Modified sprint interval training protocols. Part I. Physiological responses

Hashim Islam, Logan K. Townsend, and Tom J. Hazell

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 postexercise, as well as postexercise fat oxidation rates. Nine active males completed a resting control session (CTRL) and 3 SIT sessions in randomized order: (i) 30:240 (4 × 30-s bouts, 240-s recovery); (ii) 15:120 (8 × 15-s bouts, 120-s recovery); (iii) 5:40 (24 × 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 postexercise. 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. Postexercise 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). Postexercise fat oxidation was similar (P = 0.650) after 15:120 (0.104 g·min⁻¹) and 30:240 (0.116 g·min⁻¹) and both were greater (P < 0.030) than 5:40 (0.072 g·min⁻¹) and CTRL (0.049 g·min⁻¹). In conclusion, shorter SIT work bouts that target peak power generation increase exercise EE without compromising postexercise EE, though longer bouts promote greater postexercise fat utilization.

Key words: high-intensity interval training, energy expenditure, excess postexercise oxygen consumption, fat oxidation, repeated sprint exercise, peak power generation.

Résumé : On observe les adaptations à l’entraînement par intervalle au sprint (< 15 s) au moyen de brèves (<15 s) séances d’exercice, soulignant ainsi que la production d’une puissance de pointe peut révéler un important stimulus métabolique. Cette étude évalue les effets lors d’un SIT de la modification de la durée des périodes de travail et de repos sur la dépense énergétique (< EE>) durant et après l’exercice et sur le taux postexercice d’oxydation des graisses. Neuf hommes actifs participent à une séquence de contrôle au repos (< CTRL >) et à trois séances SIT dans un ordre aléatoire : (i) 30:240 (4 × 30 s d’exercice, 240 s de récupération), (ii) 15:120 (8 × 15 s d’exercice, 120 s de récupération) et (iii) 5:40 (24 × 5 s d’exercice, 40 s de récupération). Les protocoles apparaissent la durée totale de travail (2 min) et de récupération (16 min) de même que le ratio travail/récupération (1:8 s). On détermine EE et le taux d’oxydation des graisses par l’analyse des échanges gazeux avant, pendant et durant 3 h postexercice. Tous les protocoles suscitent une augmentation de EE par rapport à CTRL (P < 0.001). EE à l’effort est plus grande (P < 0.001) dans le protocole 5:40 (209 kcal) comparativement à 15:120 (163 kcal) et 30:240 (138 kcal) et plus grande dans le protocole 15:120 (P < 0.001) par rapport à 30:240. EE postexercice est plus grande (P = 0.014) dans le protocole 15:120 (313 kcal) comparativement à 5:40 (294 kcal), mais ces deux protocoles suscitent une EE similaire (P > 0.077) à 30:240 (309 kcal). Le taux d’oxydation des graisses postexercice est semblable (P = 0.650) après 15:120 (0,104 g·min⁻¹) et 30:240 (0,116 g·min⁻¹), mais ces taux sont plus élevés (P < 0.030) que dans le protocole 5:40 (0,072 g·min⁻¹) et dans CTRL (0,049 g·min⁻¹). De courtes séances SIT ciblant la puissance de pointe suscitent une plus grande EE à l’effort, et ce, sans affecter EE postexercice, mais de plus longues séances génèrent une plus grande utilisation des graisses postexercice. [Traduit par la Rédaction]

Mots-clés : entraînement par intervalle d’intensité élevée, dépense énergétique, consommation d’oxygène postexercice en surplus, oxydation des graisses, exercice de sprint répétée, production de puissance de pointe.

Introduction

High-intensity interval training (HIIT) involves brief repeated bouts of near maximal exercise (80%-100% maximal heart rate (HRmax)) interspersed with short recovery periods and has been shown to elicit comparable health and performance benefits to moderate-intensity (~70% maximal oxygen consumption (V̇O2max)) 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% V̇O2max) 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, in-

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creased 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., $V_{O2max}$ 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 s (Hazell et al. 2010), 15 s (Zelt et al. 2014), and 20 s (Ma et al. 2012; 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 ~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 a 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. 2017), 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% $V_{O2max}$ (~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 energy expenditure (EE) is remarkably similar after both protocols because of a greater protracted increase in resting metabolism (i.e., increased $O_2$ utilization) that occurs after SIT (Hazell et al. 2012). This excess postexercise 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 postexercise 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 postexercise fat oxidation (Whyte et al. 2010; Chan and Burns 2013; Beaulieu et al. 2015) and chronically upregulate 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 postexercise) and/or influence substrate utilization in the postexercise 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 at 3 h postexercise, as well as postexercise fat oxidation using 3 SIT protocols (traditional 30-s SIT and 2 modified protocols). All 3 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 because of 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 oxygen consumption ($V_{O2}$) 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 postexercise period because of 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 (Kiens and Richter 1998).

## Materials and methods

### Participants

Nine recreationally active males (age, 23.3 ± 3.0 years; height, 178.4 ± 5.4 cm; weight, 78.3 ± 9.0 kg; body mass index, 24.6 ± 2.2 kg·m⁻²; $V_{O2max}$, 48.9 ± 5.3 mL·kg⁻¹·min⁻¹) volunteered to participate in the current study. Participants were nonsmokers and healthy as assessed by the Physical Activity Readiness Questionnaire (PAR-Q) health questionnaire. Although all participants were physically active (≤3 times/week), 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 4 experimental sessions (~4.5 h each) during which $V_{O2}$, carbon dioxide production ($V_{CO2}$), and heart rate (HR) were measured (Fig. 1). Experimental sessions consisted of 1 control session (CTRL; no exercise) and 3 exercise sessions using 1 of the 3 SIT protocols: (i) traditional SIT with 30-s work bouts, (ii) modified SIT with 15-s work bouts, or (iii) modified SIT with 5-s work bouts. To avoid learning effects, all experimental sessions were separated by ≥21 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 $V_{O2max}$ determined during a graded exercise test to exhaustion performed on a motorized treadmill (4Front, Woodway, Wis., USA), $V_{O2}$ and $V_{CO2}$ were measured continuously using an online breath-by-breath gas collection system (MAX-II; AEL Technologies, Pa., USA), which 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 miles/h (1 mile = 1.6 km)) with incremental increases in grade (2%) applied every 2 min until volitional fatigue). HR was recorded beat-to-beat throughout the test using an integrated HR monitor (FTI: Polar Electro, Que., Canada). $V_{O2max}$ was taken as the greatest 30-s average in presence of a plateau in $V_{O2}$ values (<1.35 mL·kg⁻¹·min⁻¹ increase) despite increasing workload, or 2 of the following criteria: (i) a respiratory exchange ratio (RER) value >1.10, (ii) achievement of a HRmax (>10 beats-min⁻¹ of age-predicted maximum (220 – age)), and/or (iii) voluntary exhaustion. After a 5-min cooldown followed by sufficient rest (>20 min), participants were allowed to practice all-out running efforts on a specialized self-propelled treadmill (HiTrainer, Que., Canada) on which all the exercise sessions would be performed.
Fig. 1. Experimental session timeline. SIT, sprint interval training.

Experimental session
Participants arrived at the laboratory at 0800 h after having consumed a standardized light breakfast at 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 (Fig. 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 an HR monitor (Polar FT1) and silicon face-mask (Vmask, Hans Rudolph Inc., Kans., USA) for the continuous measurement of gas exchange (\(\dot{V}O_2\) and \(\dot{V}CO_2\)) from 0830–0845 h (baseline), 0845–0915 h (exercise period), and 0915–0945 h (30 min postexercise). Hereafter, gas exchange was measured (Fig. 1) during the last 15 min of each hour postexercise from 1000–1015 h (first hour postexercise), 1100–1115 h (second hour postexercise), and 1200–1215 h (third hour postexercise). 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) following by an 18-min SIT session and 5-min cooldown (30 min total). Warm-up and cooldown 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 1 of the following 3 SIT protocols: (i) 30:240 (traditional SIT; 4 × 30-s bouts followed by 240 s (4 min) of rest); (ii) 15:120 (8 × 15-s bouts followed by 120 s (2 min) of rest); or (iii) 5:40 (24 × 5-s bouts followed by 40 s of 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 (m·s\(^{-1}\)) attained during each sprint in 0.5-s intervals.

\(\dot{V}O_2\)
\(\dot{V}O_2\) (L·min\(^{-1}\)) was recorded as 30-s averages over the entire duration of each gas collection period. \(\dot{V}O_2\) during exercise (excluding warm-up and recovery) was taken as the average \(\dot{V}O_2\) over the duration of each SIT protocol (18 min). Postexercise \(\dot{V}O_2\) was determined by plotting the average \(\dot{V}O_2\) at each time-point (30 min postexercise and last 15 min of the first, second, and third hours postexercise), and calculating the area under the curve using the trapezoid method. The rate of \(\dot{V}O_2\) was multiplied by the duration of each distinct gas collection period to obtain total \(\dot{V}O_2\) (L). The immediate 30-min postexercise measurement was included as part of the first postexercise hour \(\dot{V}O_2\) calculation. EPOC was calculated by subtracting total \(\dot{V}O_2\) during the CTRL session from corresponding postexercise gas collection periods in each exercise session. Total EE (kcal) was calculated from total \(\dot{V}O_2\) (L) during and postexercise, assuming 5 kcal per litre O\(_2\) consumed given the limitations of using RER during exhaustive, nonsteady-state exercise (Laforgia et al. 1997).

Fat oxidation
The rate of fat oxidation was calculated during each hour postexercise using the following formula (Péronnet and Massicotte 1991):

\[
\text{fat oxidation} = 1.695 \times \dot{V}O_2 - 1.701 \times \dot{V}CO_2
\]

where fat is in g·min\(^{-1}\) and \(\dot{V}O_2\) and \(\dot{V}CO_2\) are in L·min\(^{-1}\).

Statistical analysis
All data were analyzed using Sigma Stat for Windows (version 3.5). Two-way repeated-measures ANOVA was used to determine differences in \(\dot{V}O_2\), HR, RER, and fat oxidation among the 4 treatments at all time points. A 1-way repeated-measures ANOVA was used to determine differences in total \(\dot{V}O_2\), HR, and RER (exercise, postexercise, and entire session) as well as fat oxidation and EPOC. Tukey’s honestly significant difference tests were used for post hoc analysis where necessary. Significance was set at \(P < 0.05\). All data are presented as means ± SD.

Results
\(\dot{V}O_2\) and EE
There was a significant \((P < 0.001)\) interaction effect (session × time) for average \(\dot{V}O_2\). As expected, \(\dot{V}O_2\) in the exercise period (Fig. 2A) was elevated \((P < 0.001)\) during all 3 SIT sessions compared with CTRL (0.273 ± 0.05 L·min\(^{-1}\)). Specifically, exercise \(\dot{V}O_2\) was...
greater \( (P < 0.001) \) during 5:40 \((2.326 \pm 0.258 \text{ L.min}^{-1})\) compared with both 15:120 \((1.793 \pm 0.238 \text{ L.min}^{-1})\) and 30:240 \((1.528 \pm 0.187 \text{ L.min}^{-1})\), while 15:120 was also greater \( (P < 0.001) \) than 30:240. EE based on total \( \dot{V}O_2 \) \((\text{L} \cdot \text{min}^{-1})\) and 5:40 was greater \( (P < 0.001) \) compared with 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 \( \dot{V}O_2 \) during the first hour postexercise (Fig. 3A) was similar \( (P > 0.532) \) in all 3 SIT sessions \((5:40: 0.349 \pm 0.031 \text{ L.min}^{-1}; 15:120: 0.382 \pm 0.044; 30:240: 0.374 \pm 0.038)\) and remained elevated \( (P < 0.020) \) versus CTRL \((0.273 \pm 0.040 \text{ L.min}^{-1})\), \( \dot{V}O_2 \) during the second and third hours postexercise was not different \( (P > 0.153) \) between experimental sessions (Fig. 3A). Total \( \dot{V}O_2 \) \((\text{L})\) over the entire 3-h postexercise period (Fig. 3B) was greater \( (P < 0.001) \) in all 3 SIT sessions compared with CTRL \((48.8 \pm 5.8 \text{ L})\). Total session \( \dot{V}O_2 \) was greater \( (P = 0.014) \) after 15:120 \((62.6 \pm 6.7 \text{ L})\) compared with 5:40 \((58.8 \pm 4.9 \text{ L})\), though not different \( (P = 0.863) \) between 15:120 and 30:240 \((61.7 \pm 5.1 \text{ L})\) or between 30:240 and 5:40 \((P = 0.078) \). The 3-h EPOC (Fig. 3B) for 15:120 \((162.6 \pm 19.2 \text{ kcal})\) and 30:240 \((129.2 \pm 2.6 \text{ kcal})\) was similar \( (P = 0.667) \) and both were greater \( (P < 0.040) \) compared with 5:40 \((10.0 \pm 2.9 \text{ kcal})\). EE over the entire experimental session (Fig. 4) was greater \( (P < 0.001) \) during all 3 SIT sessions versus CTRL \((348.3 \pm 45.7 \text{ kcal})\). Total session EE was similar \( (P = 0.195) \) between 5:40 \((647.8 \pm 60.0 \text{ kcal})\) and 15:120 \((626.4 \pm 65.5 \text{ kcal})\) and both were greater \( (P < 0.008) \) compared with 30:240 \((588.5 \pm 56.0 \text{ kcal})\).

**HR**

There was a significant \( (P < 0.001) \) interaction effect (session \( \times \) time) for average HR (Table 1). All 3 SIT sessions resulted in an elevated \( (P < 0.001) \) HR during the exercise period compared with CTRL. HR during exercise was similar \( (P = 0.374) \) between 5:40 and 15:120, though greater \( (P = 0.036) \) with 5:40 compared with 30:240. HR during the first hour postexercise was similar \( (P > 0.902) \) between the 3 SIT sessions and all were greater \( (P < 0.001) \) compared with CTRL. HR in the second hour postexercise was greater \( (P = 0.030) \) only after 15:120 compared with CTRL, and HR during the third hour postexercise was not different \( (P > 0.156) \) between the experimental sessions. Total session HR was elevated for all 3 SIT sessions versus CTRL \( (P < 0.001) \) with no differences between sessions.

**RER**

There was a significant \( (P < 0.001) \) interaction effect (session \( \times \) time) for average RER (Table 1). All 3 SIT sessions resulted in a greater \( (P < 0.001) \) RER during the exercise period compared with CTRL. Exercise RER was greater \( (P < 0.001) \) during 30:240 compared with both 15:120 and 5:40, while 15:120 was also greater \( (P < 0.001) \) compared with 5:40. RER during the first hour postexercise was similar \( (P = 0.261) \) between 15:120 and 30:240, and both were lower compared with 5:40 \( (P < 0.002) \) and CTRL \( (P < 0.001) \). RER during the second hour postexercise was similar \( (P > 0.053) \) between the 3 SIT protocols, though 15:120 and 30:240 both remained lower \( (P < 0.01) \) compared with CTRL. There were no differences \( (P > 0.263) \) in RER between the experimental sessions during the third hour postexercise. Total session RER was lower for both 15:120 \((P = 0.023)\) and 30:240 \((P = 0.049)\) versus CTRL (Table 1).

**Fat oxidation**

There was a significant \( (P < 0.001) \) interaction effect (session \( \times \) time) for fat oxidation rates in the postexercise period (Fig. 5). Fat oxidation during the first hour postexercise was similar \( (P = 0.659) \) between 15:120 and 30:240, and both were greater \( (P < 0.001) \) compared with CTRL, though only 30:240 was greater \( (P = 0.011) \) compared with 5:40. Fat oxidation during the second hour postexercise was similar \( (P = 0.625) \) between 15:120 and 30:240, and both were greater compared with 5:40 \( (P < 0.004) \) and CTRL \( (P < 0.001) \). Fat oxidation during the third hour postexercise was not different between the 3 SIT protocols \((P > 0.120)\), though 15:120 and 30:240 were greater \( (P < 0.013) \) compared with CTRL. Fat oxidation over the entire 3-h postexercise period was similar \( (P = 0.650) \) with 15:120 and 30:240 and both were greater compared with 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 \((7.2 \pm 0.5 \text{ m.s}^{-1})\) compared with both 15:120 \((6.6 \pm 0.7 \text{ m.s}^{-1})\) and 30:240 \((6.4 \pm 0.9 \text{ m.s}^{-1})\) (Fig. 6A), while 15:120 and 30:240 were not different \( (P = 0.638) \). Peak speed decreased \( (P < 0.015) \) by 14.3\% \((1.0 \text{ m.s}^{-1})\) and 27.5\% \((2.0 \text{ m.s}^{-1})\) from the first to the last sprint bout during 15:120 and 30:240, respectively. The decrease in peak speed during 5:40 \((4.2\%, 0.3 \text{ m.s}^{-1})\) was not significant \( (P = 0.398) \).

**Discussion**

This study investigated the effects of 3 SIT protocols with different work bout \((5–30 \text{ s})\) and recovery period \((40–240 \text{ s})\) durations on \( \dot{V}O_2/\dot{E}E \) during and 3 h postexercise, as well as postexercise fat oxidation rates. All 3 protocols were identical in terms of total exercise time \((2 \text{ min})\), recovery duration \((16 \text{ min})\), and the work/recovery ratio \((1:8 \text{ s})\) compared with traditional SIT \((30:240)\), both modified protocols \((5:40 \text{ and } 15:120)\) elicited greater EE during exercise and similar EE over the 3-h postexercise 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 with traditional 30-s SIT. Additionally, 15:120 resulted in similar fat utilization during the postexercise recovery period compared with 30:240, both of which were increased compared with 5:40 and CTRL. Collectively, these results indicate that modified SIT protocols with shorter work bouts improve exercise EE without compromising postexercise EE, though longer SIT bouts result in greater fat utilization in the postexercise period.
EE during 5:40 (209 kcal) and 15:120 (163 kcal) was increased by 52% and 18%, respectively, compared with 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; Townsend et al. 2014; Beaulieu et al. 2015). Duetoitsbriefnature,themajorityofEEwithtraditional30-s SIT is associated with the postexercise 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, 2014b; Deighton et al. 2013; Townsend et al. 2014; Beaulieu et al. 2015) and comparable to 20 min of continuous exercise (210 kcal) at a moderate (65% \( V\dot{O}_2 \text{max} \)) 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 \( \dot{V}O_2 \) 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 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 with 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 postexercise 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 \( \dot{V}O_2 \) after all 3 SIT protocols during the first hour postexercise with a return to resting values thereafter. This is in agreement with previous studies showing a relatively short-lived (30–60 min) increase in \( \dot{V}O_2 \) postexercise (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 \( \dot{O}_2 \) consumed over the entire postexercise period was greater...
Fig. 6. Peak speed (m·s⁻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. *, Significant decrease in average peak speed (P < 0.005).

with all 3 SIT protocols compared with CTRL, resulting in a 3-h EPOC response that was greater with traditional 30:240 SIT (13 L) and 15:120 (14 L) compared with 5:40 (10 L). This response is similar to that observed in previous studies (8–14 L) over an acute (<3 h) postexercise period (Chan and Burns 2013; Williams et al. 2013; Townsend et al. 2014) and suggests that the first half of a 30-s bout. *, Significant decrease in average peak speed (< 0.005).

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), resynthesis of glycogen and muscle metabolites (i.e., adenosine triphosphate (ATP), phosphocreatine (PCr)), lactate dissociation, 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 with 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 ~20% over 30 s (Bogdanis et al. 1996, 1998). Consequently, the increased glycemic/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 because of an increased ATP and/or O₂ 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 because of 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) postexercise period (Chan and Burns 2013; Beaulieu et al. 2015) as well as at 6 h (Beaulieu et al. 2015) and 24 h postexercise (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 first hour postexercise 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 second hour postexercise remained lower after 15:120 and 30:240 compared with both 5:40 and CTRL, with a corresponding increase (140–170%) in estimated fat oxidation that persisted (>70%) into the third hour postexercise. The greater fat utilization after longer duration sprint bouts (15–30 s) may be linked to a greater glycolysis 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 with 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 postexercise EE with shorter work bouts (5–15 s) compared with the traditional 30-s SIT suggests that reducing the sprint bout duration does not compromise EE 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 (Hazzell et al. 2010; Ma et al. 2013; Zelt et al. 2014) as well as cardio-metabolic health (Metcalfe et al. 2012; Gillen et al. 2014, 2016) over 2–12 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 (Hazzell 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 the first 10 s of a maximal sprint (Bogdanis et al. 1996, 1998).

Supporting the expression 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 favourable psychological responses compared with traditional 30-s SIT, which can lead to improved exercise involvement and adherence (Townsend et al. 2017). 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 statement

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

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