

UNIVERSITY OF WISCONSIN-LA CROSSE

Graduate Studies

EFFECTS OF BETA-ALANINE SUPPLEMENTATION ON SPRINT ENDURANCE

A Manuscript Style Thesis Submitted in Partial Fulfillment of the Requirements for the  
Degree of Master of Science in Human Performance

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Exercise & Sport Science

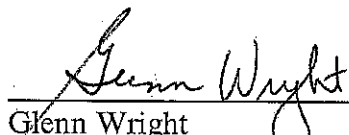
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EFFECTS OF BETA-ALANINE SUPPLEMENTATION ON SPRINT ENDURANCE

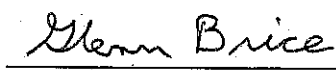
By Andrew Jagim

We recommend acceptance of this thesis in partial fulfillment of the candidate's requirements for the degree of a Master's in Human Performance – Applied Sport Science

The candidate has completed the oral defense of the thesis.

  
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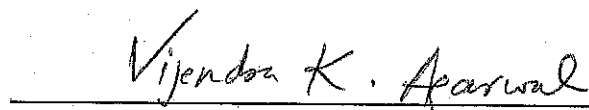
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## ABSTRACT

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The purpose of this study was to examine the effects of beta-alanine supplementation (BA) on sprint endurance at two different intensities. Twenty-one anaerobically trained [rugby players (n=4), wrestlers (n=11) and recreational athletes (n=6)] college men participated in a double blind, placebo controlled study. Subjects performed an incremental  $\text{VO}_2$  max test and two sprint to exhaustion tests set at 115% and 140% of their  $\text{VO}_2$  max on a motorized treadmill before (PRE) and after (POST) a 5 week supplementation period. During this time subjects ingested either a BA supplement or placebo (PLA). Subjects ingested 4g/day of BA or PLA during the first week and 6g/day the following 4 weeks. Capillary blood samples were taken before and after each sprint to determine blood lactate response. Following the supplementation period, no significant group (BA, PLA) x intensity (115%, 140%;  $p=0.60$ ), group by time (PRE, POST;  $p=0.72$ ), or group x intensity x time ( $p=0.74$ ) interactions were observed for time to exhaustion. Similar non-significant observations were made for lactate response to the sprints (group x intensity,  $p=0.43$ ; group x time,  $p=0.33$ , group x intensity x time,  $p=0.56$ ). It was concluded that beta-alanine supplementation did not have a significant effect on sprint endurance at supramaximal intensities.

## ACKNOWLEDGEMENTS

I would like to acknowledge the many individuals who made the completion of this project possible.

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A special thanks goes to Athletic Edge Nutrition for providing the beta-alanine supplements used throughout this study. I would like to thank the University of Wisconsin – La Crosse Graduate Research for providing funding for this project. I would also like to thank Chris Dodge and the Human Performance Lab for all their patience and assistance with the project.

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## INTRODUCTION

When a high intensity exercise is maintained longer than 20 s, the muscles can become fatigued as a result of the energy systems not being able to keep up with the demands placed upon it. 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 the resynthesis of phosphocreatine, inhibiting the rate of glycolysis, and inhibition of the contractile process itself (16).

Intramuscular buffers aid in the ability to tolerate increased  $H^+$  and allow the glycolytic process to continue for a longer period of time. Carnosine (B-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 by the enzyme carnosine synthetase. Of these two precursors, beta-alanine has been proposed to be the rate limiting substrate for the production of carnosine in the muscle (6).

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 cannot be supplemented because when it is ingested, it is hydrolyzed within the stomach to produce histadine and beta-alanine (5). Therefore, since beta alanine is likely the rate limiting substrate, studies investigating beta alanine supplementation have been done and have shown success in increasing intramuscular carnosine levels (1, 3, 6, 8). Beta-alanine is a nonessential amino acid that is found in many foods.

Higher intramuscular carnosine levels through beta-alanine supplementation may delay fatigue and increase performance. However, studies investigating the effects of beta-alanine supplementation on performance have shown mixed results. For example, studies utilizing short duration (< 60 sec), high intensity effort have been less successful in showing performance improvement (3, 13, 17) than longer duration (> 90 sec) performance durations (14, 15, 19). However, there is limited research regarding the effects of beta-alanine supplementation on different sprinting intensities to exhaustion. By investigating two different intensities, it will help determine what type/duration of intense anaerobic exercise will benefit the most from beta-alanine. The purpose of the current study is to determine the efficacy of beta-alanine supplementation on sprint endurance (sprints lasting >30 s) at two 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. Prior to the supplementation, all participants performed a maximal VO<sub>2</sub> test on a motorized treadmill. On different days subjects also

went through two familiarization sessions to determine time to exhaustion at a speed calculated to be at 140%  $\text{VO}_2$  max. Subjects were divided into two groups: a beta-alanine (BA) or placebo (PLA) supplementation group matched to their time to exhaustion (TTE) during their last familiarization trial. A PLA group was utilized rather than a crossover design because the washout time for BA is still being investigated. On two separate occasions following familiarization, subjects ran to volitional fatigue on a motorized treadmill at speeds of 115% and 140%  $\text{VO}_2$  max in a counter balanced 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 men from the University of Wisconsin-La Crosse volunteered to participate in this study (Table 1). Subjects included rugby players (n=4), wrestlers (n=11) and recreationally strength trained athletes (n=6). The wrestlers were in the late preseason stage and rugby players were in the off-season stage of their yearly training program. All participants were able to run at 140%  $\text{VO}_2$  max for at least 30 sec on a motorized treadmill. The research protocol was approved by the Institutional Review Board at the University of Wisconsin – La Crosse prior to implementation. Subjects were informed of the experimental procedures of the study and potential risks and side effects associated with supplementation and gave their verbal and signed informed consent prior to participation in the study.

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

	PLA (n=11)	BA (n=10)	All Subjects (n=21)
Age (yrs)	20 ± 2.45	20.5 ± 2.32	20 ± 2.3
Body Weight (kg)	78.46 ± 9.37	77.64 ± 11.82	78.07 ± 10.21
Height (cm)	177.10 ± 7.28	176.02 ± 6.78	176.91 ± 6.4
VO2 max (ml/kg/min)	55.8 ± 4.43	56.22 ± 6.95	56.02 ± 5.51

Values expressed as mean ± SD.

### **Supplementation**

After the familiarization trials and pretesting, participants began a five week supplementation period. Participants received either the BA supplement (Intra X Cell, Athletic Edge Nutrition, Miami FL) or a placebo (rice flour; resembling the BA supplement in looks, taste, and texture). During the first week 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 beta-alanine, 402 mg proprietary blend of N-acetylcysteine/g-lipoic acid, and 15mg vitamin E per day. During the following four weeks, participants ingested 3 capsules, 3 times a day, equaling 6 g of BA or placebo. Participants were asked 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<sub>2</sub> max.* One week prior to the familiarization trial, participants performed a VO<sub>2</sub> max test on a motorized treadmill. The VO<sub>2</sub> max test consisted of a 3-min warm-up at 93.8 m/min (3.5 mph) at 0% grade. Following the warm-up, velocity was increased to 187.6 m/min (7.0 mph) for 3 min followed by another 13.4 m/min (0.5 mph) after another 3 min. If the subjects were able to continue, grade was increased by 2% every 2 min until voluntary fatigue. Respiratory gas exchange data were measured using open-circuit spirometry (AEI, Pittsburgh, PA). All subjects reached voluntary fatigue within three stages at elevation.

*Experimental Trial.* Each experimental period consisted of two non-consecutive testing days to allow for ample recovery time between trials. Experimental trials consisted of a standardized warm up consisting of 5 min of jogging at 50% VO<sub>2</sub> max followed by 3-5 min of passive rest. Intensity for the two experimental trials was extrapolated to be approximately 140% (short duration) and 115% (long duration) VO<sub>2</sub> max. These trials took place at approximately the same hour of the day for the short and long duration run for pre- and post supplementation testing. 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 re-grasped 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.* Following the 5 min warm-up and 5 min 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  $\mu$ l of blood was immediately removed from the capillary tube and mixed with 50  $\mu$ l of NaF Triton buffer, which is used for red blood cell lyses and to prevent an increase in lactate after the whole blood sample was added to the buffer. Samples were stored in a refrigerator and analyzed within 48 hours for lactate (Yellow Springs Instruments 1500 Sport lactate analyzer, Yellow Springs, OH). Samples were analyzed in duplicate with test-retest reliability (intraclass correlation coefficient-ICC) of  $r=.996$ .

### **Statistical Analysis**

The Statistical Package for the Social Sciences (SPSS Inc. Chicago, IL), was used to analyze all of the statistical material within the study. Separate 2-way (group: BA, PLA x time: Pre, Post Supplementation) repeated measures analysis of variance (ANOVA) were used to evaluate the TTE for each test (115% and 140% VO<sub>2</sub> max). All data is reported as means  $\pm$ SD. Differences were considered significant if  $p < 0.05$ .

### **RESULTS**

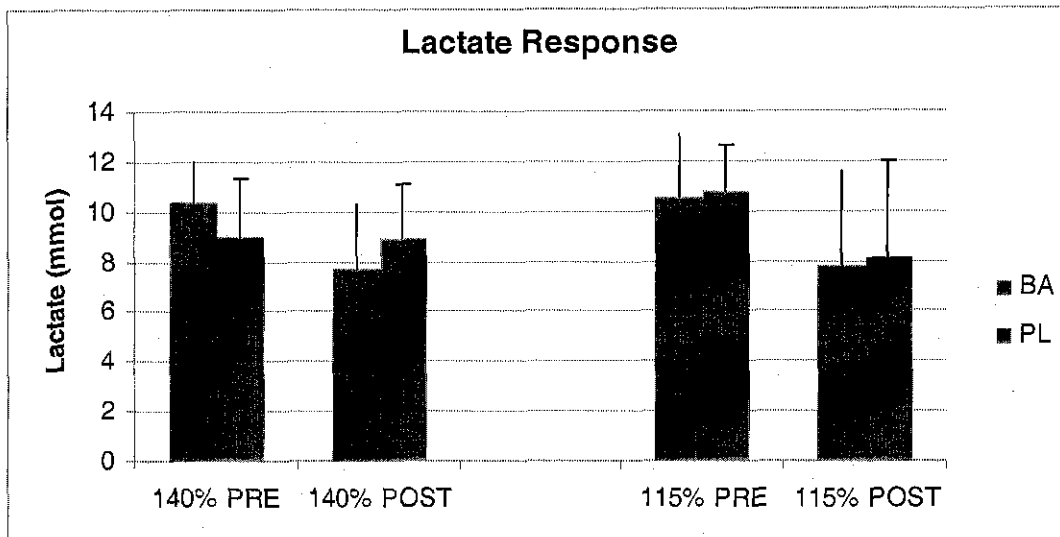
*Group Results.* Table 2 illustrates the sprint TTE pre and post-supplementation for the two different sprints at supramaximal intensities. There was no significant interaction for TTE seen by group (BA, PLA) x intensity (115%, 140%;  $p = 0.60$ ), or by group x time (PRE, POST Supplementation;  $p = 0.72$ ), or group x intensity x time ( $p = 0.74$ ).

**Table 2.** Pre and post supplementation performance times to exhaustion in supramaximal sprints at 115% and 140% VO<sub>2</sub> max. BA=Beta alanine; PLA= Placebo

	115%		140%	
	Pre (sec)	Post (sec)	Pre (sec)	Post (sec)
<b>PLA</b>	<b>154.1±35.9</b>	<b>164.8±39.8</b>	<b>69.3±24.2</b>	<b>74.78±27.5</b>
<b>BA</b>	<b>141.8 ± 51.7</b>	<b>151.5 ± 71.5</b>	<b>64.7 ± 16.5</b>	<b>66.4 ± 20.9</b>

Values are expressed as mean ± SD.

*Lactate.* Figure 1 illustrates the blood lactate values following each sprint to exhaustion analyzed pre and post-supplementation. There were no significant differences in the lactate response to the sprints (group x intensity, p = 0.43; group x time, p = 0.33, group x intensity x time, p=0.56).



**Figure 1.** Pre and post supplementation blood lactate responses following supramaximal sprints at 115% and 140%  $\text{VO}_2$  max. BA = Beta alanine; PLA = Placebo

## DISCUSSION

The purpose of this study was to determine whether sprint endurance could be improved with beta-alanine supplementation. Results showed that beta-alanine supplementation did not improve time to exhaustion in short (~1 minute) or long sprint (~2-3 min) endurance in trained, anaerobic athletes. These results are similar to those repeated by Derave et al. (3), Smith et al. (13), and Sweeney et al. (17), who also found no improvements in performance following beta-alanine supplementation.

Carnosine is an intramuscular buffer that has been demonstrated to buffer the accumulation of  $\text{H}^+$  and help delay the onset of fatigue (4). Studies have demonstrated that 4-6g of beta-alanine per day with dosing periods typically lasting from 4-6 weeks can increase carnosine concentrations within the muscle (3, 6). Derave et al. (3) showed that carnosine levels increased with beta-alanine supplementation after 4 weeks of 4.8g of

beta-alanine per day. Hill et al. (6) demonstrated a 58% increase in carnosine concentrations after 4 weeks of beta-alanine ingestion (4-6 g/day). Carnosine levels continued to increase after another 6 weeks of supplementation; however, they only increased by an additional 22%. This smaller increase suggests that there may be a limit as to how much carnosine can be stored within muscle. Because of these previous findings, similar dosing protocols were used in the current study, assuming that carnosine levels would respond similarly. The resulting increase in carnosine concentration within the muscle should have enhanced the buffering capacity of muscle cells leading to improved exercise performance in brief, high intensity bouts.

An increase in carnosine levels and a higher buffering capacity within the muscles would likely improve performance during high intensity exercise when the primary energy system utilized is fast glycolysis. An increase in the rate of fast glycolysis means a greater production of  $H^+$  in the muscle resulting in a drop in muscle pH. Previous research (9, 11), supports the idea that a drop in pH can result in muscular fatigue due to inhibition of rate-limiting enzymes involved with fast glycolysis (phosphofructokinase and phosphorylase), decreased release of  $Ca^{++}$  from the sarcoplasmic reticulum, and a decrease in cross-bridge interactions within the muscle. In the present study, pH levels were not measured; however, blood lactate levels were. Lactate is a byproduct of fast glycolysis and is often used to measure the rate at which it is occurring. Results from the current study showed moderately high blood lactate levels indicating there was a heavy reliance on the fast glycolytic system which we can assume resulted in a drop in pH within the muscles. According to Carins (Sports Med 36: 279-291, 2006), maximal efforts lasting 30 sec to > 90 sec could expect intramuscular pH in the range of 6.5-6.7.

By observing the lactate values, it can also be concluded that beta-alanine may not be successful in improving the buffering capacity of the muscles. If this would have been the case, glycolysis would have been able to proceed longer before the lowered pH within the muscle cells inhibited the rate limiting enzymes. This would have resulted in higher lactate values in the post-supplementation sprints. However, at both intensities the lactate values were similar or less than pre-supplementation lactate values following each sprint.

The assumed increase in muscle carnosine levels from beta-alanine supplementation should have improved the buffering capacity of the muscle, which in turn would improve performance during high intensity exercise. However, in the current study there were no improvements in performance seen from beta-alanine supplementation. One reason for a lack of improvement in performance from beta-alanine supplementation could be that carnosine is not a significant contributor to the total buffering capacity of the muscle during high intensity exercise. Hultman et al. (7) suggested that carnosine may contribute less than 10% to the total buffering capacity of human muscle and Mannion et al. (10) suggested it could be as low as 7%. On the other hand, Davey et al. (2) suggested that carnosine may contribute as much as 40% to total muscle buffering capacity when the physiological pH is between 6.5 and 7.5. Accordingly even a major change in carnosine concentrations wouldn't necessarily lead to an improvement in performance because it is not exactly known how much it contributes to muscle buffering capacity.

There may also be an upper limit to the amount of carnosine that can be stored within the muscle. This effect appears to be most prominent within Type II muscle fibers due to the high correlation between carnosine concentrations and the percentage of Type

II fibers within the muscle (12). Previous investigators (1, 3) have suggested that Type I fibers may be able to increase carnosine levels more than Type II fibers. Derave et al. (3) examined the effects of beta-alanine supplementation on anaerobically trained sprinters. Following 4 weeks of supplementation, Type I fibers increased carnosine levels by 47% compared to 37% in the Type II fibers in the beta-alanine group. Similar results were found in untrained subjects as well. Baguet et al. (1) supplemented untrained males with beta-alanine and found a 39% increase in carnosine concentration in Type I fibers compared to only a 23% increase in Type II fibers. These results suggest that Type I fibers may be more likely to benefit from beta-alanine supplementation and have a greater potential for increasing carnosine concentrations. This is likely due to the fact that these fiber types do not produce the anaerobic conditions that stimulate increases in carnosine seen during high intensity anaerobic training in Type II fibers. If trained subjects ingest beta-alanine to increase carnosine levels, there may be a limit to how much of an additional increase in carnosine is possible. It may be that a "ceiling" effect exists, especially within the Type II muscles of anaerobically trained individuals and this population may not benefit from the supplementation. Tallon et al. (18) found a twofold increase in carnosine concentration in trained bodybuilders when compared to untrained subjects, which may support the possibility that high intensity training can lead to such elevated carnosine levels that it becomes more difficult to synthesize further amounts. In most of the studies that have shown improvements from beta-alanine, the subjects were in an untrained state or recreationally active (1, 14, 15, 19). Conversely, the studies that have not shown improvements (3, 13, 17) including the present one, utilized anaerobically trained athletes as subjects. In the present study, anaerobically trained

athletes supplemented with beta-alanine for 5 weeks and showed no improvements in sprint performance following the supplementation period. It is possible that because of the nature of their anaerobic training background, they already had high carnosine levels within the muscle and as a result, either the dosage or the length of the supplementation period was not sufficient to increase carnosine levels further. If this were the case, they would not benefit from beta-alanine supplementation and would show no improvements in performance. Further research studying the ability for anaerobically trained muscle to increase carnosine levels by supplementation of beta alanine may be necessary.

### **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 beta-alanine supplementation. This is likely due to a ceiling effect caused by high initial levels of carnosine because of their training status.

## REFERENCES

1. Baguet, A, Reyngoudt, H., Pottier, A., Everaert, I., Callens, S., Achten, E., Derave, W. Carnosine loading and washout in human skeletal muscles. *Journal of Applied Physiology* 106: 837-842, 2009.
2. Davey, C. The significance of carnosine and anserine in striated skeletal muscle. *Archives of Biochem Biophys* 89: 303-308, 1960.
3. Derave, W, Ozdemir, M, Harris, R, Pottier, A, Reyngoudt, H, Koppo, K, Wise, J, and Achten, E. Beta alanine supplementation augments muscle carnosine content and attenuates fatigue during repeated isokinetic contraction bouts in trained sprinters. *J Appl Physiol* 103: 1736-1743, 2007.
4. Fitts, R. Cellular mechanisms of muscle fatigue. *Physiol Rev* 74: 49-94, 1994.
5. Harris, R, Tallon, M, Dunnett, M, Boobis, L, Coakley, J, Kim, H, Fallowfield, J, Hill, C, Sale, C, and Wise, J. The absorption of orally supplied beta-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. *Amino Acids* 30: 279-289, 2006.
6. Hill, C, Harris, R, Kim, H, Harris, B, Boobis, L, Sale, C, and Wise, J. Influence of beta-alanine supplementation on skeletal muscle carnosine concentration and high intensity cycling capacity. *Amino Acids* 32: 225-233, 2007.
7. Hultman, E, Sahlin, K. Acid-base balance during exercise. *Exercise & Sport Science Review* 8: 41-128, 1980.
8. Kendrick, I, Kim, J., Harris, R., Kim, C., Dang, V., Lam, T., Bui, T., Wise, J. The effect of 4 weeks b-alanine supplementation and isokinetic training on carnosine concentrations in type I and II human skeletal muscle fibres. *European Journal of Applied Physiology*. 106: 131-138, 2009.
9. Linderman, J, and Fahey T. Sodium bicarboate ingestion and exercise performance: An update. *Sports Med* 11: 71-77, 1991.
10. Mannion, A, Jakeman, P, Dunnett, M, Harris, R, and William, P. Carnosine and anserine concentrations in the quadriceps femoris muscle of healthy humans. *Eur J Appl Occup Physiol* 64: 47-50, 1992.
11. Roth, D, and Brooks, G. Lactate and pyruvate transport is dominated by a pH gradient-sensitive carrier in rat skeletal muscle sarcolemmal vesicles. *Arch Biochem Biophys* 279: 386-394, 1990.
12. Saltin, B, Gollnick, PD. Skeletal muscle adaptability: significance for metabolism and performance. In: Handbook of physiology. Baltimore: Williams & Wilkins, 1983.

13. Smith, A, Walter, A., Graef, J., Kendall, K., Moon, J., Lockwood, C., Fukuda, D., Beck, T., Cramer, J., Stout, J. Effects of B-alanine supplementation and high-intensity interval training on endurance performance and body composition in men: a double-blind trial. *Journal of the International Society of Sports Nutrition* 6: 1-9, 2009.
14. Sout, J, Cramer, JT., Zoeller, RF., Torok, D., Costa, P., Hoffman JR., Harris, RC., O'Kroy, J. Effects of beta-alanine supplementation on the onset of neuromuscular fatigue and ventilatory threshold in women. *Amino Acids* 32: 381-386, 2007.
15. Sout, J, Graves, SB., Smith, AE., Hartman, MJ., Cramer, JT., Beck, TW., Harris, RC. The effect of beta-alanine supplementation on neuromuscular fatigue in elderly (55-92 Years): a double-blind randomized study. *Journal of the International Society of Sports Nutrition* 5: 1-6, 2008.
16. Spriet, L, Lindinger, MI., McKelvie, RS., Heigenhauser, G., Jones, N. Muscle glycogenolysis and H<sup>+</sup> concentration during maximal intermittent cycling. *Journal of Applied Physiology* 66: 8-13, 1989.
17. Sweeney, K, Wright, GA., Brice, AG., Doberstein, ST. The effect of beta-alanine supplementation on power performance during repeated sprint activity. *Journal of Strength and Conditioning* 2009.
18. Tallon, M, Harris, R, Boobis, L, Fallowfield, J, and Wise, J. The carnosine content of vastus lateralis is elevated in resistance trained bodybuilders. *J Strength Cond Res* 19: 725-729, 2005.
19. Van Thienen, R, Van Proeyen, K., Vanden Eynde, B., Puype, J., Lefer, T., Hespel, P. B-alanine improves sprint performance in endurance cycling. *Medicine & Science in Sports & Exercise* 41: 898-903, 2009.

APPENDIX A  
INFORMED CONSENT

## Informed Consent

**Protocol Title:** The effects of beta-alanine supplementation on power outputs during long and short duration sprints.

**Principal Investigator:** Andrew Jagim, BS, CSCS  
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(215) 555-1212

**Emergency Contact:** Andrew Jagim, BS, CSCS  
(701) 730-4842

### ***Purpose and Procedure***

- The purpose of this study is to determine the effect of beta-alanine supplementation on power output during repeat and long duration sprints.
- My participation will involve a VO<sub>2</sub> max test, repeat sprints, and a long duration sprint (90 sec) on a cycle ergometer before and after a 5 week supplementation period. All tests will consist of a warm up of light cycling (60% of VO<sub>2</sub> Max) for 5 minutes followed by either repeat sprints or a long-duration sprint.
- The total time requirement is eight hours over a four-week period with the majority of the time being spent doing pre and post testing.
- I will ingest 4-6g/day of beta-alanine per day for 5 weeks, for a total of 9,800mg of beta-alanine.
- During the VO<sub>2</sub> max test, I will wear a snorkel-like device to analyze the gases I am breathing and a heart monitor, strapped around my chest, to monitor my heart rate and I will be asked to pedal until I reach a state of fatigue in which I can no longer continue. Testing will take place in room 225 Mitchell Hall, UW-L
- Blood will be taken from my fingertip before and after each sprint during testing to measure blood lactate.

### ***Potential Risks***

- I may experience finger and muscle soreness and substantial fatigue. Some individuals experience a slight tingling in my face due to the beta-alanine and I understand this may happen to me. The tingling sensation is short lasting and has been the only side-effect recorded.

- Individuals trained in CPR, Advanced Cardiac Life Support and First Aid will be in the laboratory, and the test will be terminated if complications occur.
- The risk of serious or life-threatening complications, for healthy individuals, like myself, is near zero.

***Rights & Confidentiality***

- My participation is voluntary.
- I can withdraw from the study at any time for any reason without penalty.
- The results of this study may be published in scientific literature or presented at professional meetings using grouped data only.
- All information will kept confidential through the use of number codes. My data will not be linked with personally identifiable information.

***Possible benefits***

- I and other athletes may benefit by understanding how buffers can help with the improvement in performance and may actually experience in improvement due to the beta-alanine supplementation.

Questions regarding study procedures may be directed to Andrew (701) 701-730-4842, the principal investigator, or the study advisor Dr. Glenn Wright, Department of Exercise and Sport Science, UW-L (608-785-4321). Questions regarding the protection of human subjects may be addressed to the UW-La Crosse Institutional Review Board for the Protection of Human Subjects, (608-785-8124 or [irb@uwlax.edu](mailto:irb@uwlax.edu)).

Participant \_\_\_\_\_ Date\_\_\_\_\_

Researcher \_\_\_\_\_ Date\_\_\_\_\_

APPENDIX B  
REVIEW OF LITERATURE

## REVIEW OF LITERATURE

### Introduction

When a high level of intense exercise is maintained longer than 20 s, the muscles can become fatigued as a result of the body's energy systems being unable to keep up with the demands placed on it. As high intensity exercise continues, hydrogen ions ( $H^+$ ) accumulate within the muscle which ultimately decreases the intramuscular pH. 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 slows the resynthesis of phosphocreatine, inhibits glycolysis, and inhibits the contractile process itself (31). Buffers aid in the ability to tolerate increased  $H^+$  production and allow the glycolytic process to continue longer. Carnosine is an example of one of these intramuscular buffers. Carnosine ( $\beta$ -alanyl-L-histidine) is a cytoplasmic dipeptide synthesized from the precursors L-histidine and beta-alanine by the enzyme carnosine synthetase. Beta-alanine is the rate limiting substrate for the production of carnosine (15). Carnosine is found in skeletal muscle, with the highest percentage found in fast-twitch muscle fibers (5). Carnosine adds to the buffering capacity of skeletal muscle to prevent the accumulation of acid and maintain pH at a more homeostatic level.

Previous studies have shown that beta-alanine supplementation can increase the concentration of beta-alanine within the muscle, allowing for an increase in the

production of intramuscular carnosine (7, 15). Since carnosine is responsible for aiding in the buffering of acid within the muscle, it is proposed that an increase in carnosine would delay fatigue and increase performance in events that rely on the anaerobic energy systems. The purpose of the current review is to examine how beta-alanine supplementation can improve the buffering capacity of muscles and, ultimately, performance during high intensity exercise.

### **Metabolism During High Intensity Exercise**

Energy for all human activity is derived from the hydrolysis of ATP to adenosine diphosphate (ADP) and inorganic phosphate ( $P_i$ ). ATP must be continually synthesized and resynthesized in order to keep up with the demands of exercise. There are three energy yielding pathways used during this process. These three energy yielding pathways are divided into an anaerobic and an aerobic component. Within the anaerobic system, there is an alactic (uses primarily phosphocreatine and stored ATP as energy source) and a lactic component which produces lactic acid through fast glycolysis. High intensity activities that rely on the alactic system do not produce significant amounts of lactic acid because activities that utilize this system as a primary means of energy production do not last long enough to stimulate abundant use of the lactic energy system. The alactic, lactic and aerobic pathways all contribute to ATP synthesis at different rates relative to the intensity and duration of exercise.

**Alactic Energy System.** The alactic metabolic system represents one of the most immediate sources for ATP resynthesis at the onset of muscular contraction. There is a minimal amount of stored ATP immediately available at the onset of muscular contraction but this can usually only provide energy up to 2 s (10). Therefore, the alactic

system is needed almost immediately at the onset of muscular contraction and is the energy yielding pathway that is able to resynthesize ATP the fastest. Although the alactic system generates ATP at a very high rate, it has a limited maximal capacity. The alactic system synthesizes ATP by donating the  $P_i$  from phosphocreatine (PCr) and combining it with ADP (the resulting molecule from the hydrolysis of ATP). During the resynthesis of ATP from PCr, a  $H^+$  is consumed ( $PCr + ADP + H^+ \xleftarrow{\text{Creatine Kinase}} ATP + \text{creatine}$ ). As a result, this reaction acts as an intramuscular buffer against the accumulation of  $H^+$  until PCr stores become depleted (8). As the resynthesis of ATP from PCR continues, levels of  $P_i$  increase within the muscle, which is an activator of the key glycolytic enzyme phosphofructokinase (PFK) (24). Following this reaction, ATP can also be resynthesized from 2 molecules of ADP through the adenylate kinase reaction ( $ADP + ADP \xleftarrow{\text{Adenylate Kinase}} ATP + AMP$ ) contributing as much as 20% of the restoration of ATP stores in the alactic system (24). AMP formed in this reaction can also be catalyzed by AMP deaminase to form IMP and  $NH_3$ . The formation of  $NH_3$  "consumes" a  $H^+$  to partially control  $H^+$  levels until glycolysis continues at a high rate ultimately leading to the accumulation of  $H^+$ .

At the onset of muscle contraction, there is an immediate need for ATP. Because PCr is the primary provider for the initial energy supply and PCr supply is limited in the muscle, PCr stores are rapidly depleted. Because of its significant contribution to the production of ATP at the onset of high intensity exercise, the concentration of PCr can be reduced to less than 40% of resting values within the first 10 s of exercise (21).

**Lactic Energy System.** As high intensity exercise duration continues, and PCr stores begin to diminish, the muscle requires an increase in the contribution of fast

glycolysis in order to meet the demands of intense exercise. It is estimated that it may take 5-6 seconds of high intensity exercise before the glycolytic pathway becomes the primary supplier of ATP (10). Intramuscular lactate and  $H^+$  increase as an indicator of the increased reliance on fast glycolysis, which gives the lactic energy system its name. As ATP hydrolysis continues to occur with muscular contraction, ADP, AMP,  $NH_3$  and  $P_i$  levels will increase. These metabolites are strong activators of the key rate limiting enzyme of glycolysis, phosphofructokinase (PFK) (10).

Jacobs et al. (17) examined the rate at which glycolysis was working during maximal exercise. The authors found that the rate of glycolysis was correlated with the concentration of lactate within the working muscle. In this study, 15 male subjects performed maximal exercise bouts lasting either 10 or 30 s on a cycle ergometer against a resistance which was standardized so that one pedal revolution resulted in 4.9 J of work per kg of body weight. Results showed that muscle lactate concentrations increased to 36 and 61 mmol per kg of dry weight after the 10 and 30 s bouts, respectively. These results show that there is indeed lactate accumulating during the initial ten seconds of high intensity exercise, suggesting that glycolysis is one source of ATP during this time frame. Researchers suggested that glycolysis has a greater capacity to provide energy over time than PCr, therefore glycolysis helps restore ATP while PCr stores are being depleted (3). This allows a smooth transition between the reliance on the alactic to the reliance on the lactic energy system.

The final product of glycolysis is pyruvate which can either be taken into the mitochondria of the cell where it is oxidized through aerobic metabolism or remain in the cytoplasm where it is converted to lactate. Pyruvate will enter mitochondria to be

consumed when there is sufficient mitochondrial activity to accept the glycolytic flux (3). However, if the glycolytic rate is faster than pyruvate can enter the mitochondria (such as during periods of high intensity exercise), pyruvate will accumulate in the cytoplasm since mitochondrial activity metabolizes pyruvate at a relatively slower rate than it is produced in the cytoplasm. Glycolysis also requires the reduction of  $\text{NAD}^+$  to  $\text{NADH}$  to allow the transfer of  $\text{H}^+$  and electrons within the cell (3). In order to prevent an accumulation of pyruvate in the cytoplasm and oxidize the  $\text{NADH}$  necessary for recycling  $\text{NAD}$  in the cytoplasm, lactate dehydrogenase (LDH) converts pyruvate into lactate to allow glycolysis to continue. Individuals can exercise up to a threshold intensity (known as the lactate threshold) with little or no accumulation of lactate in the plasma (32). However, when this critical intensity is surpassed, lactate begins to accumulate which is used as a measure to determine the rate of glycolysis occurring within the muscle.

### **$\text{H}^+$ Accumulation and Buffering Systems**

During prolonged high intensity exercise, fast glycolysis becomes self-regulating as  $\text{H}^+$ , a by-product of glycolysis, build up and lower intramuscular pH (23, 25). Previous studies have shown that 2-3 minutes of high intensity exercise to exhaustion can cause pH levels to drop from 7.0 to 6.4 (27). An accumulation of  $\text{H}^+$  has been attributed to muscle fatigue.

During high intensity exercise, the glycolytic rate can be increased up to 100 times those at rest but this rate can only be maintained for 1-4 minutes (10). Although there are a number of possibilities for this impairment of muscle functioning, the inhibition of key glycolytic enzymes appears to be a major cause. Rate limiting enzymes

in glycolysis, PFK and phosphorylase, are inhibited at a low pH (18). This occurs at different intensities in different people, depending on their training status and buffering capacity. A decrease in glycolytic enzyme activity decreases the rate of ATP synthesis which leads to muscle fatigue. Therefore, it may be beneficial for exercising muscles to reduce  $H^+$  accumulation and prevent a decrease in pH within the muscle cells (12).

Bogdanis et al. (2) examined power outputs and muscle metabolism during maximal sprints on a cycle ergometer. The authors proposed that intramuscular  $H^+$  accumulation appears to be a factor in the onset of fatigue. They determined that a high glycolytic rate found during the first 10 s of a sprint increased lactate levels within the muscle to as high as 51 mmol/kg of dry muscle. The authors found a decrease in muscle pH from 7.06 to 6.94 after 10 s and 6.82 after the last 20 s of a 30 sec sprint on a cycle ergometer. They speculated that the increase in  $H^+$  led to a decrease in the glycolytic rate during the sprint.

It has been proposed that a decrease in pH levels can have a negative effect on the glycolytic rate by inhibiting the activity of phosphorylase and PFK (19). Therefore,  $H^+$  removal (buffering) during exercise appears to be essential in order to improve performance, particularly when exercise is performed at high intensities (6). Many studies have shown that a rapid decline in force production occurs during intense exercise (commonly referred to as "fatigue") coinciding with a decline in cellular pH of active muscles (9, 12, 14). In order to counteract this accumulation of  $H^+$ , muscles have several methods that help maintain pH levels within a homeostatic range.

A method used to maintain pH within muscle cells are buffer systems. Buffers maintain pH by "accepting" the  $H^+$  ions in order to prevent their build up which would

ultimately decrease pH levels. Therefore, a greater muscle buffering capacity should allow for a more prolonged utilization of anaerobic glycolysis before a limiting pH is reached. The buffering capacity of a muscle is highly correlated with its glycolytic capacity and these two metabolic properties may possibly co-adapt (4). There are several different types of buffers utilized to maintain pH. According to Parkhouse et al. (26) the major buffering constituents of human skeletal muscle are bicarbonate, inorganic phosphate, protein, and the histidine-containing dipeptide, carnosine.

### **Carnosine**

Carnosine (B-alanyl-L-histidine) is a cytoplasmic dipeptide synthesized from the precursors L-histidine and beta-alanine by the enzyme carnosine synthetase. Histidine concentrations are high in plasma and within skeletal muscle relative to the Michaelis-Menten constant ( $K_m$ ) for carnosine synthase ( $K_m = 0.0168$  mM). Beta-alanine, on the other hand, has a lower concentration with a higher  $K_m$  for carnosine synthase ( $K_m = 1.0-2.3$  mM) (16). This leads to the notion that there is less beta-alanine available than is needed for carnosine production, making it a rate-limiting substrate.

Carnosine is found in skeletal muscle (the highest percentage found in Type II muscle fibers) and aids in the buffering of intramuscular  $H^+$ . The ability of a muscle to buffer additional  $H^+$  could be an indicator of the muscle's ability to maintain high intensity exercise, allowing for a more prolonged utilization of anaerobic glycolysis (22). It has been suggested that Type II fibers have a higher buffer capacity compared to Type I fibers (28). As previously mentioned, carnosine levels are higher in Type II fibers than in Type I. Therefore, a muscle with a predominance of Type II fibers would be better suited to buffer the accumulation of  $H^+$ . Mannion et al. (22) observed lower pH levels in

muscles with a higher percentage of Type II fibers following bouts of dynamic exercise. There is still controversy on how much carnosine contributes to the buffering capacity of human muscle. Hill et al. (15) suggested that carnosine may contribute less than 10% to the total buffering capacity of human muscle whereas other authors have reported carnosine contributing up to 20% (36).

Carnosine levels are higher in sprint athletes and bodybuilders compared to marathon runners and sedentary individuals (36). In order to determine whether or not beta-alanine supplementation itself actually increases carnosine levels, it is important to consider the training status of the subjects or measure carnosine concentrations at the start of the study. This would support the idea of whether or not beta-alanine is indeed effective in delaying fatigue. This would help determine whether or not increased carnosine levels are an effective way to improve the muscle's buffering capacity and ultimately improve performance.

Tallon et al. (36) examined the carnosine concentrations in the vastus lateralis of resistance trained bodybuilders. The bodybuilders had participated in bodybuilding at least 5 yrs with an average of 8 hrs of training per week. They hypothesized that carnosine concentrations in skeletal muscle are linked to frequent exposure to intramuscular acidosis. In this study, 6 male bodybuilders were studied as well as 6 untrained males of the same age. Results showed that carnosine concentrations were twice as high in the bodybuilders when compared to the untrained subjects. The concentrations of carnosine observed in the bodybuilders exceeded any reported in the current literature. They also found the bodybuilders had an average of 19% greater buffering capacity when compared to the average male. The authors concluded that

physiochemical buffering in type II muscle fibers was 40% higher in bodybuilders compared to untrained subjects. These results would lead one to believe that anaerobically trained individuals will have higher concentrations of carnosine within the muscle and therefore, a higher buffering capacity than non-trained individuals.

### **Beta-alanine Supplement**

Carnosine itself cannot be supplemented because when it is ingested, it is hydrolyzed within the stomach to produce histidine and beta-alanine. Therefore, ingestion of carnosine is not directly responsible for increasing intramuscular carnosine levels (11). Since beta-alanine is considered the rate limiting substrate for carnosine production in the muscle, many studies have examined the efficacy of beta-alanine supplementation to increase intramuscular carnosine levels (1, 11, 15, 20). Hill et al. (15) examined the influence of beta-alanine supplementation on skeletal muscle carnosine concentrations. In this study, 13 physically active male subjects (not involved with a structured training program), supplemented with beta-alanine (6g per day) for 4 weeks and 8 of the 13 subjects continued supplementation up to 10 weeks while 12 matched control subjects received a placebo in a double blind fashion. Muscle biopsies were taken from the vastus lateralis pre and post supplementation and categorized by fiber type. Results showed that the ingestion of beta-alanine significantly increased skeletal whole muscle carnosine concentrations from 19.9 to 30.1 and 34.7 mmol/kg of dry muscle (60 and 80% increases) after 4 and 10 weeks, respectively. These changes suggest that extended periods of supplementation, 10 weeks compared to 4, can continue to increase muscle carnosine concentration, but most of the increase takes place within the first 4 weeks. Therefore, there may be a ceiling effect in terms of the maximal amount of carnosine that can be

found within the muscle. In addition, even though carnosine was initially 1.71 times higher in type II fibers compared to type I, it increased similarly in both fiber types. These results demonstrate that muscle carnosine concentrations can be increased through beta-alanine supplementation.

Baguet et al. (1) investigated the effects of beta-alanine supplementation on carnosine levels as well as the washout period of carnosine in human skeletal muscle. Fifteen untrained subjects supplemented for 5-6 weeks with either 4.8g/day of beta-alanine or placebo. Carnosine levels were measured pre, and post-supplementation as well as 3 and 9 weeks of washout period. After the 5-6 weeks of supplementation, the beta-alanine group showed 39, 27, and 23% increases in carnosine levels in their soleus (primarily type I fibers), tibialis anterior (primarily type II), and gastrocnemius (primarily type II fibers), respectively. Carnosine levels remained constant in the placebo group. After 3 weeks of washout period carnosine levels decreased to 67% of their post-supplementation values and returned to baseline values around the 9 week period. The authors estimated that carnosine levels fell at a rate of 2-4% per week. They concluded that it may take months before a washout period of carnosine loading is complete and therefore crossover designs should be planned accordingly. It is possible that Type II fibers did not show as large of an increase in carnosine levels as Type I fibers because they were approaching the "ceiling effect" due to higher levels prior to supplementation (30).

Derave et al. (7) examined the effects of beta-alanine supplementation on carnosine concentrations. In this study, 15 sprint trained athletes were either supplemented with beta-alanine (4.8g/day) or a placebo for a period of 4 weeks. Muscle

biopsies were taken from the soleus and gastrocnemius muscle of all the athletes. Subjects were tested before and after the 4 weeks of supplementation. Results demonstrated that beta-alanine supplementation significantly increase the carnosine content in both the soleus (47%) and gastrocnemius (37%) after 4 weeks with 4.8g/day of beta-alanine. Results also showed that even subjects with a very high initial carnosine content (>12 mmol/l) were still able to elevate their concentrations by an additional 4-5 mmol/l. This suggests that there may be a "ceiling effect" and that the "normal" human muscle carnosine content is far from maximal. The authors conclude that increasing muscle carnosine through beta-alanine supplementation will help maintain the intramuscular environment during intensive exercise by countering the accumulation of  $H^+$ .

### **Beta-Alanine and Performance**

Recent studies have investigated the efficacy of beta-alanine supplementation in increasing carnosine levels to improve exercise performance. In theory, increasing carnosine will improve the buffering capacity of the muscle, delaying fatigue resulting from acidosis. This should be most effective in exercise bouts that would produce the greatest decrease in muscle pH. Suzuki et al. (34) observed that subjects that had high carnosine content in their vastus lateralis performed better during the latter stages of a 30 s Wingate test on a cycle ergometer. They also found that there was a strong positive correlation between carnosine concentration and the percentage of type II muscle fibers in the exercising muscle.

Hill et al (15) had 13 college-aged active men, not involved in a structured training program, cycle at 110% of their mean power output determined during the last

60 s of an incremental cycling test to exhaustion. Subjects ingested 4-6g of beta-alanine per day in a manner of progressing dosages as the weeks progressed. Following 4 and 10 weeks of supplementation, mean cycling times to exhaustion increased 12 and 16%, respectively.

Stout et al. (33) examined beta-alanine supplementation and its effects on physical working capacity at fatigue threshold (PWCFT), ventilatory threshold (VT), peak VO<sub>2</sub>, and time to exhaustion (TTE). Twenty – two middle aged (23-37 yrs old) women supplemented with either beta-alanine or placebo for 28 days. During the first 7 days, subjects ingested 3.2g/day of beta-alanine or placebo, followed by 6.4g/day during the remaining 21 days. Prior to the supplementation period, subjects performed a continuous, incremental cycle ergometry test to exhaustion to determine PWCFT, VT, peak VO<sub>2</sub> and TTE. Following supplementation, subjects in the beta-alanine group showed significant increases of 14, 13, and 3% in VT, PWCFT, and TTE, respectively. There were no improvements seen in the placebo group. These results suggest that bouts of exercise occurring at the fatigue threshold may be improved through beta-alanine supplementation thus giving a person the ability to maintain a certain intensity for a longer period of time before exhaustion.

Van Thienen et al. (37) examined the effect of beta-alanine supplementation on sprint performance following a bout of endurance cycling. Seventeen healthy young men that were moderately trained in cycling supplemented with beta-alanine or a placebo for an 8-week period. Subjects ingested 2g of beta-alanine per day during weeks 1 and 2, increasing to 3g per day during weeks 3 and 4, eventually increasing to 4g per day during week 5. Before and after the supplementation period, subjects completed a 10 min time

trial and a 30 s isokinetic sprint following a 110 min simulated cycling race. During the posttest, the beta-alanine group displayed significant increases of 11.4 and 5% in peak power output and mean power output, respectively, during the 30-s sprint; however, there were no significant increases shown in mean power output during the 10 min time trial. Blood lactate and pH levels were similar between both testing groups throughout all tests. These results suggest that beta-alanine supplementation can improve sprint cycling performance, specifically during short durations, even after a bout of exhaustive exercise.

Derave et al. (7) examined the effect of beta-alanine supplementation on fatigue in trained sprinters (personal best of <53 s in 400 meters). Performance tests consisted of repeated maximal isokinetic contraction bouts, isometric contractions at 45% maximal voluntary contraction, and 400 m sprint times. Fifteen male track and field athletes (400 m sprinters) supplemented with either 4.8 g/day of beta-alanine or placebo for 4 weeks. Following supplementation the beta-alanine group showed significant increases in carnosine content of the soleus and gastrocnemius of 47 and 37%, respectively. The beta-alanine group also showed significant improvements in knee torque during the 4<sup>th</sup> and 5<sup>th</sup> bouts of 30 maximal isokinetic knee extensions at a constant 180 degrees/sec whereas the placebo group showed no improvements. However, there were no improvements seen in either group in isometric endurance or 400 m race times. The authors found that increases in muscle carnosine did not improve isometric endurance or 400 m race times. However beta-alanine supplementation was a successful method of increasing the carnosine content within muscles. The beta-alanine supplementation may not have improved the isometric endurance or 400 m race times because of the training status of the subjects. It is possible that the muscles developed through their prior

training and were already reaching a ceiling effect of maximal carnosine capacity within the muscle and were therefore not able to increase carnosine concentrations as much.

Sweeney et al. (35) examined the effects of 5 weeks of beta-alanine supplementation on power outputs during high intensity, repeat sprints. Subjects included 19 physically active college-aged men. It was a double blind placebo controlled study in which the beta-alanine group consumed 4g/day of beta alanine during week 1 and 6g/day during weeks 2-5. Subjects completed 2 sets of 5x5 sec sprints with 45 s recovery between sprints and 2 min recovery between sets, pre and post supplementation. The sprints were completed on a non-motorized treadmill, using 15% of body weight as resistance. Following supplementation no differences were seen between groups for peak or mean horizontal power as well as % fatigue. The authors concluded that beta-alanine supplementation was not an effective means of increasing power during brief, repeated sprints. They suggested that PCr depletion was likely a greater limiting factor than H<sup>+</sup> accumulation and the need for intramuscular buffering.

In a similar study, Smith et al. (29) examined the effects of beta-alanine supplementation and high-intensity interval training (HIIT) on endurance performance and aerobic metabolism. In this study, 46 recreationally trained college-aged males participated in the study. Subjects completed two cycling tests including a time to peak VO<sub>2</sub> (VO<sub>2</sub>TTE) and total work done (TWD) performed at 110% of their pre-determined VO<sub>2</sub> max. Subjects also completed a ventilatory threshold (VT) and body composition test pre, mid, and post-supplementation period. Following pre-testing, subjects were assigned to either a beta-alanine or placebo group; and both groups completed 6 weeks of HIIT. Subjects supplemented with 6 g of beta-alanine per day during the first 3 weeks

and 3 g per day during the second 3 weeks. Subjects performed HIIT on an electronically braked cycle ergometer. During the first 3 weeks, subjects trained 3 days per week and completed 5 sets of 2 min intervals with 1 min rest periods at a workload of 90-110% of maximum power output. During the second 3 week period, subjects followed the same protocol except they added one more set during weeks 6 and 7. Significant improvements were seen in VO<sub>2</sub> peak, VO<sub>2</sub>TTE, and TWD after three weeks of training. Results showed improvements in VO<sub>2</sub> peak from pre to post in both groups with no significant differences seen between the two groups. However, the beta-alanine group displayed a significant increase from mid to post-testing whereas the placebo group did not. Similar results were seen in the VO<sub>2</sub>TTE test. Both groups showed similar improvements from pre to post-testing and again the beta-alanine group was the only group to significantly improve from mid to post-testing. The authors concluded that HIIT is an effective method of training for improving aerobic performances and that beta-alanine supplementation has no further enhancing effect on performance. The HIIT training could have been more successful in improving buffering capacity than the beta-alanine supplementation which would lead to the outcome of both groups increasing similarly.

### **Summary and Conclusions**

At the onset of exercise, both the aerobic and anaerobic energy systems work together in order to produce enough energy to meet the demands of the contracting muscles. However, at the onset and early stages of high intensity exercise, the anaerobic system is the primary provider of energy. Energy is derived by the hydrolysis of ATP into P<sub>i</sub>, ADP and H<sup>+</sup>. The lactic system, which is part of the anaerobic energy system,

provides the most immediate source of ATP at the onset of muscular contraction. PCr provides ATP through the creatine kinase reaction where PCr is split and donates its  $P_i$  to ADP in order to resynthesize ATP. This process can only be maintained at a high rate for roughly 10 s before PCr stores begin to diminish. At this point anaerobic glycolysis becomes the primary provider of ATP. Through this chemical process, ATP can still be resynthesized at a relatively high rate and has a greater capacity to resynthesize ATP than the PCr system. Pyruvate is a substrate produced from glycolysis and is either transported into the mitochondria where it is oxidized through aerobic metabolism or converted into lactic acid. Lactic acid is produced when pyruvate and  $H^+$  cannot be transported into the mitochondria fast enough to keep up with the rate of glycolysis. When high intensity exercise continues,  $H^+$  accumulate within the muscles. Research suggests that an accumulation of  $H^+$  will lower the pH and inhibit the rate-limiting enzyme of glycolysis which will slow down glycolysis and eventually lead to fatigue (9, 12, 13).

In order to delay fatigue, muscles have buffer systems to maintain muscle pH levels in a state of homeostasis. Carnosine is one of these intramuscular buffers that has been demonstrated to buffer the accumulation of  $H^+$  and help delay the onset of fatigue (9). Beta-alanine, a rate limiting substrate of carnosine, has been proposed to increase carnosine levels within the muscle through supplementation (11, 15). Research has been done on beta-alanine supplementation and its effect on carnosine concentrations within the muscle. It has been demonstrated that 4-6g of beta-alanine a day with dosing periods typically lasting from 4-6 weeks can increase carnosine concentrations within the muscle (7, 15).

One problem with many previous studies dealing with beta-alanine supplementation and increasing carnosine levels is the lack of control within the study's design. It has already been demonstrated that beta-alanine supplementation can increase the carnosine concentration within the muscles but many of these studies don't take into account the exercise tendencies of the subjects. It is hard to determine whether or not carnosine was elevated from strictly the beta-alanine supplementation or from high intensity exercise done by the subjects.

By understanding how beta-alanine supplementation affects carnosine levels, which ultimately should improve buffering capacity, we can have a better idea of how to improve performances done at a level of high intensity. This would especially be important in sports that require long durations of high intensity exercise, such as combative sports, track and field, hockey, and basketball. An enhanced buffering capacity would enable athletes to perform at a higher level longer before the sensation of fatigue sets in. As mentioned in the previous studies, beta-alanine supplementation has been shown to improve performances during bouts of high intensity exercise lasting from 30 to 60 sec (34, 37). Based off of these tests results and using the concepts of muscle metabolism during high intensity exercise mentioned earlier in this review, we suggest that improvements in performances could also be seen during bouts of high intensity exercise lasting from 1-3 min following beta-alanine supplementation.

## REFERENCES

1. Baguet, A, Reyngoudt, H., Pottier, A., Everaert, I., Callens, S., Achten, E., Derave, W. Carnosine loading and washout in human skeletal muscles. *Journal of Applied Physiology* 106: 837-842, 2009.
2. Bogdanis, G, Nevill, ME., Lakomy, HK., Boobis, LH. Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. *Acta Physiol Scand.* 163: 261-272., 1998.
3. Brooks, G. The lactate shuttle during exercise and recovery. *Medicine and Science of Sport and Exercise* 3: 360-368, 1986.
4. Castellini, M, Somero, GN. Buffering capacity of vertebrae muscle: correlations with potentials for anaerobic function. *Journal of Comparative Physiology* 143: 191-198, 1981.
5. Culbertson, J, Kreider, R., Greenwood, M., Cooke, M. Effects of beta-alanine on muscle carnosine and exercise performance: a review of the current literature. *Nutrients* 2: 75-98, 2010.
6. Danforth, W. Activation of glycolytic pathway in muscle. In: Control of Energy Metabolism. New York: Academic Press, 1965.
7. Derave, W, Ozdemir, M, Harris, R, Pottier, A, Reyngoudt, H, Koppo, K, Wise, J, and Achten, E. Beta alanine supplementation augments muscle carnosine content and attenuates fatigue during repeated isokinetic contraction bouts in trained sprinters. *J Appl Physiol* 103: 1736-1743, 2007.
8. Febbraio, M, Flanagan, TR., Snow, RJ., Zhao, S., Carey, MF. Effect of creatine supplementation on intramuscular TCr, metabolism and performance during intermittent, supramaximal exercise in humans. *Acta Physiol Scand.* 155: 387-395, 1995.
9. Fitts, R. Cellular mechanisms of muscle fatigue. *Physiol Rev* 74: 49-94, 1994.
10. Gatin, P. Energy system interaction and relative contribution during maximal exercise. *Sports Med* 31: 725-741, 2001.
11. Harris, R, Tallon, M, Dunnett, M, Boobis, L, Coakley, J, Kim, H, Fallowfield, J, Hill, C, Sale, C, and Wise, J. The absorption of orally supplied beta-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. *Amino Acids* 30: 279-289, 2006.

12. Hermansen, L. Effect of metabolic changes on force generation in skeletal muscle during maximal exercise. *Ciba Found Symp.* 82: 75-88, 1981.
13. Hermansen, L, Osnes, JB. Blood and muscle pH after maximal exercise in man. *Journal of Applied Physiology* 32: 304-308, 1972.
14. Hermansen, L, Stensvold, L. Production and removal of lactate during exercise in man. *Acta Physiol Scand.* 86: 191-201, 1972.
15. Hill, C, Harris, R, Kim, H, Harris, B, Boobis, L, Sale, C, and Wise, J. Influence of beta-alanine supplementation on skeletal muscle carnosine concentration and high intensity cycling capacity. *Amino Acids* 32: 225-233, 2007.
16. Horinishi, H, Grillo, M., Margolis, FL. . Purification and characterization of carnosine synthetase from mouse olfactory bulbs. *Journal of Neurochemistry* 31: 909-919, 1978.
17. Jacobs, I, Tesch, P., Bar-Or, O., Karlsson, J., Dotan, R. Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. *Journal of Applied Physiology* 55: 365-367, 1983.
18. Karlsson, J, Bonde-Peterson, F., Henriksson, J., Knuttgen, HG. Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. *Journal of Applied Physiology* 38: 763-767, 1975.
19. Karlsson, J, Sjodin, B., Thorstensson, A., Hulten, B., Frith, K. LDH isozymes in skeletal muscle of endurance and strength trained athletes. *Acta Physiol Scand.* 93: 150-156., 1975.
20. Kendrick, I, Kim, J., Harris, R., Kim, C., Dang, V., Lam, T., Bui, T., Wise, J. The effect of 4 weeks b-alanine supplementation and isokinetic training on carnosine concentrations in type I and II human skeletal muscle fibres. *European Journal of Applied Physiology.* 106: 131-138, 2009.
21. Lakomy, H. Measurement of work and power output using friction-loaded cycle ergometers. . *Ergonomics* 29: 509-517, 1986.
22. Mannion, A, Jakeman, P, Dunnett, M, Harris, R, and William, P. Carnosine and anserine concentrations in the quadriceps femoris muscle of healthy humans. *Eur J Appl Occup Physiol* 64: 47-50, 1992.
23. McCartney, N, Spriet, LL., heigenhauser, GJ. Muscle power and metabolism in maximal intermittent exercise. *Journal of Applied Physiology* 60: 1164-1196, 1986.
24. McMahon, S, Jenkins, D. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. *Sports Medicine* 32: 761-784, 2002.

25. Metzger, J, Fitts, RH. Role of intracellular pH in muscle fatigue. *Journal of Applied Physiology* 62: 1392-1397, 1987.
26. Parkhouse, W, McKenzie, DC. Possible contribution of skeletal muscle buffers to enhanced anaerobic performance: a brief review. *Medicine and Science in Sports and Exercise* 16: 328-338, 1984.
27. Robergs, R, Ghiasvand, F., Parker, D. Biochemistry of exercise-induced metabolic acidosis. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology* 287: 502-516, 2004.
28. Sahlin, K. Metabolic factors in fatigue. *J Sports Med* 13: 99-107, 1992.
29. Smith, A, Walter, A., Graef, J., Kendall, K., Moon, J., Lockwood, C., Fukuda, D., Beck, T., Cramer, J., Stout, J. Effects of B-alanine supplementation and high-intensity interval training on endurance performance and body composition in men: a double-blind trial. *Journal of the International Society of Sports Nutrition* 6: 1-9, 2009.
30. Sout, J, Graves, SB., Smith, AE., Hartman, MJ., Cramer, JT., Beck, TW., Harris, RC. The effect of beta-alanine supplementation on neuromuscular fatigue in elderly (55-92 Years): a double-blind randomized study. *Journal of the International Society of Sports Nutrition* 5: 1-6, 2008.
31. Spriet, L, Lindinger, MI., McKelvie, RS., Heigenhauser, G., Jones, N. Muscle glycogenolysis and H<sup>+</sup> concentration during maximal intermittent cycling. *Journal of Applied Physiology* 66: 8-13, 1989.
32. Stalin, B, Astrand, PO. Muscle ATP, CP, and lactate during exercise after physical conditioning. In: *Muscle metabolism during exercise*. New York: Plenum Press, 1971.
33. Stout, J, Cramer, JT., Zoeller, RF., Torok, D., Costa, P., Hoffman JR., Harris, RC., O'Kroy, J. Effects of beta-alanine supplementation on the onset of neuromuscular fatigue and ventilatory threshold in women. *Amino Acids* 32: 381-386, 2007.
34. Suzuki, Y, Ito, O, Mukai, N, Takahashi, H, and Takamatsu, K. High levels of skeletal muscle carnosine contributes to the latter half of exercise performance in 30-s maximal cycle ergometer sprinting. *Japn J Physiol* 52: 199-205, 2002.
35. Sweeney, K, Wright, GA., Brice, AG., Doberstein, ST. The effect of beta-alanine supplementation on power performance during repeated sprint activity. *Journal of Strength and Conditioning* 2009.
36. Tallon, M, Harris, R, Boobis, L, Fallowfield, J, and Wise, J. The carnosine content of vastus lateralis is elevated in resistance trained bodybuilders. *J Strength Cond Res* 19: 725-729, 2005.

37. Van Thienen, R, Van Proeyen, K., Vanden Eynde, B., Puype, J., Lefer, T., Hespel, P.  
B-alanine improves sprint performance in endurance cycling. *Medicine & Science in  
Sports & Exercise* 41: 898-903, 2009.