# KINETICS OF PULMONARY VENTILATION AND CARBON DIOXIDE OUTPUT DURING INTERMITTENT INCREASING CYCLING EXERCISE AFTER A PRIOR ANAEROBIC LOAD

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## ABSTRACT

*Background.* Research aim was to establish the influence of a prior anaerobic load on the kinetics of ventilation ($V_E$) and carbon dioxide output ($VCO_2$) during on- and off-transition phases of intermittent increasing cycling exercise.

*Methods.* The seven healthy, physically active females volunteered to estimate the influence of a prior anaerobic load on the kinetics of respiratory parameters. During the first visit $VO_2$max was evaluated using the increasing cycling exercise test. During the second testing the participants performed intermittent cycling exercise (ICE). During the other visit they performed supramaximal 30 s anaerobic exercise and after 15 min of the rest – ICE. In order to estimate the kinetics of respiratory parameters were analysed by adopting mono-exponential function.

*Results*. Mean blood lactate concentration was increased during ICE performed after prior anaerobic load in the presence of a residual metabolic acidosis. The asymptote and amplitude of monoexponential function reflecting $VCO_2$ kinetics during on- and off-transitions were not changed after prior anaerobic load. The time constant of this function was significantly longer ($p < .01$) both during on- and off-transitions at work rate below lactate threshold (LT), whereas this parameter at higher work intensities remained unchanged. The parameters of $V_E$ monoexponential function during on- and off-transitions were not changed after prior anaerobic load.

*Conclusion.* Despite similar physiological mechanism responsible for $V_E$ and $CO_2$ regulation during exercise the prior anaerobic load had different influence on the ventilation and $VCO_2$ kinetics during exercise below lactate threshold causing slowing of $VCO_2$ without changes of $V_E$ kinetics.

**Keywords:** exercise intensity, lactate threshold, pulmonary ventilation, $CO_2$ output, acidosis.

## INTRODUCTION

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 ($VO_2$) and other related parameters (heart rate (HR), carbon dioxide output ($VCO_2$), pulmonary ventilation ($V_E$)) during transitional phases between rest and exercise are associated with humans aerobic capacity, reflects its acute adaptation abilities (Whipp & Ozyener, 1998; Jones & Carter, 2000). It is known that $V_E$ kinetics at the start of moderate intensity exercise is characterised by two-phase response: after initial fast increase this parameter slowly approaches steady state at 3–4 min of exercise. During recovery $V_E$ also demonstrates fast decrease followed by slow decreasing to the rest level (Linnarsson, 1974; Whipp & Mahler, 1980). During exercise of heavy intensity $V_E$ continues slowly increasing during the whole exercise (Wasserman, Whipp, Casaburi, & Oren, 1980; Martin, Whipp, Casaburi, & Oren, 1981).

$VCO_2$ changes as monoexponential function at the onset of moderate intensity (Di Prampero, 1981; Hughson & Morrissey, 1982). Similarly to

$VO_2$, $VCO_2$ also demonstrates so called component "cardiodynamic" associated with fast changes of pulmonary circulation (Whipp & Mahler, 1980).

The kinetics of $VCO_2$ and $V_E$ is strongly interrelated and is regulated by similar physiological mechanisms (Jones & Heigenhauser, 1996). Whipp (1994) showed that $V_E$ is governed by the signals related to increased plasma $K^+$ concentration, $PCO_2$, pH ir $PO_2$ because the $V_E$ kinetics is strongly associated with that of $VCO_2$ and $V_E$ changes slower than $VO_2$. Pulmonary $CO_2$ output is additionally increased when the exercise intensity is associated with metabolic (lactic acid) acidosis. It has been found that an acute, endogenous metabolic acidaemia speeds $V_E$ kinetics in moderate exercise (Ward & Whipp, 2010). On the other hand, despite the numerous research data exact mechanisms about how ventilation is regulated during exercise, what the link between $CO_2$ kinetics and ventilation is, remain disputable (Forster, Haouzi, & Dempsey, 2012). More research is needed for investigating relationships between alterations in acid-base state and the ventilatory responses (Lindinger & Heigenhauser, 2012).

To get more insight in the relative role of mechanisms regulating different pulmonary and oxygen uptake parameters the experimental model where one exercise boot is preceded by the 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 $VO_2$ parameters during on-transition, steady state and off-transition phases of constant load exercise (Gerbino, Ward, & Whipp, 1996; MacDonald, Pedersen, & Hughson, 1997; Koppo & Bouckaert, 2000, 2001, 2002; Scheuermann, Hoelting, Noble, & Barstow, 2001; Fukuba, Hayashi, Koga, & Yoshida, 2002; Fukuba et al., 2007; Burnley, Jones, Carter, & Doust, 2000; Burnley, Doust, Carter, & Jones, 2001; Burnley, Doust, Ball, & Jones, 2002; Burnley et al., 2006; Koppo et al., 2003; Tordi, Perrey, Harvey, & Hughson, 2003; Endo et al., 2004; Moysi, Garcia-Romero, Alvero-Cruz, & Vicente-Rodriguez, 2005; Gurd et al., 2006; Jones, Berger, Wilkerson, & Roberts, 2006; Marles, Mucci, & Legrand, 2006; Marles, Perrey, Legrand, & Blondel, 2007).

**Research aim** was to establish the influence of a prior anaerobic load on the kinetics of $V_E$ and $VCO_2$ during on- and off-transition phases of intermittent increasing cycling exercise.

# METHODS

**Participants.** Seven healthy, non-smoking, physically active females 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**

| | |
|---|---|
| Sample size (n) | 7 |
| Age (years) | 22.1 (1.5) |
| Height (m) | 1.69 (0.66) |
| Weight (kg) | 56.7 (4.9) |
| $VO_2$max ($l \cdot min^{-1}$) | 2.3 (0.3) |
| $VO_2$max ($ml \cdot min^{-1} \cdot kg^{-1}$) | 41.5 (5.2) |

**Note.** Values are presented as means (SD). $VO_2$max – maximal oxygen uptake.

**Pulmonary gas exchange data collection.** Pulmonary gas exchange data ($VCO_2$ and $V_E$) were collected continuously using the automated breath-by-breath systems "Oxycon Mobile" (Jaeger, Germany). Prior to each exercise test, the gas analyser was calibrated with certificated calibration gas. The all recorded data was analysed by five seconds mean intervals using "LAB Manager" and "Microsoft Excel" programs.

**Measurement of blood lactate concentration.** The fingertip blood samples were collected into a capillary tube and subsequently analysed for blood lactate concentration as described previously (Kulis, Laurinavichyus, Firantas, & Kurtinaitienė, 1988). Lactate concentration in the blood was established by means of Eksan-G analyser using a membrane with enzyme lactaoxidase. Prior to each testing the analyser was calibrated by the standard 5 mM lactate solutions supplied by manufacturers.

**Continuous increasing cycling exercise (CCE).** 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 10 W. The pedalling rate was 50 rpm. The test was terminated when the subject was not able to keep the required pedalling rate. Throughout the CCE pulmonary $VO_2$ was measured breath-by-breath using the automated system "Oxycon Mobile" (Jaeger, Germany). The $VO_2$max 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 ICE was performed on the mechanically braked cycle ergometer (Monark 834E, Monark-Crescent AB, Sweden). The tests consisted of repeated work 3 min and passive rest 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 W 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 analysed for blood lactate concentration. The test was continued until the participants' blood lactate concentration raised over 4 mM level.

**The Wingate anaerobic test (MAL).** As a prior anaerobic load the supramaximal 30 s Wingate test was performed on Monark 834E cycle ergometer. 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.

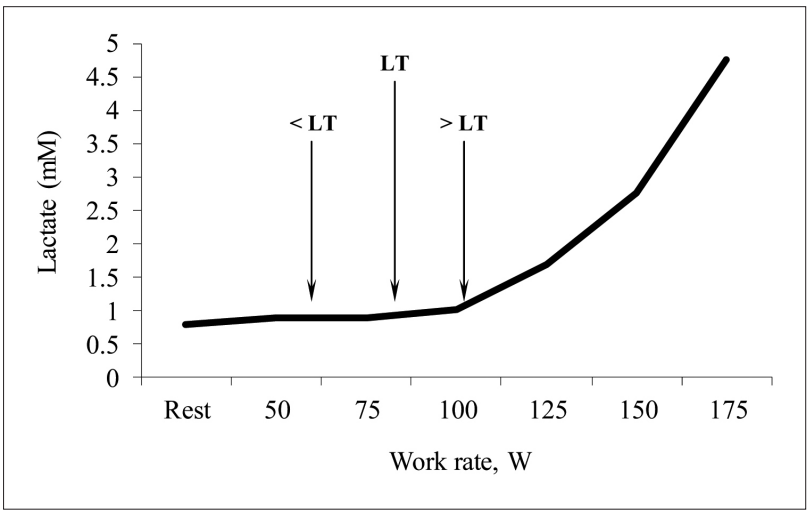
**The lactate threshold (LT) and relative work intensity estimation.** The LT was determined from visual inspection of individual plots of blood lactate concentration vs. work rate (Figure 1). The LT was considered as the work rate from [La] under rest level start raising slowly over 1–1.5 mM. The following relative intensities were chosen for the subsequent analysis: 25 W below lactate threshold (< LT); lactate threshold (LT); 25 W above lactate threshold (> LT) (Figure 1).

**The analyses of respiratory parameters kinetics.** In order to estimate the kinetics of respiratory parameters ($VCO_2$ and $V_E$) during on-transition and recovery periods were analysed by adopting mono-exponential function: $y(t) = y(b) \pm A \times (1 - e^{-t/\tau})$ where y (b) is the baseline value ($VCO_2$ or $V_E$) through the last 30 s of work or rest; A is the amplitude and $\tau$ is the time constant of the response. During on-transition the first 20 s were always removed from the analysis (Whipp, Ward, & Lamarra, 1982).

**Experimental protocol.** In order to estimate the influence of a prior anaerobic load on the kinetics of respiratory parameters ($VCO_2$ and $V_E$) each participant was tested three times on separated days. During the first visit $VO_2$max was evaluated using the CCE test (10 W·min$^{-1}$). During the second testing the participants performed ICE protocol. During the other visit they performed supramaximal 30 s anaerobic exercise (MAL) and after 15 min of the rest – ICE protocol.

**Statistical analysis.** All the values are reported as the means and standard deviations

Figure 1. **Lactate threshold (LT) and relative work intensity estimation**




**Note.** < LT represents 25 W below lactate threshold; > LT – 25 W above lactate threshold.

(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 $p < .05$.

## RESULTS

To compare the parameters between testing conditions (without and after prior anaerobic load) data were normalized to each individual's lactate threshold (LT). Figure 2 shows mean blood lactate concentration on different testing conditions indicating the presence of a residual metabolic acidosis during ICE performed after prior anaerobic load.

The asymptote, amplitude and time constant of $VCO_2$ and $V_E$ kinetics during on- and off-transitions are given in Tables 2–3.

The asymptote and amplitude of mono-exponential function reflecting $VCO_2$ kinetics during on- and off-transitions were not changed after prior anaerobic load. The time constant of this function was significantly longer ($p < .01$) both during on- and off-transitions 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 $VCO_2$ kinetics increased significantly ($p < .001$) with intensity under both testing conditions (Table 2).

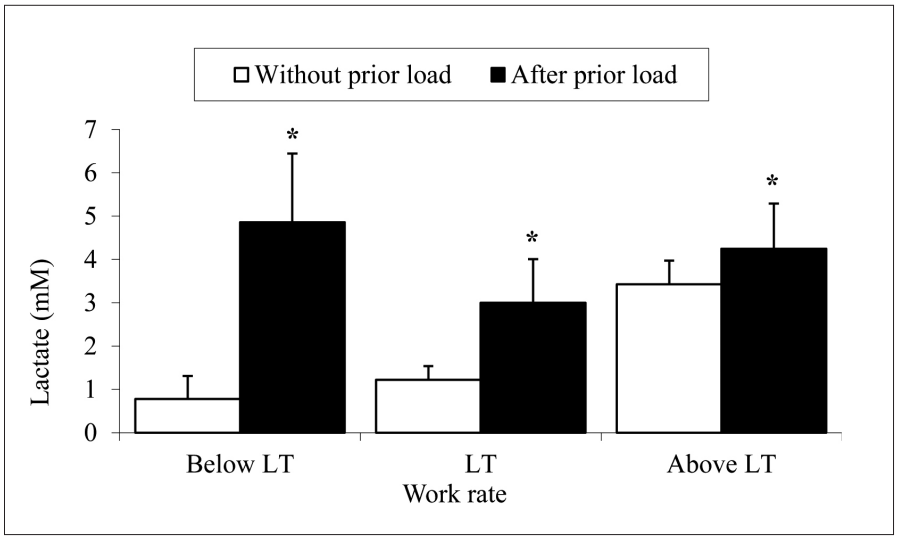



Figure 2. **Mean blood lactate concentration [La] during intermittent increasing cycling exercise on different testing conditions**

**Note.** The standard deviations presented by the bars. * – Denote significant ($p < .05$) difference between testing conditions. LT is work rate at lactate threshold.

Table 2. **Parameters of $VCO_2$ 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 prior anaerobic load | After prior anaerobic load | Without prior anaerobic load | After prior anaerobic load |
| Asymptote ($ml \cdot min^{-1}$) | < LT | 850.5 (263.4) | 787.6 (224.2) | 244.6 (67.8) | 249.9 (43.9) |
| | LT | 1308.7 (290.1) | 1246.1 (267.5) | 302.3 (84.0) | 306.5 (63.9) |
| | > LT | 1827.3 (294.7) | 1763.9 (321.9) | 413.7 (57.8) | 435.5 (86.4) |
| Amplitude, ($ml \cdot min^{-1}$) | < LT | 640.4 (240.9) | 505.5 (207.7) | 607.3 (214.8) | 536.1 (201.1) |
| | LT | 1034.9 (236.2) | 1012.4 (247.4) | 1011.1 (249.2) | 915.1 (232.9) |
| | > LT | 1492.9 (257.5) | 1403.0 (277.8) | 1405.6 (273.0) | 1322.9 (281.9) |
| Time constant (s) | < LT | 29.0 (4.4) | 39.8 (6.1) * | 42.0 (4.7) | 50.1 (5.9) * |
| | LT | 36.4 (4.4) | 37.2 (4.5) | 58.4 (5.7) | 54.9 (10.5) |
| | > LT | 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 ($p < .05$) 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.

Table 3. **Parameters of $V_E$ 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 prior anaerobic load | After prior anaerobic load | Without prior anaerobic load | After prior anaerobic load |
| Asymptote ($ml \cdot min^{-1}$) | **< LT** | **26.1 (6.4)** | **28.5 (4.8)** | **10.6 (2.2)** | **12.5 (2.2)** |
| | LT | 37.1 (6.7) | 38.7 (6.4) | 12.9 (2.9) | 14.1 (1.9) |
| | > LT | 51.3 (8.2) | 52.7 (6.8) | 17.7 (2.3) | 19.3 (3.7) |
| Amplitude, ($ml \cdot min^{-1}$) | < LT | 16.6 (5.7) | 14.4 (4.9) | 15.6 (4.9) | 16.0 (5.1) |
| | LT | 25.7 (5.7) | 27.2 (6.8) | 24.2 (6.1) | 24.3 (5.0) |
| | > LT | 37.0 (8.4) | 36.5 (6.4) | 33.4 (8.6) | 33.3 (6.9) |
| Time constant (s) | < LT | 30.2 (5.8) | 35.9 (8.5) | 46.1 (4.1) | 53.9 (9.6) |
| | LT | 36.0 (3.7) | 38.9 (6.1) | 65.1 (6.9) | 63.2 (13.0) |
| | > LT | 42.1 (2.8) | 44.6 (5.5) | 69.1 (8.3) | 64.7 (7.4) |

**Note.** The standard deviations (SD) are presented in brackets. * – Denote significant ($p < .05$) 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.

The parameters of $V_E$ 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 $V_E$ kinetics increased significantly ($p < 0.001$) with intensity under both testing conditions (Table 3).

## DISCUSSION

The $VCO_2$ time constant was significantly longer both during on- and off-transitions at work rate below LT, whereas this parameter at higher work intensities remained unchanged. On the contrary, the parameters of $V_E$ monoexponential function during on- and off-transitions were not changed after prior anaerobic load.

The latter finding contradicts the data presented by Ward and Whipp (2010) that metabolic acidaemia speeds $V_E$ kinetics in moderate exercise, consistent with carotid chemoreception contributing to the *tightness* of arterial pH-$CO_2$ regulation and the magnitude of the transient arterial hypoxaemia.

The increased blood lactate concentration at any intensity of intermittent increasing cycling after prior anaerobic load shows that exercise was performed under conditions of metabolic acidosis. Despite the 15 min of rest which separated prior anaerobic load and following intermittent exercise it was rather high (7.83 (1.36) $mmol \cdot l^{-1}$). Other authors have also shown that remarkable metabolic acidosis is observed in working muscles during intensive exercise which is persistent during following exercise and causes vasodilation and increase of oxygen supply to (Tordi et al., 2003). For instance it was established using very similar to ours‘ protocol that concentration of hydrogen in muscles was increased (Spriet, Lindinger, McKelvie, Heigenhauser, & Jones, 1989), and [La] was 5,6 $mmol \cdot l^{-1}$ (Burnley et al., 2002).

In our study only $VCO_2$ kinetics was slowed at the intensity lower than LT both during on- and of-transitions to exercise. It may be caused by increased rate of $CO_2$ production due to need to neutralize the increased amount of $H^+$ due to acidosis. The preceding load of heavy intensity is known to influence metabolism during subsequent exercise of heavy intensity (Gerbino et al., 1996; Krustrup, Gonzalez-Alonso, Quistorff, & Bangsbo, 2001). The intensified aerobic metabolism during second exercise leads to decreased anaerobic ATP resynthesis (Krustrup et al., 2001). Because of greater metabolic contribution of fatty acids and as consequence decreased respiratory exchange ratio (Green, Houston, Thomson, & Reid, 1979) $VCO_2$ may be lower during the whole duration of second exercise. During exercise $CO_2$ reserves are increased leading to ionic and osmotic changes that have effect on amount of bicarbonates and intracellular $CO_2$ (Jones & Heigenhauser, 1996). The ability of the organism to accumulate $CO_2$ improves ability to adapt to exercise (Cherniack & Longobardo, 1970). At the onset of light exercise $CO_2$ accumulates in muscles and venous. At heavy intensities when the [La] increases and concentration of $K^+$ decreases (Kowalchuk, Heigenhauser, Lindinger, Sutton, & Jones, 1988), the increase of $CO_2$ in muscles is impossible that’s

why the $CO_2$ pressure always remarkably increases. This accelerates $CO_2$ diffusion from the muscles but real $CO_2$ release is dependent on the circulation $CO_2$ dissociation curve (Jones & Heigenhauser, 1996). Differences of $CO_2$ reserves between first and second loads are contributing to changed $VCO_2$ adaptation (Hughson & Inman, 1985).

As has been established by Whipp (1994 a) $V_E$ is governed by signals that depend on increased concentration of $K^+$ ions in plasma as well as impact of $PCO_2$, pH and $PO_2$ to chemoreceptors. It is believed that increase of $V_E$ during exercise is proportional to the rate of metabolism. It remains unclear whether the rate of metabolism is dependent on $VO_2$ or $VCO_2$. When the reserves of the gases (especially $CO_2$) are changed $V_E$ is changing not due to $VCO_2$ in working muscles but because of activated $CO_2$ exchange in lungs. For that reason pH of arterial blood is regulated by $PaCO_2$ when the intensity is moderate and concentration of bicarbonates remains unchanged (Whipp & Ward, 1998). Because kinetics of $V_E$ is associated with that of $VCO_2$ at the onset of exercise $V_E$ dynamics is lower than that of $VO_2$. The rate of $CO_2$ exchange in the lungs is increased when the intensity is associated with metabolic acidosis. In such conditions because of $HCO3^-$ and $H^+$ coupling extra $CO_2$ is produced. During exercise of increasing intensity it causes $VCO_2$ acceleration in comparison with $VO_2$ (Whipp & Ward, 1998). It has been reported that significant relationship between $V_E$ and $PaCO_2$ exists during early phase of recovery (Afroundeh et al., 2013). In our case we observed some uncoupling between $VCO_2$ and $V_E$ at the light intensity after prior anaerobic load. We can speculate that $CO_2$ were too low to cause significant changes $V_E$, despite the kinetics of $VCO_2$ was slowed.

## CONCLUSIONS

After prior anaerobic load the rate of carbon dioxide output kinetics during on- and off-transition of exercise are decreased significantly when the work intensity is below lactate threshold. The kinetics of pulmonary ventilation remains unchanged under such conditions.

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