Effects of Beta-Alanine Supplementation on Sprint Endurance

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ABSTRACT

Jagim, AR, Wright, GA, Brice, AG, and Doberstein, ST. Effects of beta-alanine supplementation on sprint endurance. J Strength Cond Res 27(2): 526–532, 2013—Recent research has shown that beta-alanine (BA) supplementation can increase intramuscular carnosine levels. Carnosine is an intramuscular buffer, and it has been linked to improvements in performance, specifically during bouts of high-intensity exercise that are likely limited by muscle acidosis. Therefore, the purpose of this study was to examine the effect of BA supplementation on sprint endurance at 2 different supramaximal intensities. Twenty-one anaerobically trained (rugby players [n = 4], wrestlers [n = 11], and recreationally strength trained athletes [n = 6]) college-aged men participated in a double-blind, placebo controlled study. The subjects performed an incremental VO2max test and 2 sprint to exhaustion tests set at 115 and 140% of their VO2max on a motorized treadmill before (PRE) and after (POST) a 5-week supplementation period. During this time, the subjects ingested either a BA supplement or placebo (PLA) with meals. The subjects ingested 4 g d⁻¹ of BA or PLA during the first week and 6 g d⁻¹ the following 4 weeks. Capillary blood samples were taken before and after each sprint to determine blood lactate response to the sprint exercise. No significant group (BA, PLA) × intensity (115%, 140%; p = 0.60), group by time (PRE, POST; p = 0.72), or group × intensity × time (p = 0.74) interactions were observed for time to exhaustion. In addition, similar nonsignificant observations were made for lactate response to the sprints (group × intensity, p = 0.43; group × time, p = 0.33, group × intensity × time, p = 0.56). From the results of this study, it was concluded that beta-alanine supplementation did not have a significant effect on sprint endurance at supramaximal intensities.

KEY WORDS anaerobic performance, lactate, buffer

INTRODUCTION

When a high-intensity exercise is maintained for >20 seconds, the muscles can become fatigued as a result of the energy systems not being able to keep up with the demands placed upon them. As a result, there is an accumulation of hydrogen ions (H⁺) within muscle cells, which leads to a decrease in pH levels. These H⁺ are byproducts of anaerobic metabolism that accumulate when the muscle’s aerobic metabolic system is unable to keep up with the rapid need to resynthesize adenosine triphosphate (ATP). The formation and accumulation of H⁺ in muscle with intense exercise has been shown to affect metabolic processes by slowing down the resynthesis of phosphocreatine, inhibiting the rate of glycolysis, and inhibiting the contractile process itself (6,19).

Intramuscular buffers aid in the ability to tolerate increased H⁺ and allow the glycolytic process to continue for a longer period of time. Carnosine (β-alanyl-L-histidine) is an example of one of these intramuscular buffers. Carnosine is found in skeletal muscle with the highest percentage found in fast-twitch muscle fibers. Carnosine is a cytoplasmic dipeptide synthesized from the precursors L-histidine and beta-alanine (BA) by the enzyme carnosine synthetase. Of these 2 precursors, BA has been proposed to be the rate limiting substrate for the production of carnosine in the muscle (9).

Researchers have proposed that increasing carnosine content in the muscle may increase its buffer capacity and, therefore, performance at intensities where muscle acidosis may be a limiting factor. Carnosine itself can be supplemented; however, when it is ingested, it is hydrolyzed within the stomach producing histidine and BA (8). Therefore, because BA is likely the rate limiting substrate, studies investigating BA supplementation have shown success in increasing intramuscular carnosine levels (1,5,9,12).

Higher intramuscular carnosine levels through BA supplementation may delay fatigue and increase performance. However, studies investigating the effects of BA supplementation on performance have shown mixed results. For example, studies using short duration (<60 seconds), high-intensity effort have been less successful in showing performance improvement (4,17,24) than longer duration (>90 seconds) performance tasks (18,21,27). The notion of using trained, particularly anaerobically trained, or untrained subjects has also led to contradicting results. Studies using
untrained subjects seem to show greater increases in carnosine levels and performance after BA supplementation (7). However, there is limited research regarding the effects of BA supplementation on different sprinting intensities to exhaustion with anaerobically trained subjects. By investigating 2 different intensities, it will help determine what type and duration of intense anaerobic exercise will benefit the most from BA. The purpose of this study is to determine the efficacy of BA supplementation on sprint endurance (sprints lasting >30 seconds) at 2 different intensities.

**METHODS**

**Experimental Approach to the Problem**

This study was completed over a 6-week period using a 2-group, matched, double-blind design that was placebo controlled. Before the supplementation, all the participants performed a maximal \( VO_2 \) test on a motorized treadmill. On different days, the subjects also went through 2 familiarization sessions to determine time to exhaustion (TTE) at a speed calculated to be at 140% \( VO_2 \max \). The subjects were divided into 2 groups: a BA or placebo (PLA) supplementation group matched to their TTE during their last familiarization trial. A PLA group was used rather than a crossover design because the washout time for BA has been determined to be between 6 and 15 weeks (1), which would make testing reliability problematic. On 2 separate occasions after familiarization, the subjects ran to volitional fatigue on a motorized treadmill at speeds calculated to require 115 and 140% \( VO_2 \max \) in a counterbalanced order separated by at least 48 hours. These experimental trials were performed before (PRE) and after (POST), the 5-week supplementation period.

**Subjects**

Twenty-one trained college-aged men from the University of Wisconsin-La Crosse volunteered to participate in this study (Table 1). The subjects included wrestlers \((n = 11)\), recreationally strength trained athletes \((n = 6)\), and rugby players \((n = 4)\). The wrestlers were in the late preseason stage and rugby players were in the off-season stage of their yearly training program. All the participants were able to run at 140% \( VO_2 \max \) for at least 30 seconds on a motorized treadmill. The research protocol was approved by the university’s Institutional Review Board before implementation. All the subjects were informed of risks and benefits of both exercise testing and ingestion of BA and PLA, and all the subjects gave informed consent in writing for both treatments.

**Supplementation**

After the familiarization trials and pretesting, the participants began a 5-week supplementation period. The participants received either the BA supplement (Intra X Cell, Athletic Edge Nutrition, Miami, FL, USA) or a placebo (rice flour; resembling the BA supplement in looks, taste, and texture). During the first week, the participants ingested 2 capsules, 3 times per day of the BA supplement or placebo with meals. These dosages of the supplement resulted in ingestion of 4 g of BA, 402 mg of proprietary blend of \( N \)-acetylcysteine/g-lipoic acid, and 15 mg of vitamin E per day. During the following 4 weeks, the participants ingested 3 capsules, 3 times a day, equaling 6 g of BA or placebo. The participants were instructed to abstain from any nutritional aids or supplements during the duration of the study and maintain their regular eating and exercise habits.

**Testing Procedures**

\( VO_2 \max \). One week before the familiarization trial, the participants performed a \( VO_2 \max \) test on a motorized treadmill. The \( VO_2 \max \) test consisted of a 3-minute warm-up at 93.8 m \( \cdot \) min\(^{-1}\) (3.5 mph) at 0% grade. After the warm-up, velocity was increased to 187.6 m \( \cdot \) min\(^{-1}\) (7.0 mph) for 3 minutes followed by another 13.4 m \( \cdot \) min\(^{-1}\) (5.0 mph) after another 3 minutes. If the subjects were able to continue, the grade was increased by 2% every 2 minutes until voluntary fatigue. Respiratory gas exchange data were measured using open-circuit spirometry (AEI, Pittsburgh, PA, USA). All the subjects reached voluntary fatigue within 3 stages at elevation.

**Experimental Trial**

Each experimental period (Presupplementation and Post-supplementation) consisted of 2 nonconsecutive testing days.

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**Table 1. Physical characteristics of subjects.**

<table>
<thead>
<tr>
<th></th>
<th>PLA ((n = 11))</th>
<th>BA ((n = 10))</th>
<th>All subjects ((n = 21))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (y)</strong></td>
<td>20 ± 2.45</td>
<td>20.5 ± 2.32</td>
<td>20 ± 2.3</td>
</tr>
<tr>
<td><strong>Body weight (kg)</strong></td>
<td>78.46 ± 9.37</td>
<td>77.64 ± 11.82</td>
<td>78.07 ± 10.21</td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
<td>177.10 ± 7.28</td>
<td>176.02 ± 6.78</td>
<td>176.91 ± 6.4</td>
</tr>
<tr>
<td><strong>( VO_2 \max ) (ml·kg(^{-1})·min(^{-1}))</strong></td>
<td>55.8 ± 4.43</td>
<td>56.22 ± 6.95</td>
<td>56.02 ± 5.51</td>
</tr>
</tbody>
</table>

*PLA = placebo; BA = beta-alanine.
†Values expressed as mean ± SD.
to allow for ample recovery time between trials. The subjects were instructed to eat similar diets the day before and of each testing session. Experimental trials consisted of a standardized warm-up of 5 minutes of jogging at 50% \( V_O^{2\max} \) followed by 3–5 minutes of passive rest. Intensity for the 2 experimental trials was extrapolated to be approximately 140% (short duration) and 115% (long duration) \( V_O^{2\max} \). These trials took place in our Human Performance laboratory and were conducted at similar temperatures (72°C to 76°C) and the same hour of the day for the short and long duration runs, presupplementation and post supplementation. The subjects were familiarized how to get on a moving treadmill at the predetermined speed by holding onto the side rails of the treadmill as their feet came in contact with the moving belt. The time of effort started when the subject released the hand rails (1–2 seconds after foot contact) and ended when they regrasped the hand rails at exhaustion. No feedback for time of performance was given on any trial. The dependent variable was time to exhaustion (TTE). When the subject reached exhaustion, the speed of the treadmill was quickly reduced to 67 m/min (2.5 mph) for 4.5 minutes to allow the subject to walk and recover.

**Blood Lactate**

After the 5-minute warm-up and 5 minutes after the high-intensity runs, a fingertip blood sample was taken. After puncturing the skin of the fingertip, the first drop of blood was wiped from the skin. The succeeding blood flow was collected in a heparinized capillary tube. Twenty-five microliters of blood was immediately removed from the capillary tube and mixed with 50 μl of NaF Triton buffer, which is used for red blood cell lysing and to prevent an increase in lactate after the whole blood sample was added to the buffer. The samples were stored in a refrigerator and analyzed within 48 hours for lactate (Yellow Springs Instruments 1500 Sport lactate analyzer, Yellow Springs, OH, USA). Samples were analyzed in duplicate with test-retest reliability (intraclass correlation coefficient [ICC]) of \( r = 0.996 \).

**Statistical Analyses**

The Statistical Package for the Social Sciences (Version 16, SPSS Inc. Chicago, IL, USA), was used to analyze all of the statistical material within the study. Separate 2-way (group: BA, PLA × time: Presupplementation, Postsupplementation) repeated measures analysis of variance (ANOVA) were used to evaluate the TTE for each test (115 and 140% \( V_O^{2\max} \)). All data are reported as mean ± SD. Differences were considered significant if \( p \leq 0.05 \).

**RESULTS**

**Group Results**

Table 2 illustrates the sprint TTE presupplementation and postsupplementation for the 2 different sprints at supramaximal intensities. There was no significant interaction for TTE seen by group (BA, PLA) \( \times \) intensity (115%, 140%; \( p = 0.60 \)), or by group \( \times \) time (presupplementation, postsupplementation; \( p = 0.72 \)), or group \( \times \) intensity \( \times \) time (\( p = 0.74 \)).

**Lactate**

Figure 1 illustrates the blood lactate values after each sprint to exhaustion analyzed Presupplementation and Postsupplementation. There was a significant main effect by time (\( p < 0.01 \)) where Presupplementation (10.4 ± 1.9 mmol) was found to be higher than Postsupplementation (8.4 ± 2.9 mmol). There were no significant interactions by group in the lactate response to the sprints (group \( \times \) intensity, \( p = 0.43 \); group \( \times \) time, \( p = 0.33 \); group \( \times \) intensity \( \times \) time, \( p = 0.56 \)).

**TABLE 2.** Presupplementation and postsupplementation performance times to exhaustion in supramaximal sprints at 115 and 140% \( V_O^{2\max} \).* †

<table>
<thead>
<tr>
<th></th>
<th>115% Pre (s)</th>
<th>115% Post (s)</th>
<th>140% Pre (s)</th>
<th>140% Post (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLA</td>
<td>154.1 ± 35.9</td>
<td>164.8 ± 39.8</td>
<td>69.3 ± 24.2</td>
<td>74.8 ± 27.5</td>
</tr>
<tr>
<td>BA</td>
<td>141.8 ± 51.7</td>
<td>151.5 ± 71.5</td>
<td>64.7 ± 16.5</td>
<td>66.4 ± 20.9</td>
</tr>
</tbody>
</table>

*BA = beta alanine; PLA = placebo.
†Values are expressed as mean ± SD.
The purpose of this study was to determine the effectiveness of BA supplementation on sprint endurance in anaerobically trained subjects. The results showed that 1 week of 4 g d\(^{-1}\) followed by 4 weeks of 6 g d\(^{-1}\) of BA added to the diet did not increase TTE in short (~1 minute) or long sprint (~2–3 minutes) endurance in trained, anaerobic athletes. These results are in contrast to those of many studies (10,17,20,22,27) that found improved performance after BA supplementation. However, there have been reports by others who also found no improvements in performance after BA supplementation with trained anaerobic athletes (4,11,13,25).

Carnosine is an intramuscular dipeptide that has been demonstrated to buffer the accumulation of H\(^+\) and help delay the onset of fatigue (5). Recent studies demonstrate that 4–6 g of BA per day with dosing periods typically lasting 4 weeks can significantly increase carnosine concentrations within the muscle (4,9). For example, Hill et al. (9) demonstrated a 58% increase in carnosine concentrations after 4 weeks of BA ingestion (4–6 g d\(^{-1}\)) in untrained individuals. They estimated that the buffer capacity, attributable to carnosine, increased from 9% before supplementation to 12% of the total buffering capacity in these subjects after supplementation. Hill et al. (9) reported a 7% increase in work done during a cycle ride to exhaustion at 110% \(\text{VO}_2\)\(_{\text{max}}\) and attributed it to the increased buffer capacity after BA supplementation. As a result of the success of this dosing protocol increasing muscle carnosine levels and leading to performance improvement in a task at a similar intensity to our study, we used a similar daily dosing protocol for 1 additional week. Although we did not measure carnosine in our study, we expected an increase in carnosine concentration to the same extent as seen in the Hill study (9). As such, the lack of changes in performance TTE while running at 115 and 140% of \(\text{VO}_2\)\(_{\text{max}}\) was surprising. We can only speculate that there was an increase in intramuscular carnosine levels, however, possibly not to an extent necessary to improve performance in the well-trained anaerobic athletes in this study.

To that end, several studies (14,23,26), have shown that athletes who perform significant amounts of high-intensity training have higher levels of carnosine within the muscle when compared to untrained individuals. Hoffman et al. (11) suggested that trained individuals may need a greater relative dose of \(\beta\)-alanine or a longer supplementation period than untrained individuals to increase muscle carnosine levels high enough to see a significant improvement in performance. Hoffman et al. suggested that the lack of an effect seen in their study may have been a result of the training status of their subjects. The authors stated that during the time frame of the study, the subjects were all participating in the same strength and conditioning program, which was designed to bring the athletes to peak

<table>
<thead>
<tr>
<th>Authors</th>
<th>Population</th>
<th>Supplementation period</th>
<th>Mode of exercise</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stout et al. (20)</td>
<td>51 Men</td>
<td>28 d</td>
<td>PWCT test on a cycle</td>
<td>14.5% Improvement in PWCT compared with placebo</td>
</tr>
<tr>
<td>Stout et al. (18)</td>
<td>22 Women</td>
<td>28 d</td>
<td>PWCT test on a cycle</td>
<td>Improvement in PWCT</td>
</tr>
<tr>
<td>Stout et al. (22)</td>
<td>26 Elderly men and women</td>
<td>90 d of supplementation (7.2 g d(^{-1}))</td>
<td>PWCT graded exercise test on cycle ergometer</td>
<td>28.5% Increase in PWCT after BA supplementation improved power output at the end of the sprint</td>
</tr>
<tr>
<td>Van Thienen et al. (27)</td>
<td>17 Young healthy men</td>
<td>8 wks (~3 g d(^{-1}))</td>
<td>PWCT graded exercise test on cycle ergometer</td>
<td>No improvements seen in TTE</td>
</tr>
<tr>
<td>Derave et al. (4)</td>
<td>15 Male highly trained sprinters</td>
<td>4 wks (4.8 g d(^{-1}))</td>
<td>PWCT graded exercise test on cycle ergometer</td>
<td>No improvements seen in TTE</td>
</tr>
<tr>
<td>Sweeney et al. (25)</td>
<td>19 Physically active college-aged men</td>
<td>5 wks of supplementation (6 g d(^{-1}))</td>
<td>PWCT graded exercise test on cycle ergometer</td>
<td>No improvements seen in TTE</td>
</tr>
</tbody>
</table>

* TTE = time to exhaustion; PWCT, physical working capacity at the fatigue threshold.
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anaerobic condition. There have been several other studies that have looked at BA supplementation and have reported similar findings when using trained athletes (4,11,13,25).

In this study, the majority of the subjects were Division III wrestlers and rugby players; both sports that rely heavily on the anaerobic energy system. The athletes were involved in their respective preseason training programs that were designed to improve overall strength, power, and muscular endurance. Because of the anaerobic training, we speculate that our subjects had higher carnosine levels at the beginning of the study than subjects in studies using nonanaerobically trained subjects. Similar TTE pre and post BA supplementation supports the notion that anaerobically trained athletes may require a higher dose or longer supplementation period to produce significant changes in the already elevated carnosine that allows for a significant increase in buffer capacity in the muscle. The study by Hill et al. (9) had their “recreationally active” subjects consume between 50.1 and 80.1 mg of BA per kilogram of body weight. Our subjects used similar dosages, consuming 51.8–77.3 mg of BA per kilogram, and our supplementation period lasted one week longer than the 4 weeks used in previous studies (4,9). It is possible that one additional week of supplementation in our study was not long enough or the dosages were not high enough, to significantly augment carnosine levels compared to 4 weeks of supplementation because of the fact that our subjects were anaerobically trained.

Two different running intensities to exhaustion with the intent to produce different challenges to pH homeostasis of the muscle were used in this study. Through previous studies (9) and pilot work on lesser trained individuals, we expected TTE at 115% \( \text{V} \text{O}_{2} \text{max} \) to be between 120 and 140 seconds. On average, our subjects reached exhaustion between 141 and 154 seconds at 115% of their \( \text{V} \text{O}_{2} \text{max} \) before supplementation, which exceeded our expectations. This suggests that our subjects may have already had an enhanced buffering capacity through training alone; reiterating the fact that a higher or longer dosing strategy may have been needed.

In this study, we did not measure blood or muscle pH directly. However, high-intensity sprinting typically relies heavily on the activation of high-threshold motor units in order to increase force production. The fast-twitch fibers that make up the high-threshold motor units are characterized by greater intramuscular acidosis during their activation than slow-twitch fibers. An increased reliance on fast glycolysis, which is the primary source of energy for high-threshold motor units, will likely lead to a greater concentration of H+ ions and drop in pH. The moderately high levels of lactate seen in our study suggest an increased rate of fast glycolysis, resulting in a decrease in pH. However, without knowing the exact relative pH changes it is difficult to speculate whether or not increased carnosine levels were contributing to an enhanced buffering capacity. It is possible that the intensity used may not have been high enough to cause a significant drop in pH levels and allow for an improvement in buffering capacity to be seen. In this study, after the sprint at an intensity of 115% \( \text{V} \text{O}_{2} \text{max} \), lactate levels were only in the range of 7–9 mmol-L\(^{-1}\) (140–165 seconds in duration). In a similar study performed by Kern and Robinson (13), they found no improvements in 300-yd shuttle times after 8 weeks of BA supplementation and high-intensity training. Similar to this study, college wrestlers who were anaerobically trained, participated in the study and had similar lactate values of approximately 7–11 mmol-L\(^{-1}\) after the 300-yd shuttle. In a study done by Sweeney et al. (25), college wrestlers performed repeat high-intensity sprints, against 15% of their body weight, before and after BA supplementation and similarly found no improvements in performance. In this study lactate values averaged between 10 and 13 mmol-L\(^{-1}\) after the sprints. Even in the study done by Derave et al. (3), the subjects experienced lactate levels in the range of 15–16 mmol-L\(^{-1}\) after a 400-m run (~52 seconds in duration) and still did not see an improvement in performance. Therefore, it may just be that these intensities (400-m run, 300-yd shuttle, and high-intensity resisted sprints) and the 115% of \( \text{V} \text{O}_{2} \text{max} \) used in our study, did not stress the anaerobic system enough to lower the pH, requiring an enhanced buffering capacity.

To further study the efficacy of BA supplementation with high-intensity exercise, we had the subjects exercise at a higher intensity, one that produced exhaustion in approximately 1 minute. Doing so would allow the demonstration of the efficacy of BA as an ergogenic aid at different levels of muscle acidosis and applied to different competitive events. Exercising to exhaustion within 1 minute requires approximately 70% of the ATP provision to be supplied by the anaerobic energy system (15) and would lead to muscle pH near 6.7 (2). Before supplementation, our subjects were able to run on the treadmill at 140% intensity for 64–69 seconds before volitional fatigue set in. We expected the time frames of the TTE trials at 2 separate intensities in our study would likely stress the anaerobic systems and pH homeostasis differently. However, we found the contrary to be true as lactate levels were not different between the 2 intensities.

Surprisingly, we observed that, at both 140 and 115% \( \text{V} \text{O}_{2} \text{max} \) intensities, the postsupplementation lactate values were significantly less than presupplementation lactate values for the BA and the placebo groups. It was expected that the BA group would notice an increase in lactate accumulation in the blood as a result of improved buffering of H+ and less inhibition of rate limiting enzymes of fast glycolysis and the placebo group would have similar responses to presupplementation levels following supplementation. Although the subjects were instructed to maintain a similar diet and especially carbohydrate intake from pre to postsupplementation, it is possible that because the major population of our subjects were intercollegiate wrestlers in training (11 out of 20), these subjects may have been restricting carbohydrate or
energy intake to some degree to maintain low body weights for the upcoming season more during the postsupplementation than the presupplementation testing. It should be noted that testing was scheduled to be >3 weeks away from any need to cut weight and no drastic weight loss techniques were used during the supplementation period. It is also likely that the increase in intensity of the training program as the season approached would increase carbohydrate use and decrease storage in the muscles. Foster et al. (6) showed that the rate of increase in blood lactate is slower at similar power production in athletes in training when substrate availability is compromised. Therefore, although the significant decrease in lactate is surprising, it is likely explained by lower glycogen availability for the subjects in both groups, and therefore, likely explains why both groups decreased lactate similarly in the postsupplementation trial. A closer look at the individual lactate data (not shown) revealed that every wrestler (in both groups) produced less lactate in the posttesting than the pretesting, whereas most of the rugby players and recreational lifters produced similar or slightly higher lactate levels at both intensities.

Furthermore, the lack of an effect may be attributable to the mode of exercise selected. All of the above-mentioned studies (4,11,13,25) used some form of high-intensity running as the measure of performance. All these studies failed to show an improvement in performance after BA supplementation. Conversely, several studies have shown improvements in measures of performance when other modes of exercise have been used; cycling being the most common (16,20,22,27). Table 3 summarizes some of the recent literature on BA supplementation in which different modes of exercise were used. From the literature, it appears that studies using high-intensity running as a measure of performance failed to show improvements. Conversely, studies using high-intensity cycling seemed to show improvements in performance after BA supplementation. It may be possible that cycling uses a larger muscle mass compared to running thus placing a greater strain on the anaerobic energy system and allowing for improvements in buffering capacity to enhance performance.

**Practical Applications**

Based on the results of this study, anaerobically trained athletes that participate in sports requiring extended bouts of high-intensity exercise (i.e., long duration sprinters, rowers, speed skating, etc.) may not benefit from BA supplementation or they may need higher dosages to see the results typical of nonanaerobic trained athletes. Because anaerobically trained athletes already have elevated levels of carnosine in the muscle, the dosages used in previous studies may not be high enough to stimulate the synthesis of carnosine. It is possible that BA supplementation may have similar results to that of creatine supplementation, whereas running performances really do not seem to benefit from supplementation.

**Acknowledgments**

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