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Original Paper

Biomedical Human Kinetics, 10, 118–126, 2018
DOI: 10.1515/bhk-2018-0018

The acute effect of a single exhaustive sprint exercise session on post-exercise fat oxidation rate

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Summary

Study aim: It is well known that substrate oxidation rates are increased by exercise. The present study had two main objectives: firstly, to examine the effect of a single exhaustive exercise session on post-exercise substrate oxidation and energy expenditure; and secondly, to determine the differences between athletes and non-athletes.

Material and methods: Eighteen healthy male athletes (mean ± SD age; 19.38 ± 2.26 years, VO_{2max}; 60.57 ± 3.90 ml · kg^{-1} · min^{-1}, n = 8) and non-athletes (age; 20.30 ± 1.26 years, VO_{2max}; 44.97 ± 5.43 ml · kg^{-1} · min^{-1}, n = 10) volunteered to participate in the study. After an overnight fast, subjects performed a single sprint exercise session on a cycle ergometer with individual loads (0.075 kg per body weight) until volitional exhaustion. Energy expenditure (EE) and the substrate oxidation rate were measured at rest and during the post-exercise recovery period using indirect calorimetry.

Results: Exhaustive exercise significantly increased post-exercise fat oxidation, energy expenditure and contribution of fat to EE ($p < 0.05$). Also, it significantly decreased post-exercise carbohydrate (CHO) oxidation and the contribution of CHO to EE ($p < 0.05$). However, the changes in the substrate oxidation rate and EE after the exercise test were not different between the groups ($p > 0.05$).

Conclusions: The study results suggest that a single short-duration exhaustive exercise session causes a higher fat oxidation rate during recovery than at rest, whereas training status did not affect this situation.

Keywords: Fat oxidation – Exhaustive exercise – Energy expenditure – Post-exercise metabolism

Introduction

Relatively low rates of energy expenditure and fat oxidation predict body weight gain [48]. Chronic inactivity and inability to oxidize fat lead to lipid storage increases [40]. Although exercise has been demonstrated to provide clear physical and psychological benefits, many people continue to have an inactive life style. It is reported that barriers to exercise include a lack of adequate time, energy and motivation for exercise [25]. Additionally, many physical activities are perceived as boring or unenjoyable for many people. It is reported that people who enjoy performing an activity sustain the activity for a long time [36]. For these reasons, it is necessary to alter exercise planning so that it takes less time but is more efficient for energy expenditure and health benefits. An important aim is to increase the energy expenditure (EE) and fat oxidation rate not only during the exercise but also during the rest after the exercise. Increasing the relative contribution of lipids during the post-exercise period may be important for controlling body fat [48].

Energy expenditure is related to exercise volume and intensity. High-volume, low-intensity endurance exercise modalities have been demonstrated to increase EE [33] and the fat oxidation rate [37]. At the same exercise intensity, EE increases linearly with exercise duration [24]. Also, the magnitude of EE is related to exercise intensity [24, 42]. At low intensity exercise below 50% maximal oxygen consumption (VO_{2max}) the energy supplied is primarily from fat. However, as intensity increases the energy requirement is supplied from blood glucose, muscle glycogen and intramuscular triglyceride [7]. During high-intensity exercise, the fat oxidation rate decreases gradually, and CHO oxidation increases in parallel to exercise intensity [44].

Nevertheless, the effect of exercise on substrate metabolism and EE is not limited by exercise duration. It is

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demonstrated that the energy cost and substrate oxidation rate may be higher in the hours following exercise than at rest [42]. Several studies have reported increased post-exercise EE after resistance [33], aerobic and high-intensity interval exercise [31]. While many studies have focused on substrate mobilization during exercise [4, 7, 35], there has been limited research on post-exercise fat and CHO oxidation [2, 5, 21]. However, in these studies the exercise intensity was low (40–60% VO_{2max}) and the exercise durations were long (approximately 60 min). Kuo et al. [28] provided data showing increased fat oxidation rates at post-exercise duration. Relative exercise intensity in their study was 45–65% VO_{2max} and the exercise duration was 89–60 min. They reported that while CHO oxidation was a major source during intensive exercise, total lipid oxidation during the post-exercise period was greater than during the exercise (despite the long duration as noted above). In another study, no difference was observed in lipid oxidation after exercise at 45 and 65%VO_{2max} intensity [21].

There have been many studies focused on the metabolism after long-duration exercise. However, to our knowledge, no study has researched the effects of a short-duration single exhaustive exercise bout on post-exercise metabolism. Spending less time on exercise but gaining more in terms of EE and fat metabolism after the exercise are important. Therefore, to fully understand the effect of supramaximal intensity short-duration exercise on substrate metabolism during recovery, post-exercise recovery of normal weight individuals should be evaluated as regards the contribution of post-exercise fat oxidation to management of body fat. Primary prevention of overweight starts with maintenance of current body weight, not weight loss [11].

Most of the studies related to weight management and physical activity have been focused on overweight/obese individuals [26, 47]. However, this focus on weight management in these individuals has not extended to weight management in normal weight individuals. Few studies have been conducted with normal weight individuals, perhaps because normal weight is not considered problematic and hence is not a popular research topic [22]. It has been reported that there are some adaptations in endurance athletes that increase the fat oxidation rate through chronic training loads. Also, it is known that the fat oxidation rates are higher in endurance athletes than in non-athletes during high-intensity exercise [3].

We hypothesized that a high exercise load may cause different post-exercise metabolic changes between endurance athletes and non-athletes. For these reasons, the purpose of this study was to investigate post-exercise substrate oxidation after a single exhaustive short-duration exercise session in athletes and non-athletes.

Materials and methods

Subjects

Eight endurance trained athletes (cyclists) who compete at national and international levels and 10 sports science students who had no participation in regular physical activity within the previous year voluntarily participated in the study. Also, all subjects were young men, non-smokers and had no history of cardiovascular, metabolic, or respiratory diseases. In addition, dietary restrictions were not recommended for the subjects during the study period. The study was in accordance with the guidelines of the Helsinki Declaration. The subjects were provided with an explanation about the goals of the study and signed written, informed consent form to participate in the study, which was approved by Ethics Committee of Selçuk University Faculty of Sport Science.

Age, body weight, height, body mass index and body fat percentage were not significantly different between the groups ($p > 0.05$), while VO_{2max} was significantly higher in athletes than in non-athletes ($p < 0.05$) as shown in Table 1.

Table 1. Physical characteristics of participants

Variables	Non-athletes (n = 10)	Athletes (n = 8)
	Mean ± SD	Mean ± SD
Age [years]	20.30 ± 1.25	19.38 ± 2.26
Body weight [kg]	71.80 ± 7.57	68.94 ± 7.58
Body height [cm]	175.8 ± 4.91	176.5 ± 6.41
Body mass index [kg/m^2]	23.20 ± 1.67	22.12 ± 2.15
Body fat [%]	14.71 ± 3.43	12.16 ± 2.85
VO_{2max} [ml · kg · min^{-1}]	44.97 ±5.44	60.57 ± 3.64*

* – $p < 0.05$ – significantly different from non-athletes.

General design

The participants came to the laboratory three times. At the first visit, anthropometric measurements and the subjects' maximal pedal cadence were determined. At the second visit VO_{2max} was measured and at the last visit the exhaustive test was applied. At this stage, at rest and post-exercise substrate oxidation and EE were measured and calculated.

Furthermore, to familiarize the participants with the exercise procedures, the subjects performed the sprint exercise a week before the test.

Anthropometric measurements

The subjects' weight and height were measured with a Seca scale (Seca 711, Germany). Percent body fat was

calculated from body density using Siri's equation. Body density was calculated from four skinfold (SF) thicknesses using the equations of Durnin and Womersley [14].

$$\text{Body density} = 1.1631 - 0.0632 \cdot \log(\text{biceps SF} + \text{triceps SF} + \text{subscapular SF} + \text{suprailiac SF}),$$
$$\%\text{Body fat} = (4.95/\text{body density}) - 4.50.$$

Maximal oxygen uptake (VO_{2max})

VO_{2max} was determined with a progressive intensity cycle test (Monark 839E) by using a portable metabolic system (Cosmed, K5b[2], Rome, Italy). The athletes began the test with 100 W and the non-athlete group began with 50 W-70 rpm. Firstly, subjects warmed up at the initial loads for 3-min, then the workload was increased by 25 W every 2-min. Before each test, ambient conditions, gas analyzers and the turbine flowmeter were calibrated following the manufacturer's instructions. During the test, heart rate (HR) was monitored (S610i, Polar, Finland). Achievement of VO_{2max} was considered as the attainment of at least two of these criteria: 1) plateau in oxygen consumption (VO_2) despite increasing speed, 2) a respiratory exchange ratio (RER) (volume of carbohydrate production/volume of oxygen consumption) above 1.10, and 3) a HR within 10 beats per minute of age-predicted maximum HR (220 – age) [23].

Exhaustive exercise test

On the day before exhaustive exercise each subject consumed the same diet, which was designed as approximately 65% carbohydrate, 20% fat and 15% protein, and the subjects were not allowed to do any exercise.

Before the exhaustive test, the participants were required to come to the laboratory at 08.00 a.m. after an overnight fast (12 h). The subjects rested quietly for approximately 15 min and resting blood lactate levels were measured. Subjects then took a seat and resting respiratory values were measured with a facemask breath by breath.

After 15 min resting measures, a standardized warm-up of 5 min 50 W-60 rpm was applied before the exercise. Then, each subject undertook a single sprint exercise session on a cycle ergometer (Monark, 894E, Sweden) with individual loads (0.075 kg per body weight) until volitional exhaustion. Every test was begun with the individual predetermined maximal pedal rate ± 10 rpm. The subjects were encouraged to pedal in an "all-out" manner throughout the exercise. When the subjects could not maintain appropriate cadence (>50 rpm) the test ended. After the exercise, participants remained seated in the chair for 45 min during which breath-by-breath pulmonary gas exchange was measured continuously via the mask. All respiratory changes and substrate oxidation rates were calculated as the 15 min average before the test and 15, 30, and 45 min after the test.

HR was recorded continuously during the whole experimental period. Blood lactate levels were measured immediately after the test and after 30 min of recovery by capillary blood samples collected from a fingertip (Nova Biomedical Lactate Plus, Waltham, MA).

Calculations

The first 10 min of resting data were discarded, and the last 5 min of data were averaged. Post-exercise calculations were averaged every 15 min.

Substrate oxidation rates before and after the exercise were calculated according to the stoichiometric equations [15] assuming that the urinary nitrogen excretion rate was negligible:

$$\text{CHO oxidation } (g \cdot min^{-1}) = 4.55 \times VCO_2\ (l \cdot min^{-1}) - 3.21 \times VO_2\ (l \cdot min^{-1}).$$
$$\text{Fat oxidation } (g \cdot min^{-1}) = 1.67 \times VO_2\ (l \cdot min^{-1}) - 1.67 \times VCO_2\ (l \cdot min^{-1}).$$

Energy expenditure at rest and the 45-min recovery period after the exercise test were calculated from Weir's equation [46].

All ventilatory changes and fat-CHO oxidation rates and EE were averaged at 15 min before and after the test.

The relative contributions of fat and CHO oxidation to energy expenditure were calculated using the following equations: [13]

$$\%\text{Fat} = [(1\text{-RER})/0.29] \times 100$$
$$\%\text{CHO} = [(\text{RER-}0.71)/0.29] \times 100$$

Statistical analysis

A two-way split-plot ANOVA (mixed ANOVA) with repeated measures was used to test the effect of exhaustive exercise on substrate oxidation. The two factors were group (athletes and non-athletes) and time (15-min means before and after the exercise). When the repeated measures effect (time) was significant in split-plot ANOVA, one-way repeated measures analysis of variance with the post hoc Bonferroni test was applied to identify the effect of exhaustive exercise on metabolism. Statistical significance was set at a level of $\alpha < 0.05$ and data were expressed as the means ± standard deviation.

Results

The test time, peak power, mean power, ventilation (VE), RER, and HR were similar in the athletes and non-athletes during the exercise test ($p > 0.05$). However, VO_2 and VCO_2 were significantly different between groups ($p < 0.05$). The lactate at rest and after the test were similar in both groups ($p > 0.05$) and lactate after 30 min was lower in the athletes than in non-athletes ($p < 0.05$).

The oxygen uptake was significantly different among the measurement periods (at rest and three recovery

Table 2. Exhaustive exercise test variables and lactate levels

Variables	Non-athletes (n = 10)	Athletes (n = 8)
	Mean ± SD	Mean ± SD
Peak power [watt]	878.3 ± 237.8	929.5 ± 328.8
Mean power [watt]	502.3 ± 61.35	477.3 ± 135.4
Test duration [sec]	48.70 ± 6.99	46.88 ± 9.67
Expired minute volume [l/min]	129.6 ± 20.81	143.7 ± 14.79
Oxygen uptake [ml/min]	2423 ± 329.2	3156 ± 340.0*
Carbon dioxide production [ml/min]	3562 ± 495.7	4439 ± 363.5*
Respiratory exchange ratio	1.47 ± 0.19	1.42 ± 0.14
Heart rate [beat/min]	169.9 ± 15.31	170.7 ± 14.76
Lactate at rest [mmol/l]	1.48 ± 0.32	1.20 ± 0.23
Lactate after test [mmol/l]	12.92 ± 1.93	12.99 ± 1.53
Lactate 30 min after recovery [mmol/l]	9.68 ± 1.58	7.35 ± 1.81*

* – $p < 0.05$, significantly different from non-athletes.

phases) for the athletes and non-athletes (time effect; $F_{3,48} = 281.66$, $p < 0.05$). Oxygen uptake was significantly higher after the exercise test during all recovery periods compared with at rest. Afterwards, it decreased significantly during the recovery period ($p < 0.05$). Oxygen uptake at rest and during recovery periods were not significantly different between the groups (group effect; $F_{1,16} = 0.02$, $p > 0.05$). However, changes in oxygen uptake during the measurement periods were significantly different between the athletes and the non-athletes (group-time interaction effect; $F_{3,48} = 3.39$, $p < 0.05$). Oxygen uptake after a 30 min recovery period returned to the resting levels in athletes, while oxygen uptake after 45 min of recovery was still significantly higher than at resting levels in non-athletes ($p < 0.05$).

The respiratory exchange ratio (RER) was similar in both groups (group effect; $F_{1,16} = 2.72$, $p > 0.05$). RER decreased after 30 min of recovery in the groups (time effect; $F_{3,48} = 504.52$). The changes in RER at rest and during recovery were not different between the groups (group-workout interaction effect; $F_{3,48} = 0.79$, $p > 0.05$).

Fat oxidation rates significantly increased after a 30 min recovery period compared to at rest in the groups (time effect; $F_{3,48} = 109.39$, $p < 0.05$) (3- and 2.3-fold for the non-athletes and athletes, respectively). The fat oxidation rate in each period and its changes during the experiment were not different between groups (group effect; $F_{1,16} = 0.43$, group-time interaction effect; $F_{3,48} = 0.99$, $p > 0.05$) (Fig. 1a). Likewise, the changes in CHO oxidation rate during the experiment were similar for the groups (group effect; $F_{1,16} = 0.02$, group-time interaction effect; $F_{3,48} = 1.33$, $p > 0.05$). CHO oxidation significantly increased in the first 15 min then it decreased after the second 15 min during recovery (time effect; $F_{3,48} = 109.39$,

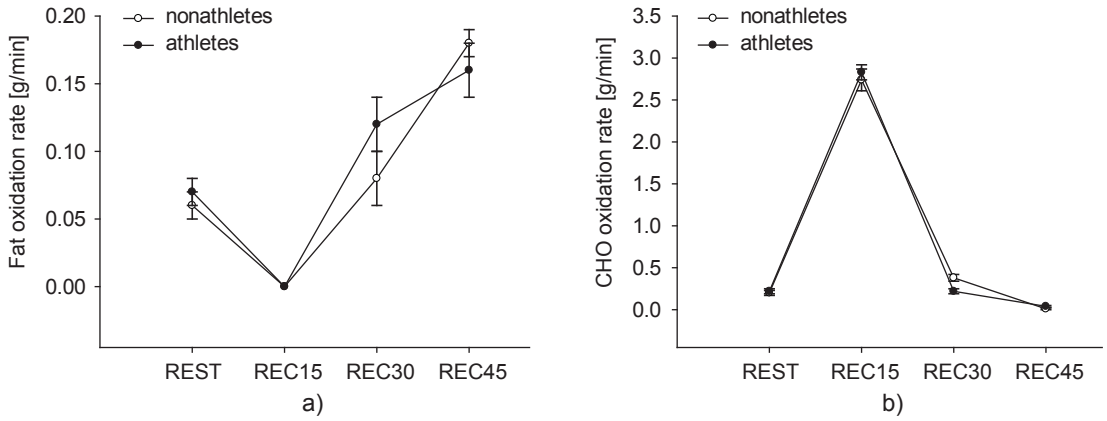

Fig. 1. a) Fat oxidation rate at rest and during recovery; b) Carbohydrate oxidation rate at rest and during recovery

$p < 0.05$) (20- and 5.5-fold for the non-athletes and athletes, respectively) (Fig. 1b).

EE at rest and in recovery periods were not significantly different between the groups (group effect; $F_{1,16} = 0.01$, $p > 0.05$). On the other hand, the changes in EE during the measurement periods were significantly different between the groups (group-time interaction effect; $F_{3,48} = 3.28$, $p < 0.05$). EE was significantly higher during all the recovery periods compared with at rest (time effect; $F_{3,48} = 412.22$, $p < 0.05$) (Fig. 2).

EE increased during the first 15 min recovery period ~300% for both groups although the fat oxidation contribution to EE was zero. The EE was higher in the second 15 min recovery period in the young adult athletes (~31%) and non-athletes (~64%) compared to resting values. However, the fat contribution to energy expenditure was ~60% in the athletes and ~40% in the non-athletes. During the third 15 min in recovery, the EE was still higher than at rest in the athletes (~7%) and the non-athletes (~3%), while the fat contribution to energy expenditure was ~97% in both groups.

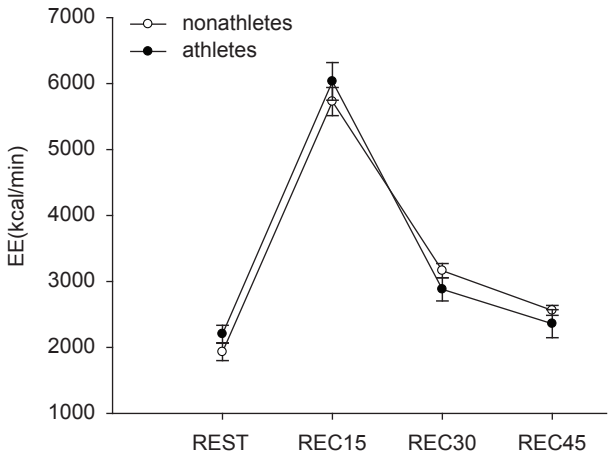

Fig. 2. Energy expenditure during at rest and recovery periods

Discussion

The main finding of the present study was that a single exhaustive sprint exercise session increased post-exercise fat oxidation (3- and 2.3-fold for the non-athletes and athletes, respectively), energy expenditure and contribution of fat to EE (51.7 and 47.9% for the non-athletes and athletes, respectively) despite decreased post-exercise CHO oxidation (20- and 5.5-fold for the non-athletes and athletes, respectively) and contribution of CHO to EE.

During high-intensity exercise, substrate oxidation rates which are quantified by indirect calorimetry can lead to overestimation of CHO and underestimation of fat oxidation [34]. For this reason, we ignored the substrate oxidation rates during exercise. We focused on post-exercise substrate oxidation rates and EE. Although there have been many studies on the effect of any exercise on post-exercise oxygen consumption, there have been limited studies examining the effect of exercise on post-exercise recovery substrate oxidation rates [6, 29, 43]. Chan and Burns [9] found increased 2-h post-exercise oxygen consumption and fat oxidation rates after interval exercise comprising four 30 s sprints. Similar studies using interval sprint exercises during several weeks have reported increased fat oxidation during rest and/or exercise [8, 16, 50]. The most important difference of this study is that only a single sprint exercise session caused an increase in post-exercise recovery fat metabolism and EE and fat contribution to total EE. To our knowledge there has not been any study using a short-duration single supramaximal sprint exercise session.

After cessation of exercise, the VO_2 does not recover and remains elevated above resting values, referred to as excess post-exercise oxygen consumption (EPOC) [18]. This elevated post-exercise metabolism contributes to phosphagen resynthesis, lactate removal, restoration of body temperature level and triacylglycerol-fatty acid cycling. EPOC magnitude and duration are influenced by exercise intensity and duration [6, 32, 38]. In this study, during the post-exercise recovery, VO_2, VCO_2, RER, and the fat-CHO oxidation rate changed. As substrate oxidations were calculated from VO_2 and VCO_2, the magnitude of the post-exercise oxygen consumption is important. After the cessation of exercise, the CHO oxidation rate was higher than the fat oxidation rate for 15 min after exercise. After 30 min, the fat oxidation rate was higher than resting values and CHO decreased. This study showed that during the post-exercise recovery following a single exhaustive sprint, the fat oxidation rate increased significantly even though the CHO oxidation rate decreased.

The exercise intensity was reported as the major effect as a stimulus on post-exercise EE [20]. Treuth et al. [42] demonstrated that high-intensity exercise increased EE more than low-intensity exercise but there was similar substrate oxidation 24 h after exercise. It is important to note that increased EE may be supplied by fat oxidation. Also, the percentage contribution of fat increased significantly in post-exercise recovery. Warren et al. researched the effects of different duration and intensity exercises on post-exercise metabolism and they found that after high-intensity exercise fat oxidation increased [45]. Nevertheless, they used 85% VO_{2max} intensity and 12–15 min exercise duration. In this study, we measured higher fat oxidation rates but used lower total exercise duration. Kimber et al. [27] observed increased free fatty acid levels after exhaustive exercise (90 min) in endurance trained men. Whyte et al. [49] demonstrated that cycle sprint of ~200 s increased

Table 3. Changes in substrate oxidation for non-athlete and athlete groups at rest and during the recovery periods

Variables	Time	Non-athletes (n = 10)	Athletes (n = 8)	ANOVA F value		
		Mean ± SD	Mean ± SD	T	G	T × G
VO_2 [ml]	Rest	273.55 ± 57.96^{abcd}	312.34 ± 18.32^{ab}			
	15 min recovery	700.40 ± 81.76^{bacd}	740.62 ± 39.30^{bacd}	281.66*	0.02	3.39*
	30 min recovery	445.44 ± 50.57^{cabd}	412.07 ± 26.27^{cbd}			
	45 min recovery	376.34 ± 34.75^{dabc}	345.72 ± 31.15^{dbc}			
RER	Rest	0.86 ± 0.05^{abd}	0.86 ± 0.04^{abd}			
	15 min recovery	1.48 ± 0.09^{bacd}	1.45 ± 0.11^{bacd}	504.52*	2.72	0.79
	30 min recovery	0.89 ± 0.06^{cbd}	0.83 ± 0.06^{cbd}			
	45 min recovery	0.70 ± 0.03^{dabc}	0.69 ± 0.06^{dabc}			
Fat oxidation [g/min]	Rest	0.06 ± 0.02^{abd}	0.07 ± 0.02^{abcd}			
	15 min recovery	0.00 ± 0.00^{bacd}	0.00 ± 0.00^{bacd}	109.39*	0.43	3.29
	30 min recovery	0.08 ± 0.05^{cbd}	0.12 ± 0.05^{cabd}			
	45 min recovery	0.18 ± 0.02^{dabc}	0.16 ± 0.05^{dabc}			
Carbohydrate oxidation	Rest	0.20 ± 0.08^{abcd}	0.22 ± 0.08^{abd}			
[g/min]	15 min recovery	2.74 ± 0.43^{bacd}	2.83 ± 0.24^{bacd}	833.95*	0.02	1.33
	30 min recovery	0.38 ± 0.13^{cabd}	0.22 ± 0.08^{cbd}			
	45 min recovery	0.01 ± 0.02^{dabc}	0.04 ± 0.04^{dabc}			
Energy expenditure	Rest	1931.40 ± 415.98^{abcd}	2203.39 ± 373.63^{abc}			
[kcal/min]	15 min recovery	5727.56 ± 674.03^{bacd}	6032.34 ± 807.26^{bacd}	412.22*	0.01	3.28*
	30 min recovery	3163.29 ± 342.46^{cabd}	2881.10 ± 497.67^{cabd}			
	45 min recovery	2561.01 ± 239.03^{dacd}	2359.88 ± 599.06^{dcb}			
Fat oxidation contribution	Rest	47.29 ± 16.90^{abcd}	49.01 ± 14.88^{abcd}			
to energy expenditure [%]	15 min recovery	0.00 ± 0.00^{bacd}	0.00 ± 0.00^{bacd}	156.67*	2.38	2.71
	30 min recovery	39.54 ± 21.93^{cabd}	60.22 ± 21.00^{cabd}			
	45 min recovery	98.97 ± 2.11^{dabc}	96.86 ± 5.48^{dabc}			
Carbohydrate oxidation contribution to energy expenditure [%]	Rest	52.71 ± 16.90^{abd}	50.99 ± 14.88^{abd}			
	15 min recovery	100.00 ± 0.00^{bacd}	100.00 ± 0.00^{bacd}	156.67*	2.38	2.71
	30 min recovery	60.46 ± 21.93^{cbd}	39.78 ± 21.00^{cbd}			
	45 min recovery	1.03 ± 2.11^{dabc}	3.14 ± 5.48^{dabc}			

$^{a–d}$ The same superscripts in the same column indicate significant differences (repeated one-way ANOVA with Bonferroni, $p < 0.05$). * – $p < 0.05$, significant main and/or interaction effect (split-plot ANOVA with repeated measures). T = time effect, G = group effect, T × G = time-group interaction.

the insulin sensitivity index and post-exercise fat oxidation rate in overweight and/or obese men. It has been reported that increased catecholamine release is a potential stimulus for the post-exercise fat oxidation rate [41]. We did not measure catecholamines but high rates of lipolysis after supramaximal intervals are explained by increased catecholamine levels.

One of the major findings of this study was the high lactate levels in both groups after cessation of exercise. The high blood lactate values after exercise are consistent with glycolysis. Although post-exercise lactate values were similar in both groups, the trained group had lower lactate levels than the untrained group at the 30th min of recovery. During maximal sprint exercises, the anaerobic glycolysis and phosphocreatine breakdown meet the energy demands first [51]. Lactate, an end product of the anaerobic glycogen, fluxes to the blood. The higher blood lactate concentrations at 30 min of the post-exercise period in the untrained group may have occurred because of slower recovery than in trained individuals. Trained subjects demonstrated better fast recovery of lactate removal. The increased lactate carrier proteins via chronic

endurance training may affect the post-exercise lactate recovery [12, 17]. It was reported that after chronic endurance training, lactate elimination occurred faster [10] because of the increased capillarization [12] and lactate carrier proteins. Also, increased mitochondrial volume and density accelerates the elimination of lactate from the blood [39].

While there have been extensive studies focused on the fat and CHO oxidation rates during different types of exercise, less research has been conducted concerning metabolic adaptations in post-exercise recovery. Moreover, there has been limited research addressing the effect of short-duration exhaustive exercise on post-exercise lipid metabolism. In the literature, exhaustive exercises were applied for long durations, as mentioned above. After the exercise, fat oxidation rate, EE, and % contribution of fat increased significantly. This is important for individuals who lack sufficient time for long-duration exercise and who want to maintain their body mass in the normal range without spending a long time on exercise. It is stated that the magnitude and duration of the post-exercise recovery metabolism depend on the exercise intensity [20]. Increased post-exercise fat oxidation might be useful for weight management to achieve a negative energy balance.

The regulation of the substrate used during exercise is complex and controlled by several factors. It has been demonstrated that higher fat oxidation rates can occur during low to moderate intensity long-duration exercise [19]. For this reason, many people assume that in order to reduce body fat the exercise intensity must be moderate or low. However, this study showed that post-exercise fat oxidation could increase by ~300%. This fat oxidation and EE increase during recovery is very important for people who have inadequate time or lack motivation for lengthy exercises.

It has been shown that the substrate oxidation rate is different in trained and untrained individuals during exercise. Some studies showed that trained individuals have a higher maximal fat oxidation rate than untrained subjects [1]. Moreover, it was reported that trained individuals have a higher fat oxidation rate at the same exercise intensity with different durations [30]. Nevertheless, there has been limited information about the effect of physical form on the post-exercise substrate oxidation rate. In this study, we did not measure the maximal fat oxidation rate, but this study shows that before and after the exhaustive exercise, a similar fat oxidation rate was recorded in both groups.

This study has some limitations: the number of subjects was low, and it would have been better to test this method with a wide range of subjects. Participants were healthy and of normal weight, so it may be informative to conduct this study in overweight subjects. Also, we used the Wingate cycle ergometer in this study. This is not practical for everyone, so this method could be used with other sprint protocols. Additionally, we tested the effect of exhaustive sprint exercise on the post-exercise period only for a single session. We do not have any information about the effects of this exercise when applied periodically. Thus, there is a need for further studies about this situation. Also, the participants were fasting overnight, and there is a need to test post-exercise fat oxidation several hours after nutrition or when food is given in the recovery period.

In this study, elevated post-exercise metabolism was similar in endurance athletes and non – athletes. This increased metabolism is important to achieve fat mobilization for weight management. This study examined acute responses of substrate oxidation to a single exhaustive sprint exercise session and found increases in fat oxidation and EE and a decrease in CHO oxidation rate after exercise. These changes may contribute to the effects of a single sprint exercise session on body fat and composition [50]. Additional studies are still necessary to evaluate the effects of daily single exhaustive exercise sessions on body fat when applied as part of a regular training program.

Conflict of interest: Authors state no conflict of interest.

References

1. Achten J., Jeukendrup A.E. (2003) Maximal fat oxidation during exercise in trained men. *Int. J. Sports. Med.,* 24: 603-608.
2. Al Mulla N., Simonsen L., Bulow J. (2000) Post-exercise adipose tissue and skeletal muscle lipid metabolism in humans: the effects of exercise intensity. *J. Physiol.,* 524: 919-928.
3. Aslankeser Z., Balcı Ş.S. (2017) Re-examination of the contribution of substrates to energy expenditure during high-intensity intermittent exercise in endurance athletes. *Peer J.*, e3769. DOI: 10.7717/peerj.3769.
4. Bergman B.C., Brooks G.A. (1999) Respiratory gas-exchange ratios during graded exercise in fed and fasted trained and untrained men. *J. Appl. Physiol.,* 86: 479-487.
5. Bielinski R., Schutz Y., Jéquier E. (1985) Energy metabolism during the postexercise recovery in man. *Am. J. Clin. Nutr.*, 42: 69-82.
6. Børsheim E., Bahr R. (2003) Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. *Sports Med.,* 33: 1037-1060.
7. Brooks G.A., Mercier J. (1994) Balance of carbohydrate and lipid utilization during exercise: the "crossover" concept. *J. Appl. Physiol.,* 76: 2253-2261.
8. Burgomaster K.A., Howarth K.R., Phillips S.M., Rakobowchuk M., Macdonald M.J., McGee S.L.,

Gibala M.J. (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *J. Physiol.,* 586: 151-160.
9. Chan H.H., Burns S.F. (2013) Oxygen consumption, substrate oxidation, and blood pressure following sprint interval exercise. *Appl. Physiol. Nutr. Metab*., 38: 182-187.
10. Cipryan L., Tschakert G., Hofmann P. (2017) Acute and post-exercise physiological responses to high-intensity interval training in endurance and sprint athletes. *J. Sports. Sci. Med.,* 16: 219-229.
11. Donnelly J.E., Blair S.N., Jakicic J.M., Manore M.M., Rankin J.W., Smith B.K. (2009) American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med. Sci. Sports. Exerc.*, 41: 459-471.
12. Dubouchaud H., Butterfield G.E., Wolfel E.E., Bergman B.C., Brooks G.A. (2000) Endurance training, expression, and physiology of LDH, MCT1, and MCT4 in human skeletal muscle. *Am. J. Physiol. Endocrinol. Metab.,* 278: E571-579.
13. Dumortier M., Thöni G., Brun J.F., Mercier J. (2005) Substrate oxidation during exercise: impact of time interval from the last meal in obese women. *Int. J. Obes.* (Lond.)., 29: 966-974.
14. Durnin J.V., Womersley J. (1974) Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br. J. Nutr.,* 32: 77-97.
15. Frayn K.N. (1983) Calculation of substrate oxidation rates in vivo from gaseous exchange. *J. Appl. Physiol. Respir. Environ. Exerc. Physiol.,* 55: 628-634.
16. Freese E.C., Levine A.S., Chapman D.P., Hausman D.B., Cureton K.J. (2011) Effects of acute sprint interval cycling and energy replacement on postprandial lipemia. *J. Appl. Physiol.,* 111: 1584-1589.
17. Frey G.C., Byrnes W.C., Mazzeo R.S. (1993) Factors influencing excess postexercise oxygen consumption in trained and untrained women. *Metabolism,* 42: 822-828.
18. Gaesser G.A., Brooks G.A. (1984) Metabolic bases of excess post-exercise oxygen consumption: a review. *Med. Sci. Sports. Exerc*., 16: 29-43.
19. González-Haro C., Galilea P.A., González-de-Suso J.M., Drobnic. F, Escanero J.F. (2007) Maximal lipidic power in high competitive level triathletes and cyclists. *Br. J. Sports. Med.*, 41: 23-28.
20. Gore C.J., Withers R.T. (1990) The effect of exercise intensity and duration on the oxygen deficit and excess post-exercise oxygen consumption. *Eur. J. Appl. Physiol. Occup. Physiol.,* 60: 169-174.
21. Henderson G.C., Fattor J.A., Horning M.A., Faghihnia N., Johnson M.L., Mau T.L., Luke-Zeitoun M., Brooks G.A. (2007) Lipolysis and fatty acid metabolism in men and women during the postexercise recovery period. *J. Physiol.,* 584: 963-981.
22. Hernandez C.A., Hernandez D.A., Wellington C.M., Kidd A. (2016) The experience of weight management in normal weight adults. *Appl. Nurs. Res.,* 32: 289-295.
23. Howley E.T., Bassett D.R., Welch H.G. (1995) Criteria for maximal oxygen uptake: review and commentary. *Med. Sci. Sports. Exerc.,* 27: 1292-1301.
24. Hunter G.R., Weinsier R.L., Bamman M.M., Larson D.E. (1998) A role for high intensity exercise on energy balance and weight control. *Int. J. Obes. Relat. Metab. Disord.,* 22: 489-493.
25. Justine M., Azizan A., Hassan V., Salleh Z., Manaf H. (2013) Barriers to participation in physical activity and exercise among middle-aged and elderly individuals. *Singapore Med. J.,* 54: 581-586.
26. Keating S.E., Johnson N.A., Mielke G.I., Coombes J.S. (2017) A systematic review and meta-analysis of interval training versus moderate-intensity continuous training on body adiposity. *Obes. Rev.;* 18: 943-964.
27. Kimber N.E., Cameron-Smith D., McGee S.L., Hargreaves M. (2013) Skeletal muscle fat metabolism after exercise in humans: influence of fat availability. *J. Appl. Physiol.*, 114: 1577-1585.
28. Kuo C.C., Fattor J.A., Henderson G.C., Brooks G.A. (2005) Lipid oxidation in fit young adults during postexercise recovery. *J. Appl. Physiol.,* 99: 349-356.
29. LaForgia J., Withers R.T., Gore C.J. (2006) Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. *J. Sports Sci.,* 24: 1247-1264.
30. Lima-Silva A.E., Bertuzzi R.C., Pires F.O., Gagliardi J.F., Barros R.V., Hammond J., Kiss M.A. (2010) Relationship between training status and maximal fat oxidation rate. *J. Sports. Sci. Med*.; 9: 31-35.
31. Malatesta D., Werlen C., Bulfaro S., Chenevière X., Borrani F. (2009) Effect of high-intensity interval exercise on lipid oxidation during postexercise recovery. *Med. Sci. Sports Exerc.,* 41: 364-374.
32. Melanson E.L., Sharp T.A., Seagle H.M., Horton T.J., Donahoo W.T., Grunwald G.K., Hamilton J.T., Hill J.O. (2002) Effect of exercise intensity on 24-h energy expenditure and nutrient oxidation. *J. Appl. Physiol.,* 92: 1045-1052.
33. Melby C., Scholl C., Edwards G., Bullough R. (1993) Effect of acute resistance exercise on postexercise energy expenditure and resting metabolic rate. *J. Appl. Physiol.,* 75: 1847-1853.
34. Romijn J.A., Coyle E.F., Hibbert J., Wolfe R.R. (1992) Comparison of indirect calorimetry and a new breath 13C/12C ratio method during strenuous exercise. *Am. J. Physiol.,* 263: 64-71.
35. Romijn J.A., Coyle E.F., Sidossis L.S., Rosenblatt J., Wolfe R.R. (2000) Substrate metabolism during different

exercise intensities in endurance-trained women. *J. Appl. Physiol.,* 88: 1707-1714.
36. Russel W.D., Newton M. (2008) Short-term psychological effects of interactive video game technology exercise on mood and attention. *Educ. Tech. Soc.,* 11: 294-308.
37. Schrauwen P., van Aggel-Leijssen D.P., Hul G., Wagenmakers A.J., Vidal H., Saris W.H., van Baak M.A. (2002) The effect of a 3-month low-intensity endurance training program on fat oxidation and acetyl-CoA carboxylase-2 expression. *Diabetes*, 51: 2220-2226.
38. Sedlock D.A., Fissinger J.A., Melby C.L. (1989) Effect of exercise intensity and duration on postexercise energy expenditure. *Med. Sci. Sports Exerc.,* 21: 662-666.
39. Thomas C., Sirvent P., Perrey S., Raynaud E., Mercier J. (2004) Relationships between maximal muscle oxidative capacity and blood lactate removal after supramaximal exercise and fatigue indexes in humans. *J. Appl. Physiol*., 97: 2132-2138.
40. Thyfault J.P., Kraus R.M., Hickner R.C., Howell A.W., Wolfe R.R., Dohm G.L. (2004) Impaired plasma fatty acid oxidation in extremely obese women. *Am. J. Physiol. Endocrinol. Metab.*, 287: 1076-1081.
41. Trapp E.G., Chisholm D.J., Boutcher S.H. (2007) Metabolic response of trained and untrained women during high-intensity intermittent cycle exercise. *Am. J. Physiol. Regul. Integr. Comp. Physiol.,* 293: 2370-2375.
42. Treuth M.S., Hunter G.R., Williams M. (1996) Effects of exercise intensity on 24-h energy expenditure and substrate oxidation. *Med. Sci. Sports Exerc*., 28: 1138-1143.
43. Tucker W.J., Angadi S.S., Gaesser G.A. (2016) Excess postexercise oxygen consumption after high-intensity and sprint interval exercise, and continuous steady-state exercise. *J. Strength. Cond. Res*., 30: 3090-3097.
44. Venables M.C., Achten J., Jeukendrup A.E. (2005) Determinants of fat oxidation during exercise in healthy men and women: a cross-sectional study. *J. Appl. Physiol.*, 98: 160-167.
45. Warren A., Howden E.J., Williams A.D., Fell J.W., Johnson N.A. (2009) Postexercise fat oxidation: effect of exercise duration, intensity, and modality. *Int. J. Sport Nutr. Exerc. Metab.,* 19: 607-623.
46. Weir J.B. (1949) New methods for calculating metabolic rate with special reference to protein metabolism. *J. Physiol.,* 109:1-9.
47. Wewege M., van den Berg R., Ward R.E., Keech A. (2017) The effects of high-intensity interval training vs. moderate-intensity continuous training on body composition in overweight and obese adults: a systematic review and meta-analysis. *Obes. Rev.,* 18: 635-646.
48. Weyer C., Pratley R.E., Salbe A.D., Bogardus C., Ravussin E., Tataranni P.A. (2000) Energy expenditure, fat oxidation, and body weight regulation: a study of metabolic adaptation to long-term weight change. *J. Clin. Endocrinol. Metab.,* 85: 1087-1094.
49. Whyte L.J., Ferguson C., Wilson J., Scott R.A., Gill J.M. (2013) Effects of single bout of very high-intensity exercise on metabolic health biomarkers in overweight/obese sedentary men. *Metabolism,* 62: 212-219.
50. Whyte L.J., Gill J.M., Cathcart A.J. (2010) Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metabolism,* 59: 1421-1428.
51. Withers R.T., Sherman W.M., Clark D.G., Esselbach P.C., Nolan S.R., Mackay M.H., Brinkman M. (1991) Muscle metabolism during 30, 60 and 90 s of maximal cycling on an air-braked ergometer. *Eur. J. Appl. Physiol. Occup. Physiol*., 63: 354-362.

Received 21.03.2018
Accepted 30.07.2018

Acknowledgments

The study was supported by Selçuk University, Scientific Research Projects (S.Ü.-BAP Project No. 17401076).