ELECTROMECHANICAL DELAY AND ITS MECHANISMS ARE NOT IMPAIRED FOLLOWING ECCENTRIC EXERCISE

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The aim of the present study was to assess the effect of exercise-induced muscle damage on both electrochemical and mechanical components involved in the electromechanical delay in the *gastrocnemius medialis* muscle. 15 healthy participants completed 10 sets of 30 maximal eccentric contractions of the plantar flexor muscles at a constant angular velocity of 45°.s^{-1}. Delayed onset muscular soreness, maximal isometric torque, and electromechanical delay were measured before, 1h, and 48h following eccentric exercise. The present study revealed that the time required for both electrochemical and mechanical process involved in electromechanical delay are not impaired by exercise induced muscle damage. This study suggests that the long lasting reduction in force after eccentric exercise cannot be associated to an alteration of the force transmission efficiency.

KEY WORDS: electromechanical delay, muscle damage, force transmission, ultrafast ultrasound.

INTRODUCTION: Intense or unaccsutomed eccentric exercise generates muscle damage. Indeed, for extensive damage, some myofiber areas or the overall myofiber are disrupted, leading to an irreversible loss of strength in the few days after eccentric exercise (Proske & Morgan, 2001). Interestingly, a strong correlation between the number of damaged muscle fibers and the changes in maximal knee extensor torque after an eccentric exercise has been found (r = 0.92; Raastad et al., 2010). The decrease in muscle force after exercise is mainly attributable to the reduction in the number of active sarcomeres. Additionally, it is plausible that the structural alterations induced by eccentric exercise (e.g., loss of desmin; Proske & Allen, 2005) contribute to force transmission impairments around the disrupted sarcomeres, and in turn decrease force at the joint level. However, the muscle force transmission after eccentric exercise has never been investigated *in vivo.*
The efficiency of the musculotendinous complex to create and transmit force can be assessed by measuring the electromechanical delay (EMD), which corresponds to the time lag between onsets of muscle activation and force production (Cavanagh & Komi, 1977). Using very high frame rate ultrasound, the relative contribution of both electrochemical (synaptic transmission, excitation-contraction coupling) and mechanical components (force transmission) involved in the EMD has been recently characterized in humans (Nordez et al., 2009). This method has been already used to describe the influence of passive tension on muscle force transmission (Lacourpaille et al., 2013) and to better understand the effect of Duchenne muscular dystrophy on contraction efficiency (Lacourpaille et al., 2014). The aim of the present study was to assess the effect of exercise-induced muscle damage on both electrochemical and mechanical components of the electromechanical delay in the *gastrocnemius medialis* muscle.

METHODS: This study was performed on fifteen healthy participants. Participants were lying prone on a Con-Trex isokinetic dynamometer and completed 10 sets of 30 maximal isokinetic

eccentric contractions of the plantar flexor muscles. Delayed onset muscular soreness, maximal isometric torque, and EMD were measured before (PRE), 1h (1H), and 48h (48H) following eccentric exercise.
For each test session, the maximal isometric torque was measured. Participants underwent two electrically evoked contractions of *gastrocnemius medialis.* During the two electrically evoked contractions, the ultrasound probe was first placed over the *gastrocnemius medialis* muscle belly (muscle delay), parallel to the muscle fascicles, and second, over the previously localized distal myotendinous junction of the *gastrocnemius medialis* (tendon delay) (Nordez et al., 2009). A very high frame rate ultrasound scanner (Aixplorer, version 7.0, Supersonic Imagine, Aix-en-Provence, France), coupled with a linear transducer array (4–15 MHz, SuperLinear 15–4, Vermon, Tours, France) was used in “research” mode to acquire raw radio-frequency signals at 4 kHz. As previously described in Lacourpaille et al. (2013a, 2013b), the detection of the onset of muscle fascicle motion (muscle delay), myotendinous junction motion (tendon delay) and external force production (EMD) was defined visually.
First, two one-way analysis of variance with repeated measures were performed on maximal voluntary contractions torque and soreness. Second, a two-way analysis of variance with repeated measures [factors = time (PRE, 1H, 48H) × delay (muscle delay, tendon delay and EMD)] was used to test whether eccentric exercise induced muscle damage altered muscle, tendon and electrochemical delays. *Post hoc* analyses were performed when appropriate using Scheffe’s method. The statistical significance was set at $p < 0.05$.

RESULTS: A significant main effect of time on muscle soreness was found ($p<0.0001$). More precisely, *post hoc* analysis revealed that muscle soreness was significantly increased at 48H (4.6/10) compared to PRE (0.5/10; $p<0.0001$). Isometric maximal voluntary contraction was reduced at 1H (- 43.3 ± 15.2 %; $p<0.0001$) and 48H post-exercise (-12.7 ± 14.1 %; $p<0.03$). Figure 1 depicts the results obtained for muscle, tendon and electromechanical delays. Muscle delay (6.5 ± 1.1 ms) was lower than tendon delay (8.3 ± 1.5 ms) ($p<0.0001$). Both muscle and tendon delays were lower than EMD (15.3 ± 1.9 ms) ($p<0.0001$). No significant main effect of time on *gastrocnemius medialis* delays ($p=0.062$) and no significant time × delay interaction were found ($p=0.310$).

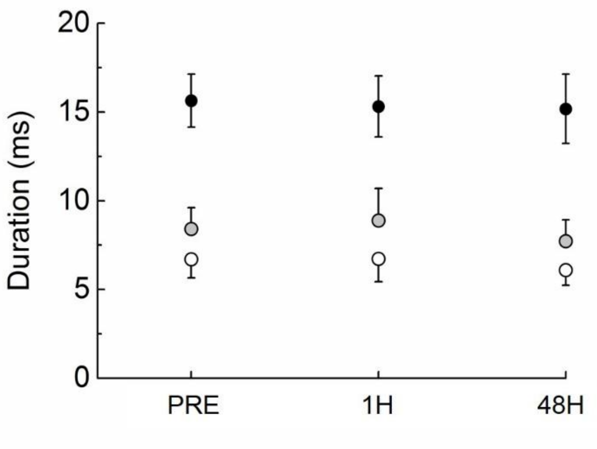

Figure 1. *Gastrocnemius medialis* delays obtained before, 1 hour and 48 hours after eccentric exercise. Time course of muscle delay (delay between muscle activation and the onset of *gastrocnemius medialis* fascicle motion; white circles), tendon delay (delay between muscle activation and the onset of *gastrocnemius medialis* myotendinous junction motion; grey circles) and electromechanical delay (EMD: delay between muscle activation and force production; black circles), before (PRE), 1 hour (1H) and 48 hours (48H) after eccentric exercise. Values are means ± SD.

DISCUSSION: The present study showed that both electrochemical and mechanical components involved in electromechanical delay are not altered after exercise-induced muscle damage. More precisely, although muscle force was significantly reduced immediately and 48 hours after the eccentric exercise, the time delays between electrical stimulation and the onsets of *gastrocnemius medialis* fascicle motion, myotendinous junction motion and force production were not affected.
A reversible loss of strength is observed in the first time following an eccentric exercise. Numerous *in vitro* studies demonstrated that this symptom is mainly due to the failure of the excitation-contraction coupling process (for review, see Warren et al., 2001). This finding has been indirectly confirmed *in vivo* by comparing the force production with low-frequency stimulations to high-frequency stimulations after an eccentric exercise (20:50 Hz force ratio) (Raastad et al., 2010; Martin et al., 2004). However, it is important to note that this method (20:50 Hz force ratio) can also be affected by the change of series compliance due to overstretching sarcomeres (Gregory et al., 2007; Raastad et al., 2010). Thus, the originality of this study resides in the direct assessment of the efficiency of muscle electrochemical process *via* the characterization of the delay between electrical stimulation and onset of fascicle motion (i.e., muscle delay). Surprisingly, muscle delay was not impaired after the eccentric exercise. Consequently, our results suggest that the efficiency of the electrochemical processes might be affected independently of its duration. This is in line with a previous study showing that muscle delay was not significantly impaired in patients with Duchenne muscular dystrophy (Lacourpaille et al., 2014) while excitation-contraction coupling failure has been largely demonstrated *in vitro* (Woods et al., 2004, 2005; De Luca et al., 2001; Capote et al., 2010).
In presence of muscle damage the long lasting decrease in muscle force has been mainly attributed to the reduction in the number of active sarcomeres. Nonetheless, considering the structural alterations induced by eccentric exercise (e.g., loss of desmin; Proske & Allen, 2005) we hypothesized that the force transmission around the disrupted sarcomeres could be impaired, leading to increase the time to transmit the force from muscle to the bone. Indeed, Lacourpaille et al. (2014) recently showed that the delay between the onsets of *biceps brachii* fascicle motion and force production is significantly increased in patients with Duchenne muscular dystrophy, highlighting the impairment of muscle force transmission previously inferred from *in vitro* studies (Claflin & Brooks, 2008; Ramaswamy et al., 2011). However, contrary to our expectation, the present study showed that the delay between muscle electrical stimulation and the onsets of myotendinous junction motion (tendon delay) and force production (EMD) were not elongated. Taking these elements together, we can speculate that, in healthy skeletal muscle, the structural arrangement of the aforementioned pathways (i.e., 'cross bridges', 'titin' and 'costamere'; Patel & Lieber, 1997) allow the preservation of the force transmission even in presence of muscle damage.

CONCLUSION: This study revealed that EMD and its electrochemical and mechanical components are not impaired by eccentric exercise induce muscle damage. This result suggests that the long lasting reduction in force after eccentric exercise cannot be associated to an alteration of the force transmission efficiency, and underline the putative preservation of 'costamere' pathway.

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