Effect of exercise duration on postprandial hypertriglyceridemia in men with metabolic syndrome

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Zhang JQ, Ji LL, Fogg DL, Fretwell VS. Effect of exercise duration on postprandial hypertriglyceridemia (PHTG) and insulin resistance in individuals with metabolic syndrome. J Appl Physiol 103: 1339–1345, 2007. First published July 19, 2007; doi:10.1152/japplphysiol.00181.2007.—We examined the effect of exercise on postprandial hypertriglyceridemia (PHTG) and insulin resistance in individuals with metabolic syndrome. Subjects were 10 hypertriglyceridemic men with insulin resistance (age = 35.0 ± 1.8 yr, body weight = 90.7 ± 3.3 kg, fasting triglyceride (TG) = 2.6 ± 0.4 mmol/l, peak oxygen consumption (V̇O₂peak) = 36.0 ± 1.3 ml·kg⁻¹·min⁻¹, and homeostatic model assessment of insulin resistance (HOMA-IR) = 3.1 ± 0.3). Each participant performed a control trial (Ctr; no exercise) and three exercise trials at 60% of their V̇O₂peak for 30 min (30 min-Ex), 45 min (45 min-Ex) and 60 min (60 min-Ex). All subjects had a fat meal in each trial. In the exercise trials, the subject jogged on a treadmill for a designated duration of 12 h before ingestion of a fat meal. Blood samples were taken at 0 h (before the meal) and at 2, 4, 6, and 8 h after the meal. The plasma TG, area score under TG concentration curve over an 8-h period (TG AUC) after the meal, and HOMA-IR were analyzed. The TG AUC scores in both the 45 min-Ex and 60 min-Ex trials were lower than the Ctr (P < 0.02). There were no significant differences in TG AUC scores between the 30 min-Ex and the Ctr (P > 0.05). There were no trial differences in the fasting plasma glucose concentration (P > 0.05). HOMA-IR values in the 30 min-Ex, 45 min-Ex, and 60 min-Ex trials were lower than the Ctr (P < 0.03), but no significant differences were found in HOMA-IR among the exercise trials. The results suggest that for physically inactive individuals with metabolic syndrome, exercising at moderate intensity for 45 min effectively attenuates PHTG while exercise for 30 min is sufficient to improve insulin action.

Exercise duration; postprandial; hypertriglyceridemia; insulin resistance

As part of the metabolic syndrome, hypertriglyceridemia (HTG) and insulin resistance are strongly associated with cardiovascular disease (27). Individuals with HTG tend to have a prolonged postprandial hypertriglyceridemia (PHTG) after a fat-meal challenge (38). An exaggerated PHTG response indicates poor triglyceride (TG) clearance from the bloodstream and is often associated with atherosclerosis, insulin resistance, low high-density lipoprotein cholesterol (HDL-C), increased low-density lipoprotein-cholesterol (LDL-C), and obesity (14). Therefore, PHTG has been proposed as a potential risk factor for cardiovascular disease (21). Humans spend a great deal of time in postprandial state, and the repeated episodes of aggregated PHTG response may amplify the TG-rich lipoprotein insult to the arterial wall. In people on high-fat diets, the arterial wall may be exposed to PHTG for a much longer time. The exaggerated PHTG response is due to an increased influx and/or a decreased efflux of TG-rich lipoproteins from the bloodstream. Insulin promotes hepatic secretion of very low-density lipoprotein-TG (VLDL-TG). Insulin-resistant state is concomitantly associated with hyperinsulinemia and, therefore, results in elevated VLDL-TG production (32). Consequently, individuals with insulin resistance often demonstrate HTG and amplified PHTG (52).

Studies have shown that exercise training improves insulin sensitivity (5). Endurance-trained individuals exhibit a significantly lower PHTG response to a fat-rich meal compared with sedentary individuals (56). A single bout of aerobic exercise attenuated PHTG in young and healthy adults (15, 50, 55). However, most of the current studies (22, 50) focused on the effect of exercise intensities on PHTG in healthy individuals. The dose response of exercise duration on PHTG and insulin resistance in individuals with metabolic syndrome (such as HTG, insulin resistance, low HDL-C, and obesity) has not yet been systematically assessed. Because individuals with the characteristics of metabolic syndrome are more susceptible to cardiovascular disease, formulating an exercise-oriented therapeutic measure for such a population would be clinically important. Therefore, the purpose of the present study was to investigate the effect of exercise duration on PHTG and insulin resistance in men with metabolic syndrome. We hypothesize that improved PHTG following exercise will be greater as exercise duration increases.

Materials and methods

Subjects. Ten sedentary male subjects with metabolic syndrome participated in this study (age = 35.0 ± 5.5 yr, body weight = 90.7 ± 10.4 kg, percent body fat = 23.6 ± 3.1%, body mass index (BMI) = 30.0 ± 3.1 kg/m², waist-to-hip ratio = 0.92 ± 0.0, peak oxygen consumption (V̇O₂peak) = 36.0 ± 4.1 ml·kg⁻¹·min⁻¹). Subjects were informed of the risks associated with the study and were required to complete an informed consent form. The study protocol was approved by the University of Texas at San Antonio Institutional Review Board. Before participating in the study, subjects completed a medical history questionnaire and a physical activity questionnaire. The power calculation and sample size determination were based on the comparison of TG area under the curve (AUC) score (the area score under TG concentration curve over an 8-h period) in our laboratory’s previous study (55). The calculated effective size was 0.98. Accordingly, the sample size of 10 subjects.
was sufficient for the repeated-measures design of the present study at power = 0.80 and α = 0.05 (48).

**Initial measurements.** At the initial visit, BMI, and waist-to-hip ratio were measured with subjects wearing light clothing. Waist and hip circumferences were measured as described by Bray and Gray (6). Body composition was assessed using the sum of three skinfold measurements specific for men (chest, abdomen, and thigh). The mean of three measurements at each site was used to estimate body density and percent body fat (19). Subjects’ fasting blood was screened for lipids and homeostatic model assessment of insulin resistance (HOMA-IR), an index of insulin resistance (4). Subjects’ fasting TG concentrations (2.6 ± 0.4 mmol/l) were within the range of 2.3–3.6 mmol/l. In addition to having a high levels of plasma TG and total cholesterol (5.4 ± 0.5 mmol/l), subjects exhibited low HDL-C (0.9 ± 0.3 mmol/l) and were considered insulin resistant (HOMA-IR = 3.1 ± 0.3) (5). The subject inclusion criteria were based on the classification by the International Diabetes Federation (1) the subjects had characteristics of metabolic syndrome (fasting TG ≥ 1.7 mmol/l, fasting HDL-C < 1.03 mmol/l, BMI ≥ 30, and waist-to-hip ratio > 0.90).

**Experimental design.** After the initial assessment, the qualified subjects performed a treadmill test to determine $V_{O2\text{peak}}$. One week after a $V_{O2\text{peak}}$ test, each of the subjects performed a control trial (Ctr; no exercise) and three exercise trials at 60% of their $V_{O2\text{peak}}$ for 30 min (30 min-Ex), 45 min (45 min-Ex), and 60 min (60 min-Ex). The order of the trials was randomized. Subjects were given 1–2 wk for recovery after each trial. In each trial, subjects had a fat-rich meal containing 100 g of fat after 12 h of fasting (see Test meal section). In the exercise trials, the subjects jogged on a treadmill for a designated time period 12 h before the fat-rich meal ingestion. Choosing exercise 12 h before fat loading was based on previous findings (53, 55) documented that exercising ~12 h before fat loading more effectively attenuated postprandial PHTG than 24 h before or 1 h after a fat-meal intake. The exercise regimens were well tolerated by all of the participants. Blood samples were taken at baseline (before the fat-rich meal) and then at 2-h intervals for four times after the meal while the subjects remained at quiet rest and only allowed to walk a short distance.

**Preparatory dietary and physical activity control.** To reduce intra-subject variability, each participant completed a 24-h dietary record during the day immediately before the first trial. A copy of this diet record was given back to the participant before each subsequent trial. Subjects’ dietary intake the day before the exercise bout consisted of 9.6 ± 3.9 MJ with 47.0 ± 8.7% carbohydrate, 17.7 ± 6.8% protein, and 36.1 ± 5.4% fat. Fat intake consisted of 11.0 ± 2.6% saturated, 12.4 ± 3.2% monounsaturated, and 5.6 ± 2.5% polyunsaturated. Because of our repeated-measures design, subjects were required to replicate their diet during the 24-h period before each subsequent trial to avoid dietary variations in blood lipids. Telephone calls were made to each participant 2 days before each trial to remind them to follow the same 24-h diet that they recorded before their first trial. No exercise or alcohol was allowed 3 days before the experimental trials, and no caffeine intake was permitted 24 h before the trials. The fat-loading test was started at the same time of day for each trial to avoid possible time-induced metabolic variations. Subjects were only allowed to drink plain water during each testing period and were also restricted from exercise during each testing period except for the designated exercise testing.

**$V_{O2\text{peak}}$ test and exercise trials.** Each participant performed a graded $V_{O2\text{peak}}$ test on a treadmill to determine the exercise intensities used in subsequent exercise sessions. Briefly, subjects warmed up for 5 min on a treadmill. The initial speed of the treadmill was 1.79 m/s for the first 2 min of the test. After the initial 2 min, the treadmill speed was increased 0.22 m/s every minute until the treadmill speed was up to 2.91 m/s. Thereafter, the speed remained constant, and the treadmill grade was raised by 2% every minute until exhaustion (55). Oxygen consumption ($V_{O2}$) at time of exhaustion was considered as $V_{O2\text{peak}}$ if subject had also reached ±5 beats/min of their age-predicted maximal heart rate (HR). Respiratory gas exchange ($V_{O2}$ and carbon dioxide production) was measured via an indirect calorimetry (model 17670, Vista Min-CPX, Ventura, CA). HR was recorded using a Polar HR monitor (Polar USA, Woodbury, NY). The peak $V_{O2}$ obtained was considered the participant’s $V_{O2\text{peak}}$.

The initial workloads needed to elicit 60% of a participant’s $V_{O2\text{peak}}$ were interpolated from the subject’s $V_{O2\text{peak}}$ and workload obtained from the $V_{O2\text{peak}}$ test. Five minutes after the exercise was started, the workload was adjusted accordingly to maintain the target level of $V_{O2}$ throughout the exercise session. The average of $V_{O2}$ and the respiratory quotient (RQ) in the steady state of each exercise session was used for caloric expenditure calculation. The RQ values were used to determine the kilocalorie equivalent and the percentage of caloric expenditure derived from carbohydrate and fat. The rate of caloric expenditure was calculated by using Lusk’s equation (kcal/min = [4.686 + 1.232×(RQ – 0.707)]×$V_{O2}$; (33)).

**Test meal.** After 12-h fasting, a participant was given a fat-rich meal in a milkshake form, which was consumed within 10 min in each trial. The milkshake consisted of a combination of 270 ml of whipping cream and 65 g of specialty ice cream with walnuts. The milkshake provided 4.1 MJ (980 kcal; 0.05 MJ/kg body weight), 100 g fat (1.1 g/kg; 66.5 g saturated, 29.5 g monounsaturated, 4.0 g polyunsaturated), 17 g carbohydrate (0.2 g/kg), and 3 g protein (0.03 g/kg). This meal has been successfully used in previous studies to induce FHTG (55, 56).

**Blood sampling and analysis.** All blood samples were drawn from an antecubital vein. Ten milliliters of blood was drawn for each sample and collected in a Vacutainer containing EDTA. Blood samples were separated by centrifugation at 4°C for 15 min at 2,000 g. Plasma was stored at –80°C until analysis. Plasma TG and cholesterol concentrations were measured enzymatically using diagnostic kits (Infinity TG Reagent; Cholesterol reagent, procedure no. 353, Sigma, St. Louis, MO). Plasma glucose levels were measured using a diagnostic kit (Infinity Glucose Reagent, Sigma). Insulin levels were analyzed using a 125I RIA kit (ICN Pharmaceuticals, Costa Mesa, CA). Total HDL-C was measured by precipitating apolipoprotein B-containing lipoproteins with heparin-MnCl2. All plasma samples were diluted with 0.9% saline (1:1) before precipitation. The supernatant was used to assay HDL-C (54). Cholesterol content of the resulting solution was measured enzymatically (Cholesterol reagent, procedure no. 353, Sigma). To eliminate interassay variability, all samples from a single participant were analyzed together in each assay. As a surrogate measure of insulin resistance, HOMA-IR demonstrates a strong correlation to euglycemic clamp-measures of total glucose disposal (r = -0.820, P < 0.0001) (4, 23). HOMA-IR was evaluated using fasting plasma glucose and fasting insulin (4, 23) and calculated using Microsoft Excel based HOMA-IR 2 calculator provided by the Oxford Center for Diabetes, Endocrinology and Metabolism (31). The intra-assay coefficients of variation for TG, total cholesterol, HDL-C, glucose, and insulin were 1.5, 0.8, 1.4, 1.9, and 2.8%, respectively.

**Data analysis.** A two-way (trial × time) ANOVA with repeated measures was performed to test the effects of exercise duration on TG and insulin data. The magnitude of total TG response was also quantified as the TG AUC score calculated by the trapezoidal rule using the following formula: TG AUC score = $2\times(L_2 + L_4 + L_6 + L_8 - 7L_0)$, where $L_0$ is the TG concentration at n hours (37). The TG AUC score is a conventional index indicating plasma TG response to a fat-meal intake. Data of TG AUC score, fasting plasma glucose, and HOMA-IR were analyzed using one-way ANOVA with repeated measures. An ANOVA with significant F ratios (P < 0.05) was followed by Newman-Keuls post hoc tests. Additionally, a one-way ANOVA was performed for fasting insulin and fasting TG (0 h) to identify differences due to the effects of exercise duration. SAS software (version 8.02, Institute, Cary, NC) was used to perform the analysis. All data are reported as means ± SD unless otherwise noted.
RESULTS

The fasting blood status of participants were indicative of metabolic syndrome (fasting TG = 2.6 ± 0.4 mmol/l, total cholesterol = 5.4 ± 0.5 mmol/l, HDL-C = 0.9 ± 0.3 mmol/l, plasma insulin = 21.2 ± 2.2 μU/ml). The total caloric expenditures in the three exercise trials were different from each other (P < 0.001), but there were no differences in percent carbohydrate and fat expenditure or average exercise HR among the three exercise trials (Table 1). Figure 1A shows the plasma TG concentrations over time among the four trials. Fasting TG was significantly lower 12 h following 45 min-Ex and 60 min-Ex compared with Ctr (P < 0.04). However, fasting TG 12 h following 30 min-Ex was not significantly different compared with Ctr. Fasting plasma insulin was lower 12 h following all three exercise trials compared with the Ctr (P < 0.001). There was a significant trial main effect (F = 5.91, P = 0.004), and the post hoc analysis revealed that TG concentrations in both the 45 min-Ex and the 60 min-Ex were significantly lower than the Ctr (P < 0.03). At 0 h (before the fat-meal intake), TG concentration was lower than Ctr following 45 min-Ex and 60 min-Ex but not 30 min-Ex (P < 0.04). TG level following 60 min-Ex was lower than 30 min-Ex (P < 0.04), but not different from 45 min-Ex. There was no difference in TG at 0 h between 45 min-Ex and 30 min-Ex. At 2 h, TG concentrations did not significantly differ among the four trials. At 4, 6, and 8 h after the meal, both the 45 min-Ex and the 60 min-Ex had lower TG levels than the Ctr and 30 min-Ex (P < 0.05). There were no trial differences between Ctr and 30 min-Ex and between 45 min-Ex and 60 min-Ex over any time point. The data for TG AUC scores are illustrated in Fig. 1B. The TG AUC score in both 45 min-Ex (P = 0.016) and 60 min-Ex (P = 0.017) was ~30% lower than the Ctr. The TG AUC score in the 60 min-Ex was lower than the 30 min-Ex (P = 0.031), but it was compatible with 45 min-Ex. The TG AUC score in 45 min-Ex was lower than the 30 min-Ex, but it did not reach significant differences (P = 0.057).

Figure 2A presents the effect of different exercise durations on insulin concentrations over the 8-h period after a fat meal. There was a significant trial main effect (F = 5.89, P = 0.004), and the post hoc analysis revealed that insulin concentrations in all the three exercise trials were lower than the Ctr (P < 0.03). All exercise durations resulted in a significantly lower fasting (0 h) plasma insulin value compared with the nonexercise Ctr (P < 0.001). At 2 h, the insulin concentrations in all of the exercise trials were lower than the Ctr (P < 0.03); insulin concentration in the 60 min-Ex was lower (P < 0.03) than both the 30 min-Ex and the 45 min-Ex; however, the latter did not differ from each other (P > 0.05). Figure 2B illustrates fasting plasma glucose concentrations. There were no trial differences in the fasting plasma glucose concentration (P > 0.05). HOMA-IR data are presented in Fig. 3. HOMA-IR values in each exercise trial were lower than the Ctr (P < 0.03). There were no significant differences in HOMA-IR among the exercise trials.

Table 1. Metabolic and heart rate responses during the exercise sessions

<table>
<thead>
<tr>
<th>Exercise Trials</th>
<th>Total, MJ/session</th>
<th>Total CHO, %</th>
<th>Total Fat, %</th>
<th>Average Heart Rate, beats/min</th>
<th>%Estimated HRmax</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 min-Ex</td>
<td>1.3 ± 0.0a</td>
<td>80.3 ± 9.9</td>
<td>19.7 ± 9.9</td>
<td>139 ± 3.1</td>
<td>74 ± 5.4</td>
</tr>
<tr>
<td>45 min-Ex</td>
<td>1.9 ± 0.1b</td>
<td>82.0 ± 6.6</td>
<td>18.0 ± 6.6</td>
<td>138 ± 3.4</td>
<td>74 ± 5.5</td>
</tr>
<tr>
<td>60 min-Ex</td>
<td>2.5 ± 0.1c</td>
<td>82.6 ± 7.1</td>
<td>17.4 ± 7.1</td>
<td>145 ± 3.2</td>
<td>78 ± 5.1</td>
</tr>
</tbody>
</table>

Values are means ± SD, 30 min-Ex, 45 min-Ex, and 60 min-Ex, exercise at 60% of peak oxygen consumption for 30, 45, and 60 min, respectively. CHO, carbohydrate; Means not sharing a common lowercase letter are significantly different from each other over the trials (P < 0.001). %estimated HRmax, % of estimated maximal heart rate (220 − age).
Postprandial lipidemia is an integrative marker of TG metabolic capacity, resulting from production and clearance of TG-rich lipoproteins by various tissues. The major finding of this study is that men with metabolic syndrome exercising at 60% $V_{\text{O}_2}\text{peak}$ for 45 min or longer 12 h before to a fat-rich meal demonstrate reduced PHTG, whereas exercising for 30 min does not elicit such an effect. However, exercising for 30 min or more significantly lowers fasting plasma insulin, improves insulin action as represented by the HOMA-IR, and lowers the plasma insulin response to a fat-rich meal 12 h postexercise. Subjects in the present study had metabolic syndrome characterized by HTG accompanied with overweight, abdominal obesity, low level of HDL-C, and insulin resistance (1). Furthermore, these subjects demonstrated a dramatic PHTG response to ingestion of a fat-rich meal. Their plasma TG increased from 2.6 to 5.0 mmol/l in ~4 to 6 h following the meal in the Ctr trial and stayed at a high level for a long period (e.g., TG concentration was still 4.0 mmol/l at 8 h). Our findings correspond with those of Lewis et al. (32) in which the cumulative-increment in total plasma TG was about threefold higher in obese subjects than their nonobese counterparts following a fat-rich meal. By comparison, the plasma TG of healthy individuals approaches fasting level at 8 h after a fat-rich meal (55, 56).

Enhanced clearance of TG-rich lipoproteins is likely to be the major determinant of postexercise decreases in PHTG in our study (2). Understanding how exercise quality and quantity can best influence TG metabolism has widespread relevance. The present study is the first to directly assess the effect of exercise duration on PHTG in male subjects with metabolic syndrome.

We have shown that exercise at 60% $V_{\text{O}_2}\text{peak}$ for 45 min or 60 min 12 h preceding ingestion of a fat-rich meal significantly attenuated the fat-rich meal-induced PHTG response as reflected by both the TG concentration (Fig. 1A) and TG AUC score (Fig. 1B). Compared with Ctr, 45 min-Ex and 60 min-Ex had 31 and 33% PHTG reduction, respectively, as indicated by the TG AUC ($P < 0.02$), whereas the 30 min-Ex only had 8% PHTG reduction ($P > 0.05$). Furthermore, compared with 30 min-Ex, 60 min-Ex and 45 min-Ex had similar PHTG-lowering effect (The TG AUC scores were 27 and 25% lower than 30 min-Ex, respectively). However, compared with 30 min-Ex, the reduction in TG AUC score for 45 min-Ex did not reach statistical significance, although its $P$ value was nearly significant ($P = 0.057$). Nevertheless, the TG concentrations in 45 min-Ex were significantly lower than 30 min-Ex for most of the time during the 8-h postprandial period (Fig. 1A). These results indicate that moderate-intensity exercise for 45 min is necessary to effectively attenuate the exaggerated PHTG response in male subjects with metabolic syndrome. The elevated PHTG response in this population may result from low lipoprotein lipase (LPL) activity (49) and/or accumulation of both hepatically derived TG-rich VLDL and intestinally derived chylomicrons following a fat-rich meal (30).

Adipose tissue normally contributes to greater postprandial TG disposal than skeletal muscle (17). Following exercise, however, TG clearance by skeletal muscle increases to a greater extent than in adipose tissue (44). Enhanced muscle LPL mRNA following moderate-intensity exercise persists for nearly 20 h (24, 45). This increase may correspond to intra-
muscular triglyceride (IMTG) reduction during the previous exercise bout (25, 47). Although not assessed in the present study, higher activity of muscle LPL and lower IMTG levels in the 45 min-Ex and 60 min-Ex groups could be explained by enhanced chylomicron or VLDL-delivered lipid disposal into muscle to replenish IMTG while providing carbohydrate-sparing oxidative substrate (51).

In the present study, exercise for 30–60 min at 60% \( \dot{V}O_{2\text{peak}} \) likely had moderate effects on muscle glycogen and IMTG (42), resulting in increased whole body fat oxidation during recovery from exercise compared with the resting state (25, 26, 43). Although the relative contributions of IMTG and plasma TG to recovering muscle fat oxidation are unclear (25, 26), circulating plasma lipids could contribute significantly to increased muscle oxidation postexercise and could account for the improved PHTG following 45 and 60 min in our study. Intuitively, an increased total glycogen and fat utilization during as well as increased fat utilization following 45 min-Ex and 60 min-Ex compared with 30 min-Ex could account for enhanced lipid uptake by recovery muscle following a fat-rich meal. Modest effects of previous exercise on adipose LPL activity and gene expression (16, 44) may further act to help redirect circulating TG from adipose towards skeletal muscle and liver following exercise. This mechanism would greatly improve the inappropriate disposal of circulating fatty acids associated with insulin resistance (9).

The 12-h interval between the exercise bout and the fat-meal makes an effect on chylomicron appearance rate an unlikely explanation for the lower plasma TG levels in our study. Prior exercise does not delay gastric emptying (13) or peak postprandial chylomicron appearance in the plasma (11, 35) a day after exercise. Reduced hepatic VLDL secretion may contribute significantly to the improved PHTG 12 h postexercise because TG clearance and reduced chylomicron appearance cannot entirely account for this reduction. Indeed, indirect evidence for reduced hepatic VLDL appearance has been provided by Fukada et al. (10) and others (11, 34). Furthermore, fasting and postprandial nonesterified fatty acid concentrations are elevated a day following exercise (2), providing substrate for hepatic VLDL packaging (46).

Insulin resistance is an important risk factor for Type 2 diabetes and cardiovascular disease (41). Insulin resistance and hyperinsulinemia promote hepatic VLDL-TG secretion (32) and inhibit LPL-mediated clearance of TG-rich lipoproteins (29), which results in fasting and postprandial HTG. Exercise increases insulin sensitivity (21), lowers serum TG in subjects with HTG and insulin resistance (28), and attenuates the hyperinsulinemia following a fat-rich meal (52). Consequently, the exercise-induced decline in plasma insulin concentration could disinhibit muscle LPL activity (29) and promote lipid oxidation in skeletal muscles (8). The attenuated insulin response may also reduce hepatic secretion of VLDL-TG (20).

Previous studies demonstrated that energy expenditure is an important determinant of the magnitude of the exercise-induced reduction in PHTG (12). Our data showed that an exercise session (30 min-Ex) with caloric expenditure of 1.3 MJ (306 kcal) did not significantly reduce PHTG. This finding is consistent with the results from previous studies using a similar exercise regimen in normolipidemic subjects (3). In contrast, a recent study by Petitt et al. (39) did not demonstrate a significant reduction in TG AUC 16 h following a 90-min bout of walking exercise (1.6 MJ total expenditure) in young, lean subjects. It is possible that the lower exercise intensity of walking (11 vs. 26 \( \text{m}^2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \)) and use of smaller muscle mass than the running protocol used in the present study could have accounted for our conflicting findings. In the present study, exercise sessions expending 1.9 MJ (461 kcal; 45 min-Ex) or 2.5 MJ (606 kcal; 60 min-Ex) effectively mitigated PHTG. It is noteworthy that the effectiveness of PHTG reduction in 45 min-Ex and 60 min-Ex was comparable (Fig. 1A), suggesting that caloric expenditure at \(-1.7 \text{MJ (400 kcal)}\) may be sufficient to effectively reduce PHTG response. This finding is in line with other studies utilizing a single exercise duration (40).

In men exhibiting the metabolic syndrome, exercise at 60% \( \dot{V}O_{2\text{peak}} \) for 30 min or longer significantly mitigated fasting plasma insulin (Fig. 2A) and HOMA-IR (Fig. 3) in addition to attenuating the hyperinsulinemia following the fat-rich meal. Whereas 30 min-Ex and 45 min-Ex had similar insulin lowering effects, 60 min-Ex had the lowest insulin concentration at 2 h after the meal. It is noteworthy that the three exercise durations used in the present study resulted in similar reductions in HOMA-IR, suggesting that exercise for 30 min is sufficient for improving insulin action following a fat-rich meal in men with metabolic syndrome.

The exercise protocol used in the present study did not affect fasting plasma glucose 12 h following the exercise bout, similar to a previous study (52) showed exercising at 60% maximal \( \dot{V}O_{2} \) (\( \dot{V}O_{2\text{max}} \)). This suggests that the volume of an exercise bout may play a role in blood glucose homeostasis due to greater glycogenolysis and elevation of glycogen synthase activity (18). Because fasting plasma glucose did not change significantly, the attenuated insulin resistance observed in the exercise trials of the present study was primarily due to the reduction in insulin levels.

Limitations to the current study include use of HOMA-IR as a measure of acute exercise-mediated improvements in whole body insulin action. Although HOMA-IR corresponds to clamp-derived measurements of insulin sensitivity (4), it may not accurately define the exercise-mediated improvement in insulin sensitivity reported in the present study due to the relative small-scale study. Additionally, the pretrial self-reported dietary recall may not completely reflect the subjects’ true energy intake. Underreporting of energy intake associated with dietary recall may exceed 25% of daily energy intake (~400 kcal/day) (7, 36) and could also result in inaccurate estimates of macronutrients. It is important to point out that the prevalence of underreporting seems to be higher in overweight individuals (7, 36).

In conclusion, our results demonstrate that a moderate exercise bout [60% \( \dot{V}O_{2\text{max}} \); ~75% of estimated maximal HR (220 – age)] before a fat-rich meal ingestion for 45 min effectively attenuates PHTG response in men with metabolic syndrome. A 30-min exercise session noticeably reduced insulin resistance, although exercise for 60 min tends to lessen a meal-induced hyperinsulinemia more effectively. These data may provide precision to an exercise-oriented therapeutic measure for individuals with metabolic syndrome and give direction for further research to define the mechanisms behind exercise-mediated improvements in PHTG.
REFERENCES


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