Excess Postexercise Oxygen Consumption After High-Intensity and Sprint Interval Exercise, and Continuous Steady-State Exercise

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Abstract

Tucker, WJ, Angadi, SS, and Gaesser, GA. Excess postexercise oxygen consumption after high-intensity and sprint interval exercise, and continuous steady-state exercise. *J Strength Cond Res* 30(11): 3090–3097, 2016–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 with continuous steady-state exercise (SSE) training. We compared EPOC after HIE, SIE, and SSE. Ten recreationally active men (age 24 ± 4 years) participated in this randomized crossover study. On separate days, subjects completed a resting control trial and 3 exercise conditions on a cycle ergometer: HIE (four 4-minute intervals at 95% peak heart rate (HR_{peak}), separated by 3 minutes of active recovery), SIE (six 30-second Wingate sprints, separated by 4 minutes of active recovery), and SSE (30 minutes at 80% of HR_{peak}). Oxygen consumption ($\dot{V}O_2$) was measured continuously during and for 3 hours after exercise. For all conditions, $\dot{V}O_2$ was higher than resting control only during the first hour postexercise. Although 3-hour EPOC and total net exercise energy expenditure (EE) after exercise were higher ($p = 0.01$) for SIE (22.0 ± 9.3 L; 110 ± 47 kcal) compared with 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 those for SIE (54.2 ± 12.0 L; 271 ± 60 kcal). Corresponding values for HIE were not significantly different from SSE or SIE. Excess postexercise oxygen consumption 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.

Key Words 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. High-intensity interval exercise generally consists of exercise bouts lasting approximately 1–4 minutes at ~90–95% of maximum heart rate, separated by 1–3 minutes of active recovery, whereas SIE typically includes 2–6 supramaximal efforts lasting ~30 seconds (i.e., Wingate cycling tests), with 3–4.5 minutes 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 (EE). Thus, HIE and SIE training may produce greater fat loss per unit of exercise EE than traditional, steady-state exercise (SSE) (22,35,36).

The reasons for the greater fat loss per unit of EE for interval exercise training are not well established, although greater excess postexercise oxygen consumption (EPOC) has been proposed (4,5,22). Compared with 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 because of 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 3 of the studies measuring EPOC for <120 minutes (7,8,34). Two of the studies reported 24-hour EPOC (13,32), but one of these (13) did not collect oxygen consumption ($\dot{V}O_2$) for 105 of the initial 180 minutes postexercise. Data from 24-hour whole-room calorimeter assessments indicate that all 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 2 and 5 bouts of SIE, with 5 of the studies using either two (7), three (34),

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or four (8,13,40) 30-second 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-second bouts during each training session over the final weeks of training. Because no studies have examined EPOC after SIE consisting of six 30-second sprint interval bouts, and no studies have compared EPOC after HIE, SIE, and SSE compared with 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-hour 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 3 separate exercise protocols (HIE, SIE, and SSE) and 1 control condition in random order with at least 72 hours 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 hours after each trial, $\dot{V}O_2$ and EE were measured to assess differences between conditions for net O_2 consumed and EE. Subjects were instructed to not exercise or consume caffeine or alcohol >48 hours before each visit. Trial order for the 4 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) were recruited by flyers posted around the Arizona State University campuses. Of the 13 subjects enrolled, 10 completed the study (mean ± *SD*: age 24 ± 4 years; height 171.6 ± 5.1 cm; weight 73.1 ± 8.2 kg; body mass index 24.8 ± 1.9 $kg \cdot m^{-2}$; percent body fat 13.5 ± 4.4%; $\dot{V}O_2$peak 45.9 ± 7.2 $ml \cdot min^{-1} \cdot kg^{-1}$). Three subjects were unable to complete the study because of 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 before participation.

Preliminary Visit

Before the first experimental test day, subjects reported to the laboratory to have anthropometrics and peak oxygen consumption ($\dot{V}O_2$peak) assessed. Body composition was assessed by Bod Pod (Cosmed, Concord, CA, USA) using air-displacement plethysmography. Standing height (in centimeters) was measured to within 0.1 cm against a wall-mounted stadiometer (Seca, Hamburg, Germany). Weight (in kilograms) was measured using an electronic scale, which is integrated with the Bod Pod.

$\dot{V}O_2$peak was determined using a ramp protocol ($30\ W \cdot min^{-1}$) on an electronically braked cycle ergometer (Viasprint 150P; Ergoline, Bitz, Germany). After a 5-minute warm-up at 50 W, the resistance increased continuously (1 W every 2 seconds) until subjects reached volitional exhaustion. Peak heart rate (HR_{peak}) 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 $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), and respiratory exchange ratio (RER). The Oxycon Mobile was calibrated as per manufacturer specifications before each baseline test and exercise/control visit. Heart rate was measured using a Polar Heart Rate monitor (Polar, Lake Success, NY, USA). $\dot{V}O_2$peak was defined as the average of the 2 highest consecutive 15-second 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 hours to avoid carryover effects. Subjects were instructed to not exercise or consume caffeine or alcohol for 48 hours before 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 hours on the day of each laboratory visit. Subjects recorded what they ate and were instructed to consume the same meal before every visit. Thereafter, subjects reported to the research laboratory at 1130 hours. Pulmonary ventilation and gas exchange were collected during seated rest from 1145 to 1200 hours (baseline), from 1200 to 1235 hours (during exercise or during seated rest for the control condition), and continuously for 3 hours postexercise (or for a time-matched 3 hours 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 $\dot{V}O_2$peak. During the 3-hour postexercise period, subjects were permitted to remove the facemask at fixed time points to periodically drink water.

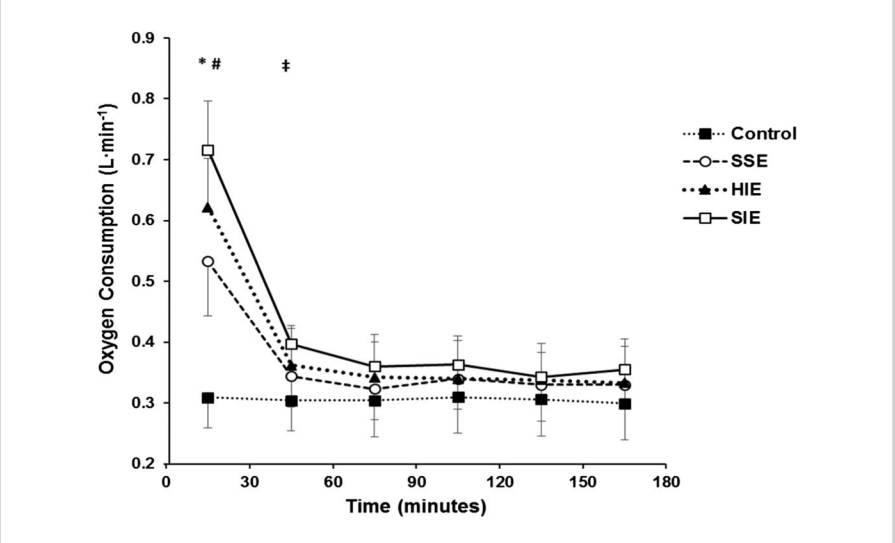

Figure 1. Postexercise oxygen consumption (in liters per minute) for continuous steady-state exercise (SSE), high-intensity interval exercise (HIE), and sprint interval exercise (SIE). *$p < 0.001$ indicates significant difference in all exercise conditions vs. control. #$p < 0.001$ indicates significant difference SIE vs. SSE. ‡$p \leq 0.05$ indicates significant difference SIE and HIE vs. control.

Exercise Sessions

Each of the 3 exercise conditions included a 5-minute warm-up at 50–60% HR_{peak}. SIE consisted of six 30-second sprints (Wingate) with the resistance set at 0.075 × subject body weight (in kilograms), followed by 4 minutes of active recovery (60% HR_{peak}) (23 minutes of total exercise time). A mechanically braked, calibrated cycle ergometer (Ergomedic 828E; Monark, Vansbro, Sweden) was used for SIE in accordance with recommendations for mechanically braked ergometers (3). High-intensity interval exercise (HIE) consisted of four 4-minute intervals at 95% HR_{peak}, with 3 minutes of active recovery (60% HR_{peak}) in between intervals (25 minutes of total exercise time). The SSE session consisted of 30 minutes of continuous exercise at 80% HR_{peak} (30 minutes of total exercise time). All HIE and SSE sessions were performed on the same electronically braked cycle ergometer that was used for the assessment of $\dot{V}O_2$peak.

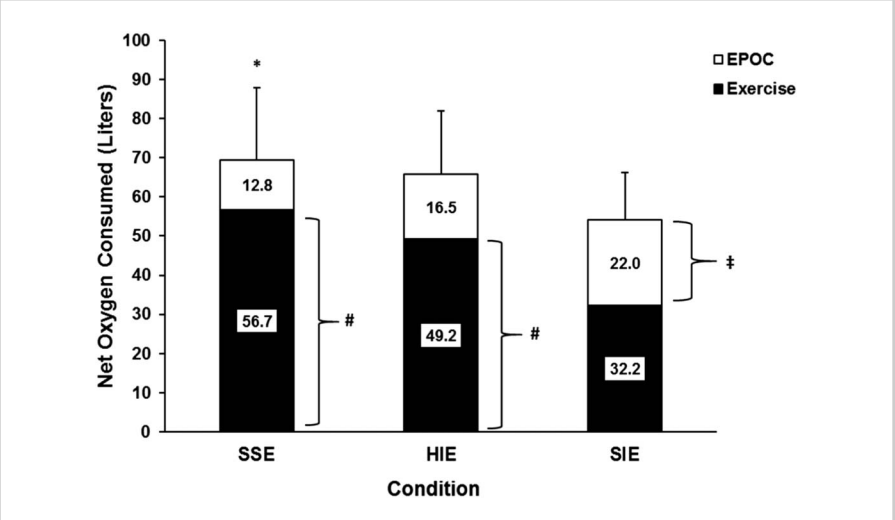

Figure 2. Net oxygen consumed (in liters) during exercise and postexercise periods for continuous steady-state exercise (SSE), high-intensity interval exercise (HIE), and sprint interval exercise (SIE). *$p \leq 0.05$ indicates significantly greater overall net O_2 consumed in SSE compared with SIE. #$p < 0.001$ indicates significantly greater net O_2 consumed during exercise in SSE and HIE compared with SIE. ‡$p \leq 0.05$ indicates significantly greater 3-hour excess postexercise oxygen consumption (EPOC) in SIE compared with SSE.

Calculations

Net O_2 consumed during and after (EPOC) each exercise condition was calculated by subtracting O_2 consumed during the time-matched control condition from the O_2 consumed during and after each of the exercise conditions. Because of the limitations of using RER to estimate substrate utilization during periods of non-steady-state $\dot{V}O_2$ (i.e., during HIE and SIE and immediately postexercise), energy expenditure (EE, in kcal) was calculated by assuming that 1 L of O_2 consumed corresponds to 5 kcal (33,40). However, because blood bicarbonate levels have been reported to return to resting levels within 30 minutes after cessation of high-intensity exercise (29), and arterial CO_2 partial pressure has been shown to be not different from resting control conditions from 60 to 120 minutes after HIE (24), we used $\dot{V}O_2$ and $\dot{V}CO_2$ data to estimate fat oxidation during the second and third hour postexercise (10):

Fat oxidation rate ($g \cdot min^{-1}$) = 1.67 ($\dot{V}O_2$ $L \cdot min^{-1}$) − 1.67 ($\dot{V}CO_2$ $L \cdot min^{-1}$).

Statistical Analyses

All data were analyzed using SPSS Software (SPSS 21.0; IBM Corporation, Armonk, NY, USA). Descriptive variables are presented as mean ± *SD*,

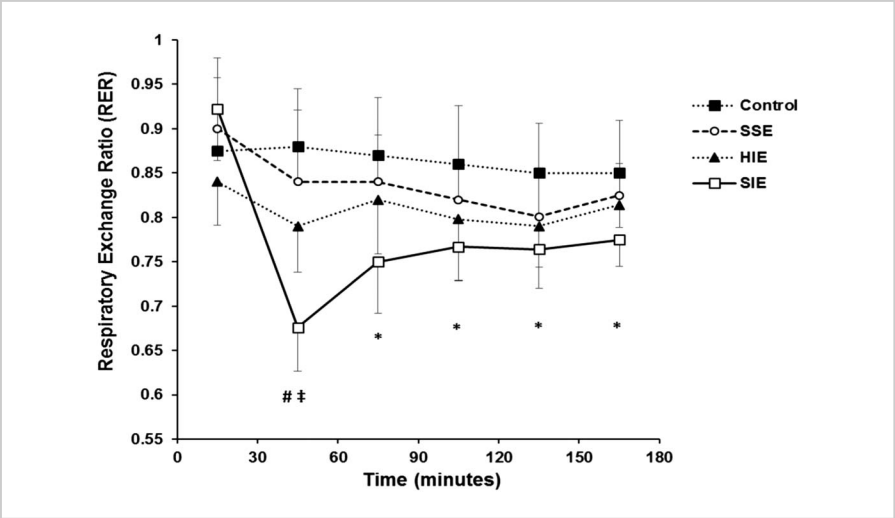

Figure 3. Postexercise respiratory exchange ratio (RER) for control, continuous steady-state exercise (SSE), high-intensity interval exercise (HIE), and sprint interval exercise (SIE). *$p \leq 0.05$ indicates that SIE is significantly lower than control and SSE. #$p < 0.001$ indicates that SIE is significantly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

and significance was set at $p \leq 0.05$. A 1-way repeated-measures analysis of variance (ANOVA) was used to compare all 4 conditions for baseline $\dot{V}O_2$, to compare exercise conditions for net O_2 consumed and net EE during exercise, and to compare total combined (exercise + postexercise) net O_2 consumed and EE. A 2-way repeated-measures ANOVA (condition × time) was used to determine differences between mean $\dot{V}O_2$, 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 mean values for significant interaction and protocol effects.

Results

Baseline Measurements

There were no differences between conditions for baseline $\dot{V}O_2$ ($L \cdot min^{-1}$) (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 $\dot{V}O_2$ during SSE (1.9 ± 0.4 $L \cdot min^{-1}$) and HIE (2.0 ± 0.4 $L \cdot min^{-1}$) were significantly greater than that of SIE (1.4 ± 0.3 $L \cdot min^{-1}$) ($p < 0.001$). Net O_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 $\dot{V}O_2$ ($p = 0.77$), net O_2 consumed ($p = 0.38$), or net EE during exercise ($p = 0.39$).

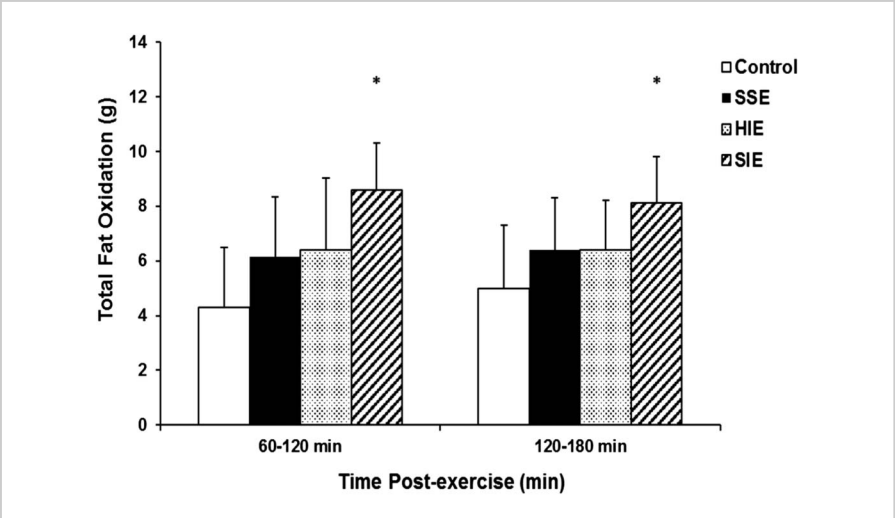

Figure 4. Total fat oxidation (in grams) during the second and third hour after continuous steady-state exercise (SSE), high-intensity interval exercise (HIE), sprint interval exercise (SIE), and time-matched control condition. *$p \leq 0.05$ indicates that SIE is significantly greater than control.

Postexercise $\dot{V}O_2$, EPOC, and Energy Expenditure

There was a significant condition ($p < 0.001$) and time ($p < 0.001$) effect for 3-hour postexercise $\dot{V}O_2$ and a time × condition interaction ($p < 0.001$) (Figure 1). Postexercise $\dot{V}O_2$ was significantly ($p < 0.001$) greater in all exercise conditions compared with CON during the initial 30 minutes following exercise (SSE: 0.53 ± 0.09 $L \cdot min^{-1}$; HIE: 0.62 ± 0.10 $L \cdot min^{-1}$; SIE: 0.72 ± 0.08 $L \cdot min^{-1}$ vs. CON: 0.31 ± 0.05 $L \cdot min^{-1}$). $\dot{V}O_2$ was also significantly higher in SIE compared with SSE during the initial 30 minutes postexercise ($p < 0.001$). In addition, $\dot{V}O_2$ after SIE and HIE were

significantly ($p \leq 0.05$) greater than CON between 30 and 60 minutes postexercise (SIE: 0.40 ± 0.05 L·min^{-1}; HIE: 0.36 ± 0.06 L·min^{-1} vs. CON: 0.30 ± 0.05 L·min^{-1}). Between 60 and 180 minutes postexercise, there were no significant differences in $\dot{V}O_2$ between conditions.

Cumulative 3-hour EPOC and net EE were greater ($p = 0.01$) after SIE (22.0 ± 9.3 L; 110 ± 47 kcal) compared with SSE (12.8 ± 8.5 L; 64 ± 43 kcal) (Figure 2). Cumulative 3-hour 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 first hour after exercise (SIE 77 ± 11 kcal, HIE 57 ± 11 kcal, and SSE 42 ± 11 kcal) (Figure 1).

Overall Net O_2 Consumed and Energy Expenditure (Exercise + Postexercise)

Combined exercise and postexercise net O_2 consumed and net EE were significantly higher for SSE (69.5 ± 18.4 L; 348 ± 92 kcal) compared with SIE (54.2 ± 12.0 L; 271 ± 60 kcal) ($p = 0.03$) (Figure 2), and there was a trend for higher net O_2 and net EE for HIE (65.7 ± 16.3 L; 329 ± 82 kcal) compared with SIE ($p = 0.07$).

Postexercise RER and Fat Oxidation

There was a significant condition ($p = 0.003$), time ($p < 0.001$), and time × condition interaction ($p < 0.001$) effect for postexercise RER. Post-hoc analyses revealed that RER was significantly lower during the 3-hour postexercise period following SIE compared with control ($p = 0.002$) and SSE ($p = 0.04$) (Figure 3). Respiratory exchange ratio was not significantly different between HIE and SIE during the 3-hour postexercise period ($p = 0.65$). Compared with CON, total fat oxidation after SIE was 4.3 g higher during the second hour (SIE: 8.6 ± 1.7 g, CON: 4.3 ± 2.2 g; $p = 0.001$) and 3.1 g higher during the third hour (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.

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 6-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 because of greater EPOC after interval exercise.

The EPOC we observed after SIE is higher than that reported in previous studies. This is likely because of our use of six 30-second sprint intervals. Williams et al. (40), whose SIE protocol included four 30-second sprints, reported a 3-hour EPOC of approximately 8 L and a net postexercise EE of 40.6 kcal. Our 3-hour EPOC and net postexercise EE were 22.0 L and 110 kcal, respectively. The 2.7-fold greater 3-hour 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 (because a full recovery is not reached during the 4-minute active recovery period after a supramaximal effort). Because the subjects for both studies had similar values for $\dot{V}O_2$peak (3.4 L·min^{-1}), EPOC comparisons are not confounded by differences in aerobic fitness. In addition, our EPOC and postexercise EE values after 30 minutes (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-second sprint intervals. Finally, our EPOC and postexercise EE values after 120 minutes (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-second sprints. Neither study reported $\dot{V}O_2$peak values of their subjects.

We measured EPOC for 3 hours postexercise based on studies that demonstrated that postexercise $\dot{V}O_2$ after SIE had returned to levels not significantly different from resting $\dot{V}O_2$ during the first 3 hours after exercise cessation (7,8,21,32,34). Although postexercise $\dot{V}O_2$ was not statistically significantly different from CON for any exercise condition after the first 60 minutes postexercise, the second and third hour contributed 30–35% to the total 3-hour EPOC, with approximately equal contribution from both the second and the third hour (Figure 1). Thus, we may have underestimated the magnitude of the EPOC by not continuing measurement beyond 3 hours. However, because the contribution to net EE during the third hour differed by ≤4 kcal between the 3 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-second sprint exercise bouts had returned to resting baseline within the first 4 hours after the SIE session and was not different for the remainder of the 24 hours spent in the whole-room calorimeter. Our results are consistent with their findings and show that even with 6-interval bouts, the duration of the EPOC is relatively short, with postexercise $\dot{V}O_2$ no longer significantly different from resting control after 60 minutes postexercise.

Sprint interval exercise was the only exercise condition that resulted in increased postexercise fat oxidation compared with the control trial. Increased fat oxidation is typically observed after 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-hour fat oxidation, and overall 24-hour 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). Sprint interval exercise acutely depresses appetite (4,9,40) but does not result in reduced energy intake within the initial 24 hours 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-second sprints, 24-hour O_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-second sprints. The discrepancy may be because of the inherent limitations of extrapolating discrete measurements using the trapezoidal method as compared with whole-room calorimetry. In the study by Hazell et al. (13), $\dot{V}O_2$ was not measured for 105 of the initial 180 minutes postexercise and thereafter was only measured at 6 and 24 hours 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 because of the extra sprint interval. In view of these findings, and those of others (8,34,40), the EPOC associated with SIE seems 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 through mechanisms other than EPOC. For example, resting energy expenditure (REE) has been reported to be elevated for 17–24 hours after a vigorous aerobic exercise session (15,23,37), in part, because of 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 hours postexercise.

It is difficult to compare our results with others who have used HIE and SSE protocols because of 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) with 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. In contrast, we found no significant difference between HIE and SSE for EPOC, possibly because of 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).

Sprint interval exercise was poorly tolerated in our study, with 3 of 13 subjects unable to complete the study because of nausea, lightheadedness, and vomiting experienced during SIE. These side effects have been cited by others using the all-out Wingate protocol (8,32). In contrast, the HIE and SSE conditions were well tolerated. The SIE protocols may not be suitable for many individuals, particularly clinical populations, because of 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-second 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 $\dot{V}O_2$ continuously throughout exercise and the entire 3-hour postexercise period and therefore did not have to estimate $\dot{V}O_2$ during non-measurement periods. We also included a non-exercise control trial rather than relying on pre-exercise resting $\dot{V}O_2$ 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-minute 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 male participants. Thus, applicability of our findings to other populations, such as female populations, 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 with traditional SSE exercise, total net EE (exercise + postexercise) is less than HIE and SSE. The 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 because of the fact that this protocol was relatively poorly tolerated in this cohort of recreationally active young men.

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