Research in Exercise Nutrition
ISSN: 2980-8960

RESEARCH ARTICLE

Research in Exercise Nutrition 5(1), 47-54

Short-Term Beta-Alanine and Markers of Muscle Damage after Eccentric versus Concentric Exercise in Trained Men

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Open Access

REN

Submitted:
19 January 2026
Revised:
08 March 2026
Accepted:
09 March 2026
Published:
09 March 2026

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Research in Exercise Nutrition:

2026, Volume 5 (Issue1), Pages 47-54

Publisher: **University of Kurdistan**

ISSN: 2980-8960

Abstract

Introduction: Eccentric exercise induces muscle damage, delayed-onset muscle soreness (DOMS), and release of calcitonin gene-related peptide (CGRP), a neurogenic mediator of pain and inflammation. We evaluated the effect of short-term beta-alanine (BA) supplementation on serum CGRP, DOMS, and rating of perceived exertion (RPE) after a single eccentric/concentric resistance exercise session.

Methods: In this counterbalanced crossover study, 20 resistance-trained men (20–25 years) completed two resistance protocols (eccentric- or concentric-emphasised, 3:1 tempo) 2 weeks apart. BA or placebo was ingested for three days before each session (single-blind; last dose 60 min pre-exercise). Serum CGRPα (ELISA), DOMS (VAS), and RPE (0–10) were assessed up to 48 h post-exercise (two-way repeated-measures ANOVA).

Results: CGRPα 24 h post-exercise was lower with BA than placebo ($p = 0.001$) and higher after eccentric than concentric contractions ($p = 0.01$); it rose 24 and 48 h after eccentric exercise ($p = 0.001$). DOMS and RPE at 24 and 48 h were lower with BA than placebo ($p = 0.001$).

Conclusions: Short-term BA supplementation attenuated the CGRP response, soreness, and perceived exertion after intense resistance exercise, more markedly after eccentric contractions, possibly via improved buffering of acidity.

Key Words: Eccentric contraction; Neurogenic inflammation; Carnosine; Muscle pain; Resistance training

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Citation:
Nourian E, Mehrabani J. Effect of short-term beta-alanine supplementation on CGRP, muscle soreness, and perceived exertion after an eccentric/concentric resistance exercise in strength-trained men. **Research in Exercise Nutrition.** 2026 Mar 21;5(1), 47-54**.** Doi: https://doi.org/10.22034/ren.2026.145517.1160

1. Introduction

Resistance exercise with a heavy training load, particularly exercise involving intense eccentric phases, is a strong stimulus for favourable muscular adaptations. However, such exercise can cause exercise-induced muscle damage (EIMD). A key indicator of EIMD is delayed-onset muscle soreness (DOMS), which usually peaks 24–72 h after exercise and can impair athletic performance and quality of life [1]. Dynamic resistance exercise comprises eccentric and concentric phases that, through changes in the muscle length–tension relationship, cause micro-level cellular and molecular damage, resulting in pain and inflammation and, with continued training, increased muscle mass. During the eccentric phase the muscle produces force while being lengthened, which imposes very high mechanical tension per unit of cross-sectional area, particularly on non-contractile components, compared with concentric contractions [2,3].

This condition leads to inflammation in muscle tissue. Among the inflammatory factors, calcitonin gene-related peptide (CGRP) has attracted attention as a key neurogenic mediator of inflammation and of the transmission of pain signals after tissue injury [4]. CGRP is reported to be released in response to muscle damage induced by eccentric exercise and, together with pain, may serve as a sensitive biomarker of the metabolic and inflammatory strain imposed by training. Under such circumstances, sport supplements may reduce pain and damage-related factors [2]. Beta-alanine (BA) supplementation increases muscle carnosine concentration [2,5] and improves performance [6]. Carnosine also has antioxidant and calcium-regulating properties [2] that may reduce oxidative stress and possibly modulate exercise-induced inflammatory and pain signals. Although the positive effect of chronic BA supplementation (>4 weeks) on performance in 1–4 min activities is clearly established [3], the literature on its effect on indices of muscle damage and pain, particularly with acute dosing, is inconsistent: some studies report reductions in markers such as creatine kinase (CK) or in pain [6], whereas others found no significant difference from placebo [7]. This heterogeneity may reflect differences in the supplementation design (chronic vs. acute), the type and intensity of the exercise protocol, and sample characteristics.

Despite a growing understanding of the role of CGRP in the regulation of pain and inflammation [8,9] and evidence on the buffering effects of BA, a considerable gap exists between these two fields. The central question is whether short-term, acute BA intervention can modulate the neuro-inflammatory response to an intense muscular challenge such as a single eccentric/concentric session. Eccentric exercise, even in trained individuals, is reported to impose greater metabolic and mechanical tension than the concentric phase, increasing muscle cell damage, acute inflammation, related markers, and muscle pain. It is therefore unclear whether increasing muscle buffering capacity through acute BA intake can modulate post-exercise CGRP release, reduce DOMS, and lower the rating of perceived exertion (RPE) during intense exercise. Most previous studies have focused on direct performance indices (e.g., strength) or common markers of muscle damage such as CK [10,11]; to our knowledge, the concurrent examination of the neuro-inflammatory pain axis (CGRP) together with perceived exertion and pain is novel. RPE, a valid subjective scale of perceived work intensity that can be influenced by metabolic factors such as acidosis, may help clarify the link between physiological changes and the perception of training strain.

Accordingly, the present study was designed to investigate the effect of short-term beta-alanine supplementation on the serum CGRP response, DOMS severity, and perceived exertion after a single eccentric/concentric resistance exercise session in resistance-trained men.

2. Methods

2.1. Participants

Twenty young male athletes (age range 20–25 years) participated in this study (Table 1). Sample size was estimated with G*Power software (see Section 2.3). Volunteers active in bodybuilding gyms who met the inclusion criteria were invited to participate. The inclusion criteria were: good general physical health, assessed with a medical–sports questionnaire; no muscle or joint injury; at least six months of resistance-training experience; normal body weight and body mass index (BMI); body fat below 18%; and no use of medication, caffeine-containing substances, or dietary supplements. After the initial assessments

and completion of the American College of Sports Medicine (ACSM) health questionnaire, participants were randomly allocated to two groups.

2.2. Study design

This was a semi-experimental study with a counterbalanced crossover design. All participants took part in two separate resistance exercise protocols separated by a 2-week rest/washout period (Figure 1). The protocols were designed so that the nature of muscle contraction was the main manipulated factor. Supplementation (beta-alanine or placebo) was administered over the three days leading up to each exercise session, and the last dose was taken 60 min before the resistance protocol. Rating of perceived exertion and muscle soreness were assessed immediately after exercise and at 24 and 48 h; blood samples were taken at baseline (after a period without training), after the supplementation period (pre-exercise), and 24 and 48 h after exercise. The controlled manipulation of contraction type allowed the effects of each contraction type to be examined separately within a counterbalanced design [1].

2.3. Sample size calculation

The sample size was estimated using G*Power software, yielding a total of 20 participants.

2.4. Nutritional intervention / Supplementation protocol

Supplementation was acute and was carried out over the three days preceding each exercise session. Participants received daily 4 g/kg body weight [12] of beta-alanine (CarnoSyn®, NAI, California, USA) or placebo (capsules containing chickpea flour) in a single-blind manner [2]. The last dose was consumed 60 min before the resistance exercise protocol.

2.5. Exercise protocol

Both protocols were performed as follows: three sets of 16 repetitions at 80% of one-repetition maximum (1-RM) and one set at 50% 1-RM to failure (Figure 1). In the first protocol, the main movement was performed with a 3:1 time ratio favouring the eccentric phase relative to the concentric phase (eccentric exercise). In the second protocol, the same ratio was reversed, i.e., 3:1 in favour of the concentric phase (concentric exercise).

2.6. Outcome measurements

Anthropometric characteristics were measured and recorded before the interventions. Height was measured with a wall-mounted seca stadiometer to the nearest 0.1 cm, with participants standing barefoot, with their back to the wall, looking straight ahead. Body weight, BMI, and body fat percentage were measured by bioelectrical impedance analysis (BIA) with an InBody 770 body composition analyser (South Korea) after standardisation of conditions (controlled hydration, relative fasting, and bladder emptying), with participants barefoot and in minimal clothing.

Rating of perceived exertion (RPE): Immediately after completion of both resistance protocols, and 24 and 48 h later, after performing full back squat and machine leg extension movements, RPE was measured using the 0–10 scale specific to resistance exercise. This valid, commonly used pictorial-numerical scale quantifies the exertion that the athlete perceives. Participants chose a number from 0 (very, very easy) to 10 (very, very hard) corresponding to their sensation. Recording RPE immediately after exercise prevents loss of measurement validity due to forgetting.

Muscle soreness (visual analogue scale, VAS): Muscle pain intensity was assessed immediately after the exercise session and at 24 and 48 h using a 100-mm horizontal VAS whose endpoints were defined as no pain (score 0) and worst possible pain (score 100). Participants marked their pain on the line, and the distance from the start of the line to the mark gave the quantitative pain score.

Calcitonin gene-related peptide (CGRP): For a more objective evaluation of muscle damage and inflammation, serum CGRP was measured. Blood sampling was performed at four time points: baseline (after the period without training), after the supplementation period (pre-exercise), and 24 and 48 h after the exercise protocol (before the full back squat and machine leg extension tests). A 5-mL blood sample was drawn from the brachial vein of each participant. To preserve sample integrity, the separated sera were immediately frozen at −80 °C, and all analyses were performed within a short period after collection. For accuracy, all samples were measured in duplicate and the mean was recorded. Serum CGRP was measured by sandwich ELISA with a Zellbio kit (Germany) with a sensitivity coefficient of 0.96 and an intra-assay coefficient of variation (CV) below 8%. The α isoform was measured.

2.7. Dietary control and physical activity standardization

Participants' diet was monitored using a 24-h dietary recall questionnaire, and on the test day a standardized breakfast was provided (295 kcal: 16.7% protein, 23.5% fat, 59.9% carbohydrate).

2.8. Statistical analysis

The Shapiro–Wilk test was used to examine the normality of data distribution. Two-way repeated-measures analysis of variance (ANOVA) was used to

 https://doi.org/10.22034/ren.2026.145517.1160

examine the interaction effects of resistance exercise (two conditions: eccentric/concentric) and supplement (two conditions: beta-alanine/placebo) on the dependent variables at the different measurement times (three or four time points). When a significant main or interaction effect was observed, the Bonferroni post hoc test was used for pairwise comparisons. The significance level was set at $p < 0.05$. Data were analysed with SPSS version 26.

3. Results

3.1. Participants' characteristics

The characteristics of the participants are presented in Table 1.

3.2. Primary outcome: CGRPa

Changes in CGRPα under beta-alanine or placebo conditions in the eccentric/concentric contraction conditions are shown in Figure 2. Serum CGRPα 24 h after exercise was lower with beta-alanine than with placebo ($p = 0.001$). In both supplementation conditions, CGRPα values were higher after eccentric than after concentric contractions ($p = 0.01$).

3.3. Secondary outcomes: RPE and muscle soreness

RPE and muscle soreness (VAS) at the different measurement times under beta-alanine or placebo in the eccentric/concentric conditions are presented in Table 2. Pain and RPE 24 and 48 h after exercise were lower with beta-alanine than with placebo ($p = 0.001$). In both supplementation conditions, the levels of these two variables were lower after eccentric than after concentric contractions ($p = 0.01$).

Table 1. Characteristics of the participants (mean ± SD, n = 20).

Variable	Mean ± SD
Age (years)	23.17 ± 2.48
Height (cm)	173.35 ± 5.67
Body weight (kg)	71.84 ± 4.29
Body mass index (kg/m²)	22.53 ± 1.88
Body fat (%)	16.33 ± 2.01
Training experience (months)	9.14 ± 2.23

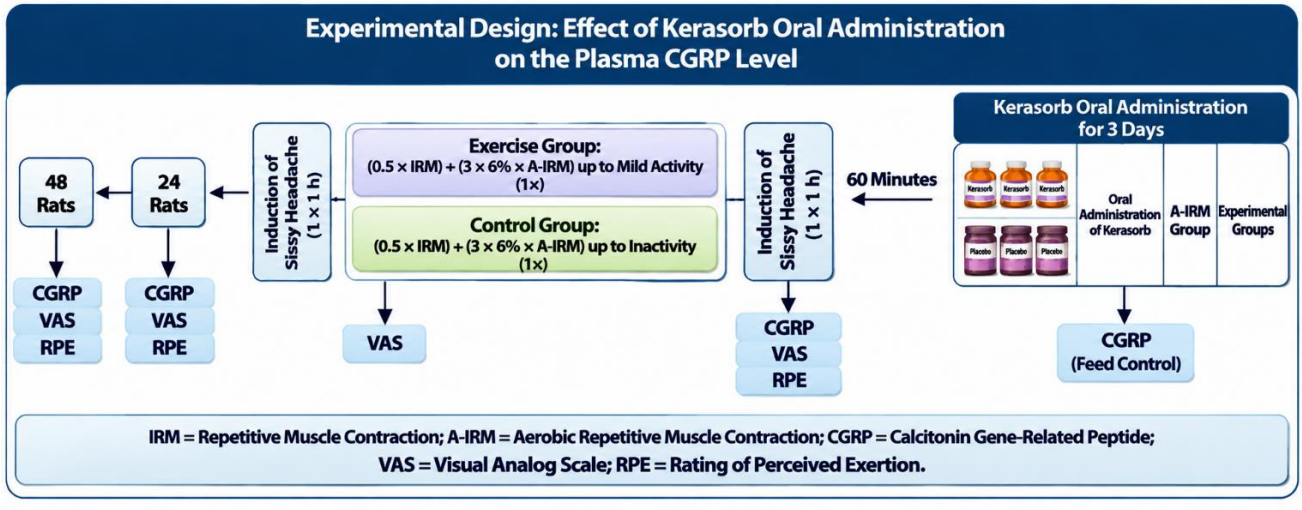


Figure 1. *Study design and experimental protocol.*

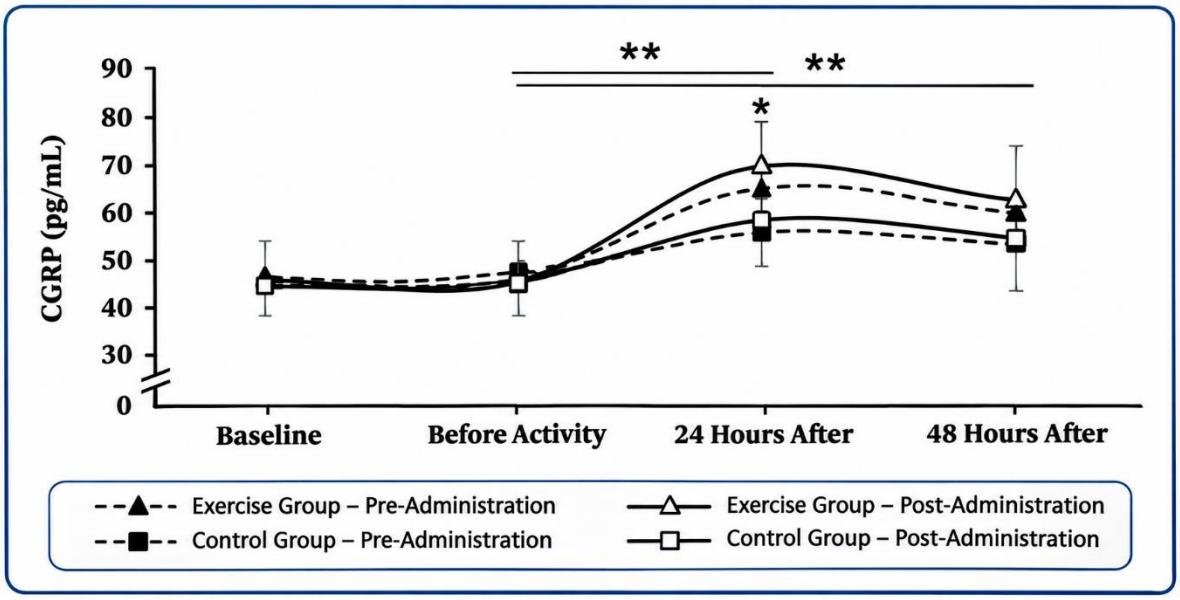


Figure 2*. Serum CGRPα values. *Significant difference 24 h after exercise (p = 0.01) between beta-alanine and placebo conditions; **significant increase 24 and 48 h after eccentric exercise relative to pre-exercise values (p = 0.001).*

Table 2. Rating of perceived exertion (RPE) and muscle soreness (VAS) at the different measurement times (mean ± SD).

Supplement	Contraction	Time point	RPE (0–10)	VAS (0–100 mm)
Beta-alanine	Eccentric	Immediately post	8.9 ± 0.48	66.14 ± 7.75
		24 h post	9.2 ± 0.36*	80.28 ± 5.36*
		48 h post	9.3 ± 0.55*	75.86 ± 8.43**
	Concentric	Immediately post	8.8 ± 0.28	68.71 ± 6.92
		24 h post	8.9 ± 0.71*	77.34 ± 6.57**
		48 h post	8.5 ± 0.43*	72.68 ± 8.43
Placebo	Eccentric	Immediately post	8.9 ± 0.59	67.25 ± 6.39
		24 h post	9.4 ± 0.61*	82.12 ± 8.81
		48 h post	9.3 ± 0.39**	77.09 ± 7.43**
	Concentric	Immediately post	8.9 ± 0.74	69.44 ± 7.63
		24 h post	9.0 ± 0.45	79.51 ± 5.09
		48 h post	9.1 ± 0.47	78.36 ± 7.34

*Significant difference 24 h after exercise (p = 0.01) between beta-alanine and placebo conditions; **significant increase 24 and 48 h after eccentric exercise relative to pre-exercise values (p = 0.001).

4. Discussion

Calcitonin gene-related peptide (CGRP) is a key neuropeptide of the sensory nervous system that plays an important and complex role in the physiology and pathophysiology of the body's response to various stimuli, including intense exercise. Released from type C nerve terminals, it is a highly potent vasodilator and also participates in inflammation, pain transmission, and tissue repair. The aim of the present study was to examine the acute effect of a single intense resistance exercise session, with emphasis on different phases of muscle contraction (eccentric and concentric), and of beta-alanine supplementation on serum CGRP levels in young athletes. The results showed that CGRP values 24 and 48 h after exercise were higher than pre-exercise values in the eccentric protocol.

The findings showed that a single intense resistance exercise session, irrespective of the type of contraction protocol, significantly increased serum CGRP levels relative to baseline. This finding is consistent with the nature and probable role of CGRP as a mediator in response to stress and injurious stimuli [13]. The mechanism of this increase is mainly

 https://doi.org/10.22034/ren.2026.145517.1160

activation of TRPV1 and TRPA1 receptors on sensory neurons. During intense exercise, factors such as the accumulation of metabolites like hydrogen ions (reduced local pH) and lactate, increased tissue temperature, and direct mechanical tension on muscle fibres and nerve terminals activate these channels. Their activation causes calcium influx into the cell and, ultimately, release of CGRP from storage vesicles in nerve terminals into the circulation [14]. This rapid physiological response is part of the body's adaptive and protective mechanisms; for example, the potent vasodilatory effect of CGRP may contribute to increased blood supply to active muscles during exercise [13]. Other research evidence supports this acute response pattern: an animal study showed that even a single acute endurance exercise session significantly increased CGRP concentrations in serum and cerebrospinal fluid [15]. Overall, it can probably be concluded that CGRP increases after exercise, indicating involvement of the neuro-sensory system in the response to the metabolic and mechanical stresses of exercise.

One finding of this study was a different pattern of CGRP change in response to the contraction types. The acute protocol emphasising eccentric contraction produced the largest increase in CGRP in blood samples taken 24 and 48 h after exercise, whereas the concentric protocol showed a smaller peak, generally occurring 24 h after exercise. This difference can be explained by the different biomechanics and greater tissue damage resulting from eccentric contractions [16]. In the eccentric phase, the muscle produces force while being lengthened, imposing very high mechanical tension per unit of cross-sectional area, particularly on non-contractile components (such as the sarcolemma, titin, and actin and myosin filaments), which causes greater microscopic damage to muscle fibre structure than concentric contractions [16]. Tissue damage is a strong stimulus for inflammatory processes. Local inflammation is accompanied by the release of cytokines and growth factors and by an acidic environment (reduced pH), all of which can act as additional stimuli for activating sensory nerve terminals and for greater and more prolonged CGRP release. Therefore, the delayed and more pronounced rise in CGRP after eccentric exercise probably reflects greater muscle damage and a more complex inflammatory response to this type of contraction [17]. This finding underlines the importance of considering the nature of contraction in training programming and recovery management, particularly to minimise delayed-onset muscle soreness (DOMS).

Examination of CGRP levels at different times after exercise (24 and 48 h) revealed a dynamic response pattern of this neuropeptide. In all conditions, CGRP peaked 24 h after exercise, coinciding with the VAS report. This coincidence reinforces the potential role of CGRP as an important mediator in the acute phase of post-exercise pain and inflammation [17]. After this peak, CGRP levels began to decline at 48 h, although the decline was less steep after eccentric exercise. This time course is consistent with the known kinetics of muscle damage and repair. Continued but decreasing CGRP secretion may contribute to the repair process: evidence suggests that, in addition to its vasodilatory effects, CGRP has immunomodulatory properties and can affect the function of immune cells such as macrophages [17]. This neuropeptide can also stimulate fibroblast proliferation and increase angiogenesis, both of which are essential for repair of damaged tissue [13]. Therefore, the longer elevation of CGRP after eccentric exercise may not only reflect greater damage but may also be part of a complex adaptive response coordinating the repair and remodelling of damaged tissue.

The present study showed that beta-alanine supplementation for three days before the exercise session had a considerable modulating effect on the CGRP response. With beta-alanine, the increase in CGRP at the post-exercise measurement times (24 and 48 h) was smaller than with placebo. This effect was observed in both exercise protocols but was statistically more prominent in the eccentric protocol. The probable mechanism can be sought in the main function of beta-alanine, the precursor for intramuscular carnosine synthesis [18]. Carnosine acts as an important intracellular buffer and increases the muscle's buffering capacity against H^+ produced during anaerobic glycolysis [7,18]; however, it was not assessed in the present study, and it is suggested that future studies address it. By reducing H^+ accumulation, the intramuscular metabolic acidosis induced by intense exercise decreases. Because a fall in pH (acidification of the environment) is one of the strong and direct stimuli for activation of the TRPV1 receptor and subsequent CGRP release, beta-alanine, by reducing this principal stimulus, can indirectly prevent excessive CGRP release [13]. This finding is of practical importance because it suggests that beta-alanine supplementation may be useful not only for improving performance and delaying fatigue but also as a strategy to modulate the inflammatory response and neuromuscular stress of very intense training sessions, particularly those with a high eccentric component.

This study has limitations. Muscle carnosine content and intramuscular pH were not measured, so the proposed buffering mechanism remains inferential; participants were young resistance-trained men followed for only 48 h; the supplementation was single-blind; and other inflammatory and muscle damage markers were not measured concurrently. Future studies with longer follow-up, concurrent

measurement of other inflammatory and damage markers, and determination of the optimal dose and duration of beta-alanine supplementation are recommended.

5. Conclusion

Intense resistance exercise is a strong stimulus for increasing serum levels of the neuropeptide CGRP. This response depends on the nature of muscle contraction: eccentric contractions, because they cause greater structural damage, produce a more prolonged CGRP increase. Short-term beta-alanine supplementation attenuated the CGRP response, probably through mechanisms related to buffering of acidity. These findings emphasise the complexity of the neuromuscular response to exercise and the importance of considering contraction type and nutritional interventions in training programming and recovery strategies.

6. Highlights

1. Short-term beta-alanine supplementation reduced the CGRP rise, an inflammatory marker, more after intense eccentric than after concentric contractions.

2. Muscle pain during recovery was also reduced more in the eccentric mode than in the concentric mode.

Statements

Funding

No funding was received for this study.

Acknowledgments

The authors thank the participants who took part in this study.

Conflicts of Interest

The authors declare no conflict of interest.

Authors' Contributions

All authors contributed equally to this work.

Supplementary Materials

Not applicable.

Data Availability Statement

Data are available from the corresponding author upon reasonable request.

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