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SHORT COMMUNICATION

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Effects of training and creatine supplement on muscle strength and body mass

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Abstract The purpose of this study was to test the effect of creatine supplement on the size of the extra- and intracellular compartments and on the increase of isokinetic force during a strength training-program. Twenty-five healthy male subjects (age 22.0 ± 2.9 years) participated in this experiment. Seven subjects formed the control-group. They did not complete any training and did not have any dietary supplement. The eighteen other subjects were randomly divided into a creatine- (n = 8) and a placebo-group (n = 10). They were submitted to a controlled strength-training program for 42 days followed by a detraining period of 21 days. Creatine and placebo were given over a period of 9 weeks. The size of the body water compartments was assessed by bio-impedance spectroscopy and the isokinetic force was determined during a single squat by means of an isokinetic dynamometer. These measurements were completed beforehand, at the end of the training period, and after the determining period. Both placebo- and creatine-group increased the isokinetic force by about 6% after the training period, showing that creatine ingestion does not induce a higher increase of the force measured during a single movement. No change in body mass was observed in the control- and placebo-groups during the entire experiment period while the body mass of the creatine-group was increased by 2 kg ($P < 0.001$). This change can be attributed partially to an increase ($P = 0.039$) in the body water content (+1.11), and more specifically, to an increase ($P < 0.001$) in the volume of the inter-cellular compartment (+0.61). Nevertheless, the relative volumes of the body water compartments remained constant and therefore the gain in body mass cannot be attributed to water retention, but probably to dry matter growth accompanied with a normal water volume.

Key words Phosphocreatine · Bio-impedance · Muscle water content · Force · Isokinetics

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Introduction

During the last few years, athletes expecting to improve their physical performance have consumed dietary creatine supplements. To the best of our knowledge, no adverse consequence associated with oral creatine supplementation has been yet reported in healthy human subjects. The only documented secondary effect seems to be an increase of the body mass and more specifically of the fat free mass (Earnest et al. 1995). This change in body composition could be explained in two ways. Firstly, the increase of free creatine concentration in the cells could induce water retention and thus modify the relative volume of the intra-cellular compartment. Secondly, creatine in itself, could induce muscle growth. Indeed, creatine, as an end-product of contraction, was suggested to be an intra-cellular signal coupling increased muscle activity and increased muscle growth (Ingwall 1976). Therefore, creatine supplementation could improve muscle strength via an up-regulation of the contractile protein synthesis.

The purpose of this study was to test these hypotheses in human subjects by measuring the isokinetic force and the space of the body water compartments during a six weeks strength training-program followed by a three weeks detraining period.

Methods

Twenty five healthy male subjects (age 22.0 ± 2.9 years; height 179.7 ± 5.2 cm) volunteered to participate in this experiment, the

protocol of which was approved by the Ethics Committees of the ''Université Catholique de Louvain'' and the ''Université Libre de Bruxelles''. All the subjects were physically active but none of them followed a regular strength-training program before the experiment.

The subjects were divided into three groups. Seven subjects formed the control-group. They did not complete any training and did not consume any dietary supplement neither creatine, nor placebo. The eighteen other subjects were randomly divided into a creatine- ($n = 8$) and a placebo-group ($n = 10$). They were submitted to a controlled strength-training program for 42 days followed by a detraining period of 21 days. The experimental protocol was double-blinded to the examiners and the subjects.

Each participant in the creatine-group took 21 g creatine monohydrate daily, distributed in 7 g doses in the early morning, at noon, and in the evening for 5 consecutive days. Then, the daily dose was reduced to 3 g for 58 additional days. The placebo-group followed the same protocol but with maltodextrine.

At the first day of the experiment (day 1), all the subjects came to the laboratory at 8.30 a.m. after a 12 hour fast without exercise. They were told to have a hydration as normal as possible and not to consume alcohol and caffeine 24 hours prior to the test. Body water compartments were assessed by bio-impedance spectroscopy using a BODYSTAT DUALSCAN 2005 apparatus which was validated in a previous study (Hannan et al. 1995). This method allows for measurements of the extra-cellular water (ECW) and of the total body water (TBW). By deduction, the intra-cellular water (ICW) was calculated.

The subjects were resting in the supine position for 10 minutes. Afterwards, a sinusoidal current of 500 μA (R.M.S.) was applied to the subjects at frequencies of 5 kHz and 200 kHz, successively, and the respective impedance values were recorded ($\pm 1\ \Omega$). A program supplied by the manufacturer of the analyser estimated the sizes of TBW, ECW and ICW from the impedance values.

Then the participants warmed-up before measuring the isokinetic force. The latter was determined during a single squat movement by means of an ARIEL isokinetic dynamometer. The linear velocity of the movement was $0.1\ m \cdot s^{-1}$ and the force was recorded over a distance of 0.5 m, starting when the knee angle was ~90°. The subjects were instructed to develop the highest force possible during the whole movement. The computer controlling the dynamometer displayed the mean value (F) of the recorded isokinetic force. Measures were repeated twice with 3 minutes recovery between each bout. The highest F value was taken into consideration. A six week (from day 1 to day 42) training-program was established from the result of the isokinetic test. The subjects were instructed to train three times a week. The first week, the subjects accomplished each time 6 series of 6 squats with a load equal to 30% of the F value measured at day 1. Then, the workload was increased progressively. The last week, each training session consisted of 8 series of 6 squats with 42.5% of the F value.

At the end of the training programme, the body water content and the isokinetic force were measured in the same conditions as described above. Then, the subjects were instructed to stop the strength training-program but to continue the ingestion of creatine during three weeks (from day 42 to day 63) after which the body water content and the isokinetic force were determined again.

The results are expressed as means ± standard deviation. The statistical significance of differences observed between means was assessed by an ANOVA design for repeated measures after verifying the normality of the distribution and the lack of difference between variances. When ANOVA showed significant changes, contrast analyses were computed to compare the data-points two-by-two. The limit of statistical significance was taken to be 0.05. All statistical analyses were performed by using the SYSTAT software (Systat Inc, 60201 Evanston, IL).

Results

The upper panel of Table 1 presents the mean values of isokinetic force measured before and at the end of the training period. The control-group did not show any significant variation of the mean force developed during a single squat, while both the placebo- and creatine-groups increased by 6% ($P = 0.022$ and 0.008, respectively). This indicates that creatine ingestion during a training period does not induce a higher increase in isokinetic force measured during a single movement.

During the detraining period (3 weeks), the isokinetic force did not return to its initial value in the three groups (Table 1, lower panel).

The results of body mass and the bio-impedance measurements are presented in Table 2. No change nor in body mass, neither in body composition was observed both in the control- and placebo-group during the entire experiment (training + detraining) while the body mass of the creative-group increased by 2.9% ($P < 0.001$). In the latter group, the 200 kHz impedance value decreased ($P = 0.038$) indicating an extension of the total body water content ($P = 0.039$). On the contrary, the 5 kHz impedance value did not change significantly rejecting any modification of the extra-cellular compartment. Thus, the origin of the total body water increase can be attributed to an enlargement of the intra-cellular volume ($P < 0.001$).

These changes in body mass, TBW, and ICW were observed at the end the training period ($P = 0.019$, $P = 0.01$, and $P < 0.001$, respectively) but remained unchanged during the detraining period.

The magnitude of the TBW increase (+1.11) could explain the 55% of body mass gain and the 30% of the ICW enlargement (+0.61). These relative values (55% and 30%) of body mass *gain* are comparable to the relative values of *total* body mass, measured at day 1:

Table 1 Effects of creatine supplementation on isokinetic force measured during a single squat movement

	Control group			Placebo group			Creatine group		
	day 1	day 42	ANOVA	day 1	day 42	ANOVA	day 1	day 42	ANOVA
Training period Force (N)	1658 ± 216	1776 ± 216	NS	1657 ± 167	1756 ± 128	0.022	1619 ± 284	1727 ± 245	0.008
	day 42	day 63	ANOVA	day 42	day 63	ANOVA	day 42	day 63	ANOVA
Detraining period Force (N)	1776 ± 216	1638 ± 167	NS	1756 ± 128	1756 ± 255	NS	1727 ± 245	1766 ± 284	NS

Table 2 Effects of nine week creatine supplementation on body composition estimated by bio-impedance

Variable	Control group				Placebo group				Creatine group			
	day 1	day 42	day 63	ANOVA	day 1	day 42	day 63	ANOVA	day 1	day 42	day 63	ANOVA
Body mass (kg)	77.5 (±8.0)	77.9 (±8.5)	77.5 (±8.6)	NS	71.9 (±7.3)	72.4 (±6.4)	72.6 (±6.7)	NS	69.8 (±8.9)	72.1 (±9.0)	71.8 (±9.0)	<0.001
Z 5 kHz (Ω)	599 (±51)	587 (±53)	609 (±62)	NS	578 (±28)	583 (±46)	566 (±59)	NS	599 (±74)	572 (±56)	579 (±78)	NS
Z 200 kHz (Ω)	448 (±35)	446 (±45)	456 (±51)	NS	452 (±24)	444 (±23)	436 (±33)	NS	469 (±53)	432 (±42)	439 (±55)	0.038
TBW (l)	41.2 (±2.9)	41.5 (±3.5)	41.0 (±3.4)	NS	39.6 (±2.5)	39.7 (±2.1)	40.1 (±2.2)	NS	38.0 (±3.9)	39.7 (±3.4)	39.1 (±4.1)	0.039
TBW (%)	53.3 (±2.1)	53.5 (±2.0)	53.0 (±1.8)	NS	55.3 (±2.5)	55.0 (±2.8)	55.4 (±3.1)	NS	54.6 (±2.4)	55.3 (±3.2)	54.8 (±2.0)	NS
ECW (l)	19.0 (±1.4)	19.3 (±1.5)	18.8 (±1.5)	NS	18.5 (±1.2)	18.5 (±1.1)	18.9 (±1.2)	NS	17.7 (±1.9)	18.4 (±1.5)	18.2 (±2.0)	NS
ECW (%)	24.5 (±1.3)	24.8 (±1.3)	24.4 (±1.0)	NS	25.9 (±1.3)	25.7 (±1.7)	26.1 (±2.0)	NS	25.4 (±1.3)	25.7 (±2.1)	25.5 (±1.3)	NS
ICW (l)	22.2 (±1.5)	22.3 (±2.0)	22.1 (±1.9)	NS	21.1 (±1.3)	21.1 (±1.2)	21.2 (±1.2)	NS	20.3 (±2.1)	21.3 (±2.1)	20.9 (±2.2)	<0.001
ICW (%)	28.7 (±1.1)	28.6 (±0.7)	28.7 (±0.9)	NS	29.5 (±1.2)	29.3 (±1.2)	29.3 (±1.3)	NS	29.2 (±1.2)	29.6 (±1.2)	29.3 (±0.9)	NS

54.6% and 29.2%, respectively. Consequently, when TBW and ICW are expressed in percentage of body mass, no significant difference can be observed, and these relative values of the body water compartment sizes remained constant.

Discussion

Obviously the ingestion of creatine increases the muscle creatine content, and therefore the creatine phosphate concentration (Harris, 1992). Creatine is a highly polar molecular playing a major role in the regulation of osmolarity in the cell. Without exchange of other ions, an increase of the free creatine content should increase the intracellular osmolarity. Consequently, water should enter into the cell modifying the relative volume of the intra-cellular compartment and increasing the total body mass. However data from the literature do not indicate any change of total body mass during short-term creatine supplementation (Grindstaff et al. 1997). In contrast, most previous studies and the present results (Table 2) showed that medium-term creatine supplementation increases total body mass by 1-2 kg. The values of TBW, ECW and ICW reported here (Table 2) are in good agreement with those presented elsewhere using a dilution technique (Amstrong et al. 1997). The control- and the placebo-group did not change TBW, ECW and ICW significantly during the six weeks of the experimentation. The creatine-group increased the absolute value of TBW and ICW, but not the relative value, which indicates that the body mass gain following a medium-term creatine supplementation is not due to intra-cellular water retention but probably to dry matter growth accompanied with a normal volume of intracellular water. These observations on intra-cellular water are in good agreement and extend previous observations, showing a gain in total body mass after creatine supplementation without any change in the percentage of total body water (Kreider et al. 1996).

Different hypotheses can be formulated to explain the link between creatine supplementation and gain in dry matter. Growth hormone concentration is well known to be affected by increase in levels of certain circulating amino acids, such as arginine, from the dietary intake. This could play the role of an external signal coupling the rise of plasma creatine concentration and an increase of growth factors. On the other hand, creatine, as an end-product of contraction, was suggested to be an intra-cellular signal coupling increased muscle activity and increased muscle growth (Ingwall 1976). Therefore, the rise of the muscle free creatine concentration induced by high level of creatine in the diet could also be an internal signal for muscle growth supporting the gain in the total body mass after creatine supplementation.

Placebo and creatine groups showed a similar increase (6%) in isokinetic force after 6 weeks of training, while the control group was unchanged (Table 1). There was measurable change in any group during the detraining period which may be due to the short duration (3 weeks). Changes in muscle force were however assessed using a single measure of a « squat » movement on an isokinetic dynamometer. It was not possible therefore to confirm true maximality although it should be noted that our subjects were very well habituated to the measured task. Clearly any conclusions regarding the lack of an enhanced training effect on muscle force due to creatine supplementation should be treated with caution. The results of the present investigation do not permit to validate the hypothesis of an increase of body mass related to a growth of muscle contractile proteins. Further experiments are needed to elucidate the link between creatine supplementation and body mass increase.

To summarize, the major finding of our study is to emphasize, for the first time, that body mass gain observed after medium-term creatine supplementation is not due to water retention in the cell but probably to dry matter gain.

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