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Effects of roller massager on muscle recovery after exercise-induced muscle damage

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ABSTRACT

Two experiments (n = 10) were conducted to determine the effects of roller massager (RM) on ankle plantar flexor muscle recovery after exercise-induced muscle damage (EIMD). Experiment 1 examined both functional [i.e., ankle plantar flexion maximal isometric contraction and submaximal (30%) sustained force; ankle dorsiflexion maximal range of motion and resistance to stretch; and medial gastrocnemius pain pressure threshold] and morphological [cross-sectional area, thickness, fascicle length, and fascicle angle] variables, before and immediately, 1, 24, 48, and 72 h after an EIMD stimulus. Experiment 2 examined medial gastrocnemius deoxyhaemoglobin concentration kinetics before and 48 h after EIMD. Participants performed both experiments twice: with (RM) and without (no-roller massager; NRM) the application of a RM (6 × 45 s; 20-s rest between sets). RM intervention did not alter the functional impairment after EIMD, as well as the medial gastrocnemius morphology and oxygenation kinetics (P > 0.05). Although, an acute increase of ipsilateral (RM = + 19%, NRM = −5%, P = 0.032) and a strong tendency for contralateral (P = 0.095) medial gastrocnemius pain pressure threshold were observed. The present results suggest that a RM has no effect on plantar flexors performance, morphology, and oxygenation recovery after EIMD, except for muscle pain pressure threshold (i.e., a soreness).

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KEYWORDS
Delayed onset muscle soreness increase; performance; muscle morphology; pain pressure threshold; muscle oxygenation

Introduction

The speed of physical recovery after exercise-induced muscle damage (EIMD) is a crucial aspect for performance optimisation (Howatson, Glyn, & van Someren, 2008). It has been reported that EIMD decreases muscle strength and joint flexibility (Matsuo et al., 2014; Ye, Beck, & Wages, 2015). These impairments peak at 24–72 h following EIMD and the recovery rate thereafter is thought to improve when using different modalities (Torres, Ribeiro, Alberto Duarte, & Cabri, 2012). However, a recent systematic review has reported a lack of effectiveness for most existing methods, whereas massage proved to be slightly effective in the relief of EIMD symptoms (Torres et al., 2012).

Foam rolling has been proposed as a recovery method to reduce EIMD symptoms (e.g., delayed onset muscle soreness increase and decreases in muscle strength and joint flexibility), by using a foam roller or a roller massager (RM) (Cheatham, Kolber, Cain, & Lee, 2015; Schroeder & Best, 2015). Foam rolling is a mechanical technique that mimics therapeutic massage by using a dense roller to apply pressure on soft tissues. It has been demonstrated that RM attenuates decrements in lower extremity muscle performance (Jay et al., 2014; MacDonald, Button, Drinkwater, & Behm, 2014; Pearcey et al., 2015a). However, the underlying mechanisms of its effects in reducing functional impairments after EIMD are still unclear. It has been suggested that RM increases blood flow into the damaged tissue enhancing blood lactate removal, reducing tissue oedema, and promoting a higher and faster oxygen delivery to the muscle (Cheatham et al., 2015; Schroeder & Best, 2015). This non-tested hypothesis can be verified by measuring the muscular deoxyhaemoglobin (HHb) extraction using near-infrared reflectance spectroscopy (Ferrari, Binzoni, & Quaresima, 1997).

The effects of RM on joint flexibility and muscle morphology have not been fully examined. Previous studies have not assessed the joint resistance to stretch to understand whether the effects on flexibility are attributed to alterations of muscle–tendon complexes stiffness. Furthermore, although MacDonald et al. (2014) have indirectly assessed muscle swelling recovery through limb girth, a sonographic measurement of muscle cross-sectional area gives a more valid assessment. In addition, it is possible to determine whether swelling effects are due to variations in muscle architecture [i.e., fascicles angles increase without changes in length].

It has been reported that EIMD itself with no treatment induces contralateral (i.e., crossover) effects (Starbuck & Eston, 2012). For instance, Starbuck and Eston (2012) demonstrated a contralateral repeated bout effect after observing a reduced strength loss in the contralateral elbow flexors 24 h after EIMD. RM itself also induces a contralateral effect on pain. Aboodarda, Spence, and Button (2015) observed a pain pressure threshold increase in the contralateral plantar flexors after

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rolling the ipsilateral plantar flexors with a RM; and thus suggesting that perceived pain recovery may be mediated by the central nervous system. However, it is unknown if RM contralateral effects on pain occur following EIMD.

Two experiments were conducted to: (i) determine the effectiveness of a RM in changing the magnitude of EIMD on functional [i.e., maximal voluntary isometric contraction (MVIC), submaximal sustained force, maximal range of motion (ROM), resistance to stretch, pain pressure threshold, and muscle activity] and morphological [i.e., cross-sectional area, and architecture (i.e., thickness, fascicle length and fascicle angle)] characteristics; (ii) examine RM contralateral effects on pain pressure threshold; and (iii) examine the RM effects in muscular HHb kinetics. We hypothesised that: (i) RM would decrease the negative functional and morphological effects induced by EIMD; (ii) contralateral effects would occur in the pain pressure threshold; and (iii) the prophylactic RM effect would relate to the HHb response.

Methods

Participants

Ten participants (8 male and 2 female; age: 24.9 ± 5.4 years; body mass: 67.9 ± 8.6 kg; height: 171 ± 7.6 cm; mean ± SD) volunteered for experiment 1, and another 10 participants (all male; age: 24.5 ± 4.9 year; body mass: 69.4 ± 7.2 kg; height: 173.3 ± 3.9 cm; mean ± SD) volunteered for experiment 2. This sample size was determined using G*Power statistical program (version 3.1.9.2) based on previous reliability testing performed on muscle morphological variables ($r > 0.8$), for a power of 80%, an expected medium effect size (i.e., $f = 0.25$), and for a level of significance of 5%. All participants were athletes with occasional experience of resistance training, had no history of ankle injuries in the past 6 months, and were instructed not to perform intense exercise at least 5 days before and during the experiment. Written informed consent was obtained from all the participants. The local Ethics committee approved the study.

Design

Participants were first familiarised with the experimental protocol in 1 session before any testing. In both experiments, an EIMD stimulus was randomly induced unilaterally to the participant's ankle plantar flexors (experimental leg), while the opposite limb was used as control (i.e., did not exercised), in 2 random balanced conditions: (1) with RM, and (2) without RM (no-roller massager; NRM) (Figure 1-a). One testing session was performed for each condition, and sessions were separated by at least 2 weeks. In the second session both legs changed roles (experimental vs. control), due to the lower contralateral repeated bout effect (Starbuck & Eston, 2012). In experiment 1, the functional and morphological variables were assessed before, immediately after, 1, 24, 48, and 72 h after the EIMD. The tests were performed in the following order: medial gastrocnemius cross-sectional area, muscle thickness, medial gastrocnemius architecture (i.e., fascicle length and fascicle angle), passive ankle dorsiflexion flexibility

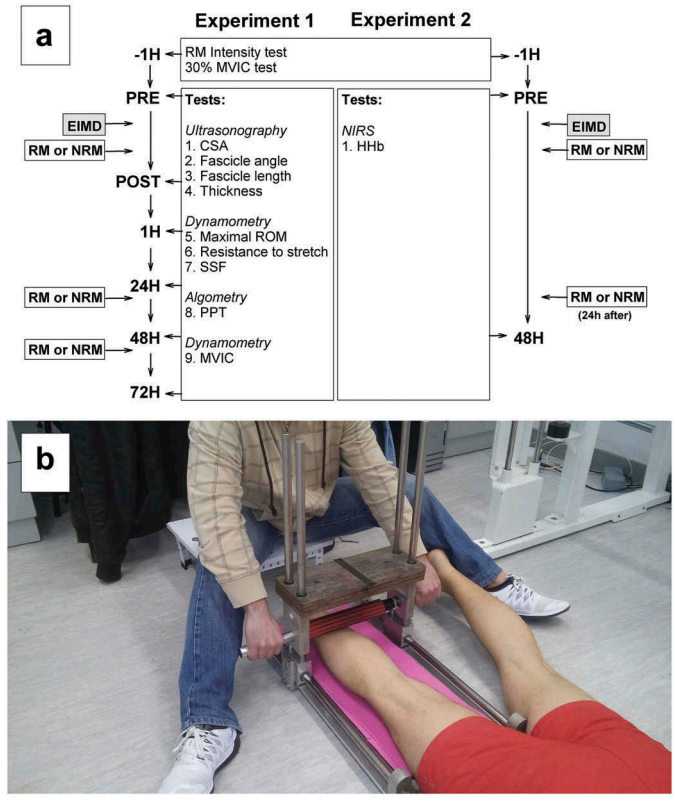

Figure 1. (a) Study design. (b) Roller massager intervention setup. The device that incorporated the roller massager ensured consistent pressure and frequency of rolling; eliminating the variations that would be typical if each participant applied the roller action to the limb.
CSA: cross-sectional area; EIMD: exercise-induced muscle damage; RM: roller massager; NIRS: near-infrared spectroscopy; PPT: pain pressure threshold; MVIC: maximal voluntary isometric contraction; ROM: range of motion; SSF: submaximal sustained force.

(i.e., maximal ROM and resistance to stretch), submaximal sustained force, medial gastrocnemius pain pressure threshold, and ankle plantar flexion MVIC. The medial gastrocnemius was selected to be examined among other plantar flexors muscles since is reported to be the muscle that most contributes to plantar flexors resistance to stretch (Taniguchi, Shinohara, Nozaki, & Katayose, 2015). All measurements were first performed in the experimental leg, followed by the control leg. In experiment 2, the muscular HHb response during a submaximal sustained force task was assessed before and 48 h after the EIMD. One hour before the first session of both experiments, 30% of plantar flexors MVIC (3 reps, 1-min rest between) and RM pressure intensity (set as the point of discomfort) were determined. EIMD stimulus consisted of 5 × 20 unilateral calf raises (1-min rest between sets), within the maximal active ROM, at a cadence of 2 s in both concentric and eccentric phases. A strength and condition professional supervised the exercise.

The RM intervention consisted of 6 × 45-s rolling bouts targeting the full gastrocnemius length (20-s rest between bouts) administrated by a strength and condition professional. Each roll lasted 2 s and was performed with a constant-pressure rolling device (Technical Services, Memorial University, St John's, Canada) that incorporated a Theraband® RM (Bradbury-Squires et al., 2015). Briefly, the RM consisted of a hard rubber

material (24-cm long and 1.4 cm in circumference), with low-amplitude, longitudinal grooves surrounding a plastic cylinder (Figure 1-b). Its rigid design allows for both superficial and deep tissue massage when it is rolled over the muscle. In 3 sets, the roller was centred over the medial gastrocnemius mid belly, and the remaining 3 sets over the lateral gastrocnemius. The RM was performed immediately after the EIMD stimulus in both experiments, and immediately after the second and third testing sessions (i.e., 24 and 48 h) in experiment 1; and 24 h after the EIMD stimulus in experiment 2.

Equipments and variables

Dynamometry

An isokinetic dynamometer (Biodex system 3 research, Shirley, NY, USA) was used to assess joint maximal ROM, resistance to stretch (i.e., passive torque), MVIC, and submaximal sustained force. In all testing, participants laid prone fixed by the ankle joint, hips, and upper back region to avoid any movement, with the lateral malleolus aligned to the dynamometer axis. Data were recorded at a sampling rate of 1000 Hz (MP100, BIOPAC, Goleta, California, USA). Maximal ROM was determined by moving the ankle at $2° \cdot s^{-1}$ from a plantar flexion position (30°) to point of discomfort, after performing 4 ankle conditioning cycles (i.e., until 80% of maximal ROM at $5° \cdot s^{-1}$). MVIC and submaximal sustained force tests were performed with the ankle at 0°. Participants produced maximal force for 2 s, in 3 plantar flexion and 1 dorsiflexion MVIC actions (60 s between repetitions); and produced isometric force at 30% of MVIC for 90 s in the submaximal sustained force test. Visual biofeedback was given during the strength tests in order to sustain the same level of force during submaximal sustained force test and for motivation purposes during the MVIC contractions.

Algometry

A digital dynamometer (1-cm^2 surface area; Lafayette, Model 01165) was used to assess pain pressure threshold. Four measurements were taken with constant loading ($1 kg \cdot s^{-1}$) (Ylinen, Nykänen, Kautiainen, & Häkkinen, 2007) at 60% of medial gastrocnemius length in 2 sites 1 cm apart, until the point when the sensation of pressure turned to pain. These sites were carefully measured, determined and marked in the first session in order to accurately use the same points during the next sessions.

Ultrasonography

An ultrasound scanner (EUB-7500; Hitachi Medical Corporation, Chiyoda-ku, Tokyo, Japan) was used in B-mode with a 6-cm 10-MHz linear probe to assess the medial gastrocnemius fascicle angle, fascicle length, cross-sectional area, and muscle thickness. Measurements were taken at 60% of medial gastrocnemius proximal-to-distal myotendinous junctions' length, with the participants laid prone and ankle fixed in neutral position (0°). For fascicle angle, fascicle length, and muscle thickness measurements, probe was placed according to fascicles orientation (Freitas, Andrade, Larcoupaille, Mil-Homens, & Nordez, 2015). Cross-sectional area assessment was performed using the extended-field-of-view setting, with slow and constant-velocity scans (Chan, Newton, & Nosaka, 2012). Three images were collected for each variable.

Near-infrared spectroscopy

A near-infrared reflectance spectroscopy equipment (NIMO, Nirox srl, Brescia, Italy) was used to estimate the muscle HHb response during submaximal sustained force test. Focus was given for the HHb concentration since its signal is less dependent of changes in blood flow, and thus can be used as an indicator of fractional O_2 extraction within the microvascular level (Ferrari et al., 1997). The probe was placed at 60% of medial gastrocnemius proximal-to-distal length and covered with a dense elastic tape. HHb measurements were corrected for the subcutaneous fat thickness assessed in sonograms. Before testing, participants rested for 2 min.

Electromyography

Surface electromyography (EMG) was performed to ensure passive condition during flexibility tests (i.e., EMG < 1% of MVIC). Electrodes were placed according to SENIAM guidelines over lateral gastrocnemius, soleus, and tibialis anterior muscles (Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). The EMG signals were acquired using a telemetric system (Plux, Lisbon, Portugal) according to the International Society of Electromyography and Kinesiology (ISEK) recommendations (Merletti, 2016).

Data processing

Mechanical (i.e., angle and torque) and EMG data were synchronised using a trigger, recorded using the BIOPAC MP100 Acquisition System (Santa Barbara, USA), and processed using MATLAB® v12.0 software (The Mathworks Inc., Natick, Massachusetts, USA). Torque was filtered [Butterworth second-order low-pass filter (8 Hz)] and corrected for gravity.

For MVIC testing, the highest value (i.e., the mean value from a 100-ms window where the midpoint was the highest peak) of 3 plantar flexion attempts was considered for analysis. For submaximal sustained force testing, the coefficient of variation [CV = standard deviation (SD)/mean × 100] of torque was calculated during the middle 80 s of contraction and used as an index of torque fluctuation (Kato, Vieillevoye, Balestra, Guissard, & Duchateau, 2011). For flexibility testing, as the maximal ROM changed across testing sessions, the resistance to stretch was determined as the passive torque at the highest common joint angle performed in the participant sessions. For algometry data, the average value of the 4 pain pressure threshold attempts of the 2 pressure sites was considered for analysis.

The medial gastrocnemius ultrasound variables (cross-sectional area, fascicle length, fascicle angle, muscle thickness) were digitised using ImageJ (version 1.48v, Wayne Rasband National Institutes of Health, USA). Digitising procedures are reported elsewhere (Freitas et al., 2015). Three fascicles were analysed in each image. The average value of each sonogram variable was considered for analysis. We previously observed (n = 13, unpublished data) a very high test–retest reliability for the ultrasound outcomes: cross-sectional area (r = 0.99, intraclass correlation coefficient ($ICC_{2,1}$) = 0.99, standard error of measurement (SEM) = 0.238 cm^2 (2.0%), fascicle angle (r = 0.99, ICC = 0.99, SEM = 0.07° (0.4%), fascicle length (r = 0.99, ICC = 0.99, SEM = 0.03 cm (0.7%), and muscle thickness (r = 0.99, ICC = 0.99, SEM = 0.01 cm (0.6%).

The medial gastrocnemius HHb data were normalised to resting values, and processed using IBM Statistics software (SPSS Inc., v20, Chicago, IL). The HHb response consists of a "time delay" (TD) at the onset of exercise, followed by an "exponential-like" HHb signal increase (DeLorey, Kowalchuk, & Paterson, 2003). The TD was determined using second-by-second data and corresponded to the time, after the onset of exercise, at which the HHb signal began the exponential-like increase (Barker, Jones, & Armstrong, 2010). The muscle HHb response was modelled using a mono exponential function (Equation 1), starting from the TD instant until the end of the contraction:

$$HHb(t) = HHb_{baseline} + A1\left(1 - exp^{-(t-TD)/\tau}\right) \quad (1)$$

where HHb (t) represents the HHb at a given time (t), $HHb_{baseline}$ represents the HHb at rest (i.e., average of HHb in the 2 min before the onset of exercise), and A1 and τ to the amplitude and time constant of the exponential HHb curve, respectively. The deoxygenation kinetics were then assessed by determining the "effective" time constant (τ'), which corresponded to the sum of TD and τ (Barker et al., 2010).

EMG signals were processed according to the ISEK recommendations [band-pass filtered (20–450 Hz), full-wave rectified and smoothed with a low-pass filter (8 Hz, 4th order Butterworth)] (Merletti, 2016), and normalised to maximal value obtained from the MVIC pre-testing.

Statistical analysis

All data were analysed using IBM SPSS Statistics 22.0 (IBM Corporation, New York, USA). Normal distribution was first confirmed using the Shapiro–Wilk test. *T*-tests were performed to ensure no differences exist between legs and pre-testing. A two-way ANOVA [condition (RM, NRM) × time (pre, post, 1 h, 24 h, 48 h, 72 h)] was performed on MVIC, submaximal sustained force, maximal ROM, resistance to stretch, cross-sectional area, fascicle angle, fascicle length, muscle thickness, and pain pressure threshold; and a two-way ANOVA [condition (RM, NRM) × time (pre, 48 h)] was performed on HHb A1 and τ', in both experimental and control legs. Post hoc analysis was performed using one-way repeated measures with contrast (simple set for the pre values) for time analysis, and *t*-test for condition analysis. The Eta Squared (η^2) was calculated from the repeated measures ANOVA output, and used to estimate the effect size between conditions (Levine & Hullett, 2002). The relation between the extent of force loss and degree of lower pain pressure tolerance at 24 h after EIMD was determined by the Pearson correlation coefficient. Significance was defined as $P \leq 0.05$. Data are presented as normalised (i.e., to baseline) values as mean ± SE, except for demographic and RM intensity variables (mean ± SD).

Results

Due to file corruption problems, ultrasound data of 1 participant were excluded from analysis. The average RM pressure intensity in both experiments was ~24.2% of body weight (16.81 ± 3.71 kg). No differences were found for all variables between pre-testing and between limbs of both conditions in both experiments ($P > 0.05$).

Table 1. *P*-values of the two-way ANOVA factors for the tested variables.

	Experimental			Control		
	C	T	C × T	C	T	C × T
Maximal voluntary isometric force	0.875	**0.004**	0.074	0.608	**<0.001**	0.665
Submaximal sustained force	0.21	**<0.001**	0.89	0.739	0.843	0.748
Maximal range of motion	0.72	**<0.001**	0.43	0.797	0.106	0.819
Resistance to stretch	0.331	**<0.001**	0.546	0.685	0.252	0.399
Cross-sectional area	0.593	**<0.001**	0.988	0.935	0.063	0.739
Muscle thickness	**0.025**	**0.003**	**0.023**	0.638	0.06	0.669
Fascicles length	0.374	0.312	0.599	0.781	0.122	0.685
Fascicles angle	0.859	**0.002**	0.58	0.781	0.072	0.685
Pain pressure threshold	0.082	**<0.001**	0.285	0.171	0.166	0.604
HHb A1	0.255	0.066	0.306	-	-	-
HHb τ'	0.075	0.075	0.382	-	-	-

C: condition; T: time; C × T: condition × time. *p*-Values below 0.05 are in bold.

P-values from ANOVA analysis for all variables tested are shown in Table 1.

The effects on plantar flexors functional variables (i.e., strength and flexibility) and medial gastrocnemius morphological variables of the experimental limb are presented in Figure 2. The experimental limb MVIC decreased within 1 h in both conditions [RM: −16% at post, and −14% at 1 h ($P = 0.02$); NRM: −15% at post ($P = 0.02$)], and increased in the contralateral limb in both conditions [RM: +6% at 1 h, +7% at 24 h, +12% at 48 h, and +19% at 72 h ($P = 0.01$–0.048); NRM: +10% at 1 h, +15% at 24 h, +10% at 48 h, and +11% at 72 h ($P = 0.03$–0.046)]. The experimental limb submaximal sustained force increased within 1 h in both conditions [RM: +324% at post, and +255% at 1 h ($P < 0.03$); NRM: +385% at post, and +276% at 1 h ($P < 0.005$)].

The experimental limb maximal ROM increased within 1 h in both conditions [RM: +20% at post, and +20% at 1 h ($P = 0.005$–0.04); NRM: +16 % at post, and +14% at 1 h ($P = 0.04$–0.05)]; and decreased in the consequent days [RM: −22% at 48 h, and −33% at 72 h ($P = 0.02$–0.05); NRM: −14% at 24 h, −24% at 48 h, and −25% at 72 h ($P = 0.002$–0.05)]. The resistance to stretch increased at all times in both conditions [RM: +26% at post, +31% at 1 h, +51% at 24 h, +50% at 48 h, and +50% at 72 h ($P = 0.001$–0.05); NRM: +25% at post, +18% 1 h, +28% at 24 h, +32% at 48 h, and +35% at 72 h ($P = 0.001$–0.01)]. A low muscle activation was observed during resistance to stretch measurements for all muscles (mean %EMG: lateral gastrocnemius = 0.35, soleus = 0.11, tibialis anterior = 0.37).

The medial gastrocnemius cross-sectional area increased in both conditions [RM: +11.4% at post, +9.4%% at 24 h, +8.1% at 48 h, and +8.2% at 72 h ($P < 0.03$); NRM: +13.3% at post, +4.8% at 1 h, +10% at 24 h, +10% at 48 h, and +9.2% at 72 h ($P < 0.02$)]. The fascicle angle increased in both conditions [RM: +14.9% ($d = 0.89$; $P < 0.001$) at post, +10.5% at 1 h, +11.8% at 24 h, +14.5% at 48 h, and +13.9% at 72 h ($P < 0.01$); NRM: +13.8% at post, +10.5% at 1 h, +14.3% at 24 h, +10.9% at 48 h, and +11.5% at 72 h ($P < 0.02$)]. No changes were observed in the fascicle length ($P > 0.05$). The muscle thickness increased in both conditions [RM: +6.1% at post, +1.9% at 1 h, +6% at 24 h, +6% at 48 h, and +7.1% at 72 h ($P < 0.05$); NRM: +13.2% at post, +6.5% at 1 h, +12.8% at 24 h, +9.6% at 48 h, and +10.1% at 72 h ($P < 0.01$)]; however, a significant effect was seen for the condition (Table 1).

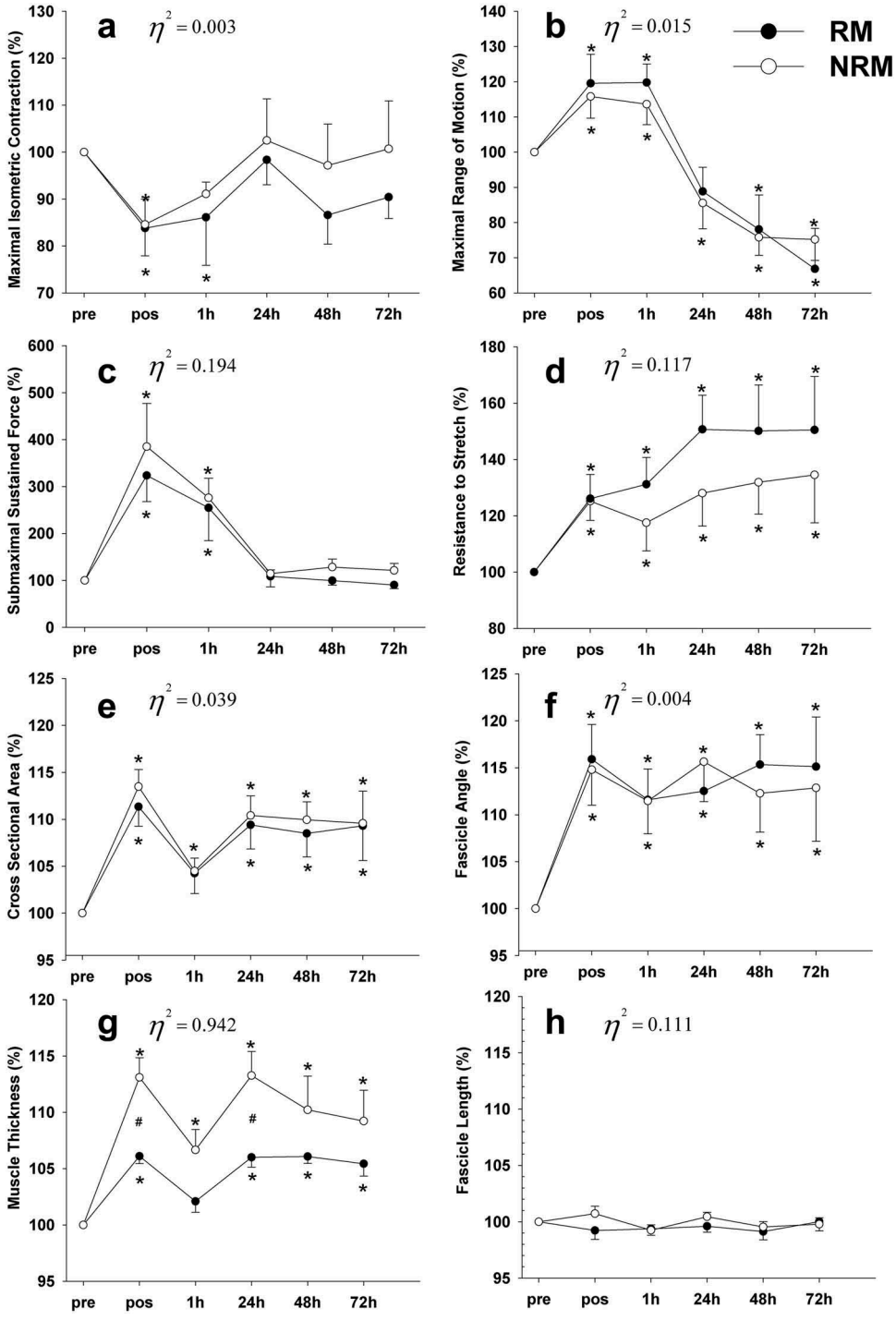

Figure 2. (a) Maximal isometric voluntary force, (b) submaximal (30%) sustained force, (c) maximal ankle dorsiflexion range of motion, (d) plantar flexors resistance to stretch, (e) Cross-sectional area, (f) muscle thickness, (g) fascicle angle, and (h) fascicle length of the medial gastrocnemius responses after exercise-induced muscle damage stimulus in both roller massager (RM) and no-roller massager (NRM) conditions. Values are normalised to baseline values, and presented as mean ± standard error. No differences were observed between conditions, except for muscle thickness.
η^2 : Eta Square for the condition.
*: statistically different from baseline ($P < 0.05$).
#: statistically different between groups ($P < 0.05$).

The effects on pain pressure threshold of both limbs are presented in Figure 3. A decreased pain pressure threshold was observed in both conditions in the consequent days [RM: −27% at 24 h, −24% at 48 h, and −28% at 72 h ($P < 0.02$); NRM: −40% at 24 h, −42% at 48 h, and −39% at 72 h ($P < 0.001$)].

A typical example of HHb response during the submaximal sustained force task is presented in Figure 4 (participant #1). No differences between conditions were observed in both τ' (mean ± SE; RM: pre = 28.56 ± 2.97, 48 h = 30.08 ± 2.17; NRM: pre = 29.14 ± 3.58, 48 h = 33.32 ± 3.26); and A1 (RM:

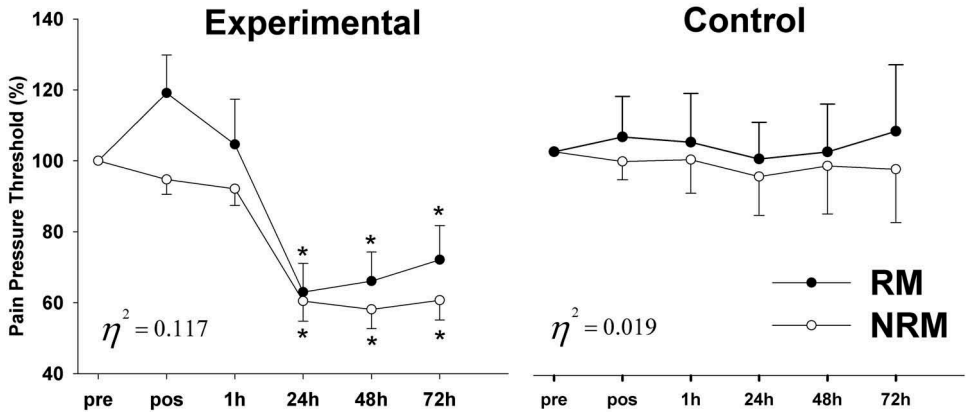

Figure 3. Pain pressure threshold of the medial gastrocnemius in the experimental and control limbs after exercise-induced muscle damage stimulus in both roller massager and no-roller massager conditions. Values are normalised to baseline values, and presented as mean ± standard error. Although, two-way (2 × 6) ANOVA did not revealed differences between protocols ($P = 0.082$), although a paired t-test revealed a statistical ($P = 0.032$) difference between RM (+19%) and NRM (−5%) interventions immediately after EIMD.
η^2 : Eta Square for the condition.
*: statistically different from baseline ($P < 0.05$).

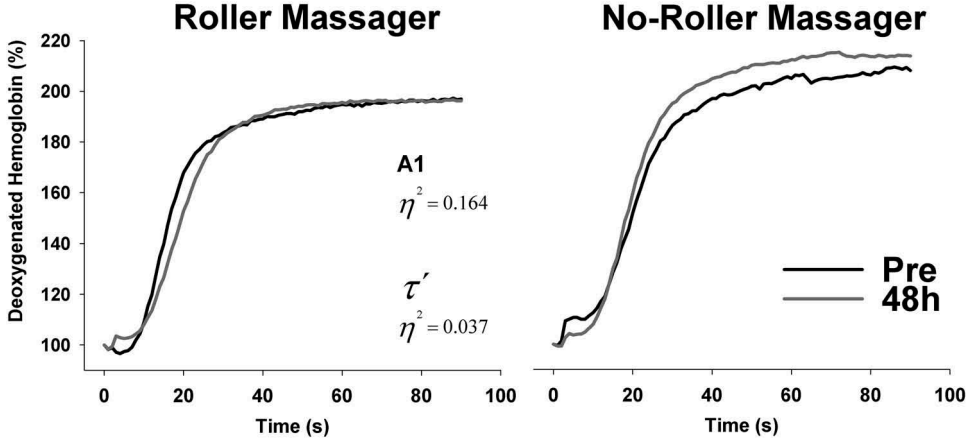

Figure 4. Deoxygenated haemoglobin response during the submaximal sustained force test after exercise-induced muscle damage stimulus in both roller massager (RM) and no-roller massager (NRM) conditions. Values are normalised to baseline values, and presented as mean ± standard error.
η^2 : Eta Square for the condition.

pre = 80.56 ± 6.95, 48 h = 96.76 ± 12.35; NRM: pre = 73.49 ± 7.92, 48 h = 77.97 ± 7.89).

A significant correlation was found between the extent of force loss and degree of lower pain pressure tolerance in both RM ($r^2 = 0.42$) and NRM ($r^2 = 0.47$) conditions at 24 h after EIMD.

Discussion

The present study investigated the effectiveness of RM in changing effects of EIMD in muscle functional, morphological and oxygenation variables. The main findings were: (1) RM did not reduce the negative effects on ankle plantar flexors functional and morphological variables induced by EIMD; (2) a tendency was observed for a RM ipsilateral and contralateral effect on pain pressure threshold immediately after the EIMD; and (3) RM did not change the muscular oxygenation response 48 h after EIMD stimulus.

In the present study, strength was assessed by MVIC, and submaximal sustained force at 30% of MVIC. Both variables were affected within 1 h after EIMD, and responses did not change with RM intervention. Results are in accordance with MacDonald et al. (2014). However, the reduced MVIC found in the present study was only observed within 1 h after the EIMD, whereas at 24 h no statistical difference was found. In addition, there was a RM contralateral effect on MVIC within 1 h and 72 h after EIMD. This result suggests that a learning effect occurred (despite participants being young athletes), since previous studies have reported that lower limbs MVIC impairment after EIMD occurs up to 7 days (Byrne, Christopher, & Roger, 2002). Meldrum, Cahalane, Keogan, and Hardiman (2003) suggested that a learning effect occurs in relatively new users of MVIC testing. Thus, extensive familiarisation is necessary. We consider this as a potential study limitation, since only 1 session was given for familiarisation purposes. Nonetheless, the present results suggest that RM is not effective at reducing plantar flexor MVIC impairment after EIMD.

We also observed a submaximal sustained force increase immediately and up to 1 h after EIMD in both conditions. Such observation is consistent with Lavender and Nosaka (2006). Submaximal sustained force measurements have been associated with intra- and intermuscular coordination ability. Specifically, a higher force fluctuation is an indicator of a greater variability within and

between muscles recruitment to sustain an isometric contraction. This ability relates to a better balance performance (Oshita, Kazushige, & Sumio, 2010). Due to the expected positive RM effect on soreness and fatigue markers (Cheatham et al., 2015; Schroeder & Best, 2015), it was hypothesised that RM could attenuate the submaximal sustained force increase after EIMD. No effects were observed in the present study, suggesting that RM is not effective in attenuating the acute submaximal sustained force increases after EIMD.

Previous studies have shown that RM can increase joint flexibility and attenuate the maximal ROM impairment induced by EIMD (Cheatham et al., 2015; Schroeder & Best, 2015). However, maximal ROM response did not differ between conditions. These findings contrast with MacDonald et al. (2014), who found significant benefits from foam rolling in knee flexion (at 48 and 72 h) and hip flexion (at 72 h) flexibility. The different results may be explained by the different joints targeted (i.e., ankle vs. knee/hip). In addition, previous studies have not measured the joint resistance to stretch (i.e., indicator of overall muscle–tendon group stiffness). Given the fact that RM has been shown to induce positive joint flexibility effects, it was expected that joint resistance to stretch could return faster to baseline, or have a lower response, since it is known that EIMD increases muscle stiffness (Lacourpaille et al., 2014). However, no significant effect was observed for the RM intervention. These results are in accordance with observations by Pournot, Tindel, Testa, Mathevon, and Lapole (2016), where local massage vibration did not benefit recovery of muscle stiffness.

As expected, EIMD produced a medial gastrocnemius swelling response (~8.6% cross-sectional area increase that lasted up to 72 h) (Cleak & Eston, 1992; Hart, Swanik, & Tierney, 2005; MacDonald et al., 2014). Such change was seen to be attributable to a ~15% fascicle angle increase, since no changes were observed in fascicle length. However, RM intervention did not change the swelling response. This result is in accordance to previous studies (Hart et al., 2005; MacDonald et al., 2014). Muscle thickness also increased (~6–12%) after EIMD; however, muscle thickness was significantly lower for RM immediately and 24 h after the EIMD, compared with the NRM condition. This finding contrasts to cross-sectional area response, suggesting twofold: (i) that swelling may not be homogeneous within the medial gastrocnemius muscle; or (ii) RM effects are not homogeneous along the medial gastrocnemius muscle. Future studies should examine these hypotheses.

As expected, pain pressure threshold had a similar response to Pearcey et al. (2015b) and Jay et al. (2014). Although ANOVA outcome showed a $P = 0.082$ for condition effect, a detailed analysis revealed a ~19% pain pressure threshold increase immediately after EIMD for RM intervention, versus a ~5% decrease for NRM intervention (paired *t*-test, $P = 0.032$), with a tendency ($P = 0.108$) to last up to 1 h after EIMD. The reason for this statistical discrepancy is related to the number of testing moments in the two-way ANOVA (2×6) test, that concealed the difference immediately after EIMD between protocols, as compared, for example, with a 2×2 ANOVA. Nonetheless, the result found in the present study suggests that RM increases the capacity to tolerate pain. In addition, Aboodarda et al. (2015) observed a ~15% pain pressure threshold increase of the control limb immediately after massage intervention. Although, there is no contralateral effect on pain pressure threshold at any time ($P = 0.171$), we observed a tendency (paired *t*-test, $P = 0.095$) for a higher (~9%) pain pressure threshold immediately after the RM intervention, compared with NRM (−6%). These results support the previous finding that RM induces contralateral effects on pain pressure threshold (Aboodarda et al., 2015). Assuming that massage modulates pain tolerance systemically, it is possible that massage affects the central pain modulatory systems. Massage-like mechanical pressure may provide analgesic effects on the muscle via the ascending pain inhibitory system (gate theory of pain) or the descending anti-nociceptive pathway (diffuse noxious inhibitory control) leading to a reduction in the sensation of pain following RM (Aboodarda et al., 2015). Furthermore, the massage-like mechanical stress from the RM may have removed or reduced trigger points (a source of musculoskeletal pain) which may also partially explain these results. However, future research is needed because the mechanism underlying this reduced pain effect is unknown.

In the present study, we observed a relationship between the extent of force loss and degree of lower pain pressure tolerance in both RM ($r^2 = 0.42$) and NRM ($r^2 = 0.47$) conditions at 24 h after EIMD. Physical performance impairment occurs in the presence of muscle soreness and massage seems to acutely increase pain tolerance. We speculate that physical performance may be less affected by EIMD if RM is applied immediately before (as a warm-up strategy), due to an increase in pain tolerance capacity. This may become a recovery strategy for training or sports competition purposes. Further research should examine this hypothesis.

The present study also tested the hypothesis that RM would increase muscle oxygenation into the damaged muscles after EIMD, promoting a faster recovery (Cheatham et al., 2015; Schroeder & Best, 2015). Our results did not show any effect in muscle O_2 extraction kinetics with the RM application. However, we did observe a tendency for a slower ($P = 0.075$) but higher amplitude ($P = 0.066$) HHb kinetics during a 90-s submaximal sustained force task 48 h after EIMD, which was similar to that reported by Davies et al. (2008), who showed similar response in HHb kinetics 48 h after EIMD.

In conclusion, the application of RM on gastrocnemius with constant pressure (~24% of body weight) did not seem to improve recovery after EIMD in the plantar flexors functional properties and gastrocnemius swelling and oxygenation response. However, roller massage might be useful to acutely increase pain pressure threshold within 1 h after EIMD. This suggests that roller massage may have a positive effect on recovery if applied immediately before a physical task in order to decrease muscle soreness and increase maximal capacity in physical tasks. Future research should examine this.

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