Effects of muscle-damaging exercise on physiological, metabolic, and perceptual responses during two modes of endurance exercise

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Abstract

Research on submaximal endurance exercise after exercise-induced muscle damage (EIMD) has produced equivocal findings. One possible explanation for this is that the responses are likely to be sensitive to the mode of endurance exercise adopted. Therefore, this study examined the influence of exercise mode (cycling vs. running) after EIMD. Twenty-four and 48 hours after EIMD, 10 male participants were measured for perceived muscle soreness, creatine kinase (CK) activity, knee extensor strength, and physiological (VO2, ventilation), metabolic (blood lactate), and perceptual [rating of perceived exertion (RPE)] responses during two counterbalanced 10-minute cycling and running bouts at lactate threshold. The muscle-damaging exercise was effective (p < 0.05) in increasing muscle soreness, elevating CK activity, and decreasing knee extensor strength at 24 and 48 hours. Similarly, increases in VO2, ventilation, and RPE were observed during both cycling and running after EIMD (p < 0.05), while blood lactate responses remained unchanged (p > 0.05). There was an interaction of time × mode for ventilation (p < 0.05), reflecting a higher relative increase for running at 24 hours post EIMD than cycling, but not for VO2, RPE, or blood lactate responses. The increased VO2 response during running could be attributed to changes in lower limb kinematics (stride length and stride frequency) and a decreased ability to utilize the stretch-shortening cycle, while the unexpectedly increased VO2 cycling response might be due to the recruitment of auxiliary muscles after EIMD. It is postulated that the differences in ventilation between exercise modes are due to different stimuli activating afferent fibers post-EIMD.

Keywords: Effort perception; Exercise-induced muscle damage; Oxygen uptake; Submaximal endurance exercise; Ventilation

Introduction

A body of evidence has emerged that the development of neuromuscular characteristics from resistance-based exercise can further enhance endurance exercise performance. However, while prolonged exposure to resistance training may improve performance in the long term, a consequence of such training, particularly when it is unaccustomed, is the immediate and long-lasting appearance of symptoms associated with exercise-induced muscle damage (EIMD) in the days following. Such symptoms include increases in muscle soreness, swelling, elevated muscle proteins in the blood, impaired muscle function, and reduced neuromuscular control. Furthermore, while maximal oxygen uptake appears to be unaltered following muscle-damaging exercise, several studies have observed no change in submaximal oxygen uptake after EIMD, suggesting that exercise economy remains stable; in contrast, others have observed increases in submaximal VO2.

The challenge of interpreting these opposing results is made more difficult by the facts that studies have used different exercise protocols to induce muscle damage and that the physiological and perceptual responses after EIMD might be sensitive to the mode of endurance exercise adopted. Indeed, those studies showing no change in submaximal VO2 typically used cycling rather than running exercise. Therefore,
it might be that alterations in limb kinematics [stride length (SL) and stride frequency (SF)] after EIMD have a greater impact on running than on cycling. Several studies have shown that EIMD provokes changes to these parameters during running and have associated these changes with increases in VO₂. The inference from such research is that muscle damage causes changes in a person’s stride pattern in order to limit the level of discomfort, and consequently an increase in oxygen cost occurs. Given that SL is unlikely to deviate during cycling, it is unlikely that oxygen cost will increase due to such changes.

Alterations in lactate metabolism as a consequence of EIMD have not been reported consistently, with some studies reporting no change in blood lactate ([La]) concentration during submaximal exercise and others reporting an increase. Elevated [La] responses may reflect an increased metabolic demand on undamaged fibers during exercise or that damage may occur to type II muscle fibers during eccentric exercise, resulting in an increased activation of nondamaged type II fibers to enable the same level of force production. However, as is the case with the equivocal VO₂ responses observed following muscle-damaging exercise, the different methods used to evoke symptoms of EIMD, and the subsequent endurance exercise adopted make comparisons between such studies problematic.

Changes in the rating of perceived exertion (RPE) during submaximal exercise suggest that participants demonstrate an altered sense of effort as a consequence of EIMD. Such responses may be associated with an increase in muscle pain following eccentric exercise, which has clearly been shown to heighten the sense of effort during force-matching tasks. Similarly, increases in RPE have also coincided with an increased ventilatory response, which may transcend from a disruption to nerve afferents located in and around the blood vessels of exercising muscle that control ventilation. It is known that ventilation and muscle pain are strong determinants of an individual’s RPE response to exercise, although whether this is consistent for different endurance exercise modes or not is unknown.

Given the uncertainties highlighted above, the purpose of this study was to evaluate the effects of the same bout of muscle-damaging exercise on physiological, metabolic, and perceptual responses to submaximal running and cycling exercise.

**Methods**

**Experimental design and participants**

Ten healthy males (age 22.8 ± 2.5 years, stature 1.77 ± 0.06 m, body mass 75.9 ± 8.8 kg), all of whom engaged in regular physical activity but had not undertaken any form of lower limb resistance exercise in the 6 months prior to assessment, volunteered to participate in the study. Prior to the study, each participant completed a written informed consent form and a health questionnaire, and received a verbal explanation of the risks associated with the experimental procedures. Ethical approval was obtained from the University’s Faculty of Health and Applied Sciences Research Ethics Committee. Participants performed a cycling and a running incremental exhaustive test in a counterbalanced order, 1 week apart, to determine lactate threshold (LT) and VO₂peak for each exercise mode (see Table 1). Subsequent measurements associated with two counterbalanced, 10-minute cycling and running exercise bouts at individual LT were recorded at baseline, and then at 24 and 48 hours after the lower limb resistance exercise (see Fig. 1). All participants were asked to refrain from any strenuous exercise 24 hours prior to each test, maintain their normal diet, and avoid using any analgesic agents.

**Procedures**

**Assessment of LT and peak oxygen uptake (VO₂)**

Two incremental protocols to exhaustion were administered through an electronically braked cycle ergometer (Lode Excalibur Sport, Lode Medical Technology, Groningen, The Netherlands) and a motorized treadmill (Woodway PPS 55 sport-I, Woodway, Waukesha, Wisconsin, USA), in order to determine mode-specific LT and VO₂peak. The protocols commenced at a workload of 50 W (cycling) or at a speed of 9 km/hour with a 1% incline (running) and increased by 25 W or 0.5 km/hour every 4 minutes until the LT was reached. Individual LT was accepted as the power output or speed at which [La] increased by 1 mmol/L above the baseline values. At this point, the resistance was increased by 25 W/min (cycling) and the speed by 1 km/hour/minute (running) until volitional exhaustion. Volitional exhaustion was defined as the point at which participants could no longer maintain either a cycling cadence between 60 and 80 rev/minute or the speed of the treadmill. [La] concentrations were obtained from the Lactate Pro analyzer (Arkray, Kyoto, Japan) using fingertip capillary blood samples taken during the 30-second periods of forced inactivity between each 4-minute exercise bout.

Expired air was collected continuously throughout each exhaustive trial using an online metabolic system calibrated prior to each test with a span gas mixture of 16% O₂ and 5% CO₂ and a 3 L syringe (Hans Rudolph Inc., Kansas City, MO, USA). Gas exchange variables such as oxygen uptake (VO₂) were recorded breath by breath and later averaged over 30 seconds. Heart rate (HR), collected via telemetry (Polar Electro, Polar Beat Oy, Kempele, Finland), and RPE were recorded breath by breath and later averaged over 30 seconds. Heart rate (HR), collected via telemetry (Polar Electro, Polar Beat Oy, Kempele, Finland), and RPE were recorded breath by breath and later averaged over 30 seconds. Heart rate (HR), collected via telemetry (Polar Electro, Polar Beat Oy, Kempele, Finland), and RPE were recorded breath by breath and later averaged over 30 seconds. Heart rate (HR), collected via telemetry (Polar Electro, Polar Beat Oy, Kempele, Finland), and RPE were recorded breath by breath and later averaged over 30 seconds.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Cycling</th>
<th>Exercise mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂peak (mL/kg/min)</td>
<td>44.44 ± 7.33</td>
<td>52.01 ± 6.11</td>
</tr>
<tr>
<td>HRpeak (beats/min)</td>
<td>181 ± 9</td>
<td>191 ± 5</td>
</tr>
<tr>
<td>LT (mmol/L)</td>
<td>2.8 ± 1.0</td>
<td>3.9 ± 1.6</td>
</tr>
<tr>
<td>LTWR</td>
<td>135.0 ± 33.8 W</td>
<td>11.3 ± 1.3 km/h</td>
</tr>
<tr>
<td>LTRPE</td>
<td>13.4 ± 1.1</td>
<td>13.6 ± 1.0</td>
</tr>
</tbody>
</table>

HRpeak = peak heart rate at peak oxygen uptake; LT = lactate threshold; LTRPE = rating of perceived exertion at lactate threshold; LTWR = work rate corresponding to lactate threshold; SD = standard deviation; VO₂peak = peak oxygen uptake.
recorded in the final 15 seconds of each exercise bout. VO2peak was accepted as the highest VO2 averaged over 30 seconds.

**Muscle-damaging exercise**

To induce symptoms of muscle damage, participants performed 10 sets of 10 Smith-machine squats at 80% of body mass, with a 2-minute recovery period between each set. Previous research has shown this protocol to be successful in inducing symptoms of muscle damage to the knee extensors.5,7,10,30

**Indirect markers of muscle damage**

With hands on hips and squatting to an approximate knee angle of 90°, each participant was asked to provide his perceived level of muscle soreness of the knee extensors using a visual analog scale (VAS). The VAS was numbered from 0 to 10 (on the reverse of the scale, unseen by the participant), wherein 0 indicated no muscle soreness, 5 signified that the muscles felt sore upon movement, and 10 indicated that the muscles were too sore to move. This scale has been used successfully in previous research9 and has been established as a valid and reliable tool to measure soreness.41

Isokinetic knee extensor torque was measured using a Biodex dynamometer (Biodex, Shirley, NY, USA) at a velocity of 60°/s. Testing was preceded by a standardized warm-up of 3-minute cycling at 50 W (Monark, 874E, Monark, Varberg, Sweden). Participants performed five maximal efforts, from which the highest value (N m) was recorded.

Plasma creatine kinase (CK) activity was assessed from a fingertip capillary sample with the participant being in a seated position. After prewarming the hand by immersing it in warm water (≈42 °C), a 30 µL sample of blood was collected, immediately pipetted to a test strip, and analyzed for CK using a colorimetric assay procedure (Reflotron, Boehringer Mannheim, Mannheim, Germany).

**Submaximal exercise protocols**

The two 10-minute submaximal exercise protocols were performed in a counterbalanced order, separated by a 30-minute rest period, on an electronically braked cycle ergometer (Lode Excalibur Sport, Lode Medical Technology) and a motorized treadmill (Woodway PPS 55sport-I, Woodway GmbH), at an intensity corresponding to each participant’s LT. Expired air, HR, RPE, and [La] were recorded in the manner described above. The treadmill gradient was also set at a 1% incline to reflect the energetic cost of outdoor running.42

**Limb kinematics**

A high-speed video camera (Casio Exilim Pro Ex-F1) recorded sagittal plane images at 100 Hz during the final 10 seconds of each 10-minute submaximal running bout. The 10-second video recordings were then downloaded and digitized using motion analysis software (Quintec Biomechanics 9.03 v14). SL and SF during each running bout were determined from the method previously used by Braun and Dutto18 and Chen et al.17 SL was calculated using the following formula:

\[
SL (\text{m}) = \frac{\text{velocity (m/s)}}{\text{stride time (seconds)}}
\]

SF was calculated as the number of full strides divided by the time between the first and last heel contacts.

**Statistical analysis**

Descriptive statistics (means ± SD) were calculated for all the dependent variables, and their distributions were checked for normality using the Shapiro–Wilk test. Changes over time in the markers of muscle damage, SL, SF, and cycling revolutions per minute (RPM) were subsequently analyzed using separate one-way repeated measures analysis of variance (ANOVAs). Two-way (time [3] × mode [2]) repeated measures ANOVAs were applied to the physiological, metabolic, and perceptual data and interpreted with respect to the assumption of sphericity (using Mauchly’s test), with any violations adjusted with the Greenhouse–Geisser correction. Post hoc Tukey tests, modified for repeated measures,43 were used to determine where significant differences occurred between conditions. The alpha level was initially set at \( p \leq 0.05 \).
Table 2
Changes in the indirect markers of muscle damage (mean ± SD) following 100 squats.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>24 h</th>
<th>48 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceived muscle soreness</td>
<td>0.4 ± 0.5</td>
<td>5.4 ± 1.5*</td>
<td>6.7 ± 1.3*</td>
</tr>
<tr>
<td>Peak torque (N m)</td>
<td>212.8 ± 38.5</td>
<td>185.4 ± 47.2*</td>
<td>183.0 ± 51.8*</td>
</tr>
<tr>
<td>CK activity (U/L)</td>
<td>78.1 ± 30.4</td>
<td>238.9 ± 181.6*</td>
<td>143.1 ± 83.9</td>
</tr>
</tbody>
</table>

*Significantly different to baseline (p < 0.05).

CK = creatine kinase; SD = standard deviation.

Results

Indirect markers of muscle damage

The squatting exercise was successful in inducing significant changes in all the measured indirect markers of muscle damage. Perceived muscle soreness increased \( F_{(2,18)} = 114.2, p < 0.0005 \) over time, with values being higher after 24 and 48 hours post-baseline, whereas peak isokinetic knee extensor torque decreased \( F_{(2,18)} = 10.1, p = 0.011 \), with values being lower after 24 hours than the baseline value. The effect of time on CK activity \( F_{(2,18)} = 7.2, p = 0.021 \) was due to values being higher than the baseline value after 24 hours, but not after 48 hours (see Table 2).

Submaximal cycling responses to muscle-damaging exercise

Table 3
Mean (±SD) physiological, metabolic, perceptual, and kinematic responses to submaximal exercise at LT following 100 squats.

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>24 h</th>
<th>48 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2 (ml/kg/min)</td>
<td>28.08 ± 5.58</td>
<td>28.81 ± 6.02</td>
<td>29.67 ± 6.53*</td>
</tr>
<tr>
<td>VCO2 (ml/kg/min)</td>
<td>26.91 ± 4.60</td>
<td>26.99 ± 5.66</td>
<td>28.12 ± 6.16</td>
</tr>
<tr>
<td>VE (L/min)</td>
<td>58.4 ± 10.18</td>
<td>60.9 ± 15.13</td>
<td>66.2 ± 16.94*</td>
</tr>
<tr>
<td>VE/VO2</td>
<td>27.72 ± 2.49</td>
<td>27.89 ± 2.98</td>
<td>29.45 ± 2.81*</td>
</tr>
<tr>
<td>VE/VCO2</td>
<td>28.81 ± 2.17</td>
<td>29.7± ± 2.48</td>
<td>30.99 ± 1.73*</td>
</tr>
<tr>
<td>RPE</td>
<td>13.5 ± 1.4</td>
<td>15.2 ± 1.6*</td>
<td>15.4 ± 1.8*</td>
</tr>
<tr>
<td>fR (breaths/min)</td>
<td>27.9 ± 5.2</td>
<td>30.6 ± 5.95</td>
<td>35.3 ± 6.33*</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>132.9 ± 16.7</td>
<td>137.4 ± 15.0</td>
<td>140.4 ± 17.9*</td>
</tr>
<tr>
<td>[La] (mmol/L)</td>
<td>3.48 ± 1.53</td>
<td>3.40 ± 1.57</td>
<td>3.69 ± 1.58</td>
</tr>
<tr>
<td>RPM</td>
<td>79.8 ± 15.7</td>
<td>75.6 ± 18.3</td>
<td>77.8 ± 18.4</td>
</tr>
<tr>
<td>SL (m)</td>
<td>—</td>
<td>—</td>
<td>2.24 ± 0.26</td>
</tr>
<tr>
<td>SF</td>
<td>—</td>
<td>—</td>
<td>14.1 ± 0.6</td>
</tr>
</tbody>
</table>

*Significantly different to baseline (p < 0.05).

fR = breathing frequency; HR = heart rate; [La] = blood lactate concentration; RPE = rating of perceived exertion; RPM = revolution per minute; SF = stride frequency; SL = stride length.

Submaximal running responses to muscle-damaging exercise

There was a main effect of time on \( \dot{V}_O2 \) \( F_{(2,18)} = 6.4, p = 0.008 \), \( V_E \) \( F_{(2,18)} = 12.7, p = 0.0001 \), \( V_E/\dot{V}_O2 \) \( F_{(2,18)} = 11.4, p = 0.001 \), \( f_R \) \( F_{(2,18)} = 7.3, p = 0.005 \), and RPE \( F_{(2,18)} = 11.9, p = 0.001 \), with values being higher than the baseline value after 24 and 48 hours. \( V_E/\dot{V}_O2 \) \( F_{(1,19)} = 1.9, p = 0.002 \) was also increased after muscle damage, although only after 24 hours. No effect was observed for \( \dot{V}_O2 \) \( F_{(2,18)} = 3.2, p = 0.066 \), HR \( F_{(2,18)} = 2.1, p = 0.154 \), or [La] response \( F_{(2,18)} = 2.1, p = 0.148 \). These changes were accompanied by decreases in SL \( F_{(1,19)} = 9.2, p = 0.012 \) and increases in SF \( F_{(1,19)} = 6.9, p = 0.023 \) after 24 and 48 hours following the squatting exercise (see Table 3).

Submaximal responses to muscle-damaging exercise—cycling versus running

The time \( \times \) mode effect on \( \dot{V}_O2 \) \( F_{(2,18)} = 1.3, p = 0.308 \), \( \dot{V}_C O2 \) \( F_{(2,18)} = 2.0, p = 0.167 \), \( f_R \) \( F_{(1,11)} = 4.5, p = 0.057 \), HR \( F_{(2,18)} = 1.4, p = 0.280 \), RPE \( F_{(2,18)} = 0.9, p = 0.414 \), or [La] response \( F_{(2,18)} = 2.5, p = 0.108 \) was not significant after muscle-damaging exercise. However, a time \( \times \) mode interaction on \( V_E \) \( F_{(2,18)} = 5.3, p = 0.016 \) reflected a higher relative increase for running after 24 hours post-EIMD than for cycling. Similarly, \( V_E/\dot{V}_O2 \) \( F_{(2,18)} = 6.2, p = 0.009 \) and \( V_E/\dot{V}_C O2 \) \( F_{(2,18)} = 7.0, p = 0.006 \) were also found to be higher after 24 hours during running compared to those during cycling (see Table 4).

Discussion

The changes observed in perceived muscle soreness, muscle function, and plasma CK indicate that the squatting protocol used in this study was effective in inducing symptoms...
associated with EIMD. These observations concur with previous studies that have adopted similar and contrasting modes of muscle-damaging exercise. The CK response peaked after 24 hours and not after 48 hours (as with perceived muscle soreness and muscle function). This pattern of response has been observed with previous research and may reflect an accelerated clearance of CK from the blood after 24 hours.

It is notable that the physiological and perceptual responses to submaximal cycling and running exercise in the current study were altered as a result of EIMD. In terms of \( \text{VO}_2 \), increases occurred during both modes of exercise, although the time course appeared to be different. That is, the \( \text{VO}_2 \) response during running was significantly elevated above pre-EIMD values after 24–48 hours, which concurred with previous findings, whereas the cycling \( \text{VO}_2 \) response was significantly higher only after 48 hours. This is a novel and unexpected finding in light of past research and deserves further scrutiny.

Cavanagh and Williams reported an increased \( \text{VO}_2 \) response when runners deviated from their optimal SL. Therefore, it is feasible that the elevated \( \text{VO}_2 \) response during submaximal running was due, in part, to changes in lower limb kinematics following muscle-damaging exercise. Indeed, the findings that this increased oxygen cost of running coincided with alterations in SL and SF, and that changes in \( \text{VO}_2 \) during submaximal running were found to be inversely correlated with changes in SL after EIMD for seven of the 10 participants \((r = -0.71 \text{ to } -0.92)\) reinforce the findings of previous running-related studies. Moreover, the outcome that our participants experienced heightened muscle soreness 24 and 48 hours after the squatting exercise might explain the shortened SL as a strategy to limit their discomfort. However, while kinematic changes could have influenced the time course response of running \( \text{VO}_2 \) after EIMD, the observation of Hamill et al that minor kinematic changes occurred without any concomitant rise in oxygen cost implies that other mechanisms were involved.

Chen et al attributed post-EIMD decrements in running economy between 24 and 72 hours to an impaired ability to utilize the stretch-shortening cycle (SSC). Previous research has demonstrated that improvements in running economy are a result of increased musculotendinous stiffness, allowing the muscle to absorb and utilize elastic energy more effectively during the SSC. However, EIMD has been shown to reduce stretch reflex sensitivity and muscle stiffness regulation, leading to a decline in the force potential of the SSC. Therefore, it is possible that reductions in musculotendinous stiffness following squatting exercise could have occurred as early as 24 hours post-EIMD, leading to a decreased ability to absorb and utilize elastic energy during running, creating an increased energy cost. Although, such changes have yet to be demonstrated empirically during running, this is an area for future investigation.

The aforementioned post-EIMD increase in \( \text{VO}_2 \) during submaximal cycling is at odds with previous research. Adopting a similar mode of muscle-damaging exercise, with comparable decrements in muscle function, both Davies et al and Moysi et al reported no changes in \( \text{VO}_2 \) during submaximal cycling. Studies using plyometrics and eccentric bench stepping to induce muscle damage also reported unchanged \( \text{VO}_2 \) responses during submaximal cycling. Greater physiological responses have been reported during cycling at slower pedaling cadences. However, changes in cycling cadence could not explain the increase in \( \text{VO}_2 \) as RPM remained unchanged (see Table 3). The elevation in \( \text{VO}_2 \) might have been due to the activation of auxiliary muscles after muscle-damaging exercise. For example, knee extensor strength had decreased by approximately 15% 48 hours after muscle damage, suggesting that increased motor unit activation may have been evident in order to generate the same level of force required during the fixed-intensity cycling. Indeed, as Bigland-Ritchie and Woods have observed a linear relationship between EMG activity and oxygen cost, it is possible that elevations in \( \text{VO}_2 \) during cycling might be explained by increased recruitment of motor units after EIMD. This requires further investigation. The only other study to observe an increase in \( \text{VO}_2 \) during submaximal cycling after muscle-damaging exercise postulated that it might have been due to an increase in ventilation. As our current elevations in \( \text{VO}_2 \) during both cycling and running occurred alongside increases in \( V_E \) (see Tables 3 and 4), it is possible that the
lungs evoked an added metabolic cost in order to facilitate the augmented $V_E$ response.

The elevated $V_E$ responses concur with previous studies during both submaximal running\textsuperscript{16,18,51} and cycling exercise.\textsuperscript{29,10,27,30,52} As with the $VO_2$ response, the differing time course of $V_E$ and $f_R$ during running and cycling after EIMD was not hypothesized and is difficult to explain. However, the following are offered as possible contributing factors. In contrast to cycling, running utilizes the SSC, and there is strong evidence that the stretch reflex has a vital role in SSC activity and contributes to force generation between eccentric and concentric phases.\textsuperscript{53} The reduction in stretch-reflex sensitivity following muscle damage is thought to be mediated by the activation of fine myelinated (Group III) and unmyelinated (Group IV) afferent fibers.\textsuperscript{47,54} These small afferents could also exert a significant influence on $V_E$ during dynamic exercise.\textsuperscript{7,55} Therefore, it is possible that activation of Group III and IV afferents inhibiting stretch-reflex sensitivity as a consequence of EIMD may have led to the early augmentation of $V_E$ during running. Group III and IV afferent fibers are often described as being polymodal, in that they are sensitive to several parameters associated with fatigue or muscle damage.\textsuperscript{34,37} Muscle pain has also been shown to provoke an increased ventilatory response through activating nociceptive muscle afferents.\textsuperscript{56–58} Therefore, such pain (soreness) following the squatting exercise might have also stimulated afferent fibers, contributing to the increased ventilatory response during running and cycling. Furthermore, the heightened muscle soreness response after 48 hours could have been responsible for the increase in $V_E$ and $f_R$ during cycling after 48 hours.

Alternatively, an increased ventilatory response while exercising with muscle damage has been attributed to alterations in metabolic pathways.\textsuperscript{14} Since [La] values in the current study were not statistically different during either cycling or running after muscle damage, the increase in $V_E$ could not be attributed to an increased respiratory response to cope with increased acidosis. Furthermore, post-EIMD $V_E/VO_2$ was also elevated during both modes of exercise, indicating that $V_E$ was not increased to excrete accumulating $CO_2$ after squatting exercise.\textsuperscript{9}

Although there was no difference in RPE response, both modes of fixed-intensity exercise revealed significant increases in effort perception 24 and 48 hours after EIMD. These results are consistent with previous research investigating the effects of EIMD during cycling\textsuperscript{9,10} and running.\textsuperscript{17,25} Jameson and Ring\textsuperscript{37} suggested that effort perception during endurance exercise is based on a combination of both peripheral (increased leg pain) and central (feelings of breathlessness) feedback. The increased knee extensor soreness reported in the current study could have provided the peripheral cue, while the elevated $V_E$ and $f_R$ responses reported during both modes of exercise provided the central cue.\textsuperscript{10} Moreover, activation of mechanoreceptors in the chest wall, lungs, and airways might have increased breathing rate, influencing the participants in perceiving exercise to be harder following squatting exercise.\textsuperscript{39} Marcora\textsuperscript{59} challenged the view that effort perception during exercise is dependent on feedback from the skeletal muscle, heart, and lungs, suggesting that an increase in RPE during exercise is centrally governed from the brain. However, Amann et al\textsuperscript{60} reported that a reduction in $V_E$ and RPE responses occurred through blocking the afferent feedback response from the locomotor muscles. Arguably, therefore, muscle afferents in the current study might have influenced RPE responses during both cycling and running modes after muscle-damaging exercise.

Although attempts were made to ensure that the exercise intensities between running and cycling were similar, it is possible that our findings were influenced by the participants’ training history. While they were engaged in regular endurance exercise (2–3 sessions per week), this mainly consisted of running-based activity with limited cycling training. Unsurprisingly, symptoms associated with EIMD are dependent on training history, with greater responses being reported in less active muscle groups than in those regularly exposed to exercise.\textsuperscript{26,61} Furthermore, individuals more accustomed with running are able to exercise at a higher intensity for a given RPE compared to those accustomed with cycling.\textsuperscript{62} Therefore, it is possible that participants in the current study tolerated running with muscle damage due to their greater familiarity with the exercise modality, while the unexpected increase in $VO_2$ during cycling might have occurred due to the participants being less familiar with this type of exercise.

In conclusion, submaximal oxygen uptake and ventilatory responses were increased during both running and cycling modes after EIMD. However, the time course of these appeared to be mode specific. It is posited that the elevated $VO_2$ responses observed during running were due to changes in lower limb kinematics and a decreased ability to utilize the SSC, while the recruitment of auxiliary muscles after EIMD might have led to the unexpected increase in cycling $VO_2$ response. The differences in ventilatory response between exercise modes after muscle damage were possibly due to different stimuli activating afferent muscle fibers. Future studies should examine whether alterations in muscle recruitment are responsible for changes in physiological responses during submaximal endurance exercise and whether such changes occur in participants trained in both cycling and running. From an applied perspective, given that long-term resistance exercise can improve endurance performance, individuals considering concurrent training should be aware of the consequences that unaccustomed resistance exercise can have on submaximal cycling and running exercise performed in the days following.

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References


