©Journal of Sports Science and Medicine (2018) **17,** 279-288
http://www.jssm.org

Research article

Creatine Supplementation Supports the Rehabilitation of Adolescent Fin Swimmers in Tendon Overuse Injury Cases

Imre Juhasz [1]✉, Judit Plachy Kopkane [2], Pal Hajdu [3], Gabor Szalay [3], Bence Kopper [4] and Jozsef Tihanyi [4]

[1] University of Physical Education, School of Doctoral Studies, Hungary; [2] University of Miskolc, Faculty of Health Care, Hungary; [3] Eszterhazy Karoly University of Applied Sciences, Institute of Sport Sciences, Hungary; [4] University of Physical Education, Department of Biomechanics, Hungary

Abstract

Our purpose was to investigate the effect of creatine (Cr) supplementation on regeneration periods in tendon overuse injury rehabilitation of adolescent fin swimmers. The participants of this study were injured adolescent competitive fin swimmers (n = 18). The subjects were randomly assigned the creatine (CR) or placebo (PL) groups with a double-blind research design. The subjects were given Cr supplementation or received the placebo as part of the conservative treatment of the tendinopathy. We measured the segmental lean mass (SLM;kg), the ankle plantar flexion peak torque (PFT;N·m), the pain intensity (NRS;values), prior to immobilization, after immobilization (R2) and after the 2nd (R4) and 4th (R6) weeks of the rehabilitation period of the injured limb. The creatine kinase (CK; U/L) enzyme levels were measured before immobilization, and then every 24 hours for four days. There was a significant decrease in SLM (CR by 5.6% vs. PL by 8.9%; $p < 0.03$) after two weeks of immobilization in both groups ($p < 0.001$). After four weeks rehabilitation the SLM significantly increased in both groups (CR by 5.5% vs. PL by 3.8%; $p < 0.01$). The percent changes in PFT after supplementation in R4 ($p < 0.001$) and R6 ($p < 0.03$) were significantly different between groups. There was a significant percent increase measured in the CR group (R4 by 10.4%; $p < 0.001$; R6 by 16.8%; $p < 0.001$), whereas significant, but lower growth found in the PL group also took place (R4 by 7.1%; $p < 0.001$; R6 by 14.7%; $p < 0.001$) after four weeks of rehabilitation. Significantly faster decrease were found in NRS of CR versus PL group during treatment ($p < 0.02$). We detected significantly lower CK levels increase at the CR group compared to the PL group. The results of this study indicate that Cr supplementation combined with therapeutic strategy effectively supports the rehabilitation of tendon overuse injury of adolescent fin swimmers.

Key words: Tendinopathy, pain, therapeutic strategy.

Introduction

Using fins, swimming becomes more effective concerning speed of movement, because the contact area of the feet is increased, allowing greater compression force and exertion on the water (Zamparo et al., 2002). However, the plantar and dorsal flexors should produce considerable greater force, to push forward the body with higher acceleration, than in normal swimming. Therefore, the load on the tendons and ligaments is increased, that may cause overuse effect and as a consequence, may result in tendon injury, i.e. acute or chronic tendinopathy (Verni et al., 1999). In fact, it was observed that child and adolescent fin swimmers often suffer from tendon damage of the long big toe flexor (musculus flexor hallucis longus; FHL) (Sereni et al., 1981).

There are several training methods and physiotherapy interventions which help prevent tendon injury or curing, by reconstructing the damaged tissues. Nowadays, creatine administration is increasingly spreading in muscles and tendon rehabilitation is based on the following findings. The oral creatine (Cr) supplementation increases muscle performance, enhances muscle mass and muscle strength during high-intensity exercise (Kreider et al., 1998; Terjung et al., 2000). In recent years, Cr supplementation for various diseases, muscle damage and its effect on their rehabilitation have been the focus of researchers. They observed that Cr strengthened the functional capacity of the muscles in neuromuscular diseases such as muscular dystrophy (Tarnopolsky and Martin, 1999; Walter et al., 2000). Kley et al. (2013) demonstrated that Cr supplementation significantly increased the maximum muscle contraction and the lean body mass in cases of muscular dystrophy patients. Recent evidence has shown that Cr supplementation is an effective therapeutic strategy in the treatment of muscle and ligament injuries caused by physical activity, and it supports unused muscle atrophy rehabilitation (Cooke et al., 2009; Hespel and Derave, 2007; Hespel et al., 2001; Op 't Eijnde et al., 2001; Pearlman and Fielding, 2006; Tarnopolsky and Martin, 1999; Walter et al., 2000), with the moderation of the appearance of inflammatory markers caused by muscle injury among others (Santos et al., 2004).

It is well documented in the literature, that delayed onset of muscle soreness is accompanied with high creatine kinase (CK) activity in the blood (Clarkson et al., 1986; Hartmann and Mester, 2000; Hortobagyi and Denahan, 1989). However, no CK elevation was observed during tendinopathy. So that on the basis of CK content muscle and tendon injury can be distinguished.

Studies have proved that Cr administration is widespread not only in adult athletes, but also among young athletes (under 18 years of age) who use Cr for the purpose of physical condition enhancement (Evans et al., 2012; Metzl et al., 2001). However, to the best of our knowledge, no data are available regarding injured adolescents athletes' creatine consumption.

Therefore we aimed to investigate the effects of Cr supplementation on the recovery of tendinopathy, of the FHL, in adolescent fin swimmers. We hypothesized that Cr

Received: 22 January 2018 / Accepted: 10 April 2018 / Published (online): 14 May 2018

supplementation would prevent muscle mass and strength loss during immobilization and would reduce pain caused by inflammation. Also, we hypothesized that during the treatment, Cr administration would increase the effect of therapy.

Methods

Participants

The participants of this study were injured adolescent male and female competitive fin swimmers (n = 18; male = 10, female = 8; years = 15.1 ± 1.5, range: 12-18 years; body mass = 60.8 ± 8.9, range: 50.5-82.5 kg; height: 1.71 ± 0.06 range: 1.59 -1.84 m). The subjects were randomly assigned to the Cr (CR; n = 9, male = 5, female = 4; years = 15.5 ± 1.4) or placebo (PL; n = 9, male = 5, female = 4; years = 14.8 ± 1.6) group with a double-blind research design. The subjects were given Cr supplementation (CR), or received placebo (PL) as part of the conservative treatment of the soft tissue (tendinopathy of the FHL). We calculated the biological age of the subjects using methods of Mészáros et al. (1990) and found no statistically significant difference between biological and chronological age in either groups. Therefore, we assumed that the maturity status equally influenced the intervention effects in both groups in average. The exclusion criteria were abnormal renal function, albuminuria, amino acid supplementation or use of acute medication during the study. Each subject and their legal representatives signed a letter of consent after receiving a description of the study. The study was approved by the research ethics committee of the university. The study is aligned with the revised directives of the Declaration of Helsinki (1964) 2013, and the International Society of Sports Nutrition (ISSN) resolution which was adopted in 2017 (Kreider et al., 2017).

Experimental conditions

Table 1 contains the treatment phases, the measured variables, the duration of the creatine supplementation, and the time of measurements.

The acute phase of tendinopathy treatment was carried out at the homes of the injured participants in line with the specialist requirements. The recovery and maintenance phase of the rehabilitation exercise program was carried out independently according to the instructions of a physiotherapist. Treatment is based on sound principles but was individualized to suit particular needs. The physiotherapy exercises and methods were used identically in both groups for all subjects. The physiotherapist was the same in every case. Individual nutrition was compiled by a nutritionist for all subjects during treatment. The subjects kept a treatment log.

Tendinopathy treatment plan

Physical examination was performed under clinical conditions. The physical examination included inspection for muscle atrophy, asymmetry, swelling, erythema, and joint effusions. Range-of-motion testing was often limited on the symptomatic side. If the diagnosis was unclear, additional imaging procedures were performed. All subjects were diagnosed with subacute (the duration of symptoms 4-6 weeks) FHL tendinopathy due to overuse (Mueller-Wohlfahrt et al., 2013). Tendinopathy is a clinical condition in which the tendon and tendon area become inflamed due to overuse (Sharma and Maffulli, 2005). Following the consultation with a medical specialist, in line with the clinical recommendations (Wilson and Best, 2005), the entire rehabilitation period was determined for six weeks, which was divided into three phases (acute, recovery, and maintenance). The acute phase consisted of a two-week relative immobilization period, during which the injured body part was fixed with an elastic bandage (casting or other rigid forms of immobilization are not considered good medical management today), and home recovery was prescribed with raising and icing of the damaged leg, and crutches had to be used for walking. During the first week of the acute phase every subject followed the R.I.C.E. (Rest, Ice, Compression, Elevation) principle (Järvinen et al., 2007) – which is also used in clinical practice for the immediate treatment of soft tissue injuries. A progressive isometric workout was prescribed for the second week of the relative immobilization period and adapted to pain intensity (early mobilization). Recovery phase: After the acute phase, rehabilitation should emphasize appropriate loading of the tendon and its muscle to provide proper stimuli for healing. Healing and recovering muscle strength and flexibility are most important at this stage. Protected motion is gradually increased to full passive, then active range of motion. During the 2nd phase, two weeks of physical therapy, mobilization of the muscles of the lower limbs, isometric, isotonic, and isokinetic exercises were performed. Maintenance phase: The last two weeks, the final phase of rehabilitation, is the most important for restoring maximum performance and minimizing the risk of reinjury. Strength and flexibility must be fully restored. Sport-specific stretching can be added when strength is adequate. Table 2 contains the recommended exercises and methods. The rehabilitation status was individually controlled by a physiotherapist.

Table 1. The process of the study.

Intervention		Immobilization		Rehabilitation	
		Acute Phase		Recovery Phase	Maintenance Phase
Duration		(1-2 weeks)		(3-4 weeks)	(5-6 weeks)
Cr supplement		5 days loading phase followed by a 37 days maintenance phase			
Variables	**SLM**	Baseline	R2	R4	R6
	PFT		R2	R4	R6
	NRS	Baseline	R2	R4	R6
	CK	Baseline, 24, 48, 72, 96			

NRS-numeric rating scale; SLM-segmental lean mass; PFT-plantar flexion torque; CK- creatine kinase), the duration of the creatine supplementation (Cr supplement), and the time of measurements (Baseline-prior the immobilization; R2 - after 2 weeks of the acute phase; R4 - after 2 weeks of the recovery phase; R6 - after 2 weeks of the maintenance phase; 24, 48, 72, and 96 hours after baseline.

Table 2. Physiotherapy treatment plan of FHL tendinopathy for adolescent fin swimmers.

PHASES	PERIODE (programme per week)	EXERCISE, METHODS (main exercises)	FREQUENCY (pc/block/day)	AIM
Acute	1-2	**RICE and Isometric Exercises:** Back location: -Flex the musculus quadriceps femoris; hold for 5 sec. than relax 2 sec. -Flex the leg extensor muscles; hold for 5 sec. than relax 2 sec. Hallux is in neutral state.	5-10 exercises 3-8 times per day (at home)	Pain relief; Holding muscle strength
Recovery	3-4	**Improving Range of Motion:** Stretching with PIR and PNF technique: -Hallux and ankle extension; hold for 5 sec.; than relax 2 sec. -Active strengthening, own muscle strength: -Hallux and ankle flexion: hold for 5 sec. than relax 2 sec.	3 PIR ex/10-20 pc 2 PNF ex/10-20 pc (with physiotherapist) 5 Strength ex/10-20pc (at home)	Holding the ankle and toe mobility Strength the plantar flexor muscles with own, active muscle exercises
Maintenance	5-6	**Strengthening:** Own muscle strength: -Hallux and ankle flexion: hold for 5 sec. than relax 2 sec. Resistance ex. with bands: -Hallux and ankle flexion: hold for 5 sec. than relax 2 sec. Sport-specific physiotherapy: -Prone position; flex the knees, uncles and toes than relax. With bands also. Strength and balance ex. with dyn-air: -Step up to the dyn-air with the patient leg, flex the hallux and the ankle and step back Strength and flexibility with Fit-Ball: -Sit on the Fit-Ball; suspension on the ball, step forward and flex the toe and ankle to the floor, step back - Sit on the Fit-Ball; lower extremity extended, toe and ankle extended, hold for 5 sec. than relax 2 sec.; step back	5ex/10-20pc (with physiotherapist) 5ex/10-20pc (with physiotherapist) 2ex/10-20pc 2ex/10-20pc 2ex/5-10pc	Strength the plantar flexor muscles with gymnastic devices

The rehabilitation status was individually controlled by a physiotherapist. PIR - postisometric relaxation; PNF - proprioceptive neuromuscular facilitation; ex - exercises; pc - pieces.

Subjects were instructed not to take part in any other treatments such as non-steroidal analgesic and anti-inflammatory drugs, or ultrasound during treatment. The subjects signed informed consent forms agreeing with the requirement.

Creatine supplementation protocol

The definition of Cr supplementation was adjusted to reference (Kreider et al, 2017), and to our earlier study (Juhasz et al., 2009). We asked the subjects of the CR group to take 20g 100% micronized Cr monohydrate (Bio-Tech, Inc., Ft. Lauderdale, FL, US) during the first five days (loading phase). The total daily dose was divided into 4 x 5 g portions. A dose of the total weight was 12g including 5g Cr monohydrate, 7g dextrose, and 0.075g ascorbic acid. The subjects were instructed to consume the dissolved mixture after getting up, before breakfast, 30 minutes before lunch, in the afternoon at 4 pm., and before going to bed. The PL group consumed a dextrose, ascorbic acid, flour mixture, with the taste, texture, and appearance equivalent with the mixture of the CR group. The mixture had to be dissolved in 0.4 liter of water prior to usage. During the remaining 37 days (maintenance phase) the total daily dose was 1 x 5 g Cr or placebo were given daily before breakfast in mixture described above. Participants reported no side effects during treatment.

Direct Segmental Multi-Frequency Bioelectrical Impedance Analysis (DSM-BIA)

We measured the segmental lean mass (SLM; kg) of the injured limb with the DSM-BIA (Bartels et al., 2015) method prior to immobilization, immediately after immobilization and during the 2nd and 4th week of the subsequent period of rehabilitation. The subjects were measured in

each case: in the morning, on an empty stomach, after using the toilet and, in underwear. The InBody 720 (Biospace Co., Ltd., Seoul, Korea) was used to measure DSM-BIA (1–1000 kHz; $r^2 = 0.99$ compared with DEXA (Lim et al., 2009)). Testing was conducted according to the manufacturer instructions. The subject stepped on the foot electrodes barefoot and stood still until SLM was measured. The subject grasped the hand electrode cables, and gently held on to the thumb electrode and the palm electrode. Hands were held 15° away from the body, until measuring was completed. The inbuilt software was used to calculate SLM values.

Experimental procedure for the measurement of Plantar Flexion Torque (PFT)

A custom-made dynamometer measured the ankle plantar flexion isometric peak torque. The dynamometer consisted of an aluminum plate, and two alloy aluminum single point load cells (type: AG100; Lorenz Messtechnik GmbH, Alfdorf, Germany), which were placed below the plate. The non elastic strap with metal reinforcement was pulled through the aluminum plate, and was fixed to the load cell. The nominal force which was measured by the device (combination of two load cells) is 2000 Newtons (N); its precision is 1 N and its resolution is 0.1 N. The dynamometer was easily portable (since its weight was less than 3 kg with small external dimensions 40 × 40 × 10 cm). The transducer signal was conditioned with an electronic board equipped with an onboard analogic low pass filter (cut-off frequency: 10 Hz). A digital screen could either continuously display the force or retain the maximal value in the flexion direction.

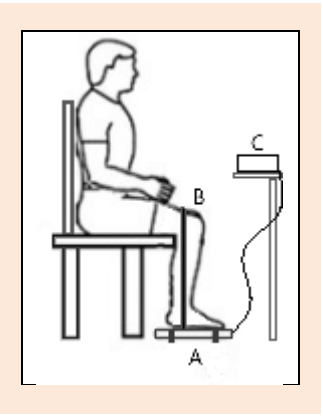


Figure 1. The Measurement of Plantar Flexion Torque. The Experimental Setup for Measurements of Ankle Plantar Flexion Torque (PFT; Mmax; N·m). Dynamometer (A), Non Elastic Strap (B), Digital Screen (C).

The subjects were seated on a chair adjusted to the height of the subject in order to obtain a right angle at the hip, knee and ankle joints. The feet were held flat on the dynamometer. For ankle plantar-flexion measurement, the strap was placed and held distally on the thigh and passed directly over the external malleolus. The subject was asked to pull against the strap by extending his ankle while pushing with the sole of their foot while trying to lift the heel. The experimental setup for maximum PFT measurements is shown in Figure 1. PFT was measured in the sitting position with the hip and knee joints at 90 degree angles and the ankle in a neutral position (Figure 1).

Subjects were verbally encouraged to produce their maximal ankle plantar-flexion strength. Two trials were recorded, consisting of two 2-4 second maximal contractions separated by a 30 second rest period. If the relative difference between these two maximal voluntary contractions (MVC) was within 10%, no additional trials were required. If not, additional trials were proposed as long as two reproducible MVCs were obtained. The maximum value of the two reproducible trials was retained for further analyses. The peak torque was computed. The experimental conditions were the same in all cases. Two independent evaluators performed the measurement to assess reliability.

Numeric rating scale for pain assessment

Instructions (McCaffery and Beebe, 1993)**:**

1. The patients were asked the following questions:
   - What number would you give your pain right now?
   - What number on a scale from 0 to 10 would you give your pain when it is the worst that it gets and when it is the best that it gets?
   - At what number is the pain at an acceptable level for you?
2. When the explanation suggested in #1 above was not sufficient for the patient, it was sometimes helpful to give further explanation or to conceptualize the Numeric Rating Scale in the following manner:
   - 0 = No Pain
   - 1-3 = Mild Pain (nagging, annoying, interfering little with ADLs)
   - 4–6 = Moderate Pain (interferes significantly with ADLs)
   - 7-10 = Severe Pain (disabling; unable to perform ADLs)

   (ADLs: Activities of Daily Living)
3. Our team, in collaboration with the adolescent/family (if appropriate), could determine appropriate interventions in response to the Numeric Pain Ratings.

Blood sampling and metabolite measurements

Creatine kinase (CK) was assessed before the immobilization (baseline), and then every 24 hours for four days. Every time, before sampling, subjects sat quietly for 5 minutes. For serum CK, blood was drawn from the antecubital vein into a 10 mL collection tube via a Vacutainer apparatus. The blood samples were allowed to clot at room temperature for 10 minutes and centrifuged for 15 minutes. Serum was separated and frozen at -20°C for subsequent analysis. Total CK was determined by Beckman DU 640 spectrophotometer (Beckman Instruments, Inc., Fullerton, CA, US) in duplicate, at 25°C, using a commercial test kit (Labtest, Sao Paulo, Brazil).

Statistical analysis

Because of the complexity of the study, the limited number of subjects available with similar injury and the basis of previous studies using similar number of subjects, we concluded that for the purpose of determining statistical

significant differences the limited sample size will be sufficient. All statistical computations were run on the measured raw datasets. The Shapiro-Wilk's W test was carried out for each variable for normality. All of the variables were normally distributed. Fisher's exact test was used to compare the homogeneity of the variances. Two-way analysis of variance (ANOVA) was applied when the effect of the immobilization and the rehabilitation program was tested (specifically for PFT a 2x3, for DSM-BIA and for NRS a 2x4 and for CK a 2x5 model was used for the comparison of the measured data). Repeated measures ANOVA was used to compare values within the groups, and also on the basis of the repeated measures ANOVA results intraclass correlation coefficient-ICC, standard error of measurement-SEM and minimal difference-MD, was calculated for CR and PL, to verify the reliability of the procedure, in accordance with Vincent and Weir (2012) and Weir (2005). Tukey HSD post hoc analysis was carried out for the groups when the ANOVA confirmed significant difference. Statistica 12.6 (StatSoft Inc., Tulsa, US) software served for statistical analysis. All data in tables, figures, and texts are given as means ± SD. A value of $p < 0.05$ was considered significant and indicated in the text.

Results

Direct Segmental Multi-Frequency Bioelectrical Impedance Analysis (DSM-BIA)

After two weeks of relative immobilization of the injured leg, the SLM significantly decreased ($p < 0.01$) in both groups. The SLM decreased by 8.9 ± 0.9% (-0.65 ± 0.09 kg) in PL and by 5.6 ± 0.5% (-0.43 ± 0.05 kg) in CR, respectively. We found a significant difference ($p < 0.05$) between the two groups after immobilization (statistical analysis was calculated using a 2X4 ANOVA, interaction between groups: $F=57.47$, $p < 0.01$). The next four weeks of the active rehabilitation program increased the injured leg's SLM in both groups. During the four weeks active rehabilitation period, we detected a significant increase of 5.5 ± 0.6% in the CR group (+0.4 ± 0.04 kg; $p < 0.01$), and also a significant, but lower growth of 3.8 ± 0.8% in the PL group (+0.25 ± 0.06 kg; $p < 0.01$), compared to the values after the immobilization. After the 4-week period of active rehabilitation, the SLM was significantly different from baseline in PL (-0.4 ± 0.04kg; $p < 0.01$). In contrast, CR group reached a state of baseline. We found a significant difference ($p < 0.01$) between the two groups after four weeks of active rehabilitation. For CR ICC = 0.99, SEM = 0.86kg, MD = 2.37kg; for PL ICC = 0.99, SEM=0.58kg, MD = 1.6kg (Table 3).

The Plantar Flexion Peak Torque (PFT)

The PFT (Mmax; N·m) values were not measurable prior to immobilization. There was a significant increase measured in the CR group (R4 by 10.4 ± 2.9%; $p < 0.01$; R6 by 16.8 ± 1.7%; $p < 0.01$), whereas significant but lower growth found in the PL group also took place (R4 by 7.1 ± 2.3%; $p < 0.01$; R6 by 14.7 ± 2.3%; $p < 0.01$) after four weeks of active rehabilitation (Figure 2). The percentage changes in PFT were significantly different between the experimental groups after treatments (statistical analysis was calculated using a 2X3 ANOVA, interaction between groups: $F = 24.6$, $p < 0.01$) CR vs PL; R2-R6; 28.8 ± 3.1% vs. 22.8 ± 2.8%; $p < 0.01$). There was a significant difference in the PFT (CR vs. PL; R2 = 103.2 ± 10.8 vs. 95.9 ± 5.5, $p < 0.05$; R4= 113.8 ± 11.1 vs. 102.7 ± 4.6, $p < 0.01$;

Table 3. Effect of creatine supplementation on the injured leg's lean mass (SLM, kg) during immobilization and rehabilitation. Values are mean (± SD).

Conditions	Immobilization			Rehabilotation weeks		
	Baseline	R2	P value	R4	R6	P value
CR group	7.76 (.92)	7.33 (.89)	.001	7.47 (.90)	7.74 (.91)	.001
PL group	7.29 (.59)	6.64 (.53) *	.001	6.75 (.55) *	6.89 (.57) *	.001

The injured leg's segmental lean mass (SLM; kg); prior to the immobilization (Baseline) and after acute (R2); recovery (R4) and the maintenance (R6) phase of the rehabilitation of the creatine (CR) and placebo (PL) groups. The P values refer to the treatment effect during relative immobilization and active rehabilitation; * Indicates a significant difference between the CR and PL groups ($p < 0.05$).

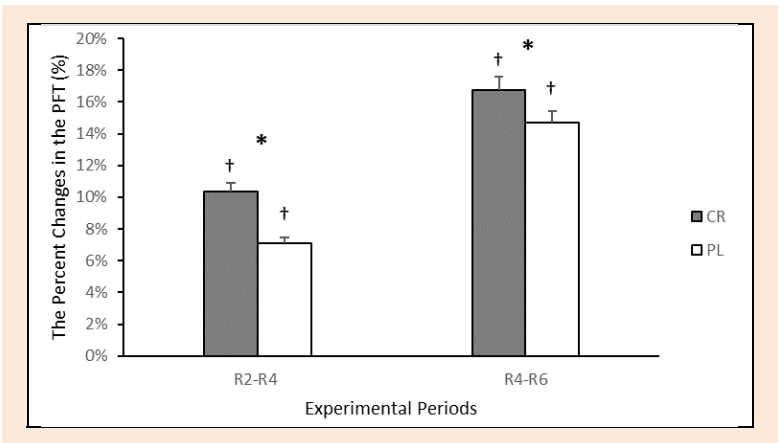


Figure 2. The percentage (5) change of the plantar flexion peak torque at the recovery (R2-R4) and during the maintenance phases (R4-R6) in the CR and PL groups. * Indicates significant differences between the CR and PL groups ($p<0.05$). † Indicates significant relative change in the experimental groups ($p<0.05$). Values are means ± SD.

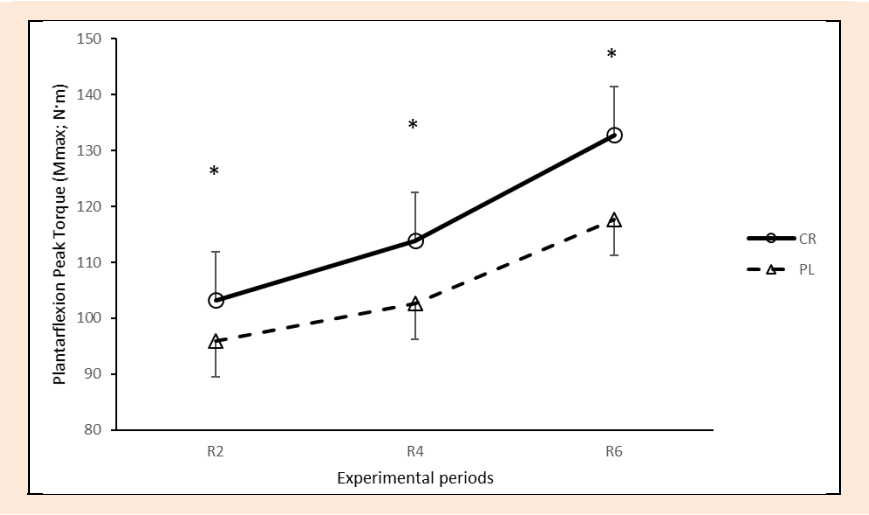


Figure 3. The plantar flexion peak torque (PFT; Mmax; N·m) of the injured foot in the experimental periods. The PFT of the injured foot after the acute (R2), recovery (R4) and maintenance (R6) phase of rehabilitation in the creatine (CR) and placebo (PL) groups. * Indicates significant differences between the CR and PL groups ($p < 0.05$). Values are means ± SD.

R6 = 132.8 ± 12.4 vs. 117.7 ± 5.2, $p < 0.01$) after two weeks of relative immobilization followed by four weeks of active rehabilitation between the two groups. For CR ICC = 0.99, SEM = 1.55N·m, MD = 4.28N·m; for PL ICC = 0.97, SEM = 1.8N·m, MD = 4.97N·m. (Figure 3).

Numeric Rating Scale (NRS; 0-10) for pain assessment

The pain intensity (NRS) was measured on a scale ranging from 0-10. Before the immobilization (Baseline), and after the acute (R2), recovery (R4) and maintenance (R6) phase of rehabilitation. The pain intensity was significantly lower two weeks after relative immobilization (Baseline-R2; decreased by 64.4 ± 9.6%; $p < 0.01$), after the recovery (Baseline-R4; decreased by 93.1 ± 8.2%; $p < 0.01$) and the maintenance (Baseline-R6; decreased by 98.4 ± 4.8%; $p < 0.01$) phases of the active rehabilitation in the CR group. The result in the PL group was about the same but the decrease of pain intensity happened in a slower pace during the experimental periods (Baseline-R2; decreased by 57.7 ± 9.4%; Baseline-R4 by 72.4 ± 8%; Baseline-R6 by 88.8 ± 9.6%; $p < 0.01$). In the percentage change there was a significant difference between groups during active rehabilitation (statistical analysis was calculated using a 2X4 ANOVA, interaction between groups: $F = 6.39$, $p < 0.01$). Significantly faster decrease were found in the CR group during rehabilitation versus the PL group (CR vs PL; R2-R6; 94.4 ± 16.7% vs. 75 ± 20.4%; $p < 0.02$). For CR ICC = 0.14, SEM = 2.49, MD = 6.88; for PL ICC = 0.88, SEM = 0.81, MD = 2.25 (Figure 4).

Creatine Kinase (CK)

In the CR group the CK significantly elevated by 3.2 ± 1.7% ($p < 0.01$) during the first 24 hours, then significantly decreased by 10.1 ± 7.1% ($p < 0.01$) during the next three days. In the PL group the CK significantly increased further by 12.9 ± 5.3% ($p < 0.01$) during the first two days, then significantly decreased by 9.3 ± 3.1% ($p < 0.00$) during the next two days. A significantly relative difference was found 48 hours after the beginning of treatment between the experimental groups (CR vs PL; 24 – 48 hours; -0.1 ± 1.7% vs. +6.0 ± 3.1%; $p < 0.01$). We observed no significant difference in CK (U/L) levels between the two groups (statistical analysis was calculated using a 2X5 ANOVA, interaction between groups: $F=13.82$, $p < 0.01$) before the start of treatment (CR vs PL; Baseline = 444.2 ± 184.3 vs. 428.9 ± 146.8), and 24 (456.4 ± 184.7 vs. 453.8

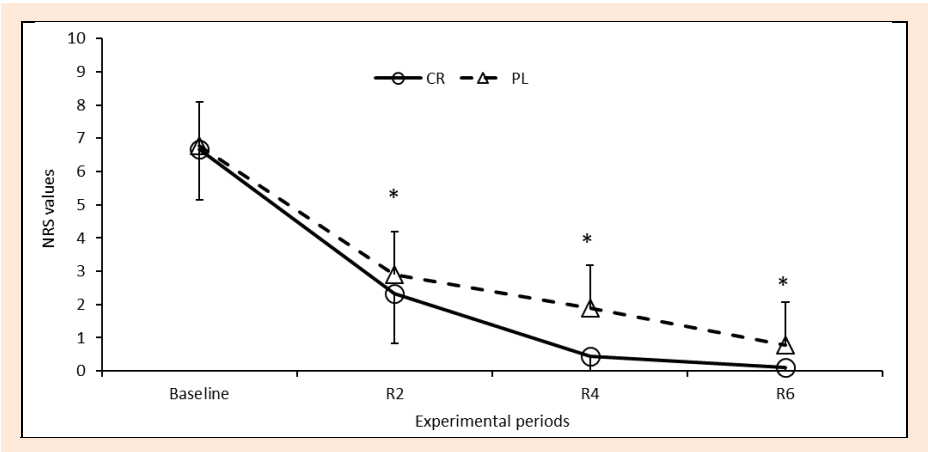


Figure 4. The intensity of pain (Numeric Rating Scale- NRS; 0-10 values) during the experimental periods. Values of NRS before immobilization (Baseline) and after acute (R2), recovery (R4) and maintenance (R6) phases of creatine (CR) and placebo (PL) groups. * Indicates a significant difference between the PL and the CR groups ($p < 0.05$). Values are means ± SD.

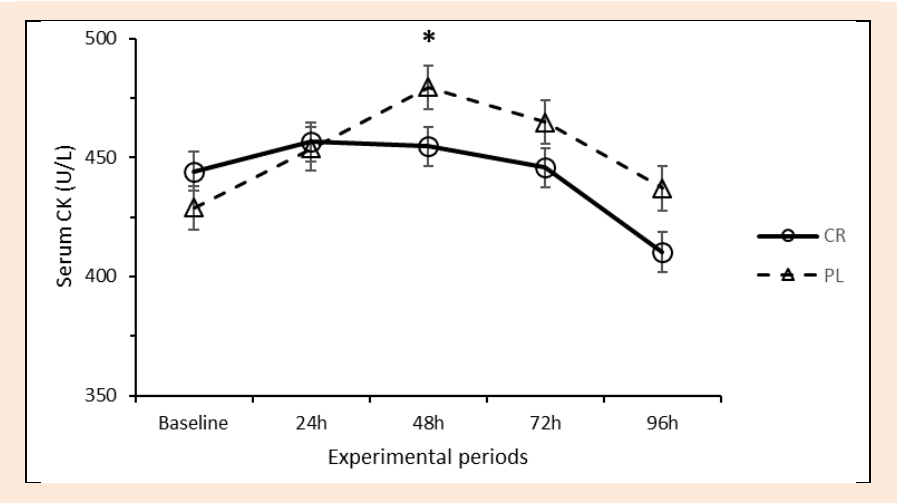


Figure 5. Serum creatine kinase (CK; U/L) in the experimental periods. CK prior to treatment (baseline) and after the start of treatment at 24, 48, 72, and 96 hours, creatine (CR) and placebo (PL) groups. * Indicates significant relative change between groups ($p < 0.05$). Values are means ± SD.

± 149.9), 72 (445.7 ± 181.3 vs. 464.8 ± 155.3) 96 (410.3 ± 192.2 vs. 437.0 ± 149.3) or hours after the beginning of treatment. For CR ICC = 0.99, SEM = 3.48 U/L, MD = 9.6 U/L; for PL ICC = 0.99, SEM = 5.44 U/L, MD = 15.01 U/L (Figure 5).

Discussion

Our paper presents a novel research on the effect of Cr supplementation on regeneration periods in tendon overuse injury rehabilitation of adolescent fin swimmers. The results in our present study demonstrate that a therapy-strategy combined with Cr supplementation efficiently supports the tendinopathy rehabilitation of adolescent fin swimmers. It moderates the muscle and strength loss during rehabilitation after injury; decreases pain intensity and significantly shortens the entire rehabilitation period. The limitation of this study is the small sample size. However, our results give some preliminary basis for further research.

Relatively little data is available regarding young athletes' Cr consumption. We know that more and more athletes under 18 years of age use Cr supplements for the purpose of physical enhancement (Evans et al., 2012; Metzl et al., 2001). Some authors do not recommend creatine consumption for children and adolescent (Metzl et al., 2001; Unnithan et al., 2001), in contrast, no international organization prohibits the use, since there is no evidence that the use of creatine would be harmful to young athletes' physical or mental health (Kreider et al., 2017). In this regard, while planning our study we considered the 2017 resolution of the ISSN authoritative (Kreider et al., 2017).

Compared to the number of writings about the swimmers' motion system damage, we found only a few studies that discussed the active motion system injuries affecting fin swimmers, their causes and treatment options (Sereni et al, 1981; Verni et al., 1999; Zamparo et al., 2002). The muscle and tendon damage caused by omission, physical inactivity and decreased muscle function results in muscle atrophy, which in turn significantly increases the time of rehabilitation until the athlete is able to reach an active performance levels again. The strategies are of great importance for athletes which can reverse or prevent significant functional deterioration caused by muscular dystrophy. The data presented in this study demonstrates for the first time, that Cr supplementation combined with therapeutic strategy effectively supports the rehabilitation of tendon overuse damage of adolescent fin swimmers.

The lower limb muscles and tendons provide the primary propulsive force in fin swimming. The muscles and tendon injuries are primarily due to overuse and wrong technical implementation. The identification and accurate diagnosis, the treatment and rehabilitation of adolescent athletes, requires more than just conservative treatment and rest. There is evidence that young athletes better and more quickly regenerate after muscle and tendon injury (Best, 1995), but there is a likelihood that these injuries become chronic, if the adolescent athletes undergo excessive or repeated physical stress. However, proper treatment and careful monitoring can minimize the possible irreversible damage (Valovich McLeod et al., 2011).

The FHL muscle is the strongest muscle among the deep digital flexor muscles, is involved in plantar flexion, supination and approximation of the foot (Langley et al., 1974). The FHL muscle is part of the propulsion power transmission during fin swimming. If the tendon is damaged, the person usually feels pain in the whole ankle. This area is swollen, warm and painful to touch. In mild cases, pain may occur during rest after a strenuous activity, but in severe cases, pain comes during exercise and immediately after. The injury's acute, gradually appearing form, might considerably affect performance, but it responds well to conservative medical treatment. However, the healing of the chronic type may take time due to the lack of treatment and inappropriate rehabilitation (Kannus et al., 2002; Sereni et al., 1981).

The preservation of the skeletal muscle mass plays an important role in the rehabilitation of muscle and tendon damage. In our study, which supports the results of previous studies (Cooke et al., 2009; Hespel et al., 2001; Johnston et al., 2009; Pearlman and Fielding, 2006), we found that Cr supplementation can reduce muscle and strength loss during two-weeks of relative immobilization. The

four-week active rehabilitation period, which follows the relative immobilization period, increases muscle hypertrophy and strength, and in this way considerably shortens the entire rehabilitation time. Reduction in SLM was significantly greater in PL than in CR, indicating less loss in the muscle mass. The attenuated reduction most probably can be attributed to the Cr supplementation. In light of this result, it could be assumed that the CR group, would increase SLM to a greater extent during therapy applied after immobilization, than the PL group. Indeed, combining specific therapy with creatine supplementation the CR group increased SLM significantly and approached the base level.

The gains in body mass observed are likely due to water retention during supplementation. Cr is an osmotically active substance. Thus, any increase in the body's Cr content should result in increased water retention (Hultman et al., 1996; Volek et al., 1997) and consequent gains in total body water (TBW) and SLM.

Because Cr is primarily stored intramuscularly (95%), it is more likely that the increase in TBW would be intracellular because of the direct influx of water into the muscle cell. Increase in cell volume appears to be an anabolic proliferative signal, which may be the first step in muscle protein synthesis (Haussinger et al., 1994; Haussinger, 1996).

In this present study we have not examined TBW content. It is also important to know that the BIA method is very sensitive to the change in this indicator, and even with a slight increase in TBW, this results in a higher SLM.

Our results suggest that Cr supplementation attenuates the muscle mass loss, and also supports the faster recovery of muscle size. Larger mass and more muscle fibers would potentially have a greater total capacity to store and exploit ingested Cr (Brault and Terjung, 2003). Therefore, it is possible that fin swimmers' lower limb high muscle mass, have a great potential to respond to Cr supplementation related protective effects.

After any injury of muscle or delayed onset muscle soreness (DOMS), the force generation capacity of the muscle decreases. The reason of this force reduction is partly due to the pain that inhibits the muscle to exert maximum force. Unfortunately, we do not have data about the maximum strength of the FHL, that could have been observed before injury, to estimate the force reduction caused by the tendinopathy. In our study, the torque increased almost linearly during the immobilization in both groups that can be attributed to the decreasing pain. As the rate of force increase is similar in both groups, we cannot state that Cr supplementation caused this elevation in torque. During therapy, both groups increased further torque production, but CR enhanced force with greater rate than PL did, which may be the consequence of Cr supplementation.

We first investigated the pain intensity change of overuse tendon damage to alternative treatment strategy in adolescent fin swimmer cases. After two weeks of immobilization, the pain decreased in both groups, but swimmers in the CR group reported significantly less pain. Actually, the CR group had only minor pain after the two-week therapy program, which disappeared by the end of the experiment. The PL group recovered slower and the difference was significant in all measurements between the two groups. Previous studies suggest that Cr supplementation reduces the increase of inflammatory cytokines concentration (Bassit et al., 2008). If we consider that the pain was due to the inflammation, then we can assume that the decrease of pain can be attributed partly to Cr supplementation. There was a significant decrease in pain intensity in the CR group that may be related to its effect of Cr on inflammatory markers.

The most widely studied marker of muscle damage induced by physical exercise is CK (Brancaccio et al., 2007). It is a fact that athletes use amino acid supplements that can reduce the CK levels and muscle pain after strenuous exercise (Greer et al., 2007). In our study, having examined the level of CK, we detected a significantly lower relative serum-level increase in the CR group compared to the PL group, but this difference was not significant over the next two days. Our results complete Santos et al's (2004) results. Baseline CK level was higher in both groups, compared to the reference values of healthy athletes (Hartmann and Mester, 2000). Increased CK is most likely due to greater sarcolemma and sarcoplasmic reticulum membrane instability as a result of mechanical stress from the eccentric exercise (Rawson et al., 2001). However, there is no direct evidence that the elevated CK and inflammation of the tendons are related.

It is noted that the application of the CK enzyme as a damage marker in sports medicine received several criticisms because of the large variability within and among individuals, and there is a diversity in gender, and sports activities (Hortobagyi and Denahan, 1989; Kuipers, 1994).

Muscular overuse is associated with structural damage of the contractile elements and reflected in DOMS. Mechanical overstress is supposed to be the major contributing factor for inducing muscle damage. The initial damage is followed by an inflammatory response and eventually by regeneration. Calcium is assumed to play an important role in triggering the inflammatory changes (Kuipers, 1994). With exercise-induced muscle damage, there is injury to the cell membrane which triggers the inflammatory response, leading to the synthesis of prostaglandins and leukotrienes (Connolly et al., 2003). Additionally, alterations in sarcolemma and sarcoplasmic reticulum membranes are evident. This damage may result in increased intracellular calcium levels which may be associated with muscle degradation (Rawson et al., 2001).

Thus, ingestion of exogenous Cr may provide protective effects via increased phosphocreatine synthesis which, in turn may aid in stabilizing the sarcolemma membranes and thereby reducing the extent of damage. The presumed anti-inflammatory effect of Cr behind the mechanisms is not known. Further research is needed to clarify the possible systematic effects on muscle Cr.

Conclusion

Altogether, the observations presented in our paper indicate that Cr supplementation combined with therapeutic strategy effectively supports the rehabilitation of tendon overuse damage of adolescent fin swimmers. Our results suggest that the Cr supplementation, combined with specific therapy, is a good way to accelerate the recovery of

the injured tendons and ligaments. Furthermore, it can be assumed that oral supplementation of Cr applied in the most severe training periods, may prevent overuse injury, i.e. tendinopathy.

Acknowledgements

We appreciate the athletes' patience, perseverance, and the possible unpleasant test procedures that made it possible to conduct the study. Thanks to Eva Kalman and Zoltan Guba for providing language help. Thanks to Tracy Lloyd and Paul Illand for language proofreading. IJ designed the study, oversaw data collection, data analysis and manuscript preparation. JPK assisted with study design, data analysis and manuscript preparation. PH assisted with designing the study, manuscript preparation and statistical analysis. GSZ assisted with manuscript design and helped to draft the manuscript. BK assisted with statistical analysis. JT assisted with study design, data analysis and manuscript preparation. All authors have read and approved the final version of the manuscript, and agreed with the order of presentation of the authors. None of the authors declare competing financial interests. This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors. The research was approved by the Ethics Committee of the Eszterhazy Karoly University. The studies were conducted in accordance with the declaration of Helsinki. Each participant and parent gave their written, informed consent after explanation of the study purpose, experimental procedures, possible risks and benefits. All the volunteers signed a free and informed consent term before participation.

References

Bartels, E.M., Sørensen, E.R. and Harrison, A.P. (2015) Multi-frequency bioimpedance in human muscle assessment. *Physiological Reports* **3,** e12354.

Bassit, R.A., Curi, R. and Costa Rosa, L.F. (2008) Creatine supplementation reduces plasma levels of pro-inflammatory cytokines and PGE2 after a half-ironman competition. *Amino Acids* **35,** 425-31.

Best, T.M. (1995) Muscle-tendon injuries in young athletes. *Clinics in Sports Medicine* **14,** 669-686.

Brancaccio, P., Maffulli, N. and Limongelli, F.M. (2007) Creatine kinase monitoring in sport medicine. *British Medical Bulletin* 81-82, 209-30.

Brault, J.J. and Terjung, R.L. (2003) Creatine uptake and creatine transporter expression among rat skeletal muscle fiber types. *American Journal of Physiology. Cell Physiology* **284,** C1481-1489.

Clarkson, P.M., Byrnes, W.C., McCormick, M., Turcotte, L.P. and White, J.S. (1986) Muscle soreness and serum creatine kinase activity following isometric, eccentric, and concentric exercise. *International Journal of Sports Medicine* **7,** 152-155.

Connolly, A.J., Sayers, S.P. and McHugh, M.P. (2003) Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research* **17,** 197-208.

Cooke, M.B., Rybalka, E., Williams, A.D., Cribb, P.J. and Hayes, A. (2009) Creatine supplementation enhances muscle force recovery after eccentrically-induced muscle damage in healthy individuals. *Journal of the International Society of Sports Nutrition* **6,** 13.

Evans, M.W., Ndetan, H., Perko, M., Williams, R., and Walker, C. (2012) Dietary supplement use by children and adolescents in the United States to enhance sport performance: results of the national health interview survey. *The Journal of Primary Prevention* **33,** 3-12.

Greer, B.K., Woodard, J.L., White, J.P., Arguello, E.M. and Haymes, E.M. (2007) Branched-chain amino acid supplementation and indicators of muscle damage after endurance exercise. *International Journal of Sport Nutrition and Exercise Metabolism* **17,** 595-607.

Hartmann, U. and Mester, J. (2000) Training and overtraining markers in selected sport events. *Medicine and Science in Sports and Exercise* **32,** 209-215.

Haussinger, D., Lang, F. and Gerok, W. (1994) Regulation of cell function by the cellular hydration state. *American Journal of Physiology* **267,** 343-355.

Haussinger, D. (1996) The role of cell hydration in the regulation of cell function. *Biochemical Journal* **313,** 697-710.

Hespel, P. and Derave, W. (2007) Ergogenic effects of creatine in sports and rehabilitation. *Subcellular Biochemistry* **46,** 245-259.

Hespel, P., Eijnde, B.O., Leemputte, M.V., Ursø, B., Greenhaff, P.L., Labarque, V., Dymarkowski, S., Hecke, P.V. and Richter, E.A. (2001) Oral creatine supplementation facilitates the rehabilitation of disuse atrophy and alters the expression of muscle myogenic factors in humans. *The Journal of Physiology* **536,** 625-633.

Hortobágyi, T. and Denahan, T. (1989) Variability in creatine kinase: Methodological, exercise, and clinically related factors. *International Journal of Sports Medicine* **10,** 69-80.

Hultman, E., Soderlund, K., Timmons, J.A., Cederblad, G. and Greenhaff, P.L. (1996) Muscle creatine loading in men. *Journal of Applied Physiology* **81,** 231-37.

Järvinen, T.A., Järvinen, T.L., Kääriäinen, M., Aärimaa, V., Vaittinen, S., Kalimo, H. and Järvinen, M. (2007) Muscle injuries: optimising recovery. *Best Practice and Research Clinical Rheumatology* **21,** 317-331.

Johnston, A.P., Burke, D.G., MacNeil, L.G. and Candow, D.G. (2009) Effect of creatine supplementation during cast-induced immobilization on the preservation of muscle mass, strength, and endurance. *Journal of Strength and Conditioning Research* **23,** 116-120.

Juhász, I., Györe, I., Csende, Z., Rácz, L. and Tihanyi, J. (2009) Creatine supplementation improves the anaerobic performance of elite junior fin swimmers. *Acta Physiologica Hungarica* **96,** 325-336.

Kannus, P., Paavola, M., Paakkala, T., Parkkari, J., Järvinen, T., and Järvinen, M. (2002) Pathophysiology of overuse tendon injury. *Der Radiologe* 42, 766-70.

Kley, R.A., Tarnopolsky, M.A. and Vorgerd, M. (2013) Creatine for treating muscle disorders. *The Cochrane Database of Systematic Reviews* **6,** CD004760.

Kreider, R.B., Ferreira, M., Wilson, M., Grindstaff, P., Plisk, S., Reinardy, J., Cantler, E. and Almada, A.L. (1998) Effects of creatine supplementation on body composition, strength, and sprint performance. *Medicine and Science in Sports and Exercise* **30,** 73-82.

Kreider, R.B., Kalman, D.S., Antonio, J., Ziegenfuss, T.N., Wildman, R., Collins, R., Candow, D.G., Kleiner, S.M., Almada, A.L. and Lopez, H.L. (2017) International Society of Sports Nutrition position stand: safety and efficacy of creatine supplementation in exercise, sport, and medicine. *Journal of the International Society of Sports Nutrition* **14,** 18.

Kuipers, H. (1994) Exercise-induced muscle damage. *International Journal of Sports Medicine* **15,** 132-135.

Langley, LL., Telford, I.R. and Christensen, J.B. (1974) *Dynamic anatomy physiology*. New York: McGraw-Hill Book Campany.

Lim, J.S., Hwang, J.S., Lee, J.A., Kim, D.H., Park, K.D., Jeong, .JS. and Cheon, G.J. (2009) Cross-calibration of multi-frequency bioelectrical impedance analysis with eight-point tactile electrodes and dual-energy X-ray absorptiometry for assessment of body composition in healthy children aged 6-18 years. *Pediatrics International* **51,** 263-268.

McCaffery, M. and Beebe, A. (1993) *Pain: Clinical Manual for nursing practice*. Baltimore, MD: V.V. Mosby Company.

Meszaros, J., Farmosi, I., Frenkl, R. and Mohacsi, J. (1990) *The biological basis of the children's sports*. Sport, Bp. 53-57.

Metzl, J.D., Small, E., Levine, S.R. and Gershel, J.C. (2001) Creatine use among young athletes. *Pediatrics* **108,** 421-425.

Mueller-Wohlfahrt, H.W., Haensel, L., Mithoefer, K., Ekstrand, J., English, B., McNally, S., Orchard, J., van Dijk, C.N., Kerkhoffs, GM., Schamasch, P., Blottner, D., Swaerd, L., Goedhart, E. and Ueblacker, P. (2013) Terminology and classication of muscle injuries in sport: The Munich consensus statement. *British Journal of Sports Medicine* **47,** 342-350.

Op 't Eijnde, B., Urso, B., Richter, E.A., Greenhaff, P.L. and Hespel, P. (2001) Effect of oral creatine supplementation on human muscle GLUT4 protein content after immobilization. *Diabetes* **50,** 18-23.

Pearlman, J.P. and Fielding, R.A. (2006) Creatine monohydrate as a therapeutic aid in muscular dystrophy. *Nutrition Reviews* **64,** 80-88.

Rawson, ES., Gunn, B., and Clarkson, PM. (2001) The effects of creatine supplementation on exercise-induced muscle damage. *Journal of Strength and Conditioning Research* **15,** 178-184.

Santos, R.V., Bassit, R.A., Caperuto, E.C. and Costa Rosa, L.F. (2004) The effect of creatine supplementation upon inflammatory and muscle soreness markers after a 30km race. *Life Sciences* **75,** 1917-1924.

Sereni, G., Reggiani, E. and Odaglia, G. (1981) Physiopathology of fin swimming. II. Clinical data. *Minerva Medica* **72,** 1405-1408.
Sharma, P. and Maffulli, N. (2005) Tendon Injury and Tendinopathy: healing and repair. *The Journal of bone and joint surgery. American volume* **87,** 187-202.
Tarnopolsky, M. and Martin, J. (1999) Creatine monohydrate increases strength in patients with neuromuscular disease. *Journal of Neurology* **52,** 854-857.
Terjung, R.L., Clarkson, P., Eichner, E.R., Greenhaff, P.L., Hespel, P.J., Israel, R.G., Kraemer, W.J., Meyer, R.A., Spriet, L.L., Tarnopolsky, M.A., Wagenmakers, A.J. and Williams, M.H. (2000) American College of Sports Medicine roundtable. The physiological and health effects of oral creatine supplementation. *Medicine and Science in Sports and Exercise* **32,** 706-717.
Unnithan, V.B., Veehof, S.H., Vella, C.A. and Kern, M. (2001) Is there a physiologic basis for creatine use in children and adolescents? *Journal of Strength and Conditioning Research* **15,** 524-528.
Valovich McLeod, T.C., Decoster, L.C., Loud, K.J., Micheli, L.J., Parker, J.T., Sandrey, M.A. and White, C. (2011) National Athletic Trainers' Association position statement: prevention of pediatric overuse injuries. *Journal of Athletic Training* **46,** 206-220.
Verni, E., Prosperi, L., Lucaccini, C., Fedele, L., Beluzzi, R. and Lubich, T. (1999) Lumbar pain and fin swimming. *The Journal of Sports Medicine and Physical Fitness* **39,** 61-65.
Vincent, W.J. and Weir, JP. (2012) *Statistics in Kinesiology*. Human Kinetics publishing. 213-228.
Volek, J.S., Kraemer, W.J., Bush, J.A., Boetes, M., Incledon, T., Clark, K.L. and Lynch, J.M. (1997) Creatine supplementation enhances muscular performance during high intensity resistance exercise. *Journal of the American Dietetic Association* **97,** 765-770.
Walter, M.C., Lochmüller, H., Reilich, P., Klopstock, T., Huber, R., Hartard, M., Hennig, M., Pongratz, D. and Müller-Felber, W. (2000) Creatine monohydrate in muscular dystrophies: a double-blind, placebo-controlled clinical study. *Journal of Neurology* **54,** 1848-1850.
Weir, J.P. (2005) Quantifying test-retest reliability using the interclass correlation coefficient and the SEM. *Journal of Strength and Conditioning Research* **19,** 231-240.
Wilson, J.J. and Best, T.M. (2005) Common overuse tendon problems: A review and recommendations for treatment. *American Family Physician* **72,** 811-818.
Zamparo, P., Pendergast, D.R., Termin, B. and Minetti, A.E. (2002) How fins affect the economy and efficiency of human swimming. *Journal of Experimental Biology* **205,** 2665-2676.

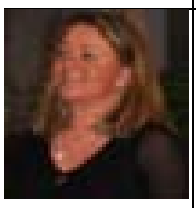
Key points

- The strategies are of great importance for athletes which can reverse or prevent significant functional deterioration caused by muscular dystrophy.
- Relatively little data is available regarding young athletes' creatine supplementation.
- We first investigated the pain intensity change of overuse tendon damage to alternative treatment strategy in adolescent fin swimmer cases.
- The limitation of this study is the small sample size. However, our results give some preliminary basis for further research.

✉ **Imre Juhasz**
University of Physical Education, School of Doctoral Studies, Hungary

AUTHOR BIOGRAPHY

Imre JUHASZ
Employment
University of Physical Education, School of Doctoral Studies
Degree
PhD candidate
Research interests
The effect of creatine supplement on physical performance and rehabilitation; Sport nutrition for young swimmers
E-mail: juhasz.imre@uni-miskolc.hu



Judit Plachy KOPKANE
Employment
Associate Professor, University of Miskolc, Faculty of Health Care
Degree
PhD
Research interests
Physical rehabilitation of sports injuries; Physical activity of elderly people
E-mail: efkplachy@uni-miskolc.hu

Pal HAJDU
Employment
Ass. Prof., Eszterhazy Karoly Univ. of Applied Sciences, Inst. of Sport Sciences
Degree
MSc
Research interests
Physical activity for young athletes
E-mail: hajdu.pal@uni-eszterhazy.hu

Gabor SZALAY
Employment
Ass. Prof., Eszterhazy Karoly Univ. of Applied Sciences, Inst. of Sport Sciences
Degree
MSc
Research interests
Physical training for young athletes
E-mail:
szalay.gabor@uni-eszterhazy.hu

Bence KOPPER
Employment
Assoc. Prof., University of Physical Education, Department of Biomechanics
Degree
PhD
Research interests
Biomechanics of the musculoskeletal system, movement analysis, mathematical modelling and optimization of sports movements, biomechanical aspects of sports injuries
E-mail: kopper.tf@gmail.com

Jozsef TIHANYI
Employment
Professor, University of Physical Education, Department of Biomechanics
Degree
PhD, DSc
Research interests
Functional biomechanics. The effect of creatine on aerobic and anaerobic performance; Eccentric exercise induced muscle damage, muscle fiber adaptation
E-mail: tihanyi.jozsef@tf.hu