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Postexercise Fat Oxidation: Effect of Exercise Duration, Intensity, and Modality

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Postexercise fat oxidation may be important for exercise prescription aimed at optimizing fat loss. The authors examined the effects of exercise intensity, duration, and modality on postexercise oxygen consumption (VO_2) and substrate selection/respiratory-exchange ratio (RER) in healthy individuals. Three experiments (n = 7 for each) compared (a) short- (SD) vs. long-duration (LD) ergometer cycling exercise (30 min vs. 90 min) matched for intensity, (b) low- (LI) vs. high-intensity (HI) cycling (50% vs. 85% of VO_{2max}) matched for energy expenditure, and (c) continuous (CON) vs. interval (INT) cycling matched for energy expenditure and mean intensity. All experiments were administered by crossover design. Altering exercise duration did not affect postexercise VO_2 or RER kinetics ($p > .05$). However, RER was lower and fat oxidation was higher during the postexercise period in LD vs. SD ($p < .05$). HI vs. LI resulted in a significant increase in total postexercise energy expenditure and fat oxidation ($p < .01$). Altering exercise modality (CON vs. INT) did not affect postexercise VO_2, RER, or fat oxidation ($p > .05$). These results demonstrate that postexercise energy expenditure and fat oxidation can be augmented by increasing exercise intensity, but these benefits cannot be exploited by undertaking interval exercise (1:2-min work:recovery ratio) when total energy expenditure, duration, and mean intensity remain unchanged. In spite of the apparent benefit of these strategies, the amount of fat oxidized after exercise may be inconsequential compared with that oxidized during the exercise bout.

Keywords: exercise metabolism, EPOC, recovery, weight management

The increasing prevalence of obesity-related morbidity and the declining rates of exercise participation have led to growing interest in strategies to optimize the metabolic benefits of an exercise bout. In particular, an exercise strategy that optimizes fat utilization both during and after exercise is sought. At the same time, recommendations by expert committees need to be taken into account. The optimal exercise protocol must be a compromise between what is achievable, safe, and physiologically most advantageous.

Given that energy expenditure may remain elevated after exercise for 12–24 hr (Maehlum, Grandmontagne, Newsholme, & Sejersted, 1986), the postexercise period is potentially significant for exercise prescription designed to elicit weight

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loss. However, when compared with effectors of substrate oxidation during exercise, there has been significantly less research concerning the characteristics of an exercise bout that affect postexercise metabolism, particularly fat oxidation (Kuo, Fattor, Henderson, & Brooks, 2005). As a result, possible implications of this for current exercise guidelines remain equivocal (Kuo et al.).

Although not unanimous (Maresh et al., 1992), most previous research has shown that when exercise energy expenditure is increased by increasing exercise duration, postexercise energy expenditure, as indicated by increased excess postexercise oxygen consumption (EPOC) measured by indirect calorimetry, is elevated (Bahr, Ingnes, Vaage, Sejersted, & Newsholme, 1987; Bielinski, Schutz, & Jequier, 1985; Chad & Wenger, 1988; Gore & Withers, 1990; Hagberg, Mullin, & Nagle, 1980; Sedlock, Fissinger, & Melby, 1989). It also appears likely that this phenomenon only occurs beyond a critical exercise intensity, typically ~50% of VO_{2max} (Gore & Withers). The available evidence shows that substrate selection, as noted by the respiratory-exchange ratio (RER), is not significantly altered by changing exercise duration (Bahr et al.). However, most of the aforementioned studies report EPOC but not substrate selection during this period (Gore & Withers; Hagberg et al.; Maresh et al.).

It is often reported that postexercise energy expenditure increases proportionally with exercise intensity (Bahr & Sejersted, 1991; Gore & Withers, 1990; Hagberg et al., 1980; Smith & Mc Naughton, 1993). Nonetheless, a number of published studies documenting the effects of exercise intensity on exercise substrate oxidation have been confounded by differences in exercise energy expenditure (Bahr & Sejersted; Chad & Quigley, 1991; Gore & Withers; Hagberg et al.; Smith & Mc Naughton), did not report substrate selection/RER values (Gore & Withers; Hagberg et al.; Smith & Mc Naughton), or employed exercise intensities (Maresh et al., 1992; Melanson et al., 2002; Mulla, Simonsen, & Bulow, 2000; Thompson, Townsend, Boughey, Patterson, & Bassett, 1998) outside of those recommended in current guidelines (American College of Sports Medicine [ACSM], 1998). Inspection of studies that have matched for energy expenditure shows conflicting findings that postexercise energy expenditure increases (Phelain, Reinke, Harris, & Melby, 1997; Sedlock et al., 1989) or is unchanged (Kuo et al., 2005; Melanson et al.; Mulla et al.; Thompson et al.) by an increase in exercise intensity. Of the studies to report postexercise substrate selection, results suggest that RER decreases during the postexercise period when exercise intensity is increased (Phelain et al.), but many report no effect of intensity on postexercise RER (Melanson et al.; Mulla et al.; Thompson et al.), especially when exercise bouts are tightly matched for energy expenditure (Kuo et al.).

If raising exercise intensity or duration does increase postexercise fat oxidation, a combination of these factors may optimize postexercise fat oxidation. However, a prolonged bout of high-intensity exercise (≥75% of VO_{2max}) is generally beyond the tolerance level of healthy but non-endurance-trained adults, for whom the current recommendations are indicated (ACSM, 1998). By incorporating high-intensity intervals interspersed with low-intensity recovery periods, individuals can exploit the benefits of high-intensity exercise while expending the same total amount of energy as in a continuous moderate-intensity exercise bout (Astrand, Rodahl, Dahl, & Stromme, 2003). Given the suggestion that exercise intensity is the greatest stimulus for the increase in postexercise energy expenditure (Gore & Withers, 1990), there has been increasing interest in this stochastic-type exercise training. Recent experimental evidence suggests that this modality of exercise may promote higher rates of

adipose lipolysis, which may enhance fat oxidation (Trapp, Chisholm, & Boutcher, 2007). Although reports regarding the effect of interval versus continuous exercise on substrate selection in the postexercise period are conflicting (Laforgia, Withers, Shipp, & Gore, 1997; McGarvey, Jones, & Petersen, 2005), EPOC often increases with interval-type exercise (Brockman, Berg, & Latin, 1993; Laforgia et al., 1997).

The aim of this study, therefore, was to investigate the effects of exercise duration, intensity, and modality on postexercise metabolic rate (VO_2), substrate selection, and thus the whole-body rate of fat oxidation. We hypothesized that (a) when compared with short-duration exercise, long-duration exercise of the same intensity would increase postexercise fat oxidation; (b) when compared with low-intensity exercise, high-intensity exercise matched for energy expenditure would increase postexercise fat oxidation; and (c) when compared with continuous exercise, interval exercise of equal duration and energy expenditure would increase postexercise fat oxidation.

Methods

Participants

This study comprised three experiments, each of which examined the effect of a different component of the exercise bout on postexercise fat oxidation via cross-over design: Experiment A (duration, $n = 7$), Experiment B (intensity, $n = 7$), and Experiment C (modality, $n = 7$). Separate groups of participants were employed for each experiment. Participants were recreationally active and engaged in aerobic exercise 1–4 days per week. All female participants were eumenorrheic and were not in the menstrual phase of the menstrual cycle during the time of the experimental trials. Physical characteristics of participants are indicated in Table 1. Participants were informed about the nature and potential risks of the experimental procedures before their written informed consent was obtained. The study was approved by the Northern Tasmanian Health and Medical Research Ethics Committee.

Table 1 Participant Characteristics, *M* (*SE*)

	Experiment A		Experiment B		Experiment C	
Participants	7 (4M:3F)		7 (6M:1F)		7 (6M:1F)	
Age, years	20.6 (0.6)		30.4 (2.0)		27.3 (2.4)	
VO_{2max}, ml · kg^{-1} · min^{-1}	39.6 (2.8)		57.5 (3.0)		45.7 (3.7)	
Trial	**SD**	**LD**	**LI**	**HI**	**CON**	**INT**
Body mass (kg)	72.6 (5.5)	72.6 (5.5)	75.0 (3.6)	75.2 (3.4)	73.8 (4.7)	74.0 (4.5)
BMI, kg/m^2	23.4 (1.1)	23.4 (1.1)	23.6 (0.7)	23.6 (0.7)	23.4 (0.8)	23.5 (0.7)
Body fat, %	20.3 (2.2)	20.3 (2.2)	18.9 (1.5)	17.9 (1.6)	22.8 (2.3)	22.9 (2.3)

Note. M = male; F = female; VO_{2max} = maximal oxygen consumption; BMI = body-mass index; SD = short duration; LD = long duration; LI = low intensity; HI = high intensity; CON = continuous; INT = interval.

Preliminary Testing

One week before the exercise-intervention phases of the study, maximal oxygen uptake (VO_{2max}) during cycling exercise was determined for each participant via electronically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands). The test required the participant to cycle at four 5-min submaximal steady-state power outputs, followed by an incremental increase in power (30 W/min) until volitional fatigue. Throughout the test participants breathed through a reusable silicone mouth and nasal breathing mask (Series 7930, Hans Rudolph, Kansas City, MO). Expired ventilation volume and expired O_2 and CO_2 fractions were continuously measured by metabolic cart (Model 17670 Vista Mini-CPX, VacuMed, California) and used to calculate VO_2 and VCO_2. All volumes were corrected to standard temperature and pressure, dry. VO_2 and VCO_2 analyzers were calibrated immediately before each test using gases of known composition, and the pneumotach, which contained a turbine flow sensor, was calibrated with a Hans Rudolph 3-L syringe (Model 5530, Kansas City, MO). Heart rate was monitored throughout exercise by telemetry (Polar Electro, Finland). All participants achieved VO_{2max} according to standard ACSM criteria. External power output and VO_2 attained during the final 2 min of each submaximal workload and the maximal ramp were used to formulate regression equations, from which workloads for the exercise bouts were derived.

Pretrial Standardization

To standardize endogenous substrate availability before experimental exercise interventions, participants were instructed to maintain activities of daily living and avoid all forms of recreational exercise for 24 hr and to record their physical activity and diet during the 48 hr preceding their first experimental trial. This exercise and diet regimen was replicated before all subsequent interventions for each individual. For further standardization, all participants consumed a high-carbohydrate (73% carbohydrate, 2% fat, 20% protein) meal providing 1.5 g carbohydrate/kg body weight the evening before each experimental trial. This meal was consumed no later than 10 hr before trial commencement, after which participants refrained from food and beverages (ad libitum water) until completion of the experimental trial the following day. Diet composition was quantified via Foodworks (Xyris Software, Melbourne, Australia).

Experimental Protocol

In each experiment, participants undertook two acute exercise interventions separated by at least 3 days. The order of the experimental trials was administered by crossover design (in each experiment 4 participants completed one trial first while the other 3 participants completed the other trial first). To minimize circadian effects on metabolism the experimental trials were performed at the same time of day. All trials consisted of a preexercise rest period (30 min), an exercise bout (~15–90 min), and a postexercise recovery period (90 min). Respiratory gases were collected periodically throughout the trial via a two-way, high-velocity, low-resistance breathing valve (Hans Ruldoph, Kansas City, MO), which was held in place by headgear and connected to a Douglas bag by lightweight extension tubing. Expired respiratory-gas samples were collected for 4 min during preexercise and

3 min during exercise and postexercise periods, and mean substrate oxidation was calculated. O_2 and CO_2 fractions of expired air were measured using the O_2 and CO_2 sensors as described. Both analyzers were calibrated immediately before each measurement. Pulmonary-ventilation volume was measured by manual evacuation through a 7-L calibration syringe (Model 4900, Hans Rudolph) and corrected to standard temperature and pressure, dry. VO_2 and VCO_2 were determined as outlined previously. Throughout the trial, heart rate was measured over 1-min periods at the same time as expired-gas collection. Participants were permitted to drink water ad libitum during and after the exercise bout.

On presenting to the laboratory in the morning (7 a.m.), participants voided their bladder, and thereafter anthropometric (height and weight) and bioelectrical impedance (percentage body fat; Model 5582A, Homedics) measurements were recorded. After being fitted with a heart-rate monitor and breathing-apparatus headpiece, participants rested in a supine or prone position in a quiet room. They were counseled on the importance of minimizing movement, particularly during expired-air collection. After 15 min, the breathing mouthpiece was inserted, and, for 5 min before sampling, participants wore nose clips and breathed through the mouthpiece. After ~20 min, three consecutive samples of expired gas were collected. The mean of these samples was used to determine basal (preexercise) measurements of metabolic rate and substrate selection.

After basal measurements, participants either began exercising or sat quietly while reading a book or watching television for a period of 60–75 min before commencing the exercise bout (according to their treatment allocation). During all exercise bouts, participants cycled on the same electronically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) that was used for the preliminary testing at a cadence of >60 rpm, which was consistent between trials. Three minutes worth of expired respiratory gas was collected at 15-min intervals during exercise (~6-min interval in Experiment B) to determine whole-body substrate oxidation. The time of exercise initiation was adjusted in all trials so that the postexercise period began at the same time of day. During the postexercise period participants rested in the supine position while expired air was sampled for 3-min periods at 6-min intervals until 30 min after exercise cessation, and then at 10-min intervals between 30 and 90 min postexercise.

Experiment A. Experiment A was designed to examine the effect of exercise duration on postexercise fat oxidation by comparing short- versus long-duration ergometer cycling exercise. Participants cycled for 30 min in the short-duration bout and 90 min in the long-duration bout. The exercise trials were matched for exercise intensity and were performed at a workload designed to elicit 50% of VO_{2max}. This intensity was selected because it is considered the minimum recommended to improve body composition and achieve positive adaptations in aerobic power (ACSM, 1998).

Experiment B. Experiment B was designed to examine the effect of exercise intensity on postexercise fat oxidation by comparing low- versus high-intensity ergometer cycling exercise. In the low-intensity bout participants cycled for 30 min at a workload designed to elicit 50% of VO_{2max}. In the high-intensity bout they cycled at a workload designed to elicit 85% of VO_{2max} for a duration that matched the low-intensity trial for external energy expenditure (~12–15 min).

Experiment C. Experiment C was designed to examine the effect of exercise modality on postexercise fat oxidation by comparing continuous versus interval ergometer cycling exercise. In the continuous bout participants cycled for 90 min at a workload designed to elicit 50% of VO_{2max}. In the interval bout they undertook 90 min of stochastic-effort ergometer cycling that alternated between 60-s submaximal workloads (85% of VO_{2max}) and 120-s active-recovery workloads (30% of VO_{2max}). Continuous and interval trials were matched for exercise duration and external energy expenditure.

Analytical Procedures and Calculations

Total fat-oxidation rates were calculated using the nonprotein respiratory quotient (Peronnet & Massicotte, 1991):

$$\text{Fat oxidation rate} = 1.695 \times VO_2 - 1.701 \times VCO_2$$

VO_2 and VCO_2 in L/min and oxidation rates were expressed in g/min. In addition, fat-oxidation rates were expressed in $mg \cdot kg^{-1} \cdot min^{-1}$.

Oxidative energy expenditure and non-protein-assumed carbohydrate or fat oxidation during exercise and in the postexercise period were measured by calculating the area under the curve for VO_2 and VCO_2 above the basal rate versus time. Energy expenditure was calculated by assuming that 1 L of O_2 consumption is required to fully metabolize 21 kJ of substrate.

Statistics

All measures before and postexercise were compared by two-way repeated-measures analysis of variance (ANOVA). Where significant main effects (group or time) were identified, post hoc analyses were performed via paired Student's *t* tests with a Bonferroni correction for multiple comparisons to confirm significant differences from baseline. Paired Student's *t* tests were used to compare substrate oxidation during exercise between treatments. EPOC and total fat oxidation in the 90-min postexercise period were determined by the area-under-the-curve method. This was chosen to account for instances in which the rate of fat oxidation or energy expenditure was consistently elevated after one exercise trial in the absence of a significant difference in postexercise kinetics. Total EPOC and postexercise fat oxidation were compared between trials by paired Student's *t* tests. Reported dietary macronutrient intake was assessed via Foodworks, with differences between treatments compared via paired Student's *t* tests. Statistical significance was accepted at $p < .05$. Calculations were performed using SPSS for Windows Version 14. All values are expressed as mean ± *SE*.

Results

Dietary Intake and Anthropometry

Participants remained weight stable between trials in all experimental conditions ($p > .05$ for all, Table 1). They maintained a moderate carbohydrate-mixed diet comprising ~50% carbohydrate, 30% fat, and 15% protein for the 48 hr before all experimental trials. There were no significant differences in total energy consumption or macronutrient intake before the trials in any experimental condition ($p > .05$ for all).

Basal Measurements

There was no significant difference in basal VO_2 between trials in any experiment ($p > .05$, Figure 1). Similarly, basal nonprotein RER was statistically similar between trials in all experiments ($p > .05$, Figure 2). Therefore, the basal rate of whole-body fat oxidation was not different between trials in Experiment A, B, or C ($p > .05$, Figure 3). Basal heart rate was not different between trials in any experiment ($p > .05$).

Exercise Measurements

Experiment A. Total oxidative energy expenditure during exercise was significantly higher in the long- versus short-duration trial ($p < .01$), and mean RER during exercise was significantly lower in the long- versus short-duration trial ($p < .05$). Total fat and carbohydrate oxidation during exercise were both significantly higher in the long- versus short-duration trial ($p < .05$).

Experiment B. Total oxidative energy expenditure during exercise was slightly but significantly higher in the low- versus high-intensity trial ($p < .05$), and mean RER during exercise was significantly lower in the low- versus high-intensity trial ($p < .01$). Total fat oxidation was significantly higher and carbohydrate oxidation was significantly lower during exercise in the low- versus high-intensity trial ($p < .01$).

Experiment C. Total oxidative energy expenditure during exercise was not different between the continuous and interval trials ($p > .05$), and mean RER during exercise was significantly lower in the continuous versus interval trial ($p < .01$). Thus, total fat oxidation was significantly higher and carbohydrate oxidation during exercise was significantly lower in the continuous versus interval trial ($p < .05$).

Postexercise Measurements

The relative effects of short- versus long-duration, low- versus high-intensity, and continuous versus interval trials on postexercise VO_2, RER, and fat oxidation are summarized in Table 2 and Figure 4. The time courses of VO_2, RER, and fat-oxidation change after exercise are shown in Figures 1–3, respectively.

VO_2, Experiment A. There was no significant Trial × Time interaction in mean VO_2 kinetics throughout the recovery period for short- versus long-duration exercise ($p > .05$, Figure 1[a]). Although VO_2 after long-duration exercise was slightly higher than after short-duration exercise throughout the entire 90-min recovery period, there was no significant difference in total EPOC between trials (35 ± 5 ml O_2/kg and 41 ± 8 ml O_2/kg for short and long duration, respectively; $p > .05$; Figure 1[a]).

VO_2, Experiment B. A significant Trial × Time interaction was observed ($p < .01$), with postexercise VO_2 in high intensity significantly greater than low intensity for almost the first 40 min of recovery (Figure 1[b]). Total EPOC was significantly higher in the high- (79 ± 9 ml/kg) versus low-intensity trial (24 ± 7 ml/kg, $p < .05$, Figure 1[b]).

VO_2, Experiment C. No significant Trial × Time interaction in EPOC was observed for continuous versus interval exercise ($p > .05$, Figure 1[c]). There was no significant difference in EPOC between trials (38 ± 8 ml/kg and 51 ± 8 ml/kg for continuous and interval, respectively; $p > .05$; Figure 1[c]).

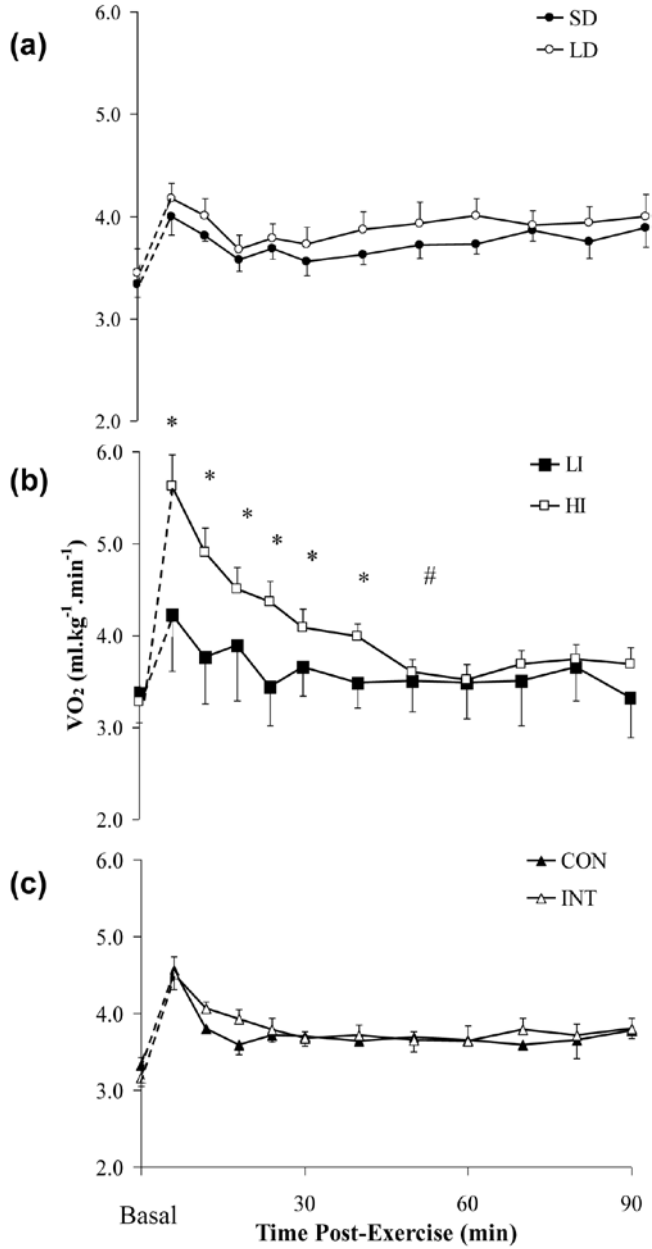

Figure 1 — Effect of (a) short- (SD) versus long-duration (LD) cycling, (b) low- (LI) versus high-intensity (HI) cycling, and (c) continuous (CON) versus interval (INT) cycling on whole-body oxygen consumption (VO_2) during 90 min postexercise. Values are mean ± *SE*; $n = 7$ participants. #Significant Treatment × Time interaction (VO_2 kinetic), $p < .01$. *Total energy expenditure significantly different from alternate trial, $p < .05$.

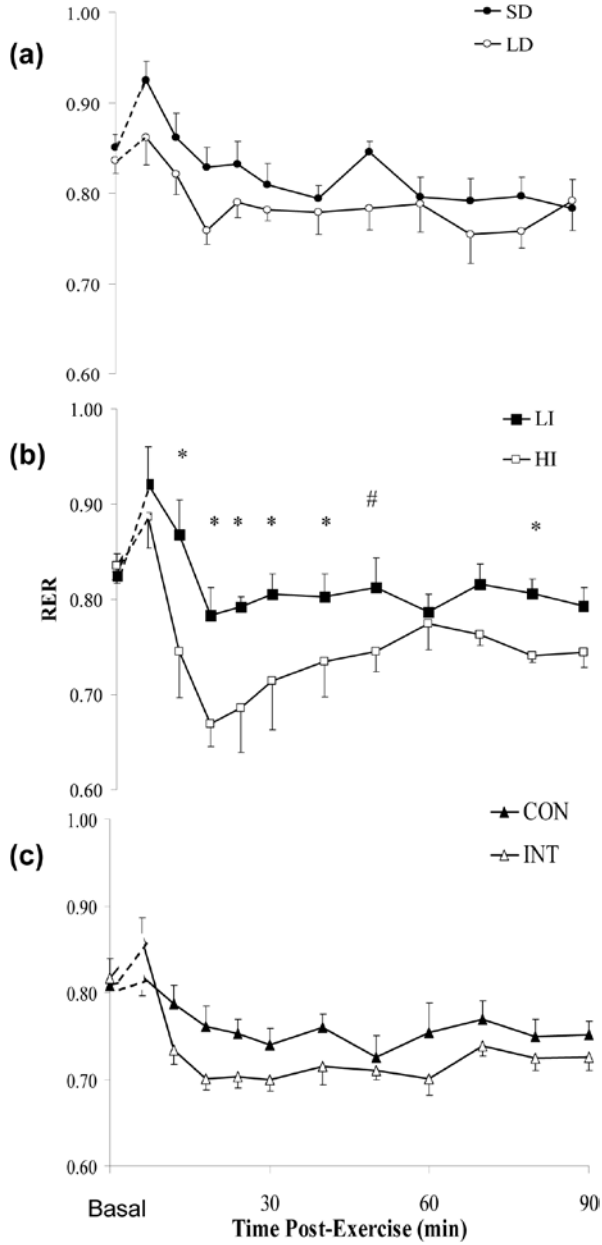

Figure 2 — Effect of (a) short- (SD) versus long-duration (LD) cycling, (b) low- (LI) versus high-intensity (HI) cycling, and (c) continuous (CON) versus interval (INT) cycling on whole-body respiratory-exchange ratio (RER) during 90 min postexercise. Values are mean ± *SE*; $n = 7$ participants. #Significant Treatment × Time interaction (RER kinetic), $p < .05$. *Total energy expenditure significantly different from alternate trial, $p < .05$.

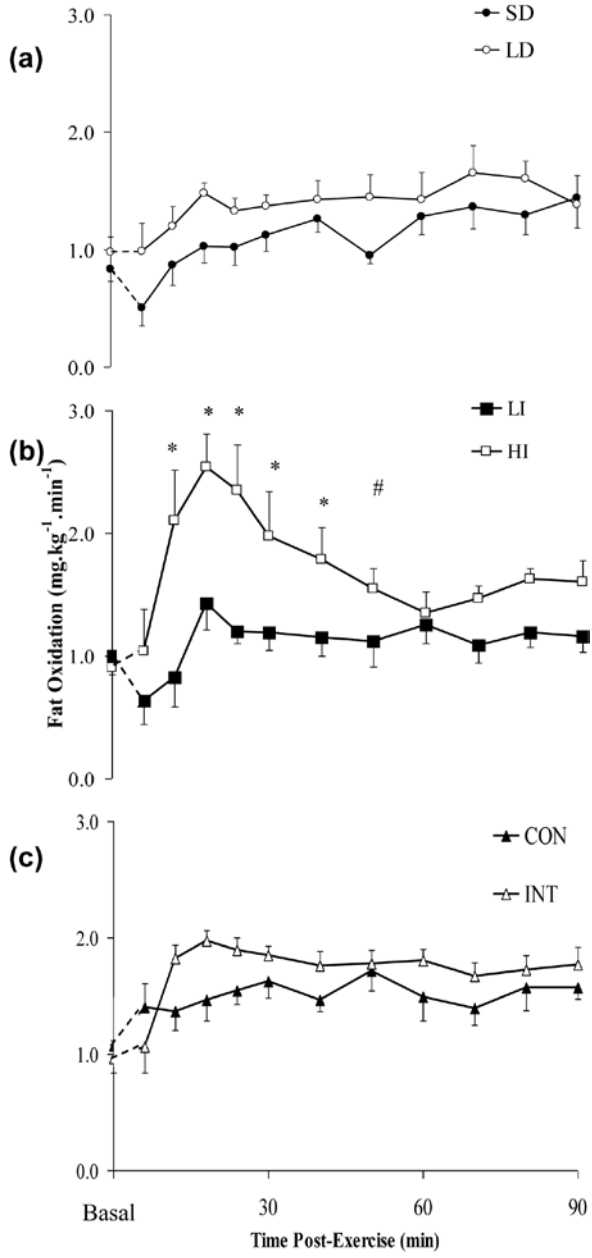

Figure 3 — Effect of (a) short- (SD) versus long-duration (LD) cycling, (b) low- (LI) versus high-intensity (HI) cycling, and (c) continuous (CON) versus interval (INT) cycling on whole-body relative fat oxidation during 90 min postexercise. Values are mean ± *SE*; $n = 7$ participants. #Significant Treatment × Time interaction (fat-oxidation kinetic), $p < .05$. *Total energy expenditure significantly different from alternate trial, $p < .05$.

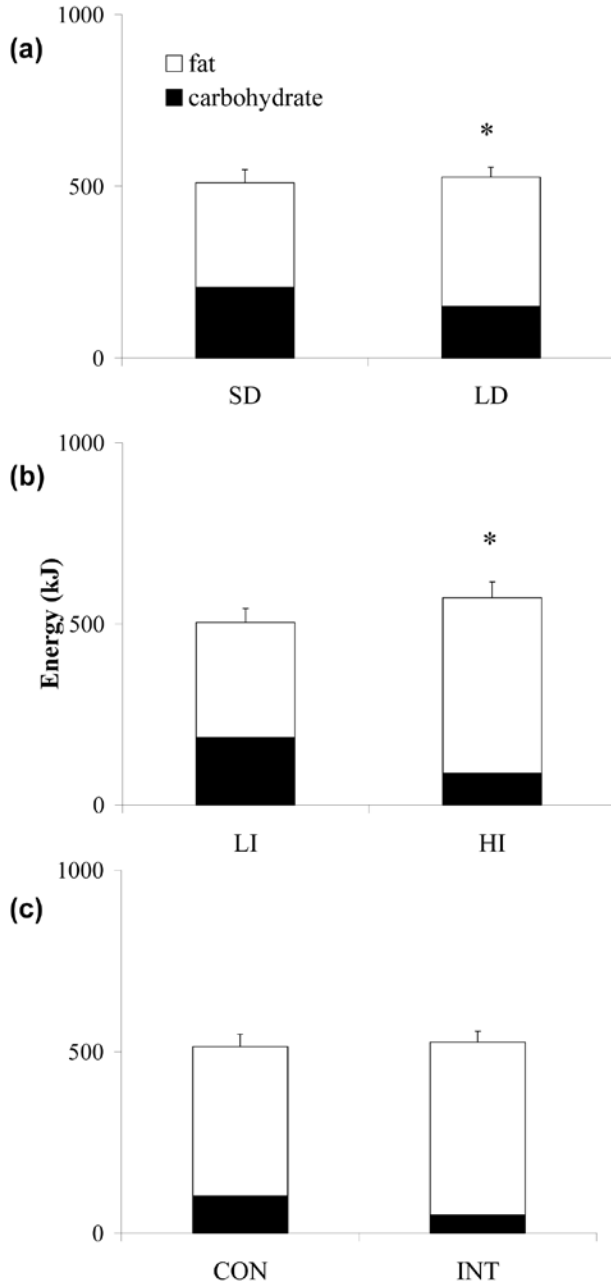

Figure 4 — Effect of (a) short- (SD) versus long-duration (LD) cycling, (b) low- (LI) versus high-intensity (HI) cycling, and (c) continuous (CON) versus interval (INT) cycling on total energy expenditure during 90 min postexercise. Values are mean ± *SE*; $n = 7$ participants. *Total postexercise fat oxidation significantly different from alternate trial, $p < .05$.

Table 2 Summary of the Relative Effect of Exercise Duration, Intensity, and Modality on Postexercise Energy Expenditure, RER, and Fat Oxidation

Experimental vs. control trial	VO_2		RER		Fat Oxidation	
	Kinetics	Value	Kinetics	Value	Kinetics	Value
LD vs. SD	—	—	—	* ↓	—	* ↑
HI vs. LI	*	* ↑	*	* ↓	*	* ↑
INT vs. CON	—	—	—	—	—	—

Note. VO_2 = oxygen consumption; RER = respiratory-exchange ratio; SD = short duration; LD = long duration; LI = low intensity; HI = high intensity; CON = continuous; INT = interval.
*Significant difference in postexercise kinetics (Time × Treatment effect) or absolute values. —No difference in postexercise kinetics (Time × Treatment effect) and/or absolute values. ↑/↓Direction of effect in first vs. second listed trial, respectively.

RER, Experiment A. Mean RER declined during the 90-min postexercise period to below the basal value after both short- and long-duration exercise ($p < .01$, Figure 2[a]). No significant difference in postexercise RER kinetics was observed (Trial × Time interaction, Figure 2[a]).

RER, Experiment B. A significant Time × Trial interaction was observed between the low- and high-intensity trials for postexercise RER ($p < .05$, Figure 2[b]). After low-intensity exercise, RER was not different from basal. However, after high-intensity exercise RER declined below basal values for almost the entire 90-min postexercise period (Figure 2[b]). At the end of the recovery period from high-intensity exercise, RER was still decreased below the basal value by ~13% ($p < .01$).

RER, Experiment C. Mean RER declined during the 90-min postexercise period to below the basal value after both continuous and interval exercise ($p < .01$, Figure 2[c]). No significant difference between the continuous and interval trials was observed in postexercise RER kinetics (Trial × Time interaction, $p > .05$, Figure 2[c]).

Fat-Oxidation Rate, Experiment A. The mean rate of fat oxidation tended to increase with time to above basal rates after both short- and long-duration exercise ($p < .01$). At the end of the 90-min postexercise period, mean fat-oxidation rates for short- and long-duration trials were above the basal value by ~86% and 55%, respectively. Although the time course of postexercise fat oxidation was not different between conditions ($p > .05$), when compared with short-duration exercise, mean fat oxidation after long-duration exercise tended to be higher at all times (Figure 3[a]). Total fat oxidation in the 90-min postexercise period was significantly greater after long- (126 ± 9 mg/kg) versus short-duration exercise (100 ± 9 mg/kg, $p < .01$, Figures 3[a] and 4[a]).

Fat-Oxidation Rate, Experiment B. The mean rate of fat oxidation increased with time to above the basal rate after high- but not low-intensity exercise ($p < .01$). At the end of the 90-min postexercise period, the mean rate of fat oxidation was elevated above the basal value by ~104% after high-intensity exercise. A significant Time × Trial interaction was found ($p < .01$), such that the rate of fat oxidation was significantly higher after high- than low-intensity exercise until 40 min postexercise

($p < .01$, Figure 3[b]). Total fat oxidation in the 90-min postexercise period was significantly greater after high- (173 ± 6 mg/kg) versus low-intensity exercise (106 ± 11 mg/kg, $p < .01$, Figures 3[b] and 4[b]).

Fat-Oxidation Rate, Experiment C. The mean rate of fat oxidation tended to increase with time to above basal rates after both continuous and interval exercise ($p < .01$). At the end of the 90-min postexercise period, mean fat-oxidation rates for the continuous and interval trials were above the basal value by ~54% and 90%, respectively. The time course of postexercise fat oxidation was not different between conditions ($p > .05$, Figure 3[c]). Total fat oxidation in the 90-min postexercise period was not significantly different after continuous (132 ± 9 mg/kg) and interval exercise (146 ± 3 mg/kg, $p > .05$, Figures 3[c] and 4[c]).

Discussion

The purpose of this investigation was to systematically examine the effects of altering exercise duration, intensity, and modality on postexercise metabolic rate (VO_2), substrate selection, and thus the rate of whole-body fat oxidation using exercise doses that lie within acceptable and achievable levels. For the same intensity of exercise, increasing exercise duration increased total energy expenditure and fat oxidation during and after the exercise bout. When energy expenditure during exercise was matched, increasing exercise intensity reduced total fat oxidation during exercise but increased energy expenditure and fat oxidation during the 90 min after exercise. However, this beneficial effect of increasing exercise intensity could not be exploited by undertaking stochastic interval-type exercise matched for total energy expenditure and duration.

Although few previous studies have reported the effects of exercise duration on postexercise substrate selection (RER), the weight of evidence suggests that increasing the duration of the exercise bout results in higher rates of EPOC (Bahr et al., 1987; Bielinski et al., 1985; Chad & Wenger, 1988; Gore & Withers, 1990; Hagberg et al., 1980; Sedlock et al., 1989). We observed a larger postexercise fat oxidation after long- versus short-duration exercise as a consequence of a relative elevation of EPOC in combination with a consistently lower RER. The fact that higher fat oxidation was not concurrent with specific alteration of the postexercise RER kinetic may suggest that the benefit imparted by increasing exercise duration is, on the whole, a consequence of bulk increase in substrate oxidation, perhaps reflective of greater net glycogen depletion (Kuo et al., 2005). In contrast, the observed higher rate of fat oxidation after high- versus low-intensity exercise was a reflection of both augmented EPOC and specific alteration of the postexercise RER kinetic suggestive of a higher relative fat oxidation. Although on the basis of indirect calorimetry measurement we cannot exclude the possibility that alteration of postexercise RER after the high-intensity bout in part reflects CO_2 retention as a consequence of replenishment of the bicarbonate pool (see later discussion), there is good physiological support for an elevation in the rate of fat oxidation after high-intensity exercise. Unlike glucose oxidation—triglyceride lipolysis—free-fatty-acid uptake (FFA) and oxidation are not as tightly regulated in relation to metabolic requirements. FFA availability, which, in part, influences fat oxidation (Issekutz, Bortz, Miller, & Paul, 1967), is affected by hormone-sensitive lipase activity (both adipose and intramyocellular; Watt et al., 2004). The greater catecholamine

response to higher intensity exercise is thus a potential stimulant of postexercise mitochondrial respiration, increased adipose lipolysis and oxidation (Mulla et al., 2000), and/or increased oxidation of intramyocellular triglyceride-derived fatty acids (Kiens & Richter, 1998). We did not measure circulating catecholamines, but the substantially higher heart rate after high- versus low-intensity exercise (data not shown) is likely to reflect this. When combined with the increased rate of glycogen degradation during high-intensity exercise (Bielinski et al.; Kuo et al.; Thompson et al., 1998), this may explain the higher postexercise rates of fat oxidation.

More recently, augmentation of the catecholamine response to high-intensity exercise was proposed to explain higher rates of adipose lipolysis (possibly leading to enhanced fat oxidation) after 8–24 s of supramaximal cycling intervals interspersed with 12–36 s of recovery (Trapp et al., 2007). Nonetheless, when we attempted to exploit this phenomenon by employing stochastic-type exercise involving 60-s submaximal work periods (85% of VO_{2max}) interspersed with 120-s recovery periods at 30% of VO_{2max}, we failed to observe any significant change in EPOC, RER, or postexercise fat oxidation. Given the highly variable nature of interval-exercise prescription it is difficult to resolve this conflict with other studies in literature, although previous reports of enhanced energy expenditure after interval versus continuous exercise may have been confounded by differences in exercise duration (Brockman et al., 1993) or the supramaximal work bouts used (Trapp et al.).

Our results continue a long line of metabolic studies that have inferred rates of fat and carbohydrate oxidation during and after exercise on the basis of indirect calorimetry. In all of those studies, however, the data should be viewed prudently, particularly if fat oxidation was derived from RER measures early (~0–30 min) in the postexercise period. With increasing intensity of exercise, acidosis is known to erroneously underestimate the rate of fat oxidation because of increases in the excretion of nonmetabolic CO_2. The kinetics of nonmetabolic CO_2 and the labile CO_2 store (in body fluids) have been elegantly modeled during exercise by Rowlands (2005), but no such data yet exist for the postexercise period. Although, conversely, there is some degree of CO_2 retention in the postexercise period (to replenish the labile CO_2 store), the precise magnitude and duration of this remains unknown, particularly with respect to changing exercise dose. To help mitigate this issue we collected our first postexercise expiratory-gas samples after 6 min of resting and note that our observation of an increased rate of fat oxidation in high- versus low-intensity exercise persists irrespective of any transient underestimation of RER early after exercise because of the dramatic increase in energy expenditure (high-intensity trial) during this time. Furthermore, although such considerations are acknowledged with regard to Experiment C, our observation of no difference in postexercise energy expenditure or fat oxidation between interval and continuous trials would only be confirmed if postexercise RER were underestimated in the interval trial. By using continuous tracer infusion of ^{13}C bicarbonate, Henderson et al. (2007) recently showed that postexercise bicarbonate/CO_2 retention does increase above preexercise values in high- versus low-intensity exercise, although the magnitude of this effect is relatively small (~10%) and thus we would expect our findings to remain. Nevertheless, such findings do not necessarily imply that postexercise VCO_2 underestimates metabolic CO_2 production (Henderson et al., 2007). Until tracer-based evidence becomes available to definitively confirm or dispute our indirect calorimetry findings and those of our predecessors, we believe that our data contribute new insights using exercise doses that are relevant to current recommendations.

In contrast to most previous research, the emphasis of our current study was to elucidate the most physiologically advantageous methods to optimize fat oxidation during and after the exercise bout. We also emphasized the important practical caveat that the exercise dose must comply with current recommendations for developing and maintaining cardiorespiratory fitness while being achievable and safe (ACSM, 1998). In this respect, the results indicate that when exercise bouts are matched for total energy expenditure, higher exercise intensities result in higher postexercise fat oxidation. In our protocol, however, this benefit was more than offset by the inhibition of fat oxidation during exercise at 85% of VO_{2max} (which may in part be overestimated by nonmetabolic CO_2 excretion as a result of bicarbonate buffering as a consequence of systemic acidosis). For instance, assuming an energy yield of 37 kJ per gram of fat oxidized in the current study, mean fat oxidation during the 30 min of exercise at 50% of VO_{2max} was ~10.5 g. Despite a substantially higher postexercise rate of fat oxidation after high- versus low-intensity exercise, nearly 2.5 hr of this enhanced postexercise fat oxidation would be required to merely offset the apparent loss of fat oxidation incurred during high- versus low-intensity exercise. With the restricted (90-min) postexercise period used in the current study (longer periods were considered unfeasible given the strict requirement to minimize motion during the phase; Kuo et al., 2005), we cannot rule out the possibility that the metabolic benefit of high-intensity exercise may outweigh that of low-intensity exercise >4–5 hr after exercise. Nonetheless, like EPOC/energy expenditure (Gore & Withers, 1990; Laforgia et al., 1997), the low rates of fat oxidation in the postexercise versus exercise period indicate that any metabolic benefit would be unlikely or, at best, trivial. The compelling outcome of this investigation therefore is that, like energy expenditure per se, efforts to maximize fat oxidation should focus on the exercise bout rather than the postexercise period (Laforgia, Withers, & Gore, 2006). Furthermore, in the context of prescribing exercise using intensities compliant with current guidelines, any alteration in exercise modality also has little impact. Arguably the best strategy to maximize fat-loss benefits from exercise is to expend as much energy as possible, at an intensity associated with high rates of fat oxidation (45–65% VO_{2max}; Achten, Gleeson, & Jeukendrup, 2002).

In conclusion, this study examined the effect of altering exercise duration, intensity, and modality on postexercise energy expenditure and fat metabolism. Of these variables, only intensity was found to significantly affect EPOC and postexercise fat metabolism. However, the additional energy expended after high-intensity exercise is trivial when energy expenditure that occurs during the exercise bout is considered.

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