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RESEARCH

Order effect of an 8-week concurrent training program on the maximal fat oxidation

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Abstract

Background Concurrent training (CT) is a common method used in health-oriented training programs. However, its design needs to be explored in order to inform practitioners about the most effective methods to improve different health-related markers (e.g., maximal fat oxidation [MFO]). Thus, this study aims to determine the order effect of an 8 week CT on the MFO of recreationally trained young adults.

Methods Twenty participants were allocated in two different groups to perform the CT differing only in the exercise sequence. The endurance training (ET) consisted of 4 to 6 repetitions of 30 s all-out running sprints with 4 min of active recovery. The resistance training (RT) consisted of 4 to 6 sets at 60 to 80% of the one-repetition maximum with 5 to 1 repetition in reserve of back squat and bench press exercises. 15 min of rest were established between exercise modes. Previous and after the CT program, participants performed a graded exercise test where MFO was determined.

Results There was a significant time effect on MFO ($p = 0.044$). A moderate increase was observed in both ET + RT (Mean change: 0.11 [− 0.02 to 0.25] g/min; Effect size: 0.61 [− 0.12 to 1.35]) and RT + ET (Mean change: 0.07 [− 0.01 to 0.16] g/min; Effect size: 0.62 [− 0.12 to 1.36]) groups. No significant interaction was observed ($p = 0.658$).

Conclusions The 8 week CT program improved the muscle oxidative capacity of recreationally trained young adults regardless of the exercise sequence.

Keywords Resistance training · Sprint interval training · Physical fitness · Fat oxidation

Introduction

The capacity to oxidize fat (i.e., fat oxidation [FO]) during exercise has been a used in clinical settings as an impairment in its maximum rate (i.e., maximal fat oxidation [MFO]) is associated with the development of metabolic syndrome [1]. FO is regulated through different steps. It begins with the adipose tissue lipolysis, followed by the transport of free fatty acids to muscle and across the cell membrane, and ends through the beta-oxidation process carried out by the enzymes that regulate the Krebs cycle and the electron transport chain [2]. Thus, health training programs should consider those exercises that maximize the protein expression of those elements involved in FO.

Traditionally, moderate-intensity continuous training (MICT) has been used for improving FO. However, it has been established that high-intensity interval training (HIIT) or sprint interval training (SIT) can be as effective as MICT in almost half of the training time [3]. The mechanism by which SIT improves FO relies on the activation of the signaling pathways of mitochondrial biogenesis [4, 5]. The glycolytic metabolism required during the high-intensity bouts involves the accumulation of free radicals, ions and metabolites (i.e., lactate, creatine, AMP, H^+), resulting in the phosphorylation of the AMP-activated protein kinase (AMPK) and, thus, the

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expression of the peroxisome proliferator-activated receptor coactivator-1α (PGC-1α), the main transcriptional cofactor that mediates mitochondrial biogenesis [4]. Of note, it seems that the AMPK activation is also bound to the on–off pattern of interval training [6].

Albeit in a lesser degree, it has been shown that resistance training (RT) can also have an impact on FO through the phosphorylation of the AMPK [7, 8]. As it occurs during endurance training (ET), the AMPK is activated through markers of low cellular energy and, thus, the load and the volume established on RT can modulate the signaling of the biogenesis pathways. Porter et al. [7] observed an improvement in mitochondrial respiration after 12 weeks of whole-body RT conducted 3 times per week at an intensity of 60–80% of the one-repetition maximum (1RM), with multiple sets performed to failure. Thus, it seems that in order to stimulate mitochondrial biogenesis, RT should be focused on inducing a high metabolic stress [9].

A high metabolic stress could be also induced by combining ET and RT within the same training program (i.e., concurrent training [CT]). However, the synergistic effect of both exercise modes is determined by the exercise principles employed. The exercise sequence has been postulated as one of the potential factors that mediates mitochondrial biogenesis. Wang et al. [10] observed an enhanced expression of marker genes related to mitochondrial biogenesis (i.e., PGC-1α) and substrate regulation (i.e., PDK4) when ET (i.e., one hour cycling at 65% of the maximum oxygen uptake [VO_{2max}]) was followed by RT (i.e., six sets of leg press at 70–80% 1RM up to 15 repetitions) compared to ET alone in recreationally active adults. Likewise, Coffey et al. [11] observed that PGC-1α raised after CT (ET: 30 min of cycling at 70% of VO_{2max}; RT: eight sets of leg extension at 80% 1RM), being slightly higher when ET was followed by RT in regular training adults.

The acute molecular signaling is the first adaptive response to training. However, the exercise-training-induced adaptions are the result of the summed effects of repeated training sessions that require to be analyzed in the long term in order to determine the efficacy of a training program on improving the muscle oxidative capacity. Likewise, the adaptive response to CT requires the analysis of further combinations of exercise modes, intensities, and volumes, as well as the population recruited [12]. Therefore, this study aims to determine the order effect of an 8 week CT program composed of SIT and lower- and upper-body resistance exercises on the MFO of recreationally trained young adults.

Materials and methods

Experimental design

A longitudinal pre-post design was used to compare the effect of altering the exercise sequence of a CT program (i.e., ET followed by RT [ET + RT] vs. RT followed by ET [RT + ET]) on MFO. Before starting the training program, participants attended to the pre-tests where body composition, back squat and bench press 1RM, MFO, and VO_{2max} were determined. Standardized groups were created considering VO_{2max}. Then, participants began with an 8 week CT program composed of three sessions per week of 60 to 90 min duration with all-out running SIT, back squat, and bench press resistance exercises. After checking their compliance with the training program, participants followed the post-test sessions. All testing and training sessions were conducted in a research center with at least 48–72 h of rest, at a consistent time of day for each subject (± 1 h) and under similar environmental conditions (~ 20 °C and ~ 60% humidity).

Participants

A group of 20 recreationally trained healthy young adults were initially enrolled. All participants were required to meet the following inclusion criteria: (i) being between 18 and 30 years old, (ii) free from any injuries within the six months before data collection, and (iii) being physically active according to the guidelines of the American College of Sports Medicine (ACSM) [13]. After assessing for eligibility and determining the baseline level of fitness of participants, they were allocated in two groups differing only in the exercise sequence: ET + RT ($n = 10$) or RT + ET ($n = 10$) to conduct an 8 week CT program. However, 3 participants dropped out from the training intervention for reasons unrelated to the study and 3 participants were discarded due to the lack of compliance with training. Thus, 7 participants (3 males and 4 females) in the ET + RT group (age: 21.0 [2.0] years; height: 170 [5.9] cm; body mass: 64.1 [8.0] kg; fat mass: 10.9 [3.2] kg; fat-free mass [FFM]: 53.2 [9.9] kg; back squat one-repetition maximum [1-RM]: 1.40 ± 0.31 kg/kg_{BM}; bench press 1-RM: 0.90 ± 0.30 kg/kg_{BM}; training experience: 5 ≥ years; training frequency: 2 ≥ sessions per week) and 7 participants (5 males and 2 females) in the RT + ET group (age: 21 [1.5] years; height: 178 [11] cm; body mass: 77.5 [10.9] kg; fat mass: 15.5 [6.9] kg; FFM: 62.2 [10.7] kg; back squat 1-RM: 1.50 ± 0.25 kg/kg_{BM}; bench press 1-RM: 0.75 ± 0.20 kg/kg_{BM}; training experience: 5 ≥ years; training frequency: 2 ≥ sessions per week) completed the entire intervention process (Table 1). A post hoc analysis of the achieved power for this sample size was conducted using G*power (version 3.1), given $\alpha = 0.05$, $ES = 0.60$, total sample size = 14, statistical tests = means: difference between dependent means. This analysis revealed a power of 0.55 for the time effect. All participants were informed about the research purpose and procedures of the study prior to signing a written informed consent form. The study protocol

Table 1 Baseline characteristics of training groups

	ET + RT	RT + ET	*p*-value
MFO (g/min)	0.60 (0.15)	0.68 (0.12)	0.579
MFO_{rel} (g/kg FFM/min)	0.011 (0.003)	0.011 (0.002)	0.793
Fatmax (% of VO_{2max})	53.9 (7.5)	55.7 (5.5)	0.620
VO_{2max} (ml/kg/min)	52.1 (5.1)	49.6 (5.0)	0.367

Values are mean ± SD; *ET* endurance training; *RT* resistance training; *MFO* maximal fat oxidation; MFO_{rel} MFO relativized to fat-free mass; *Fatmax* MFO relative intensity; VO_{2max} maximum oxygen uptake; *FFM* fat-free mass

adhered to the tenets of the declaration of Helsinki and was approved by the institutional review board (ref. 2546/CEIH).

Body composition

The anthropometric characteristics of the participants (body mass [kg], fat mass [kg] and FFM [kg]) were obtained using the bioimpedanciometer Inbody 230 (Inbody, Seoul, Korea), which has been previously validated by a dual-energy X-ray system [14]. Participants were encouraged to follow the recommendations of the American Society of Exercise Physiologists before the test [15].

MFO, fatmax, VO_{2max}

The graded exercise test (GXT) was conducted on a treadmill (WOODWAY Pro XL, Woodway, Inc., Waukesha, WI, USA). Participants warmed up for 5 minutes at a self-selected speed with the premise of not exceeding an intensity at which they could not talk. Then, participants were fitted with the validated portable metabolic analyzer (PNOE, ENDO Medical, Palo Alto, CA) [16], which was previously calibrated according to the manufacturer's instructions. The GXT was customized to determine MFO and VO_{2max} in the same session and according to the experience and level of the participants. From a starting velocity of 5 km/h, the velocity was increased by 1 km/h every 3 min until the respiratory gas exchange rate reached 1.0. After that, the velocity remained constant, and the incline was increased by 2% every minute until volitional exhaustion.

The breath-by-breath data of each record were exported into an Excel spreadsheet. To exclude errant breaths, values outside the 95% confidence interval of the local mean were removed. Then, breath-by-breath data were linearly interpolated to give 1 s values. Average values of oxygen uptake (O_2; L/min) and dioxide production (CO_2; L/min) for the last 60 s in each 3 min step were used to determine FO (gr/min) through the Frayn equation [17] with the assumption that the urinary nitrogen excretion rate (n) was negligible:

$$FO = 1.67*O_2 - 1.67*CO_2 - 1.92*n.$$

For each subject, the calculated values for FO in each step were depicted graphically as a function of exercise intensity (%VO_{2max}) and a 2nd polynomial curve with an intersection in (0,0) was constructed to determine MFO (g/min) and the intensity that elicits the MFO (Fatmax) (% VO_{2max}). VO_{2max} was determined as the highest 30 s rolling mean value.

CT program

Both groups completed an 8 week CT program composed of three sessions of 60 to 90 min per week with running SIT, as well as back squat and bench press resistance exercises, differing only in their exercise sequences (ET + RT vs. RT + ET) (Table 2). Relative loads were applied according to the 1RM pre-test (Table 1). Back squat and bench press 1RM were estimated from the individual and exercise-specific load-velocity profile [18]. The training program progressed every 2 weeks in volume and load for running SIT (i.e., 4–6 intervals) and resistance exercises (i.e., 60–80% 1RM), respectively. Since sessions were created with a time-efficient purpose, only 15 min of rest was established between exercise modes. To minimize the potential effect of fatigue on the subsequent exercise to be performed, the ET + RT group conducted the bench press exercise first after the running SIT, and the RT + ET group began with the back squat before the bench press exercise and the running SIT. An

Table 2 8 week concurrent training program

3 days/week	SIT	RT
Week 1	4 × 30″ all out 4´active rest	4–5 × 60% 1RM, RIR 5–6, rest 2´
Week 2	4 × 30″ all out 4´active rest	4–5 × 60% 1RM, RIR 5–6, rest 2´
Week 3	5 × 30″ all out 4´active rest	5–6 × 70% 1RM, RIR 3–4, rest 2´
Week 4	5 × 30″ all out 4´active rest	5–6 × 70% 1RM, RIR 3–4, rest 2´
Week 5	6 × 30″ all out 4´active rest	5–6 × 80% 1RM, RIR 2–3, rest 2´
Week 6	6 × 30″ all out 4´active rest	5–6 × 80% 1RM, RIR 2–3, rest 2´
Week 7	6 × 30″ all out 4´active rest	6 × 80% 1RM, RIR 1–2, rest 2´
Week 8	6 × 30″ all out 4´active rest	6 × 80% 1RM, RIR 1–2, rest 2´

SIT sprint interval training; *RT* resistance training; *1RM* one-repetition maximum; *RIR* repetitions in reserve

instructor assisted and encouraged the participants in each session of the training program. Sessions were conducted with at least 48 h of recovery in between. Participants were encouraged to follow their habitual dietary patterns over the entire training program.

Statistical analysis

Data are presented as mean (SD) or (95% confidence interval). The normal distribution and homogeneity of variance were confirmed through the Shapiro–Wilk test and Levene's tests, respectively. The baseline characteristics of each training group were compared through an independent sample student's *t* test. A one-way mixed model (group x time) analysis of variance (ANOVA) was used to determine the effect of altering the exercise sequence (i.e., ET + RT or RT + ET) on the MFO, MFO relativized to FFM (MFO_{rel}), Fatmax, and VO_{2max}. The normality of the residuals was confirmed through the inspection of Q–Q plots. The homogeneity of variances was also confirmed through the Levene test. Pairwise effect size (ES) was determined as (mean change/SD change) [19], and interpreted as follows: trivial (< 0.2), small (0.2–0.59), moderate (0.60–1.19), large (1.20–2.0), and extremely large (> 2.0) [20]. Statistical analyses were performed using the software package SPSS (IBM SPSS, version 25.0; IBM, Chicago, IL). Alpha was set at 0.05.

Results

Training groups presented similar baseline characteristics regarding MFO (ET + RT: 0.60 [0.15] g/min; RT + ET: 0.68 [0.12] g/min; $p = 0.579$), MFO_{rel} (ET + RT: 0.011 [0.003] g/min/kg FFM; RT + SIT: 0.011 [0.002] g/min/kg FFM; $p = 0.793$), Fatmax (ET + RT: 53.9 [7.5] % VO_{2max}; RT + ET: 55.7 (5.5) % VO_{2max}; $p = 0.620$), and VO_{2max} (ET + RT: 52.1 [5.1] ml/kg/min; RT + ET: 49.6 [5.0] ml/kg/min; $p = 0.367$) (Table 1).

There was a significant time effect on MFO ($F_{(1,12)} = 5.07$; $p = 0.044$) (Fig. 1). A moderate increase was observed in both ET + RT ($p = 0.08$; mean change: 0.11 [− 0.02 to 0.25] g/min; ES: 0.61 [− 0.12 to 1.35]) and RT + ET ($p = 0.227$; mean change: 0.07 [[− 0.01 to 0.16] g/min; ES: 0.62 [− 0.12 to 1.36]) groups. MFO_{rel} did not reach a significant time effect ($F_{(1,12)} = 3.79$; $p = 0.075$) although a small increase was observed in both ET + RT ($p = 0.107$; mean change: 0.002 [− 0.001 to 0.004] g/min/kg FFM; ES: 0.54 [− 0.20 to 1.28]) and RT + ET ($p = 0.333$; mean change: 0.001 [0.000

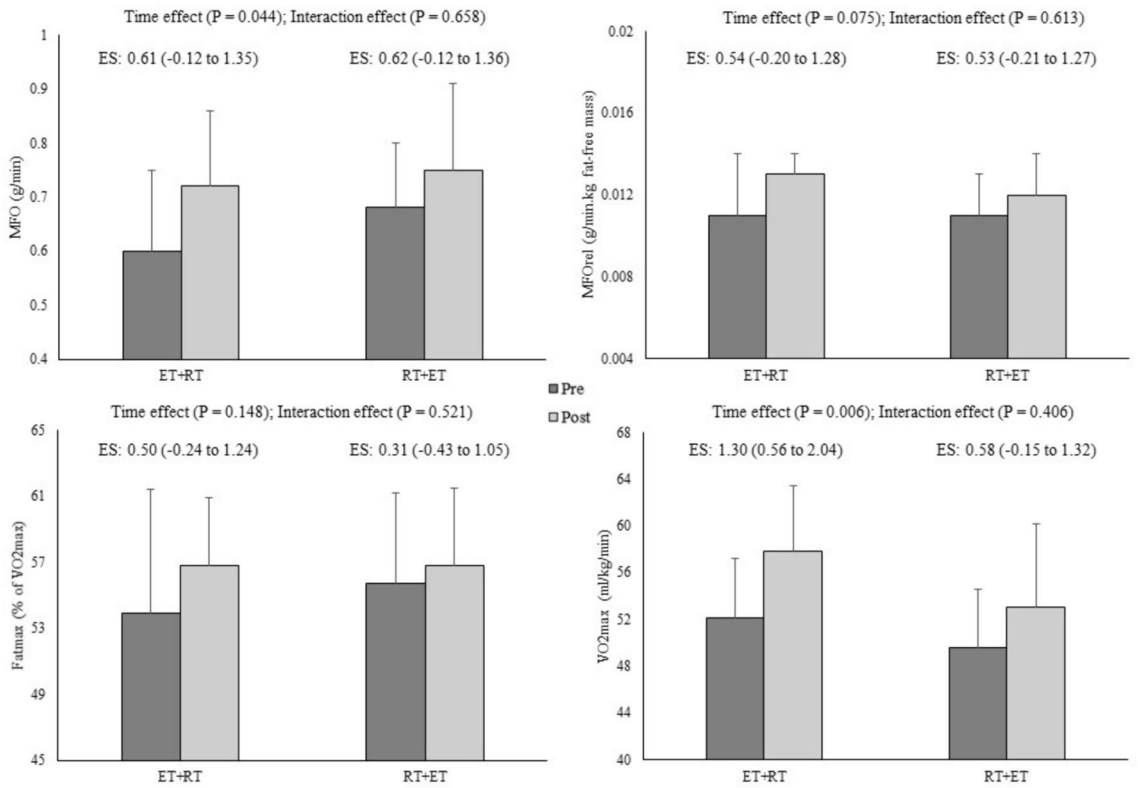

Fig. 1 Effect of the exercise sequence of the concurrent training program. Values are mean ± SD; *ET* endurance training; *RT* resistance training; *MFO* maximal fat oxidation; MFO_{rel} MFO relativized to fat-free mass; *Fatmax* MFO relative intensity; *VO2max* maximum oxygen uptake; *FFM* Fat-free mass

to 0.002] g/min/kg FFM; ES: 0.53 [− 0.21 to 1.27]) groups. A non-significant time effect was also obtained on Fatmax ($F_{(1,12)}$ = 2.38; p = 0.148), although a small increase was observed in both ET + RT (p = 0.145; mean change: 2.86 [− 1.41 to 7.12] % of VO_{2max}; ES: 0.50 [− 0.24 to 1.24]) and RT + ET (p = 0.544; mean change: 1.14 [− 1.61 to 3.89] % of VO_{2max}; ES: 0.31 [− 0.43 to 1.05]) groups. There was a significant time effect on VO_{2max} ($F_{(1,12)}$ = 11.04; p = 0.006). A large increase was observed in ET + RT group (p = 0.012; mean change: 5.69 [2.46 to 8.92] ml/kg/min; ES: 1.30 [0.56 to 2.04]) and a small increase in RT + ET group (p = 0.107; mean change: 3.35 [− 0.89 to 7.59] ml/kg/min; ES: 0.58 [− 0.15 to 1.32]). Non-significant interactions were observed in the aforementioned variables ($F_{(1,12)}$ < 0.740; p > 0.406).

Discussion

This study aimed to determine the order effect of an 8 week CT program composed of SIT and low and upper-body resistance exercises on the MFO of recreationally trained young adults. The results revealed that the CT program improved the MFO in both training groups in a similar manner, and thus, no interferences were observed regarding the exercise sequences on improving the muscle oxidative capacity.

Both training groups improved MFO in a similar magnitude (ET + RT: 0.11 [− 0.02 to 0.25] g/min; RT + ET: 0.07 [− 0.01 to 0.16] g/min). While training-induced increases of 1-MET have been considered of clinical relevance [21], there are no consolidated thresholds for interpreting MFO changes. In this regard, Atakan et al. [3] have recently proposed that any increases greater than 0.07 g/min would be meaningful, being such changes observed in HIIT and SIT interventions of 4 to 12 weeks (0.05 to 0.13 g/min). These MFO changes are derived from the molecular signaling that the high-intensity exercise induces, activating the AMPK and, thus, the expression of PGC1-α, the main mediator of mitochondrial biogenesis [4]. Using the same SIT protocol of 30 s all-out, Gibala et al. [22] observed an acute twofold increase in PGC1-α mRNA above resting values. However, the AMPK phosphorylation can be also achieved through RT as long as it involves an overload stimulus [23]. Therefore, several studies support that the molecular signaling of mitochondrial biogenesis is enhanced with CT compared to each exercise mode alone [10, 24–26]. This is of particular interest for practitioners since it has been observed that CT (RT: 4 × 8 leg extension at 70% 1RM; ET: 20 min cycling at 55% of peak aerobic power output) can elicit a similar response to the double ET time (40 min cycling at 55% of peak aerobic power output) [25], which could give variety to the sessions of a training program with the same aim.

Given the effectiveness of CT compared to isolated exercises, it is necessary to determine if such benefits are due to the effect of an additional exercise or due to a synergistic effect. In line with the results of the present study, Coffey et al. [11] determined the acute molecular response to a CT (RT: 8 × 5 leg extension at 80% 1RM; ET: 30 min cycling at 70% of VO_{2max}), observing a significant time effect for PGC-1α, but not an interaction between both sequences. However, when the CT was composed of the same RT and a cycling SIT (10 × 6 s), there was a moderate exercise order effect in favor of RT followed by SIT [27].

The potential benefits derived from CT or a particular exercise sequence appear to reside in the metabolic environment that the previous exercise leaves in the other. It has been shown that a low carbohydrate availability augments the early signaling responses that mediate mitochondrial biogenesis [28, 29]. This enhanced response is well-established in ET [29]. However, this has been also observed in RT [30], which in conjunction with its glycogen depletion effects [31], reinforces its role in enhancing the muscle oxidative capacity and its synergistic effect with ET.

Conclusion

The results obtained revealed that the 8 week CT program composed of SIT and lower- and upper-body resistance exercises improved the muscle oxidative capacity of recreationally trained young adults regardless of the exercise sequence.

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Author contributions All authors have contributed equally in the elaboration of the manuscript.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no conflict of interest.

Human rights The study protocol adhered to the tenets of the Declaration of Helsinki and was approved by the institutional review board (ref. 2546/CEIH).

Informed consent All participants were informed about the research purpose and procedures of the study prior to signing a written informed consent form.

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