**Level of hamstrings damage depending on force-generating capacity and creatine kinase activity**

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**Introduction**

Although direct evidence of muscle damage is histological, in the sports context, easy measurable proxy markers are needed. According to Paulsen et al. (2012), muscle function measured as force-generating capacity (e.g., maximal isometric strength) is considered to be a reliable marker of the degree of muscle damage:

- *mild exercise-induced muscle damage* corresponds with a decline in force-generating capacity of no more than 20% and recovery within 48 hours.
- *moderate exercise-induced muscle damage* corresponds with a 20-50% decline in force-generating capacity, and/or full recovery between 48-168 hours.
- *severe exercise-induced muscle damage* corresponds with a decline in force generating capacity of more than 50%, and/or recovery of force-generating capacity exceeds 168 hours.

Furthermore, serum creatine kinase (CK) activity may be used to separate subjects with mild muscle damage (< 1,000 IU/L) and severe muscle damage (> 10,000 IU/L) ( Clarkson and Newham, 1995).

**Methods**

**Participants**

Thirteen young and healthy recreational sport practisers (21.3 ± 0.5 yrs, 74.6 ± 4.1 kg, 178 ± 1.7 cm) with no previous hamstring injuries one year before the study.

**Eccentric exercise**

Each participant performed 6 x 10 eccentric unilateral hamstring curls at 120% of their 1-repetition maximum (Kubota et al., 2007; Mendiguchia et al., 2013).

**Procedures**

**Force-generating capacity**

Force-generating capacity was measured as maximum voluntary contractions (MVCs) (maximal isometric strength); i.e. average force in a 1-s window once a force plateau had been established. MVC of the knee flexors muscles were measured with a force gauge connected to an A/D converter system (MuscleLab™, Engedal AS, Langesund, Norway). Subjects were prone with the hip joint at 40° of flexion, with the knee joint at 30° of flexion. Measurements were done at baseline (Pre), and at 24, 48 and 72 hours post-exercise. The leg which showed the greatest loss of MVC was used for further analyses.

**Creatine kinase**

8mL blood samples were obtained at the baseline (Pre), and at 24, 48, and 72 hours post-exercise for the later analysis of CK activity in serum. CK was used as a biomarker of fibre membrane disruptions (Baird et al., 2012) induced by the eccentric exercise. This study was approved by the Ethics Committee of the Catalan Autonomous Government (Sports Council).

**Results**

Conclusions

The same intensive eccentric exercise protocol induced different levels of muscle damage. According to the categorization proposed by Paulsen et al. (2012) based on the loss of force-generating capacity, the eccentric exercise induced severe muscle damage in 8 of the 13 subjects. Those subjects, categorized as high responders, also showed the greatest CK activity at every time point analyzed. Interestingly, 2 subjects were moderate responders since they showed a loss of the force-generating capacity between the 20 and 50% of the MVC. However, the fact that the force-generating capacity was not fully recovered 1 week after the exercise and the CK showed a clear increasing trend (serum peak at 168 hours post-exercise), suggested a more severe muscle damage process. Surprisingly, 3 subjects were low responders since they showed an slight decrease of the force-generating capacity (< 20% MVC) an were recovered one week after the exercise. Moreover, the CK serum activity at every time point analyzed were the lowest in this group (< 1,000 IU/L). Nevertheless, in low responders, CK peaked at 24 hours post-exercise and at 168 hours post-exercise showing a very different time course and magnitude of changes than the rest of the participants.

Once the traditional statistics have been performed, the categorization of subjects according to its loss in force-generating capacity seems to allow for better interpretations. While in high responders the time course of force-generating capacity and CK activity showed a similar recovery trend, in moderate and low responders only the force-generating capacity showed a recovery trend.

**References**


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**Figure 1.** Data are presented as mean ± SEM (n = 13). (a) Time-related changes (one-way repeated measures ANOVA followed by paired t test with Bonferroni correction) on percentage of Maximum Voluntary Contraction (MVC). (b) Time-related changes (Friedman’s one-way ANOVA followed by Wilcoxon matched-pairs signed-rank test for the repeated measures). Green line, mild muscle damage threshold; Red line, severe muscle damage threshold. **‘p** < 0.05, ‘p” < 0.001, respectively.

**Figure 2.** (a) Categorization of subjects as ‘low’, ‘moderate’ and ‘high’ responders according to the loss of Maximum Voluntary Contraction (MVC). (b1) Creative kinase activity from high responders and (b2) moderate and low responders. Green line, mild muscle damage threshold; Red line, severe muscle damage threshold.

**Figure 3.** Categorization of force-generating capacity. **b1** High responders (n = 8) 48-168 hours post-exercise; **b2** Moderate responders (n = 2) 0-48 hours post-exercise; Low responders (n = 4) 0-48 hours post-exercise.