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Original Article

# Impacts of combined or single supplementation of branched-chain amino acids on delayed onset muscle soreness and muscle damage following resistance exercise

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**Summary.** The purpose of this investigation was to determine and compare the effects of leucine (Leu), valine (Val), isoleucine (Ilue), and combined branched chain amino acids (BCAA) supplementation, on markers of muscle damage following resistance exercise. It was hypothesized that supplementation would attenuate the levels of indirect muscle damage markers and muscle soreness following exercise-induced muscle damage, and facilitate the restoration of muscle function. A total of 50 untrained males were randomly divided into five groups; Leu (n = 10), Ilue (n = 10), Val (n = 10), BCAA (n = 10), and placebo (n = 10). The muscle damage protocol specific to non-athletes is used in the current study. A 500 ml of group-specific solution was given to each group in two time points, 30 minutes before and immediately after performing the exercise. Multivariate analysis showed significant time differences for LDH in all groups ($p<0.05$); there were significant multivariate time differences in all groups regarding creatine kinase (CK) ($p=0.000$ for all groups). A significant time differences observed in all groups regarding repetition maximum [RM] ($p<0.01$) and pain ($p=0.000$ for all groups). The results of this investigation show that compared to combined BCAA, Ilue or Val; Leu provides more protective effect on attenuating the immediate increase in biochemical markers of muscle damage following eccentric-based resistance exercise, while combined BCAA may aid to maintain the range of motion. Additionally the results showed that perceived pain by individuals in the placebo group was significantly higher compared to all other groups. Hence, BCAA intake, especially Leu, in the dosage used in this investigation is ergogenic for untrained males.

**Key words:** creatine kinase, lactate dehydrogenase, muscle soreness; muscle damage, branched-chain amino acid, leucine, exercise

## Introduction

It is well documented that exercise, including resistance or long distance exercise consisting of lengthening (eccentric) contractions, causes muscle damage and discomfort compounded with delayed onset of muscle soreness (DOMS), and edema may be lasted over time and chronically (1). The eccentric phase of resistance exercise has been found to induce the greatest level of muscle damage (2). Muscle damage can also change physical performance and reduce the ability of the muscle to contract with maximal force (3).

One way to attenuate this damage is through supplementation with nutrients that have been shown to influence cellular processes including proteolytic pathways and inflammation(4). Amino acids are required to produce various proteins, including those needed for tissue repair, thus increasing the abundance of amino

acids within the body would seem to be a reasonable choice to promote skeletal tissue recovery. Additionally, amino acids through the mediation of a number of cellular processes associated with regulating skeletal muscle metabolism, can influence recovery (5). Many of investigators have evaluated the effect of amino acid supplements to attenuate levels of muscle damage, and subsequent reductions in functionality of the muscle, following exercise, and among them branched amino acids, due to their relative abundance in skeletal muscle, have received considerable attention (6-8).

Creatine kinase (CK) and lactate dehydrogenase (LDH) are metabolic enzymes and their activity in serum has been used widely as an indirect method of determining skeletal muscle damage (8). Coombes and McNaughton (8) observed significantly lower CK levels at 2, 3, 4, 24, 72, and 120 h post exercise in the group that consumed 12 gr branched chain amino acids (BCAA) per day for 14 days. Similarly, Greer et al (7) found that acute ingestion of 50 g of BCAAs resulted in significantly lower CK levels at 4, 24, and48 h following a cycling protocol. BCAA supplementation has also demonstrated the ability to decrease the level of delayed-onset muscle soreness (DOMS) experienced 24 h post-exercise and attenuated the decrease in leg-flexion torque 48 h post exercise (7). Another study (9) also showed that a single high dose of BCAAs (100 mg/kg) immediately before seven sets of 20 squats significantly attenuated DOMS and decreases in isometric muscle force in the days following exercise. On the other hand, Jackman et al (6) recently reported no effect of BCAA supplementation on CK, Mb, or force production following 120 eccentric contractions of the knee extensors. Stock et al (10) had subjects perform six sets of squats to fatigue at 75% of the subject's 1 repetition maximum (1RM) while consuming either 22.5 mg/kg of leucine or placebo with 0.25 g/kg of carbohydrate solution. They reported no effect of leucine on CK, LDH or muscle soreness in the 72 h following exercise, as well as no differences in performance when the same exercise protocol was repeated 72 h post-exercise.

With respect to these literatures, it remains unclear whether BCAA supplementation, combined or alone, have different effects, if any, for attenuating the muscle damage response followed by exercise. Therefore, the purpose of this investigation was to determine and compare the effects of Leu, Ilue, Val, and combined BCAA, on markers of muscle damage following resistance exercise. It was hypothesized that supplementation would attenuate the levels of indirect muscle damage markers and muscle soreness following exercise-induced muscle damage, and help to facilitate the restoration of muscle function.

## Materials and Methods

Statistical population of the study included 50 non-athlete men who voluntarily participated and randomly assigned into five groups of supplements Leu (n=10), Ilue (n=10), Val (n=10), BCAA (n=10) and placebo (n=10). The reason for selecting non-athletes men was to observe obvious increments in muscle damage indices and to see possible supplementation effect (9). The muscle damage protocol specific to non-athletes is used in the current study (17).

At the first testing session, aims, details and probable risks of performing the exercise explained to the participants and then they provided with a written consent. Height measured to the nearest 0.1 centimeter using a wall-mounted stadiometer. To measure weight, a precise scale (Camry, model: 9003 EB) was used to the nearest 0.1 kilograms. At the same session, participants' leg presses recorded and one repetition maximum (1RM) calculated by the following formula:

1RM: W / [1.0278-(0.0278r)]; which r is the number of repetitions performed and W is the amount of weight used.

All subjects were university students, used to eat dormitory foods and recommended to avoid highintensity exercises, use of drugs particularly sedative, supplements and caffeine one week pre-/ and posttesting session. They were told not to alter their usual daily diet and have a comfortable non-stressful 8 hours of sleep, the night before testing.

To reduce the effect of bruising occurred in recording session, there was an interval of 7 days before testing session. Before implementing exercise protocol, 5 ml of fasting blood taken from antecubital vein of participants in sitting position, to test for enzymes creatine kinase and lactate dehydrogenase. Perceived

muscle soreness completed using a PAS 6-point standard scale (a combination of graphic and numeral scales) (19, 20). According to Shilaja and colleagues (2003), the validity of PAS scale determined using correlation coefficient with visual analogue scale of 0.82 (at the p level of 0.01) (19). In the pretesting step, all participants had normal level of blood parameters without feeling of muscle soreness. A 500 ml of group-specific solution was given to each group in two times, 30 minutes before and immediately after performing the exercise. Supplemental groups at each time consumed certain amount of solution (group L: 10 mg/Kg.BW of leucine), (group I: 10 mg/Kg.BW of isoleucine), (group V: 10 mg/Kg.BW of valine), (group B: 10 mg/Kg.BW of BCAAs [in 1:1:1 ratio]), (and group Placebo: 30 mg/Kg.BW of maltodextrin). BCAAs and maltodextrin used in the current research prepared from PNC (Karen Pharma & Food Supplement Co., Iran). Supplements and placebo weighted using a digital scale (Sartorious model: Bp221s, Germany) to the nearest thousandth of a gram.

Delayed onset muscle soreness (DOMS) and muscle damage in lower body developed using a leg squat machine with a weight equivalent to 75% of 1RM similar to Stock's statement (2010). After explaining the procedure to the participants, six sets of leg press attempt with 75% of 1RM performed by subjects to fatigue and voluntary inability margin. Positive movement implemented by examiner, where the leg raised to a 0° knee angle and the negative movement (eccentric contraction) performed by subjects. There was 3 minutes of rest between sets. Blood indices (LDH and CK), as well as perceived muscle soreness measures repeated 24 (TIME 1), 48 (TIME 2) and 72 (TIME 3) hours later using PAS scale.

Laboratory measures included activity of LDH and CK enzymes. Serum samples were prepared by 5 ml blood drawn from antecubital vein in sitting position. Samples, then, incubated at laboratory temperature for 20 minutes to coagulate and immediately after centrifuged for 10 minutes at 3000 rpm. Serum CK determined using colorimetric assay based on Jaffe reaction with sensitivity of 1 U/L and CV of 1.6% (Colorimetric Kit, Pars Azmoon Co., Tehran, Iran). LDH activity measured using enzymatic colorimetry with sensitivity of 5 U/L and CV of 1.2% (Colorimetric Kit, Pars Azmoon Co., Tehran, Iran). Both CK and LDH stated as unit per liter.

All statistical analyses were carried out using SPSS version 21.0 (SPSS Inc., Chicago, Illinois, USA). Five separate repeated measure analysis of variance (ANOVAs) were used to define whether CK, LDH, RM and pain values for the control group showed significant change during the testing period. If a significant time effect was observed, Tukey post hoc analyses were performed to determine where significance was obtained. All values are expressed as mean ± SD.

## Results

Anthropometric measurements (age, weight, height and BMI) were obtained in the first testing session (Table 1). There were no significant baseline differences between groups in terms of LDH, RM, CK and Pain (Table 2).

**Table 1.** Anthropometric values for each group (mean ± SD)

| Group | Age (yrs) | Weight(kg) | Height (m) | BMI |
|---|---|---|---|---|
| BCAA (n=10) | 22.23±1.53 | 73.3±7.82 | 1.77±0.075 | 23.26±1.250 |
| Lue (n=10) | 20.55±2.27 | 74.59±6.64 | 1.77±0.073 | 23.58±0.519 |
| ILue (n=10) | 23.17±2.42 | 73.88±6.33 | 1.77±0.068 | 23.46±0.609 |
| Val (n=10) | 23.37±2.38 | 70.43±7.07 | 1.75±0.069 | 22.89±0.865 |
| Placebo (n=10) | 22.06±1.85 | 72.58 | 1.75±0.043 | 23.61±0.860 |
| p-value (n=10) | 0.038 | 0.662 | 0.861 | 0.327 |

*All values are expressed as mean ± SD.*
*BCAA; branched chain amino acids, Leu; leucine, Ilue; isoleucine; Val; valine. Yrs; years, kg; kilograms, m; meters, BMI; body mass index.*

**Table 2.** Baseline values for LDH, RM, CK and Pain.

| Variable /Group | BCAA | Lue | ILue | val | placebo | p-value |
|---|---|---|---|---|---|---|
| pain | 0 | 0 | 0 | 0 | 0 | |
| RM | 134.20 ± 2.700 | 132.10±4.383 | 132.70±3.129 | 133.40±2.716 | 131.40±2.633 | 0.333 |
| CK (IU/L) | 113.30± 7.79 | 115.90± 5.425 | 119.40± 10.458 | 121.30± 9.878 | 122.50 ± 5.740 | 0.083 |
| LDH (IU/L) | 265.49±71.044 | 251.00±46.366 | 294.74±74.886 | 226.53±49.818 | 376.46±134.719 | 0.603 |

*All values are shown as mean ± SD.*
*BCAA; branched chain amino acids, Leu; leucine, Ilue; isoleucine; Val; valine. RM; repetition maximum, CK; creatine kinase, LDH; lactate dehydrogenase.*

**LDH:** All groups showed significant time differences for LDH ($p<0.05$). However, in the placebo group mean value was significantly higher than other groups at time 2 and 3.

In all groups mean LDH value at time 1 displayed a significant higher mean from pre-exercise value. However, the minimum values for LDH were observed in leucine supplemented group (323.30±47.951), followed by BCAA and Isoleucine, 324±73.309 and 327.31±58.745 respectively. Multivariate analysis showed significant time differences for serum marker of LDH in all groups ($p<0.05$). Further inspection revealed significant differences between T1 with T2 (p=0.046 [BCAA], p=0.000 [Leu], p=0.011 [Ileu], p=0.000 [Val] & p=0.010 [Placebo]) and T1 with T3 (p=0.028 [BCAA], p=0.081 [Leu], p=0.010 [Ileu], p=0.000 [Val] & p=0.005 [Placebo])), but there was no significant difference between T2 with T3 for all groups. The peak LDH level for BCAA group occurred at Time 3, but for other groups it peaked at Time 2 and declined thereafter at Time 3. The LDH levels for five groups are shown in figure 1. No significant between groups differences were observed for LDH at Time2 and Time3 measures. Multiple comparisons for LDH at two-time points are presented in tables 3 and 4.

**CK:** There were significant multivariate time differences in all groups regarding CK (p=0.000 for all groups). Inspecting the means showed significant differences between T1 with T2 (p=0.000 for all groups), T1 with T3 (p=0.000 for all groups) and difference between T2 with T3 for Leu (p=0.000), Ileu (p=0.002), Val (p=0.003) and placebo (p=0.00), but there was no significant differences for BCAA group, comparing

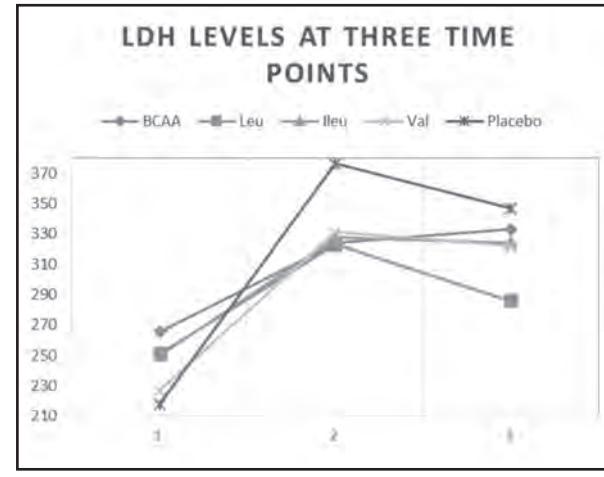


**Figure 1.** Changes in LDH levels post-resistance exercise protocol

T1 and T3. The peak CK level for all groups occurred at Time 2. Figure 2 displays CK levels at three time points. No significant between group differences were observed for CK at Time 2. However at Time 3 measurements a significant multivariate between group difference was revealed (p=0.01). Tukey's post hoc results showed that CK levels were significantly higher for BCAA compared to all other groups. Multiple comparisons for CK at two-time points are presented in tables 3 and 4.

**RM:** A significant time differences observed in all groups regarding RM ($p<0.01$). Further inspection showed significant differences between T1 with T2 (p=0.000 [BCAA, Leu, Ileu & Placebo] and p=0.002 [Val]), T1 with T3 (p=0.000 for all groups) and T2 with T3 (p=0.047 [BCAA], p=0.007 [Val] & p=0.048 [Placebo]), but there was no significant difference between T2 with T3 in Leu and Ileu groups (p=0.168

**Table 3.** Mean differences for LDH, CK, RM and pain in the first 24-hour (Time2) measurements.

| | Groups | BCAA | Leu | Ileu | Val | Placebo |
|---|---|---|---|---|---|---|
| LDH | BCAA | | 0.70 | -3.31 | -7.09 | -52.45 |
| | Leu | | | -4.01 | -7.79 | -53.16 |
| | Ileu | | | | -3.78 | -49.14 |
| | Val | | | | | -45.36 |
| | Placebo | | | | | |
| | | BCAA | Leu | Ileu | Val | Placebo |
| CK | BCAA | | 5.50 | 20.70 | 12.70 | -8.20 |
| | Leu | | | 15.20 | 7.20 | -13.70 |
| | Ileu | | | | -8.00 | -28.90* |
| | Val | | | | | -20.90 |
| | Placebo | | | | | |
| | | BCAA | Leu | Ileu | Val | Placebo |
| RM | BCAA | | 1.00 | 1.40 | -1.90 | 2.00 |
| | Leu | | | 0.40 | -2.90 | 1.00 |
| | Ileu | | | | -3.30 | 0.60 |
| | Val | | | | | 3.90* |
| | Placebo | | | | | |
| | | BCAA | Leu | Ileu | Val | Placebo |
| Pain | BCAA | | 1.60 | 0.80 | 2.40* | -0.20 |
| | Leu | | | -0.80 | 0.80 | -1.80* |
| | Ileu | | | | 1.60 | -1.00 |
| | Val | | | | | -2.60* |
| | Placebo | | | | | |

*Post hoc comparisons using Tukey's LSD. Mean differences shown.*
** shows mean difference is significant at the 0.05 level*

**Table 4.** Mean differences for LDH, CK, RM and pain in the first 24-hour (Time2) measurements.

| | Groups | BCAA | Leu | Ileu | Val | Placebo |
|---|---|---|---|---|---|---|
| LDH | BCAA | | 47.81 | 9.61 | 11.69 | -12.97 |
| | Leu | | | -38.20 | -36.12 | -60.79 |
| | Ileu | | | | 2.08 | -22.59 |
| | Val | | | | | -24.67 |
| | Placebo | | | | | |
| | | BCAA | Leu | Ileu | Val | Placebo |
| CK | BCAA | | 54.10* | 61.70* | 59.80* | 58.20* |
| | Leu | | | 7.60 | 5.70 | 4.10 |
| | Ileu | | | | -1.90 | -3.50 |
| | Val | | | | | -1.60 |
| | Placebo | | | | | |
| | | BCAA | Leu | Ileu | Val | Placebo |
| RM | BCAA | | 4.30* | 4.60* | 4.80* | 6.70* |
| | Leu | | | 0.30 | 0.50 | 2.40 |
| | Ileu | | | | 0.20 | 2.10 |
| | Val | | | | | 1.90 |
| | Placebo | | | | | |
| | | BCAA | Leu | Ileu | Val | Placebo |
| Pain | BCAA | | 1.20 | 1.00 | 1.20 | -2.00* |
| | Leu | | | -0.20 | 0.00 | -3.20* |
| | Ileu | | | | 0.20 | -3.00* |
| | Val | | | | | -3.20* |
| | Placebo | | | | | |

*Post hoc comparisons using Tukey's LSD. Mean differences shown.*
** shows mean difference is significant at the 0.05 level*

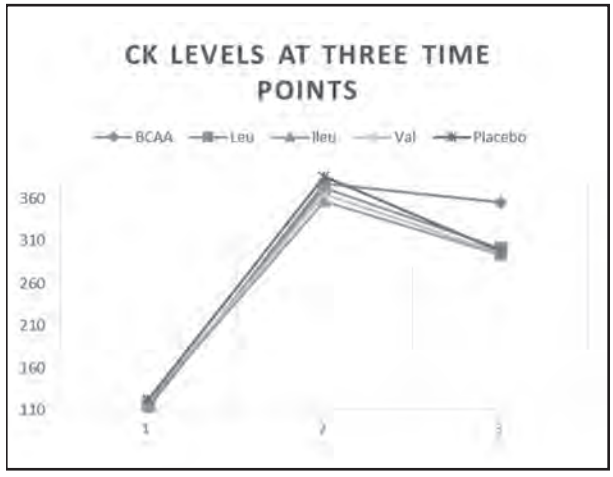


**Figure 2.** Changes in CK activity post-resistance exercise protocol

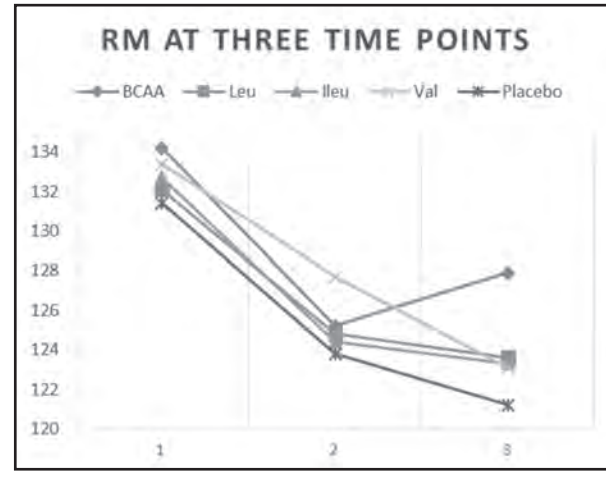


**Figure 3.** Changes in RM post-resistance exercise protocol

and p=0.292, respectively). Lowest RM level for BCAA group occurred at Time 2, but for other groups it peaked at Time 3 and declined thereafter at Time 3. RM at three time points are displayed in figure 3. There were no significant between group differences for RM at Time2, whereas a significant multivariate between group difference was observed ($p<0.01$). Tukey's post hoc results showed that RM was significantly higher for BCAA compared to all other groups. Tables 3 and 4 show the multiple comparisons for RM.

**Pain:** There were significant multivariate time differences in all groups regarding Pain ($p=0.000$ for all groups). Detailed results showed significant differences between T1 with T2 ($p=0.000$ for all groups), T1 with T3 ($p=0.000$ for all groups) and difference between T2 with T3 only for ILeu ($p=0.010$), but there was no significant differences for BCAA, Leu, Val and Placebo. The peak pain in all groups occurred at Time 2. Pain scores for groups at three time points are shown in figure 4. There were no significant between group differences for pain at Time2, whereas a significant multivariate between group difference was found ($p<0.01$). Tukey's post hoc results showed that pain was significantly perceived significantly higher by individuals in the Placebo compared to all other groups. Tables 3 and 4 show the multiple comparisons for pain perception. Post hoc comparisons using the Tukey's LSD tests are stated as mean difference in the table 3 and 4.

## Discussion

The aim of this study was to determine if combined or single BCAA supplementation would ameliorate muscle damage from eccentric exercise. It was hypothesized that BCAA supplementation, single or in combination would decrease post exercise concentrations of CK, LDH and the loss of RM.

We found some supports for our assumptions. Indeed combined or single BCAA were able to attenuate the increases in LDH; it should be noted that in this regard Lue was the most effective agent. Leucine is an anti-proteolytic amino acid, as well as a potent stimulus

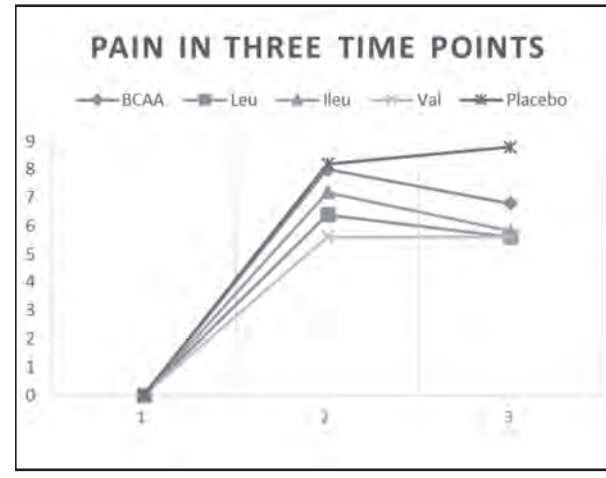


**Figure 4.** Changes in pain post-resistance exercise protocol

for protein synthesis and When it is co-administrated with the other BCAAs, protein synthesis becomes stimulated (11). However, when leucine is removed from the mixture, protein synthesis is abated (12), Therefore, it can be argued that leucine is the rate-limiting BCAA controlling protein synthesis during the recovery period following exercise (11). Since leucine supplementation activates protein synthesis in skeletal muscle (13), enhancing frequency of anabolic processes would seem to be a reasonable method of accelerating the recovery process in physical performance (11). In this context, a recent review (14) have reported that both BCAAs and leucine are effective supplements in the attenuation of exercise-induced muscle damage, leucine intake promotes regeneration of the cell membranes that were damaged by the exercise by increasing the endogenous synthesis of HMβ and its metabolite HMG-CoA.

Combined BCAA supplementation failed to decrease CK activity as effective as single amino acids; indeed the highest values of CK at time 3 was observed in BCAA group. There were no significant differences for CK between other experimental groups. CK was significantly elevated from pre-exercise levels for all groups at 24h; and significantly decreased from 24h at 48h. In line with our findings, Jackman et al (6) reported no effect of BCAA supplementation on CK, Mb, following 120 eccentric contractions of the knee extensors, Similarly, more recently in Foure A (15), et al study the BCAA supplementation did affect neither the plasma CK activity nor the amino acids concentrations changes resulting from the damaging exercise In addition, Ra et al (16) reported that BCAA supplementation alone failed to inhibit muscle damage and soreness after eccentric exercise.

Contrariwise, Shimomura et al (9) recently showed that a single dose of BCAAs (100 mg/kg) instantly before seven sets of 20 squats significantly attenuated DOMS and declines in the force of isometric muscle in the days following exercise. Previous studies have shown that reduced protein degradation is associated with a lower CK value (17, 18) and that supplementation with BCAA (8) can partially attenuate exerciseinduced muscle damage and/or proteolysis. Similarly; one recent study (19) proposed that the administration of BCAA supplementation is useful to amend muscle soreness and fatigue induced by different types of exercise. Likewise, Greer et al (7) observed that acute ingestion of 50 g of BCAAs attenuated circulating CK at 4, 24, and 48 h following a 90 min cycling protocol.

These discrepancies in findings may be related to the differences in the dosage of BCAA, as Jackman et al (6) only consumed 14.6 g of BCAAs in close to the exercise protocol while Greer et al (7) used the 50 g dosage during exercise. In this regard, it has been indicated that ingestion of BCAA both acutely before, and immediately following intensive resistance training offsets the decrement in muscle function and alleviates symptoms of muscle soreness. These findings demonstrates a potential dose-response effect, with a higher dose having a considerably greater effect compared to a lower dose (20). Additionally, the extent of muscle damage may explain some of the inconsistencies in these findings, as the investigations showing beneficial effects of supplementation in the modest levels of muscle damage (7-9). Since in our subjects, there were no within group differences for CK in BCAA group; it appears to be responders and non-responders to elevations in this biochemical molecule, also it should be noted that due its location within the cytosol, CK is indicator of membrane disruption and are not necessarily representative of damage to the myofibrils (11).

However, regarding the observed effects of single administration of Leu, it has been previously (21) reported that an essential amino acid-based formula containing β-hydroxy-β-methylbutyrate (the metabolite of leucine) beneficially affected training-induced changes in markers of muscle damage and decreased the CK. Likewise, a recent study (22) reported that Leucine- enriched essential amino acids promote the rate of protein synthesis and improves muscle soreness after eccentric exercise. However, there is evidence that high-dose leucine supplementation was unable to attenuate the increased markers of muscle damage that follows eccentric-based resistance exercise (11). Stock et al (10) reported that Leu had no effect on squats to fatigue 72 h after exercise. However, in this mentioned investigation a comparatively low dose of leucine has been used and also no supplementation in the days following the exercise protocol has been used. Low doses may be advantageous when consumed chronically over a period. Of note, higher doses ([150 mg/kg) may be required to exert any useful effect on intense exercise (7). Kirby et

al (11) showed that leucine provides no protective effect on reducing the instant increase in biochemical markers of muscle damage following eccentric-based resistance exercise. It is possible that leucine may help in the maintenance of isometric muscle function following muscle damage; however, the ergogenic effect was only noticeable when comparing the mean decrease at all-time points. The physiological application of these results may be insignificant, since no effect was observed on dynamic muscle function during the static jump. Further investigations should focus on direct markers of myofibril damage, as well as some of the structures involved in process responsible for force output. Furthermore, investigation of the immunological impacts of ILeu following muscle damage may provide information regarding the possible role in muscle recovery (11).

A significant time differences observed in all groups regarding RM, The primary finding of this investigation is that BCAA supplementation, in combination or alone, may aid in the maintenance of RM. All experimental groups showed a similar decrease in isometric RM across all time points; however, the BCAA and leucine groups mean RM value were significantly higher than the other groups at time 3. The results from present study corroborate the findings of a great deal of previous studies (8, 21, 23, 24); which reported promoting effect of amino acid supplementation on maximal voluntary contraction and range of motion. Supporting our results, there is evidence (25) that BCAA supplementation is associated with maintained lean mass in addition to significant improvement in participants' 1RM squat and 1RM bench press from pre-test. BCAA supplementation has been stated to decline exercise-induced protein degradation possibly by promoting an anti-catabolic hormonal profile (26). This result may be explained by the fact that, exercise-induced muscle damage is a manifestation of neuromuscular function reduction, reduced range of motion, increased muscle soreness, limb swelling and the elevation of intramuscular proteins in blood(1, 27, 28); thus any attempt to reduce the negative effects of exercise-induced muscle damage including BCAA would have beneficial effect on RM (29).

DOMS-induced pain decreases muscle strength gains and exercise performance for up to three weeks (30); thus, effective interventions to prevent or diminish DOMS and/or promote recovery of muscle function after exercise are reasonable strategy to reduce pain. A recent review suggested that BCAA supplementation could promote interesting effects on muscle repair by promoting muscle sarcomerogenesis, reducing protein oxidation and improving muscle functional status (31). Previous studies suggested that BCAA supplementation may be effective in decreasing muscle soreness in many types of exercises, in addition to stimulating an anabolic environment that is often preferred with resistance training. However an exact amount for optimum BCAA supplementation is not recognized, it is suggested that in physically active individuals, the ingestion of large amounts of BCAA is not required to reduce muscle soreness (32). Instead, it appears that consistent ingestion of BCAA s is useful in reducing DOMS(33). However a recent study (34) reported no benefit the increased BCAAs availability could improve alterations of muscle function (DOMS) and the changes in the related metabolic states (e.g. mitochondrial function and pH homeostasis,). In addition, the BCAA supplementation did change neither the plasma CK activity nor the amino acids concentrations changes resulting from the damaging exercise; however the authors suggested that, a short BCAAs supplementation period could have been ineffectual to decrease the acute damage of exercise. Additionally, Amirsasan R et al (35) described that the two different dosages of BCAA did not reduce the DOMS associated with heavy resistance exercise, but it should be noted that their subjects were trained wrestlers that is different with our subjects. Furthermore; Danielle T et al (36) found that BCAA plus glucose supplement, in comparison with an equivalent glucose containing placebo, reduced exercise-induced DOMS in relatively inactive female adults , their participants were who engaged in no more than one hour physical activity with light to moderate intensity per week, that is comparable with our participants.

## Conclusion

From the present study we concluded that single or coadministration of BCAAs may play critical physiological functions such as stimulation of muscle protein synthesis after exercise. Additionally, the benefi

cial effect of consuming a BCAA supplement in the prevention of muscle damage is suggested. Therefore; after resistance training, protein supplements and/or a meal containing sufficient levels of proteins should be ingested immediately. Further investigations are required to investigate the effect of different dosage of BCAA intake on muscle damage indices.

## Limitation

In this study, the intervention has been conducted on single dose, and the duration of amino acid consumption may play an important role for these results.

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