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Creatine supplementation does not improve physical performance in a 150 m race

C. Javierre, M. A. Lizarraga, J. Ll.Ventura[1], E. Garrido and R. Segura

Unidad de Metabolismo y Nutrición, Depto. de Ciencias Fisiológicas y Nutrición, Facultad de Medicina, Universidad de Barcelona, Campus de Bellvitge and [1]Ciudad Sanitaria y Universitaria de Bellvitge, 08907 L'Hospitalet de Llobregat/Barcelona (Spain)

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Creatine supplementation has been shown by several authors to improve physical performance in very high intensity, intermittent, exercises. The effect on performance, as well as in plasma creatine and lactate concentrations has been studied in a group of twelve sprinters of national class when running a distance of 150 m on two occasions, before and after creatine (or placebo) supplementation for the previous three days. The most important differences in the biochemical parameters analyzed have been in plasma creatinine concentration, wich increased substantially both before and after the race in the group that had received a daily supplement of 25 grams of creatine monohydrate for the previous three days. Creatine supplementation, therefore, did not improve physical performance, in the conditions, when running a 150 m distance.

Key words: Creatine, Performance, Ergogenic.

Sprinters muscles show, generally, a higher concentration of phosphorylcreatine than those of long distance runners. On the other hand, depletion of phosphorylcreatine muscle stores has been associated with the appearance of peripheral fatigue (9, 10)

Correspondence to C. Javierre (Tel.: 93-4024517; Fax: 93-4024213).

Oral creatine supplementation increased plasma creatine concentration as well as the creatine content in skeletal muscle (7), a change that was the more important the lower the initial values were. Creatine uptake by muscle appears to be higher during the first days of supplementation, the renal excretion progressively increasing thereafter. The creatine content, which is present at approximately 60 % as phosphorylcreatine, increases

after supplementation in relation to the amount of exercise performed by each particular muscle group (6, 7). A daily 20 g creatine supplementation for 5 days is followed by a muscle phosphorylcreatine content increase, observed both by analyses of muscle biopsy samples and by magnetic resonance spectroscopy scan, while accelerating the rate of phosphorylcreatine resynthesis (4).

The changes observed in sport performance after creatine supplementation appear to be quite variable according to the scant literature on this topic. Thus, BALSOM *et al.* (1) have observed that in dynamic high intensity exercises, of 4 to 24 minutes of duration, performance was lower after creatine supplementation. On the other hand, (8) a significant improvement in the final run of a 4 by 300 m race and in the trials of a 4 by 1000 m race has been reported (8), as well as an improvement on muscle torque during repeated bouts of maximal voluntary contraction (3).

The aim of the present study was to determine if oral creatine supplementation would enhance the performance of high intensity, continuous exercise in contrast to that of highly intense but intermittent exercises. As phosphorylcreatine is the most important source of energy during a substantial part of a high speed race, an improvement in performance in this type of trial would be expected, had the muscle a larger amount of both creatine and phosphocreatine, available.

Materials and Methods

Twelve sprinters with an average of 21.4 (SD 3.0) years of age, a body mass of 65.8 (SD 8.6) kg and a height of 175.0 (SD 5.1) cm took part in this study after having been informed about the aim and the characteristics of the same. All participants had been sport practitioners for more than six years with an average training volume of 20 h per week and their record performance was at national Spanish category level.

All the trials were done outdoors and separately for each individual who ran a distance of 150 m, on two occassions, on a track of tartan, at the highest speed possible. In each case, the different tests were performed at the same day's time.

All participants performed one trial without creatine supplementation, used as a control. Two weeks later they repeated the same trial having half of them ingested, during the previous 3 days, 25 g of creatine monohydrate per day (in dosages of 5 g) dissolved in a total volume of 275 ml of water flavoured with lemon juice, in order to mask the sample, and the other half having ingested "supplementation" of plain water flavoured in the same way. The individuals were assigned to their respective group at random and the administration of the different beverages was done following a double blind procedure.

During each trial session, blood samples were obtained from an antecubital vein on resting conditions, just before the warming up period, as well as fifteen minutes after finalising the trial. In each blood sample glucose and creatinine concentration were determined by means of suitable enzymatic methods, using a "dry chemistry" procedure (Reflotron, Boehringer Mannheim GmbH, Mannheim, Germany).

In addition, capillary blood samples were obtained from the ear lobe immediately before the trial and 3, 5 and 7 min after it during the recovery period. Capillary blood samples were used to determine blood lactate concentration by means of an enzymatic method that uses lactate oxidase immobilised within a three layer membrane that fits into a platinum electrode (YSI 23L Blood lactate analyser,

YSI Inc, Yellow Springs, Ohio, USA). The amount of sample used for lactate determination was 25 µl and that required for each of the blood chemistries was 32 µl.

Data were analysed using a two-way analysis of variance (ANOVA) with repeated measures. Null hypothesis was rejected for $p < 0.05$.

Results

In the creatine group no statistically significant differences have been observed between the performance times of the first and the second trials (mean difference = –0.14 s, $p > 0.05$), although a tendency for a better performance during the second trial has been observed. The same results took place in the placebo group (mean difference = –0.26 s, $p > 0.05$). The performance times of the components of the two groups have been compared with each other. Those corresponding to the first trial showed no significant differences between the "placebo" and the "creatine" group (mean difference 0.1 s, $p>0.05$). The times achieved during the second trial were practically identical despite the fact that the components of the "creatine" group had ingested a supplement of 25 g of creatine daily during the three days previous to the test.

The plasma lactate concentration of the samples obtained before the trials and at 3, 5 and 7 min of the recovery period showed no significant differences between the two groups either in the first trial or the second one.

The creatinine plasma concentration in the samples obtained during the second trial showed a substantial increase, after the daily 25 g creatine ingestion for the previous three days, both in those corresponding to resting conditions (with a mean increment of 83.4 mmol/l with respect to those of the first trial, $p < 0.01$) as well as in those corresponding to the samples obtained 15 min after finishing the trial (with a mean difference of 98.6 mmol/l, $p = 0.01$) (table I)

In relation to the plasma creatinine values, there were no statistically significant differences in the samples obtained before the supplementation between the two groups (the placebo and the creatine

Table I. *Performance time (s) and plasma lactate, glucose and creatine values (mmol/l) before and after supplementation* (mean ± SE).

	Before supplementation		After supplementation	
	Placebo	Creatinine	Placebo	Creatinine
Time	17.9 ± 0.8	17.8 ± 0.7	17.7 ± 0.7	17.7 ± 0.7
Lactate Pre-Race	3.02 ± 0.22	2.70 ± 0.14	3.70 ± 0.31	3.46 ± 0.34
3 min Lact.	7.62 ± 0.29	7.23 ± 0.51	8.08 ± 0.43	8.44 ± 0.62
5 min Lact.	9.01 ± 0.36	8.09 ± 0.41	9.91 ± 0.27	9.45 ± 0.66
7 min Lact.	9.20 ± 0.29	8.18 ± 0.45	9.66 ± 0.32	8.60 ± 0.58
Glucose Pre-race	4.74 ± 0.2	4.48 ± 0.17	4.62 ± 0.33	4.14 ± 0.33
Glu.Recovery	6.10 ± 0.4	5.70 ± 0.28	6.06 ± 0.19	5.90 ± 0.36
Creatinine Pre-Race	92.1 ± 4.4	100.0 ± 5.2	101.8 ± 7.0	183.4 ± 24.6*
Creatinine Recov.	104.4 ± 6.1	112.8 ± 5.2	132.5 ± 6.1	211.4 ± 27.2*

*$p < 0.05$.

groups). However, and as expected, plasma concentration values were much higher in the creatine group than in the placebo one after the three days of supplementation with a mean difference between the samples obtained immediately before the trial of 81.6 mmol/l ($p = 0.009$) and a difference of 78.9 mmol/l ($p = 0.02$) between the samples obtained in each group after finishing the race.

Discussion

Oral creatine supplementation has been shown to be able to increase muscle creatine and phosphocreatine availability (7). The increased phosphorylcreatine content induced by such dietetic manipulation would delay the development of fatigue during repeated bouts of maximal exercise in man (3) and, eventually, improve physical performance. The availability of phosphorylcreatine appears to be the limiting factor for the continuation of a maximal intensity physical effort, the depletion of the phosphorylcreatine reserve muscle during intensive exercise being associated with the onset of muscle fatigue (9, 10).

Data obtained by HARRIS *et al.* (7), indicate that creatine ingestion for a period of time resulted in its muscle content increase, within a few days, and that a further increase in the muscle store was possible when supplementation was combined with regular exercise; at any rate, the total creatine maximun concentration that can be achieved appears to be around 150 mmol/kg dry weight (from a mean initial value of 124 mmol/kg dry weight). Of the total amount of creatine present in muscle, around 67 % appears to be in the form of phosphorylcreatine, before the oral supplementation, with a mean content of 84.2 mmol/kg dry weight muscle and 61 % after creatine supplementation, with a mean content of 91 mmol/kg dry weight muscle. Muscle ATP content is not affected by changes in total creatine content (7).

Although GREENHAFF *et al.* (5) have shown that a group of six subjects were all able to sustain peak isokinetic torque production at a high level during repeated bouts of maximal voluntary contraction after a regime of oral creatine supplementation and HARRIS *et al.* (7) have reported an improvement in repeated running times with creatine supplementation, we have not observed any improvement in physical performance after supplementation with oral creatine for three days before a sprint race, with a daily intake of 25 g of creatine monohydrate, ingested in doses of 5 g. The performance times when running a distance of 150 m on two different occasions (before and after supplementation with a placebo or with creatine) were minimal, with a mean difference of −0.10 s ($p = 0.5$) for the race before supplementation and no difference after supplementation (< 0.01 s difference).

It could be argued that the period of supplementation was shorter than that used by HARRIS *et al.* (8) who administered oral creatine for 6 days. However, plasma creatinine concentration rose about 80 % in basal conditions and around 60 % after finishing the race when on the creatine supplementation, which would suggest that the creatine levels achieved have been high enough to influence muscle total creatine concentration.

Although the components of each group have performed the same amount of work, it can be observed that lactate accumulation tends to be lower in the group that had ingested a supplement of creatine during the previous three days (peak lactate accumulation 9.96 mM in the "placebo" group versus 9.56 mM in the "creatine" group). At the same time, the Δ-lactate values tend to be lower but not significant, in the group that had received

the creatine (6.26 for the placebo versus 6.10 for the creatine group). In this respect, it has been observed (11) that immediately after the fifth repetition of a standardised 6 s bout of high intensity exercise, the muscle lactate accumulation was 70 % lower after than before creatine supplementation. On the other hand, no differences in peak blood lactate accumulation has been observed, when performing three 30 s bouts of maximal isokinetic cycling, between a placebo group and one that had received a 20 g/day creatine supplement (2).

Creatine supplementation may improve the performance of intermittent high intensity exercises due to the greater availability of total creatine which would allow a higher rate of phosphorylcreatine resynthesis. As this is done through the transfer of a high energy phosphate from ATP -generated by oxidative phosphorylation in the mitochondria- to creatine, a minimum time is needed for the oxidative systems to replenish the stores of phosphorylcreatine, depleted by the bout of high intensity exercise, approximately 2 min, according to GREENHAFF *et al* (3). In contrast, in single high intensity continuous exercises, such as that performed in our study, the high rate of ATP utilization for a longer period of time cannot be satisfied by a corresponding rate of phosphorylcreatine resynthesis, even when larger amounts of creatine are available. In these circumstances, the limiting factor appears to be the rate of ATP regeneration, by the creatine kinase transference system, and not to the total amount of phosphorylcreatine available or the capacity or rate of the mitochondria to regenerate phosphorylcreatine.

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C. JAVIERRE, M. A. LIZARRAGA, J. LL. VENTURA, E. GARRIDO and R. SEGURA. *La suplementación dietética con creatina no aumenta el rendimiento físico en carreras de 150 m.* J. Physiol. Biochem. (Rev. esp. Fisiol.), 53 (4), 343-348, 1997.

Se ha sugerido que la suplementación con creatina puede dar lugar a una mejoría en el rendimiento físico en esfuerzos de carácter intermitente de alta intensidad. En el presente trabajo se estudia el rendimiento, así como las concentraciones plasmáticas de creatinina y de lactato, en un grupo de 12 velocistas de nivel nacional que corrieron una distancia de 150 m, a la máxima velocidad posible, en dos ocasiones distintas, una antes y otra después de la suplementación con 25 g de monohidrato de creatina, o placebo, durante los tres días previos a la prueba. Las diferencias más importantes en los parámetros bioquímicos analizados se han observado en la concentración de creatinina plasmática que, en el grupo que recibió el suplemento de creatina, aumentó sustancialmente en las muestras obtenidas tanto antes como después de la carrera. En las condiciones en las que se ha realizado este trabajo, la suplementación con creatina no da lugar a un aumento en el rendimiento en una carrera de 150 m, a velocidad máxima.

Palabras clave: Creatina, Rendimiento, Ayudas ergogénicas.

References

1. Balsom P. D., Harridge, S. D. R., Sjödin, K. and Ekblom, B. (1993): *Acta Physiol. Scand.*, 149, 521-523.
2. Birch, R., Noble, D. and Greenhaff, P. L. (1994): *Eur. J. Appl. Phisiol.*, 69, 268-270.
3. Greenhaff, P. L., Casey, A., Short, A. H., *et al.* (1993): *Clin. Sci.*, 84, 565-571.
4. Greenhaff, P. L., Bodin, K., Harris, R. C., Hultman, E., Jones, D. A., Mcintyre, D. B., Söderlund, K. and Turner, D. L. (1994): *J. Physiol.*, 467, 75P.
5. Greenhaff, P. L., Constantin-Teodosiu, D. and Hultman, E. (1994): *J. Physiol.*, 478, 84P.
6. Harris, R. C., Hultman, E. and Nrdesjon, L. D. (1974): *Scand. J. Clin. Invest.*, 33, 109-120.
7. Harris, C., Söderlund, K. and Hultman, E. (1992): *Clin. Sci.*, 83, 368-374.

8. Harris, C., Viru, M., Greenhaff, P. L. and Hultman, E. (1993): *J. Physiol.*, 467, 74P.
9. Hultman, E. (1967): *Scan. J. Clin. Lab. Invest.*, 19 suppl. 94, 1-64.
10. Hultman, E., Bergtrom, J. and Anderson, N. M. (1967): *Scan. J. Clin. Lab. Invest.*, 19, 56-66.
11. Söderlund, K., Balsom, P. B. and Ekblom, K. (1994): *Clin. Sci.*, 87 Suppl. 120.