Effect of Creatine Supplementation on Training for Competition in Elite Swimmers

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ABSTRACT

PEYREBRUNE, M. C., K. STOKES, G. M. HALL, and M. E. NEVILL. Effect of Creatine Supplementation on Training for Competition in Elite Swimmers. *Med. Sci. Sports Exerc.*, Vol. 37, No. 12, pp. 2140–2147, 2005. **Purpose:** The study was conducted to examine the effects of oral creatine supplementation on training for competition in 20 elite swimmers. **Methods:** Subjects performed a maximal sprint test (8 × 50 yd (45.72 m), T1) before loading with creatine (Cr, 20 g·d^{-1} Cr monohydrate for 5 d), 1 wk later (T2), and following a 22- to 27-wk period of training and competition (T3). Following T2, subjects supplemented with either Cr (3 g + glucose 7 g·d^{-1}) or placebo (glucose 10 g·d^{-1}; double blind) for the remainder of the 22- to 27-wk season and then both groups supplemented once more with 20 g·d^{-1} Cr monohydrate for 5 d before their major competition. Venous and capillary blood samples were obtained pre- and posttest during the repeated sprint tests to determine blood metabolites and hormones. Competition times were recorded, and changes in subjects' best times were used to compare the effect of training and supplementation on competitive performance. **Results:** Mean competition times in the Cr and control groups changed by +1.90 ± 1.91 and +0.72 ± 1.64% for short course (SC, 25-m pool) and by +0.14 ± 1.14 and −0.59 ± 0.82% long course (LC, 50-m pool), respectively (Cr vs control, NS). No differences between groups were found in blood metabolites, although the human growth hormone (hGH) response to repeated sprints was blunted following Cr loading (T1, 30.42 ± 14.60 and 28.95 ± 18.27 μg·L^{-1}; T2, 21.48 ± 13.96 and 14.24 ± 7.32 μg·L^{-1} for Cr and control groups, respectively $P < 0.05$). **Conclusion:** No statistically significant differences in performance were observed between groups after long-term maintenance during training, although small differences were observed that might be meaningful for elite performers. **Key Words:** SPRINT SWIMMING, BLOOD METABOLITES, HUMAN GROWTH HORMONE, INSULIN-LIKE GROWTH FACTOR-I

Oral creatine (Cr) supplementation has been demonstrated to increase resting concentrations of Cr and phosphocreatine in skeletal muscle (7,9), and to improve performance during intermittent high-intensity exercise through an increased recovery rate (1,5). Improvements in performance, however, have not been identified during single sprint bouts (1,6) or in elite competitive swimmers at 25-, 50-, and 100-m race distances (13). Swimming performance in repeated sprints has been found to improve following Cr supplementation (8,16). The benefit of this ergogenic effect in swimming is likely to be in enhancing training, where repeated bouts of high-intensity swimming are regularly performed, rather than in competition, where a single sprint performance prevails.

Ten weeks of strength training combined with Cr supplementation has been shown to result in an enhanced gain in high-intensity, intermittent isokinetic arm flexion exercise capacity in previously sedentary subjects compared with a control group (28). In contrast, a recent study did not identify any differences in the performance of elite swimmers following a Cr "loading" period compared with either a low dosage "maintenance" period or no supplementation (27). This study, however, did not investigate the physiologic responses of Cr supplementation to training and was conducted over a relatively short, 8-wk period. To date, no longer term (>10 wk) training studies have investigated the physiologic effects of oral Cr supplementation in elite swimmers.

Sprint exercise has been shown to be a potent stimulus for human growth hormone (hGH) secretion (14,25,26), whereas 18 wk of swimming training has been found to enhance the hGH response following an endurance training set (2). Acute Cr supplementation (a single 20-g dose) has also been shown to elicit an hGH response in a similar manner to that observed following exercise (21). The addi-

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Submitted for publication November 2004.
Accepted for publication July 2005.

0195-9131/05/3712-2140/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/01.mss.0000179101.38913.73

tion of acute Cr administration to either maximal, submaximal (29), or resistance (15) exercise, however, does not appear to alter the hGH response to exercise, and it has been reported that short-term Cr supplementation does not alter the hGH response to exercise (15). The effects of a longer training period (>8 wk) combined with a commonly used Cr supplementation regimen (e.g., 20 g·d^{-1} for 5 d) on the hGH response to sprint swimming, or markers of 24-h hGH secretion, has not yet been considered.

This study was conducted to determine the effects of two oral Cr supplementation regimens during 22–27 wk of training for competition on swimming performance and metabolic and hormone responses to a sprint swimming set in elite swimmers. It is hypothesized that maintaining a Cr dose of 3 g·d^{-1} over a 7-month training period will enhance training and, consequently, improve performance.

METHODS

Subjects. Twenty-three swimmers (14 men, 9 women) from Loughborough University swimming club began the training study. Three subjects dropped out or their results were disregarded during the course of the 28-wk training period because of injury (2 men) and illness (1 woman), leaving 12 male and 8 female swimmers. Further illnesses observed were minor, with less than 2 d of training disruption at any stage. All subjects were involved in a regular training and competitive program (8–10 swimming sessions per week) and were British National Championship medalists or had swum at senior or junior international level in the previous 12 months. Subjects were informed of the purpose of the study and any known risks involved before giving their written consent to participate. The protocol was approved by the ethical committee of Loughborough University who were satisfied that no known or reported side effects would occur from Cr supplementation at the doses (20 g·d^{-1} of Cr for 5 d, or 3 g·d^{-1} for ≤ 196 d) prescribed for this study (22).

Swimmers had regular experience of competition swimming and were familiar with the sprint and endurance tests used in this study. All subjects undertook initial sprint and endurance tests, followed by a period (20 g·d^{-1} Cr monohydrate + 20 g·d^{-1} glucose for 5 d) of Cr supplementation and then repeated the sprint test. Subjects were then assigned (initially matched for time, event, and standard) to an experimental (Cr supplementation, Cr; $N = 9$) and control (glucose; $N = 11$) group. Mean ± SD ($N = 20$) subject characteristics for Cr and control groups were: age, 20 ± 1 and 20 ± 2 yr; height, 180.2 ± 11.1 and 179.1 ± 9.4 cm; body mass, 74.7 ± 15.2 and 73.7 ± 11.6 kg, respectively (all NS).

Personal best times were all recorded within the previous year in a 25-m pool. Personal best times for 100-m freestyle were 52.6 ± 1.3 and 54.2 ± 1.8 s for men, and 57.9 ± 1.8 and 58.5 ± 1.8 s for women in the Cr and control groups, respectively.

Experimental procedures and protocol. Subjects performed five testing sessions during the course of the swimming season: On two occasions a typical speed-lactate swimming endurance test (ET) comprising 5 × 200 yd (182.88 m) incremental repeats (adapted from (17)), and on three occasions a maximal sprint training set of 8 × 50 yd (45.72 m) (T; (16)). Test sessions took place before (both tests, T1 and ET1) and after a 5-d supplementation period (8 × 50-yd test only, T2), and both tests (T3 and ET2) after a 22- to 27-wk training period that culminated in the GB World Championship Trials or other appropriate event within a 2-wk period and after a second 5-d supplementation period. The supplementation period involved two high-dosage 5-d loading phases and a low-dosage 22- to 27-wk maintenance phase.

Repetitions during the 8 × 50-yd test took place with a dive from racing blocks and were timed from an official start. Subjects were asked to exert maximal effort on each repetition, while maintaining a constant stroking technique. The repeats used in the speed-lactate test were performed from a "push" start in the water and were incremental beginning at approximately 75% of personal best pace and increasing in even stages to maximal effort on the final 200 yds. "Antiwave" lane ropes were used to divide the lanes to reduce unnatural water resistance as they would be in competition. Experienced timekeepers using chronograph stopwatches recorded swimming times in duplicate during the testing sessions. Competition data were recorded electronically at all competitions between October 2000 and April 2001.

Subjects were required to refrain from consuming alcohol for the day before all tests, and performed only light exercise (4000–5000 m·d^{-1} swimming training) in the 2 d before each test. All test sessions took place in the Sports Science Research Laboratories and Sports Hall swimming pool (25 yd) at Loughborough University. Average pool temperature was 29 ± 1°C. A standardized warm-up (25 min) was performed in preparation for each test. Repeated tests were carried out at the same time and same day of the week. The timing of the test protocol is illustrated in Figure 1.

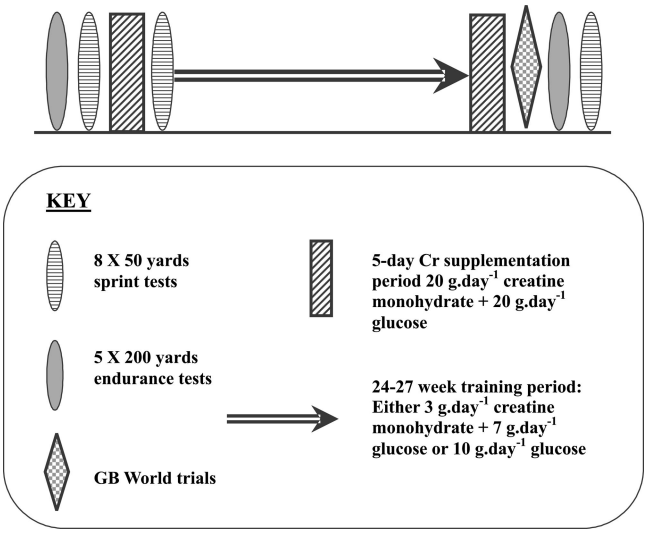

FIGURE 1—Schematic illustration of the training, competition and test protocols.

Supplementation. All subjects were provided with 20 premeasured supplement packets (5 g Cr (i.e., Creatine, Optima Health Care, Cardiff, UK) + 5 g glucose) and instructed to mix the powder in hot water and cordial for immediate consumption at 9:00, 12:00, 15:00, and 18:00 h for each of the 5 d during the loading supplementation period. Immediately following T2, subjects were assigned (using a double-blind protocol) to either a Cr (3 g Cr + glucose 7 $g \cdot d^{-1}$) or placebo (glucose 10 $g \cdot d^{-1}$) group. Subjects consumed one packet per day at 12:00 h every day for a period of 22–27 wk. Total intake of Cr by the Cr group over this period was ≤ 546 g. Subjects were given a 1-wk supply of packets clearly labeled and a checklist grid to complete after taking each packet. This compliance grid was checked by the researchers on a weekly basis, along with any comments regarding health. During the loading phases only, some subjects reported feeling bloated or had some gastrointestinal disturbance.

One week before the GB World Championship Trials (or relevant alternative event), all subjects were again provided with 20 premeasured supplementation packets (as above) and conducted a similar loading regimen. The only difference between the groups, and therefore the intervention, was the maintenance Cr dose or placebo during the training period.

Equipment and measurements. Height and body mass (and, where possible, skinfold measurements (4)) were measured on each visit to the laboratory in swimming attire. Heart rate was recorded at rest and immediately following (within 3 s of completion) each repetition during all exercise phases of the investigation by short-range telemetry (Polar Electro PE3000 Kempele, Finland). A transmitter was held to the chest, level with the sternum, and contact between electrodes and skin was ensured. Heart rate ($beats \cdot min^{-1}$) was recorded from the receiver's display within 3–5 s of contact.

Blood collection and analysis. In the 8 × 50-yd tests, antecubital venous blood samples (10 mL) were obtained at rest before the standardized warm-up, and 1 min after cessation of exercise. All samples were taken with the subject supine and postexercise samples were obtained after the swimmers had climbed out of the pool and walked approximately 20 m to the laboratory. Samples were dispensed into plain, lithium heparin and calcium heparin tubes. Blood pH was measured immediately using a pH blood gas monitor or analyzer (Radiometer ABL 5, Copenhagen, Denmark). Hematocrit (Hct) concentration was determined in triplicate using 30-μL microhematocrit tubes, which were centrifuged and read (Hawksley Micro-Hct Instruments). Hemoglobin (Hb) was measured using the Cyanmethaemoglobin method (Boehringer Mannheim, GmbH Test-combination, Mannheim, Germany). Changes in plasma volume were estimated from the Hb and Hct values using the method described by Dill and Costill (3).

Whole blood (1 mL) was dispensed into a calcium heparinized tube, centrifuged, and the plasma supernatant was stored at −70°C. Within 48 h, samples were thawed and assayed enzymatically for ammonia (Sigma Diagnostics, 171-C Ammonia Kit). An aliquot of the venous blood sample (4–5 mL) was allowed to clot for 30 min in the plain tube. This was then centrifuged, and the serum stored at −70°C for later analysis of hGH and IGF-1. The serum was analyzed for hGH by routine enzyme-linked immunosorbent assay (ELISA) (Biosource Cytoscreen, Nivelles, Belgium) that had a sensitivity of 0.11 $mU \cdot L^{-1}$ and intra- and interassay coefficients of variation for repeated analysis of 2.1–3.6% and 6.8–7.1%, respectively. The IGF-1 analysis was carried out by routine ELISA (R&D Systems Europe) that had a sensitivity of 0.026 $ng \cdot L^{-1}$ and intra- and interassay coefficients of variation for repeated analysis of 3.5–4.5% and 7.5–8.3%, respectively.

In the 8 × 50-yd test, duplicate 20-μL samples of blood were also obtained pre- and postexercise from a thumb prick to determine blood lactate concentration. Blood samples were dispensed into tubes containing 200 μL of 2.5% perchloric acid, mixed, and centrifuged for 3 min. Tubes were then stored at −20°C and assayed enzymatically using the method described by Maughan (12). Intraassay coefficients of variation for repeated analysis were 1.9–2.6% for blood lactate concentration.

Analysis of results. A three-way analysis of variance (ANOVA) or two-way ANOVA with repeated measures (SPSS) was used, where appropriate, to examine differences between the control and Cr group (main effect group), between all subjects during T1, T2, and T3 (main effect trial) and to examine the response of all subjects over time (main effect time). For significant *F*-ratios, a paired Student's *t*-test was used to determine the cause of the variance using the *Bonferroni* correction. Pearson product moment correlation was used to identify any relationship between variables. Differing responses between the groups as a result of supplementation were identified by group-trial and group-trial-time interactions. Practical significance curves (12) were also generated for performance times by calculating confidence limits for each possible size of benefit of one intervention over the other (i.e., Cr vs control). Values are presented as mean ± standard deviation (SD) and significance set at the $P < 0.05$ level. Changes in performance time are presented as a positive (faster) or negative (slower) percentage in the results.

Performance data for all subjects were collected, although because of one subject dropout, one subject's fear of needles, and spoilt samples, metabolic and hormone data were not available for all subjects. The number of subjects included for each analysis for both groups are detailed in the table and figure legends.

Because the main effect—time—was always statistically significant at the $P < 0.01$ level (except for two repetitions during the 8 × 50-yd test), this main effect is not referred to in the tables or figures. Times have been combined for male and female subjects to avoid small group sizes ($N = 4$), because statistically, no gender-group differences were observed.

RESULTS

Body composition. Body mass for T1, T2, and T3 was 75.0 ± 15.5, 74.8 ± 15.5, and 75.6 ± 15.6 kg for the Cr group ($N = 9$), and 73.7 ± 11.7, 73.7 ± 11.4, and 72.9 ±

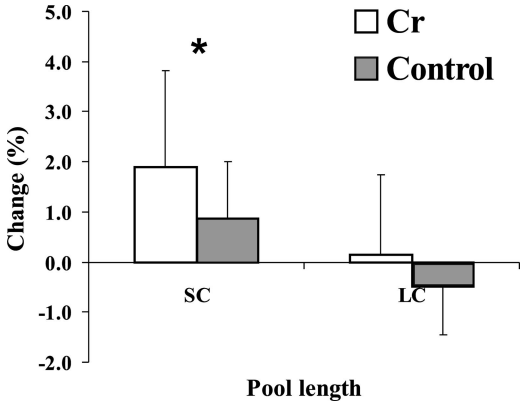

FIGURE 2—Competition best-event performance changes conducted throughout the study (Cr $N = 9$; control $N = 11$; mean ± SD). * $P < 0.05$ Short course pre- vs posttraining.

9.8 kg for the control group ($N = 11$) (group and trial main effects, NS). The four-site sum of skinfolds was 36.3 ± 13.1, 36.7 ± 12.8, and 37.9 ± 11.5 mm for the Cr group, and 50.1 ± 17.5, 50.6 ± 17.1, and 47.1 ± 9.4 mm for the control group in the three trials, respectively (group × trial interaction, NS).

Training and competition. No differences in training volume were observed between groups throughout the 22- to 27-wk period (Cr 31,150 ± 11,750 vs control 30,050 ± 11,950 yd·wk^{-1}, NS). These values include periods of taper and the Christmas holiday where training volumes were significantly reduced.

Mean performance times for the subjects' best event in the Cr and control groups changed by +1.90 ± 1.91 and +0.86 ± 1.60% for short course (SC, 25-m pool), and by +0.14 ± 1.14 and −0.49 ± 0.95% for long course (LC, 50-m pool), respectively (SC, pre- vs posttraining, $P < 0.05$; group × trial interaction and LC times, NS, Fig. 2). Figure 3 shows the practical significance curve for LC times to indicate the level of probability associated with improvements in performance when comparing Cr and control. For LC performances, there is a 90% likelihood of some benefit (i.e., any improvement in performance) as a result of maintenance of Cr supplementation.

Performance times. Performance times recorded for the first repetition in the 8 × 50-yd sprint test were 23.88

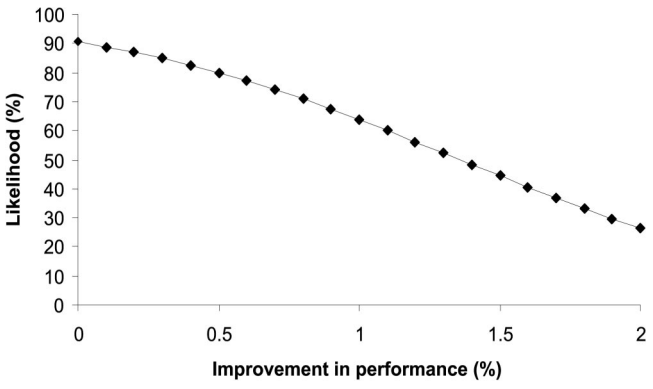

FIGURE 3—Practical significance curve for long-course competition performances.

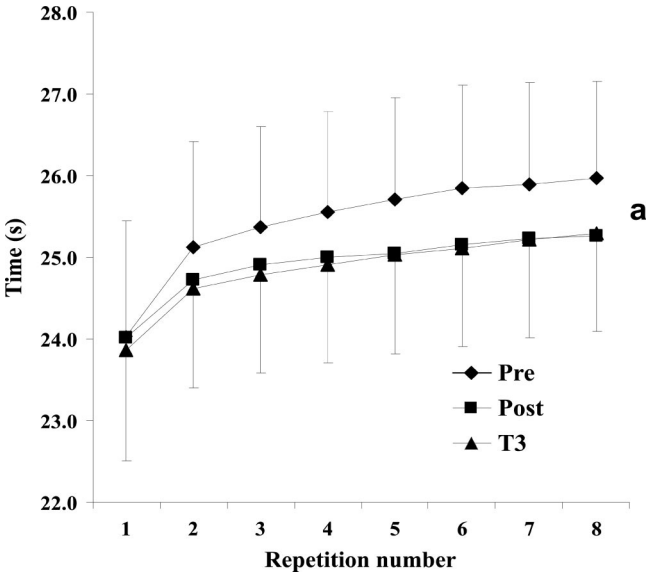

FIGURE 4—Mean performance times for all subjects in the three 8 × 50-yd tests ($N = 20$; mean ± SD). a. $P < 0.05$ T1 vs T2 and T3.

± 1.68 s in T1, 23.84 ± 1.50 s in T2, and 23.70 ± 1.58 s in T3 for the Cr group, and 24.20 ± 1.09, 24.22 ± 1.34, and 24.00 ± 1.20 s for the control group (group and trial main effects, NS, Figs. 4 and 5).

During the repeated sprint test, mean times were faster in all subjects (repetition number 1 to 8) by +8.1 ± 3.1, +5.3 ± 2.5, and +6.0 ± 2.8% in T1, T2, and T3, respectively (T1 vs T2 and T3, $P < 0.05$: Fig. 4). Total sprint time for all subjects was 203.5 ± 9.9 s in T1 versus 199.2 ± 9.5 s in T2 and 198.8 ± 9.6 s in T3 ($P < 0.05$: Fig. 4).

No differences in faster performance times (repetition 1 to 8 during the 8 × 50-yd test) between Cr and control groups were observed (Cr +8.2 ± 2.8, +5.3 ± 2.2, and

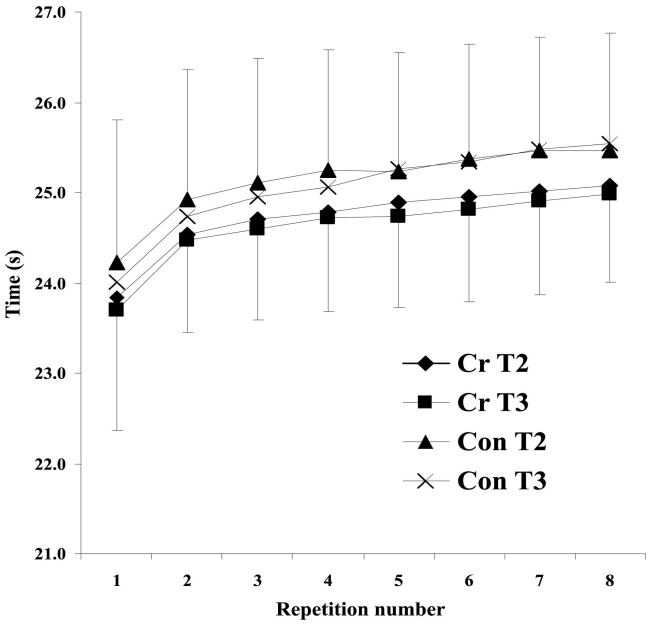

FIGURE 5—Mean performance times for Cr and control groups pre- and posttraining in the 8 × 50-yd tests (Cr $N = 9$; control $N = 11$; mean ± SD).

TABLE 1. Postexercise hormone, metabolic, and heart rate responses to 8 × 50 yd of sprint swimming in elite swimmers (hGH: Cr $N = 7$, control $N = 8$; capillary blood and heart rate Cr $N = 9$; control $N = 11$; all other metabolites Cr $N = 8$; control $N = 11$; mean ± SD).

	8 × 50 yd					
	Control			Creatine		
	T1	T2	T3	T1	T2	T3
Heart rate (beats·min^{-1})	184 ± 7	185 ± 6	183 ± 8	185 ± 9	185 ± 7	182 ± 8
Capillary blood lactate (mmol·L^{-1})	14.5 ± 2.1	15.0 ± 2.3	15.1 ± 4.2	14.6 ± 2.2	15.0 ± 2.1	14.4 ± 2.2
Venous blood lactate (mmol·L^{-1})	14.3 ± 2.4	14.9 ± 2.7	15.4 ± 4.6	14.8 ± 2.3	15.1 ± 2.3	15.5 ± 2.0
Ammonia (μmol·L^{-1})	200.7 ± 56.0	193.7 ± 67.8	213.7 ± 94.9	226.7 ± 48.7	213.5 ± 63.9	192.9 ± 85.3
Blood pH	7.10 ± 0.09	7.07 ± 0.10	7.07 ± 0.11	7.07 ± 0.06	7.07 ± 0.09	7.06 ± 0.04
Hemoglobin (g·dL^{-1})	15.1 ± 1.0	15.3 ± 0.9	15.7 ± 2.2	15.2 ± 1.3	15.3 ± 1.2	15.8 ± 1.4
Hematocrit (%)	46.1 ± 2.6	47.2 ± 2.1	47.4 ± 2.1	46.8 ± 3.6	47.5 ± 3.1	47.8 ± 4.1
Plasma volume (% change)	−2.3 ± 2.0	−4.3 ± 2.4	−5.9 ± 5.0	−3.2 ± 1.9	−4.6 ± 2.0	−6.9 ± 4.6

+5.5 ± 3.0%; control +8.1 ± 3.5, +5.3 ± 2.8, and +6.5 ± 2.6% for T1, T2, and T3, respectively, NS, Fig. 5). Values for T2 and T3 only for Cr and control groups have been represented in Figure 5 for clarity.

Endurance tests. Speeds corresponding to a fixed blood lactate concentration of 4 mmol·L^{-1} (V4) changed between tests from 1.57 ± 0.05 and 1.58 ± 0.05 yd·s^{-1} to 1.60 ± 0.11 and 1.54 ± 0.09 yd·s^{-1} for Cr and control groups, respectively (Cr vs control, NS). The change in values represents changes of +1.54 ± 5.56% in the Cr group and −2.33 ± 4.78% for control subjects. A significant ($P < 0.05$) correlation was found between the changes in V4 values and SC (25 m) times ($r = 0.50$), but not between V4 and LC (50 m) changes ($r = 0.28$).

Metabolic and hormone responses. Metabolic and cardiovascular responses of both groups in all tests are shown in Table 1. No significant differences were found with any variable between groups or treatments. Hb and Hct are shown as metabolites are reported uncorrected for changes in plasma volume. Plasma ammonia and blood pH are included to indicate the extent of the metabolic stress following repeated maximal repeated sprinting.

Serum growth hormone (GH) concentrations (Fig. 6) increased significantly from rest to postexercise in all tests ($P < 0.01$). In T1, T2, and T3, postexercise serum GH concentrations were 29.0 ± 18.3, 14.2 ± 7.3, and 22.6 ± 17.8 μg·L^{-1}, and 30.4 ± 14.6, 21.5 ± 14.0, and 23.8 ± 16.2 μg·L^{-1} for Cr ($N = 7$) and control ($N = 8$) groups, respectively (test × time interaction effect $P < 0.05$: Fig. 6). Resting serum IGF-I concentrations in T1 and T3 were 117 ± 21 and 113 ± 22 ng·mL^{-1} and 114 ± 31 and 107 ± 21 ng·mL^{-1} for Cr ($N = 8$) and control ($N = 10$), respectively (group and trial main effects, NS).

DISCUSSION

The main finding of the present study was that 22–27 wk of oral Cr supplementation at maintenance levels during training does not statistically significantly improve competitive swimming performance or performance during a repeated sprint test in elite competitors more than a 5-d Cr loading regimen. All subjects improved their repeated sprint performance after an initial loading phase, but those who continued to ingest Cr failed to improve their competitive performances statistically significantly more than the control group. The small differences identified between the groups, however, may be of practical significance to the elite competitor. Cr loading resulted in an apparent attenuation of GH response to repeated sprints, but longer term maintenance of Cr supplementation did not appear to have any effect on the hGH response to exercise. Metabolic and cardiovascular responses to repeated sprints were similar before and after training in both the Cr and control groups.

All subjects improved repeated sprint performance as a result of the initial loading phase, in agreement with previous findings (16,27). Because of the design of the present study, however, it is possible (but unlikely on the basis of previous work, 16) that this improvement was a result of a placebo effect, because all athletes knew that they were following a period of Cr loading. The main focus of this study was the effect of longer term maintenance of Cr supplementation on performance and, therefore, these results are of more interest. During the 22–27 wk of the study, subjects improved their best SC times by +1.90 ± 1.91 and +0.72 ± 1.64% in the Cr and control groups, and their best LC times changed by +0.14 ± 1.14 and −0.49 ± 0.95%, respectively. An improvement of +1.9% reflects approximately 1 s faster 100-m freestyle performance time in this group of swimmers, the equivalent of the difference between first and sixth place in the 2004 Olympic 100-m freestyle finals (men, 48.17 vs 49.23; women, 53.84 vs 54.81). Despite potential masking of results, the net difference in LC performances between the groups of 0.73% is

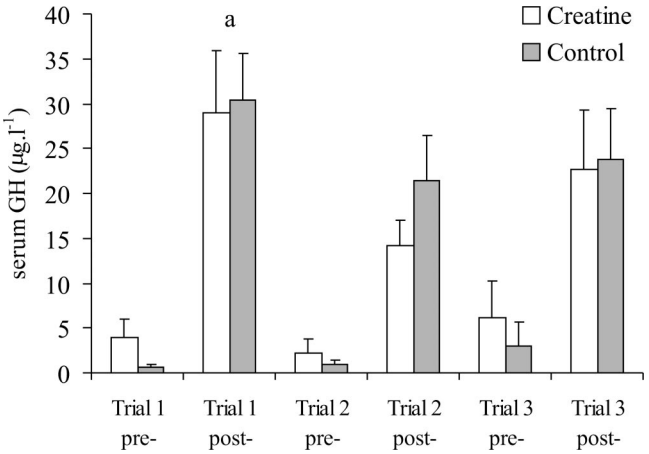

FIGURE 6—Human growth hormone responses to 8 × 50-yd sprints pre- and postcreatine supplementation and posttraining for Cr and control groups (Cr $N = 7$; control $N = 8$; mean ± SD). a. $P < 0.05$ T1 vs T2 and T3.

substantial, particularly because the outcome of international races is often determined by a few hundredths of a second.

Short-course swimming is characterized by a greater proportion of the race time spent pushing off the wall using near-maximal leg extensions, and may have been more familiar to these swimmers who train in this length of pool. The improvements were small, but reflect the difficulty that elite swimmers have when continually trying to improve as they reach their own maximal performance levels (17). It is likely that the training *per se* will lead to greater improvements than supplementing with Cr without carrying out any training, and these effects may disguise more subtle changes between groups in this study.

The likelihood of improvements in performance times as a result of Cr maintenance is shown by the practical significance curve in Figure 3. The generation of these practical significance curves allows independent assessment of probability associated with the benefits of maintaining Cr supplementation (23). For LC times, a 90% likelihood exists that maintaining Cr results in any improvement in performance, whereas an approximately 64% likelihood of a performance improvement of 1% and an approximately 26% likelihood that Cr maintenance results in a performance improvement of at least 2%.

In this group of elite swimmers, subjects responded to loading supplementation (T1 to T2), but not to additional maintenance supplementation during 22–27 wk of training. Lemon et al. (11) suggested that 35 d after Cr supplementation, some subjects may still have elevated muscle TCr levels. This can occur if a high meat or fish diet is consumed following the supplementation period (although this was not monitored in the present study). If some of the subjects in the control group in this study had elevated muscle TCr levels for 4–8 wk following supplementation, they might have received similar potential benefits as the Cr group, although at least 14 wk remained during which the Cr group could have gained benefits from the maintenance protocol.

Analysis of the speed-lactate test results (5 × 200-yd endurance test) showed a large interindividual variation in the change in endurance training status (represented by V4: the swimming speed corresponding to a blood lactate concentration of 4 mmol·L^{-1}) over the course of the study. This metabolic index has previously been used to describe seasonal changes during competitive swimming periods (17) and is strongly correlated to performance in endurance swimming events (19). It was expected that most subjects would improve their V4 during the course of the season and, although some subjects improved, many others did not (Cr +1.54 ± 5.56%, control −2.33 ± 4.78%, NS). Although every effort was made to test swimmers as soon as possible after the main competition, some swimmers may have spent 3–4 wk in a state of reduced training before their main event (a normal swimming taper) followed by little or no training for 7–10 d. This may have resulted in a reduction in endurance capacity.

No significant differences were observed in blood metabolites between the training Cr and control groups. Balsom et al. (1) have observed lower blood lactate and plasma ammonia concentrations during maximal exercise following Cr supplementation, suggesting a reduction in energy contribution from anaerobic glycolysis and an increase from either aerobic metabolism or PCr degradation. This effect has been observed following training, although the second loading supplementation may have masked any differences between the groups. Adenosine triphosphate resynthesis from adenosine diphosphate (ADP) and PCr consumes a hydrogen ion (H^+) during the reaction. Blood pH values were similar between the groups, suggesting again that Cr supplementation has a greater impact on this aspect of metabolism than any benefits gained from training.

In the present study, postexercise serum hGH concentrations were similar to those found in other studies, with mean values slightly higher than those reported by Stokes et al. (25) following a 30-s cycle ergometer sprint (15–25 $\mu g \cdot L^{-1}$), but lower than in male sprint-trained athletes following a 30-s treadmill sprint (35–50 $\mu g \cdot L^{-1}$ (14)). The hGH response showed greater interindividual differences with ranges of approximately 5–52 $\mu g \cdot L^{-1}$, approximately 6–42 $\mu g \cdot L^{-1}$, and approximately 2–55 $\mu g \cdot L^{-1}$ in T1, T2, and T3. This range in values was similar in both male and female swimmers and was not related to sprint or endurance specialization. This is at odds with previous reports (14), although many of the swimmers were neither sprint nor endurance trained, and would fall into a category between those proposed by Nevill et al. (14). Previous studies have demonstrated great interindividual variation in the hGH response to exercise, with a large range of peak hGH concentrations and time to peak concentrations in addition to differences in total release (18,26). In the present study, because of the elite nature of the subjects, only one blood sample was taken approximately 1 min postexercise, which provides only limited information about the hGH response. The postexercise hGH value was recorded, however, at +1 min after the 8 × 50-yd test, which represents a time point approximately 13 min following the first repetition and is sufficient time for serum hGH concentrations to rise.

Previous studies considering the effect of training on the GH response to exercise have provided equivocal results, with reported increases (2), decreases (24,30), and no change (10) in postexercise hGH concentrations following training. The present study demonstrated a blunted hGH response to repeated sprints following 1 wk of Cr supplementation. Whether this was caused by a learning effect of the test, the training undertaken during the week, the Cr supplementation, or a combination of these factors is not clear. Following a longer period of training (22–27 wk), however, the hGH response was partially restored toward values similar to those in the presupplementation trial. A total of 18 wk of mixed training in elite swimmers has been found to augment the hGH response to exercise (2) and, if T2 is taken as the base line in the present study, some agreement exists in the findings of the two studies. When considering the effect of training in the present study from T1 to T3, however, a significant decrease was seen in the hGH response to exercise following training (Fig. 6), which

is in agreement with the findings of Weltman et al. (30) and Stokes et al. (24).

Schedel et al. (21) observed an hGH response following a single 20-g ingestion of Cr, without exercise, which they suggested might be caused by an increased intracellular ADP concentration. A hGH response is observed following exercise and linked to the adenylate kinase (AK) reaction and the activation of creatine kinase (CK) (21), although similar ammonia values (despite different hGH concentrations) before and after supplementation in the present study do not support this mechanism. The hGH responses following Cr supplementation (21), however, were considerably smaller than those observed in the present study, and acute administration of Cr before exercise does not alter the hGH response to exercise (15,29).

An apparent attenuation of the hGH response to exercise was found after 1 wk of training combined with Cr loading. In contrast, previous research has shown that 5 d of Cr supplementation at a similar dose to that in the present study has no effect on the hGH response to resistance exercise (15). The reason for this disparity in findings is not clear, although in the present study both groups carried out the initial Cr loading phase, making it difficult to draw conclusions. Of more importance in the present study is the effect of long-term training combined with maintenance of Cr administration. Although, as mentioned, the 22–27 wk of swim training attenuated the hGH response to exercise, and no effects were seen of long-term maintenance of Cr supplementation on postexercise hGH concentrations.

Circulating IGF-I concentrations have been suggested as an appropriate measure of integrated 24-h hGH secretion (20). Only 2 wk of endurance training increased plasma IGF-I values (20), suggesting that short-term training results in increased integrated 24-h hGH secretion. The present study is the first to consider the effect of a longer period of training, combined with Cr supplementation, in elite athletes on resting serum IGF-I concentrations. These did not change, suggesting that 22–27 wk of training combined with Cr supplementation, whether via discrete loading phases or loading phases as well as maintenance, does not alter integrated 24-h hGH secretion.

In summary, the findings of the present study confirm that oral Cr supplementation (20 $g \cdot d^{-1}$ for 5 d) improves repeated sprint swimming performance, but that an additional supplementation protocol during 22–27 wk of training does not significantly enhance the competitive performance of elite swimmers to a greater extent than training alone. Despite this, small differences between groups identified in this study suggest that elite level swimmers may derive practically important benefits from Cr maintenance during training. This prolonged period of Cr supplementation had no effect on the hGH response to exercise.

The authors would like to acknowledge the assistance of Antonis Tsampoukos, Paul Cherry, Gabbi Scanlon, Spencer Newport (all Loughborough University) and Paul Whitaker (Leicester Royal Infirmary) in the collection and analysis of data in this study. Sincere thanks to all the National and International swimmers at Loughborough University who participated in this study and to their coaches Ian Armiger and Ben Titley for their co-operation throughout.

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