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Excess Post-Exercise Oxygen Consumption and Substrate Oxidation Following High-Intensity Interval Training: Effects of Recovery Manipulation

MÁRCIO A. G. SINDORF‡, MOISÉS D. GERMANO‡, WELLINGTON G. DIAS†, DANILO R. BATISTA†, TIAGO V. BRAZ‡, MARLENE A. MORENO‡, and CHARLES R. LOPES‡

Department of Human Movement Science, Methodist University of Piracicaba, Piracicaba, São Paulo, BRAZIL

†Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 14(2): 1151-1165, 2021. The recovery manipulation during high-intensity interval training (HIIT) may have the potential to modulate the responses of post-exercise energy metabolism. The purpose of this study was to investigate how the type (*i.e.*, passive and active) and duration (*i.e.*, short and long) of the recovery between the intervals in HIIT affect the excess post-exercise oxygen consumption (EPOC) and oxidation of fats and carbohydrates during the post-exercise recovery. Eight physically active men performed a maximal incremental test, to determine the peak oxygen consumption ($\dot{V}O_2$peak) and the first ventilatory threshold (VT), and four HIIT exercise sessions on a treadmill. The HIIT exercise sessions consisted of 5 intervals interspersed with 4 recovery periods; each interval was sustained until exhaustion, and the intensity was set at the $\dot{V}O_2$peak velocity; recoveries were passive, active (VT velocity), short (2-min), or long (8-min). The HIIT exercise sessions were performed in a random and crossed manner. After the HIIT exercise sessions, EPOC and oxidation of fats and carbohydrates were measured during the 120-min of post-exercise recovery. There were no differences in the EPOC among the exercise sessions (p = 0.56). There were no differences among the exercise sessions in the amount of energy expended on the oxidation of fats (p = 0.78) and carbohydrates (p = 0.91) during the post-exercise recovery. The recovery manipulation during HIIT does not affect the EPOC and post-exercise fat and carbohydrate oxidation. One can choose the type and duration of recovery, knowing that the post-exercise substrate oxidation and EPOC responses will be preserved.

KEY WORDS: Energy Expenditure, Respiratory Exchange Ratio, Body Weight, Fitness Trends

INTRODUCTION

High-intensity interval training (HIIT) and sprint interval training (SIT) protocols have been used as physical exercise strategies to improve resting energy metabolism via responses of excess post-exercise oxygen consumption (EPOC) and substrate oxidation, thus favoring the control of body weight (14, 15, 20, 32, 34). The prescription of HIIT and SIT involves the manipulation of at least nine variables (3). The intensity and duration of the recovery between the intervals in HIIT and SIT are important, because the manipulation of these variables can

regulate the contribution of the energy systems to energy production, maintenance of pH, kinetics of oxygen consumption ($\dot{V}O_2$) during subsequent intervals, metabolic power, and performance (3, 7, 29). The responses of EPOC and post-exercise (Post-Ex) substrate oxidation are modulated by means of physiological responses from the exercise session (34); therefore, the manipulation of the recovery between the intervals in HIIT may have the potential to modulate the responses of EPOC and Post-Ex fat and carbohydrate (CHO) oxidation.

Several studies have investigated the EPOC in HIIT and SIT. These studies have compared the EPOC of HIIT and SIT with steady state continuous exercise protocols (5, 12, 14, 18, 21, 22, 26, 32, 34, 35), and with other HIIT and SIT protocols (15-17, 21, 32). As far as it is known by the authors, no previous study investigated how the type (*i.e.*, passive and active) and duration (*i.e.*, short and long) of the recovery between the intervals in HIIT affect the EPOC.

Previous studies have shown that HIIT or SIT practice can increase and decrease the Post-Ex fat and CHO oxidation, respectively (4, 6, 14, 15, 17, 18, 20, 22, 32, 34, 35), however, there are few studies that compare this response in HIIT and SIT protocols (8, 15, 17, 32), and no previous study investigated how the type (*i.e.*, passive and active) and duration (*i.e.*, short and long) of the recovery between the intervals in HIIT affect the Post-Ex fat and CHO oxidation.

A survey of trends in the world of fitness for 2020 was published, with the interval training ranked second in a list of 20 positions (30); however, little is known about the effects of recovery manipulation, during HIIT, in the Post-Ex energy metabolism. Therefore, the purpose of this study was to investigate how the type and duration of the recovery between the intervals in HIIT affect the EPOC and Post-Ex fat and CHO oxidation. Based on previous studies that analyzed the Post-Ex energy metabolism in interval training protocols and found no differences (8, 9, 15-17, 21, 32), the authors hypothesize that there are no significant differences in the proposed measures.

METHODS

Participants

Eight physically active men (weight: 75.00 ± 10.52 kg; height: 1.74 ± 0.04 m; age: 28.00 ± 3.78 years; body mass index: 24.72 ± 3.22 kg/m^2) participated in this study. The participants were non-smokers, free of diseases, did not use any medication, and had been performing regular endurance and weight-lifting training for at least one year. All participants had already experienced some form of interval training in their regular endurance training. The participants responded to a health history questionnaire before the study's procedures. After explaining the project, the participants signed the informed consent form. This study was approved by the local research ethics committee (protocol n^o 523/2010), and was in accordance with the declaration of Helsinki and its resolutions. This research was carried out fully in accordance to the ethical standards of the International Journal of Exercise Science (23).

Sample Size Calculation: The sample size calculation was performed using a partial eta-squared (η^2_p) of 0.226, of our study, for the $\dot{V}O_2$ group-time interaction during the pre-exercise (Pre-Ex)

and Post-Ex times, which is the primary outcome. The G*Power software v.3.1.9.2 (10) with alpha of 0.05, beta of 0.80, effect size f of 0.54, and family F-test (ANOVA repeated measures within-between interaction) was used, and the inclusion of six participants was indicated. Eight participants were chosen to agree with the minimum number of participants in previous studies that analyzed the Post-Ex energy metabolism in HIIT and SIT (8, 9, 12, 14, 18, 34).

Protocol

Experimental Design: The participants performed five visits to the research laboratory on non-consecutive days to avoid residual effects of fatigue. The first visit was designed to explain the research project to the participants, answer a health history questionnaire, sign a free and informed consent form, and perform a maximal incremental treadmill test to determine the peak oxygen consumption ($\dot{V}O_2$peak) and the first ventilatory threshold (VT). The other four visits were designed to perform four experimental protocols, carried out in a random and crossed manner (8, 9, 11, 20, 32, 35), consisting of HIIT exercise sessions. The first visit and the first experimental protocol were separated by 48-72-h, and the experimental protocols were separated by 5-7 days.

The participants were asked not to perform vigorous physical exercises and not to consume any type of stimulant (alcohol, soft drinks, caffeine, etc.) in the 24-h preceding the maximal incremental test and the experimental protocols. Participants were asked to refrain from any change in diet during the study and keep their standard nutrition habits (8, 15, 20, 22, 24, 32). They were instructed not to eat food for 1-h preceding the visits to the laboratory, and water was *ad libitum*. The participants arrived at the laboratory during the daytime from 08:00 to 13:00-h, and respecting the same time of day in relation to the initial visit. The ambient temperature and relative air humidity in the laboratory were maintained at between 22-24°C and 40-60%, respectively.

Maximal Incremental Test: The participants performed a maximal incremental test on a treadmill (Inbrasport-ATL, Porto Alegre, RS, Brazil), according to Lourenço et al. (19). The protocol began with a warm-up of 3-min at 6 km/h; after warming-up, the velocity was increased to 7 km/h with increments of 0.3 km/h every 25-s under fixed inclination at 1%, until exhaustion. After exhaustion, the participants underwent a 5-min recovery phase, characterized by decreases in the maximal velocity reached (60%, 55%, 50%, 45%, and 40%) at each minute to avoid possible discomfort. Measurements of $\dot{V}O_2$, carbon dioxide production, and pulmonary ventilation were performed directly through an expired gas analyzer (MedGraphics VO2000, St. Paul, Minnesota, USA), using 25-s averages to match the time of the test stages (19). The $\dot{V}O_2$peak was considered as the highest $\dot{V}O_2$ value achieved in the test and the VT was determined by means of pulmonary ventilation and ventilatory equivalent for $\dot{V}O_2$ analysis (2). The heart rate was measured at each stage of the test using a heart rate monitor (Polar RS800CX, Kempele, Finland).

HIIT Exercise Sessions: The HIIT exercise sessions were performed on a treadmill (Inbrasport-ATL, Porto Alegre, RS, Brazil) with 1% inclination, and the cardiorespiratory variables of the

participants were monitored by means of an expired gas analyzer (MedGraphics VO2000, St. Paul, Minnesota, USA).

The HIIT sessions consisted of 5 intervals interspersed with 4 recovery periods. The intensity of the intervals was maintained at the external workload (*i.e.*, velocity) corresponding to the $\dot{V}O_2$peak, and each interval was sustained until exhaustion (1). For each HIIT session, a specific type and duration of recovery were adopted between the intervals. Before the exercise sessions, warm-up was performed on the treadmill for 3-min at 90% of the velocity corresponding to the VT, followed by 2-min of passive rest for the beginning of the exercise session.

The recovery employed between the intervals was passive, active, short, or long. During the passive recovery, the participants rested by sitting on a chair positioned on the treadmill; during the active recovery, the participants continued to run at the velocity corresponding to the VT. The short and long recovery times used were 2 and 8-min, respectively. The recovery time of 2-min was chosen because it is considered a short recovery time for HIIT (3), and provides adequate recovery time (28). The recovery time of 8-min was used to characterize a long recovery time for HIIT (3), and provides complete restoration of the phosphocreatine stocks when performed passively (31). The active recovery was performed at the VT, as this is the upper limit of workloads during the exercise, which can be sustained over a prolonged period of time without progressively increasing blood lactate and consequent pulmonary hyperventilation (13). The recoveries between the intervals were denominated as short-passive recovery (SPR), long-passive recovery (LPR), short-active recovery (SAR), and long-active recovery (LAR). Based on previous studies that used 4 to 6 intervals in the HIIT and SIT protocols (9, 24, 28, 29, 32), it was chosen to use 5 intervals in the present study.

Measures: Before each exercise session (including the warm-up), the participants rested dorsally for 30-min on a stretcher. After this rest period, the participants remained to lie in the dorsal position for a further 12-min for the Pre-Ex measurements. The first 2-min were discarded and the final 10-min were averaged to quantify the Pre-Ex values of $\dot{V}O_2$, respiratory exchange ratio (RER), and oxidation of fats and CHO (5, 8, 20, 22, 24, 26, 34).

Soon after the last interval of the exercise session, the participants were moved to a chair where they remained seated for 5-min; after this period, the participants were moved to a stretcher where they remained lying in the dorsal position until completing the 120-min of Post-Ex recovery (14, 22). Throughout the Post-Ex recovery, the participants remained connected to the expired gas analyzer, where at the times of 30 and 60-min, a period of 10-min occurred in which the participants could take off the mask and drink water (20).

The acquisition of the cardiorespiratory data during the entire collection period was performed using 10-s averages (11). The intensity of the exercise sessions was analyzed by means of the peak $\dot{V}O_2$ average of the 5 intervals and its percentage in relation to the $\dot{V}O_2$peak of the maximal incremental test (*i.e.*, $\dot{V}O_2$%$\dot{V}O_2$peak) (8, 9). The EPOC was calculated by means of the area under the curve using the trapezoidal method, and the Pre-Ex $\dot{V}O_2$ was subtracted. GraphPad Prism software v.6.01 (GraphPad Software, La Jolla, CA, USA) was used to calculate the area under

the curve (24). To calculate the energy expenditure (EE) during the exercise and in EPOC, the caloric equivalent of 5-kcals was used for each liter of oxygen consumed (14, 15, 35). The oxidation of fats and CHO during the Pre-Ex and Post-Ex times were calculated by means of the following equations (4, 6):

$$\text{CHO (\%)} = [(\text{RER} - 0.707)/0.293] \ (100)$$
$$\text{CHO (kcal/min)} = [(\%\text{CHO}/100) \ (\dot{V}O_2)] \ (5.05 \text{ kcal/L}O_2)$$
$$\text{Fats (kcal/min)} = [(1-\%\text{CHO}/100) \ (\dot{V}O_2)] \ (4.7 \text{ kcal/L}O_2)$$

These equations assume a steady-state condition, which cannot be observed immediately after a HIIT exercise session (20). However, the blood bicarbonate levels have been reported to return to resting levels within 30-min after cessation of high-intensity exercise, and arterial CO_2 partial pressure has been shown to be not different from resting control conditions from 60-120-min after HIIT (32), therefore, the $\dot{V}O_2$ and $\dot{V}CO_2$ data were used to estimate the substrate oxidation during the 60-120-min Post-Ex, when the RER was already normalized (*i.e.*, RER values ≥0.70 and ≤1.00) (6, 32).

During the Pre-Ex measurements and immediately after the last interval of the exercise session, 25-µl of blood was collected from the fingertips using a heparinized capillary tube and transferred to a microtube containing 50-ml of 1% sodium fluoride for analysis on a lactate analyzer (YSI-2300 Data Analyzer; Yellow Springs Instruments, Yellow Springs, OH, USA).

Statistical Analysis

All data were analyzed using the SPSS software v.22.0 (IBM Corp., Armonk, NY, USA). The normality and homogeneity of the data were tested using the Shapiro-Wilk and Levene tests, respectively. The data are presented as mean ± SD. The ANOVA one-way repeated measures test was used to analyze the differences among the experimental protocols for the Pre-Ex measures, EE of the exercise sessions, $\dot{V}O_2$ average of the intervals, lactate, EPOC, and the amount of energy expended on the oxidation of CHO during the Post-Ex recovery. The amount of energy expended on the oxidation of fats during the Post-Ex recovery and the $\dot{V}O_2$%$\dot{V}O_2$peak presented non-normal distribution, therefore, these variables were analyzed using the Friedman's test. The exhaustion time during the first interval of the HIIT sessions was analyzed, with the ANOVA one-way repeated measures test, as this interval is not influenced by the recovery manipulation and represents 100% of the exhaustion time at the velocity corresponding to the $\dot{V}O_2$peak (1). The ANOVA two-way repeated measures test (group vs time) was used to analyze the differences among the experimental protocols for the kinetics of $\dot{V}O_2$, RER, and oxidation of fats and CHO during the Pre-Ex and Post-Ex times. The ANOVA one-way and two-way repeated measures tests were used to analyze the performance of the intervals. The Bonferroni post-hoc test was used, and a statistical significance level of 5% was adopted. The Greenhouse-Geisser correction was used when necessary.

The ANOVA's effect size (*i.e.*, η^2_p) was analyzed and interpreted as 0.00-0.04 no effect, 0.05-0.25 minimum effect, 0.26-0.63 moderate effect, and ≥ 0.64 strong effect (33). The Friedman's effect size (*i.e.*, Kendall's W) was analyzed and interpreted as 0.20–0.49 small effect, 0.50–0.79 medium effect, and ≥ 0.80 large effect (25).

RESULTS

Maximal Incremental Test: The results of the maximal incremental test were as follows: $\dot{V}O_2$peak: 49.51 ± 5.96 ml/kg/min; velocity at the $\dot{V}O_2$peak: 14.95 ± 1.66 km/h; $\dot{V}O_2$ at the VT: 33.69 ± 5.01 ml/kg/min; velocity at the VT: 10.08 ± 1.47 km/h; VT%$\dot{V}O_2$peak: 67.88 ± 3.91 %.

Pre-Ex Measures and Exhaustion Time during the First Interval: There were no significant differences among the experimental protocols in the Pre-Ex measures for the $\dot{V}O_2$ ($F_{(3, 21)}$ = 0.139; p = 0.93; η^2_p = 0.019 [no effect]) (Figure 1b), blood lactate concentration ($F_{(3, 21)}$ = 0.971; p = 0.42; η^2_p = 0.122 [minimum effect]) (Figure 2a), RER ($F_{(3, 21)}$ = 0.014; p = 0.99; η^2_p = 0.002 [no effect]) (Figure 3a), and oxidation of fats ($F_{(3, 21)}$ = 0.014; p = 0.99; η^2_p = 0.002 [no effect]) (Figure 3b) and CHO ($F_{(3, 21)}$ = 0.238; p = 0.86; η^2_p = 0.033 [no effect]) (Figure 3c).

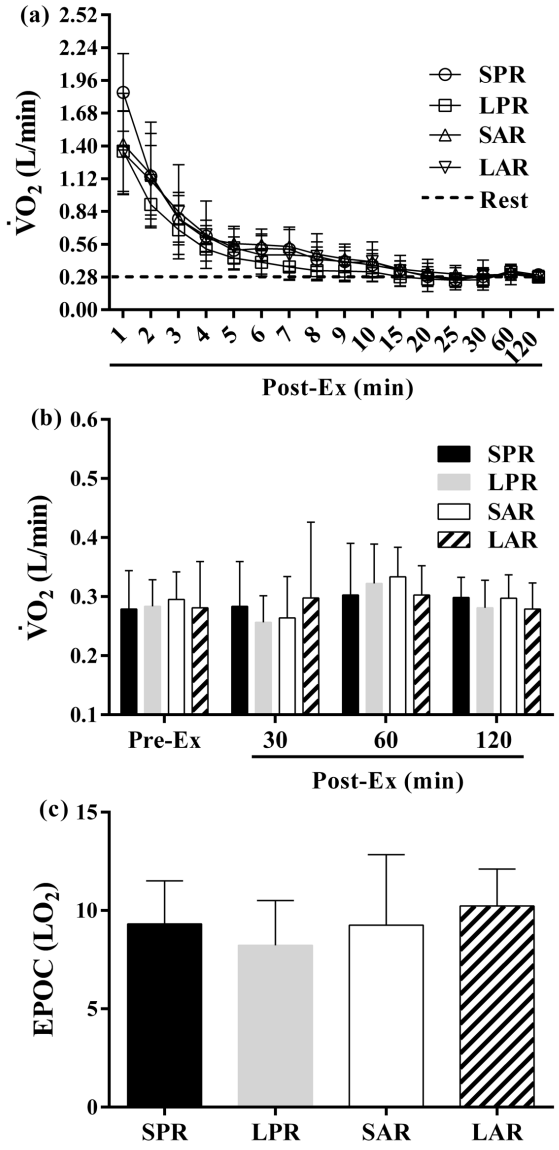

Figure 1. The data are presented as mean ± SD. Descriptive analysis of the oxygen consumption ($\dot{V}O_2$) kinetics during the post-exercise (Post-Ex) recovery (a). The $\dot{V}O_2$ during the pre-exercise (Pre-Ex) and Post-Ex recovery times (b). The excess post-exercise oxygen consumption (EPOC) (c). SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. Note: the rest value in the figure "a" is the average of the Pre-Ex values presented in the figure "b", since there were no differences among them in the Pre-Ex time (26). There was no group effect in the three figures (p > 0.05).

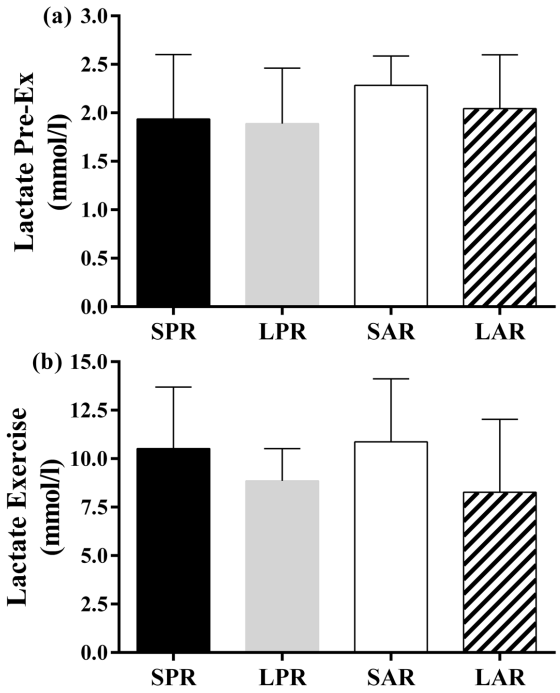

Figure 2. The data are presented as mean ± SD. The blood lactate concentration in the pre-exercise (Pre-Ex) time (a) and immediately after the last interval of the exercise session (b). SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. There were no differences among the protocols in the two figures ($p > 0.05$).

The exhaustion time during the first interval was 238.63 ± 61.92, 222.88 ± 55.48, 217.75 ± 57.14, and 208.13 ± 61.93 seconds, for the experimental protocols SPR, LPR, SAR, and LAR, respectively, and without significant differences among them ($F_{(3, 21)} = 1.468$; $p = 0.25$; $\eta^2_p = 0.173$ [minimum effect]).

Performance of the Intervals: There was an effect for the interval ($F_{(3, 21)} = 17.344$; $p < 0.00$; $\eta^2_p = 0.712$ [strong effect]), interval number ($F_{(1.292, 9.045)} = 31.385$; $p < 0.00$; $\eta^2_p = 0.818$ [strong effect]), and interaction ($F_{(3.823, 26.760)} = 6.174$; $p < 0.00$; $\eta^2_p = 0.469$ [moderate effect]) (Table 1). There was a main effect for the percentage of variation ($F_{(3, 21)} = 38.732$; $p < 0.00$; $\eta^2_p = 0.847$ [strong effect]), with the LPR protocol showing better performance maintenance (Table 1).

Table 1. The time in seconds for each of the five intervals and the percentage of variation (%Δ) from the first to the last interval. Data are presented as mean and standard deviation in parentheses.

	Interval Number					%Δ
Protocol	1st	2nd	3rd	4th	5th	
SPR	238.63 (61.92)	148.38 (33.64)	131.88 (27.33)	137.50[a] (27.45)	118.63[a] (11.44)	- 46.89[b] (16.77)
LPR	222.88 (55.48)	202.50 (61.42)	172.88[a] (43.27)	159.88[a] (32.97)	180.13[a] (40.51)	- 17.74[c,d] (11.49)
SAR	217.75 (57.14)	112.63[a] (17.03)	102.75[a] (10.74)	82.38[a] (17.34)	84.00[a] (20.87)	- 60.53 (8.45)
LAR	208.13 (61.93)	104.00 (27.12)	77.63[a] (35.24)	71.63[a] (23.69)	68.63[a] (30.87)	- 66.75 (13.00)

The data are presented as mean ± SD. SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. a: different from the first interval ($p < 0.05$); b: different from the corresponding long recovery ($p < 0.05$); c: different from the corresponding active recovery ($p < 0.05$); d: unlike all other recoveries ($p < 0.05$).

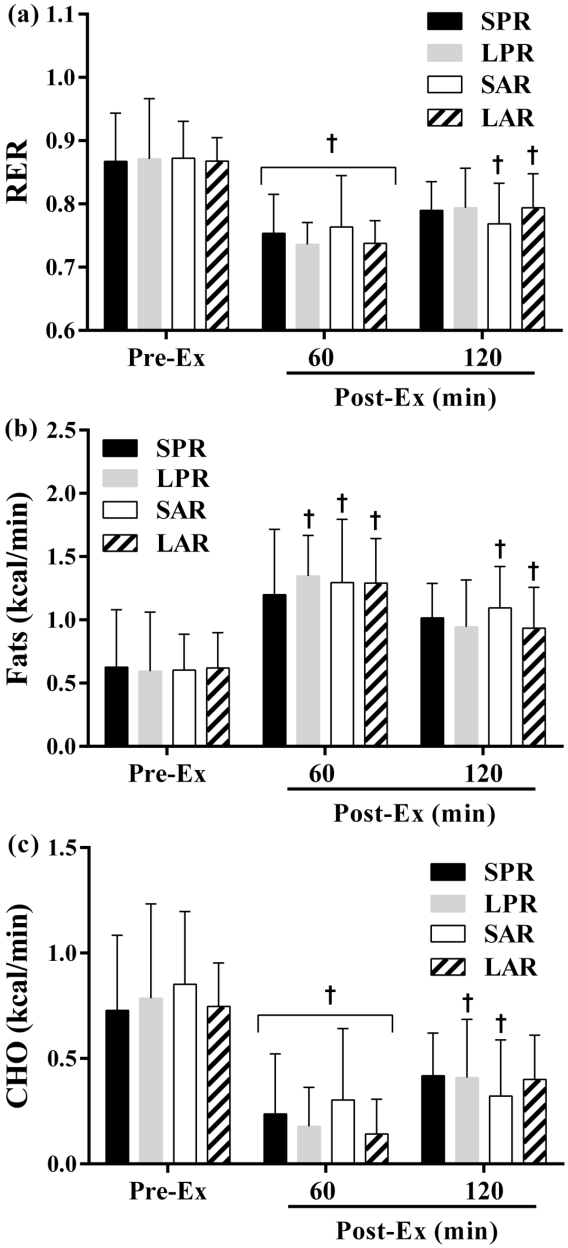

Figure 3. The data are presented as mean ± SD. The respiratory exchange ratio (RER) (a) and oxidation of fats (b) and carbohydrates (CHO) (c) during the pre-exercise (Pre-Ex) and post-exercise (Post-Ex) recovery times. SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. †: different from the Pre-Ex time ($p < 0.05$). There was no group effect in the three figures ($p > 0.05$).

$\dot{V}O_2$ Average of the Intervals, $\dot{V}O_2$%$\dot{V}O_2$peak, and Lactate of the Exercise: There were no significant differences among the experimental protocols in the $\dot{V}O_2$ average of the intervals ($F_{(1.198, 8.385)} = 1.344$; $p = 0.28$; $\eta^2_p = 0.161$ [minimum effect]) and $\dot{V}O_2$%$\dot{V}O_2$peak ($\chi^2_{(3)} = 2.684$; $p = 0.44$; W = 0.112 [small effect]) of the exercise (Figure 4a,b). There were no significant differences in the blood lactate concentration of the exercise ($F_{(3, 21)} = 2.136$; $p = 0.12$; $\eta^2_p = 0.234$ [minimum effect]) (Figure 2b).

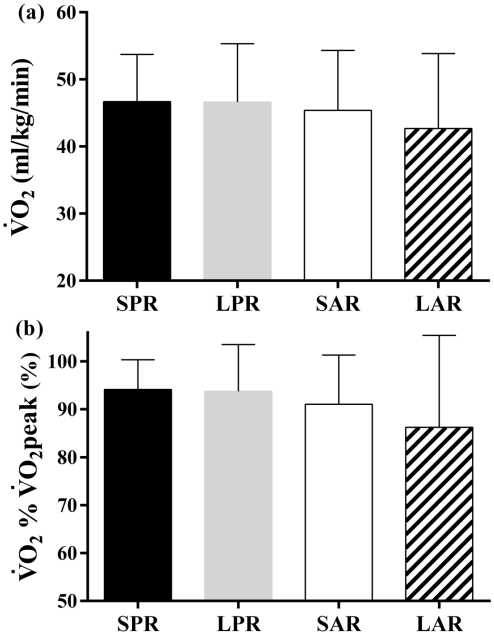

Figure 4. The data are presented as mean ± SD. The peak $\dot{V}O_2$ average of the five intervals (a) and its percentage in relation to the $\dot{V}O_2$peak of the maximal incremental test (b). $\dot{V}O_2$: oxygen consumption; $\dot{V}O_2$peak: peak oxygen consumption; SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. There were no differences among the protocols in the two figures ($p > 0.05$).

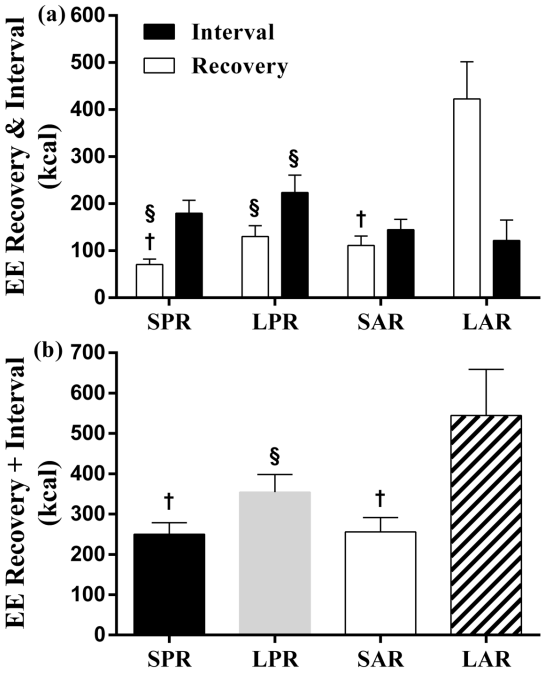

Figure 5. The data are presented as mean ± SD. The energy expenditure (EE) of the intervals and recoveries that make up the high-intensity interval training sessions (a). The sum of the intervals and recoveries EE values (b). SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. †: different from the corresponding long recovery ($p < 0.05$); §: different from the corresponding active recovery ($p < 0.05$).

EE of the HIIT Sessions: There was a main effect for the EE of the intervals ($F_{(3, 21)} = 18.108$; $p < 0.00$; $\eta^2_p = 0.721$ [strong effect]), however, the pairwise comparisons test only found a significant difference in the comparison between LPR vs LAR ($p < 0.00$) and LPR vs SAR ($p < 0.00$), the other comparisons were not significantly different (Figure 5a). All the comparisons for the EE of the recoveries were significantly different ($F_{(1.268, 8.875)} = 108.510$; $p < 0.00$; $\eta^2_p = 0.939$ [strong effect]), except for the comparison between LPR vs SAR ($p = 0.95$) (Figure 5a). The EE of the HIIT sessions

was significantly different for all the comparisons ($F_{(1.484,\ 10.391)}$ = 37.720; p < 0.00; η^2_p = 0.843 [strong effect]), except for the comparison between SPR vs SAR (p = 0.99) (Figure 5b).

Post-Ex $\dot{V}O_2$ and EPOC: Figure 1a shows a descriptive analysis of the $\dot{V}O_2$ kinetics during the Post-Ex recovery. There was no group and group-time interaction effect for the $\dot{V}O_2$ during the Pre-Ex and Post-Ex times (group $F_{(3,\ 21)}$ = 1.780; p = 0.18; η^2_p = 0.203 [minimum effect]; group-time interaction $F_{(4.356,\ 30.490)}$ = 2.044; p = 0.10; η^2_p = 0.226 [minimum effect]), but there was a time effect ($F_{(2.031,\ 14.217)}$ = 129.589; p < 0.00; η^2_p = 0.949 [strong effect]). At 30-min of Post-Ex recovery, the $\dot{V}O_2$ had returned to the Pre-Ex value for all the experimental protocols (Figure 1b). There were no significant differences in the comparison of the EPOC among the experimental protocols ($F_{(3,\ 21)}$ = 0.688; p = 0.56; η^2_p = 0.090 [minimum effect]) (Figure 1c). The EPOC, in terms of EE, was 46.58 ± 10.97, 41.13 ± 11.42, 46.27 ± 17.97, and 51.16 ± 9.39 kcals, for the experimental protocols SPR, LPR, SAR, and LAR, respectively.

Post-Ex RER, Fat, and CHO: There was no group and group-time interaction effect, but there was a time effect during the Pre-Ex and Post-Ex times for the RER (group $F_{(3,\ 21)}$ = 0.019; p = 0.99; η^2_p = 0.003 [no effect]; time $F_{(2,\ 14)}$ = 67.658; p < 0.00; η^2_p = 0.906 [strong effect]; group-time interaction $F_{(2.966,\ 20.762)}$ = 0.635; p = 0.59; η^2_p = 0.083 [minimum effect]), and oxidation of fats (group $F_{(3,\ 21)}$ = 0.088; p = 0.96; η^2_p = 0.012 [no effect]; time $F_{(2,\ 14)}$ = 51.924; p < 0.00; η^2_p = 0.881 [strong effect]; group-time interaction $F_{(6,\ 42)}$ = 0.344; p = 0.90; η^2_p = 0.047 [no effect]) and CHO (group $F_{(3,\ 21)}$ = 0.189; p = 0.90; η^2_p = 0.026 [no effect]; time $F_{(2,\ 14)}$ = 57.349; p < 0.00; η^2_p = 0.891 [strong effect]; group-time interaction $F_{(6,\ 42)}$ = 0.660; p = 0.68; η^2_p = 0.086 [minimum effect]) (Figure 3a,b,c).

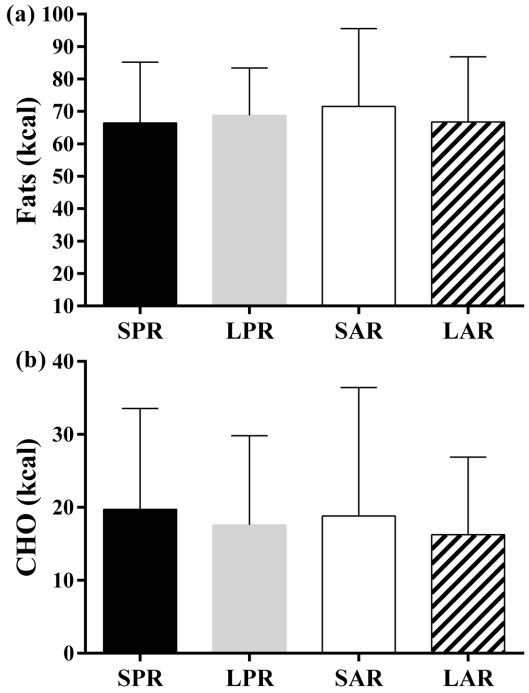

Figure 6. The data are presented as mean ± SD. The amount of energy expended on the oxidation of fats (a) and carbohydrates (CHO) (b) during the post-exercise recovery. SPR: short-passive recovery; LPR: long-passive recovery; SAR: short-active recovery; LAR: long-active recovery. There were no differences among the protocols in the two figures (p > 0.05).

There were no significant differences among the experimental protocols in the amount of energy expended on the oxidation of fats ($\chi^2_{(3)} = 1.050$; $p = 0.78$; W = 0.044 [small effect]) and CHO ($F_{(3, 21)} = 0.166$; $p = 0.91$; $\eta^2_p = 0.023$ [no effect]) during the Post-Ex recovery (Figure 6a,b).

DISCUSSION

The initial hypothesis of the study was confirmed. The main finding of this study was to show that the recovery manipulation in the HIIT sessions does not provide significant differences in the EPOC and Post-Ex fat and CHO oxidation (Figures 1, 3, 6), although there were significant differences in the EE of the HIIT sessions (Figure 5b). It was also possible to observe that the recovery manipulation in the HIIT sessions does not provide significant differences in the $\dot{V}O_2$ (Figure 4a,b) and blood lactate concentration (Figure 2b) of the exercise.

There were no significant differences in the Pre-Ex measures (*i.e.*, $\dot{V}O_2$, lactate, RER, and oxidation of fats and CHO measures), as expected, and this emphasizes the high adherence of the participants to the study's recommendations, and indicates that the participants began the experimental protocols in comparable physiological states (8, 20, 22, 26, 34). The lack of significant differences in the exhaustion time during the first interval of the HIIT sessions also emphasizes the high adherence of the participants to the study's recommendations.

There were no significant differences in the $\dot{V}O_2$ and $\dot{V}O_2\%\dot{V}O_2peak$ of the exercise (Figure 4a,b). Smilios et al. (29) analyzed the effects of different active recovery times (*i.e.*, 4, 3, and 2-min of recovery) in a HIIT protocol, and found no differences in the $\dot{V}O_2\%\dot{V}O_2peak$ and time spent at high rates of $\dot{V}O_2$ during the exercise. Other studies investigating the type (7) and duration (9) of recovery in interval training protocols, also found no differences in the $\dot{V}O_2$ and $\dot{V}O_2\%\dot{V}O_2peak$ of the exercise. The studies mentioned above corroborate the findings of the present study. The recovery manipulation alters the kinetics of $\dot{V}O_2$ during subsequent intervals (29); however, this does not significantly alter the $\dot{V}O_2$ and $\dot{V}O_2\%\dot{V}O_2peak$ of the exercise.

There were no significant differences in the blood lactate concentration of the exercise (Figure 2b), and this is in line with previous studies. Eigendorf et al. (8) compared HIIT and SIT protocols with SAR and different external workloads but equalized by the internal workload (*i.e.*, $\dot{V}O_2$), and found no difference in the lactate of the exercise. Other studies investigating the type (7) and duration (28) of recovery in interval training protocols, also found no differences in the lactate of the exercise. Although it has been postulated that the recovery manipulation could alter the contribution of the energy systems to energy production (29), the results of the present study show that the glycolytic contribution to energy production is not significantly altered.

The differences found in the total EE of the HIIT sessions (Figure 5b) were already expected, because the authors manipulated the recovery in the training sessions. The recovery manipulation had little effect in the EE of the intervals, since there was no significant difference between short vs long recovery times (regardless of the type of recovery) and between SPR vs SAR (Figure 5a). The lack of significant differences in the EE of the intervals can be explained by

the short recovery time used being adequate for maintaining performance, as described by Seiler and Hetlelid (28). The difference found in the EE of the intervals between LPR vs LAR is in accordance with that described in the literature, which shows that the use of LAR provides loss of performance (3).

The EPOC duration found in the present study is in agreement with the findings of Cabral-Santos et al. (5) and Schaun et al. (26), who used HIIT and SIT protocols with SPR, respectively, and observed the EPOC during the first 30-min of Post-Ex recovery. There were no differences in the magnitude of the EPOC among the experimental protocols (Figure 1c), and the values obtained are in agreement with those reported in other studies that employed HIIT (5, 21, 22) and SIT (21, 26, 35) protocols.

It is not clear why there were no differences in the EPOC among the experimental protocols, but two explanations may exist. Firstly, the lack of significant differences in the interval intensity among the protocols (Figure 4a,b) may have resulted in this lack of difference in the EPOC. Warren et al. (34) showed that the EPOC is highly affected by the exercise intensity and less affected by the exercise volume. The second hypothesis is a possible plateau response of the EPOC. Scott et al. (27) reported an EPOC plateau during the investigation of different resistance exercise protocols, as supported by previous studies with rats that performed sprint exercises with different intensities and volumes, and found no differences in the EPOC (27).

A study by Warren et al. (34) demonstrated that the exercise volume exerts influence in the amount of fat oxidation during the Post-Ex recovery, however, this was not observed in the present study (Figure 6a). It is also noteworthy that no significant differences were observed among the experimental protocols in the amount of CHO oxidation during the Post-Ex recovery (Figure 6b). The explanation for this result is unclear, but it may be related to the lack of significant differences in the interval intensity among the protocols (Figure 4a,b), since Eigendorf et al. (8) demonstrated that the average intensity (*i.e.*, $\dot{V}O_2$) of the intervals is an important factor to regulate the responses of energy metabolism in interval training protocols.

In the present study, there were no significant differences among the experimental protocols for the EPOC and Post-Ex fat and CHO oxidation; however, it is important to note that all the experimental protocols were efficient in providing EPOC and Post-Ex fat and CHO oxidation responses. In this sense, the choice of recovery to be used will depend on secondary factors, such as performance (Table 1).

The present study has some limitations. In the present study, the open-circuit indirect calorimetry was used, which made it impossible to measure the substrate oxidation during the HIIT sessions, as well as in the first 59-min of Post-Ex recovery; such measures would provide a better understanding of the responses of energy metabolism during and after the HIIT sessions. A specific sample of physically active men was used; therefore, caution should be taken when extrapolating the results to subjects at the extremes of the physical conditioning variation, such as sedentary subjects with overweight/obesity and endurance athletes. The authors established a short period (2-h) for the Post-Ex recovery, and therefore some of the prolonged

effects of the protocols (~ 24-h) may not have been observed; however, the period used was enough to observe the return of the $\dot{V}O_2$ to the Pre-Ex value (5, 26), and the result on substrate oxidation is unlikely to change (8, 17, 32).

In conclusion, the recovery manipulation during HIIT does not affect the EPOC and Post-Ex fat and CHO oxidation. The findings of the present study can be used to develop HIIT protocols for body weight control in physically active men. The HIIT is among the main trends in the world of fitness (30); however, little is known about the effects of recovery manipulation, during HIIT, in the Post-Ex energy metabolism. The findings show that one can choose the type and duration of recovery, knowing that the Post-Ex substrate oxidation and EPOC responses will be preserved.

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