MUSCLE CARnosine loading by Beta-alanine supplementation is
more pronounced in trained vs. untrained muscles

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B.A. B.T. had full access to all the data in the study and takes responsibility for the integrity of the
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Running Head: Training and muscle carnosine loading

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ABSTRACT

Purpose. Carnosine occurs in high concentrations in human skeletal muscle and assists working capacity during high-intensity exercise. Chronic beta-alanine (BA) supplementation has consistently been shown to augment muscle carnosine concentration, but the effect of training on the carnosine loading efficiency is poorly understood. The aim of the present study was to compare muscle carnosine loading between trained and untrained arm and leg muscles.

Methods. In a first study (n=17), reliability of carnosine quantification by proton magnetic resonance spectroscopy (1H-MRS) was evaluated in deltoid and triceps brachii muscles. In a second study, participants (n=35; 10 nonathletes, 10 cyclists, 10 swimmers and 5 kayakers) were supplemented with 6.4g/day of slow-release BA for 23 days. Carnosine content was evaluated in soleus, gastrocnemius medialis and deltoid muscles by 1H-MRS. All the results are reported as arbitrary units.

Results. In the nonathletes, BA supplementation increased carnosine content by 47% in the arm and 33% in the leg muscles (NS). In kayakers, the increase was more pronounced in arm (deltoid) vs. leg (soleus + gastrocnemius) muscles (0.089 vs. 0.049), whereas the reverse pattern was observed in cyclists (0.065 vs. 0.084). Swimmers had significantly higher increase in carnosine in both deltoid (0.107 vs. 0.065) and gastrocnemius muscle (0.082 vs. 0.051) compared to nonathletes.

Conclusions. We showed that 1) carnosine content can be reliably measured by 1H-MRS in deltoid muscle, 2) carnosine loading is equally effective in arm vs. leg muscles of nonathletes, and 3) carnosine loading is more pronounced in trained versus untrained muscles.

KEYWORDS: histidine-containing dipeptides, muscle contractions, sport supplements
INTRODUCTION

There is a growing interest in the molecule carnosine and its role in skeletal muscle in the field of exercise physiology (1, 6, 23). Carnosine (β-alanyl-L-histidine) is a dipeptide synthesized from the precursors L-histidine and beta-alanine (BA) by carnosine synthase (5). It is stored in high concentrations in human skeletal muscle (~5 mmol/kg wet muscle) (6). The chronic oral ingestion of BA, the rate-limiting precursor in carnosine synthesis, has been shown to elevate the muscle carnosine content by 40 – 80 % (2, 4, 7, 13, 14, 26). Interestingly, the muscle carnosine loading strategy is ergogenic in high-intensity exercise (for meta-analysis see Hobson et al. (15)), which is likely related to its function in skeletal muscle, as a proton buffer (3) and calcium regulator (8).

Since augmented muscle carnosine has many applications in sports and possibly also in health (10, 22), it is necessary to generate a better understanding of the determinants of muscle carnosine loading. First, the absolute increase in muscle carnosine is strongly dependent upon the total BA amount supplemented over a certain period of time (26). Meanwhile, Stegen et al. (25) demonstrated that coingestion of BA with meals can beneficially influence muscle carnosine loading, suggesting that insulin could play a role in muscle carnosine loading.

It could be hypothesized that exercise training can also facilitate muscle carnosine loading, by analogy with creatine, another popular nutritional supplement, for which a higher increase in creatine concentration was demonstrated in the trained compared to the untrained leg after creatine supplementation (12, 21). In the latter study, one-legged training consisted of 1 hour hard exercise per day for 7 days. To date, the effect of concomitant training during BA supplementation on the carnosine loading efficiency is poorly understood. Only one training study is available (17), which found that carnosine loading after BA supplementation was not influenced by a limited volume of one-legged isokinetic training, consisting of 10 x 10 maximal isokinetic contractions, 3 to 4 training sessions per week, over 4 weeks (17).

The present study aims to explore the effects of training on BA-induced muscle carnosine loading in an observational rather than interventional design. For this purpose we used a novel training study
approach, in which we investigated the effects of BA on carnosine content in arms and legs in
nonathletes versus athletes who train either upper body or lower body, or both.

We recently developed a non-invasive method for quantification of carnosine in human lower leg
muscles using proton magnetic resonance spectroscopy ($^1$H-MRS) (19). To our knowledge, $^1$H-MRS-
based metabolite quantification in a single muscle of the upper body has never been investigated,
neither for creatine nor for carnosine loading. In the present study, we first investigated in which large
muscle of the upper body carnosine is reliably measurable by $^1$H-MRS. Then, we hypothesized that
muscle carnosine loading via BA supplementation is of the same magnitude in arm and leg muscles in
a non-active control group. Finally and most importantly, we aimed to investigate whether carnosine
loading would be more pronounced in trained versus untrained muscles, by comparing loading in arm
and leg muscles that are specifically trained in different athlete groups.
MATERIALS AND METHODS

This study consists of 2 parts. For study A, carnosine was measured in 2 muscles of the arm to investigate in which muscle of the upper body carnosine is reliably measurable by $^1$H-MRS. Study B was a nutritional intervention study focusing on the difference in loading between arm and leg muscles in nonathletes and different athlete populations.

Study A

Subjects: Seventeen students (15 males and 2 females) volunteered to participate in this study. All subjects were physically active and were involved in different sports (athletics, football, swimming, cycling…). The subjects’ age, weight, and height were 22.0 ± 1.0 yr, 72.4 ± 6.4 kg, and 178.0 ± 8.9 cm, respectively.

Study protocol: Eleven subjects (9 men and 2 women) were measured twice on the same day to test the methodological variation. The subjects were measured, taken out of the scanner and immediately back in the scanner and were measured again. Six subjects underwent 2 tests, separated by 3 – 4 weeks, to test the additional biological variation of muscle carnosine content over that period. In the seventeen subjects, muscle carnosine content was measured separately in deltoid and triceps brachii muscles by $^1$H-MRS, as specified below.

Study B

Subjects: Thirty-five men (10 healthy nonathletes, 10 road cyclists, 10 swimmers and 5 flat-water kayakers) participated in this study. Table 1 shows some details about the training history and current training load of the athletes. All the athletes were well-trained at baseline (recreational and regional class athletes) and trained at least 8 hours a week in their specific sports (cycling, swimming or kayaking) during the supplementation period, whereas the nonathletes were inactive throughout. All subjects had a normal diet and none of them was a vegetarian. None of the subjects took supplements in the 3 months prior to the study and during the study, except from the supplement as part of the experimental intervention.
Study protocol: All the participants were supplemented for 23 days with 6.4 g slow release beta-
alanine (SR-BA) (4x/day 2 tablets of 800 mg, Carnosyn, Natural Alternatives International). None of
the subjects reported side effects due to the supplementation. Before and after the supplementation
period muscle carnosine content was measured by $^1$H-MRS in soleus, gastrocnemius medialis and
deltoid muscles. Due to the better reproducibility of the deltoid in study A, we decided to exclude
triceps brachii in study B.

Both studies were approved by the local ethical committee (Ghent University Hospital, Ghent,
Belgium). All subjects gave their written informed consent to take part in the studies and were aware
that they were free to withdraw from the experiment at any point.

**Muscle carnosine quantification by $^1$H-MRS**

All the MRS measurements were performed on a 3-T whole body MRI scanner (Siemens Trio,
Erlangen). Carnosine content was measured by $^1$H-MRS, as described by Baguet et al. (2). The
subjects were lying in supine position on their back. To measure the calf muscles, the lower leg was
fixed in a spherical knee-coil with the angle of the ankle at 20° plantar flexion. A shoulder coil was
used to measure carnosine in the deltoid and triceps muscle (Fig 1A). Single voxel point-resolved
spectroscopy (PRESS) sequence with the following parameters was used; repetition time (TR) of
2.000 ms, echo time (TE) of 30 ms, number of excitations is 128, 1.024 data points, spectral
bandwidth of 1.200 Hz, and a total acquisition time of 4.24 min. The average voxel size for the soleus,
gastrocnemius, deltoid and triceps brachii muscle was 40 mm x 12 mm x 30 mm, 40 mm x 12 mm x
30 mm, 40 mm x 13 mm x 30 m and 30 mm x 9 mm x 27 mm, respectively. The line width of the
water signal for the soleus, gastrocnemius, deltoid and triceps brachii muscle was on average
respectively 25.5 Hz, 26.8 Hz, 27.4 Hz and 25.2 Hz, following shimming procedures. The integral of
the C2-H peak (at ~8 ppm) was quantified relative to the water peak integral (x1000) and reported as
arbitrary units (11) (Fig 1B). The reason why we could not calculate into millimolar concentrations is
because the shoulder coil was a receive-only coil, whereas the knee coil was a transmit/receive coil.

With a receive-only coil it is not possible to use the phantom method to calculate the millimolar
concentrations, as we usually do. In order to allow comparison between the leg muscles and the
deltoid, we expressed the concentration relative to the water peak (arbitrary units) for all muscles. We
found significant correlation coefficients for the water signal pre and post supplementation in the three
muscles groups: 0.684 (p < 0.001) for soleus, 0.632 (p < 0.001) for gastrocnemius and 0.661 (p < 0.001)
for deltoid. The variation coefficients were respectively 4.23% for soleus, 4.59% for gastrocnemius
and 7.1% for deltoid. There was no significant difference in water signals between pre and post
supplementation for the different groups in the different muscles. These results showed that muscle
water content was not influenced during supplementation which gives us the possibility to use these
arbitrary units.

Statistics

The reliability of measuring carnosine in a single muscle of the upper body in study A was evaluated
by Intraclass Correlation Coefficient. The variation coefficient (CV) was calculated by following
equation: CV = (SD/Mean)*100.

For study B, a one-way ANOVA was performed to compare the difference in baseline muscle
carnosine concentration and the absolute carnosine increase after BA supplementation between the 4
groups. A post hoc analysis (Tukey) was done for multiple group comparison and a paired sample t-
test was done to investigate the difference in the absolute carnosine increase within the groups. All
analyses were done with SPSS statistical software (SPSS 21, Chicago, IL). All values are reported as
mean ± SD and statistical significance was set at p < 0.05.
RESULTS

Reliability of arm muscle carnosine quantification (Study A).

The CV of the carnosine concentration was calculated for each muscle. Deltoid muscle had a smaller variation than the triceps brachii (6.6% vs. 9.2%; Table 2). Intraclass correlation coefficients (ICC) were significant for both deltoid (ICC: 0.903, p < 0.01) (Fig 1C) and triceps muscle (ICC: 0.656, p = 0.01), respectively. Over a time period of 4 weeks, we found a CV of 13.3% in the deltoid and a correlation coefficient of 0.734 (p = 0.03).

BA-induced carnosine loading in arm vs leg muscles (Study B).

For the nonathletes, there was a significant increase in carnosine concentration in both arm (deltoid) and leg muscles (soleus + gastrocnemius) after supplementation, but the absolute and relative increase in carnosine in the arm vs. leg muscles showed no significant difference (p = 0.286 and p = 0.230, respectively) (Fig 2). The relative increase was 47.44 ± 32.29% in arm muscle and 33.01 ± 23.71% in the leg muscles.

There were no differences between the athletic groups concerning height and weight (Table 1). Yet, swimmers were significantly younger than the cyclists (p = 0.019). There were no differences in baseline deltoid carnosine concentration between the groups. Baseline carnosine concentration in the leg muscles (soleus + gastrocnemius) was significantly lower in the cyclists than in nonathletes (p = 0.028) and kayakers (p = 0.047).

In gastrocnemius muscle, the absolute increase in carnosine (expressed as arbitrary units) was significantly higher in the cyclists (0.082 ± 0.035) and the swimmers (0.076 ± 0.015), compared to the nonathletes (0.043 ± 0.026) and the kayakers (0.035 ± 0.014) (Fig 3 and 4). The swimmers (0.107 ± 0.029) had a higher absolute increase in the deltoid muscle in comparison to cyclists (0.065 ± 0.040) and the nonathletes (0.065 ± 0.035) (Table 3). There were no differences between the groups in the soleus. For mean muscle carnosine in the legs (soleus and gastrocnemius), the cyclists (0.084 ± 0.027) and swimmers (0.082 ± 0.017) had a higher absolute increase than the nonathletes (0.051 ± 0.031). We
found no significant correlations between training volume or years of training and increases in carnosine concentration with BA supplementation within nor across athlete groups.

In the kayakers, their deltoid muscle (0.089 ± 0.026) had a higher absolute increase than their leg muscles (soleus + gastrocnemius) (0.049 ± 0.019, p = 0.001). An opposite pattern was observed with the cyclists where the leg muscles (0.084 ± 0.027) showed a tendency to a higher absolute increase than the deltoid muscle (0.065 ± 0.040, p = 0.078).

Taking all the subjects together (n = 35), there was a negative correlation between baseline carnosine content and the absolute increase in muscle carnosine in the leg muscles (soleus + gastrocnemius) (p = 0.020; r = -0.404) and deltoid muscle (p = 0.005; r = -0.476) following BA supplementation. The lower the baseline muscle carnosine content was, the higher was the increase in muscle carnosine, following supplementation. For each group individually, there was only a significant negative correlation between baseline carnosine content and the absolute increase in the deltoid muscle in the cyclists group (p = 0.010; r = -0.765).
DISCUSSION

The first goal of our study was to determine in which muscle of the upper body carnosine can be reliably measured via $^1$H-MRS. For both the deltoid and triceps brachii muscles, the C2-peaks (at ~8 ppm) in the proton MRS spectra were sufficiently large to allow quantification. We found relatively low CVs (6.6 % for deltoid and 9.2 % for triceps brachii) in the test-retest condition within the same day, indicating that the carnosine concentration in the arm can be reliably measured. When adding the biological variability (3-4 weeks apart), the CV in the deltoid increased from 6.6 % to 13.3 %, which is in agreement with Baguet et al. (4), who found a variation of 9.8 % in the soleus muscle and 14.2 % in the gastrocnemius muscle, when measured several weeks apart, compared to 4.3 % and 7.6 %, respectively, when measured twice on the same day (19). This first part of the study gave us the opportunity to compare supplementation-induced changes in muscle carnosine concentrations within one subject in different parts of their body in the second part of the study. Due to the better reproducibility of the deltoid, we decided to exclude triceps brachii in study B.

A subsequent goal was to compare BA-induced muscle carnosine loading in arm vs. leg muscles in a non-active control group. To date, the increase of carnosine concentration after BA supplementation has not yet been investigated in the upper body musculature, despite several reports of ergogenic effects in exercise types that entirely (27) or predominantly (24) depend on this part of the body. Our results indicate that BA supplementation is equally effective in raising carnosine concentrations in upper vs. lower body muscles in nonathletes. Based on the above results, we started measuring athletes from different sports disciplines to compare carnosine loading in specifically trained and untrained muscles. We selected three well-trained athletic populations: swimmers, kayakers and cyclists. Kayakers almost exclusively train their upper body, cyclists the lower body whereas swimmers train the entire body. It was hypothesized, based on the positive effects of exercise training on muscle creatine loading (12, 21), that trained muscles would have a higher absolute increase in carnosine concentration when compared to untrained muscles. In line with this hypothesis, the results of current observational study clearly indicate that there is an effect of training on the carnosine loading as we observed a nearly doubling of the increase in muscle carnosine content after supplementation in
specifically trained muscles compared to the untrained muscles (77.95% vs. 42.88%, respectively). In soleus, we did not find a greater accumulation in cyclists compared to nonathletes and kayakers. Soleus is a tonic muscle that is recruited very frequently also in daily activities like walking and standing in nonathletes. Therefore the difference in activity degree between the groups is smaller for soleus than for gastrocnemius. In the gastrocnemius, we found a higher absolute increase in carnosine concentrations in the swimmers and cyclists compared to the nonathletes. Vice versa, the deltoid of the swimmers had a higher absolute increase in carnosine concentration in comparison to the nonathletes. This was further supported when comparing loading after supplementation in different muscles within each athlete group: cyclists were able to accumulate more carnosine in their leg muscles (soleus + gastrocnemius) than arm (deltoid) muscles, whereas a reverse pattern was observed in kayakers. Even though we did not conduct a training intervention (only nutritional intervention) in the current study, these results confirm that trained muscles are more efficiently loaded with carnosine than untrained muscles (Fig 4).

Our findings seem to be in contrast with Kendrick et al. (17) who found no difference in carnosine loading between a trained and untrained leg in a single-leg training study. However, it is worthy to note that training volume (10 x 10 maximal knee extensions, 3 – 4 times per week) was limited in that study. The athletes in our study trained at least 8 hours per week in their specific sport. If we estimate the amount of contractions, the current study had approximately 130x the number of contraction cycles compared to Kendrick’s study. Kendrick et al. (16) probably did not undertake enough contractions to really assess whether training had an effect on muscle carnosine synthesis. We suspect that the apparent contradiction between both studies is attributable to differences in training modalities, which should be subject to further investigation. The observed differences in carnosine loading in trained vs. untrained muscles could either be attributed to the acute effects of muscle contractile activity, i.e. the effect of the actual exercise during the supplementation period, or to the beneficial structural and metabolic properties of muscle induced by prior training. The current study design is not able to distinguish between training status vs. exercise effects. However, an advantage of the current study design is that –similar to the one-leg training studies- the comparison occurs within one subject,
ensuring identical nutritional and environmental factors, and identical circulating hormone and BA concentrations. Yet in contrast to the one-leg training studies, effects of larger training load and volumes can be investigated in the currently adopted design.

Although this is still the first study to document a training effect on muscle carnosine loading, it is tempting to speculate on its underlying physiological mechanisms. A possible explanation for the acute effect of exercise training is the increased blood flow in contracting muscles, which results in better BA delivery to the muscle cells. Another possibility is that there is a contraction-induced stimulation of transporters such as TauT and PAT1, which are expressed in human skeletal muscle (9), to take up BA in the myocyte. This would be in accordance with other transsarcolemmal metabolite transporters, such as the glucose and fatty acid transporter (GLUT4 and FAT/CD36), which are recruited by a contraction stimulus (16, 18). On the other hand, if it is the result of the training status, rather than the contractile activity, it could be related to the endurance training-induced increases in the capillary density (20), which ensures also better BA delivery in the trained muscles. Consequently, this same training-induced alteration can also increase the expression of transporters involved in BA uptake and enzymes involved in carnosine synthesis (9). However, all the proposed explanations are speculative at present.

From a practical point-of-view, this scientific finding can be translated into attractive implications to athletes. It seems wise to supplement BA during a period of substantial training volume, rather than in a rest or recovery period, in order to optimize supplementation effectiveness throughout a training season. In addition, the physiological carnosine loading mechanism is probably more effective in trained vs. untrained individuals. This is opposite to some other ergogenic supplements, where biological activity and effectiveness is often less pronounced in a trained population.

In summary, we managed to measure carnosine reliably and non-invasively in a single muscle of the upper body (deltoid) by $^1$H-MRS. We also showed that muscle carnosine loading via BA supplementation is of the same magnitude in arm and leg muscles in a non-active control group. Finally, we demonstrated that carnosine loading is more pronounced in the trained versus untrained
muscles of athletes. These findings suggest that training is a possible determinant of carnosine loading,
but it remains to be determined whether these effects are due to the acute exercise effects and/or to
chronic adaptations of training.
ACKNOWLEDGEMENTS

The contribution of Bauters Jeroen, Debosschere Dennis, Charle Pieter and Monkerhey Griet is greatly acknowledged. We thank Roger Harris and Natural Alternatives International (NAI) for generously providing the beta-alanine supplements.

GRANTS

This study was financially supported by grants from the Research Foundation – Flanders (FWO G.0243.11 and G.0352.13N).


FIGURE CAPTIONS

Figure 1A: Representative image (coronal plane) of voxel placement in the deltoid muscle.

Figure 1B: C2 and C5 proton resonances of the imidazole ring of carnosine can be identified at ~8ppm and ~7ppm, respectively, in the proton MRS spectrum of the human deltoid muscle (representative spectrum).

Figure 1C: Study A: Reliability of the carnosine concentration in the deltoid muscle, measured twice on the same day. * p < 0.05. ICC = 0.903.

Figure 2: Study B: Absolute increase in muscle carnosine concentration in arms (deltoid) and legs (average of soleus and gastrocnemius) in the nonathletes.* p < 0.05 vs pre.

Figure 3: Study B: Absolute increase in muscle carnosine concentration in deltoid and gastrocnemius muscle in the nonathletes, cyclists, kayakers and swimmers. Plus and minus signs indicate whether this limb is actively trained (+) or not (-) in this group.

Figure 4: Study B: Absolute increase in muscle carnosine concentration in deltoid and gastrocnemius muscle in the cyclists, nonathletes, kayakers and swimmers.
Table 1: Baseline values of the four groups of study B and details of the training of the athletes.

Table 2: Coefficient of variation (CV) of the carnosine content (study A).

a. The coefficient of variation (CV) of the carnosine content in deltoid and triceps muscle measured twice on one day (n = 11).

b. The coefficient of variation (CV) of the carnosine content in deltoid muscle at week 0 and 4 (n = 6).

Table 3: Muscle carnosine concentration pre and post supplementation and absolute increase in muscle carnosine concentrations after BA supplementation in the nonathletes, cyclists, kayakers and swimmers.
Figure 1C

M. Deltoid

Retest (arb. unit)

Test (arb. unit)
Figure 2

![Graph showing changes in Carnosine levels in leg and arm](image-url)
Figure 3

Carnosine (arb. unit)

- Nonathletes
- Cyclists
- Kayakers
- Swimmers
Figure 4

Carnosine increase in deltoid (arb. unit)

Carnosine increase in gastrocnemius (arb. unit)

Swimmers

Kayakers

Nonathletes

Cyclists
**Table 1:** Baseline values of the four groups of study B and details of the training of the athletes.

<table>
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<tr>
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<th>Nonathletes</th>
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<th>Kayakers</th>
<th>Swimmers</th>
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<td>10</td>
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<tr>
<td>Age (yr)</td>
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<td>21.7 (± 3.4)</td>
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<td>Height (cm)</td>
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<td>Training hours/ week (h)</td>
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<td>9.6 (± 2.6)</td>
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There are no significant differences between the groups, except for age in the swimmers. Swimmers’ age was significant lower than the age of the cyclists and kayakers (* p < 0.05). Data are means ± SD.
**Table 2:** Coefficient of variation (CV) of the carnosine content (study A)

**a.** The coefficient of variation (CV) of the carnosine content in deltoid and triceps muscle measured twice on one day (n = 11)

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<td>0.141</td>
<td>0.021</td>
<td>14.9</td>
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<td>0.116</td>
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<td>0.113</td>
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<td>3.9</td>
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<td>0.169</td>
<td>0.170</td>
<td>0.170</td>
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<td>0.6</td>
</tr>
<tr>
<td>6</td>
<td>0.132</td>
<td>0.167</td>
<td>0.150</td>
<td>0.025</td>
<td>16.7</td>
</tr>
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<td>7</td>
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<td>0.176</td>
<td>0.186</td>
<td>0.015</td>
<td>7.9</td>
</tr>
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<td>8</td>
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<td>0.182</td>
<td>0.178</td>
<td>0.006</td>
<td>3.3</td>
</tr>
<tr>
<td>9</td>
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<td>0.104</td>
<td>0.126</td>
<td>0.031</td>
<td>24.9</td>
</tr>
<tr>
<td>10</td>
<td>0.137</td>
<td>0.136</td>
<td>0.136</td>
<td>0.001</td>
<td>0.4</td>
</tr>
<tr>
<td>11</td>
<td>0.137</td>
<td>0.167</td>
<td>0.152</td>
<td>0.021</td>
<td>14.0</td>
</tr>
<tr>
<td>Mean</td>
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<td>0.155</td>
<td></td>
<td></td>
<td>9.2</td>
</tr>
<tr>
<td>Stdev</td>
<td>0.030</td>
<td>0.031</td>
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</table>

**b.** The coefficient of variation (CV) of the carnosine content in deltoid muscle at week 0 and 4 (n = 6)

<table>
<thead>
<tr>
<th>Deltoid</th>
<th>0w</th>
<th>4w</th>
<th>Mean</th>
<th>Stdev</th>
<th>CV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.069</td>
<td>0.102</td>
<td>0.086</td>
<td>0.023</td>
<td>27.29</td>
</tr>
<tr>
<td>2</td>
<td>0.149</td>
<td>0.133</td>
<td>0.141</td>
<td>0.011</td>
<td>8.02</td>
</tr>
<tr>
<td>3</td>
<td>0.131</td>
<td>0.129</td>
<td>0.130</td>
<td>0.001</td>
<td>1.09</td>
</tr>
<tr>
<td>4</td>
<td>0.089</td>
<td>0.111</td>
<td>0.100</td>
<td>0.016</td>
<td>15.56</td>
</tr>
<tr>
<td>5</td>
<td>0.133</td>
<td>0.168</td>
<td>0.151</td>
<td>0.025</td>
<td>16.44</td>
</tr>
<tr>
<td>6</td>
<td>0.089</td>
<td>0.076</td>
<td>0.083</td>
<td>0.009</td>
<td>11.14</td>
</tr>
<tr>
<td>Mean</td>
<td>0.110</td>
<td>0.120</td>
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<td></td>
<td>13.3</td>
</tr>
<tr>
<td>Stdev</td>
<td>0.032</td>
<td>0.031</td>
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</tr>
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</table>
Table 3: Muscle carnosine concentration pre and post supplementation and absolute increase in muscle carnosine concentrations after BA supplementation in the nonathletes, cyclists, kayakers and swimmers.

<table>
<thead>
<tr>
<th>M. Soleus</th>
<th>Pre</th>
<th>Post</th>
<th>Δ (post – pre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonathletes</td>
<td>0.149 (± 0.029)</td>
<td>0.209 (± 0.055)</td>
<td>0.060 (± 0.041)</td>
</tr>
<tr>
<td>Cyclists</td>
<td>0.098 (± 0.032)</td>
<td>0.183 (± 0.035)</td>
<td>0.085 (± 0.023)</td>
</tr>
<tr>
<td>Kayakers</td>
<td>0.155 (± 0.066)</td>
<td>0.216 (± 0.041)</td>
<td>0.061 (± 0.027)</td>
</tr>
<tr>
<td>Swimmers</td>
<td>0.140 (± 0.033)</td>
<td>0.227 (± 0.032)</td>
<td>0.087 (± 0.023)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>M. Gastrocnemius</th>
<th>Pre</th>
<th>Post</th>
<th>Δ (post – pre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonathletes</td>
<td>0.171 (± 0.050)</td>
<td>0.214 (± 0.052)</td>
<td>0.043 (± 0.026)</td>
</tr>
<tr>
<td>Cyclists</td>
<td>0.121 (± 0.043)* †</td>
<td>0.202 (± 0.039)</td>
<td>0.082 (± 0.035)* †</td>
</tr>
<tr>
<td>Kayakers</td>
<td>0.178 (± 0.038)</td>
<td>0.214 (± 0.030)</td>
<td>0.037 (± 0.013)</td>
</tr>
<tr>
<td>Swimmers</td>
<td>0.157 (± 0.042)</td>
<td>0.233 (± 0.040)</td>
<td>0.076 (± 0.015)* †</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>M. Deltoideus</th>
<th>Pre</th>
<th>Post</th>
<th>Δ (post – pre)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonathletes</td>
<td>0.149 (± 0.032)</td>
<td>0.214 (± 0.036)</td>
<td>0.065 (± 0.035)</td>
</tr>
<tr>
<td>Cyclists</td>
<td>0.142 (± 0.036)</td>
<td>0.207 (± 0.027)</td>
<td>0.065 (± 0.040)</td>
</tr>
<tr>
<td>Kayakers</td>
<td>0.135 (± 0.033)</td>
<td>0.223 (± 0.028)</td>
<td>0.089 (± 0.026)</td>
</tr>
<tr>
<td>Swimmers</td>
<td>0.131 (± 0.039)</td>
<td>0.238 (± 0.045)</td>
<td>0.107 (± 0.029)* S</td>
</tr>
</tbody>
</table>

Data are means ± SD. * p < 0.05 versus control group. † p < 0.05 versus kayakers. * p < 0.05 versus cyclists.