Post-exercise fat oxidation: effect of exercise duration, intensity and modality

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SUMMARY

Post exercise fat oxidation may be of importance for exercise prescription aimed at optimizing fat loss. We examined the effects of exercise intensity, duration and modality on post-exercise oxygen consumption (VO\textsubscript{2}) and substrate selection/respiratory exchange ratio (RER) in healthy individuals. Three experiments (n = 7 for each) comparing: A) short (SD) vs. long duration (LD) ergometer cycling exercise (30 min vs. 90 min, respectively) matched for intensity; B) low (LI) vs. high intensity (HI) cycling (50 % vs. 85 % of VO\textsubscript{2}max, respectively) matched for energy expenditure; and C) continuous (CON) vs. interval (INT) cycling matched for energy expenditure and mean intensity. All experiments were administered by cross-over design. Altering exercise duration did not affect post-exercise VO\textsubscript{2} or RER kinetics (P >0.05). However, RER was lower and fat oxidation was higher during the post-exercise period in LD vs. SD (P <0.05). HI vs. LI resulted in a significant increase in total post-exercise energy expenditure and fat oxidation (P <0.01). Altering exercise modality (CON vs. INT) did not affect post-exercise VO\textsubscript{2}, RER or fat oxidation (P >0.05). These results demonstrate that post-exercise energy expenditure and fat oxidation can be augmented by increasing exercise intensity, however, these benefits cannot be exploited by undertaking interval exercise (1 min: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
INTRODUCTION

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 the exercise bout. In particular, an exercise strategy which 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 optimum 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 h (Maehlum, Grandmontagne, Newsholme, & Sejersted, 1986), the post-exercise period is of potential significance for exercise prescription designed to elicit weight loss. However, when compared with effectors of substrate oxidation during exercise, there exists significantly less research concerning the characteristics of the exercise bout which affect post-exercise 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., 2005).

Whilst not unanimous (Maresh et al., 1992), most previous research has shown that when exercise energy expenditure is increased by increasing exercise duration, post-exercise energy expenditure, as indicated by increased excessive post-exercise 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 may only occur beyond a critical exercise intensity, typically ~ 50% of VO$_2$max (Gore & Withers, 1990). 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., 1987). However, the majority of the aforementioned studies report EPOC but not substrate selection during this period (Gore & Withers, 1990; Hagberg et al., 1980; Maresh et al., 1992).
It is often reported that post-exercise energy expenditure increases proportionally with exercise intensity (Bahr & Sejersted, 1991; Gore & Withers, 1990; Hagberg et al., 1980; Smith & McNaughton, 1993). Yet, 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, 1991; Chad & Quigley, 1991; Gore & Withers, 1990; Hagberg et al., 1980; Smith & McNaughton, 1993), did not report substrate selection/respiratory exchange ratio values (Gore & Withers, 1990; Hagberg et al., 1980; Smith & McNaughton, 1993), 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, 1998). Inspection of those studies which have matched for energy expenditure show conflicting findings that post-exercise energy expenditure increases (Phelain, Reinke, Harris, & Melby, 1997; Sedlock et al., 1989) or is unchanged (Kuo et al., 2005; Melanson et al., 2002; Mulla et al., 2000; Thompson et al., 1998) by an increase in exercise intensity. Of those studies to report post-exercise substrate selection, results suggest that RER decreases during the post-exercise period when exercise intensity is increased (Phelain et al., 1997) but many report no effect of intensity on post-exercise RER (Melanson et al., 2002; Mulla et al., 2000; Thompson et al., 1998), especially when exercise bouts are tightly matched for energy expenditure (Kuo et al., 2005).

If raising exercise intensity or duration does increase post-exercise fat oxidation, a combination of these factors may optimize post-exercise fat oxidation. However, a prolonged bout of high intensity exercise (≥75% of VO2max) is generally beyond the tolerance level of healthy but non-endurance trained adults for whom the current recommendations are indicated (American College of Sports Medicine, 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 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 post-exercise 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 post-exercise 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 duration, intensity and exercise modality on post-exercise metabolic rate (VO$_2$), substrate selection and thus the whole-body rate of fat oxidation. It was hypothesized that: 1) when compared with short duration exercise, long duration exercise of the same intensity increases post-exercise fat oxidation; 2) when compared with low intensity exercise, high intensity exercise matched for energy expenditure increases post-exercise fat oxidation; and 3) when compared with continuous exercise, interval exercise of equal duration and energy expenditure increases post-exercise fat oxidation.
METHODS

Subjects
This study comprised three experiments, each of which examined the effects of different components of the exercise bout on post-exercise fat oxidation via cross-over design: Experiment A (duration, n = 7); Experiment B (intensity, n = 7); Experiment C (modality, n = 7). Separate groups of subjects were employed for each experiment. Subjects were recreationally active and participated 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 each experimental trial. Physical characteristics of subjects are indicated in Table I. Subjects 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.

Preliminary testing
One week prior to participation in the exercise intervention phases of the study, maximal oxygen uptake (VO$_{2\text{max}}$) during cycling exercise was determined for each participant via electronically-braked cycle ergometer (Lode Excalibur, Groningen, Netherlands). The test required the subject to cycle at four five-minute submaximal steady-state power outputs, followed by an incremental increase in power (30 watts/min) until volitional fatigue. Throughout the test subjects breathed through a re-usable mouth and nasal silicone breathing mask (Series 7930, Hans Rudolph, Kansas City). Expired ventilation volume ($V_E$) 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 STPD. 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-Litre syringe (Model 5530, Kansas City). Heart rate was monitored throughout exercise by telemetry (Polar Electro, Finland). All participants achieved VO$_{2\text{max}}$ according to standard American College of Sports Medicine (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.

**Pre-trial standardization**

In order to standardize endogenous substrate availability prior to experimental exercise interventions, subjects were instructed to maintain activities of daily living and avoid all forms of recreational exercise for 24-h and to record their physical activity and diet during the 48-h preceding their first experimental trial. This exercise/diet regime was replicated prior to all subsequent interventions for that individual. For further standardization all subjects consumed a high carbohydrate (73 % carbohydrate; 2 % fat, 20 % protein) meal providing 1.5g CHO/kg body weight the evening prior to each experimental trial. This meal was consumed no later than 10-h before trial commencement after which subjects 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, subjects undertook two acute exercise interventions (described below), separated by at least three days. The order of the experimental trials was administered by crossover design (in each experiment four participants completed one trial first while the other three participants completed the alternate trial first). To minimize circadian effects on metabolism the experimental trials were performed at the same time of day. All trials consisted of a pre-exercise rest period (30-min), an exercise bout (~ 15 to 90-min) and a post-exercise recovery period (90-min). Respiratory gases were collected periodically throughout the trial via two-way, high-velocity, low-resistance breathing valve (Hans Rulolph, Kansas City), 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 pre-exercise and 3 min during exercise and post-exercise periods, and mean substrate oxidation calculated. O$_2$ and CO$_2$ fractions of expired air were measured using the O$_2$ and CO$_2$ sensors as described above.
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, Kansas City) and corrected to STPD. VO\textsubscript{2} and VCO\textsubscript{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. Subjects were permitted to drink water *ad libitum* during and following the exercise bout.

On presenting to the laboratory in the morning (0700 h), subjects 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, subjects rested in a supine or decubitus position in a quiet room. Subjects 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, subjects wore nose clips and breathed through the mouthpiece. After ~ 20-min, 3 consecutive samples of expired gas were collected. The mean of these samples was used for determination of basal (pre-exercise) measurements of metabolic rate and substrate selection.

Following basal measurements, subjects either began exercising or sat quietly while reading a book or watching television for a period of 60-75 minutes before commencing the exercise bout (according to their treatment allocation). During all exercise bouts, subjects cycled on the same electronically-braked cycle ergometer (Lode Excalibur, Groningen, Netherlands) that was used for the preliminary testing at a cadence of > 60 rpm, which was consistent between trials. Three minutes 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 post-exercise period began at the same time of day. During the post-exercise period subjects rested in the supine position whilst expired air was sampled for 3-min periods at 6-min intervals until 30 min following exercise cessation, and at 10 minute intervals between 30-90 min post-exercise.
Experiment A. This experiment was designed to examine the effect of exercise duration on post-exercise fat oxidation by comparing short versus long duration ergometer cycling exercise. Subjects 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$_{2\text{max}}$. This intensity was selected because it is considered to be the minimum recommended to improve body composition and achieve positive adaptations in aerobic power (American College of Sports Medicine, 1998).

Experiment B. This experiment was designed to examine the effect of exercise intensity on post-exercise fat oxidation by comparing low versus high intensity ergometer cycling exercise. In the low intensity bout subjects cycled for 30-min at a workload designed to elicit 50% of VO$_{2\text{max}}$. In the high intensity bout subjects cycled at a workload designed to elicit 85% of VO$_{2\text{max}}$ for a duration that matched the low intensity trial for external energy expenditure (~12 to 15-min).

Experiment C. This experiment was designed to examine the effect of exercise modality on post-exercise fat oxidation by comparing continuous versus interval ergometer cycling exercise. In the continuous bout subjects cycled for 90-min at a workload designed to elicit 50% of VO$_{2\text{max}}$. In the interval bout subjects undertook 90-min of stochastic effort ergometer cycling which alternated between 60 second submaximal workloads (85% of VO$_{2\text{max}}$) and 120 second active recovery workloads (30% of VO$_{2\text{max}}$). 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 non-protein respiratory quotient (Peronnet & Massicotte, 1991):

\[
\text{Fat oxidation rate} = 1.695 \times \text{VO}_2 - 1.701 \times \text{VCO}_2
\]
VO₂ and VCO₂ in L/min and oxidation rates were expressed in g/min. In addition, fat oxidation rates were expressed in mg/kg/min.

Oxidative energy expenditure and non-protein assumed carbohydrate/fat oxidation during exercise and in the post-exercise period was measured by calculation of the area under the curve (AUC) for VO₂ and VCO₂ above the basal rate versus time. Energy expenditure was calculated by assuming that 1 L O₂ consumption is required to fully metabolise 21 kJ of substrate.

Statistics
Comparison between all measures prior to and post-exercise were performed 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 t-tests with a Bonferroni correction for multiple comparisons to confirm significant differences from baseline. Paired Student t-tests were used to compare substrate oxidation during exercise between treatments. EPOC and total fat oxidation in the 90 min post-exercise period were determined by the AUC method. This was chosen in order to account for instances in which the rate of fat oxidation or energy expenditure was consistently elevated following one exercise trial in the absence of a significant difference in post-exercise kinetics. Total EPOC and post-exercise fat oxidation were compared between trials by paired Student t-tests. Reported dietary macronutrient intake was assessed via Foodworks® with differences between treatments compared via paired Student t-tests. Statistical significance was accepted at P <0.05. Calculations were performed using SPSS for Windows Version 14. All values are expressed as means ± SE.
RESULTS

Dietary Intake and Anthropometry
Subjects remained weight stable between trials in all experimental conditions (P>0.05 for all, Table I). Subjects maintained a moderate CHO mixed diet comprising ~ 50% CHO; 30 % fat and 15 % protein for the 48 h prior to all experimental trials. There were no significant differences in total energy consumption or macronutrient intake prior to the trials in any experimental condition (P>0.05 for all).

Basal Measurements
There was no significant difference in basal VO₂ between trials in any experiment (P >0.05, Fig. 2). Similarly, basal non-protein RER was statistically similar between trials in all experiments (P >0.05, Fig. 3). Therefore, the basal rate of whole-body fat oxidation was not different between trials in either Experiment A, B or C (P >0.05, Fig. 4). Basal heart rate was not different between trials in any experiment (P >0.05).

Exercise Measurements
Experiment A. Total oxidative energy expenditure during exercise was significantly higher in the long versus short duration trial (P< 0.01) whilst mean RER during exercise was significantly lower in the long versus short duration trial (P<0.05). Total fat and carbohydrate oxidation during exercise were both significantly higher in the long versus short duration trial (P<0.05).

Experiment B. Total oxidative energy expenditure during exercise was slightly but significantly higher in the low versus high intensity trial (P<0.05) whilst mean RER during exercise was significantly lower in the low versus high intensity trial (P<0.01). Total fat oxidation was significantly higher, and carbohydrate oxidation was significantly lower during exercise in the low versus high intensity trial (P<0.01).

Experiment C. Total oxidative energy expenditure during exercise was not different between the continuous and interval trial (P>0.05) whilst mean RER during exercise was significantly lower in the continuous versus interval trial (P<0.01). Thus, total fat
oxidation was significantly higher, and carbohydrate oxidation during exercise was significantly lower in the continuous versus interval trial (P <0.05).

Post-Exercise Measurements
The relative effects of short versus long duration, low versus high intensity and continuous versus interval trials on post-exercise VO$_2$, RER and fat oxidation are summarized in Table II and Figure 4. The time course of VO$_2$, RER and fat oxidation change following exercise is shown in Figures 1-3, respectively.

Oxygen Consumption
Experiment A. There was no significant trial x time interaction in mean VO$_2$ kinetics throughout the recovery period for short versus long duration exercise (P>0.05, Fig. 1A). While VO$_2$ following long duration exercise was slightly higher than following 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>0.05, Fig. 1A).

Experiment B. A significant trial x time interaction was observed (P<0.01) with post-exercise VO$_2$ in high intensity significantly greater than low intensity for almost the first 40 min of recovery (Fig. 1B). Total EPOC was significantly higher in the high (79 ± 9 ml/kg) versus low intensity trial (24 ± 7 ml/kg) (P< 0.05, Fig. 1B).

Experiment C. No significant trial x time interaction in EPOC was observed for continuous versus interval exercise (P>0.05, Fig. 1C). There was no significant difference in EPOC between trials (38 ± 8 ml/kg and 51 ± 8 ml/kg for continuous and interval, respectively; P>0.05, Fig. 1C).

Respiratory Exchange Ratio
Experiment A. Mean RER declined during the 90 min post-exercise period to below the basal value following both short and long duration exercise (P<0.01; Fig. 2A). No significant difference in post-exercise RER kinetics was observed (trial x time interaction) (Fig. 2A).
Experiment B. A significant time x trial interaction was observed between the low intensity and high intensity trial for post exercise RER (P<0.05, Fig. 2B). Following low intensity exercise, RER was not different from basal. However, following high intensity exercise RER declined below basal values for almost the entire 90-minute post-exercise period (Fig. 2B). At the end of the recovery period from high intensity exercise, RER was still decreased below the basal value by ~ 13 % (P<0.01).

Experiment C. Mean RER declined during the 90 min post-exercise period to below the basal value following both continuous and interval exercise (P<0.01, Fig. 2C). No significant difference between the continuous and interval trial was observed in post-exercise RER kinetics (trial x time interaction; P>0.05, Fig. 2C).

Fat Oxidation Rate

Experiment A. The mean rate of fat oxidation tended to increase with time to above basal rates following both short and long duration exercise (P<0.01). At the end of the 90 min post-exercise period, mean fat oxidation rates for short and long duration trials were above the basal value by ~ 86 % and 55 %, respectively. While the time course of post-exercise fat oxidation was not different between conditions (P>0.05), when compared with short duration exercise, mean fat oxidation following long duration exercise tended to be higher at all times (Fig. 3A). Total fat oxidation in the 90 min post-exercise period was significantly greater following long (126 ± 9 mg/kg) versus short duration exercise (100 ± 9 mg/kg) (P<0.01, Figs. 3A and 4A).

Experiment B. The mean rate of fat oxidation increased with time to above the basal rate following high but not low intensity exercise (P<0.01). At the end of the 90 min post-exercise period, the mean rate of fat oxidation rate was elevated above the basal value by ~ 104 % following high intensity exercise. A significant time x trial interaction was found (P<0.01), such that the rate of fat oxidation was significantly higher following high than low intensity exercise until 40 min post-exercise (P<0.01, Fig. 3B). Total fat oxidation in the 90 min post-exercise period was significantly greater following high (173 ± 6 mg/kg) versus low intensity exercise (106 ± 11 mg/kg) (P<0.01, Figs. 3B and 4B).

Experiment C. The mean rate of fat oxidation tended to increase with time to above basal rates following both continuous and interval exercise (P<0.01). At the end of the 90 min
post-exercise 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 post-exercise fat oxidation was not different between conditions (P>0.05, Fig. 3C). Total fat oxidation in the 90 min post-exercise period was not significantly different following continuous (132 ± 9 mg/kg) and interval exercise (146 ± 3 mg/kg) (P<0.01, Figs. 3C and 4C).
DISCUSSION
The purpose of this investigation was to systematically examine the effects of altering exercise duration, intensity and modality on post-exercise metabolic rate (VO$_2$), substrate selection and thus the rate of whole-body fat oxidation using exercise doses which 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 post-exercise substrate selection (RER), the weight of evidence suggests that increasing the duration of the exercise bout results in higher rates of excessive post-exercise oxygen consumption (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 post-exercise fat oxidation following long versus short duration exercise as a consequence of a relative elevation of EPOC in combination with a consistently lower RER. That higher fat oxidation was not concurrent with specific alteration of the post-exercise 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 following high- versus low-intensity exercise was a reflection of both augmented EPOC and specific alteration of the post-exercise 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 post-exercise RER following 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 is 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 (HSL) activity (both adipose and intramyocellular) (Watt et al., 2004). The greater catecholamine response to higher intensity exercise is thus a potential stimulant of post-exercise 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 following 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., 1985; Kuo et al., 2005; Thompson et al., 1998), this may explain the higher post-exercise 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). Yet, when we attempted to exploit this phenomenon by employing stochastic-type exercise involving 60-sec submaximal “work” periods (85 % of VO$_{2\text{max}}$) interspersed with 120-sec “recovery” periods at 30 % of VO$_{2\text{max}}$, we failed to observe any significant change in EPOC, RER or post-exercise fat oxidation. Given the highly variable nature of interval-exercise prescription it is difficult to resolve this conflict with other literature, although previous reports of enhanced energy expenditure following interval versus continuous modality exercise may have been confounded by differences in exercise duration (Brockman et al., 1993) or the supra-maximal work bouts used (Trapp et al., 2007).

Our results continue a long line of metabolic studies which have inferred rates of fat and carbohydrate oxidation during and after exercise on the basis of indirect calorimetry. In all of these studies however, the data should be viewed prudently, particularly if fat oxidation is derived from RER measures early (~ 0-30 min) in the post-exercise period. With increasing intensity of exercise, acidosis is known to erroneously underestimate the
rate of fat oxidation due to increases in the excretion of non-metabolic CO\textsubscript{2}. Modelling of the kinetics of non-metabolic CO\textsubscript{2} and the labile CO\textsubscript{2} store (in body fluids) has been elegantly performed during exercise by Rowlands (2005), but no such data yet exist for the post-exercise period. Whilst, conversely, there is some degree of CO\textsubscript{2} retention in the post-exercise period (to replenish the labile CO\textsubscript{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 post-exercise expiratory gas samples after 6 min of resting, and highlight 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. Further, whilst such considerations are acknowledged with regard to Experiment 3, our observation of no difference in post-exercise energy expenditure or fat oxidation between interval and continuous trials would only be confirmed if post-exercise RER was underestimated in the interval trial. By using continuous tracer infusion of $^{13}$C bicarbonate, Hendersen et al. (2007) have recently shown that post-exercise bicarbonate/CO\textsubscript{2} retention does increase above pre-exercise values in high versus low-intensity exercise, although the magnitude of this effect is relatively small ($\sim$ 10\%) and thus we would expect our findings to remain. Nevertheless such findings do not necessarily imply that post-exercise VCO\textsubscript{2} underestimates metabolic CO\textsubscript{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 which are relevant to current recommendations.

In contrast to the majority of previous research, the emphasis of our present 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 cardio-respiratory fitness, whilst being achievable and safe (American College of Sports Medicine, 1998). In this respect, the results indicate that when exercise bouts are matched for total energy expenditure, higher exercise intensities result in higher
post-exercise fat oxidation. Yet, in our protocol, this benefit was more than offset by the inhibition of fat oxidation during exercise at 85% of VO\textsubscript{2max} (which may in part be overestimated by non-metabolic CO\textsubscript{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 present study, mean fat oxidation during the 30-min of exercise at 50% of VO\textsubscript{2max} was ~10.5 g. Despite a substantially higher post-exercise rate of fat oxidation after high- versus low-intensity exercise, nearly 2.5-h of this enhanced post-exercise 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) post-exercise period used in the present study (longer periods were considered to be 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 h after exercise. Yet, like EPOC/energy expenditure (Gore & Withers, 1990; Laforgia et al., 1997), the low rates of fat oxidation in the post- 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 post-exercise period (Laforgia, Withers, & Gore, 2006). Further, in the context of prescribing exercise using intensities compliant with current guidelines, any alteration in exercise modality also imparts 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\textsubscript{2max}) (Achten, Gleeson, & Jeukendrup, 2002).

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


FIGURES/LEGENDS

Figure 1. Effect of A) short- (SD) versus long- (LD) duration cycling; B) low- (LI) versus high- (HI) intensity cycling and; C) continuous (CON) versus interval (INT) cycling on whole-body oxygen consumption (VO₂) during 90-min post-exercise. Values are means ± SE; n = 7 subjects. # Significant treatment × time interaction (VO₂ kinetic), P < 0.01. * Total energy expenditure significantly different from alternate trial, P < 0.05

Figure 2. Effect of A) short- (SD) versus long- (LD) duration cycling; B) low- (LI) versus high- (HI) intensity cycling and; C) continuous (CON) versus interval (INT) cycling on whole-body respiratory exchange ratio (RER) during 90-min post-exercise. Values are means ± SE; n = 7 subjects. # Significant treatment × time interaction (RER kinetic), P < 0.05. * Total energy expenditure significantly different from alternate trial, P < 0.05

Figure 3. Effect of A) short- (SD) versus long- (LD) duration cycling; B) low- (LI) versus high- (HI) intensity cycling and; C) continuous (CON) versus interval (INT) cycling on whole-body relative fat oxidation during 90-min post-exercise. Values are means ± SE; n = 7 subjects. # Significant treatment × time interaction (fat oxidation kinetic), P < 0.05. * Total energy expenditure significantly different from alternate trial, P < 0.05

Figure 4. Effect of A) short- (SD) versus long- (LD) duration cycling; B) low- (LI) versus high- (HI) intensity cycling and; C) continuous (CON) versus interval (INT) cycling on total energy expenditure during 90-min post-exercise. Values are means ± SE; n = 7 subjects. * Total post-exercise fat oxidation significantly different from alternate trial, P < 0.05.
**TABLES**

**Table I. Subject characteristics**

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<td>Subjects</td>
<td>7 (4M:3F)</td>
<td>7 (6M:1F)</td>
<td>7 (6M:1F)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>20.6 (0.6)</td>
<td>30.4 (2.0)</td>
<td>27.3 (2.4)</td>
</tr>
<tr>
<td>VO$_{2\text{max}}$, ml/kg/min</td>
<td>39.6 (2.8)</td>
<td>57.5 (3.0)</td>
<td>45.7 (3.7)</td>
</tr>
<tr>
<td><strong>Trial</strong></td>
<td><strong>SD</strong></td>
<td><strong>LD</strong></td>
<td><strong>LI</strong></td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>72.6 (5.5)</td>
<td>72.6 (5.5)</td>
<td>75.0 (3.6)</td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>23.4 (1.1)</td>
<td>23.4 (1.1)</td>
<td>23.6 (0.7)</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>20.3 (2.2)</td>
<td>20.3 (2.2)</td>
<td>18.9 (1.5)</td>
</tr>
</tbody>
</table>

Table II. Summary of the relative effect of exercise duration, intensity and modality on post-exercise energy expenditure, respiratory exchange ratio and fat oxidation.

<table>
<thead>
<tr>
<th>Experimental vs. Control Trial</th>
<th>VO$_2$ kinetics</th>
<th>value</th>
<th>RER kinetics</th>
<th>value</th>
<th>Fat Oxidation kinetics</th>
<th>value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LD vs. SD</td>
<td>-</td>
<td>-</td>
<td>* ↓</td>
<td>-</td>
<td>* ↑</td>
<td>-</td>
</tr>
<tr>
<td>HI vs. LI</td>
<td>* ↑</td>
<td>*</td>
<td>* ↓</td>
<td>*</td>
<td>* ↑</td>
<td>*</td>
</tr>
<tr>
<td>INT vs. CON</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

VO$_2$: oxygen consumption. RER: respiratory exchange ratio. SD: short duration. LD: long duration. LI: low intensity. HI: high intensity. CON: continuous. INT: interval. ↔: indicates no significant difference between trials. *: indicates significant difference in post-exercise kinetics (time x treatment effect) or absolute values. -: indicates no difference in post-exercise kinetics (time x treatment effect) and/or absolute values. ↑/↓: indicates direction of effect in first vs. second listed trial, respectively.
Energy (kJ)

SD

LD

LI

HI

CON

INT

fat

carbohydrate

*