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Original Research

β-Alanine Plus Sodium Bicarbonate Co-Supplementation Does Not Decrease Neuromuscular Fatigue in Swimming

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ABSTRACT

International Journal of Exercise Science 17(2): 648-659, 2024. This study aimed to investigate the effects of chronic β-alanine (βA) plus acute sodium bicarbonate (SB) co-supplementation on neuromuscular fatigue during high-intensity intermittent efforts in swimming. Eleven regional and national competitive-level young swimmers performed a neuromuscular fatigue assessment before and immediately after two 20 × 25-m front crawl maximal efforts every 90 s, performed at pre- and post-4-week co-supplementation. Neuromuscular fatigue was evaluated by percutaneous electrical stimuli through the twitch interpolation technique on the *triceps brachii* and *quadriceps femoris*. Performance was defined by the mean time of the 20 efforts and blood samples to lactate concentrations were collected every four efforts. Participants supplemented 3.2–6.4 g·day^{-1} of chronic βA or placebo (PL) during four weeks, and acute 0.3 g·kg^{-1} of SB or PL 60 min before the second assessment (allowing βA+SB and PL+PL groups). No statistical changes were found in neuromuscular fatigue of *triceps brachii*. In the *quadriceps femoris*, a main effect of time was found in potentiated twitch delta values in pooled groups, showing a statistical increase of 19.01% after four weeks (Δ = 13.05 [0.35 - 25.75] N; p = 0.044), without time × group interactions. No statistical difference was found in the swimming performance. Blood lactate increased by 25.06% only in the βA+SB group (Δ = 6.40 [4.62 - 8.18] mM; p_{Bonf} < 0.001) after the supplementation period. In conclusion, 4-week βA and SB co-supplementation were not able to reduce neuromuscular fatigue levels and improve performance in high-intensity intermittent efforts, but statistically increased blood lactate levels.

KEY WORDS: Ergogenic aids, buffering capacity, voluntary activation

INTRODUCTION

Exercise-induced neuromuscular fatigue is a multifactorial physiological phenomenon, composed of central and peripheral factors that influence the muscle force production capacity (3). Central fatigue is characterized by decreased muscle voluntary activation, caused by a reduction in the motor unit firing rates during muscle force production due to impairment in the primary motor cortex activation and propagation of the command from the central nervous system to the motoneurons (28). Peripheral fatigue, on the other hand, is characterized by a decrease in the contractile strength of muscle fibers. This is caused by the inhibition of the cross-bridge interaction due to the accumulation of inorganic phosphate and hydrogen ions in the sarcolemma (18), lower calcium release (29), and decreases in available metabolic substrates (12).

In swimming, the use of nutritional ergogenic aids is a very common strategy to try to prevent neuromuscular fatigue and consequently improve performance. Among different dietary strategies, β-alanine (βA) and sodium bicarbonate (SB) supplementations (both isolated or combined) are often used in swimming (4, 5, 8, 10, 14, 17, 21, 27, 31) due to the ability to improve intracellular (11) and extracellular (2) buffering capacity, respectively. βA is an important amino acid in the endogenous synthesis of intramuscular carnosine (β-alanyl–L-histidine) (11). Abundant in skeletal muscle, intramuscular carnosine content has important physiological roles at intracellular levels, such as an increase in the capacity of contractile apparatus, calcium sensitivity regulation, and hydrogen ions buffering (6, 11). On the other hand, SB ($NaHCO_3^-$) can increase plasmatic bicarbonate (HCO_3^-) levels, which in turn has an important physiological role at the extracellular level, allowing better acid-base balance through higher hydrogen ions diffusion (2).

As mentioned above, several studies have investigated the effects of the βA and SB supplementation in order to greater buffering capacity in swimming, but the premise that this would attenuate the exercise-induced fatigue has not been experimentally tested using a neuromuscular approach. In addition, isolated βA and SB supplementations can improve swimming performance in high-intensity intermittent efforts (4, 8, 10, 27, 31) and maximal 100-m (5, 17) and 200-m (5, 14) race-pace events, however, the combination of βA plus SB in a co-supplementation protocol was tested only in short-distance race-pace events, and not in high-intensity intermittent efforts (an ecological context closer to sports training). Therefore, there is a research gap in the literature concerning the potential effects of the combination of βA and SB on neuromuscular fatigue and swimming performance during high-intensity intermittent efforts.

Thus, this study aimed to investigate the effects of 4-week chronic βA plus acute SB co-supplementation on neuromuscular fatigue and swimming performance during 20 × 25-m front crawl maximal efforts every 90 s in regional and national competitive-level young swimmers. The hypothesis is that the combination of βA and SB can reduce the levels of fatigue established during high-intensity intermittent efforts, improving swimming performance.

METHODS

Participants

Eleven regional and national competitive-level young swimmers voluntarily participated in this study. The participants' characteristics are shown in Table 1. They were randomly divided into two groups: co-supplemented with chronic βA plus acute SB (βA+SB; n = 6) or chronic placebo plus acute placebo (PL+PL; n = 5). The inclusion criteria for participants were swimmers with at least two years of competitive experience, with a training routine of at least five sessions per week, and no history of metabolic or heart diseases. All procedures were previously approved by the Ethics Committee of the University of Sao Paulo (protocol no: 14713719.6.0000.5659), conducted in accordance with the Declaration of Helsinki (30) and ethical standards of the International Journal of Exercise Science (19).

Table 1. Mean ± standard deviation of participants' characteristics.

Characteristics	βA+SB (n = 6)	PL+PL (n = 5)
Gender (male/female)	5/1	4/1
Age (years)	20.00 ± 3.22	19.25 ± 4.99
Height (cm)	172.33 ± 8.45	175.75 ± 9.29
Body mass (kg)	65.92 ± 11.42	69.53 ± 5.06
100-m front crawl performance (s)	62.33 ± 17.83	63.80 ± 10.23

Note: no between-subjects effects were found in all variables ($p \geq 0.544$).

Protocol

To investigate the effects of βA and SB co-supplementation on neuromuscular fatigue and swimming performance, two double-blind, randomized, and placebo-controlled trials were performed (experimental design is shown in Figure 1). Participants attended the laboratory for evaluations on two occasions, separated by 4 weeks. On the first day (PRE), they were randomly divided into the experimental groups and then performed the neuromuscular assessment before and after the swimming performance. After 4 weeks of supplementation (POST), participants returned to the laboratory and repeated the neuromuscular assessment and swimming performance. In all cases, participants were instructed to avoid drinking caffeine, alcohol, and energy drinks for at least 12 hours before each evaluation.

The 4-week co-supplementation protocol was defined as the chronic intake of βA and acute intake of SB as proposed by previous studies (4, 11). In the first week, participants ingested 3.2 g·day^{-1} of βA (99.9% pure β-alanine, CarnoSyn, CA, USA) or placebo taken in four daily doses (800 mg·capsule^{-1}) every four hours. In the following three weeks, the daily supplement content was doubled to 6.4 g·day^{-1} in the same four daily doses (2 × 800 mg·capsule^{-1}). One day after the 4-week βA supplementation protocol ends, participants received 0.3 g·kg^{-1} of body mass of SB or placebo 60 min before the neuromuscular assessment and swimming performance. Both βA and SB were compounded in acid-resistant hypromellose capsules (both identical in appearance and weight (DRcaps, Capsugel, USA) to slow absorption into the bloodstream and avoid paresthesia and gastrointestinal discomfort.

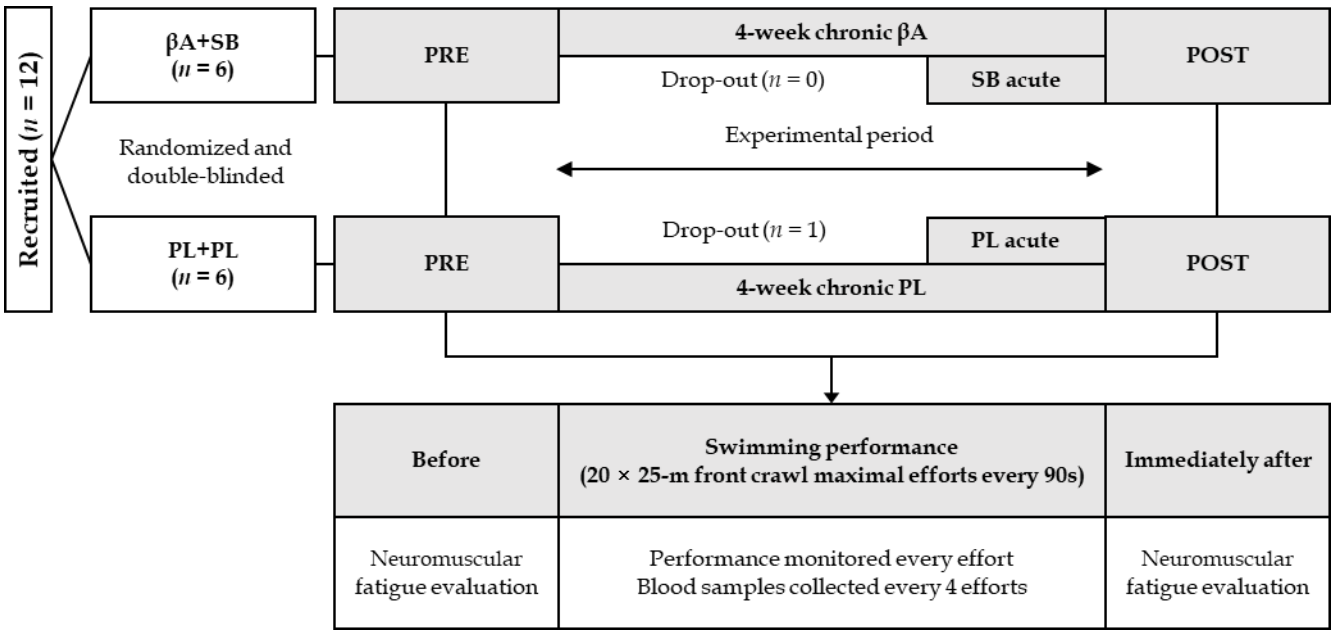

Figure 1. Experimental design of the study.

Swimming performance was characterized by high-intensity intermittent efforts, composed of 20 × 25-m front-crawl maximal efforts every 90 s with passive intervals. This exercise design was chosen for its ability to provoke a high activation of the glycolytic metabolic pathway, and because it is typically used in the sports training context and practical field (16). Performance was always monitored by the same researcher at each effort through a manual stopwatch and characterized by the mean time of the 20 efforts. To determine blood lactate concentrations, 25-µL blood samples were collected from the earlobe every four efforts during the swimming performance, and at the 3rd, 5th, and 7th min during recovery. Blood samples were collected through glass capillary microtubes containing heparin and immediately dispensed and homogenized in Eppendorf tubes containing 1% sodium fluoride (NaF), which were stored at –12 ºC for later analysis in a lactate analyzer (YSI 2300 STAT, Yellow Springs, USA). All procedures were performed in a short-course indoor pool (25 m long × 12 m wide) at a temperature of 27 ± 1 °C. The participants were instructed and verbally encouraged to give maximal effort during swimming performances.

Neuromuscular fatigue (*i.e.*, central and peripheral component), was determined by the Twitch Interpolation Technique (20), characterized by percutaneous electrical stimuli during isometric voluntary contractions performed in elbow and knee extensor musculature (*triceps brachii* and *quadriceps femoris*, respectively) before and immediately after (at most 1.5 min) the swimming performance (*i.e.*, presented as delta values; Δ = POST – PRE). The assessments were performed at an adjustable custom-built strength chair specially designed for this protocol, possessed in an experimental setup next to the swimming pool, allowing assessment of the upper and lower limbs in a 90° flexed position. The force signal data were acquired at 1000 Hz using load cells (CSAZL-100kg, MK Control and Instrumentation, BRA) attached to the wrist and ankle of the participants. The force signal was amplified by a signal amplifier (MKTC-05, MK Control and

Instrumentation, BRA), converted by an acquisition data board (USB-6501, National Instruments, USA), and processed by a specific algorithm in the Matlab software (R2018a, The MathWorks Inc, USA).

Neuromuscular fatigue in the *triceps brachii* was defined through percutaneous electrical stimuli during a 30-s submaximal isometric contraction (SIC_{TRI}, N) and monitored using real-time visual force feedback, as previously proposed (22). The central fatigue component was defined by the average superimposed twitch force (ST_{TRI}, N), evoked by six superimposed pulses applied every five seconds during the SIC_{TRI} (22). The peripheral fatigue component was defined by the potentiated twitch force (PT_{TRI}, N), evoked by a single potentiated pulse, applied to the relaxed musculature, at the end of the SIC_{TRI} (22). The percutaneous electrical stimuli (double pulses with 10 ms interpulse interval) were applied by two small-size self-adhesive electrodes (3 × 3 cm; ValuTrode, Arktus, BR) positioned on the *triceps brachii* muscle belly (medial head; distally 15-20 cm above the olecranon) through a constant-current stimulator (Bio Electric Stimulator Prototype, Inside, BR).

Neuromuscular fatigue in the *quadriceps femoris* was defined through percutaneous electrical stimuli during a 5-s maximal voluntary contraction (MVC_{QUA}, N) (15). The central fatigue component was defined by the superimposed twitch force (ST_{QUA}, N), evoked by a single superimposed pulse applied at 2-s during the MVC (15). The peripheral fatigue component was defined by the potentiated twitch force (PT_{QUA}, N), evoked by a single potentiated pulse, applied to the relaxed musculature, at the end of the MVC_{QUA} (15). The percutaneous electrical stimuli (double pulses with 10 ms interpulse interval) were applied by two large-size self-adhesive electrodes (5 × 9 cm; ValuTrode, Arktus, BR) positioned on the *quadriceps femoris* muscle belly (*vastus lateralis*; distally 15-20 cm above the femur lateral epicondyle) through a constant-current stimulator (Bio Electric Stimulator Prototype, Inside, BR).

Voluntary activation of both *triceps brachii* (VA_{TRI}, %) and *quadriceps femoris* (VA_{QUA}, %) was defined through the relationship between the amplitude of force signals evoked by the percutaneous electrical stimuli during and after the contractions (20), as presented in Expression 1,

$$VA(\%) = \left\{1 - \left[ST \cdot \left(\frac{\left(\frac{F_{ST}}{PF}\right)}{PT}\right)\right]\right\} \cdot 100 \quad (1)$$

where ST is the supramaximal force evoked by the superimposed twitch, FST is the signal amplitude before the superimposed pulse, PF is the peak force achieved during the contraction, and PT is the force evoked by the potentiated twitch. For both limbs, this equation allows correct cases in which the percutaneous electrical stimulus was applied slightly before or after the peak torque plateau.

To define the optimal intensities of percutaneous electrical stimuli for both *triceps brachii* and *quadriceps femoris*, before the assessments a protocol of consecutive incremental double pulses (increment pulses: 5 mA; interpulse interval: 10 ms) was applied through the constant-current stimulator on the relaxed muscles until reaching the peak force plateau. The optimal intensity of electrical stimulations was increased by 20% to ensure supramaximal activation in the *quadriceps femoris* (15), but not in the *triceps brachii* due to *biceps brachii* co-contraction (22).

Statistical Analysis

Data are presented as mean and ± 95% confidence interval (–95% to +95% CI). Using the Jamovi software (version 2.3.28.0), a generalized linear mixed model (GLMM) was used to statistically analyze the data set. Time (PRE × POST) and group (βA+SB × PL+PL) were set as fixed effects, and the subjects as random effects. Performance and blood lactate were tested as covariates using Pearson's correlation for *triceps brachiis* (SIC_{QUA}, PT_{TRI}, ST_{TRI}, and VA_{TRI}) and *quadriceps femoris* (MVC_{QUA}, PT_{QUA}, ST_{QUA}, and VA_{QUA}) dependent variables. The model with the lowest AIC (Akaike information criterion) was chosen for analysis, with the statistical significance level set as $p < 0.05$ with Bonferroni *post hoc* multiple comparisons also set as $p_{Bonf} < 0.05$ when necessary.

RESULTS

Neuromuscular fatigue data of *triceps brachii* and *quadriceps femoris* are shown in Figure 2. No statistical changes were found in the neuromuscular variables of the *triceps brachii*. In *quadriceps femoris*, a main effect of time showed a statistical increase only in ΔPT_{QUA} after the supplementation period (Δ = 13.05 [0.35 - 25.75] N; p = 0.044), without time × group interaction.

Performance and blood lactate data are shown in Figure 3. There were no statistical differences in swimming performance values. A main effect of time showed a statistical increase in blood lactate after the supplementation period (Δ = 3.89 [2.78 - 5.06] mM; $p < 0.001$), and a time × group interaction was found (Δ = 5.02 [2.72 - 7.32] mM; $p < 0.001$), with the *post hoc* multiple comparisons showed statistically higher values only in the βA+SB (Δ = 6.40 [4.62 - 8.18] mM; $p_{Bonf} < 0.001$).

DISCUSSION

This study aimed to investigate the effects of 4-week βA and SB co-supplementation on neuromuscular fatigue and swimming performance in regional and national competitive-level young swimmers. The main results indicated that, despite statistically increasing blood lactate, the co-supplementation of βA and SB was not able to decrease neuromuscular fatigue levels and improve swimming performance during high-intensity intermittent efforts.

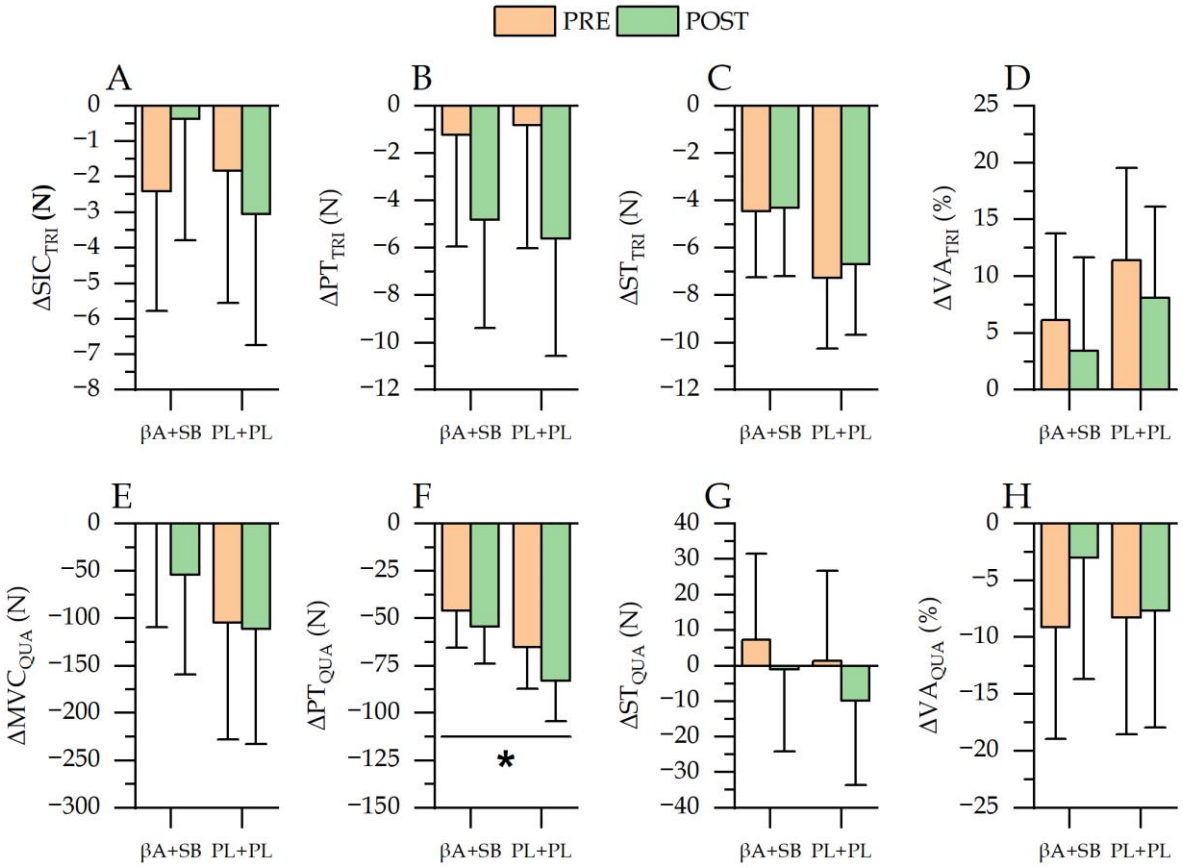

Figure 2. Generalized linear mixed model with gamma distribution. Mean and ± 95%CI of **(A)** submaximal isometric contraction, **(B)** potentiated twitch, **(C)** superimposed twitch (covariated for blood lactate), and **(D)** voluntary activation (covariated for blood lactate) of *triceps brachii*; and **(E)** maximal voluntary contraction, **(F)** potentiated twitch, **(G)** superimposed twitch (covariated for performance), and **(H)** voluntary activation (covariated for blood lactate) of *quadriceps femoris*. * denotes main effect of time ($p < 0.05$).

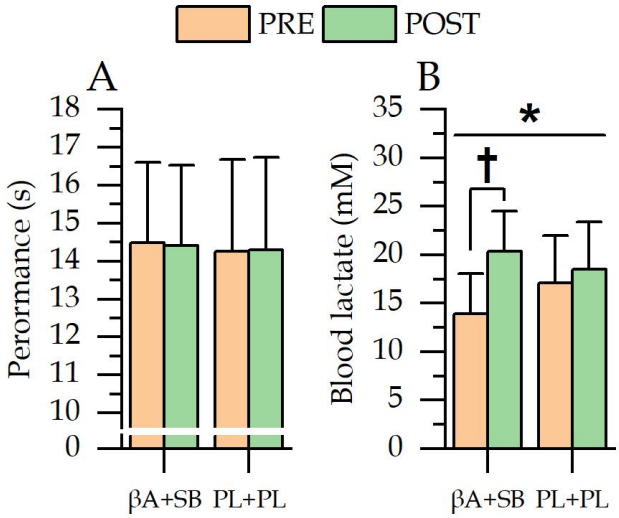

Figure 3. Generalized linear mixed model with gamma distribution. Mean and ± 95%CI of **(A)** swimming performance and **(B)** blood lactate (covariated for performance and *triceps brachii* submaximal isometric contraction). * denotes main effect of time ($p < 0.05$) and † denotes time × group interaction ($p_{Bonf} < 0.05$).

Some studies have proposed to investigate the chronic effect of βA combined with SB acute intake in order to improve swimming performance, reaching an ergogenic effect employed by a possible attenuation in fatigue through higher intra and extracellular buffering capacity (5, 17), which until now had not been experimentally tested through a neuromuscular approach. Therefore, to the best of our knowledge, this is the first study to investigate the effects of co-supplementation of βA and SB on neuromuscular fatigue through percutaneous electrical stimuli in swimming. In this way, our results showed a 19.01% increase in ΔPT_{QUA} values (p = 0.044) after 4 weeks in pooled groups, and the time × group non-interaction showed that the co-supplementation was not statistically effective in reducing or avoiding the onset of neuromuscular fatigue after high-intensity intermittent efforts. On the other hand, this finding may indicate that different variables (*e.g.*, training load) may also negatively influence the accumulation of peripheral fatigue during four weeks.

Swimming performance during the 20 × 25-m front crawl maximal efforts was also not influenced statistically by co-supplementation, since no between-subjects effects were found. Our results are in agreement with other studies, which have investigated the same co-supplementation protocol in maximal 100-m and 200-m swimming performances (5, 17). Mero, Hirvonen, Saarela, Hulmi, Hoffman and Stout (17), despite showing a statistical increase in the pH, bicarbonate, and base excess levels after the co-supplementation, indicating better buffering capacity, found no statistical improvements in the swimming performance of two 100-m front-crawl maximal efforts (12-min passive rest interval) in national and international competitive-level male swimmers. In addition, they found no statistical effects of the co-supplementation in the blood lactate levels, unlike the results found in our study, very possibly explained by the difference between the type and duration of the efforts. Similarly, de Salles Painelli, Roschel, De Jesus, Sale, Harris, Solis, Benatti, Gualano, Lancha Jr and Artioli (5) also observed no statistical effects of the co-supplementation on the 100-m and 200-m maximal swimming performances in junior-standard swimmers; despite this, the 100-m and 200-m performance times were about 1.4 and 0.63% faster in the co-supplemented group compared to the placebo group, respectively. Different from our study (since it was not our aim), both authors mentioned above also investigated the effects of isolates βA and SB supplementations in their studies. Alone, 4-week βA supplementation was not able to improve performance in 100-m and 200-m maximal efforts (5, 17), while SB acute intake was able to statistically mitigate the worsening of performance in two 100-m repeated maximal efforts (17) and improve single 200-m front-crawl swimming performance (5).

Although the co-supplementation was not able to improve neuromuscular fatigue levels and swimming performance in high-intensity intermittent efforts, a statistical increase of 25.06% in the blood lactate was observed in βA+SB but not in PL+PL ($p_{Bonf} < 0.001$), indicating a possible improvement in buffering capacity induced by the 4-week co-supplementation protocol. In similar high-intensity intermittent swimming protocols, isolated SB supplementation increased blood lactate levels by 16.47% in 8 × 50-m maximal front-crawl efforts every 5 min (10), 16.61% in 4 × 50m front-crawl maximal efforts with 1-min interval in-between (31), and 12.06% in 6 × 100-m maximal front-crawl efforts every 6 min (4). Although βA supplementation has not been

investigated alone in high-intensity intermittent swimming protocols, our results showed that the βA may have a possible additional effect on increasing blood lactate when combined with SB during swimming performance.

Although not assessing neuromuscular parameters, several studies have also investigated the βA and SB co-supplementation in different sports fields (1, 13, 23, 26). In cycling, the co-supplementation seems to be able to statistically increase the time to exhaustion during a high-intensity cycling capacity test (at 110% of maximum power) by 16.2% in physically active males; however, this result was not statistically additional to the improvement provided (12.1%) by the alone βA supplementation (23). In a 4-min maximal cycling effort, the co-supplementation could statistically improve the maximal power output by 3.3% in highly-trained cyclists; considered a statistically additional effect to alone βA supplementation, which in turn did not statistically improve performance in this test (1). In rowing, the co-supplementation had a possibly beneficial additional effect on 2000-m time trial performance in well-trained rowers; however, this effect was small compared to the very likely beneficial effect provided by alone β-alanine supplementation (13). In repeated sprint ability under hypoxia conditions (2,500 m altitude; %O2 = 15.5), the co-supplementation increased peak power output by 4.93% in 5 × 6-s maximal sprint performance on a non-motorized treadmill in recreationally active male games players; alone, βA supplementation did not influence the repeated sprint performance (26). Finally, even though different sports fields and experimental protocols have been investigated, and the co-supplementation be recommended during high-intensity exercises lasting between 30 s and 10 min, the possible ergogenic effects and neuromuscular responses of βA combined with SB are not yet fully understood (for a systematic review, see Gilsanz, López-Seoane, Jiménez and Pareja-Galeano (9)).

Since our study was a pioneer in investigating the ergogenic effects of βA plus SB co-supplementation on neuromuscular fatigue in swimming during high-intensity intermittent efforts, we understand that the resolution of some important limitations present in this study may be an interesting factor for future research with similar approaches. Firstly, our analysis of only 11 athletes (considering 1 drop-out due to muscle injury) divided into two groups was not segmented by gender or competitive level. In addition, once we study regional and national competitive-level swimmers, the investigation of elite competitive-level swimmers is necessary to also characterize the ergogenic effects of βA and SB on performance and neuromuscular responses in this athlete category. Furthermore, the investigation of co-supplementation on neuromuscular fatigue in middle-distance maximal efforts (*e.g.*, 400-m swimming performance), threshold, and maximal aerobic power-intensity intermittent efforts can also be interesting due to the time of exercise when the buffering effects through intramuscular carnosine seem to be most effective (~ 4 min) (25). Besides, the implementation of a taper period can be interesting for neuromuscular responses, since in most cases the supplementation is provided in high-training load during swimming periodization to take advantage of the glycolytic metabolic stimuli of the high-intensity periods, the intensified training can compromise the neuromuscular performance (7). Although a 4-week βA supplementation period seems to be sufficient for increasing the intramuscular carnosine content levels (11), this supplementation load does not

seem to be the best to provide consistent ergogenic effects, since new evidence has shown that peak intramuscular carnosine synthesis seems to occur within a 20-week supplementation period (24). Finally, in future studies, this neuromuscular assessment approach should also be investigated with isolated βA and SB supplementations.

Regarding the data mentioned above, we believe that our results can help athletes and swim coaches to make decisions from a practical perspective, both in relation to the ergogenic effects of the supplementation protocol to be chosen and the level of neuromuscular fatigue induced by high-intensity intermittent efforts. In conclusion, 4-week βA plus SB co-supplementation increases blood lactate but does not decrease neuromuscular fatigue and improves swimming performance in regional and national competitive-level young swimmers.

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