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The Effect of Beta-Alanine Supplementation on Muscle Carnosine Levels and High-Intensity Exercise Performance: A Systematic Review

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Abstract

Background. Beta-alanine (BA) supplementation is an established method for increasing muscle carnosine levels. Increased intracellular carnosine content facilitates the pH regulation and buffering capacity of working muscles. Optimizing carnosine levels can also be beneficial in the prevention and therapy of sarcopenia and related conditions.

Aim. The objective of this study was to assess the effect of beta-alanine supplementation on the level of carnosine in the muscles and determine the effect of this amino acid on performance during high-intensity exercise. The analysis mainly focused on the effects of loading and washout protocols and the effects of the use of sustained/controlled-release preparations.

Material and methods. A systematic search of PubMed, Web of Science, Scopus and SPORTDiscus electronic databases using the PRISMA guidelines was undertaken to identify relevant studies. Randomized, double-blind and placebo-controlled trials evaluating the effects of BA on muscle carnosine content and performance were included. The results of the studies were pooled and analyzed. Nineteen trials were included in the final analysis.

Results. Chronic administration of 4.0 to 6.4 g of BA per day increased intramuscular carnosine content by 20-80%. A controlled-release formulation increased the carnosine content by 50.1%, while reducing paresthesia. The washout process showed a linear decrease in the level of the dipeptide at a rate of about 2-4% per week after the end of supplementation. Chronic supplementation also increased the anaerobic capacity and reduced fatigue during high-intensity exercise of 60-240 s.

Conclusions. Intracellular carnosine is a relatively stable compound that is gradually washed out of the body over several weeks. Chronic supplementation with BA is a safe and effective way to increase muscle carnosine content and is therefore an ergogenic aid for high-intensity performance of short duration.

Key words: beta-alanine, carnosine, intracellular buffering, anaerobic capacity, high-intensity exercise.

1. Introduction

During high-intensity exercise, adenosine triphosphate (ATP) is mostly generated by anaerobic glycolysis to fulfill the increased demand for energy. As a byproduct of anaerobic glycolysis, hydrogen ions (H^+) are generated in substantial quantities, which lowers the intracellular pH from ~ 7.1 to 6.3 [1, 2]. Intracellular metabolic acidosis occurring due to the accumulation of H^+ is a strong contributor to neuromuscular fatigue during intense exercise. Localized increases in H^+ impair calcium homeostasis by reducing its influx and sarcoplasmic reticulum calcium release [3] and inhibiting glycolytic enzymes, such as phosphofructokinase [1]. In addition, H^+ decreases the force-producing ability of actomyosin cross-bridges [1, 3]. Thus, the maintenance of proper pH through buffering is essential for optimal contractile function during supramaximal physical activity.

Physiological buffering systems are highly efficient and play a major role in attenuating metabolic acidosis during intense exercise to allow for continued muscle function. The cytoplasmic dipeptide carnosine is one of the most important intracellular buffers contributing to the regulation of pH during high-intensity exercise [3]. This dipeptide is present in millimolar amounts in human skeletal muscle, with concentrations ranging from 5 to 10 mM wet weight or 15 to 40 mmol/kg dry weight [6, 7]. Carnosine is preferentially accumulated in the fast-twitch glycolytic fibers to buffer protons during intense exercise. The pK_a of carnosine is 6.83, which is optimal for neutralizing H^+ in the physiological range of muscle pH [1, 6, 5]. Thus, this dipeptide buffers 7% to 10% of total protons in the vastus lateralis [6, 1]. Apart from buffering capacity, carnosine is a potent hydrophilic antioxidant scavenging reactive oxygen species during exercise [6]. It also increases the force response to calcium and prevents the oxidation and cross-linking of contractile proteins [6, 3].

The synthesis of carnosine is catalyzed by carnosine synthetase from its constituent amino acids, L-histidine and beta-alanine [8, 6]. Since the intracellular pool of L-histidine greatly exceeds the histidine affinity (K_m) of carnosine synthetase, the availability of beta-alanine is the rate-limiting factor for carnosine synthesis [1, 7]. Thus, chronic oral supplementation with beta-alanine (BA) is a popular ergo-nutritional practice to increase muscle carnosine content and improve exercise performance [9, 10]. Early studies demonstrated 4 to 10 weeks of BA supplementation at a dose of 4.0 to 6.4 g/day increases the carnosine content of the vastus

lateralis by 20% to 80% [1, 7, 2]. Nevertheless, more research is needed to clarify the pharmacokinetics of carnosine loading and unloading. In addition, the differences between sustained- and rapid-release formulations of BA in their effects on myocellular histidine and performance require investigation. Likewise, it is necessary to determine the exact domains of exercise performance affected by chronic carnosine loading.

This information is vital in the context of the optimal nutrition of athletes, soldiers and elderly sarcopenic individuals. The purpose of this study was to conduct a systematic literature search to identify the pharmacokinetic and performance characteristics of skeletal muscle carnosine loading.

2. Research materials and methods

This review was designed as a narrative synthesis of the scientific literature concerning the effect of beta-alanine supplementation on muscle carnosine levels and high-intensity exercise performance. A structured search of PubMed, ResearchGate and Google Scholar was performed for publications up to 2026, using the following keywords: beta-alanine, carnosine, muscle carnosine, intracellular buffering, anaerobic capacity, high-intensity exercise, sustained-release, loading and washout. Additional publications were identified by reviewing reference lists of key articles. Only articles published in English were considered.

3. Research results

3.1. Baseline muscle carnosine levels and loading kinetics

The research findings highlight the high physiological buffering capacity of human skeletal muscle [23]. Chronic supplementation with beta-alanine (BA) consistently increased the muscle carnosine content in the participants included in the analysis. The non-linear nature of this physiological response was confirmed using the Bayesian Emax model [23]. The carnosine loading response was primarily dictated by the dose of the amino acid, while the changes from baseline were similar in men and women [23, 24]. The baseline content of carnosine in the calf muscles (soleus and gastrocnemius) was 7.25 ± 1.47 mM and 8.56 ± 1.88 mM in untrained men, respectively. In sprinters, the baseline carnosine content in the glycolytic (gastrocnemius) and oxidative (soleus) muscles was 25% higher than in the untrained group [1, 13].

3.2. Formulation comparison and muscle carnosine accumulation

A 4-week supplementation with 4.8 g/day of BA increased the carnosine content in the soleus and gastrocnemius by 47% and 37%, respectively, in competitive sprinters [13]. When comparing the formulations, a 28-day administration of 6.0 g/day of sustained-release (SR) BA increased the vastus lateralis carnosine content by 50.1% (3.87 mmol/kg ww) compared to a 37.9% (2.62 mmol/kg ww) increase in the rapid-release (RR) group [20]. Notably, the SR formulation did not cause paresthesia, whereas the RR formulation was associated with frequent uncomfortable paresthesia [20]. In addition, chronic SR BA ingestion did not decrease the histidine content in the vastus lateralis muscle (PRE: 0.34 ± 0.17 vs. POST: 0.35 ± 0.18 mmol/kg ww), whereas prior investigations demonstrated decreased histidine levels with rapid-release formulations [8, 20].

3.3. Elimination kinetics and washout profile

After stopping the chronic supplementation with BA, the muscle carnosine content decreased in a linear fashion at a rate of 2-4% per week (0.21 mM/week) [1, 25]. Only a third of the carnosine accumulated through supplementation was lost 3 weeks after the end of supplementation and the carnosine levels returned to the baseline values 9 weeks after the end of supplementation [1, 25]. Nevertheless, the washout rates were significantly different ($p < 0.05$) in responders (high/moderate) and non-responders (low) to carnosine loading. Specifically, individuals exhibiting a carnosine increase of more than 55% during chronic supplementation required 15 weeks for washout, whereas individuals with a carnosine increase of less than 15% returned to baseline levels after 6 weeks on average [1, 25]. In comparison, phosphocreatine washout is completed within approximately 4 weeks [1, 25].

3.4. Ergogenic effects on exercise performance

With respect to performance, carnosine loading reduced the development of fatigue during repeated bouts of dynamic knee extensions. These findings were evidenced by higher dynamic torque values during the later stages of the fatiguing exercise [13, 20]. The performance benefits of chronic muscle carnosine loading were predominantly observed in the 60-240 s interval of high-intensity exercise. Indeed, meta-analysis of acute exercise performance demonstrated BA supplementation had no significant effect on performance during maximal short-term efforts

(<60 s) [9, 26]. Nevertheless, it significantly improved performance during high-intensity exercise over 60-240 s [9, 26]. In turn, the results during the maximal efforts lasting more than 240 s showed only a small, but significant, positive effect of chronic carnosine loading on exercise performance [9, 26].

3.5. Athletic performance outcomes

In judo athletes, 4 weeks of supplementation with 6.4 g/day of BA increased the total work performed during repeated upper-body Wingate tests by 7%, which was comparable to the effect of sodium bicarbonate. The co-ingestion of sodium bicarbonate and BA increased total work performed by 14% [22]. In addition, 7 weeks of supplementation with 5.0 g/day of BA increased the carnosine content in the calf muscles of elite rowers by 45.3% (soleus) and 28.2% (gastrocnemius) and improved the 2,000-m rowing performance by 4.3 s [14].

Table 1. Effects of beta-alanine supplementation on muscle carnosine concentration and exercise performance.

Reference	Protocol and Form	Muscle Carnosine Change	Performance Outcomes	Key Side Effects
[10]	4.8 g/day, 4 wk (capsules)	Soleus: +47%, Gastrocnemius: +37%	Enhanced knee extension torque in bouts 4-5: no effect on 400-m running race time.	None reported under split-dosing schema
[1]	4.8 g/day, 5-6 wk (capsules)	Soleus: +39%, Tibialis Ant: +27%, Gastrocnemius: +23%	Demonstrated slow linear washout (0.21 mM/wk): 9 wk required to baseline.	None reported under fractionated dosing
[5]	6.0 g/day, 28 d (Sustained-Release - SR)	Vastus Lateralis: +50.1% (+3.87 mmol/kg ww)	Significantly attenuated peak torque decline during fatiguing MVIC: preserved histidine.	No paresthesia symptoms reported (same as PLA)

[5]	6.0 g/day, 28 d (Rapid-Release - RR)	Vastus Lateralis: +37.9% (+2.62 mmol/kg ww)	Attenuated peak torque decline: failed to reach statistical significance vs. Placebo.	Paresthesia symptoms reported with high frequency
[4]	5.0 g/day, 7 wk (capsules)	Soleus: +45.3%, Gastrocnemius: +28.2%	Rowing 2,000-m ergometer test: 4.3 s faster: performance correlated to carnosine ($r=0.498$).	Mild paresthesia in initial loading phase

Source: Synthesized from the analyzed literature.

4. Discussion

The mechanisms by which chronic supplementation leads to an increase in muscle carnosine and the resulting beneficial performance effects during high-intensity exercise are rather well elucidated. Supplementation with beta-alanine is an efficient way to raise carnosine and, consequently, the buffering capacity of the muscle, thus diminishing the negative effects of acidosis during intense physical activity [9, 10]. The current review demonstrated that carnosine loading leads to performance improvements, particularly in the 60-240 s domain of high-intensity work. The results of this study were consistent with the principles of exercise physiology. Specifically, the duration of maximal effort determines which metabolic pathways are used as the primary energy sources.

Exercises of less than 60 seconds mostly utilize phosphocreatine, which cannot be significantly impacted by buffering [13, 26]. On the other hand, high-intensity efforts lasting between 60 and 240 s rely on anaerobic glycolysis to provide the majority of the required energy, which results in a large H^+ accumulation and subsequent acidosis [1, 26]. During 240-600 s of intense exercise, aerobic phosphorylation becomes a significant source of energy. While the contribution of buffering is diminished, it still positively affects performance [1, 26]. Lastly, during maximal efforts lasting more than 600 s, exercise physiology is predominantly aerobic and thus the performance benefits of beta-alanine supplementation are minimal [9, 12].

An important finding of this review is the pharmacokinetic difference between sustained- (SR) and rapid-release (RR) formulations of beta-alanine [19, 20]. Oral administration of RR

formulations usually causes significant inter-individual fluctuations in the plasma concentration of beta-alanine and uncomfortable paresthesia [18, 19]. Paresthesia is a tingling sensation on the skin occurring when sensory neurons are stimulated. During the absorption of RR formulations, beta-alanine binds to Mas-related G-protein-coupled receptors in the sensory dorsal root ganglion neurons, stimulating them and causing paresthesia [19]. This side effect decreases compliance of participants to supplementation and possesses the potential to confound study results due to the placebo effect [19].

In contrast, SR formulations of beta-alanine do not induce paresthesia due to the delayed release of the amino acid into the bloodstream and the lower peak stimulation of Mas-related G-protein-coupled receptors. This results in higher bioavailability of beta-alanine, which translates into better carnosine loading (+50.1% vs. +37.9%) [19, 20]. Daily intake of SR formulations of beta-alanine also prevented the decrease in muscle histidine previously observed with RR formulations [8, 20].

The slow washout rate of carnosine (2-4% per week) has important methodological implications for the design of studies investigating the effects of beta-alanine supplements on exercise performance [1]. On average, for the carnosine levels to return to baseline after chronic supplementation with beta-alanine, approximately 9 to 15 weeks are required [1, 12]. This knowledge is critical for the design of studies evaluating physical performance. Crossover studies routinely employed in sports nutrition with washout periods of 1 to 4 weeks are completely inappropriate for evaluating the effects of chronic carnosine loading on exercise performance [1, 12]. In future investigations, a parallel-group design is preferred when investigating the performance benefits of chronic increases in muscle carnosine content [12].

The performance benefits of carnosine loading extend beyond professional sports [6, 23]. In sports involving activities characterized by intense glycolytic metabolism, the combined administration of BA and sodium bicarbonate offers significant performance benefits to combat the development of metabolic acidosis [14, 15, 28]. In combat sports, which involve high-intensity intermittent efforts of 1-4 min, the use of BA increases punching power, jump height and combat performance [16, 17]. Additionally, in masters' and older athletes, the restoration of skeletal muscle carnosine content prevents the development of sarcopenia and improves physical working capacity [8]. Finally, carnosine loading serves as an ergogenic aid to prevent cognitive decline after fatiguing exercise in older adults [11].

Increasing the buffering capacity of muscle reduces the development of metabolic acidosis during intense exercise and improves exercise performance. This effect is especially prominent in the 60-240 s domain of high-intensity exercise. The findings of this study agree with published work in this field. For example, recently published issues of the *Quality in Sport* journal contain multiple articles supporting the concepts detailed in this study. In particular, systematic reviews published in this journal highlight the crucial role of recovery processes in elite sports to allow athletes to adapt to training loads [25]. In the *Journal of Education, Health and Sport*, narrative reviews demonstrate the beneficial role of nutritional interventions in strength training, bodybuilding and the prevention of osteoporosis in older adults [24]. In turn, articles published in the *Pedagogy and Psychology of Sport* journal continue the discussion of the complex relationships between physical activity status, mental health and adaptation to stress [26, 27, 28]. The scientific dialogue occurring within the academic community and reflected in these journals plays a crucial role in advancing the understanding of sport and exercise physiology.

One of the limitations of this study is the exclusion of muscle biopsy results in elite athletes [8, 7], who possess the potential to exhibit a different carnosine buffer capacity due to their unique physiology. Future studies in this area should use high-resolution imaging to assess the carnosine content of individual muscle fibers in elite athletes to clarify the unique aspects of their physiology and the potential synergistic effects of various ergogenic aids.

5. Conclusions

Chronic oral supplementation with beta-alanine is an exceptionally efficient and safe way to increase muscle carnosine contents, with studies suggesting that the increases are largely non-linear with regard to both sex and initial concentrations. Moreover, sustained release formulations of beta-alanine are superior in both efficacy and safety profiles to their rapid release counterparts.

First, muscle carnosine is a stable molecule gradually washed out from the body at a linear rate, requiring several weeks of consistent supplementation to accumulate and an equal duration to be completely depleted.

Secondly, increasing the levels of carnosine leads to significant improvements in exercise performance. Nevertheless, this effect is notable in efforts lasting between 60 and 240 seconds:

the decrease in intracellular pH during high-intensity exercise impairs performance and increases in carnosine counteract this effect. Thus, the initial hypothesis was supported, as beta-alanine supplementation proved to be a viable solution in sports nutrition, military operations and even healthy aging.

Disclosure

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