Amino Acids (2012) 43:39–47
DOI 10.1007/s00726-012-1268-0

ORIGINAL ARTICLE

Effect of beta-alanine supplementation on repeated sprint performance during the Loughborough Intermittent Shuttle Test

Bryan Saunders · Craig Sale · Roger C. Harris · Caroline Sunderland

Received: 23 December 2011 / Accepted: 6 March 2012 / Published online: 21 March 2012

Abstract The aim of this study was to examine the effect of β-alanine supplementation on repeated sprint performance during an intermittent exercise protocol designed to replicate games play. Sixteen elite and twenty non-elite game players performed the Loughborough Intermittent Shuttle Test (LIST) on two separate occasions. Trials were separated by 4 weeks of supplementation with either β-alanine (BA) or maltodextrin (MD). There was no deterioration in sprint times from Set 1 to Set 6 of the LIST in either group prior to supplementation (elite: $P = 0.92$; non-elite: $P = 0.12$). Neither BA nor MD supplementation affected sprint times. Blood lactate concentrations were elevated during exercise in both groups, with no effect of supplementation. β-Alanine supplementation did not significantly improve sprint performance during the LIST. Neither group showed a performance decrement prior to supplementation, which might have masked any benefit from increased muscle buffering capacity due to β-alanine supplementation.

Keywords Beta-alanine · Carnosine · Intermittent exercise · Repeated sprint performance

Introduction

Team sports, such as football, hockey and rugby, are characterised by intermittent bouts of exercise involving low and high-intensity running. Match play requires players to continually reproduce maximal and near maximal sprints with short periods of recovery over an extended period of time, a fitness component termed repeated sprint ability (Dawson et al. 1997; Bishop et al. 2001). High-intensity efforts of this nature increase energy demand on the muscle, with the link between chemical energy and mechanical work being mediated by adenosine-5′-triphosphate (ATP). To meet the increased energy demand, ATP is hydrolysed to adenosine-5′-diphosphate (ADP), although the ATP store is limited and must be continually replenished. The aerobic rate of ATP resynthesis is quickly exceeded by the rate of ATP hydrolysis (14.9 ± 2.2 mmol kg^{-1}dm s^{-1}, Gaitanos et al. 1993), meaning that the shortfall in ATP must be met by the hydrolysis of PCr and anaerobic glycolysis (Hultman and Sjöholm 1983). When the glycolytic rate in muscle is higher than the rate of pyruvate oxidation, lactic acid is produced to facilitate the continuation of muscle contraction but this causes acidification following dissociation to the lactate anion (Lac^-) and hydrogen cation (H^+).

Reduced intracellular pH (pHi) interferes with several metabolic processes; explaining a reduction in force production and the onset of fatigue (Spriet et al. 1989). As a result, a decline in repeated sprint performance might be attenuated in those individuals able to buffer the reduced pHi more effectively. Increased repeated sprint ability has been claimed to be associated with a greater H^+ buffering capacity in elite female hockey players (Bishop et al. 2003), recreational team sport females (Bishop and Edge 2006), untrained females (Bishop et al. 2004) and professional and amateur male footballers (Rampinini et al. 2009), although this is based on the titration of muscle homogenates. An intervention designed to increase intracellular buffering capacity may thus be of benefit to repeated sprint ability and team sport performance.

B. Saunders · C. Sale · C. Sunderland (✉)
Sport, Health and Performance Enhancement (SHAPE) Research Group, School of Science and Technology, Nottingham Trent University, Clifton Lane, Nottingham NG11 8NS, UK
e-mail: caroline.sunderland@ntu.ac.uk

R. C. Harris
Junipa Ltd, Newmarket, Suffolk CB8 8HD, UK

Carnosine (β-alanyl-L-histidine) is a naturally occurring dipeptide found in high concentrations in skeletal muscle (Harris et al. 2006; Hill et al. 2007), and due to its pKa (6.83), it is a suitable buffer over the exercise pHi transit-range (Bate-Smith 1938). β-alanine supplementation has been shown to be effective in increasing muscle carnosine levels (Harris et al. 2006; Hill et al. 2007), thereby increasing muscle buffering capacity, with the potential to improve exercise performance and capacity that is limited by the accumulation of H^+ (Hill et al. 2007; Sale et al. 2011). In a meta-analysis of the literature, Hobson et al. (In press) showed that β-alanine supplementation was effective in improving high-intensity exercise of durations between 60 and 240 s ($P = 0.001$) and in excess of 240 s ($P = 0.05$), but not <60 s ($P = 0.3$). A timeframe between 60 and 240 s is a period when anaerobic energy sources can contribute between 20 and 60 % of the total energy requirement (Maughan et al. 1997), resulting in a large accumulation of H^+. It has been suggested that an exercise duration of <60 s may not be sufficient to induce reductions in pHi that will limit exercise (Sale et al. 2010), although repeated short duration exercise may increase the sensitivity to reduced pHi (Katz et al. 1984).

Previous studies have not shown a significant effect of β-alanine supplementation on repeated sprint performance (Hoffman et al. 2008; Sweeney et al. 2010). However, these studies did not determine repeated sprint performance during simulated or actual games play and, thus, did not consider the implications of the additional metabolic demand of the entire activity. Therefore, the aim of this investigation was to examine the effects of 4 weeks β-alanine supplementation on multiple sprint performance during the Loughborough Intermittent Shuttle Test (LIST) (Nicholas et al. 2000). Furthermore, to determine any differences of β-alanine supplementation on game players of varying standard, both elite and non-elite game players were recruited to the study, since reports have suggested improved buffering capacities in trained compared with recreational athletes (Sahlin and Henriksson 1984; Parkhouse et al. 1985; Edge et al. 2006). We hypothesised that β-alanine supplementation would result in an improvement in sprint performance during the LIST.

Materials and method

Participants

Twenty elite and twenty non-elite male game players volunteered for the study and were split into β-alanine and placebo groups, matched for estimated VO_{2max}. Four elite players withdrew from the study due to injury, meaning that 16 elite players were included in the final data set (Table 1). The elite population consisted of national hockey players, all of whom had represented their country at U18, U21 or full international level. The non-elite population were recreationally active individuals who engaged in team sports (football and hockey) 1–2 times per week. A health screening procedure was repeated prior to each laboratory visit to ensure that the health status of the participants had not changed. Participants had not taken any supplement in the 3 months prior to the study and had not taken β-alanine for at least 6 months. None of the participants were vegetarian and, therefore, would have encountered small amounts of β-alanine in their diet from the hydrolysis of carnosine and its methyl derivatives in meat. All participants were requested to maintain similar levels of physical activity and dietary intake for the duration of the study, and the compliance with this request was verbally confirmed with participants prior to commencement of the post-supplementation trial. The study was approved by the institution's Ethical Advisory Committee.

Experimental design

Participants attended the laboratory on four separate occasions. The first two visits comprised of the multistage fitness test, a progressive shuttle run to volitional exhaustion, and a habituation of the LIST. The remaining two sessions were main trials comprising of the LIST, each trial separated by 4 weeks of supplementation. Participants maintained a food diary in the 24-h period before the first main trial, and this was subsequently used to replicate the diet prior to the second main trial.

Participants were supplemented with either 6.4 g day^{-1} of β-alanine (CarnoSyn^{TM}, NAI, USA) or placebo

Table 1 Physical characteristics of the participants (mean ± 1SD)

	Elite		Non-elite	
	Placebo ($N = 8$)	β-Alanine ($N = 8$)	Placebo ($N = 10$)	β-Alanine ($N = 10$)
Age (years)	19 ± 2	20 ± 1	22 ± 3	22 ± 2
Height (m)	1.77 ± 0.05	1.80 ± 0.06	1.81 ± 0.07	1.79 ± 0.08
Body mass (kg)	72.1 ± 7.1	75.0 ± 11.0	84.9 ± 10.9	81.0 ± 11.5
Estimated VO_{2max} (ml kg min^{-1})	59.4 ± 2.6	58.6 ± 2.4	50.7 ± 5.0	50.5 ± 4.4
Compliance (%)	94 ± 5	87 ± 10	96 ± 4	96 ± 6

(maltodextrin; NAI, USA) in tablet form over a 4-week period. Participants were split into the β-alanine or placebo group matched for estimated VO_{2max} (Table 1). The dosing regimen consisted of two 800 mg β-alanine or placebo tablets ingested four times per day at 3–4 h intervals. The compliance of supplementation was monitored using supplementation logs, with a high degree of compliance being reported in all groups (Table 1). There were no reports of symptoms of paraesthesia from any of the participants in either group. All supplements were tested by HFL Sports Science prior to use to ensure no contamination with steroids or stimulants according to ISO 17025 accredited tests.

Experimental protocols

Preliminary testing

Height and body mass (Seca, UK) were recorded upon arrival to the first session, before participants performed a progressive shuttle run test to exhaustion (Ramsbottom et al. 1988). A 5 min standardised warm-up was performed, consisting of light jogging and running, followed by 5 min of self-selected stretching. Participants were then required to run between markers set 20 m apart at increasing speeds dictated by an audio signal. The test was ended if the participant failed to reach the designated line within the given time frame on two consecutive occasions or at volitional exhaustion. The final level attained by the participant was used to estimate maximal oxygen uptake (Ramsbottom et al. 1988).

All participants performed a habituation trial to avoid any learning effects during the main trials. A 5 min standardised warm-up was performed, consisting of light jogging and running, followed by 5 min of self-selected stretching. Participants then performed two sets of the LIST to familiarise themselves with the protocol.

Main trials

All tests were conducted in a sports hall with an ambient temperature of 18.8 ± 0.9 °C and relative humidity of 42.8 ± 4.7 %. Participants performed a warm-up as described for the habituation, before performing the LIST (Nicholas et al. 2000). The LIST requires participants to run between markers set 20 m apart at varying speeds dictated by an audio signal. The test consisted of six exercise sets approximately 15 min long separated by periods of 3 min rest (Fig. 1). Within each set was an exercise pattern (Fig. 1) repeated 11 times, incorporating walking, sprinting (over 15 m), recovery, cruising and jogging; cruising and jogging were defined as 95 and 55 % of an individual's estimated VO_{2max}. These corresponding running speeds were calculated using the tables for predicted VO_{2max} values (Ramsbottom et al. 1988). Nicholas et al. (2000) determined the reliability of the LIST, with

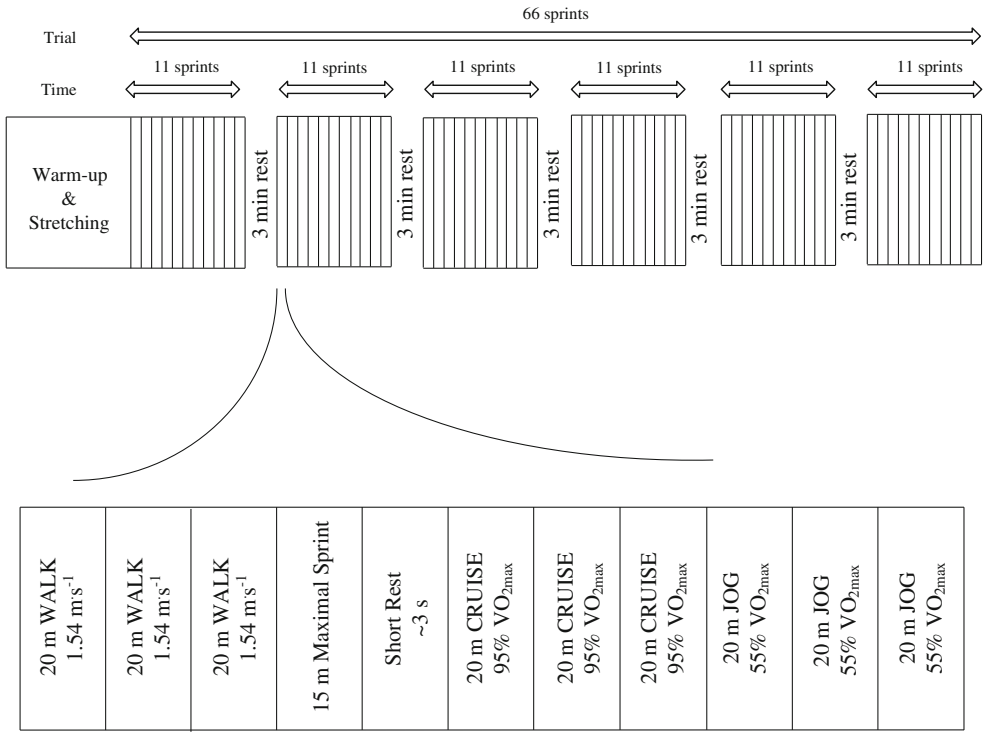

Fig. 1 The Loughborough Intermittent Shuttle Test

similar average times for 15 m sprints during two trials separated by 1 week (2.42 ± 0.04 s and 2.43 ± 0.04 s). The 95 % limits of agreement for sprint times were −0.14 to 0.12 s.

Individual sprint times over 15 m were recorded for each of the 11 sprints per set (Brower Timing Systems IRD-T173, Utah, USA), with a total of 66 sprints completed during the LIST (Fig. 1). Sprint times were averaged every set to allow the analysis of sprint performance throughout the LIST (time), and over the entire trial to allow comparisons between pre- and post-supplementation (trial). Heart rate was recorded every 5 s throughout exercise (Polar, Polar Electro Oy, Finland) and participants indicated their overall ratings of perceived exertion (RPE) during the last walking stage of each set on a 14 point scale (Morgan and Borg 1976). Fingerprick blood samples were taken during the 3 min rest periods between sets, immediately following the final sprint, and analysed for blood lactate concentration (Lactate Pro, Arkray, Japan). Pre- and post-exercise body mass was measured (Seca, UK). Subjects were allowed to drink water ad libitum throughout; total fluid ingested during the exercise protocol was recorded.

Statistical analysis

All data were analysed using Statistica 9 (Statsoft, USA) and are presented as mean ± 1SD for 8 participants in the two elite supplementation groups and 10 participants in the non-elite groups except for heart rate data, which are presented for 8 participants in both groups due to heart rate monitor malfunction. Sprint data were filtered every set to remove any non-maximal sprint times; any sprint time more than two SD outside of the mean of the corresponding set were removed from the data. *P* plots and Cochran's *Q* were used to confirm normality and homogeneity of variance of the data. A three-way factorial ANOVA (supplement × trial × time) was used to determine any difference in sprint times, blood lactate, heart rate and RPE. Fisher LSD tests were used for post hoc analyses where appropriate and statistical significance was accepted at the $P \leq 0.05$ level.

Results

Elites

Sprint times

There was no effect of supplementation on sprint performance across each set of the LIST (supplement × trial × time, $P = 0.99$), or when taking into consideration all sprints (supplement × trial, $P = 0.63$). There was no main effect of time ($P = 0.92$) indicating that sprint times did not change significantly as the number of sprints performed increased (Fig. 2).

Measurements

Blood lactate concentration and RPE were increased over time ($P \leq 0.001$), but not heart rate ($P = 0.76$) (Table 2),

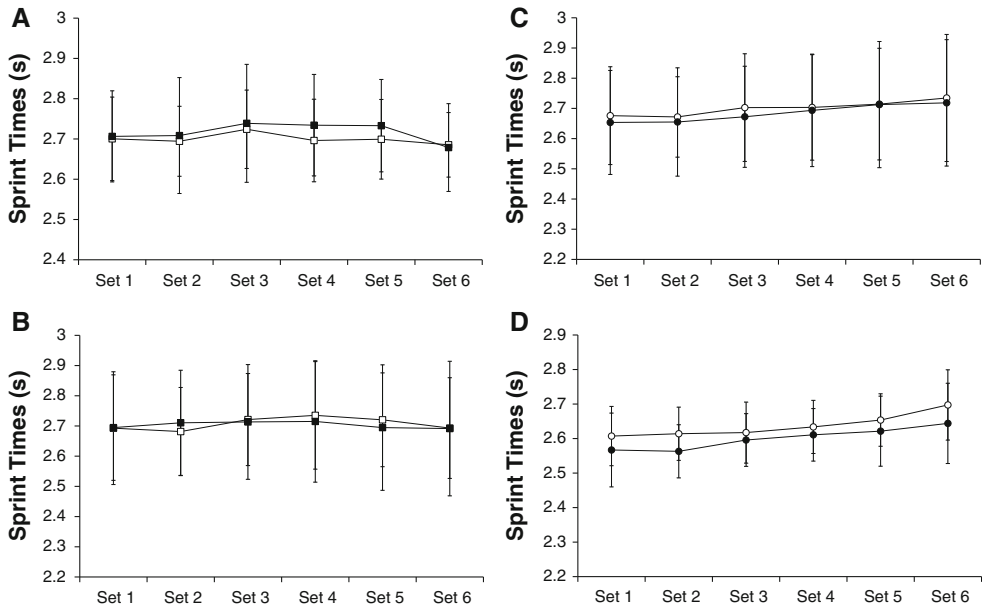

Fig. 2 Panels **a** and **b** display sprint times during the LIST for elite participants in the placebo (**a**) and β-alanine (**b**) groups both pre- (*white*) and post (*black*)-supplementation. Panels **c** and **d** display sprint times during the LIST for non-elite participants in the placebo (**c**) and β-alanine (**d**) groups both pre- (*white*) and post (*black*)-supplementation

Table 2 Blood lactate, heart rate and RPE during the LIST for elite and non-elite participants (mean ± 1SD)

	Elite							Non-elite						
	Baseline	Set 1	Set 2	Set 3	Set 4	Set 5	Set 6	Baseline	Set 1	Set 2	Set 3	Set 4	Set 5	Set 6
Lactate (mmol L^{-1})														
Pre-MD	1.4 ± 0.5	2.7 ± 0.9*	2.5 ± 0.7*	2.7 ± 1.1*	2.7 ± 0.8*	3.2 ± 1.1*	2.9 ± 0.9*	1.8 ± 0.4	5.8 ± 2.2*	7.4 ± 3.4*	5.9 ± 2.6*	6.2 ± 2.7*	6.6 ± 2.7*	7.0 ± 3.0*
Post-MD	2.0 ± 1.0	3.0 ± 1.4	2.7 ± 1.0	3.0 ± 1.5	2.9 ± 1.2	2.8 ± 1.0	3.6 ± 1.3*	1.5 ± 0.4	5.3 ± 2.3*	6.1 ± 2.6*	6.1 ± 2.8*	5.8 ± 2.7*	6.2 ± 3.5*	6.2 ± 3.4*
ΔPre–post-MD	0.6 ± 1.3	0.3 ± 1.8	0.2 ± 0.8	0.3 ± 1.3	0.2 ± 1.0	−0.4 ± 1.9	0.7 ± 2.1	−0.3 ± 0.5	−0.5 ± 1.9	−1.3 ± 2.7	0.1 ± 1.5	−0.4 ± 2.2	−0.4 ± 3.1	−0.8 ± 3.2
Pre-BA	1.6 ± 0.7	3.3 ± 1.6*	2.7 ± 1.3	2.9 ± 1.7*	3.7 ± 1.3*	2.9 ± 0.7*	3.5 ± 1.5*	1.6 ± 0.6	6.2 ± 1.7*	5.7 ± 2.2*	5.1 ± 2.1*	5.8 ± 3.0*	4.5 ± 1.8*	4.9 ± 2.0*
Post-BA	1.4 ± 0.5	2.9 ± 1.1*	3.2 ± 1.6*	4.0 ± 1.7*	2.6 ± 0.9*	2.1 ± 0.7	2.5 ± 0.9	1.6 ± 0.5	6.1 ± 2.7*	5.3 ± 3.8*	4.5 ± 3.0*	4.5 ± 2.1*	3.9 ± 2.1*	4.5 ± 2.6*
ΔPre–post-BA	−0.2 ± 1.0	−0.3 ± 1.7	0.5 ± 2.0	1.1 ± 2.3	−1.1 ± 1.6	−0.8 ± 0.8	−1.0 ± 0.9	−0.1 ± 0.5	−0.2 ± 2.3	−0.4 ± 2.1	−0.6 ± 2.9	−1.3 ± 2.9	−0.6 ± 2.7	−0.4 ± 1.6
Heart rate (beats min^{-1})														
Pre-MD	–	159 ± 8	163 ± 10	162 ± 11	161 ± 10	159 ± 8	159 ± 7	–	161 ± 11	166 ± 10	168 ± 9	170 ± 10	170 ± 10	170 ± 10
Post-MD	–	156 ± 10	160 ± 12	160 ± 13	158 ± 12	158 ± 13	160 ± 14	–	161 ± 11	166 ± 12	168 ± 9	168 ± 10	167 ± 10	169 ± 10
ΔPre–post-MD		−4 ± 8	−3 ± 6	−2 ± 7	−3 ± 9	−1 ± 9	1 ± 10		0 ± 4	0 ± 5	0 ± 5	−2 ± 5	−2 ± 5	−1 ± 6
Pre-BA	–	155 ± 5	159 ± 6	157 ± 5	156 ± 5	157 ± 5	160 ± 5	–	167 ± 12	171 ± 13	172 ± 12	172 ± 13	171 ± 14	170 ± 10
Post-BA	–	152 ± 10	156 ± 11	155 ± 10	154 ± 10	154 ± 10	154 ± 10	–	161 ± 11	166 ± 12	166 ± 12	166 ± 11	167 ± 12	167 ± 10
ΔPre–post-BA		−2 ± 8	−2 ± 6	−1 ± 7	−2 ± 7	−3 ± 7	−4 ± 8		−6 ± 6	−5 ± 7	−6 ± 7	−6 ± 6	−5 ± 7	−3 ± 5
RPE														
Pre-MD	–	11 ± 2	12 ± 1	14 ± 2^	15 ± 1^	16 ± 1^	17 ± 1^	–	14 ± 2	15 ± 1	16 ± 2^	17 ± 2^	18 ± 1^	19 ± 1^
Post-MD	–	10 ± 1	13 ± 1^	14 ± 2^	14 ± 2^	15 ± 2^	16 ± 2^	–	14 ± 2	15 ± 2	16 ± 2	16 ± 2^	17 ± 2^	18 ± 2^
ΔPre–post-MD		−1 ± 1	0 ± 1	−1 ± 2	−1 ± 1	−1 ± 2	−1 ± 2		0 ± 2	0 ± 1	−1 ± 1	−1 ± 2	−1 ± 2	−1 ± 2
Pre-BA	–	11 ± 2	12 ± 1	13 ± 1^	14 ± 1^	15 ± 1^	15 ± 1^	–	13 ± 1	14 ± 2^	15 ± 1^	16 ± 2^	16 ± 2^	18 ± 2^
Post-BA	–	11 ± 1	12 ± 2	13 ± 2	13 ± 3^	14 ± 2^	15 ± 2^	–	12 ± 2	13 ± 2	15 ± 2^	15 ± 2^	17 ± 2^	17 ± 2^
ΔPre–post-BA		0 ± 2	−1 ± 2	0 ± 2	−1 ± 3	0 ± 2	0 ± 2		0 ± 2	−1 ± 2	−1 ± 1	−1 ± 1	0 ± 2	0 ± 2

Pre-MD pre-supplementation in the maltodextrin group, *Post-MD* post-supplementation in the maltodextrin group, *Pre-BA* pre-supplementation in the β-alanine group, *Post-BA* post-supplementation in the β-alanine group, Δ*Pre–post* within individual change from pre- to post-supplementation at the corresponding time-point

* $P \leq 0.05$ from baseline; ^ $P \leq 0.05$ from Set 1

with no effect of supplementation on any of these variables. Body mass, as a percentage of pre-exercise body mass, was well maintained following exercise in both supplementation groups: pre (placebo: -0.8 ± 0.8 %, β-alanine: -0.6 ± 0.8 %) and post-supplementation (placebo: -0.9 ± 0.8 %, β-alanine: -0.3 ± 0.5 %) with no significant differences between trials ($P = 0.49$) or groups ($P = 0.37$).

Non-elites

Sprint times

There was no effect of supplementation on sprint times across each set of the LIST (supplement × trial × time, $P = 0.99$) or when taking into consideration all sprints (supplementation × trial, $P = 0.58$). There was no main effect of time in the non-elites ($P = 0.12$) indicating that sprint times did not become significantly slower as the number of sprints performed increased (Fig. 2).

Measurements

Lactate was increased from baseline following every set of the LIST ($P \leq 0.001$) (Table 2) in both groups prior to supplementation, with no effect of supplementation. Heart rate remained stable over the duration of the LIST with no effect of supplementation ($P = 0.19$) (Table 2). There was a time effect on RPE ($P \leq 0.001$), increasing throughout the LIST, although there was no effect of supplementation in either group. The level of dehydration was well controlled in both supplementation groups: pre (placebo: -1.0 ± 0.4 %, β-alanine: -1.0 ± 0.6 %) and post-supplementation (placebo: -1.3 ± 0.5 %, β-alanine: -1.2 ± 0.6 %) with no significant differences between trials ($P = 0.06$) or groups ($P = 0.8$).

Discussion

This is the first study to investigate the effects of β-alanine supplementation on repeated sprint performance during prolonged intermittent activity simulating team sport game play. Contrary to our hypothesis, β-alanine did not have an effect on sprint performance during the LIST. The lack of a deterioration in sprint times during the LIST, in either group prior to supplementation might, however, have masked any effects of an increase in muscle buffering capacity brought about by elevated muscle carnosine content. Indeed, Balsom et al. (1992) showed that 15 m sprint performance could be maintained over forty exercise bouts separated by 30 s rest, suggesting that changes in the intracellular environment for this type of exercise were not sufficient to induce fatigue. An increase in blood lactate concentration following the second sprint was, however, reported by Balsom et al. (1992) suggesting that anaerobic glycolysis contributed to every sprint. In the present study, participants completed a total of 66 sprints, albeit with a different recovery profile than Balsom et al. (1992), in addition to the increased demand of the additional intermittent activity between sprints. For this reason we hypothesised that a greater decrement in performance would occur during our protocol as the result of H^+ accumulation and that, as a result, β-alanine supplementation would attenuate any decline in sprint performance. However, no significant decrement in sprint performance was observed in any group and the blood lactate values in the current investigation are similar to those reported by Balsom et al. (1992). They are also lower (3–6 mmol L^{-1}) than previous LIST studies (>6 mmol L^{-1}; Nicholas et al. 2000) and repeated sprint activity studies that have shown a correlation to H^+ buffering capacity (>8 mmol L^{-1}; Bishop et al. 2003, 2004). As such, we suggest that the intensity of the LIST and the duration of the sprints and high-intensity running may not have been sufficient to induce reductions in performance due to a reduced pHi, which would then subsequently have been influenced by β-alanine supplementation.

In contrast to the present study, other studies using the LIST have shown a deterioration in sprint performance as the number of sprints performed increased in trained (Sunderland and Nevill 2005) and youth (Phillips et al. 2010) game players. The decline in sprint times in those studies (>0.09 s) are higher than the maximum shown by the elites (+0.02) and non-elites (+0.09), which may explain some of the differences. McGregor et al. (1999) showed semi-professional footballers could maintain sprint performance throughout the LIST when fluid was administered, although sprint times worsened when fluid was restricted in the same population. In the present study, the elite population demonstrated inconsistent sprint times across the test including the ability to improve performance in the final set. Sprint times in the non-elites showed a more consistent, but still non-significant, decline in sprint times across the LIST, and, as such, may be a truer reflection of their inclination to perform each sprint maximally. The elite athletes may have adopted a pacing strategy to delay fatigue and optimise performance, thereby subconsciously affecting the performance outcome of the study.

The ability to perform repeated sprints has been associated with H^+ buffering capacity (Bishop et al. 2003, 2004; Bishop and Edge 2006; Rampinini et al. 2009) as a large accumulation in intramuscular H^+ can negatively impact upon muscle function. Disruption of PCr recovery (Harris et al. 1976), inhibited glycolysis (Trivedi and

Daniforth 1984) and disruption of the muscle contractile machinery (Donaldson and Hermansen 1978; Fabiato and Fabiato 1978) have all been reported with reduced pHi, as well as an increased perception of effort during high-intensity intermittent exercise (Price and Moss 2007). Male and female game players of a high standard have increased H^+ buffering capacity compared to game players of a lower standard (Edge et al. 2006; Rampinini et al. 2009) and untrained females (Edge et al. 2006). In addition, higher levels of muscle carnosine have been shown in runners, rowers (Parkhouse et al. 1985) and bodybuilders (Tallon et al. 2005) than in their endurance trained or untrained counterparts. Although muscle carnosine concentrations were not directly determined in this study, it can be hypothesised that baseline carnosine concentration was higher in the elite versus the non-elite population. Nonetheless, β-alanine supplementation has been shown to significantly increase muscle carnosine concentrations in trained sprinters (Derave et al. 2007), with the dose of β-alanine used in the present study being higher than that employed by Derave et al. (2007). Whilst the fact that we did not determine muscle carnosine concentrations means that we cannot confirm a direct effect of β-alanine supplementation on muscle carnosine in either group, supplementation has consistently been shown to increase muscle carnosine, with all individuals showing a response to supplementation (for a review see Sale et al. 2010). Therefore, a lack of response to β-alanine supplementation in terms of elevated muscle carnosine concentrations is unlikely to explain the lack of an effect on sprint performance in this study.

Suzuki et al. (2002) showed a positive correlation between muscle carnosine content and power output during the last two 5 s periods of a 30 s maximal cycling bout, although the area occupied by type II muscle fibres is likely to have been of more importance in their study. Indeed, Hill (2007) showed no effect of β-alanine supplementation on mean power output, peak power output or fatigue index during three repeated 30 s maximal sprint cycles. It has been suggested that muscle buffering capacity does not affect performance during exercise <60 s in duration (Bogdanis et al. 1998) and would therefore be unaffected by increased levels of muscle carnosine brought about by β-alanine supplementation; a suggestion supported by a recent meta-analysis of the literature (Hobson et al., In Press). Nonetheless, an increased repeated sprint ability, consisting of repeated 6 s maximal bouts every 30 s, has been shown to be positively correlated to H^+ buffering capacity (Bishop et al. 2003, 2004), which suggests that repeated short duration exercise bouts could well be affected by increased buffering capacity. Indeed, several studies have shown an ergogenic effect of pre-exercise alkalosis using sodium bicarbonate supplementation on repeated high-intensity bouts (Price et al. 2003; Bishop and Claudius 2005; Artioli et al. 2007).

Previous studies have examined the effect of β-alanine supplementation on isolated repeated sprint performance (Hoffman et al. 2008; Sweeney et al. 2010), although not as part of simulated game activity as in the present study. Hoffman et al. (2008) showed no effect of β-alanine supplementation on repeated 200 yard line drills in collegiate football players; although they only compared differences between groups, since no baseline measurements were taken. Similarly, Sweeney et al. (2010) showed no effect of β-alanine supplementation on two sets of repeated 5 × 5 s sprints, although the short recovery period (45 s) between sprints which may not have been enough to restore PCr to initial levels (Bogdanis et al. 1996), and may have contributed more to fatigue than reduced pHi. Due to the increased period between sprints and additional metabolic demand of the active recovery, it was hypothesised that the LIST would be a more suitable repeated sprint protocol, likely to be affected by large accumulations of H^+. Furthermore, the ecological validity of the LIST (Nicholas et al. 2000) makes it a more suitable protocol to investigate the effect of β-alanine supplementation on team sport performance. Similar to previous studies, however, β-alanine supplementation did not have an effect on performance lasting <60 s in duration.

In conclusion, the ingestion of 6.4 g day^{-1} β-alanine over 4 weeks did not improve repeated sprint performance during the LIST. The lack of a significant finding may be due to the lack of deterioration in performance, in both groups, prior to supplementation, which might have masked any effect of increased muscle carnosine content. Future investigation should incorporate Part B of the LIST (Nicholas et al. 2000), performed following five sets of the LIST, and intended to exhaust participants within 10 min. Alternating shuttles at speeds corresponding to 55 and 95 % VO_{2max} are performed continuously until participants fail to reach the line on two consecutive occasions at the higher speed level. A capacity test of this sort may be more sensitive to changes in muscle buffering capacity than the performance measures in the main part of the LIST (Hobson et al., In press).

Acknowledgments The authors would like to thank National Alternatives International, San Marcos, California for providing the β-alanine (Carnosyn™) and Maltodextrin supplements. The results of the present investigation do not constitute endorsement by Amino Acids.

Conflict of interest We declare that we received β-alanine and maltodextrin supplies from NAI to undertake this study, though no additional funding was provided. Roger Harris is an independent paid consultant of NAI, is named as an inventor on patents held by NAI, and is in receipt of other research grants awarded by NAI.

References

Artioli GG, Gualano B, Coelho DF, Benatti FB, Gailey AW, Lancha AH Jr (2007) Does sodium-bicarbonate ingestion improve simulated judo performance. Int J Sport Nutr Exerc Metab 17:206–217

Balsom PD, Seger JY, Sjödin B, Ekblom B (1992) Physiological responses to maximal intensity intermittent exercise. Eur J Appl Physiol 65:144–149

Bate-Smith EC (1938) The buffering of muscle in rigour: protein, phosphate and carnosine. J Physiol 92:336–343

Bishop D, Claudius B (2005) Effects of induced metabolic alkalosis on prolonged intermittent-sprint performance. Med Sci Sport Exerc 37:759–767

Bishop D, Edge J (2006) Determinants of repeated-sprint ability in females matched for single-sprint performance. Eur J Appl Physiol 97:373–379

Bishop D, Spencer M, Duffield R, Lawrence S (2001) The validity of a repeated sprint ability test. J Sci Med Sport 4:19–29

Bishop D, Lawrence S, Spencer M (2003) Predictors of repeated sprint ability in elite female hockey players. J Sci Med Sport 6:199–209

Bishop D, Edge J, Goodman C (2004) Muscle buffer capacity and aerobic fitness are associated with repeated-sprint ability in women. Eur J Appl Physiol 92:540–547

Bogdanis G, Nevill M, Boobis L, Lakomy H (1996) Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. J Appl Physiol 80:876–884

Bogdanis GC, Nevill ME, Lakomy HKA, Boobis LH (1998) 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

Dawson B, Goodman C, Lawrence S, Preen D, Polglaze T, Fitzsimons M, Fournier P (1997) Muscle phosphocreatine repletion following single and repeated short sprint efforts. Scand J Med Sci Sport 7:206–213

Derave W, Ozdemir MS, Harris RC, Pottier A, Reyngoudt H, Koppo K, Wise JA, Achten E (2007) β-Alanine supplementation augments muscle carnosine content and attenuates fatigue during repeated isokinetic contraction bouts in trained sprinters. J Appl Physiol 103:1736–1743

Donaldson SKB, Hermansen L (1978) Differential direct effects of H^+ and Ca^{2+}-activated force of skinned fibres from the soleus, cardiac, adductor magnus muscle of rabbits. Eur J Physiol 376:55–65

Edge J, Bishop D, Hill-Haas S, Dawson B, Goodman C (2006) Comparison of muscle buffer capacity and repeated sprint ability of untrained, endurance-trained and team-sport athletes. Eur J Appl Physiol 96:225–234

Fabiato A, Fabiato F (1978) Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from cardiac and skeletal muscles. J Physiol 276:233–235

Gaitanos GC, Williams C, Boobis LH, Brooks S (1993) Human muscle metabolism during intermittent maximal exercise. J Appl Physiol 75:712–719

Harris RC, Edwards RHT, Hultman E, Nordesjo LO, Nylind B (1976) The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. Eur J Physiol 367:137–142

Harris RC, Tallon MJ, Dunnett M, Boobis LH, Coakley J, Kim HJ, Fallowfield JL, Hill CA, Sale C, Wise JA (2006) The absorption of orally supplied β-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. Amino Acids 30:279–289

Hill CA (2007) β-Alanine supplementation and high-intensity exercise. Unpublished doctoral thesis, University of Southampton

Hill CA, Harris RC, Kim HJ, Harris BD, Sale C, Boobis LH, Kim CK, Wise JA (2007) Influence of β-alanine supplementation on skeletal muscle carnosine concentrations and high intensity cycling capacity. Amino Acids 32:225–233

Hobson RM, Saunders B, Ball G, Harris RC, Sale C. Effects of β-alanine supplementation on exercise performance: a review by meta-analysis. Amino Acids (In Press)

Hoffman JR, Ratamess NA, Faigenbaum AD, Ross R, Kang J, Stout JR, Wise JA (2008) Short duration β-alanine supplementation increases training volume and reduces subjective feelings of fatigue in college football players. Nutr Res 28:31–35

Hultman E, Sjöholm H (1983) Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. J Physiol 345:525–532

Katz A, Costill DL, King DS, Hargreaves M, Fink WJ (1984) Maximal exercise tolerance after induced alkalosis. Int J Sports Med 5:107–110

Maughan RJ, Gleeson M, Greenhaff PL (1997) Biochemistry of exercise training. Oxford University Press, Oxford

McGregor SJ, Nicholas CW, Lakomy HKA, Williams C (1999) The influence of intermittent high-intensity shuttle running and fluid ingestion on the performance of a soccer skill. J Sport Sci 17:895–903

Nicholas CW, Nuttall FE, Williams C (2000) The Loughborough Intermittent Shuttle Test: a field test that simulates the activity pattern of soccer. J Sport Sci 18:97–104

Parkhouse WS, McKenzie DC, Hochachka PW, Ovalle WK (1985) Buffering capacity of deproteinized human vastus lateralis muscle. J Appl Physiol 58:14–17

Phillips SM, Turner AP, Gray S, Sandersonm MF, Sproule J (2010) Ingesting a 6 % carbohydrate-electrolyte solution improves endurance capacity, but not sprint performance, during intermittent, high-intensity shuttle running in adolescent team games players aged 12–14 years. Eur J Appl Physiol 109:811–821

Price M, Moss P (2007) The effects of work:rest duration on physiological and perceptual responses during intermittent exercise and performance. J Sport Sci 25:1613–1621

Price MJ, Moss P, Rance S (2003) Effects of sodium bicarbonate ingestion on prolonged intermittent exercise. Med Sci Sport Exerc 35:1303–1308

Rampinini E, Sassi A, Morelli A, Mazzoni S, Fanchini M, Coutts AJ (2009) Repeated-sprint ability in professional and amateur soccer players. Appl Physiol Nutr Metab 34:1048–1054

Ramsbottom R, Brewer J, Williams C (1988) A progressive shuttle run test to estimate maximal oxygen uptake. Brit J Sport Med 22:141–144

Sahlin K, Henriksson J (1984) Buffer capacity and lactate accumulation in skeletal muscle of trained and untrained men. Acta Physiol Scand 122:331–339

Sale C, Saunders B, Harris RC (2010) Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. Amino Acids 39:321–333

Sale C, Saunders B, Hudson S, Wise JA, Harris RC, Sunderland CD (2011) Effect of beta-alanine plus sodium bicarbonate on high-intensity cycling capacity. Med Sci Sport Exerc 43:1972–1978

Spriet LL, Lindinger MI, McKelvie RS, Heigenhauser GJF, Jones NL (1989) Muscle glycogenolysis and H^+ concentration during maximal intermittent cycling. J Appl Physiol 66:8–13

Sunderland C, Nevill ME (2005) High-intensity intermittent running and field hockey skill performance in the heat. J Sport Sci 23:531–540

Suzuki Y, Ito O, Mukai N, Takahashi H, Takamatsu K (2002) High levels of skeletal muscle carnosine contributes to the latter half of exercise performance during maximal cycle ergometer sprinting. Jap J Physiol 52:199–205

Sweeney KM, Wright GA, Brice AG, Doberstein ST (2010) The effects of β-alanine supplementation on power performance during repeated sprint activity. J Strength Cond Res 24:79–87

Tallon MJ, Harris RC, Boobis LH, Fallowfield JL, Wise JA (2005) The carnosine content of vastus lateralis is elevated in resistance-trained bodybuilders. J Strength Cond Res 19:725–729

Trivedi B, Daniforth WH (1984) Effect of pH on the kinetics of frog muscle phosphofructokinase. J Biol Chem 241:4110–4112