Creatine Supplementation Enhances Intermittent Work Performance

Michael C. Prevost, Arnold G. Nelson, and G. Stephen Morris

To determine the impact of creatine supplementation on high-intensity, intermittent work, 18 participants each performed 2 sets of 4 different work bouts to exhaustion. For the first set of work bouts, all participants received a placebo (5 g of calcium chloride daily). For the second set of work bouts, 9 participants again received the placebo, while the other 9 received creatine supplementation (18.75 g creatine monohydrate daily for 5 days prior to and 2.25 g creatine daily during testing). The four work bouts in each set consisted of cycling to exhaustion at 150% peak oxygen uptake (VO2 peak) either nonstop (A), intermittently for either 60-s work/120-s rest periods (B), 20-s work/40-s rest (C), or 10-s work/20-s rest (D). Creatine supplementation significantly increased (p < .01) the total work time of all bouts. Protocol D showed the greatest increase (>100%); C increased 61.9%; B increased 61.0%; and A increased 25.3%. These results demonstrate that creatine supplementation significantly extends one's capacity to maintain a specific level of high-intensity, intermittent exercise.

Key words: exhaustion, anaerobic exercise, lactate, ergogenic aid

Many types of sport, work, and physical activities require repeated maximal or supramaximal levels of exertion. These high-intensity work outputs can be maintained for only brief time periods before fatigue occurs. The performance of high-intensity work, however, can be significantly extended if brief periods of rest are interspersed among the work periods (i.e., intermittent work) (Astrand & Rodahl, 1986). While intermittent work does increase the time one can sustain high-intensity work, fatigue will ultimately occur, reflecting an inability of the working muscle's creatine phosphate (PCr) stores to supply the sufficient amount of Adenosine triphosphate (ATP) necessary to maintain the elevated work output (Bogdanis, Nevill, Lakomy, & Boobis, 1993; Essen, 1978; Gaitanos, Williams, Boobis, & Brooks, 1993; Saltin, Essen, & Pendersen, 1976).

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Recent research has shown that oral creatine supplementation can increase skeletal muscle PCr levels (Balsom, Söderlund, Sjödin, & Ekblom, 1995; Febbraio, Flanagan, Snow, Zhao, & Carey, 1995; Greenhaff, Bodin, Söderlund, & Hultman, 1994; Harris, Söderlund, & Hultman, 1992) and that such supplementation has ergogenic potential (Balsom et al., 1995; Balsom, Ekblom, Söderlund, Sjödin, & Hultman, 1995; Greenhaff et al., 1993). For example, Greenhaff et al. (1993) showed that, following creatine supplementation, peak torque production was higher in Bouts 2, 3, and 4 of a five-bout work protocol of 30 minutes voluntary isokinetic contractions interspersed with a 60-s rest. Balsom et al. (1995) showed that, following creatine supplementation, the power output of a single high-intensity 10-s exercise period preceded by five 6-s work bouts (each separated by a 30-s rest) was greater than it had been prior to creatine supplementation. Later, Balsom et al. (1995) reported that creatine supplementation significantly improved the capacity to maintain a given pedal speed during intermittent cycling consisting of ten 6-s high-intensity work bouts interspersed with a 30-s rest.

Because creatine supplementation can increase peak performance of short-term, intermittent work protocols and the magnitude of an "all out" work bout at the end of an intermittent protocol, one would expect that creatine supplementation also to increase the total
number of intermittent work bouts that could be performed at a specific high intensity. If so, then creatine supplementation would benefit the performance of physical or sporting activities which entail bursts of highly intense activity interspersed with periods of reduced or lower activity (e.g., basketball, soccer, and hockey) by enabling all participants to play longer before becoming fatigued and, thus, requiring fewer rests for top players. Unfortunately, no creatine supplementation study has investigated whether this practice will delay fatigue or increase the number of high-intensity work bouts that can be performed or whether the ergogenic benefits of creatine supplementation are optimal for any particular high-intensity/low-intensity duty cycle. Thus, the primary purpose of this study was to determine if creatine supplementation would benefit the performance of activities which require repeated, intermittent maximal or supramaximal exertion levels by enabling an individual to perform more intermittent activities in succession. A second purpose was to determine if any specific intermittent high-intensity/low-intensity duty cycle benefited more by the supplementation. These purposes were met by comparing the impact of creatine supplementation versus a placebo on the time required to reach exhaustion of one continuous and three intermittent high-intensity exercise bouts of differing lengths, but with the same work-to-rest ratio. We hypothesized that creatine supplementation would increase the time to exhaustion and that a greater benefit would be realized as the combined length of the work-rest cycle was shortened.

Methods

Participants

Participants were physically active healthy men (10) and women (8) volunteers recruited from various university physical education activity classes (see Table 1 for participant characteristics). To ensure that an individual’s exercise practices did not influence study results, only those individuals who had not experienced an alteration in their exercise habits or activity levels over the preceding 3 months and who were willing to maintain their current activity levels over the full duration of the study were included in the study. The experimental protocol was approved by the appropriate institutional review board, and each individual gave written consent to act as a participant in this experiment.

Experimental Protocol

The experiment was divided into two phases: initial (Phase 1), and supplement (Phase 2). Preliminary discussions with the participants indicated that most were aware of the ergogenic potential of creatine supplementation. Because this notion could bias the tests results in favor of creatine supplementation, it was decided that the participants would be given some form of supplementation for both phases of the experiment and be informed that the intent of the study was to evaluate two different dosages of creatine supplementation. For Phase 1, all participants received a placebo (see below) and underwent four different work protocols. Each of the four work protocols was performed on separate days in random order. For Phase 2, the participants were divided into two groups, a control (CON) group which again received the placebo and a supplementation (PCR) group which received the actual creatine supplementation (see below). Each group consisted of 5 men and 4 women.

The testing proceeded as follows (see Figure 1): Day 1—start of Phase 1 supplementation (placebo) regimen and performance of a peak workload test; Days 5, 7, 9, 11—testing sessions consisting of either protocol A, B, C, or D (see below); Day 11—end of Phase 1; Days 12–19—rest period; Day 20—start of Phase 2 supplementation regimen; Days 25, 27, 29, 31—testing sessions consisting of either protocol A, B, C, or D (same order as days 5–11); Day 31—end of Phase 2.

Peak workload was determined on the first day of Phase 1 testing to obtain a standardized workload of high intensity for the ensuing Phase 1 and 2 work bouts. Participants were instructed to refrain from rigorous physical exercise the day before each test and to fast for at least 4 hours prior to a test session. All exercise tests were performed on a Monark 818E cycle ergometer (GHJ, Stockholm, Sweden). During the peak workload test, the seat height was adjusted to participant satisfaction and recorded to ensure the same position for each succeeding test. Prior to each test, participants engaged in a standardized warm-up procedure consisting of 5 min of pedaling at a low-tension level.

<table>
<thead>
<tr>
<th>Table 1. Subject characteristics (mean ± standard deviation)</th>
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<tbody>
<tr>
<td><strong>PCR</strong></td>
</tr>
<tr>
<td>Age (years)</td>
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<tr>
<td>Weight (kg)</td>
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<tr>
<td>V02 peak (ml·kg⁻¹·min⁻¹)</td>
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<td>Workload @ V02 peak (W)</td>
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Note. PCR = creatine supplementation group; CON = placebo supplementation group.
Participants began the peak workload test immediately after the warm up by pedaling at 100 rpm against zero load for 1 min. The load was then increased by 50 W each minute until the participant was no longer able to maintain the required 100-rpm pedaling rate. During the test, the expired gases were continuously monitored, and oxygen consumption was calculated every 20 s. The workload associated with the last completed full stage (last 1-min stage) was considered the peak workload.

The exercise bouts consisted of four different exercise protocols to exhaustion. To ensure that the same criterion for exhaustion was used for each individual and each work bout, one person administered all the exercise bouts. One exercise protocol was performed per testing session, and the exercise protocols were assigned in random order during Phase 1, with the same order repeated during Phase 2. Following warm up, each participant pedaled 100 rpm at 150% of their peak workload following one of the four exercise protocols outlined below until the individual was exhausted (unable to maintain 100 rpm for 10 s). The four protocols consisted of: Protocol A—continuous cycling; Protocol B—repeated bouts of 30 s of cycling, with each bout separated by 60 s of rest; Protocol C—repeated bouts of 20 s of cycling, with each bout separated by 40 s of rest; and Protocol D—repeated bouts of 10 s of cycling, with each bout separated by 20 s of rest. The total time spent working at each protocol was recorded, and for each protocol the participants were uninformed about the exact length of time that they had worked.

Supplementation

Placebo supplementation (PCR Phase 1, CON Phases 1 and 2) consisted of 1 g of calcium chloride (five tablets; Oyster Shell Calcium, Spring Valley, Allegan, MI) five times daily for 5 days, followed by 0.6 g of calcium chloride (three tablets) once daily for 6 days. Calcium chloride was chosen as the placebo, because it was available in a tablet form indistinguishable in appearance (identical in size, shape, and color) from the creatine tablets. During Phase 2, participants in the PCR group ingested 3.75 g creatine monohydrate (five tablets of 0.75 g each; Creatabolin C10, Around the World Inc., Greensboro, NC) five times daily (total of 18.75 g per day) for 5 days. They then ingested 2.25 g creatine monohydrate (three tablets) once daily for 6 days. All participants were unaware of which supplement they received during either test phase, and during Phase 2 the test administrator did not know which individuals were creatine supplemented. For both the calcium chloride and the creatine tablets, the participants were told to ingest as much water as necessary to swallow the tablets, and neither the CON or PCR participants reported any gastric distress from taking the supplements.

Blood Sampling and Analysis

Multiple blood samples were collected during each exercise test. Each sample consisted of 100 μl of blood collected in heparinized microcapillary tubes by the finger-prick method. Blood samples were taken on the following schedule: Protocol A (continuous bout)—immediately prior to exercise, immediately after exercise, and 3 min post exercise; Protocol B (30-60 s intermittent bout)—immediately prior to exercise, immediately after the first 30-s pedaling period (30 s), immediately after the second 30-s pedaling period (2 min), and immediately post exercise; Protocol C (20-40 s intermittent bout)—immediately prior to exercise, immediately after the second 20-s pedaling period (2 min), immediately after the fourth 20-s pedaling period (4 min), and immediately post exercise; Protocol D (10-20 s intermittent bout)—immediately prior to exercise, immediately after the fourth 10-s pedaling period (2 min), immediately after the eighth 10-s pedaling period (4 min), and immediately post exercise.

Blood samples were placed in an ice-water bath and centrifuged immediately following the exercise test. The resulting plasma was stored at -70°C for later analysis. Blood plasma lactate concentration was determined using the Analox GM7 microstat analyzer (Analox Instruments Ltd., London, England). The microstat analyzer functions by measuring the oxygen change when oxygen reductase enzymes react with their substrates under controlled semi-anerobic conditions.

Statistical Analysis

For each work bout (Protocols A–D), total work time was calculated from the time to exhaustion by subtracting out the rest period times. Total work time was analyzed using an analysis of variance (ANOVA) with repeated measures (group x phase). Post-ANOVA analysis involved, where appropriate, the use of Tukey's range
test. The experimental error rate was set at .05, and was maintained throughout all post-ANOVA tests. Each bout was also analyzed independently for differences in lactate at the specific time intervals, using ANOVA with repeated measures (group x phase x time). In addition to the ANOVA, the relationships between peak oxygen uptake (VO_{peak}) and increases in total work output and between body weight and increases in total work output were examined using simple linear regression. Prior to drawing conclusions concerning the relationships, the regression model was examined for departure from the linear model (i.e., lack of error variance linearity or constancy, presence of outliers, lack of error term normality, etc.) using established diagnostic tests.

Results

Total Work Time

Creatine supplementation significantly increased the time to exhaustion and total work time (and, thus, total work output), regardless of work protocol (see Figure 2). Protocol D was impacted more than the other protocols, $F(1, 16) = 180, p < .01, \omega^2 = .21$ with PCR participants showing a greater than 100% increase in total work time, while CON participants showed no significant change between Phases 1 and 2. For all participants, Protocol D in Phase 2 stopped when the participant reached exhaustion or achieved at least twice the performance time reached during Phase 1, whichever came first. This action was taken, because the first two participants to perform Protocol D in the Phase 2 work bout expressed an inclination to remove themselves from study after working for a period nearly three times the length of their Phase 1 activity. On completing the testing, half the participants ($n = 9$) had been able to double their previous time, and all in the PCR group. No PCR participant reported feeling fatigued at the termination of Protocol D in Phase 2, and many felt capable of continuing the work indefinitely. For Protocols C, B, and A, however, the PCR participants did reach a point of fatigue. Nevertheless, Protocol C was also significantly impacted by creatine supplementation, $F(1, 16) = 25.9, p < .01, \omega^2 = .20$. For this exercise protocol, PCR participants in Phase 2 saw a 61.9% greater total work time over PCR participants in Phase 1, while CON participants showed no significant difference between Phases 1 and 2. Likewise, when exercising according to Protocol B, $F(1, 16) = 36.1, p < .01, \omega^2 = .17$, PCR participants showed a 61% increase in total work time (Phase 2 over Phase 1), while CON participants doing Protocol B showed no significant change between the two phases. Finally, total work time using exercise Protocol A was significantly prolonged by creatine supplementation, $F(1, 16) = 5.8, p < .05, \omega^2 = .06$, with the PCR participants experiencing a 23.5% increase in Phase 2 as compared to Phase 1, and the CON participants showing no significant change.

Plasma Lactic Acid

As expected, plasma lactic acid concentration increased with exercise for all treatment and exercise combinations (Bout A: $F(3, 48) = 635, p < .01, \omega^2 = .86$; Bout B: $F(3, 48) = 682, p < .01, \omega^2 = .95$; Bout C: $F(3, 48) = 798, p < .01, \omega^2 = .94$; Bout D: $F(3, 48) = 751, p < .01, \omega^2 = .86$). However, in Protocol A post-exercise plasma lactic acid levels were significantly lower following creatine supplementation, while consumption of the placebo did not alter this measure ($F(3, 48) = 19.8, p < .01, \omega^2 = .006$) (see Figure 3). The ability of creatine supplementation to significantly blunt the exercise-induced rise in plasma lactic acid levels was also present during Protocol B, $F(3, 48) = 7.04, p < .01, \omega^2 = .003$ (see Figure 4); Protocol C, $F(3, 48) = 6.85, p < .01, \omega^2 = .001$ (see Figure 5); and Protocol D, $F(3, 48) = 20.1, p < .01, \omega^2 = .006$ (see Figure 6).

Regression of Body Weight and Fitness

Body weights did not change from Phase 1 to 2, $F(1, 35) = 2.95, p > .05$. Neither body weight ($r = .074$) nor VO_{peak} test ($r = .038$) were good predictors of the mag-
magnitude of improvement in total work output due to creatine supplementation for A, B, or C (D, which was stopped at twice the performance time of Phase 1, was not analyzed).

Discussion

Creatine supplementation (5 g of creatine monohydrate 4–6 times a day for 2 or more days) significantly increases the total creatine content of the human quadriceps femoris muscle (Harris et al., 1992; Greenhaff et al., 1994). An increase in creatine and PCR has been shown to: (a) blunt the decline in ATP concentration following high intensity exercise (Balsom et al., 1995); (b) increase the rate of PCR resynthesis following high intensity exercise (Greenhaff et al., 1994; Febbraio et al., 1995); and (c) reduce plasma lactate acid accumulation during high-intensity, intermittent exercise (Balsom et al., 1993; Balsom et al., 1995; Febbraio et al., 1995). Functionally, creatine supplementation increases both the total work output in a single high-intensity exercise bout preceded by a series of repetitive high-intensity work bouts (Balsom et al., 1993) and increases the peak performance during a series of brief, repetitive high-intensity work bouts (Balsom et al., 1995; Greenhaff et al., 1993). It is unknown if the increases in peak performance and terminal bout work output also mean that an athlete will be able to perform a greater number of high-intensity bouts before the onset of fatigue or, in other words, be able to perform at near peak for a longer time. In addition, if creatine supplementation can extend the onset of fatigue during intermittent work, it is not known if a specific high-intensity/low-intensity duty cycle demonstrates this benefit to a greater degree than any other. Results from the current study demonstrate that creatine supplementation extends the performance of both continuous and intermittent high-intensity exercise. The benefits, however, are more pronounced in short-term, intermittent, high-intensity bouts. Furthermore, the data from the current study suggest at least two possible mechanisms through which the elevated energy needs were better met following creatine supplementation.

First, the improved performance could have been achieved by simply increasing the concentration of stored phosphagens. (While we did not measure muscle creatine and PCR levels, it is reasonable to assume that each participant experienced an increase in intramuscular PCR stores similar to those documented by previous researchers (Balsom et al., 1995; Febbraio et al., 1995; Greenhaff et al., 1994; Harris et al., 1992), as we employed a dose, 18.75 g per day, similar to theirs, 20–25 g per day.) It is generally accepted that the energy (ATP) requirements of brief (< 20 s), high-intensity exercise are met principally by utilizing available phos-
phagen stores. Hence, any increase in the concentration of stored phosphagens would allow more work to be done before the high-energy phosphate stores become sufficiently depleted to compromise work output. Therefore, shorter intermittent work periods (especially < 20 s) should derive the greatest benefit from creatine supplementation. This is precisely what was seen in this experiment. Protocol D (10 s of work) yielded the greatest increases in total work time following creatine supplementation, with the participants expressing no signs of fatigue despite doubling their total work time. Such results suggest that the creatine supplementation increased the intramuscular PCR stores to a level exceeding that necessary to perform the 10 s of work, and the rest period allowed adequate replenishment of the stores.

In addition, creatine supplementation may have improved total work output by decreasing the working muscle’s reliance on glycolysis to provide ATP. This metabolic shift would then reduce lactate accumulation and the ergolytic effect of the accompanying reduction in muscle pH. As high-intensity exercise is initiated, there is a rapid degradation of high-energy phosphates (Gaitanos et al., 1993; Bogdanis et al., 1993). This reduction in PCR and ATP activates glycolysis to buffer the drop in ATP levels. For instance, Gaitanos et al. (1993) found that glycolysis provided as much as 50% of the ATP required for 6 s of maximal work. Furthermore, as the work period or repetitive work bouts increases, it would be expected that the contribution from glycolysis would increase as well. This increasing reliance on glycolysis to maintain ATP levels during intermittent work may contribute to the onset of fatigue. Again, Gaitanos et al. (1993) saw ATP production from glycolysis during 10 bouts of 6-s sprints drop from 14.9 mM·kg⁻¹·dry wt-min⁻¹ (initial rate) to 5.3 mM·kg⁻¹·dry wt-min⁻¹ (10th bout rate). The authors proposed that the large increase in lactate and the concomitant decrease in muscle pH was responsible for reducing glycolytic rates. In the present study, plasma lactate accumulation was reduced following creatine supplementation. Assuming that lactate efflux and clearance were not influenced by creatine supplementation, the lower plasma lactate values suggest a reduced reliance on anaerobic glycolysis following creatine supplementation. This decreased reliance on glycolysis and concomitant lower lactate production would result in a smaller reduction in intramuscular pH. Because a reduction in pH has been linked to a reduced ability of glycolysis to generate ATP (Gaitanos et al., 1993), a reduced decline in pH might extend time to exhaustion by maintaining a faster glycolytic ATP production rate. However, a creatine supplementation-induced drop in lactic acid levels is probably not the primary reason for the improvement in time to exhaustion. If the capacity to continue the intermittent bouts was primarily linked to anaerobic glycolysis, one would expect similar lactate levels at exhaustion. The plasma lactate levels at exhaustion, however, were not the same in protocols A, B, and C.

A reduced reliance on glycolysis might also help explain the greater impact of creatine supplementation on protocol D. Due to the relatively short work periods during protocol D (10 s), it is likely that glycolysis contributed less to ATP production during this protocol than

![Graph](image)

**Figure 5.** Protocol C plasma lactate concentrations for both the creatine supplementation (PCR) and placebo (CON) groups for both experimental phases. The plotted data points are means ± standard deviation. * denotes a Phase 2 mean of a specific protocol which is significantly (p<0.01) higher than its corresponding Phase 1 mean.

![Graph](image)

**Figure 6.** Protocol D plasma lactate concentrations for both the creatine supplementation (PCR) and placebo (CON) groups for both experimental phases. The plotted data points are means ± standard deviation. * denotes a Phase 2 mean of a specific protocol which is significantly (p<0.01) higher than its corresponding Phase 1 mean.
during the other protocols (note that lactate accumulation was lower during protocol D than during the other protocols). Therefore, even a small decline in the glycolytic rate would represent a relatively large reduction in the contribution of glycolysis during protocol D. Consequently, during longer work bouts, a similar small decline in the already large contribution from glycolysis would be expected to have less effect.

It has been proposed that fitness levels may affect the response to creatine supplementation, because exercise training increases PCr stores (MacDougall, Ward, Sale, & Sutton, 1977), and creatine uptake due to creatine feeding is greatest in participants who have a lower initial level of creatine (Harris et al., 1992). Hence, the relationship between fitness level (highest VO_2 during VO_2peak test) and the magnitude of response to creatine supplementation was also investigated. No significant relationship between these two variables, however, was found. Perhaps the chosen index of fitness has little relationship to muscle creatine levels; or the difference between participants' fitness and initial creatine levels did not vary enough interindividually to establish a strong correlation; or, in all probability, the initial creatine levels are more related to dietary practices (protein intake) rather than fitness levels. Alternatively, the amount of the creatine supplement may have been sufficient to impact performance regardless of initial creatine levels. As stated previously, no relationship between body weight and the response to creatine supplementation was observed. The participants, however, encompassed a broad range of body weights from 52.2 kg to 131.5 kg. Hence, the lightest participant in the study received nearly 2.5 times the relative dose of the heaviest subject, and, thus, the dose administered to the lighter participants far exceeded that necessary to produce the ergogenic response.

In summary, creatine supplementation significantly delayed or prevented fatigue in individuals performing high-intensity intermittent work. These findings support the argument that creatine supplementation can be of ergogenic benefit. Available data argue that creatine supplementation probably works by enhancing intramuscular phosphagen levels or delaying excessive dependence on anaerobic glycolysis. Because creatine supplementation had its smallest effect on continuous, high-intensity activity, the ergogenic benefit appears to be greatest for intermittent work bouts. As such, its benefit may lie in enhancing recovery from high-intensity work as much as delaying the onset of fatigue during the high-intensity work itself. Because the participants experienced the least amount of fatigue during the 10-s work bouts, this study substantiates the supposition of Febbraio et al. (1995) that creatine supplementation would yield its greatest impact on work that relied primarily on creatine phosphate as the chief energy source. Hence, it would appear that creatine supplementation would prove beneficial to those athletes involved in sports in which high-intensity activity levels are interspersed with periods of low activity.

References


Notes

1. Data available on request from Michael Prevost, NOMI ASTC, MCAS El Toro, P.O. Box 94011, Santa Ana, CA 92709-4011.
Authors' Notes

The authors thank Around The World, INC., of Greensboro, NC, for providing the creatine supplement. Address all correspondence regarding this article to Arnold Nelson, Department of Kinesiology, 112 Long Field House, Louisiana State University, Baton Rouge, LA 70803.