The impact of soccer match play on the muscle damage response in youth female soccer athletes.

Original Investigation

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Abstract

Post-match assessment of creatine kinase (CK) activity and delayed onset muscle soreness (DOMS) are common markers of exercise-induced muscle damage and recovery status in soccer players. These responses have not been examined in youth female players. This study examined the effect of competitive match play on CK activity and DOMS in elite youth players. Thirtyfour elite female players, divided into three chronological age groups (U13, n = 11; U15, n = 10; U17 n = 12). Players completed baseline testing for CK and DOMS that was repeated immediately (for DOMS), 80, 128 and 168h post competitive match play for CK. Significant time effects were reported for CK ($P = 0.006$) and DOMS ($P < 0.01$). Significant differences between baseline and 168h post-match were reported for CK ($P < 0.01$), with significant group differences between U13 and U17's for CK ($P < 0.01$). All parameters returned to baseline in U17's at 168h but increased CK was evident for U13 and U15's at 168h. In conclusion seven days may be insufficient for biochemical recovery in youth female athletes. Therefore, monitoring strategies to assess muscle damage between training and match play should be considered to track recovery and potentially reduce muscular injury risk.

Key Words: Creatine kinase, maturation, recovery, football

Introduction

Exercise-induced muscle damage (EIMD) occurs following exercise where there are a high proportion of eccentric muscle actions, such as repeated sprints and change of direction associated with soccer match play [14]. The signs and symptoms of EIMD are well characterized and associated with disruption to normal sarcomere arrangement of a muscle fibre [22] cytoskeletal and membrane disruption [11, 17], and loss of force production [2]. However, there is a paucity of literature in youth athletes examining EIMD and in particular changes in creatine kinase (CK).

Soccer match play has been shown to cause micro trauma to muscle, which may be exacerbated with multiple matches throughout a season [15]. A number of studies have examined the acute effects of match play and concurrent fatigue in elite adult soccer players and referees [1, 28]. It has been reported that there is a significant increase in CK up until 48hr after competitive match play in elite adult male players, but that CK returned to pre-match levels at 72h [15]. Significant increases in CK in male adult players have also been reported, which peaked at 48h and remained significantly higher than baseline levels up until 94h post-match play only returning to baseline levels at 120h post-match play [15]. There are very few studies that have explore the effects of match play on muscle damage determined from CK in female soccer players [1].

It has been reported that there is a 152% increase in CK immediately following match play, indicating muscle cell damage during a match in elite female adult soccer players [1]. CK levels approached pre-match levels at the commencement of the next match (69h later). The change in CK in active muscles after match play reflects structural alterations within the muscles. Whether this pattern is evident in youth female soccer players remains to be identified. To our knowledge

there are no studies that have examined the time course recovery of CK following match play in elite female youth players.

The hormone oestrogen is known to have a stabilising effect on skeletal muscle membranes [16]. It is shown that it does this via interactions with the phospholipid double layer at the cell membrane, thereby increasing its structural integrity [9, 31], therefore this combination of factors may explain the role of oestrogen in attenuating muscle damage and CK leakage following exercise [4]. With increases in oestrogen at the onset of puberty, it could be expected that the older age groups may demonstrate a lower CK response compared to the younger age groups. This might reflect differing protective effects of oestrogen on CK activity in female youth players. Therefore, whether young girls follow a similar trend and appear as low CK responders, across a range of chronological ages and maturational stages, remains to be determined. To our knowledge no studies have explored the effects of competitive match play on muscle damage (using CK as a marker of damage) in female youth players, throughout the recovery period until the next competitive match.

Method

Elite youth female soccer players aged 11-17 years old were recruited from a centre of excellence participating in the English FA Women's Super League. Participants were split into three age groups U13 (n = 11), U15 (n = 10) and U17 (n = 12) years (Table 1). The project received ethical approval by the University's Research Ethics committee and it meets the ethical standards of the International Journal of Sports Medicine [13], and participants completed both a participant consent and physical activity readiness questionnaire (PARQ) which were obtained prior to testing.

Table 1: Group mean (±SD) participant characteristics by age group

	U13	U15	U17
Age (years)	12.19 ± 0.63	13.82 ± 0.62	15.78 ± 0.35
Standing Height (cm)	152.40 ± 8.66	161.44 ± 3.61	166.76 ± 3.44
Sitting Height (cm)	114.22 ± 4.61	120.33 ± 2.08	123.80 ± 3.50
Body Mass (kg)	40.66 ± 7.82	51.80 ± 7.43	58.57 ± 4.72
Maturity offset (PHV)	-0.31 ± 0.76	1.22 ± 0.49	2.61 ± 0.36

Study Design & Experimental Procedures

Participants attended four separate sessions distributed over a seven-day period and during a typical week that consisted of a competitive soccer match and routinely scheduled training sessions at their club (Figure 1). All matches were played on outdoor natural grass pitches in accordance with English Football Association rules. Matches were all played on the same day (Temp 7°C; relative humidity 72%) in a structured league setting so that opposition were deemed to be of the same standard as the team being measured. No structured recovery or training interventions were put in place by the researchers as the objective was not to try to control training/weekly load. However, physical activity was recorded over the week so that this could be factored into any biological response to a typical training week. The same testing battery was used in all four sessions to try and establish a potential recovery profile of biochemical measures post-match play. Baseline measures were obtained one hour prior to the first soccer match after 80 h of rest. In order to establish the player's recovery, the players were tested 1 hour prior to the second match (168 h post).

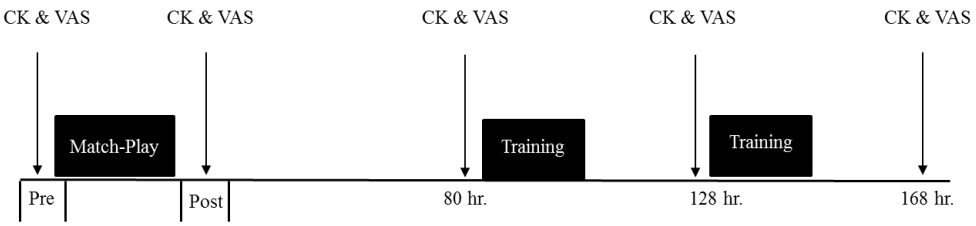

Figure 1: Schematic representation of research design

Anthropometric Measures

Standing and sitting height was measured to the nearest mm using a stadiometer (Holtain Limited, Crymych). Leg length was calculated from standing height minus sitting height. Participant's exact age was calculated using their date of birth and the date of baseline testing. Maturation as determined by age from peak height velocity (APHV) was done using known equations [20].

Physical Activity

Self-reported 3-day physical activity questionnaires (3dPAR) was used to monitor participant's exposure to physical activity which has been found to be a reliable measure of physical activity in adolescents [26]. In a simplified version of the 3dPAR participants were asked to report the type, duration and intensity of any physical activities that they participated in within the past three days. Questionnaires were handed out for the week prior to data collection. During the data collection week questionnaires were provided on each testing session to report their exposure in the three days prior (pre-match, 80, 128 and 168h post). Although participants were instructed to avoid strenuous physical activity in the 48h prior to data collection, they were encouraged to continue their normal routine of physical activity outside of the Centre of Excellence training and match fixtures. Players were not expected to report any training sessions delivered by the centre of

excellence, instead coaches were asked to fill out a training log for each football session carried out within the seven-day testing period.

Creatine Kinase Sampling

Approximately 30µL of capillary blood was collected from a finger via a prick made with a spring-loaded disposable lancet (Accu-Check, Roche Diagnostics, Germany). The blood sample was immediately analysed using a colorimetric assay procedure using a Reflotron® systems spectrophotometer (F. Hoffman-La Roche Ltd, Basel, Switzerland) for plasma CK activity. The coefficient of variation for inter and intra-assay variation were 3.5 and 3.7% respectively.

Delayed Onset of Muscle Soreness (DOMS)

Using a visual analogue scale (VAS) participants provided a rating of perceived muscle soreness on a subjective scale. The scale was 10cm in length, with 0 (no soreness) and 10 (very, very sore) representing the extreme ends of the scale [5]. Participants were instructed to mark a cross along the line that relates to the amount of muscle soreness that they felt at that current time. To avoid potential bias from previous measurements a blank VAS scale was provided at each testing session.

Match Intensity Measures

Within half an hour post-match, participants were asked to rate their perceived level of exertion during the match. As each team had differing match durations in accordance with English Football Association rules (U13 = 2 x 35min halves, U15 = 2 x 40min halves and U17 = 2 x 45min), the Borg scale was used as it has been previously reported to be a reliable measure of rate of perceived

exertion (RPE) regardless of age [10], or gender and easily learnt by older children and adolescents [33]. The simplified fixed ten point Borg CR-10 Scale [23] was used to allow an easy method of measure the level of intensity on a scale ranging from zero to ten, zero representing low intensity/rest up to ten representing maximal effort. *Statistical Analysis*

Statistical analysis was performed using SPSS (v19.0 for Windows; SPSS Inc, Chicago). Descriptive statistics were calculated for each variable including means and standard deviation (±SD). A 4 (Time) × 3 (Group) Repeated Measures Analysis of Variance (RMANOVA) was performed to determine interaction and main effects for outcome variables: CK and VAS. A oneway analysis of variance (ANOVA) was performed to determine significant group effects in total weekly activity exposure and match intensity. Bonferroni-corrected post hoc t-tests were performed if significant interaction and main effects were determined, to establish where the statistical differences occurred. Significance level was set at $P < 0.05$. The classification of effect sizes for pre-match to 80h was conducted on CK measures and was determined by Cohen's *d*.

The effect size was classified as small ($0.00 \leq d \leq 0.49$), medium ($0.50 \leq d \leq 0.79$), and large ($d \geq 0.80$) [6].

Results

Creatine Kinase

Mean (±SD) CK by age and time are shown in Figure 2. A significant main effect of time was evidenced ($F_{(1.89,\ 45.34)} = 5.89$, $P = 0.006$). Post hoc analysis revealed that CK was significantly higher at 80h and 120h compared with pre-match levels, and between 80h and 168h post-match for all age groups. All groups showed increase in the 80h post testing values with a small effect

sizes demonstrated for the U15 (ES = 0.45) and a large effect size shown for both the U13 (ES = 0.72) and U17 (ES = 1.59) groups. A statistically significant group difference was reported between the U13 and U17 groups at the 168h measure ($P < 0.01$). The absolute % change in CK from baseline to 168hr post-match play was +287% for the U13, +156% for the U15 and -23% for the U17.

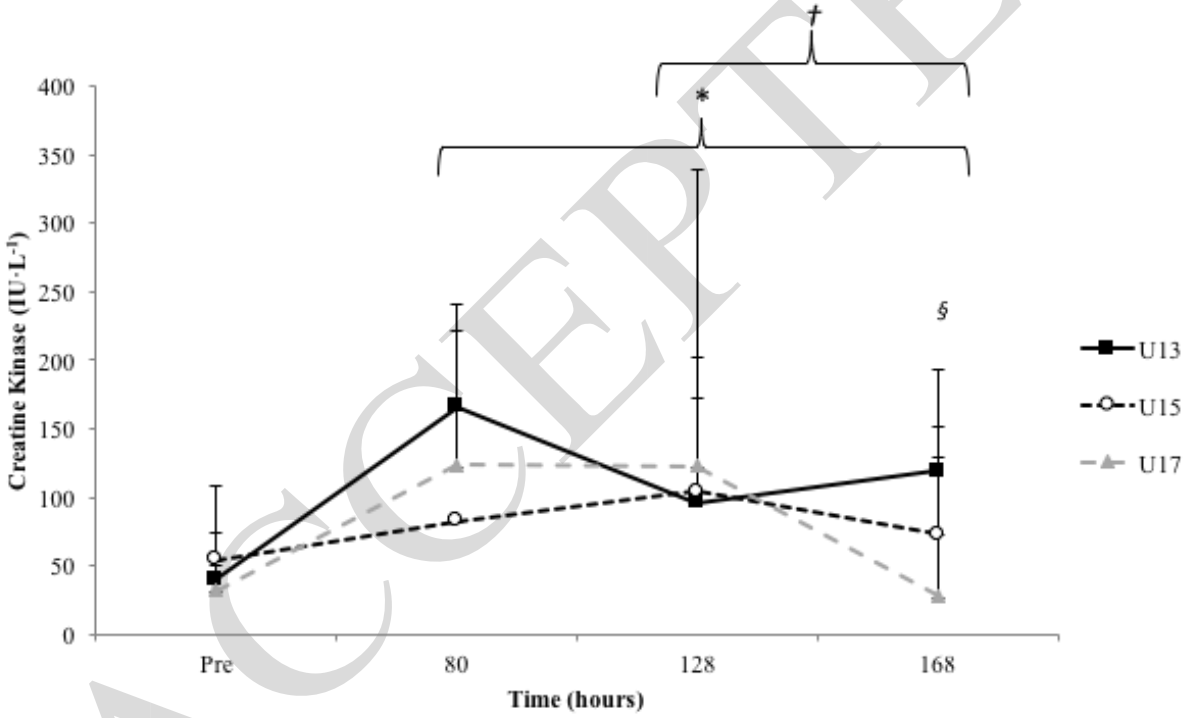

Figure 2: Mean (±SD) values for CK (I·Ul-1) pre- and post-match play per age group

* Significant differences compared to baseline
† Significant difference compared to 80h
§ Significant difference compared to U13

Perceived muscle soreness (VAS)

Mean (±SD) VAS is displayed in Table 2 according to age and time. A significant main effect for time was reported ($F_{(2.15,\ 62.31)}$ = 37.02, P < 0.01). Post hoc analysis revealed that VAS was significantly higher at 80h and 128h compared with pre-match levels, and between 80h and 168h post-match for all groups. No significant group interaction (P = 0.07) for perceived muscle soreness was observed.

Table 2: Mean (±SD) VAS (cm) pre- and post-match play per age group

Group	Baseline	Post-Match	80 h	128 h	168 h
U13	0.03 ± 0.95	3.88 ±2.69*	0.79 ± 1.22*	0.20 ± 0.39*	1.10 ± 1.58*†
U15	0.17 ± 0.47	3.68 ±2.45*	0.79 ± 0.89*	1.24 ± 1.19*	1.18 ± 1.18*†
U17	0.85 ± 0.97	5.33 ± 2.52*	1.64 ± 2.23*	2.43 ± 2.24*	1.13 ± 1.10*†

* *Significant differences compared to baseline*

† *Significant difference compared to 80h*

Weekly physical activity and match intensity

Mean (SD) weekly exposure to physical activity during data collection week (Table 3) and match intensity ratings (Table 4) are presented. No significant group differences were reported in total exposure to physical activity across the training week. A significant between group effect for hard intensity exercise ($F_{(2,\ 30)}$ = 4.32, P = 0.02) was reported, but not for moderate or very hard intensity. Post hoc analysis revealed the U13 to significantly perform more hard intensity exercise compared to the U17s (P = 0.02). A between group significant effect was reported for match

intensity ($F_{(2,28)} = 6.24$, $P < 0.01$). Post hoc analysis determined statistical differences between the U15 and U17 groups only ($P < 0.01$). VAS (cm) pre- and post-match play per age group

Table 3: Mean (±SD) values for VAS (cm) pre- and post-match play per age group

Group	Baseline	Post-Match	80 h	128 h	168 h
U13	0.03 ± 0.95	3.88 ±2.69*	0.79 ± 1.22*	0.20 ± 0.39*	1.10 ± 1.58*†
U15	0.17 ± 0.47	3.68 ±2.45*	0.79 ± 0.89*	1.24 ± 1.19*	1.18 ± 1.18*†
U17	0.85 ± 0.97	5.33 ± 2.52*	1.64 ± 2.23*	2.43 ± 2.24*	1.13 ± 1.10*†

* *Significant differences compared to baseline*

† *Significant difference compared to 80h*

Table 4: Mean (±SD) values for RPE (AU) post-match play per age group

Group	Match play
U13	8.18 ± 0.68
U15	8.63 ± 0.44*
U17	7.25 ± 1.22

* *Significantly different to U17*

The main findings of the current study indicate that competitive soccer match play significantly compromises skeletal muscle integrity and elevates perceptions of soreness in elite youth female soccer players. We also observed that recovery rates differed between the chronological age groups with the U17's recovering to baseline levels at 168 h. The data from this study suggest that recovery from competitive match play in youth female players is age dependent and may need to be adequately monitored and managed to reduce any subsequent injury risk.

Although the focus of this study was not on the effects of acute fatigue, a significant increase in self-reported pain was evidenced post competitive match play demonstrating potentially, the

immediate muscle damage response in elite female youth athletes. Elevated CK values were reported up to 128 h (U17) and 168 h post competitive match play (U13 and U15) highlighting delayed recovery of CK. In comparison to the limited research in paediatric populations, the acute and residual muscle damage response demonstrated in the current study is greater than previously determined [8, 30, 32]. This is in contrast to previous reports highlighting youth populations as low CK responders compared to adult populations [19] due to a potentially greater resistance to fatigue during exercise [7].

It has been previously indicated that muscle damage can influence stretch shortening cycle (SSC) function [2], and a muscles ability to produce force [27]; therefore, residual muscle damage may influence an athlete's ability to re-perform during subsequent training sessions and match fixtures within a 168-hour period. However, research investigating the muscle damage response to residual fatigue up to 168 h post competitive match play is very limited, particularly in paediatric populations. Therefore, this is the first study to investigate the muscle damage response to residual fatigue post-competitive match play to next competitive match play in elite female youth soccer.

The biochemical muscle damage response reported in the current study could be attributed to the nature of the exercise protocol involving competitive match play rather than a simulated fatiguing protocol. It has been indicated that exercise-induced muscle damage can be influenced by the type, intensity and duration [24], with greater muscle damage induced through movements involving eccentric muscle actions and physical contact. Therefore, the greater increase in CK activity compared to previous literature could be attributed to eccentric actions (e.g. during braking, side-stepping, jump landing) and increased contact involved in competitive match play [12, 21].

Muscle damage post-competitive match play in female adult soccer players has been evidenced via increased CK activity up to 18 h post-competitive match play [12]. The current study demonstrates a greater CK activity 80 h post-competitive match play. However, baseline values were greater [12] than the current study (150 vs 40 IU/L). Therefore, although greater peak CK values were reported [12], the actual CK increase was lower than that in the current study. It has been previously suggested that training status can impact an individual's response to exercise [3], with trained individuals demonstrating higher baseline values, and a lower exercise-induced increase in muscle damage compared to untrained individuals [8]. Regular exposure to physical activity may develop protective mechanisms to withstand structural damage to the muscle [8]. Therefore, it was suggested that the higher baseline values reported may be attributed to the increased exposure to exercise demonstrated in the trained group [12]. Furthermore, greater muscle mass has also been highlighted as a potential influential factor to greater baseline CK values [8]. Therefore, as this is one of the first studies to investigate the muscle damage response in elite female youth populations the lower baseline CK activity is can be attributed to youth female players having lower exposure to training, combined with smaller muscle mass make this study's data supportive of the research in adult elite females [1, 12] and trained pre-pubescent boys [8].

Residual effects of muscle damage were evident with a 295%, 251% and 113% greater CK values compared to baseline in the U13, U17 and U15 respectively. This extended CK activity is in association with previous research reporting evidence of muscle damage up to 69 h post exercise in adult female soccer players [1] and 96 h post exercise in untrained adult males [18]. Chronological age specific differences were apparent with residual fatigue. Initially, the U13 and

U17 demonstrated a similar CK response post-match at 80 and 128 h post-match play; however, the recovery of muscle damage markers was significantly different between the U13 and U17 at 168 h post-match play, with U17 showing CK activity below baseline values by 168 h postmatch, while elevated CK values were still evident in the U13 and U15 age groups. Therefore, in addition to subsequent training sessions 80 and 128 h post-match play, the younger participants may be at a greater risk for injury and underperformance during the second match fixture with muscle damage still evident. Therefore, it would appear that elite female youth soccer players are high CK responders demonstrating residual muscle damage up to 168 h post competitive match play.

Oestrogen can act as a protective mechanism for skeletal muscle [16] assisting in the maintenance of the cell membrane during exercise, preventing the leakage of CK [9, 31]. Therefore, with an influx of oestrogen demonstrated at the onset of puberty continually increasing during puberty until adulthood, it could be expected that the older age groups may demonstrate a lower magnitude of change in CK response compared to the younger age groups. However, the U17's demonstrated a large effect size 80 h post-match play, this was not isolated as the U13 group also showed a large effect of match play on CK activity. If oestrogen played a large contributory role in the muscle damage response to exercise, then age related differences would be evident immediately post exercise. However, no significant age differences were reported in the CK response 80 and 128 h post-match play. Therefore, alternative factors may be influencing these varied age-specific responses to residual fatigue. Whilst oestrogen can act as a protective mechanism for muscle damage during moderate intensity exercise, it may be unable to cope with higher intensity exercise [4]. The rating of perceived exertion self-reported in the current study on match play demonstrated

high values across the three age groups (Table 4). Therefore, it may be that the higher intensity movements associated with soccer match play will reduce the effect of oestrogen [4]. Although the influence of oestrogen cannot be disregarded, it is likely that additional factors contribute to the age-related difference reported 168 h post-match.

The potential influence of external factors including training intensity and exposure to physical activity needs to be acknowledged. It is worth noting that although this study utilised measures of training load and match intensity (3dPar and match RPE), they are wholly subjective measures. The use of these as sole monitoring tools may not fully elucidate the external metrics from match play (e.g. accelerations, decelerations and time spent at max velocity) that are specific to exacerbating the mechanical stress experienced by the players. The exposure data recorded highlighted that the U13′s reported a greater total weekly physical activity load compared to the older age groups, however, this difference was not significant. Further analysis revealed a significant difference in the amount of hard intensity exercise reported between the U13′s and U17′s. With exercise intensity associated with the amount of muscle damage induced [24], it could be expected that higher exposure to higher intensity exercise will induce greater muscle damage. Therefore, irrespective that the total duration of exposure of physical activity was similar, the actual type and intensity of exercise can influence an individual′s muscle damage response. With previous indications that an association exists between exposure to physical activity and injury risk [29], it could be argued that all age groups actual work load should be tracked to help individualise the work completed in training to help reduce any risk for injury during the subsequent training sessions and match fixtures within the seven-day period post-match play.

Irrespective of age, elite female adolescent athletes report muscle damage up to 128 h and 168 h post exercise. These findings reinforce that competitive match play and weekly training load are most likely responsible for the elevated CK by the next competitive match. It has been well reported that accumulated muscle damage is likely to contribute to increased risk of ACL injury, through the muscle damage negatively impacting an athletes' rate of force development and proprioceptive capability, thereby reducing joint stability and increasing risk of ACL rupture [25]. Therefore, the levels of accumulated and chronic muscle damage found in the current study presents a serious risk factor for injury and neuromuscular readiness to re-perform in these young female players. Coaching staff working with these populations should be encouraged to look at the structure and content of their training sessions following match play to better manage the training load and help to reduce the risk of injury.

Limitations of the present study include the small sample size within each age group studied which may have affected the statistical power of the analyses. Participants also experienced varying match and training demands prior to and during the data collection process. Issues such as these are a feature of performing research on athletes in-season and are, as such, unavoidable. However, this could have been mitigated by the use of GPS to track and collate distances and intensities of activity during match play and training, thereby allowing a more comprehensive analysis of work load to be conducted. Future research is warranted to examine the contribution of acceleration, deceleration and high velocity running actions to the acute and temporal recovery response to match play and typical training weeks in female youth players. Further research could also be undertaken to assess a recovery intervention and how it might transfer to variables such as acceleration and sprinting performance during training and subsequent match play.

The current study is the first to demonstrate that biochemical markers of fatigue are present in female youth soccer players following competitive match play, and that it persists throughout the training week. This potentially identifies them as an "at-risk" group and therefore an emphasis of intervention programs must be to develop recovery strategies to mitigate the risk of prolonged muscle damage. Importantly, these recovery strategies must include components that develop fatigue resistance. Well structured, developmentally appropriate strength and conditioning work should be viewed as a season-long commitment to offset the negative effects of accumulated metabolic fatigue. Early intervention is critical in developing correct recovery protocols to assist the reduction in metabolic fatigue in these athletes.

References

1. Andersson HM, Raastad T, Nilsson J, Paulsen G, Garthe I, Kadi F. Neuromuscular fatigue and recovery in elite female soccer: effects of active recovery. Med Sci Sports Exerc. 2008: 40: 372-380.
2. Byrne C, Twist C, Eston R. Neuromuscular function after exercise-induced muscle damage. Sports Med. 2004: 34: 49-69.
3. Chevion S, Moran DS, Heled Y, Shani Y, Regev G, Abbou B, Berenshtein E, Stadtman ER, Epstein Y. Plasma antioxidant status and cell injury after severe physical exercise. Proc Natl Acad Sci. 2003: 100: 5119-5123.
4. Clarkson PM, Sayers SP. Etiology of exercise-induced muscle damage. Can J Appl Physiol. 1999: 24: 234-248.
5. Cleak MJ, Eston RG. Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. Br J Sports Med. 1992: 26: 267-272.
6. Cohen J (ed). Statistical Power Analysis for the Behavioral Sciences. New York, NY: Routledge Academic, 1988.
7. De Ste Croix MBA. and Deighan MA. (2011). Development of joint stability during childhood. pp. 233 In M. De Ste Croix and T. Korff (Eds), Paediatric Biomechanical and Motor Control: Theory and Application. Routledge
8. Elamaran M. and Muhammed Musthafa M. The changes on creatine kinase in response to aerobic exercise among novice and trained soccer players of different ages. Int J Phys Ed Fit Sports. 2013 2: 29-33.
9. Enns DL and Tiidus PM. The influence of estrogen on skeletal muscle. Sports Med. 2010: 40: 41-58.
10. Eston RG and Williams JG. Exercise intensity and perceived exertion in adolescent boys. Br J Sports Med. 1986: 20: 27-30.
11. Feasson L, Stockholm D, Freyssenet D, Richard I, Duguez S, Beckmann JS, Denis C. Molecular adaptations of neuromuscular disease---associated proteins in response to eccentric exercise in human skeletal muscle. J Physiol (Lond). 2002: 543: 297-306.
12. Gravina L, Ruiz F, Lekue JA, Irazusta J, Gil SM. Metabolic impact of a soccer match on female players. J Sports Sci. 2011: 29: 1345-1352.
13. Harriss DJ, Atkinson G. Ethical standards in sports and exercise science research: 2016 update. Int J Sports Med 2015; 36: 1121-1124
14. Howatson G. and Milak A. Exercise-induced muscle damage following a bout of sport specific repeated sprints. J Str & Cond Res 2009: 23: 2419-2424.
15. Ispirlidis I, Fatouros IG, Jamurtas AZ, Nikolaidis MG, Michailidis I, Douroudos I, Margonis K, Chatzinikolaou A, Kalistratos E, Katrabasas I, Alexiou V. Time-course of changes in inflammatory and performance responses following a soccer game. Clin J Sport Med. 2008: 18: 423-431.

16. Kendall B, Eston R. Exercise-induced muscle damage and the potential protective role of estrogen. Sports Med. 2002: 32: 103-123.

17. Lovering RM and De Deyne PG. Contractile function, sarcolemma integrity, and the loss of dystrophin after skeletal muscle eccentric contraction-induced injury. Am J PhysiolCell Physiol 2004: 286: C230-C238.

18. Magal M, Dumke CL, Urbiztondo ZG, Cavill MJ, Triplett NT, Quindry JC, McBride JM, Epstein Y. Relationship between serum creatine kinase activity following exerciseinduced muscle damage and muscle fibre composition. J Sports Sci. 2010: 28: 257-266.

19. Marginson V, Rowlands AV, Gleeson NP, Eston RG. Comparison of the symptoms of exercise-induced muscle damage after an initial and repeated bout of plyometric exercise in men and boys. J Appl Physiol. 2005: 99: 1174-1181.

20. Mirwald RL, Baxter-Jones AD, Bailey DA, Beunen GP. An assessment of maturity from anthropometric measurements. Med Sci Sports Exerc. 2002: 34: 689-694.

21. Nédélec M, McCall A, Carling C, Legall F, Berthoin S, Dupont G. Recovery in Soccer. Sports Med. 2012: 42: 997-1015.

22. Newham DJ, Mills KR, Quigley BM, Edwards RH. Pain and fatigue after concentric and eccentric muscle contractions. Clin Sci. 1983: 64: 55-62.

23. Noble BJ, Borg GA, Jacobs I, Ceci R, Kaiser P. A category-ratio perceived exertion scale: relationship to blood and muscle lactates and heart rate. Med Sci Sports Exerc. 1983: 15: 523-528.

24. Nosaka K, Aldayel A, Jubeau M, Chen TC. Muscle damage induced by electrical stimulation. Eur J Appl Physiol. 2011: 111: 2427-2437.

25. Owings TM, Grabiner MD. Motor control of the vastus medialis oblique and vastus lateralis muscles is disrupted during eccentric contractions in subjects with patellofemoral pain. Am J Sports Med. 2002; 30: 483- 487.

26. Pavlidou S., Michalopoulou M., Aggelousis N. and Taxildari K. Validation of a 3 day physical activity record and the SW pedometer in Greek children. Biol Exerc 2011: 7: 2539

27. Raastad T, Risøy BA, Benestad HB, Fjeld JG, Hallén J. Temporal relation between leukocyte accumulation in muscles and halted recovery 10 -20 h after strength exercise. J Appl Physiol. 2003: 95: 2503-2509.

28. Silva, JR., Ascensao, A., Marques, F., Seabra, A., Rebelo, A., & Magalhães, J. Neuromuscular function, hormonal and redox status and muscle damage of professional soccer players after a high-level competitive match. Eur J Appl Physiol, 2013: 13: 21932201. doi: 10.1007/s00421-013-2633-8

29. Silvers HJ and Mandelbaum BR. Prevention of anterior cruciate ligament injury in the female athlete. Br J Sports Med. 2007: 41: 152-159.

30. Soares, JMC, Mota, P., Duarte, JA., Appel, HJ. Children are less susceptible to exerciseinduced muscle damage than adults: a preliminary investigation. Pediatr Exerc Sci. 1996: 8: 361–367.

31. Tiidus PM. Can estrogens diminish exercise induced muscle damage? Can J Appl Physiol. 1995: 20: 26 –38.

32. Webber LM, Byrnes WC, Rowland TW, Foster VL. Serum creatine kinase activity and delayed onset muscle soreness in prepubescent children: a preliminary study. Pediatr Exerc Sci. 1989: 1: 351– 359.

33. Williams JG, Eston RG, Stretch C. Use of the rating of perceived exertion to control exercise intensity in children. Pediatr Exerc Sci. 1991: 3: 21-27.