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Acute high-intensity endurance exercise is more effective than moderate-intensity exercise for attenuation of postprandial triglyceride elevation

Justin R. Trombold, Kevin M. Christmas, Daniel R. Machin, Il-Young Kim, and Edward F. Coyle

The Human Performance Laboratory, Department of Kinesiology and Health Education, The University of Texas at Austin, Austin, Texas

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Trombold JR, Christmas KM, Machin DR, Kim I, Coyle EF. Acute high-intensity endurance exercise is more effective than moderate-intensity exercise for attenuation of postprandial triglyceride elevation. *Am J Physiol Endocrinol Metab* 114: 792–800, 2013. First published January 31, 2013; doi:10.1152/japplphysiol.01028.2012.—Acute exercise has been shown to attenuate postprandial plasma triglyceride elevation (PPTG). However, the direct contribution of exercise intensity is less well understood. The purpose of this study was to examine the effects of exercise intensity on PPTG and postprandial fat oxidation. One of three experimental treatments was performed in healthy young men ($n = 6$): nonexercise control (CON), moderate-intensity exercise (MIE; 50% $\dot{V}O_{2peak}$ for 60 min), or isoenergetic high-intensity exercise (HIE; alternating 2 min at 25% and 2 min at 90% $\dot{V}O_{2peak}$). The morning after the exercise, a standardized meal was provided (16 kcal/kg BM, 1.02 g fat/kg, 1.36 g CHO/kg, 0.31 g PRO/kg), and measurements of plasma concentrations of triglyceride (TG), glucose, insulin, and β-hydroxybutyrate were made in the fasted condition and hourly for 6 h postprandial. Indirect calorimetry was used to determine fat oxidation in the fasted condition and 2, 4, and 6 h postprandial. Compared with CON, both MIE and HIE significantly attenuated PPTG [incremental AUC; 75.2 (15.5%), $P = 0.033$, and 54.9 (13.5%), $P = 0.001$], with HIE also significantly lower than MIE ($P = 0.03$). Postprandial fat oxidation was significantly higher in MIE [83.3 (10.6%) of total energy expenditure] and HIE [89.1 (9.8) %total] compared with CON [69.0 (16.1) %total, $P = 0.039$, and $P = 0.018$, respectively], with HIE significantly greater than MIE ($P = 0.012$). We conclude that, despite similar energy expenditure, HIE was more effective than MIE for lowering PPTG and increasing postprandial fat oxidation.

cycling; energy expenditure; fat oxidation; postprandial plasma triglycerides

IN MODERN SOCIETY, WHERE LIFESTYLE IS CHARACTERIZED by an increase in sedentary activities as well as the ease with which food is obtained, most individuals spend the majority of their day in the postprandial state, when physical activity is low. Compared with fasting plasma triglyceride (TG) measurement, the transient increase in plasma TG following a meal rich in fat [i.e., postprandial plasma TG (PPTG)] may be a better predictor of cardiovascular disease incidence, including nonfatal myocardial infarction and ischemic stroke as well as other fatal cardiovascular events (4, 53). Furthermore, low resting fat oxidation is an independent predictor of gain in body fat (34, 54) as well as a determinant of the magnitude of PPTG (12, 13, 27). Acute endurance exercise (30–75% $\dot{V}O_{2max}$) is an effective means to attenuate PPTG and increase fat oxidation following consumption of a high-fat meal (3, 14, 15). The plasma triglyceride-lowering effects of acute exercise have been thought to be mediated predominantly by the total energy expended (14, 52), total carbohydrate oxidation during the most recent exercise bout (18), postexercise fat oxidation (13), and/or muscle lipoprotein lipase (mLPL) activity (20). However, the independent effects of exercise intensity on PPTG are not well understood (22, 45, 47).

It is unclear whether elevation from low-intensity (i.e., ~30% $\dot{V}O_{2max}$, walking) to moderate-intensity exercise (i.e., ~60% $\dot{V}O_{2max}$, jogging) further reduces PPTG when energy expenditure is similar (22, 47). Furthermore, investigation of an exercise intensity effect on triglyceride metabolism at intensities greater than 50–65% $\dot{V}O_{2max}$ has yielded mixed results when assessed in both the postprandial (10, 45, 48) or fasting condition (7, 8). These discrepancies may be explained by the nature of the diet and activity controls (10, 48), timing of the high-fat tolerance test (10, 22, 47), the energy expended during the most recent exercise session (52), the intensities being compared, or whether the TG assessment was performed in the fasting or postprandial condition.

Both acute (40) and chronic (2 wk) (32) consumption of a low-carbohydrate diet reduces both fasting plasma TG and VLDL secretion rate from the liver compared with an isoenergetic high-carbohydrate diet, suggesting that TG metabolism is altered by relative carbohydrate intake independent of energy balance. Despite this, the specific contribution of a carbohydrate or energy deficit to the postexercise condition and PPTG is controversial (18, 28). However, acute exercise oxidizes glycogen from the liver and skeletal muscle, increases postexercise fat oxidation, and reduces fasting TG, an effect that is abolished when a carbohydrate-rich postexercise meal is provided (33). Similar to altering carbohydrate balance by diet alone, increasing exercise intensity from 50 to 90% $\dot{V}O_{2max}$ increases total carbohydrate oxidation (plasma glucose and intramuscular glycogen) two- to fourfold (5, 41, 49). Consequently, increasing carbohydrate oxidation during exercise by increasing intensity may further attenuate PPTG and increase resting fat oxidation the morning after exercise despite similar energy expenditure.

The purpose of this study was to examine the effects of acute, continuous moderate-intensity endurance exercise (MIE; 50% $\dot{V}O_{2peak}$) and intermittent high-intensity endurance exercise (HIE; 90% $\dot{V}O_{2peak}$) on PPTG and postprandial fat oxidation. We hypothesized that both HIE and MIE will attenuate PPTG compared with a nonexercise control (CON), with greater effect in the HIE treatment compared with MIE. Ad-

Address for reprint requests and other correspondence: E. F. Coyle, Univ. of Texas at Austin, One University Station, Bellmont Hall, Rm. 222K, Austin, TX 78712 (e-mail: coyle@mail.utexas.edu).

ditionally, we hypothesized that the PPTG-lowering effects of exercise will be related to postprandial fat oxidation.

METHODS

Subjects

Six healthy men with no prior history of cardiovascular disease, metabolic dysfunction, or current orthopedic injury were recruited from the university community to participate in this study [age: 25.0 (2.9) yr; body mass: 81.5 (10.3) kg; body fat: 15.5 (4.3)%; height: 178.6 (7.4) cm; peak oxygen consumption while cycling ($\dot{V}O_{2peak}$): 55.5 (1.3) ml·kg^{-1}·min^{-1}]. Subjects were recreationally active (i.e., each performed regular physical activity but were not formally training for an endurance competition of any kind), which may explain the moderately high maximal aerobic capacity. Two of the six subjects were considered overweight (BMI of 28 and 27, respectively) but had relatively low body fat percentage (14 and 20%, respectively). 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 written informed consent was obtained.

Experimental Protocol

Each subject performed three 3-day treatments in a randomized order, with a minimum of 1 wk between treatments. Each treatment consisted of 2 days of diet and activity controls, where subjects consumed a laboratory-provided diet, performed no outside exercise, and monitored their daily step count. On the evening of *day 2*, each subject performed one of three treatments in a randomized order. Either ~60 min of cycling at 50% $\dot{V}O_{2peak}$ (MIE) or an isoenergetic cycling exercise for ~40–45 min consisting predominantly of intervals alternating between 2 min at 25% and 2 min at 90% $\dot{V}O_{2peak}$ (HIE; described below) was performed. The other trial was a CON. On the morning of *day 3*, subjects reported to the laboratory following an 11-h overnight fast. Upon arrival at the laboratory, fasting blood was collected for measurements of plasma TG, glucose, insulin, and β-hydroxybutyrate (β-HB) as well as hematocrit and hemoglobin. Immediately after the blood collection, resting energy expenditure and fat oxidation were determined in the supine position. After completion of the fasting measurements, a large mixed meal [16 kcal/kg, 1.02 g fat/kg, 1.36 g carbohydrate (CHO)/kg, and 0.31 g pro/kg] was consumed. Blood draws were repeated hourly for 6 h postprandial, with resting energy expenditure and fat oxidation measurements repeated at 2, 4, and 6 h postprandial.

Approximately 1 wk prior to the start of the study, each subject reported to the laboratory for familiarization and for necessary prestudy testing (FAM1) to be performed. During this visit, resting indirect calorimetry was performed to ensure that each subject was comfortable with the measurement and for calculation of resting metabolic rate. Following the resting measurements, height, weight, and body composition (skinfold measurement) were determined. After anthropometric measurements were taken, submaximal and maximal cycling protocols were performed to determine the work rate and target energy expenditure for the exercise treatments in HIE and MIE.

Exercise Protocols

Submaximal and peak $\dot{V}O_2$ determination. One week prior to the initiation of the first treatment, $\dot{V}O_{2peak}$ while cycling was determined by first having each subject perform a submaximal cycling protocol with four 5-min stages at 75, 125, 175, and 225 watts (Lode, Gronigen, The Netherlands). Following a 10- to 15-min rest period, each subject performed a $\dot{V}O_{2peak}$ test. Briefly, the first stage was set at a work rate to achieve ~75–80% of predicted $\dot{V}O_{2peak}$ based on the submaximal heart rate and oxygen consumption vs. work rate regression equations and age-predicted maximum heart rate (208 − 0.7 × age) (46). Following the first 4-min stage, the work rate was increased every 2 min until 10 min and then increased at 1-min increments thereafter with the aim of reaching each subject's predicted $\dot{V}O_{2peak}$ at 6–8 min. The subject was verbally encouraged to continue the maximal cycling test until volitional fatigue. Peak oxygen consumption was determined from the final 30 s prior to exhaustion during the maximal protocol. 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, and Beck Integrative Physiology Systems; Hans Rudolph, Kansas City, MO).

The regression equation of oxygen consumption vs. work rate from the submaximal test and the $\dot{V}O_{2peak}$ determined during the maximal oxygen consumption test was used to set the work rates for MIE and HIE that correspond to 50 and 90% $\dot{V}O_{2peak}$, respectively. Additionally, $\dot{V}O_2$ and $\dot{V}CO_2$ measurements from the submaximal protocol were used to determine the target energy expenditure for the first exercise trial based on the energy cost of cycling at 50% $\dot{V}O_{2peak}$ for 60 min (26):

$$\text{Exercise energy expenditure (EE; kcal/min)} = \left[3.716 \times \left(\text{Vo}_2\ \text{l/min}\right)\right] + \left[1.332\ \left(\text{Vco}_2\ \text{l/min}\right)\right] - \text{resting energy expenditure}$$

One to three days following FAM1, each subject returned to the laboratory to perform a work rate verification trial (FAM2). Briefly, each subject cycled for ~5–7 min at 50 and 90% of $\dot{V}O_{2peak}$ to verify the target oxygen consumption and energy expenditure determined from FAM1.

Exercise intervention protocols. At 1900 on *day 2* of the MIE and HIE treatments, subjects reported to the laboratory for the exercise protocol following an ~6-h fast. For the first exercise treatment (MIE or HIE), the target energy expenditure for the exercise treatment was determined as described above, with the duration of the second exercise protocol matched to the energy expenditure from the first exercise treatment (26). For the MIE treatment, subjects cycled at ~50% $\dot{V}O_{2peak}$ for ~60 min. For the HIE treatment, subjects cycled at 40% $\dot{V}O_{2peak}$ for 4 min, followed immediately by 5 min at ~90% $\dot{V}O_{2peak}$ and then 3 min at 25% $\dot{V}O_{2peak}$. Immediately following this first 12 min, each subject performed intervals consisting of 2 min at 90% $\dot{V}O_{2peak}$ and 2 min at 25% $\dot{V}O_{2peak}$. During the exercise trials, indirect calorimetry was used to measure energy expenditure using real-time breath-by-breath $\dot{V}O_2$ and $\dot{V}CO_2$ measurements collected from *minutes 5–10*, *25–30*, and *45–50* for MIE and *minutes 0–12*, *20–24*, and *36–40* for HIE (MA Tech Services and Beck Integrative Physiology Systems). The duration of the second exercise treatment was adjusted to match the energy expenditure from the first exercise bout.

Resting Energy Expenditure and Substrate Oxidation

Resting energy expenditure was determined using indirect calorimetry by gas collection for 15 min following 10 min of rest in the supine position. This was accomplished using carbon dioxide and oxygen analyzers in conjunction with a mixing chamber and calibrated dry gas meter for expired volume determination (models S-3A/I and CD-3A, respectively; Applied Electrochemistry, Pittsburgh, PA). These measurements were performed in the fasted condition on FAM1 as well as on *day 3* of each treatment immediately following the blood draw in the fasted condition and then repeated 2, 4, and 6 h postprandial. For metabolic data collection during FAM1 and on *day 3*, total postprandial energy expenditure (as calculated above for the exercise data) and relative contributions of fat and carbohydrate oxidation were calculated as (2, 26) follows:

$$\text{Resting energy expenditure (REE; kcal/min)}$$

$$\text{REE} = \left[3.716 \times \left(\text{Vo}_2\ \text{l/min}\right)\right] + \left[1.332\ \left(\text{Vco}_2\ \text{l/min}\right)\right]$$

$$\text{Carbohydrate oxidative rate}\ \left(\text{kcal/min}\right) = \left(\left(\left(\text{NPRQ} - 0.707\right)/0.293\right) \times 100\right) \times \text{REE}$$

$$\text{Fat oxidation rate}\ \left(\text{kcal/min}\right) = \text{REE} - \text{carbohydrate oxidation rate}$$

$$\text{Total energy expenditure}\ \left(\text{TEE; kcal/6 h}\right)$$

$$\text{TEE} = \left(\text{fasting REE} \times 60\right) + \left(2\ \text{h REE} \times 120\right) + \left(4\ \text{h REE} \times 120\right) + \left(6\ \text{h REE} \times 60\right)$$

The nonprotein respiratory quotient (NPRQ) was collected using the relationship between $\dot{V}O_2$ and $\dot{V}CO_2$ under the assumption that protein oxidation would be determined by their 24-h protein intake (which was similar for all 3 treatments) and that protein oxidation during the meal test on *day 3* would not change based on the treatment. Total fat and total carbohydrate oxidation during the fasting and postprandial period were calculated using the above equation for TEE by substitution of REE (kcal/min) for the temporally appropriate fat and carbohydrate oxidation rates as calculated above.

Daily Activity

During the entire testing period, subjects were instructed to maintain their normal activity level and refrain from both formal and recreational exercise. Each subject wore a pedometer at all times when not sleeping for *days 1–3* for all three treatments (Yamax Digi-Walker SW-200 pedometer; Great Performance, London, UK). Subjects were instructed to keep their daily step count between 7,000 and 9,000 steps on *days 1* and *2* for each testing period, with the step count evenly spaced out throughout the day. 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 FAM1, daily caloric requirement was determined from resting oxygen consumption and carbon dioxide production in conjunction with the Harris-Benedict equation, with an activity correction of 1.3 and fat-free mass determined by skinfold measurement (23). The estimated daily caloric requirement was used to prepare standardized meals matched for both energy and macronutrient content (isocaloric, daily macronutrients: 60% carbohydrate, 15% protein, and 25% fat) for consumption on *days 1* and *2* for each testing period to achieve energy balance during the CON treatment. As a result, both MIE and HIE were in a similar negative energy balance relative to CON. Meals were consumed at a similar time each day for all treatments. On *day 1*, meals were provided between 0700 and 0800, 1200 and 1300, and 1800 and 1900, with similar energy and macronutrient content in all three meals [833 (77) kcal, 126 (12) g of carbohydrate, 23 (2) g of fat, and 31 (3) g of protein]. On *day 2*, breakfast and lunch were provided at similar times as on *day 1* but had higher carbohydrate content [1,006 (92) kcal, 168 (15) g of carbohydrate, 23 (2) g fat, and 31 (3) g of protein]. The evening meal on *day 2* was provided after the exercise bout at ~2,100 and had similar fat and protein content compared with dinner on *day 1* but less carbohydrate [503 (46) kcal, 42 (4) g of carbohydrate, 23 (2) g of fat, and 31 (3) g of protein]. Subjects were kept in close communication by phone and email to ensure compliance with all aspects of the dietary controls. Alcohol and caffeine consumption were prohibited starting the evening before *day 1* of each treatment.

High-fat tolerance test. The high-fat tolerance test (HFTT) meal was given to the subject on the morning of *day 3* following fasting measurements, as described above. The meal consisted of a mixture of "Half and Half" and ice cream (Hill Country Farm; H.E.B., San Antonio, TX). The energy content of the HFTT was determined by the subject's body mass (~16 kcal/kg body mass, 1.02 g fat/kg, 1.36 g CHO/kg, 0.31 g pro/kg). This meal has been shown in our laboratory to significantly increase plasma TG levels in healthy young men. The subject consumed the meal in ≤5 min and remained comfortably seated, where they were allowed to read or watch movies during the 6-h postprandial period. Subjects were also instructed to consume ~250 ml of water in the morning prior to the HFTT, with water consumption ad libitum for the first HFTT, with the volume matched within 50 ml for the subsequent treatments.

Biochemical Analysis

On *day 3*, blood was collected in the fasted state and hourly for 6 h postprandial using a venous catheter from the antecubital vein of the right arm. Blood samples were collected in K_2 EDTA tubes (Vacutainer, Franklin Lakes, NJ) and centrifuged immediately for 10 min at 2,000 *g* at 4°C. Plasma was aliquoted and stored at −80°C for later metabolic analysis. TG, glucose, and β-HB were analyzed using commercially available assays (Point Scientific, Canton, MI). Plasma insulin was analyzed using a commercially available ELISA kit (Alpco Diagnostics, Salem, NH). Both total (TG AUC) and incremental area under the curve for plasma triglyceride (TG_I AUC) were calculated using the trapezoidal method and reported as a percent of the CON value. The incremental AUC is the area under the postprandial curve, excluding the fasting value, whereas total AUC is the area under the postprandial curve extending to zero. Plasma insulin and glucose metabolism were measured in the fasted state using homeostatic model of insulin resistance (HOMA-IR). Postprandial insulin and glucose responses were quantified by the insulin sensitivity index (ISI), using a modification to a traditional oral glucose tolerance test to account for the less frequent plasma sampling in this study (29). Additionally, fasting whole blood was used to measure hematocrit and hemoglobin to assess changes in plasma volume prior to the HFTT for each treatment (9). Intra-assay coefficients of variation for TG, glucose, insulin, and β-HB were 4.1, 3.7, 3.7 and 2.7%, respectively.

Statistical Analysis

TG_I AUC and TG AUC, postprandial energy expenditure, and the relative contributions of postprandial fat and carbohydrate oxidation were analyzed using a one-way ANOVA, with repeated measurements used for comparisons between treatments. Fasting and postprandial plasma glucose, insulin, and β-HB were assessed with a two-way repeated-measures ANOVA for treatment and treatment × time interactions. If the treatment × time interaction was found to be significant, post hoc pairwise comparisons were made. A two-tailed Pearson product moment correlation analysis was used to assess the relationship between TG_I AUC and total postprandial fat oxidation as well as TG AUC and HOMA-IR. A Student *t*-test was used to compare total $\dot{V}O_2$ (liters), $\dot{V}O_2$ (l/min), percent $\dot{V}O_{2peak}$, and total energy expenditure during the exercise trials, and when applicable, Bonferroni's post hoc correction was applied. For all tests, significance was found at $P < 0.05$. All analysis was performed using SPSS software (Chicago, IL). Unless otherwise indicated, all data are reported as means (SD).

RESULTS

Exercise Treatment

TEE during the exercise trials, after correction for resting measurements, was not different between MIE and HIE [660.5 (35.7) kcal and 654.8 (30.6) kcal, respectively, $P = 0.42$]. The average rate of oxygen consumption was significantly lower in MIE [2.19 (0.28) l/min, 48.8 (1.2) $\%\dot{V}O_{2peak}$] compared with HIE [3.34 (0.5) l/min, 74.7 (6.1) $\%\dot{V}O_{2peak}$, $P < 0.05$]. During the final minute of the 5 min at 90% $\dot{V}O_{2peak}$ in the warmup phase of HIE, $\dot{V}O_2$ was 4.13 (0.6) l/min (~91.3% $\dot{V}O_{2peak}$). Exercise time was significantly greater in MIE [66.5 (6.0) min]

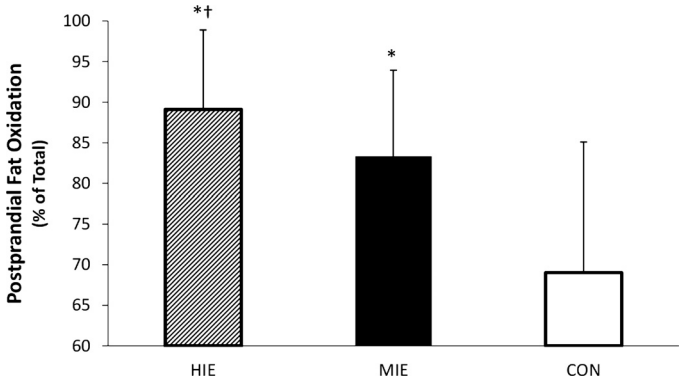

Fig. 1. Fasting and postprandial fat oxidation (%total resting energy expenditure). Treatments were a nonexercise control (CON), moderate-intensity endurance exercise (MIE), and high-intensity endurance exercise (HIE). Values are expressed as means (SD). *Significantly greater than CON; †significantly greater than MIE.

compared with HIE [42.0 (4.0) min, $P < 0.05$]. Total mechanical work was significantly greater in MIE [622.2 (24.4) kJ] compared with HIE [462.7 (44.4) kJ, $P < 0.05$].

Daily Activity

Daily step counts were not statistically different across any treatments for *day 1* [7,839 (607), 7,786 (604), and 7,950 (620); CON, MIE, and HIE, respectively], *day 2* [7,896 (515), 7,856 (354), and 8,031 (231); CON, MIE, and HIE, respectively], or *day 3* [913 (170), 939 (174), and 981 (261); CON, MIE, and HIE, respectively, $P > 0.05$ for all treatments on each day].

REE and Substrate Oxidation

Total REE on *day 3* during the 6-h postprandial period of the HFTT was not statistically different between treatments [453 (69), 453 (74), and 459 (66) kcal; CON, MIE, and HIE, respectively, $P > 0.05$ for all]. The relative contribution of fat oxidation during this period was significantly greater than CON [69.0 (16.1)%] in both MIE [83.3 (10.6)%, $P = 0.039$] and HIE [89.1 (9.8)%, $P = 0.018$], with HIE significantly greater than MIE ($P = 0.012$) (Fig. 1). Conversely, the relative contribution of carbohydrate oxidation during this period was significantly less than CON [31.0 (16.1)%] in both MIE [16.7 (10.8)%, $P = 0.036$] and HIE [10.9 (9.8)%, $P = 0.018$], with HIE less than MIE ($P = 0.015$).

Plasma Analysis

TG. The total postprandial plasma TG responses are presented in Fig. 2. In response to the HFTT on *day 3*, the TG_I AUC was significantly lower in both MIE [75.2 (15.5)% of CON, $P = 0.033$] and HIE [54.9 (13.5)% of CON, $P = 0.001$] compared with CON, with HIE lower than MIE ($P = 0.03$) (Fig. 3*B*). TG AUC was only significantly lower in HIE [69.4 (17.1)% of CON, $P = 0.021$] relative to CON, with no significant difference between MIE [81.1 (16.0)% of CON, $P = 0.102$] and CON. Furthermore, there was no significant difference between MIE and HIE ($P = 0.276$) when analyzed as total area under the curve (Fig. 3*A*).

Total postprandial fat oxidation (kcal/6 h) was significantly inversely correlated with TG_I AUC ($mg \cdot dl^{-1} \cdot 6\ h^{-1}$, $r = -0.67$, $r^2 = 0.45$, $P < 0.01$; Fig. 4). To account for potential type I error by pseudoreplication, a one-tailed Pearson product correlation evaluation was performed separately for CON ($r = -0.672$, $P = 0.074$), MIE ($r = -0.674$, $P = 0.071$), and HIE ($r = -0.738$, $P = 0.047$).

β-HB. There was no significant difference between treatments in plasma β-HB (overall treatment effect). However, there was a significant treatment × time interaction ($P < 0.05$). Fasting β-HB was significantly greater in HIE [0.175 (0.07) mmol/l] compared with CON [0.099 (0.05) mmol/l, $P = 0.024$], with no significant difference between MIE [0.134 (0.10) mmol/l, $P = 0.155$] and CON. Fasting β-HB was not different between HIE and MIE ($P = 0.29$).

Insulin and glucose. Plasma insulin and glucose concentrations were not significantly different between treatments (treatment and treatment × time interactions, $P > 0.05$; Table 1). Glucose and insulin metabolism were assessed by ISI [10.5 (4.4), 11.9 (4.6), and 11.9 (4.6) for CON, MIE, and HIE, respectively, $P > 0.05$ for all] and HOMA-IR [2.03 (0.86), 1.57 (0.48), and 1.38 (0.51) for CON, MIE, and HIE, respectively, $P > 0.05$ for all] and were not significantly different between treatments.

Plasma volume. There were no significant changes in fasting plasma volume on *day 3* across any of the three treatments

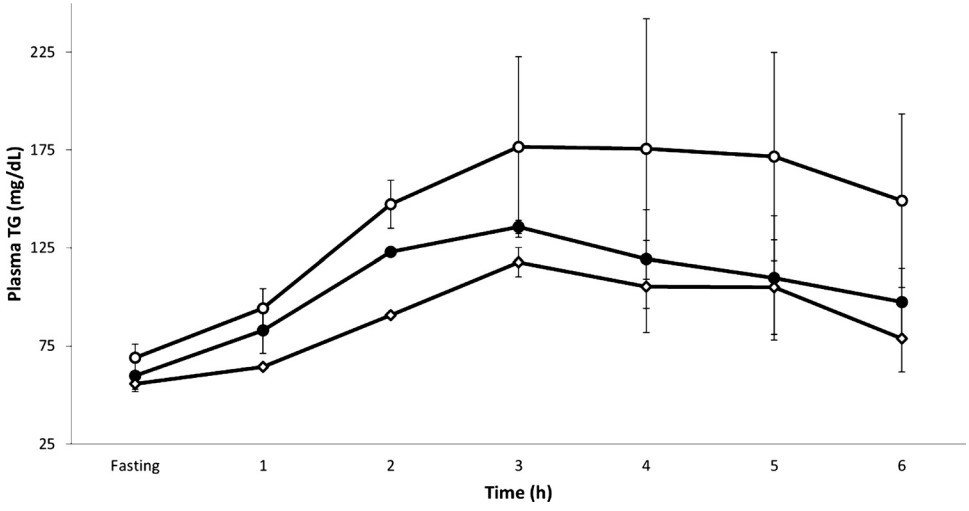

Fig. 2. Plasma triglyceride (TG; mg/dl). Treatments were CON (○), MIE (●), and HIE (◇). Values are reported as means ± SE.

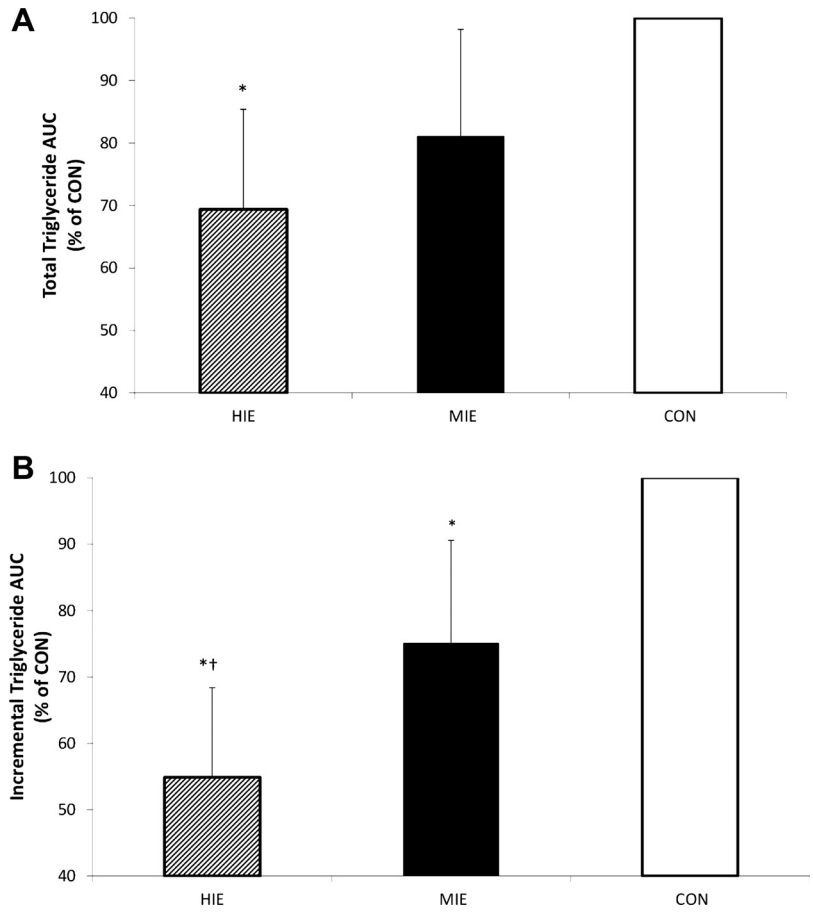

Fig. 3. Plasma TG total (TG AUC; *A*) and incremental area under the curve (TG_I AUC; *B*). Treatments were CON, MIE, and HIE. Values are expressed as %CON. *Significantly less than CON; †Significantly less than MIE. Data are reported as means (SD).

[99.2 (3.4) and 98.7 (2.7)% of CON for MIE and HIE, respectively, $P > 0.05$ for all].

DISCUSSION

The major finding of this study was that intermittent HIE was significantly more effective than continuous MIE in lowering the incremental postprandial plasma TG elevation (Fig. 3). To the best of our knowledge, this is the first study to show that when energy expenditure is similar, high-intensity endurance exercise is more effective than moderate-intensity endurance exercise for acutely lowering incremental PPTG. Furthermore, both MIE and HIE increased significantly the relative contribution of postprandial fat oxidation compared with CON, with HIE significantly greater than MIE (Fig. 1).

Acute exercise has been shown consistently to lower PPTG, with the magnitude dependent largely on the energy expenditure during the most recent exercise bout (14, 52). However, investigation into the independent effect of exercise intensity when comparing low- (~25–30% $\dot{V}O_{2peak}$) to moderate-intensity exercise (~50–65% $\dot{V}O_{2peak}$) (22, 47) as well as moderate-

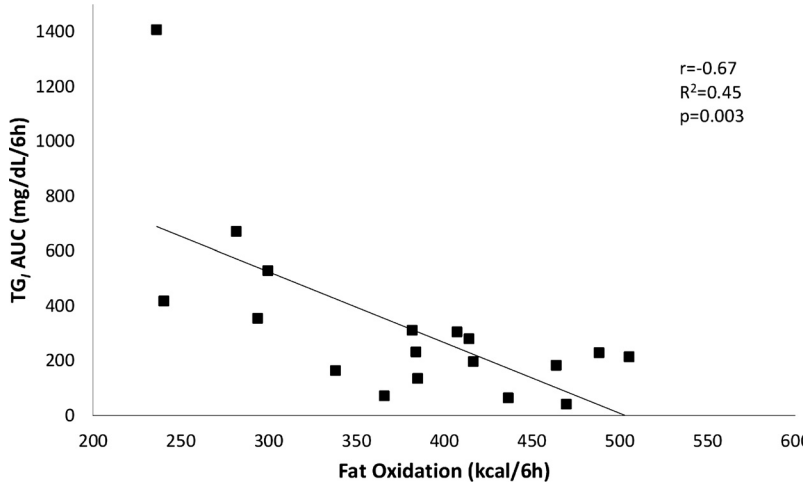

Fig. 4. Relationship between plasma TG_I AUC and total postprandial fat oxidation (kcal/6 h). ■, Data from an individual subject generated during one of the 3 trials.

Table 1. *Plasma insulin and glucose during fasted and postprandial conditions*

Time, h	Fasting	1	2	3	4	5	6
Insulin, μU/ml							
HIE	6.9 (2.1)	37.7 (22.7)	17.8 (10.5)	14.3 (6.4)	7.4 (3.0)	5.1 (3.1)	5.6 (5.5)
MIE	8.0 (3.2)	30.7 (17.6)	14.5 (7.3)	14.4 (7.1)	6.4 (4.3)	6.0 (3.5)	4.7 (3.4)
CON	7.8 (2.9)	44.8 (26.7)	12.1 (3.4)	16.2 (14.7)	9.0 (6.0)	7.2 (4.3)	7.24 (3.1)
Glucose, mg/dl							
HIE	87.3 (7.1)	101.9 (26.7)	102.6 (7.6)	125.0 (12.1)	114.6 (15.6)	117.4 (17.4)	108.2 (16.6)
MIE	85.7 (7.1)	91.4 (13.9)	99.4 (15.3)	123.5 (15.7)	113.9 (15.4)	120.9 (19.8)	112.6 (16.5)
CON	90.4 (9.3)	87.7 (15.8)	98.7 (15.8)	124.1 (15.6)	116.2 (23.4)	126.5 (30.5)	123.6 (20.1)

Values are expressed as means (SD). HIE, high-intensity exercise; MIE, moderate-intensity exercise; CON, nonexercise control.

to high-intensity interval exercise (48) has yielded equivocal findings. Katsanos et al. (22) showed a significant lowering of PPTG AUC of 39% when the test meal was provided 1 h following 60 min of acute exercise targeted to expend ~1,100 kcal at 65% $\dot{V}O_{2max}$ compared with a nonexercise control. In the same study, isoenergetic exercise at 25% $\dot{V}O_{2max}$ resulted in a nonsignificant 9% decrease in PPTG, indicating an intensity effect of exercise performed immediately prior to PPTG assessment. In contrast, Tsetsonis and Hardman (47) showed a significant lowering of PPTG when the test meal was provided the morning after acute exercise, expending ~1,000 kcal at both 32 and 63% $\dot{V}O_{2max}$, with no significant differences between the two intensities. This discrepancy may be due to the timing of the HFTT in relation to the exercise bout related to the time course of postexercise mLPL (rate-limiting enzyme for TG hydrolysis) activation (24). However, Petitt et al. (37) showed no decrease in PPTG compared with a nonexercise control when the test meal was provided 16 h after an exercise bout performed at ~25% $\dot{V}O_{2peak}$ (11.6 $ml \cdot kg^{-1} \cdot min^{-1}$; data not provided directly in the study) at an energy expenditure of ~400 kcal. In the same study, energy-matched resistance exercise lowered PPTG by ~20% compared with both control and low-intensity exercise (37). With the use of a similar protocol, acute resistance-type exercise was more effective than isoenergetic exercise at 30% $\dot{V}O_{2peak}$ (~400 kcal energy expenditure for both) in decreasing plasma VLDL concentration as well as mean plasma residence time of VLDL triglycerides in the circulation in the fasted state the morning after the exercise session (27). This is somewhat contradictory to the meal-timing hypothesis; however, energy expenditure of ~480 kcal at 60% $\dot{V}O_{2peak}$ significantly lowers PPTG 12 h postexercise, whereas 310 kcal at the same intensity does not (52), suggesting a requisite exercise energy expenditure to lower PPTG. In contrast, Ferreira et al. (10) reported that continuous moderate-intensity running (~73% $\dot{V}O_{2max}$) to expend ~500 kcal was equally effective at lowering PPTG as an isoenergetic high-intensity interval protocol (3 min intervals at ~95% $\dot{V}O_{2max}$), when the HFTT was administered 30 min postexercise. This finding may refute the meal-timing and energy expenditure hypothesis; however, there was no difference between the two treatments in postexercise fat oxidation, an outcome that may explain the absence of an exercise intensity effect on PPTG. Taken together, these studies suggest the importance of the timing of the postexercise meal, total energy expenditure during exercise, postexercise fat oxidation, or magnitude of exercise intensity for a PPTG-lowering effect of exercise effect to be present. On the basis of these discrepant findings, we planned the current protocol so that MIE would be sufficient by both intensity (50% $\dot{V}O_{2peak}$) and energy expenditure (~650 kcal) to lower PPTG but not so high an intensity that we might not observe an effect between MIE and HIE.

The primary focus of the present study was to describe the overall PPTG-lowering effect achieved by increasing exercise intensity, with less emphasis on the mechanisms responsible. Despite this, in agreement with previous research (13), postexercise postprandial fat oxidation is significantly inversely correlated with PPTG ($r = -0.67$, $r^2 = 0.45$, $P < 0.01$), suggesting that higher postprandial fat oxidation might have contributed to the exercise-induced lowering of PPTG. In fact, increased liver fat is associated with an overproduction and secretion of hepatic VLDL triglyceride (1), an effect that may contribute to elevated PPTG and is modulated at least partially by increased fat oxidation (21). A similar interaction may occur in peripheral tissues such as skeletal muscle, where intracellular fatty acid availability is related to mLPL activity and TG uptake (36); however, the exact interaction of skeletal muscle fat oxidation and subsequent TG uptake is not entirely clear. In the context of the present study, postexercise muscle glycogen content is inversely related to resting fat oxidation (31, 43), and increasing exercise intensity from 50 to 90% $\dot{V}O_{2max}$ increases carbohydrate oxidation during exercise approximately two- to fourfold, mainly from intramuscular sources (5). Although not directly measured, it can be inferred that greater skeletal muscle glycogen oxidation occurred during the exercise in HIE compared with MIE. If this is indeed the case, greater glycogen depletion in skeletal muscle may have contributed to the observed increase in postprandial fat oxidation and subsequent lowering of PPTG as both a general exercise effect and an additional contribution of exercise intensity. Alternatively, increasing exercise intensity from moderate to high intensity (65–85% $\dot{V}O_{2peak}$) results in a relatively greater depletion of glycogen from fast-twitch fibers (16). This is of importance, insomuch as contraction-induced increases in mLPL seem to be specific to fast-twitch fibers in rodents (17), suggesting that greater activation of mLPL may have occurred during the HIE treatment, an effect that could facilitate greater TG uptake by the exercised muscle fibers.

The general observation of an increase in postexercise fat oxidation is in agreement with previous work following both resistance exercise and endurance exercise (13, 19, 31); however, the independent contribution of exercise intensity to postexercise macronutrient partitioning and fat oxidation is not entirely clear. Kiens and Richter (25) exercised subjects and depleted muscle glycogen significantly, with no difference in postexercise intramuscular triglyceride content. In the same study, during the first 30 h of recovery, muscle glycogen

increased, whereas intramuscular triglyceride decreased with a coordinate decrease in respiratory exchange ratio and an increase in mLPL activity, suggesting a partitioning of carbohydrate toward storage as skeletal muscle glycogen and fat toward oxidation. However, to the best of our knowledge, no study has measured postexercise muscle glycogen content in coordination with fat oxidation when comparing differing exercise intensities. Saris and Schrauwen (42) showed that exercise at 80% $\dot{V}O_{2peak}$ resulted in a trend for decreased postexercise respiratory exchange ratio ($P = 0.06$) compared with an isoenergetic exercise bout at 40% $\dot{V}O_{2peak}$, with a similar finding reported when exercise at 65% $\dot{V}O_{2peak}$ was compared with interval-type exercise at 90% $\dot{V}O_{2peak}$, similar to the present study (30). However, this finding is not consistent across the literature, as acute exercise at 25 or 85% $\dot{V}O_{2peak}$, expending ~315 kcal with a postexercise meal provided to replace the exercise energy expenditure, resulted in similar fat oxidation the next morning (i.e., energy balance with the control trial), with both exercise treatments higher than a nonexercise control (50). The combination of relatively low energy expenditure and the postexercise meal replacement may have masked an intensity effect to increase fat oxidation the next morning. In a separate study, exercise at 45 or 65% $\dot{V}O_{2peak}$ in both men and women resulted in a similar increase in postexercise fat oxidation despite a higher carbohydrate oxidation during exercise in the 65% $\dot{V}O_{2peak}$ treatment (19). It is possible that the range of intensities examined [i.e., 40% difference in the present study and that of Saris and Schrauwen (42) compared with a 20% difference in the study of Henderson et al. (19)] may describe this discrepancy. Alternatively, since muscle glycogen oxidation increases exponentially with increasing exercise intensity (41), the intensities compared may be critical to have a large enough difference in glycogen utilization during exercise (i.e., comparisons at 40–60% compared with 60–80% $\dot{V}O_{2peak}$). Regardless of this uncertainty, it is clear that more research is needed to understand the interaction between acute exercise intensity and postexercise fat oxidation.

Dietary controls leading up to the test day when PPTG was assessed are of importance. In the present study, HIE and MIE were in a relatively negative energy balance compared with CON (−655 and −660 kcal, respectively). Therefore, we cannot differentiate between the effects of exercise per se and the negative energy balance. However, in the present study, lower PPTG assessed by incremental methods in HIE compared with MIE suggests that exercise may have an effect beyond that of strictly energy deficit. Similarly, we are unable to determine whether the intensity or general exercise effect would persist if all of the energy or carbohydrate expended during exercise were replaced prior to the test meal. With this in mind, provision of a postexercise mixed meal to match the energy but not the carbohydrate expended (24 g of carbohydrate in the meal) during exercise retains at least a portion of the PPTG-lowering effect of acute exercise (11). However, replacement of 110% of the carbohydrate oxidized during exercise abolished the exercise effect completely (18). This suggests that the amount of carbohydrate oxidation during exercise and the amount of carbohydrate consumed after exercise are important in determining the PPTG-lowering effect of exercise. It should be noted that in all treatments, an identical meal in both energy and macronutrient content was provided at ~2,100 on *day 2* of each treatment (postexercise for MIE and HIE). This meal was low to moderate in carbohydrate (~42 g, 36% energy from carbohydrate) and may have contributed, along with the relatively negative energy deficit in HIE and MIE, to the observed exercise intensity effect on PPTG by keeping skeletal muscle or hepatic glycogen relatively low. As a result, regardless of the matched postexercise meal in both MIE and HIE, it is unclear whether the intensity effect would persist if a higher-carbohydrate postexercise meal is provided. From a public health perspective, it is unclear whether chronic lowering of PPTG would be better accomplished by regular high-intensity exercise or moderate-intensity exercise combined with a diet low in carbohydrates.

In clinical populations, insulin resistance has been suggested as a key regulator in the development of hypertriglyceridemia by impairing glycogen storage in skeletal muscle and increasing hepatic de novo lipogenesis (35), a condition that is improved after an acute bout of endurance exercise (39). In the later study (39), increased postexercise skeletal muscle glycogen storage was accompanied by a coordinate decrease in de novo lipogenesis and PPTG; those authors proposed that increased storage of carbohydrate as glycogen in skeletal muscle redirects carbohydrate away from the liver and decreases VLDL synthesis and secretion from de novo sources. In contrast, in the present study, despite differences in PPTG, there was no difference in ISI or HOMA-IR between treatments, suggesting that changes in insulin sensitivity did not contribute to the attenuation of PPTG. We did not measure postexercise muscle glycogen directly; however, despite the same energy expenditure and macronutrient intake, on the morning after exercise resting, postprandial carbohydrate oxidation was significantly lower in HIE compared with MIE. This suggests an increase in nonoxidative carbohydrate disposal (i.e., glycogen storage) in HIE compared with MIE (31), due possibly to differential macronutrient trafficking in peripheral tissues such as skeletal muscle (25) despite similar fasting and postprandial plasma glucose and insulin. Additionally, our subjects were healthy young men that were not insulin resistant, so glucose tolerance in this population is less likely to be impaired or related to PPTG. Moreover, this study was not designed to assess directly the effects of exercise intensity on insulin sensitivity, as the meal provided and the timing of plasma collection were not the standardized oral glucose tolerance test, as described elsewhere (29). It is also possible that the nonspecific nature of assessing insulin sensitivity using measurements of plasma insulin and glucose, although correlated to the hyperinsulinemic euglycemic clamp (29), may not provide robust determination of tissue-specific insulin sensitivity per se (44).

It is generally accepted that excess postexercise oxygen consumption (EPOC) occurs for several hours after an acute exercise bout and may be dependent on the duration and intensity of the exercise performed. Exercise at 50% $\dot{V}O_{2peak}$ results in lower EPOC compared with exercise at 75% $\dot{V}O_{2peak}$, when exercise energy expenditure is matched (4.8 vs. 9.0 liters over a 3-h postexercise period) (38). Although not measured in this study, this would represent ~4% of our overall exercise oxygen consumption, making it unlikely but not impossible to account for the ~27% difference we observed between MIE and HIE in TG_I AUC. Furthermore, in the present study, indirect calorimetry was also performed during the "easy"

portion of each interval and was factored into our energy expenditure calculation, potentially minimizing the confounding effects of EPOC. In fact, continuous moderate-intensity exercise at 65% $\dot{V}O_{2peak}$ compared with isoenergetic interval-type exercise with 2 min at 90% $\dot{V}O_{2peak}$ and 3 min at 30% $\dot{V}O_{2peak}$ results in similar EPOC (30). This suggests that although HIE may have had increased EPOC, compared with MIE the increase was likely to be at least partially accounted for in our calculations but otherwise comprised a relatively small amount of the total exercise energy expenditure.

This study has several limitations. It is important to note that there was no significant difference between MIE and HIE when PPTG was assessed by total TG AUC. However, because HIE was significantly lower than CON, with no difference between CON and MIE, and the incremental method has been suggested to better represent the magnitude of the postprandial plasma TG elevation compared with total TG AUC (6), the intensity effect is still at least partially present. From a dietary control standpoint, we selected an activity correction of 1.3 to estimate the energy intake required for each subject in the present study. It is possible that this is an underestimation given their daily step counts; however, this "underfeeding" was unlikely to significantly influence our findings since it was matched in all three treatments.

Another limitation of the present study is that, despite the similar exercise energy expenditure between treatments, mechanical work while cycling was ~35% higher in MIE compared with HIE, suggesting decreased efficiency in HIE. We are unable to say for certain why this discrepancy exists; however, it is important to note that this suggests that work accomplished may have been lower in HIE compared with MIE, a result that would strengthen the overall finding of more effective lowering of PPTG in HIE compared with MIE. It is also possible as well that the use of intermittent as opposed to continuous indirect calorimetry during the exercise trials may have influenced the quantification of exercise energy expenditure. Despite this, there was no evidence of drift in $\dot{V}O_2$ or energy expenditure in either MIE or HIE (data not reported), indicating that the intermittent collection method was likely to be an appropriate means to quantify total exercise energy expenditure. Of final consideration, it is difficult to ascertain the clinical relevance of lower PPTG and higher fat oxidation in HIE compared with MIE. The use of relatively healthy male subjects may limit the application of these findings to women and to less fit clinical populations. It is also possible that individuals, especially clinical populations, may be unable or unwilling to perform this type of high-intensity exercise. However, comparison in moderate (50% $\dot{V}O_{2max}$) and high-intensity (70–80% $\dot{V}O_{2max}$) exercise training in postinfarction patients with heart failure showed an improvement in subjective assessment quality of life following the high-intensity compared with the moderate-intensity training (51). This suggests the possibility for practical applicability as well as greater treatment efficacy of interval-type exercise training in both healthy and clinical populations.

The finding that both HIE and MIE were more effective than CON at lowering PPTG and increasing fat oxidation confirms previous findings that exercise is an effective means to acutely improve postprandial triglyceride metabolism. The further improvement observed in incremental PPTG with HIE compared with MIE is of particular interest. The exact clinical implications are beyond the scope of the present study; however, this illustrates a "proof of concept" that an exercise intensity effect persists beyond comparisons of low- (~25%) and moderate-intensity exercise (~50–65%). In conclusion, both HIE and MIE attenuated incremental PPTG compared with CON; however, HIE was more effective compared with MIE.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

J.R.T., K.M.C., D.R.M., I.-Y.K., and E.F.C. contributed to the conception and design of the research; J.R.T., K.M.C., and D.R.M. performed the experiments; J.R.T. and E.F.C. analyzed the data; J.R.T., K.M.C., D.R.M., and E.F.C. interpreted the results of the experiments; J.R.T. prepared the figures; J.R.T. drafted the manuscript; J.R.T., K.M.C., D.R.M., I.-Y.K., and E.F.C. edited and revised the manuscript; J.R.T., K.M.C., D.R.M., I.-Y.K., and E.F.C. approved the final version of the manuscript.

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