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THE EFFECTS OF CREATINE SUPPLEMENTATION ON SPRINT RUNNING PERFORMANCE AND SELECTED HORMONAL RESPONSES

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ABSTRACT

The purpose of this study was to determine the influence of short-term creatine supplementation on sprint running performance (100 and 200 m) and circulating hormone [growth hormone (GH), testosterone and cortisol] concentrations. Twenty amateur male runners were randomly divided into a creatine supplementation group, or placebo group. Subjects were provided with capsules containing either creatine monohydrate or identical powdered cellulose placebo. Daily creatine monohydrate supplementation was 20 g/day parceled into three equal dosages to be consumed with each major meal. Subjects were tested for performance and resting blood hormone concentrations before and after six days. A double-blind research design was employed in this study. After this creatine loading, the mean running performance time of the creatine supplementation group decreased significantly in the 100 m, but not the 200 m. Serum GH, testosterone, and cortisol concentrations were not affected by creatine supplementation. It can therefore be concluded that although short-term creatine supplementation was found to improve sprint performance in the 100 m in amateur runners, this performance improvement did not appear to be hormonally mediated.

Key words: Sprint performance; Creatine supplementation; Hormonal responses; Creatine loading.

INTRODUCTION

Creatine is a popular dietary supplement that is used by athletes to increase muscle mass and strength and especially to improve sports performance (Kreider, 2003; Rawson & Persky, 2007). Supplementation thereof has been demonstrated to increase resting concentrations of creatine and phosphocreatine in skeletal muscle (Navratil *et al*., 2009).

Gotshalk *et al.* (2008) reported that creatine supplementation (0.3 g/kg/day for seven days) resulted in a significant increase in the amount of work performed during five sets of bench press and jump squats in comparison to a placebo group. Mujika *et al*. (2000) found that creatine supplementation (20 g/day for six days) improved repeated sprint performance (6×15 m sprints with 30 sec. recovery) and jumping ability in soccer players. In a study by Skare

and Skadberg (2001) creatine supplementation (20 g/day) also decreased 100 m sprint times and reduced the total time of 6 × 60 m sprints in a group of well-trained adolescent competitive runners.

Not all previous studies have, however, found that creatine supplementation enhances exercise performance. Op't Eijnde *et al*. (2001) reported that creatine (20 g/day for five days) did not enhance stroke performance or 70 m agility sprint performance in well-trained tennis players. Improvements in performance were also not identified during single or repetitive sprint bouts (Greenhaff *et al*., 1993; Kinugasa *et al*., 2004) or in swimmers in 25, 50, and 100 m race distances (Mujika *et al.,* 1996; Mendes *et al*., 2004). The scarcity of published reports concerning single sprints lasting 6-60 sec., the lack of standardization of exercise protocols and variations in individual training levels may account for these discrepant results and suggest that additional studies are needed.

As creatine supplementation rapidly increases body mass and fat-free mass (Rawson & Persky, 2007; Gotshalk *et al.,* 2008), it has been hypothesised that creatine induces hypertrophy through endocrine mechanisms. Few studies evaluating the effects of creatine supplementation on performance have, however, included additional analysis of hormonal responses on consecutive days and these have produced conflicting results. Volek *et al*. (1997) assessed circulating testosterone and cortisol concentrations immediately post-exercise (five sets of bench presses and jump squats) in creatine (25 g/d for seven days) and placebo supplemented subjects and found no effect. Schedel *et al*. (2000), however, found increased serum growth hormone (GH) concentrations (83%) in response to a 20 g oral creatine bolus.

These discrepancies in the findings may primarily be attributed to variations in the performance level of subjects (amateur vs. elite), experimental protocol, gender and age. The purpose of this study was therefore to determine the influence short-term creatine supplementation on performance and hormonal responses to sprint running performance in subjects who were modestly trained.

METHOD

Subjects

Twenty healthy young male amateur runners (mean age: 21 years) volunteered to participate in this study. Because less intensively trained athletes may have a greater capacity to increase their intramuscular stores of creatine than the elite athletes (Selsby *et al.,* 2003), amateur runners were selected to participate in this study. All subjects were informed of the purpose, procedures and possible risks of the investigation before they gave written consent to participate in the study. They were also required to confirm that they had not taken any anabolic supplements or drugs during the previous year and had refrained from creatine supplementation for at least three months before the start of this study. The Institutional Review Board of the University approved the research protocol. The subjects had been doing sprint training (100 and 110 m), twice per week for a period of at least three months and had previously taken part in club sport activities (such as mini-football). The subjects refrained from any additional nutrition supplementation and exercise during this study and were encouraged to adhere to their usual dietary patterns. Before the study, subjects were assigned

to a creatine supplementation (CR) or a placebo (PL) group using a randomized double-blind design.

Experimental design

A double-blind, randomized study was employed using two experimental groups (creatine or placebo supplementation) who underwent six days supplementation. After pre-testing (one day later), subjects were provided with capsules containing either creatine monohydrate (Creatine Fuel, Twin Laboratories, Inc., Hauppague, NY) or identical powdered cellulose placebo. Daily creatine monohydrate supplementation was 20 g/day parceled into three equal dosages to be consumed with each major meal. The subjects consumed the supplements for six days.

Testing occurred before and at the end of six days of supplementation. Performance tests in 100 and 200 m sprint were started after the subjects underwent a standard warm-up. Fifteen minutes of recovery was given between tests. Participations were asked to refrain from exercise and from consumption of alcohol for 48 hours prior to each protocol day.

Body composition

Body composition was determined from seven skinfold sites (triceps, subscapular, midaxillary, chest, suprailiac, abdomen, and thigh) according to the method of Lohman, *et al*. (1988) using a Lange skinfold caliper. Skinfold measurements were based on the average of two trials and obtained on the right side in serial fashion by the same investigator. Body density was estimated using the age-adjusted equation of Pollock and Jackson (1984). The three-compartment Siri equation was used for % body fat (Siri, 1961). Height and body mass were assessed by digital scale (Japan) and height rod (Iran).

Blood collection and analyses

Blood samples were obtained via venipuncture, after five minutes in a supine position, from an antecubital vein by using a 20-guage needle and vacutainer tubes for the determination of serum testosterone, cortisol and GH concentration. Blood samples were obtained, pre and after six days of supplementation (immediately after running tests), in the early morning hours, and after a 10 hour overnight fast and occurred during a standardized time of day for each subject in order to minimize the effects of diurnal hormonal variations. The blood was processed and centrifuged, and the resultant serum was stored at −80°C until analyzed. Total serum testosterone, cortisol and GH were determined in duplicate by using standard radioimmunoassay procedures and were assayed via kits (Yellow Spring, OH).

Statistical analyses

Data are reported as mean ± SEM. A two-way analysis of variance (ANOVA) with repeated-measures design was used to establish whether PL and CR treatments differed with time. In the case of a significant *F* value, a Fisher's least significant difference (LSD) *post hoc* test was used to locate the exact time point of the differences between means. The level of significance for this investigation was set at $P<0.05$.

RESULTS

Physical characteristics and dietary intakes of the subjects

There were no significant differences between groups in terms of mean (± SD) physical characteristics. In the CR group these included age (21.75 ± 1.32 years), height (176.32 ± 6.35 cm), body mass (69.16 ± 8.65 kg) and percent body fat (16.12 ± 4.12%), whereas in the PL group, age (20.83 ± 1.73 years), height (75.60 ± 3.22 cm), body mass (69.12 ± 10.46 kg) and percent body fat (16.92 ± 5.25%).

No significant differences were observed between the CR and PL groups regarding the composition of carbohydrate, protein, and fat in the diet during the supplementation period.

Performance

The mean changes in running performance times in CR and PL groups are shown in Table 1. They were significantly decreased in the CR group in the 100 m ($P = 0.04$), but not in the 200 m ($P>0.05$).

TABLE 1. **RUNNING PERFORMANCE TIMES DURING THE PRE AND POST-SUPPLEMENTATION PERIOD IN THE PL (*N*=10) AND CR (*N*=10) GROUPS. DATA PRESENTED AS MEAN ± SEM**

	Pre	Post	Pre	Post
	CR		PL	
100 m (sec)	11.96 ± 3.9	11.23 ± 1.8*	11.82 ± 3.7	11.79 ± 3.2
200 m (sec)	22.82 ± 4.9	22.47 ± 6.4	22.79 ± 5.3	22.71 ± 5.7

(CR: creatine supplementation group, PL: placebo supplementation group)
* Significant difference ($P<0.05$) to Pre-test

Body composition

The CR group gained significantly more body mass (0.79 ± 0.11 kg) and fat-free mass (0.54 ± 0.05 kg) than the PL group (Table 2).

TABLE 2. **MEASURES OF BODY COMPOSITION IN THE PL (*N*=10) AND CR (*N*=10) GROUPS DURING THE PRE AND POST-SUPPLEMENTATION PERIOD. DATA PRESENTED AS MEAN ± SEM**

	CR group	PL group
Body mass (kg)		
Pre	69.16 ± 8.65	69.12 ± 10.46
Post	69.95 ± 9.76*	69.20 ± 11.12
Body fat (%) #		
Pre	16.12 ± 4.12	16.92 ± 5.25
Post	15.97 ± 4.67	16.65 ± 5.89
Body fat (kg) #		
Pre	11.23 ± 4.51	11.55 ± 6.48
Post	11.48 ± 4.84	11.69 ± 6.53
Fat-free mass (kg) #		
Pre	57.93 ± 5.68	57.57 ± 7.27
Post	58.47 ± 5.23*	57.51 ± 7.55

Values are mean ± SE obtained from skinfold analyses (based on the average of two trials and obtained on the right side)
*$P < 0.05$ from corresponding Pre value for the CR group only

Hormonal responses

The hormonal responses measured are presented in Table 3. No significant changes were observed in serum GH, testosterone and cortisol concentrations from before to after-supplementation in both groups of CR and PL (P>0.05).

TABLE 3. **SERUM GROWTH HORMONE, TESTOSTERONE AND CORTISOL CONCENTRATIONS IN THE PL (*N*=10) AND CR (*N*=10) GROUPS DURING THE PRE AND POST-SUPPLEMENTATION PERIOD. DATA PRESENTED AS MEAN ± SEM**

	Pre	Post	Pre	Post
	CR		PL	
Serum GH (ng/ml)	11.19 ± 2.03	11.23 ± 2.76	11.64 ± 2.26	11.67 ± 2.35
Serum Testosterone (ng/ml)	6.21 ± 1.37	6.18 ± 1.55	6.57 ± 2.24	6.64 ± 2.85
Serum Cortisol (mg %)	19.35 ± 2.17	19.30 ± 3.57	20.17 ± 2.33	19.98 ± 3.64

DISCUSSION

It has been well established that increasing dietary availability of creatine serves to increase total creatine and phosphocreatine concentrations in the muscle (Kreider, 2003). It is also known that the availability of creatine and phosphocreatine play a significant role in contributing to energy metabolism particularly during intense exercise. Theoretically, increasing the availability of phosphocreatine would enhance cellular bioenergetics of the

phosphagen system that is involved in high-intensity exercise performance of very short duration (Kreider, 2003), or the resynthesis of phosphocreatine during recovery (Greenhaff *et al*., 1993). The results of this study indicated that creatine supplementation 20 g/day (three times a day) for six days with no physical training decreased sprint running time (100 m) in the 20 amateur runners assessed in this study. The present study supports the finding Skare and Skadberg (2001), who also reported that short-term creatine supplementation (20 g/day) decreased 100-m sprint times in runners.

Although the findings of our study support those of previous investigations (Mujika *et a*l. 2000; Skare & Skadberg, 2001; Anomasiri *et al*., 2004; Hoffman *et al.,* 2005; Kraemer *et al.,* 2007; Gotshalk *et al.,* 2008) and suggest that short-term creatine supplementation can significantly increase exercise performance, they do conflict with others (Greenhaff *et al*., 1993; Mujika *et al.,* 1996; Kinugasa *et al*., 2004; Mendes *et al*., 2004) that did not replicate this difference. A possible explanation for the contrasting findings may be related to the calibre of the subjects examined. It is interesting to the note that Greenhaff *et al*. (1993), Mujika *et al.(*1996), Kinugasa *et al*.(2004) and Mendes *et al*.(2004) used competitive or elite athletes as subjects whereas our subjects were amateur. Harris *et al.* (1992) and Greenhaff *et al.* (1994) indicate that the extent of creatine uptake into the muscle is inversely related to an individual's initial muscle creatine content. The higher the initial intramuscular creatine concentration, the more difficult it is to increase stores (Harris *et al*., 1992; Greenhaff *et al*., 1994). Therefore, it is possible that our amateur runners had a greater capacity to increase their intramuscular stores of creatine than their elite counterparts (Greenhaff *et al*., 1993; Mujika *et al.,* 1996; Kinugasa *et al*., 2004; Mendes *et al*., 2004), who may already have had maximal intramuscular creatine concentrations. However, in this study intramuscular stores of creatine were not measured.

Based on the role of creatine supplementation in elevating intramuscular phosphocreatine stores and sustaining ATP production during muscle contraction (Kreider, 2003), the expectation was that creatine supplementation would have decreased both 100 m and 200 m performance. However, a significant improvement in mean 200 m times was not apparent. This may have been due to the large intersubject variability and small sample size. Participants of this study were, however, also in the collegiate amateur category. It is therefore possible that their poor technique and coordination may have been affecting performance results. Since the 100 m is shorter than 200 m, performance difference in 100 m would be less. It is therefore likely that large intersubject variability and or poor technique and coordination may have had less of an effect during the 100 m event. The small observed changes may however be meaningful for competitive running performers.

As creatine supplementation results in a rapid increase in body mass and fat-free mass (Rawson & Persky, 2007; Gotshalk *et al.,* 2008), it has been hypothesised that creatine induces muscle hypertrophy through endocrine mechanisms. Blood concentrations of GH and testosterone stimulate muscle protein accretion (Kraemer *et al.,* 2007) as GH stimulates protein synthesis by activating ribosomal initiation factors and improving translational efficiency (Bush *et al*., 2003). Testosterone also increases protein synthesis by binding to the androgen receptor for the complex to become a transcription factor and thirdly by possibly activating muscle satellite cells, which is important because gene transcription is an initial target for the modulation of protein synthesis (Herbst & Bhasin, 2004; Olsen *et al*., 2006).

No significant effects of creatine supplementation on serum GH, testosterone and cortisol responses at rest were found. The unchanged GH and cortisol after creatine supplementation is also consistent with other reports. For example, Op't Eijnde and Hespel (2001) found that creatine supplementation (20 g/day for five days) could not alter cortisol and GH responses to a single bout of heavy resistance exercise. Moreover, Volek *et al*. (1997) assessed testosterone and cortisol immediately post-exercise (five sets of bench presses and jump squats) in creatine (25g/day for seven days) and placebo-supplemented subjects, and found no effect of creatine on testosterone and cortisol hormones status. Results of this study and previous data indicate that it is unlikely that creatine supplementation is hormonally mediated. Furthermore, it is possible that creatine supplementation may affect other protein synthesis factors. Deldicque *et al*. (2005) reported that creatine supplementation (21g/day for five days) can facilitate muscle anabolism through increase of IGF-I (30%) and IGF-II (40%) mRNA in muscle.

Six days of creatine supplementation resulted in a small but significant increase in both body mass (0.79 ± 0.11 kg) and fat-free mass (0.54 ± 0.05 kg). These results are similar to the previous findings of Gotshalk *et al*. (2002 & 2008). A limitation of the current study was that muscle mass and body water were not measured. Nevertheless, the acute increase in body mass is most likely due to an increase in total body water (Ziegenfuss *et al*. 1998) and not an increase in muscle protein or muscle mass (Gotshalk *et al.,* 2008).

In conclusion, the data suggest that short-term creatine supplementation increases sprint running performance in amateur runners. An association between creatine supplementation and serum testosterone or decreased serum cortisol concentrations was, however, not found and the possibility that creatine supplementation is hormonally mediated by, a systemic change in these hormonal alterations is not supported.

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