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 Physiological Responses**

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Modified sprint interval training protocols. Part I: physiological responses.**Hashim Islam, Logan K. Townsend, & Tom J. Hazell**

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ABSTRACT

Adaptations to sprint interval training (SIT) are observed with brief (≤ 15 s) work bouts highlighting peak power generation as an important metabolic stimulus. This study examined the effects of manipulating SIT work bout and recovery period duration on energy expenditure (EE) during and post-exercise, as well as post-exercise fat oxidation rates. Nine active males completed a resting control session (CTRL) and three SIT sessions in randomized order: 1) 30:240 (4 x 30 s bouts, 240 s recovery); 2) 15:120 (8 x 15 s bouts, 120 s recovery); 3) 5:40 (24 x 5 s bouts, 40 s recovery). Protocols were matched for the total duration of work (2 min) and recovery (16 min), as well as the work-to-recovery ratio (1:8 s). EE and fat oxidation rates were derived from gas exchange measured before, during, and for 3 h post-exercise. All protocols increased EE versus CTRL ($P < 0.001$). Exercise EE was greater ($P < 0.001$) with 5:40 (209 kcal) versus both 15:120 (163 kcal) and 30:240 (138 kcal), while 15:120 was also greater ($P < 0.001$) than 30:240. Post-exercise EE was greater ($P = 0.014$) with 15:120 (313 kcal) versus 5:40 (294 kcal), though both were similar ($P > 0.077$) to 30:240 (309 kcal). Post-exercise fat oxidation was similar ($P = 0.650$) after 15:120 ($0.104 \text{ g} \cdot \text{min}^{-1}$) and 30:240 ($0.116 \text{ g} \cdot \text{min}^{-1}$) and both were greater ($P < 0.030$) than 5:40 ($0.072 \text{ g} \cdot \text{min}^{-1}$) and CTRL ($0.049 \text{ g} \cdot \text{min}^{-1}$). Shorter SIT work bouts targeting peak power generation increase exercise EE without compromising post-exercise EE, though longer bouts promote greater post-exercise fat utilization.

Key Words: High-intensity interval training; energy expenditure; excess post-exercise oxygen consumption; fat oxidation; repeated sprint exercise; peak power generation.

INTRODUCTION

High-intensity interval training (HIIT) involves brief repeated bouts of near maximal exercise (80-100% HR_{max}) interspersed with short recovery periods and has been shown to elicit comparable health and performance benefits to moderate-intensity ($\sim 70\%$ VO_{2max}) continuous training, albeit with much less time-commitment and exercise volume (Gibala et al. 2014). Similar benefits are achieved with a more intense form of intermittent exercise known as sprint interval training (SIT) that involves supramaximal ($>100\%$ VO_{2max}) work bouts, traditionally structured as four to six 30 s “all-out” efforts separated by 4 min of recovery (Gibala et al. 2014). The potent physiological effects of SIT are highlighted by numerous studies reporting central (cardiovascular) and peripheral (muscular) adaptations that facilitate increases in both aerobic (Gibala et al. 2006; Burgomaster et al. 2008; MacPherson et al. 2011; Hazell et al. 2014a) and anaerobic (MacDougall et al. 1998; Hazell et al. 2010; Zelt et al. 2014) performance. Additionally, SIT has been shown to improve body composition (i.e. decreased fat mass, increased lean mass) following 2-6 weeks of training, despite only 2-3 min of actual exercise performed per session (Whyte et al. 2010; MacPherson et al. 2011; Hazell et al. 2014a).

Interestingly, SIT-induced improvements in aerobic and anaerobic parameters (i.e. VO_{2max} , time trial performance, Wingate power) as well as cardiometabolic health (i.e. muscle oxidative capacity, insulin sensitivity) are not compromised with even shorter work bouts involving 10 (Hazell et al. 2010), 15 (Zelt et al. 2014), and 20 s (Ma et al. 2013; Metcalfe et al. 2012; Gillen et al. 2014; 2016) of supramaximal exercise. When considering the metabolic demands of SIT (predominantly anaerobic), a traditional 30 s bout is characterized by rapid peak power generation during the initial seconds of exercise (<10 s) followed by a precipitous power decline over the remainder of the effort. Although it is unclear which portion of this effort drives the adaptive mechanisms, the aforementioned improvements with shorter SIT bouts (≤ 20 s) suggest that peak power generation early in the bout may be a more important metabolic stimulus than the attempted maintenance of power output that follows (Hazell et al. 2010). This appears logical as $\sim 45\%$ of the total work during a 30 s sprint is performed within the first 10 s (Bogdanis et al. 1996) and as little as 4 s of repeated sprint running activates signaling pathways

associated with mitochondrial remodeling in muscle (Serpiello et al. 2012). As detailed in our companion article (Townsend et al. accepted), shorter work bouts may also be more psychologically appealing given their ability to improve exercise-related parameters such as affect, self-efficacy and enjoyment.

The traditional 30 s SIT protocol expends less energy (~175 kcal) than a 30 min bout of continuous aerobic exercise at 70% $\text{VO}_{2\text{max}}$ (~440 kcal), which is not surprising given the drastically lower amount (2 min vs. 30 min) of exercise involved (Hazell et al. 2012). However, 24 h EE is remarkably similar after both protocols due to a greater protracted increase in resting metabolism (i.e. increased O_2 utilization) that occurs after SIT (Hazell et al. 2012). This excess post-exercise oxygen consumption (EPOC) is a consequence of the greater metabolic perturbations created during intense exercise (Laforgia et al. 2006) and has been shown to increase post-exercise EE with both SIT (Hazell et al. 2012; Chan and Burns 2013; Townsend et al. 2014; Beaulieu et al. 2015) and HIIT (Skelly et al. 2014). Additionally, these protocols have been shown to acutely increase post-exercise fat oxidation (Whyte et al. 2010; Chan and Burns 2013; Beaulieu et al. 2015) and chronically up-regulate various enzymes and proteins involved in fat oxidation (Burgomaster et al. 2008; Gillen et al. 2013; 2014) and transport (Perry et al. 2008). Thus, fat loss after SIT may be attributable to EPOC driven elevations in resting EE combined with a substrate shift towards increased fat utilization, although there is some controversy regarding these effects (Williams et al. 2013). Given that shorter bouts of SIT drive similar adaptations to the traditional 30 s protocol, it is possible that reducing work bout duration may improve EE (during and post-exercise) and/or influence substrate utilization in the post-exercise period. The optimal combination of SIT work bout and recovery period duration for targeting these parameters has not been established.

The purpose of the present study was to determine the effects of manipulating sprint bout and recovery period duration on EE during and 3 h post-exercise, as well as post-exercise fat oxidation using three SIT protocols (traditional 30 s SIT and two modified protocols). All three protocols were matched for total duration of work (2 min) and recovery (16 min) by maintaining the established work-to-recovery ratio (1:8 s) from the traditional SIT protocol. We hypothesized that the protocols with shorter SIT work

bouts (5 or 15 s) would improve EE due to an enhanced regeneration of peak power during successive bouts (i.e. greater number of higher quality efforts per session) as well as shorter rest periods that prevent the return of VO_2 to resting values (Hazell et al. 2014b). On the other hand, the attempted maintenance of power output over longer work bouts (15 or 30 s) would likely elicit greater fat oxidation in the post-exercise period due to a greater decrease in muscle glycogen (Bogdanis et al. 1996; 1998) and the subsequent priority given to its resynthesis such that fat utilization covers metabolic demands (Kleins and Richter 1998).

METHODS

Participants. Nine recreationally active males (23.3 ± 3.0 y, 178.4 ± 5.4 cm, 78.3 ± 9.0 kg, 24.6 ± 2.2 kg·m⁻²; 48.9 ± 5.3 ml·kg⁻¹·min⁻¹) volunteered to participate in the current study. Participants were non-smokers and healthy as assessed by the PAR-Q health questionnaire. Although all participants were physically active (≤ 3 times/wk), none were currently involved in a systematic training program nor had they been for at least 4 months prior to data collection. Participants were not taking any dietary supplements at the time of the study. The experimental procedures were explained in detail to all participants and all provided written informed consent before any data collection. The Research Ethics Board at Wilfrid Laurier University approved this study in accordance with the ethical standards of the 1964 Declaration of Helsinki.

Study Design. Participants completed four experimental sessions (~4.5 h each) during which oxygen consumption (VO_2), carbon dioxide production (VCO_2), and heart rate (HR) was measured (Figure 1). Experimental sessions consisted of one control session (CTRL; no exercise) and three exercise sessions using one of the three SIT protocols: 1) Traditional SIT with 30 s work bouts; 2) Modified SIT with 15 s work bouts; or 3) Modified SIT with 5 s work bouts. To avoid learning effects, all experimental sessions were separated by ≥ 1 week and administered in a balanced randomized exposure to treatment order. Participants were instructed to refrain from physical activity, alcohol, and caffeine for at least 48 h before

each experimental session. Diet was maintained by having all participants record their breakfast prior to the first experimental session, and replicate this intake for all subsequent sessions.

Pre-experimental Procedures. All participants completed a laboratory familiarization session (>5 days) before data collection to introduce testing procedures and reduce any learning effects during subsequent experimental sessions. Participants also had their maximal oxygen consumption ($\text{VO}_{2\text{max}}$) determined during a graded exercise test to exhaustion performed on a motorized treadmill (4Front, Woodway, WI, USA). VO_2 and VCO_2 were measured continuously using an online breath-by-breath gas collection system (MAX-II, AEI technologies, PA, USA) that was calibrated with gases of known concentrations and a 3-L syringe for flow. Following a 5-min treadmill warm-up, each participant ran at a self-selected pace (5-7 mph) with incremental increases in grade (2%) applied every 2 min until volitional fatigue. Heart rate (HR) was recorded beat-to-beat throughout the test using an integrated HR monitor (FT1, Polar Electro, QC, Canada). $\text{VO}_{2\text{max}}$ was taken as the greatest 30 s average in presence of a plateau in VO_2 values ($<1.35 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ increase) despite increasing workload, or two of the following criteria: 1) a respiratory exchange ratio (RER) value >1.10 ; 2) achievement of a maximal HR (<10 bpm of age-predicted maximum [$220-\text{age}$]) and/or; 3) voluntary exhaustion. After a 5-min cool down followed by sufficient rest (>20 min), participants were allowed to practice “all-out” running efforts on a specialized self-propelled treadmill (HiTrainer, QC, Canada) on which all the exercise sessions would be performed.

Experimental Session. Participants arrived at the laboratory at 0800 h after having consumed a standardized light breakfast 30 min prior to arrival (0730 h) and limited their activity while commuting to the laboratory (i.e. drove or used public transit). They remained in the laboratory for the next ~ 4.5 h (Figure 1). Upon arrival participants rested quietly (sitting in a chair) for 30 min prior to any data collection to ensure that a rested state was achieved. Participants were then fitted with a HR monitor (Polar FT1) and silicon facemask (Vmask™, Hans Rudolph Inc., KS, USA) for the continuous measurement of gas exchange (VO_2 & VCO_2) from: 0830-0845 h (baseline), 0845-0915 h (exercise

period), and 0915-0945 h (30 min post-exercise). Hereafter, gas exchange was measured (Figure 1) during the last 15 min of each hour post-exercise from: 1000-1015 h (1st h post-exercise), 1100-1115 h (2nd h post-exercise), and 1200-1215 h (3rd h post-exercise). Participants rested quietly while seated and read between gas collection periods. All gas exchange measurements were made in a temperature-controlled room (21 °C) using the gas collection system described earlier (AEI MAX-II). Identical experimental procedures were followed during the CTRL session with the exception of the exercise period (0845-0915 h), during which participants rested quietly.

Exercise Protocols. All exercise protocols began with a 7 min warm-up (at 3 mph) followed by an 18 min SIT session and 5 min cool-down (30 min total). Warm-up and cool-down were performed on a motorized treadmill (Woodway 4Front) for speed consistency while SIT was performed on a specialized self-propelled treadmill (HiTrainer). Exercise sessions involved “all-out” running sprints using one the following three SIT protocols: 1) 30:240 (traditional SIT; 4 x 30 s bouts followed by 240 s (4 min) rest); 2) 15:120 (8 x 15 s bouts followed by 120 s (2 min) rest) or; 3) 5:40 (24 x 5 s bouts followed by 40 s rest). The treadmill interface provided audio prompting to begin and stop running bouts and verbal encouragement was provided for the entirety of all sprints. Customized treadmill software recorded the speed ($\text{m}\cdot\text{sec}^{-1}$) attained during each sprint in 0.5 s intervals.

VO₂. Oxygen consumption ($\text{L}\cdot\text{min}^{-1}$) was recorded as 30 s averages over the entire duration of each gas collection period. VO₂ during exercise (excluding warm-up & recovery) was taken as the average VO₂ over the duration of each SIT protocol (18 min). Post-exercise VO₂ was determined by plotting the average VO₂ at each time-point (30 min post-exercise, and last 15 min of the 1st, 2nd and 3rd h post-exercise), and calculating the area under the curve using the trapezoid method. The rate of VO₂ was multiplied by the duration of each distinct gas collection period to obtain total VO₂ (L). The immediate 30 min post-exercise measurement was included as part of the first post-exercise hour VO₂ calculation. EPOC was calculated by subtracting total VO₂ during the CTRL session from corresponding post-

exercise gas collection periods in each exercise session. Total EE (kcal) was calculated from total VO_2 (L) during and post-exercise, assuming 5 kcal per L O_2 consumed given the limitations of using respiratory exchange ratio (RER) during exhaustive, non-steady-state exercise (Laforgia et al. 1997).

Fat Oxidation. The rate of fat oxidation was calculated during each hour post-exercise using the following formula (Péronnet and Massicotte 1991): $\text{Fat (g}\cdot\text{min}^{-1}) = 1.695 \times \text{VO}_2 (\text{L}\cdot\text{min}^{-1}) \times 1.701 \times \text{VCO}_2 (\text{L}\cdot\text{min}^{-1})$.

Statistical Analysis. All data were analyzed using Sigma Stat for Windows (Version 3.5). Two-way repeated measures analysis of variance (ANOVA) was used to determine differences in VO_2 , HR, RER, and fat oxidation among the 4 treatments at all time points. A one-way repeated measures ANOVA was used to determine differences in total VO_2 , HR, and RER (exercise, post-exercise, and entire session) as well as fat oxidation and EPOC. Tukey's HSD tests were used for post-hoc analysis where necessary. Significance was set at $P < 0.05$. All data are presented as means $\pm$ standard deviation (SD).

Results

VO_2 & EE. There was a significant ($P < 0.001$) interaction effect (session $\times$ time) for average VO_2 . As expected, VO_2 in the exercise period (Figure 2A) was elevated ($P < 0.001$) during all three SIT sessions compared to CTRL ($0.273 \pm 0.05 \text{ L}\cdot\text{min}^{-1}$). Specifically, exercise VO_2 was greater ($P < 0.001$) during 5:40 ($2.326 \pm 0.258 \text{ L}\cdot\text{min}^{-1}$) compared to both 15:120 ($1.793 \pm 0.238 \text{ L}\cdot\text{min}^{-1}$) and 30:240 ($1.528 \pm 0.187 \text{ L}\cdot\text{min}^{-1}$), while 15:120 was also greater ($P < 0.001$) than 30:240. EE based on total VO_2 (L; Figure 2B) was predictably greater ($P < 0.001$) during the exercise period in all three SIT sessions compared to CTRL ($24.6 \pm 4.5 \text{ kcal}$). EE during 5:40 ($209.4 \pm 4.5 \text{ kcal}$) was greater ($P < 0.001$) compared to both 15:120 ($162.6 \pm 19.2 \text{ kcal}$) and 30:240 ($137.5 \pm 16.9 \text{ kcal}$), while 15:120 was also greater ($P < 0.001$) than 30:240. Average VO_2 during the 1st h post-exercise (Figure 3A) was similar ($P > 0.532$) in all three SIT sessions (5:40: $0.349 \pm 0.031 \text{ L}\cdot\text{min}^{-1}$; 15:120: 0.382 ± 0.044 ; 30:240: 0.374 ± 0.038) and remained elevated

($P < 0.020$) versus CTRL ($0.273 \pm 0.040 \text{ L} \cdot \text{min}^{-1}$). VO_2 during the 2nd and 3rd h post-exercise was not different ($P > 0.153$) between experimental sessions (Figure 3A). Total VO_2 (L) over the entire 3 h post-exercise period (Figure 3B) was greater ($P < 0.001$) in all three SIT sessions compared to CTRL (48.8 ± 5.8 L). Total post-exercise VO_2 was greater ($P = 0.014$) after 15:120 (62.6 ± 6.7 L) compared to 5:40 (58.8 ± 4.9 L), though not different ($P = 0.863$) between 15:120 and 30:240 (61.7 ± 5.1 L) or between 30:240 and 5:40 ($P = 0.078$). The 3-h EPOC (Figure 3B) for 15:120 (13.8 ± 5.2 L) and 30:240 (12.9 ± 2.6 L) was similar ($P = 0.667$) and both were greater ($P < 0.040$) compared to 5:40 (10.0 ± 2.9 L). EE over the entire experimental session (Figure 4) was greater ($P < 0.001$) during all three SIT sessions versus CTRL (348.3 ± 45.7 kcal). Total session EE was similar ($P = 0.195$) between 5:40 (647.8 ± 60.0 kcal) and 15:120 (626.4 ± 65.5 kcal) and both were greater ($P < 0.008$) compared to 30:240 (588.5 ± 56.0 kcal).

HR. There was a significant ($P < 0.001$) interaction effect (session x time) for average HR (Table 1). All three SIT sessions results in an elevated ($P < 0.001$) HR during the exercise period compared to CTRL. HR during exercise was similar ($P = 0.374$) between 5:40 and 15:120, though greater ($P = 0.036$) with 5:40 compared to 30:240. HR during the 1st h post-exercise was similar ($P > 0.902$) between the three SIT sessions and all were greater ($P < 0.001$) compared to CTRL. HR in the 2nd h post-exercise was greater ($P = 0.030$) only after 15:120 compared to CTRL, and HR during the 3rd h post-exercise was not different ($P > 0.156$) between the experimental sessions. Total session HR was elevated for all three SIT sessions vs. CTRL ($P < 0.001$) with no differences between sessions.

RER. There was a significant ($P < 0.001$) interaction effect (session x time) for average RER (Table 1). All three SIT sessions resulted in a greater ($P < 0.001$) RER during the exercise period compared to CTRL. Exercise RER was greater ($P < 0.001$) during 30:240 compared to both 15:120 and 5:40, while 15:120 was also greater ($P < 0.001$) compared to 5:40. RER during the 1st h post-exercise was similar ($P = 0.261$) between 15:120 and 30:240, and both were lower compared to 5:40 ($P < 0.002$) and CTRL ($P < 0.001$). RER during the 2nd h post-exercise was similar ($P > 0.053$) between the three SIT protocols, though 15:120

and 30:240 both remained lower ($P<0.01$) compared to CTRL. There were no differences ($P>0.263$) in RER between the experimental sessions during the 3rd h post-exercise. Total session RER was lower for both 15:120 ($P=0.023$) and 30:240 ($P=0.049$) vs CTRL (Table 1).

Fat Oxidation. There was a significant ($P<0.001$) interaction effect (session x time) for fat oxidation rates in the post-exercise period (Figure 5). Fat oxidation during the 1st h post-exercise was similar ($P=0.659$) between 15:120 and 30:240, and both were greater ($P<0.001$) compared to CTRL, though only 30:240 was greater ($P=0.011$) compared to 5:40. Fat oxidation during the 2nd h post-exercise was similar ($P=0.625$) between 15:120 and 30:240, and both were greater compared to 5:40 ($P<0.004$) and CTRL ($P<0.001$). Fat oxidation during the 3rd h post-exercise was not different between the three SIT protocols ($P>0.120$), though 15:120 and 30:240 were greater ($P<0.013$) compared to CTRL. Fat oxidation over the entire 3-h post-exercise period was similar ($P=0.650$) with 15:120 and 30:240 and both were greater compared to 5:40 ($P<0.03$) and CTRL ($P<0.001$), which were not different ($P=0.125$).

Training Data. Average peak speed attained was greater ($P<0.043$) during 5:40 (7.2 ± 0.5 m·sec⁻¹; Figure 6A) compared to both 15:120 (6.6 ± 0.7 m·sec⁻¹; Figure 5B) and 30:240 (6.4 ± 0.9 m·sec⁻¹; Figure 6C), while 15:120 and 30:240 were not different ($P=0.638$). Peak speed decreased ($P<0.005$) by 14.3% (1.0 m·sec⁻¹) and 27.5% (2.0 m·sec⁻¹) from the 1st to the last sprint bout during 15:120 and 30:240, respectively. The decrease in peak speed during 5:40 (4.2%, 0.3 m·sec⁻¹) was not significant ($P=0.398$).

Discussion

This study investigated the effects of three SIT protocols with different work bout (5-30 s) and recovery period (40-240 s) durations on VO_2/EE during and 3 h post-exercise, as well as post-exercise fat oxidation rates. All three protocols were identical in terms of total exercise time (2 min), recovery duration (16 min) and the work:recovery ratio (1:8 s). Compared to traditional SIT (30:240), both modified protocols (5:40 and 15:120) elicited greater EE during exercise and similar EE over the 3 h post-

exercise period (though 15:120 was slightly greater than 5:40). Consequently, EE over the entire experimental session was higher with the modified SIT protocols compared to traditional 30 s SIT. Additionally, 15:120 resulted in similar fat utilization during the post-exercise recovery period compared to 30:240, both of which were increased compared to 5:40 and CTRL. Collectively, these results indicate that modified SIT protocols with shorter work bouts improve exercise EE without compromising post-exercise EE, though longer SIT bouts result in greater fat utilization in the post-exercise period.

EE during 5:40 (209 kcal) and 15:120 (163 kcal) was increased by 52% and 18% respectively, compared to the traditional 30:240 SIT protocol (138 kcal), despite no differences in total exercise (2 min) or recovery (16 min) time. Although EE during traditional 30 s SIT is typically higher (140-178 kcal) than our 30:240 protocol, the values attained during 15:120 are comparable to previous studies involving traditional SIT (Hazell et al. 2012; Deighton et al. 2013; Hazell et al. 2014b; Townsend et al. 2014; Beaulieu et al. 2015). Due to its brief nature, the majority of EE with traditional 30 s SIT is associated with the post-exercise recovery period (Hazell et al. 2012) and our present data demonstrate a modified version of SIT can expend a considerable amount of calories during the actual exercise session as well. In fact, EE during our 5:40 protocol is 17-49% greater than previously reported with traditional SIT (Hazell et al. 2012; Deighton et al. 2013; Hazell et al. 2014b; Townsend et al. 2014; Beaulieu et al. 2015) and comparable to ~20 min of continuous exercise (210 kcal) at a moderate (65% $\text{VO}_{2\text{max}}$) intensity (Deighton et al. 2013), despite 90% less exercise time. This may be attributable to the short recovery periods (40 s) in this protocol, which do not allow for the fall in VO_2 that is observed during the 4 min recovery periods with traditional 30 s SIT (Hazell et al. 2014b). Additionally, performing shorter work bouts allows for the improved regeneration of peak power during successive bouts, resulting in a greater number of higher quality efforts (i.e. more work performed) during an exercise session (Hazell et al. 2010) as evidenced by the 5:40 group's ability to maintain peak speed across many bouts.

Due to the relatively low EE during a traditional 30 s SIT session compared to moderate-intensity continuous aerobic training, improvements in body composition (Whyte et al. 2010; MacPherson et al. 2011; Hazell et al. 2014a) have been partly attributed to protracted increases in post-exercise metabolism

(i.e. EPOC) observed after SIT (Hazell et al. 2012; Chan and Burns 2013; Williams et al. 2013; Townsend et al. 2014; Beaulieu et al. 2015), HIIT (Skelly et al. 2014), and other variations of supramaximal interval exercise (Bahr et al. 1992; Laforgia et al. 1997). We observed an elevated VO_2 after all three SIT protocols during the 1st h post-exercise with a return to resting values hereafter. This is in agreement with previous studies showing a relatively short-lived (30-60 min) increase in VO_2 post-exercise (Chan and Burns 2013; Williams et al. 2013; Beaulieu et al. 2015), though our group has also shown a more prolonged (180 min) effect with continuous measurement of gas exchange (Townsend et al. 2014). However, total O_2 consumed over the entire post-exercise period was greater with all three SIT protocols compared to CTRL resulting in a 3 h EPOC response that was greater with traditional 30:240 SIT (13 L) and 15:120 (14 L) compared to 5:40 (10 L). This response is similar to that observed in previous studies (8-14 L) over an acute (<3 h) post-exercise period (Chan and Burns 2013; Williams et al. 2013; Townsend et al. 2014) and suggests that the first half of a 30 s “all-out” effort is sufficient for provoking acute metabolic perturbations that increase EPOC. Although subtle increases in VO_2 during each individual time-point may not be significant (Williams et al. 2013), the cumulative effect of each h post-exercise can increase EE over a prolonged measurement period, as supported by the increased EPOC response (~65 L) observed over a 24 h period after both SIT (Hazell et al. 2012) and HIIT (Skelly et al. 2014). Similarly, two studies involving supramaximal bouts of interval cycling (1-3 x 2 min bouts at 108% $\text{VO}_{2\text{max}}$) and running (20 x 1 min bouts at 105% $\text{VO}_{2\text{max}}$) have both shown an increase in EPOC measured over 4 (16 L) and 9 (15 L) h, respectively (Bahr et al. 1992; Laforgia et al. 1997). While the energy deficit due to EPOC in the acute post-exercise period (40-65 kcal) may be insufficient for achieving substantial fat loss (Williams et al. 2013), it is unlikely that this brief time frame fully encapsulates the magnitude and/or duration of response, which clearly extends well beyond this point (Hazell et al. 2012; Skelly et al. 2014).

The mechanisms responsible for EPOC are likely due to the metabolic perturbations that arise from SIT and the subsequent processes required for restoring physiological equilibrium. These processes involve the replenishment of oxygen stores (in blood and tissues), re-synthesis of glycogen and muscle

metabolites (i.e. ATP, PCr), lactate dissipation, normalization of body temperature and pH, increases in muscle protein turnover, catecholamine release, and increased triglyceride/fatty acid cycling (Laforgia et al. 2006). As anaerobic energy yield from PCr and glycolysis peaks within the first 15 s of “all-out” exercise (Smith and Hill 1991), shorter SIT bouts may sufficiently disrupt myocellular energy status to stimulate some of these processes, which is consistent with the similar EPOC response after 15:120 and traditional 30:240 SIT. The greater EPOC after both of these protocols compared to 5:40 SIT may be attributable to a greater cost of glycogen resynthesis associated with prolonged efforts, as maximal sprinting reduces muscle glycogen by ~12% over 10 s, ~18% over 20 s, and >30% over 30 s (Bogdanis et al. 1996; 1998). Consequently, the increased glycolytic/glycogenolytic flux during longer sprint bouts should result in higher lactate formation, a greater reduction in muscle-buffering capacity (H^+ accumulation), and lower pH levels (both intramuscular and in blood) (Bogdanis et al. 1996; 1998). These impairments in anaerobic energy production would also increase reliance on aerobic metabolism during successive bouts (Gaitanos et al. 1993), which would likely be sustained by intramuscular triglyceride/fatty acid cycling (McCartney et al. 1986). Though the 5:40 protocol would still be expected to elicit similar energetic disturbances (though of lesser magnitude as suggested by the EPOC response), interval exercise using shorter work bouts and recovery periods relies more heavily on PCr, which supplies the majority of ATP during the initial seconds (<3 s) of maximal exercise (even over successive bouts) and is rapidly resynthesized in recovery (~50% of pre-exercise values after ~30 s) (Gaitanos et al. 1993). Additionally, as SIT involves a high degree of type II fibre recruitment it is also possible that an increased contribution of inefficient fast-twitch fibres and subsequently greater fatigue over longer duration sprint bouts would elicit greater metabolic disturbances due to an increased ATP and/or O_2 cost of exercise (Casey et al. 1996). Finally, the increase in circulating catecholamines in response to SIT (Williams et al. 2013), which is greater with longer duration bouts (Trapp et al. 2007) may have also played a role due to potential increases in triglyceride/fatty acid cycling via β -adrenoreceptor stimulation (Zouhal et al. 2008).

Several studies have shown increased fat oxidation in the immediate (<2 h) post-exercise period (Chan and Burns 2013; Beaulieu et al. 2015) as well as 6 h (Beaulieu et al. 2015) and 24 h post-exercise (Whyte et al. 2010). Similar to previous studies (Hazell et al. 2012; Chan and Burns 2013; Williams et al. 2013; Beaulieu et al. 2015) we observed a depressed RER in the 1st h post-exercise after both 15:120 and 30:240, which is reflective of CO₂ retention to replenish bicarbonate stores used for lactate buffering (Laforgia et al. 2006). RER during the 2nd h post-exercise remained lower after 15:120 and 30:240 compared to both 5:40 and CTRL, with a corresponding increase (140-170%) in estimated fat oxidation that persisted (>70%) into the 3rd h post-exercise. The greater fat utilization after longer duration sprint bouts (15-30 s) may be linked to a greater glycogen depletion (Bogdanis et al. 1996; 1998) and the higher metabolic priority given to its resynthesis such that energy demands must be covered by triglyceride breakdown (Kiens and Richter 1998). Additionally, catecholamine-induced increases in lipolysis and metabolism (Zouhal et al. 2008) may have been more pronounced with longer compared to shorter sprint bouts (Trapp et al. 2007). Although this lipolytic effect has not always been reported (Williams et al. 2013), several studies have shown increases in catecholamines (Trapp et al. 2007; Williams et al. 2013), free fatty acids (Peake et al. 2014), and glycerol (McCartney et al. 1986; Trapp et al. 2007) after intense intermittent exercise, all of which are indicative of increased fat oxidation (Whyte et al. 2010; Chan and Burns 2013; Beaulieu et al. 2015). These observations are further supported by the improvements in muscle fat oxidative capacity achieved after training (Burgomaster et al. 2008; Perry et al. 2008; Gillen et al. 2013; 2014).

The observed improvements in exercise EE and similar post-exercise EE with shorter work bouts (5-15 s) compared to traditional 30 s SIT, suggests that reducing the sprint bout duration does not compromise energy expenditure and fat oxidation after SIT. While the current investigation involved acute exercise, SIT protocols involving 10, 15, and 20 s bouts of intense exercise have shown significant improvements in aerobic and anaerobic performance (Hazell et al. 2010; Ma et al. 2013; Zelt et al. 2014), as well as cardiometabolic health (Metcalf et al. 2012; Gillen et al. 2014; 2016) over 2-6 weeks of training. Therefore, our acute data combined with these chronic training adaptations emphasize the

importance of peak power generation during the initial seconds (≤ 15 s) of a SIT bout, which may be a more potent metabolic stimulus for driving adaptive mechanisms than the maintenance of power output that follows (Hazell et al. 2010). This seems logical as nearly half the work performed and the majority of ATP depletion occurs within the first 10 s of a maximal sprint (Bogdanis et al. 1996; 1998). Consequently, shorter sprints are likely sufficient for stimulating energy sensing mechanisms (i.e. AMPK mediated activation of PGC1- α) that facilitate metabolic remodeling associated with SIT (Gibala et al. 2009; Serpiello et al. 2012).

While the current study provides valuable insight into the acute metabolic effect of modified SIT protocols, there are some limitations that must be considered. First, the intermittent measurement of gas exchange (i.e. last 15 min of each hour) may have influenced our results, though previous work from our lab shows similar results using a continuous 2 h gas collection period (Townsend et al. 2014). Second, CO₂ retention in the immediate post-exercise period may have influenced our RER-derived estimates of fat oxidation as the duration of this effect is unknown and there may have been potential differences in lactate buffering between the three SIT protocols. Third, while each participant recorded and replicated their intake prior to each session we did not provide a standardized test meal to all participants. Finally, as the current study involved healthy and active young adults, it is difficult to generalize our findings to other populations (i.e. unfit, overweight, sedentary).

SUMMARY

In summary, shorter SIT bouts (5-15 s) promote greater increases in exercise EE as well as comparable post-exercise EE to the traditional 30 s SIT protocol, though longer bouts (15-30 s) are required to increase post-exercise fat utilization. These findings have important implications for long-term energy balance and weight management as these modified SIT protocols potentially stimulate metabolism in a time-efficient manner (2 min of exercise). Nevertheless, the significant improvements in body composition following SIT (Whyte et al. 2010; MacPherson et al. 2011; Hazell et al. 2014a) cannot be attributed to EE alone, but rather the potential combination of an elevated metabolic rate, greater

suppression of appetite (Hazell et al. 2016), and increased fat oxidation (Chan and Burns 2013) among other factors that facilitate the energy deficit required for fat loss. SIT protocols with shorter work bouts may also elicit more favorable psychological responses compared to traditional 30 s SIT, which can lead to improved exercise involvement and adherence (Townsend et al. accepted). Future studies should investigate if training regimens involving these modified SIT protocols can promote similar benefits as traditional SIT, and establish the minimum dose of exercise required to achieve these results.

CONFLICT OF INTEREST

The authors report no conflicts of interest associated with this manuscript.

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Table 1 – Average heart rate/RER during and post-exercise exercise.

	CTRL	5:40	15:120	30:240
<i>HR (b·min⁻¹)</i>				
During	67±10	158±11 ^{ad}	151±14 ^a	145±9 ^a
1 h post-exercise	62±8	83±7 ^a	85±13 ^a	82±20 ^a
2 h post-exercise	59±8	71±11	73±12 ^a	71±10
3 h post-exercise	59±9	67±10	69±9	68±9
Total post-exercise	62±8	86±7 ^a	88±10 ^a	87±8 ^a
<i>RER</i>				
During	0.93±0.05	1.11±0.06 ^a	1.33±0.09 ^{ab}	1.43±0.16 ^{abc}
1 h post-exercise	0.90±0.06	0.87±0.08	0.77±0.09 ^{ab}	0.72±0.06 ^{ab}
2 h post-exercise	0.88±0.04	0.85±0.07	0.80±0.08 ^a	0.78±0.04 ^a
3 h post-exercise	0.86±0.04	0.84±0.05	0.83±0.07	0.81±0.05
Total post-exercise	0.90±0.03	0.87±0.05	0.83±0.06 ^a	0.84±0.05 ^a

Note: ^a different versus CTRL; ^b different versus 5:40; ^c different versus 15:120; ^d different versus 30:240 (P<0.05).

FIGURE CAPTIONS

Figure 1. Experimental session timeline. SIT; Sprint interval training.

Figure 2. During exercise oxygen consumption. A) VO_2 ($\text{L}\cdot\text{min}^{-1}$). B) Total L O_2 consumed (excluding warm-up and cool-down). *Note:* Unlike letters indicate significantly different mean values ($P<0.05$).

Figure 3. Post-exercise oxygen consumption. A) VO_2 ($\text{L}\cdot\text{min}^{-1}$) during each h of recovery. B) Total L O_2 consumed and EPOC (gray shaded areas) over the entire 3-h post-exercise period. *Note:* Unlike letters indicate significantly different mean values. * - denotes significantly greater EPOC vs 5:40.

Figure 4. Total session (including baseline, warm-up, exercise, cool-down, recovery) energy expenditure estimated from total L O_2 ($5 \text{ kcal}\cdot\text{L}^{-1}$). *Note:* Unlike letters indicate significantly different mean values ($P<0.05$). CTRL; control.

Figure 5. Estimated fat oxidation rates post-exercise. *Note:* ^a different versus CTRL; ^b different versus 5:40.

Figure 6. Peak speed ($\text{m}\cdot\text{sec}^{-1}$) output during each exercise session: A) 5:40; B) 15:120; and C) 30:240. Percentages in parentheses indicate the decline in peak speed from the first to the last work bout. *Note:* * - denotes significant decrease in average peak speed ($P<0.005$)

Figure 1. Experimental session timeline. SIT; Sprint interval training.

108x48mm (300 x 300 DPI)

Draft

Figure 2. During exercise oxygen consumption. A) VO_2 ($\text{L} \cdot \text{min}^{-1}$). B) Total L O_2 consumed (excluding warmup and cool-down). Note: Unlike letters indicate significantly different mean values ($P < 0.05$).

172x187mm (300 x 300 DPI)

Figure 3. Post-exercise oxygen consumption. A) VO_2 (L·min⁻¹) during each h of recovery. B) Total L O_2 consumed and EPOC (gray shaded areas) over the entire 3-h post-exercise period. Note: Unlike letters indicate significantly different mean values. * - denotes significantly greater EPOC vs 5:40.

227x88mm (300 x 300 DPI)

Figure 4. Total session (including baseline, warm-up, exercise, cool-down, recovery) energy expenditure estimated from total $L O_2$ ($5 \text{ kcal} \cdot L^{-1}$). Note: Unlike letters indicate significantly different mean values ($P < 0.05$). CTRL; control.

Figure 5. Estimated fat oxidation rates post-exercise. Note: *a* different versus CTRL; *b* different versus 5:40.

82x68mm (300 x 300 DPI)

Figure 6. Peak speed ($\text{m}\cdot\text{sec}^{-1}$) output during each exercise session: A) 5:40; B) 15:120; and C) 30:240. Percentages in parentheses indicate the decline in peak speed from the first to the last work bout. Note: * - denotes significant decrease in average peak speed ($P < 0.005$)

105x235mm (300 x 300 DPI)