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Effects of 28 days of beta-alanine and creatine monohydrate supplementation on aerobic power, ventilatory and lactate thresholds, and time to exhaustion

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Summary. The effect of beta-alanine (β-Ala) alone or in combination with creatine monohydrate (Cr) on aerobic exercise performance is unknown. The purpose of this study was to examine the effects of 4 weeks of β-Ala and Cr supplementation on indices of endurance performance. Fifty-five men (24.5 ± 5.3 yrs) participated in a double-blind, placebo-controlled study and randomly assigned to one of 4 groups; placebo (PL, $n = 13$), creatine (Cr, $n = 12$), beta-alanine (β-Ala, $n = 14$), or beta-alanine plus creatine (CrBA, $n = 16$). Prior to and following supplementation, participants performed a graded exercise test on a cycle ergometer to determine $\dot{V}O_{2peak}$, time to exhaustion (TTE), and power output, $\dot{V}O_2$, and percent $\dot{V}O_{2peak}$ associated with VT and LT. No significant group effects were found. However, within groups, a significant time effect was observed for CrBa on 5 of the 8 parameters measured. These data suggest that CrBA may potentially enhance endurance performance.

Keywords: Beta-alanine – Creatine – Exercise – Lactate threshold – Ventilatory threshold

Introduction

It is well documented that athletes involved in sports that require high-intensity intermittent exercise performance ingest supplements containing creatine (Cr) in an effort to enhance anaerobic performance. The performance enhancing effects of Cr supplementation have been attributed to several factors including increasing skeletal muscle phosphocreatine (PCr) content, improved PCr resynthesis, increased buffering capacity, and greater shuttling of mitochondrial ATP into the cytoplasm (Harris et al., 1992; Hultman et al., 1996). In support, a few studies have demonstrated that supplementation with Cr resulted in a 2–5% decrease in blood lactate accumulation (Balsom et al., 1995; Febbraio et al., 1995; Prevost et al., 1997), and a 2–3% lower oxygen consumption ($\dot{V}O_2$) (Balsom et al., 1995) associated with repeat bouts of anaerobic exercise. In contrast, only a few studies have evaluated the effects of Cr supplementation on submaximal exercise performance. Nelson et al. (2000) showed that seven days of Cr supplementation significantly decreased submaximal $\dot{V}O_2$ and increased $\dot{V}O_2$ and percent $\dot{V}O_{2peak}$ at the ventilatory threshold as well as time to exhaustion. Jones et al. (2002) found a significantly decreased $\dot{V}O_2$ during submaximal exercise above the ventilatory threshold with 10 days of Cr supplementation.

Oral beta-alanine (β-Ala) supplementation has also been suggested to improve high intensity exercise performance (Harris et al., 2003; Hill et al., 2005). Recent studies by Hill et al. (2005) and Harris et al. (2005) have demonstrated that 28 days of β-Ala supplementation ($4–6\,g\,d^{-1}$) increased intramuscular levels of carnosine (beta-alanyl-L-histidine) by approximately 60%. The proposed physiological roles of carnosine in skeletal muscle are many and include pH buffering (Begum et al., 2005; Severin et al., 1953; Harris et al., 1990), functioning as an anti-oxidant (Boldyrev et al., 1987), regulating muscle contractility by exerting effects on Ca^{2+} cycling (Batrukova and Rubstov, 1997), Ca^{2+} sensitivity (Lamont and Miller, 1992) and excitation-contraction coupling (Begum et al., 2005; Petukhov et al., 1976), inhibiting protein glycation (Hipkiss et al., 1995), and preventing formation of protein–protein cross links (Hipkiss, 2000).

Several studies suggest that carnosine's role as a pH buffer may be an important factor in determining high-intensity exercise performance (Suzuki et al., 2002; Harris

et al., 2003). Suzuki et al. (2002) suggested that carnosine serves as a buffer and helps maintain skeletal muscle acid-base homeostasis when large quantities of hydrogen ions (H^+) are produced during a 30 sec sprint. Harris et al. (2003) demonstrated improvements in performance during a 4-minute all-out/maximal cycle ergometry test in men after supplementing with β-Ala ($3.2\,g\,d^{-1}$) for 5 weeks. The authors concluded that the improvements may have been due to an enhanced H^+ buffering capacity as a result of increased muscle carnosine levels after β-Ala supplementation.

Hill et al. (2005) investigated the potential synergistic effect of Cr and β-Ala on high-intensity exercise performance by comparing total work performed at 110% of pre-determined maximal power out on a cycle ergometer under four supplementation conditions: 1) β-Ala ($4.0\,g\,d^{-1}$ increasing to $6.4\,g\,d^{-1}$ at week 4), 2) β-Ala (same dose) and Cr ($20\,g\,d^{-1}$ during week 4), 3) placebo, and 4) Cr ($20\,g\,d^{-1}$ during week 4). With the exception of the placebo condition, an increase in total work was seen in all groups, but the effects were not additive. A synergistic effect of β-Ala and Cr supplementation on aerobic exercise performance has also been proposed, but no studies have been conducted on the effect of β-Ala, either alone or in combination with Cr, on indices of cardiorespiratory endurance. Therefore, the purpose of this study was to examine the effects of 4 weeks of β-Ala and/or Cr supplementation on $\dot{V}O_{2peak}$, lactate and ventilatory thresholds, and time to exhaustion measured during a graded exercise test on a cycle ergometer.

Materials and methods

Subjects

Fifty-five men (mean age ± SD = 24.5 ± 5.3 yrs, height = 171.9 ± 27.9 cm and weight = 82.0 ± 7.1 kg) volunteered for this investigation. Before initiating the study, all participants were informed of all procedures and risks associated with the study and signed an informed consent in accordance with the Florida Atlantic University Institutional Review Board for Human Subjects Experimentation.

Supplementation protocol

None of the subjects had ingested creatine, or any other dietary supplements, for a minimum of 12 weeks before the initiation of the study. During the course of the study, the subjects were instructed to maintain their current exercise and dietary patterns and abstain from other nutritional supplements, nonprescription drugs, and caffeine. After pre-testing, the subjects were randomly assigned to one of four treatment conditions using a double-blind design: a) placebo (PL; 34 g of dextrose; $n = 13$), b) creatine (Cr; 5.25 g of creatine monohydrate plus 34 g of dextrose; $n = 12$), c) beta-alanine (Carnosyn™, NAI, San Marcos, CA) (β-Ala; 1.6 g beta-alanine plus 34 g of dextrose; $n = 14$), or d) beta-alanine plus creatine (Phosphagen Elite™, EAS, Golden, CO) (CrBA; 5.25 g of creatine monohydrate; 1.6 g beta-alanine plus 34 g of dextrose; $n = 16$). The supplements were identical in taste and appearance, and were dissolved in 16 oz of water and ingested four times per day for 6 consecutive days, then twice per day for 22 days before post-testing.

Exercise tests

Prior to and following the supplementation protocol, subjects performed a continuous graded exercise test (GXT) on an electronically braked cycle ergometer (Excalibur Sport, Groningen, Netherlands) to determine $\dot{V}O_{2peak}$, ventilatory threshold (VT) and lactate threshold (LT). Pre- and post-supplementation testing took place at the same time of day for each subject and on the same equipment. Post-supplementation GXT was administered within 24 h after termination of the supplementation protocol. Subjects reported to the lab after fasting for 3 h prior to each test. For each GXT, the initial power output was set at 30 watts and increased 30 watts every two minutes until the subject could not maintain the required power output at a pedaling rate of 70 rpm or volitional termination due to fatigue. Gas analyzers were calibrated with room air and gases of known concentration prior to each test. Respiratory gases ($\dot{V}_E$, $\dot{V}O_2$, $\dot{V}CO_2$), and RER were measured continuously with open-circuit spirometry (True One 2400® Metabolic Measurement System, Parvo-Medics Inc., Provo, UT) and data was averaged over 30 sec intervals. The highest 30 sec values for respiratory/metabolic data measured during the GXT were recorded as maximal values. Indicators used to confirm that $\dot{V}O_{2peak}$ was attained included a plateau in heart rate (HR) values or attainment of HR values within 10% of age-predicted HR_{max}, a plateau in oxygen uptake (defined by an increase of not more than $150\,ml\,min^{-1}$) and a RER value greater than 1.15.

Heart rate was measured continuously using a Polar heart rate monitor (a_1, model # 1902690, Polar Electro Inc, Lake Success, NY). Blood samples were obtained by a fingerstick for the determination of blood lactate concentration immediately prior to each GXT, during the last 15 sec of each exercise stage, at termination, and four minutes post-exercise. Blood samples were analyzed for blood lactate concentration with a YSI 2300 STAT PLUS Lactate Analyzer (Yellow Springs Inc., Yellow Springs, OH).

Determination of lactate and ventilatory thresholds

Lactate threshold was determined from a plot of blood lactate values against power output values as previously described by Weltman et al. (1990). Lactate threshold was defined as the highest power output attained during the GXT's prior to a nonlinear increase in blood lactate concentration. The ventilatory threshold was determined from a plot of ventilation ($\dot{V}_E$) against $\dot{V}O_2$ as described previously (Orr et al., 1982). The determination of lactate and ventilatory thresholds was performed solely by the principle investigator who was blinded as to the identity of the subjects and the experimental condition. Examples of LT and VT determination are presented in Fig. 1. The power output, $\dot{V}O_2$, and percent $\dot{V}O_{2peak}$ associated with the LT and VT were recorded. These six values, along with $\dot{V}O_{2peak}$ and time to exhaustion (TTE) measured during the GXT were used as the indices of cardiorespiratory endurance.

Statistical analysis

Separate one-way analyses of variance (ANOVA) were used to analyze pre-test data for group differences in the indices of cardiorespiratory endurance. The independent variable, group, included four levels: PL, β-Ala, Cr, and CrBA. Data were then analyzed with a 2 by 4 (time by group) repeated measures analysis of co-variance (ANCOVA) using pre-supplementation variables demonstrating significant between-group differences as covariates. When appropriate, Bonferoni-adjusted post-hoc pairwise comparisons were used to examine differences between groups.

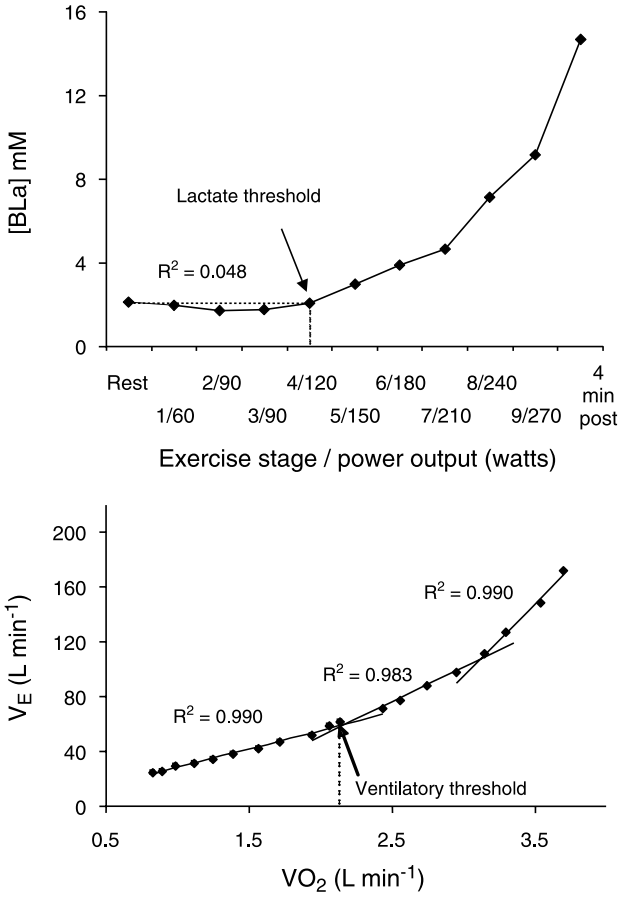

Fig. 1. Examples of lactate and ventilatory threshold determination

An alpha of $P \leq 0.05$ was established a priori. SPSS version 13 (SPSS, Inc., Chicago, IL) was used for all statistical analyses.

Results

Table 1 contains the mean ± SD values for the pre- and post-supplementation test results. ANOVA of pre-supplementation data revealed a between-group difference for percent $\dot{V}O_{2peak}$ at the LT and this variable was used as a covariate as described above. ANCOVA did not indicate a significant difference ($P > 0.05$) among the group means for the post-supplementation values for the indices of cardiorespiratory endurance after adjusting for the pre-supplementation differences. However, within groups, significant ($P < 0.05$) time effects (pre- to post-supplementation) were seen as indicated by $*$ in Table 1. Supplementation with CrBA demonstrated a significant improvement in five out of the eight indices of cardiorespiratory endurance, specifically $\dot{V}O_2$ and power output at LT and VT, as well as percent $\dot{V}O_{2peak}$ at VT. Supplementation with Cr alone only showed improvement in TTE and power output at VT. Only power output at LT was

Table 1. Indices of endurance performance, pre- and post-supplementation (mean ± SD)

	Placebo		Cr		CrBA		β-Ala	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
VO_{2peak} (L/min)	3.27 ± 0.76	3.42 ± 0.80	3.17 ± 0.65	3.27 ± 0.59	3.15 ± 0.72	3.20 ± 0.79	2.83 ± 0.71	2.86 ± 0.78
LT (VO_2 (L/min))	1.94 ± 0.54	1.81 ± 0.51	1.83 ± 0.48	1.93 ± 0.45	1.74 ± 0.40	1.84 ± 0.44*	1.94 ± 0.63	1.84 ± 0.56
LT (watts)	143.1 ± 49.2	126.9 ± 44.4*	126.4 ± 39.3	137.1 ± 32.7	125.6 ± 36.7	136.9 ± 37.9*	130.0 ± 43.1	142.5 ± 42.7*
LT (% VO_{2peak})	61.6 ± 6.8	53.2 ± 9.6*	57.6 ± 5.8	58.7 ± 7.3	57.4 ± 10.7	58.6 ± 8.4	64.5 ± 11.4	65.5 ± 10.4
VT (VO_2 (L/min))	2.18 ± 0.62	2.18 ± 0.49	1.99 ± 0.57	2.12 ± 0.57	2.02 ± 0.50	2.18 ± 0.42*	1.88 ± 0.66	1.80 ± 0.55
VT (watts)	175.4 ± 54.6	170.8 ± 39.5	147.9 ± 44.8	162.9 ± 45.1*	161.3 ± 45.0	178.1 ± 37.1*	142.5 ± 57.4	157.5 ± 54.5
VT (% VO_{2peak})	68.9 ± 4.9	64.2 ± 6.8	61.7 ± 10.2	64.3 ± 10.8	64.7 ± 10.5	69.8 ± 11.4*	62.6 ± 11.5	67.4 ± 9.2
TTE (sec)	977.8 ± 253.9	1002.5 ± 258*	914.7 ± 174.1	951.2 ± 171*	944.4 ± 204.9	964.9 ± 201.0	831.4 ± 202.0	864.0 ± 221.5

improved with β-Ala supplementation. The placebo group showed a significant *decrease* in percent $\dot{V}O_{2peak}$ and power output at the LT, but an increase in TTE.

Discussion

The most noteworthy finding of this study was the significant increase in five of eight indices of cardiorespiratory endurance with CrBA supplementation. Individually, supplementation with Cr showed improvements in power output at VT and TTE, while β-Ala only demonstrated an improvement in power output at LT. A significant improvement in TTE was seen in the placebo group, but this was accompanied by decreases in power output and percent $\dot{V}O_{2peak}$ at LT. The improvement in TTE seen in the placebo group appears to have been driven by relatively large increases in four of the subjects. These individuals demonstrated increases in TTE of 40, 45, 62, and 63 sec compared with a non-significant decrease of 15.4 ± 7.2 sec in the remainder of the group. However, any conclusions based on these findings must be tempered by the fact that there were no significant between-group effects.

Regardless, the present data at least suggest that supplementation with CrBA may enhance the potential for submaximal endurance performance as measured by the lactate and ventilatory thresholds. Very recently, Harris et al. (2006) demonstrated that supplementation with CrBA, as used presently, resulted in significant increases ($9.5\,mmol\,kg^{-1}$ dry mass, $P<0.01$) in muscle carnosine content. The authors concluded that "The increase in muscle carnosine with β-Ala supplementation did not appear to be reduced by the presence of taurine (or creatine) present in the commercial supplement." Further, the increased muscle carnosine content resulting from CrBA supplementation was associated with an improvement in $\dot{V}O_2$ at VT and TTE in response to a maximal graded exercise test performed on a cycle ergometer.

The results of several investigations of the effect of CrBA supplementation on supramaximal exercise have also been reported. Hill et al. (2005) found that supplementation with both Cr and β-Ala improved cycling performance (TTE) at 110% $\dot{V}O_{2max}$ however, the effects were not additive. Supplementation with β-Ala alone increased average power output during all-out cycling exercise of four minutes duration (Harris et al., 2003). The improvement in performance, however, was limited to the first minute of exercise, before full cardiovascular adjustment. The authors concluded that this was due to H^+ buffering by carnosine during this transitional period. Presently, our data demonstrate that the number of significant improvements in the performance indices with CrBA supplementation were greater than would be expected based on the results seen with Cr or β-Ala alone and did not appear to be additive.

In contrast to β-Ala, the effects of Cr supplementation on submaximal exercise performance have been the focus of several investigations. Nelson et al. (2000) found that Cr supplementation significantly lowered submaximal $\dot{V}O_2$ as well as heart rate and delayed the onset of the ventilatory threshold (expressed as either absolute or relative $\dot{V}O_2$). The authors suggested that Cr supplementation may have increased the relative contribution of the anaerobic energy system(s). They further proposed that this, in turn, reduced the aerobic contribution to ATP resynthesis resulting in reduced submaximal $\dot{V}O_2$ and heart rate. Jones et al. (2002) examined the effects of Cr supplementation on $\dot{V}O_2$ during repeat bouts of submaximal cycling exercise of 6 min duration at moderate (defined as the power output corresponding to 80% VT) and high intensities (defined as the power output midway between VT and $\dot{V}O_{2max}$). They found that Cr lowered $\dot{V}O_2$ during high intensity, but not moderate intensity exercise. In contrast, Stroud et al. (1994) found no change in submaximal $\dot{V}O_2$ during incremental treadmill exercise. The current investigation did not examine $\dot{V}O_2$ at specific submaximal workloads, but did demonstrate a significant improvement in power output at VT.

Recognizing that supplementation with Cr often results in increased body mass (Earnest et al., 1996; Greenhaff et al., 1993; Hultman et al., 1996), data were reported using absolute values to remove the influence of body weight. Additionally, we further investigated the potential confound of changes in body weight by 1) using matched-pair *t*-tests to determine if there were significant changes in body weight pre- to post-supplementation within each condition and 2) using correlation analysis to search for significant relationships ($P<0.05$) between changes in body weight and changes in the indices of endurance performance. The results of these analyses revealed that only the CrBA group demonstrated a significant change in body weight (1.05 kg, $P<0.05$). While the Cr group showed an apparent increase in body weight (1.31 kg), it was not significant ($P>0.05$). Correlation analysis showed no relation between changes in body weight with changes in any of the indices of endurance performance in any group with the sole exception of a positive relation ($r=0.58$, $P<0.05$) between change in body weight with change in time to exhaustion in the Cr group. As such, we conclude that the changes in body weight seen presently had little if any relationship to the changes in the indices of performance.

The supplementation protocols for the present study have been demonstrated to significantly increase intramuscular levels of Cr (Greenhaff et al., 1993) and β-Ala (Harris et al., 2005). However, there is evidence that uptake of these supplements into skeletal muscle is not uniform among all individuals. Greenhaff et al. (1993) found that approximately 20% of individuals were "non-responders" in that intramuscular levels of creatine did not increase appreciably ($<10\,mmol\,kg^{-1}$ dry mass) in response to oral Cr supplementation. These authors further suggest that the increase in muscle creatine content may be directly related to pre-supplementation levels of intramuscular creatine. Specifically, the greatest increases were seen in individuals with a total intramuscular creatine content of $<120\,mmol\,kg^{-1}$ dry mass. There may be an upper limit for creatine stores in skeletal muscle of about $160\,mmol\,kg^{-1}$ dry mass. Harris et al. (2005) showed variability in the response of muscle carnosine content to four weeks of supplementation with β-Ala. Twenty percent of these subjects (3 out of 15) exhibited changes in carnosine levels that were clearly different in magnitude from the other subjects. Interestingly, there did not seem to be a specific pattern, as one subject showed no change, while the response of the other two was 2.5–3 times greater than any of the other subjects. Given that we were unable to measure muscle creatine or carnosine content, the possibility remains that the findings of the present study may have been influenced by the documented variability in the skeletal muscle response to oral supplementation with Cr and/or β-Ala.

In summary, four weeks of supplementation with CrBA demonstrated significant improvements in five of eight indices of cardiorespiratory endurance measured during incremental cycle ergometry. Supplementation with Cr and β-Ala resulted in improvements in two and one of the indices, respectively. The placebo group demonstrated decreases in two indices and improvement in one. While it is important to reiterate that the improvements were not significant when compared between groups, these data at least suggest that supplementation with CrBA especially may delay the onset of the VT and LT during incremental cycle exercise in men. Future studies should examine muscle carnosine and/or PCr levels along with blood lactate concentration during submaximal fatiguing exercise with and without β-Ala and/or Cr supplementation.

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