Postexercise Macronutrient Intake and Subsequent Postprandial Triglyceride Metabolism

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ABSTRACT

TROMBOLD, J. R., K. M. CHRISTMAS, D. R. MACHIN, D. W. VAN PELT, T.H. CHOU, I.-L. KIM, and E. F. COYLE. Postexercise Macronutrient Intake and Subsequent Postprandial Triglyceride Metabolism. *Med. Sci. Sports Exerc.*, Vol. 46, No. 11, pp. 2099–2106, 2014. Acute endurance exercise has been shown to lower postprandial plasma triglyceride (PPTG) concentrations; however, whether this is due to the negative energy and/or CHO deficit from the exercise bout is not well understood. **Purpose**: This study aimed to examine the effects of a postexercise meal consisting of either high or low CHO content on PPTG and postprandial fat oxidation the morning after an exercise bout. **Methods**: Healthy young men (n = 6) performed each of four experimental treatments: 1) nonexercise control (CON), 2) 80 min of cycling with either no meal replacement (EX), 3) a high-CHO postexercise meal (EX+HCHO), or a 4) low-CHO postexercise meal (EX+LCHO). A standardized meal for PPTG determination was provided (16.0 kcal·kg^{-1} body mass, 1.02 g fat·kg^{-1}, 1.36 g CHO·kg^{-1}, 0.31 g protein·kg^{-1}) 12 h after the exercise, and measurements of plasma triglyceride (TG) concentration and whole-body resting fat oxidation were made in the fasted condition and during the 4-h postprandial period. **Results**: The total area under the curve for plasma TG was significantly lower in EX+LCHO (325 (63) mg·dL^{-1} per 4 h) compared with that in EX+HCHO (449 (118) mg·dL^{-1} per 4 h, P = 0.03). Postprandial fat oxidation during this period was significantly greater in EX+LCHO (257 (58) kcal per 4 h, P = 0.003) compared with that in EX+HCHO (209 (56) kcal per 4 h). The change in total postprandial fat oxidation (kcal per 4 h) relative to CON was significantly and inversely correlated with the change in the total TG area under the curve relative to CON (mg·dL^{-1} per 4 h, ΔTG AUC, R^2 = 0.37, P = 0.008). **Conclusions**: The low CHO composition of the postexercise meal contributes to lower PPTG and increased fat oxidation, with lower PPTG related to an increase in fat oxidation. **Key Words:** CARBOHYDRATE, FAT OXIDATION, GLUCOSE TOLERANCE, PHYSICAL ACTIVITY, TRIGLYCERIDE

BASIC SCIENCES

In developed countries where food availability is relatively high and advances in technology and automation lead to reduced physical activity, understanding the interaction between diet and physical activity on metabolic function is an increasing public health interest. Compared with fasting plasma triglyceride (TG) measurement, the transient increase in plasma TG after a meal rich in fat (i.e., postprandial plasma TG (PPTG)) may be a better predictor of cardiovascular disease incidence including nonfatal myocardial infarction, ischemic stroke, and other fatal cardiovascular events (3,36). Furthermore, low resting fat oxidation is an independent predictor of gain in body fat (23,37) and a determinant of the magnitude of PPTG elevation (11). As such, understanding TG elevation and fat oxidation after a mixed meal and after exercise can provide insight into the efficacy of lifestyle interventions related to treatment or preservation of metabolic health.

Acute endurance exercise (e.g., 30%–85% $\dot{V}O_{2peak}$) is an effective means to attenuate PPTG and increase fat oxidation after consumption of a high-fat meal (2,12,32). The PPTG-lowering effect of acute exercise has been thought to be mediated predominately by the total energy expended during the most recent exercise bout (12,35). However, exercise at intensities greater than 25%–40% $\dot{V}O_{2peak}$ is characterized by an increased reliance on CHO as an energy source (28,33), specifically from the skeletal muscle and liver glycogen. Furthermore, postexercise CHO status (i.e., glycogen depletion) has been shown to be significantly inversely correlated with resting fat oxidation (29), an effect that may drive the exercise-induced attenuation of PPTG elevation (11).

Both acute (26) and chronic (2 wk) (21) consumption of a low-CHO diet lowers both fasting and postprandial (31) plasma TG and VLDL secretion rate from the liver. This

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suggests that TG metabolism is lowered not just by increased negative energy balance but also by relative CHO intake or balance. However, the specific contribution of the CHO or energy deficit characteristic to the postexercise condition on PPTG remains controversial (5,10,14,18). It is clear that a high-CHO postexercise meal abolished the PPTG-lowering effects of exercise; however, it is unclear if this was due to energy or CHO replacement (14). To the best of our knowledge, a similarly designed study testing both a low- and high-CHO postexercise meal has not been performed with PPTG as the primary metabolic outcome.

We therefore investigated whether consumption of two different postexercise meals, differing only in CHO and fat content, would alter the PPTG-lowering effects of acute endurance exercise. We hypothesized that despite restoring energy balance, consumption of a low-CHO postexercise meal would preserve the PPTG-lowering effects of exercise.

METHODS

Subjects

Six healthy men, with no history of cardiovascular disease or metabolic dysfunction, were recruited from the university community to participate in this study (age, 25.3 (5.4) yr; body mass, 76.6 (12.8) kg; body fat, 14.1% (4.4%); height, 177.5 (5.7) cm; peak oxygen consumption ($\dot{V}O_{2peak}$), 49.3 (7.4) $mL \cdot kg^{-1} \cdot min^{-1}$). Throughout the duration of the study, subjects were asked to maintain their normal physical activity and eating patterns. This study was conducted under a protocol approved by the University of Texas at Austin institutional review board, and a written informed consent was obtained from each subject before participation.

Experimental Protocol

Each subject performed four 3-d treatments in a randomized order, with a minimum of 1 wk in between treatments (Fig. 1). Each treatment consisted of 2 d of diet and activity controls where subjects consumed the same laboratory-provided diet, performed no outside exercise, and monitored their daily step count. For three of the four treatments, on the evening of day 2, each subject performed an 80-min exercise session on a cycle ergometer, with 60 min performed at a moderate intensity (approximately 65% $\dot{V}O_{2peak}$), followed by ten 2-min high-intensity (approximately 80%–90% $\dot{V}O_{2peak}$) intervals (EX, EX+LCHO, and EX+HCHO). After the exercise session, each subject consumed either a high-CHO (EX+HCHO), an isoenergetic low-CHO meal (EX+LCHO), or no postexercise meal (EX). In addition, each subject performed a nonexercise control trial in which no exercise was performed on the evening of day 2 (CON). On the morning of day 3, fasting and postprandial blood was collected for measurements of plasma TG concentration, insulin concentration, glucose concentration, nonesterified fatty acids (NEFA) concentration, β-hydroxybutyrate (β-H) concentration, hematocrit count, and hemoglobin concentration. Blood draws were repeated hourly for 4 h and at 30 and 90 min postprandial. Resting energy expenditure and fat oxidation measurements were collected in the fasted condition and at 0.5, 1, 2, and 4 h postprandial.

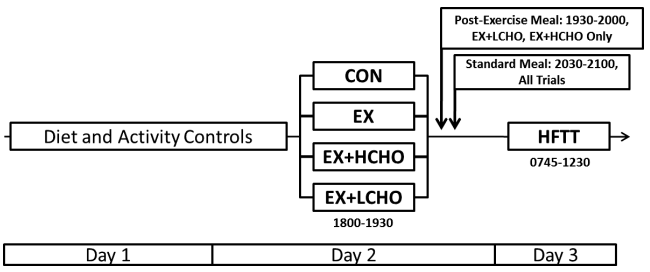

FIGURE 1—Overall protocol design. Each subject performed all four experimental trials, differing only in the treatment on the evening of day 2: CON, EX, EX+LCHO, and EX+HCHO. See text for details.

Exercise Protocols

Submaximal and $\dot{V}O_{2peak}$ determination. One week before the initiation of the first treatment, $\dot{V}O_{2peak}$ while cycling was determined by having each subject perform a submaximal cycling protocol with four 5-min stages at approximately 75, 125, 175, and 225 W (Lode, Groningen, Netherlands). After a 10- to 15-min rest period, each subject performed a $\dot{V}O_{2peak}$ test, as previously described (32). Oxygen consumption and carbon dioxide production were monitored during both submaximal and maximal cycling protocols by real-time breath-by-breath $\dot{V}O_2$ and $\dot{V}CO_2$ analysis using a mass spectrometer and a dual inspired–expired pneumotach (MA Tech Services, St. Louis, MO; Beck Integrative Physiology Systems). These data were used in concert to establish the exercise protocols for each subject.

Exercise intervention protocols. At approximately 1800–1830 hours on day 2 of the EX, EX+HCHO, and EX+LCHO treatments, subjects reported to the laboratory for the exercise protocol after an approximately 2-h fast. For all three exercise treatments, each subject cycled at 65% $\dot{V}O_{2peak}$ for 60 min followed immediately by ten 2-min intervals. Each interval consisted of 1-min "hard" and 1-min "easy" cycling. The "hard" portion was set at a work rate of approximately 95% $\dot{V}O_{2peak}$. A similar protocol has been used to examine the role of postexercise CHO intake and was shown to significantly decrease skeletal muscle glycogen (14). During the exercise trials, indirect calorimetry was used to estimate energy expenditure and CHO oxidation using real-time breath-by-breath $\dot{V}O_2$ and $\dot{V}CO_2$ measurements collected from minutes 0–10, 25–35, and 45–55 and throughout the 10 intervals (60–80 min) (MA Tech Services, St. Louis, MO; Beck Integrative Physiology Systems). The calculated rates were then summated to provide the total energy expenditure and CHO oxidation throughout the exercise protocol, as described previously (1,17).

Resting Energy Expenditure and Fat Oxidation

Resting energy expenditure was determined using indirect calorimetry by gas collection for 15 min after 10 min of rest in the supine position (models S-3A/I and CD-3A, respectively; Applied Electrochemistry, Pittsburgh, PA). These measurements were performed in the fasted condition during a familiarization visit to the laboratory, on day 3 of each treatment in the fasted condition, and 0.5, 1, 2, and 4 h postprandial. Total postprandial energy expenditure and fat oxidation were calculated.

The nonprotein respiratory quotient was collected using the $\dot{V}O_2$ and $\dot{V}CO_2$ relation under the assumption that protein oxidation would be determined by their 24-h protein intake (which was similar for all four treatments) and that protein oxidation during the meal test on day 3 would not change on the basis of the treatment. Total resting fat oxidation was calculated by substitution of resting energy expenditure ($kcal \cdot min^{-1}$) for fat oxidation rates.

Daily Activity

During the entire testing period, subjects were instructed to maintain their normal activity level during each treatment period. Each subject wore a pedometer during all waking hours during days 1–3 for all four treatments (Yamax Digi-Walker SW-200 pedometer; Great Performance Ltd., London, United Kingdom). After the first treatment, subjects were provided with their step count from days 1 and 2 and instructed to replicate this activity for the subsequent treatments.

Nutrition

Daily intake. On the familiarization visit, daily caloric requirement was determined as described previously with a physical activity correction of 1.5 (15). The estimated daily caloric requirement was used to prepare standardized meals matched for both energy and macronutrient content (isocaloric, 60% CHO, 15% protein, and 25% fat) for consumption on days 1 and 2 for each testing period to achieve energy balance during the CON treatment. Meals were consumed at a similar time each day for all treatments (800, 1200, 1600, and 2000 for day 1 and 800, 1200, 1600, and 2100 for day 2). The last meal before the high fat tolerance test (HFTT) was similar for all four treatments and was consumed between 2030 and 2100 hours and contained approximately 500 kcal, with 33% from CHO, 42% from fat, and 25% from protein. Alcohol and caffeine consumption were prohibited starting the evening before day 1 of each treatment.

HFTT. The HFTT meal was provided to the subject on the morning of day 3 (starting between 0730 and 0800 hours) after the fasting measurements. The meal consisted of a mixture of "half and half" and ice cream (Hill Country Farm; H. E. B., San Antonio, TX; approximately 16 $kcal \cdot kg^{-1}$ body mass, 1.02 g $fat \cdot kg^{-1}$, 1.36 g $CHO \cdot kg^{-1}$, and 0.31 g $protein \cdot kg^{-1}$). The subject remained comfortably seated where they were allowed to read or watch movies during the 4-h postprandial period.

Postexercise meal. In addition to the mixed meal consumed at approximately 2100 hours on day 2 for all four treatments, in both EX+HCHO and EX+LCHO, a postexercise meal was provided by the laboratory to be consumed within 10 min of completion of the exercise protocol on day 2 (approximately 1930–2000 hours). The caloric content of this meal was based on CHO oxidation during the first exercise trial, as described previously (1,9,17).

The postexercise supplement for EX+HCHO consisted of white bread, a 6%–10% sucrose drink (Kool-Aid; Kraft Foods, Northfield, IL), and soy protein powder (EX+HCHO: 878 kcal, 82.9% CHO, 4.4% fat, and 12.8% protein). The postexercise supplement for EX+LCHO was designed to be isoenergetic to the EX+HCHO supplement and consisted of peanuts (Planter's; Kraft Foods, Northfield, IL), heavy whipped cream (Hill Country Farm; H.E.B., San Antonio, TX), and water (EX+LCHO: 879 kcal, 12.5% CHO, 72.2% fat, and 10.2% protein). The treatments were designed for the EX trial to be in relative negative energy balance compared with those in CON, EX+HCHO, and EX+LCHO (i.e., approximately −880 kcal). CON and EX+HCHO were designed to achieve CHO balance, whereas EX and EX+LCHO were in negative CHO balance. EX+LCHO and EX+HCHO were designed to be matched in energy content.

Biochemical Analysis

On day 3, blood was collected in the fasted state and periodically over 4 h postprandial using a venous catheter in the antecubital vein of the right arm. Blood samples were collected in K_2 EDTA tubes (Vacutainer; BD, Franklin Lakes, NJ) and centrifuged immediately for 10 min at 2000*g* at 4°C. Plasma was aliquoted and stored at −80°C for later analysis (TG, insulin, NEFA, and β-HB concentration) (Pointe Scientific, Canton, MI; Alpco Diagnostics, Salem, NH; Wako Chemicals, Richmond, VA). Two additional plasma samples were collected for NEFA, insulin, and glucose analysis at 30 and 90 min postprandial. Both total and incremental area under the curve for plasma TG (TG AUC and TG_I AUC, respectively) were calculated using the trapezoidal method. In addition, fasting whole blood was used to measure hematocrit and hemoglobin counts to assess changes in plasma volume before the HFTT for each treatment (7) and was not different between trials. Intraassay coefficient of variation for TG, NEFA, β-H, insulin, and glucose level were 2.5%, 3.8%, 1.9%, 4.6%, and 1.9%, respectively.

Statistical Analysis and Calculations

Variables with single point outcomes (e.g., plasma TG AUC, fat oxidation, etc.) were analyzed using a one-way ANOVA with repeated measures. Fasting and postprandial plasma outcomes were analyzed with a two-way repeated-measures ANOVA for treatment and treatment–time interactions. If the treatment–time interaction was found to

be significant, pairwise comparisons were made. The change in total and incremental TG AUC (ΔTG AUC and ΔTG_I AUC), postprandial fat oxidation (ΔFO), fasting β-H (Δβ-H), energy balance (ΔEB), CHO balance (ΔCB), and average postprandial and fasting FFA (ΔNEFA) relative to CON was calculated by subtracting the EX, EX+LCHO, or EX+HCHO values from the CON value for each subject and each outcome. A two-tailed Pearson product-moment correlation analysis was used to assess the relation between ΔTG AUC compared with ΔFO and Δβ-H as well as ΔEB and ΔCB relative to ΔTG AUC and ΔFO. When applicable, the Fisher least significant difference *post hoc* correction was applied. Because of analysis of multiple outcomes and treatments, this may increase the chance for type I error. For all tests, significance was set at $P < 0.05$. Before the start of the study, an *a priori* power analysis was performed on the basis of a predicted difference in TG AUC of 100 $mg \cdot dL^{-1}$ per 4 h between EX+HCHO and EX+LCHO, with an SD of 80 for each outcome. All analyses were performed using SPSS software (SPSS Inc., Chicago, IL). Unless otherwise indicated, all data are reported as mean (SD).

RESULTS

Exercise Treatment

Total energy expenditure during the exercise trials were not different between EX, EX+LCHO, and EX+HCHO (193 (33), 197 (34), and 197 (34) L $\dot{V}O_2$, respectively, $P > 0.05$, and 876 (149), 898 (146), and 917 (136) kcal, respectively, $P > 0.05$). Total CHO oxidation during the exercise treatments was not different between EX, EX+LCHO, and EX+HCHO (634 (181), 668 (200), and 635 (134) kcal, respectively, $P > 0.05$).

Energy and Macronutrient Balance

There were no differences in overall relative energy balance between EX+LCHO and EX+HCHO (+20 (106) and +17 (125) kcal relative to CON, $P > 0.05$, respectively) (Fig. 1). These values were calculated on the basis of daily intake and exercise expenditure for total energy and CHO intake/expenditure. As designed, EX exhibited a negative energy balance that was significantly different from CON, EX+LCHO, and EX+HCHO (−866 (166) kcal relative to CON, $P < 0.05$ for all). Relative CHO balance in CON was significantly different from that in EX, EX+LCHO, and EX+HCHO (−640 (188), −542 (239), and +158 (117) kcal relative to CON, respectively, $P < 0.05$ for all). Both EX and EX+LCHO were significantly lower than EX+HCHO in terms of CHO balance ($P < 0.05$ for all) but not significantly different from each other ($P > 0.05$). These data confirm that our study design was successful in matching CON, EX+LCHO, and EX+HCHO for energy balance while attaining a relative CHO deficit in EX+LCHO.

Daily Activity

Daily step counts were not significantly different across all treatments for day 1 (8446 (1543), 8122 (1927), 7930 (1576), and 7895 (1692) steps for CON, EX, EX+HCHO, and EX+LCHO, respectively), day 2 (7463 (2247), 7076 (1490), 7357 (1447), and 7384 (997) steps for CON, EX, EX+HCHO, and EX+LCHO, respectively), and day 3 (494 (132), 570 (252), 493 (79), and 560 (115) steps for CON, EX, EX+HCHO, and EX+LCHO, respectively, $P > 0.05$ for all treatments on all days).

Resting Energy Expenditure and Fat Oxidation

Total resting energy expenditure on day 3 during the fasted and 4-h postprandial period of the HFTT was not significantly different between treatments (345 (59), 353 (37), 356 (57), and 342 (62) kcal for CON, EX, EX+LCHO, and EX+HCHO, respectively, $P > 0.05$ for all), except for that in EX+LCHO, which was significantly greater than that in CON. Fat oxidation during this period was significantly greater than that in CON (180 (51) kcal) in both EX (243 (48) kcal, $P = 0.005$) and EX+LCHO (257 (58) kcal, $P = 0.003$), with that in EX+HCHO (209 (56) kcal) not significantly different from that in CON ($P = 0.144$) (Fig. 2). Furthermore, postprandial fat oxidation in EX+HCHO was significantly lower than that in both EX ($P = 0.003$) and EX+LCHO ($P = 0.002$), but no significant difference was observed between that in EX and that in EX+LCHO ($P = 0.07$) (Fig. 2). Furthermore, ΔFO was significantly inversely correlated with ΔCB ($R^2 = 0.37$, $P = 0.007$) but not ΔEB ($R^2 = 0.06$, $P = 0.32$). These data suggest that higher postprandial fat oxidation is related to the magnitude of postexercise CHO deficit either from exercise or from postexercise dietary intake.

Plasma Analysis

Plasma TG. The total PPTG responses from day 3 are presented in Figure 3. There was an overall treatment–time interaction effect ($P = 0.004$). In the fasted condition, both of

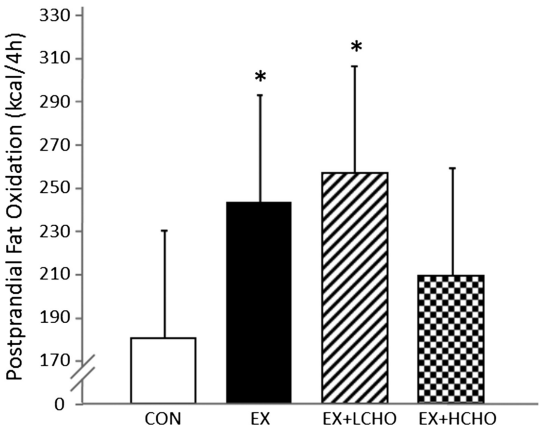

FIGURE 2—Fasting and postprandial fat oxidation (kcal per 4 h). Treatments were CON, EX, EX+LCHO, and EX+HCHO. Values expressed as mean (SD). *Significantly greater than CON and EX+HCHO.

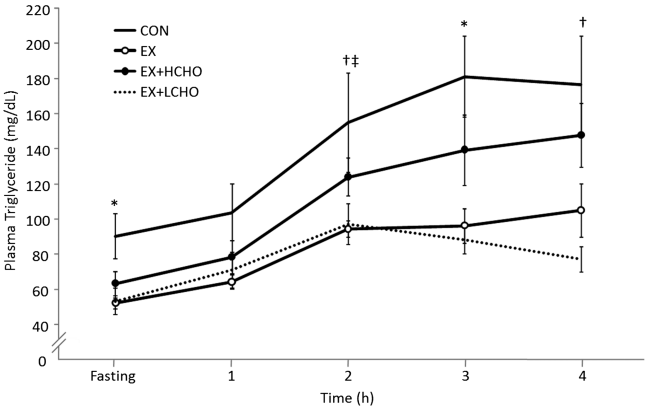

FIGURE 3—Plasma TG concentration (mg·dL^{-1}) during the 4-h postprandial TG test. Treatments were CON, EX, EX+LCHO, and EX+HCHO. Values reported as mean ± SE. *EX and EX+LCHO were significantly lower than CON. †EX+LCHO was significantly lower than CON and EX+HCHO. ‡EX+HCHO was significantly greater than EX.

those in EX and EX+LCHO were significantly lower than those in CON (P = 0.021 and P = 0.043, respectively) and there was a trend for those in EX+HCHO to be less than those in CON (P = 0.050). In response to the HFTT, the total plasma TG AUC was significantly lower in EX (334 (32) mg·dL^{-1} per 4 h, P = 0.03), EX+LCHO (325 (63) mg·dL^{-1} per 4 h, P = 0.01), and EX+HCHO (449 (118) mg·dL^{-1} per 4 h, P = 0.02) compared with that in CON (583 (188) mg·dL^{-1} per 4 h). EX+LCHO and EX+HCHO were not significantly different than EX (P = 0.81 and P = 0.09, respectively), but EX+LCHO was significantly lower than EX+HCHO (P = 0.03) (Fig. 4). There were no significant differences in incremental AUC between all treatments, although the general effect was similar to total TG AUC.

The change in total postprandial fat oxidation (kcal per 4 h) relative to CON (ΔFO) was significantly inversely correlated with the change in the total and incremental TG AUC (mg·dL^{-1} per 4 h; ΔTG AUC and ΔTG_I AUC, R^2 = 0.37, P = 0.008, and R^2 = 0.28, P = 0.023, respectively)

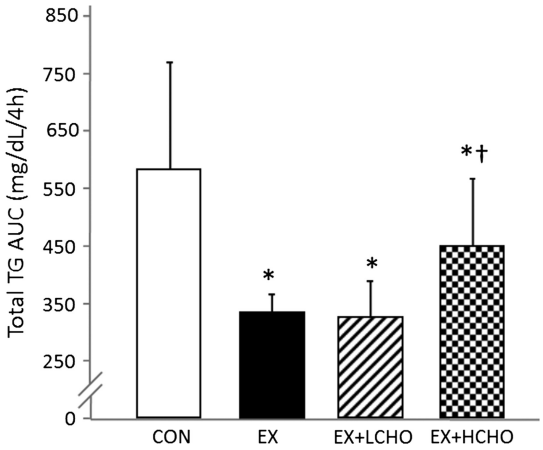

FIGURE 4—Total plasma TG AUC. Treatments were EX, EX+LCHO, and EX+HCHO. Data are reported as mean (SD). *Significantly less than CON. †Significantly greater than EX+LCHO.

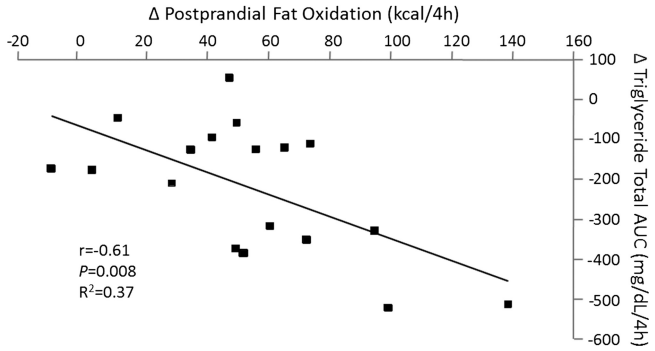

FIGURE 5—Relation between the change in total plasma TG AUC relative to CON (mg·dL^{-1} per 4 h, ΔTG AUC) and the change in total postprandial fat oxidation relative to CON (kcal per 4 h; ΔFO, r = −0.61, R^2 = 0.37, P = 0.008).

(Fig. 5). Furthermore, both ΔTG AUC were significantly directly correlated with ΔCB (R^2 = 0.26, P = 0.03) but not ΔEB (R^2 = 0.06, P = 0.25). This suggests that the magnitude of decrease in PPTG postexercise is at least partially related to an increase in postprandial fat oxidation, an effect likely to be mediated by CHO deficit, either by diet and/or exercise.

Plasma glucose and insulin. Plasma insulin and concentrations were not significantly different across all four treatments, fasting and postprandial (Fig. 6B and C, respectively).

Fasting β-H. There were no significant differences in fasting β-H (surrogate marker for hepatic fat oxidation) concentration across all four treatments (0.159 (0.05) mM, 0.252 (0.11) mM, 0.253 (0.09) mM, and 0.155 (0.018) mM for CON, EX, EX+LCHO, and EX+HCHO, respectively, P > 0.05 for all), with the exception of that in EX being significantly greater than that in CON (P = 0.033). Both EX and EX+LCHO were not significantly different from EX+HCHO (P = 0.067 and P = 0.053, respectively). There was a significant inverse relation between ΔTG AUC and Δβ-H (R^2 = 0.39, P = 0.006). This supports the relation observed between high postprandial whole-body fat oxidation and low PPTG observed postexercise.

Plasma NEFA. There was a significant overall treatment effect (P < 0.05) but no significant treatment–time interaction for plasma NEFA (P = 0.07) (Fig. 6A). Average fasting and postprandial plasma NEFA concentrations were significantly higher in EX (0.382 (0.143) mM), EX+LCHO (0.405 (0.097), mM) and EX+HCHO (0.324 (0.088) mM) compared with those in CON (0.281 (0.081) mM, P = 0.024, P = 0.001, and P = 0.011, respectively). Furthermore, EX+HCHO was significantly lower than EX+LCHO (P = 0.015). In addition, ΔNEFA from CON was significantly positively related to ΔFO (R^2 = 0.28, P = 0.026) and β-H (R^2 = 0.51, P = 0.001).

DISCUSSION

The primary finding of the present study was that despite similar energy balance, consumption of a postexercise low-CHO meal resulted in lower PPTG and increased fat oxidation

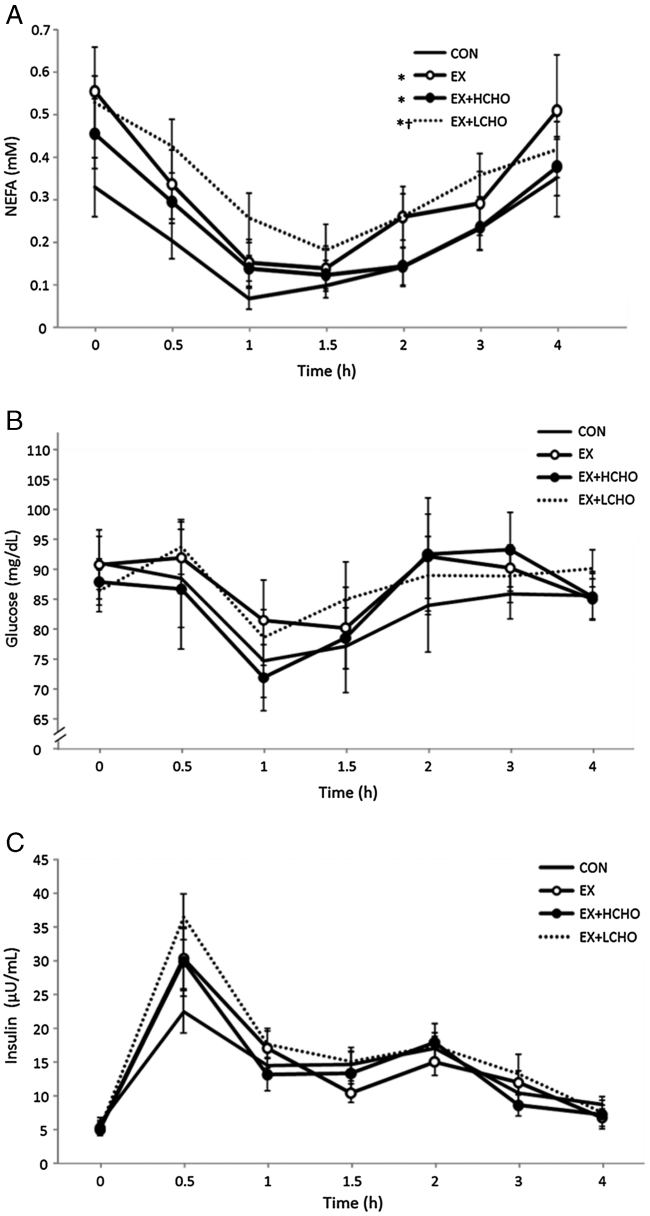

FIGURE 6—Plasma NEFA concentration (mM) (A), glucose concentration (mg·dL^{-1}) (B), and insulin (μU·mL^{-1}) (C). Treatments were EX, EX+LCHO, and EX+HCHO. All significance notations are reported for treatment effects. Data are reported as mean ± SE. *Significantly greater than CON. †Significantly greater than EX+HCHO.

compared with those in consumption of an isoenergetic postexercise high-CHO meal. Furthermore, the improvement in PPTG was related to increases in fat oxidation and CHO deficit but not energy deficit. To the best of our knowledge, this is the first study to show that the PPTG response is at least partially related to changes in fat oxidation and CHO balance rather than energy balance alone.

Acute endurance exercise has consistently been shown to attenuate PPTG, with the magnitude of the exercise effect thought to be mostly dependent on the energy expenditure during the most recent exercise bout (12,35). Furthermore, postexercise energy replacement with a mixed meal (approximately 48% CHO) has been shown to prevent the PPTG-lowering effect of exercise (energy expenditure, approximately 450 kcal) (5). Conversely, Freese et al. (10) reported that postexercise PPTG was significantly lower than a nonexercise control condition despite 100% of the exercise energy expenditure being replaced with a postexercise mixed meal (approximately 200 kcal, approximately 46% CHO). However, after 90 min of exercise at 60%–65% $\dot{V}O_{2peak}$, maintenance of the postexercise CHO deficit by refeeding a low-CHO meal resulted in significantly lower fasting plasma TG to refeeding an isoenergetic higher CHO meal (22). In the postprandial state, using a very similar experimental design as the present study, Harrison et al. (14) provided a postexercise CHO replacement meal that abolished the exercise-induced attenuation of PPTG elevation compared with that when no postexercise meal was provided (i.e., maintenance of the energy deficit). This study indicates a more prominent role of CHO deficit over fat deficit to mediate the exercise effects on postprandial metabolism; however, without an energy-matched low-CHO group, this interaction was left unresolved (10). In the present study, despite similar energy balance, PPTG was significantly higher in EX+HCHO compared with that in EX+LCHO when assessed by total AUC (449 (118) and 325 (63) kcal per 4 h) and peak postprandial plasma TG concentration (156.9 (41.9) mg·dL^{-1} and 106.0 (21.5) mg·dL^{-1}, respectively). These findings suggest a unique effect of restoration of the CHO deficit from exercise to at least partially prevent the PPTG-lowering effects of exercise.

Despite the recent findings indicating that CHO depletion during exercise plays an important role in determining the TG-lowering effects of acute exercise (14,22), not all research supports this conclusion. Administration of the lipolysis inhibitor, acipimox, before 90 min of running at approximately 60% $\dot{V}O_{2peak}$, resulted in a similar attenuation of PPTG compared with that in placebo despite a modest increase of CHO oxidation by approximately 33 g or 132 kcal during the exercise bout in the acipimox treatment (18). Conversely, when CHO oxidation during exercise is relatively large and not refed (approximately 700- to 1000-kcal CHO deficit), CHO balance seems to be strongly associated with the exercise-induced lowering of fasting and postprandial plasma TG concentration (14,22). In the present study, EX+LCHO and EX+HCHO were in energy balance but EX+LCHO had a relative CHO deficit of −700 kcal compared with that in EX+HCHO, and this coincided with lower PPTG. Interestingly, despite refeeding all of the energy and having a significantly greater CHO balance compared with that in CON (+158 kcal), TG AUC was significantly lower in EX+HCHO compared with that in CON, suggesting that exercise may lower PPTG, independent of both energy and CHO deficit.

Postprandial fat oxidation is inversely related to PPTG (8) and CHO balance (i.e., glycogen content in the liver and skeletal muscle) when manipulated by diet and exercise (29). These findings are in agreement with the present study where the change in fat oxidation compared with that in

CON was significantly inversely correlated with the change in both the total TG AUC (R^2 = 0.37, P = 0.008) and CHO balance (R^2 = 0.37, P = 0.007). Furthermore, in agreement with previous work (11), the change in β-H compared with that in CON, a surrogate marker of hepatic CHO status (27), was significantly inversely correlated with ΔTG AUC (R^2 = 0.38, P = 0.006). Conversely, increased fat oxidation may simply be a compensatory response from increased fatty acid availability because both ΔFO and Δβ-H were significantly correlated with ΔNEFA. Importantly, these are just static plasma NEFA measures, and it does not account for fatty acid flux into the skeletal muscle and hepatic cells. Taken together, increased fat oxidation seems to at least partially determine the PPTG-lowering effects of exercise.

Both acute and chronic consumption of a high-CHO/low-fat diet without exercise lead to increased plasma TG concentration in both the fasted and postprandial states (21,24,26,31). This effect is likely a result of decreased hepatic fatty acid oxidation contributing to increased VLDL output from the liver (21,30) and decreased peripheral TG uptake (24). These findings, paired with the known effects of insulin to inhibit fat oxidation (6) and to decrease R_aFFA (25), may explain why high CHO intake could elevate plasma TG concentration while decreasing plasma NEFA concentration. In light of this, in the previously mentioned study by Malkova et al. (18), PPTG may have been similar in the acipimox trial not only because of the relatively small CHO deficit but also because CHO balance was adjusted without changing CHO intake and, thus, not changing insulin as much as after a high-CHO meal. In the present study, despite similar fasting insulin across all four treatments, average plasma NEFA concentrations were significantly different between EX+LCHO (0.405 (0.097) mM) and EX+HCHO (0.324 (0.088) mM, P = 0.015), reflecting a potential lasting effect of insulin on lipolysis. In the present study, the energy and macronutrient content of the last meal on day 2 of each treatment was controlled (i.e., approximately 2100 in the evening of day 2, 11 h before HFTT) and the postexercise meals in EX+LCHO (approximately 25 g CHO) and EX+HCHO (approximately 200 g CHO) were both consumed 12 h before the HFTT. The direct effects of insulin from the evening meal in EX+HCHO to inhibit fat oxidation may have been "washed out" before the HFTT. With this in mind, an alternate explanation is that if postexercise glycogen remains low (i.e., EX and EX+LCHO), more of the CHO provided in the HFTT would be stored as glycogen (4,16) rather than oxidized (20), resulting in increased fat oxidation and decreased PPTG in these trials compared with those in CON and EX+HCHO.

This study has several limitations. First of all, the use of a 4-h postprandial instead of the more common 6- to 8-h test may have masked some potential findings; however, Weiss et al. (34) demonstrated that the 4-h test is significantly correlated with the 8-h test for measurement of total and incremental TG AUC (R^2 = 0.92 and 0.89, respectively). Another limitation is that because this is an acute study, despite statistical significance, it is difficult to ascertain the long-term clinical relevance of attenuating PPTG and increasing fat oxidation by reducing postexercise CHO intake. Exercise training in combination with a reduced CHO diet has been shown to be an effective means to attenuate PPTG in obese individuals (19); however, to our knowledge, no study has been conducted comparing this with an exercise training program with an isoenergetic higher-CHO meal. Lastly, this study was conducted in relatively young healthy men and does not necessarily extend to women or individuals that are obese or have preexisting metabolic impairment, although both lean and obese men show an attenuation of PPTG after acute endurance exercise (13).

In conclusion, the present study indicates that a low CHO composition of a postexercise meal contributes to reduced PPTG and increased fat oxidation, independent of energy content, and the increase in fat oxidation is related to the postprandial TG-lowering effects of exercise.

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