Effect of creatine loading on neuromuscular fatigue threshold

JEFFREY STOUT,1 JOAN ECKERSON,1 KYLE EBERSOLE,2 GERI MOORE,1 SHARON PERRY,2 TERRY HOUSH,2 ANTHONY BULL,2 J OEL CRAMER,2 AND ASH BATHEJA3

1Exercise Science Department, Creighton University, Omaha 68178; 2Center for Youth Fitness and Sports Research, University of Nebraska, Lincoln 68588; and 3Department of Physical Therapy, University of Nebraska Medical Center, Omaha, Nebraska 68198

A NUMBER OF INVESTIGATIONS have used surface electromyographic (EMG) procedures to identify the power output associated with the onset of neuromuscular fatigue (NMF) during cycle ergometry (3, 4, 7–9, 14, 21). NMF is typically characterized by an increase over time in the electrical activity of the working muscles (2, 4, 14, 15). Moritani et al. (15) suggested that the fatigue-induced increase in EMG amplitude is a result of progressive recruitment of additional motor units (MU) and/or an increase in the firing frequency of MUs that have already been recruited. Theoretically, work bouts at power outputs at or below the NMF threshold can be maintained continuously without EMG evidence of fatigue (i.e., no significant increase in EMG amplitude over time).

DeVries et al. (3, 4) developed an incremental cycle ergometer test called “the physical working capacity at the fatigue threshold” (PWCFT), which utilizes EMG fatigue curves to identify the power output that corresponds to the onset of the NMF threshold. The PWCFT represents the highest power output that results in a nonsignificant (P > 0.05) increase in the electrical activity of the thigh muscles over time. Whereas the PWCFT test has been shown to be reliable (2, 4), valid (2), and sensitive to changes in fitness level (2), the physiological mechanism responsible for the increase in EMG amplitude over time during a fatiguing task is unknown. Two potential mechanisms, however, include the accumulation of metabolic by-products (lactate, H+, Pi, and ammonia) and/or the depletion of stored energy substrates [ATP, phosphocreatine (PCr), and glycogen] (13). Housh et al. (8, 9) have reported that manipulation of blood acid-base balance with ammonia chloride and sodium bicarbonate, as well as glycogen depletion and supercompensation, did not affect the onset of NMF as measured by the PWCFT test. However, McCartney et al. (12) have suggested that “alterations in the blood acid-base state have little influence on muscle pH.” In addition, there is evidence to suggest that skeletal muscle PCr may serve as a temporal energy buffer as well as a modulator of glycolysis and, therefore, may influence NMF (22). The effect of PCr manipulation on EMG fatigue curves, however, is unknown. Therefore, the purpose of the present study was to determine the effect of Cr loading on the onset of NMF, as measured by the PWCFT test in women athletes.

METHODS

Subjects. Fifteen female members of the university crew team [age 19.0 ± 2.0 (SD) yr] volunteered as subjects for this investigation. All procedures were approved by the Institutional Review Board before the initiation of the study, and each subject was advised of any possible risks before providing informed consent.

Supplementation protocol. None of the subjects had ingested Cr, or any other dietary supplements, for a minimum of 12 wk before the initiation of the study. During the course of the study, the subjects were asked to maintain their current dietary patterns and abstain from other nutritional supplements, nonprescription drugs, and caffeine. After pretesting, the subjects were randomly assigned to one of two treatment conditions using a double-blind design: 1) 20 g of flavored dextrose powder as a placebo (PI, n = 8); or 2) 5.0 g of Cr monohydrate plus 20 g of dextrose in a flavored powder blend (Cr, n = 7) (Creatine Edge Effervescent, Fortress Systems, Omaha, NE). The powders, identical in taste and appearance, were dissolved in 16 oz of water and ingested four times per day for 5 consecutive days before posttesting.

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
Electrode placement and EMG instrumentation. A bipolar (2.54-cm center-to-center) surface electrode (Quinton Quick prep silver-silver chloride) arrangement was placed on the right thigh over the lateral portion of the vastus lateralis (VL), midway between the greater trochanter and the lateral condyle of the femur. The reference electrode was placed over the iliac crest. Interelectrode impedance was kept below 2,000 $\Omega$ by careful abrasion of the skin. The EMG signal was preamplified (gain: $3 \times 1,000$) by using a differential amplifier (EMG 100, Biopac System, Santa Barbara, CA). The EMG signal was sampled at 1,000 points/s and filtered at 10–500 Hz. The root mean square EMG amplitude values were calculated for the 10-s time frame for each sample taken (MP100, Biopac Systems).

Determination of PWC$\text{FT}$ . The PWC$\text{FT}$ values were determined from the VL muscle by using the protocol of deVries et al. (3). Figure 1 illustrates how the PWC$\text{FT}$ was determined using the data from subject 7 in the Cr group (Table 1). The subjects began pedaling (with toe clips) at 60 W (70 rpm) on a calibrated, electronically braked cycle ergometer (Corval 400, Quinton Instruments, Seattle, WA). The power output was then increased by 30 W every 2 min until the subject could no longer maintain 70 rpm. During each 2-min interval, six 10-s EMG samples were recorded from the VL. The PWC$\text{FT}$ was determined by averaging the highest power output that resulted in a nonsignificant ($P > 0.05$; single-tailed $t$-test) slope value for the EMG amplitude vs. time relationship, with the lowest power output that resulted in a significant ($P < 0.05$) slope value (Fig. 1).

Reliability of the PWC$\text{FT}$ was determined by using a subsample of subjects ($n = 11$) measured 7 days apart. The test-retest intraclass correlation coefficient (R) was 0.94 (SE $\pm 6$ W), which is similar to values reported by deVries et al. (2, 3) in older ($R = 0.976$) and younger male subjects ($R = 0.947$). In addition, the test-retest mean difference for the PWC$\text{FT}$ values 0.5 W was not statistically significant ($t = 0.09; P > 0.05$).

Statistical analysis. Changes in body weight (BW) as a result of supplementation were analyzed by using a $2 \times 3 \times 2$ [treatment (Pl, Cr) $\times$ time (pretest, postest)] mixed factorial ANOVA. Differences in the mean posttest PWC$\text{FT}$ value were determined by using analysis of covariance, with pretest PWC$\text{FT}$ serving as the covariate. Data were considered significantly different when the probability was $P \leq 0.05$.

RESULTS

The descriptive characteristics of the subjects, as well as the changes in BW and PWC$\text{FT}$ for the two groups, are shown in Table 1. There were no significant changes in BW from pretesting to posttesting for either group. However, the adjusted mean posttest PWC$\text{FT}$ value for the PI group (mean $= 155$ W) was significantly less than that of the Cr group (mean $= 186$ W).

Table 1. Characteristics of the subjects ($n = 15$)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>BW-Pre, kg</th>
<th>BW-Post, kg</th>
<th>PWC$\text{FT}$-Pre, W</th>
<th>PWC$\text{FT}$-Post, W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>18</td>
<td>163</td>
<td>75.5</td>
<td>75.0</td>
<td>165</td>
<td>135</td>
</tr>
<tr>
<td>2</td>
<td>19</td>
<td>165</td>
<td>41.4</td>
<td>51.8</td>
<td>135</td>
<td>135</td>
</tr>
<tr>
<td>3</td>
<td>17</td>
<td>167</td>
<td>56.4</td>
<td>56.0</td>
<td>135</td>
<td>135</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>170</td>
<td>59.0</td>
<td>59.6</td>
<td>135</td>
<td>135</td>
</tr>
<tr>
<td>5</td>
<td>17</td>
<td>164</td>
<td>80.5</td>
<td>81.8</td>
<td>135</td>
<td>135</td>
</tr>
<tr>
<td>6</td>
<td>19</td>
<td>160</td>
<td>60.5</td>
<td>59.0</td>
<td>105</td>
<td>105</td>
</tr>
<tr>
<td>7</td>
<td>19</td>
<td>163</td>
<td>56.0</td>
<td>56.0</td>
<td>105</td>
<td>105</td>
</tr>
<tr>
<td>Mean $\pm$ SD</td>
<td>$19.0 \pm 1.2$</td>
<td>$164.9 \pm 3.1$</td>
<td>$63.7 \pm 10.4$</td>
<td>$63.6 \pm 10.6$</td>
<td>$146.3 \pm 22.3$</td>
<td>$146.3 \pm 22.3$</td>
</tr>
<tr>
<td>Creatine group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>17</td>
<td>172</td>
<td>74.3</td>
<td>76.4</td>
<td>165</td>
<td>225</td>
</tr>
<tr>
<td>2</td>
<td>21</td>
<td>154</td>
<td>66.7</td>
<td>68.2</td>
<td>165</td>
<td>195</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>163</td>
<td>67.3</td>
<td>65.0</td>
<td>195</td>
<td>225</td>
</tr>
<tr>
<td>4</td>
<td>22</td>
<td>159</td>
<td>64.0</td>
<td>64.5</td>
<td>165</td>
<td>195</td>
</tr>
<tr>
<td>5</td>
<td>18</td>
<td>166</td>
<td>78.9</td>
<td>78.7</td>
<td>225</td>
<td>225</td>
</tr>
<tr>
<td>6</td>
<td>18</td>
<td>167</td>
<td>62.7</td>
<td>63.4</td>
<td>165</td>
<td>165</td>
</tr>
<tr>
<td>7</td>
<td>20</td>
<td>178</td>
<td>65.5</td>
<td>64.7</td>
<td>105</td>
<td>135</td>
</tr>
<tr>
<td>Mean $\pm$ SD</td>
<td>$19.4 \pm 1.8$</td>
<td>$165.6 \pm 8.0$</td>
<td>$68.5 \pm 5.9$</td>
<td>$68.7 \pm 6.3$</td>
<td>$169.3 \pm 36.4$</td>
<td>$195* \pm 34.6$</td>
</tr>
</tbody>
</table>

PWC$\text{FT}$, physical working capacity at fatigue threshold; BW, body weight; Pre, before treatment; Post, after treatment. *Mean PWC$\text{FT}$-Post values significantly different from mean PWC$\text{FT}$-Pre values ($P < 0.05$).
Intra- and extracellular ammonia, may be responsible for fatigue-induced increases in MU recruitment and the corresponding increase in EMG amplitude. In agreement, Taylor et al. (19) also found that, for incremental cycle ergometry, the accumulation of plasma lactate and ammonia was associated with an increase in EMG amplitude measured from the rectus femoris muscle. Therefore, there is evidence to suggest that a reliance on anaerobic glycolysis leads to an increase in EMG amplitude from the working muscles as a result of changes in muscle and blood lactate levels and the corresponding decrease in pH.

In the present study, Cr loading resulted in a delay in the onset of NMFF (as measured by the PWCFT test), which may have been due to the effect of elevated muscle PCr on the transition from aerobic to anaerobic metabolism. Prevost et al. (17) and Volek and Kraemer (22) have hypothesized that increasing muscle PCr content by Cr loading may decrease the reliance on anaerobic glycolysis, reduce intramuscular lactate accumulation, and, therefore, delay the onset of fatigue. Thus the results of the present study suggest that during incremental cycle ergometry Cr loading may delay the onset of NMFF and the fatigue-induced increase in EMG at submaximal power outputs by reducing the reliance on anaerobic glycolysis and attenuating the accumulation of lactate and ammonia in the working muscles and blood.

In summary, Cr loading resulted in a significantly higher PWCFT value (186 W) compared with a PI (155 W), indicating that Cr loading may delay the onset of NMFF during incremental cycle ergometry in female athletes. The delay in NMFF may have been due to augmented PCr levels in the muscle, which may have resulted in a greater capacity to delay anaerobic glycolysis (16, 17, 22). Future studies that would directly measure muscle PCr, lactate, and ammonium levels are warranted to validate these results.

We thank Fortress International (Omaha, NE) for funding this study.

Address for reprint requests and other correspondence: J. R. Stout, Creighton Univ., Exercise Science Dept., 2500 California Pl., Omaha, NE 68178 (E-mail: jrstout@creighton.edu).

Received 14 May 1999; accepted in final form 31 August 1999.

REFERENCES

5. Febbraio, M., T. Flanagan, R. Snow, S. Zhao, and M. Carey. Effect of creatine supplementation on intramuscular TCr, metabo-