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ORIGINAL ARTICLE

Assessing the Effectiveness of Anaerobic Threshold and Respiratory Compensation Point on Fat and Carbohydrate Oxidations During Exercise in Sedentary Males

Anaerobic Threshold and Substrate Oxidation

Seda Ugras[1] Sermin Algul[2], Oguz Ozcelik[3]

[1] Department of Physiology, Bozok University, Faculty of Medicine, Yozgat, Turkey, email: sedaugras@hotmail.com (seda.ugras@bozok.edu.tr)

[2] Department of Physiology, Van Yuzuncu Yil University, Faculty of Medicine, Van, Turkey, e-mail: serminalgul@hotmail.com or serminalgul@yyu.edu.tr

[3] Department of Physiology, Kastamonu University, Faculty of Medicine, Kastamonu, Turkey, e-mail: oozcelik@kastamonu.edu.tr

Abstract

Study Objectives: Anaerobic threshold (AT) and respiratory compensation point (RCP) are two important metabolic set points. We aimed to determine the effects of exercise intensity at AT and RCP on the balance of substrate oxidation rates. ***Methods:*** Eleven male participants performed an incremental exercise test to exhaustion on a cycle ergometer to estimate AT and RCP. Subsequently, we conducted three 30 minute constant load exercise tests at AT (W_{AT}), RCP (W_{RCP}) and 25% below AT ($W_{<AT}$) in a randomized order. Pulmonary gas exchange parameters measured breath-by-breath. We estimated substrate oxidation rate by using Frayn equations. ***Results:*** We found that AT and RCP occurred at a mean intensity of 60% (range between 53-64% of VO_2peak) and 72% of VO_2peak (range between 66-76% of VO_2peak) respectively. Fat oxidation was found to be 0.221 ±0.01 g/min at $W_{<AT}$ and this significantly increased to 0.340±0.01 g/min at W_{AT} and 0.326±0.03 g/min at W_{RCP} ($p<0.05$). ***Conclusion:*** We found that carbohydrate oxidation was 1.621±0.03 g/min ($W_{<AT}$), 1.961±0.02 g/min (W_{AT}) and 2.417±0.1 g/min (W_{RCP}) ($p<0.05$). AT and RCP provides optimal metabolic strain to all participant and stimulate more fat oxidations. Thus clinicians should consider using exercise intensity at AT and RCP to achieve the rate of highest fat oxidation.

Key words: Exercise, Anaerobic Threshold, Respiratory Compensation Point, Fat Oxidation, Carbohydrate Oxidation, Respiratory Quotient, Metabolism

Introduction

During exercise, carbohydrate and fat are two dominant substrates oxidized by skeletal muscles for energy production[1]). However, absolute and relative contributions of fat and carbohydrate oxidations may be affected by many factors, including exercise intensity, exercise duration, substrate availability, gender differences, sympathetic activity and training status[2]). It is commonly believed that an impaired ability to oxidize fat and carbohydrate may be an important factor in the etiology of obesity[3]), and diabetes[4]).

Importantly, exercise intensity is perceived as a main factor in determining the rate of fat and carbohydrate oxidation[5]). Optimal fat oxidation can be achieved across a range of work rates between low and moderate exercise intensity[6, 7]). Research studies have shown that increasing workload from moderate to high intensity results in a shift in substrate utilization from fat to carbohydrate and carbohydrate becomes obligatory metabolic substrate during severe anaerobic exercise[5]). Exercise intensity is generally determined by a certain fixed percentage of an individuals' maximal O_2 uptake (VO_2max) (i.e. 25, 50, 65, 70, 85 % of VO_2max)[6, 8, 9]) or maximal exercise capacity (i.e. 50% of Wmax)[10]). However, application of a fixed workload (as a percentage of VO_2max or Wmax) to individuals may not be assumed to result in similar metabolic strain being placed upon all of them. It has been suggested that to prescribe exercise intensity, researchers and clinicians should consider the metabolic demands of an exercise by applying a threshold concept rather than the concept of a fixed percentage of maximal values[11]). A threshold concept defines specific metabolic phases during exercise and thus it might reflect the individual metabolic stress levels of participants[1, 12]). The anaerobic threshold (AT) and respiratory compensation point (RCP) are two specific set points that widely used in clinical and sports medicine for establishing optimal exercise intensity for each subject[1]).

It is important to establish fat and carbohydrate oxidation under similar metabolic intensity rather than fixed percentage of workload or intensity for each individual[3]). To the best of our knowledge there have been no studies comparatively evaluated the rate and amount of fat and carbohydrate oxidations at intensities corresponding to AT and RCP. In the present study, we intended to evaluate impact of specific metabolic intensities of single exercise sessions at the AT and RCP (and also 25% <AT) on the balance of fat and carbohydrate oxidations in a sample of young, sedentary participants.

Material and Method

Participants

Eleven healthy young male (mean±SD age: 20.8±1.9 years, height: 184±9 cm, weight 74.9±5.8 kg, and body mass index: 22±2 kg/m^2) participated in this study. The study protocol was approved by the Ethical Committee of Firat University by the ethical guidelines of the 1964 Declaration of Helsinki). All participants gave written informed consent based on university approved documents after the nature of the exercise trials and all potential risks and benefits were explained to them.

We screened all participants before the study to ensure that they were free from illness and any physical and metabolic limitations. Exclusion criteria were as follows: taking medications and having a history of any diseases related to the cardiovascular, respiratory, liver, renal, musculoskeletal, neuromuscular and metabolic systems. We asked all participants to refrain from eating, smoking, consuming caffeine, drugs or alcohol, taking any ergogenic aid and strenuous exercise. In addition, participants were asked to fill in a 3-day food and liquid intake details (for breakfast, lunch dinner and any additional meals) and they were asked to not change this diet before all subsequent experiments.

All participants attended the Human Exercise Physiology Laboratory before the study between 8:00 am to 9:00 am, following an overnight fasting state. The subject was introduced to the experimental procedures and anthropometric measurements (i.e. height weight, body mass and body composition) were taken (Body Fat Analyser, Tanita, TBF 300, Japan). The conditions of the Human Exercise Physiology Laboratory were controlled so that air temperature and humidity were the same for all exercise-testing sessions.

Exercise Tests Procedures

Each subjects completed four test sessions. Each subject initially performed a standardised rapid incremental exercise cycling protocol using an electromagnetically-braked cycle ergometer (VIA sprintTM 150/200P, Germany). Participants were able to adjust the saddle height so that it was in the most suitable position for their leg. As a warm-up period, we started the exercise work protocol with participants cycling at 20 W (60 rpm) for a period of four minutes. Then, we increased the work rate by a 15 W/min using a work rate controller and continued to the participants' limit of tolerance[14]). Participants were carefully controlled in order to avoid hyperventilation and to achieve a physiological steady-state condition during the warm-up period so that we could make a valid estimate of AT[15]). In addition, prior to exercise, it is strictly avoided from an acute negative energy balance and reduced muscle glycogen levels that may have effects on fat oxidation during exercise[16]).

All participants were tested in a random order in three constant load exercise test conditions separated by approximately three days. For the subsequent tests, as a warm-up period, participants performed an initial work load of 20 W for four minutes, then we increased the work using their individually determined work rate to their AT (W_{AT}), respiratory compensation point (W_{RCP}) and 25% below their AT ($W_{<AT}$) and they continued to cycling for 30 minutes. The all trials were always performed in the morning (between 8:00 a.m. to 9:00 a.m.) after an overnight fast and at the same time to avoid circadian variance.

Estimation of AT and RCP

The peak VO_2 (VO_2peak) was taken as the highest VO_2 at the end of the ramp test. AT was estimated from the V-slope relationship, i.e. the relationships showing increased CO_2 output (VCO_2) as a function of O_2 uptake (VO_2)[17]). In addition, we used other conventional methods also used to validate our estimation of AT. These were the increased ventilatory equivalent for VO_2 (V_E/VO_2) and the end tidal partial pressure of O_2 ($P_{ET}O_2$)[18, 19]). The RCP was estimated from the systematic increased ventilatory equivalent for VCO_2 (V_E/VCO_2) and the systematic decrease in end tidal partial pressure of CO_2 ($P_{ET}CO_2$)[20, 21]). Heart rate was monitored continuously beat-by-beat from the interval of a standard 12-lead electrocardiogram during testing[12]).

Metabolic Measurements

During the exercise test, pulmonary and ventilator gas exchange parameters were measured breath-by-breath using a respiratory gas analyser (Master Screen CPX, Germany)[22]). Ventilation measurement were done using a precise, bidirectional, digital volume sensor (Triple V-Volume Sensor). Before the each test, we calibrated the gas and volume of the system and calibration of the volume and gas analyser was done automatically.

Substrate utilization

During exercise tests, to estimate of fat and carbohydrate oxidation, we performed breath-by-breath measurements of VO_2 and VCO_2 and respiratory quotient (RQ) using a metabolic gas analyser (Master Screen CPX, Germany). We calculated fat and carbohydrate oxidation rates using Frayn equations[23]). The urinary nitrogen excretion rate was accepted as negligible.

Fat oxidation (g/min) = 1.67 x VO_2 (L/min) - 1.67 x VCO_2 (L/min)

Carbohydrate oxidation (g/min) = 4.55 x VCO_2 (L/min) – 3.21 x VO_2 (L/min)

To avoid potential instability of plasma bicarbonate during RCP test, we used indirect calorimetry for the determination of substrate oxidation rates when constant V_E and VCO_2 and end-tidal CO_2 partial pressure ($P_{ET}CO_2$) responses were observed[24]).

Statistical Analyses

We have presented the values for all descriptive data as mean (±SD). We identified substrate utilisation rates for fat and carbohydrate among the three different exercise intensities using repeated-measures ANOVA with post hoc t-test. A p value <0.05 was considered to be statistically significant.

Results

Participants' values of VO_2 and work rate response to incremental exercise task are given in Table 1. The mean VO_2 at the AT was 24.03 ± 3.9 ml/min/kg and the mean VO_2peak for each kg of body weight was 40.24 ± 5 ml/min/kg. AT and RCP occurred at an intensity of 60% (range 53-64%) and 72% of VO_2peak (range 66-76%), respectively. Work rates for each kilogram of body weight at AT and at the Wmax were 2.103±0.3 W/kg and 2.956±0.3 W/kg, respectively. During steady-state constant load exercise (average of last 10 minutes), the mean (±SD) RQ was 0.922±0.006 for $W_{<AE}$, 0.903±0.003 for the W_{AE} and 0.920±0.009 for the W_{RCP}. The highest fat oxidation ratio was in workload associated with W_{AT} ($p<0.05$), but there was no statistically significant difference between $W_{<AE}$ and W_{RCP} ($p=0.5$).

Rate of fat and carbohydrate oxidations during each minute of three constant load exercise tests are shown in Figure 1. During the constant load exercise (average of last 10 minutes) at $W_{<AT}$, the amount of fat and carbohydrate oxidation was 0.221±0.01g/min and 1.621±0.03 g/min, respectively. Increasing exercise intensity to W_{AT} resulted in a significant increase in total fat (0.340±0.01 g/min) and total carbohydrate oxidation (1.961±0.02 g/min), ($p<0.05$). During the constant load exercise, increasing exercise intensity up to W_{RCP} resulted in a marked increase in total carbohydrate oxidation compared to that of other exercise intensities (2.417±0.1 g/min ($p<0.05$) (Figure 1). The amount of fat oxidation (0.326±0.03 g/min) was significantly higher compared to that at $W_{<AT}$ ($p< 0.05$). However, there were no significantly differences in amount of fat oxidation between W_{AT} and W_{RPC} tests.

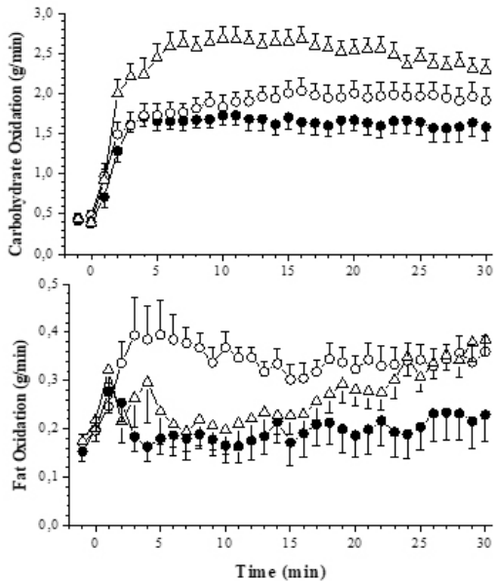

Figure 1. The rate of carbohydrate (upper graph) and fat oxidation (lower graph) (mean values) for each minute of three constant load exercise tests: ● shows $W_{<AT}$, ○ shows W_{AT} and Δ shows W_{RCP}.

Figure 2 shows standardisation of fat and carbohydrate oxidation rates for each W work rates at the $W_{<AT}$, W_{AT} and W_{RCP}. The rate of substrate oxidation with regarding each W work productions showed marked differences compared the total substrate oxidation rates. Standardisation of fat oxidation rate at W_{AT} was significantly higher than $W_{<AT}$ and W_{RCP} ($p<0.05$) (Figure 2).

Table 1. During incremental exercise test, mean (±SD) values of O_2 uptake at maximal exercise (VO_2peak L/min), O_2 uptake at anaerobic threshold (VO_2AT, L/min). The work rate below anaerobic threshold ($W_{<AT}$, W), at the anaerobic threshold (W_{AT}, W), at the respiratory compensation point (WRCP, W) and at maximal exercise (Wmax, W). The heart rate at maximal exercise (HRmax, beat/min).

Variables	
VO_2peak L/min	3.00± 0.3
VO_2AT (L/min)	1.78±0.2
$W_{<AT}$ (W)	98±15
W_{AT} (W)	130±21
W_{RCP} (W)	157±24
Wmax (W)	217±27
HRmax (beat/min)	189±4
HR_{AT} (beat/min)	150±12

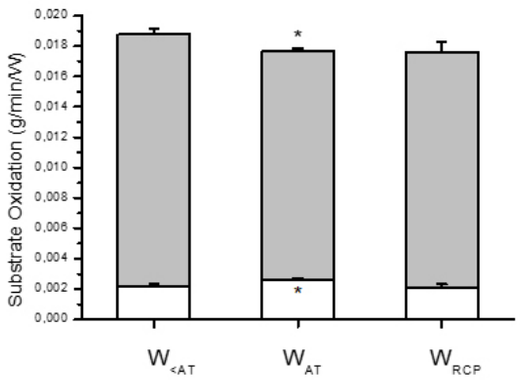

Figure 2. Standardised the rate of fat (white column) and carbohydrate (light grey column) oxidations (mean±SD, n=11) in response to each watt work rate at exercise intensity below anaerobic threshold ($W_{<AT}$), at the anaerobic threshold (W_{AT}) and at the respiratory compensation point (W_{RCP}).

*Reflects statistically significant differences.

Discussion

Strategies for the regulation of energy balance and substrate oxidation rates at rest and during various type of exercise are a high health priority. There is much interest among the investigators to understand the effect of exercise intensity on rate and amount of substrate oxidations. Although, shift in substrate oxidations during exercise has been studied previously across a wide range of work rates, little attention has been paid to specific exercise protocols at W_{AT} and W_{RCP}. To the best of our knowledge there have been no studies comparatively evaluated the rate of fat and carbohydrate oxidations at intensities corresponding to W_{AT} and W_{RCP}. A greater understanding of how fat and carbohydrate metabolism is affected by preceding work performed at the onset of anaerobic metabolism and before exercise hyperventilation would be advantageous and might have implications for the design of efficient exercise programs to achieve weight loss and weight maintenance in participants with metabolic imbalance.

Clinically optimal exercise intensity for energy regulation should focus fundamentally on promoting a high energy expenditure associated with high fat oxidation rates. In the current study, the impact of specific metabolic intensity protocols (i.e. $W_{<AT}$, W_{AT} and W_{RCP}) on fat and carbohydrate oxidation rates were evaluated comparatively during individually standardized constant load exercise tests. The constant load exercise tests corresponded to $W_{<AT}$, W_{AT} and W_{RCP} are provides metabolic steady state condition[1, 25]) that is important issue to accurately predict substrate utilisation. The lowest level of RQ was found to be at W_{AT} that indicate highest fat oxidation ratio compared to work intensity at W_{RCP} and $W_{<AT}$[26]). We did not count protein oxidation in the present study which is contributing energy production approximately 5% during prolonged exercise[27]).

During constant load exercise fat oxidation increases as exercise intensity increases until highest fat oxidation is reached at intensity of specific metabolic point of W_{AT} and W_{RCP}. In the present study, we observed large variations in fat oxidation rate between 0.2 g/min and 0.6 g/min among participants[6]), even though they performed exercise at their specific metabolic intensities at W_{AT} and W_{RCP}. The highest rate of fat oxidation during constant load exercise was observed at the W_{AT} and W_{RCP} in untrained healthy young males (Figure 1). In the present study, the mean fat oxidation rates of young male were 0.340±0.01 g/min and 0.326±0.03 g/min which occurred at the exercise intensity associated with AT (53-64% of Wmax and averaged 60% of Wmax) and RCP (66-76% of Wmax and averaged 72% of Wmax), respectively. It should be emphasised that application of fixed work protocol (i.e. 60% or 70% of Wmax) may cause substantially variable acute metabolic challenge to energy metabolic systems among the subjects. Despite marked difference in work rate and intensities, W_{AT} (and W_{RCP}) indicate identical exercise strain on the metabolic systems of all individuals[12]); and are widely used for specific exercise prescriptions[21, 28]). Increased fat oxidation rate during exercise at W_{AT} and W_{RCP} might be related to the increase in circulating free fatty acid which is shown to be promoting greater lipid oxidation[29]). Interestingly, fat oxidation rate becomes significantly higher in W_{AT} when standardised with each W of work rate (Figure 2).

In previous studies, a peak fat oxidation rate has been reported to occur at around 60% of VO_2peak, which is similar to the W_{AT} results[30]). However, exercise intensities expressed as a fixed percentage of maximal values might not result in metabolic responses that

are homogenous across the sample, especially in this case where the work intensity varied between 53-64% of Wmax in W_{AT}. In literature, a strong relationship between blood lactate and fat oxidation rate during an incremental exercise has been reported[6, 31, 32]). The highest fat oxidation rate at 65% of VO_2 peak which did not coincide with AT was reported[33]). In contrast, the results of a study requiring participants to exercise at 45% to 65% of VO_2peak showed no significant differences in fat oxidation[34]). An increase in carbohydrate oxidation and decrease in contribution of fat oxidation to participants' energy supplies during exercise intensity above 60% to 75% of VO_2max have been reported5, 6). However, in trained participants, a high fat oxidation rate has been reported at 75% of VO_2max which was associated with AT[26, 35]).

In contrast, an increased fat oxidation rate at a work intensity of 35-48 of %VO_2max (i.e. moderate intensity) and a reduction in fat oxidation when exercise intensity increased above 48% have been reported[8]). Similarly, another study showed greater fat oxidation at low exercise intensity (33% of VO_2max) compared to moderate exercise intensity (66% of VO_2max). However, the results of the present study do not support the concept that low to moderate intensity aerobic exercise promotes greater fat oxidation (Figure 1 and Figure 2). The $W_{<AT}$ reflecting low exercise intensity[36]) varied between 40-48% of Wmax (averaged 45% of Wmax) and this resulted in the lowest percentage of fat oxidation with regard to total substrate oxidation and the lowest rate of fat oxidation (Figure 1).

Conclusion

As a conclusion, considering energy metabolic substrate oxidation studies, exercise intensity corresponded to AT provide higher fat oxidation rates in totally and normalising for each watt of applied work rate. Thus researchers and clinicians should consider using specific metabolic intensity, especially at W_{AT} (and W_{RCP}) to achieve optimal substrate oxidation rates, instead of using fixed percentage of work protocols. However, further investigations with higher number of samples size including obese participants are warranted.

Conflicts of interest

The authors declare that there is no conflict of interest in this manuscript.

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Correspondence:

Seda UGRAS
Department of Physiology, Bozok University, Faculty of Medicine, Yozgat, Turkey,
email: sedaugras@hotmail.com
(seda. ugras@bozok.edu.tr)