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EFFECT OF DAMAGING EXERCISE ON ELECTROMECHANICAL DELAY

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ABSTRACT: *Introduction:* In this study we aimed to quantify the effect of exercise-induced muscle damage on both the electrochemical and mechanical components of electromechanical delay using very-high-frame-rate ultrasound. *Methods:* Fifteen participants underwent electrically evoked contractions of the medial gastrocnemius muscle with an ultrasound transducer on the muscle belly and on the myotendinous junction, before, 1 hour, and 48 hours after eccentric exercise of the plantar flexor muscles. *Results:* Maximal isometric plantar flexor torque was significantly lower at 1 hour ($-41.1 \pm 14.9\%$; $P = 0.0001$) and 48 hours ($-11.9 \pm 14.9\%$; $P = 0.038$) post-exercise compared with pre-exercise. However, the delay between electrical stimulation and the onset of muscle activation, the delay between electrical stimulation and myotendinous junction motion, and the electromechanical delay were not altered significantly by eccentric exercise ($P = 0.063$). *Conclusions:* These findings suggest that moderate muscle damage does not affect the time for the electrochemical or mechanical components of electromechanical delay.

Unaccustomed or repetitive eccentric contractions are recognized as a major cause of destructive changes in muscle ultrastructure.^{1,2} It has been hypothesized that disruption of the muscle structures involved in force production and transmission, followed by activation of calcium-dependent degradative pathways (for review, see Warren *et al.*³), is the cause, but the exact mechanisms of muscle fiber microinjury are not fully understood. *In vitro* studies have demonstrated that muscle damage may impair excitation-contraction coupling immediately after eccentric contractions, with a slow recovery over the next 7 days.⁴ At the same time, the time-courses of protein loss (contractile and cytoskeletal) and extracellular matrix disruption show non-significant changes immediately after exercise, followed by a peak value between 1 and 5 days later, depending on the marker

used.⁵⁻⁷ These cytoskeletal alterations are classically considered to be the main contributors of strength loss after eccentric exercise.^{3,8} However, to date, the consequences of excitation-contraction coupling failure and protein loss after eccentric exercise on the force-generating and/or -transmitting processes have not been investigated *in vivo*.

Electromechanical delay (EMD) corresponds to the time lag between the onset of muscle activation and force production⁹ and is influenced by both electrochemical (synaptic transmission, excitation-contraction coupling) and mechanical processes (force transmission).^{9,10} The relative contributions of both electrochemical and mechanical components have been quantified *in vivo* using very-high-frame-rate ultrasound^{10,11} or mechanomyography.^{12,13} More precisely, the delay between muscle electrical stimulation and the onset of muscle fascicle motion is mainly due to electrochemical processes.¹⁰ The delay between the onset of fascicle motion and the onset of force production has been attributed to force transmission along the series-elastic components, which can be split into 2 parts (i.e., aponeurosis and tendon) by detecting the onset of myotendinous junction motion.¹⁰ Using ultrafast ultrasound, Lacourpaille *et al.*¹⁴ showed that patients with Duchenne muscular dystrophy exhibit a longer delay between the onset of muscle fascicle motion and force production compared with healthy, age-matched participants, suggesting altered muscle force transmission. Further characterization of the time required for production and transmission of muscle force could provide a better understanding of electrochemical and mechanical consequences of muscle damage. Thus, the aim of this study was to assess the effect of exercise-induced muscle damage on both the electrochemical and mechanical components of electromechanical delay in the medial gastrocnemius muscle. We used very-high-frame-rate ultrasound to determine the onset of both muscle fascicle and myotendinous junction motion during electrically evoked contractions. We hypothesized that: (1) EMD would be lengthened 1 hour after eccentric exercise, mainly because of a longer delay associated with electrochemical processes; and (2) both electromechanical

Abbreviations: 1H, 1 hour after exercise; 48H, 48 hours after exercise; ANOVA, analysis of variance; Dm, muscle delay; DOMS, delayed-onset muscle soreness; Dt, tendon delay; EMD, electromechanical delay; I_{max}, intensity inducing the maximal plantar flexion force; MDC, minimally detectable change; MVC, maximal voluntary contraction; Pre, pre-exercise test session

Key words: eccentric contraction; excitation-contraction coupling; exercise; muscle damage; muscle force transmission; high-frame-rate ultrasound

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and mechanical components of EMD would be increased 48 hours after eccentric exercise.

METHODS

Fifteen healthy volunteers participated in this experiment (8 men and 7 women; age 24.7 ± 4.1 years, height 1.73 ± 0.08 m, weight 70.6 ± 13.1 kg). They were informed regarding the nature, aims, and risks associated with the experimental procedures before providing written consent. The study was approved by the local ethics committee (No. 120656), and all procedures conformed to the 2004 Declaration of Helsinki.

Experimental Design. The experiment was conducted on 3 separate days. Participants first performed an initial test session (Pre) 2 days before they performed eccentric exercise of the plantar flexor muscles. A similar session was repeated 1 hour (1H) and 48 hours (48H) after the eccentric exercise and at the same time of day. The peak torque produced during an isometric maximal voluntary contraction, delayed-onset muscular soreness (DOMS), and EMD were measured during each session (Pre, 1H, and 48H).

Eccentric Exercise. Before the exercise, the maximal range of motion in dorsiflexion was determined. Participants lay prone (full hip extension = 0° , full knee extension = 0°) on a Con-Trex isokinetic dynamometer (CMV AG, Dübendorf, Switzerland) with the right ankle fixed to the dynamometer's accessory with non-compliant straps to prevent heel rise. The input axis of the dynamometer was carefully adjusted to the axis of rotation of the right ankle joint. Eccentric contractions were performed over the total range of motion for each participant (from 18° in dorsiflexion to 42° in plantar flexion, on average). Participants completed 10 sets of 30 maximal eccentric contractions of the plantar flexor muscles at a constant angular velocity of $45^\circ/\text{s}$. Between each contraction, the foot was passively repositioned to the starting position. A 2-min passive recovery period was included between each set. Enthusiastic verbal encouragement was given to ensure maximum effort of participants in each repetition.

Muscle Soreness. DOMS was evaluated at the beginning of each test session using an illustrated visual analog scale numbered from 0 ("My muscles do not feel sore at all") to 10 ("My muscles feel so sore that I do not want to move them") with a sliding pointer. Subjects were asked to report the soreness level on the scale after palpations of the calf muscles by the same investigator on different sites of the triceps surae, the muscle insertions, and the muscle bellies of 3 plantar flexor muscles.¹⁵

Maximal Isometric Torque. After a warm-up of 20 isokinetic repetitions, maximal plantar flexor tor-

que was evaluated on a Con-Trex ergometer, during 5-s maximal isometric voluntary contractions (MVCs) at a 90° ankle angle. A total of 3 trials was performed, and the trial with the highest isometric torque was considered for further analysis. Mechanical signals provided by the dynamometer (angle, velocity, and torque) were digitized by a 12-bit analog-to-digital converter (DT 9804; Data Translation, Marlboro, Massachusetts) at a sampling frequency of 5 kHz and low-pass filtered (third-order zero-lag Butterworth filter, cut-off frequency 10 Hz).

Electromechanical Delay. *Ergometer.* A home-made ergometer was used to measure the force produced by the plantar flexors.¹⁰ Subjects lay prone with their legs fully extended. The right foot was fixed securely in a rigid cycling shoe on an adjustable system connected to a force transducer (SML-50; range 0–50 lbf, sensitivity 2 mV/V, interface) near the metatarsal joint. A rigid cycling shoe was chosen to avoid possible dynamics in coupling between the shoe and the force sensor. The force signal was digitized at a sampling rate of 5 kHz (MP36; BIOPAC, Goleta, California).

Electrical Stimulation. Percutaneous electrical stimulations were applied over the medial gastrocnemius to elicit contraction. A constant-current stimulator (DS7A; Digitimer, Letchworth Garden City, UK) delivered a single electrical pulse (pulse duration 500 μs , 400 V) through 2 electrodes (2×1.5 cm; Compex, Annecy-le-Vieux, France), 1 placed on the main motor point (previously determined as the location inducing the strongest twitch with the lowest electrical stimulation) and the other on the distal portion of the medial gastrocnemius.

Ultrasonography. A very-high-frame-rate ultrasound scanner (Aixplorer, version 5.0; 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 radiofrequency signals at 4 kHz. Force and ultrasound data were synchronized using transistor-transistor logic pulses, as described elsewhere.^{11,16}

Protocol. Participants underwent 4 electrically evoked contractions of the medial gastrocnemius, with 1-min rest in between, at 70% of the intensity that induced maximal plantar flexion force (I_{max}) with a 1-min rest in between. Briefly, I_{max} was determined by increasing the output current (incremental step of 5 mA) until the maximum force output was achieved. Seventy percent of I_{max} was chosen to limit the discomfort associated with the stimulation and because it was demonstrated elsewhere that EMD is not affected by an increase in stimulus intensity above this threshold.¹⁶ The

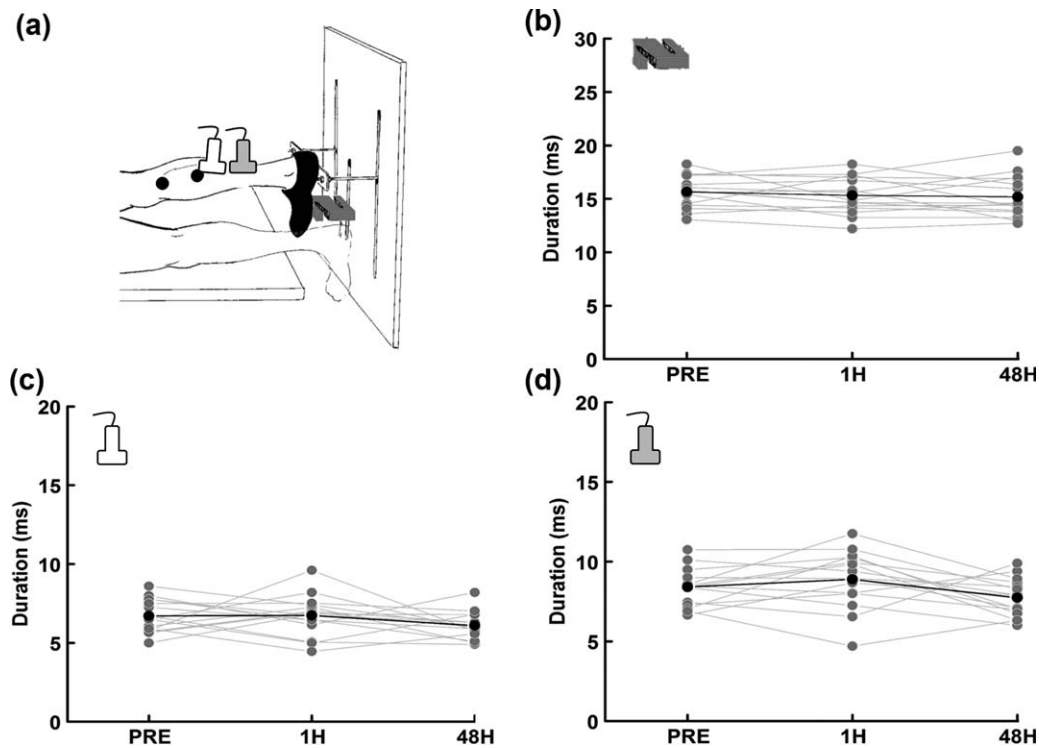


FIGURE 1. Experimental set-up (a) and individual medial gastrocnemius delays obtained before (PRE), and 1 hour (1H) and 48 hours (48H) after eccentric exercise (b–d). (a) Two electrical stimulation electrodes were placed over the medial gastrocnemius (black circles). Probe locations for muscle delay (Dm: delay between muscle activation and the onset of medial gastrocnemius fascicle motion; white transducer) and tendon delay (Dt: delay between muscle activation and the onset of medial gastrocnemius myotendinous junction motion; gray transducer). Individual time-course of electromechanical delay [EMD: delay between muscle activation and force production (b)], muscle delay [Dm (c)], and tendon delay [Dt (d)], before (PRE), and 1 hour (1H) and 48 hours (48H) after eccentric exercise, are represented with gray lines and circles. Mean time-courses are represented with black circles and lines.

ultrasound transducer was placed over the medial gastrocnemius muscle belly (2 muscle trials) and over the previously localized distal myotendinous junction of the muscle (2 tendon trials) (Fig. 1a). Participants were instructed to be fully relaxed before each stimulation.

Data Processing. EMD data were processed using MATLAB scripts (The Mathworks, Inc., Natick, Massachusetts). Ultrasound B-mode images were used to determine the region of interest for each contraction (between the 2 aponeuroses of the medial gastrocnemius for muscle trials and on the myotendinous junction for tendon trials).¹⁰ The displacements along the ultrasound beam axis were calculated using a 1-dimensional cross-correlation of the windows of consecutive radiofrequency signals.^{17,18} Thus, the tissue motion between the 2 consecutive images (i.e., particle velocity) was measured with micrometric precision. Displacements were averaged over the region of interest and then used to detect the onset of muscle and myotendinous junction motion. As previously

described by Lacourpaille *et al.*,¹⁴ detection of onset of both muscle fascicle and myotendinous junction motion and external force production were determined visually and blinded (the experimenter was not aware of the condition when doing the detection). We defined the EMD as the time delay between electrical stimulation and the onset of force production. The delays between muscle electrical stimulation and the onsets of muscle fascicle motion [referred to as time delay for muscle contraction (Dm, for muscle trials)] and myotendinous junction motion [referred to as time delay for tendon motion (Dt, for tendon trials)] were determined. Then, the difference between Dm and Dt (aponeurosis delay) and between Dt and EMD (tendon delay) were calculated as pure indicators of force transmission along aponeurosis and tendon, respectively. The between-session reliability of Dm, Dt, and EMD was previously determined on the biceps brachii muscle.¹⁶ The standard errors of measurement (SEM) were 0.51 ms, 0.34 ms, and 0.66 ms for Dm, Dt, and EMD, respectively. Considering these results, the minimum

detectable change (MDC) of each delay (MDC = standard error in measurement $\times$ 2.77)¹⁹ considered in this study was 1.41, 0.94, and 1.82 ms for Dm, Dt, and EMD, respectively.

Statistical Analysis. All analyses were performed with Statistica version 7.0 (StatSoft, Inc., Tulsa, Oklahoma). Normality testing (Kolmogorov–Smirnov) was consistently passed, and therefore values are reported as mean $\pm$ standard deviation. First, 3 separate 1-way (time effect) analyses of variance (ANOVAs) with repeated measures were performed on MVC torque, electrically evoked force, and DOMS. Second, a 2-way ANOVA with repeated measures [within-subject factors = time (Pre, 1H, 48H) $\times$ delays (Dm, Dt, and EMD); categorical factor = gender (men and women)] was used to test whether eccentric exercise-induced muscle damage altered Dm, Dt, and EMD. Third, a 2-way ANOVA with repeated measures [within subject factors = time (Pre, 1H, 48H) $\times$ delays (aponeurosis and tendon)] was used to test whether eccentric exercise affects force transmission independently of electrochemical process. The sphericity assumption in repeated-measures ANOVA was violated (Mauchly test), and therefore the Geisser–Greenhouse correction was used for EMD, Dm, and Dt. *Post-hoc* analyses were performed when appropriate using the Newman–Keuls method. Statistical significance was set at $P < 0.05$.

RESULTS

Gender Effect. A significant interaction was found between delays and gender ($P = 0.04$). *Post-hoc* analysis revealed that EMD was significant longer in men compared with women ($P = 0.01$; + 10.2%). However, no significant gender $\times$ delays $\times$ time interaction was found ($P = 0.25$).

Presence of Muscle Damage. Time was a significant main effect on muscle soreness ($P < 0.0001$). More precisely, *post-hoc* analysis revealed that DOMS was significantly increased at 48H (4.6 of 10) compared with Pre (0.5 of 10; $P < 0.0001$), whereas no significant difference was found at 1H (0.9 of 10; $P = 0.58$). ANOVA revealed a significant main effect of time ($P < 0.0001$) on maximal voluntary torque. Maximal isometric plantar flexor torque was significantly lower at 1H ($-41.1 \pm 14.9\%$; $P = 0.0001$) and 48H ($-11.9 \pm 14.9\%$; $P = 0.038$) after exercise compared with Pre. The electrically evoked force of the medial gastrocnemius was significantly altered at 1H after eccentric exercise ($-48.4 \pm 20.8\%$; $P < 0.0001$) and returned close to baseline values at 48H ($-5.0 \pm 19.8\%$; $P = 0.28$).

Consequences of Muscle Damage on EMD. Figure 1 depicts the mean and individual time-courses of EMD (Fig. 1b), Dm (Fig. 1c), and Dt (Fig. 1d).

Neither a significant main effect of time ($P = 0.063$) nor an interaction of time $\times$ delays ($P = 0.310$) could be found. Thus, EMD, Dm, and Dt were not significantly affected by exercise-induced muscle damage, but a main effect of delay was shown ($P < 0.0001$). Indeed, Dm was shorter (6.5 ± 1.1 ms) than both Dt (8.3 ± 1.5 ms; $P < 0.0001$) and EMD (muscle trial: 15.3 ± 1.9 ms; $P < 0.0001$). Dt was also significantly shorter than EMD (tendon trial: 15.3 ± 1.6 ms; $P < 0.0001$). No significant difference was found between the EMD in muscle trials and tendon trials. ANOVA revealed no significant effect of time on the calculated force transmission delays (aponeurosis and tendon; $P = 0.575$). The time required for force transmission along the aponeurosis was significantly shorter (1.8 ± 1.1 ms) than along the tendon (7.0 ± 1.9 ms; $P < 0.0001$). No significant interaction between force transmission delays and time was found ($P = 0.175$).

DISCUSSION

In this study we have shown that both the electrochemical and mechanical components involved in EMD are not altered significantly by exercise-induced muscle damage. More precisely, although maximal muscle strength was significantly reduced at 1H and 48H after eccentric exercise, the time delays between electrical stimulation and the onsets of medial gastrocnemius fascicle motion, myotendinous junction motion, and force production were not affected significantly.

Before eccentric exercise, EMD, Dm, and Dt values were 15.6 ± 1.5 , 6.7 ± 1.1 , and 8.4 ± 1.2 ms, respectively. Using the same experimental protocol, Nordez *et al.*¹⁰ reported similar values for Dm and Dt, but shorter EMD (11.6 ms). This discrepancy may be explained by different gender representation between the studies (i.e., only men in Nordez *et al.*¹⁰ vs. 53% men in our study).²⁰ The higher tendon stiffness exhibited in men compared with women^{21,22} may contribute to increased force transmission velocity between the muscle and bone and, in turn, decreased EMD. However, our results do not support this hypothesis. It should be kept in mind that the sample size for the gender comparison was small (8 men and 7 women), leading to a small statistical power. Further studies are needed to explore the gender effect on EMD and its components.

According to the literature,^{23,24} the decrease in maximal voluntary isometric torque measured 48 hours post-exercise provides a good estimate of the amount of muscle damage. Recently, Raastad *et al.*⁷ reported a strong relationship between the percentage of fibers with myofibrillar disruptions and changes in force-generating capacity 6 hours after

exercise ($r=0.92$). The 11.9% decrease in maximal peak isometric torque observed at 48 hours after exercise is similar to that reported during exercises classically used to induce muscle damage, such as downhill backward walking (e.g., -14%).²⁵ We are therefore confident that the task used in this study provoked a significant amount of muscle damage.⁸ Despite the presence of moderate damage, according to the classification of Paulsen *et al.*,⁸ there was no significant effect of exercise-induced muscle damage on muscle delays ($P=0.063$).

It is important to consider that the absence of statistical significance may not provide definitive evidence of a lack of difference. This is because our study may have been underpowered. Using the same technique as used in this study, Lacourpaille *et al.*¹⁶ determined the between-session reliability of EMD on the biceps brachii muscle. Based on the reported standard error of measurement (SEM) data, the minimum detectable difference was 1.41, 0.94, and 1.82 ms for Dm, Dt, and EMD, respectively. In the present study, however, the maximal change was well below this threshold (i.e., 0.61 ± 1.23 , 0.69 ± 1.52 , and 0.47 ± 1.34 ms, for Dm, Dt, and EMD respectively), and therefore we are confident that the absence of a significant difference is true and not related to a lack of statistical power. It is also important to note that the absence of a significant effect of exercise-induced muscle damage on EMD may be explained by a lack of sensitivity of our technique to detect subtle changes in EMD. Using a similar technique, however, Rampichini *et al.*¹³ reported a significant lengthening of both electrochemical (+1.4 ms) and mechanical (+3.3 ms) delays at 1 min immediately after a fatiguing task. During recovery, electrochemical delay returned to baseline 7 min after exercise, while mechanical delay remained elevated. In the same way, both Sasaki *et al.*¹² and Lacourpaille *et al.*¹¹ demonstrated the influence of small changes in muscle passive tension on the time required to transmit force from muscle to bone. In addition, an increased time delay between the onset of muscle fascicle motion and force production (aponeurosis and tendon delay) has been shown in children with Duchenne muscular dystrophy (+6.7 ms compared with age-matched healthy participants).¹⁴ Taken together, these results suggest that the technique used in our study was sufficiently sensitive to detect even small changes in EMD.

Some hypotheses can be proposed to explain the absence of significant changes of EMD and its components (i.e., electrochemical and mechanical) after exercise-induced muscle damage. *In vitro* studies demonstrated a large deficit in maximal

strength production immediately after eccentric contractions, whereas only a small volume of tissue was affected by structural damage.²⁶ As most of the strength loss is restored if the muscle is exposed to caffeine, thus increasing Ca^{2+} release from the sarcoplasmic reticulum, numerous studies have shown immediate excitation-contraction coupling failure after eccentric exercise (for review, see Warren *et al.*³). The current study findings do not indicate any significant change between electrical stimulation and the onset of muscle fascicle motion (Dm) after eccentric exercise. Interestingly, despite altered excitation-contraction coupling suggested in children with Duchenne muscular dystrophy, our previous work also failed to show an effect on duration, further suggesting that efficiency may be affected independently of duration. However, a significant increase in force transmission (aponeurosis and tendon delay) has been reported in children with Duchenne muscular dystrophy compared with healthy, age-matched participants (+75%),¹⁴ indicating involvement of muscle structural abnormalities. Due to muscle damage, some force transmitters between myofibrils (e.g., desmin)²⁷ or muscle fibers (e.g., extracellular matrix)²⁸ are disrupted, as evidenced by the histological myofibril distortion observed.²⁹ Our study suggests that moderate muscle damage does not affect the time required to transmit forces from muscle to bone. This finding corroborates the hypothesis proposed by Patel and Lieber,³⁰ which suggests the importance of force transmission redundancy within the muscle cell (both longitudinal and lateral), especially after muscle damage. Nonetheless, further studies are required to better describe the effect of muscle ultrastructural changes (e.g., severe muscle damage, muscle strain injury, and aging) on force transmission in skeletal muscle.

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