 22 (8 premenopausal); 3 bouts/wk of 4–8, 30-s all-out cycle sprints with 4-min active recovery] or a nonexercise control condition [n = 23 (10 premenopausal); CON]. Participants randomized to SIT completed a session of SIT for familiarization prior to the first session of SIT. For premenopausal women enrolled in the trial, all meal challenges during baseline, pre-, and posttesting were completed during days 5–14 of the follicular phase to control for confounding effects of the menstrual cycle. All participants completed a baseline high-fat meal challenge (B-HFMC) prior to the intervention. To determine the acute effect of SIT, participants randomized to SIT then completed another HFMC, 2–5 days after the B-HFMC, with an acute bout of SIT the evening prior (~14–16 h) to the high-fat meal challenge (Pre-HFMC). Between 3 and 5 days following completion of the 6-wk intervention, participants in SIT completed another HFMC paired with an acute bout of SIT the evening prior (~14–16 h) to the high-fat meal challenge while participants in CON completed another HFMC without prior exercise (Post-HFMC).

**Body composition.** Participants’ body composition was assessed prior to and after the 6-wk intervention using dual-energy X-ray absorptiometry (iDXA, GE Healthcare, Fairfield, CT). The fat-free mass (FFM) was used to estimate the cycle resistance for SIT and the amount of food consumed in the test meal during the HFMC. Additionally, body weight was assessed prior to and then once weekly throughout the 6-wk intervention to ensure participants remained weight stable, which was defined as ±2 kg from baseline weight.

**High-fat meal challenges.** All HFMC were administered after a 12-h overnight fast and were similar to other experiments performed in our laboratory (16, 35). All participants refrained from alcohol ingestion for 48 h prior to administration of the HFMC while participants not randomly assigned an acute SIT session refrained from all planned exercise outside of daily activities for 48 h. Participants arrived in the laboratory in a fasted state in the morning, performing as little physical activity as possible. An intravenous catheter was then inserted into an antecubital vein and kept patent with 0.5 ml of 10 USP units/ml heparin lock flush. A fasting blood sample was obtained, and then the participant consumed the high-fat test meal. The high-fat test meal was composed of 1.6 g fat, 1.3 g carbohydrate, and 0.7 g protein/kg FFM and provided ~84 kJ/kg FFM. The high-fat test meal was a commercially available breakfast that consisted of a croissant, an omelet, two slices of cheese, 4 sausage patties (Jimmy Dean, Hillshire Brands), and a Little Debbie Honey Bun (Little Debbie, McKee Foods). To keep the macronutrient content of the meal consistent across participants and trials, each component of the meal was individually weighed and cut to account for differences in FFM among the participants. Total caloric content of the high-fat meal ranged from 807 to 1,153 kcals. Water consumption was held constant across all trials to 4.3 ml/kg body mass to ensure consistent hydration. Following consumption of the test meal, participants neither ate nor drank anything for the following 3 h. Blood samples were taken at 0, 30, 60, 120, and 180 min postprandial. Participants rested in a sitting position throughout the 3-h postprandial period, getting up only to use the restroom as needed.

**Acute bout of sprint interval training.** To determine the difference between the acute and chronic effects of SIT on PPL, participants randomized to SIT completed a 2-day testing session before and after the 6-wk intervention. On day 1 of this testing, participants completed an acute bout of SIT between 1400 and 1900. SIT consisted of four, 30-s, all-out sprints with 4 min of active recovery between each sprint. SIT was administered on a mechanically braked, stationary cycle ergometer (Monarch Egomedic 874E; Monark, GIM, Stockholm, Sweden) against a fixed resistance of 0.09 kg/kg FFM (9% of FFM), similar to protocols previously used in our laboratory (15, 16, 38). Following a 5-min warm-up pedaling against no resistance, participants were instructed to pedal as fast as possible, with resistance applied to the flywheel after maximal speed was attained and continued for 30 s. The number of revolutions and the total work performed on each sprint was quantified using an optical sensor (Sports Medicine Industries, St. Cloud, MN) that 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). Immediately following each sprint, heart rate (Polar Vantage XL) and subjective rating of perceived exertion (RPE) (4) were recorded. Four minutes of active recovery on the cycle was completed prior to the administration of the next sprint. During active recovery, participants pedaled against no resistance at a self-selected speed. Following completion of the acute bout of SIT, participants were instructed to rest at home for the evening and consume a typical evening meal. The following morning (day 2), participants arrived in the laboratory in a fasted state and completed a HFMC.

**Sprint interval training.** Participants randomized to SIT completed three training sessions per week for 6 wk. Training sessions consisted of repeated 30-s all-out cycling sprints (4–8 sprints/session). The same protocol was applied as used during acute SIT sessions (described above). The intervals were repeated until the prescribed number of sprints was completed. Participants completed four sprints per session during the first 2 wk of training. The number of sprints per training session was then increased by one sprint bout every week thereafter until participants completed eight sprints per session during the final week of training.

Participants in all groups were instructed to maintain their prestudy physical activity outside of the intervention. Weekly physical activity logs were reviewed and assessed for adherence to physical activity requirements.

**Analytic methods.** In the fasted state, 14 ml of blood was collected, and while in the postprandial state, 10 ml of blood was collected. The first 2 ml of blood was discarded and the rest was collected into BD Vacutainer 4.0-ml serum separation tubes and 3.0-ml sodium-heparin tubes (Becton Dickinson, Franklin Lakes, NJ) for preparation of...
serum and plasma, respectively. Plasma samples were centrifuged at 2,000 RPM at 5°C for 10 min, and resulting plasma removed, divided into aliquots, and stored at −70°C until analyzed for insulin, glucose, and nonesterified fatty acids (NEFA). Serum separation tubes were allowed to clot for 30 min prior to being centrifuged at 2,000 RPM 5°C for 10 min, separated and divided into aliquots, and stored at −70°C to be analyzed for TG. Enzymatic, colorimetric assays were used to measure serum TG (Wako L-Type TG M assay, Wako Chemicals, Richmond, VA), plasma glucose (Wako Glucose C2 assay, Wako Chemicals), and plasma NEFA [Wako NEFA-HR (2) assay, Wako Chemicals]. Plasma insulin was measured using a radioimmunoassay (RIA Kit, Human Specific Insulin, Millipore, Billerica, MA). Inter-assay coefficients of variation were 2.5% for TG, 2.8% for glucose, 3.3% for NEFA, and 7.8% for insulin.

Statistical analyses. All analyses were performed using IBM SPSS for Windows (SPSS 21.0). The dependent variables exhibited normal distributions at baseline. Postprandial responses for TG, insulin, glucose, and NEFA were quantified by summing the 3 h area under the curve (AUC) for serum/plasma concentrations vs. time using the trapezoidal rule to obtain the total AUC response (tAUC). With \( n \) measurements \( y_i \) at time \( t \) (0, 0.5, 1, 2, and 3 h), the AUC was calculated as follows: \( 0.5 \times [(y_0 + y_1)/2] + 0.5 \times [(y_1 + y_2)/2] + 1.0 \times [(y_2 + y_3)/2] + 1.0 \times [(y_3 + y_4)/2] \). Incremental AUC (iAUC) was calculated by subtracting the fasting value from the postprandial values before calculating the AUC. An independent \( t \)-test was used to assess differences among groups at baseline in physical characteristics and all outcome variables. The magnitude of all effects was calculated using Cohen’s \( d \). An alpha-level of 0.05 was used for all tests of significance. All results are expressed as means ± SD.

To compare the difference between an acute bout of SIT prior to and after the 6-wk intervention, a one-way repeated-measures ANOVA was performed on fasting, tAUC, and iAUC serum/plasma concentrations of TG, insulin, glucose, and NEFA with follow-up modified Bonferroni post hoc tests to determine differences between responses to B-HFMC, Pre-HFMC, and Post-HFMC. A two-way (treatment \( \times \) time) repeated-measures ANOVA was performed at each time point, and then follow-up modified Bonferroni post hoc tests were performed.

To determine the chronic effects of SIT, a two-way repeated-measures group (SIT vs. CON) \( \times \) session (Pre vs. Post) ANOVA was conducted on fasting, tAUC, and iAUC serum/plasma concentrations of TG, insulin, glucose, and NEFA. A three-way (session \( \times \) time \( \times \) group) repeated-measures ANOVA was performed on postprandial serum/plasma concentrations of TG, insulin, glucose, and NEFA to determine differences at specific postprandial time points.

RESULTS

Participants. A flowchart outlining recruitment, enrollment, and participant withdrawals can be found in Fig. 1. Of the 160 participants eligible for participation in this randomized clinical trial, 82 withdrew prior to randomization and/or no longer met inclusion criteria while 76 were randomized to 1 of 4 treatment groups. Of these 76 women who were randomized, 15 withdrew prior to baseline testing and did not participate in the intervention and were therefore excluded from analysis. Furthermore, 12 withdrew or were excluded during the intervention, 3 were excluded due to failure to insert the IV catheter during posttesting, and 1 was excluded because her baseline fasting, tAUC, and iAUC TG responses were considered outliers (>3 SD from the mean). One participant withdrew because of an injury that did not occur during SIT.

Participant characteristics did not differ between groups at baseline (Table 1; \( P > 0.05 \)). Overall, the participants in this trial had a mean age of 51.9 ± 9.2 yr, weight of 85.3 ± 19.3 kg, waist circumference of 100.3 ± 11.8 cm, body mass index (BMI) of 31.3 ± 6.5 kg/m², FFM of 46.0 ± 7.2 kg, and relative body fat percentage of 45.1 ± 5.8% at baseline. There were no significant group \( \times \) time interactions, or significant effects of time on fat mass, FFM, body weight, body fat percentage, or BMI following the intervention (all \( P > 0.05 \)).

Fasting and postprandial responses to the B-HFMC were not different between groups for TG, glucose, insulin, or NEFA (all \( P > 0.05 \)). The fasting and postprandial responses for premenopausal and postmenopausal women were not statistically significantly different at baseline (\( P > 0.05 \)).

Sprint interval training. A paired \( t \)-test to compare responses during the pre- and postintervention acute bouts of SIT revealed average power output increased significantly (219 ± 56 vs. 249 ± 56 W, respectively; \( P < 0.01 \)) while RPE decreased significantly (18 ± 1 vs. 16 ± 2, respectively; \( P < 0.01 \)). Average peak heart rate tended to be lower in the postintervention acute bout of SIT (161 ± 16 vs. 158 ± 15 beats/min; \( P = 0.09 \)).

Chronic responses to SIT. Fasting concentrations of insulin, NEFA, and glucose at baseline and following the 6-wk intervention can be found in Table 2. The two-way ANOVA revealed a significant group \( \times \) session interaction for fasting NEFA concentrations (\( P < 0.05 \)). Fasting NEFA concentrations increased in SIT (Cohen’s \( d = 0.25 \)) and decreased in CON (Cohen’s \( d = -0.15 \)) following the 6-wk intervention. There were no significant group \( \times \) session interactions for TG, insulin, or glucose or main effects of time for insulin or glucose (\( P > 0.05 \)). A significant effect of session was found for fasting TG (\( P < 0.05 \)) and NEFA (\( P < 0.01 \)).

The two-way ANOVA revealed no significant group \( \times \) session interactions for tAUC or iAUC responses of TG, insulin, glucose, or NEFA. A significant main effect of session was found for TG iAUC (\( P < 0.05 \)) but no other significant main effects for tAUC or iAUC responses of TG, insulin, glucose, or NEFA were found (all \( P > 0.05 \); Table 3).

Postprandial TG, insulin, glucose, and NEFA responses can be found in Fig. 2. The three-way repeated-measures ANOVA revealed a significant session \( \times \) time interaction for postprandial insulin (\( P = 0.04 \)); however, a lack of an interaction of group with session or time indicates SIT did not influence the postprandial insulin response compared with CON. A significant session \( \times \) time \( \times \) group interaction was found for fasting postprandial glucose (\( P < 0.05 \)) and NEFA (\( F_{4,172} = 3.96, P < 0.01 \)). There were no other session \( \times \) group, time \( \times \) group, session \( \times \) time, or session \( \times \) time \( \times \) group interactions (all \( P > 0.05 \)) for other postprandial blood responses. A significant effect of session was found for TG (\( P = 0.02 \)). A significant session effect independent of a significant group \( \times \) session interaction indicates postprandial TG were lowered following the 6-wk intervention independent of SIT. Significant effects of time were found for TG (\( P < 0.001 \)), NEFA (\( P < 0.001 \)), insulin (\( P < 0.001 \)), and glucose (\( P < 0.001 \)).

Acute vs. chronic responses to SIT. A one-way repeated-measures ANOVA on SIT participants compared B-HFMC, Pre-HFMC, and Post-HFMC and revealed a significant effect of SIT on fasting TG (\( P < 0.01 \)) and tAUC TG (\( P = 0.01 \)). Follow-up tests for simple effects revealed that compared with B-HFMC, fasting TG was significantly lower (\( P < 0.05 \)) following an acute bout of SIT before (Pre-HFMC; Cohen’s
The main finding of this trial was that although a single, acute session of SIT attenuated PPL in middle-aged women at-risk for MetS, 6 wk of SIT training did not modify the attenuation of PPL compared with an acute bout of SIT.

**DISCUSSION**

The main finding of this trial was that although a single, acute session of SIT attenuated PPL in middle-aged women at-risk for MetS, 6 wk of SIT training did not modify the attenuation of PPL compared with an acute bout of SIT.

d = −0.40) and after the 6-wk intervention (Post-HFMC; Cohen’s d = −0.30; Fig. 3A). The tAUC TG response was attenuated in Pre-HFMC (Cohen’s d = −0.32) and Post-HFMC (Cohen’s d = −0.23) compared with B-HFMC (P < 0.05; Fig. 3B). There was no difference in the fasting or tAUC TG response following an acute bout of SIT before and after the intervention (P > 0.05). There was no significant effect of acute or chronic SIT on the iAUC TG response or fasting (Table 2), tAUC, or iAUC responses (Table 3) of insulin, glucose, or NEFA (all P > 0.05).

The two-way ANOVA revealed no significant session × time interactions (P > 0.05) for the postprandial responses of TG, insulin, glucose, or NEFA following B-HFMC, Pre-HFMC, and Post-HFMC. A significant effect of session was found for TG (P < 0.01). A follow-up one-way ANOVA with post hoc tests on each postprandial time point revealed TG were lower at Pre-HFMC and Post-HFMC at 0, 30, 60 min postprandial compared with B-HFMC (P < 0.05; Fig. 3). TG concentrations were not significantly different comparing Pre-HFMC and Post-HFMC at any postprandial time point (all P > 0.05). A significant effect of time was found for TG (P < 0.001), NEFA (P < 0.001), insulin (P < 0.001) and glucose (P < 0.001).
High-intensity interval exercise (i.e., SIT) has been suggested as a low-volume training modality that induces a host of physiological adaptations that reduce MetS and CVD risk (2, 6, 12, 16, 36, 39). Our results suggest that 6 wk of SIT does not amplify the attenuation of PPL compared with the attenuation following a single bout of SIT. Previous investigations on the effect of aerobic exercise training have shown that PPL is not attenuated ~48 h after the final training bout (1) as the attenuation of PPL following exercise is thought to be acute, lasting ~24–48 h. The findings from this investigation are the first to show a single session of SIT reduces PPL in middle-aged women at-risk for MetS and adds to the existing body of literature that PPL can be attenuated following an acute bout of high-intensity interval exercise in young, healthy individuals (12, 16, 36, 39).

Table 2. Fasting plasma concentrations of glucose, insulin, and NEFA

<table>
<thead>
<tr>
<th>Group</th>
<th>SIT (n = 22)</th>
<th>CON (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, mg/dl</td>
<td></td>
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</tr>
<tr>
<td>B-HFMC</td>
<td>102.5 ± 7.4</td>
<td>104.5 ± 12.5</td>
</tr>
<tr>
<td>Pre-HFMC</td>
<td>102.6 ± 6.8</td>
<td>101.6 ± 15.1</td>
</tr>
<tr>
<td>Post-HFMC</td>
<td>100.9 ± 9.9</td>
<td>101.6 ± 15.1</td>
</tr>
<tr>
<td>Insulin, mmol/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B-HFMC</td>
<td>13.5 ± 6.7</td>
<td>16.1 ± 7.7</td>
</tr>
<tr>
<td>Pre-HFMC</td>
<td>12.5 ± 6.9</td>
<td>15.4 ± 7.1</td>
</tr>
<tr>
<td>Post-HFMC</td>
<td>12.3 ± 5.2</td>
<td>15.4 ± 7.1</td>
</tr>
<tr>
<td>NEFA, mmol/l*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B-HFMC</td>
<td>0.59 ± 0.24</td>
<td>0.64 ± 0.22</td>
</tr>
<tr>
<td>Pre-HFMC</td>
<td>0.63 ± 0.22</td>
<td>0.61 ± 0.20</td>
</tr>
<tr>
<td>Post-HFMC</td>
<td>0.66 ± 0.28</td>
<td>0.61 ± 0.20</td>
</tr>
</tbody>
</table>

Values are means ± SD. NEFA: nonesterified fatty acids. B-HFMC, baseline high-fat meal challenge; Pre-HFMC, Pre high-fat meal challenge; Post-HFMC, Post high-fat meal challenge. For further description, see Study design. *Significant chronic group × session interaction (P < 0.05). Session: defined as differences across testing conditions. Group: defined as differences between SIT and CON.
Exercise training has long been suggested to reduce risk of metabolic and cardiovascular abnormalities associated with CVD. The attenuation of PPL through exercise is thought to be acute, due to a transient (34), tissue-specific (33) increase in lipoprotein lipase (LPL) activity and reduced hepatic VLDL output, which accounts for up to 70% of TG clearance (22, 30). Individuals engaged in exercise training exhibit lower PPL compared with sedentary subjects (7, 31); however, when trained individuals were asked to abstain from exercise for 60 h prior to PPL assessment, the postprandial response was similar to untrained participants (23). Similar in finding, Herd et al. (21) examined the

Fig. 2. Postprandial serum/plasma concentrations of triglycerides (TG), glucose, insulin, and nonesterified fatty acids (NEFA) during B-HFMC, Pre-HFMC, and Post-HFMC in sprint interval training (SIT) (column 1) and control (CON) (column 2). Values are means ± SD. SIT, sprint interval training; CON, control; HFMC, high-fat meal challenge. B-HFMC, baseline high-fat meal challenge; Pre-HFMC, Pre high-fat meal challenge; Post-HFMC, Post high-fat meal challenge. For further description of groups, see Study design. *P < 0.05, Pre-HFMC vs. B-HFMC; †P < 0.05, Post-HFMC vs. B-HFMC.
The current investigation was the first to determine if there is a difference in the attenuation of PPL due to an acute bout of exercise before and after training. The magnitude of the reduction in PPL after an acute bout of SIT after the 6-wk intervention was slightly weakened compared with the acute bout of SIT prior to the intervention, although not statistically different (Cohen’s $d = -0.23$ and $-0.32$, respectively; Fig. 2). This slightly diminished benefit may be due to the reduced physiological strain during the post-acute bout of SIT. Participants in both groups significantly reduced their RPE; however, there was no significant difference in average heart rate in the post-acute bout of SIT compared with the pre-acute bout of SIT. Although training did not increase the attenuation of PPL from an acute bout of SIT (four all-out, 30-s sprints), they may have benefited from increased work capacity. Participants completed eight all-out, 30-s sprints per session during the final week of the training intervention, which may have caused a greater acute reduction in PPL, although the added benefit of these increased sprints is only speculative, as the magnitude of the reduction in PPL following eight all-out, 30-s sprints has not been investigated.

There are several limitations to the current randomized controlled trial. Strict inclusion/exclusion criteria and budget limitations limited participant enrollment. Additionally, this investigation focused on women at-risk for MetS; whether men at-risk for MetS would respond similarly is uncertain and generalizations of these results to other groups should not be made. It is plausible that the benefit of 6-wk SIT intervention is the improved exercise capacity and exercise volume. During week 6 of training, participants completed eight sprints per session, twice the number of sprints as performed per session during the first week of training. This increase in exercise volume may magnify the attenuation of PPL as previous investigations have suggested a positive correlation between energy expended during an exercise session and the magnitude of the reduction in PPL (32). The current investigation did not assess the magnitude of the reduction in PPL following eight sprints and therefore may not have fully captured the benefit of chronic SIT on attenuation PPL. Additionally, it is important to note that high-intensity exercise such as SIT is not suitable for all individuals as the risk of acute cardiovascular events during exercise increases with increasing exercise intensity, particularly in older people who are unaccustomed to exercise or have existing cardiovascular disease.

In conclusion, a single session of SIT effectively reduced PPL in middle-aged women at-risk for MetS, but 6 wk of SIT did not modify that attenuation. The results from this study highlight the efficacy of SIT as an effective mode of exercise to attenuate PPL in both young, healthy individuals and middle-aged women at-risk for MetS. Repeated acute bouts of exercise completed every 24–48 h are necessary to consistently maintain lower postprandial TG concentrations.
REFERENCES


