MUSCLE FATIGUE INCREASES METABOLIC COSTS OF ERGOMETER CYCLING WITHOUT CHANGING VO₂ SLOW COMPONENT

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ABSTRACT
The aim of the present study was to investigate effects of muscle fatigue on oxygen costs of ergometer cycling and slow component of pulmonary oxygen uptake (VO₂) kinetics. Seven young men performed 100 drop jumps (drop height of 40 cm) with 20 s of rest after each jump. After the subsequent hour of rest, they cycled at 70, 105, 140 and 175 W, which corresponded to 29.6 ± 5.4, 39.4 ± 7.0, 50.8 ± 8.4 and 65.8 ± 11.8 % of VO₂peak, respectively, for 6 min at each intensity with 4-min intervals of rest in between the exercise bouts. The VO₂ response to cycling after the exercise (fatigue condition) was compared to ergometer cycling without prior exercise (control condition). From 3rd to 6th min of cycling at 105, 140 and 175 W, VO₂ was higher (p < 0.05-0.01) when cycling in the fatigue compared to the control condition. Slow component of VO₂ kinetics was observed when cycling at 175 W in the control condition (0.17 ± 0.09, l·min⁻¹, mean ± SD), but tended to decrease in the fatigue condition (0.13 ± 0.15 l·min⁻¹). In summary, results of the study are in agreement with the hypothesis that muscle fatigue increases oxygen costs of cycling exercise, but does not affect significantly the slow component of pulmonary oxygen uptake (VO₂) kinetics.

KEY WORDS: Muscle fatigue, energy cost, oxygen uptake, oxygen consumption slow component.

INTRODUCTION
In spite of intensive research, factors determining metabolic costs of cycling remain unclear. It is known that mass of legs and rate of pedalling can affect the pulmonary oxygen uptake (VO₂) which reflects the energy metabolism in aerobic exercise (Martin et al., 2001; McDaniel et al., 2002; Neder et al., 2000). A slow and continuous increase in VO₂ is often observed after 3 min of ergometer cycling at intensities exceeding 60% VO₂max (Whipp, 1994). It has been hypothesized that this slow component of VO₂ kinetics reflects an increase in metabolic costs of cycling associated with recruitment of less efficient type II fibres (Saunders et al., 2000).

Concentric muscle contractions are impaired following exhaustive eccentric exercise which is known to induce muscle fatigue and damage (Horita et al., 2003). It is not clear if metabolic costs of ergometer cycling increase as well. An elevated
minute ventilation, breathing frequency, blood lactate, respiratory exchange ratio, heart rate, and rating of perceived exertion have been reported during ergometer cycling after eccentric exercise (Gleeson et al., 1995; 1998). However, VO₂ at submaximal intensities did not change. It has been also shown that eccentric exercise did not alter or had only a marginal effect on gross cycling efficiency in presence of marked muscle soreness (Moysi et al., 2005). At the same, there is a 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 VO₂ increased during ergometer cycling after exercise induced depletion of glycogen in type I fibres as well as during repetitive fatiguing isometric contractions (Krustedup et al., 2004; Vollestad et al., 1990). There is some evidence that muscle fatigue intensifies recruitment of less efficient type II fibres during exercise (Nakagawa et al., 2005; Krustedup et al., 2004). Overall, however, findings on the metabolic costs of ergometer cycling after fatiguing exercise are controversial and we decided to re examine them. We could not find any study investigating effects of fatiguing stretch shortening exercise on slow component of VO₂ kinetics during exercise.

The main of the present study was to investigate effects of drop jump exercise on VO₂ during ergometer cycling at several submaximal intensities. Repetitive drop jumps induce muscle fatigue of long duration with signs of muscle damage (Murfet et al., 2003; Skurvedys et al., 2000). 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). The first hypothesis of the study was that during ergometer cycling after the drop jump exercise the plateau level of VO₂ will be higher in the fatigue compared to the control condition. The second hypothesis was that the slow component of VO₂ kinetics would increase in amplitude and appear at lower exercise intensities during muscle fatigue. It is believed that progressive recruitment of type II muscle fibres can be associated with a gradual increase in metabolic cost of ergometer cycling even at 50% VO₂max (Krustedup et al., 2004).

**METHODS**

**Subjects and experiments**

Seven young men (age 24.1 ± 0.7 years, stature 1.79 ± 0.02 m, body mass 71.9 ± 3.9 kg) took part in the study that was approved by the local Ethics Committee. The subjects were students of physical education and participated in aerobic activities two-three times a week (3-5 h per week). All of them were familiar with the procedures of the study. After a detailed explanation, informed consent was obtained, and the participants took part in three different experiments. In experiment I, peak VO₂ was determined. Experiments II and III were designed to study VO₂ during ergometer cycling in rested and fatigue conditions, respectively.

**Ergometer cycling and data collection**

The mechanically braked cycle ergometer (Monark 818E, Monark-Crescent AB, Sweden) was used. The cadence was 70 revolutions per minute. Subjects breathed through low resistance mouthpiece and gas exchange was measured breath-by-breath using miniaturised telemetric gas analysis system (Cortex, Leipzig, Germany). Heart rate was recorded simultaneously (Polar Electro Oy, Kempele, Finland). Data values for these measurements were averaged over 1-min periods for statistical comparison of the exercise in the control and fatigue condition. Analysis of VO₂ kinetic was performed using the initial breath-by-breath data. A fingertip blood sample was collected into a capillary tube at the end of each 6-min bout of exercise and subsequently analysed for blood lactate concentration as described previously (Kulis et al., 1988).

**Experimental protocols**

In experiment I, peak VO₂ (VO₂peak) was evaluated using a ramp exercise test (21 W·min⁻¹). 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 VO₂ was determined. Experiments II and III were performed without any prior exercise (control condition). Then, after at least three days, the ergometer cycling was repeated 60 min after the repetitive drop jump exercise (experiment III, fatigue condition). 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 (Skurvedys et al., 2000). At the same time 60 min of rest after repetitive exercise would allow a recovery.
Table 1. Expiratory pulmonary ventilation (\(V_E\)), heart rate (HR) and pulmonary oxygen uptake (\(VO_2\)) after subtraction of oxygen uptake of cardiac and respiratory muscles (see methods for details). All the data are averages over the last 3 min of cycling at each intensity in control and fatigue conditions. Values are means (±SD).

<table>
<thead>
<tr>
<th>Workload (W)</th>
<th>(VE) (l·min(^{-1})) Control</th>
<th>Fatigue</th>
<th>HR (beats·min(^{-1})) Control</th>
<th>Fatigue</th>
<th>Corrected (VO_2) (l·min(^{-1})) Control</th>
<th>Fatigue</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>24.1 (4.7)</td>
<td>26.6 (1.9)</td>
<td>109 (14)</td>
<td>113 (7)</td>
<td>1.00 (.12)</td>
<td>1.09 (.08)</td>
</tr>
<tr>
<td>105</td>
<td>33.1 (5.6)</td>
<td>37.7 (5.6)</td>
<td>125 (16)</td>
<td>131 (8)</td>
<td>1.34 (.13)</td>
<td>1.50 (.15) *</td>
</tr>
<tr>
<td>140</td>
<td>43.3 (7.6)</td>
<td>51.2 (10.7) *</td>
<td>140 (19)</td>
<td>149 (13)</td>
<td>1.71 (.18)</td>
<td>1.90 (.24) *</td>
</tr>
<tr>
<td>175</td>
<td>60.7 (11.8)</td>
<td>71.5 (17.5) **</td>
<td>158 (20)</td>
<td>165 (13)</td>
<td>2.23 (.28)</td>
<td>2.41 (.25) **</td>
</tr>
</tbody>
</table>

* and ** denote significant (\(p < 0.05\) and \(p < 0.01\), respectively) differences between control and fatigue conditions.

of muscle temperature and metabolites which could interfere with \(VO_2\) responses to exercise (Ratkevicius et al., 1998b; Saugen and Vøllestad, 1995; Tordi et al., 2003).

D\(\text{rop jump exercise}\)

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 to 130-150 heart beats min\(^{-1}\). Then the subjects performed 10 squat-stands. Thereafter, the subject performed 100 drop 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.

\(VO_2\) components

The amplitude of the slow component of \(VO_2\) kinetics was calculated as the difference between the mean value of \(VO_2\) over the last 30 s of the 6th min of exercise and that of the last 30 s of the 3rd min of the test (Whipp et al., 1994). This was apparently the most reliable method of assessment, as mathematical modelling of the slow component did not produce reliable results.

Oxygen uptake of heart and respiratory muscles contributes to \(VO_2\), and changes in pulmonary ventilation and heart rate could have a significant impact on \(VO_2\) response to repetitive exercise. An attempt was made to evaluate importance of these factors to \(VO_2\) (Table 1). The oxygen costs of cardiac work were assumed to be 0.2 ml·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:

\[
W_b = -0.251 + 0.0382V_E + 0.00176V_E^2
\]

where \(W_b\) is work of breathing, and \(V_E\) is expiratory pulmonary ventilation.

Then oxygen uptake of respiratory muscles was estimated as:

\[
V_{RM}O_2 = 34.9 + 7.45W_b
\]

where \(V_{RM}O_2\) is \(O_2\) uptake of the respiratory muscles, \(W_b\) is work of breathing.

Statistical analysis

Two-factor repeated measures analyses of variances

Table 2. Blood lactate and slow component of \(VO_2\) kinetics (s. c. \(VO_2\)) when cycling in control and fatigue conditions. Values are means (±SD).

<table>
<thead>
<tr>
<th>Workload (W)</th>
<th>Blood lactate (mM) Control</th>
<th>Fatigue</th>
<th>s. c. (VO_2) (l·min(^{-1})) Control</th>
<th>Fatigue</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>2.33 (.71)</td>
<td>2.15 (1.02)</td>
<td>.04 (.10)</td>
<td>.07 (.09)</td>
</tr>
<tr>
<td>105</td>
<td>2.22 (.47)</td>
<td>2.15 (.77)</td>
<td>.06 (.08)</td>
<td>.08 (.03)</td>
</tr>
<tr>
<td>140</td>
<td>2.42 (.73)</td>
<td>2.75 (.95)</td>
<td>.08 (.07)</td>
<td>.11 (.11)</td>
</tr>
<tr>
<td>175</td>
<td>3.93 (1.39) #</td>
<td>3.15 (.13) #</td>
<td>.17 (.09) #</td>
<td>.13 (.15)</td>
</tr>
</tbody>
</table>

# Significant (\(p < 0.05\)) differences from the values at 70 W.
Metabolic costs of cycling

Figure 1. Pulmonary oxygen uptake (VO₂) during ergometer cycling at 70 W, 105 W, 140 W and 175 W. Value are means with standard deviations (SD) indicated by the bars.

(ANOVAs) were used when evaluating effects of exercise time and repetition of exercise bout. The F ratios were considered statistically significant when p < 0.05. If significant effects were found, post hoc testing was performed applying paired t-tests with Bonferroni correction for multiple comparisons. Statistical significance of all tests was set at p < 0.05. The results are presented as means ± standard deviations (SD).

RESULTS

VO₂ in repetitive exercise
The VO₂ peak of the volunteers was 51.7 ± 5.6 ml·min⁻¹·kg⁻¹. All the participants cycled at 70, 105, 140 and 175 W in the control and fatigue conditions, respectively. The VO₂ data from these experiments are presented in Figure 1. In the control condition, VO₂ increased with exercise intensity (p < 0.001) reaching 29.6 ± 5.4, 39.4 ± 7.0, 50.8 ± 8.4 and 65.8 ± 11.8% of VO₂ peak at 70, 105, 140 and 175 W, respectively. The prior drop jump exercise tended to amplify the VO₂ response to exercise. From 3rd to 6th min of cycling at 105, 140 and 175 W, VO₂ was higher (p < 0.05-0.01) in the fatigue compared to the control condition. This could be due to changes in pulmonary ventilation (Vₑ) and heart rate (HR). Data on Vₑ, HR and VO₂ corrected for oxygen uptake of cardiac and respiratory muscles are presented in Table 1. Indeed, Vₑ and HR tended to increase when cycling was performed in the damage condition. However, this increase in Vₑ and HR could account for only up to 10% of the overall increase in VO₂ during cycling in the fatigue relative to the control condition. At 105, 140 and 175 W of cycling, VO₂ remained higher (p < 0.05-0.01) in the fatigue compared to the control condition even when oxygen uptake of cardiac and respiratory muscles was subtracted from the VO₂ values.

Slow component of VO₂ kinetics and blood lactate
Data on blood lactate and the amplitude of slow component of VO₂ kinetics are presented in Table 2. 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 VO₂. 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 VO₂. In the control condition, the slow component of VO₂ kinetics was significant (p < 0.05) at 175 W as all seven subjects showed an increase in VO₂ from 3rd min to 6th min of cycling. However, it tended to decrease in the fatigue condition.
**Respiratory exchange ratio (RER)**

The data on RER during exercise are presented in Table 3. The two-way ANOVA showed that RER increased with exercise intensity (p < 0.05), but there were no differences between experiments in the control and fatigue conditions.

<table>
<thead>
<tr>
<th>Workload (W)</th>
<th>Control</th>
<th>Fatigue</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>0.83 (.06)</td>
<td>0.82 (.05)</td>
</tr>
<tr>
<td>105</td>
<td>0.86 (.04)</td>
<td>0.83 (.04)</td>
</tr>
<tr>
<td>140</td>
<td>0.86 (.04)</td>
<td>0.84 (.03)</td>
</tr>
<tr>
<td>175</td>
<td>0.88 (.04)</td>
<td>0.86 (.05)</td>
</tr>
</tbody>
</table>

**Drop jump exercise**

Drop jump exercise was used to induce muscle fatigue which would lead to a change in oxygen costs of the subsequent cycling exercise. During repetitive jumping, VO₂ increased over initial 3 min and then was maintained at the level of 1.38 ± 0.22 l min⁻¹ or approximately 38% of VO₂peak recorded during cycling in experiment I. Data on heights of these jumps are presented in Figure 2. The jump heights tended to decrease during the exercise, but the decrease did not reach the significance level.

![Figure 2](image.png)

**DISCUSSION**

The study 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. Overall, VO₂ increased to higher plateau levels when ergometer cycling was repeated in the fatigue condition. It is argued that compensatory recruitment of type II fibres and impaired force transmission due to damage of structural proteins contributed to this phenomenon. On the other hand results of the study fail to link muscle fatigue and damage to the slow component of VO₂ during cycling exercise.

After the drop jump exercise, VO₂ increased significantly when ergometer cycling was performed at the three highest intensities and a similar tendency was observed at the lowest intensity as well. Each subject cycled under the two conditions on separate occasions. Thus it is unlikely that the increase in VO₂ was accidental. An increase in O₂ costs of cycling at 50% VO₂max has been observed after 3 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 VO₂ and the largest increase in VO₂ 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).

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 (Clarkson and Hubal, 2002; Lieber and Frieden, 1999). Changes in force generating capacity of skeletal muscles were not measured in this study, but our experiments were performed using the same equipment with participation of similar volunteers as in previous studies (Skurvydas et al. 2000, Streckis et al. 2005). Changes in height of repetitive jumps also followed a similar pattern. Other authors employing similar protocols of exercise also reported muscle fatigue and damage (Strojnik et al., 2000). Thus, it is reasonable to assume that muscle fatigue was induced in the present study. It is also likely that some degree of muscle damage was present. Our volunteers did report a mild pain in their muscles when walking the stairs for a few days after the experiment.
Cycling exercise was performed at 70, 105, 140 and 175 W which corresponded to approximately 30, 40, 50 and 65% VO\(_2\)peak. Primarily type I fibres are recruited at intensities below 50% VO\(_2\)max and recruitment of type II fibres increases progressively at higher intensities (Greig et al., 1985; Krstrup et al., 2004). This is in agreement with our data on blood lactate that showed a significant increase only during exercise at the highest intensity (see Table 2). 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). Studies on single fibre preparation and whole muscles suggest that type II fibres have significantly higher ATP costs of isometric force generation than type I fibres (Horrowitz et al., 1994; Nakagawa et al., 2005; Stienen et al. 1996). Thus increased involvement of type II could lead to an increase in average metabolic costs of work. 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). In addition, indirect evidence suggests that mechanical interactions between motor units are involved in reducing ATP costs of force production during isometric contractions (Nakagawa et al., 2005). Damage to structural proteins is likely to impair such interactions and increase metabolic costs of muscle work in addition to the possible effects of type II fibre recruitment.

Exercise induced muscle pain and soreness could also be associated with increased metabolic costs of exercise though evidence is contradictory (Moysi et al., 2005). There was no attempt to quantify muscle pain in the present study, but exercise was performed one hour after repetitive drop jumps. Muscle pain and inflammation are mild at this time point of recovery and become marked 1-3 days after muscle damage inducing exercise (Clarkson and Newham, 1995). Thus it is unlikely that muscle pain was a significant factor in this study.

Muscle temperature could also potentially affect metabolic costs of cycling exercise (Edwards et al., 1972). Muscle temperature was not measured in our study. However, a moderate increase in muscle temperature did not increase VO\(_2\) when ergometer cycling was performed at similar exercise intensities (Koga et al., 1997). In this study, cycling exercise was carried out 1 hour after the drop jump exercise when major recovery of muscle temperature should have occurred (Saugen and Vøllestad, 1995). Furthermore, VO\(_2\) did not differ significantly between control and fatigue conditions in the first bout of cycling when the largest differences in muscle temperature are expected. In view of all these considerations, it is unlikely that temperature was the major factor in our study.

It appears also that 1 hour of rest is sufficient for a major metabolic recovery since repetitive jumping can be considered as an exercise of moderate intensity (<40% VO\(_2\)peak). RER was similar during exercise in the control and fatigue conditions, and differences in VO\(_2\) between exercise in the control and fatigue conditions could hardly be due to differences in the metabolic state and blood flow at the beginning of exercise.

The slow component of VO\(_2\) response to exercise was evaluated in this study as there was a slow increase in VO\(_2\) during the forth bout of exercise at 175 W (~ 65% VO\(_2\)peak). The protocol of ergometer cycling was possibly not optimal for full manifestation of this phenomenon as higher exercise intensities (75% VO\(_2\)max) are associated with a larger drift in VO\(_2\) during exercise (Pringle et al., 2003). Incomplete metabolic recovery between repeated exercise bouts could also affect VO\(_2\) kinetics (Tordi et al., 2003). However, exercise bouts at 70, 105 and 140 W were associated with little accumulation of blood. There is evidence that recovery of ATP, phosphocreatine and intracellular pH are essentially complete within 4 min even after exercise of much higher intensity (Ratkevicius et al., 1998b). Thus it seems unlikely that prior exercise compromised VO\(_2\) slow component to a significant degree as it appeared in all seven subjects. Interestingly, the drop jump exercise did not increase the amplitude of the slow component of VO\(_2\). If anything, it became smaller. This suggests that recruitment of type II fibres is not always linked to appearance of the slow component of VO\(_2\). If anything, it became smaller. This suggests that recruitment of type II fibres is not always linked to appearance of the slow component of VO\(_2\). If anything, it became smaller. This suggests that recruitment of type II fibres is not always linked to appearance of the slow component of VO\(_2\).
Results of our results do not necessarily contradict findings of Krstrup et al. (2004). Glycogen depletion and muscle damage after repetitive drop jumps could produce different patterns of muscle fibre recruitment. Glycogen depletion is likely to increase fatigue rate in the affected muscle fibres and compensatory recruitment of type II fibres would accelerate after a few minutes of exercise. Muscle damage is expected to cause a failure to produce any force in a number of fibres and a larger number of type II fibres would be recruited right from the beginning of exercise.

CONCLUSIONS

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 slow component of VO₂ kinetics.

ACKNOWLEDGEMENT

All the experiments of this study comply with the current laws of Lithuania where the study was performed. Authors would like to thank volunteers who enthusiastically participated in this study.

REFERENCES


KEY POINTS

- Repetitive fatiguing exercise induce an increase in metabolic costs of ergometer cycling exercise.
- It is argued that muscle pain, muscle temperature, elevated pulmonary ventilation and heart rate, shift towards from carbohydrate to fat metabolism are of minor importance in this phenomenon.
- Increased recruitment of type II fibres and impaired force transmission between muscle fibres due to damage of structural proteins appear to play the major role in reducing efficiency of ergometer cycling.

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