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Main title: Mechanistic and methodological perspectives on the impact of intense interval training on post-exercise metabolism

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Abstract

The post-exercise recovery period is associated with an elevated metabolism known as excess post-exercise oxygen consumption (EPOC). The relationship between exercise duration and EPOC magnitude is thought to be linear whereas the relationship between EPOC magnitude and exercise intensity is thought to be exponential. Accordingly, near-maximal and supramaximal protocols such as high-intensity interval training (HIIT) and sprint interval training (SIT) protocols have been hypothesized to produce greater EPOC magnitudes than submaximal moderate-intensity continuous training (MICT). This review updates previous reviews by focusing on the impact of HIIT and SIT on EPOC. Research to date suggests small differences in EPOC post-HIIT compared to MICT in the immediate (<1 h) recovery period, but greater EPOC values post-HIIT when examined over 24 h. Conversely, differences in EPOC post-SIT are more pronounced, as SIT tends to produce a larger EPOC vs. MICT at all time points. We discuss potential mechanisms that may drive the EPOC response to interval training (e.g. glycogen resynthesis, mitochondrial uncoupling, and protein turnover among others) and also consider the role of EPOC as one of the potential contributors to fat loss following HIIT/SIT interventions. Lastly, we highlight a number of methodological shortcomings related to the measurement of EPOC following HIIT and SIT.

Keywords: high-intensity interval training; sprint interval training, excess post-exercise oxygen consumption, EPOC, energy expenditure

Introduction

High-intensity interval training (HIIT) refers to protocols prescribing a "near-maximal" training stimulus where the target intensity is ~80-100% of maximal oxygen consumption ($\dot{V}O_{2max}$) or maximal heart rate (HR_{max}), interspersed with periods of low-intensity activity or rest [1]. A more intense version of HIIT is sprint interval training (SIT), involving repeated supramaximal exercise efforts where the absolute workload is greater than the workload required to elicit 100% $\dot{V}O_{2max}$, often performed as "all-out" efforts [2]. Both types of exercise result in similar aerobic adaptations to traditional moderate-intensity continuous training (MICT) [2-7], as well as anaerobic benefits unique to this modality [3,6,8]. Importantly, these training adaptations are achieved with a lower time-commitment and exercise volume than MICT [2,7,9] and despite this reduction in training volume, include improvements in body composition [4,10-12].

An important factor to consider is the magnitude and duration of the oxygen uptake ($\dot{V}O_2$) response that occurs after cessation of both interval and continuous training. During exercise, $\dot{V}O_2$ elevates to support the increased energy demand and remains elevated after exercise, resulting in a $\dot{V}O_2$ above baseline. This elevated $\dot{V}O_2$ is known as excess post-exercise oxygen consumption (EPOC) [13]. The EPOC, and thus elevated metabolic rate, leads to increases in energy expenditure that contributes to the total metabolic cost of the exercise session. The magnitude and duration of EPOC is variable, and many research efforts have focused on elucidating these factors to better understand the contribution of EPOC to total daily energy expenditure [14]. Currently, it is known that the magnitude of the EPOC response depends on both the intensity and duration of exercise, and it appears that exercise intensity has a greater impact [14-17]. Evidence suggests that while there is a linear relationship between EPOC and exercise duration at intensities greater than 50-60% of $\dot{V}O_{2max}$, the relationship between exercise intensity and EPOC magnitude is exponential [14,17], and a significant body of recent work has examined the effect of HIIT and SIT protocols on the EPOC response. This review will update previous reviews that have examined submaximal endurance exercise by focusing on the impact of near-maximal (HIIT) and supramaximal (SIT) interval training on EPOC, including an updated perspective on the potential mechanisms that drive the EPOC response. Further consideration will include the relevance of EPOC for fat loss, as this is a controversial topic. Lastly,

we will consider the methodological inconsistencies and limitations between studies examining the impact of HIIT and SIT on EPOC to date.

Effects of HIIT on Post-Exercise Metabolism

As seen with MICT [14,16], $\dot{V}O_2$ increases during a single HIIT session and remains elevated above resting levels post-exercise. The characteristics of studies examining EPOC following HIIT alone or compared to MICT are outlined in Tables 1 and 2 respectively, and demonstrate a consistent, albeit small, presence of EPOC. For instance, in the only study to compare two HIIT protocols to a no exercise control session without a comparison to MICT (Table 1), both protocols elicited an EPOC of ~2.8 L within 1 h post-exercise [18]. As seen in Table 2, most HIIT studies demonstrate a similar EPOC value with an average of ~3.3 L/h in the 0.5 – 3 h post-exercise period, and report significantly greater $\dot{V}O_2$ values compared to baseline levels [19-21] or a no-exercise control session [18,22-24] within 1 h post-exercise, though others did not report this information [25-27]. Beyond 1 h, the EPOC magnitude and duration varies greatly, with some reporting a sustained EPOC between 2-5 h [20,22,23] or 12 h [28] post-HIIT, while others do not report significantly elevated $\dot{V}O_2$ beyond 1 h [18,19,24]. Collectively, these results strongly support to ability of HIIT to increase $\dot{V}O_2$ compared to baseline levels within 1 h post-exercise. Beyond this time frame, EPOC is variable and post-exercise $\dot{V}O_2$ is not consistently elevated compared to baseline when examined on an hourly basis.

The ability of HIIT to produce a greater EPOC compared to MICT is less clear. As previously noted, exercise performed at near-maximal and supramaximal intensities appear to have a greater, more sustained EPOC than those of lower intensities [14,16]. However, the majority of available studies that have included direct between-protocol comparisons present equivocal findings, with some reporting significant differences in EPOC magnitude following HIIT compared to MICT in the hour immediately following exercise [20-23], and others not [19,24-26,29]. Of the eight studies that investigated the effects of HIIT and MICT on EPOC within a 0.5 – 3 h timeframe post-exercise (Table 2), the average total magnitude equalled to ~6.2 L and ~4.5 L following HIIT and MICT, respectively [19-26,29]. Three of the studies recorded a significantly elevated EPOC following HIIT vs. MICT [20,21,23], with a mean EPOC of ~3.6 L/h post HIIT and ~1.6 L/h post MICT. However, of the remaining five studies reporting no significant differences, mean EPOC was found to be ~3.1 L/h following HIIT, and ~2.7

L/h following MICT [19,24-27]. Upon extrapolation from all eight papers, the mean EPOC magnitude per hour was found to be ~3.3 L/h following HIIT and ~2.3 L/h following MICT, suggesting the differences between MICT and HIIT in the majority of studies are consistently small over a short post-exercise period. When the measurement period is extended to 5 h post-exercise, HIIT EPOC was more than double MICT EPOC suggesting the small difference can accumulate over time, but only one study has measured in this time frame at the present [22].

Interestingly, when measurements were made over 24 h after completion of either HIIT or MICT, similar total $\dot{V}O_2$ values and 24-h energy expenditure were achieved between protocols [30]. However, the exercise bouts were not isocaloric, and the $\dot{V}O_2$ consumed during MICT was significantly greater than that which was consumed for HIIT. As such, a similar total $\dot{V}O_2$ at 24 h indicates that HIIT induced a larger EPOC and minimized the disparity between $\dot{V}O_2$ levels slowly over several hours. This data suggests that extended post-exercise data collection periods may be necessary to identify changes in $\dot{V}O_2$ and energy expenditure induced by HIIT compared to MICT, where the small differences in $\dot{V}O_2$ between protocols accumulate over time to produce a greater EPOC magnitude. Accordingly, two studies measuring post-exercise metabolic rate in a room calorimeter identified that resting energy expenditure remained significantly elevated above control levels at 22 h [31] and 24 h [20] following a bout of HIIT work-matched with MICT, with greater post-exercise increases in HIIT (~103-160 kcal) compared to MICT (~64 kcal) [20,31], while another group reported elevated $\dot{V}O_2$ compared to both baseline and MICT at 21 h post-exercise [28]. Due to the brief post-exercise collection period utilized in most studies comparing the two protocols (≤ 5 h), it is possible that the EPOC response following HIIT is often underestimated, with most studies only capturing a small portion of the changes induced by HIIT when the measurement period is limited to the few hours post-exercise [31].

Additionally, longer post-exercise measurement periods may more accurately represent the true energetic cost of a HIIT session. A single bout of MICT is typically associated with a significantly greater net $\dot{V}O_2$ (and thus energy expenditure) compared with a single session of HIIT [2,4,6,30,32] and is largely attributed to the greater amount of time spent exercising in MICT. For example, a 75 kg individual with a relative $\dot{V}O_{2max}$ of 40 $mL \cdot kg^{-1} \cdot min^{-1}$ consumes ~117 L of O_2 (~585 kcal; all energy expenditure equivalents are presented with an assumed relationship of 5 kcal/L

of O_2 consumed) during a 60 min bout of MICT at 65% $\dot{V}O_{2max}$ compared to ~42 L of O_2 (~210 kcal) during a 20 min session of low-volume HIIT (10 x 1 min at 90% $\dot{V}O_{2max}$), including the oxygen cost of the recovery periods between intervals. In this scenario, ~75 L of O_2 must be consumed post-exercise (~375 kcal) in order for the session of HIIT to match the energy expended from MICT. While a value of this magnitude is certainly not attained in the few hours immediately post-exercise, similar total $\dot{V}O_2$ values are achieved by 22 h [30], suggesting the prolonged post-exercise period following HIIT is integral to the overall energy expenditure, as well as to decreasing the disparity in energy expenditure associated with the MICT itself.

Effects of SIT on Post-Exercise Metabolism

As with HIIT, there is strong evidence supporting the ability of SIT to elevate $\dot{V}O_2$ above control levels post-exercise. Studies examining EPOC following SIT compared to no exercise or MICT with various protocols over several time frames are outlined in Tables 1 and 3 and demonstrate a consistent presence of EPOC in the recovery period compared to baseline levels [25,33-36] or a no-exercise control day [24,32,37-45], with one exception [46]. Average total EPOC magnitude equates to ~6.8 L/h, ~1.7 L/h, and 2.6 L/h from 0.5 – 3 h, 9 h, and 24 h post-SIT, respectively (Table 3). This data suggests while there is a large elevation in EPOC immediately following SIT that diminishes over time, the EPOC response remains over an extended period, contributing potentially important increases in energy expenditure to that which is induced during exercise. Specifically, at the minimum magnitude of 2.6 L/h that is sustained at 24 h post-exercise (Table 3) over the course of the day, ~55 L of O_2 (~275 kcal) are consumed.

In contrast to HIIT, many studies demonstrate larger EPOC magnitudes after SIT directly compared to MICT [24,25,33-35,40,42,43], with few others reporting no differences [32,44]. On average, the total EPOC magnitude equates to ~9.1 L, or ~6.3 L/h in the 0.5 – 3 h following SIT, compared to ~5.9 L or ~3.8 L/h following MICT. Similar values are reported in studies examining EPOC following Wingate-based SIT vs. MICT or control (Tables 1 and 3), with an average total EPOC magnitude of ~17.7 L and a per h average of ~5.4 L across ten studies [24,32,34,36,38-41,44,46]. Again, these values are greater than that which is seen following MICT at ~8.4 L total and ~2.9 L/h, demonstrating the ability of SIT to induce EPOC above control levels or compared to MICT post-exercise. Seven of the studies

recorded a significantly elevated EPOC following SIT vs. MICT [24,25,33-35,40,42,43], with the mean EPOC totalling to ~6.74 L/h. However, of the remaining three studies reporting no significant differences compared to control [46], or MICT [32,44], mean EPOC was found to be ~2.7 L/h following SIT, and ~1.7 L/h following MICT. It is evident that majority of the data support the notion that the greater exercise intensity associated with supramaximal workloads lead to larger EPOC values.

Importantly, one group did not identify a measurable difference between SIT and MICT $\dot{V}O_2$ in any of the 30 min measures made over the course of a 24 h period where 24-h $\dot{V}O_2$ was similar following both types of exercise, totalling to ~498.0 L and ~500.2 L of O_2 for SIT and MICT, respectively [44]. As anticipated, total $\dot{V}O_2$ during the exercise session was significantly greater with MICT (~87.6 L) than SIT (~35.1 L), demonstrating that greater EPOC throughout the post-exercise period with SIT (~62.8 L) compared to MICT (~12.0 L) resulted in similar total uptakes at 24 h. Notably, due to the pattern of $\dot{V}O_2$ uptake with HIIT/SIT relative to MICT, most of the O_2 consumed with HIIT/SIT occurs in the recovery period, while majority of $\dot{V}O_2$ with MICT occurs during the exercise session itself [44]. More recently, others also reported equivalent 24 h energy expenditure following SIT and MICT despite a significantly lower energy expenditure during the bout of SIT compared to MICT, though a greater $\dot{V}O_2$ was observed post-SIT, and remained significantly elevated for up to 8 h [35]. These findings again suggest that small differences protracted over long durations are potentially important contributors to the total oxygen cost of the session, and may require longer post-exercise data collection periods to be identifiable.

Possible Mechanisms for HIIT and SIT-induced EPOC

The mechanisms underlying the elevated metabolism that occurs in the hour immediately following prolonged steady-state exercise (the "rapid" component) are well understood, and include oxygen replenishment in blood and muscle, adenosine triphosphate (ATP) and creatine phosphate resynthesis, lactate dissipation, and normalization of blood pH, body temperature, circulation, and ventilation [10,14,16]. Less well understood are the mechanisms responsible for the protracted post-exercise $\dot{V}O_2$ over the course of the day (the "slow" component). As with MICT, the mechanisms are likely attributable to the processes required for restoring physiological equilibrium post-exercise,

however they may be differentially affected following HIIT/SIT due to the intermittent nature, higher intensity, and greater metabolic stress associated with near-maximal and supramaximal protocols.

Increases in body temperature, circulation, and ventilation persisting beyond the rapid component may be a small contributing factor [16,37]. Compared to lower intensity exercise, higher intensity exercise stimulates larger increases in HR, ventilation, and body temperature, and may be more relevant to the EPOC response from HIIT/SIT compared to the MICT. Even so, these factors are likely responsible for a relatively small proportion of EPOC, as the oxygen cost of these processes is low [16]. Similarly, higher intensity exercise is associated with greater levels of glycogen depletion as observed with maximal sprinting [47,48], suggesting that greater EPOC magnitudes observed following longer exercise durations [39] may be attributable to greater increases in lactate formation, H^+ accumulation, lower pH levels, and glycogen resynthesis [47,48]. Furthermore, greater levels of glycogen depletion may be linked to greater post-exercise fat utilization to sustain energetic demands while glycogen resynthesis occurs [49], though substrate use in the recovery period is not the focus of many EPOC studies. Those who have examined substrate use reported greater fat utilization in the recovery period following SIT compared to MICT [40] and following both HIIT/SIT compared to MICT with no differences between interval training protocols [24], while others have found no difference between HIIT/SIT and MICT [20,22,23,27,46]. Recruitment of inefficient fast-twitch fibres may also contribute to EPOC due to the high levels of type II fibre recruitment in SIT, subsequently leading to greater fatigue and metabolic disturbances associated with the increased ATP and/or O_2 cost of exercise [50].

Others have suggested catecholamine-induced increases in metabolism and lipolysis to be potential factors involved in EPOC [44]. The lipolytic effects of epinephrine are well understood, and both HIIT [31] and SIT [32] have demonstrated increases in the concentration of circulating epinephrine and norepinephrine to a greater extent than from MICT. However, despite greater increases in post-exercise catecholamine concentrations, significant increases in free fatty acid availability and oxidation were not found compared to MICT suggesting that enhanced lipolysis may not play a large role in HIIT/SIT EPOC [32].

Additional insight in to the molecular mechanisms of EPOC after HIIT/SIT may be found at the level of the mitochondria. Due to the inherent inefficiencies in mitochondrial respiration,

uncoupling of $\dot{V}O_2$ from ATP production due to proton leakage results in dissipation of some of the available free energy as heat [51]. In rats, protein leak across the inner mitochondrial membrane contributes to ~50% of resting respiration rate in perfused skeletal muscle and accounts for an estimated 25% of whole-body basal metabolic rate [52]. As such, it is plausible that enhanced respiratory uncoupling may contribute to the slow component of EPOC, particularly after high-intensity exercise protocols that elicit profound changes in intracellular metabolites/ions (e.g. ADP, Pi, Ca^{2+}) [53,54] involved in the control of mitochondrial respiration [55]. However, studies examining the impact of acute high-intensity exercise on respiratory uncoupling have produced contradictory findings of increased [56], decreased [57-59], or unchanged [31] uncoupled respiration in skeletal muscle mitochondria, and the limited evidence presently available makes it difficult to support or refute the contribution of altered mitochondrial function to prolonged EPOC.

An upregulation of hypoxia inducible factor 1 alpha subunit (HIF-1α) has been demonstrated in human skeletal muscle during recovery from a single exercise bout [60,61]. As a master regulator of the cellular homeostatic response to hypoxia, this transcription factor facilitates increased O_2 delivery to tissue [60]. Individuals with an autosomal recessive disorder (Chuvash polycythemia) expressing increased HIF-1α accumulate more lactate compared to control patients [60]. As lactate removal is a well-known mechanism underlying the EPOC response, it is plausible that an upregulation of HIF-1α following HIIT/SIT may influence EPOC. Alternatively HIF-1α may activate glycolytic pathways resulting in reduced mitochondrial function and $\dot{V}O_2$ [60]. Thus, reduced $\dot{V}O_2$ during HIIT/SIT as a result of increased HIF-1α may potentially augment the EPOC response. Presently, we are unaware of a study that has concurrently measured HIF-1 activation and EPOC following acute exercise, thereby making it difficult to further speculate on the relationship between these two variables.

Finally, increases in muscle damage and protein turnover may contribute to the HIIT/SIT EPOC response. Elevated muscle protein synthesis has been demonstrated in recovery from both MICT and HIIT/SIT in older men [62], and as the energy cost associated with protein synthesis is significant [63] it is logical that an increased rate may account for a portion of the prolonged HIIT/SIT EPOC response. Indeed, one group reported greater EPOC following a bout of resistance training compared to a bout of MICT and suggested the greater EPOC was a consequence of the energy requirement associated with protein repair/synthesis [28]. Further research investigating both muscle

protein breakdown and synthesis is required to determine if they account for a significant part of the prolonged HIIT/SIT-induced EPOC.

Relevance of EPOC for Fat Loss

Recent work has identified small to moderate improvements in body composition following HIIT/SIT that are similar to and, in some cases, greater than that which is seen following MICT. Indeed, a recent meta-analysis indicated that while MICT, HIIT and SIT are similarly effective at reducing body fat percentage, interval training results in superior reductions in total absolute fat mass by ~28.5% compared to MICT [12]. Others have reported equal effectiveness at reducing total body fat percentage and absolute fat mass, though only when protocols are matched for total work [64]. When protocols are not matched, MICT tends to produce greater reductions in total body fat percentage compared to HIIT and SIT as a whole, though Wingate-based SIT (performed in an "all-out" manner) on its own produces body fat reductions comparable to MICT in less time [64]. Nevertheless, the ability of HIIT/SIT to reduce fat mass irrespective of MICT is well-supported, and these improvements in body composition have been demonstrated in several populations, including recreationally active women [11,65] and men [65], untrained women [66], inactive overweight women [67-70], and overweight or obese men [70-74].

The mechanisms underlying the observed fat loss are not well understood, though it has been proposed that the EPOC induced by HIIT/SIT is a potential contributor [10,30,38,44]. Due to the intense nature of the protocol, it is often hypothesized that HIIT/SIT elevates EPOC to a greater extent than MICT, and over the course of the day may result in an energy deficit not accounted for in the estimated caloric expenditure associated with the session itself [38]. However, as discussed in the present review, greater EPOC values have been consistently documented following SIT only [24,25,33,34,42,43,75], and equivocally following HIIT [20-23], compared to MICT, with the resultant increases in energy expenditure negligible in the immediate hour post-exercise. Furthermore, these increases in energy expenditure are similar to [30], and sometimes greater than [20] that which is induced by MICT at 24 h. Thus, an increased EPOC is unlikely to be the sole contributor to the similar reductions in body fat percentage demonstrated with interval training as has been purported in some literature. Rather, it is more likely that the EPOC response is a small part in the overall fat loss process, and contributes to

a combination of exercise-related changes to energy balance over repeated sessions of interval training that results in the HIIT/SIT-induced fat loss. For example, evidence of appetite suppression following HIIT and SIT has been reported in several studies [32,76-82], and it appears that high-intensity exercise can induce suppression to a greater extent than MICT [76,83]. Together, the combination of a negative energy balance plus decreased appetite following HIIT/SIT may partially account for the observed improvements in body composition. Other potential mechanisms may include increased fat oxidation following HIIT and SIT, as these protocols result in significant glycogen depletion [47,48] and increase plasma glycerol concentrations after exercise [66], suggesting lipids as an important energy source to meet energetic demands while glycogen resynthesis occurs [49]. Additionally, HIIT and SIT may stimulate significant increases in circulating catecholamines and other hormones promoting fat oxidation, such as adiponectin and interleukin-6 (IL-6) [32,76,84].

Methodological Inconsistencies and Limitations

Although trends in the EPOC response following HIIT/SIT have been identified in this review, there are several methodological inconsistencies within the field that may underlie the conflicting results surrounding the magnitude and duration of EPOC, thus limiting the inferences we can draw from results. A significant source of inconsistency is the heterogeneity between exercise protocols used to examine characteristics of the EPOC response. Unlike MICT, which involves a submaximal bout of exercise at an easily verified intensity that can be standardized across individuals, HIIT and SIT are infinitely variable in terms of the number, intensity, and duration of the work bouts, as well as the work-to-rest ratio [85]. Large variability exists within the thirteen studies examining EPOC following HIIT (Tables 1 and 2), as three studies utilized the ten-by-one protocol (10 x 1 min at ~90% HR_{max} with 1 min rest or recovery periods), and the remaining ten involved other variations of near-maximal exercise bouts separated with varying amounts of rest or recovery, though notably at similar intensities. With respect to SIT, the most consistently used protocol in the literature is the traditional Wingate protocol (30-s bouts of "all-out" exercise separated with 4 min of rest), however, comparison across even these studies is challenging, as the number of work bouts vary between two to six. The variability within protocols is important as it has been shown that the volume of HIIT/SIT affects the EPOC magnitude and duration [19], yet the impact of manipulating other SIT variables on EPOC

remains inconclusive. Thus, it is difficult to compare results across studies with wide-ranging differences. Relatedly, several groups work-matched the HIIT/SIT and MICT protocols, while others did not. Of the studies examining HIIT vs. MICT, eight groups made the trials isocaloric, and of those examining SIT vs. MICT, only one included isocaloric trials, further contributing to the heterogeneity between protocols. Lastly, studies involving Wingate-based SIT have utilized a range of external loads (6.5 – 10% of body mass) [32,38,44] which may have confounded comparisons of the EPOC magnitude between studies involving Wingate-based SIT. While preliminary evidence [86] suggests no difference in the aerobic contribution to a single Wingate effort between the intended Wingate resistance and higher values (i.e. 9.5%), future research is necessary to determine if higher or lower resistance values generate different effects on EPOC.

A collective feature of Wingate-based SIT studies is the "all-out" nature of the exercise bout, however the degree to which this effort is monitored over the duration of the sprint is often inadequately described or not reported at all. Currently, there is no consensus as to what constitutes an "all-out" effort, and it is unlikely that the exercise stimulus is consistent across all individuals due to the highly subjective nature of working "as hard as possible". This is particularly important as the effort put forth during a sprint bout will directly impact the subsequent $\dot{V}O_2$. As such, studies that seek to characterize the EPOC response following various SIT protocols may benefit from recording the power/speed output of the entire duration of a SIT bout. Alternatively, the use of a fatigue index criteria that must be met in order for a bout to be confirmed as "all-out" may be utilized, as either method may provide some indication that a maximal effort was exerted throughout. The use of measureable criteria to characterize the effort in a sprint bout will assist in the standardization of effort across various SIT protocols, and mitigate concerns about conclusions that can be drawn from the resultant findings.

Further confounding this issue is the active or passive nature of the HIIT/SIT recovery, as some studies do not sufficiently standardize the periods in between work bouts, allowing some participants complete rest while others perform active recovery. With regards to traditional Wingate-based SIT, these periods account for >85% of the protocol duration, and differences between active or passive recovery may impact the return of $\dot{V}O_2$ to resting values, as directly evidenced by recent work using a Wingate-based SIT protocol [87]. The degree to which the various protocols, including

rest/recovery periods, are administered as planned or standardized across individuals is not often reported and as such, warrants the question of whether the EPOC observed is due to methodological inconsistencies with measurement, or due to the protocol administration itself.

Other concerns exist with regards to the nature of $\dot{V}O_2$ measurement over the recovery period. Specifically, some investigations have employed continuous $\dot{V}O_2$ monitoring, while others have used intermittent monitoring at discrete time points, leading to discrepancies regarding when $\dot{V}O_2$ returns to resting levels. In terms of the determination of baseline values, some studies have used resting $\dot{V}O_2$ levels taken on a separate control day when no exercise was performed, whereas others use pre-exercise values where participants rest quietly for a period of time, and the $\dot{V}O_2$ in the final few minutes is used to represent baseline values. It is possible that the latter method may lead to a falsely high estimate of baseline $\dot{V}O_2$ due to the effect of anticipation or nerves regarding the upcoming exercise session, and can lead to an underestimation of the EPOC response [16]. Furthermore, there is a circadian effect on $\dot{V}O_2$ and it is possible that EPOC magnitudes may be influenced by these natural fluctuations throughout the day rather than an effect of the exercise bout. However, data from studies conducted in our lab demonstrates a negligible difference between pre-exercise $\dot{V}O_2$ levels and a no-exercise control day (0.026 L/min difference, or ~7 kcal across a 30 min measure) [44], suggesting the differences between study designs is a small methodological limitation.

It has been suggested that differences in EPOC magnitude and duration may arise when varying modalities are used. For instance, running is a more whole-body exercise involving greater muscle mass recruitment for a given submaximal workload, and this exercise modality may elevate $\dot{V}O_2$ to a greater extent than cycling [88,89]. However, we have previously demonstrated similar EPOC values following a running and cycling SIT session [41] suggesting mode has little effect on EPOC, though the post-exercise data collection period was brief (2 h) and cannot be generalized to the late post-exercise period. Differences in fitness level may also contribute to EPOC, though the evidence is equivocal. Some have reported an inverse correlation between fitness and EPOC [25], suggesting that trained individuals demonstrate a more rapid return to resting $\dot{V}O_2$ levels [16], though others have suggested training status has a negligible influence on EPOC [14]. For example, 24 h resting energy expenditure post-HIIT and post-MICT was examined at baseline and following 16 weeks of aerobic

training (40 min cycling at ~80% maximal heart rate, 3 times per week) and no significant differences from pre- to post-training were found [31].

Lastly, an additional concern is the conditions of the post-experimental period, as these conditions are not always well-controlled or reported, particularly with regards to participants' activity and nutrition. To prevent unrelated increases in $\dot{V}O_2$ over the recovery period, it is necessary that activity levels are strictly controlled and mimic baseline activity conditions. Many studies in the current review controlled the post-exercise period to mimic baseline activity conditions, as well as described the measures taken to do so (providing transportation to the laboratory, prohibiting unnecessary walking, etc.), though some did not, which may contribute to the equivocal results between studies. With regards to nutrition, it is evident that the thermic effect of feeding elevates post-exercise $\dot{V}O_2$ and may result in overestimation of the EPOC response [14]. We previously measured $\dot{V}O_2$ for 30 min immediately following a meal, and reported increases in $\dot{V}O_2$ from ~0.242 L/min pre-meal to ~0.354 L/min post-meal demonstrating a 3.36 L increase over 30 min due to the thermic effect of feeding on $\dot{V}O_2$ [44]. Thus, caution should be used when interpreting EPOC values where a post-exercise meal was consumed. In the present review, all of the studies examining EPOC at 12 – 24 h either provided standardized meals at identical time points, instructed participants to replicate meals from food logs, or held the macronutrient content of each meal constant to account for the thermic effect of feeding on the measurement of EPOC. Of the groups examining EPOC within a shorter time frame, several provided standardized meals at identical time points, only one group did not describe the content of the participants' nutrient intake, and the remainder did not provide a meal during the post-exercise measurement period. In contrast, a pre-exercise meal is less important, and performing MICT or SIT in the fed or fasted state does not affect EPOC or energy expenditure in a 3-h recovery period [90,91]. Lastly, limited sample sizes may contribute to some of the variance in findings. Of the 28 studies included in Tables 1-3, the number of participants per study ranged from 6 to 33 with a mean of 11, and only 9 studies had a sample size greater than 10. As the ability to make meaningful conclusions on the effect of the treatment depends on the power of the study (and thus, sample size) [92], it is possible that many studies are underpowered to detect a difference in the EPOC response following HIIT/SIT vs. MICT.

Conclusions

In summary, both HIIT and SIT increase $\dot{V}O_2$ levels up to 1 h post-exercise, resulting in a significant EPOC compared to pre-exercise levels or a no-exercise control day. Beyond this time frame, $\dot{V}O_2$ is greatly variable and decreases towards baseline values following both types of interval training. Compared to MICT, the differences in $\dot{V}O_2$ post-HIIT are inconsistent, with some studies reporting greater EPOC compared to MICT within 5 h post-exercise [20-23] and others reporting no differences between the two protocols within the same time-period [19,24-27]. Furthermore, some studies suggest that HIIT results in elevated $\dot{V}O_2$ compared to MICT and baseline at ~21 h post-exercise [28,31], while others have reported a return to baseline $\dot{V}O_2$ levels within 1 h post-exercise [18,19,24,32]. In contrast, the differences in $\dot{V}O_2$ post-SIT compared to MICT are more pronounced, and SIT tends to produce greater EPOC magnitudes vs. MICT at all time points [24,25,33,34,42,43,75], with few exceptions [32,44]. These data support the established trend that SIT produces greater EPOC magnitudes than MICT protocols. Conversely, the suggestion that HIIT produces greater EPOC magnitudes vs. MICT does not appear to be supported by current evidence in direct comparisons when short post-exercise data collection periods are utilized. However, similar 24 h energy expenditure has been found following MICT and HIIT [30], suggesting HIIT may produce a larger EPOC magnitude over an extended period of time. This larger EPOC magnitude over an extended period is also seen in response to SIT suggesting longer post-exercise measurement periods are necessary to more accurately assess EPOC.

Potential mechanisms underlying the prolonged EPOC component include protracted increases in ventilation, circulation, body temperature, and catecholamines, a greater cost of glycogen resynthesis associated with maximal sprint efforts, recruitment of inefficient fast-twitch fibres, enhanced respiratory uncoupling in skeletal muscle mitochondria, upregulation of HIF-1α, and increased rates of muscle protein synthesis. While some of the mechanisms underlying the rapid component are well defined and are similar between both MICT and HIIT/SIT, some may be specific to interval training, and more work is required to elucidate the factors underlying HIIT and SIT-induced EPOC. Lastly, much of the conflicting results regarding the EPOC response following HIIT/SIT may be attributed to the heterogeneity in methodologies that exist between studies. These inconsistencies include, but are not limited to, the wide-ranging differences between protocols used to examine the EPOC response, the lack of standardization of such protocols and how well they are described to enable replication, the measurement of the EPOC response, length of the post-exercise O_2 collection period, and control of participants' post-exercise activity and feeding.

Perspectives

This review updates previous reviews [14,16] on the characteristics of EPOC by focusing on the impact of HIIT/SIT protocols, which is currently lacking in the literature. Evidence in direct comparisons demonstrate a consistent, but small EPOC in the immediate post-exercise period that is sometimes greater than that induced by MICT post-HIIT, and more commonly greater post-SIT. Similar total $\dot{V}O_2$ values are observed at 24 h between MICT, HIIT, and SIT, suggesting that the EPOC associated with a bout of interval training is understated when evaluated only in the immediate hours post-exercise, as small magnitudes on a per-hour basis accumulate over 24 h. This elevated post-exercise metabolism over the course of the day increases the energy expenditure derived from a single exercise session but is not likely to solely explain the fat losses associated with interval training. Like MICT, the mechanisms underlying EPOC are likely attributable to processes required for restoring physiological equilibrium following exercise but may be differentially affected following HIIT/SIT due to the intermittent nature, higher intensity, and greater metabolic stress associated with such protocols. Variability in findings may be attributable to the heterogeneity between protocols used and technical aspects related to EPOC measurement.

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Table 1. Characteristics of studies examining EPOC following HIIT or SIT alone.

Reference [#]	Age (y)	*n* (♀)	$\dot{V}O_{2peak/max}$ (mL·kg^{-1}·min^{-1})	Protocol	Collection period	Total EPOC (L of O_2)	EPOC (L/h)
HIIT							
Kelly et al. 2013 [18]	21.0	9 (0)	44.0	C: 10 × 1 min @~85% $\dot{V}O_{2max}$^^^ 1 min recovery	1 h; CM	~2.8 [a]	~2.8
				C: 10 × 4 min @~85% $\dot{V}O_{2max}$^^^ 2 min recovery		~2.9 [a]	~2.9
Mean±SD	-	-	-			***~2.9 ±0.1***	***~2.9 ±0.1***
Kelly et al. 2013 [18]	21.0	9 (0)	44.0	C: 10 × 1 min @~85% $\dot{V}O_{2max}$^^^ 1 min recovery	11 h; 1 h CM 9.75 h I	~8.5[a]	~0.8
				C: 10 × 4 min @~85% $\dot{V}O_{2max}$^^^ 2 min recovery		~10.6[a]	~1.0
Mean±SD	-	-	-			***9.5 ±1.4***	***0.9 ±0.1***
SIT							
Bahr et al. 1992 [37]	23.0	6 (0)	49.9	C: 1 × 2 min @108% $\dot{V}O_{2max}$ 3 min recovery	1 h [c]; CM	5.6	5.6
				C: 2 × 2 min @108% $\dot{V}O_{2max}$ 3 min recovery		6.7	6.7
				C: 3 × 2 min @108% $\dot{V}O_{2max}$ 3 min recovery		7.8	7.8
Burns et al. 2012 [46] [b]	17.2	3 (7)	NR	C: 2 × 30 sec "all out" bouts (7.5% BM) 4 min recovery	1.5 h; CM	4.8	3.2
Chan & Burns 2013 [38] [b]	23.3	10 (0)	NR	C: 4 × 30 sec "all out" bouts (6.5% BM) 4.5 min recovery	2 h; CM	13.6	6.8

Islam et al. 2017 [39] [b]	23.3	9 (0)	48.9	R: 4 × 30 sec "all-out" bouts 4 min recovery	3 h; CM	12.9	4.3
				R: 8 x 15 sec "all-out" bouts 2 min recovery		13.8	4.6
				R: 24 x 5 sec "all-out" bouts 40 s recovery		10.0	3.3
Townsend et al. 2014 [41] [b]	24.4	8 (0)	NR	C: 4 × 30 sec "all-out" bouts (7.5% BM) 4 min recovery	2 h; CM	9.0	4.5
				R: 4 × 30 sec "all-out" bouts 4 min recovery		13.0	6.5
	22.4	0 (8)	NR	C: 4 × 30 sec "all-out" bouts (7.5% BM) 4 min recovery		12.0	6.0
				R: 4 × 30 sec "all-out" bouts 4 min recovery		9.0	4.5
Mean±SD	*22.3 ±2.6*	*7±3 (8±1)*	*49.4 ±0.7*			***10.6 ±3.6***	***5.3 ±1.5***
Sevits et al. 2013 [36] [b]	26	12 (0)	53.0	C: 5 × 30 sec "all-out" bouts (7.5% BM) 4 min recovery	24 h; CM	45.2	1.9
Tsuji et al. 2017 [45]	23	10 (0)	52.1	R: 6-7 × 20 sec @170% $\dot{V}O_{2max}$ 10 s recovery	21 h; CM	8.0	0.4
Mean±SD	*24.5 ±2.1*	*11 ±1*	*52.2 ±0.6*			***26.6 ±26.3***	***1.1 ±1.1***

Note: [a] = data presented as energy expenditure (kJ) and converted to L of O_2; BMI: body mass index; [b] = Wingate-based SIT study; [c] = EPOC magnitudes reported for both 1 h and 14 h post-exercise; C: cycling; CM: continuous measure; HIIT: high-intensity interval training; I: intermittently; NR: not reported; R: running; SIT: sprint interval training; $\dot{V}O_{2max}$: maximal oxygen uptake. ^^^ - 90% HR_{max}.

Table 2. Characteristics of studies examining EPOC following HIIT vs. MICT.

Reference [#]	Age (y)	*n* (♀)	$\dot{V}O_{2peak/max}$ (mL·kg^{-1}·min^{-1})	HIIT protocol	MICT protocol	Collection period	Total EPOC magnitude (L of O_2)		EPOC magnitude (L/h)	
							HIIT	MICT	HIIT	MICT
0.5 – 3 h post exercise										
Larsen et al. 2014 [19]	56.7	7 (0)	33.2	R: 1 × 4 min bouts ~85% $\dot{V}O_{2max}$^^^ 3 min AR periods (55% $\dot{V}O_{2max}$^)	47 min @ 55% $\dot{V}O_{2max}$^,†	~30 min[a]; CM	1.4	1.4	2.8	2.8
	56.7	7 (0)	33.2	R: 4 × 4 min bouts ~85% $\dot{V}O_{2max}$^^^ 3 min AR periods (55% $\dot{V}O_{2max}$^)	47 min @ 55% $\dot{V}O_{2max}$^,†	~1 h[a]; CM	2.9		2.9	
Malatesta et al. 2009 [23]	24.2	12 (0)	52.3	C: 1 min bouts ~85% $\dot{V}O_{2max}$^^^ 1 min recovery periods (WM with MICT)	1 h @ ~45% $\dot{V}O_{2max}$ †	3h; CM	4.9	0.5	1.6	0.2
Matsuo et al. 2012 [25]	24.0	10 (0)	52.0	C: 3 × 3 min bouts ~80-90% $\dot{V}O_{2max}$ 2 min AR periods (50% $\dot{V}O_{2max}$)	40 min @ 65% $\dot{V}O_{2max}$	3 h; I (6 x 10 min)	4.5	2.9	1.5	1.0
McGarvey et al. 2012 [26]	30.0	12 (0)	49.0	C: 7 x 2 min bouts ~90% $\dot{V}O_{2max}$ 3 min AR periods (30% $\dot{V}O_{2max}$)	30 min @ 65% $\dot{V}O_{2max}$†	~2 h[b]; CM	7.6	7.0	3.8	3.5
Treuth et al. 1996 [20]	28.1	0 (8)	39.3	C: 15 x 2 min bouts ~100% $\dot{V}O_{2max}$ 2 min recovery periods	60 min @ ~50% $\dot{V}O_{2max}$	2 h; CM	10.3[c]	7.5[c]	5.2	3.8

Note: [a] = EPOC measured until baseline oxygen consumption was established; AR: active recovery; [b] = EPOC measured until 5 min average of recovery $\dot{V}O_2$ equaled to baseline; BMI: body mass index; [c] = data presented as energy expenditure (kJ) and converted to L of O_2; C: cycling; CM:

Tucker et al. 2016 [24]	24.0	10 (0)	45.9	C: 4 × 4 min bouts @~85% $\dot{V}O_{2max}$^^^ 3 min AR periods (60% HR_{peak})	30 min @ 70% $\dot{V}O_{2max}$^^	3 h; CM	16.5	12.8	5.5	4.3
Warren et al. 2009 [27]	27.3	6 (1)	45.7	C: 1 min bouts ~85%$\dot{V}O_{2max}$ 2 min recovery periods (30% $\dot{V}O_{2max}$)	90 min @ ~50% $\dot{V}O_{2max}$†	1.5 h; I	3.7	2.8	2.5	1.9
Wingfield et al. 2015 [21]	24.6	0 (20)	NM	R: 10 x 1 min bouts ~85%$\dot{V}O_{2max}$ 1 min recovery periods	30 min @ ~45-55% HRR	1 h; I	4.2[d]	0.8[d]	4.2	0.8
Mean±SD	*29.9± 11.1*	*9.5± 2.5 (9.7± 9.6)*	*45.3± 7.0*	-	-	-	***6.2 ±4.7***	***4.5 ±4.3***	***3.3 ±1.4***	***2.3 ±1.5***
5 h post-exercise										
Karstoft et al. 2016 [22]	60.3	7 (3)	24.4	IW: 10 x 3 min bouts ~89% $\dot{V}O_{2peak}$	1 h CW @ ~73% $\dot{V}O_{2peak}$†	5 h; I (4 x 30 min)	8.4	3.7	2.1	0.9
21 – 24 h post-exercise										
Greer et al. 2015 [28]	22.0	10 (0)	34.5	C: 15-18 × 30 s bouts ~90% $\dot{V}O_{2max}$ 2-3 min recovery period[e]	43 min @ ~40% $\dot{V}O_{2max}$†	21 h; 30 min CM @ 12 & 21 h	82.8[f]	3.6[f]	3.5	0.2
Hunter et al. 2017 [31]	32.6	0 (33)	25.3	C: 144 sec bouts 84% $\dot{V}O_{2max}$ 103 s recovery periods (WM with MICT)	60 min @ 50% $\dot{V}O_{2peak}$†	22 h; CM	20.6[g]	12.8[g]	0.9	0.6

Skelly et al. 2014 [30]	21.0	9 (0)	46.0	C: 10 × 1 min bouts ~85% $\dot{V}O_{2max}$ ^^^ 1 min active recovery periods (50 W)	50 min @ 55% $\dot{V}O_{2max}$ ^	24 h; I (7 x 30 min)	27.2	19.8	1.1	0.8
Mean±SD	*25.2 ±6.4*	*9.5 ±0.7 (-)*	*35.3 ±10.4*	-	-	-	***43.5 ±34.2***	***12.1 ±8.1***	***1.8 ±1.4***	***0.5 ±0.3***

continuous measure; CW: continuous walking; [d] = data presented as kcal/d, we extrapolated; [e] = No set # of intervals. Session stopped when EE matched the MICT session; [f] = data presented as 30 min measures at 12 and 21 h post-exercise, we extrapolated; [g] = data presented as resting energy expenditure over 24 h extrapolated from gas exchange data measured over 1 h; HIIT: high-intensity interval training; I: intermittently; IW: intermittent walking; NM: not measured; NR: not reported; MICT: moderate-intensity continuous training; R: running; $\dot{V}O_{2max}$: maximal oxygen uptake; ^ – 70% HR_{max}; ^^ – 80% HR_{max}; ^^^ – 90% $HR_{max;}$ ^^^^ – 95% HRR; † – isocaloric exercise conditions.

Table 3. Characteristics of studies examining EPOC following SIT vs. MICT.

Reference [#]	Age (y)	*n* (♀)	$\dot{V}O_{2peak/max}$ (mL·kg^{-1}·min^{-1})	SIT protocol	MICT protocol	Collection period	EPOC magnitude (L of O_2)		EPOC magnitude (L/h)	
							SIT	MICT	SIT	MICT
0.5 – 3 h post-exercise										
Islam et al. 2018 [40] [a]	23.0	8 (0)	51.2	R: 4 × 30 sec "all-out" bouts 4 min recovery periods	30 min @ 65% $\dot{V}O_{2max}$	2 h; I	10.0	6.0	5.0	3.0
Matsuo et al. 2012 [25]	24.0	10 (0)	52.0	C: 7 × 30 sec bouts @ 120% $\dot{V}O_{2max}$ 15 s recovery periods	40 min @ 65% $\dot{V}O_{2max}$	3 h; I	6.8	2.9	2.3	1.0
Metcalfe et al. 2015 [42]	26.0	8 (0)	51.0	C: 2 x 20 sec "all-out" bouts 5 min recovery periods (60 W)	50 min @ 50% $\dot{V}O_{2max}$	1.5 h: I	5.1	4.1	3.4	2.7
Schaun et al. 2018 [33]	22.3	0 (11)	38.2	UR: 8 × 20 sec bouts @ 130% $\dot{V}O_{2max}$ 10 s recovery periods	30 min @ ~70% $\dot{V}O_{2max}$	0.5 h; CM	4.2	4.6	8.3	9.1
Townsend et al. 2013 [34] [a]	23.3	6 (0)	40.2	C: 3 × 30 sec "all-out" bouts [b] 4 min recovery periods	30 min @ 55% $\dot{V}O_{2max}$ [^]	0.5 h; CM	7.5	1.8	15.0	3.6
Tucker et al. 2016 [24] [a]	24.0	10 (0)	45.9	C: 6 × 30 sec "all-out" bouts (7.5% BM) 4 min recovery periods (60% HR_{peak})	30 min @ 70% $\dot{V}O_{2max}$ [^^]	3 h; CM	22	12.8	7.3	4.3
Williams et al. 2013 [32] [a]	23.6	18 (0)	41.0	C: 4 × 30 sec "all-out" bouts (7.5% BM) 4.5 min active recovery periods	60 min @ 65% $\dot{V}O_{2peak}$	3 h; I	8.1	8.8	2.7	2.9

Mean± *SD*	23.7 ±1.4	10.0 (-) ±4.2 (-)	45.6 ±5.9	-	-	-	***9.1 ±6.0***	***5.9 ±3.8***	***6.3 ±4.5***	***3.8 ±2.5***
9 h post-exercise										
Laforgia et al. 1997 [43]	21.2	8 (0)	69.2	R: 20 × 1 min bouts @ 105% $\dot{V}O_{2max}$ 2 min recovery periods	30 min @ 70% $\dot{V}O_{2max}$ †	9 h; CM	15.0	9.0	1.7	1.0
21 – 24 h post-exercise										
Hazell et al. 2012 [44] [a]	23.0	8 (0)	52.0	C: 4 × 30 sec "all-out" bouts (10% BM) 4 min recovery periods	30 min @ 70% $\dot{V}O_{2max}$	24 h; I	62.8	12.4	2.6	0.5

Note: [a] = Wingate-based SIT study; [b] = resistance not reported in study; BMI: body mass index; C: cycling; CM: continuous measure; I: intermittently; MICT: moderate intensity continuous training; SIT: sprint interval training; $\dot{V}O_{2max}$: maximal oxygen uptake. ^ – 70% HR_{max}; ^^ - 80% HR_{max}; ^^^ - 90% $HR_{max;}$ ^^^^ - 95% HRR; † – isocaloric exercise conditions.