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ORIGINAL RESEARCH

Beta-Alanine Supplementation Improves Jumping Power and Affects Severe-Intensity Performance in Professional Alpine Skiers

Micah Gross, Kathrin Bieri, Hans Hoppeler, Barbara Norman, and Michael Vogt

Introduction: Supplementation with beta-alanine may have positive effects on severe-intensity, intermittent, and isometric strength-endurance performance. These could be advantageous for competitive alpine skiers, whose races last 45 to 150 s, require metabolic power above the aerobic maximum, and involve isometric muscle work. Further, beta-alanine supplementation affects the muscle force-frequency relationship, which could influence explosiveness. We explored the effects of beta-alanine on explosive jump performance, severe exercise energy metabolism, and severe-intensity ski-like performance. ***Methods***: Nine male elite alpine skiers consumed 4.8 g/d beta-alanine or placebo for 5 weeks in a double-blind fashion. Before and after, they performed countermovement jumps (CMJ), a 90-s cycling bout at 110% VO_2max (CLT), and a maximal 90-s box jump test (BJ90). ***Results***: Beta-alanine improved maximal (+7 ± 3%, $d = 0.9$) and mean CMJ power (+7 ± 2%, $d = 0.7$), tended to reduce oxygen deficit (-3 ± 8%, $p = .06$) and lactate accumulation (-12 ± 31%) and enhance aerobic energy contribution (+1.3 ± 2.9%, $p = .07$) in the CLT, and improved performance in the last third of BJ90 (+7 ± 4%, $p = .02$). These effects were not observed with placebo. ***Conclusions***: Beta-alanine supplementation improved explosive and repeated jump performance in elite alpine skiers. Enhanced muscle contractility could possibly explain improved explosive and repeated jump performance. Increased aerobic energy production could possibly help explain repeated jump performance as well.

Keywords: carnosine, skiing, box-jump test, buffering capacity, energy production

Oral supplementation with the amino acid beta-alanine increases muscle content of the dipeptide carnosine (beta-alanyl-L-histidine) and has positive effects on some short high-intensity exercise tasks. Several studies using supplementation protocols of 3.2 to 6.4 g/d over 4–10 weeks have shown increases in muscle carnosine content of 23 to 80% in muscles of the upper and lower leg (Baguet et al., 2010; Baguet et al., 2009; Derave et al., 2007; Harris et al., 2006; Hill et al., 2007; Kendrick et al., 2008). Supplementation has also been associated with improved cycling time to exhaustion (~2.5 min) at 110% of maximal aerobic capacity (VO_2max) (Hill et al., 2007; Sale et al., 2011; Smith et al., 2009), maximal 2000-m rowing performance (Hobson et al., 2013), and intermittent running (Saunders et al., 2012b), and isometric endurance capacity (Sale et al., 2012). Others have reported no performance improvement after beta-alanine supplementation in repeated sprint exercise (Saunders et al., 2012a), isometric endurance and 400-m sprinting (Derave et al., 2007), or cycling time to exhaustion at intensities greater that 110% VO_2max (Jagim et al., 2013). These findings all seem to agree with the conclusion of the meta-analysis performed by Hobson et al. (Hobson et al., 2012), namely that maximal exercise lasting between 1 and 4 min is likely to improve with beta-alanine supplementation, whereas those lasting less than 60 s are not.

Improvements to severe-intensity exercise of ~1 to 4 min duration following beta-alanine supplementation may be related to enhanced aerobic energy contribution (Gross et al., 2014). Indeed, we recently showed an increase in aerobic energy production and reduced reliance on anaerobic energy at the same severe intensity for a fixed duration (90 s) following 38-day beta-alanine supplementation (Gross et al., 2014). Additional ergogenic effects associated with muscle carnosine include enhancing Ca^{2+} release from the sarcoplasmic reticulum, or helping maintain it during muscle acidification (Rubtsov, 2001), increasing Ca^{2+} sensitivity of the contractile elements of human vastus lateralis and rat muscle (Dutka & Lamb, 2004; Dutka et al., 2012; Rubtsov, 2001), increasing intracellular acid buffering (Hill et al., 2007; Hobson et al., 2012; Sale et al., 2010), and reducing reliance on anaerobic glycolysis (Boldyrev, 2012). Further, beta-alanine supplementation has been shown to positively affect fatigue resistance and the force-frequency relationship in mouse muscle (Everaert et al., 2013).

Gross, Bieri, Hoppeler, and Vogt are with the Institute for Anatomy, University of Bern, Switzerland. Norman is with the Dept. of Laboratory Medicine at the University Hospital Huddinge, Karolinska Instituet, Stockholm, Sweden. Address author correspondence to Micah Gross at micah.gross@baspo.admin.ch.

Competitive alpine skiing typically involves bouts lasting 45 s to 2.5 min at intensities exceeding VO_2max (Saibene et al., 1985; Veicsteinas et al., 1984) and involving large amounts of isometric and eccentric muscle work (Berg & Eiken, 1999; Berg et al., 1995), as well as precise coordination. From a metabolic standpoint, performance is most likely limited by restrictions to leg blood flow, insufficient aerobic energy supply, and glycolytic metabolite accumulation leading to muscular fatigue and compromised motor control (Ferguson, 2010). At the same time, leg power is important for ski performance, which is evident in the jumping prowess of competitive skiers (Breil et al., 2010; Gross et al., 2009; Patterson et al., 2009). This being the case, alpine skiers might experience positive effects on performance from beta-alanine supplementation.

In this study, we explored the effects of beta-alanine supplementation on explosive jump performance, severe-intensity cycling energy metabolism and severe-intensity repeated-jump performance in professional alpine skiers. We hypothesized that beta-alanine supplementation would enhance explosiveness, increase aerobic energy contribution, decrease reliance on anaerobic glycolysis and improve performance during severe-intensity exercise.

Methods

Subjects

Participants for the study were nine male skiers from the Swiss national ski federation (age: 19.5 ± 1.1 y, weight: 79.0 ± 6.1 kg, height: 180 ± 5 cm, VO_2max: 53.1 ± 6.1 ml/min/kg), who were competing internationally at the European Cup level. All subjects provided written consent to all study procedures, which had been approved by the ethical review board of the canton of Bern, Switzerland.

Intervention

The study took place between August and October, in the second half of the skiers' off-season conditioning period. Skiers were divided into two groups, which were matched for VO_2max, height and weight. In a double-blind fashion, 400 mg gel capsules containing either beta-alanine (BAL) or maltodextrine (placebo, PLA) were distributed to the subjects. Coaches, who were also blinded to the group affiliation, took responsibility for controlling supplement compliance. Subjects consumed four capsules three times daily for a daily dose of 4.8 g over a period of five weeks. They were advised to consume the capsules at the three main meal times (Stegen et al., 2013) and before going to sleep. Capsules contained purified beta-alanine or maltodextrine and were produced noncommercially for this study (Pharma Futura SA, 3979 Grône, Switzerland). All subjects performed the same training before and throughout the study, which was confirmed by subjects' training logs detailing training time and perceived intensity (RPE, 1 to 10 scale) for all performed sessions. Training load was quantified by multiplying training time in minutes by session RPE (Foster et al., 2001). Training during this period included high volumes of strength and conditioning training and on-snow ski training.

Laboratory Tests

Before (T1) and after the supplementation period (T2), subjects reported to the laboratory for a series of tests and measurements. Upon arrival, body weight and body fat percentage via seven skinfolds (Jackson & Pollock, 1978) were assessed. Subjects then performed a self-paced 10-min warm-up on a cycle ergometer.

After warming up, subjects performed three maximal countermovement jumps (CMJ) on a QuattroJump force plate (Kistler Instruments, Winterthur, Switzerland) according to standardized procedures. Briefly, they were instructed to jump as high as possible with hands fixed at the hips. QuattroJump software (version 1.0.9.2) supplied jump height (in cm) and maximal (P_{max}, in W/kg) and average concentric power (P_{avg}, in W/kg) for each jump and averaged for the three jumps.

Thereafter, a constant-workload (110% of peak incremental power output [PPO]), fixed-duration (90 s) cycling test (CLT) was performed for assessing VO_2 on-kinetics, oxygen deficit and proportions of aerobic and anaerobic energy contribution. Breath-by-breath respiratory data were collected using the Oxycon Pro spirometry system (Erich Jaeger GmbH, Höchberg, Germany). Heart rate (HR) was measured continuously (Polar Electro Oy, Kempele, Finland). Workload was determined from incremental cycling tests (30 W/3 min) performed outside of the study less than 3 months before the study; CLT workload was 110% of the maximal incremental workload. After a 3-min warm up at 40% of maximal incremental workload, subjects cycled at 50 W and 60 rpm for 3 min (baseline phase). The baseline workload of 50 W was chosen because it is the lowest at which the Ergoline can provide continuous resistance at 60 rpm. Thereafter, cadence was increased to 105 rpm and workload was adjusted to 110% VO_2max within approximately 3 s. This was maintained for 90 s. Blood lactate was measured at the finger 30 s before, 15 s, after and 4 min after the 90-s test (Biosen C-line sport, EKF-diagnostic GmbH, Barleben/Magdeburg, Germany). The cadence during the test was chosen to ensure that subjects could pedal smoothly and complete the exercise task with minimal upper body motion. VO_2peak in the CLT was defined as the highest 15-s average of breath-by-breath data. The time constant (τ) of the primary component of VO_2 on-kinetics was determined using an automated calculation table (Microsoft Excel 2010). This table tested integers between 1 and 30 for τ and time delay, to find the best (minimal residual) exponential fit to the VO_2 data (reduced to 5-s averages and manually filtered of outliers) based on the following equation:

$$VO_2(t) = VO_2(b) + A \cdot (1 - e^{-[t - TD/\tau]})$$

where $VO_2(t)$ is VO_2 at time t during the 90-s test; $VO_2(b)$ is baseline VO_2 during the minute preceding the test (ie,

at 50 W); A is the amplitude of VO_2 increase, i.e., peak VO_2—VO_2(b); TD is the time-delay; and τ is the time constant. At 110% VO_2max, a monoexponential has been shown to describe VO_2 kinetics sufficiently (Ozyener et al., 2001). However, because data appeared to be clearly biphasic in most cases, the first 15 s of the test were omitted for calculating of the primary phase τ, to avoid contamination of the cardiodynamic phase. In a pilot study, the CV for τ during CLT was 13% (95% c.i. ± 2.3 s).

Oxygen deficit was taken as the difference between O_2 consumption during the 90-s test and the estimated O_2 demand (Bangsbo et al., 1990). O_2 consumption was the area under the curve of gross VO_2 between 0 and 90 s (ie, mean VO_2·1.5 min). O_2 demand at 110% PPO was estimated by interpolating data from the most recent incremental test (using stages before plateau in VO_2 only) and correcting the y-intercept based on steady-state VO_2 during the warm-up before the CLT. The following equation was used for estimating O_2 demand:

$$O_2 \text{ demand} = (m \cdot PO_{110\%} + [b - m \cdot PO_{40\%}]) \cdot 1.5 \text{ min}$$

where m is the slope of the VO_2/PO relationship (ml/min/W) established from the maximal incremental test, b is the steady-state VO_2 (ml/min) averaged over the second half of the warm-up preceding the 90-s test, and $PO_{110\%}$ and $PO_{40\%}$ are the workloads (W) applied during test and warm-up, respectively. The percentage of aerobic energy contribution was calculated as O_2 consumption/O_2 demand · 100%. Finally, the excess postexercise O_2 consumption in the 5 min following the test ($EPOC_5$) was taken as the net O_2 consumption (above resting value) during this period. In our laboratory, CV in this test for O_2 consumption, O_2 deficit, aerobic energy contribution and $EPOC_5$ are 2% (95% c.i. ± 88 ml), 3% (95% c.i. ± 115 ml), 2% (95% c.i. ± 1.1 percentage points) and 8% (95% c.i. ± 242 ml), respectively (Märzendorfer, 2011).

After recovering for 30 min, subjects concluded the laboratory session by performing the 90-s box jump test (BJ90). The BJ90 is a simple indoor performance test typically used in alpine skiing to simulate the metabolic and biomechanical demands of the sport (Andersen & Montgomery, 1988). The test goal is to perform as many lateral jumps as possible onto and off of a bench (width 50 cm, height 44 cm), alternating left to right on the jump down, within 90 s. Jumps were tallied by an investigator after 30, 60, and 90 s.

Muscle Biopsies

Before and after 90-s CLT, muscle biopsies from *m. vastus lateralis* were taken. Before warm-up, two incisions were made under local anesthesia induced with Lidocaine, approximately 1 cm apart in the midthigh portion of *m. vastus lateralis* of the right le.g., and the first (preexercise) biopsy was extracted using a Bergström needle (Bergstrom, 1975). A second (postexercise) biopsy was extracted using a Pro-Mag 2.2 automatic biopsy instrument (MD Tech, Gainsville, FL, USA) exactly 15 s after completion of the CLT. The Bergström technique was used for the pre- exercise biopsy to attain sufficient tissue (which was used for additional analyses not published here) while the automatic biopsy instrument was used postexercise to ensure the desired fast extraction. Both biopsies were immediately frozen in isopentane cooled by liquid nitrogen, then preserved in liquid nitrogen until further analysis.

From the resting biopsies, cross-sectional slices (12 µm) were stained for determining muscle fiber-type composition using procedures described by Billeter et al. (Billeter et al., 1980). Samples were preincubated at pH 4.75 instead of pH 4.35, as this produced better contrast in pilot stains. Stained samples were photographed with 20× magnification (ColorView 3U CCD Color Camera in a Leica DMRB light microscope). Reasonably cross-sectioned fibers were visually classified as type I (darkly stained) or type II (unstained or faintly stained) muscle fibers. In all, 374 ± 139 (range 172 to 546) and 245 ± 122 (range 91 to 458) fibers per subject were analyzed for T1 and T2, respectively. From the same photos, cross-sectional area of a subset of cross-sectioned fibers (92 ± 40, range 40 to 142 and 56 ± 59, range 21 to 200 for T1 and T2, respectively) was assessed using the area tool in cell^D software (version 3.4, Olympus Soft Imaging Solutions GmbH).

At each time point, portions of resting and postexercise biopsies were freeze-dried, dissected under a microscope until freed from blood and connective tissue, and weighed for analysis of muscle pH, buffering capacity and muscle lactate (see below).

One portion of freeze-dried muscle (1 to 9 mg) was designated for determination of muscle pH and buffering capacity, based on the methods of Bishop et al. (Bishop et al., 2008). In short, muscle was homogenized by hand on ice in a 10 mM NaF solution at a dilution of 30 mg muscle / ml solution. Twenty-five- or 30-µl portions were transferred to Eppendorf tubes, vortexed, and warmed to 37°C on a Thermomixer (Vadaux-Eppendorf, Basel, Switzerland) for measurement of pH using a microelectrode (MI-410, Microelectrodes Inc., USA) connected to a pH-meter (Model 320, Mettler-Toledo, USA) double-calibrated at pH 7.0 and 4.01. After initial pH determination, solutions from preexercise samples were titrated with 10 mM HCl until pH decreased to 6.3. pH was measured after each 2-µl addition of HCl, and the mean slope was calculated from the serial pH-measurements and normalized to express muscle buffering capacity in terms of mmol H^+/kg dry muscle/pH unit.

Another portion (0.7 to 6.8 mg) was submerged in 0.4 M $HClO_4$ on ice and broken into small pieces by hand to extract metabolites. After 20 min, the solution was neutralized using 2 M $KHCO_3$ and centrifuged for 10 s. Muscle lactate was analyzed in the supernatant by a fluorometric enzymatic method (Lowry & Passonneau, 1972).

Side Effects

At the conclusion of the study, subjects filled out a questionnaire regarding side effects (frequency and severity) they may have experienced during the study.

Data Analysis

All performance variables from T1 and T2 laboratory tests were compared within groups using Wilcoxon signed ranks tests. Only for parameters where *n* for each group was at least four, groups were compared and interaction effects were assessed by comparing delta values between groups via independent *t* tests (W.G. Hopkins, 2003; Hopkins et al., 2009). Pearson's correlations were performed between changes in CMJ power parameters and subjects' percentage of type II muscle fibers. The level of significance was set at $p = .05$. Data are reported as mean ± standard deviation, with effect sizes (*d*) which were calculated as the mean change relative to the standard deviation of the first measurement.

Table 1 Anthropometric Data

Measurment/Time Point	BAL (*n* = 5)	PLA (*n* = 4)
Bodyweight (kg)		
T1	76.4 ± 3.9	81.6 ± 8.3
T2	76.5 ± 3.5	81.0 ± 8.3
p time	0.88	0.47
Sum of 7 skinfolds (mm)		
T1	47.5 ±13.3	66.6 ± 10.2
T2	45.8 ± 10.2	59.1 ± 5.2
p time	0.43	0.11
Body fat (%)		
T1	11.1 ± 2.4	14.4 ± 1.8
T2	10.8 ± 1.8	13.1 ± 0.9
p time	0.44	0.12

Note. Data presented as mean ± s.d. for before (T1) and after (T2) supplementation with beta-alanine (BAL) or placebo (PLA).

Results

Laboratory Tests

The CMJ and CLT were performed by nine subjects at both test points and biopsies were taken from eight, as one subject objected to the biopsy procedures. The BJ90 was however performed by only seven subjects, as two (both in PLA) were recovering from injury at the onset of the study. In addition, one subject failed to complete the training log throughout the study. There were no changes after supplementation in bodyweight, the sum of seven skinfolds or body fat percentage (Table 1).

In the CMJ, BAL improved P_{max} (+7.0 ± 2.5%, $d = 0.9$, $n = 5$, $p = .04$) and P_{avg} (+7.0 ± 2.4%, $d = 0.7$, $n = 5$, $p = .04$) significantly following supplementation, whereas no change was seen in PLA (P_{max}: +1.0 ± 3.5%, $d = 0.4$, $n = 4$, $p = .47$; P_{avg}: 0.4 ± 2.6%, $d = 0.1$, $n = 4$, $p = .72$). Interaction effects for both variables were significant. On the other hand, there was no change in either BAL (-0.7 ± 2.3%, d=-0.1, $n = 5$, $p = .59$) or PLA (+5.0 ± 8.1%, d=-0.9, $n = 4$, $p = .27$), and no interaction effect for jump height. These data are displayed in Table 2 and Figure 1. Changes in P_{avg} in BAL tended to be correlated with the percentage of type II muscle fibers ($r = .83$, $p = .08$, $n = 5$).

In the CLT, oxygen deficit remained unchanged in BAL (-2.5 ± 7.5%, $d = 0.4$, $n = 5$, $p = .50$) but tended to increase in PLA (+6.6 ± 2.8%, $d = 0.5$, $n = 4$, $p = .07$) resulting in a tendency for an interaction effect ($p = .06$). A similar pattern was seen for blood lactate accumulation (difference pre-CLT to 4 min post-CLT), aerobic energy contribution, and $EPOC_5$. Blood lactate accumulation remained unchanged in BAL (-11.5 ± 31.1%, $d = 0.6$, $n = 5$, $p = .35$) but tended to increase in PLA (+34.6 ± 20.1%, $d = 1.7$, $n = 4$, $p = .07$), leading to

Table 2 Countermovement Jump Variables

	P_{max} (W/kg)			P_{avg} (W/kg)			Jump Height (cm)		
	T1	T2	*p* time	T1	T2	*p* time	T1	T2	*p* time
BAL (*n* = 5)	57.6 ± 4.7	61.7 ± 6.1	.01	34.0 ± 3.3	36.4 ± 3.9	<.01	55.8 ± 4.5	55.5 ± 5.5	.59
PLA (*n* = 4)	57.0 ± 1.5	57.6 ± 3.3	.58	35.7 ± 1.5	35.8 ± 1.6	.81	51.1 ± 2.7	53.5 ± 3.6	.27
p deltas	.03			.01			.20		

Note. Data presented as mean ± s.d. for before (T1) and after (T2) supplementation with P_{max}: maximal concentric power. P_{avg}: average concentric power.

Table 3 Muscle Lactate Measurement from Biopsies Taken Before and Immediately After Severe-Intensity Cycling

	Muscle Lactate, Pre[a]			Muscle Lactate, Post[a]			Δ Muscle Lactate[a]		
	T1	T2	*p* time	T1	T2	*p* time	T1	T2	*p* time
BAL (*n* = 5)	2.1 ± 1.7	1.1 ± 0.8	0.35	47.9 ± 11.5	49.8 ± 9.7	0.36	43.3 ± 10.8	45.2 ± 8.1	.50
PLA (*n* = 3)	3.9 ± 1.8	2.0 ± 3.4	0.29	38.8 ± 7.6	40.9 ± 5.9	0.59	32.3 ± 7.6	35.3 ± 4.3	.59
p deltas	.61			.97			.78		

Note. Data presented as mean ± s.d. for before (T1) and after (T2) supplementation with beta-alanine (BAL) or placebo (PLA). Cycling test comprised 90 s at 110% VO_2max.

[a]mmol/kg d.w.

a tendency for an interaction effect (p = .07). Aerobic energy contribution remained unchanged in BAL (+1.3 ± 2.9%, d = 0.4, n = 5, p = .47) and tended to decrease in PLA (-2.1 ± 1.3%, d = 1.0, n = 4, p = .07), leading to a tendency for an interaction effect (P = .07). Net $EPOC_5$ did not change in BAL (-6.3 ± 17.9%, d = 0.9, n = 5, p = .42) but tended to increase in PLA (+15.2 ± 11.3%, d = 1.3, n = 4, p = .07); p for the interaction was 0.07.

There was a tendency for improved overall performance (+2.6 ± 2.4%, d = 0.6, n = 5, p = .08) in the BJ90 following beta-alanine supplementation (Figure 2). Whereas pacing for the initial 60 s was nearly identical, BAL tended to perform more jumps in the final 30 s (+6.8 ± 4.2, d = 0.9, n = 5, p = .06). Changes in BJ90 overall performance for the two subjects in PLA were +1 and -3 jumps. Change in the final 30 s was -4 and +1 jumps.

Muscle Biopsies

Histochemical determination of fiber-type distribution revealed that subjects had 61 ± 11% (range: 44 to 79%, n = 8) type I fibers and 39 ± 11% (range 21 to 56%, n = 8) type II fibers, with no difference between groups (p > .57) and no changes occurring during the study (p > .15). Mean fiber cross-sectional areas were 6367 ± 731 μm^2 and 7311 ± 1534 μm^2 for type I and type II fibers, respectively (n = 8), and did not differ between groups or change during the study.

There were no effects of intervention on muscle pH before the CLT or on postexercise pH (p > .16). Pooled for all subjects, muscle pH before and after the CLT was 6.87 ± 0.05 and 6.64 ± 0.07, respectively (n = 8). Neither were there effects (p > .16) of intervention on buffering capacity (Figure 3). There were no effects of intervention on pre- or postexercise muscle lactate, or on exercise-induced muscle lactate accumulation (Table 3).

Side Effects

Four out of five subjects receiving beta-alanine reported that they had experienced no side effects. However, one subject reported having frequent and severe paresthesia as well as some digestion problems.

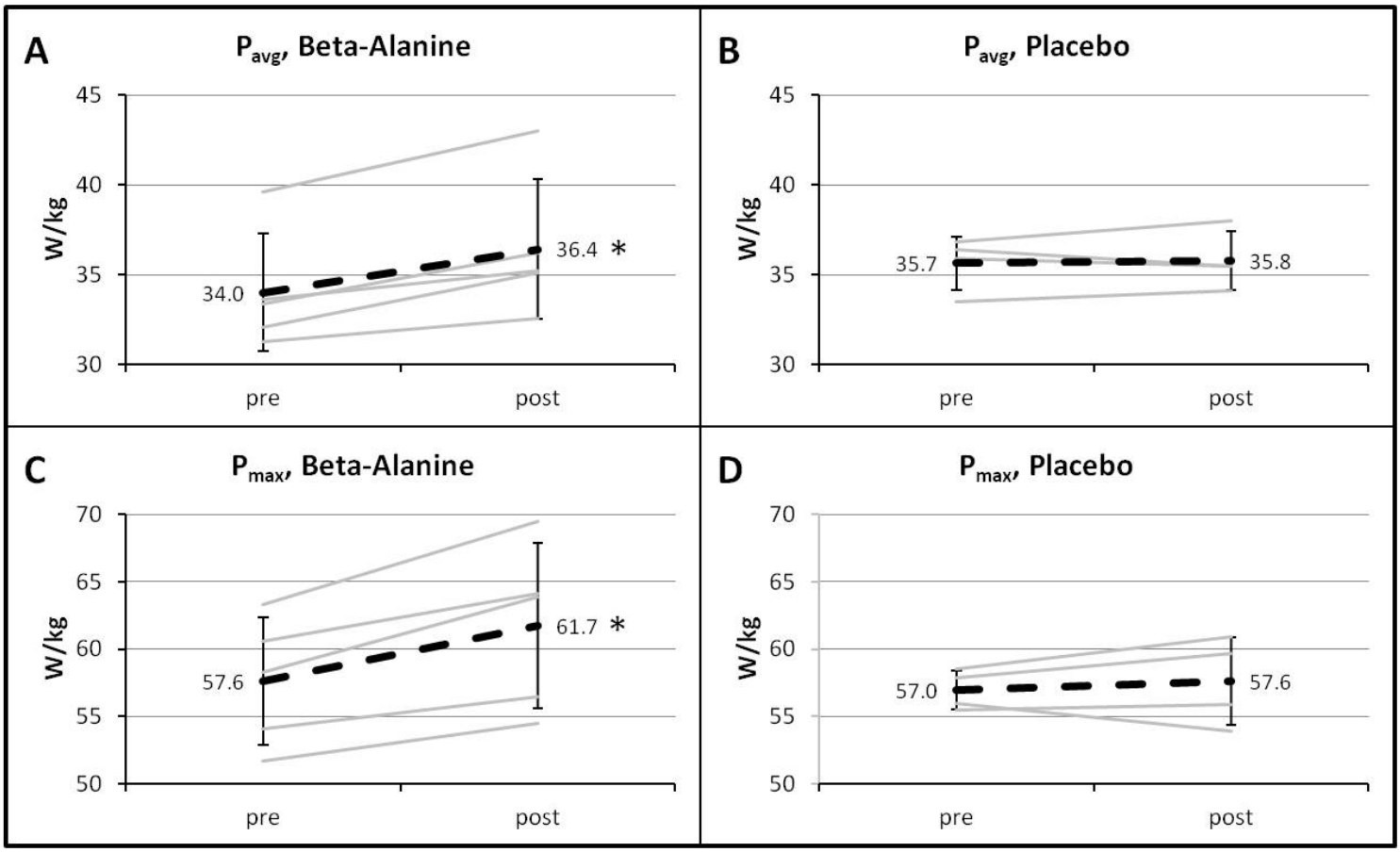

Figure 1 — Individual changes (gray lines) and group changes (mean ± s.d., black dashed line) in countermovment jumping power with beta-alanine (n = 5) or placebo (n = 4). P_{avg}, P_{max}: mean and maximal concentric power.

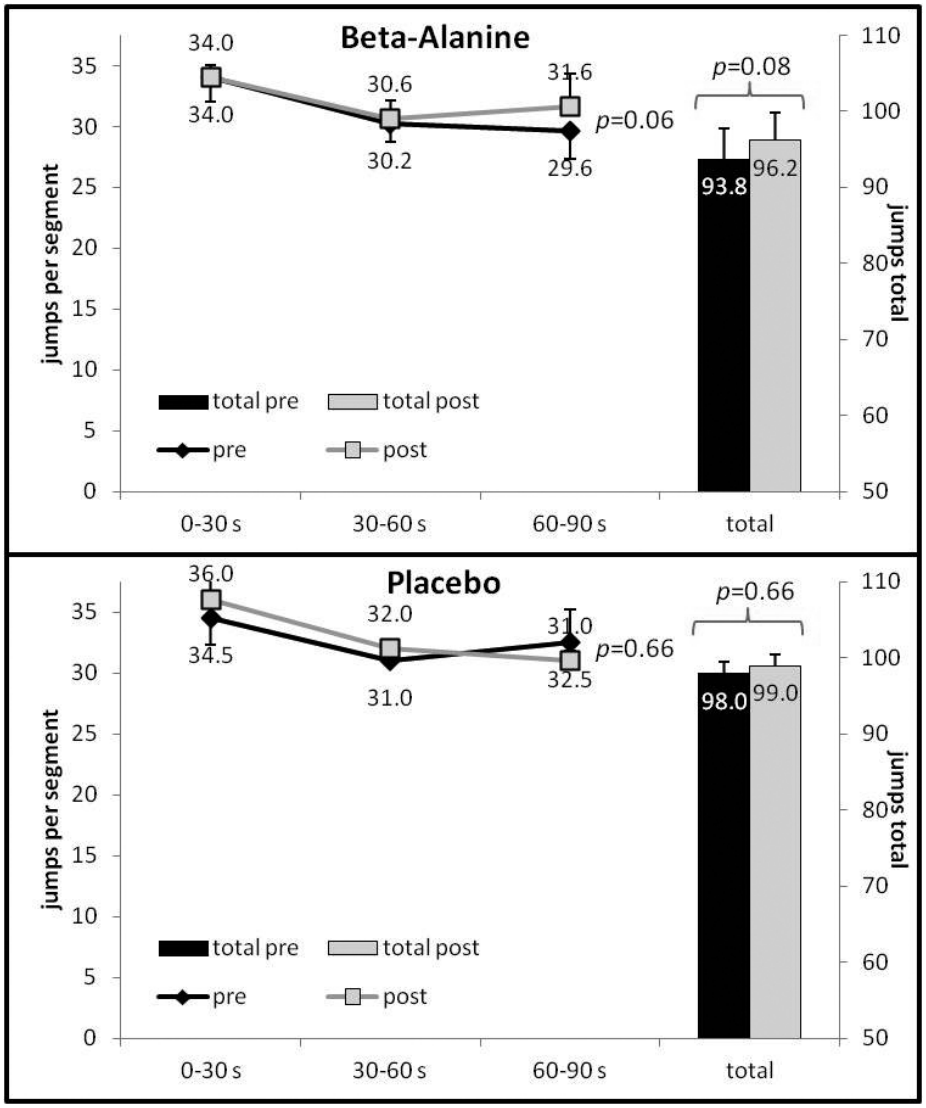

Figure 2 — Jumps performed per 30-s segment and in total during the 90-s box jump test, before and after supplementation with beta-alanine (n = 5) or placebo (n = 2, see text).

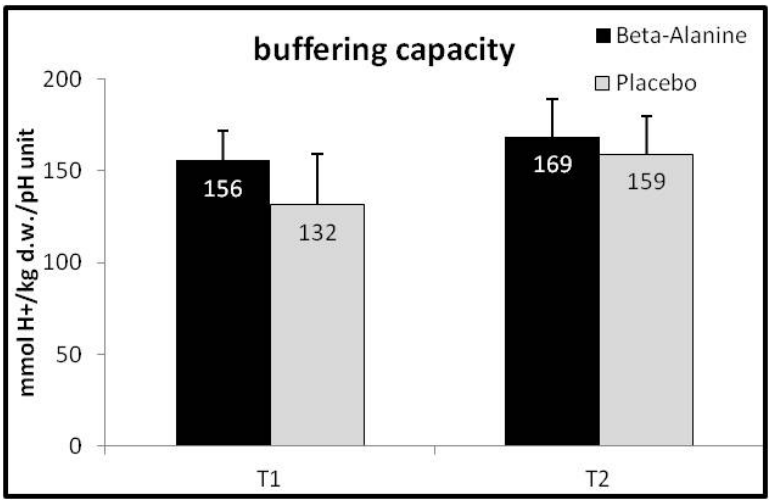

Figure 3 — Muscle buffering capacity before (T1) and after supplementation (T2) with beta-alanine (n = 5) or placebo (n = 3). There was no significant change in either the beta-alanine (p = .16) or placebo group (p = .44).

Discussion

The main finding of this study was that beta-alanine supplementation improved explosive jumping power in elite alpine skiers. In addition, 90-s repeated box jump performance tended to improve while, during 90-s severe-intensity constant-load cycling, there were tendencies in several parameters suggesting a shift toward aerobic energy contribution, which could help explain the changes in box jump performance.

Several parameters failed to reach statistical significance, which is partly attributable to the small *n*. While the small sample size presents the main limitation of the current study, this was the trade-off for performing the study on professional skiers. In addition, the performance-enhancing effect of beta-alanine seems to be a combination of several mechanisms, which probably also led to the small effects reported here. Another limitation was that we did not measure muscle carnosine, but instead made the rather safe assumption that beta-alanine supplementation like that employed in the current study increases carnosine (for review, see Artioli et al., 2010; Sale et al., 2010). Nonetheless, if we had been able to correlate changes in carnosine to changes in performance, stronger conclusions could have been possible.

Changes in power generation during countermovement jumping have not been shown previously in response to beta-alanine supplementation. Hoffman et al. (Hoffman et al., 2008) measured maximal and average power output during 1-repetition maximum barbell squats, and although there were increases in both following beta-supplementation, these were not significant. However, Everaert et al. show that beta-alanine supplementation shifts the force-frequency curve of the fast-twitch muscle EDL of mice to the left (2013). Further, Dutka and Lamb show improved Ca^{2+}-sensitivity in skinned rat muscle fibers (2004), while Dutka et al. show enhanced Ca^{2+}-sensitivity in skinned human slow- and fast-twitch fibers (2012), in both cases in a dose-dependent manner upon addition of carnosine. Although these authors did not show any improvement of the maximal force production with more carnosine present in the myoplasm, the better Ca^{2+}-sensitivity of the contractile apparatus may have increased the muscle contraction velocity. Indeed, for the same maximal force production, the Ca^{2+} released from the sarcoplasmic reticulum may bind to the troponin C at a faster rate. Since we observed a tendency for changes in CMJ P_{avg} to be correlated to the percentage of fast-twitch fibers, it is feasible that enhanced contractility of fast-twitch muscle, in accordance to the findings of Everaert et al. (2013) could be a mechanism behind improved CMJ power following beta-alanine supplementation.

There was an increase in jumps performed in the last 30 s of the BJ90 and overall performance (total jumps) tended to improve with beta-alanine supplementation. Although this did not occur in PLA, only two subjects performed the test at both time points, which means attributing the improvement in BAL solely to the supplement should be done with caution. Nonetheless, previous studies report delayed fatigue (improved time to exhaustion) following beta-alanine supplementation during severe-intensity cycling (~2.5 min) (Hill et al., 2007; Sale et al., 2011; Smith et al., 2009) and isometric exercise (Sale et al., 2012), as well as increased contractile apparatus Ca2+-sensitivity in the presence of carnosine (Dutka et al., 2012), findings which would agree with the changes we observed in BJ90. Alternatively, in light of changes in CMJ power which can be more confidently attributed to beta-alanine, improvements in BJ90 could plausibly be related to increased maximal jumping power, which allowed BJ90 to be performed at a lower relative power.

Whereas many postulate that beta-alanine supplementation improves severe exercise performance by increasing the buffering capacity of muscle carnosine (e.g., Hill et al., 2007; Hobson et al., 2012; Sale et al., 2010), no change in total muscle buffering capacity was detectible in the current study. This was also the case in a previous study from our laboratory (Gross et al., 2014); however, as the titration technique does have its limitations, it is possibly incorrect to conclude from the current study that buffering capacity is not affected by beta-alanine supplementation. On the other hand, it is also known that carnosine can enhance muscle activity directly, in vitro (Boldyrev, 2012) and in vivo in animals (Stvolinskii et al., 1992), or maintain muscle fiber Ca^{2+}-sensitivity in the presence of acidification (Rubtsov, 2001). For example, rats injected with carnosine ran 25 to 30% longer while accumulating less lactate than control animals (Stvolinskii et al., 1992), due perhaps to reduced energy expenditure per unit work or improved mitochondrial coupling (Boldyrev 2012). Observations of such effects (termed "Severin's phenomenon") are not new, but have received relatively little appreciation in recent exercise-related studies on beta-alanine and carnosine. An exception to this oversight is the work of Dutka and Lamb (2004), who show improved Ca^{2+}-sensitivity of the contractile apparatus in skinned rat muscle fibers, and that of Dutka et al. (2012), who showed the same thing in mechanically skinned human muscle fibers, in both cases in a dose-dependent manner upon addition of carnosine. In light of these effects, decreased reliance on anaerobic glycolysis, entailing slower accumulation of fatigue-inducing glycolytic metabolites, and better-maintained Ca^{2+}-sensitivity could reveal a further mechanism behind changes in BJ90 performance following beta-alanine supplementation.

Although the increased reliance on aerobic energy in BAL did not reach statistical significance, the interaction effect compared with PLA was nearly significant, suggesting that beta-alanine could support a more optimal energy provision. Thus, whereas a negative training adaptation in relation to aerobic energy provision at posttesting occurred in PLA, this was counteracted by beta-alanine.

Perspectives

Previous studies on beta-alanine supplementation have focused mostly on severe exercise time-to-exhaustion,

sprint or strength performance, and have accentuated the increased buffering effect of carnosine as the main factor leading to improvement. In the current study, we show that beta-alanine supplementation improved explosive jumping power and 90-s repeated jump performance in elite alpine skiers, although results from repeated jumping cannot be considered controlled. We suspect that increased Ca2+-sensitivity of the contractile apparatus can explain improvements in explosive jumping power and 90-s repeated jump performance, and that enhanced aerobic energy production can help explain repeated jump performance as well. Future research should explore these mechanisms of performance improvement following beta-alanine supplementation, in addition to those related to buffering capacity.

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