Effects of creatine supplementation on the energy cost of muscle contraction: a $^{31}$P-MRS study

SINCLAIR A. SMITH,1 SCOTT J. MONTAIN,2 RALPH P. MATOTT,2 GARY P. ZIENTARA,3 FERENC A. J OLESZ,3 AND ROGER A. FIELDING1

1Department of Health Sciences, Sargent College of Health and Rehabilitation Sciences, Boston University, Boston 02215; 2United States Army Research Institute of Environmental Medicine, Natick 01760; and 3Department of Radiology, Brigham and Women’s Hospital and Harvard Medical School, Boston, Massachusetts 02115


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Dietary creatine supplementation (0.3 g·kg$^{-1}$·day$^{-1}$ for 5 days) has been shown to increase muscle phosphocreatine (PCr) concentration and hydrolysis (31) and improve muscle performance during intermittent high-intensity exercise (1, 3, 6, 14, 20–22). Although PCr availability and use are enhanced throughout intermittent exercise bouts, few studies report improvements in performance during initial intermittent bouts or during single bouts of exercise after supplementation (2, 11, 15, 16, 30, 32, 33, 39). A potential explanation for these findings may be that creatine supplementation alters the energy cost of muscle force production (ATP cost of contraction) at the onset of exercise by increasing muscle PCr concentration.

The ATP cost of contraction varies depending on muscle fiber type, substrate availability, and contraction frequency and duration. Muscle ATP costs have been found to be higher at the onset of contraction (9, 31), increase as contraction frequency increases, and decrease as contraction duration increases (4, 10, 13, 36, 37), suggesting that muscle excitation-relaxation processes influence the ATP cost of muscle contraction. In addition, human and animal studies have reported that fast-twitch (type II) muscle fibers have higher PCr concentrations and hydrolysis rates than slow-twitch (type I) fibers (5, 28) and a greater ATP cost of contraction (8, 12, 24, 34). Conversely, a depletion of ATP and PCr stores in rat muscle has been shown to reduce the cost of contraction (17, 18). These findings suggest that there may be a positive relationship between muscle PCr concentration and the cost of contraction. The effect of increasing muscle PCr via creatine supplementation on the ATP cost of contraction, however, has not been investigated. A study of this design may further define the degree of dependence between muscle PCr concentration and cost of contraction and assist in determining possible mechanisms responsible for the variations observed in muscle cost of contraction.

The purpose of this study was to determine the effects of creatine supplementation and exercise on skeletal muscle ATP costs of contraction by measuring energy expenditure during brief static and dynamic exercise bouts performed before and after exhaustive exercise. In addition, the effects of creatine supplementation on pH, phosphagen kinetics, and muscle endurance were investigated. Intramuscular pH and phosphagen compounds were measured noninvasively throughout exercise and recovery by using $^{31}$P-magnetic resonance spectroscopy (MRS), which greatly enhances measurement resolution over muscle biopsy techniques (29). We hypothesized that 1) creatine supplementation would influence muscle ATP cost of contraction by increasing PCr availability, 2) muscle cost of contraction would decline after exercise, and 3) creatine supplementation would improve muscle endurance. The possible association of PCr creatine supplementation and cost of muscle contraction has yet to be investigated.

Methods

Subjects. Six women and three men participated in the study (Table 1). All subjects were physically active, free from chronic disease, and used no regular medications, as determined by a medical history questionnaire. The study was approved by the appropriate institutional review boards, and all subjects gave their voluntary and informed consent before participation.
Creatine supplementation. Two single-blind exercise trials were performed: a placebo trial, which was followed by a creatine trial 7–14 days later. The trials were not randomized, as skeletal muscle creatine levels can remain elevated above basal levels for 4–5 wk after supplementation stops (23). Five days before each trial, the subjects began consuming 0.3 g·kg$^{-1}$·day$^{-1}$ of either a placebo (granulated sugar) or 0.3 g·kg$^{-1}$·day$^{-1}$ of creatine monohydrate (Phosphagen, Experimental and Applied Sciences, Pacific Grove, CA) combined with 0.3 g·kg$^{-1}$·day$^{-1}$ of a flavored powder drink mix. The relative creatine dosage was determined by using a mean body weight of 70 kg at a dose of 20 g/day (22, 23). The mixture was dissolved in water and consumed four times per day. The subjects were blinded as to whether they were receiving the placebo or creatine, and the mixtures were similar in taste, texture, and color.

Exercise. Both groups performed single-leg knee-extension exercise to exhaustion while lying supine inside a whole body 1.5-T magnetic resonance system (General Electric SIGNA, General Electric Medical Systems, Milwaukee, WI). Exhaustion was defined as the time when the subject could not maintain the rate and/or range of motion after being given verbal encouragement by the investigators. The exercise apparatus provided concentric resistance via a lever arm-and-pulley system integrated with a flywheel and resistance strap, as illustrated elsewhere (35). An elastic cord returned the lever arm to the starting position after each knee extension. Knee extensions were performed from ~110 to ~145° of knee extension at 37 contractions/min set by an audible metronome. Power output during exercise was determined by measuring the velocity and tension applied to an in-line pulley and by estimating leg mass (41). Both legs were tested in each experimental condition.

Before the experimental trials, two to three exercise practice sessions were performed to familiarize the subjects with the experimental procedures and to determine the appropriate exercise intensity. The maximum flywheel resistance at which each subject was able to perform 3–4 min of exercise was used. Therefore, the resistance for each subject was dependent on the subject’s muscle endurance capacity. Between the placebo and creatine trials, however, the resistance was kept constant for each subject.

Before the exhaustive exercise bouts (preexercise) and at select intervals during recovery, measurements of ATP cost of contraction were obtained during static or dynamic contractions. The arrows in Figs. 1 and 2 illustrate the preexercise and recovery contraction sequence. To determine the quantity of muscle ATP use per joule of work performed, the subjects performed six dynamic knee extensions (~8 s) with their left leg at 100 and 50 s before exhaustive exercise, at 30 s of recovery, and every minute thereafter through 5 min of recovery. The resistance and cadence for these brief dynamic bouts were the same as those used during the exhaustive

### Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Subjects</th>
<th>n</th>
<th>Age, yr</th>
<th>Placebo Weight, kg</th>
<th>Creatine Weight, kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women</td>
<td>6</td>
<td>28 ± 1.4</td>
<td>57.0 ± 1.2</td>
<td>57.0 ± 1.3</td>
</tr>
<tr>
<td>Men</td>
<td>3</td>
<td>33 ± 2.1</td>
<td>68.8 ± 4.8</td>
<td>69.0 ± 5.7</td>
</tr>
</tbody>
</table>

Values are means ± SE.
exercise bout. To determine the quantity of ATP use per newton of force generated, the subjects performed a static 5-s maximal voluntary contraction (MVC) with their right leg at \(110^\circ\) of knee extension at 100 and 50 s before exhaustive exercise, every 30 s of recovery for 2 min, and every minute thereafter through 5 min of recovery. In addition, the time to recover to 75% of the mean preexercise MVC value was determined. The preexercise and recovery knee extensions and MVCs were performed within a 10-s \(^{31}\)P-MRS sampling period.

\(^{31}\)P-MRS. \(^{31}\)P spectra were collected continuously from rest throughout exercise and recovery by a \(^1\)H/\(^{31}\)P dual radio-
frequency transmit/receive 11-cm surface coil (USAsia, Columbia, OH) placed over the quadriceps muscles. 31P data were acquired by using a hard-pulse 25.85-MHz excitation (pulse width 600 μs), repetition time 1,000 ms, spectral width 2,000 Hz, and 1,024 sampled free induction decay (FID) points. Before exercise, a proton magnetic resonance image was acquired axially by using the H1/31P surface coil to verify coil placement and muscle group participation. A linear gradient shim procedure was performed to reduce field inhomogeneity within the sensitive volume. Surface coil transmitter and receiver gains for 31P-MRS were set once to maximize PCR signal acquired from the muscle and kept constant throughout the study. Ten FID signals were averaged producing one spectrum every 10 s. Care was taken to ensure that exercise began and ended at the onset of an FID cycle. The magnetic resonance system was calibrated by using known standards on each testing day.

FID processing consisted of apodization of 10-Hz line broadening, zero-filling to 4,096 points and Fourier transformation, followed by zero- and first-order phasing. Relative concentrations of Pγ, PCR, and βATP were determined from spectral peak areas. PCR/βATPrest, Pγ/βATPrest, and βATP/βATPrest ratios were converted to millimoles per kilogram wet weight by assuming that the area of βATPrest was equivalent to 5.5 mmol·kg wet wt−1·s−1 (37). βATPrest was the mean area of the two initial resting βATP peaks. Finally, pH was calculated by using the chemical shift between the Pγ and PCR frequency (38).

The rate of PCR resynthesis and a time constant (τ) were determined from a monoexponential curve fit to the PCR recovery data after exhaustive exercise (25–27, 40). The following monoexponential equation was used: y = a[1 – exp(bx)] + c, where y represents the PCR value at any given time x, a is the change in PCR during recovery, b is the rate constant (1/τ = τ), and c is the initial PCR value at the onset of recovery. The values for initial PCR resynthesis rate were determined from the slope of the initial 10 s of the monoexponential curve fit (24, 38). Before fitting, the PCR data were modified, eliminating two data points after each knee extension or MVC bout during recovery, as indicated by a in Fig. 3. A pilot study determined that this procedure provided recovery results comparable to those obtained without intermittent dynamic and static exercise (n = 4, r = 0.92, P < 0.01). The quantity of PCR hydrolysis (ΔPCR) was determined for preexercise and recovery contractions. To account for the ongoing resynthesis of PCR during recovery, the quantity of PCR resynthesized during each brief bout was derived from the monoexponential curve and added to the actual change in PCR to determine the total ΔPCR as illustrated in Fig. 3. For each subsequent dynamic and static bout, the monoexponential curve was shifted 20 s to the right, and ΔPCR was calculated. The total PCR hydrolysis during the exhaustive exercise bout was determined as the change in PCR from rest to the end of exhaustive exercise.

ATP cost of contraction during the brief static and dynamic bouts was determined by calculating ATP use derived from PCR hydrolysis (7, 31) and by measuring the force or work produced during the contractions. For bouts of <10 s, the change in PCr has been shown to accurately reflect ATP use (19). Potential production of ATP from anaerobic glycolysis was monitored via the changes in muscle pH (31), and mitochondrial oxidative phosphorylation has been shown to be delayed ~10 s from the onset of exercise (7, 19). The ATP cost of contraction was represented as the millimoles per kilogram of ATP used per newtons of force produced multiplied by seconds (ΔATP/N; mmol·kg·N·s−1) for the brief static bouts and as the millimoles per kilogram of ATP used per joule of work produced (ΔATP/J; mmol·kg−1·J−1) for the brief dynamic bouts. During the final 10 s of exhaustive dynamic exercise, ATP cost of contraction was determined by calculating ATP use derived from PCR, glycogen, and oxidative phosphorylation (31).

Analysis. For variables pertaining to the exhaustive exercise bouts (ΔPCR, initial PCR resynthesis rate, Trec, mean power output, and time to exhaustion), repeated-measures ANOVA tests were used with treatment (creatine vs. placebo) and leg (right vs. left) as factors. For variables pertaining to the preexercise and recovery dynamic and static contractions (PCR, Pγ, ATP, ΔATP/N, ΔATP/J, and peak MVC), repeated-measures ANOVA tests were used with treatment (creatine vs. placebo) and time (preexercise and recovery bouts) as factors. Newman-Kuels post hoc tests were used to determine mean differences between and within factors. A paired t-test was used to determine mean differences in body weight and time to 75% MVC recovery between the placebo and creatine trials. The significance level was set at P < 0.05 for all tests, and results are presented as means ± SE.

RESULTS

Creatine supplementation. Figures 1 and 2 illustrate the relationship between PCR and pH kinetics during the placebo and creatine trials for the static (5-s MVCs) and dynamic (6 extensions) exercise protocols. Tables 2 and 3 contain phosphagen kinetic and muscle performance results obtained during exhaustive exercise and

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**Table 2. Muscle PCR, Pγ, ATP, and pH results**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Trial</th>
<th>Rest</th>
<th>End Exercise</th>
<th>Recovery (3 min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCR, mmol/kg</td>
<td>Placebo</td>
<td>40.7±1.8</td>
<td>11.7±2.0</td>
<td>31.6±1.6</td>
</tr>
<tr>
<td>Pγ, mmol/kg</td>
<td>Placebo</td>
<td>46.6±1.1</td>
<td>11.1±2.1</td>
<td>34.7±1.5</td>
</tr>
<tr>
<td>ATP, mmol/kg</td>
<td>Placebo</td>
<td>5.5</td>
<td>3.97±0.24</td>
<td>4.51±0.27</td>
</tr>
<tr>
<td>pH</td>
<td>Placebo</td>
<td>7.13±0.07</td>
<td>6.46±0.07</td>
<td>6.70±0.07</td>
</tr>
<tr>
<td>Creatine</td>
<td>5.5</td>
<td>4.45±0.27</td>
<td>4.91±0.20</td>
<td></td>
</tr>
<tr>
<td>Creatine</td>
<td>7.12±0.07</td>
<td>6.49±0.07</td>
<td>6.45±0.07</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE. Placebo and creatine trial results for phosphocreatine (PCR), Pγ, ATP, and pH for subjects’ right and left quadriceps at rest, at the end of exhaustive exercise, and after 3 min of recovery. There were no differences or interactions with regard to leg, as indicated by repeated-measures ANOVA. *Significant difference from control placebo value (P = 0.03).

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**Table 3. Muscle performance and PCR metabolic results**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Placebo</th>
<th>Creatine</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean power, W</td>
<td>17.9±0.5</td>
<td>18.0±0.5</td>
<td>0.98</td>
</tr>
<tr>
<td>ΔPCR, mmol/kg wet wt</td>
<td>29.6±2.4</td>
<td>34.1±2.8</td>
<td>0.02</td>
</tr>
<tr>
<td>Pγrate, mmol·kg−1·min−1</td>
<td>27.0±1.8</td>
<td>32.1±2.0</td>
<td>0.19</td>
</tr>
<tr>
<td>Trec, s</td>
<td>49.1±3.6</td>
<td>46.6±3.7</td>
<td>0.66</td>
</tr>
<tr>
<td>Time to exhaustion, s</td>
<td>268±17</td>
<td>286±20</td>
<td>0.25</td>
</tr>
<tr>
<td>Time to 75% MVC recovery, s</td>
<td>63.3±17.6</td>
<td>82.5±25.8</td>
<td>0.71</td>
</tr>
</tbody>
</table>

Values are means ± SE. Results for mean power output during exhaustive exercise, PCR hydrolysis (ΔPCR), initial PCR resynthesis rate (Pγrate; mmol·kg wet wt−1·min−1), PCR resynthesis time constant (Trec), time to exhaustion, and time to 75% maximal voluntary contraction (MVC) recovery for placebo and creatine trials. Data from both quadriceps were included. There were no differences or interactions with regard to leg, as indicated by repeated-measures ANOVA.
include bilateral data for each subject. There were no

differences or interactions with regard to right vs. left
leg for any of these results, as indicated by the repeated-
measures ANOVA. After creatine supplementation, rest-
ing muscle PCr was increased by 15% ($P < 0.04$, Table
2), and total PCr hydrolysis during the exhaustive
exercise bout was increased by 13% ($P < 0.02$, Table 3)
while the mean exercise power output remained con-
stant (Table 3). During recovery, the initial PCr resyn-
thesis rate and $T_c$ were not significantly affected by
creatine (Table 3). ATP levels declined during exercise
($P < 0.01$) and tended to be greater overall after
creatine supplementation ($P = 0.08$); however, there
were no interactions between time and treatment fac-
 tors (Table 2). There were no differences in $P_i$ and pH
kinetics after creatine supplementation (Table 2). There
was, however, a consistent increase in pH and reduc-
tion in PCr values ($P < 0.01$) as a result of the static
and dynamic bouts performed during preexercise and recov-
ery, as illustrated in Figs. 1 and 2. The duration of
recovery data collection varied depending on each
subject’s time to exhaustion. After 180 s of recovery, the
subject number represented by the data begins to
decline, which causes some fluctuation in the PCr and
pH recovery data in Figs. 1 and 2.

Figures 4 and 5 illustrate the static, $\Delta$ATP/N, and
dynamic, $\Delta$ATP/J, ATP costs of contraction during
preexercise and recovery for the placebo and creatine
trials. Neither $\Delta$ATP/N nor $\Delta$ATP/J was significantly
affected by creatine supplementation ($P = 0.98$). ATP
was derived from PCr hydrolysis during preexercise,
initial 10 s of exhaustive exercise, and recovery. Measur-
able contributions from glycolysis and oxidative phos-
phorylation were not detected, which is consistent with
previous studies (7, 19). In contrast, most of the ATP
($>95\%$) utilized during the final 10 s of exhaustive
exercise (Fig. 6) was derived from oxidative phosphory-
lation. The ATP cost of contraction calculated at the end
of exhaustive exercise (Fig. 6) was not different from
ATP costs calculated during dynamic recovery derived
from $\Delta$PCr (Fig. 5). Creatine supplementation, how-
ever, increased the ATP cost of contraction during the
final 10 s of exhaustive exercise compared with the
placebo value (Fig. 6).

The indicators of muscle performance, time to exhaus-
tion, time to 75% MVC recovery, and peak MVC, illus-
trated in Table 2 and Fig. 7, were not significantly
influenced by creatine supplementation. All the sub-
jects were verbally encouraged by the investigators
during exercise and appeared to give their maximal
effort. The work performed during the preexercise and
recovery six extension dynamic bouts (not shown) was
consistent throughout both trials.

Exhaustive exercise. The ATP cost of contraction for
the brief static and dynamic bouts declines after the
initial preexercise bout. The initial preexercise $\Delta$ATP/N
value at $-100$ s was greater than the recovery values
for 30 through 240 s ($P < 0.01$, Fig. 4). Similarly, the
initial preexercise $\Delta$ATP/J at $-100$ s was greater than
the preexercise $-50$ s value and all the $\Delta$ATP/J recov-
ery values ($P < 0.01$, Fig. 5). These results indicate that
during the initial brief dynamic and static exercise
bouts the quadriceps muscles had a higher ATP cost of
contraction compared with subsequent ATP costs mea-
sured during preexercise and during recovery from
exhaustive exercise.

Fig. 4. ATP cost of contraction per N·s
of force produced ($\Delta$ATP/N; mmol·kg wet wt
$^{-1}$·N·s$^{-1}$) for 5-s MVC bouts
performed before (preexercise) exhaustive
exercise and during recovery. Values are
means ± SE. *Significant difference from
preexercise $-100$-s bout for placebo and
creatine results ($P < 0.01$).

Fig. 5. ATP cost of contraction per J
of work produced ($\Delta$ATP/J; mmol·kg
wet wt $^{-1}$·J$^{-1}$) for bouts of 6 dynamic knee
extensions performed before (preexer-
cise) exhaustive exercise and during
recovery. Values are means ± SE. *Signif-
ificant difference from preexercise
$-100$-s bout for placebo and creatine
results ($P < 0.01$).
DISCUSSION

This study investigated the effects of oral creatine supplementation and exhaustive exercise on muscle ATP cost of contraction. In addition, the effects of creatine supplementation on muscle phosphagen metabolism, pH, and endurance were examined. \(^{31}\)P-MRS was used to measure quadriceps muscle PCr, P\(_i\), ATP, and pH throughout exercise and recovery during a placebo and creatine trial. Muscle ATP cost of contraction was measured during 5-s static and 8-s dynamic exercise bouts performed before and after exhaustive exercise and derived from the \(\Delta\)ATP and force output of the muscle. The brief static and dynamic bouts were used to minimize ATP production by glycolytic and oxidative systems, so that PCr hydrolysis would reflect ATP use by contracting muscle fibers (7, 19). We hypothesized that 1) creatine supplementation would increase muscle ATP cost of contraction by increasing PCr availability and utilization, 2) the muscle cost of contraction would be reduced after exhaustive exercise, and 3) creatine supplementation would improve muscle performance.

Creatine supplementation. Our results show a 15% increase in resting muscle PCr and a 13% increase in total PCr hydrolysis during exhaustive exercise after creatine supplementation (Tables 2 and 3), which is consistent with previous results (35). However, the increase in PCr availability and overall use brought about by creatine supplementation did not affect muscle ATP cost of contraction calculated from brief static and dynamic exercise bouts (Figs. 4 and 5). Studies have reported that fast-twitch muscle fibers have greater resting PCr levels, PCr hydrolysis rates, and ATP cost of contraction than slow-twitch fibers (5, 8, 12, 24, 28, 34). Conversely, a depletion of ATP and PCr stores in rat muscle by hadacidin, an adenylosuccinate synthase inhibitor, has been shown to reduce the cost of contraction (17, 18). Furthermore, PCr levels and ATP cost of contraction differ widely among individuals (8). Given that no change was observed in ATP cost of contraction after creatine supplementation, our results suggest that PCr availability and utilization do not appear to be associated with differences in ATP cost of contraction observed across muscle fiber types and individuals. Other factors, such as cross-bridge cycling rate and cost of excitation and relaxation processes, inherent to muscle fiber types, most likely account for the differences observed in the cost of contraction.

Creatine supplementation increased ATP costs of contraction in the last 10 s of exhaustive dynamic exercise (Fig. 6). However, ATP hydrolysis during this 10-s period was predicted primarily from the initial PCr resynthesis rate during recovery, which was found to be increased by creatine supplementation in middle-aged persons (35). Although the initial PCr resynthesis rate was not significantly influenced by creatine supplementation in this study (Table 3), nonsignificant increases in PCr resynthesis rate may account for the difference in ATP cost of contraction. This raises some question as to the validity of using PCr resynthesis rate to predict ATP production from oxidative phosphorylation when muscle creatine is manipulated by creatine supplementation. That is, the change in ATP production after creatine supplementation calculated from the PCr resynthesis rate may have resulted from changes in PCr recovery kinetics, not from changes in mitochondrial oxidative capacity, as the calculation was intended to measure (9, 31). Further study is required to elucidate the relationship between changes in PCr concentration after creatine supplementation and PCr resynthesis rate.
Creatine supplementation tended to spare ATP stores (P = 0.08, Table 2) during exhaustive exercise, which is consistent with previously reported trends (3, 16). However, the muscle performance indicators, peak MVC force (Fig. 7), time to exhaustion, and time to 75% MVC recovery (Table 3) were not significantly influenced. Although creatine supplementation increased PCr concentration by 15%, one-half of the total PCR hydrolyzed during the ~277-s exhaustive exercise bouts was consumed in the first 30 s (Figs. 1 and 2), indicating that muscle ATP requirements were supplied primarily from glycolytic and oxidative systems. While studies have shown that creatine supplementation improves brief intermittent exercise performance (1, 3, 6, 14, 20–22), most findings indicate that creatine does not significantly improve single-bout exercise performances as in this study (2, 11, 15, 16, 30, 32, 33, 39).

Exhaustive exercise. Figures 4 and 5 illustrate that the ATP cost of contraction for brief static (i.e., ΔATP/N) and dynamic (i.e., ΔATP/J) exercises declined after the initial bout at ~100 s preexercise and was less after exhaustive exercise. Our results are consistent with studies in which ATP cost of contraction during continuous and intermittent static contractions of the gastrocnemius/soleus muscles was reported to decline after the onset of a contraction sequence (9, 31). In addition, studies comparing static intermittent and continuous exercise of the quadriceps muscles report higher ATP cost of contraction and fatigue rates during intermittent vs. continuous contractions (4, 10, 37). The results of these studies and ours suggest that changes in ATP use by excitation/relaxation mechanisms during the course of exercise may account for the differences in ATP cost of contraction.

During muscle contraction, there are several sites of ATP utilization, actomyosin cross bridges, Ca2+ pumps, Na+–K+ pumps, and phosphorylation processes that may influence the cost of contraction during preexercise bouts. At the onset of exercise, the Ca2+ flux across the sarcoplasmic membrane is greater than that during continuous exercise, which increases Ca2+ pump ATP consumption and has been suggested as a mechanism for the increased cost of contraction at the onset of exercise (4, 10, 37). In addition, the cross-bridge turnover rate, particularly for fast-twitch muscles, appears to be greater at the onset of a contraction, leading to a higher initial ATP consumption (8, 10, 24). It has been estimated that the total ATP costs for a 1-s quadriceps muscle contraction is 60% greater than the ATP cost during continuous contraction (4).

It has also been suggested that the higher ATP cost at the onset of exercise may be a result of underestimation of ATP costs later in exercise, when ATP for contraction is supplied predominantly from glycolytic and oxidative energy stores (9). In this study, the brief (<10 s) preexercise and recovery bouts were used to minimize the muscle ATP consumption from glycolytic and oxidative stores. Given the intensity and duration of these bouts, the ΔPCr should accurately predict ATP consumption by the muscle (7, 19). The pH values during preexercise and recovery increase after each bout, indicating H+ consumption by the creatine kinase reaction. Significant ATP generation by anaerobic glycolysis should mask this effect. Activation of oxidative phosphorylation has been shown to be delayed ~10 s from the onset of exercise (7, 19). Furthermore, there is consistency between cost of contraction values derived from ΔPCr at 30 s of recovery (Fig. 5) and values derived primarily from oxidative phosphorylation at the end of exhaustive exercise (Fig. 6).

Conclusion. This study investigated the effects of creatine supplementation and exhaustive exercise on ATP cost of contraction and the effects of creatine supplementation on muscle endurance and phosphagen metabolism. Quadriceps muscle pH and phosphagen kinetics were measured by using 31P-MRS. The ATP cost of contractions was determined by measuring ΔATP and force production during brief dynamic and static contractions performed at select intervals before and after exhaustive exercise.

Creatine supplementation increased muscle PCr concentration; however, this did not affect muscle ATP cost of contraction, as we hypothesized. These results suggest that differences in ATP cost of contraction observed between individuals (7) and muscle fiber types (5, 12, 24, 28, 34) are unaffected by increases in PCr availability resulting from creatine supplementation. In addition, muscle performance, as determined by time to exhaustion, time to 75% MVC recovery, and peak MVC, was not significantly affected by creatine supplementation. The ATP cost of contraction for both conditions was greatest at the onset of exercise. These results are consistent with previous studies (9, 31) and suggest that the increased ATP cost of contraction at the onset of exercise is associated with muscle excitation and relaxation processes.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision.

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Address for reprint requests: S. A. Smith, Belmont Univ., 1900 Belmont Blvd., Nashville, TN 37212-3757 (E-mail: smiths@mail.belmont.edu).

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