Excess postexercise oxygen consumption after high-intensity and sprint interval exercise, and continuous steady-state exercise

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ABSTRACT

Higher excess postexercise oxygen consumption (EPOC) after high-intensity interval exercise (HIE) and sprint interval exercise (SIE) may contribute to greater fat loss sometimes reported after interval training compared continuous, steady-state exercise (SSE) training. We compared excess postexercise oxygen consumption (EPOC) after HIE, SIE, and SSE. Ten recreationally active males (age 24 ± 4 y) participated in this randomized crossover study. On separate days, subjects completed a resting control trial and three exercise conditions on a cycle ergometer: HIE (four 4-min intervals at 95% HR_{peak} separated by three min of active recovery); and SIE (six 30-s Wingate sprints, separated by four min of active recovery); and SSE (30 min at 80% of peak heart rate (HR_{peak})). Oxygen consumption (VO_2) was measured continuously during and for 3 h after exercise. For all conditions, VO_2 was higher than resting control only during the 1st h postexercise. Although 3-h EPOC and total net EE after exercise were higher (p=0.01) for SIE (22.0 ± 9.3 L; 110 ± 47 kcal) compared to SSE (12.8 ± 8.5 L; 64 ± 43 kcal), total (exercise + postexercise) net O_2 consumed and net EE were greater (p=0.03) for SSE (69.5 ± 18.4 L; 348 ± 92 kcal) than for SIE (54.2 ± 12.0 L; 271 ± 60 kcal). Corresponding values for HIE were not significantly different from SSE or SIE. EPOC after SIE and HIE is unlikely to account for the greater fat loss per unit EE associated with SIE and HIE training reported in the literature.

KEYWORDS: High-intensity interval exercise, Wingate testing, endurance exercise, recovery oxygen uptake, fat oxidation.
INTRODUCTION

Meta-analyses of high-intensity interval exercise (HIE) training (18, 30, 38) and sprint interval exercise (SIE) training (11) have confirmed the effectiveness of low-volume, vigorous exercise for improving aerobic fitness and a number of cardiometabolic risk markers. HIE generally consists of exercise bouts lasting ~1-4 min at ~90-95% of maximum heart rate, separated by 1-3 min of active recovery, whereas SIE typically includes 2-6 supramaximal efforts lasting ~30 s (i.e., Wingate cycling tests), with 3-4.5 min of rest in between. These modes of exercise training have also been shown to be equally (22, 26, 28, 31) or more (35, 36) effective for body fat reduction, despite less total exercise time and exercise energy expenditure. Thus HIE and SIE training may produce greater fat loss per unit of exercise energy expenditure (EE) than traditional, steady-state exercise (SSE) (22, 35, 36).

The reasons for the greater fat loss per unit EE for interval exercise training are not well established, although greater excess postexercise oxygen consumption (EPOC) has been proposed (4, 5, 22). Compared to SSE, HIE has been reported to produce a greater EPOC in some studies (20, 21) but not others (24, 25, 33). Because the magnitude of EPOC is influenced more by exercise intensity than exercise duration (2, 12, 20), the potential for SIE to affect EPOC may be greater than that of HIE.

Several studies have reported EPOC after SIE (7, 8, 13, 32, 34, 40). Comparisons are difficult due to differences in experimental design. Three of the studies included SIE as the only exercise condition (7, 8, 32). Measurement of EPOC also varied considerably, with three of the studies measuring EPOC for <120 min (7, 8, 34). Two of the studies reported 24-h EPOC (13, 32), but one of these (13) did not collect oxygen consumption (VO₂) for 105 of the initial 180 min postexercise. Data from 24-h whole-room calorimeter assessments indicate that all of the EPOC after SIE occurs during the first few hours postexercise (32).
The most significant limitation of the studies of EPOC after SIE is the fact that they all used between two and five bouts of SIE, with five of the studies using either two (7), three (34), or four (8, 13, 40) 30-s bouts of SIE. Conclusions from these studies suggest that the EPOC is relatively short-lasting and quantitatively small. However, it has been shown that the number of intervals performed (i.e., volume) affects EPOC (21, 32). This is relevant to the interpretation of SIE training studies because significant decreases in body fat (22) and waist and hip circumference (39), or increases in skeletal muscle fat oxidation enzymes (6), have been reported when SIE training progressed to six 30-s bouts during each training session over the final weeks of training. Since no studies have examined EPOC after SIE consisting of six 30-s sprint interval bouts, and no studies have compared EPOC after HIE, SIE and SSE compared to a no-exercise control trial, the purpose of the study was to fill this gap in the literature. A better understanding of EPOC and EE following different exercise protocols may also assist fitness professionals and coaches with exercise prescription for weight loss or weight maintenance.

We hypothesized that SIE would elicit a greater 3-h EPOC than both HIE and SSE, and that SIE would result in the greatest postexercise fat oxidation.

METHODS

Experimental Approach to the Problem

A randomized, crossover with repeated measures design was used for this study. Each subject performed three separate exercise protocols (HIE, SIE and SSE) and one control condition in random order with at least 72 h between trials to avoid carryover effects. This design strengthened internal validity and allowed us to test our hypothesis and ensure practical application of the results. During and for 3 h after each trial VO2 and EE were measured to assess differences between conditions for net O2 consumed and EE. Subjects were instructed
to not exercise or consume caffeine or alcohol >48 hours prior to each visit. Trial order for the four experimental conditions was randomized for all subjects using a random number generator. Sample size (n=10) was determined based on previous studies that have assessed EPOC and fat oxidation differences between exercise protocols (7, 8, 13, 33).

Subjects

Recreationally active, nonsmoking men (19-32 years old) were recruited via flyers posted around the Arizona State University campuses. Of the 13 subjects enrolled, ten (mean ± SD: age 24 ± 4 y; height 171.6 ± 5.1 cm; weight 73.1 ± 8.2 kg; body mass index 24.8 ± 1.9 kg·m$^2$; percent body fat 13.5 ± 4.4 %; VO$_{2\text{peak}}$ 45.9 ± 7.2 ml·min$^{-1}$·kg$^{-1}$) completed the study. Three subjects were unable to complete the study due to nausea, lightheadedness and/or vomiting experienced during the SIE protocol. This study was approved by the Arizona State University Institutional Review Board and study procedures were carried out in a climate-controlled research laboratory. All subjects provided informed written consent prior to participation.

Preliminary visit

Prior to the first experimental test day, subjects reported to the laboratory to have anthropometrics and peak oxygen consumption (VO$_{2\text{peak}}$) assessed. Body composition was assessed by Bod Pod$^\text{TM}$ (Cosmed, Concord, CA, USA) using air-displacement plethysmography. Standing height (cm) was measured to within 0.1 cm against a wall-mounted stadiometer (Seca, Hamburg, Germany). Weight (kg) was measured using an electronic scale which is integrated with the Bod Pod.

VO$_{2\text{peak}}$ was determined using a ramp protocol (30 W·min$^{-1}$) on an electronically-braked cycle ergometer (Viasprint 150P, Ergoline, Bitz, Germany). After a 5-min warm up at 50 W, the
resistance increased continuously (1 W every 2 s) until subjects reached volitional exhaustion. Peak heart rate (HR<sub>peak</sub>) was recorded and used for exercise prescription. Ventilation and pulmonary gas exchange were measured continuously using the Oxycon Mobile™ portable breath-by-breath metabolic measurement system (Carefusion, San Diego, CA, USA) for determination of VO<sub>2</sub>, carbon dioxide production (VCO<sub>2</sub>) and respiratory exchange ratio (RER). The Oxycon Mobile™ was calibrated as per manufacturer specifications prior to each baseline test and exercise/control visit. Heart rate was measured using a Polar Heart Rate monitor (Polar, Lake Success, NY, USA). VO<sub>2peak</sub> was defined as the average of the 2 highest consecutive 15-s averages achieved during the ramp protocol.

**Experimental test days**

Each subject reported to the laboratory on 4 occasions, each separated by a minimum of 72 h to avoid carryover effects. Subjects were instructed to not exercise or consume caffeine or alcohol for 48 h prior to each visit. The 4 experimental conditions included a control (no exercise) day and 3 exercise conditions. Condition order was randomized. Subjects were asked to eat the same breakfast of their choice at 0800 h on the day of each laboratory visit. Subjects recorded what they ate and were instructed to consume the same meal prior to every visit. Thereafter, subjects reported to the research laboratory at 1130 h. Pulmonary ventilation and gas exchange were collected during seated rest from 1145 to 1200 h (baseline), from 1200 to 1235 h (during exercise or during seated rest for the control condition), and continuously for 3 h postexercise (or for a time-matched 3 h during the seated rest condition). All non-exercise measurements were taken while subjects rested quietly in a comfortable chair.

During all experimental conditions the subjects wore the same lightweight, portable metabolic measurement system used in the test to determine VO<sub>2peak</sub>. During the 3-h
postexercise period subjects were permitted to remove the facemask at fixed time points to periodically drink water.

Exercise Sessions

Each of the 3 exercise conditions included a 5-min warm-up at 50-60% HR\textsubscript{peak}. SIE consisted of six 30-s sprints (Wingate) with the resistance set at 0.075 x subject body weight (kg), followed by 4 min of active recovery (60% HR\textsubscript{peak}) (23 min total exercise time). A mechanically-braked, calibrated cycle ergometer (Monark Ergomedic 828E, Monark, Sweden) was used for SIE in accordance with recommendations for mechanically-braked ergometers (3). High-intensity interval exercise (HIE) consisted of four 4-min intervals at 95% HR\textsubscript{peak}, with 3 min of active recovery (60% HR\textsubscript{peak}) in between intervals (25 min total exercise time). The SSE session consisted of 30 min of continuous exercise at 80% HR\textsubscript{peak} (30 min total exercise time). All HIE and SSE sessions were performed on the same electronically-braked cycle ergometer that was used for the assessment of VO\textsubscript{2peak}.

Calculations

Net O\textsubscript{2} consumed during and after (EPOC) each exercise condition was calculated by subtracting O\textsubscript{2} consumed during the time-matched control condition from the O\textsubscript{2} consumed during and after each of the exercise conditions. Due to the limitations of using RER to estimate substrate utilization during periods of non-steady-state VO\textsubscript{2} (i.e., during HIE, SIE and immediately postexercise), energy expenditure (EE, in kcal) was calculated by assuming that 1L O\textsubscript{2} consumed corresponds to 5 kcal (33, 40). However, because blood bicarbonate levels have been reported to return to resting levels within 30 min after cessation of high-intensity exercise (29), and arterial CO\textsubscript{2} partial pressure has been shown to be not different from resting control
conditions from min 60-120 after HIE (24), we used VO\textsubscript{2} and VCO\textsubscript{2} data to estimate fat oxidation during the 2\textsuperscript{nd} and 3\textsuperscript{rd} h postexercise (10):

\[
\text{Fat oxidation rate (g/min\textsuperscript{-1})} = 1.67 (\text{VO}_2 \text{ L-min\textsuperscript{-1}}) - 1.67 (\text{VCO}_2 \text{ L-min\textsuperscript{-1}})
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**Statistical Analysis**

All data were analyzed using SPSS Software (SPSS 21.0, IBM Corporation, Armonk, NY). Descriptive variables are presented as Mean ± SD and significance was set at P<0.05. A one-way repeated measures analysis of variance (ANOVA) was used to compare all four conditions for baseline VO\textsubscript{2}, to compare exercise conditions for net O\textsubscript{2} consumed and net EE during exercise, and to compare total combined (exercise + postexercise) net O\textsubscript{2} consumed and EE. A two-way repeated measures ANOVA (Condition x Time) was used to determine differences between mean VO\textsubscript{2}, RER and total fat oxidation by condition over time during the postexercise period. If the sphericity assumption was violated (Greenhouse-Geisser \(\varepsilon < 0.75\)), degrees of freedom (df values) for within-subject effects were adjusted using the Greenhouse-Geisser correction. Bonferroni correction was used for post-hoc pairwise comparison of means for significant interaction and protocol effects.

**RESULTS**

**Baseline measurements**

There were no differences between conditions for baseline VO\textsubscript{2} (L-min\textsuperscript{-1}) (CON 0.35 ± 0.08; SSE 0.33 ± 0.04; HIE 0.31 ± 0.04; SIE 0.33 ± 0.06; p=0.22).

**Oxygen consumption and energy expenditure during exercise**

Net VO\textsubscript{2} during SSE (1.9 ± 0.4 L-min\textsuperscript{-1}) and HIE (2.0 ± 0.4 L-min\textsuperscript{-1}) were significantly greater than SIE (1.4 ± 0.3 L-min\textsuperscript{-1}) (p<0.001). Net O\textsubscript{2} consumed and net EE during exercise
were significantly higher for SSE (56.7 ± 11.6 L; 284 ± 58 kcal) and HIE (49.2 ± 9.0 L; 246 ± 45 kcal) compared with SIE (32.2 ± 6.5 L; 161 ± 33 kcal) (p<0.001). There was no significant difference between SSE and HIE for net VO$_2$ (p=0.77), net O$_2$ consumed (p=0.38) or net EE during exercise (p=0.39).

**Postexercise VO$_2$, EPOC, and energy expenditure**

There was a significant condition (p<0.001) and time (p<0.001) effect for 3-h postexercise VO$_2$ as well as a time x condition interaction (p<0.001) (Figure 1). Postexercise VO$_2$ was significantly (p<0.001) greater in all exercise conditions compared to CON during the initial 30 min following exercise (SSE: 0.53 ± 0.09 L·min$^{-1}$; HIE: 0.62 ± 0.10 L·min$^{-1}$; SIE: 0.72 ± 0.08 L·min$^{-1}$ versus CON: 0.31 ± 0.05 L·min$^{-1}$). VO$_2$ was also significantly higher in SIE compared to SSE during initial 30 min postexercise (p<0.001). In addition, VO$_2$ after SIE and HIE were significantly (p<0.05) greater than CON between 30 and 60 min postexercise (SIE: 0.40 ± 0.05 L·min$^{-1}$; HIE: 0.36 ± 0.06 L·min$^{-1}$ versus CON: 0.30 ± 0.05 L·min$^{-1}$). Between 60 min and 180 min postexercise, there were no significant differences in VO$_2$ between conditions.

Cumulative 3-h EPOC and net EE were greater (p=0.01) after SIE (22.0 ± 9.3 L; 110 ± 47 kcal) compared to SSE (12.8 ± 8.5 L; 64 ± 43 kcal) (Figure 2). Cumulative 3-h EPOC and net EE after HIE (16.5 ± 9.2 L; 83 ± 46 kcal) were not different (p>0.05) from SIE or SSE. The majority (65-70%) of excess EE occurred during 1st h after exercise (SIE 77 ± 11 kcal, HIE 57 ± 11 kcal, SSE 42 ± 11 kcal) (Figure 1).

*** (Figure 1 and 2 about here)
Overall net O\textsubscript{2} consumed and energy expenditure (Exercise + postexercise)

Combined exercise and postexercise net O\textsubscript{2} consumed and net EE were significantly higher for SSE (69.5 ± 18.4 L; 348 ± 92 kcal) compared to SIE (54.2 ± 12.0 L; 271 ± 60 kcal) (p=0.03) (Figure 2), and there was a trend for higher net O\textsubscript{2} and net EE for HIE (65.7 ± 16.3 L; 329 ± 82 kcal) compared to SIE (p=0.07).

Postexercise RER and fat oxidation

There was a significant condition (p=0.003), time (p<0.001) and time x condition interaction (p<0.001) effect for postexercise RER. Post-hoc analyses revealed that RER was significantly lower during the 3-h postexercise period following SIE compared to control (p=0.002) and SSE (p=0.04) (Figure 3). RER was not significantly different between HIE and SIE during the 3-h postexercise period (p=0.65). Compared to CON, total fat oxidation after SIE was 4.3 g higher during the second h (SIE: 8.6 ± 1.7 g, CON: 4.3 ± 2.2 g; p=0.001) and 3.1 g higher during the third h (SIE: 8.1 ± 1.8 g, CON: 5.0 ± 2.3 g; p=0.01) (Figure 4). There were no significant differences between exercise trials for postexercise fat oxidation.

*** (Figure 3 and 4 about here)

DISCUSSION

Our results confirmed our hypothesis that SIE elicited a greater EPOC and postexercise fat oxidation than both SSE and HIE. However, because the exercise EE for SIE, even with six interval exercise bouts, was significantly less than that for SSE and HIE, combined exercise + postexercise net EE was lowest for SIE. This result, in addition to our finding that exercise and postexercise EE were not different for SSE and HIE, suggests that it is unlikely that the greater
fat loss observed after interval exercise training reported in some studies (22, 35, 36) is due to greater EPOC after interval exercise.

The EPOC we observed after SIE is higher than reported in previous studies. This is likely due to our use of six 30-s sprint intervals. Williams et al. (40), whose SIE protocol included four 30-s sprints, reported a 3-h EPOC of approximately 8 L, and a net postexercise EE of 40.6 kcal. Our 3-h EPOC and net postexercise EE were 22.0 L and 110 kcal, respectively. The 2.7-fold greater 3-h EPOC and net postexercise EE in our study highlights the effect of greater interval number (volume) on EPOC magnitude. Each additional sprint interval may trigger a greater systemic perturbation than the previous interval resulting in an additive effect (since a full recovery is not reached during the 4-min active recovery period after a supramaximal effort). Since the subjects for both studies had similar values for VO\textsubscript{2peak} (3.4 L min\textsuperscript{-1}), EPOC comparisons are not confounded by differences in aerobic fitness. In addition, our EPOC and postexercise EE values after 30 min (12.3 L; 62 kcal) were greater than those reported by Townsend et al. (34) (8 L; 37.5 kcal), who had subjects perform only three 30-s sprint intervals. Lastly, our EPOC and postexercise EE values after 120 min (18.7 L; 94 kcal) were greater than those reported by Chan and Burns (8) (13.6 L; 64.6 kcal), whose SIE protocol included four 30-s sprints. Neither study reported VO\textsubscript{2peak} values of their subjects.

We measured EPOC for 3 h postexercise based on studies that demonstrated that postexercise VO\textsubscript{2} after SIE had returned to levels not significantly different from resting VO\textsubscript{2} during the first 3 h after exercise cessation (7, 8, 21, 32, 34). Although postexercise VO\textsubscript{2} was not statistically significantly different from CON for any exercise condition after the first 60 min postexercise, the second and third h contributed 30-35% to the total 3-h EPOC, with approximately equal contribution from both the second and third h (Figure 1). Thus we may have underestimated the magnitude of the EPOC by not continuing measurement beyond 3 h. However, because the contribution to net EE during the third h differed by ≤4 kcal between the
three exercise conditions, extending the postexercise measurement period would not likely change our primary conclusion. Furthermore, in an experiment using a whole-room calorimeter, Sevits et al. (32) demonstrated that EPOC after five 30-s sprint exercise bouts had returned to resting baseline within the first 4 h after the SIE session, and was not different for the remainder of the 24 h spent in the whole-room calorimeter. Our results are consistent with their findings, and show that even with six interval bouts, the duration of the EPOC is relatively short, with postexercise VO\textsubscript{2} no longer significantly different from resting control after 60 min postexercise.

SIE was the only exercise condition that resulted in increased postexercise fat oxidation compared to the control trial. Increased fat oxidation is typically observed after either HIE or SIE (2, 4, 19, 20, 24, 25, 40). Even though fat oxidation was increased after SIE, the quantitative significance is uncertain. Whole-room calorimeter data indicated that SIE did not increase 24-h fat oxidation, and overall 24-h fat balance was not changed (32). Thus it is unlikely that EPOC and increased fat oxidation explain the greater fat loss per unit EE of exercise training sessions as reported previously (22, 35, 36). SIE acutely depresses appetite (4, 9, 40), but does not result in reduced energy intake within the initial 24 h after SIE (4). It remains to be determined if long-term SIE training affects energy intake.

Only one study has demonstrated a substantial EPOC after SIE (13). In that study, which included four 30-s sprints, 24-h O\textsubscript{2} consumption was increased by 98 L (~475 kcal). This is more than twice as great as that reported by Sevits et al. (32), who used five 30-s sprints. The discrepancy may be due to the inherent limitations of extrapolating discrete measurements using the trapezoidal method as compared to whole-room calorimetry. In the study of Hazel et al. (13), VO\textsubscript{2} was not measured for 105 of the initial 180 min postexercise, and thereafter was only measured at 6 h and 24 h postexercise. Our results compare favorably with the whole-room calorimeter data of Sevits et al. (32). Their SIE session elevated EE by 225 kcal. Our SIE protocol increased EE by 271 kcal. The higher EE in our study could be expected due to the
extra sprint interval. In view of these findings, and those of others (8, 34, 40), the EPOC associated with SIE appears to be relatively minor and does not offset the lower EE cost of the exercise session itself.

It is possible that high-intensity exercise could facilitate fat loss and long-term weight control via mechanisms other than EPOC. For example, resting energy expenditure (REE) has been reported to be elevated for 17-24 h after a vigorous aerobic exercise session (15, 23, 37), in part due to an increase in sympathetic tone (15). Vigorous aerobic exercise may also improve ease of locomotion (16, 17) and increase nonexercise activity thermogenesis (NEAT) (14). To our knowledge, the specific effects of the HIE and SIE protocols used in our study on REE and NEAT have not been published. However, the whole-room calorimeter study of Sevits et al. (32) suggests that SIE does not elevate REE at 24 h postexercise.

It is difficult to compare our results with others who have used HIE and SSE protocols due to differences in interval number and duration, intensity and duration of the SSE protocol, and differences in subject population. We are aware of only one other study that compared HIE (same as used in our study) to SSE (21). Larsen et al. (21) showed that HIE elicited a significantly higher EPOC than SSE (EE matched at 70% HR_{peak}) in men with metabolic syndrome. By contrast, we found no significant difference between HIE and SSE for EPOC, possibly due to a comparatively higher intensity in our SSE group (80% HR_{peak}). Results from other studies comparing HIE and SSE are inconsistent. Although one study reported that HIE produced a greater EPOC than SSE (20), most indicated that HIE and SSE produced EPOC of similar magnitude (24, 25, 33). Our finding that HIE and SSE produced net exercise EE and EPOC that were not different is consistent with findings that HIE and SSE result in similar fat loss after training (26, 28, 31).
SIE was poorly tolerated in our study, with three out of 13 subjects unable to complete the study due to nausea, lightheadedness and vomiting experienced during SIE. These side effects have been cited by others utilizing the all-out Wingate protocol (8, 32). In contrast, the HIE and SSE conditions were well tolerated. SIE protocols may not be suitable for many individuals, particularly clinical populations, due to the side effects and high levels of motivation necessary to complete this type of exercise. The HIE protocol we used has been shown to improve cardiorespiratory fitness and be safe and tolerable in a range of populations including those with congestive heart failure and middle-aged adults with metabolic syndrome (1, 27, 41).

Our study has several strengths. By including six 30-s sprint intervals in our SIE protocol, our net exercise EE and EPOC data were more directly applicable to the interpretation of SIE training studies that reported significant decreases in body fat (22) and waist and hip circumference (39), or increases in skeletal muscle fat oxidation enzymes (6). We measured VO$_2$ continuously throughout exercise and the entire 3-h postexercise period and therefore did not have to estimate VO$_2$ during non-measurement periods. We also included a non-exercise control trial rather than relying on pre-exercise resting VO$_2$ as a baseline from which to calculate EPOC differences between exercise conditions (21, 34).

A limitation of our study is that our exercise trials were not matched for total EE. However, we selected commonly used HIE and SIE protocols that, by their design, precluded matching for EE. Our 30-min SSE protocol was selected on the basis of previous studies comparing EPOC after interval and continuous exercise protocols (13, 20, 25, 34). Furthermore, using exercise protocols not matched for EE made our results applicable to training studies wherein SIE produced greater fat loss per unit EE (22, 35, 36). Our study included only young, recreationally active males. Thus applicability of our findings to other populations, such as females, older adults and patients with chronic diseases, is uncertain.
PRACTICAL APPLICATIONS

The results of our study provide practical insights for fitness professionals and coaches prescribing SSE, HIE or SIE for purposes of increasing the total energy cost of exercise. Although SIE elicits greater EPOC compared to traditional SSE exercise, total net EE (exercise + postexercise) is less than HIE and SSE. EPOC is unlikely to be the major contributor to fat loss and body composition changes previously observed following high-intensity interval exercise training. Finally, it is important to acknowledge that SIE may have limited utility due to the fact that this protocol was relatively poorly tolerated in this cohort of recreationally active young men.

REFERENCES


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FIGURE LEGENDS

Figure 1. Postexercise oxygen consumption (L·min⁻¹) for continuous steady-state exercise (SSE), high-intensity interval exercise (HIE) and sprint interval exercise (SIE). * p <0.001, indicates significant difference in all exercise conditions versus control. # p < 0.001, indicates significant difference SIE versus SSE. ‡ p <0.05, indicates significant difference SIE and HIE versus control.

Figure 2. Net oxygen consumed (L) during exercise and postexercise periods for continuous steady-state exercise (SSE), high-intensity interval exercise (HIE) and sprint interval exercise (SIE). * p < 0.05, indicates significantly greater overall net O₂ consumed in SSE compared to SIE. # p<0.001, indicates significantly greater net O₂ consumed during exercise in SSE and HIE compared to SIE. ‡ p<0.05, indicates significantly greater 3-h excess postexercise oxygen consumption (EPOC) in SIE compared to SSE.

Figure 3. Postexercise respiratory exchange ratio (RER) for control, continuous steady-state exercise (SSE), high-intensity interval exercise (HIE) and sprint interval exercise (SIE). * p <0.05, SIE significantly lower than control and SSE. # p<0.001, SIE significantly lower than all other conditions. ‡ p<0.05, indicates HIE significantly lower than control.

Figure 4. Total fat oxidation (g) during the 2nd and 3rd h after continuous steady-state exercise (SSE), high-intensity interval exercise (HIE) and sprint interval exercise (SIE), and time-matched control condition. * p < 0.05, indicates SIE significantly greater than control.
Figure 1.
Figure 2.
Figure 3.
Figure 4.