Relative Aerobic and Anaerobic Energy Contributions during Short-Duration Exercise Remain Unchanged over A Wide Range of Exercise Intensities

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The present study aimed to examine whether different exercise intensities, ranging from submaximal to supramaximal, modulate the relative contributions of aerobic and anaerobic energy systems during short-duration exercise. Eight competitive male track and field athletes (22.3 ± 1.0 years) performed a 30-s pedaling test at seven different intensities corresponding to O₂ demands of 40, 50, 60, 70, 80, 90, and 100 ml/kg/min. The power outputs required at each O₂ demand were determined from the extrapolated linear relationships between power and the steady-state O₂ uptake obtained during submaximal-intensity exercise. The VO₂max test and 30-s Wingate anaerobic test were also performed. Relative aerobic and anaerobic energy contributions were estimated by the ratio of O₂ uptake and O₂ deficit, the latter being calculated as the difference between O₂ demand and O₂ uptake. The exercise intensity of the 30-s pedaling test ranged from 73.4 ± 7.4 to 180.9 ± 18.2% VO₂max. As exercise intensity increased, O₂ uptake (13.9 ± 2.1 to 26.8 ± 2.1 ml/kg/min) and O₂ deficit (26.9 ± 2.1 to 73.7 ± 2.2 ml/kg/min) during the 30-s pedaling test increased (P < 0.05). However, the relative aerobic (34.1 ± 5.1 to 26.7 ± 2.0%) and anaerobic (65.9 ± 5.1 to 73.3 ± 2.0%) energy contributions during the 30-s pedaling test did not differ across all O₂ demands (P > 0.05). These results suggest that the relative aerobic and anaerobic energy contributions during short-duration exercise remain nearly constant over a wide range of exercise intensity.

Keywords: energy system contribution, exercise intensity, short-duration exercise, O₂ uptake, O₂ deficit

1. Introduction

Energy utilized for exercise is provided via the aerobic and anaerobic energy systems. Traditionally, the aerobic energy supply during exercise has been determined by measuring O₂ uptake (Åstrand and Saltin, 1961). On the other hand, the estimation of anaerobic energy supply had not been established until Medbø et al. (1988) introduced the concept that the accumulated O₂ deficit, as determined by the difference in estimated O₂ demand and measured O₂ uptake, may reflect the anaerobic energy supply during exercise. Since then, researchers evaluated O₂ uptake and O₂ deficit to estimate aerobic and/or anaerobic energy contributions during short-duration high-intensity exercise (Gastin, 2001). For example, Medbø and Tabata (1989) demonstrated that the aerobic energy contributions during exhaustive cycling lasting 30 sec, 1 min, and 2-3 min were 30 ± 1%, 47 ± 2%, and 65 ± 2%, respectively. A similar response was also confirmed in subsequent studies employing different exercise modalities such as swimming (Ogita et al., 2003) and running (Duffield and Dawson, 2003). However, since both exercise duration and intensity varied in the above studies, it cannot be concluded whether exercise intensity modulates the relative energy contributions. Fixed exercise duration experiment is necessary to assess the influence of exercise intensity on relative aerobic and anaerobic energy contributions during exercise.
Some insights can be gleaned from previous work regarding the influence of exercise intensity on the relative aerobic and anaerobic energy contributions with a fixed exercise duration. Based on the data reported by Spencer and Gastin (2001), aerobic energy contributions during the first 20 sec of 200-m, 400-m, 800-m, and 1500-m maximal running on a treadmill were 34.4%, 32.0%, 39.5%, and 59.1%, respectively. While this indicates that the relative aerobic energy contribution during exercise increases as exercise intensity decreases, the aforementioned study by Spencer and Gastin (2001) tested highly trained athletes of the selected each event. Since the physical fitness and/or training status of participants is a determinant of relative energy system contributions (Calbet et al., 2003; Granier et al., 1995), a within-participant design is necessary to better understand the influence of exercise intensity on relative energy system contributions as reported in two previous studies (Gastin et al., 1995; Peyrebrune et al., 2014). Gastin et al. (1995) demonstrated that in physically active males the relative aerobic energy contribution during the first 30 s of constant-intensity exercises (107 ± 1%VO₂max and 125 ± 2%VO₂max) and 90-s all-out exercise (149 ± 5%VO₂max) was 41.2%, 38.1%, and 34.1%, respectively. This result suggests that a higher exercise intensity leads to a lower aerobic energy contribution, a finding that is consistent with the above-mentioned findings by Spencer and Gastin (2001). In contrast, Peyrebrune et al. (2014) reported that in competitive swimmers the aerobic energy contribution during a 30-s exercise at 154 ± 6 and 165 ± 2%VO₂max was 25 ± 4% vs. 33 ± 8%, respectively. Therefore, it remains inconclusive how different exercise intensities modulate relative energy system contributions. It should also be noted that the exercise intensities employed in the above studies were supramaximal only (> 100%VO₂max). Hence, it remains to be determined if relative aerobic and anaerobic energy contributions differ over a wide range of exercise intensities from submaximal (< 100%VO₂max) to supramaximal. Assessing relative aerobic and anaerobic energy contributions during short-duration exercises over a wide range of exercise intensities is important since exercise intensities of training programs for anaerobic type athletes such as sprinters often vary from submaximal to supramaximal.

Therefore, the present study aimed to examine whether different exercise intensities ranging from submaximal to supramaximal modulate the relative energy system contributions during 30-s exercise duration. We hypothesized that the relative contribution of aerobic energy during short-duration exercise would decrease as exercise intensity increases. We elected to adopt 30-s exercise as estimated accumulated oxygen deficit method during shorter duration of exercise (e.g., 10-20 s of exercise) can be more affected by O₂ store and/or time delay required for increased muscle O₂ consumption to be reflected in expiratory gases (Medbø and Tabata, 1989; Medbø, 2010). The present study would advance our fundamental knowledge of metabolic responses during short-duration exercises, which would help assess and design training programs for athletes and coaches.

2. Methods

2.1. Participants

The participants were eight competitive male track and field athletes, including seven sprinters and one decathlete (100-m sprint time, mean ± standard deviation, 11.09 ± 0.38 sec). All participants were tested during off season. Their age, height, body mass, and body fat percentage were 22.3 ± 1.0 years, 1.77 ± 0.04 m, 70.1 ± 6.1 kg, and 10.5 ± 3.2%, respectively. Body fat percentage was assessed using a multifrequency segmental body composition analyzer (MC-190 SV, TANITA Co., Tokyo, Japan). All participants visited the laboratory before commencing the experiment to get accustomed with exercising on a cycle ergometer. The seat and handlebar heights were recorded and kept constant in all test. The present study was approved by the human ethical committee of the University of Tsukuba, in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to participation.

2.2. Procedures

This study consisted of one preliminary session and seven subsequent experimental sessions. All preliminary and experimental sessions were completed within three weeks, each separated by ≥ 48 h. The order of the seven experimental sessions was randomized (Table 1). All tests were performed using a mechanically braked cycle ergometer (Power max VIII, Combi Co., Tokyo, Japan) in an experimental room regulated to a room temperature of 24-26°C and relative humidity of ~ 60%. Before each session, the participants
were instructed to avoid strenuous exercise, caffeine, and alcohol for at least 24 h. The participants were also instructed not to eat any food two hours before and during each session. They arrived at the laboratory at the same time for each session.

### 2.3. Preliminary session

#### 2.3.1. Submaximal test

Upon arriving at the laboratory, the participants initially performed a 3-min warm-up with a gradually increasing intensity protocol (from 40 to 240 W at a rate of 40 W/30 sec). Thereafter, they performed a submaximal cycling test to estimate the O₂ demands at the different exercise intensities. An initial workload for the submaximal cycling was set at 40 W and was increased by 40 W every 4 min interspersed with 2-min rest periods. The exercise intensity, duration, and rest periods employed were determined based on studies by Finn et al. (2000) and Green and Dawson (1996). Blood samples were obtained from a warmed fingertip immediately after each exercise stage to evaluate blood lactate concentrations. The exercise was terminated when the blood lactate concentration exceeded 4 mM since O₂ demand could be overestimated at intensities exceeding lactate/ventilatory threshold or respiratory exchange ratio (Hill and Vingren, 2011; Wilkerson et al. 2004). Workload (kp) and the pedal cadence during the submaximal test were determined using the following equations:

\[
\text{Pedal cadence (rpm)} = 0.14 \times \text{Power (W)} + 37 \quad \text{(equation 1)}
\]

\[
\text{Workload (kp)} = \frac{\text{Power (W)}}{[\text{Pedal cadence (rpm)} \times 0.98]} \quad \text{(equation 2)}
\]

Optimum pedal cadence exists at each workload as reflected by the efficient cadence that requires the smallest increase in O₂ uptake (Böning et al., 1984; Coast and Welch., 1985). Moreover, the optimum pedal cadence increases as power increases (Coast and Welch, 1985). Accordingly, pedal cadence was increased as workload increased in the submaximal test of the present study. The above equations were derived based on our pilot work, coupled with the previous data (Coast and Welch, 1985). For each individual, a linear relationship between power and O₂ demand was established from the measured power and the steady-state O₂ uptake at each stage of cycling with the blood lactate < 4 mM and the respiratory exchange ratio < 1.00 in the submaximal test.

#### 2.3.2. VO₂max test

Twenty minutes after the submaximal cycling test, the participants performed an incremental cycling test to obtain VO₂max, an index of aerobic capacity. This test began with an initial workload of 2.3 kp, which was increased by 0.1 kp every 1 min. The pedal cadence was maintained at 90 rpm throughout the cycling until exhaustion. Exhaustion was defined as an inability to maintain a pedal cadence of > 85 rpm for 5 sec. All participants exhibited a levelling off of O₂ uptake despite increases in intensity (difference < 2.0 ml/kg/min). VO₂max was defined as the highest VO₂ value averaged over a 30-s interval.

### 2.4. Experimental session

#### 2.4.1. 30-s pedaling test

After arriving at the laboratory, the participants initially performed a 10-min warm-up at a 1.0 kp workload with a pedal cadence of 90 rpm. Thereafter, they completed a 20-min post-exercise rest period and then performed a 30-s pedaling test (Figure 1). Ten seconds before commencing the test, the participants started cycling with a workload of 0 kp and a gradual increase in cadence to 100 rpm (so-called rolling start) (Minahan et al., 2007). Subsequently, the participants
cycled at a pre-determined workload and a pedal cadence for 30 sec at an $O_2$ demand of 40, 50, 60, 70, 80, 90, or 100 ml/kg/min. This 30-s pedaling test was repeated seven times with different target workloads and pedal cadences to obtain all seven $O_2$ demand data.

2.4.2. Wingate Anaerobic Test

The participants conducted the Wingate Anaerobic Test (WAnT) approximately 20 min after completing the 30-s pedaling test at an $O_2$ demand of 50 ml/kg/min. The workload for WAnT was 7.5% of the body weight × kp. WAnT was undertaken with a rolling start as with the 30-s pedaling test. Once the 30-s WAnT commenced, the participants pedalled with maximal effort.

2.5. Measurements

An expired gas analysis was continuously performed using a gas analyzer (EXP mode, AE-310s, Minato Medical Science Co., Osaka, Japan) using the computerized standard open circuit technique. The obtained respiratory data were averaged over 5-s time intervals. Blood lactate concentrations were assessed using a lactate analyser (YSI 1500 SPORT L-Lactate Analyser, YSI Inc., Yellow Springs, OH, USA). Heart rate was measured using standard telemetry (Polar S610i, Polar Electro Japan, Tokyo, Japan).

2.6. Data analyses

The expired gas in the submaximal test, $\dot{V}O_2$max test, and 30-s pedaling test was analysed from 3 min before each exercise. Mean pedal cadence, mean power, steady-state $O_2$ uptake, heart rate, and respiratory exchange ratio during each stage in the submaximal test were evaluated as an average value over the last 2-min period. The $O_2$ demand during each 30-s pedaling test was estimated by linear extrapolations of power data and steady-state $O_2$ uptake obtained in the submaximal test. The $O_2$ deficit during each 30-s pedaling test was calculated as the difference between $O_2$ demand and $O_2$ uptake (Figure 2). Relative aerobic and anaerobic energy system contributions were estimated by ratio of $O_2$ uptake and the $O_2$ deficit. $O_2$ demand, $O_2$ uptake, and $O_2$ deficit during each 30-s pedaling test were utilized to estimate energy demand and aerobic and anaerobic energy supplies. The linear extrapolations were used to calculate the relative intensi-

![Figure 1](image1.png)

**Figure 1** The experiment protocol of 30-s pedaling test ↓: Measurements of the blood lactate concentration

![Figure 2](image2.png)

**Figure 2** A model of calculating $O_2$ demand and $O_2$ deficit for the 30-s pedaling test

The $O_2$ demand during each 30-s pedalling test was estimated by linear extrapolations of power data and steady-state $O_2$ uptake obtained in the submaximal test. The $O_2$ deficit during 30-s pedalling test was calculated as the difference between $O_2$ demand and $O_2$ uptake.
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2.7. Statistical analyses

Values are presented as mean ± standard deviation. The linear relationship between the power and the O₂ demand was analysed using the coefficient of determination. All variables presented in Table 3 were analysed using repeated-measures one-way analysis of variance with a factor of the O₂ demand. When a main effect of O₂ demand was detected, a post hoc analysis was performed using a Holm-Bonferroni test to determine differences between O₂ demands. The significance level for all comparisons was set at < 0.05. The statistical analyses were performed using R version 3.4.4 (The R Foundation for Statistical Computing, Vienna, Austria).

3. Results

The mean, peak power of WAnT, and VO₂ max were 10.3 ± 0.3 W/kg, 13.1 ± 0.4 W/kg, and 56.0 ± 5.6 ml/kg/min.

The mechanical and metabolic variables assessed during the submaximal test are presented in Table 2. The R² value of the linear relationship between the power and the O₂ demand was 0.994 ± 0.005.

The mechanical and metabolic variables assessed during the 30-s pedaling test are presented in Table 3. The peak blood lactate concentration was defined as the peak. Mean power output during the 30-s pedaling test was presented relative to that of the WAnT (%).

### Table 2  Mechanical and metabolic variables during the submaximal test

| Workload (Kp) | Mean pedal cadence (rev/min) | Mean Power (W) | Steady-state O₂ uptake (ml/kg/min) | %VO₂ max | Mean Heart rate (beats/min) | Post Blood lactate concentration (mM) | Respiratory Exchange ratio |
|---------------|-----------------------------|----------------|-----------------------------------|-----------|-----------------------------|-------------------------------------|--------------------------|
| Stage 1       | 1.0                         | 41.4 ± 0.9     | 42.0 ± 0.9                         | 10.9 ± 0.9| 19.6 ± 1.8                  | 82 ± 11.8                           | 1.0 ± 0.6                |
| Stage 2       | 1.7                         | 46.1 ± 0.4     | 80.0 ± 0.7                         | 16.0 ± 1.3| 28.7 ± 2.5                  | 93 ± 10.7                           | 1.0 ± 0.3                |
| Stage 3       | 2.3                         | 51.7 ± 0.1     | 121.0 ± 0.3                        | 21.8 ± 1.6| 39.1 ± 3.8                  | 107 ± 11.0                         | 1.4 ± 0.4                |
| Stage 4       | 2.7                         | 56.5 ± 0.3     | 156.0 ± 0.7                        | 28.1 ± 2.1| 50.5 ± 4.0                  | 122 ± 11.1                         | 1.9 ± 0.5                |
| Stage 5       | 3.1                         | 62.5 ± 0.6     | 198.0 ± 1.7                        | 35.4 ± 2.6| 63.5 ± 5.0                  | 141 ± 12.6                         | 3.0 ± 0.7                |
| Stage 6       | 3.5                         | 68.5 ± 0.4     | 245.0 ± 1.4                        | 43.4 ± 3.1| 77.9 ± 6.5                  | 161 ± 9.5                          | 5.2 ± 1.2                |

Date are mean ± standard deviation

### Table 3  Mechanical and metabolic variables during the 30-s pedaling test

| O₂ demand (ml/kg/min) | 40 | 50 | 60 | 70 | 80 | 90 | 100 |
|-----------------------|----|----|----|----|----|----|-----|
| O₂ demand (ml/kg/min) | 40.8 ± 0.3 | 50.9 ± 0.8 | 60.5 ± 0.5 | 70.1 ± 0.7 | 80.5 ± 1.2 | 90.0 ± 1.1 | 100.5 ± 1.3 * |
| % mean power relative to WAnT | 33.7 ± 1.0 | 43.1 ± 1.3 | 51.9 ± 1.4 | 60.8 ± 1.4 | 70.4 ± 2.0 | 79.1 ± 1.5 | 88.8 ± 2.7 * |
| %VO₂ max | 73.4 ± 7.4 | 91.7 ± 9.6 | 108.9 ± 10.9 | 126.3 ± 13.9 | 145.0 ± 15.5 | 162.1 ± 16.8 | 180.9 ± 18.2 * |
| Mean pedal cadence (rev/min) | 70.1 ± 7.5 | 81.1 ± 3.2 | 89.7 ± 4.3 | 98.4 ± 4.9 | 108.3 ± 5.8 | 116.9 ± 5.8 | 127.4 ± 7.3 * |
| Mean heart rate (beats/min) | 13.9 ± 2.1 | 15.1 ± 1.5 | 17.4 ± 1.6 | 20.0 ± 2.4 | 21.8 ± 1.5 | 24.0 ± 1.4 | 26.8 ± 2.1 † |
| Mean heart rate (beats/min) | 26.9 ± 2.1 | 35.8 ± 1.3 | 43.1 ± 1.5 | 50.1 ± 2.6 | 58.7 ± 2.0 | 66.1 ± 1.8 | 73.7 ± 2.2 * |
| Mean heart rate (beats/min) | 96.8 ± 4.4 | 101.6 ± 7.5 | 105.5 ± 9.8 | 107.9 ± 5.9 | 112.8 ± 6.7 | 116.6 ± 10.9 | 121.7 ± 13.6 † *
| Peak blood lactate concentration (mM) | 1.5 ± 0.2 | 2.3 ± 0.6 | 2.9 ± 0.5 | 3.7 ± 0.6 | 4.9 ± 0.8 | 5.8 ± 0.7 | 7.7 ± 1.0 |
| Aerobic energy contribution (%) | 34.1 ± 5.1 | 29.6 ± 2.8 | 28.7 ± 2.6 | 28.6 ± 3.5 | 27.1 ± 1.9 | 26.6 ± 1.6 | 26.7 ± 2.0 |

Date are mean ± standard deviation

* all values differed each other (P < 0.05)
† all values differed each other with the exception that O₂ demands of 40 vs. 50-60 ml/kg/min, 60 vs 70 ml/kg/min, and 70 vs. 80 ml/kg/min (P < 0.05)
‡ all values differed each other with the exception that O₂ demands of 40 vs. 50-100 ml/kg/min, and 50 vs. 80 ml/kg/min (P < 0.05)
during the 30-s pedaling test are presented in Table 3. The main effect of O₂ demand on the relative aerobic energy contribution was significant (F = 7.39, P < 0.01). However, the relative aerobic energy contribution during the 30-s pedaling test did not differ across all O₂ demands (Table 3, Figure 3). Both O₂ uptake and O₂ deficit during each exercise increased with increasing O₂ demand (main effects of O₂ demand on aerobic and anaerobic energy supplies were F = 72.1, P < 0.01 and F = 727.1, P < 0.01, respectively; Table 3). O₂ uptake was different between an O₂ demand of 40 vs. 70-100 ml/kg/min, 50 vs. 60-100 ml/kg/min, 60 vs. 80-100 ml/kg/min, 70 vs. 90-100 ml/kg/min, 80 vs. 90-100 ml/kg/min, and 90 vs. 100 ml/kg/min (all P < 0.05; Table 3). O₂ deficit differed across all O₂ demands (P < 0.01; Table 3).

4. Discussion

The purpose of the present study was to examine if different exercise intensities ranging from submaximal to supramaximal modulate the relative energy system contributions during a fixed duration of exercise. We demonstrated that the O₂ uptake and O₂ deficit during each exercise increased as O₂ demand increased. However, the relative aerobic and anaerobic energy system contributions during short-duration pedaling exercises at different intensities were nearly constant.

In the present study, O₂ uptake during the 30-s pedaling test increased as exercise intensity increased from 73.4 ± 7.4 to 180.9 ± 18.2%VO₂max (Table 3). This result suggests that aerobic energy supply increases in an exercise intensity-dependent manner. Similarly, Wilkerson et al. (2004) compared the O₂ uptake kinetics of physically active males at seven different intensities (60% of the gas exchange threshold to 120%VO₂max) and indicated that the O₂ uptake at 80 sec into each exercise was higher as intensity increased. Furthermore, Sousa et al. (2017) demonstrated that O₂ uptake of competitor male swimmers during the first 30 sec of constant-intensity exercises increased as exercise intensity increased from 95% to 105%VO₂max. Noteworthy, our results demonstrated that exercise intensity-dependent elevations in O₂ uptake occurred above 100%VO₂max levels. This may reflect exercise intensity-dependent rapid increases in O₂ uptake upon the initiation of exercise, which requires future scrutiny.

The O₂ deficit during the 30-s pedaling test increased with exercise intensity as was observed for aerobic energy supply (Table 3). Along these lines, the peak blood lactate concentration, an indirect indicator of anaerobic energy supply (Jacobs, 1986), increased with elevations in intensity (1.5 ± 0.2 to 7.7 ± 1.0 mM, P < 0.05) (Table 3). Hence, these results suggest that anaerobic energy supply from the glycolysis system increased with elevations in exercise intensity.

The relative aerobic and anaerobic energy system contributions during the 30-s pedaling test did not differ across all O₂ demands (Table 3, Figure 3). It is generally thought that the relative aerobic energy contribution decreases as exercise intensity increases (Hoffman, 2002), but this is not true according to our results. The lack of changes in relative energy system contributions over a wide range of exercise intensities was due to the fact that both aerobic and anaerobic energy supplies increased as exercise intensity increased, as discussed above.

We do not know why both aerobic and anaerobic energy supplies increased in an exercise intensity-dependent manner, but our results are in line with those of studies showing that the O₂ uptake during exercise relates to anaerobic energy supply (de Aguiar et al., 2015; Korzeniewski and Zoladz, 2004; Whipp et al. 1999). Whipp et al. (1999) reported that changes in O₂ uptake paralleled those in creatine phosphate, the latter reflecting anaerobic energy supply. Similarly, other studies supported this result (Barstow et al., 1994; Rossiter et al., 2002). Additionally, the mitochondria, which play a critical role in aerobic energy production, are related to intracellular metabolites such as adenosine diphosphate, creatine phosphate, and inorganic phosphate (Korzeniewski and Zoladz,
2004). Thus, the O₂ uptake response could be stimulated by increases in the metabolites involved in the anaerobic energy system (de Aguiar et al., 2015). In addition, muscle temperature would increase as exercise intensity increased due to elevated heat production in the active muscles. Elevated muscle temperatures could cause a rightward shift of the oxygen dissociation curve, resulting in greater release of O₂ from haemoglobin and thus O₂ uptake. Elevations in muscle temperature can also enhance anaerobic energy supplies (Febbraio et al., 1996). Therefore, the O₂ uptake and the O₂ deficit can similarly increase with elevations in exercise intensity.

Previous studies reported relative energy contribution with narrower range of exercise intensities of 154 ± 6 to 165 ± 2%VO₂,max in competitive swimmers (Peyrebrune et al., 2014) and 107 ± 1%VO₂,max to 125 ± 2%VO₂,max in physically active males (Gastin et al., 1995). Although Spencer and Gastin (2001) reported data obtained with a wide range of exercise intensity of 103 ± 6 to 201 ± 3%VO₂,peak, they tested highly trained athletes of the selected each running event. Also, Gastin et al. (1995) and Spencer and Gastin (2001) did not perform a statistical analysis to compare values between different exercise intensities. We compared relative energy contributions across a wide range of intensities from submaximal to supramaximal (73.4 ± 7.4 to 180.9 ± 18.2%VO₂,max; Table 3) using the same male sprinters, providing important insights into relationship between exercise intensity and relative contributions of aerobic and anaerobic energy supplies.

4.1. Limitations

The most participants recruited in the present study were sprinters. Some studies (Calbet et al., 2003; Granier et al., 1995) reported that sprinters exhibit higher rates of anaerobic energy contributions during WanT, compared with endurance athletes. Therefore, it appears that relative energy contributions during high-intensity exercise can be influenced by individual’s training background. Accordingly, our results obtained in male sprinters may not be simply applicable to other populations such as endurance athletes.

We examined the relative energy system contributions during 30-s pedaling test at fixed O₂ demands, as O₂ deficit, but not O₂ uptake, is a main component of O₂ demand during short-duration exercises (Gastin, 2001). Additionally, O₂ uptake appears to be modulated by absolute peripheral O₂ utilization therefore increases in anaerobic and hydrolysis metabolites at the onset of exercises (Korzeniewski and Zoladz, 2004; Rossiter et al., 2002; Sahlin et al., 1988). Nevertheless, had we assessed responses at fixed relative exercise intensity of %VO₂,max, our results may have been different.

Our results suggest that the relative energy contributions during short-duration exercise remain nearly constant over a wide range of exercise intensity. However, caution is needed to interpret our results since relative energy system contribution could be influenced by exercise duration (Medbø and Tabata, 1989). Had we examined responses with longer exercise durations, results may have been different.

5. Conclusion

We show that the relative aerobic and anaerobic energy contributions during short-duration (i.e., 30-s) exercise remained nearly constant over a wide range of exercise intensities from 73.4 ± 7.4 to 180.9 ± 18.2%VO₂,max.

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