The Influence of Recovery Duration on High-Intensity Exercise Performance After Oral Creatine Supplementation

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Catalogue Data

Key words: ergogenic aids, cycle ergometry, short-term fatigue
Mots-clés: facteurs ergogènes, ergocycle, fatigue à court terme

Abstract/Résumé
The purpose of this study was to determine the effects of creatine supplementation on the ability to reproduce and maintain a high percentage of peak power output during the second of two bouts of high-intensity cycle sprinting following four different recovery intervals. Eighty healthy, active male subjects were randomly assigned to one of two groups (creatine or placebo) and one of four recovery intervals (30, 60, 90, or 120 s). Two maximal cycle ergometer sprints, separated by the assigned recovery interval were performed before and after a 5-day supplementation protocol in which 20 g/day of creatine (plus 4 g/day glucose) or 24 g/day glucose placebo were ingested by subjects from creatine and placebo groups, respectively. Maximal peak power output (PP) and the absolute time to fatigue (TTF) were compared pre- versus postsupplementation. No significant group interactions were noted in this study. Specifically, creatine supplementation had no effect on subjects’ ability to reproduce or maintain a high percentage of PP during the second bout of exercise.

Le but de cette étude est de vérifier si un supplément de créatine permet de maintenir un haut régime de travail au cours d’une deuxième épreuve de sprint sur ergocycle après 4 différentes périodes de récupération. Quatre-vingts vigoureux hommes en bonne santé sont aléatoirement répartis dans deux groupes (créatine ou placebo) et dans les quatre sous-groupes de récupération (30, 60, 90, et 120 s). Deux épreuves maximales de sprint sur
ergocycle intercalées d’une des quatre périodes de récupération sont accomplies par tous les sujets avant et après 5 jours d’ingestion d’un supplément nutritionnel (créatine: 20 g par jour additionné de 4 g de glucose; placebo: 24 g de glucose par jour). Le régime maximal de travail (PP) et le temps d’effort jusqu’à époussasion (TTF) sont comparés. L’analyse statistique ne révèle aucune interaction significative des groupes. Plus particulièrement, le supplément de créatine n’améliore pas la PP au cours de la deuxième épreuve.

Introduction

Creatine phosphate (CP) is utilized to maintain high rates of ATP turnover in skeletal muscle during brief bouts of high-intensity exercise (Hultman et al., 1983). The inevitable decline in power output observed during this type of activity has been attributed, in part, to a depletion of intramuscular CP stores (Sahlin, 1986). It has been suggested that high rates of ATP utilization might be better maintained, and therefore high-intensity exercise positively affected, if the preexercise levels of CP could be raised beyond normal (Balsom et al., 1994).

Recent studies have shown that ingesting approximately 20 g/day of creatine (Cr) for 5–6 consecutive days will lead to significant increases in total intramuscular creatine (Cr + PCr) (Harris et al., 1992; Hultman et al., 1996). However, although several investigators have reported a beneficial effect of Cr supplementation on high-intensity exercise performance (Balsom et al., 1993; Birch et al., 1994; Greenhaff et al., 1993), others have been unable to confirm these findings (Cooke et al., 1995; Febbraio et al., 1995; Mujika et al., 1996). Consequently, the ergogenic potential of increased ingestion of Cr is unresolved. Nevertheless, two primary hypotheses have emerged to explain how Cr supplementation might benefit athletes engaged in high-intensity short-duration activities: (a) Cr supplementation may stimulate the formation and storage of “excess” CP within the muscle prior to exercise (i.e., substrate loading), resulting in an increased intramuscular phosphoryl-transfer potential, or (b) Cr supplementation may increase intramuscular free Cr, and thereby accelerate the rate of CP resynthesis following fatiguing bouts of high-intensity exercise.

With regard to the first hypothesis, it has been shown that resting intramuscular CP concentrations are not significantly increased after Cr supplementation (Harris et al., 1992; Hultman et al., 1996). Although some reports seem to indicate a beneficial effect of Cr supplementation on performance, the data suggesting normal CP after supplementation, argue against the likelihood of any immediate ergogenic effect. For example, we previously reported that a 5-day supplementation regimen known to increase total Cr levels in skeletal muscle (Febbraio et al., 1995; Harris et al., 1992; Hultman et al., 1996) had no ergogenic effect on power output or fatigue during a single bout of high-intensity cycle sprinting (Cooke et al., 1995). Similar negative results have been described by other authors (Barnett et al., 1996; Febbraio et al., 1995; Mujika et al., 1996). Thus, it would seem that the practice of ingesting large amounts of Cr prior to a single bout of exercise may be of questionable value.

Alternatively, dietary Cr supplementation has been shown to produce an increase in resting levels of intramuscular free-Cr (Harris et al., 1992; Hultman et al., 1996). This increase is further augmented if the Cr ingestion is preceded by fatiguing bouts of exercise (Harris et al., 1992). Elevations in intramuscular free-Cr
have long been known to stimulate CP resynthesis rates in vitro (Jacobs and Lehniger, 1973; Seraydarian et al., 1976). Only recently have the effects of nonphysiologic concentrations of intramuscular Cr been investigated in the intact organism. Greenhaff et al. (1994) demonstrated that Cr supplementation increases the rate of CP resynthesis during recovery from electrically evoked maximal contractions in humans. This stimulatory effect of Cr on CP resynthesis seems to occur during the first few minutes following CP depletion. Therefore, the benefits of higher relative CP levels at the start of a second bout of exercise would most likely be evident following recovery durations within this time frame. The relationship between Cr supplementation and recovery duration during repeated bouts of high-intensity exercise has not yet been established.

The purpose of this study was to determine the effects of Cr supplementation on the ability to reproduce and maintain a high percentage of peak power output during secondary bouts of high-intensity, cycle exercise. Additionally, variations in intertrial recovery time were compared to determine if the proposed ergogenic effect of Cr supplementation might depend upon the duration of recovery allowed between consecutive bouts of exercise.

Methods

SUBJECTS

This study was approved by the Committee for the Protection of Human Subjects in Research, Texas A&M University. Eighty male subjects (age = 24.2 ± 3.3 yr, height = 179.3 ± 6 cm, weight = 81 ± 10.7 kg) were recruited for this investigation. All subjects gave their written informed consent and completed brief health histories. Individuals with a history of heart disease or hypertension, or who used excessive alcohol or tobacco products were eliminated from consideration. Subjects answered questions regarding their weekly exercise protocol (e.g., quantity of aerobic vs. anaerobic exercises), and categorized themselves as either (a) active–highly trained, (b) active–not highly trained, or (c) not active–not highly trained. Daily exercise was not controlled, but because exercise seems to affect Cr entry into skeletal muscle (Harris et al., 1992), subjects who chose the third choice (c) were eliminated from further consideration. In addition, because vegetarians seem to possess lower total-body creatine concentrations compared to meat eaters (Delanghe et al., 1989), vegetarians were excluded from this study. In an effort to insure subjects assigned to different groups were similar with respect to the amount of meat servings ingested per day, subjects were asked to record a representative 3-day diet during the supplementation period. The results of our random distribution of subjects throughout each group with respect to factors discussed above is presented in Table 1.

It is unclear whether males and females differ with respect to total intramuscular Cr levels. However, at least one study reported that females possessed higher total Cr levels relative to tissue weight than males (Forsberg et al., 1991). For this reason, we chose to study only males. Finally, previous work with the cycle ergometer showed that the reproducibility of our cycle sprint test was compromised when subjects failed to produce a peak power output of at least 600 W. Subjects unable to meet this criterion during the orientation sessions were not included in the study.
<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Weight</th>
<th>Height</th>
<th>Aerobic/wk</th>
<th>Anerobic/wk</th>
<th>Conditioning (subjective)</th>
<th>Meat/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>P30</td>
<td>24.0 ± 1.1</td>
<td>81.6 ± 2.9</td>
<td>181.9 ± 1.9</td>
<td>2.7 ± 0.5</td>
<td>2.5 ± 0.7</td>
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<td>8</td>
</tr>
<tr>
<td>Cr30</td>
<td>25.2 ± 1.1</td>
<td>78.1 ± 3.4</td>
<td>178.2 ± 2.0</td>
<td>1.7 ± 0.6</td>
<td>3.2 ± 0.6</td>
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<tr>
<td>P60</td>
<td>22.9 ± 0.8</td>
<td>79.2 ± 2.7</td>
<td>178.1 ± 1.4</td>
<td>2.4 ± 0.6</td>
<td>2.2 ± 0.8</td>
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<td>7</td>
</tr>
<tr>
<td>Cr60</td>
<td>24.0 ± 0.9</td>
<td>79.6 ± 3.2</td>
<td>180.6 ± 1.9</td>
<td>1.9 ± 0.5</td>
<td>3.3 ± 0.8</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>P90</td>
<td>23.2 ± 0.8</td>
<td>82.2 ± 2.9</td>
<td>181.5 ± 1.2</td>
<td>2.6 ± 0.5</td>
<td>2.2 ± 0.6</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Cr90</td>
<td>24.5 ± 1.2</td>
<td>80.7 ± 2.8</td>
<td>178.6 ± 2.2</td>
<td>1.2 ± 0.7</td>
<td>3.1 ± 0.7</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>P120</td>
<td>24.2 ± 0.9</td>
<td>83.1 ± 2.8</td>
<td>182.1 ± 1.3</td>
<td>1.7 ± 0.5</td>
<td>2.4 ± 0.4</td>
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<td>10</td>
</tr>
<tr>
<td>Cr120</td>
<td>22.5 ± 0.8</td>
<td>80.3 ± 2.5</td>
<td>178.5 ± 1.7</td>
<td>2.4 ± 0.4</td>
<td>1.3 ± 0.6</td>
<td>2</td>
<td>8</td>
</tr>
</tbody>
</table>

*Note.* Values are $M ± SEM$. ($N = 80; n = 10$ per group unless specified). P = placebo; Cr = creatine; HT = highly trained; NHT = not highly trained. Recovery intervals are 30, 60, 90, and 120 s.
CYCLE ERGOMETER

The device used in this investigation was a standard Monark cycle ergometer with the loading mechanism removed. An electronic load cell (LeBow model 3167, 0-900 N) was mounted to the ergometer frame and aligned with the tangent of the outside of the flywheel. The flywheel resistive strap was attached to the load cell, wrapped around the flywheel and over an externally mounted pulley. Previous work with a similar device demonstrated that a resistance of about 11 kg was sufficient for comparable subjects to produce peak power output (Cooke et al., 1995; Williams et al., 1988). Based on this previous work, in the present study a lead disk weighing 11.3 kg was attached to the resistive strap. A small wheel, with a circumference 1/9 that of the flywheel was fitted with a rubber O-ring and mounted to the ergometer frame in a position that allowed it to be driven by the flywheel. A continuous-turn potentiometer (Ace Electronics, Houston, TX) was mounted to the small wheel and calibrated to measure distance. The entire apparatus was bolted to the floor to provide maximum stability. The device is shown diagrammatically in Figure 1.

DATA COLLECTION AND ANALYSIS

Amplified signals (Grass Instruments, Quincy MA) from the load cell and potentiometer were digitized and sampled by a das-8 analog-to-digital converter (Metrabyte, Taunton MA.). Raw data were analyzed on line by microcomputer. Data collection was externally triggered when a force threshold (20% change from calibrated value) was reached during the first pedal revolution.

To determine power output, the following calculations were made: flywheel distance was calculated from the maximal voltage produced from each 360° rotation of the potentiometer. Each rotation was equivalent to 0.18 m traveled by the

Figure 1. Modified cycle ergometer.
flywheel. The true frictional force applied to the flywheel (N) was determined as the difference between the calibration value recorded before initiation of the test and the average force registered by the load cell (80–84 N). To minimize oscillations in power output frequently observed with instantaneous power measurements (Lakomy, 1986), while at the same time retaining a high degree of sensitivity, average force was computed for every four revolutions of the potentiometer. The time taken for four revolutions was recorded by the computer’s internal timers. Average force was multiplied by the distance traveled and divided by the time taken to cover the distance; this resulted in power being computed for every 0.72 m traveled by the flywheel. A computer analysis program identified peak power and compared subsequent power values to the peak. When power declined by 30%, the computer signaled the end of the exercise bout.

The following variables were computed: (a) peak power (PP) in watts, the highest power output value recorded during the test, and (b) time to fatigue (TTF) in seconds, the time taken from peak power to 30% decline from peak power.

EXPERIMENTAL PROTOCOL.

After having read and signed the informed consent form, and after successful completion of the health history and activity questionnaire, subjects were randomly assigned to one of two groups, creatine (Cr) or placebo (P) (n = 40 per group), and one of four recovery intervals (30, 60, 90, or 120 s; n = 10 per group). Because the sprint protocol used in this study was highly taxing, we attempted to maximize our chances of obtaining a truly maximal effort by assigning each subject to only one recovery duration. The seat of the cycle ergometer used in the power tests was individually adjusted to allow a 10–15° bend at the knee joint when a subject’s legs were fully extended. Subjects were then moved to a standard Monark ergometer, their feet were secured on the pedals with toe straps, and they performed a standardized warm-up consisting of pedaling at 75 W/min for 5 min.

Before the start of the sprint tests, subjects were instructed to grasp the bottom of the handlebars and remain seated throughout the test. At a verbal command, subjects pedaled as fast as possible until signaled to stop by the computer. Verbal encouragement was provided throughout the test. A 20-min rest period followed, and then a second trial was performed. These first two trials served as learning trials, and were performed to ensure that each subject became familiar with the testing procedures and the dynamics of the testing device. The reproducibility of PP and TTF were assessed in a pilot study (Cooke et al., unpublished observations), which confirmed that two learning trials were sufficient to control for effects due to unfamiliarity. We tested 15 subjects twice on Day 1, separated by a 20-min recovery period; subjects then reported to the laboratory 48 hr later and performed a third trial. The variable means for Tests 1, 2, and 3 (850.8 ± 28.4, 851.6 ± 27.4, and 848.6 ± 27.6 W for PP; 7.6 ± 0.36, 7.7 ± 0.41, and 7.7 ± 0.46, s for TTF, respectively) were not significantly different (p > .01). Intraclass correlation coefficients were high (.98 for tests administered on the same day and days for PP; .94 for tests administered on the same day and .93 for tests administered on different days for TTF). Although subjects may differ widely in their responses to constant-load ergometry, we have shown previously (Cooke et al., 1995; Williams et al., 1988) and recently (Cooke et al., unpublished observations) that this type of test is highly reproducible within subjects across time points.
All subjects reported to the laboratory 2 days after their initial learning trials. Because of the potential of this test to produce nausea if performed after a meal, subjects reported to the laboratory in the morning, after an overnight fast. Subjects were weighed upon arrival, and then performed the standardized warm-up as mentioned above. Following the warm-up, subjects performed two consecutive cycle sprints separated by either 30, 60, 90, or 120 s of recovery, depending on interval affiliation. The rest period was initiated immediately after the end of the first sprint, and the recovery duration was monitored by computer. Subjects remained seated on the ergometer during the rest period and were allowed to watch the countdown on the computer screen. At the end of the recovery period, the computer signaled, and subjects immediately began the second bout.

Following the pretests, subjects were given a supplement package corresponding to their treatment group and recovery interval affiliation. Beginning the morning after the pretests, subjects dissolved a 6 g dose of Cr (5 g creatine, 1 g glucose), or P (6 g glucose placebo) in water and consumed it. This procedure was repeated three additional times during the day at 3- to 4-hr intervals for 5 consecutive days. This supplementation protocol has been shown to result in a significant increase in total Cr concentrations measured from muscle biopsy samples (Harris et al., 1992; Hultman et al., 1996).

On the morning following the final day of supplementation, subjects returned to the laboratory for the posttests. The posttesting procedures were identical to the pretesting procedures.

STATISTICAL ANALYSES

Peak power and time to fatigue were evaluated for the two groups (Cr and P) separated into four different recovery intervals (30, 60, 90, and 120 s) for two trials at two time points (pre- and postsupplementation) using a $2 \times 4 \times 2 \times 2$ factorial analysis of variance (ANOVA), with repeated measures on the time and trial factors. Analysis of simple main effects was used to evaluate significant interactions. The alpha level for the experiment was set at $p = .05$. Bonferroni’s adjustment for two dependent variables was incorporated for the separate analysis of peak power and time to fatigue (Maxwell and Delaney, 1990). Therefore, the significance level for the analysis with respect to these variables was established at $p = .025$.

Total body weight was examined for the two groups (P and Cr) at two time points (pre- and postsupplementation) using a $2 \times 2$ factorial ANOVA, with repeated measures on the time factor. Significant interactions were investigated by analyzing the simple main effects. Significance was established at $p = .05$.

Results

Sample power curves for 2 subjects taken from performance data recorded during the first trial before supplementation are presented in Figure 2. As may be seen, PP was similar for both individuals (940–940 W) but the ability to maintain a high percentage of peak power (e.g., TTF) varied considerably. The selection of a percentage decline in PP (i.e., 30%) to serve as the criterion for test termination represented an effort to equate the degree of fatigue each subject would experience during a bout of exercise.
Figure 2. Comparative power curves for two subjects before supplementation. Subject A shown with closed circles, Subject B with closed squares.

CREATINE SUPPLEMENTATION AND BODY WEIGHT

Cr supplementation resulted in a significant increase in total body weight for subjects in the Cr group (80.6 ± 1.9 pre- vs. 81.6 ± 1.2 kg postsupplementation; n = 40 per group; p < .05). Evaluation of the simple main effects demonstrated that total body weight was significantly different for the Cr group postsupplementation (p < 0.05). Similar changes in body weight were not evident for the P group (80.5 ± 1.8 pre- vs. 80.6 ± 1.1 postsupplementation; p > .05).

CREATINE SUPPLEMENTATION AND SINGLE BOUTS OF EXERCISE

Cr supplementation had no effect on the capacity to produce maximum power during single bouts of high-intensity exercise. Trial 1 pre- and Trial 1 postsupplementation were not significantly different (n = 40 per group; p > .025). The effects of Cr supplementation on PP are shown in Table 2.

Likewise, Cr supplementation had no effect on the ability to maintain peak power output during single bouts of high-intensity exercise. TTF was not significantly different during Trial 1 after, compared to before supplementation (n = 40 per group; p > .025). The effects of Cr supplementation on TTF are shown in Table 3.

CREATINE SUPPLEMENTATION AND REPEATED BOUTS OF EXERCISE

Because the restoration of muscular force after a single fatiguing bout of exercise follows the same time course as CP resynthesis (Sahlin and Ren, 1989), it was of interest to determine the extent of PP restoration following different recovery
Table 2  Peak Power Production Before and After Glucose Placebo or Creatine Ingestion

<table>
<thead>
<tr>
<th></th>
<th>Presupplementation</th>
<th>Postsupplementation</th>
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<tbody>
<tr>
<td></td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td>P30</td>
<td>878.7 ± 31.4</td>
<td>803.8 ± 26.9*</td>
</tr>
<tr>
<td>Cr30</td>
<td>874.5 ± 40.7</td>
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</tr>
<tr>
<td>P60</td>
<td>872.5 ± 34.4</td>
<td>845.5 ± 28.4*</td>
</tr>
<tr>
<td>Cr60</td>
<td>890.4 ± 22.6</td>
<td>838.1 ± 20.5*</td>
</tr>
<tr>
<td>P90</td>
<td>853.0 ± 41.0</td>
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</tr>
<tr>
<td>Cr90</td>
<td>890.7 ± 33.9</td>
<td>863.5 ± 34.4*</td>
</tr>
<tr>
<td>P120</td>
<td>901.2 ± 38.8</td>
<td>887.4 ± 34.3</td>
</tr>
<tr>
<td>Cr120</td>
<td>884.8 ± 40.6</td>
<td>876.2 ± 38.0</td>
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<td></td>
<td>880.1 ± 33.0</td>
<td>814.6 ± 31.1*</td>
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<td>887.7 ± 44.1</td>
<td>811.9 ± 37.2*</td>
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<td>905.7 ± 32.3</td>
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<td>902.4 ± 48.2</td>
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<tr>
<td></td>
<td>888.5 ± 36.3</td>
<td>887.3 ± 32.3</td>
</tr>
</tbody>
</table>

Note. Values are M ± SEM expressed in watts. n = 10 per group. P = placebo; Cr = creatine. Trials are separated by 30, 60, 90, or 120 s of recovery.
*p < .05 from Trial 1 to Trial 2.

Table 3  Absolute Time Taken to Fatigue Before and After Glucose Placebo or Creatine Ingestion

<table>
<thead>
<tr>
<th></th>
<th>Presupplementation</th>
<th>Postsupplementation</th>
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<tbody>
<tr>
<td></td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td>P30</td>
<td>7.1 ± 0.32</td>
<td>4.9 ± 0.26*</td>
</tr>
<tr>
<td>Cr30</td>
<td>6.9 ± 0.69</td>
<td>5.2 ± 0.42*</td>
</tr>
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<td>P60</td>
<td>7.0 ± 0.57</td>
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<td>6.6 ± 0.45</td>
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<tr>
<td>P90</td>
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<tr>
<td>Cr90</td>
<td>6.9 ± 0.62</td>
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</tr>
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<td>6.7 ± 0.47</td>
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<td>5.8 ± 0.52</td>
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<td>6.0 ± 0.54</td>
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<td>6.2 ± 0.73</td>
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<td>6.4 ± 0.60</td>
<td>5.6 ± 0.46*</td>
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</table>

Note. Values are M ± SEM expressed in secpmds. n = 10 per group. P = placebo; Cr = creatine. Trials are separated by 30, 60, 90, or 120 s of recovery.
*p < .05 within the treatment period.

Durations. Analysis of the presupplementation data revealed significant differences between PP in Trial 1 and the PP achieved during Trial 2 after the three shortest recovery intervals (30, 60, and 90 s; n = 10 per group; p < .025). However, PP for Trials 1 and 2 was not significantly different following 120 s of recovery (n = 10 per group; p > .025).

Cr supplementation had no significant effect on the restoration of PP during Trial 2 after 30, 60, 90, or 120 s of recovery (n = 10 per group; p > .025). These results are shown in Table 2.
TTF was significantly decreased during Trial 2 compared to Trial 1 for all recovery intervals ($p < .025$). Trend analysis data indicated that, although none of the recovery durations were sufficient to allow complete recovery of endurance capacity, TTF was linearly related to recovery time ($r = .84$).

Cr supplementation had no significant effect on the restoration of TTF during Trial 2 following all recovery intervals ($p > .025$). These results are shown in Table 3.

**Discussion**

The results presented here confirm our previous finding that dietary supplementation with Cr monohydrate (20 g per day for 5 days) does not enhance peak performance during single bouts of high-intensity cycle exercise (Cooke et al., 1995). The present results, obtained from a larger group of subjects, seem to support our earlier contention that ingestion of large amounts of Cr prior to a single maximal sprint-type activity has little ergogenic value.

While this interpretation may apply to single bouts of high-intensity exercise, it does not necessarily preclude the possibility that dietary ingestion of Cr may beneficially affect other types of athletic performance. For instance, it has been proposed that Cr supplementation may accelerate or in some way enhance recovery from certain fatiguing bouts of exercise (Balsom et al., 1994). A specific suggestion is that Cr supplementation may increase the rate of CP resynthesis during the recovery interval between successive bouts of high-intensity exercise (Greenhaff et al., 1994). The basis for this hypothesis resides in the fact that elevating free Cr in vitro stimulates the rate of CP resynthesis by altering the mass action ratio of the Lohmann Reaction. Consistent with this notion is the fact that (a) resting mitochondrial respiration depends on both the concentrations of ATP and Cr (Bessman and Fonyo, 1966), and (b) postcontraction mitochondrial respiration is related to changes in free Cr (Saks et al., 1978; Seraydarian et al., 1976). Presumably, as Cr increases the rate of the Lohmann Reaction in the direction of CP synthesis, the resulting ADP serves to maintain sufficiently high rates of ATP production to support this reaction. Evidence of this effect in humans was demonstrated by the work of Greenhaff et al. (1994), who showed that CP concentrations were 30% higher at the end of a 2-min recovery from electrically evoked maximal contractions in Cr-supplemented subjects. Such a stimulatory effect of Cr on postexercise CP resynthesis may underlie the reported ergogenic effects of Cr supplementation during repeated bouts of high-intensity exercise (Balsom et al., 1993, 1995; Greenhaff et al., 1993).

In order to further investigate this possibility, we first compared PP and TTF between two successive bouts of high-intensity cycle exercise prior to Cr supplementation. Our results indicated that a 2-min recovery interval between bouts was sufficient for subjects to reproduce the PP obtained during Trial 1. These data are in agreement with those of Sahlin and Ren (1989), who showed that a 2-min recovery was required for complete isometric force restoration following fatiguing exercise. It has been estimated that approximately 75 to 90% CP restoration occurs during the first 2 min after exercise-induced CP depletion (Harris et al., 1976; Piiper and Spiller, 1970). Similarly, the restoration of muscular force follows the same time course, and is directly related to the degree of CP resynthesis (Sahlin
and Ren, 1989). Based on these findings, we anticipated that the restoration of power output would be compromised following recovery durations less than about 2 min. In the present study, subjects were unable to reproduce PP following rest periods of 30, 60, and 90 s.

Thus, our results compare favorably with those of Balsom et al. (1992), who found that maintaining running speed during repeated 40-m sprints depended upon adequate recovery between bouts. Likewise, Hirvonen et al. (1987) suggested that the decrease in running velocity typically seen during sprinting corresponds closely to the onset of significant CP depletion. Interestingly, TTF during the second bout of exercise in the present study was always significantly less than the initial trial, irrespective of the recovery duration employed (30, 60, 90, or 120 s). Thus, while 120 s was sufficient for PP restoration, it seems subjects require more time to completely regain their resistance to fatigue during this type of exercise.

Theoretically, an increase in CP availability could alter the capacity to produce and maintain power output during repeated bouts of high-intensity exercise. However, the data presented in this report showed that Cr supplementation had no significant effect on PP or TTF during the second of two successive bouts of cycle-sprint exercise. This was true irrespective of recovery duration. Evidently, the anticipated increase in sarcoplasmic free Cr known to occur after Cr supplementation (Harris et al., 1992; Hultman et al., 1996) was not sufficient to promote more rapid recovery. It has been assumed that the extra free Cr available to the muscle cell consequent to Cr supplementation and exercise would be used at the mitochondria, stimulating the resynthesis of CP from mitochondrial-derived ATP (Greenhaff et al., 1994). However, it is also possible that high concentrations of Cr might remain close to the contractile apparatus. Saks et al. (1978) suggested that high concentrations of Cr at the myofibrils could inhibit CP utilization through competitive binding for the same active site on creatine kinase. Further research is necessary to determine whether high intramuscular Cr concentrations are potentially beneficial or detrimental to muscle energetics. In the present study, the notion that Cr supplementation accelerates recovery from high-intensity exercise was not supported.

Although Cr supplementation was not found to have any ergogenic influence during two repeated bouts of exercise in the present investigation, our results do not preclude the possibility that experimental augmentation of intramuscular Cr stores might be an effective strategy to delay the onset of fatigue during numerous bouts of exercise. Evidence of lower muscle (Balsom et al., 1995) and blood (Balsom et al., 1993) lactate concentrations, as well as decreased plasma ammonia (Birch et al., 1994; Greenhaff et al., 1993) and plasma hypoxanthine (Balsom et al., 1993) concentrations measured after numerous prefatiguing exercise bouts suggest that increased muscle buffering capacity, rather than an increase in energy substrate availability per se may underlie the ergogenic potential of Cr.

It could be argued that the negative results reported in this and previous reports (Barnett et al., 1996; Cooke et al., 1995; Mujika et al., 1996) might be due to inadequate levels of Cr supplementation. Indeed, intramuscular phosphagen concentrations were not measured in this investigation, and it is possible that the Cr supplementation procedure used did not result in a significant increase in intramuscular free Cr. Although several investigators have confirmed from muscle biopsy material that comparable subjects possessed significantly elevated total Cr concentrations after similar Cr supplementation procedures (Balsom et al., 1995;
Harris et al., 1992; Hultman et al., 1996), we concede that interpretation of our data is limited by our lack of biochemical support. The fact that Cr supplementation resulted in a significant increase in total body weight, however, implies enhanced Cr uptake in the present study, as suggested by other investigators (Balsam et al., 1993; Greenhaff et al., 1994; Stroud et al., 1994). In addition, caffeine consumption was not controlled by our subjects. Recent evidence suggests that caffeine ingestion may counteract the ergogenic effects of creatine supplementation (Vanderbergh et al., 1996). Whether moderate intake of caffeine produces effects like those seen with caffeine supplementation (5 mg · kg⁻¹ · day⁻¹; Vanderbergh et al., 1996) is not known. In any event, from a practical standpoint, it is of interest that a large group (N = 80) of healthy volunteers (the same population generally targeted by athletic supplement companies) derived no performance-enhancing effect from dietary creatine ingestion.

In summary, the results presented here confirm our earlier finding that Cr ingestion lends no performance advantage to individuals engaged in single bouts of high-intensity, cycle sprint exercise (Cooke et al., 1995). Furthermore, these current data indicate that Cr supplementation does not enhance the recovery of maximal power output between two repeated bouts of high-intensity exercise. Certainly, it remains possible that the proposed ergogenic effect of dietary Cr supplementation is highly task specific and that the cycling activities employed in these two studies were not appropriate for demonstrating the performance-enhancing potential of Cr. Additional research using different modes of exercise and different testing protocols seems warranted.

References


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