RELEASE OF MUSCLE $\alpha$-ACTIN INTO SERUM AFTER INTENSIVE EXERCISE

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ABSTRACT: Purpose: To study the effects of high-level matches on serum alpha actin and other muscle damage markers in teams of rugby and handball players. Methods: Blood samples were drawn from 23 sportsmen: 13 rugby players and 10 handball players. One sample was drawn with the player at rest before the match and one immediately after the match. Immunoassays were used to determine troponin I, troponin T, LDH, and myoglobin concentrations. Western blot and densitometry were used to measure $\alpha$-actin concentrations. Muscle injury was defined by a total CK value of > 500 IU/L (Rosalki method). Results: Mean pre- and post-match serum alpha-actin values were, respectively, 7.16 and 26.47 µg/ml in the handball group and 1.24 and 20.04 µg/ml in the rugby team. CPK, LDH and myoglobin but not troponin 1 levels also significantly differed between these time points. According to these results, large amounts of $\alpha$-actin are released into peripheral blood immediately after intense physical effort. Possible cross-interference between skeletal and cardiac muscle damage can be discriminated by the combined use of $\alpha$-actin and troponin I. Conclusion: The significant increase in alpha-actin after a high-level match may be a reliable marker for the early diagnosis and hence more effective treatment of muscle injury.

KEY WORDS: $\alpha$-actin, muscle injury, release, immunoblotting

INTRODUCTION

Intense physical exercise induces muscle-fibre injury, whose severity depends on the duration and characteristics of the exercise and the training stage of the athlete [5]. The type of muscle contractions produced by exercise also influences the severity and extent of injury. Thus, repeated eccentric muscle contractions (contraction of muscle while it lengthens) can cause persistent diffuse pain. Dynamic exercises that involve an eccentric component are a risk factor for muscle injury [14].

The clinical assessment of post-exercise muscle injury is a major challenge and its severity and progression are highly variable. It is characterized by remnant pain in muscle masses and sometimes by a feeling of persistent heaviness in lower limbs [8]. Because the clinical diagnosis of this condition is non-specific, its impact in sports has been poorly studied.

In males, physical exercise-induced muscle injuries are characterized by delayed pain, muscle tension, mobility limitation, poorer performance and the presence of muscle enzymes in blood [20]. The biological diagnosis of these lesions remains difficult due to the lack of specificity, sensitivity and reproducibility of available markers. Histologically, exercise-related muscle injuries first appear as lesions concentrated in myofibrils and cytoskeleton [12, 13].

Under electron or high-power microscopy, the lesions appear as sarcomere-devoid patches that resemble the disruption of fibre-supporting protein structures, e.g., desmin and laminin. Initially limited to sarcomeres, the lesions develop very quickly and produce an inflammatory reaction that gives rise to a tissue repair process. In more severe cases, damaged cells enter cell death and are replaced with newly-formed fibres from satellite cells [27].

The aetiology of muscle injury has not been well defined, and mechanical, metabolic and/or vascular origins have been proposed [20]. The mechanical hypothesis has prevailed to date, based on the negative effects of tension on cell structures. As mentioned above, muscle injury mainly occurs after exercise that has a major eccentric component. It has been demonstrated that the peak tension in this
type of contraction corresponds to a greater length than corresponds to actin-myosin cross-bridges, and lesions may be produced by the greater tension applied to each actin-myosin cross-bridge during physical exercise [20]. Another proposition is based on the heterogeneous sarcomere length during eccentric type contraction, with some sarcomeres stretched beyond the resistance of the elastic fibre structures, disrupting their sarcolemma. A change in the structure of sarcomeres is also observed and is often at the origin of lesions.

However, some physical activities such as unloading have also been associated with muscle injury [17]. Although these lesions are less severe, they inspired the hypothesis that other factors besides mechanical mechanisms might explain the development of lesions in exercise. There has also been interest in the relationship between apoptosis and exercise-related muscle injury, and a defective apoptotic process has been proposed as a possible explanatory factor [6]. In short, the mechanisms underlying exercise-induced muscle lesions have yet to be fully elucidated.

It has been previously reported α-actin to be a novel and reliable marker of skeletal muscle damage in sportspeople, with a high sensitivity of 63.3-100% [3,22,23].

With this background, the present study was designed to determine α-actin levels and muscle damage indexes in the serum of high-level handball and rugby players before and after a competitive match.

**MATERIALS AND METHODS**

**Subjects.** The study population comprised 13 members of the University of Granada male handball team with a mean age of 24.5 yrs (handball group) and 10 members of the City of Granada City male rugby team with a mean age of 27.7 yrs (rugby group).

**Design.** Six dependent variables were measured: serum troponin I (cTnI), troponin T (cTnT), Actin (ACT), creatine kinase (CK) and lactate dehydrogenase (LDH) levels. Two independent variables were studied: sport (handball or rugby) and measurement time (before or after match).

**Experimental procedures.** Muscle damage indexes were determined in serum before and after a high-competition handball or rugby match. All subjects signed their informed consent to participation in the study, which was approved by The Ethical Committee of The University of Granada.

**Blood sampling.** Before the match (with the player at rest) and again immediately after the match, qualified nurses drew 5-ml venous blood samples from each participant into tubes with separator gel (Venoject II, Terumo Europe, Leuven, Belgium).

**Determination of total CK activity.** Total CK activity was determined by using the enzymatic kinetic method, in which the CK catalyzes the transfer of a phosphate group from creatine phosphate to adenosine diphosphate (ADP). The subsequent formation of adenosine triphosphate (ATP) is measured by using two associated reactions catalyzed by hexokinase (HK) and glucose-6-phosphate dehydrogenase (G6PDH), which produce nicotinamide adenine dinucleotide (NAD). This CK assay contains the activator monothioglycerol.

**Immunoassay of troponin I (cTnI), troponin T (cTnT) and myoglobin.** Troponin I, troponin T and myoglobin were determined by chemiluminescence immunoassay. For cTnI and MYO, a sandwich-type immunoenzymatic assay (Beckman Instruments) was used. A sample was added to a glass reaction vessel with alkaline phosphatase-conjugated anti-troponin I and anti-myoglobin monoclonal antibodies along with paramagnetic particles coated with anti-troponin I and anti-myoglobin monoclonal antibodies. Cardiac troponin and human myoglobin bind to the antibody in the solid phase, whereas the antibody-alkaline-phosphatase conjugate reacts with different antigenic sites on cardiac troponin and myoglobin molecules. After incubation, materials not bound to the solid phase are removed by separation in a magnetic field and washing. A chemiluminescent substrate, Lumi-Phos 530, was added to the reaction vessel and a luminometer was used to measure the light generated by the reaction. The production of photons is inversely proportional to the amount of enzymatic conjugate present at the end of the reaction and, consequently, to the concentration of cardiac troponin I and myoglobin in the sample. The amount of analyte in the sample was determined by using a multipoint calibration curve.

For troponin T determination, the Elecsys 2010 troponin T test (Roche Diagnostic) was used, which has a total duration of 18 min at 37°C. In a first incubation step, a sandwich-type complex was formed by the sample, a specific biotinylated monoclonal antibody against troponin T, and a ruthenium chelate-labelled (chelate tris(2,29-bipyridul)ruthenium(II), (Ru(bpy)2+3) specific monoclonal antibody against troponin T.

**Detection of α-actin by Western-blot.** Sarcomeric α-actin was determined by Western blot. A sample of serum (5 μl) from each study subject was dissolved in Laemmli’s sample buffer (62.76 mm Tris-HCl pH 6.8, 1% 2-mercaptoethanol, 1% SDS, 10% glycerol, and 0.01% bromophenol blue) at 1:5 ratio, boiled for 5 min, microfuged for 1 min and analyzed by SDS-PAGE (sodium dodecylsulphate polyacrylamide gel electrophoresis) in Mini Protean II cell (Biorad) at 60 mA for 1 h at room temperature. Gels with serum samples were run in duplicate in all cases. Gels for immunoblot analyses were separated electrophoretically and transferred to a nitrocellulose membrane by applying a current of 20 V at room temperature for 30 min. The blots were treated with blocking solution (20mM Tris, 0.9 NaCl, 10% non-fat milk) for 3 h at room temperature and then reacted with a 1:2000 dilution of anti-sarcomeric α-actin monoclonal antibody (Alpha-Sr-1 Clones. Dako, Denmark). Primary antibodies were incubated overnight at 4°C. Membranes were washed (15 min in 5% TBST) and incubated with horseradish peroxidase-conjugated rabbit anti-mouse IgG (1:2000, Sigma-Aldrich, Inc) for 1 h at room temperature, followed by additional washes (15 min in 5% TBST). Proteins were visualized by enhanced chemiluminescence (ECL, Bonus, Amersham).

**Densitometric analysis of α-actin.** Densitometric analysis was carried out by scanning X-ray images of the membranes (Fluorine-S Multimager, Bio-Rad) at an image resolution of 100 pixels per inch.


TABLE 1. DETERMINATION OF LEVELS OF MYOGLOBIN (MYO), CREATINE PHOSPHOKINASE (CPK), LACTATE DEHYDROGENASE (LDH), TROPONIN T (cTnT), TROPONIN I (cTnI), AND α-ACTIN IN SERUM.

<table>
<thead>
<tr>
<th>Sport</th>
<th>Measurement time point</th>
<th>Pre-match Mean (SD)</th>
<th>Post-match Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>cTnI (ng/mL)</td>
<td>Handball</td>
<td>0.019 (0.0095)</td>
<td>0.019 (0.0166)</td>
</tr>
<tr>
<td></td>
<td>Rugby</td>
<td>0.016 (0.0084)</td>
<td>0.016 (0.0107)</td>
</tr>
<tr>
<td>MYO (ng/mL)</td>
<td>Handball</td>
<td>34.92 (13.00)</td>
<td>158.35 (69.06)</td>
</tr>
<tr>
<td></td>
<td>Rugby</td>
<td>34.23 (6.829)</td>
<td>323.69 (130.94)</td>
</tr>
<tr>
<td>cTnT (ng/mL)</td>
<td>Handball</td>
<td>0.0032 (0.008)</td>
<td>0.0369 (0.0103)</td>
</tr>
<tr>
<td></td>
<td>Rugby</td>
<td>0.0032 (0.008)</td>
<td>0.0067 (0.0038)</td>
</tr>
<tr>
<td>α-ACT (µg/ml)</td>
<td>Handball</td>
<td>7.1692 (5.8406)</td>
<td>26.47 (9.7298)</td>
</tr>
<tr>
<td>CPK (IU/L)</td>
<td>Handball</td>
<td>308.61 (149.31)</td>
<td>409.692 (191.34)</td>
</tr>
<tr>
<td></td>
<td>Rugby</td>
<td>235.40 (73.33)</td>
<td>391.10 (111.23)</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>Handball</td>
<td>326.85 (31.70)</td>
<td>394.38 (38.18)</td>
</tr>
<tr>
<td></td>
<td>Rugby</td>
<td>300.50 (32.81)</td>
<td>501.30 (72.50)</td>
</tr>
</tbody>
</table>

Note: Means and standard deviations of the six dependent variables as a function of the sport and measurement time. Handball and Rugby.

A densitometry image analysis software package (Quantity 1, Bio-Rad) was used. The procedure was repeated for each study sample. The digital image obtained was formed by 66 wells: 33 with control group samples and 33 with skeletal muscle damage group samples. A standard curve was obtained by immunoblotting with an amount of known proteins (ng of pure α-actin) and densometrically quantifying the bands obtained.

Statistical analysis. Data were entered into a personal computer and statistical procedures were performed using the SPSS statistical package (v.11.5). Statistical significance was preset at the p<0.001 level.

Because of the quasi-experimental design of the study, a mixed two-factor MANOVA was used after testing that the appropriate assumptions were met by using Bartlett’s test of sphericity and the Kolmogorov-Smirnov test of normality. Because Levene’s test for equality of variances was significant for only two of the dependent variables, the more conservative Pillai Trace (PT) statistic was used.

RESULTS

Table 1 shows the mean values and SDs for the six dependent variables as a function of the sport and measurement time point.

In the multivariate analyses, the sports (handball vs. rugby) variable (PT = 0.923; F(6,16)= 24.864; p = 0.0001; Eta² = 0.962), measurement time variable (PT = 0.962; F(6,16)= 68.085; p=0.0001; Eta² = 0.962) and interaction between them (PT = 0.852; F(6,16)= 15.390; p=0.0001; Eta² = 0.852) were all significant.

Differences between handball and rugby groups were only found in MYO, cTnT, ACT and LDH (Table 2). Both groups showed differences between before and after the match in all variables except for TnI and Ck (Table 3). MYO, cTnT and LDH values were significantly influenced by the interaction between Measurement Time and Sport (Table 4).

Figure 1 shows the higher level of all markers (except for cTnT) after the handball and rugby matches, observing that the greatest increase was in α-actin. α-actin levels were highly similar between the groups before the match, with the handball players showing a higher post-match increase versus the rugby players.

In Figure 2, the first gel (A) represents pre-match α-actin levels in sera from the handball group, with a band at 43 kDa. Lane 1 corresponds to the positive control (80 ng/µl of pure actin) and lanes 2-9 to the pre-match α-actin levels of the handball group. The second gel (B) represents post-match α-actin levels in sera from the handball group. Lane 1 corresponds to the positive control (80 ng/µl of pure actin) and lanes 2-9 to the post-match α-actin levels of the same group.

TABLE 2. F(1,21) VALUE FOR SPORT VARIABLE VERSUS DEPENDENT VARIABLES.

<table>
<thead>
<tr>
<th>Variable</th>
<th>F</th>
<th>Significance</th>
<th>Partial square</th>
</tr>
</thead>
<tbody>
<tr>
<td>TnI</td>
<td>0.865</td>
<td>0.363</td>
<td>0.040</td>
</tr>
<tr>
<td>MYO</td>
<td>13.913</td>
<td>0.001*</td>
<td>0.399</td>
</tr>
<tr>
<td>cTnT</td>
<td>104.025</td>
<td>0.000*</td>
<td>0.832</td>
</tr>
<tr>
<td>Act</td>
<td>9.308</td>
<td>0.006</td>
<td>0.307</td>
</tr>
<tr>
<td>CPK</td>
<td>0.601</td>
<td>0.447</td>
<td>0.028</td>
</tr>
<tr>
<td>LDH</td>
<td>14.695</td>
<td>0.001*</td>
<td>0.412</td>
</tr>
</tbody>
</table>

Note: * - Significant difference (p<0.01).

TABLE 3. VALUE OF GREENHOUSE-GEISSER STATISTIC FOR MEASUREMENT TIME VERSES DEPENDENT VARIABLES.

<table>
<thead>
<tr>
<th>Variable</th>
<th>F</th>
<th>Significance</th>
<th>Partial square</th>
</tr>
</thead>
<tbody>
<tr>
<td>TnI</td>
<td>0.000</td>
<td>1.000</td>
<td>0.000</td>
</tr>
<tr>
<td>MYO</td>
<td>102.883</td>
<td>0.0001*</td>
<td>0.830</td>
</tr>
<tr>
<td>cTnT</td>
<td>58.197</td>
<td>0.0001*</td>
<td>0.735</td>
</tr>
<tr>
<td>Act</td>
<td>103.938</td>
<td>0.0001*</td>
<td>0.832</td>
</tr>
<tr>
<td>CPK</td>
<td>113.704</td>
<td>0.0001*</td>
<td>0.844</td>
</tr>
<tr>
<td>LDH</td>
<td>159.866</td>
<td>0.0001*</td>
<td>0.884</td>
</tr>
</tbody>
</table>

Note: * - Significant difference (p<0.01).

TABLE 4. GREENHOUSE-GEISSER VALUES FOR THE INTERACTION OF SPORT AND MEASUREMENT TIME VERSUS DEPENDENT VARIABLES.

<table>
<thead>
<tr>
<th>Variable</th>
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<td>0.363</td>
<td>0.040</td>
</tr>
<tr>
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<td>13.913</td>
<td>0.001*</td>
<td>0.399</td>
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<td>104.025</td>
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<td>0.832</td>
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<td>Act</td>
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<td>0.006</td>
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<td>0.601</td>
<td>0.447</td>
<td>0.028</td>
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<tr>
<td>LDH</td>
<td>14.695</td>
<td>0.001*</td>
<td>0.412</td>
</tr>
</tbody>
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Note: * - Significant difference (p<0.01).
Measurement of the release of muscle enzymes is used to estimate the amount of muscle damage after sports exercise, since this release is assumed to be related to the amount of muscle enzymes and hence to the muscle damage caused by the exercise [2]. Armstrong demonstrated that more muscle enzymes were released by untrained rats than trained rats during exhausting physical exercise [4]. Release of these intracellular proteins has been attributed to permeability changes and muscle membrane rupture [18]. Some authors found no correlation between muscle enzyme release and histologically-evaluated muscle damage, supporting the proposition that increased enzyme release is permitted by increased membrane permeability [30]. However, this mechanism has not been fully elucidated and the cause of any increased permeability has not been established. In aerobic activities of long duration, e.g., marathons, an increase in plasma creatine phosphokinase and myoglobin is frequently observed as an expression of muscle damage, leading some authors to propose these increases as markers of overtraining [19]. However, similar increases have been reported after acute training or competition in athletes without overtraining syndrome [11,26]. The objective of the present study was to determine the release of proteins after intense physical activity (high-level competitive match), considering levels detected during pre-match rest as reference values.

Myoglobin levels were markedly and significantly higher after the match in both sports groups. This increase was higher in the rugby group, possibly because a rugby match involves more physical aggression and stronger action with continual body contact [29]. On the other hand, these factors also vary according to the position of each player in their team. In fact, the considerable inter-individual variability found means that myoglobin levels cannot serve to support an early diagnosis of fatigue and muscle damage. This protein is also considered to be highly non-specific because its blood concentration can be increased in both skeletal and cardiac damage [21]. It is released very early into the blood, and high levels were found in the players’ sera immediately after the match.

The data obtained in this study agree with previous reports that myoglobin is detected in the blood much earlier than CK and therefore reaches its peak sooner [21,20]. At present, the most widely used biochemical indicator to assess muscle damage is the release of intramuscular CK enzyme into
the blood. The length of the delay between the end of acute exercise and the increase in blood CK activity is controversial. Some authors described a peak immediately after exercise [30, 1] but others reported longer intervals, with one group even observing two peaks of CK activity at 0 hrs and at 24-36 hrs after a downhill race [4]. Van der Meulen et al. highlighted the considerable difference between CK activity levels in rats after 1.5 hrs and 2.5 hrs of exercise [30].

Muscle proteins (e.g., CK and myoglobin) are known to be released from damaged muscle into the blood after high-intensity physical exercise [7]. The present study confirms that high-intensity sports activity increases CK levels, which reached 409.6 IU/L after the handball and 391.1 IU/L after the rugby match. Account should be taken of the high CK levels in these players before the match: 308.6 IU/L in the handball players and 235.4 IU/L in the rugby players. These elevated pre-match levels indicate a possible fatigue accumulation due to intense daily exercise, which may induce muscle injury and the degradation of somatic tissue proteins during the match.

Various authors have shown that the release of these enzymes induces muscle inflammation and skeletal muscle collapse, allowing them to be used as markers of exercise-induced skeletal muscle damage and muscle fatigue [19]. The increase in these enzymes during intense exercise is higher in untrained than in trained individuals [9]. This was reflected in the present study by the lower increase in LDH and myoglobin levels in the handball players, who train more regularly than the rugby players (data not shown), conferring their muscles with a higher degree of protection against muscle damage [9, 28].

According to Nosaka et al., the greatest drawback to using blood muscle protein levels as a muscle damage marker is the wide inter-individual variability in their release, with CK levels ranging from 236 to 25,244 IU/L [25]. Individuals with higher CK activity in blood generally had larger muscle damage according to magnetic resonance imaging (MRI) findings but the authors reported that this was not a perfect relationship.

A recent study demonstrated an association between serum CK and glutathione activities, and the authors suggested that glutathione might act as a CK protector in the blood [15]. This and other factors may eventually explain the wide variability in the response of CK to exercise. At any rate, CK activity in the blood only provides an indirect qualitative marker of muscle damage and may be influenced by various non-muscular factors, including genetic factors [7].

A 95% increase in plasma LDH activity was observed in the rugby players after the match, similar to the 85% increase reported by Komulainen et al. [18]. In contrast, the handball players showed higher baseline levels but only a small post-match increase [24].

Plasma troponin levels were very low, and no significant differences in cTnT were observed between before and after the match. Minimal amounts of cTnT were detected, and no significant difference was observed between these time points. Serum cTnT levels are very low or undetectable in the absence of disease, and maximum cTnT levels for healthy subjects are around 0.06 ng/mL, compared with maximum values of 0.03 ng/mL in the present study population [24].

A significant increase (p<0.001) in α-actin levels was observed after the match in both the handball group (from 7.16 to 26.47 μg/mL) and rugby group (from 1.24 to 20.04 μg/mL). These findings contradict the report by Féasson et al., using immunoblotting, that α-actin levels were not modified either immediately after, at one day after, or at 14 days after eccentric exercise [10]. This discrepancy with the results obtained in this study may be explained by differences in technique or the type of antibody. Thus, Féasson et al. used an antibody from a different clone (AC-40, from Sigma) at a different dilution (1/3000). We confirmed the reliability of the present study by comparing the results of multiple assays [10].

CONCLUSIONS

The results of this study confirm that large amounts of α-actin are released into the peripheral blood immediately after an intense physical effort. The measurement of α-actin appears to offer a sensitive instrument to detect muscle lesions in sportspersons at risk of injury.

Acknowledgements

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