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Time-course of recovery of peak oxygen uptake after exercise-induced muscle damage

Christopher D. Black[a,b,*], Alexander R. Gonglach[a], Robert E. Hight[a], Jessica B. Renfroe[a]

[a] Department of Health and Exercise Science, University of Oklahoma, Norman, OK USA
[b] Department of Health, Exercise Science, and Recreation Management, University of Mississippi, Oxford, MS, USA

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ABSTRACT

$\dot{V}O_2$ peak has been shown to be reduced 48 h following exercise-induced muscle damage (EIMD), but it is unclear how long this reduction may persist. In this study eight endurance trained participants (21.5 ± 1.1 years old) performed a maximal exercise tests over 10-days followings EIMD. Cardiorespiratory variables were collected via open-circuit spirometry and soreness, maximal strength (MVC), motor-unit recruitment, and contractile properties were assessed prior to each test. MVC was reduced for up to 4-days ($p \leq 0.05$) and soreness was evident for 10-days in the quadriceps ($p < 0.05$). $\dot{V}O_{2peak}$ was reduced 7.4% 2-days post EIMD (55.5 ± 6.0 vs. 51.3 ± 5.8; $p = 0.006$) and remained reduced in 6 of 8 participants at 10-days post ($p = 0.005$). No relationship was found between changes in MVC, soreness, motor-unit recruitment, and contractile properties and changes in $\dot{V}O_{2peak}$ ($p > 0.05$). EIMD resulted in small, but prolonged reductions in $\dot{V}O_{2peak}$. Our findings suggest mechanisms aside from force loss and soreness are primarily responsible for the reductions in $\dot{V}O_{2peak}$ after EIMD.

1. Introduction

High-intensity resistance (RT) and explosive (e.g., plyometric) training (ET), when performed in combination with endurance training, have been shown to improve endurance performance [see (Yamamoto et al., 2010; Yamamoto et al., 2008) for review]. However, a potential negative consequence of RT and ET, especially at the beginning of training and/or when intensity and volume-load are progressively increased, could be exercise-induced muscle damage (EIMD). EIMD typically occurs following the performance of novel, high force eccentric exercise which is experienced during RT and ET. The disruption of sarcomere and contractile ultrastructure following EIMD leads to a well characterized sequelae of signs and symptoms including elevated serum creatine kinase (Newham et al., 1983a), inflammation and localized edema (Black et al., 2008; Black and McCully, 2008a), a decreased ability to generate force (Newham et al., 1983b), and heightened pain sensitivity—often termed delayed-onset muscle soreness (DOMS) (Newham et al., 1983b). A growing body of evidence clearly indicates EIMD negatively impacts endurance exercise performance as demonstrated by reductions in time-trial performance (Burt and Twist, 2011; Marcora and Bosio, 2007; Twist and Eston, 2009) and time-to-exhaustion (Asp et al., 1998; Carmichael et al., 2006, 2010; Davies et al., 2011a, 2008, 2009).

While the exact mechanism(s) underlying the reduced endurance performance remain unclear, EIMD been shown to impact metabolism in a manner consistent with greater stress being placed upon uninjured muscle fibers and to alter perceptual responses to exercise. EIMD leads to increases in the ratio of inorganic phosphate (P_i) to phosphocreatine (PCr) (Davies et al., 2011a; Foure et al., 2014) and blood lactate [La] at rest (Asp et al., 1998). Movement economy (oxygen cost per work rate) has also been shown to be reduced following EIMD (Burt et al., 2013; Burt et al., 2014; Chen et al., 2009, 2007). In a previous study, our lab demonstrated reductions in cycling $\dot{V}O_2$ peak 48-h following EIMD of the quadriceps (Black and Dobson, 2012). Our findings that participants were unable to reach similar heart rates at exercise cessation combined with similar reductions in strength and $\dot{V}O_2$ peak (~10%) suggest reduced strength and/or motivational factors rather than direct impairments of cardiorespiratory or mitochondrial function may be at play. Consistent with this idea, EIMD leads to heightened ratings of perceived exertion (RPE) (Black and Dobson, 2013; Burt et al., 2012, 2013; Davies et al., 2009, 2011b; Gleeson et al., 1995; Twist and Eston, 2009) and heightened ratings of muscle pain (Black and Dobson, 2013) in humans during exercise.

Previous studies have focused on the 24–48 h immediately following eccentric exercise when force loss and DOMS, indirect

* Corresponding author at: Department of Health and Exercise Science University of Oklahoma Norman, OK 73069, USA.
E-mail address: cblack@ou.edu (C.D. Black).

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markers commonly used to assess EIMD, are most pronounced. A recent study (Burt et al., 2013) used the repeated-bout effect, where an initial bout of damaging exercise reduces damage in subsequent exercise, to manipulate the magnitude of EIMD and associated force loss and DOMS and demonstrated an attenuation of the increases in RPE, [La], $\dot{V}_E$, and $\dot{V}O_2$ during submaximal running when EIMD was reduced following the "repeated bout." Indicating the physiological and perceptual responses during exercise may be related to force loss and/or DOMS. Force and soreness recover (e.g., return to baseline values) in the days following EIMD along a well characterized time-course and tend to returned to baseline levels within 7–10 days following moderate EIMD (Black and McCully, 2008b; Prasartwuth et al., 2006; Sayers et al., 2003). While perhaps not the single best indicator of endurance performance, $\dot{V}O_2$ peak is nonetheless an integrative measure of aerobic metabolic function, and is associated with endurance performance (Grant et al., 1997). Extending upon our previous findings and determining if the commonly observed changes in cardiorespiratory and perceptual variables also recover following EIMD along a similar time-course as force loss and DOMS could provide further insight into the role they may play in reduced endurance performance following EIMD. As such the purpose of the present experiment was to examine the effects of EIMD on $\dot{V}O_2$ peak and its relationship to maximal voluntary strength (MVC), muscle contractile properties, and DOMS over the course of 7–10 days following eccentric exercise.

2. Materials and methods

2.1. Participants

Eleven healthy, college-aged men and women volunteered to participate. A mixed sex sample was chosen as, in humans, men and women have been shown to respond similarly to EIMD (Hubal et al., 2008; Rinard et al., 2000). The participants were experienced runners who self-reported running at least 4 days per week covering a total distance of at least 16 miles and did not perform high-intensity resistance exercise on a regular basis. The participants were screened for medical conditions, use of prescription pain and psychiatric medication, and any condition that would preclude performance of strenuous running and resistance exercise. Instructions were given to participants to limit alcohol use and physical activity during the study and to refrain from the use of anti-inflammatory medication or any rehabilitative modality (ice, massage, heat, etc.) that might affect sensations of pain/soreness and affect recovery of exercise performance. Compliance with these instructions was confirmed by a questionnaire completed prior to each testing session. Participants were also asked to refrain from consuming caffeine 8 h before each testing session. The participants completed recall questionnaires before each testing session to indicate compliance with these instructions. The experimental methods were approved by a University Institutional Review Board, and written informed consent was obtained from each participant prior to testing. Two participants withdrew due to competition commitments and data from one female was removed from analysis due to a failure to demonstrate a clear loss of isometric force (indicating EIMD may not have occurred)—leaving the total number of participants analyzed to eight (3 women, 5 men). A sample of 8 was sufficient to detect a moderate (≥0.40 SD) difference in $\dot{V}O_{2peak}$ using a repeated-measures ANOVA at an alpha level of 0.05 and a power of 0.80, assuming a correlation between repeated trials of ≥0.95. Powering the study in order to detect an effect of this magnitude was based upon previous findings from our lab (Black and Dobson, 2012) where $\dot{V}O_{2peak}$ was reduced ~0.43 SD 48-h following EIMD.

2.2. Experimental approach

This study used a within-participant, repeated measures design to determine the effects of EIMD on metabolic and perceptual responses during exercise and the effects of EIMD on muscle contractile properties. A control group was not used as $\dot{V}O_2$ peak has been shown to be highly reliable between testing sessions (Katch et al., 1982). Participants completed 5 or 6 testing sessions over the course of 10 days, with all testing sessions occurring at a similar time of day ±2 h. Three familiarization sessions were completed prior to the initial experimental testing session. During these sessions, participants were familiarized with the MVC and interpolated twitch protocol as well as running on a treadmill while wearing the equipment for assessing $\dot{V}O_{2peak}$. On experimental test day 1, MVC and muscle contractile properties were assessed followed by a $\dot{V}O_{2peak}$ test. On test day 2 (48 h later) participants performed high-intensity eccentric exercise (front loaded split squats) to induce EIMD. Immediately following eccentric exercise, MVC and muscle contractile properties were assessed followed by an assessment of $\dot{V}O_{2peak}$. Subsequently, all measures were retested 2, 4, and 7 days after the induction of EIMD. If each participant's $\dot{V}O_{2peak}$ had not returned to baseline levels (defined as within ±2.1 ml kg min^{-1} (Taylor et al., 1955) of the value obtained during baseline testing) 7 days following EIMD three additional days of recovery were provided and participants were retested on the 10th day following EIMD.

2.3. Peak exercise tests

Peak exercise tests were performed on a computer-driven treadmill (Trackmaster TMX22; Full Vision; Newton, KS) and $\dot{V}_E$, $\dot{V}O_2$, $\dot{V}CO_2$, and RER were measured every 15 s via open-circuit spirometry (Parvomedics; Sandy, UT). Prior to testing, instructions were provided for using the 6–20 Borg RPE scale and a 0–10 muscle pain rating scale (Cook et al., 1997). During the test, participants performed a 5-min warm-up at a self-selected speed that equated to either a "light jog" (typically 5.0–6.0 mph) or brisk walk (4.0–4.5 mph). Following the warm-up period, the treadmill speed was increased to a self-selected "comfortable," but brisk running speed (which ranged from 6.0–9.0 mph). Running speed was held constant for the duration of the test and every two minutes the treadmill grade was increased by 2% until volitional exhaustion was reached. Strong verbal encouragement was provided throughout the test. All subsequent tests were performed using the same treadmill velocity and same timing of increase in treadmill grade. Oxygen and carbon dioxide analyzers were calibrated before each measurement with known gas concentrations and a flow meter calibration was performed using a 3-L syringe. $\dot{V}O_2$ and $\dot{V}CO_2$ were standardized to standard temperature and pressure dry (STPD). Heart rate (HR) was continuously measured during the test (Polar Electro Oy, Kempele, Finland). RPE and muscle pain were reported during the last 10 s of each minute and blood lactate (La) was measured via finger stick 2 min after the test ended (Lactate Plus, Nova Biomedical, Waltham, MA). Gas exchange threshold (GET) was determined from visual inspection of plots of the following measures: (1) the initial disproportionate increase in $\dot{V}CO_2$ when $\dot{V}CO_2$ was graphed against $\dot{V}O_2$ (i.e., the V-slope method) and (2) an increase in $\dot{V}_E/\dot{V}O_2$ when graphed against $\dot{V}O_2$ without a corresponding increase in $\dot{V}_E/\dot{V}CO_2$ (Beaver et al., 1986). Each set of plots were evaluated by two investigators blinded to the participant and testing time point. $\dot{V}O_{2peak}$ was defined by a plateau in $\dot{V}O_2$ (change of <2.1 ml kg^{-1} min^{-1}) (Taylor et al., 1955) with an increase in work rate or the attainment of three of the following four criteria: RER ≥ 1.1, peak HR within 10 bpm of age-predicted maximum, La ≥ 8mmol, and an RPE of ≥ 18.