Effect of Beta-alanine Supplementation on the Onset of Blood Lactate Accumulation (OBLA) During Treadmill Running: Pre/post 2 Treatment Experimental Design

Judith Lukaszuk

Thomas Jordan

Mark Misic

Josephine Umoren

Follow this and additional works at: https://huskiecommons.lib.niu.edu/allfaculty-peerpub

Original Citation

This Article is brought to you for free and open access by the Faculty Research, Artistry, & Scholarship at Huskie Commons. It has been accepted for inclusion in Faculty Peer-Reviewed Publications by an authorized administrator of Huskie Commons. For more information, please contact jschumacher@niu.edu.
Effect of beta-alanine supplementation on the onset of blood lactate accumulation (OBLA) during treadmill running: Pre/post 2 treatment experimental design

Thomas Jordan¹, Judith Lukaszuk*¹, Mark Misic² and Josephine Umoren¹

Abstract
Background: Beta-Alanine (βA) has been shown to improve performance during cycling. This study was the first to examine the effects of βA supplementation on the onset of blood lactate accumulation (OBLA) during incremental treadmill running.

Methods: Seventeen recreationally-active men (mean ± SE 24.9 ± 4.7 yrs, 180.6 ± 8.9 cm, 79.25 ± 9.0 kg) participated in this randomized, double-blind, placebo-controlled pre/post test 2-treatment experimental design. Subjects participated in two incremental treadmill tests before and after 28 days of supplementation with either βA (6.0 g·d⁻¹) (βA, n = 8) or an equivalent dose of Maltodextrin as the Placebo (PL, n = 9). Heart rate, percent heart rate maximum (%HRmax), %VO₂max@OBLA (4.0 mmol.L⁻¹ blood lactate concentration) and VO₂max (L.min⁻¹) were determined for each treadmill test. Friedman test was used to determine within group differences; and Mann-Whitney was used to determine between group differences for pre and post values (p < 0.05).

Results: The βA group experienced a significant rightward shift in HR@OBLA beats.min⁻¹ (p < 0.01) pre/post (161.6 ± 19.2 to 173.6 ± 9.9) but remained unchanged in the PL group (166.8 ± 15.8 to 169.6 ± 16.1). The %HRmax@OBLA increased (p < 0.05) pre/post in the βA group (83.0% ± 9.7 to 88.6% ± 3.7) versus no change in the PL group (86.3 ± % 4.8 to 87.9% ± 7.2). The %VO₂max@OBLA increased (p < 0.05) in the βA group pre/post (69.1 ± 11.0 to 75.6 ± 10.7) but remained unchanged in the PL group (73.3 ± 7.3 to 74.3 ± 7.3). VO₂max (L.min⁻¹) decreased (p < 0.01) in the βA group pre/post (4.57 ± 0.8 to 4.31 ± 0.8) versus no change in the PL group (4.04 ± 0.7 to 4.18 ± 0.8). Body mass kg increased (p < 0.05) in the βA group pre/post (77.9 ± 9.0 to 78.3 ± 9.3) while the PL group was unchanged (80.6 ± 9.1 to 80.4 ± 9.0).

Conclusions: βA supplementation for 28 days enhanced sub-maximal endurance performance by delaying OBLA. However, βA supplemented individuals had a reduced aerobic capacity as evidenced by the decrease in VO₂max values post supplementation.

Background
Running is a popular form of exercise in the United States and for many it is considered a competitive sport. While performance goals can range from simply finishing a race to competition in an Olympic event, it is likely that many participants seeking to improve performance use various nutritional supplements.

One such supplement that has recently received interest in improving exercise performance is Beta-Alanine (βA) [1-10]. βA is a non-proteinogenic amino acid that is synthesized in the liver as the final metabolite of uracil and thymine degradation. While produced endogenously, the primary source of βA in humans comes from their diet. Meat is the primary source of dietary βA, with highest concentrations found in chicken and turkey [11]. The performance enhancing potential of βA supplementation lies in its effect on increasing muscle carnosine levels.
due to its role as the limiting factor in the muscle carnosine synthesis [12-14].

Carnosine (β-alanyl-L-histidine) is a dipeptide found in muscle tissue that acts as an intramuscular buffer of [H+] [4,7,8,12]. During high intensity exercise, a greater reliance on the glycolysis and phosphagen systems to supply ATP to working muscles results in an accumulation of [H+] which leads to exercise-induced metabolic acidosis [15]. A decline in pH has been implicated as a cause of muscle fatigue and decreased muscle contractile function [16]. Attenuating exercise induced acidosis is purported to result in performance improvements in activities requiring prolonged bouts of high intensity work. This is supported by findings that muscle carnosine concentrations are higher in sprinters [17], bodybuilders [18], and team sport athletes regularly participating in high intensity intermittent exercise [19,20] than in their sedentary counterparts.

Previous studies investigating the effect of βA on performance measures have shown improvements in total work done (TWD) [4,10], time to exhaustion (TTE) [1,4,10], physical working capacity at fatigue threshold (PWCFT) [1,3], power output at lactate threshold (LT) [5], attenuated fatigue during repeated bouts of resistance training [7], and final 30 second sprint performance during a 2 hour time trial [9]. Research has however been conducted using primarily cycle ergometry [1-5,9,10], so it remains to be determined if βA supplementation would have an ergogenic effect during running performance. Therefore, we hypothesized that βA supplementation would delay OBLA. Therefore, the purpose of this study was to determine the effects of 4 weeks of βA supplementation on HR@OBLA, %HRmax@OBLA, %VO2max@OBLA, VO2max during incremental treadmill running.

Methods

Subjects

Seventeen men who were recreationally active and running at least 3 times per week and had not taken any sports supplements for at least 6 weeks volunteered to participate in this study (Table 1). Subjects provided signed consent to participate and all study procedures were approved by the Northern Illinois University Institutional review board prior to enrollment in the study.

Experimental Design

This study used a double-blind-placebo-controlled pre/post test 2-treatment experimental design. On days 1 and 29, subjects reported to the exercise lab for anthropometric collection and to perform an incremental treadmill running protocol. During the 28 day study, subjects were randomly assigned to consume a supplement containing either βA (6.0 g·d⁻¹) or Placebo (PL) Maltodextrin (6.0 g·d⁻¹). Pre- and post-supplementation testing took place at the same time of day for each subject and on the same equipment. Subjects were asked to fast for 2 hrs prior to each test. Subjects were asked to abstain from taking any other dietary supplements and to maintain their regular diet and exercise patterns for the duration of the study. Subjects were also required to abstain from caffeine or vigorous exercise for 24 hrs before exercise testing.

Anthropometric data were recorded in light exercise clothing and bare feet using a wall mounted stadiometer and calibrated digital scale (Tanita Body Composition Analyzer TBF-300A, Tanita Corp, Arlington Heights, IL). Subjects were connected to an automated metabolic measurement system (Parvomedics TrueMax 2400, Concentius Technologies, Sandy, UT) via mouthpiece and headset and fitted with a telemetric heart rate monitor (Polar F6, Finland) in seated position for resting variables prior to testing.

Participants performed 3 minutes of walking on the treadmill at 6.4 km.hr⁻¹ (4.0 mph) to acclimate to the apparatus. The treadmill was then set at a fixed 9.6 km.hr⁻¹ (6.0 mph) for the duration of the test. Every 3 minutes, the treadmill incline was increased by 2% grade. After stage 5, any remaining stages ensued at 3% grade increase (stages: 0%, 2%, 4%, 6%, 8%, 11%, 14%, 17%). The test continued until the participant reached volitional exhaustion. Oxygen uptake was obtained every 30 seconds (s) throughout the test. VO2max was recorded as the highest 30 s average recorded prior to volitional exhaustion. Criteria for VO2max was attainment of at least two or more of the following: reaching a plateau in VO2 (< 2.1 ml.kg⁻¹.min⁻¹ increase) the final two stages of the test, achieving

<table>
<thead>
<tr>
<th>Table 1: Physical Characteristics of Subjects.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Variable</strong></td>
</tr>
<tr>
<td>Age (yr)</td>
</tr>
<tr>
<td>Height (cm)</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
</tr>
<tr>
<td>BMI</td>
</tr>
</tbody>
</table>

Values are means ± SE
a respiratory exchange ratio (RER ≥ 1.10) and/or reaching a HR within 5 beats per min⁻¹ of predicted maximal value (220 - age). In the final 30 s of each stage, participants were asked to report an overall body rating of perceived exertion (RPE) using a 6–20 numeric scale [21], heart rate was recorded, and a capillary blood lactate sample was collected. Subjects were oriented to the RPE scale prior to initiation of the test.

A fixed marker of 4.0 mmol·L⁻¹ blood was used to define the onset of blood lactate accumulation (OBLA). This fixed lactate measurement provides the most reasonable and accurate lactate analysis relative to the scope of this study and has been shown to be a valid evaluation of physiological changes with specificity to endurance performance [17], and improvements in endurance fitness [18].

Immediately following RPE and HR data collection with 30 s remaining in each stage, subjects while running were asked to extend their left index finger, which was prepared with an alcohol pad and dried with gauze. After preparation, a lancet device was applied to the fingertip and samples were collected in capillary tubes. All lactate samples were immediately analyzed in duplicate using an Accutrend Lactate Analyzer (F. Hoffman-La Roche Ltd, Basel, Switzerland). After compiling the data, the stage that elicited 4.0 mmol·L⁻¹ blood lactate which has been previously identified as the OBLA [22] was used to determine lactate threshold. OBLA, VO₂max@OBLA and HR@OBLA were all calculated using linear interpolation between relevant data points as has been previously explained by Neville et al. [23].

The treadmill protocol continued until volitional exhaustion was attained and the highest heart rate experienced during the test was recorded as Max Heart Rate (MHR). OBLA was then also identified by the percentage of maximum heart rate (%MaxHR@OBLA) at which it occurred.

**Supplementation**

During the study, subjects were asked to refrain from taking any other dietary supplements or making changes to their regular dietary and exercise patterns. The participants were randomly assigned in a double-blind manner to receive either β-Alanine or Placebo. The supplements were supplied by Athletic Edge Nutrition, Miami, Florida. Subjects received βA supplement (6.0 g·d⁻¹ βA, 600 mg N-Acetylcysteine, 2.7 mg alpha-lipoic acid, 45 IU Vitamin E) or a PL (6.0 g·d⁻¹ Rice Flour Maltodextrin). Both groups followed the same supplementation protocol of 3 capsules 3 times daily with meals.

Supplementing with 6.4 g·d⁻¹ of βA for 28 days has been shown to increase carnosine levels by 60% [4,12] so it can be assumed that supplemented subjects in this study experienced a significant increase in intramuscular carnosine concentration. Three of the eight subjects in the βA supplemented group reported tingling in their fingers and hands. No other side effects were reported by those individuals supplemented with βA and subjects in the PL group reported no side effects.

**Statistical Analysis**

Because of the degree of non-normality in the distributions, data transformation could not be done to obtain statistical normality. For this reason, nonparametric statistical methods were used to analyze the data. The Friedman test was used to determine within group differences; and the Mann-Whitney test was used to determine between group differences. Data were analyzed using SPSS for Windows (Version 16.0, 2007 Chicago, IL) Prior to initiation of the study the alpha level was set at p < 0.05 to determine statistical significance. Data are presented as means ± standard error (SE).

**Results**

**Participant Characteristics**

At baseline there were no differences in age, height, body mass, BMI, absolute VO₂max L·min⁻¹ (4.57 ± 0.8 βA vs. 4.04 ± 0.7 PL) relative VO₂max ml.kg⁻¹.min⁻¹ (58.7 ± 50.0 βA vs. 50.0 ± 5.2 PL) or max HR beats. min⁻¹ (195 ± 10.2 βA vs. 193.4 ± 14.9 PL) between subjects in the two groups (Table 1). Table 2 presents the mean and standard error values for VO₂max (L·min⁻¹), VO₂max (ml.kg⁻¹.min⁻¹), %VO₂max @ OBLA, VO₂@ OBLA (L·min⁻¹) MaxHR (beats.min⁻¹), HR@OBLA (beats. min⁻¹), and %HRMax@OBLA for both treatment groups at pre- and post-testing.

**Absolute (L.min⁻¹) and Relative VO₂ max (ml.kg⁻¹.min⁻¹)**

On day 1 pre-supplementation there were no significant differences in VO₂max between subjects in βA and the PL groups (p=.154). On day 29 (post-supplementation) subjects in the βA group had significant decreases in both absolute and relative VO₂max values (p = 0.005), while no changes were observed in the PL group.

%VO₂max@OBLA

On day 1 pre-supplementation there were no significant differences in %VO₂max@OBLA between subjects in the βA and PL groups. On day 29 (post-supplementation) subjects in the βA group had a significant increase (p = 0.034) in %VO₂max@OBLA while no changes were observed in the PL group.

VO₂@OBLA

On day 1 pre-supplementation there were no significant differences in VO₂@OBLA (L·min⁻¹) between subjects in
the βA and PL groups. On day 29 (post-supplementation) no changes were observed in the βA group or PL group.

Heart Rate@OBLA and %HRmax@OBLA
On day 1 pre-supplementation there were no significant differences in heart rate at OBLA (HR@OBLA), or percent maximum heart rate at OBLA (%HRmax@OBLA) between subjects in the two groups. On day 29 (post-supplementation) subjects in the βA group had a significant increase (p = 0.005) in HR@OBLA and %HRmax@OBLA (p = 0.005), while no changes were observed in the PL group. HR@OBLA increased in 8/8 βA supplemented subjects versus 7/9 increased for PL and 2/9 (PL) remained the same post versus pre supplementation. Percent HRmax@OBLA increased in 7/8 (βA) and decreased in 1/8 βA subjects, whereas 6/9 increased for the PL subjects and 3/9 (PL) decreased post versus pre supplementation.

Body Mass
There was a statistically significant increase in mean body mass for the βA group (p = 0.034) post supplementation while there was no change in the PL group. Mean body mass for the βA group increased by 0.4 kg (77.9 ± 9.0 to 78.3 ± 9.3 kg) following the 28 day supplementation period, while no change occurred in the placebo group (80.6 ± 9.1 to 80.4 ± 9.0 kg).

Body Mass Index (BMI)
There was a change in BMI values pre/post supplementation (p = 0.034)(βA 23.7 ± 2.3 vs. PL 23.8 ± 2.3) versus post supplementation (βA 24.9 ± 1.8 vs PL 24.8 ± 1.7).

Rate of Perceived Exertion (RPE)
There were no changes in the final RPE numbers obtained at test termination in the βA group pre/post (18.50 ± .42 to 17.50 ± .82) versus the PL group (18.56 ± .44 to 18.78 ± .32).

Discussion
While previous studies have suggested an ergogenic effect with βA supplementation in cyclists, this was the first study using running as the exercise protocol. In the current study, results showed that βA supplementation delayed OBLA as illustrated by significant increases in HR@OBLA, %HRmax @ OBLA compared to the PL group. These findings are in part consistent with Zoeller et al. who noted an improvement in power output at lactate threshold on a cycle ergometer [5]. These researchers observed no change in ventilatory measures (VO2peak at OBLA), however, it should be noted that Zoeller et al. used a much lower dose of βA (3.2 g·d-1) versus the 6.0 g·d-1 used in this study [5].

While muscle levels of carnosine were not measured for this study previous research has indicated that 4-10 weeks of βA supplementation (2.4-6.4 g·d-1) increased muscle carnosine levels 37-80% [4,7,8,12] and that a significant relationship exists between carnosine concentration and high intensity exercise performance [19]. Furthermore, carnosine levels are higher in trained athletes [20,24,25] and body builders [26] and have been shown to increase in response to high intensity exercise such as sprint training [27].

Ergogenic Mechanism of Carnosine
Physically active individuals have higher muscle carnosine concentrations than their sedentary counterparts [20,24,25] and it is clear that both supplementation with βA [4,7,8,12] and high intensity exercise [28] independently increase muscle carnosine levels. While the exact mechanism of action concerning carnosine and exercise

<table>
<thead>
<tr>
<th>Exercise Parameters Pre and Post Supplementation.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Day 1</strong></td>
</tr>
<tr>
<td>βA</td>
</tr>
<tr>
<td>VO2max (L·min⁻¹)</td>
</tr>
<tr>
<td>VO2max (ml·kg·min⁻¹)</td>
</tr>
<tr>
<td>VO2@OBLA (L·min⁻¹)</td>
</tr>
<tr>
<td>%VO2max@OBLA (%)</td>
</tr>
<tr>
<td>Max HR (beats·min⁻¹)</td>
</tr>
<tr>
<td>HR @OBLA(beats·min⁻¹)</td>
</tr>
<tr>
<td>%HRmax @OBLA (%)</td>
</tr>
</tbody>
</table>

Values are means ± SE; *p < 0.05 Pre Supplementation βA vs. Post Supplementation βA **p < 0.01 Pre Supplementation βA vs. Post Supplementation βA
The effects of βA on OBLA during incremental stages of running. After 28 days of 6.0 g·d⁻¹ of βA supplementation, the βA group had a delay in OBLA as determined by decreases in HR@OBLA and %MaxHR@OBLA. The findings of this study are consistent with previously discussed studies showing a delay in fatigue after βA supplementation [1-5,7,9,10]. A delay or rightward shift in OBLA during a high intensity exercise offers a significant advantage to an athlete trying to maintain repeated or prolonged high intensity muscle contractions. In addition to the HR findings, there was also an observed increase in %VO₂max@OBLA within the βA group. However, the authors feel this may be misleading as there was also a decrease in the VO₂max values post supplementation within the βA group. Therefore, the increase in %VO₂max@OBLA may simply be due to a decrease in the VO₂max value. This decrease in VO₂max was an unexpected finding as it is indicative of a reduced aerobic capacity and is not a typical training response.

**Implications of Study Results**

The present study is the first to our knowledge to examine the effects of βA on OBLA during incremental stages of running. After 28 days of 6.0 g·d⁻¹ of βA supplementation, the βA group had a delay in OBLA as determined by decreases in HR@OBLA and %MaxHR@OBLA. The findings of this study are consistent with previously discussed studies showing a delay in fatigue after βA supplementation [1-5,7,9,10]. A delay or rightward shift in OBLA during a high intensity exercise offers a significant advantage to an athlete trying to maintain repeated or prolonged high intensity muscle contractions. In addition to the HR findings, there was also an observed increase in %VO₂max@OBLA within the βA group. However, the authors feel this may be misleading as there was also a decrease in the VO₂max values post supplementation within the βA group. Therefore, the increase in %VO₂max@OBLA may simply be due to a decrease in the VO₂max value. This decrease in VO₂max was an unexpected finding as it is indicative of a reduced aerobic capacity and is not a typical training response.
tem. More specifically, OBLA was delayed based on higher HR@OBLA and %HRmax@OBLA in the group of individuals receiving the βA versus the PL. Future research is needed to confirm these results and to test for performance related outcomes specific to distance running.

Future Research
Future studies should focus on looking at the effects of βA on 10-20 km simulated endurance road race performance. With this, a close examination of VO2max should be considered. It would also be of interest to determine the ergogenic effects of βA on intermittent sports, such as soccer, hockey, basketball or football, which require a combination of endurance and sprint performance.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
TJ was the primary author of the manuscript and played an important role in the data collection and assessment. JL, MM and JU played an important role in data collection and manuscript preparation. All authors have read and approved the final manuscript.

Acknowledgements
The authors would like to thank Athletic Edge Nutrition 3109 Grand Avenue #280 Miami, Fl, http://www.aenutrition.com for donating the products and 3000.00 US dollars for lactate measurements. No other funding was received.

The authors would like to thank Dr. Paul Luebbers, of Emporia State University, for his editorial assistance. The mention of any dietary supplement ingredient in this paper does not constitute an endorsement by the authors.

Author Details
1School of Family, Consumer, and Nutrition Sciences. Northern Illinois University, DeKalb, IL, USA and 2Department of Kinesiology and Physical Education, Northern Illinois University, DeKalb, IL, USA

Received: 13 October 2009 Accepted: 19 May 2010
Published: 19 May 2010

References


doi: 10.1186/1550-2783-7-20
Cite this article as: Jordan et al., Effect of beta-alanine supplementation on the onset of blood lactate accumulation (OBLA) during treadmill running: Pre/post 2 treatment experimental design. Journal of the International Society of Sports Nutrition 2010, 7:20