Amino Acids (2006)
DOI 10.1007/s00726-006-0474-z
Printed in The Netherlands

Amino Acids

Effects of β-alanine supplementation on the onset of neuromuscular fatigue and ventilatory threshold in women

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Received October 4, 2006
Accepted October 30, 2006
Published online November 30, 2006;

Summary. This study examined the effects of 28 days of β-alanine supplementation on the physical working capacity at fatigue threshold (PWC_{FT}), ventilatory threshold (VT), maximal oxygen consumption ($\dot{V}O_{2\text{-MAX}}$), and time-to-exhaustion (TTE) in women. Twenty-two women (age ± SD 27.4 ± 6.1 yrs) participated and were randomly assigned to either the β-alanine (CarnoSyn™) or Placebo (PL) group. Before (pre) and after (post) the supplementation period, participants performed a continuous, incremental cycle ergometry test to exhaustion to determine the PWC_{FT}, VT, $\dot{V}O_{2\text{-MAX}}$, and TTE. There was a 13.9, 12.6 and 2.5% increase ($p < 0.05$) in VT, PWC_{FT}, and TTE, respectively, for the β-alanine group, with no changes in the PL ($p > 0.05$). There were no changes for $\dot{V}O_{2\text{-MAX}}$ ($p > 0.05$) in either group. Results of this study indicate that β-alanine supplementation delays the onset of neuromuscular fatigue (PWC_{FT}) and the ventilatory threshold (VT) at submaximal workloads, and increase in TTE during maximal cycle ergometry performance. However, β-alanine supplementation did not affect maximal aerobic power ($\dot{V}O_{2\text{-MAX}}$). In conclusion, β-alanine supplementation appears to improve submaximal cycle ergometry performance and TTE in young women, perhaps as a result of an increased buffering capacity due to elevated muscle carnosine concentrations.

Keywords: Carnosine – β-Alanine – Ergogenic aids – Electromyography – Cycle ergometry

Introduction

A number of studies have used surface electromyographic (EMG) procedures to identify the power output associated with the onset of neuromuscular fatigue (NMF) during cycle ergometry (deVries et al., 1987, 1990; Halal et al., 1987; Housh et al., 1990, 1991; Matsumoto et al., 1990). Neuromuscular fatigue is typically characterized by an increase in the electrical activity of working muscles over time (deVries et al., 1987, 1989; Matsumoto et al., 1990; Moritani et al., 1993). Moritani et al. (1993) suggested that the fatigue-induced increase in EMG amplitude is a result of progressive recruitment of additional motor units (MU) and/or increase in the firing frequency of the active MUs. In theory, therefore, cycle ergometry exercise at power outputs at or below the NMF threshold can be maintained continuously without a significant increase in EMG amplitude over time.

Based on this model, deVries et al. (1987, 1990) developed an incremental cycle ergometer test called the *physical working capacity at fatigue threshold* (PWC_{FT}), which utilizes the relationship between EMG amplitude and fatigue during submaximal cycle ergometry to identify the power output that corresponds to the onset of NMF. The PWC_{FT} represents the highest power output that results in a non-significant ($p > 0.05$) increase in muscle activation of the vastus lateralis over time. The PWC_{FT} test has been demonstrated as reliable (deVries et al., 1987, 1989; Stout et al., 2000), valid (deVries et al., 1989), and sensitive to changes in fitness level (deVries et al., 1989). Furthermore, deVries et al. (1982) and Moritani et al. (1993) have reported strong relationships between NMF and ventilatory threshold (VT). The VT is defined as a disproportionate increase in pulmonary ventilation with oxygen consumption ($\dot{V}O_2$) during incremental exercise and is thought to represent the ventilatory response to the increased production of CO_2 caused by the buffering of lactic acid as it accumulates in the blood during exercise. It is possible that the association between the PWC_{FT} (increased muscle activation) and VT (increased pulmonary ventilation) during fatigue is related to a greater

number of motor units being activated resulting in an increased level of ventilation, however, the specific physiological mechanism responsible for this association still remains unclear.

It has been proposed that exercise-induced decreases in intramuscular pH, due to increases in [H^+], may interfere with the excitation-contraction coupling process of skeletal muscle, which, in turn, leads to decreases in power output and fatigue (Fitts and Holloszy, 1976). In order to maintain pH homeostasis, various buffering systems are involved, including the bicarbonate system. In addition, at least one animal study has shown that increases in [H^+] may activate the type III and IV afferent nervous system pathways (Dempsy et al., 1995), which may also contribute to the abrupt increase in ventilation rates during incremental exercise (VT) via their stimulatory actions of the respiratory center in the medulla.

It has been shown that intramuscular carnosine (β-alanyl-L-histidine) may be an often overlooked, yet effective physiological H^+ buffer. Hill et al. (2006) recently demonstrated that β-alanine supplementation can significantly increase muscle carnosine levels, which directly corresponded to an improvement in exercise performance. It was hypothesized that the increases in muscle carnosine concentrations caused by β-alanine supplementation improved the buffering capacity of the participants, which improved their performance. Similarly, Stout et al. (2006), reported significant increases in PWC_{FT} in untrained men after 28 days of β-alanine supplementation ($3.2\,g \cdot d^{-1}$). Furthermore, Zoeller et al. (2006) reported a significant increase (7%) in the VT in a similar sample of untrained men after ingesting a supplement containing β-alanine ($3.2\,g \cdot d^{-1}$) for 28 days. Moreover, Kim et al. (2006) demonstrated a significant increase in VT in highly trained young male cyclists after 12 weeks of β-alanine ($4.8\,g \cdot d^{-1}$) supplementation and training, which corresponded with a 58.8% increase in muscle carnosine.

In addition to the demonstrated changes in PWC_{FT} and VT, Zoeller et al. (2006) and Kim et al. (2006) examined the effects of β-alanine supplementation on improvements in maximal oxygen consumption ($\dot{V}O_{2\text{-}MAX}$) and time to exhaustion (TTE). Neither study reported any significant changes in $\dot{V}O_{2\text{-}MAX}$, and Zoeller et al. (2006) found no change in TTE. Kim et al. (2006), however, did report a significant increase in TTE. The difference between the Zoeller et al. (2006) and Kim et al. (2006) investigations regarding the TTE is unknown, but the outcome may have been related to the duration of β-alanine supplementation (4 vs. 12 weeks), supplement dosage ($3.2\,g \cdot d^{-1}$ vs. $4.8\,g \cdot d^{-1}$) or differences in subjects' training status (untrained vs. trained).

In theory, increasing skeletal muscle carnosine concentrations using β-alanine supplementation may delay fatigue (as measured by VT and PWC_{FT}) by increasing H^+ buffering capacity during exercise (Harris et al., 2006), which has been demonstrated in both trained and untrained men. To date, however, no studies have examined the effects of β-alanine supplementation on exercise performance in women. There is evidence that suggests differences between men and women in ventilatory responses to incremental exercise (Kilbride et al., 2003), anaerobic indices calculated during cycle ergometry (Bulbulian et al., 1996), $\dot{V}O_{2\text{-}MAX}$ (Vogel et al., 1986), muscle fatigability and TTE (Clark et al., 2005). Thus, we felt there was a need to examine the influence of β-alanine supplementation in women and, hence, the purpose of this study was to examine the effects of 28 days of β-alanine supplementation on the PWC_{FT}, VT, $VO_{2\text{-}MAX}$, and TTE in women.

Materials and methods

Subjects

Twenty-two females volunteered for this investigation and their demographic information is presented in Table 1. All procedures were approved by the Florida Atlantic University Institutional Review Board for Human Subjects Experimentation before the initiation of the study, and each

Table 1. Age, height and body mass of the subjects at the start of the study, and body mass at the end of 4 weeks supplementation with β-alanine or placebo

		Age (yrs)	Height (cm)	Body mass (kg)	
				0 weeks	4 weeks
β-Alanine	Mean	28.9	161.1	58.0	58.0
$n = 11$	SD	8.1	6.4	10.5	10.4
Placebo	Mean	25.8	164.4	62.2	62.0
$n = 11$	SD	4.0	6.7	10.1	9.7

Table 2. Dosing strategies employed for the two treatment groups

Week	Dosing times (mg)				Per day (g)
	9 am	12 am	3 pm	6 pm	
1	800	800	800	800	3.2
2	1600	1600	1600	1600	6.4
3	1600	1600	1600	1600	6.4
4	1600	1600	1600	1600	6.4

Total: 156.8 g β-alanine (β-Ala) or maltodextrin (P)

participant was advised of any possible risks before providing written informed consent.

Experimental design

This was a double-blind, randomized, placebo-controlled, parallel design. None of the participants had ingested creatine, or any other dietary supplements, for a minimum of 12 weeks before the initiation of this study. During the course of the study, the participants were asked to maintain their current exercise and dietary patterns and abstain from other nutritional supplements, nonprescription drugs, and caffeine. Prior to the testing, each participant was randomly assigned to either the β-alanine ($n = 11$) or placebo (PL, $n = 11$) group. After the pre-supplementation testing, the β-alanine or placebo was administered each day in 4 divided doses. During days 1–7, $3.2\,g \cdot d^{-1}$ were consumed, and $6.4\,g \cdot d^{-1}$ were taken for days 8–28. The dosing strategy is provided in Table 2. The β-alanine (CarnoSyn™) was obtained from Natural Alternatives International, San Marcos, USA.

Exercise tests

Prior to and following the supplementation protocol, participants performed a continuous graded exercise test (GXT) on an electronically braked cycle ergometer (Excalibur Sport, Groningen, Netherlands) to determine $\dot{V}O_{2\text{-MAX}}$, ventilatory threshold (VT), physical working capacity at fatigue threshold (PWC_{FT}) and time to exhaustion (TTE). Pre- and post-supplementation testing took place at the same time of day (±2 h) for each subject using the same equipment. The post-supplementation GXT was administered within 24 h after termination of the supplementation protocol. Participants reported to the lab after fasting for 3 h prior to each test. During each GXT, the initial power output was set at 40 W and increased by 20 W every 3 min until the participant could no longer maintain the required power output at a pedaling rate of 70 rpm or volitional termination due to fatigue.

Oxygen consumption measurements

Respiratory gases were monitored and continuously analyzed with open-circuit spirometry and used to calculate variables including minute ventilation ($\dot{V}_E$), oxygen consumption rate ($\dot{V}O_2$), carbon dioxide expiration rate ($\dot{V}CO_2$), and respiratory exchange ratio (RER) with a metabolic cart and the manufacturer's software (True One 2400® Metabolic Measurement System, Parvo-Medics Inc., Provo, UT). The data was averaged over 30-s intervals. The metabolic cart was calibrated prior to each test with room air for flow rate and gases of known volume and concentration for the O_2 and CO_2 analyzers. The highest 30-s $\dot{V}O_2$ value during the GXT was recorded as the maximal oxygen uptake ($\dot{V}O_{2\text{-MAX}}$) if it coincided with at least two of the following criteria: (a) plateau in heart rate (HR) or HR values within 10% of the age-predicted HR_{max}, (b) plateau in $\dot{V}O_2$ (defined by an increase of not more than $150\,ml \cdot min^{-1}$), and/or (c) RER value greater than 1.15 (Day et al., 2003).

Determination of ventilatory threshold

The ventilatory threshold (VT) was determined from a plot of ventilation ($\dot{V}_E$) against $\dot{V}O_2$ as described previously (Orr et al., 1982). Two linear regression lines were fit to the lower and upper portions of the $\dot{V}_E$ vs. $\dot{V}O_2$ curve, before and after the break point, respectively. The intersection of these two lines was defined as the VT. The determination of VT was performed by two independent and experienced exercise physiologists who were blinded to the identity of the subjects and the experimental condition. The $\dot{V}O_2$ that was associated with the VT break point was recorded as the representative value.

Test-retest reliability for the VT was determined by using 12 female participants measured 28 days apart. Using the recommendations of Weir (2005), the intraclass correlation coefficient was 0.91 (SE = $0.11\,l \cdot min^{-1}$), which was similar to that reported by Amann et al. (2004) in young trained cyclists ($r = 0.95$). In addition, there was no significant difference ($p > 0.05$) between the mean VT values from trial 1 (mean ± SE $1.41 \pm 0.06\,l \cdot min^{-1}$) to trial 2 ($1.40 \pm 0.07\,l \cdot min^{-1}$).

Electromyographic (EMG) measurements

A bipolar (2.54 cm center-to-center) surface electrode (Quinton Quick prep silver–silver chloride) arrangement was placed on the right thigh over the lateral portion of the vastus lateralis muscle, midway between the greater trochanter and the lateral condyle of the femur. The reference electrode was placed over the iliac crest. Interelectrode impedance was kept below 5000 Ω by careful abrasion of the skin. The raw EMG signals were pre-amplified (gain: ×1000) using a differential amplifier (EMG100C, Biopac Systems, Inc., Santa Barbara, CA), sampled at 1000 Hz, and stored on a personal computer for off-line analysis. The EMG signals were later bandpass filtered from 10–500 Hz (2nd order Butterworth filter) and expressed as root mean square (rms) amplitude values (μVrms) by software (AcqKnowledge v3.7, Biopac Systems, Inc., Santa Barbara, CA).

Determination of PWC_{FT}

The PWC_{FT} values were determined using the EMG amplitude values from the vastus lateralis muscle from the methods described by deVries et al. (1987, 1990). The subjects began pedaling (with toe clips) at 40 W (70 rpm) on a calibrated, electronically-braked cycle ergometer (Lode Excalibur Sport Cycle Ergometer, Groningen, The Netherlands). As previously described, the power output was then increased by 20 W every 3 min until the subject could no longer maintain 70 rpm. During each 3-min interval, nine 10-s EMG samples were recorded from the vastus lateralis. The PWC_{FT} was determined by averaging the highest power output that resulted in a nonsignificant ($p > 0.05$; single-tailed t-test) slope value for the EMG amplitude vs. time relationship with the lowest power output that resulted in a significant ($p \leq 0.05$) slope value (deVries et al., 1987, 1990; Stout et al., 2000).

Test-retest reliability for the PWC_{FT} test was determined by using 12 female subjects measured 28 days apart. The intraclass correlation coefficient was 0.964 (SE 11.66 W), which was similar to values previously reported by Stout et al. (2000) and deVries et al. (1989, 1990) in young athletic women ($r = 0.940$), young men ($r = 0.947$) and older men ($r = 0.976$), respectively. In addition, there was no significant difference ($p > 0.05$) between the mean PWC_{FT} values from trial 1 (110.8 ± 8.7 W) to trial 2 (107.5 ± 9.2 W).

Dietary analysis

In order to analyse the dietary data, 3-day dietary recalls were evaluated during the 1st and 4th week using Nutritrac Dietary Analysis software (Version 3.0). The dietary recalls were analyzed for total kilocalorie intake (kcal) and macronutrient percentages (carbohydrate, protein, and fat).

Statistical analyses

Four separate two-way mixed factorial ANOVAs (time [pre- vs. post-supplementation]) × group [β-alanine vs. placebo]) were used to analyze the VT, PWC_{FT}, $VO_{2\text{-MAX}}$, and TTE data. Follow-up analyses included either dependent or independent-sample t-tests. In addition, independent-samples t-tests were used to analyze the caloric intake (kcal) calculated from the dietary analyses as well as the body mass (kg) data. Prior to all statistical analyses, the alpha level was set to $p \leq 0.05$ to determine statistical significance. Data were statistically analysed using SPSS version 12.0® (SPSS Inc., Chicago, IL) software.

Table 3. Mean and standard error (SE) values for ventilatory threshold, the physical working capacity at fatigue threshold, maximal oxygen consumption, and time-to-exhaustion for the pre- and post-supplementation trials with either the placebo or β-alanine

		Ventilatory threshold ($l \cdot min^{-1}$)		Physical working capacity at fatigue threshold (W)		Maximal oxygen consumption ($l \cdot min^{-1}$)		Time-to-exhaustion (s)	
		Placebo	β-Alanine	Placebo	β-Alanine	Placebo	β-Alanine	Placebo	β-Alanine
Pre-supplementation	mean	1.43	1.30	111.82	113.64	1.90	1.91	1133.55	1117.55
	SE	0.06	0.10	9.52	12.45	0.11	0.16	91.53	118.98
Post-supplementation	mean	1.42	1.51**	108.18	130.00**	1.91	1.91	1133.36	1146.73*
	SE	0.08	0.13	10.10	12.99	0.15	0.15	98.15	110.27

* $p < 0.05$, ** $p > 0.001$ for pre- to post-difference

Results

Table 3 shows the mean and standard error values for VT, PWC_{FT}, $\dot{V}O_{2\text{-}MAX}$, and TTE. The VT (Fig. 1A, data indicated a significant two-way interaction ($p < 0.001$). The dependent-samples *t*-tests showed a 13.9% increase in VT from pre- to post-supplementation for the β-alanine treatment ($p < 0.001$), but no change for the placebo treatment ($p > 0.05$).

There was a significant two-way interaction ($p < 0.001$) for the PWC_{FT} values (Fig. 1B). Follow-up analyses indicated a 12.6% increase in PWC_{FT} power output from pre- to post-supplementation for the β-alanine treatment ($p < 0.001$), but no changes for the placebo treatment ($p > 0.05$).

There was no two-way interaction ($p > 0.05$) for $\dot{V}O_{2\text{-}MAX}$ (Fig. 1C), no main effect for group ($p > 0.05$), and no main effect for time ($p > 0.05$). Similar results were found for TTE (Fig. 1D), with no two-way interaction ($p > 0.05$), no main effect for treatment ($p > 0.05$), however significant change for time ($p < 0.05$) in the β-alanine group.

There was no significant ($p > 0.05$) difference between groups for their compliance rate (89.5% β-alanine group; 91.7% for the PL group). This represents a $5.0\,g \cdot d^{-1}$ intake for the β-alanine group. Analyses of the dietary recalls showed no significant differences between the β-alanine or PL groups in daily caloric intake (1903 ± 233 and 2118 ± 266 Kcal, respectively). Analysis of dietary composition showed that the daily diet for both groups was comprised of 52% carbohydrates, 31% fat, and 17% protein. No significant ($p > 0.05$) changes in body mass were observed from pre- to post-supplementation in either the β-alanine or PL groups (Table 1).

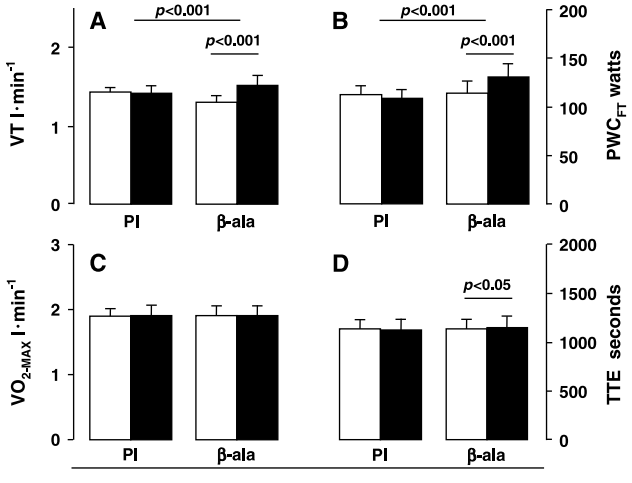

Fig. 1. **A** Ventilatory threshold (VT; $l \cdot min^{-1}$), **B** physical working capacity at fatigue threshold (PWC_{FT}; W), **C** maximal oxygen uptake ($\dot{V}O_{2\text{-}MAX}$; $l \cdot min^{-1}$), **D** time to exhaustion (TTE; s) for the β-Alanine and placebo treatments during the pre- (white) and post-supplementation (black) assessments. Columns are mean ± SE, and the lines represent individual responses. Significant changes with time and time x treatment are indicated

Discussion

Several recent investigations (Harris et al., 2006; Hill et al., 2006) using young men (25.4 ± 2.1 yrs) have shown that 28 days of β-alanine supplementation ($65\,mg \cdot kg^{-1} \cdot d^{-1}$) significantly elevated whole muscle carnosine levels by an average of 60%. The women in the present study were similar in age (Table 1) and consumed either β-alanine or PL with dosages similar to the study by Harris et al. (2006) (Table 2). However, while the total amount of β-alanine (140 g) was similar to the Hill et al. (2006) study (i.e., 146 g given over 28 days), the dosage relative to body mass for the women in this study was 24% higher ($86\,mg \cdot kg^{-1} \cdot d^{-1}$). Therefore, although muscle carnosine levels were not directly measured in the present study, the results based from previous investigations (Harris et al., 2006; Hill et al., 2006) suggest that the β-alanine supplementation increased muscle carnosine levels.

The findings of this study indicate that β-alanine supplementation delays the onset of NMF during incremental cycle ergometry in young women as indicated by increased PWC_{FT} (12.6%), VT (13.9%) and TTE (2.5%) (Fig. 1; Table 3). These results support previous work that demonstrated, a similar 14.5% increase in the PWC_{FT} following 28 days of β-alanine ($49\,mg \cdot kg^{-1} \cdot d^{-1}$) supplementation in untrained men (Stout et al., 2006). In addition, Zoeller et al. (2006) reported a significant increase in VT (7%), but no changes in $\dot{V}O_{2\text{-}MAX}$ or TTE after β-alanine supplementation ($49\,mg \cdot kg^{-1} \cdot d^{-1}$) over a similar supplementation period. Furthermore, Kim et al. (2006) reported significant increases in VT, TTE, and muscle carnosine in highly trained male cyclists after supplementing β-alanine ($65\,mg \cdot kg^{-1} \cdot d^{-1}$) and exercise training for twelve weeks. Neither Zoeller et al. (2006) nor Kim et al. (2006) reported changes in $\dot{V}O_{2\text{-}MAX}$, which is consistent with the present study. However, Kim et al. (2006) demonstrated increases in TTE, whereas Zoeller et al. (2006) no significant change. In agreement with Kim et al. (2006) the present findings showed a significant ($p < 0.05$) increase (2.5%) in TTE with β-alanine supplementation. The inconsistent findings regarding TTE are unclear, but may have been related to (a) the length of supplementation, (b) dosage, (c) concurrent exercise training, and/or (d) the training status of the participants. Future research is needed to determine the interactions among training status, concurrent exercise training, and β-alanine supplementation in women. Additional studies may also be needed to determine the optimal doses of β-alanine for men and women that will increase muscle carnosine concentrations and improve exercise performance. Currently, it would appear that a minimum effective dose of $65–86\,mg \cdot kg^{-1} \cdot d^{-1}$ is needed for performance enhancement in young men and women.

Recently, Hill et al. (2006) examined the effects of β-alanine supplementation ($65\,mg \cdot kg^{-1} \cdot d^{-1}$) on muscle carnosine levels in relation to the work completed during an exhaustive cycle ergometry about at 110% of estimated maximal power in men. The authors reported that supplementing with β-alanine significantly increased muscle carnosine (58.8%) which corresponded to a 12.4% increase in total work compared with no change in the placebo group. Harris et al. (2006), Hill et al. (2006), and Suzuki et al. (2002) have all suggested that increasing muscle carnosine (with β-alanine supplementation or exercise training) may increase the H^+ buffering capacity in skeletal muscle, which may also contribute to the improvement in exercise performance by delaying the onset of fatigue.

McClaren et al. (1989) have suggested that a decrease in muscle pH, as a result of H^+ accumulation, may be responsible for fatigue-induced increases in muscle activation and the corresponding increase in EMG amplitude. Taylor et al. (1997) also found that, for incremental cycle ergometry, the accumulation of plasma lactate was associated with an increase in EMG amplitude measured from the rectus femoris muscle. This evidence supports the hypothesis that increased blood lactate during exercise and the subsequent H^+ accumulation may trigger an increase in muscle activation (EMG amplitude) due to a decrease in pH. Furthermore, it has been suggested that a decrease in muscle and blood pH (via H^+ accumulation) during intense exercise may directly stimulate the pulmonary ventilation rate to remove CO_2 and/or indirectly stimulate the type III and IV afferent inputs to the respiratory control center in the medulla (Svedahl and MacIntosh, 2003; Dempsy et al., 1995).

In the present study, 28 days of β-alanine supplementation resulted in a significant increase in $PWC_{FT,}$ VT, and TTE which may have been due to an increase in muscle carnosine concentrations. It has been suggested that this mechanism may improve intramuscular H^+ buffering capacity (Harris et al., 1990, 2006; Hill et al., 2006). Harris et al. (2006) and Hill et al. (2006) have hypothesized that increasing muscle carnosine through β-alanine supplementation will help maintain the intramuscular pH environment during intense exercise by countering the accumulation of H^+. The results of the present study supported this hypothesis and suggested that β-alanine supplementation may delay the fatigue-induced increases in EMG amplitude and ventilation rate during incremental cycle ergometry at sub-maximal workloads, even without concurrent exercise training. In addition, β-alanine supplementation appears to improve TTE at maximal intensity, however, may not improve $\dot{V}O_{2\text{-}MAX}$ which is less dependent on improvements in buffering capacity. Future studies should include exercise training regimens in conjunction with β-alanine supplementation so that specific recommendations can be made for exercise prescription and β-alanine consumption.

In summary, our findings indicate that β-alanine supplementation ($86\,mg \cdot kg^{-1} \cdot d^{-1}$) for 28 days delays the onset of neuromuscular fatigue and VT, leading to increases in submaximal performance and TTE during cycle ergometry in young women. The delay in neuromuscular fatigue, VT and improvement in TTE is likely the result of elevated muscle carnosine levels, which provides a greater capacity to buffer H^+ during exercise. Although recommendations must await further clinical trials, these find-

ings may be useful for nutritionists, sports scientists, and athletes searching for alternative nutritional supplements that improve exercise performance. In addition, these findings may provide a foundation for future studies to examine dosage- and exercise training-related issues in conjunction with β-alanine supplementation to further elucidate the proposed mechanism of action involving muscle carnosine concentrations and improved H^+ buffering capacity within skeletal muscle in women.

Acknowledgements

The authors wish to thank Natural Alternatives International, San Marcos, USA, for donating the β-alanine (CarnoSyn™). We also wish to thank the subjects who volunteered for this study and Robert Curran and Dr. Joseph O'Kroy for assisting with the data collection.

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