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**TITLE**

CREATINE INGESTION INCREASES ANAEROBIC CAPACITY AND MAXIMUM ACCUMULATED OXYGEN DEFICIT

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# *Creatine Ingestion Increases Anaerobic Capacity and Maximum Accumulated Oxygen Deficit*

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## Abstract/Résumé

*The purpose of this study was to test the hypothesis that ingestion of creatine monohydrate increases anaerobic exercise capacity, as reflected by the maximal accumulated oxygen deficit (MAOD). Subjects were assigned, double-blind, to placebo (PL, n = 12) or creatine (CR, n = 14) groups and ingested 5-g doses 4 times daily of artificial sweetener or artificially sweetened creatine monohydrate, respectively, for 5 days. On a separate day subjects exercised to exhaustion at 125% $VO_2max$. After two familiarization trials, MAOD was again determined before treatment, after 5 days of PL or CR treatment, and 7 days later. MAOD increased after CR treatment from 4.04 ± 0.31 to 4.41 ± 0.34 L ($p < .001$) and remained elevated for another 7 days (4.31 ±0.33, $p < .001$). Time to exhaustion also increased in CR from 130 ±7 to 141 ±7 s ($p < .01$) and remained increased for another 7 days (139 ±8 s, $p < .01$). These data demonstrate that ingesting creatine monohydrate for 5 days increases the MAOD, and is likely to have an ergogenic effect on supramaximal exercise performance that persists for at least a week after treatment.*

*Le but de l'étude est de tester l'hypothèse à l'effet que l'ingestion de monohydrate de créatine améliore la capacité d'effort anaérobie estimée par la mesure du déficit maximal d'oxygène accumulé (MAOD). Les sujets, répartis à double insu dans le groupe placebo (PL, n = 12) ou dans le groupe expérimental (CR, n = 14) consomment, selon le cas, 4 fois par jour durant 5 jours, 5 g d'édulcorant artificiel ou de monohydrate de créatine sucré*

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*artificiellement. Quelques jours plus tard, les sujets participent à une épreuve à 125% du $\dot{V}O_2$max jusqu'à épuisement. Après deux épreuves de familiarisation, le MAOD est évalué encore une fois avant le traitement, après 5 d de traitement (PL ou CR) et 7 d plus tard. Le MAOD du groupe CR passe de 4,04 ±0,31 à 4,41 ±0,34 L après le traitement (p < ,001) et demeure au même niveau 7 d plus tard (4,31 ±0,83, p < ,001). Le TE du groupe CR passe aussi de 130 ±7 à 141 ±7 s (p < ,01) et reste au même niveau après 7 d (139 ±8 s, p < ,01). Ces observations indiquent que l'ingestion de monohydrate de créatine durant cinq jours améliore le MAOD et procure vraisemblablement au cours d'un effort supramaximal un effet ergogène qui persiste au moins une semaine après le traitement.*

## Introduction

Intramuscular total creatine concentration ([TCr]) is about 118 mmol · kg$^{-1}$ dry muscle in healthy subjects, and dietary creatine supplementation with 20 g of creatine monohydrate for 5 days has been reported to result in 20–50% increases in [TCr] (Gordon et al., 1995; Greenhaff et al., 1994; Harris et al., 1992). Several studies have reported ergogenic effects after such creatine supplementation, although there is no information available concerning the persistence of these effects after stopping creatine supplementation (for reviews, see Balsom et al., 1994; Greenhaff, 1995; Maughan, 1995). The maintenance of force generation during voluntary (Earnest et al., 1995; Greenhaff et al., 1993) and electrically evoked (Harridge et al., 1994) muscle contractions is apparently improved. The rate of decline of power output that occurs during repeated bouts of supramaximal-intensity cycle exercise was significantly less after creatine supplementation (Balsom et al., 1993a; Birch et al., 1994). There is also one report, in abstract form only, of improved running performance in trained middle distance runners during repeated 300-m or 1000-m interval runs (Harris et al., 1993). Such improvements in performance have not been universally found, however, as is exemplified by the studies of the effects of creatine treatment on performance of the 30-s Wingate test (Cooke et al., 1995), and sprint performance of competitive swimmers (Mujika et al., 1996).

The mechanism of action by which creatine supplementation may cause improved performance has not yet been clearly identified. It has been suggested that creatine supplementation sustains the duration during which free creatine can be maintained above the [Km] for creatine kinase for the resynthesis of phosphocreatine (PCr) (Greenhaff et al., 1994a). Such an effect would explain the increased rate of PCr resynthesis reported after exercise (Greenhaff et al., 1994a), and perhaps the higher preexercise muscle PCr concentrations sometimes reported in association with the higher [TCr] (Gordon et al., 1995; Greenhaff et al., 1993; Harris et al., 1992). Such higher initial PCr levels may permit more work to be accomplished during intense exercise by reducing the rate of intramuscular ATP depletion, as has been recently reported (Greenhaff et al., 1994b).

Aerobic exercise performance is not affected by creatine supplementation (Balsom et al., 1993b; Stroud et al., 1994), suggesting that the performance improvements described above were primarily due to increases in the contribution of anaerobic energy metabolism to exercise performance. There has not been any attempt, however, to verify the relative changes in aerobic or anaerobic metabolism contributions to the improved exercise performance attributed to creatine supplementation. Therefore, this investigation was carried out to test the hypothesis that 5 days of creatine treatment would increase the capacity for dynamic exercise fueled by anaerobic energy metabolism and that this increase would be reflected in an increase in the maximum accumulated oxygen deficit (MAOD). A supplementary hypothesis was that any observed ergogenic effect would be sustained for one week after stopping creatine supplementation. This study did not include measurements of intramuscular creatine concentrations.



## Methods

Permission to undertake this study was obtained from institutional human ethics committees. A priori consideration of sample size was based on the premise that an ergogenic effect could be considered demonstrated if the short treatment period with creatine caused an increase in MAOD that was similar to the 10% increase that has been observed after 6 weeks of training (Medbø and Burgers, 1990). Pilot testing with some of the subjects who volunteered to participate gave a rough indication of what the pretraining MAOD value would be. Presuming a pretraining mean ± *SD* MAOD of 4.0 ± 1.2 L, and a test-retest reliability coefficient of 0.95, it was calculated that 10 subjects in the treatment group would yield a statistical power of 0.91 for an alpha level of .05 (Howell, 1985, p. 179). Several more subjects per treatment group were recruited to compensate for envisaged subject "dropout." Thirty subjects volunteered and gave their informed written consent to participate in this study.

All subjects were familiar with exhaustive exercise, but their training status varied substantially. Most subjects engaged in regular recreational fitness activities and some were training for competitive sports. Although creatine is synthesized endogenously it is also consumed as a naturally occurring dietary component in animal flesh; although vegetarians were not a priori excluded from participation, none of the subjects were vegetarians. All subjects were medically screened to ensure good health before commencing experimentation. They were assigned to either the placebo (PL) or creatine (CR) groups in a double-blind fashion by a technician not present during the exercise testing. The technician was instructed to match the groups for the MAOD values recorded during familiarization trials. The same technician also distributed to each subject their placebo or creatine to be ingested during the treatment period described below.

During the course of the study, 1 of the CR subjects and 3 of the PL subjects withdrew because of illness, scheduling difficulties, loss of interest, or some combination of these. Data are presented here only for those subjects who completed all trials, that is, 14 subjects in CR (11 males and 3 females) and 12 subjects in PL (10 males and 2 females). Their mean ± *SD* age, weight, height, and maximal aerobic power ($\dot{V}O_2$max) during cycle exercise were 24.5 ± 3.8 years, 78.3 ± 2.1 kg, 1.78 ± 0.07 m, and 48 ± 6 ml · kg$^{-1}$ · min$^{-1}$, respectively.

TEST OF ANAEROBIC CAPACITY

Anaerobic capacity was evaluated by determining the maximum accumulated oxygen deficit (MAOD) during a short maximal-effort cycle exercise test. Earlier studies demonstrated that exercise leading to exhaustion in about 2 min is sufficient to elicit a maximum oxygen deficit and that an appropriate intensity to cause exhaustion in 2 min during cycle ergometry is about 125% of $\dot{V}O_2$max (Gastin and Lawson, 1994; Medbø and Tabata, 1989). Therefore, a day before the first determination of the MAOD, the $\dot{V}O_2$max of each subject was defined as the highest oxygen consumption measured during cycle ergometry using a continuous incremental protocol to exhaustion on an electrically braked ergometer (Ergomed 930, Siemens, Germany) which maintained power output independent of pedaling frequency within the range of 50–130 rev · min$^{-1}$. Intensity was increased by 30 W · min$^{-1}$ for the male subjects and 20 W · min$^{-1}$ for the female subjects. After a 45-min recovery period, subjects exercised for 4 min at each of four different submaximal power outputs estimated to be approximately 50, 65, 75, and 85% of $\dot{V}O_2$max.

Oxygen consumption was measured during the last minute of the incremental test to exhaustion and during the four submaximal power outputs by directing the expired respiratory gases into a 350-L wet spirometer (Collins Gasometer, Braintree, MA) for the measurement of expired ventilatory volumes. After determining the volume and temperature of the expired gas, a sample line directed a sample from the spirometer to oxygen and carbon dioxide analyzers (Ametek models S3A and CD3A, Pittsburgh, PA) for the determination of gas fractions. Individual subject linear regression equations of the rate of oxygen consumption ($\dot{V}O_2$) versus power output were calculated from the four submaximal power outputs described above, and the power output equivalent of 125% $\dot{V}O_2$max was calculated by extrapolation of this regression equation. All subsequent tests of MAOD were performed at this intensity.

The subjects were informed prior to the MAOD test that they should attempt to maintain a pedaling rate of 110 rev · min$^{-1}$, and that they would be warned once when the rate decreased below 60. They viewed the pedaling rate indicator continuously during exercise, and the MAOD test was stopped when the pedaling rate decreased to 60 rev · min$^{-1}$ a second time. There were no clocks or other indications of elapsed time visible to the subjects during testing. The MAOD test consisted of an initial 3-min warm-up at 60 W, after which intensity was increased immediately (i.e., in less than 1 s) to the individual's MAOD wattage. It was at this point that the measurement of exercise time to exhaustion (TE) and the collection of expired respiratory gases commenced. Expired respiratory gases were directed to the wet spirometer, and after exercise was completed, the total oxygen consumption ($\dot{V}O_2$) during exercise was calculated with standard formulae after determining the oxygen and carbon dioxide fractions in the expired gases as described earlier. The MAOD was calculated in oxygen consumption equivalents as the difference between the oxygen demand of exercise (from each subject's TE and linear regression described above) and the $\dot{V}O_2$. All oxygen consumption val-



DESIGN AND TREATMENTS

For familiarization purposes and to document the reproducibility of the measured variables, the subjects performed the MAOD test twice within one week before commencing the experiment. They subsequently performed the MAOD test three more times: pretreatment, posttreatment, and 7 days posttreatment. After their pretreatment test they were given their treatment to be ingested during the subsequent 5 days. Not only were the subjects "blinded" with regard to their treatment group, but attempts were made to make the two treatments similar in terms of texture, colour, size, and volume of the treatment vials.

The CR daily treatment consisted of ingesting 20 g of artificially sweetened creatine monohydrate (Triple Crown, UK) for 5 days. The purity of the creatine was compared with standard assay procedures to commercially available creatine monohydrate standards and found to be as pure as the standards. The daily dose was divided into four plastic vials, each containing 5 g of creatine and 0.2 g Nutrasweet. The PL was also divided into four vials per day for 5 days. Each vial contained 0.9 g of Nutrasweet, a volume that filled the vials to a similar height as was the case with the CR treatment. The PL tasted somewhat sweeter than the CR, but the subjects only consumed their own treatment and were thus unaware of this difference. The subjects were instructed to consume their treatment at regular intervals throughout the day by dissolving the contents of a single vial in a warm drink of their choice. On the day after their last treatment day, they reported to the laboratory for the posttreatment test. They returned again 7 days later for the final MAOD test.

The subjects were instructed to avoid hard exercise for two days prior to each MAOD test, but to otherwise maintain their normal exercise regime. They were also given a standardized liquid meal (250 ml EnsurePlus) and instructed not to eat for 3 hours prior to testing other than this liquid meal which was to be ingested 2 hours before each test. They were asked to avoid caffeinated beverages or food only on the day of testing. At the time this study was conducted, we were unaware of the recent publication suggesting that caffeine can reduce creatine uptake (Vandenberghe et al., 1996). Tests for each subject were carried out at similar times of the day to minimize the effects of diurnal rhythms.

The pretreatment values for the two groups were compared using a one-way analysis of variance (ANOVA). ANOVA for repeated measures was used to determine the significance of changes across trials within each group, and if the $F$ ratio was significant, then the trial mean values were compared using contrast of means procedures..Commercially available statistical software was used (SuperANOVA and StatView, Abacus Concepts, Berkeley, CA). Statistical significance was accepted at $p < .05$.

## Results

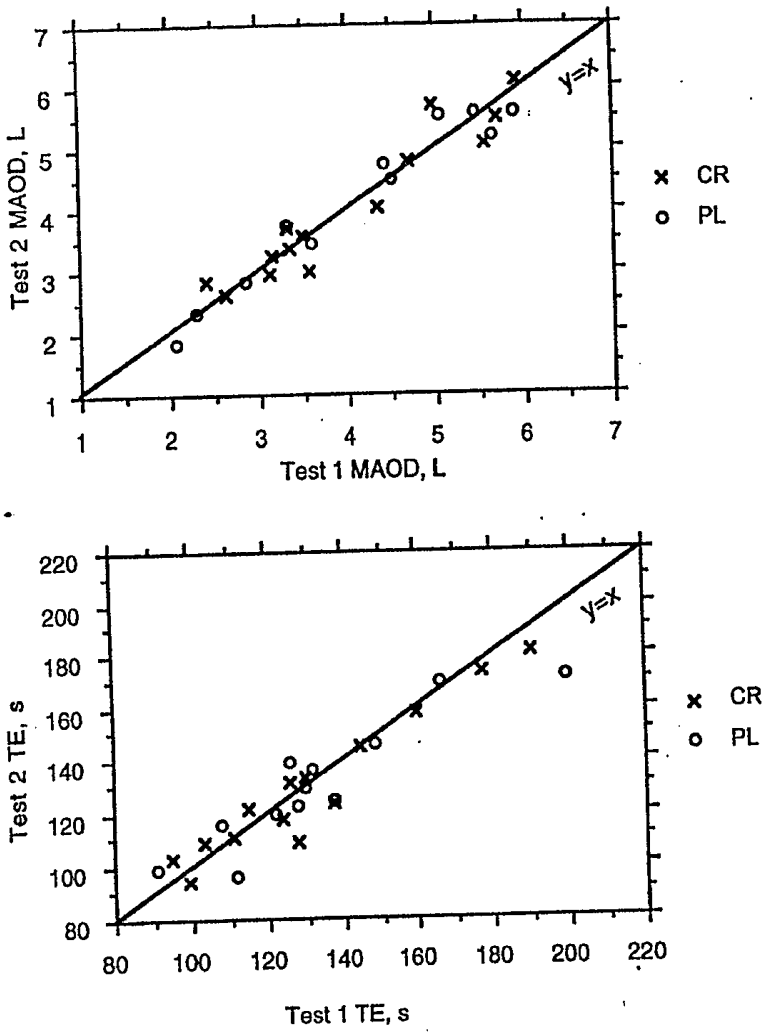


**Figure 1.** Reproducibility of the maximum accumulated oxygen deficit (MAOD) and the time to exhaustion (TE) during the MAOD test performed twice, with 3–5 days intervening between tests, for the 26 subjects participating in the study. Subjects in the creatine (CR) and placebo (PL) groups are differentiated, and the line of identity is plotted.

to their treatment group. The first test was considered a familiarization trial, and the data were not included in subsequent analyses. The data from the second trial were compared with the pretreatment trial data to demonstrate the reproducibility of the measured variables. Figure 1 shows the data from these two tests of the MAOD and time to exhaustion (TE) during the MAOD test. The mean ± *SD* values for these two trials did not differ significantly for either the MAOD (3.995 ± 1.233 vs. 4.030 ± 1.235 L, for the first and second test, respectively) or TE (132 ± 28 vs. 130 ± 24 s). The intercept of the calculated linear regression equation relating the



two tests was not significantly different from zero for the MAOD and the test-retest correlation coefficient was .97. For TE, the intercept of 20 s was different from zero ($p = .02$) with a test-retest correlation of .94.

There were no significant differences between groups for their pretreatment values for body weight, $VO_2max$, the MAOD, or TE during the MAOD test. Table 1 lists the mean values ± *SEM* for the variables of interest. There were no changes in any of the measured variables in the PL group across trials. In contrast, body weight increased by 0.7 kg after treatment in the CR group, and was still higher 7 days after treatment compared to the pretreatment values. The MAOD, TE, and the oxygen uptake during the MAOD test were also significantly increased after treatment in the CR group, and all of these variables were still significantly different from the pretreatment values 7 days after treatment; the latter two trials did not differ from each other.

Figure 2 shows the individual subject values for the MAOD on the various trials. The mean change in the MAOD from the pretreatment to the posttreatment trial was 0.367 L. Twelve of the 14 CR subjects (including two of the 3 females in this group) increased their MAOD after treatment, although the range of the change was substantial, from a slight decrease of 0.084 L to an increase of 0.988 L. Only one of the CR subjects, a female, did not have an increased TE after treatment; the changes ranged from that subject's decreased TE by 8 s to an increase of 24 s and the mean change was 11 s. Although the mean relative changes in the MAOD and TE were similar in CR, the individual changes in these variables were not significantly correlated.

## Discussion

The results of the present study support the hypotheses that the MAOD would be increased following 5 days of creatine treatment, and that the effect would persist for at least 7 days after treatment. The relative increase in the MAOD after creatine treatment was about 10%, similar to the relative change caused by 6 weeks of anaerobic training (Medbø and Burgers, 1990), and is commensurate with the 9% change in TE during the MAOD test. These results support those studies reporting ergogenic effects after similar creatine treatments (Balsom et al., 1993a; Birch et al., 1994; Earnest et al., 1995; Greenhaff et al., 1993; Harridge et al., 1994; Harris et al., 1993). With the exception of the Earnest et al. (1995) study, which reported muscular strength improvements, the other studies involved repeated bouts of exercise; our study extends the ergogenic effects to include a single bout of supramaximal intensity exercise to exhaustion.

Although all but two subjects had an increased MAOD after creatine treatment, there was variation in the extent of that increase. Such a finding is consistent with the observations that not all subjects respond to creatine supplementation with increases in [TCr]. While there are no [TCr] measurements for the present study, there are reports that those subjects with relatively low pretreatment concentrations have demonstrated the greatest increases in [TCr] after supplementation, whereas those subjects with relatively high pretreatment levels may show no change (Gordon et al., 1995; Greenhaff, 1995; Harris et al., 1992).

**Table 1 Values for Variables of Interest Before (Pre), After (Post), and After 7 Days of Treatment With Placebo or Creatine.**

| | Placebo ($n$ = 12) | | | Creatine ($n$ = 14) | | |
|---|---|---|---|---|---|---|
| | Pre | Post | 7 days post | Pre | Post | 7 days post |
| Body weight (kg) | 75.1 ± 3.1 | 75.3 ± 3.0 | 75.2 ± | 80.9 ± 2.9 | 81.6 ± 3.0* | 81.6 ± 3.0* |
| Time to exhaustion (s) | 130.9 ± 6.7 | 134.0 ± 6.2 | 131.7 ± | 129.5 ± 7.0 | 140.6 ± 7.1** | 138.5 ± 8.3** |
| MAOD (L) | 4.020 ± 0.392 | 4.028 ± 0.376 | 3.981 ± 0.390 | 4.038 ± 0.313 | 4.405 ± 0.342** | 4.314 ± 0.328** |
| Oxygen uptake (L) | 6.290 ± 0.491 | 6.488 ± 0.505 | 6.354 ± 0.462 | 6.697 ± 0.580 | 7.283 ± 0.611** | 7.203 ± 0.696* |

*Note.* Values are $M \pm SE$. MAOD = maximum accumulated oxygen deficit.
*$p < .05$ from pretreatment. **$p < .01$ from pretreatment.






**Figure 2.** The maximum accumulated oxygen deficit (MAOD) measured pretreatment, posttreatment, and 7 days posttreatment. Individual subject data are connected with a unique symbol. Mean trial values are shown as a histogram. Statistical significance for the creatine group refers to comparisons with the pretreatment value.

The increased body weight observed after creatine treatment has been reported in several other studies (Balsom et al., 1993a; Earnest et al., 1995; Greenhaff et al., 1993). There was no relationship within the CR group between body weight and the extent of the change in the MAOD after treatment, or between body weight and weight change after treatment. Body composition measurements were not included in the present study, but with such a short treatment period, our presumption is that the increased weight is a result of increased water retention.

The basic premise of our study was that any observed increase in MAOD would be caused by an increase in muscle creatine concentration, which would enhance ATP turnover via anaerobic metabolism. Since there were no muscle tissue metabolite measurements done in this study, this premise cannot be verified. Others, however, reported changes in [TCr] after creatine supplementation in the range of 24–40 mmol · $kg^{-1}$ dry muscle or about 7.5 mmol · $kg^{-1}$ wet weight, most of which has been attributed to increases in free creatine (Harris et al., 1992). Presuming, as suggested earlier, that this free creatine would be available stoichiometrically for PCr synthesis and that 20 kg of muscle are recruited during cycle exercise, then about 150 mmol of additional high energy phosphates would be available. Assuming a 6:1 P:O ratio, conversion yields about 25 mmol of oxygen or 560 ml of oxygen. Thus, the observed mean increase in the MAOD of 0. 4 L of oxygen consumption equivalents after creatine treatment does indeed seem reasonable, assuming that this increase was attributable to an increase in [TCr].

There are many studies which document the MAOD test's methodology, reproducibility, validity, and sensitivity to training (Bangsbo et al., 1990; Bleue and Jacobs, 1994; Gastin and Lawson, 1994; Medbø, 1996; Medbø and Burgers, 1990; Medbø et al., 1988; Oleson et al., 1994; Ramsbottom et al., 1994; Scott et al., 1991). There has been concern expressed, however, that the calculation of the MAOD in oxygen consumption equivalents necessitates the estimation of an oxygen cost of exercise for an intensity of exercise that exceeds maximal aerobic power. This calculation was done by extrapolating mechanical efficiency during submaximal exercise to supramaximal intensities, but the presumption of similar mechanical efficiencies across such a wide range of exercise intensities has recently been questioned (Bangsbo, 1996; Green and Dawson, 1995). In summarizing his review of the validity of the MAOD test, Graham (1996) concluded that although comparisons of the MAOD between groups of subjects may be confounded by an inappropriate calculation of the oxygen cost of exercise, the MAOD is a useful tool for studies with repeated measures designs. Thus, even if the calculation of the absolute oxygen cost of exercise during the test is not precise in the present study, the main findings would not change because of the repeated measures design of this study.

We have no reason to believe that the oxygen cost of exercise would change substantially from trial to trial. A potentially confounding factor would be if the creatine ingestion per se influenced the slope of the regression of oxygen consumption on exercise intensity, and this aspect was not investigated in the present study. However, Stroud et al. (1994) reported that creatine ingestion did not affect the oxygen cost of submaximal exercise, and Balsom et al. (1993b) reported that maximal aerobic power was not affected by creatine supplementation. Thus, there is no reason to believe that mechanical efficiency of exercise was altered simply because of the creatine treatment.

With regard to the supplementary hypothesis about the persistence of any effects after ceasing creatine treatment, Greenhaff (1995) surmised that the natural degradation of elevated intramuscular creatine stores is likely to occur over several weeks. In their study with direct measurements of muscle creatine concentration Hultman et al. (1996) reported that elevated creatine concentrations were sustained between 7 and 14 days after stopping supplementation. Both Hultman et al. (1996) and Febbraio et al. (1995) reported that muscle creatine levels were no longer elevated 28 days after stopping supplementation. These reports are consistent with the increased MAOD that was still observed in the present study 7 days after stopping the creatine supplementation.

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