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The effects of oral creatine supplementation on performance in single and repeated sprint swimming

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We studied the effects of oral creatine supplementation on sprint swimming performance in 14 elite competitive male swimmers. The subjects performed a single sprint (1×50 yards [45.72 m]) and repeated sprint set (8×50 yards at intervals of 1 min 30 s) before and after a 5 day period of either creatine (9 g creatine + 4.5 g maltodextrin + 4.5 g glucose day^{-1}) or placebo (18 g glucose day^{-1}; double-blind protocol) supplementation. Venous and capillary blood samples were taken for the determination of plasma ammonia, blood pH and lactate. Mean times recorded for the single 50 yard sprint were unchanged as a result of supplementation (creatine *vs* control, N.S.). During the repeated sprint test, mean times increased ($P < 0.01$, main effect time) during all trials, but performance was improved as a result of creatine supplementation (sprints 1–8: control pre-, 23.35 ± 0.68 to 26.32 ± 1.34 s; control post-, 23.59 ± 0.66 to 26.19 ± 1.48 s; creatine pre-, 23.20 ± 0.67 to 26.85 ± 0.42 s; creatine post-, 23.39 ± 0.54 to 25.73 ± 0.26 s; $P < 0.03$, group $\times$ trial interaction). Thus the percentage decline in performance times was reduced after creatine supplementation (control, $12.7 \pm 5.7\%$ *vs* $11.0 \pm 5.5\%$; creatine, $15.7 \pm 4.3\%$ *vs* $10.0 \pm 2.5\%$; $P < 0.05$, group $\times$ trial interaction). The metabolic response was similar before and after supplementation, with no differences in the blood lactate or pH response. Plasma ammonia was lower on the second trial ($P < 0.05$, main effect trial), but this could not be attributed to the effect of supplementation (group $\times$ trial interaction, N.S.). A further urinary analysis study supported these findings by demonstrating an approximately 67% (~26 g) retention of the administered creatine in this group of swimmers after an identical supplementation regimen. In summary, our results suggest that ingesting 9 g creatine per day for 5 days can improve swimming performance in elite competitors during repeated sprints, but appears to have no effect on a single 50 yard sprint.

Keywords: ammonia, creatine supplementation, lactate, phosphocreatine, sprint swimming.

Introduction

The decline in the rate of resynthesis of adenosine triphosphate (ATP) as a result of depletion of phosphocreatine is recognized as a possible cause of the reduction in muscular power in maximal intensity exercise (Hultman *et al.*, 1967). Recent studies have shown that oral creatine supplementation can: increase the total concentration of creatine and phosphocreatine in skeletal muscle and the rate of phosphocreatine resynthesis (Harris *et al.*, 1992; Greenhaff *et al.*, 1993a, 1994a,b); increase muscle peak and mean power output during cycling (Birch *et al.*, 1994); increase total work and reduce fatigue during repeated maximal exercise (Balsom *et al.*, 1993; Greenhaff *et al.*, 1993b, 1994a; Bogdanis *et al.*, 1996); improve running performance times (Harris *et al.*, 1993); and improve recovery in repeated bouts of high-intensity exercise (Balsom *et al.*, 1993; Greenhaff *et al.*, 1993a,b, 1994a; Bogdanis *et al.*, 1996).

These results suggest that oral creatine supplementation can improve performance in high-intensity short-term exercise by increasing resting concentrations of creatine and phosphocreatine, and by improving the rate of phosphocreatine and ATP resynthesis in skeletal muscle. However, all of these studies used high dosages (i.e. 20–30 g day^{-1}) and none examined sprint swimming performance using elite competitors.

Despite claims by the manufacturer of a creatine supplement (Ergomax™ C150) that swimming performance can be enhanced by 5% as a result of creatine

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supplementation, there are at present no reports of any swimming studies in the literature to confirm or refute such claims. In addition, the recommended dosage of their product (9 g creatine day^{-1} for 5 days) is lower than those used in the above-mentioned studies (20–25 g day^{-1} for 5–6 days). Furthermore in a recent study, Cooke *et al.* (1995) reported no significant differences in performance during the first or second of two cycle ergometer sprints separated by 20 min rest after oral creatine supplementation (20 g creatine day^{-1} for 5 days). Therefore, the efficacy of creatine supplementation for performance enhancement during a single sprint may be questioned.

In summary, very few studies have examined the effects of oral creatine supplementation on sports performance (in comparison with performance during laboratory tests such as isokinetic cycling), or have used any group of subjects other than sedentary or active recreational sportspeople. In addition, the uptake of creatine at a dosage of 9 g day^{-1} is unknown.

The aims of the present study were to establish the effects of oral creatine supplementation on the performance and metabolic responses of elite competitive swimmers during a single sprint swim of 50 yards and during a training set of 8×50 yards, and to estimate creatine uptake at 9 g day^{-1} in this group of swimmers.

Methods

Subjects

Fourteen male swimmers from Loughborough University swimming club participated in the study. All subjects were involved in a regular training and competitive programme (6–10 swimming sessions per week) and had competed in the National Championships or had swum at international level in the previous 12 months. The subjects were informed of the aims of the study and any known risks, and then gave their written consent to participate. The protocol was approved by the Ethical Committee of Loughborough University.

The swimmers had previous experience of 50 yard competition swimming, as well as sprint training sets such as the one used in this study. The subjects were randomly assigned to an experimental (creatine supplementation) or control (glucose) group. The mean ($\pm s$) physical characteristics of the subjects were as follows: age, 20 ± 2 and 21 ± 2 years; height, 181 ± 5 and 183 ± 6 cm; body mass, 75.6 ± 5.2 and 75.9 ± 9.3 kg for the creatine and control groups, respectively.

Personal best times were all recorded within the previous year and recorded in a 25 m pool. Times were converted for a 25 yard pool using the Amateur Swimming Association Standard conversion tables. Mean personal best times for 50 yards were 22.26 ± 0.37 s for the creatine group and 22.26 ± 0.33 s for the control group.

Selection of test protocol

A race distance of 50 yards was selected as the closest to a true 'sprint' in competitive swimming. Conveniently, the swimming duration time (approximately 22–23 s for males) allows for some comparison with sprint studies carried out using other modes of exercise (e.g. cycle ergometry and treadmill sprinting).

The distance and rest period for the sprint interval training set were devised after two pilot studies in which the swimmers completed a set of 5×100 yards at intervals of 4 min, and a set of 12×50 yards at intervals of 2 min. The subjects were asked to exert maximum effort on the first repetition of the set and also on subsequent repetitions. The results of both tests suggested that the ability of the swimmers warranted a shorter rest interval, and that 12 sprints were too daunting for the swimmers to give maximum effort on the first sprint. Therefore, 8×50 yards at intervals of 1 min 30 s was chosen and thus the first sprint of this set could be compared directly with the single 50 yards sprint to establish that effort was maximal at the start of the set.

Experimental procedures and protocol

The subjects undertook four test sessions: the 1×50 yard and 8×50 yard sprints were performed on consecutive days and were then repeated after a minimum of 1 week. Five days before the second set of tests, the subjects consumed either 9 g creatine + 4.5 g maltodextrin + 4.5 g glucose day^{-1} (creatine supplementation) or 18 g glucose day^{-1} (control; see below). The subjects were randomly assigned to groups using a double-blind design.

All repetitions took place with a dive from racing blocks and were timed from an official start. 'Anti-wave' lane ropes were used to divide the lanes as in competition to reduce unnatural water resistance. The subjects were asked to exert maximum effort on each repetition, while maintaining a constant stroking technique. Swimming times were recorded in duplicate by experienced timekeepers using chronograph stopwatches.

The subjects were required to refrain from consuming alcohol for 24 h before both tests, and performed light exercise (4–5 km swimming training per day controlled by the experimenter) throughout the study. During the 3 days before test 1, the subjects were required to record

all food and drink intake in a food record diary. They were then requested to follow the same (pre-test 1) diet and eating schedule before test 2. All test sessions took place in the Sports Science Research Laboratories and Sports Hall swimming pool (25 yards) at Loughborough University. The mean ($\pm s$) pool temperature was 26° $\pm$ 1°C.

A standardized warm-up (approximately 25 min) was performed in preparation for each test. Test order was conducted as above for all subjects to obtain a reference time for one 50 yard sprint. The objective in the second test was to repeat that time as closely as possible on the first 50 yard swim and then on successive repetitions. The timing of the test protocol is illustrated in Fig. 1.

The tests were conducted either 1 or 2 weeks apart to allow healing from needle punctures, full recovery after each test and standardization. Repeated tests were carried out at the same time and same day of the week.

Supplementation period (5 days)

The subjects were provided with 15 pre-measured packets of powder (either 3 g creatine + 1.5 g maltodextrin [i.e. three Powdered Ergomax™ C150 tablets] + 1.5 g glucose or 6 g glucose) and instructed to mix the powder in hot water, tea or coffee for immediate consumption at 09:00, 13:00 and 17:00 h each day during the supplementation period.

Equipment and measurements

Height and body mass were measured on each visit to the laboratory in swimming attire. Heart rate was recorded at rest and throughout all the exercise phases of the investigation using short-range telemetry (Polar Electro PE3000, Kempele, Finland). A transmitter was strapped to the chest, level with the sternum, and secured under a lycra 'triathlon top' (Speedo Europe Ltd.) to ensure constant contact between the electrodes

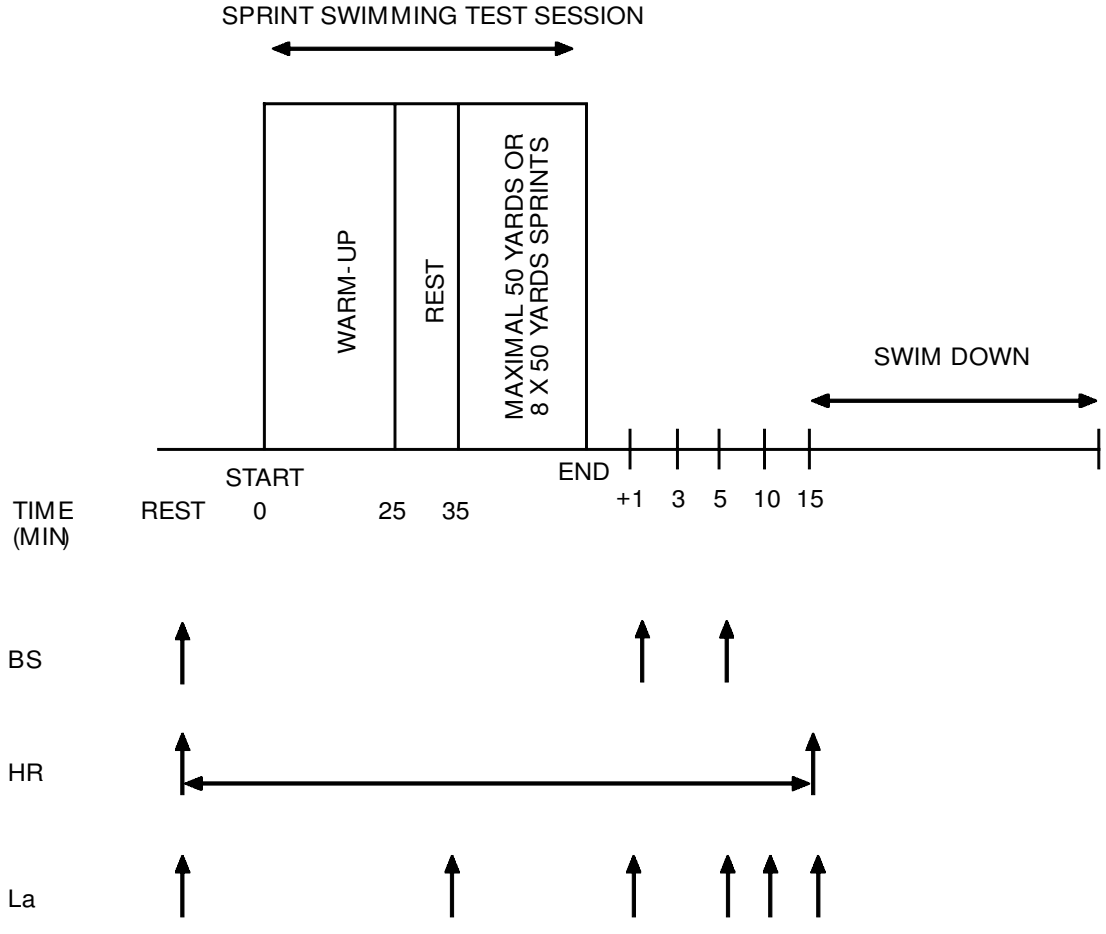

Figure 1 Schematic illustration of the 1 $\times$ 50 yard and 8 $\times$ 50 yard swimming protocols. BS, venous blood sample; HR, heart rate; La, fingertip blood sample.

and the skin. The receiver was vacuum-sealed in a waterproofed plastic bag and placed under the costume.

Blood collection and analysis

Antecubital venous blood samples (10 ml) were obtained at rest, before the standardized warm-up, and 1 and 5 min after cessation of exercise in the 8 × 50 yard test only. All samples were taken with the subject supine, but the swimmers did have to climb out of the pool and walk approximately 10 m to the laboratory. Samples were dispensed into lithium heparin and calcium heparin tubes. Blood pH was measured immediately using a pH blood gas monitor (Radiometer PHM 73 and BMS 3 Mk2, Copenhagen, Denmark). Haematocrit concentration was determined in triplicate using 30 μl microhaemotocrit tubes, which were centrifuged and read (Hawksley Micro-Haematocrit Instruments, UK). Haemoglobin was measured using the cyanmethaemoglobin method (Boehringer Mannheim, GmbH test-combination, Germany). Changes in plasma volume were estimated from the haemoglobin and haematocrit values using the method described by Dill and Costill (1974).

One millilitre of whole blood was centrifuged, the plasma supernatant drawn off and stored at –70 °C. Within 48 h, samples were thawed and assayed enzymatically for ammonia (Boehringer Mannheim, MPR 1 kit).

In all tests, duplicate 20 μl samples of blood were obtained pre-exercise and 1, 5, 10 and 15 min post-exercise from a small thumb-prick to determine lactate concentration. Blood samples were dispensed into tubes containing 200 μl of 2.5% perchloric acid, mixed and centrifuged for 3 min. The tubes were then stored at –20 °C and assayed enzymatically later using the method described by Maughan (1982).

Urinary analysis study

A subsequent study was carried out more than 6 months after the original supplementation period to determine the retention of creatine after supplementation with 9 g day^{-1} for 5 days. Six male subjects (three from the original creatine group and three from the original control group) followed an identical creatine supplementation regimen (see Supplementation period). All urine samples from the six subjects were collected over an 8 day period, the daily volumes recorded and aliquots taken from each and frozen for later analysis.

Urine was assayed enzymatically for creatine and creatinine using a commercially available kit (Boehringer Mannheim, MPR 2 Creatinine PAP). Creatine measurements were made by omitting the first creatininase step (Delanghe *et al.*, 1988) and read spectrophotometrically against standards of known concentrations. Total creatine excretion was corrected for the mean increase in creatinine in accordance with Rossiter *et al.* (1996).

Inter-assay coefficients of variation for repeated analysis on duplicate urine samples were 4.9% for creatine and 4.3% for creatinine.

Analysis of results

A three-way analysis of variance, or two-way where appropriate (Statistica/Mac), was used to examine differences between the control and creatine group (main effect of group), between all subjects before and after supplementation (main effect of trial) and to examine the response of all subjects over time (main effect of time). As the main effect of time was always statistically significant at the $P < 0.01$ level, this main effect is not referred to in the tables or figures. Differing responses between the groups as a result of supplementation were identified by group × trial and group × trial × time interactions. Values are presented as means ± standard deviation.

Results

Performance times

Mean times recorded for the single 50 yard sprint were 22.95 ± 0.51 s pre-treatment and 23.24 ± 0.70 s post-treatment for the creatine group, and 23.36 ± 0.50 s and 23.45 ± 0.58 s for the control group (creatine *vs* control, N.S.). While the performance times were not significantly different between groups or between treatments, the 50 yard times in the present study were significantly slower ($P < 0.01$) than personal best (PB) times for both groups (creatine PB, 22.26 ± 0.33 s; control PB, 22.26 ± 0.37 s). However, sprint 1 times in the repeated sprint test were the same as those achieved in the reference single sprint (single sprint time *vs* sprint 1 of 8 × 50 yards, N.S.).

During the repeated sprint test, mean times increased (repetitions 1 to 8) from 23.35 ± 0.68 s to 26.32 ± 1.34 s in the control group and from 23.20 ± 0.67 s to 26.85 ± 0.42 s in the creatine group before supplementation, and from 23.59 ± 0.66 s to 26.19 ± 1.48 s in the control group and from 23.39 ± 0.54 s to 25.73 ± 0.26 s in the creatine group after supplementation ($P < 0.03$, group × trial interaction; Fig. 2). The overall improvement was confirmed by the reduction in total sprint time in the creatine group after supplementation (204.3 ± 5.02 *vs* 200.2 ± 3.86 s), while the control group showed no improvement (204.7 ± 8.04 *vs* 204.3 ± 8.69 s; group × trial interaction, $P < 0.05$).

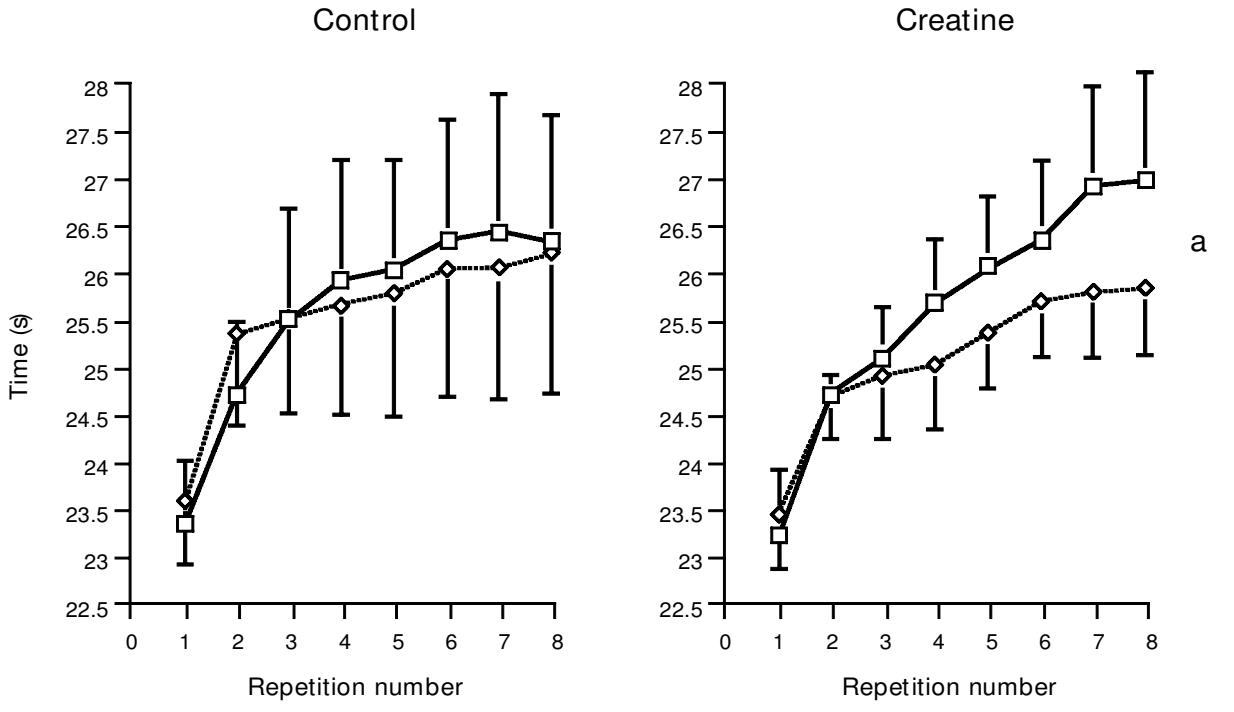

Figure 2 Mean performance times for the control and creatine groups, pre- and post-trial, in the 8 × 50 yard test (control, $n = 7$; creatine, $n = 7$; mean ± *s*). [a] $P < 0.03$ (group × trial interaction).

The percentage decline in performance times was reduced after creatine supplementation (control, 12.7 ± 5.7 *vs* 11.0 ± 5.5%; creatine, 15.7 ± 4.3 *vs* 10.0 ± 2.5%; $P < 0.05$, group × trial interaction; Fig. 3).

Metabolic responses

The metabolic responses of both groups in all tests are shown in Table 1. No significant differences were found in any metabolic variable between groups or treatments, although peak blood lactate concentration was significantly higher ($P < 0.01$) in the 8 × 50 yard test than the 1 × 50 yard test. In the creatine group, one subject recorded the same peak ammonia value pre- and post-creatine supplementation and the other six recorded lower values. Although the decrease in plasma ammonia was 53 μmol l^{-1} in the creatine group and ~39 μmol l^{-1} in the control group (main effect of trial, $P < 0.05$), these changes could not be attributed to supplementation (group × trial and group × trial × time interactions, N.S.).

Urinary analysis

Creatinine excretion was 1.63 ± 0.35 g over the first 3 days of collection and 2.21 ± 0.26 g during the 5 day period of supplementation ($P < 0.001$). No creatine was recovered in the urine during the first 3 days, but

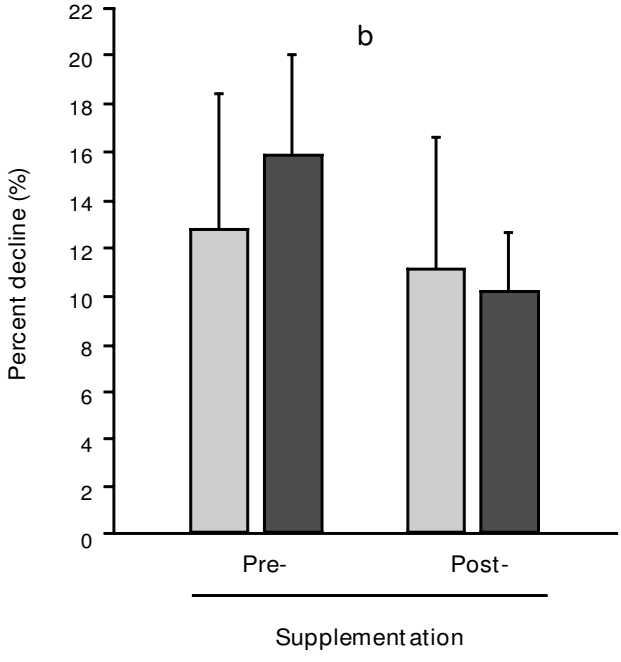

Figure 3 Percent decline in performance from repetition 1 to repetition 8 (8 × 50 yard test) for the control and creatine groups, pre- and post-trial (control, $n = 7$; creatine, $n = 7$; mean ± *s*). [b] $P < 0.05$ (group × trial interaction). ■, creatine; □, control.

Table 1 Peak (or lowest for blood pH) metabolic and heart rate responses to 1 × 50 yard and 8 × 50 yard sprint swims in elite swimmers (mean ± *s*)

	1 × 50 yard				8 × 50 yard			
	Control		Creatine		Control		Creatine	
	Pre-	Post-	Pre-	Post-	Pre-	Post-	Pre-	Post-
Heart rate (beats min^{-1})	176 ± 17	175 ± 15	180 ± 18	171 ± 9	186 ± 13	177 ± 10	188 ± 15	181 ± 17
Blood lactate (mmol l^{-1})	9.7 ± 2.4	9.6 ± 2.3	9.7 ± 1.1	9.3 ± 1.0	18.0 ± 1.9	16.8 ± 2.7	17.8 ± 3.0	17.3 ± 1.8
Ammonia (μmol l^{-1})*					217.9 ± 49.8	178.8 ± 51.1	227.5 ± 75.8	174.5 ± 20.0
Blood pH					6.98 ± 0.05	7.00 ± 0.03	6.98 ± 0.07	7.01 ± 0.04

Note: Plasma ammonia and blood pH were determined from venous samples. Blood lactate concentrations were determined from capillary samples for both trials, as venous samples were not taken during the single sprint swim (control, $n = 7$; creatine, $n = 7$). * $P < 0.05$ (main effect of trial).

rose from 1.32 ± 0.52 g on the first day of supplementation to 4.45 ± 1.03 g by the fifth day (Fig. 4).

Creatine uptake was highest on day 1 of supplementation (6.61 ± 0.52 g) and declined each day to 3.49 ± 1.04 g by day 5. Creatine retained was 86.2 ± 6.3% for day 1 and 45.9 ± 12.7% for day 5 of the intake value. The total amount of creatine retained was 26.1 ± 2.0 g or 66.9 ± 5.1% of the total administered over 5 days (Table 2). The range of creatine retention was 23.4–27.8 g or 59.2–71.5%.

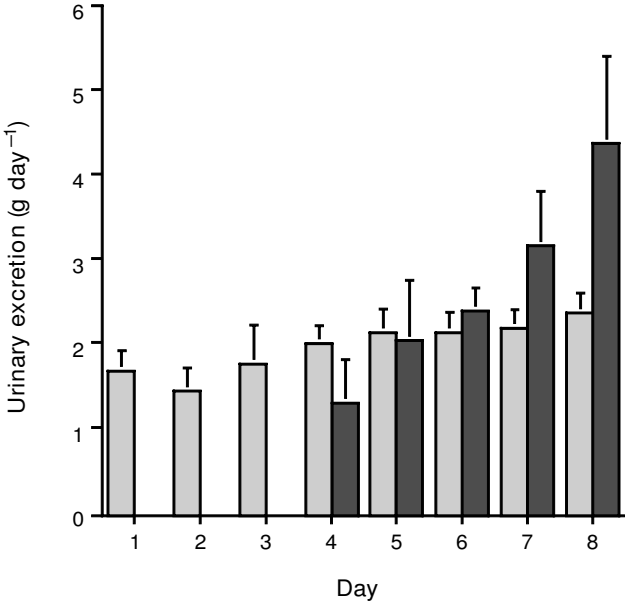

Figure 4 Urinary creatine and creatinine excretion over an 8 day period ($n = 6$). Creatine supplementation (9 g day^{-1}) took place on days 4–8. ■, creatine; □, creatinine.

Discussion

The main findings of the present study are that oral creatine supplementation at approximately half the dosage used in previous studies can improve swimming performance in elite competitors during repeated sprints, but appears to have no effect on a single 50 yard sprint. The metabolic responses to single and repeated sprints were similar, even though performance times in the repeated sprints test were faster after creatine supplementation.

The time recorded in the 1 × 50 yard sprint was used as a reference for the current sprint performance status of the swimmer. No significant differences were observed between the 1 × 50 yard time and the first repetition in the 8 × 50 yard set, an indication that a maximal sprint was recorded in both tests and that the swimmers were highly motivated. However, personal best times were significantly faster than the 1 × 50 yard time and the first repetition of the 8 × 50 yard set of sprints. This was probably due to lack of specific competition preparation; that is, a lack of

Table 2 Creatine uptake during creatine supplementation of 9 g creatine monohydrate (7.92 g creatine) per day for 5 days ($n = 6$; mean ± *s*)

Creatine uptake	Day 4	Day 5	Day 6	Day 7	Day 8	Total
% of intake	86.2 ± 6.3	74.8 ± 9.4	69.28 ± 3.3	58.8 ± 7.5	45.9 ± 12.7	66.9 ± 5.1
g	6.61 ± 0.52	5.85 ± 0.71	5.48 ± 0.26	4.69 ± 0.63	3.49 ± 1.04	26.12 ± 2.01
g kg^{-1}	0.086 ± 0.008	0.077 ± 0.012	0.072 ± 0.006	0.061 ± 0.011	0.046 ± 0.016	0.342 ± 0.046

'taper' and 'shaving down' (Sharp and Costill, 1989; Costill *et al.*, 1992). Improvements of 3–4% in performance time after a taper have been reported (Costill *et al.*, 1992).

The results of the present study support the findings of others who observed no differences in peak power output or performance in single exercise bouts: Balsom *et al.* (1993) in the first 6 s bout of maximal cycling exercise; Bogdanis *et al.* (1996) in the first 10 s treadmill sprint of a series; Cooke *et al.* (1995) in the first of two 15 s cycle ergometer sprints; Greenhaff *et al.* (1993b) in maximal isometric leg extensor torque; and Odland *et al.* (1994) in short-term maximal sprint cycle performance.

Although performance in a single bout of exercise appears to be unaffected by creatine supplementation in this and earlier studies, it has been shown that the total creatine content of skeletal muscle is increased by 20–50%, of which approximately 20% could be accounted for as phosphocreatine (Harris *et al.*, 1992). Increasing the phosphocreatine concentration of the muscle would appear, therefore, to have no effect on peak power production or performance in a single bout of short duration exercise, despite the view that the availability of phosphocreatine may be a limitation in the recovery of power output (Bogdanis *et al.*, 1995). An explanation for this observation may involve the speed of the creatine kinase reaction, which is, in part, determined by substrate concentration. At the start of exercise, the speed of this reaction will be close to maximum, and therefore to increase the substrate (phosphocreatine) concentration further will not affect the speed of this reaction and consequently peak power output and short-term swimming performance.

However, creatine supplementation did improve performance towards the end of the 8×50 yard test in this group of elite swimmers. As performance was improved, it seems possible that even 9 g creatine day^{-1} for 5 days may raise the muscle creatine content, thus providing a mechanism for the improvement. Evidence for this suggestion is provided by the urinary analysis data, which showed an approximate 67% (or ~26 g) retention of creatine over the 5 day period. The percentage retention of creatine was higher than in previous studies (15–32%, Greenhaff *et al.*, 1994b; 25–55%, Rossiter *et al.*, 1996), but the total amount of creatine retained and the estimated muscle creatine uptake (28.0 ± 3.8 mmol per kg dry weight), assuming a muscle mass estimate of 40% of body mass and muscle water content of 3.3 litres per 4.3 kg wet muscle weight (Harris *et al.*, 1992), were similar to the values reported by Rossiter *et al.* (~35 g and 38.1 ± 10 mmol per kg dry weight). In addition, a recent study has shown that creatine uptake into the muscle occurs even at lower dosages (3 g day^{-1}) after 14 days of supplementation (Hultman *et al.*, 1996). In the present study, uptake of creatine may have been facilitated by increased blood flow as a result of the supervised training (4–5 km per day) undertaken by the swimmers during the supplementation period. For example, Harris *et al.* (1992) found that subjects who performed single-legged exercise showed muscle creatine increases of approximately 44 mmol per kg dry weight in the exercised leg compared with approximately 30 mmol per kg dry weight in the non-exercised leg. Also, the administration of creatine together with glucose and maltodextrin close to meals with a high carbohydrate content may have aided muscle creatine uptake by raising serum insulin levels (Green *et al.*, 1995). Furthermore, these university students on low finance may have had a dietary deficiency of creatine. Thus it would appear that 9 g day^{-1} did increase muscle creatine content. Therefore, the mechanism of improvement in repeated sprints in the present study may have been that creatine concentration was maintained above the K_m value (~19 mmol l^{-1} assuming full activation) for the creatine kinase reaction, thereby increasing the rate of phosphocreatine resynthesis between sprints (Greenhaff *et al.*, 1994b).

The improvements in the 8×50 yard test may additionally have been caused by improved buffering through the increase in muscle creatine. Adenosine triphosphate resynthesis from ADP and phosphocreatine consumes a hydrogen ion (H^+) in the process. An increase in phosphocreatine turnover rate through greater creatine content in the muscle will therefore consume more H^+ and improve muscle buffering capacity. However, in the present study, the changes in blood pH were similar before and after supplementation.

Blood lactate values recorded in the single sprint were similar to those reported by Balsom *et al.* (1993), Bonifazi *et al.* (1993) and Greenhaff *et al.* (1993b), and slightly lower than those reported by Birch *et al.* (1994). In accordance with Birch *et al.* (1994) and Greenhaff *et al.* (1993b), blood lactate values in the present study were not significantly different after creatine supplementation. These findings contradict the significant decline in blood lactate after creatine supplementation found by Balsom *et al.* (1993), although the reductions observed in that study were small (10.8 to 9.0 mmol l^{-1}). In the 8×50 yard test, peak blood lactate values were higher than those of 9–15 mmol l^{-1} reported by Holmer (1972) and Nadel *et al.* (1974) for maximal swimming over 30 s to 5 min, but similar in range to those reported by Sawka *et al.* (1979) and Pelayo *et al.* (1996) in repeated swimming sprints. Creatine supplementation did not alter the blood lactate response, even though sprint swimming performance was improved, thus suggesting that the higher rate of ATP resynthesis

required for the faster swims was achieved without any change in the contribution from glycolysis to energy supply.

Plasma ammonia values, reflecting adenosine monophosphate (AMP) deamination, measured in the 8×50 yard test, may provide further information regarding the mechanisms responsible for the improvements in performance observed in the creatine group during the later repetitions (Banister *et al.*, 1985; Greenhaff *et al.*, 1993b). This reaction is accelerated in extreme, high-intensity exercise bouts (such as in this study) to support high ATP turnover rates. Although plasma ammonia values were lower post-supplementation, there was no difference between the groups. Large inter-individual differences may have caused this effect. In addition, the smaller creatine dosage used in the present experiment may not have exaggerated the change in purine metabolism to the same extent as in other studies (Balsom *et al.*, 1993; Greenhaff *et al.*, 1993b; Bogdanis *et al.*, 1996). However, six of seven subjects in the creatine group showed a decline in plasma ammonia after supplementation. This, together with an improvement in performance times in repetitions towards the end of the 8×50 yard test, provides support for high ATP turnover rates from sources other than AMP deamination, most probably from an increased contribution from phosphocreatine degradation as a result of increased phosphocreatine resynthesis.

In summary, the findings of this study suggest that oral creatine supplementation (9 g day^{-1} for 5 days) does not affect the performance of elite swimmers over 50 yards but does improve performance during a repeated sprint swimming set (8×50 yards). Evidence for creatine supplementation as the cause of performance improvements is provided from the urinary analysis results, which showed an approximate 67% (~26 g) retention of the administered creatine. The most likely mechanism to account for the performance improvement is an increase in muscle creatine content leading to increased phosphocreatine resynthesis and thus higher pre-exercise muscle phosphocreatine content in the latter sprints of the 8×50 yard set.

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