Effect of creatine supplementation on muscle damage markers and physical performance in volleyball athletes

Efecto de la suplementación de creatina sobre marcadores de daño muscular y desempeño físico en atletas de voleibol

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Abstract

Given creatine supplementation may attenuate exercise-induced damage and directly influence the ATP-CP system, the purpose of the study is to assess the effects of creatine (Cr) supplementation on muscle damage markers; creatine kinase (CK) and lactate dehydrogenase (LDH), late-onset muscle pain (DOMS) and physical performance in volleyball athletes. A controlled study with a double blind model was performed. Fourteen participants supplemented (0.3 g / kg) of creatine or placebo during (loading phase) and (0.1 g / kg) during (maintenance phase). Significant differences were observed in total plasma creatine concentration ($p < 0.05$), body weight ($p = 0.047$) and lower pain perception ($p = 0.020$), 24 hours ($p = 0.001$), 48 hours ($p < 0.001$) and 72 hours ($p = 0.011$) in the creatine group. The evaluation of subjective perception of pain verified a significant difference in the creatine group ($p < 0.05$). It was concluded that creatine supplementation, associated with carbohydrate consumption, attenuated the perception of pain in volleyball players after the muscle damage protocol.

Key words: DOMS, muscle injury, jumping, performance.

Resumen

Dado que la suplementación con creatina puede atenuar el daño inducido por el ejercicio e influir directamente en el sistema ATP-CP, el propósito del estudio es evaluar los efectos de la suplementación con creatina (Cr) sobre los marcadores de daño muscular como; creatina quinasa (CK) y lactato deshidrogenasa (LDH), dolor muscular de aparición tardía (DOMS) y rendimiento físico en atletas de voleibol. Se realizó un estudio controlado con modelo doble ciego. Catorce participantes suplementaron (0.3 g / kg) de creatina o placebo durante (fase de carga) y (0.1 g / kg) durante (fase de mantenimiento). Diferencias significativas fueron observadas en la concentración plasmática total de creatina ($p < 0.05$), peso corporal ($p = 0.047$) y menor percepción del dolor ($p = 0.020$), 24 horas ($p = 0.001$), 48 horas ($p < 0.001$) y 72 horas ($p = 0.011$) en el grupo creatina. La evaluación de la percepción subjetiva del dolor verificó diferencia significativa en el grupo creatina ($p < 0.05$). Se concluyó que la suplementación de creatina, asociada al consumo de carbohidratos atenuó la percepción de dolor en los jugadores de voleibol después del protocolo de daño muscular.

Palabras clave: DOMS, lesión muscular, saltos, desempeño.

Introduction

Exercise can promote changes in the cellular environment, especially high intensity or close to exhaustion training can damage muscle fibers, which will increase energy expenditure and can impair the movement efficiency, strength and force production (Brancaccio et al., 2007). The degree of damage and muscle soreness may be compounded over time and persist chronically, especially in individuals frequently engaging in vigorous exercise or an overreaching phase and can induce the release of muscle enzymes in the plasma. Some of these muscle enzymes such as creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) are considered indirect biochemical markers of muscle damage (Brown et al., 1997).

It is common for volleyball players to have muscular damage due to the accomplishment of a large number of jumps per match or in training session, since after the jumps it is necessary to perform eccentric contractions in order to decelerate whole body center of gravity when returning to the ground (Komi & Bosco, 1978). Eccentric contractions are the main cause of muscle damage and may negatively affect performance during exercise. In light of this information, the importance of attenuating muscle injury in athletes caused by different mechanisms is evident (Clarkson & Hubal, 2002).

Among the great variety of supplements available, creatine (Cr) stands out as a substance that has surpassed the milestone of millions of U.S. dollars in profit in the United States per year (Terjung et al., 2000). The success of Cr was also proven in 1999, as its worldwide consumption reached 2.7 million kilos that year. This success is largely attributed to its ability to promote increased Cr and creatine phosphate (CP) stocks, which consequently optimize the energy supply of the adenosine triphosphate-phosphocreatine (ATP-CP) system (Deminice et al., 2013). Some of the ergogenic effects described in the literature includes increased muscle mass, antioxidant capacity, tamponade effect, and ability to maintain muscle cell integrity (Hultman et al., 1996).

Given that creatine supplementation can attenuate damages induced by intense physical exercise (Cooke et al.,2009; Ra et al., 2013), and directly influence the ATP-CP system, which is prevalent in volleyball (Conlee et al., 1982), this study examined the use of a seven-day trial creatine supplementation, associated with a muscle damage induction protocol, in order to investigate the ability to maintain muscle cell integrity through indirect markers of muscle damage, as well as it is influence on the physical performance tests. In this context, it is believed that Cr supplementation may reduce the level of muscle damage and improve jumping performance in volleyball athletes.

Methods

Participants

The volunteers were 14 healthy and well-trained men that were engaged in a volleyball team (under-21 years category) of the city of Sertãozinho (São Paulo-Brazil). The general characteristics of the volunteers are presented in Table 1. All volunteers were training regularly 3 ~d a week, about 2h a day. The study was approved by the Research Ethics Committee of the School of Physical Education and Sport of Ribeirão Preto. All volunteers completed a written informed consent. None of the participants were smokers or was talking any type of medication.

Experimental Design

A double-blind, randomized, placebo-controlled study was conducted. The volunteers were randomly divided into two groups: Supplemented with creatine group (CG) (n=7) or placebo group (PG) (n=7). Following recruitment, the first phase of tests was started, in which blood was collected for biochemical analysis, jump height and relative power tests were performed as a measure of performance. Immediately after the first performance tests, the period of supplementation (loading phase) was started (days 1-7), which was composed by a high creatine dose associated with carbohydrate (CHO) in the CG group and only CHO in the PG.

After this period (8th day), blood collection and jump performance test were performed, as well as the muscle injury induction protocol, followed by a muscle soreness test, blood collection, and vertical jump test.

Immediately following these procedures, a second supplementation period (maintenance phase) was started (days 8-11) and composed by a lower dose of creatine plus CHO and the PG only CHO. During the second supplementation period, all previous tests were repeated 24, 48 and 72 hours after the injury induction protocol. Fig. 1 summarizes the study design protocol.

Athletes were also instructed not to change their daily training activities and food intake during the data collection period. During the two weeks of supplementation (loading and maintenance week), the athletes quantified the training load through the Rat-

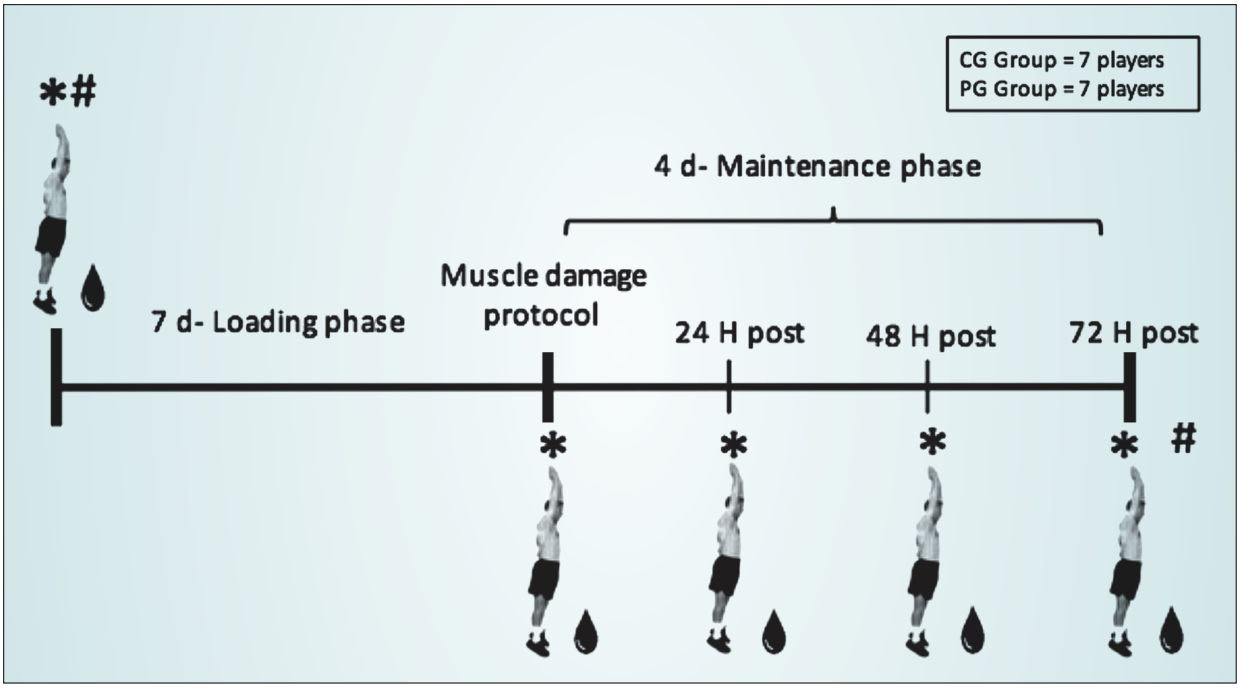

Figure 1. Experimental Design.

Notes: Blood collection for CK, LDH and Cr quantification; Jump test; # Training load quantification and food record (3 days) application;); *DOMS-visual scale; 7 d-loading phase (0.3 creatine/kg/day + 1.2 g carbohydrate/kg/day); 4 d-maintenance phase (0.1 g creatine/kg/day + 0.4 carbohydrate/kg/day). Weight and height measurements were performed using a scale and a stadiometer.

ing of perceived exertion (RPE) and reported a three-day food record to verify their energy and macronutrient intake using DietPro5i® professional software.

Supplementation Protocol

The supplementation protocol was composed by 11 days divided into two phases: 1) loading phase (7 days) in which CG subjects were supplemented with 0.3g/kg/day of creatine associated with 1.2g/kg/day of carbohydrate. The PG subjects were supplemented with 1.5g/kg/day of placebo (maltodextrin) (Greenhaff et al., 1993; Harris et al.,1992).

In the maintenance phase (which lasted 4 days) subjects in the CG received 0.1 g/kg/day of creatine plus CHO supplementation, composed of 0.4 g/day/kg maltodextrin. The PG received 0.5g/kg/day of maltodextrin, the fractionation procedure was identical to the first phase of supplementation.

The supplements were composed by creatine monohydrate or maltodextrin powder, which were packed in individual doses, according to dose proposed in each supplementation phase and were distributed to players by a non-participating collaborator of the study to ensure protocol blindness. The athletes were instructed to fractionate the daily dose packs in four doses at different times of the day (before breakfast, after lunch, during the afternoon, and before dinner) and to dilute the portion of supplement in 300 ml of water. The supplements of both groups (creatine and placebo) presented similar characteristics of color and flavor in order to blind the supplementation.

Training Load Quantification

Training intensity was monitored during periods of supplementation through training diaries. The athletes recorded the Rating of perceived exertion (RPE) using scale described by (Ferreira et al., 2011; Foster et al., 2001) and the duration in minutes of the training session. This method enables the calculation of the internal load by associating the intensity with the duration of the training.

The results were obtained through the training diaries and were used to evaluate the total load during the intervention period, being referred to as a sum, allowing the visualization of the alternation and distribution of loads. In addition to the sum of the load, the monotony of the loads was calculated by the mean value of the sum of the load divided by the standard deviation; (Monotony = Summation ÷ DP), while the density of the loads was calculated by multiplying the monotony by the sum of the loads: Density = Monotony × Sum (Foster et al., 2001).

Vertical Jumping Test

The vertical jump performance test was performed through a contact mat (Cefise®. Brazil), on which the flight time, jump power and jump height of the athletes were measured. The volunteers performed the Counter movement Jump test (CMJ) (Kirksey et al., 1999; Bosco et al.,1983; Jiménez-Reyes, Cuadrado-Peñafiel, & González-Badillo, 2011) without aid of the upper limbs, following a voice command by the researchers. Starting from the standing position, the subjects performed the knee flexion up to an angle of about 90 degrees and performed the jump with a countermovement at maximum intensity. They were advised not to flex the knee and keep the trunk upright when performing the jump, as a different execution could affect the test results. Participants performed three trials spaced by a 60-second recovery time, with only the best performance being considered.

Muscle Soreness

Delayed onset muscle soreness was determined using a 100-mm visual analogue scale (VAS), delimited by the terms "no pain" and ¨severe pain." The participants assessed their perception of the pain by performing a squat movement at a 90-degree angle, marking the pain perceived during the movement, according to the extremes of the scale. The test was applied 24h, 48h and 72 h after the injury induction protocol (Radak et al.,1999; Stanton & Abt, 2000; Nosaka et al., 2006).

Injury Induction Protocol

The athletes performed a total of 100 jumps starting from a height of 0.6 m. When landing, they were verbally encouraged to jump in the vertical plane with maximum effort. Five sets of 20 jumps were performed with a 10-second interval between each jump and 2 minutes between each series (Tofas et al., 2008; Miyama & Nosaka, 2004).

Blood collection and biochemical Analysis

Blood collection from the brachial vein was performed in the baseline period, prior to the beginning of the first phase of supplementation, at the end of the first supplementation phase, immediately after the muscle injury induction protocol, and 24 h, 48 h and 72 h after the injury induction protocol.

Creatine kinase (total CK) and Lactate dehydrogenase (LDH) were measured using the specific creatine kinase and lactate dehydrogenase kit from Blister® Life Biotechnology, with a colorimetric spectrophotometer reaction. Plasma creatine levels were assayed by the Jaffe reaction (Deminice et al., 2009).

Statistical Analysis

Data were tested for normality and sphericity using the Shapiro-Wilk and Levene tests, respectively. A two-way ANOVA model for repeated measurements, followed by the LSD post-test, was adopted in order to perform comparisons within and between groups. Time (baseline, pre-injury, post-injury, and 24 h, 48 h and 72 h after injury) and treatment (creatine and placebo) were set as independent factors for mean comparisons, and values were expressed as mean ± standard deviation (bar graphs). In the cases of non-parametric distribution (pain perception and plasma creatinine), the Friedman test was applied, followed by the Benjamini-Hochberg FDR post-test, and the values were expressed as median, maximum, and minimum (boxplot graphs). For non-spherical data, the Huynh-Feldt correction was applied. For the statistical analysis of training load data (load, monotony, and density), the Wilcoxon test was performed. Differences were significant when $p \leq 0.05$.

Results

After the creatine supplementation protocol over a period of seven days (loading phase), a significant increase in plasma Creatine concentration in CG ($p < 0.001$) was observed when compared to PG, using a Friedman's statistic test. Benjamini-Hochberg FDR post-test. Figure 3A. Regarding nutrients intake, both groups were similar regarding total calorie intake, however, it was observed it was observed a higher intake of protein ($p=0.008$) and lipid ($p=0.012$) in the Creatine group post intervention (Table 1).

Jumping height and relative jumping power were evaluated as indicators of physical performance, while blood levels of CK and LDH were evaluated as indicators of muscle damage. In this study, a decrease in jump height was observed immediately after the injury protocol ($p = 0.022$) in Creatine group, returning to baseline performance after 24 hours ($p = 0.238$). In relation to Placebo group, an increase in jump height was observed at the end of 72 hours following the injury protocol ($p = 0.008$). This effect was reflected in the relative jumping power for Creatine group immediately after the event ($p = 0.028$) and 24 hours after the event ($p = 0.200$) and for Placebo group 72 hours

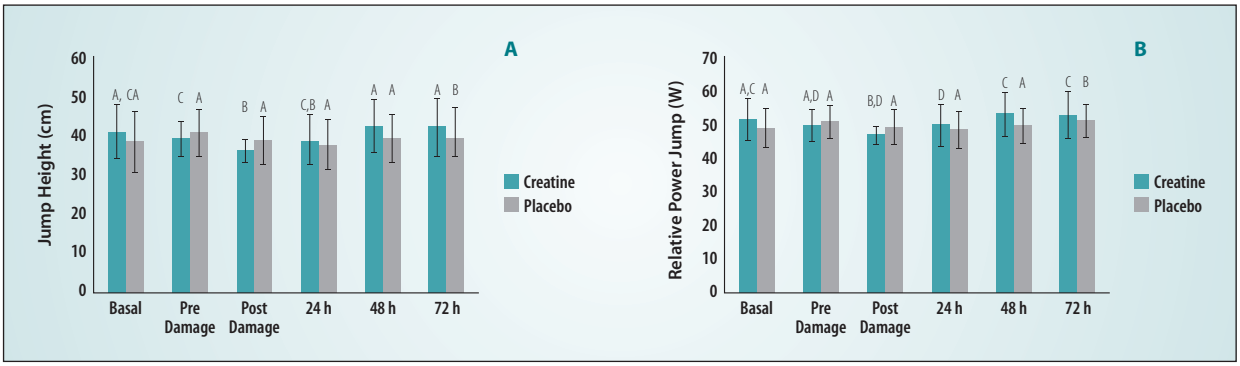

Figure 2. Jumping performance before and after the injury protocol associated with creatine supplementation or placebo. Values in mean ± SD. Different uppercase letters represent differences between the times within the groups ($p \leq 0.05$). Repeated two-way ANOVA measurements. Post-test LSD.

Table 1. Sample characteristics, food intake and muscle damage markers before and after supplementation.

Characteristics	Creatine Group					Placebo Group				
	Pre			Post		Pre			Post	
Age (y)	18±0.3					19±0.4				
Height (m)	1.83±0.04					1.80±0.06				
Weight (kg)	70.7±10.7 A,a			72.2±10.7 B,b		80.9±6.2 b			81±5.7 a	
TCI (Kcal/day)	2100±645			2540.2±815.7		1852±580.8			2023.8±488.3	
PTN (g)	100.8±27 A			137.3±27.4 B		97.4±45.7			107.6±38.2	
LIP (g)	56.3±23.3 A			76.6±23.6 B		57.2±19.2			65±23.2	
CHO (g)	297.4±92.7			311.2±87.5		236.8±70.8			252.6±77.4	
Muscle Damage	Pre Damage	Post Damage	24h	48h	72h	Pre Damage	Post Damage	24h	48h	72h
CK (U/L)	319.4±106.5	263.4±74.67 A	482.2±103.5 B	427.8±85.5 B	479,7±109.3	351.7±70.4	342.6±82.9	429.6±56.8	366.7±76	322.9±61.8
LDH (U/L)	343.6±126.7	427±162.8 A	410.6±111	448.4±74.6	455.5±153.7 B	407.2±90.42	335.2±162.5	327.2±79.5 A	441.3±130.6 B	409.8±441.8

Note: TCI, total calorie intake. Values are expressed as mean ± SD. Different lowercase letters represent differences between groups ($P \leq 0.05$). Different uppercase letters represent differences between the times within the groups ($P \leq 0.05$). Repeated two-way ANOVA measures and LSD post-test was applied.

after the event ($p = 0.016$) (Fig. 2B). Nevertheless, the jump height and relative jumping power values were similar for both Creatine group and Placebo group throughout the intervention ($p = 0.849$ and $p = 0.731$, respectively).

In relation on Delayed onset muscle soreness (DOMS), Creatine group reported a lower pain pattern in relation to Placebo group during the period of evaluation after the muscle injury protocol (difference between groups: immediate pain ($p = 0.020$), 24 hours ($p = 0.001$), 48 hours ($p < 0.001$), and 72 hours ($p = 0.011$)). There was no significant difference in pain between times in Creatine group. In Placebo group, however, there was a decrease in pain in the period of 48-72 hours after the event ($p = 0.043$) (Table 1).

No differences were observed between Creatine group and Placebo group for CK ($p = 0.719$) and LDH ($p = 0.499$) regarding biochemical markers of muscle damage after performing the muscle injury protocol. Nevertheless, CK values increased 24 h after the event in Creatine group ($p = 0.012$), equaling initial values after 48 hours ($p = 0.206$). Placebo group did not present significant differences on CK after the injury event ($p = 0.169$). On the other hand, the LDH values remained stable in Creatine group up to 48 hours after the injury protocol ($p = 0.783$), showing an increase in relation to the initial value 72 h after the protocol ($p = 0.022$). In Placebo group, LDH elevation occurred 48 hours after the injury event ($p = 0.043$) (Table 1).

Regarding the body weight of the individuals, a difference was observed between Creatine group and Placebo group before supplementation ($p = 0.047$), in which a player mean weight was 70.74 kg in Creatine group and 81.34 kg in Placebo group. After supplementation, there was no statistical difference between Creatine group and Placebo group ($p = 0.077$). Placebo group did not present changes in body weight after supplementation ($p = 0.830$), while the Creatine group increased 1.4 kg of body weight, which refers to 2% when compared to the baseline measurement ($p = 0.003$) (Fig. 4).

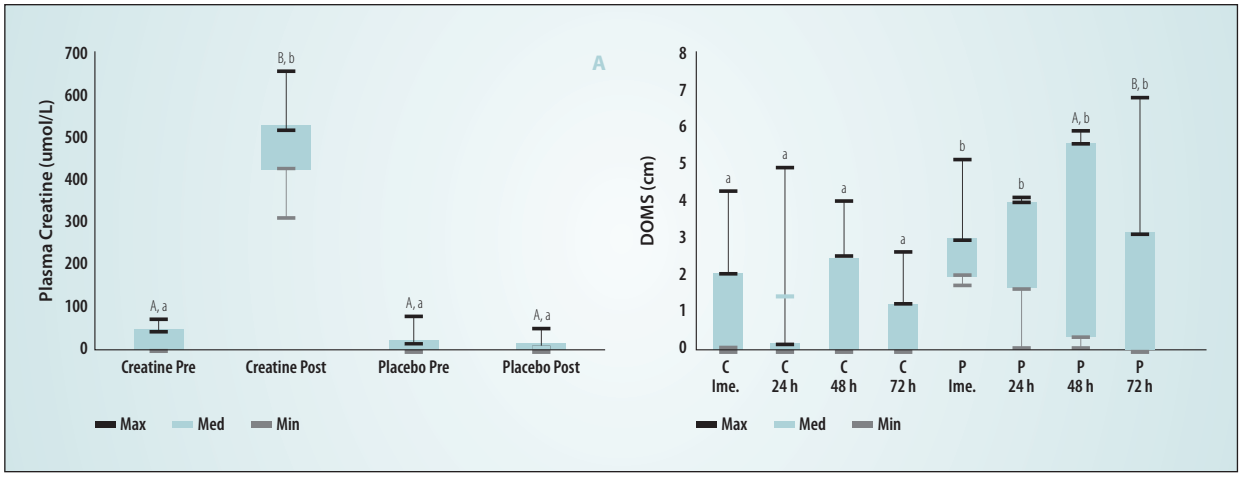

Figure 3. Plasma creatine concentration (A) and Delayed onset muscle soreness (DOMS) (B) after injury protocol (Scale represented from 0 to 5 cm), before and after supplementation with creatine or placebo. Values in median, maximum, and minimum. Different lowercase letters represent differences between groups ($p \leq 0.05$). Different uppercase letters represent differences between the times within the groups ($p \leq 0.05$). Friedman's test. Benjamini-Hochberg FDR post-test was applied to this non-parametric data.

Table 2. Training load quantification.

	Training load (a.u.)	Density (a.u.)	Monotony (a.u)
Week 1	345.0 ± 90.28	570.5 ± 174.5	1.64 ± 0.1417
Week 2	740.0 ± 129.7A	4549 ± 3404A	6.01 ± 4.219A

Values are expressed as mean ± SD. A represents difference between weeks 1 and 2 ($p \leq 0.05$).

Table 2 shows the values of the training load variables. A significant increase was observed in week two when compared to week one in terms of training load, density and monotony variables.

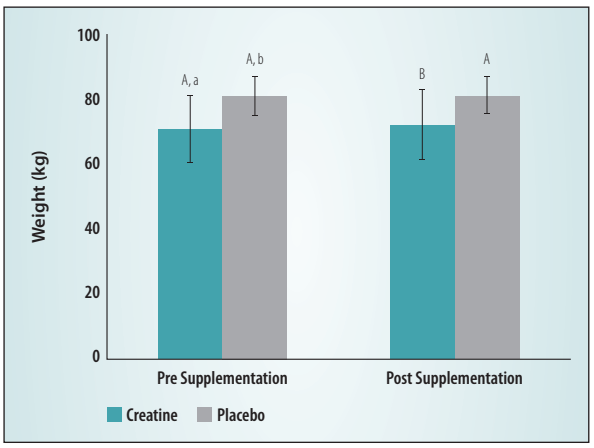

Figure 4. Body weight before and after supplementation with creatine or placebo. Values in mean ± SD. Different lowercase letters represent differences between groups ($p \leq 0.05$). Different uppercase letters represent differences between the times in the groups ($p \leq 0.05$). Repeated two-way ANOVA measures. LSD post-test.

Discussion

The main findings of this study demonstrated that although it was able to attenuate delayed onset muscle soreness, creatine supplementation does not prevent elevated damage muscle markers induced by muscle injury protocol or enhanced performance in volleyball athletes.

Despite the increased availability of plasma creatine, the seven days of supplementation of 0.3 g/kg/day of creatine associated with 1.2 g/kg/day of carbohydrate did not promote better performance in the jumping test. No significant differences were observed in the variables related to jump height and relative jumping power between Creatine group and Placebo group (Fig. 2A, B). The mentioned variables are essential for blocking, serving and attack actions that are present in matches and training of volleyball teams (Tidball, 2005). Specifically, in attack, the higher the player's point of contact with the ball, the greater the projected angle to the ground, which increases the chances of the ball going inside the opposing court, as well as increases the possibility of transposition of the block, without increasing the player's time in the air, giving them the possibility of executing various technical resources (Stanton & Abt, 2000). Our results are similar another study (Kreider et al., 2017), that demonstrated no differences in jumping performance, 1RM (1 repetition maximum), jump between creatine vs placebo supplemented in volleyball athletes.

Similar finding was presented in research (Lamontagne et al., 2011), who demonstrated no changes in jumping performance test in track and field athletes supplemented with creatine plus glutamine or placebo. Different authors attribute the result to the absence of the ergogenic effect of creatine in the maximum jump-

ing ability, as the maximum jumping performance with countermovement is an extremely fast physical exercise (less than one second), thereby possibly not influenced by the increase in creatine and creatine phosphate stocks (Hultman et al., 1996; Greenhaff et al.,1993; Santos et al.2004). According to previous findings (Goodall & Howatson, 2008), improvements in performance is mainly due to the pattern of recruitment of muscle fibers, contractile protein content, and enzymatic activity, variables that are not directly influenced by creatine supplementation. Notably, some studies have demonstrated that creatine supplementation can improve jump performance. Similar to this study (Campillo et al., 2015), with football players improved jumping performance when supplemented with creatine compared to placebo supplemented group. However, unlike our study, the athletes performed specific training to improve jumping performance (plyometric protocols), which leads us to conclude that creatine by itself does not improve performance but, in association with a specific physical training protocols, may show an improvement.

In our study, the purpose of the muscle injury induction protocol was to investigate creatine ability to maintain muscle cell integrity, which is also evaluated through the perception of delayed-onset muscle soreness, which is one of the indirect methods to evaluate muscle injury (Cheung et al., 2003). After skeletal muscle injury, either by mechanical stress or metabolic stress, an inflammation occurs acting the cells of the immune system, such as macrophages and neutrophils, among others. This can be associated with significant increases of inflammatory markers such as interleukin-6 and tumor necrose factor-alfa (Buford et al., 2009). Another fact that emphasizes that delayed-onset muscle soreness is directly related to injury and inflammation is that both pain and inflammation occur in the post-injury period (Kreider et al., 2017). Our data demonstrated a significant decrease in pain perception when volleyball athletes were supplemented with creatine in comparison to placebo supplemented athletes. Indeed, Creatine group presented lower pain perception when compared to Placebo group at all post-injury moments (Fig. 3A).

Corroborating with these results, one study showed lower pain perception in the group supplemented with creatine when compared to the placebo group (Van-Dusseldorp et al., 2018). Nevertheless, the mechanisms responsible for this effect have not yet been elucidated. It has been hypothesized that an increase in intramuscular concentrations of creatine phosphate, which may stabilize sarcolemma, favors creatine ability to reduce inflammation and oxidative stress. Furthermore, increased levels of phosphocreatine and intramuscular glycogen, which may facilitate the delivery of energy to the Ca^{2+}/ATPase pump activity during an effort, modulates calcium (Ca^{2+}) influx and thereby decreases the damage induced by the increase in intracellular Ca^{2+} (Clark, 1996; Saks & Strumia, 1993; Smith, 2004).

On the other hand, few studies in the literature did not show the effect of creatine in reducing the perception of post-exercise soreness, in which no significant difference was found between the creatine-supplemented and non-supplemented groups (Rawson et al., 2007; Rawson et al., 2001). Rawson et al. (2007) study performed a muscle injury induction protocol and concluded that creatine was not able to protect the skeletal muscle from injuries caused by eccentric exercises and that they involve a great application of muscle strength, when evaluated by enzymes released by muscle. Another study used an acute supplementation protocol similar to the present study and found no significant difference between the creatine-supplemented and placebo groups in CK concentrations up to 72 h after the injury event (Rawson et al., 2001).

Our study specifically analyzed the enzymatic activity of CK and LDH, and, no significant difference was found between groups Creatine group and Placebo group at the end of the experimental protocol. Our results were similar to the study of Rawson et al. (2001), in which no significant difference was found between the creatine and placebo-supplemented groups at all times evaluated after performing an injury induced resistance protocol at a 75% intensity of 1 RM.

In contrast to the results presented so far on the protective effect of creatine on skeletal muscle, another study demonstrated that creatine was able to decrease the total CK concentration and prevented the increase of LDH levels in long-distance running athletes (Santos et al.2004). The authors attributed this result to creatine ability to maintain muscle cell integrity by increasing cell volume through water retention, increased glycogen stores, and myofibrillar content. Unlike our study, the exercise performed in the abovementioned study was aerobic (running), and according to Rawson et al., (2007), strength exercises with strong eccentric components are extremely damaging to the skeletal muscles, which may impair any possible protective effect of creatine.

Interestingly, our study demonstrated that the total CK concentration of the placebo group did not show a difference before and after the injury protocol. Nevertheless, according to other research there is a high degree of individual biological variability in serum CK activity (Cooke et al., 2009), making it difficult to

demonstrate a statistical difference when analyzing small number of individuals (Clarkson & Hubal, 2002; Ra et al., 2013). Another interesting fact in our study is that the muscle damage measured by the serum LDH enzyme concentration was significantly higher in Placebo group after 48 h and in Creatine group after 72 h. We believe that this fact may have occurred due to the team's training routine, given that, according to Table 2, the variables of the weekly training loads were different. Furthermore, in the second week, there was an increase in all training variables (training load, density, and monotony). A previous research developed by our group showed that increases in the training load can be associated with higher levels of muscle damage markers (Galan et al., 2018). However, in the present study the injury protocol performed in the second week of training when the high training load could interfere on the CK and LDH values in both groups.

These results can elucidate many hypotheses, such as the placebo effect, as well as the association with the increase in body mass that causes greater energy expenditure, that can interfere on recovery status (Piucco & Santos, 2009) but we observed a decrease in delayed-onset muscle soreness in the creatine group, showing a positive effect of creatine supplementation. In order to monitor food intake, three-day food records were completed pre and post the two weeks of supplementation. The total caloric intake was similar between groups, however, regarding macronutrients (Table 1), Creatine group presented a higher intake of protein and lipid post intervention. Since that calorie intake of the Creatine group was similar between pre and post (p=0.063), and the difference was only in the content of macronutrient of the diet, we consider that this increase did not impact the results of the present study.

Another fact observed in our study only in the Creatine group was the increase in body weight. Researchers have attributed the increase in body weight to the ability of Cr to change cell osmolarity, resulting in water retention (Olsen et al., 2006). Our results were similar to several studies, which also observed changes in body weight in individuals with creatine supplementation (Clarkson & Hubal, 2002; Cooke et al.,2009; Campillo et al., 2015).

Body mass can directly influence speed, explosive force and the number of jumps, since that acceleration equation is calculated by force divided by mass. Movements that requires speed and explosive power, also can be influenced by other factors such as jump technique, jump performance, impact, training intensity, and another external factors to the training (Piucco & Santos, 2009) corroborating to the results found in the present study.

A previous study demonstrated that the increase in body weight promoted by creatine may cause an ergolytic effect in sports performance that are not favorable to the increase in total body mass, due to the need of a greater force to displace this mass (Branch, 2003).

In conclusion, our data demonstrated that, although the training load was increased, and consequently the muscle damage markers, without altering the athlete's performance, creatine supplementation was able to attenuate late-onset muscle pain.

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