

LITHUANIAN ACADEMY OF PHYSICAL EDUCATION

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**INFLUENCE OF PRIOR LOAD ON THE
KINETICS OF CARDIORESPIRATORY
PARAMETERS DURING CYCLING
EXERCISE OF DIFFERENT INTENSITY**

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**PRIEŠKRŪVIO POVEIKIS VEGETACINIŲ
SYSTEMŲ FUNKCIJOS RODIKLIŲ KAITAI
ATLIEKANT SKIRTINGO INTENSYVUMO
VELOERGOMETRINĮ KRŪVĮ**

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ABBREVIATIONS

ADP – adenosine diphosphate

ATP – adenosine triphosphate

CCE – continuous increasing cycling exercise

EMG – electromyogram

GET – gas exchange threshold

H^+ – hydrogen ion

HR – heart rate

ICE – intermittent increasing cycling exercise

K^+ – potassium ion

LT – lactate threshold

P_ACO_2 – partial pressure of carbon dioxide in alveolus

$PaCO_2$ – partial pressure of carbon dioxide in arterial blood

PCO_2 – partial pressure of carbon dioxide

$P_{ET}CO_2$ – partial pressure of carbon dioxide at the end of expiration

$P_{ET}O_2$ – partial pressure of oxygen at the end of expiration

PO_2 – partial pressure of oxygen

$PvCO_2$ – partial pressure of carbon dioxide in venous blood

RER – respiratory exchange ratio

\dot{Q} – cardiac output

$\dot{V}O_2 \text{ max}$ – maximum oxygen uptake

$\dot{V}CO_2$ – rate of pulmonary carbon dioxide output

$\dot{V}O_2$ – rate of pulmonary oxygen uptake

\dot{V}_E – rate of pulmonary ventilation

$\% \Delta$ – % difference between lactate threshold (in $\dot{V}O_2$) and $\dot{V}O_2 \text{ max}$

[*La*] – blood lactate concentration

[*PCr*] – phosphocreatine concentration

INTRODUCTION

The rate of metabolism is rarely constant during exercise. In the transition from rest to muscular work or during recovery, during variations in exercise intensity the rate of metabolism changes rapidly. Kinetics of pulmonary oxygen uptake ($\dot{V}O_2$) and other related parameters (HR , $\dot{V}CO_2$, \dot{V}_E) during transitional phases between rest and exercise are associated with humans aerobic capacity, reflects its acute adaptation abilities (Whipp & Ozyener, 1998; Jones & Carter, 2000). The rate at which aerobic *ATP* production in skeletal muscles meets new metabolic demands has influence on such acute adaptation characteristics as on-transition, fatigue and recovery. More rapid adaptation of aerobic *ATP* production at the start of exercise decreases homeostatic changes in cells and the whole organism (Grassi, 2001).

It is well established that at the onset of exercise $\dot{V}O_2$ increases as an exponential function (Astrand & Saltin, 1961; Casaburi et al., 1987, 1989; Poole et al., 1991; Barstow & Mole 1991; Barstow, 1994; Poole, 1994; Whipp, 1994; Gaesser & Poole, 1996; Barstow et al., 2000; Ozyener et al., 2001). The mode of $\dot{V}O_2$ response depends on the exercise intensity and may consist of two (when intensity is below *LT*) (Henry & DeMoor, 1956; Whipp, 1970; Cerretelli & Di Prampero, 1987) or three phases (above *LT*) (Whipp & Wasserman, 1972; Linnarsson, 1974; Hughson & Morrissey, 1982; Paterson & Whipp, 1991; Barstow & Mole, 1991). It remains not clear, whether the rate of $\dot{V}O_2$ kinetics is limited by the adaptation of O_2 transport to muscles or O_2 utilization mechanisms in muscles (Tschakovsky & Hughson, 1999). To get more insight in the relative role of mechanisms mentioned above the experimental model where one exercise boot is preceded by other one (prior load) is often applied. There are numerous data about the influence of the intensity or mode of prior load on the kinetics of cardiorespiratory parameters during on-transition, steady state and off-transition phases of constant load exercise (Gerbino et al., 1996; MacDonald et al., 1997; Koppo & Bouckaert, 2000-2002; Scheuermann et al., 2001; Fukuba et al., 2002, 2007; Burnley et al., 2000-2002, 2006; Koppo et al., 2003; Tordi et al., 2003; Endo et al., 2004; Moysi et al., 2005; Gurd et al., 2006; Jones et al., 2006; Marles et al., 2006, 2007). The accumulation of blood metabolites (H^+ , K^+ , and lactate) after prior heavy (i.e. intensity above *LT*) exercise is known to increase the muscle blood flow at the onset of following exercise. However, prior heavy exercise had no effect on the magnitude and rate of the $\dot{V}O_2$ kinetics during moderate exercise for young healthy individuals (Gerbino et al., 1996; MacDonald et al., 1997; Burnley et al., 2002). On the contrary, $\dot{V}O_2$ kinetics during heavy exercise is often accelerated after prior heavy exercise (Gerbino et al., 1996; MacDonald et al., 1997; Koppo & Bouckaert, 2001; Bearden &

Moffatt, 2001; Scheuermann et al., 2001, 2002; Burnley et al., 2002, 2006; Tordi et al., 2003; Jones et al., 2006; Fukuba et al., 2007). Finally, it is not quite clear how the intensity of the prior load (or warm-up) influences the kinetics of $\dot{V}O_2$ and other cardiorespiratory system functional parameters (Germino et al., 1996; MacDonald et al., 1997; Fukuba et al., 1998; Burnley et al., 2000; Koppo & Bouckaert, 2000). The role of the prior exercise mode on the changes of above mentioned parameters remains still unclear (Gaesser & Poole, 1996; Whipp, 1996; Fukuba & Whipp, 1999; Tordi et al., 2003; Fukuba et al., 2007).

The ratio between $\dot{V}O_2$ and work rate during steady state phase of exercise (work efficiency) reflects the aerobic capacity and individual's ability to perform endurance type physical loads (Whipp et al., 2002; Jones & Carter, 2000). Despite intensive studies the exact determinants of cycling exercise efficiency remain unknown. The leg mass and pedal frequency had effect on the $\dot{V}O_2$ kinetics during cycling exercise (Neder et al., 2000, Martin & Spiriduso, 2001, McDaniel et al., 2002; Migita & Hirakoba, 2006; Gonzales & Scheuermann, 2006). After the fast exponential increase throughout priming 3 min the $\dot{V}O_2$ forward slowly increased during above *LT* exercise (Whipp, 1994). It has been hypothesized that this slow component of $\dot{V}O_2$ kinetics reflects an increase in metabolic costs of cycling associated with recruitment of less efficient type II fibres (Saunders et al., 2000; Bernasconi et al., 2006). Concentric muscle contractions are impaired following exhaustive eccentric exercise, which can cause muscle fatigue and damage (Horita et al., 2003; Moysi et al., 2005). It is not clear if metabolic costs of ergometer cycling increase as well. Increases in \dot{V}_E , breathing frequency, blood lactate, respiratory exchange ratio, *HR*, and rating of perceived exertion have been reported during ergometer cycling after eccentric exercise (Gleeson et al., 1995; 1998). However, $\dot{V}O_2$ at submaximal intensities did not change. It has been also shown that eccentric exercise did not alter nor had only a marginal effect on gross cycling efficiency in presence of marked muscle soreness (Moysi et al., 2005). At the same, there is clear evidence that eccentric exercise reduces concentric contraction economy in the muscle of the mouse (Warren et al., 1996). It has been also demonstrated that $\dot{V}O_2$ increased during ergometer cycling after exercise induced depletion of glycogen in type I fibres (Osborne & Schneider, 2006) as well as during repetitive fatiguing isometric contractions (Vollestad et al., 1990; Krstrup et al., 2004). There is some evidence that muscle fatigue intensifies recruitment of less efficient type II fibres during exercise (Krstrup et al., 2004; Nakagawa et al., 2005; Deley et al., 2006). Overall, however, findings on the metabolic costs of ergometer cycling after fatiguing exercise are controversial. Also we could not find any study investigating effects of prior fatiguing stretch shortening exercise on different phases of $\dot{V}O_2$ kinetics during exercise. Repetitive drop jumps induce muscle

fatigue of long duration with signs of muscle damage (Skurvydas et al., 2000; Murfet et al., 2003). It could be expected that this would increase metabolic costs of ergometer cycling at least in part due to intensified recruitment of less efficient type II muscle fibres (Nakagawa et al., 2005; Migita & Hirakoba, 2006). It is believed that progressive recruitment of type II muscle fibres can be associated with a slow component of $\dot{V}O_2$ kinetics of ergometer cycling even at 50% $\dot{V}O_{2\max}$ (Krustrup et al., 2004).

Physiological responses to incremental and constant work rate (*CWR*) exercise tests can be used for assessment of functional capacity and for design of physical training programmers in diseased, sedentary, and athletic populations (Whipp et al., 1981; Meyer et al., 1998; Wasserman et al., 1999). Specifically, incremental or ramp exercise tests, which typically involve increases in work rate in such a way that exhaustion is reached in 8-14 minutes, are often used for the assessment of the maximal rate of aerobic *ATP* resynthesis ($\dot{V}O_{2\max}$) as well as for establishing of *LT* or *GET* (Whipp et al., 1981; Wasserman et al., 1999). To our knowledge, only changes in dynamics of several cardiorespiratory parameters during continuous incremental exercise or ramp exercise tests until exhaustion under conditions of metabolic acidosis have been previously been determined (Schneider & Berwick, 1998; Jones & Carter, 2004; Marles et al., 2006). We did not find any data about effects of prior load on above mentioned parameters relation to work rate during intermittent increasing cycling exercise test.

Hypothesis. Different prior load can result in specific changes of the blood [*La*], muscle blood flow and metabolism, metabolic or non-metabolic fatigue, and this will cause the variability in changes of kinetics of different cardiorespiratory parameters during on- and off-transition phases of following exercise. Separate phases of this kinetics may also be influenced in different kind and this influence may also depend on the intensity of following exercise.

Novelty and originality. It was estimated for the first time that prior eccentric-concentric exercise, consisting of 100 drop jumps with 20 s of rest after each jump, increased the absolute values of oxygen uptake at steady state, i. e. decreased the aerobic work efficiency, during both moderate and heavy aerobic cycling exercise, but had no effect on the kinetics of cardiorespiratory system parameters at the onset of exercise, and did not change the slow component of the $\dot{V}O_2$ kinetics. The prior incremental cycling exercise increased the absolutely values of $\dot{V}O_2$ during both moderate and heavy exercise, had no effect on the fast component, but decreased slow component of the $\dot{V}O_2$ kinetics.

Theoretical and practical application of study results.

The results of this study enlarge knowledge in such fields of exercise physiology as acute adaptation of human organism to exercise, the influence of metabolic and non-metabolic fatigue as well as warm-up mode and intensity on the aerobic work efficiency. The study provides additional knowledge in aerobic capacity testing using intermittent increasing exercise protocols.

Aim of the study was to determine the influence of prior load on cardiorespiratory parameters' kinetics during cycling exercise of different intensity.

Objectives

1. To determine the influence of a prior aerobic loads of different intensity on heart rate kinetics during intermittent increasing cycling exercise.
2. To determine the influence of a prior excentric-concentric exercise on cardiorespiratory parameters kinetics during intermittent increasing cycling exercise.
3. To establish the influence of a prior anaerobic load on the relationship among cardiorespiratory parameters, blood lactate concentration and the intensity of intermittent increasing cycling exercise.
4. To establish the influence of a prior anaerobic load on cardiorespiratory parameters during on- and off-transion phases of intermittent increasing cycling exercise.
5. To evaluate the effect of prior incremental cycling exercise on pulmonary oxygen uptake kinetics during moderate and heavy intensity exercise.

1. METHODS AND STUDY DESIGN

1.1. SUBJECTS

The thirty-six healthy, non-smoking, physically active persons volunteered to participate in the studies. Their physical and aerobic capacity characteristics are presented in Table 1. Informed consent was signed by each of the subjects after the experimental protocols and possible risks associated with participation in the studies had been explained, as approved by the Local Research Ethics Committee (in accordance with the Declaration of Helsinki). All participants reported to the laboratory rested (having performed no strenuous activity in the preceding 24 h), well hydrated and having abstained from food and caffeine for at least 3 h before testing. Tests were conducted in a well-ventilated laboratory at the same time of day for each participant, at a comfortable temperature (18–21°C).

Table 1. Physical and aerobic capacity characteristics of subjects. Values presented are means (SD).

	I study	II study	III study	IV study
Sample size (n)	8	7	7	14
Sex	Male	Male	Female	Male
Age (years)	22.5 (2.6)	23.9 (2.0)	22.1 (1.5)	20.3 (1.2)
Height (m)	1.77 (0.30)	1.79 (0.69)	1.69 (0.66)	1.82 (0.08)
Weighth (kg)	69.6 (9.8)	71.9 (2.0)	56.7 (4.9)	78.4 (8.7)
$\dot{V}O_2$ max ($l \cdot \text{min}^{-1}$)	Not measured	3.6 (0.6)	2.3 (0.3)	4.1 (0.4)
$\dot{V}O_2$ max ($\text{ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$)	Not measured	50.2 (6.4)	41.5 (5.2)	52.2 (5.0)

1.2. METHODS

Pulmonary gas exchange data collection

Pulmonary gas exchange data ($\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E , RER , $P_{ET}O_2$, $P_{ET}CO_2$, EqO_2 , $EqCO_2$) were collected continuously using the automated breath-by-breath systems “Oxycon Mobile” (Jaeger, Germany) or “Cortex” (Germany). Prior to each exercise test, the gas analyzers were calibrated with certificated calibration

gas. The all recorded data was analyzed by five seconds mean intervals using “LAB Manager“ and “Microsoft Excel“ programs.

Heart rate monitoring and recording

The heart rate (*HR*) was continuously monitored and recorded using short-range telemetry “Polar S810” (Finland) and further analyzed using “Polar Precision Performance“ and “Microsoft Excel“ programs.

Measurement of blood lactate concentration

A fingertip blood sample was collected into a capillary tube and subsequently analyzed for blood lactate concentration as described previously (Kulis et al., 1988). Lactate concentration in the blood was established by means of Eksan-G analyzer using a membrane with enzyme lactoxidase. Prior to each testing the analyzer was calibrated by the standart 5 mM lactate solutions supplied by manufactures.

Continuous increasing cycling exercise (*CCE*) and determination of $\dot{V}O_2$ max

The *CCE* was performed on the mechanically braked cycle ergometer (Monark 834E, Monark-Crescent AB, Sweden). First all the subjects exercised for 3 min with the intensity of 17 W. Thereafter the intensity was set to 70 W and increased every minute by 21 W for male subjects or set to 50 W and increased every minute by 10 W for female subjects. The pedaling rate was 70 rpm and 50 rpm for males and females, respectively. The test was terminated when the subject was not able to keep the required pedaling rate. Throughout the *CCE*, pulmonary $\dot{V}O_2$ was measured breath-by-breath using the automated system “Oxycon Mobile” (Jaeger, Germany). The $\dot{V}O_2$ max was determined as the highest mean value recorded in any 30 s period before the participant’s volitional termination of the test.

Intermittent increasing cycling exercise (*ICE*)

The subjects performed graded intermittent exercise on the mechanically braked cycle ergometer (Monark 834E, Monark-Crescent AB, Sweden). The tests consisted of repeated work (male subjects – 3 or 6 min, female subjects – 3 min) and passive rest (male subjects – 4 min, female subjects – 3 min) intervals. No special warm-up was performed. The work rate of first work period was set to 17 W. Thereafter the intensity was set to 70 W and increased by 25 or 35 W for male subjects or by 25 W for female subjects during each consecutive work period. Before test and during the last 30 s of each work period a fingertip blood sample was collected into a capillary tube and subsequently analyzed for blood lactate

concentration. The test was continued until the participants' blood lactate concentration risen over 4 mM level.

The lactate threshold (LT) and relative work intensity estimation

The *LT* was determined from visual inspection of individual plots of blood lactate concentration vs. work rate (fig. 1). The lactate threshold was considered as the work rate from which lactate concentration under rest level start raising slowly over 1–1.5 mM. The *LT* also was estimated by indirect method using absolute values of *HR* at recovery period during *ICE* test (Stasiulis, 1997).

The following relative intensities were chosen for the subsequent analysis and comparison of cardiorespiratory data in different study protocols: 50 W below lactate threshold (< 50 W *LT*); 25 or 35 W below lactate threshold (< 25 W *LT* or < 35 W *LT*); lactate threshold (*LT*); 25 or 35 W above lactate threshold (> 25 W *LT* or > 35 W *LT*); 50 W above lactate threshold (> 50 W *LT*) (fig. 1).

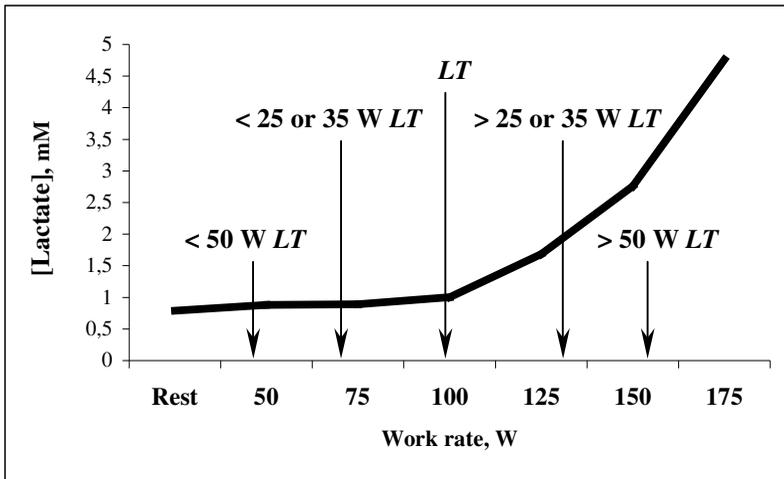


Fig. 1. The lactate threshold (*LT*) and relative work intensity estimation.

The analyses of cardiorespiratory parameters kinetics

In order to estimate the kinetics of cardiorespiratory parameters (*HR*, $\dot{V}O_2$, $\dot{V}CO_2$ and \dot{V}_E) during and after exercise that intensity was lower than *LT* responses during on-transition and recovery periods were analyzed by adopting mono-exponential function:

$$y(t) = y(b) \pm A \cdot (1 - e^{-t/\tau})$$

where $y(b)$ is the baseline value (HR , $\dot{V}O_2$, $\dot{V}CO_2$ or \dot{V}_E) through the last 30 s of work or rest; A is the amplitude and τ is the time constant of the response.

In order to estimate $\dot{V}O_2$ kinetics during on-transition to exercise that intensity was higher than LT the response was analyzed by adopting bi-exponential function:

$$\dot{V}O_2(t) = \dot{V}O_2(b) \pm A_P \cdot (1 - e^{-t/\tau_P}) \pm A_S \cdot (1 - e^{-t/\tau_S})$$

where $\dot{V}O_2(b)$ is the mean $\dot{V}O_2$ through the last 30 s of rest (base line); A_P and A_S are the amplitudes and τ_P and τ_S are the time constants of fast and slow components; t is the time interval from start of the work (s).

During on-transition the first 20 s were always removed from the analysis (Whipp et al., 1982).

Oxygen uptake of heart and respiratory muscles contributes to $\dot{V}O_2$ and changes in pulmonary ventilation and heart rate could have a significant impact on $\dot{V}O_2$ response to repetitive exercise. An attempt was made to evaluate importance of these factors to $\dot{V}O_2$. The oxygen costs of cardiac work were assumed to be $0.2 \text{ ml} \cdot \text{beat}^{-1}$ (Kitamura et al., 1972) while oxygen uptake of respiratory muscles was calculated as previously described (Carra et al., 2003). For this particular correction work of breathing was calculated at first:

$$Wb = -0.251 + 0.0382 \cdot \dot{V}_E + 0.00176 \cdot \dot{V}_E^2$$

where Wb is work of breathing and \dot{V}_E is expiratory pulmonary ventilation. Then oxygen uptake of respiratory muscles was estimated as:

$$VRMO_2 = 34.9 + 7.45 \cdot Wb$$

where $VRMO_2$ is O_2 uptake of the respiratory muscles, Wb is work of breathing.

1.3. STUDY DESIGN

1.3.1. STUDY 1

In order to estimate the influence of a prior aerobic load on the HR kinetics each subject was tested three times on separated days. During the first visit the ICE was performed (fig. 2A) on the mechanically braked cycle ergometer

(Monark 834E, Monark-Crescent AB, Sweden) and the pedalling rate was 70 rpm.

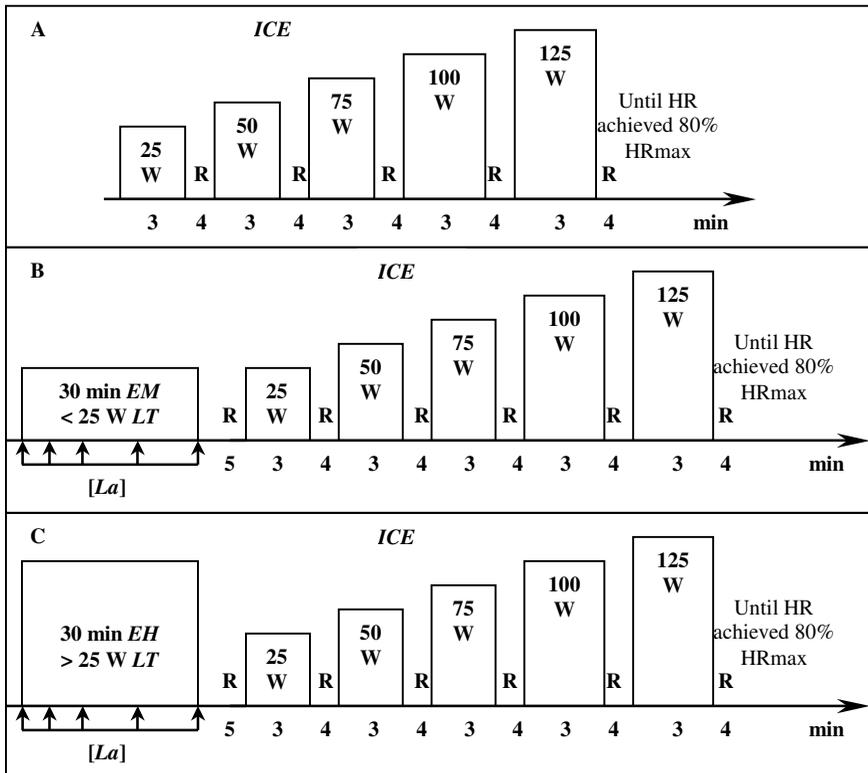


Fig. 2. Protocol of study 1

Note. EM – moderate exercise (below *LT*), EH – heavy exercise (above *LT*), ICE – intermittent increasing cycling exercise, [La] – the collection of the blood samples, R – rest, A, B, C – the occasions of testing.

The test consisted of repeated 3 min work and 4 min passive rest intervals. No special warm-up was performed. The work rate of first work period was set to 50 W. Thereafter the intensity was increased by 25 W during each consecutive work period. The *HR* was continuously monitored and recorded using short-range telemetry “Polar S810” (Finland). The *ICE* test was continued until the subjects’ *HR* was achieved over 80% of *HR* maximum predicted by his age ($220 - \text{age}$, years). The individual’s *LT* was estimated by indirect method using absolute values of *HR* at recovery period during *ICE* (Stasiulis, 1997). During the second

and third visits the subjects performed in random order moderate exercise (< 25 W *LT*) (*EM*) or heavy exercise (> 25 W *LT*) (*EH*) for 30 min and after 5 min of rest repeated *ICE* protocol (fig. 2B and 2C). At rest, 5th, 10th, 20th and 30th min of *EM* and *EH* a fingertip blood samples were collected into a capillary tube and subsequently analyzed for blood lactate concentration as described previously (Kulis et al., 1988).

1.3.2. STUDY 2

In order to estimate the influence of a prior eccentric-concentric load on the kinetics of cardiorespiratory parameters (*HR*, $\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E) each subject was tested three times on separated days. During the first visit, peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$) was evaluated using the *CCE* test (21 W min^{-1} , fig. 3A). The test was started at 70 W and continued until the intensity of cycling could not be maintained at the required level for longer than 10 s. The volunteers exercised for 12.0 ± 1.85 min^{-1} and the average value of $\dot{V}O_2$ over the last 30 s of cycling was referred to as $\dot{V}O_{2peak}$.

During the second and third visits, the subjects cycled for 6 min at intensities of 70 , 105 , 140 and 175 W with 4 min intervals of rest in between the exercise bouts. Capillary blood samples were collected during the last 30 s of cycling at each of the four intensities. First, participants cycled without any prior exercise (second visit, control condition, (fig. 3B)). Then, after at least three days the ergometer cycling was repeated 60 min after the repetitive drop jump exercise (third visit, fatigue condition, (fig. 3C)). It has been previously demonstrated that the employed protocol of repetitive drop jumps induces depression of force lasting at least 24 hours in the knee extensor muscles (Skurvydas et al., 2000). At the same time 60 min of rest after repetitive exercise would allow a recovery of muscle temperature and metabolites which could interfere with $\dot{V}O_2$ responses to exercise (Ratkevicius et al., 1998, Saugen & Vollestad 1995; Tordi et al., 2003)

Drop jump exercise

The drop jump exercise was performed as previously described (Skurvydas et al., 2000). The jumps were performed on a standard jump mat that displayed jump heights after each jump (Powertimer Testing System, Newtest, Tampere, Finland). Before repetitive jumping, each subject performed warming up exercise that consisted of 5 min running on the spot with an intensity that corresponded *HR* to 130 - 150 ($\text{beats} \cdot \text{min}^{-1}$). Then the subjects performed 10 squat-stands. Thereafter, the subject performed 100 drops jumps from a height of 40 cm to an

approximately 90 degree angle in the knees, followed by a countermovement jump. One jump was performed every 20 s. The subjects received feedback about their performance and were instructed to jump as high as possible. This protocol of exercise is known to induce a prolonged loss of muscle force accompanied with signs of muscle damage (Murfet et al., 2003; Skurvydas et al., 2000). All these procedures, including the warming up exercise and 100 drop jumps, lasted approximately 50 min.

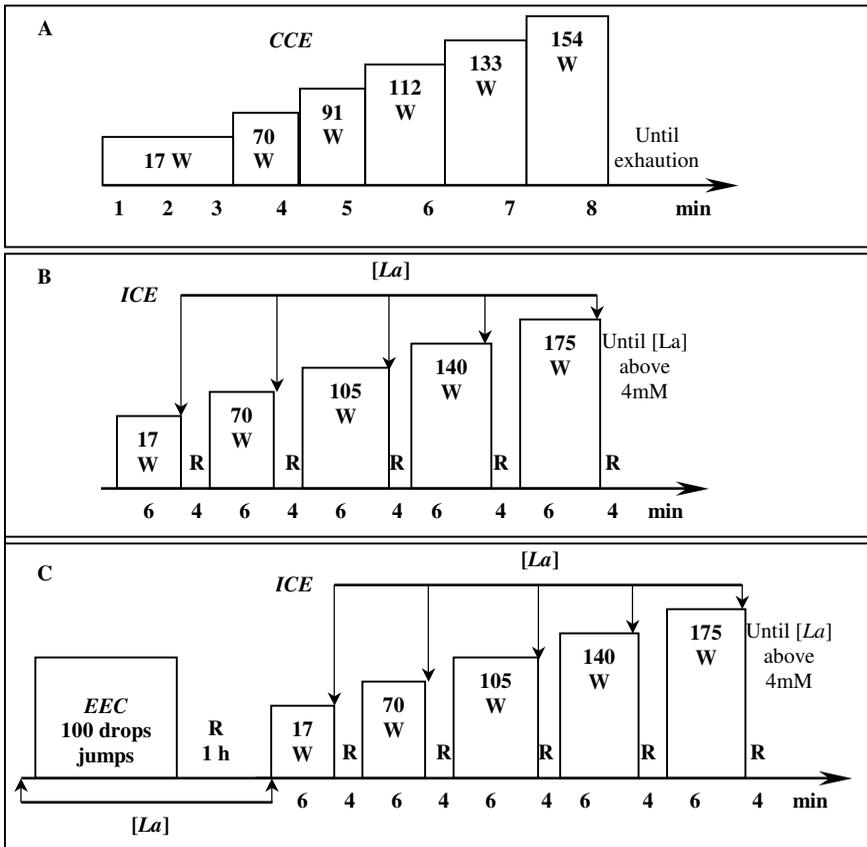


Fig. 3. Protocol of study 2

Note. *CCE* – continuous increasing cycling exercise until exhaustion. *ICE* – intermittent increasing cycling exercise until $[La]$ above 4 mM, *EEC* – eccentric-concentric exercise; $[La]$ – the collection of the blood for the estimation lactate concentration, R – rest, A, B, C – the occasions of testing.

1.3.3. STUDY 3

In order to estimate the influence of a prior anaerobic load on the kinetics of cardiorespiratory parameters (HR , $\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E) each participant was tested three times on separated days.

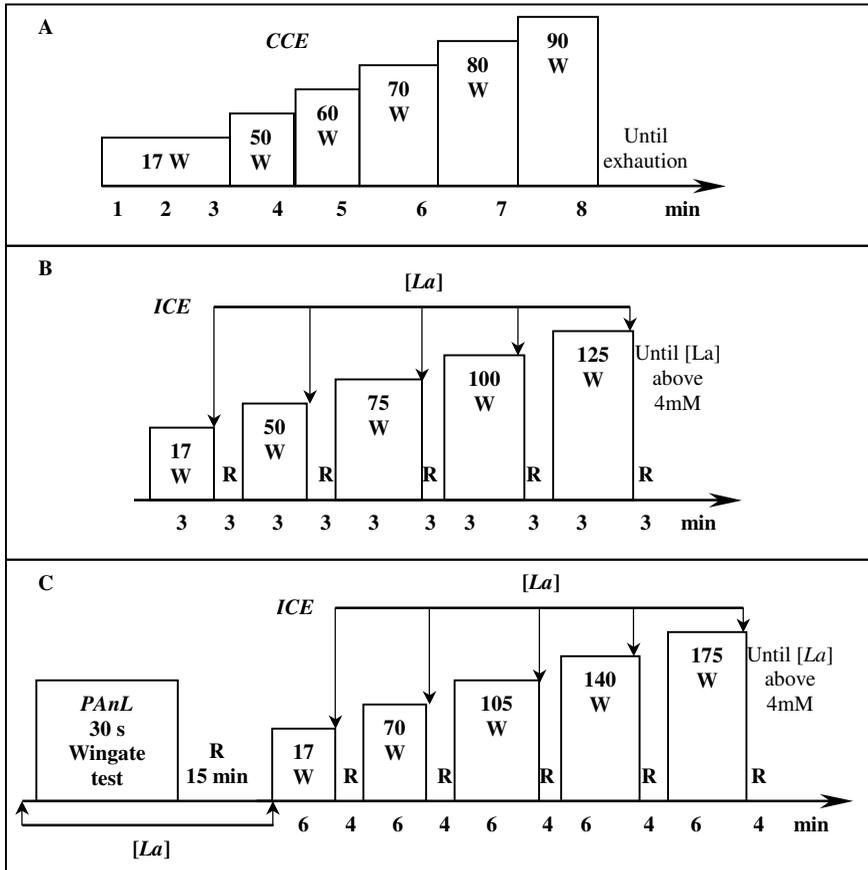


Fig. 4. Protocol of study 3

Note. *CCE* – continuous increasing cycling exercise until exhaustion. *ICE* – intermittent increasing cycling exercise, *PAnL* – prior anaerobic exercise; [La] – the collection of the blood for the estimation lactate concentration, R – rest, A, B, C – the occasions of testing.

The cycling frequency was maintained at approximately 50 rpm throughout all exercises. During the first visit $\dot{V}O_2$ max was evaluated using the *CCE* test (10 W min^{-1} , fig. 4A). The test was started at 50 W and continued until the intensity of cycling could not be maintained at the required level for longer than 10 s. During the second testing the participants performed *ICE* protocol (fig. 4B). During the other visit they performed supramaximal 30 s anaerobic exercise (*EMI*) and after 15 min of the rest – *ICE* protocol (fig. 4C). Before *ICE* test (at rest) and during the last 30 s of each work period of *ICE* a fingertip blood samples were collected into a capillary tube and subsequently analyzed for blood lactate concentration. Pulmonary gas exchange data were collected continuously using the automated breath-by-breath system “Oxycon Mobile” (Jaeger, Germany). The *HR* was monitored and recorded continuously using short-range telemetry “Polar S810” (Finland).

The Wingate anaerobic test

As prior anaerobic load the supramaximal 30 s Wingate test was performed on Monark 834E cycle ergometer (Bar-Or, 1981). The test was preceded by warm-up consisting of 5 min cycling (25-50 W) interrupted by short lasting bursts of high intensity. After this warm-up, the subjects took 1 min of rest for blood sampling and then performed 30 s all-out cycling followed by 1 min cool-down cycling with no resistance. The bicycle ergometer mechanical resistance was set at 7.5% of body mass.

1.3.4. STUDY 4

In order to estimate the influence of a prior mixed aerobic anaerobic load on the $\dot{V}O_2$ kinetics during on-transition each participant visited the laboratory on two occasions over a 1-wk period. During the first day the participants performed *CCE* protocol (fig. 5A). This visit was used for $\dot{V}O_2$ max, *GET* and the intensity of constant work rate (*CWR*) exercise determination whereas the other visit was used to complete the *CWR* exercise after preceding *CCE*. After the *CCE* the participants were randomly split in two groups: one group (n=7) completed moderate intensity (*EM* – 45% of $\dot{V}O_2$ max) *CWR* exercise (fig. 5B1), other group (n=7) completed heavy intensity (*EH* – 70% of $\dot{V}O_2$ max) *CWR* exercise (fig. 5B2). These power outputs were determined by linear regression of each participant’s $\dot{V}O_2$ on power output using data from the *CCE*. In the second examination each participant performed two *CWR* exercises (6 min cycling preceded by 2 min of unloaded cycling) interspaced with *CCE*. The time intervals of passive rest between *CWR* exercise and *CCE* were 4 min and between *CCE*

and repeated *CWR* exercise were 10 min. The cycling frequency was maintained at approximately 70 rpm throughout all exercises. Pulmonary gas exchange data were collected continuously using an automated breath-by-breath system “Oxycon Mobile” (Jaeger, Germany). The *HR* was recorded simultaneously using short-range telemetry “Polar S810” (Finland). A fingertip blood samples were collected into a capillary tube at the end of each bout of *CWR* exercise, at 5th and 10th min after *CCE* and subsequently analyzed for blood lactate concentration.

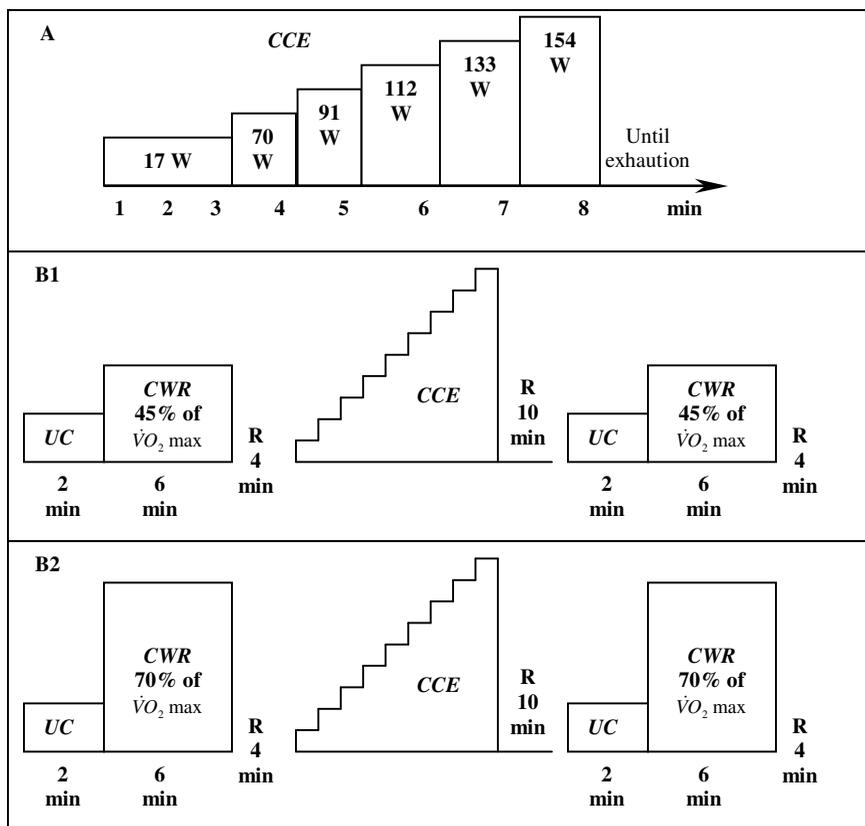


Fig. 5. Protocol of study 4

Note. *CCE* – continuous increasing cycling exercise until exhaustion. *CWR* – constant work rate cycling exercise. *UC* – unloaded cycling. *R* – rest; A, B1, B2 – days of testing.

1.4. STATISTICS

All values are reported as the means and standard deviations (SD). Examination of normality distribution was performed using Kolmogorov-Smirnov test. Comparisons of parameters between testing conditions and among different intensities were conducted using Wilcoxon matched pairs test or two-way repeated measures analyses of variance (ANOVA). If significant effects were found, post hoc testing was performed applying paired t- tests with a Bonferroni correction for multiple comparisons or Tukey's test. The limit of significance was set at $P < 0.05$.

2. RESULTS

2.1. Influence of prior aerobic exercise of different intensity on heart rate (HR) kinetics during intermittent increasing cycling exercise (ICE).

Fig. 6 presents HR data obtained during intermittent increasing cycling exercise test performed under three experimental conditions. The mean values of HR during 3rd min of work were significantly increased after both moderate and heavy prior load (fig. 6, left). This increase tended to be higher after heavy prior aerobic exercise. After heavy prior aerobic load the mean values of HR during 4th min of recovery were significantly increased at all intensities while after moderate prior aerobic load they were higher only at intensities equal to or lower than LT (fig. 6, right).

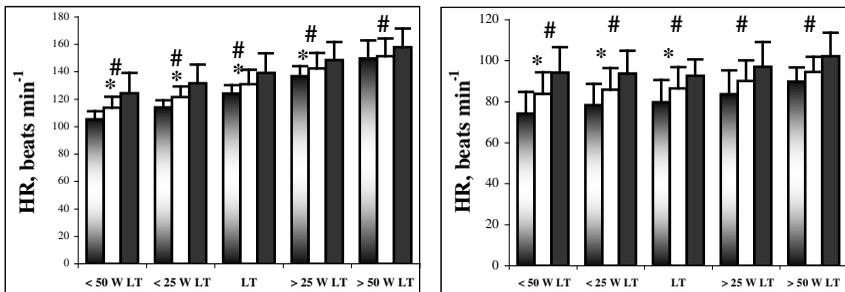


Fig. 6. The mean values of HR during 3rd min of work (on the left) and 4th min of recovery (on the right) during ICE on different testing conditions

Note. Denote grey columns – without prior load, white columns – after moderate aerobic exercise, black columns – after heavy aerobic exercise). * – Denote significant ($P < 0.05$) difference between conditions without prior load and after moderate aerobic exercise. # – Denote significant ($P < 0.05$) difference between conditions without prior load and after heavy aerobic exercise. LT is work rate at lactate threshold, <50 or <25 W LT is work rate below lactate threshold and >50 or >25 W LT is work rate above lactate threshold.

Table 2 shows mean values of the HR on- and off-kinetics assessed by fitting a mono-exponential function during ICE test on three different testing conditions.

Neither prior moderate nor heavy aerobic exercises had effect on the *HR* on- and off-kinetics parameters (amplitude and time constant) ($P > 0.05$), but time constant of *HR* off-kinetics after moderate and heavy aerobic exercise was significantly shorter ($P < 0.05$) when work rate was 50 W below *LT*, whereas this parameter was significantly faster when work rate was equal to *LT* ($P < 0.05$).

Table 2. Mean *HR* on- and off-kinetics values assessed by fitting of mono-exponential function during *ICE* on three different testing conditions.

Parameters	Work rate	Without prior load	After moderate aerobic exercise	After heavy aerobic exercise
On- transition				
<i>Amplitude</i> , beats · min ⁻¹	<50W <i>LT</i>	31.9 (12.4)	28.1 (12.0)	24.5 (14.0)
	<25W <i>LT</i>	35.7 (11.8)	36.0 (13.1)	36.1 (12.7)
	<i>LT</i>	46.6 (12.0)	46.1 (14.7)	44.8 (13.6)
	>25W <i>LT</i>	57.4 (11.6)	55.7 (13.8)	53.5 (13.8)
	>50W <i>LT</i>	61.6 (14.5)	60.5 (6.9)	58.3 (10.4)
<i>Time constant</i> , s	<50W <i>LT</i>	22.8 (11.7)	14.0 (3.7)	18.6 (5.5)
	<25W <i>LT</i>	22.2 (14.9)	19.7 (15.3)	25.9 (9.1)
	<i>LT</i>	30.9 (12.8)	25.7 (13.3)	32.8 (15.9)
	>25W <i>LT</i>	27.8 (14.3)	26.0 (7.4)	30.2 (11.2)
	>50W <i>LT</i>	37.1 (13.4)	34.3 (12.9)	32.3 (4.8)
Off- transition				
<i>Amplitude</i> , beats · min ⁻¹	<50W <i>LT</i>	33.1 (14.1)	29.8 (11.1)	30.6 (12.0)
	<25W <i>LT</i>	38.7 (13.3)	35.9 (10.9)	38.6 (11.0)
	<i>LT</i>	49.2 (12.0)	45.5 (12.8)	46.5 (14.7)
	>25W <i>LT</i>	54.4 (14.7)	56.1 (12.9)	57.9 (15.4)
	>50W <i>LT</i>	57.8 (11.3)	60.6 (7.2)	59.0 (10.6)
<i>Time constant</i> , s	<50W <i>LT</i>	19.1 (5.3)	34.3 (6.0) *	38.5 (18.2) **
	<25W <i>LT</i>	22.7 (7.8)	23.5 (8.9)	37.3 (18.3)
	<i>LT</i>	35.9 (11.0)	26.0 (7.1) *	38.0 (15.6)
	>25W <i>LT</i>	34.2 (9.4)	28.4 (6.0)	41.0 (18.6)
	>50W <i>LT</i>	47.7 (13.3)	42.5 (8.3)	41.0 (7.3)

Note. The standart deviations are presented in bracket. * – Denote significant ($P < 0.05$) difference between conditions without prior load and after moderate aerobic exercise. ** – Denote significant ($P < 0.05$) difference between conditions without prior load and after heavy aerobic exercise. *LT* is work rate at lactate threshold, <50 or <25 W *LT* is work rate below lactate threshold and >50 or >25 W *LT* is work rate above lactate threshold.

2.2. Influence of a prior eccentric-concentric exercise (EEC) on the kinetics of cardiorespiratory parameters during intermittent increasing cycling exercise (ICE).

Drop jump exercise was used to induce muscle fatigue, which would lead to a change in oxygen costs of the subsequent cycling exercise. Data on heights of these jumps are presented in Fig. 7. The jump heights tended to decrease during the exercise, but the decrease did not reach the significance level ($P > 0.05$), while after *EEC* at the 30th and 60th min of rest the heights of jumps were significantly lower ($P < 0.05$).

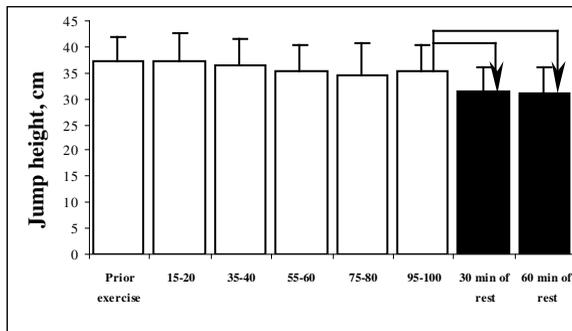


Fig. 7. Jump heights dynamic during repetitive drop jumps exercise

Note. Averages of each five consecutive jumps are presented. Values are means with standard deviations (SD) indicated by the bars. Arrows mark significant difference between values ($P < 0.05$).

Table 3. The cardiorespiratory parameters during rest before start of ICE on the different testing conditions.

Parameters	Without prior load	After prior load
Oxygen uptake ($\dot{V}O_2$) ($l \cdot \text{min}^{-1}$)	0.260 (0.052)	0.292 (0.063)
Carbon dioxide output ($\dot{V}CO_2$) ($l \cdot \text{min}^{-1}$)	0.208 (0.064)	0.224 (0.039)
Heart rate (<i>HR</i>) (beats $\cdot \text{min}^{-1}$)	74.9 (13.2)	80.2 (12.2)
Pulmonary ventilation (\dot{V}_E) ($l \cdot \text{min}^{-1}$)	7.6 (1.8)	9.0 (2.2)
Respiratory exchange ratio (<i>RER</i>)	0.781 (0.114)	0.772 (0.053)

Note. Values are means with standard deviations (SD) presented in brackets.

Table 3 shows the means of $\dot{V}O_2$, $\dot{V}CO_2$, HR , \dot{V}_E and RER during rest before start *ICE* exercise on the different testing conditions. One hour after *EEC* all above mentioned parameters were unaltered ($P > 0.05$) compared with parameters during rest before start *ICE* without prior load exercise.

All the participants cycled at 70, 105, 140 and 175 W in the control and fatigue conditions, respectively. The $\dot{V}O_2$ data from these experiments are presented in Fig. 8. In the control condition, $\dot{V}O_2$ increased with exercise intensity ($P < 0.05$) reaching 29.6 (5.4), 39.4 (7.0), 50.8 (8.4) and 65.8 (11.8) % of $\dot{V}O_2$ max at 70, 105, 140 and 175 W, respectively. The prior drop jump exercise tended to amplify the $\dot{V}O_2$ response to exercise. From 3rd to 6th min of cycling at 105, 140 and 175 W, $\dot{V}O_2$ was higher ($P < 0.05$) in the fatigue compared to the control condition. This could be due to changes in pulmonary ventilation and heart rate.

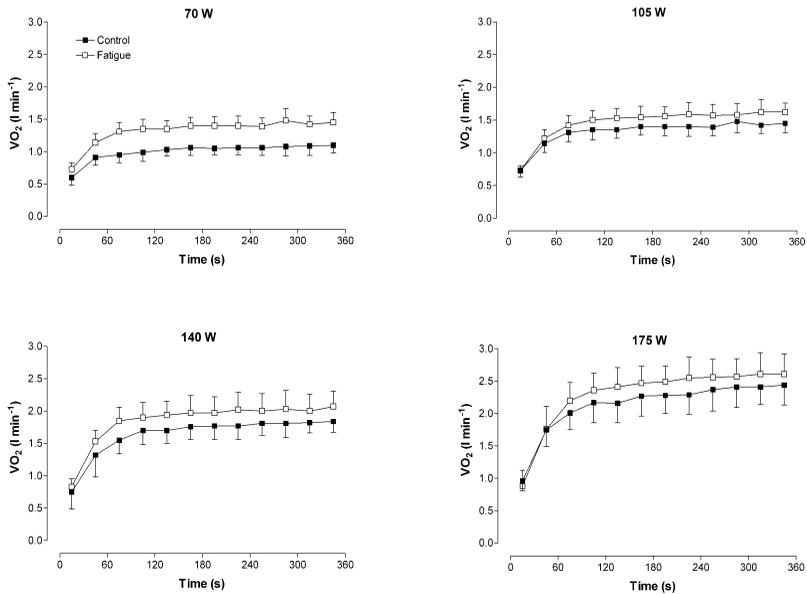


Fig. 8. Pulmonary oxygen uptake ($\dot{V}O_2$) during ergometer cycling at 70, 105, 140 and 175 W

Note. Values are means with standard deviations (SD) indicated by the bars.

Table 4. Expiratory pulmonary ventilation (\dot{V}_E), heart rate (HR) and pulmonary oxygen uptake ($\dot{V}O_2$) after subtraction of oxygen uptake of cardiac and respiratory muscles (see methods for details)

Parameters	Work rate	Without prior load	After prior load
\dot{V}_E ($l \cdot \text{min}^{-1}$)	70 W	24.1 (4.7)	26.6 (1.9)
	105 W	33.1 (5.6)	37.7 (5.6)
	140 W	43.3 (7.6)	51.2 (10.7) *
	175 W	60.7 (11.8)	71.5 (17.5) *
HR (beats $\cdot \text{min}^{-1}$)	70 W	109 (14)	113 (7)
	105 W	125 (16)	131 (8)
	140 W	140 (19)	149 (13)
	175 W	158 (20)	165 (13)
$Corrected\dot{V}O_2$ ($l \cdot \text{min}^{-1}$)	70 W	1.00 (0.12)	1.09 (0.08)
	105 W	1.34 (0.13)	1.50 (0.15) *
	140 W	1.71 (0.18)	1.90 (0.24) *
	175 W	2.23 (0.28)	2.41 (0.25) *

Note. All the data are averages over the last 3 min of cycling at *ICE* in different testing conditions. Values are means with standard deviations (SD) presented in bracket. * Denote significant ($P < 0.05$) differences between different testing conditions.

Data on \dot{V}_E , HR and $\dot{V}O_2$ corrected for oxygen uptake of cardiac and respiratory muscles are presented in Table 4. Indeed, \dot{V}_E and HR tended to increase when cycling was performed in the damage condition. However, this increase in \dot{V}_E and HR could account for only up to 10% of the overall increase in $\dot{V}O_2$ during cycling in the after prior load relative to the without prior load condition. At 105, 140 and 175 W of cycling, $\dot{V}O_2$ remained higher ($P < 0.05$) in the fatigue compared to the control condition even when oxygen uptake of cardiac and respiratory muscles was subtracted from the $\dot{V}O_2$ values.

Data on blood lactate and the amplitude of slow component of $\dot{V}O_2$ kinetics are presented in Table 5. The two-way ANOVA revealed a significant effect of the drop jump exercise neither on the blood lactate nor on the amplitude of the slow component of $\dot{V}O_2$. The blood lactate increased significantly ($P < 0.05$) only when exercise was performed at the highest intensity (175 W) in the control condition. Similar results were noted for the amplitude of the slow component of $\dot{V}O_2$. In the control condition, the slow component of $\dot{V}O_2$ kinetics was significant ($P < 0.05$) at 175 W as all seven subjects showed an increase in $\dot{V}O_2$

from 3rd min to 6th min of cycling. However, it tended to decrease in the fatigue condition.

Table 5. Blood lactate and slow component of $\dot{V}O_2$ kinetics (s. c. $\dot{V}O_2$) when cycling in different testing conditions.

Parameters	Work rate	Without prior load	After prior load
s. c. $\dot{V}O_2$ ($l \cdot \text{min}^{-1}$)	70 W	0.04 (0.10)	0.07 (0.09)
	105 W	0.06 (0.08)	0.08 (0.03)
	140 W	0.08 (0.07)	0.11 (0.11)
	175 W	0.17 (0.09) #	0.13 (0.15) #
Blood lactate (mM)	70 W	2.33 (0.71)	2.15 (1.02)
	105 W	2.22 (0.47)	2.15 (0.77)
	140 W	2.42 (0.73)	2.75 (0.95)
	175 W	3.93 (1.39) #	3.15 (1.13) #

Note. Values are means with standard deviations (SD) presented in bracket. # Denote significant ($P < 0.05$) differences from the values at 70 W.

Table 6. Mean $\dot{V}O_2$ on- and off-kinetics values assessed by fitting of mono-exponential function during ICE test on two different testing conditions

Parameters	Work rate	Without prior load	After heavy aerobic exercise
On- kinetics			
Amplitude, beats $\cdot \text{min}^{-1}$	< 35 W LT	1.168 (0.305)	1.288 (0.334) *
	LT	1.567 (0.383)	1.726 (0.412)
	> 35 W LT	2.057 (0.423)	2.254 (0.492) *
Time constant, s	< 35 W LT	16.3 (4.7)	16.9 (4.5)
	LT	22.9 (10.4)	16.2 (5.8)
	> 35 W LT	22.0 (4.8)	27.9 (12.8)
Off-kinetics			
Amplitude, beats $\cdot \text{min}^{-1}$	< 35 W LT	1.219 (0.339)	1.345 (0.352)
	LT	1.618 (0.330)	1.778 (0.404)
	> 35 W LT	2.215 (0.470)	2.309 (0.443)
Time constant, s	< 35 W LT	35.6 (7.3)	32.8 (5.0)
	LT	35.7 (5.0)	33.4 (3.9)
	> 35 W LT	40.3 (3.2)	37.8 (3.6)

Note. The standart deviations are presented in bracket. * Denote significant ($P < 0.05$) difference between two testing conditions. LT is work rate near lactate threshold, <35 W LT is work rate below lactate threshold and >35 W LT is work rate above lactate threshold.

Table 6 shows mean values of the $\dot{V}O_2$ on- and off-kinetics assessed by fitting a mono-exponential function during *ICE* test on two different testing conditions. Prior *EEC* had no effect on the $\dot{V}O_2$ on- and off-kinetics parameters (amplitude and time constant) ($P > 0.05$), but amplitude of $\dot{V}O_2$ on-kinetics after *EEC* was significantly higher ($P < 0.05$) when work rate was 35 W below and above *LT*.

Table 7 shows mean values of the *HR* on- and off-kinetics assessed by fitting of mono-exponential function during *ICE* test on two different testing conditions. Amplitude of *HR* on-kinetics was significantly increased ($P < 0.05$) after prior *EEC* when work rate was at *LT*, and time constant was significantly increased when work rate was 35 W below *LT*. Prior *EEC* had no effect on amplitude and time constant of the *HR* off-kinetics ($P > 0.05$).

Table 7. Mean *HR* on- and off-kinetics values assessed by fitting of mono-exponential function during *ICE* test on two different testing conditions.

Parameters	Work rate	Without prior load	After heavy aerobic exercise
On-kinetics			
<i>Amplitude,</i> beats · min ⁻¹	< 35 W <i>LT</i>	44.2 (12.8)	45.8 (14.7)
	<i>LT</i>	52.8 (13.4)	60.2 (16.8) *
	> 35 W <i>LT</i>	69.9 (19.0)	72.3 (16.8)
<i>Time constant,</i> s	< 35 W <i>LT</i>	18.5 (9.4)	31.1 (19.6) *
	<i>LT</i>	31.3 (13.8)	35.9 (14.5)
	> 35 W <i>LT</i>	37.3 (13.3)	49.8 (16.8)
Off-kinetics			
<i>Amplitude,</i> beats · min ⁻¹	< 35 W <i>LT</i>	42.1 (13.4)	44.5 (13.2)
	<i>LT</i>	53.9 (16.3)	58.5 (18.0)
	> 35 W <i>LT</i>	67.1 (19.7)	67.6 (15.9)
<i>Time constant,</i> s	< 35 W <i>LT</i>	35.3 (9.7)	30.3 (9.4)
	<i>LT</i>	43.4 (11.5)	51.0 (17.4)
	> 35 W <i>LT</i>	64.7 (23.2)	68.1 (19.9)

Note. The standart deviations are presented in bracket. * Denote significant ($P < 0.05$) difference between two testing conditions. *LT* is work rate near lactate threshold, <35 W *LT* is work rate below lactate threshold and >35 W *LT* is work rate above lactate threshold.

Table 8 shows mean values of the $\dot{V}CO_2$ on- and off-kinetics assessed by fitting of mono-exponential function during *ICE* test on two different testing conditions. Prior *EEC* had no effect on the amplitude and time constant of $\dot{V}CO_2$ on- and off-kinetics ($P > 0.05$), although the time constant of $\dot{V}CO_2$ off-kinetics decreased significantly after *EEC* ($P < 0.05$) when work rate was 35 W above *LT*.

Table 8. Mean $\dot{V}CO_2$ on- and off-kinetics values assessed by fitting of mono-exponential function during *ICE* test on two different testing conditions

Parameters	Work rate	Without prior load	After heavy aerobic exercise
On-kinetics			
<i>Amplitude,</i> beats · min ⁻¹	< 35 W <i>LT</i>	1.011 (0.250)	1.081 (0.257)
	<i>LT</i>	1.345 (0.312)	1.482 (0.374)
	> 35 W <i>LT</i>	1.837 (0.362)	2.015 (0.494)
<i>Time constant,</i> s	< 35 W <i>LT</i>	28.5 (10.1)	27.1 (8.5)
	<i>LT</i>	39.1 (17.1)	25.4 (7.3)
	> 35 W <i>LT</i>	39.9 (5.0)	41.3 (15.7)
Off-kinetics			
<i>Amplitude,</i> beats · min ⁻¹	< 35 W <i>LT</i>	1.025 (0.276)	1.119 (0.270)
	<i>LT</i>	1.376 (0.277)	1.500 (0.354)
	> 35 W <i>LT</i>	1.976 (0.391)	2.035 (0.495)
<i>Time constant,</i> s	< 35 W <i>LT</i>	45.3 (10.6)	39.7 (7.6)
	<i>LT</i>	44.5 (7.3)	38.9 (6.9)
	> 35 W <i>LT</i>	54.0 (8.0)	47.9 (6.7) *

Note. The standard deviations are presented in bracket. * Denote significant ($P < 0.05$) difference between two testing conditions. *LT* is work rate near lactate threshold, <35 W *LT* is work rate below lactate threshold and >35 W *LT* is work rate above lactate threshold.

Table 9 shows mean values of the \dot{V}_E on- and off-kinetics assessed by fitting of mono-exponential function during *ICE* test on two different testing conditions. Amplitude of \dot{V}_E on- and off-kinetics was significantly increased ($P < 0.05$) after prior *EEC*. Prior *EEC* had no effect on the time constant \dot{V}_E of on- and off-kinetics ($P > 0.05$), although it was significantly decreased after *EEC* ($P < 0.05$) when work rate was at *LT*.

Table 9. Mean \dot{V}_E on- and off-kinetics values assessed by fitting of mono-exponential function during *ICE* test on two different testing conditions

Parameters	Work rate	Without prior load	After heavy aerobic exercise
On- kinetics			
<i>Amplitude,</i> beats · min ⁻¹	< 35 W <i>LT</i>	24,5 (7,0)	28,5 (7,6) *
	<i>LT</i>	34,0 (10,6)	41,6 (13,3) *
	> 35 W <i>LT</i>	52,1 (13,4)	64,0 (22,1) *
<i>Time constant,</i> s	< 35 W <i>LT</i>	33,2 (9,2)	34,9 (12,8)
	<i>LT</i>	48,6 (16,9)	35,2 (10,3)
	> 35 W <i>LT</i>	57,0 (8,3)	59,5 (20,4)
Off-kinetics			
<i>Amplitude,</i> beats · min ⁻¹	< 35 W <i>LT</i>	24,9 (8,8)	29,9 (8,5) *
	<i>LT</i>	35,9 (9,3)	43,9 (13,9) *
	> 35 W <i>LT</i>	56,4 (12,5)	65,2 (22,1)
<i>Time constant,</i> s	< 35 W <i>LT</i>	48,6 (10,9)	46,3 (12,9)
	<i>LT</i>	51,2 (11,6)	42,3 (11,7) *
	> 35 W <i>LT</i>	59,8 (16,7)	53,5 (11,6)

Note. The standart deviations are presented in bracket. * Denote significant (P < 0.05) difference between two testing conditions. *LT* is work rate near lactate threshold, <35 W *LT* is work rate below lactate threshold and >35 W *LT* is work rate above lactate threshold.

2.3. Influence of a prior anaerobic load on cardiorespiratory and blood lactate parameters during intermittent increasing cycling exercise (ICE)

The dynamics of blood lactate concentration $[La]$ during ICE was changed after prior anaerobic load, e.g. under conditions of metabolic acidosis. After decrease at initial loads it started increasing at the intensities near the *LT* (Table 10, fig. 9, right). Under both conditions the *HR* increased linearly as the function of work intensity, but was significantly higher ($P < 0.05$) at lower intensities after prior anaerobic load (Table 10, fig. 9).

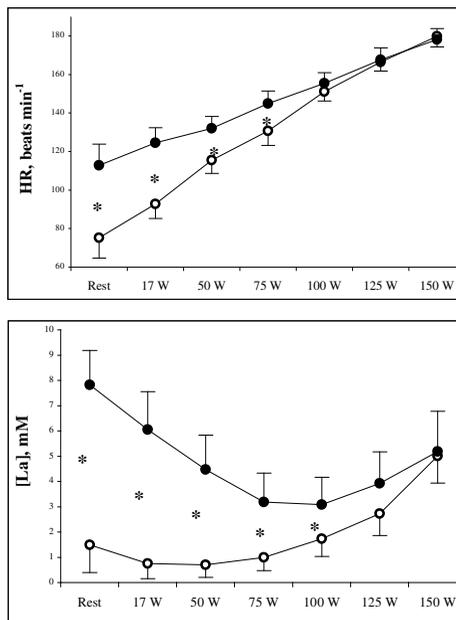


Fig. 9. The heart rate (above) and blood lactate concentration (below) dynamics during ICE on different testing conditions

Note. White circles – without prior load, black circles – after prior load. The standart deviations presented by the bars. * – Denote significant ($P < 0.05$) difference between two testing conditions. 17, 50, 75, 100, 125 and 150 W is work rate.

The two-way ANOVA revealed a significant effect (intensity – $P < 0.001$; prior load – $P < 0.001$; interaction – $P < 0.001$) of the prior anaerobic load on the blood lactate and the HR during ICE (fig. 9).

Table 10. Mean values of heart rate (HR) and blood lactate concentration [La] during ICE on different testing conditions

Work rate	HR , beats \cdot min ⁻¹		[La], mM	
	Without prior load	After prior load	Without prior load	After prior load
Rest	75.1 (10.5)	112.7 (11.0) *	1.49 (1.10)	7.83 (1.36) *
17 W	92.8 (7.5)	124.5 (7.9) *	0.76 (0.61)	6.05 (1.49) *
50 W	115.4 (6.8)	131.9 (6.2) *	0.70 (0.50)	4.47 (1.36) *
75 W	130.6 (7.4)	144.8 (6.5) *	0.99 (0.52)	3.18 (1.15) *
100 W	151.0 (4.9)	155.5 (5.4)	1.73 (0.70)	3.08 (1.08) *
125 W	166.4 (4.6)	167.5 (6.2)	2.73 (0.87)	3.92 (1.26)
150 W	179.9 (5.5)	178.2 (5.6)	5.01 (1.07)	5.19 (1.59)

Note. The standard deviations are presented in brackets. * – Denote significant ($P < 0.05$) difference between two testing conditions.

Table 11 and Fig. 10 show dynamics of the pulmonary gas exchange parameters ($\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E , RER , $P_{ET}O_2$, $P_{ET}CO_2$, EqO_2 , $EqCO_2$) during ICE on different testing conditions.

The two-way ANOVA revealed that under both conditions the pulmonary oxygen uptake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$) and pulmonary ventilation (\dot{V}_E) increased significantly as the function of work intensity ($P < 0.001$). The parameters mentioned above were significantly higher ($P < 0.05$) at rest under conditions of metabolic acidosis. However, no effect of prior anaerobic load on dynamics of $\dot{V}O_2$, $\dot{V}CO_2$ and \dot{V}_E was found.

The dynamics of RER during ICE was changed after prior anaerobic load. At rest RER was significantly higher under conditions of metabolic acidosis ($P < 0.05$). During ICE it decreased at first to loads and then started increasing but remained lower than under normal conditions. Similarly, also $P_{ET}O_2$ decreased during first two loads under both conditions but after prior anaerobic load it was higher at all intensities. The values of $P_{ET}CO_2$ and $EqCO_2$ were significantly decreased at all intensities in comparison with control values (fig. 10).

Table 11. Pulmonary gas exchange parameters ($\dot{V}O_2, \dot{V}CO_2, \dot{V}_E, RER, P_{ET}O_2, P_{ET}CO_2, EqO_2, EqCO_2$) values during ICE on different testing conditions

Work rate	$\dot{V}O_2, \text{ml} \cdot \text{min}^{-1}$		$\dot{V}CO_2, \text{ml} \cdot \text{min}^{-1}$	
	Without prior load	After prior load	Without prior load	After prior load
Rest	279.6 (35.0)	374.7 (79.7) *	234.3 (34.6)	356.5 (95.3) *
17 W	552.9 (108.6)	631.2 (98.0)	443.1 (90.5)	517.4 (103.2)
50 W	971.6 (81.3)	932.8 (91.1)	800.6 (104.8)	667.1 (72.3) *
75 W	1195.6 (90.4)	1190.5 (79.5)	1057.5 (78.6)	974.7 (70.2)
100 W	1456.0 (95.5)	1452.8 (69.3)	1380.6 (101.5)	1282.7 (47.3) *
125 W	1735.3 (93.2)	1732.7 (99.5)	1724.4 (143.4)	1649.5 (98.4)
150 W	2002.3 (85.0)	2050.8 (89.1)	2066.3 (176.6)	2053.4 (158.7)
	$\dot{V}_E, \text{l} \cdot \text{min}^{-1}$		<i>RER</i>	
Rest	10.2 (1.3)	17.1 (3.3) *	0.83 (0.06)	0.94 (0.07) *
17 W	16.5 (3.1)	22.6 (4.5) *	0.80 (0.09)	0.80 (0.08)
50 W	25.1 (4.2)	25.8 (3.2)	0.82 (0.09)	0.72 (0.05) *
75 W	33.4 (4.1)	34.1 (4.3)	0.91 (0.07)	0.84 (0.06)
100 W	41.4 (5.3)	42.9 (4.4)	0.96 (0.08)	0.90 (0.04)
125 W	50.1 (6.4)	52.0 (5.8)	1.00 (0.09)	0.97 (0.03)
150 W	63.4 (8.9)	65.3 (7.0)	1.05 (0.08)	1.01 (0.03)
	$P_{ET}O_2, [\text{kPa}]$		$P_{ET}CO_2, [\text{kPa}]$	
Rest	14.87 (0.57)	15.85 (0.23) *	4.19 (0.44)	3.69 (0.30) *
17 W	14.08 (0.58)	15.09 (0.56) *	4.74 (0.30)	3.92 (0.31) *
50 W	13.55 (0.91)	14.07 (0.49)	5.36 (0.47)	4.53 (0.46) *
75 W	13.82 (0.71)	14.13 (0.62)	5.49 (0.41)	4.89 (0.46) *
100 W	13.98 (0.79)	14.32 (0.55)	5.60 (0.47)	5.02 (0.50) *
125 W	14.25 (0.79)	14.60 (0.60)	5.52 (0.47)	5.06 (0.55)
150 W	14.78 (0.66)	15.00 (0.54)	5.25 (0.47)	4.87 (0.55)
	EqO_2		$EqCO_2$	
Rest	32.40 (2.58)	41.94 (2.60) *	39.05 (3.33)	44.92 (4.07) *
17 W	27.00 (3.10)	33.29 (4.85) *	33.70 (2.41)	41.16 (2.96) *
50 W	24.17 (3.18)	25.62 (2.75)	28.75 (2.91)	34.17 (3.48) *
75 W	25.05 (2.84)	26.02 (3.69)	27.60 (2.37)	30.98 (3.34) *
100 W	25.68 (3.23)	26.69 (3.53)	26.66 (2.22)	29.73 (3.59)
125 W	26.73 (3.98)	27.62 (3.80)	26.74 (2.18)	28.57 (3.46)
150 W	29.20 (4.45)	29.57 (3.51)	27.66 (2.64)	29.11 (3.00)

Note. The standard deviations (SD) are presented in brackets. * – Denote significant ($P < 0.05$) difference between testing conditions.

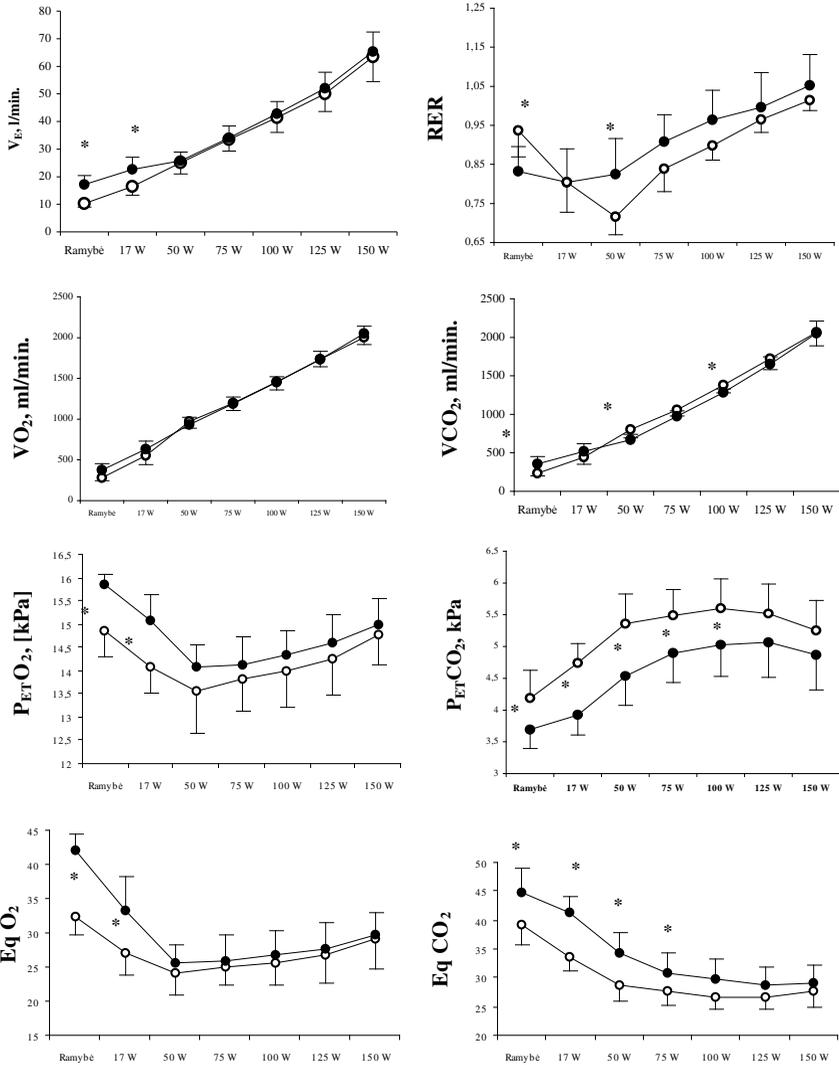


Fig. 10. Pulmonary gas exchange dynamics during ICE on different testing conditions

Note. White circles – without prior load, black circles – after prior load. The standard deviations presented by the bars. * – Denote significant ($P < 0.05$) difference between two testing conditions. 17, 50, 75, 100, 125 and 150 W is work rate.

2.4. The cardiorespiratory parameters during on- and off-transitions of intermittent increasing cycling exercise (ICE) after a prior anaerobic load

To compare the cardiorespiratory parameters between testing conditions (without and after prior anaerobic load) data were normalized to each individual's *LT*. Fig. 11 shows mean blood lactate concentration at different testing conditions indicating the presence of a residual metabolic acidosis during *ICE*.

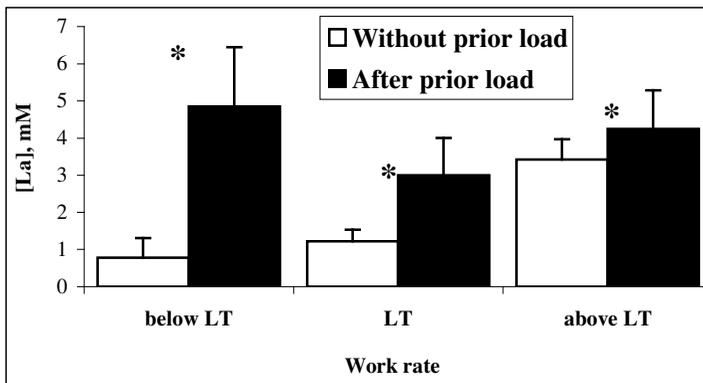


Fig. 11. Mean blood lactate concentration [La] during intermittent increasing cycling exercise on different testing conditions.

Note. The standard deviations presented by the bars. * – Denote significant ($P < 0.05$) difference between testing conditions. *LT* is work rate at lactate threshold.

The asymptote, amplitude and time constant of HR , $\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E kinetics during on- and off-transitions are given in Tables 12-15.

After prior anaerobic load the HR asymptote of monoexponential function during on-transition was significantly higher ($P < 0.01$) at the intensity below *LT*, the amplitude was significantly decreased at all work rates ($P < 0.05$) and the time constant was no changed. The amplitude and time constant of monoexponential function during off-transition were not changed after prior anaerobic load, but the asymptote was significantly higher ($P < 0.05$). The two-way ANOVA revealed that both during on- and off-transitions all parameters of monoexponential function did significantly ($P < 0.001$) increase in parallel with work intensity (tab. 12).

Table 12. Parameters of HR changes assessed by adopting monoexponential function during on- and off-transition periods of intermittent increasing exercise performed without and after prior anaerobic load

Parameters	Work rate	On-transition		Off-transition	
		Without PAnL	After PAnL	Without PAnL	After PAnL
Asymptote, beats · min ⁻¹	< LT	115.8(13.3)	134.1(8.9)*	76.3(6.7)	107.5(5.6)*
	LT	138.5(14.7)	150.3(8.2)	95.0(10.1)	112.1(6.7)*
	> LT	165.9(10.2)	167.2(9.0)	105.3(10.8)	116.8(6.3)*
Amplitude, beats · min ⁻¹	< LT	44.2(10.8)	27.6(8.4)*	39.6(10.2)	26.4(8.5)*
	LT	59.5(11.9)	39.8(6.3)*	43.0(10.2)	38.0(6.6)
	> LT	70.1(9.3)	54.5(8.9)*	60.6(9.3)	50.4(10.5)
Time constant, s	< LT	24.4(3.9)	26.9(5.4)	22.7(1.9)	26.9(5.8)
	LT	27.3(9.7)	33.4(8.2)	24.8(5.9)	34.0(9.6)
	> LT	39.1(6.0)	37.0(7.3)	52.8(7.7)	52.2(5.4)

Note. The standart deviations are presented in brackets. * – Denote significant (P < 0.05) difference between testing conditions. *LT* is work rate near lactate threshold; < *LT* is work rate below lactate threshold and > *LT* is work rate above lactate threshold.

Table 13. Parameters of $\dot{V}O_2$ kinetics during on- and off-transition periods of intermittent increasing exercise without and after prior anaerobic load.

Parameters	Work rate	On-transition		Off-transition	
		Without PAnL	After PAnL	Without PAnL	After PAnL
Asymptote, ml · min ⁻¹	< LT	994.7(268.4)	989.4(246.7)	264.8(65.6)	285.3(30.2)
	LT	1383.0(272.3)	1411.0(265.0)	286.1(76.3)	305.4(64.7)
	> LT	1808.0(269.1)	1807.6(292.5)	347.2(41.0)	390.7(69.0)
Amplitude, ml · min ⁻¹	< LT	750.9(251.6)	670.1(234.2)	732.0(224.2)	705.5(250.4)
	LT	1095.0(212.7)	1151.1(267.2)	114.6(227.0)	1087.4(264.6)
	> LT	1504.5(237.0)	1461.4(265.8)	1453.2(245.7)	1412.0(283.0)
Time constant, s	< LT	19.8 (4.9)	21.5 (3.2)	35.7 (3.5)	42.5(5.9)*
	LT	20.7 (5.4)	19.6 (3.7)	46.4 (6.1)	45.6(7.1)
	> LT	21.7 (4.7)	21.0 (4.2)	45.7 (4.8)	42.9(2.8)

Note. The standard deviations (SD) are presented in brackets. * Denote significant (P < 0.05) difference between testing conditions. *LT* is work rate at lactate threshold; < *LT* is work rate below lactate threshold and > *LT* is work rate above lactate threshold.

The prior anaerobic load had no effect on the asymptotic value and amplitude of $\dot{V}O_2$ monoexponential function during on- and off-transitions, while this values increased in parallel with work intensities ($P < 0.001$). The time constants of $\dot{V}O_2$ monoexponential function during on- and off-transitions also did not change except for recovery after below LT exercise where it was significantly longer ($P < 0.02$) after prior anaerobic load. The work rate had no effect on the $\dot{V}O_2$ time constants during on-transitions and off-transitions (tab. 13).

The asymptote and amplitude of monoexponential function reflecting $\dot{V}CO_2$ kinetics during on- and off-transitions were not changed after prior anaerobic load. The time constant of this function was significantly longer ($P < 0.01$) both during on- and off-transition at work rate below LT , whereas this parameter at higher work intensities remained unchanged. The two-way ANOVA revealed that both during on- and off-transitions all the mentioned above parameters of $\dot{V}CO_2$ kinetics increased significantly ($P < 0.001$) with intensity under both testing conditions (tab. 14).

Table 14. Parameters of $\dot{V}CO_2$ kinetics during on- and off-transition periods of intermittent increasing exercise without and after preceding anaerobic load

Parameters	Work rate	On-transition		Off-transition	
		Without <i>PAnL</i>	After <i>PAnL</i>	Without <i>PAnL</i>	After <i>PAnL</i>
Asymptote, $\text{ml} \cdot \text{min}^{-1}$	< <i>LT</i>	850.5(263.4)	787.6(224.2)	244.6(67.8)	249.9(43.9)
	<i>LT</i>	1308.7(290.1)	1246.1(267.5)	302.3(84.0)	306.5(63.9)
	> <i>LT</i>	1827.3(294.7)	1763.9(321.9)	413.7(57.8)	435.5(86.4)
Amplitude, $\text{ml} \cdot \text{min}^{-1}$	< <i>LT</i>	640.4(240.9)	505.5(207.7)	607.3(214.8)	536.1(201.1)
	<i>LT</i>	1034.9(236.2)	1012.4(247.4)	1011.1(249.2)	915.1(232.9)
	> <i>LT</i>	1492.9(257.5)	1403.0(277.8)	1405.6(273.0)	1322.9(281.9)
Time constant, s	< <i>LT</i>	29.0(4.4)	39.8(6.1)*	42.0(4.7)	50.1(5.9)*
	<i>LT</i>	36.4(4.4)	37.2(4.5)	58.4(5.7)	54.9(10.5)
	> <i>LT</i>	41.4(5.2)	41.9(5.5)	63.7(7.6)	58.6(4.1)

Note. The standard deviations (SD) are presented in brackets. * – Denote significant ($P < 0.05$) difference between testing conditions. *LT* is work rate at lactate threshold; < *LT* is work rate below lactate threshold and > *LT* is work rate above lactate threshold.

The parameters of \dot{V}_E monoexponential function during on- and off-transitions were not changed after prior anaerobic load. The two-way ANOVA revealed that both during on- and off-transitions all the mentioned above parameters of \dot{V}_E

kinetics did significantly increase ($P < 0.001$) with intensity under both testing conditions (tab. 15).

Table 15. Parameters of \dot{V}_E changes during on- and off-transition periods of intermittent increasing exercise without and after prior anaerobic load

Parameters	Work rate	On-kinetics		Off-kinetics	
		Without <i>PAnL</i>	After <i>PAnL</i>	Without <i>PAnL</i>	After <i>PAnL</i>
Asymptote, $l \cdot \text{min}^{-1}$	< <i>LT</i>	26.1(6.4)	28.5(4.8)	10.6(2.2)	12.5(2.2)
	<i>LT</i>	37.1(6.7)	38.7(6.4)	12.9(2.9)	14.1(1.9)
	> <i>LT</i>	51.3(8.2)	52.7(6.8)	17.7(2.3)	19.3(3.7)
Amplitude, $l \cdot \text{min}^{-1}$	< <i>LT</i>	16.6(5.7)	14.4(4.9)	15.6(4.9)	16.0(5.1)
	<i>LT</i>	25.7(5.7)	27.2(6.8)	24.2(6.1)	24.3(5.0)
	> <i>LT</i>	37.0(8.4)	36.5(6.4)	33.4(8.6)	33.3(6.9)
Time constant, s	< <i>LT</i>	30.2(5.8)	35.9(8.5)	46.1(4.1)	53.9(9.6)
	<i>LT</i>	36.0(3.7)	38.9(6.1)	65.1(6.9)	63.2(13.0)
	> <i>LT</i>	42.1(2.8)	44.6(5.5)	69.1(8.3)	64.7(7.4)

Note. The standart deviations (SD) are presented in brackets. * – Denote significant ($P < 0.05$) difference between two testing conditions. *LT* is work rate at lactate threshold; < *LT* is work rate below lactate threshold and > *LT* is work rate above lactate threshold.

2.5. Effect of prior incremental cycling exercise on pulmonary oxygen uptake kinetics during moderate and heavy intensity exercise

As a prior load the participants performed the continuous increasing cycling exercise test (*ICE*). The work rate during the 6 min of moderate (*EM*) and heavy (*EH*) exercise bouts averaged 115.1 (17.2) and 215.7 (43.5) W, the relative $\dot{V}O_2$ was 48.8 (3.06) and 69.4 (8.58) % of $\dot{V}O_{2\max}$, respectively. The mean blood lactate concentration just before the start of *EM* and *EH* was 6.04 (1.1) and 6.22 (1.5) mM respectively, indicating the presence of a residual metabolic acidosis. After experimental exercise it was more decreased in *EM* group than in *EH* one (Table 16).

Table 16. Blood lactate responses to control, incremental and experimental exercises in two groups.

[La], mM	<i>EM</i> group	<i>EH</i> group
At rest	1.66(0.44)	1.34(0.40)
After control exercise	1.62(0.93) #	4.28(2.16)
5 th min after <i>ICE</i>	6.78(1.32)	6.60(2.06)
10 th min after <i>ICE</i> , before experimental exercise	6.04(1.12)	6.22(1.52)
After experimental exercise	3.92(1.47) *#	5.41(2.60) *

Note. * – Significant difference between control and experimental exercise ($P < 0.05$). # – Significant difference between groups ($P < 0.05$)

The time course of mean responses for $\dot{V}O_2$ from unloaded cycling to exercise in *EM* and *EH* groups is shown in Fig. 12. The mean $\dot{V}O_2$ values at rest, during unloaded cycling and during exercise in *EM* and *EH* groups are presented in Fig. 13. The increase of mean group $\dot{V}O_2$ values at rest and during unloaded cycling was similar in both groups (170.6 and 250.6, 164.0 and 247.9 ml min^{-1} in *EM* and *EH* groups, respectively). Mean $\dot{V}O_2$ values at 3rd and 6th min of exercise were more increased in *EH* group (by 397.6 and 273.9 ml \cdot min⁻¹) as compared with *EM* group (by 225.5 and 222.5 ml \cdot min⁻¹).

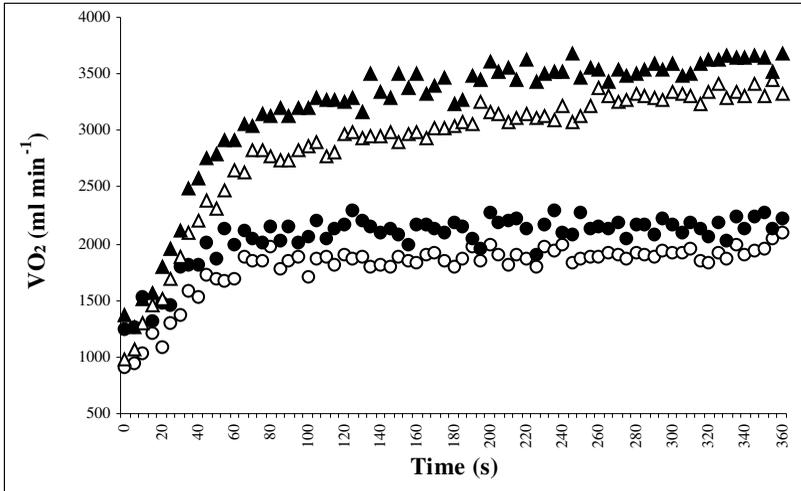


Fig. 12. The oxygen uptake ($\dot{V}O_2$) means group responses to moderate (\circ) and heavy (Δ) exercises.

Note. *Open symbols* represent the control exercise bout and *closed symbols* represent the bout performed following prior exercise.

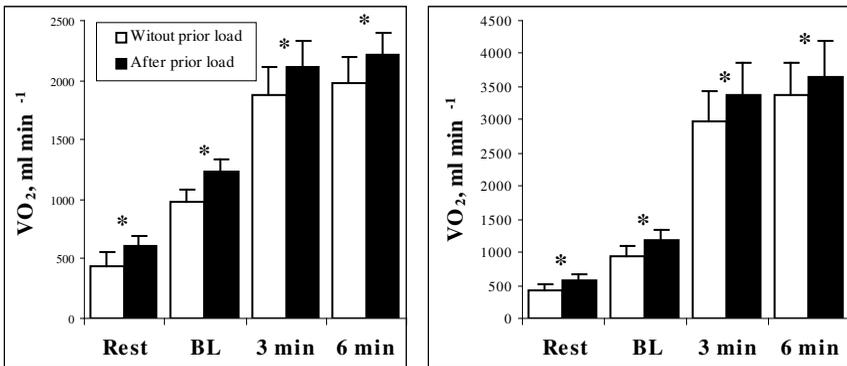


Fig. 13. Means values of the absolute $\dot{V}O_2$ at rest, during unloaded cycling (*BL*), at 3rd and 6th min of exercise.

Note. The *left figure* represents the moderate exercise group and the *right figure* represents the to heavy exercise group. Standard deviations presented by the bars. * – Denote significant difference ($P < 0.05$) between control and experimental exercises.

Table 17. Parameters of oxygen uptake responses during moderate exercises performed before and after ICE.

Parameters	Control	Experimental
A1 (ml · min ⁻¹)	906.1 (202.9)	908.2 (207.0)
τ1 (s)	18.2 (7.4)	13.8 (4.2)
BL+A1 (ml · min ⁻¹)	1889.6 (209.3)	2142.3 (204.1)*
SC ^{6⁻3[']} (ml · min ⁻¹)	103.1 (58.6)	100.1 (46.8)

Note. BL – base line of $\dot{V}O_2$, τ1 time constant of the $\dot{V}O_2$ primary response, A1 amplitude of the $\dot{V}O_2$ primary response, SC^{6⁻3[']} difference between the end exercise $\dot{V}O_2$ and 3rd min $\dot{V}O_2$, * denote significant difference between control and experimental exercise (P < 0.05).

The parameters of the $\dot{V}O_2$ response assessed by adopting exponential functions to moderate and heavy intensity exercises are summarized in Tables 17 and 18. The parameters of the primary $\dot{V}O_2$ response not affected by prior ICE in both groups. The time constant of the $\dot{V}O_2$ slow component was significantly decreased in EH group, though the amplitude of the slow component did not change. These changes resulted in the decrease of the SC^{6⁻3[']} but the change was not significant.

Table 18. Parameters of oxygen uptake responses during heavy exercises performed before and after CCE.

Parameters	Control	Experimental
A1 (ml · min ⁻¹)	1472.0 (360.6)	1474.1 (342.9)
τ1 (s)	10.7 (7.3)	10.0 (4.0)
A2 (ml · min ⁻¹)	1138.8 (722.0)	1013.8 (426.1)
τ2 (s)	176.2 (55.3)	107.8 (50.8) *
BL+A1 (ml · min ⁻¹)	2414.9 (323.2)	2664.8 (415.5) *
BL+A1+A2 (ml · min ⁻¹)	3553.7 (704.7)	3678.6 (644.6)
$\dot{V}O_{2EE}$ (ml · min ⁻¹)	3382.0 (499.9)	3650.2 (576.8) *
SC ^{6⁻3[']} (ml · min ⁻¹)	389.8 (166.9)	266.1 (150.4)

Note. BL – base line of $\dot{V}O_2$, $\dot{V}O_{2EE}$ end exercise of $\dot{V}O_2$, τ1 time constant of the $\dot{V}O_2$ primary response, τ2 time constant of the $\dot{V}O_2$ slow component, A1 amplitude of the $\dot{V}O_2$ primary response, A2 amplitude of the $\dot{V}O_2$ slow component, SC^{6⁻3[']} difference between the end exercise $\dot{V}O_2$ and 3rd min $\dot{V}O_2$, * denote significant difference between control and experimental exercise (P < 0.05).

3. GENERAL DISCUSSION

The dynamics of $\dot{V}O_2$ is considered as very important measure of organism ability to perform so called aerobic exercise, when the majority of energy is produced by aerobic *ATP* production. Four variables characterizing individual's aerobic capacity are usually determined: (1) the $\dot{V}O_2$ max, (2) the lactate or ventilatory thresholds, (3) the work efficiency and (4) the kinetics of $\dot{V}O_2$ and other cardiorespiratory parameters during exercise and recovery (Whipp et al., 1982; Jones & Carter, 2000). This study was focused mainly on the work efficiency and kinetics of cardiorespiratory parameters though some aspects of it deal with the problem of anaerobic thresholds determination. The main experimental factor used in our study was the influence of different prior load on the changes of parameters mentioned above during following exercise. Such experimental conditions are often used in many laboratories of exercise physiology and are thought to provide better understanding of factors that determine the rate of $\dot{V}O_2$ kinetics and the effect of warm-up on the acute adaptation and performance during following laboratory or competitive exercise. It is supposed that warm-up can cause faster $\dot{V}O_2$ increase during on-transition to exercise and thereby may decrease oxygen deficit, diminish the role of anaerobic *ATP* production, reduce lactate concentration in muscles and blood and consequently help to avoid to early fatigue and improve competitive results (Jones & Poole, 2005). To our knowledge this study was the first that examined the influence of prior eccentric-concentric (drop jumps) exercise and prior continuously increasing cycling exercise on the aerobic work efficiency and kinetics of many cardiorespiratory measures during on- and off-transition phases of working and recovery periods of intermittent increasing cycling exercise. We demonstrated that prior load had no influence on the fast component of $\dot{V}O_2$ kinetics at the onset of exercise, but the aerobic work efficiency was reduced after prior heavy aerobic or eccentric-concentric exercise. This may help to understand better the role of the mode and intensity of warm-up or prior load on the factors that determine human's aerobic performance.

The results of our second and fourth studies confirmed that the type of $\dot{V}O_2$ kinetics is dependent first of all on the exercise intensity (Astrand & Saltin, 1961; Casaburi et al., 1987, 1989; Poole et al., 1991; Barstow & Mole 1991; Barstow et al., 1994; Poole, 1994; Whipp, 1994; Gaesser & Poole, 1996; Barstow et al., 2000; Ozyener et al., 2001). During the aerobic exercise of moderate intensity

(below LT) the kinetics of $\dot{V}O_2$ consists of two phases (Henry & De Moor, 1956; Whipp, 1970; Cerretelli & Di Prampero, 1987). The first phase is called cardiodynamic (depends on increased venous blood return at the start of exercise) and lasts for approximately 15-20 s (Casabury et al., 1989). The second phase (fast component) is characterized by rapid increase of $\dot{V}O_2$ until reaching steady state (Whipp & Wasserman, 1972; Grassi et al., 1996; Rossiter et al., 1999; Brittain et al., 2001; Carter et al., 2000). The $\dot{V}O_2$ response in this phase is modelled by applying monoexponential function with the amplitude of 9-10 ml/min/W (Poole et al., 1992) and the time constant of 15-40 s (Whipp et al., 1982; Hughson et al., 1988; Barstow et al., 1996). During recovery after moderate intensity exercise similar monoexponential kinetics of $\dot{V}O_2$ is also observed (Linnarsson, 1974; Paterson & Whipp, 1991). During on-transition to heavy aerobic exercise (above LT) the third phase of $\dot{V}O_2$ response, so called slow component, appears and it is characterized by slow increase in $\dot{V}O_2$ (Whipp, 1987).

It remains not clear what is the relative role of oxygen supply to working muscles and that of processes determining intramuscular $\dot{V}O_2$ on the rate of pulmonary $\dot{V}O_2$ kinetics at the start of exercise (Tschakovsky & Hughson, 1999). If it were more dependent on the oxygen supply, its increase should result in fasting of pulmonary $\dot{V}O_2$. On the contrary, if the experimental results show opposite than the predominating role of intramuscular processes is emphasized (Mahler, 1980).

It is believed that prior load can change oxygen supply to working muscles during following exercise. The altered oxygen supply might be reflected in HR changes before and at the start of exercise. We analysed the changes of HR kinetics after different prior loads in first and fourth studies. We confirmed the well-known fact that HR changes in monoexponential manner at the start of exercise and during recovery and approaches the steady state level in 3-5 min (Davies et al., 1972; Hagberg et al., 1980; Astrand & Rodahl, 1986; Taylor et al., 1999). This is regulated by decreased parasympathetic and increased sympathetic influence during on-transition while opposite is true during recovery (Orizio et al., 1988; Perini et al., 1989; Pierpoint et al., 2000). The important role of hormonal and intramuscular mechanisms in the HR regulation, especially during intensive exercise, is also well documented (Christensen & Galbo, 1983; Wallin et al., 1987). The changes in HR during work and recovery were correlated with the intensity of exercise and blood catecholamine concentration (Orizio et al., 1988; Perini et al., 1989). We have also observed that the rate of HR kinetics during transitions between exercise and recovery is dependent on the exercise

intensity. During intermittent increasing cycling exercise the nonlinear (phasic) relationship between the work rate and HR at the end of recovery periods was demonstrated and this agrees with our previous findings (Stasiulis, 1997). The time constants of HR kinetics both during work and recovery increased in parallel with work intensity and this confirmed similar results published by Orizio et al. (1988) and Perini et al. (1989).

The present study (protocols 1 and 4) did not reveal any significant influence of prior heavy aerobic or anaerobic exercise on HR kinetics during transitional phases of repeated exercise. This is in accordance with data published by Bearden and Moffatt (2001). However, we measured only HR that is only one determinant of cardiac output. As was shown by Yoshida et al. (1995), both $\dot{V}O_2$ and cardiac output kinetics were speeded during repetitive one leg exercise. Recently, investigations using Doppler ultrasound have shown that speeding of the $\dot{V}O_2$ kinetics in the second bout of high-intensity knee extension exercise was mainly associated with widening of arteriovenous O_2 difference and the increase in cardiac output might contribute to this speeding only during a very early stage (the first 50 s) of the transition (Fukuba et al., 2007). So, even prior intensive load may cause metabolic acidosis, increase HR and $\dot{V}O_2$ before exercise, stimulate muscle blood flow and facilitate oxygen extraction by muscles, it does not always result in faster $\dot{V}O_2$ kinetics, especially in phase II of the response. This is confirmed by numerous data about no influence of intensive arms or legs prior exercise on the parameters (amplitude and time constant) of fast component of $\dot{V}O_2$ response during repetitive intensive exercise (Scheuermann et al., 2001; Fukuba et al., 2002; Koppo et al. 2003). Our study also showed no effect of prior exercise on the time constants of the fast component of $\dot{V}O_2$ response during exercise of both moderate and heavy intensities. Recently, A. Marles et al. (2007) have also demonstrated, that the parameters of the phase II $\dot{V}O_2$ kinetics were unaffected by prior heavy exercise, while some parameters of local muscle deoxygenation kinetics were significantly faster. On the contrary, the speeding of $\dot{V}O_2$ response was observed during repetitive running at the intensity of $\dot{V}O_2$ max (Billat et al., 2000) or during heavy exercises preceded by very intensive ones (Rossiter et al., 2001; Tordi et al., 2003). In accordance with findings of B. J. Gurd et al. (2006), faster $\dot{V}O_2$ kinetics during moderate intensity exercise performed after heavy exercise may be associated both with elevated pyruvate dehydrogenase activity and greater muscle perfusion. Prior load, especially of heavy intensity usually influences the $\dot{V}O_2$ slow component

as was demonstrated in this study as well. For estimation of $\dot{V}O_2$ slow component we calculated both parameters of biexponential function and differences in $\dot{V}O_2$ between 6th and 3rd min of heavy exercise as proposed by many investigators (Gerbino et al., 1996; MacDonald et al., 1997; Bohnert et al., 1998). The slow component of $\dot{V}O_2$ response appears only during exercise above *LT* (Paterson & Whipp, 1991). Our study has confirmed this observation. In addition, heavy prior aerobic anaerobic load (fourth study protocol) caused a decrease of time constant and amplitude of $\dot{V}O_2$ slow component by 44 and 49%, respectively. Similar effect of prior heavy exercise was demonstrated in other studies as well (Gerbino et al., 1996; MacDonald et al., 1997; Burnley et al., 2000; 2006). The mechanisms of the slow component of $\dot{V}O_2$ have not been determined. Most of investigators agree that is associated with changes in intramuscular $\dot{V}O_2$ (Poole et al., 1991; Stringer et al., 1994; Belardinelli et al., 1995). Endo et al. (2004) supposed that factors located in muscle but not that related to cardiovascular system are responsible for attenuation of $\dot{V}O_2$ slow component during repetitive heavy exercise.

In the present study, we used as heavy prior load continuously increasing exercise that is usually applied for aerobic capacity evaluation. Its effect on following exercise was similar to that observed by using single constant rate heavy exercise. Moreover, we showed that after such prior load the absolute $\dot{V}O_2$ was increased, e.g. work efficiency was decreased both during moderate and heavy constant rate exercise. J. U. Gonzales and B. W. Scheuermann (2006) have also demonstrated, that prior heavy exercise increases oxygen cost during moderate exercise without associated change in surface EMG. So, the measurement of work economy or efficiency under such conditions seems not to be advisable.

The results of our third study protocol are also related to the methodology of aerobic capacity testing. The influence of prior maximal anaerobic 30 s cycling on relationship of separate cardiorespiratory parameters and blood lactate from work rate was different. Only dynamics or values of $[La]$, *HR* and $P_{ET}CO_2$ were affected under these conditions. The relationship between $[La]$ and work rate was changed and become “U” shaped like in other studies (Davies & Gass, 1981). The character of the relationship of $\dot{V}CO_2$, $\dot{V}O_2$, \dot{V}_E , $P_{ET}CO_2$ from work rate remained unchanged and may be used for correct determination of anaerobic thresholds or $\dot{V}O_2$ max. No change in ventilatory threshold under conditions of metabolic acidosis has been previously reported (Davies & Gass, 1981). The *HR* data confirmed that after similar prior load this measure significantly increases at

the intensities below or equal to LT (Stasiulis et al., 2001). The neural mechanisms that control HR seem not to be changed at the higher intensities. The HR increase at the lower intensities may be explained by the augmented impact of catecholamine that concentration in blood is usually increased after such anaerobic and heavy exercise (Dimsdale et al., 1984; Vincent et al., 2003, 2004). The peripheral reflexes and increase of body temperature may also be important for such changes.

We demonstrated also, that absolute values of $\dot{V}O_2$ at third min of work periods during intermittent increasing cycling exercise were not changed after prior anaerobic load (30 s Wingate test) except for their increase at rest. The last increase may be explained by influence of increased body temperature (Gregson et al., 2002, 2002a), higher blood catecholamine levels (Vincent et al., 2004), changes in substrate utilization (Krustrup et al., 2004) and lactate metabolism (Roston et al., 1987). On the contrary, $\dot{V}O_2$ and work rate slope was increased during second of two consecutive maximal ramp exercises (Jones & Carter, 2004). This controversy may be explained by higher residual effect of maximal ramp test in above cited study.

The study 2 was designed to investigate effects of muscle fatigue on metabolic costs of ergometer cycling. A protocol of repetitive drop jumps was employed to induce muscle fatigue and stimulate recruitment of motor units during the subsequent ergometer cycling exercise. It has been previously demonstrated, that after the protocol of drop jumps applied in the present study, muscle force generating capacity decreases by ~30% as measured 20 min after the exercise and still shows ~15% deficit 24 hours after the exercise (Skurvydas et al., 2000, Streckis et al., 2005). In addition, there is also evidence of a significant increase in plasma creatine kinase (CK) activity 24 hours after the exercise (Murfet et al., 2003). These are typical signs of muscle fatigue and damage after exercise (Lieber & Frieden, 1999; Clarkson & Hubal, 2002). Other authors employing similar protocols of exercise also reported muscle fatigue and damage (Strojnik & Komi, 2000). So, the results of this study showed that $\dot{V}O_2$ increased to higher plateau levels when ergometer cycling was repeated in the fatigue condition.

An increase in O_2 costs of cycling at 50% $\dot{V}O_2$ max has been observed after days of exhaustive combat exercise and 65 km running (Bahr et al., 1991; Millet et al., 2000, respectively). In our study, however, volunteers performed a drop jump exercise of rather limited duration. Our calculations show that changes in heart rate and pulmonary ventilation could account for only up to 10% of the increase in $\dot{V}O_2$ and the largest increase in $\dot{V}O_2$ must have occurred in skeletal muscles. This is agreement with direct measurements of oxygen uptake in knee extensor muscles that show a significant increase in oxygen uptake during fatiguing exercise at high intensity (Poole et al., 1991). Repetitive stretching of

contracting muscles might be of importance in our study. An exercise with repetitive cycles of stretch and shortening induces muscle fatigue and damage (Lieber et al., 1996; Lieber and Frieden, 1999; Strojnik et al., 2000).

After the drop jump exercise, recruitment of type II fibres is expected to intensify at lower intensities of ergometer cycling compared to the exercise performed in the control condition (Ratkevicius et al., 1995; 1998a). Thus increased involvement of type II could lead to an increase in average metabolic costs of work (Deley et al., 2006). Muscle damage per se might also contribute to metabolic costs of exercise. It could be hypothesized that damaged muscle fibres act as an additional mechanical loading on the contracting fibres (Lieber et al., 1996; Sandercock, 2000). Interestingly, the prior drop jump exercise did not increase the amplitude of the slow component of $\dot{V}O_2$. If anything, it became smaller. This suggests that recruitment of type II fibres is not always linked to appearance of the slow component of $\dot{V}O_2$ kinetics during exercise (Garland et al., 2006). This agrees well with the findings of Scheuerman et al. (2001) and Gonzales & Scheuermann (2006) who did not observe any link between changes in *EMG*, an indicator of motor unit recruitment, and $\dot{V}O_2$ during the continuous high intensity cycling. In summary, results of the study support the hypothesis that muscle fatigue and possibly damage induces an increase in metabolic costs of ergometer cycling, but fail to link these impairments with the changes of fast or slow component of $\dot{V}O_2$ kinetics.

CONCLUSIONS

1. The heart rate (HR) values at the end of 3 min work and 4 min recovery periods of intermittent increasing cycling exercise are increased after prior 30 min long aerobic exercise of both moderate (below LT) and heavy (above LT) intensity. The relative rate and magnitude of HR changes both during on- and off transitions of exercise does not change as well.
2. Muscle fatigue and injury after prior excentric-concentric exercise increase oxygen costs of cycling exercise, i.e. decrease efficiency of aerobic work, but does not affect fast or slow component of pulmonary oxygen uptake kinetics.
3. Prior anaerobic load causes metabolic acidosis and has significant influence on magnitude and alteration pattern of blood lactate concentration, also on values of HR , peak end tidal CO_2 pressure and ventilation CO_2 equivalent during work periods of intermittent cycling exercise. The values of peak end tidal CO_2 pressure are significantly decreased at all intensities in comparison with control values. The dynamics and values of oxygen uptake, CO_2 output and pulmonary ventilation do not significantly change under conditions of metabolic acidosis.
4. Prior anaerobic load increases significantly the values of HR at the end of work and recovery periods of intermittent increasing cycling exercise, decreases the amplitude of its changes, but has no effect on the rate of HR kinetics. After such prior load the rate of $\dot{V}O_2$ kinetics during off-transition and the rate of $\dot{V}CO_2$ kinetics during on-transition off-transition of exercise are decreased significantly when the work intensity is below lactate threshold.
5. Prior incremental cycling exercise has no effect on the rate of $\dot{V}O_2$ kinetics at the start of both moderate and heavy exercises, but decreases the slow component of this kinetics during heavy exercise. The absolute values of $\dot{V}O_2$ are increased at steady state during both moderate and heavy exercises under conditions of metabolic acidosis.
6. The relative rates of changes (time constants) of the HR and $\dot{V}O_2$ are influenced neither by prior brief anaerobic nor by long and heavy aerobic exercise and nor by prior excentric-concentric exercise. This is true for both on- and off-transition periods of exercise and does not depend on its intensity in the region near the LT . On the contrary, absolute steady state $\dot{V}O_2$ and HR values are increased, e.g. aerobic work efficiency is decreased both after prior heavy aerobic and after prior excentric-concentric exercise. The slow component of $\dot{V}O_2$ response seems to be affected (decreased) only by prior heavy aerobic exercise.

SANTRAUKA

Fizinio krūvio metu žmogaus organizmas retai būna pastovaus metabolizmo sąlygose. Griaučių raumenų darbo pradžia, kurios metu vyksta organizmo įsidirbimas, ir darbo pabaiga, po kurios prasideda atsigavimas, taip pat intensyvumo kaita dirbant, yra susiję su poreikiu pakeisti metabolizmo intensyvumą. Nustatyta, kad deguonies suvartojimo ($\dot{V}O_2$) bei su juo susijusių rodiklių ($\dot{S}SD$, $\dot{V}CO_2$ ir \dot{V}_E) kaita įsidirbimo ir atsigavimo metu atspindi žmogaus aerobinį pajėgumą bei jo greitosios adaptacijos fiziniam krūviui galimybes (Whipp & Ozyener, 1998; Jones & Carter, 2000). Greitis, kuriuo aerobinė *ATP* resintezė griaučių raumenyse prisitaiko prie naujo energijos poreikio, veikia tokias greitosios adaptacijos fiziniam krūviui savybes kaip įsidirbimas, nuovargis ir atsigavimas. Greitesnė aerobinės oksidacijos adaptacija sumažina ląstelės bei organų homeostazės pokyčius darbo pradžioje ar pasikeitus intensyvumui (Grassi, 2001).

Žinoma, kad $\dot{V}O_2$ krūvio pradžioje didėja eksponentiškai (Astrand & Saltin, 1961; Casaburi et al., 1987, 1989; Poole et al., 1991; Barstow & Mole 1991; Barstow, 1994; Poole, 1994; Whipp, 1994; Gaesser & Poole, 1996; Barstow et al., 2000; Ozyener et al., 2001). Ši kaita priklausomai nuo darbo intensyvumo gali būti dviejų (kai intensyvumas mažesnis už *LaS*) (Henry & DeMoor, 1956; Whipp, 1970; Cerretelli & Di Prampero, 1987) arba trijų (kai intensyvumas didesnis už *LaS*) fazių (Whipp & Wasserman, 1972; Linnarsson, 1974; Hughson & Morrissey, 1982; Paterson & Whipp, 1991; Barstow & Mole, 1991). Nėra aišku, ar $\dot{V}O_2$ kaitos greitis labiau priklauso nuo O_2 tiekimo raumenims, ar nuo O_2 suvartojimo greičio mitochondrijose (Tschakovsky & Hughson, 1999). Siekiant geriau suprasti santykinį minėtų veiksnių vaidmenį, dažnai taikomas toks eksperimentinis modelis, kai po vieno krūvio (prieškrūvio) atliekamas kitas krūvis. Sukaupia daug duomenų apie tai, kaip skirtingo intensyvumo ar pobūdžio prieškrūvis veikia įvairių vegetacinių sistemų rodiklių kaitą įsidirbimo, pastoviosios būklės ir atsigavimo metu atliekant atskirą krūvį (Germino et al., 1996; MacDonald et al., 1997; Koppo & Bouckaert, 2000-2002; Scheuermann et al., 2001; Fukuba et al., 2002, 2007; Burnley et al., 2000-2002, 2006; Koppo et al., 2003; Tordi et al., 2003; Endo et al., 2004; Moysi et al., 2005; Gurd et al., 2006; Jones et al., 2006; Marles et al., 2006, 2007). Nustatyta, kad po sunkaus (kai intensyvumas – virš *LaS*) prieškrūvio kraujyje susikaupę metabolitai (H^+ , K^+ , laktatas) padidina raumenų kraujotaką sekančio krūvio pradžioje. Nepaisant to, jauniems sveikiems suaugusiems asmenims atliekant vidutinio intensyvumo darbą, prieš tai atliktas sunkus prieškrūvis neturi poveikio $\dot{V}O_2$ kaitos laiko konstantai ir dydžiui (Germino et al., 1996; MacDonald et al., 1997; Burnley et

al., 2002). Kita vertus, tiriamiesiems po sunkaus prieškrūvio atliekant didelio intensyvumo darbą, nustatyta, kad $\dot{V}O_2$ kaita pagreitėja (Gerbino et al., 1996; MacDonald et al., 1997; Koppo & Bouckaert, 2001; Bearden & Moffatt, 2001; Scheuermann et al., 2001, 2002; Burnley et al., 2002, 2006; Tordi et al., 2003; Jones et al., 2006; Fukuba et al., 2007). Taigi, nėra galutinai aišku, kaip $\dot{V}O_2$ ir kitų vegetacinių sistemų funkcijos rodiklių kaitą veikia prieškrūvio (ar pramankštos) intensyvumas (Gerbino et al., 1996; MacDonald et al., 1997; Fukuba et al., 1998; Burnley et al., 2000; Koppo & Bouckaert, 2000). Nėra žinoma įvairaus pobūdžio prieškrūvio įtaka minėtų rodiklių kaitai įsidirbimo, pastoviosios būklės ir atsigavimo metu (Gaesser & Poole, 1996; Whipp, 1996; Fukuba & Whipp, 1999; Tordi et al., 2003; Fukuba et al., 2007). $\dot{V}O_2$, \dot{SSD} , $\dot{V}CO_2$ ir \dot{V}_E kaita dar nėra tyrinėta atliekant kartotinį nuosekliai didinamą fizinį krūvį.

Žinoma, kad $\dot{V}O_2$ ir atliekamo darbo santykis esant pastoviajai būklei (darbo efektyvumas) atspindi žmogaus aerobinį pajėgumą ir greitos adaptacijos fiziniam krūviui galimybes (Whipp et al., 2002; Jones & Carter, 2000). Nepaisant intensyvių tyrimų, veiksniai, lemiantys veloergometrinio krūvio efektyvumą, nėra aiškūs. J. U. Gonzales ir B. W. Scheuermann (2006) nustatė, kad po didelio intensyvumo aerobinio prieškrūvio padidėja $\dot{V}O_2$, t. y. sumažėja mechaninio darbo veloergometru efektyvumas, nors *EMG* nepakinta. Žinoma, kad kojų masė ir mynimo dažnumas daro poveikį $\dot{V}O_2$ minant veloergometrą (Neder et al., 2000, Martin & Spiriduso, 2001, McDaniel et al., 2002; Migita & Hirakoba, 2006). Kai darbo intensyvumas viršija *LaS*, po greitos eksponentiškos kaitos per pirmas tris minutes $\dot{V}O_2$ ir vėliau iš lėto didėja (Whipp 1994). Iškelta hipotezė, kad šis vadinamasis lėtasis $\dot{V}O_2$ kaitos komponentas, dėl kurio darbo metabolinės sąnaudos didėja (mažėja efektyvumas), yra susijęs su II tipo raumenų skaidulų rekrutavimu (Saunders et al., 2000; Bernasconi et al., 2006). Nustatyta, kad po varginančių ekscentrinių pratimų, sukeliančių raumenų nuovargį ir pažeidą, sumažėja koncentrinių susitraukimų efektyvumas (Horita et al., 2003; Moysi et al., 2005). Neaišku, kaip tokie pratimai gali paveikti po jų atliekamo veloergometrinio krūvio efektyvumą. Pastebėta, kad tokiomis sąlygomis padidėja \dot{V}_E , kvėpavimo dažnumas, kraujo laktato koncentracija, \dot{SSD} , subjektyviai suvokiamų pastangų dydis, nors $\dot{V}O_2$ išlieka nepakitęs (Gleeson et al., 1995; 1998). Ekscentrinis prieškrūvis nežymiai veikia mynimo efektyvumą (Moysi et al., 2005). Kita vertus, pelių raumenų tyrimai parodė, kad efektyvumas sumažėja, kai ekscentriniai pratimai atliekami po koncentrinių susitraukimų (Warren et al., 1996). Pademonstruota, kad $\dot{V}O_2$ padidėja, kai veloergometrinis krūvis atliekamas prieš tai sumažinus I tipo raumenų skaidulų glikogeno atsargas (Osborne & Schneider, 2006), o taip pat kartojant keletą izometrinių pratimų

(Vollestad et al., 1990; Krustup et al., 2004). Yra duomenų, kad nuovargio metu intensyviai rekrutuojamos mažiau efektyvios II tipo raumenų skaidulos (Krustup et al., 2004; Nakagawa et al., 2005; Deley et al., 2006). Taigi, duomenys apie veiksnius darančius įtaką veloergometro mynimo efektyvumui nuovargio metu yra prieštaringi. Be to, neradome darbų, kur būtų tiriamas ekscentrinų pratimų (prieškrūvio) poveikis skirtingoms $\dot{V}O_2$ kaitos fazėms. Žinoma, kad daugkartiniai nušokimai su po to sekančiais vertikaliais šuoliais sukelia ilgalaikį raumenų nuovargį su raumenų pažeidos požymiais (Skurvydas et al., 2000, Murfet et al., 2003). Galima tikėtis, kad tai turėtų padidinti veloergometro mynimo metaboles sąnaudas dėl intensyvesnio II tipo raumenų skaidulų rekrutavimo (Nakagawa et al., 2005; Mígita & Hirakoba, 2006). Manoma, kad progresuojantis II tipo raumenų skaidulų rekrutavimas gali būti susijęs su $\dot{V}O_2$ kaitos lėtojo komponento pasireiškimu netgi esant 50% nuo $\dot{V}O_2$ max intensyvumui (Krustup et al., 2004).

Vegetacinių sistemų veiklos atsakas į nuosekliai didinamo krūvio testus yra svarbus įvertinant sergančių, nesportuojančių ir sportininkų funkcinių pajėgumą bei sudarant treniruotės programas (Whipp et al., 1981; Meyer et al., 1998; Wasserman et al., 1999). Paprastai tokie testai, kurių metu krūvis nuosekliai didinamas, kol per 8-14 min tiriamasis pasiekia visišką nuovargį, atliekami siekiant nustatyti $\dot{V}O_2$ max ir AAS (Whipp et al., 1981; Wasserman et al., 1999). Dažniausiai tokių testų metu registruojami ir analizuojami tokie kvėpavimo funkcijos ir dujų apykaitos rodikliai kaip \dot{V}_E , $\dot{V}O_2$, $\dot{V}CO_2$, taip pat $\dot{S}SD$ ir $[La]$. Mūsų žiniomis, atskirų rodiklių kaita yra tirta nepertraukiamo nuosekliai didinamo krūvio metu, po prieš tai atlikto vienos minutės maksimalaus anaerobinio intensyvumo krūvio (Schneider & Berwick, 1998; Marles et al., 2006) arba po nepertraukiamo nuosekliai didinamo krūvio iki visiško nuovargio testo (Jones & Carter, 2004). Neradome duomenų apie tai, koks prieš tai atlikto prieškrūvio poveikis minėtų rodiklių priklausomumui nuo darbo intensyvumo atliekant kartotinę nuosekliai didinamo krūvio testą.

Tyrimo hipotezė. Įvairaus prieškrūvio sukelti kraujo laktato koncentracijos, raumenų kraujotakos ir metabolizmo pokyčiai, metabolinis ar nemetabolinis nuovargis, turėtų daryti įtaką atskirų vegetacinių sistemų rodiklių kaitai įsidirbimo ar atsigavimo metu. Šis poveikis turėtų būti nevienodas skirtingose šių rodiklių kaitos fazėse ir turėtų priklausyti nuo atliekamo darbo intensyvumo.

Tyrimo originalumas. Pirmą kartą nustatyta, kad po ekscentrinio-koncentrinio prieškrūvio, (100 kas 20 s atliekamų nušokimų nuo pakylės su vertikaliais šuoliais) padidėja absoliučios deguonies suvartojimo reikšmės pastoviosios būklės metu, t. y. sumažėja vidutinio ir didelio intensyvumo aerobinio veloergometrinio darbo efektyvumas, tačiau toks prieškrūvis neturi poveikio deguonies ir kitų vegetacinių sistemų funkcijos rodiklių kaitos greičiui darbo

pradžioje, bei nepakeičia $\dot{V}O_2$ kaitos lėto komponento. Nustatyta, kad kaip prieškrūvis atliktas nuosekliai didinamo krūvio testas padidina absoliučias $\dot{V}O_2$ reikšmes ir vidutinio, ir didelio intensyvumo krūvio metu, nedaro įtakos taip vadinamo greito, bet sumažina lėtą $\dot{V}O_2$ kaitos komponentą. Parodyta, kad 30 s maksimalių pastangų anaerobinis prieškrūvis nepakeičia $\dot{V}O_2$ ir \dot{V}_E priklausomumo nuo kartotinio nuosekliai didinamo krūvio intensyvumo.

Teorinė ir praktinė tyrimo reikšmė. Tyrimo rezultatai yra svarbūs sporto ir pratimų fiziologijai, kadangi papildo žinias apie vegetacinių sistemų funkcijos rodiklių kaitą greitosios adaptacijos fiziniams krūviams metu, parodo metabolinio ir nemetabolinio nuovargio įtaką aerobinio darbo efektyvumui. Tyrimo rezultatai parodo įvairaus intensyvumo ir pobūdžio pramankštos įtaką vėlesnio aerobinio darbo efektyvumui. Tyrimo rezultatai leidžia suprasti, kaip optimizuoti aerobinio pajėgumo testavimo taikant kartotinio nuosekliai didinamo krūvio testus protokolą.

Išvados

1. Ir lengvesnis, ir sunkesnis už laktato slenkstį (atitinkamai vidutinio ir didelio intensyvumo) 30 min trunkantis aerobinis prieškrūvis daro poveikį absoliučioms $\dot{V}O_2$ reikšmėms kartotinio, iš 3 min darbo ir 4 min poilsio susidedančio, nuosekliai didinamo krūvio testo metu. Viso testo metu nepastebima nei lengvesnio, nei sunkesnio aerobinio prieškrūvio poveikio $\dot{V}O_2$ kaitos santykiniam greičiui ir pokyčio dydžiui.
2. Atliekant kartotinį, iš 6 min darbo ir 4 min poilsio susidedantį, nuosekliai didinamo krūvio testą veloergometru po ekscentrinio-koncentrinio prieškrūvio (100 nušokimų-šuolių), padidėja absoliučios $\dot{V}O_2$ reikšmės pastoviosios būklės metu, t. y. sumažėja aerobinio darbo efektyvumas. Raumenų nuovargis ir pažeida po tokio prieškrūvio neturi poveikio $\dot{V}O_2$ kaitai krūvio pradžioje ir lėtam $\dot{V}O_2$ kaitos komponentui krūvio metu.
3. Po anaerobinio prieškrūvio sukeltos metabolinės acidozės atliekant kartotinį, iš 3 min darbo ir 3 min poilsio susidedantį, nuosekliai didinamo krūvio testą, skirtingų vegetacinių sistemų funkcijos rodiklių priklausomumas nuo darbo intensyvumo pakinta nevienodai. Anaerobinis prieškrūvis reikšmingai pakeičia laktato koncentraciją ir jos kaitos pobūdį, $\dot{V}O_2$, CO_2 slėgio iškvėpimo pabaigoje ir ventiliacinio CO_2 ekvivalento dydį. Kitų tirtų rodiklių ($\dot{V}O_2$, $\dot{V}CO_2$, \dot{V}_E , RER , O_2 slėgio iškvėpimo pabaigoje ir ventiliacinio O_2 ekvivalento) absoliučios reikšmės ir priklausomumas nuo darbo intensyvumo po anaerobinio prieškrūvio išlieka panašūs kaip ir jo neatlikus.

4. Anaerobinis prieškrūvis reikšmingai padidina tik $\dot{S}SD$ reikšmes įsidirbimo ir atsigavimo pabaigoje, sumažina šio rodiklio pokyčio amplitudę, bet neturi poveikio jo kaitos greičiui. Anaerobinis prieškrūvis sumažina $\dot{V}O_2$ kaitos atsigavimo metu ir $\dot{V}CO_2$ kaitos įsidirbimo metu greitį, kai darbo intensyvumas mažesnis už LaS ($p < 0,05$).
5. Mišrus aerobinis-anaerobinis prieškrūvis neturi poveikio $\dot{V}O_2$ kaitos greičiui vidutinio ir didelio intensyvumo darbo pradžioje, bet sumažina lėtą šio rodiklio kaitos komponentą didelio intensyvumo darbe. Dėl prieškrūvio sukeltos metabolinės acidozės padidėja absoliutus $\dot{V}O_2$ ir vidutinio, ir didelio intensyvumo darbo metu.
6. $\dot{S}SD$ ir $\dot{V}O_2$ kaitos greitis įsidirbimo metu atliekant ir lengvesnius už LaS (vidutinio intensyvumo), ir sunkesnius už LaS (didelio intensyvumo) krūvius nepakinta nei po trumpo anaerobinio, nei po sunkaus aerobinio, nei po ekscentrinio-koncentrinio prieškrūvio. Labiausiai po prieškrūvio pakinta absoliučios $\dot{V}O_2$ reikšmės pastoviosios būklės metu, kurios padidėja po sunkaus aerobinio bei po ekscentrinio-koncentrinio prieškrūvių. Tai rodo aerobinio darbo efektyvumo sumažėjimą po minėtų prieškrūvių.

PUBLICATIONS

Scientific publications in continuous transactions and periodicals entered in a special list confirmed by the Lithuanian Science Council

Scientific publications on the topic of the dissertation:

1. Stasiulis, A., **Dubininkaitė, L.**, Aleksandravičienė, R. (2001). Anaerobinio prieškrūvio poveikis širdies susitraukimų dažnumo kaitai atsigauant po intensyvaus darbo (Influence of a preceding anaerobic load on the heart rate kinetics during recovery after exercise of different intensity). *Lietuvos bendrosios praktikos gydytojas*, 5 (5), 443–446.

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2. **Dubininkaitė, L.** Influence of prior aerobic load on the heart rate kinetics during intermittent increasing exercises. 8th Annual Congress of the European College of Sport Science Proceedings. Salzburg, Austria, July 9–12, 2003.

3. Stasiulis, A., **Dubininkaitė, L.**, Ratkevičius, A., Skurvydas, A. Effect of prior drop jumps on the VO₂ kinetics during exercise near or above lactate threshold. 8th Annual Congress of the European College of Sport Science Proceedings. Salzburg, Austria, July 9–12, 2003.

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