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ORIGINAL ARTICLE

Exercise-induced muscle damage on the contractile properties of the lumbar paraspinal muscles: a laser displacement mechanomyographic approach

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Abstract

Purpose This study investigated whether laser displacement mechanomyography (MMG) could detect acute injury of low back muscles following strenuous eccentric exercise.

Methods Sixteen healthy adults (10 females, 6 males, mean ± standard deviation, age 21 ± 2.90 years, BMI 21.63 ± 1.99 kg/m^2), without low back pain or low back resistance training, were recruited. Strength [maximum voluntary isometric contraction force (MVC)], pain intensity [visual analogue scale (VAS)], biological markers of muscle injury (serum myoglobin and creatine kinase levels), and MMG-derived muscle contractile properties were measured at seven different time points. Pre-exercise 'control' measures were taken prior to a strenuous eccentric exercise task, followed by an immediate post-exercise measurement and further four consecutive daily measurements. A final post-exercise measurement was completed on day 12 post-exercise.

Results Compared to pre-exercise control, MVC was lower immediately post-exercise (day 1) and on days 2–3. VAS scores were higher post-exercise (day 1) and from days 2–5. Myoglobin was significantly higher on day 4, whilst creatine kinase was significantly higher on days 4–5. MMG-derived maximum muscle displacement (D_{max}) was significantly diminished post-exercise (day 1) at all vertebral segments (L1-MT), while contraction velocity (V_c) was significantly slower at all segments except sacral multifidus. V_c recovered rapidly (by day 2), while mid-lumbar D_{max} resolved on day 12. D_{max} had moderate correlations with MVC ($R = 0.61$) and VAS ($R = -0.50$), and low correlations with myoglobin ($R = -0.36$).

Conclusion MMG appears capable of detecting changes in muscle contractile properties associated with an acute bout of low back pain.

Keywords Laser displacement mechanomyography (MMG) · Low back pain · Erector spinae · Multifidus · Acute injury · Spine

Abbreviations

CK	Creatine kinase
CV	Coefficient of variation
D_{max}	Maximal muscle belly displacement
ES	Erector spinae
LBP	Low back pain
LDS	Laser displacement sensor
MMG	Mechanomyography
MT	Multifidus
MVC	Maximum voluntary contraction
MYO	Myoglobin
SEM	Standard error mean
T_c	Contraction time
TNS	Transcutaneous neuromuscular stimulation
VAS	Visual analogue scale
V_c	Contraction velocity

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Introduction

Approximately 30% of athletes are expected to suffer from low back pain (LBP) throughout their careers (Trainor and Trainor 2004). This is, in part, due to the intensity of prolonged exercise regimes that place high mechanical stress and strain on the musculoskeletal system (Fett et al. 2017). Eccentric exercise, in particular, results in muscle soreness and stiffness, structural damage to soft tissues (microtrauma), a decline in muscle strength and range of joint motion, and neuromuscular dysfunction with more intense and prolonged muscle fatigue occurring compared with concentric and isometric exercise (Clarkson and Newham 1995). These detrimental responses to eccentric exercise can cause irregular loading of hard and soft tissues (Clarkson and Newham 1995; Cheung et al. 2003). Altered loading of these structures can potentially cause abnormal mechanics of the spinal column, which, in turn, activate nociceptive sensors in the low back for the emergence of LBP (Roy et al. 1989; Panjabi 2006). Sports with repetitive loading of the spine have been shown to have higher incidences of LBP (Sward et al. 1990; Belavý et al. 2016). In particular for eccentric movements, Howell (1984) found a positive correlation between hyperflexion motions of the lumbar spine (i.e., excessive lengthening of paraspinal musculature) and the incidence of LBP in female rowers. Thus, prolonged eccentric exercise can lead to LBP, commonly affecting structures including nerve roots, muscle, fascial structures, bones, joints, and intervertebral discs (Allegri et al. 2016).

Whilst many cases of LBP are considered non-specific and, hence, not associated with specific injury sites, if an injury site is located, then most patients will have evidence of mechanical disturbances to soft and/or hard tissues (Golob and Wipf 2014). These injuries directly cause atrophy of spinal muscles in the immediate vicinity of the injury site (Hides et al. 1994; Hodges et al. 2006; Beneck and Kulig 2012; Wan et al. 2015). Given that an emerging technique known as mechanomyography (MMG) has previously been shown to detect muscle atrophy in limb muscles (Pisot et al. 2008; Than et al. 2016), it is thought probable that MMG might also detect localised atrophy in the muscle tissues directly surrounding spinal injury sites through the abnormal contractile properties of atrophied muscle tissues. Therefore, MMG might provide clinicians with a non-invasive and inexpensive means of identifying injury sites in anatomically related LBP.

Identifying injury sites within the clinic would bypass delayed and costly diagnostic imaging, allowing faster implementation of rehabilitative programs for both athletes and the general populace. The translation of this to rehabilitative settings would subsequently allow clinicians to have targeted sites to monitor with MMG to determine the success of treatments through changes in contractile properties. By monitoring these specific sites of injury, clinicians would have informative data to assist decision-making in allowing patients to return to competition or the workforce. However, before assessing its clinical applications, the capacity of MMG to detect acute injury to the low back muscles in a laboratory setting requires investigation.

Therefore, the primary aim of this study was to investigate the utility of the MMG technique to detect changes in lumbosacral muscle contractile properties consistent with acute experimentally induced muscle injury. It was hypothesized that MMG-derived contractile properties of low back muscles, following a strenuous eccentric exercise protocol, would reflect the time-course of eccentric injury and muscle recovery.

Methods

Ethical approval for this study was obtained from the University of Queensland Human Research Ethics Committee A (HREC) (no. 2016001752). Informed written consent was obtained from each subject. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Healthy female ($n = 10$) and male university students ($n = 6$) [mean ± standard deviation, age 21 ± 2.90 years, body mass index (BMI) 21.63 ± 1.99 kg/m^2] were recruited for the 2-week exercise and recovery study (Fig. 1). Inclusion criteria consisted of being less than 30 years of age, no history of lower back resistance training and no history of LBP. No history of LBP was defined as no previously clinically diagnosed occurrence of pain in the lumbar spine. All measures were taken approximately 24 h apart, except for pre- and post-exercise being taken immediately before and after exercise, respectively. All testing procedures were conducted by the authors, except for blood collection which was conducted by a qualified professional phlebotomist.

Exercise protocol

The exercise protocol involved participants performing nine sets of body-weighted maximal repetitions in a 45° extension/flexion movement with 90 s rest between sets. A trunk flexion/extension phase tempo of 4:1 s was selected in accordance with Trost et al. (2011) to accentuate the eccentric phase (flexion) of the movement. In accordance with Larsen et al. (2017), participant arms were crossed in front of the chest whilst performing unsupported flexion

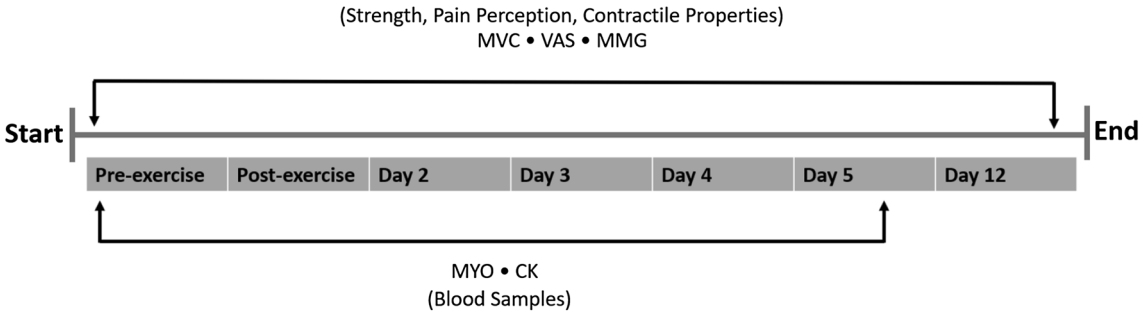

Fig. 1 Timeline of study. Strength, pain perception, and MMG-derived contractile properties were taken at all time points. Final measurements for blood sample collection were on day 5. *CK* creatine kinase, *MMG* mechanomyography, *MVC* maximum voluntary contraction force, *MYO* myoglobin, *VAS* visual analogue scale

Fig. 2 Exercise protocol using a 45° hyperextension bench. **a** Starting position before beginning the eccentric phase, **b** end position of the eccentric phase before beginning the concentric phase, and **c** manual assistance delivered by researcher during the concentric phase in return to starting position. A trunk flexion/extension phase tempo of 4:1 s was employed

(eccentric back extensor muscle activation), with manually assisted extension by the researchers (Fig. 2). The end of each set was determined when participants were no longer able to maintain tempo.

Laser displacement mechanomyography (LDS MMG)

Ten lumbar facet joints bilaterally from L1 to S1 (for erector spinae), as well as two bilateral sites over the sacrum (for multifidus), were located via palpation and ultrasound using a 7.5 MHz linear transducer probe (Mindray DP-50, Shenzhen, China). The spinous and transverse processes of each vertebrae and posterior superior iliac spine of the ilium were used as bony landmarks. The facet joints were chosen as a focus of the study due to their increasing implication in LBP (Amirdelfan et al. 2014). Participants were seated in a modified testing chair with their chest against the backrest. Straps were additionally fixed around the upper thorax to prevent unilateral spinal flexion during electrical stimulation (Fig. 3). MMG was recorded using a laser displacement sensor (LDS) (class 2 laser; model LG10A65PU) placed over the lumbar erector spinae (ES) and sacral multifidus (MT) overlying the 12 low back sites for MMG measurement. The output voltage of the laser was 0–10 V DC with a response distance of 75–125 mm. The beam dimensions on the skin

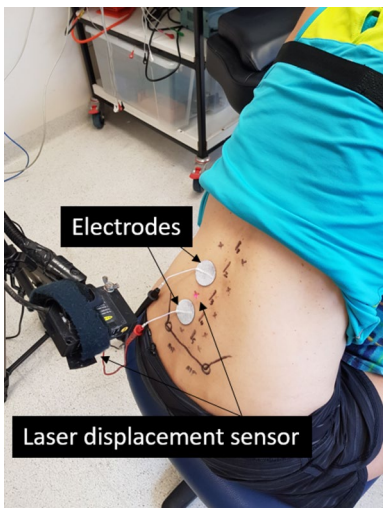

Fig. 3 LDS MMG testing position. Participants were seated in a specialised testing chair, whilst a strap was placed around the upper thorax for stabilisation. Ten lumbar facet joints and two bilateral sites over the sacrum were outlined for testing to target the lumbar erector spinae and sacral multifidus. Stimulatory electrodes delivered an electrical stimulus to cause involuntary contraction (twitch response) of the muscle. The LDS was mounted onto a modified adjustable camera tripod with a mic holder. Velcro straps used to secure the sensor to the mic holder. *LDS* laser displacement sensor, *MMG* mechanomyography

surface had a width of 0.8 mm and a height of 1.1 mm at an approximate distance of 100 mm. The resolution of the laser was approximately 90 µm on a 'fast'-response speed setting. The bandwidth was 50 Hz with a sensing beam wavelength of 650 nm.

The test–retest reliability of LDS MMG in this position has been demonstrated as clinically reliable (Than et al. 2018). The LDS was moved sequentially from proximal to distal, left side first before right, for testing. LDS MMG was selected for its accuracy, reliability, absence of manual post-processing or filtering following data collection, and general ease of use compared to other sensors (Seidl et al. 2017). Compared with contact sensors, Seidl et al. (2017) state the LDS to have greater sensitivity to temporal MMG parameters due to its noncontact nature. However, application of the LDS is, thus, dependent on controlled environments to restrict muscle movement for valid data collection.

To stimulate muscle segments at each site, two stimulatory electrodes (NeuroTrac TENS self-adhesive electrodes; 30 mm diameter) were placed on the skin in a vertical orientation overlying the facet joint (50 mm inter-electrode distance). To measure radial muscle belly displacement following transcutaneous neuromuscular stimulation, the LDS was positioned approximately 100 mm perpendicular to the muscle belly midway between the two stimulatory electrodes. Both electrodes and the LDS MMG were repositioned each time for every site tested. Muscles were then maximally stimulated following a transcutaneous twitch stimulus. Electrical square-wave stimuli of increasing current (mA) were delivered whilst maintaining a constant voltage (400 V) and duration (200 µs) (Digitimer DS7AH) until a maximum muscle contraction was observed without distortion of the sinusoidal MMG waveform (Tosovic et al. 2016). The starting stimulus was 30 mA, with subsequent stimuli increasing by 20 mA each time. The stimulus range for reaching each subject's maximum was 200–250 mA. Thirty second rest intervals between stimuli minimized fatigue from overstimulation.

MMG contractile properties were calculated as mean values from five recordings of maximal MMG waveforms. MMG waveforms were recorded in LabChart® 7 (ADInstruments), and analysis performed with the Peak Analysis module within the software. Waveforms from the LDS MMG were connected to an ADInstruments data acquisition system (16/30 channel PowerLab system, ADInstruments, ML880). A band-pass filter range of 1 Hz–1 kHz was applied for filtering and amplification with digitisation at a rate of 1000 samples s^{-1}. A 9 Hz low-pass digital filter was applied in the recording of the waveforms (Seidl et al. 2017). Two parameters were investigated from MMG waveforms for this study. Maximal muscle belly displacement (D_{max}) was calculated as the 100% peak height of the waveform (mm). Contraction time (T_c), calculated as the time interval (ms) between 10 and 90% of the peak height on the ascending slope, was not directly investigated within this study, but was required for contraction velocity (V_c) calculations. This is due to V_c being derived from D_{max} divided by T_c (mm/ms), referring to the ascending slope of the MMG waveform. Calculations of these variables from obtained MMG waveforms and their inferred physiology have been described by Than et al. (2018).

Maximum voluntary isometric contraction force (MVC)

Measures of low back muscle maximum voluntary contraction force (MVC) were performed in accordance with Essendrop et al. (2001) and Hjortskov et al. (2005). The selected methodology was found to have highly reproducible Pearson's and intra-class correlations (ICC) with R values above 0.9 for between day measures (Essendrop et al. 2001). Subjects were placed in an upright standing position with straps, fastened underneath the axilla, horizontally connected to a crane strain-gauge dynamometer fixed to the wall at the same height. The pelvis was pressed against a brace at the height of the iliac crest. Both the straps and pelvic brace were adjusted to suit individuals (Fig. 4). Participants were instructed to achieve maximum force within 3 s, with trials being performed three times. 90 s were allocated between trials, with the highest MVC (kg) selected for the respective session.

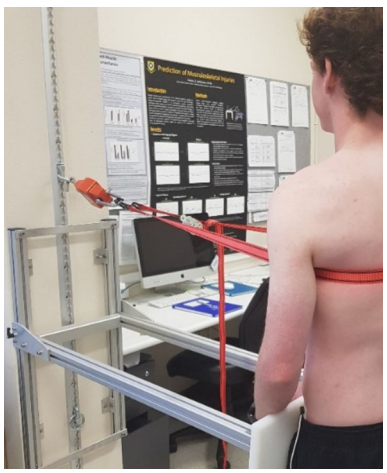

Fig. 4 Maximum voluntary contraction force (MVC). Straps, fastened below the axilla, were horizontally connected to an adjustable crane strain-gauge dynamometer that was fixated to the wall. An adjustable pelvic brace was set at the height of the iliac crest. Participants performed three trials of back extensions in this position, with 90 s allocated between trials

Myoglobin and creatine kinase analysis

Each participant had six blood samples collected for biological markers in accordance with Fig. 1. Approximately 6 mL of blood was collected from an antecubital vein by standard venepuncture techniques into a serum separation tube. Tubes were left at room temperature to clot for 30 min, before centrifugation at 1000×*g* for 10 min at 4 °C. Subsequently, serum was aliquoted and stored at − 80 °C until analysis. Myoglobin concentration and CK activity were measured on automated clinical analysers (e411, Roche Diagnostics; myoglobin, Cobas Mira, Roche; CK) and commercially available kits (Roche Diagnostics, myoglobin; and CDT14010, Thermo Fisher, CK). The coefficient of variation (CV) for myoglobin and CK was 1.95 ± 1.55% and 1.20 ± 1.72%, respectively. Myoglobin and CK were measured in duplicate to obtain these CV values.

VAS scores

The level of muscle pain and stiffness was quantified using a 100 mm visual analogue scale (VAS) in which 0 indicated "no pain or stiffness" and 100 represented "extreme pain and stiffness" (Lau et al. 2013). VAS scores were obtained before all the other measures each day, except for post-exercise which was taken immediately after exercise. Scores were simplified to out of 10.

Statistical analysis

Sample size considerations were based on 'a priori: sample size' calculations in G*Power 3.1.9.2 (University of Düsseldorf, Düsseldorf, Germany) with the statistical test chosen as 'ANOVA: Repeated measures, within factors'. Effect size *f* was set at 0.35, α error probability at 0.05, intended power at 0.80, number of groups 1, number of measurements at 7 (i.e., pre-exercise to day 12), correlations among repeated measures at 0.5, and nonsphericity correction *E* at 1.

For measures of MVC, VAS, and biological markers, a repeated-measures one-way ANOVA was performed in GraphPad Prism 7 (GraphPad Software, Inc., La Jolla, California, USA) with Bonferroni's post hoc. For each MMG parameter, linear mixed model regressions were used to examine the effect of time (× seven time points), side (× two for left/right), and segment (× six segments) using Stata 13.1 (StataCorp, Texas, USA). Time, side, and segment were entered as fixed factors and individuals as random intercept. Bonferroni's post hoc tests were used to locate significance between sides, and time points, for each segment and correct for multiple comparisons. A mixed-effects model, instead of a traditional two-way ANOVA, was chosen for MMG measures to account for correlation between repeated measures and reduce type I error rates (Gueorguieva and Krystal 2004).

Pearson's *R* correlation values were calculated in SPSS 25 (IBM Corporation IBM, Armonk, New York, USA). For MVC, VAS, and biological markers, this consisted of values across all seven time points being pooled (six time points for blood markers). For MMG variables, this consisted of the L3/L4 segment (left and right side averaged), across all seven times points, being pooled. The L3/L4 segment was selected due to its approximate centre of the lumbosacral spine where the greatest loading would occur (Harrison et al. 2005). An additional stepwise linear regression analysis was performed in SPSS where MMG (i.e., D_{max} or V_c) was considered the dependent variable and MVC, VAS, myoglobin, and creatine kinase were possible predictors. This also was done with a pooled data set across all seven time points (six time points for blood markers). MMG was also of pooled data at the L3/L4 segment (left and right side averaged), across all seven times points, for the stepwise regression. Significance was accepted as $p < 0.05$. MMG results are presented as percentages of day 1 pre-exercise (i.e., respective time point/pre-exercise baseline). All data are presented as mean ± standard error of the mean.

Results

Sample size power analysis

For 'a priori: sample size' calculations, a total *n* of 10 were required to achieve the intended power of 0.80 for within subject analysis.

Strength, pain perception and blood physiology

MVC was significantly reduced post-exercise ($p < 0.0001$) and on days 2–3 ($p = 0.0006$, $p < 0.0001$) compared to pre-exercise (Fig. 5a). VAS scores were significantly higher post-exercise and on days 2–5 compared to pre-exercise ($p < 0.0001$, $p < 0.0001$, $p < 0.0001$, $p = 0.0004$, $p = 0.0237$) (Fig. 5b). Myoglobin was significantly higher on day 4 against pre-exercise baseline ($p = 0.0208$) (Fig. 5c), whilst creatine kinase was significantly higher on days 4–5 ($p = 0.0026$, $p = 0.0005$) (Fig. 5d). Supplemental Table 1 details the statistical results of the ANOVA.

Mechanomyography

Time

D_{max} (Table 1) was significantly lower post-exercise for all vertebral levels ($p < 0.0001$ for all levels). Day 2

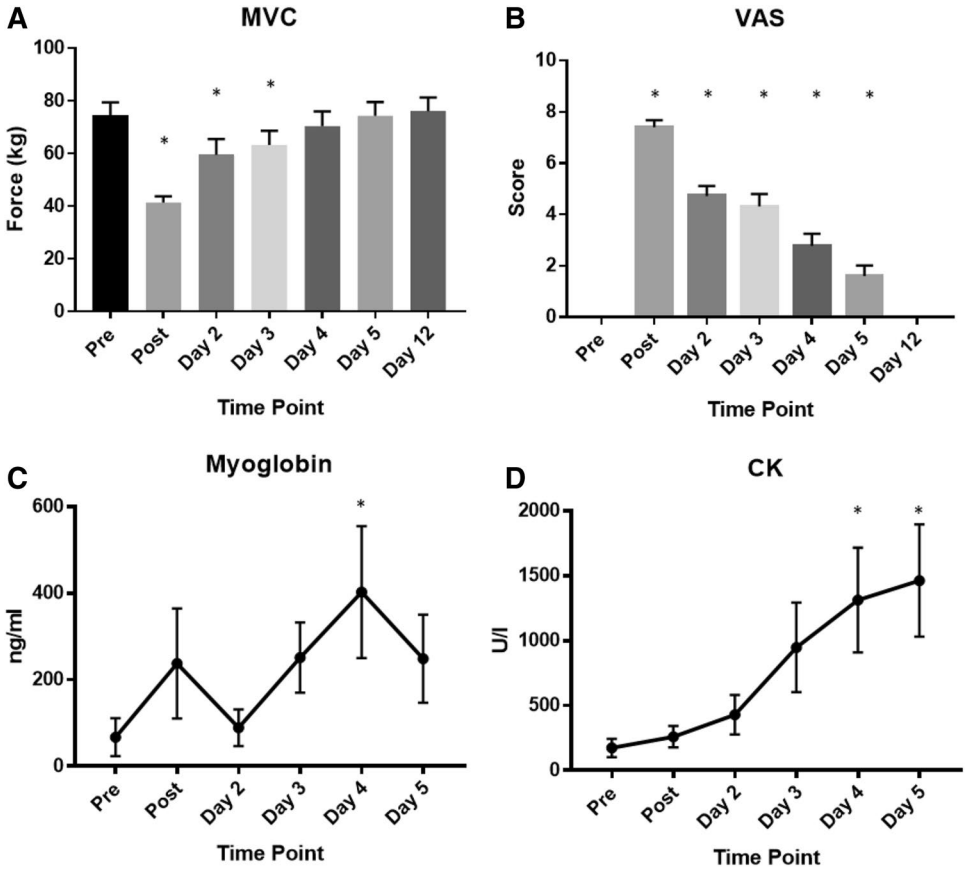

Fig. 5 Measures of strength, pain perception, and blood physiology. **a** MVC, **b** VAS score intensity of pain and stiffness in the low back, **c** myoglobin within blood plasma, and **d** CK within blood plasma. Asterisk indicates significant difference from baseline ($p < 0.05$). Errors bars indicate SEM. *CK* creatine kinase, *MVC* maximum voluntary contraction, *SEM* standard error mean, *VAS* visual analogue scale

Table 1 D_{max} averages as a percentage of 100% pre-exercise baseline

Left	Post	Day 2	Day 3	Day 4	Day 5	Day 12
L1/L2	**45.09 ± 5.57***	**88.70 ± 7.02***	91.56 ± 7.17	**86.12 ± 7.14***	90.16 ± 7.64	91.58 ± 6.18
L2/L3	**40.92 ± 4.23***	**86.82 ± 7.00***	**88.29 ± 5.92***	91.56 ± 6.60	93.28 ± 7.18	100.21 ± 6.55
L3/L4	**37.24 ± 4.63***	**84.54 ± 6.11***	**85.73 ± 5.60***	**88.17 ± 6.42***	**87.96 ± 6.98***	95.74 ± 6.51
L4/L5	**34.86 ± 4.80***	**77.79 ± 7.02***	84.61 ± 6.40	**84.93 ± 7.04***	**88.76 ± 10.21***	105.53 ± 12.26
L5/S1	**38.43 ± 4.09***	75.02 ± 5.85	76.16 ± 5.71	85.12 ± 8.47	87.80 ± 10.03	94.56 ± 9.51
MT	**50.29 ± 5.51***	79.53 ± 6.37	78.98 ± 7.24	81.20 ± 8.43	86.38 ± 8.98	109.65 ± 10.47
Right	Post	Day 2	Day 3	Day 4	Day 5	Day 12
L1/L2	**50.13 ± 5.36***	**86.85 ± 5.28***	90.17 ± 5.02	**87.59 ± 5.73***	88.89 ± 6.32	97.79 ± 6.48
L2/L3	**47.56 ± 4.81***	**84.40 ± 5.60***	**85.72 ± 4.75***	88.48 ± 5.83	89.03 ± 6.58	99.76 ± 5.65
L3/L4	**44.32 ± 5.15***	**80.82 ± 5.14***	**86.02 ± 4.25***	**85.03 ± 6.44***	**86.04 ± 7.65***	99.81 ± 6.65
L4/L5	**41.38 ± 4.77***	**81.18 ± 7.47***	85.24 ± 5.91	**88.495 ± 10.71***	**91.27 ± 12.64***	101.34 ± 8.51
L5/S1	**46.58 ± 5.56***	83.91 ± 10.62	77.16 ± 7.40	84.13 ± 8.91	87.89 ± 13.01	94.14 ± 8.93
MT	**57.97 ± 7.60***	79.58 ± 7.95	80.63 ± 7.66	86.70 ± 9.83	79.41 ± 8.83	101.73 ± 11.24

D_{max} calculated as the 100% peak height of the sinusoidal MMG waveform (mm). Averages are accompanied by SEM

D_{max} maximal muscle displacement, *MT* multifidus, *SEM* standard error mean

Significance against pre-exercise baseline indicated by * and bold text ($p < 0.05$)

showed lower D_{max} values for L1/L2 to L4/L5 ($p = 0.031$, $p = 0.001$, $p = 0.001$, $p = 0.001$). Day 3 showed lower D_{max} values for L2/L3 and L3/L4 only ($p = 0.018$, $p = 0.022$). Day 4 showed lower D_{max} for L1/L2, L3/L4 and L4/L5 ($p = 0.024$, $p = 0.023$, $p = 0.029$). Day 5 showed lower D_{max} for L3/L4 and L4/L5 ($p = 0.007$, $p = 0.038$). V_c (Table 2)

Table 2 V_c averages as a percentage of 100% pre-exercise baseline

	Post	Day 2	Day 3	Day 4	Day 5	Day 12
Left						
L1/L2	**54.95 ± 5.00***	88.14 ± 5.75	94.04 ± 5.96	88.39 ± 6.58	88.66 ± 6.80	92.00 ± 5.83
L2/L3	**53.75 ± 4.18***	87.97 ± 6.56	96.22 ± 6.85	94.31 ± 6.38	93.52 ± 6.47	101.27 ± 6.50
L3/L4	**51.0 ± 5.31***	89.82 ± 4.68	93.65 ± 4.60	94.01 ± 5.20	88.93 ± 5.05	95.78 ± 4.50
L4/L5	**44.83 ± 5.58***	85.48 ± 6.40	95.01 ± 6.42	96.28 ± 6.59	91.88 ± 8.99	107.47 ± 12.79
L5/S1	**52.19 ± 4.61***	78.87 ± 5.54	84.55 ± 6.70	96.34 ± 10.24	100.07 ± 9.40	93.73 ± 10.01
MT	69.89 ± 8.07	90.32 ± 6.96	94.75 ± 8.59	92.08 ± 8.836	94.72 ± 9.073	110.24 ± 10.29
Right						
L1/L2	**65.96 ± 6.35***	91.57 ± 4.71	101.09 ± 4.43	95.52 ± 5.700	94.72 ± 5.88	98.15 ± 7.32
L2/L3	**61.19 ± 5.47***	93.82 ± 5.29	91.32 ± 5.16	96.91 ± 6.21	92.68 ± 6.36	99.83 ± 5.91
L3/L4	**61.81 ± 6.43***	86.78 ± 4.35	96.84 ± 4.05	92.56 ± 5.70	87.28 ± 6.56	102.75 ± 6.44
L4/L5	**54.78 ± 5.30***	91.06 ± 6.65	96.00 ± 6.69	94.23 ± 8.62	94.63 ± 11.04	98.59 ± 6.94
L5/S1	**57.37 ± 4.08***	83.52 ± 5.77	81.06 ± 5.17	89.90 ± 9.75	92.23 ± 10.35	94.13 ± 8.80
MT	72.20 ± 7.68	86.16 ± 5.93	89.94 ± 5.67	92.27 ± 9.51	81.92 ± 7.91	102.53 ± 11.91

V_c calculated as the ascending slope of the sinusoidal MMG waveform. Averages are accompanied by SEM

Significance against pre-exercise baseline indicated by * and bold text ($p < 0.05$)

MT multifidus, *SEM* standard error mean, V_c velocity of contraction

was significantly slower post-exercise for L1/L2 to L5/S1 ($p < 0.0001$ for stated levels). Supplemental Table 2 details the statistical results of the linear mixed model regressions.

Side

Prior to testing, right-side dominance was self-reported by 15 of the 16 participants. Post-exercise was the only time point to show a significant difference between overall sides for D_{max} ($p = 0.03$) and V_c ($p = 0.04$) (Supplemental Table 3). For D_{max}, the left side decreased by 38.07 mm (i.e., all left-sided segments combined), whilst the right decreased by 30.71 mm (i.e., all right-sided segments combined), causing a total difference of 7.36 mm between sides (Supplemental Table 4). For V_c, the left side slowed by a total of 158.59 mm/s, whilst the right slowed by 117.01 mm/s, causing a difference of 41.58 mm/s between sides (Supplemental Table 5).

Pearson's *R* correlations and stepwise linear regression

Correlations (Table 3) were denoted as low (0.30–0.49), moderate (0.50–0.69), and high (0.70–0.90) (Mukaka 2012). For within L3/L4 segment MMG correlations, D_{max} had high correlations with V_c ($p < 0.0001$). In terms of comparison to the other measures of damage, MVC had a moderate correlation to D_{max} ($p < 0.0001$) and V_c ($p < 0.0001$). VAS scores had a moderate negative correlation to D_{max} ($p < 0.0001$). Myoglobin had negatively low correlations to D_{max} ($p = 0.0004$).

For the stepwise linear regression (Table 4), D_{max} had MVC, VAS, and myoglobin added as variables by the analysis, producing an R^2 value of 0.52 when all predictors were

Table 3 Correlation matrix

	D_{max}	V_c	MVC	VAS	Myo	Ck
D_{max}		**0.85***	**0.61***	**−0.50***	**−0.36***	0.06
V_c	**0.85***		**0.60***	**−0.29***	**−0.27***	0.04
MVC	**0.61***	**0.60***		**−0.49***	−0.19	0.07
VAS	**−0.50***	**−0.29***	**−0.49***		0.14	−0.11
Myo	**−0.36***	**−0.27***	−0.19	0.14		**0.45***
Ck	0.06	0.04	0.07	−0.11	**0.45***	

Pearson's *R* correlation values. Correlated MMG values were of an average between left and right sides at the L3/L4 segment. Correlations were between pooled values for all time points of the study (i.e., pre-exercise to day 12)

Ck creatine kinase, D_{max} maximal muscle displacement, *MVC* maximum voluntary contraction, *Myo* myoglobin, *VAS* visual analogue scale, V_c velocity of contraction

Significant correlations indicated by * and bold text ($p < 0.05$)

Table 4 Stepwise regression analysis

Model	R	R^2	Adjusted R^2	Std. error of the estimate
D_{max}				
1	0.64[a]	0.41	0.40	3.12
2	0.68[b]	0.47	0.46	2.97
3	0.72[c]	0.52	0.50	2.85
V_c				
1	0.60[d]	0.37	0.36	19.08

D_{max} maximum muscle displacement, *Std.* standard, V_c velocity of contraction

[a]Predictors: (constant), MVC

[b]Predictors: (constant), MVC, VAS

[c]Predictors: (constant), MVC, VAS, myoglobin

[d]Predictors: (constant), MVC

introduced. V_c only had MVC added with an R^2 value of 0.37.

Discussion

The aim of this study was to investigate whether the MMG technique could identify the onset, time-course, and recovery of acute injury (eccentric muscle fatigue) in the segmental low back muscles through temporal changes in contractile properties of muscle segments at any of 12 low back sites. As seen from the results, this study was able to show that MMG measures of muscle contractile properties do reflect the temporal onset and recovery of acute back muscle injury as measured by changes in back strength (MVC), the participant's perception of pain (VAS scores), and biological markers of muscle damage (myoglobin and creatine kinase).

Within this study, the time-course for strength deficits (MVC) (Cheung et al. 2003; Bishop et al. 2011), elevated pain perception (VAS) (Lau et al. 2013), and blood-borne markers (myoglobin and creatine kinase) (Sayers and Clarkson 2003; Kanda et al. 2013) all paralleled the previous literature on eccentric exercise-induced muscle damage. The delay between MVC decline and appearance of biological damage markers seen here has been described by Lee and Clarkson (2003), who describe an inflammatory response, following mechanical damage, for the late appearance (3–5 days after exercise) of myoglobin and creatine kinase in the blood.

Post-exercise, the left side of the lumbosacral spine was found to have a significantly larger decrease in MMG-derived D_{max} and V_c than the right side. As 15 of the 16 investigated participants reported right-side dominance, it appears that the non-dominant (left) lumbosacral muscles experienced greater fatigue than the dominant (right) from the exercise protocol. This finding is supported by Farina et al. (2003), who show, in upper trapezius musculature, that the non-dominant side is more fatigable than the dominant. Farina et al. (2003) attribute this as long-term preferential use of one side, with respect to the other, leading to changes in muscle fibre membrane and control properties for fatigue resistance. Williams et al. (2002) would further this by suggesting preferential use of the dominant side to lead to more fatigue resistance in certain structures/mechanisms of the neuromuscular system.

Further investigating the MMG variables, D_{max} was significantly diminished immediately post-exercise for all the muscle segments (Table 1). Each segment recovered on particular days with complete resolution in all the muscle segments by day 12. The L3/L4 vertebral level specifically showed consistently lower values through to day 5. As the L3/L4 vertebral level is at approximately the centre of the lumbosacral spine, there may have been an increased stress load placed upon the segment during the exercise task. This contention is supported by Harrison et al. (2005) who show increased compressive and shear loads at the L3/L4 level (as well as L5/S1) following anterior translation of the thoracic spine in the upright posture. Physiologically, reductions in D_{max} after muscle damage have been attributed to excitation–contraction uncoupling, increase in muscle tone or stiffness, and possible swelling of musculature dampening the MMG response (Hunter et al. 2012; Macgregor et al. 2016). Changes in recruitment patterns or changes in the temporal sequencing of muscle activation patterns after injury to muscular or connective tissue (Cheung et al. 2003) could also be a cause for diminished spatial displacement within this study.

The velocity (V_c) of the actin–myosin cross-bridging was found to be significantly slowed post-exercise. Sacral MT was the only segment to deviate from this in that it displayed no significant effect of exercise for V_c (Table 2). As the function of the MT is in providing segmental stabilisation (Dickx et al. 2010), requiring a consistent V_c in response to diminished D_{max} may be a necessity to maintain performance. The recovery of V_c by day 2, despite continued deficits in the other measures, could be explained by fibre typing. Literature has shown a high type I slow-twitch fatigue resistant fibre composition (62–65%) of the lumbosacral spine (Mannion 1999). Type I fibres have a higher mitochondrial content and a greater oxidative enzyme compliment, allowing for greater fatigue resistance (Mannion 1999). Thus, type I fibres are metabolically more capable of coping with repetitive high tension stresses (Friden and Lieber 1992). This would translate into the rapid recovery of V_c against other measures. However, this contention would have required subject biopsies to support and remains speculative in the current study.

Another explanation for the rapid recovery of V_c, in comparison to strength and pain perception at later time points could be due to greater damage to surrounding connective tissues rather than the muscle fibres themselves (Nogueira Ade et al. 2011). This is supported by Brown et al. (1997) who found eccentric exercise to elicit markers suggestive of connective tissue damage within muscle. Stiffness in the musculotendinous unit may also explain the diminished MVC up to 48 h post-exercise (day 3) seen within the study, despite supposedly recovered V_c (Kawczynski et al. 2018). It may, therefore, be beneficial to investigate tendon stiffness in the low back during acute injury in future to confirm this outcome. Techniques such as shear wave elastography (SWE), which Creze et al. (2017) have shown to be practicable for quantifying lumbar muscle stiffness, may be ideal for such quantifications.

When investigating relationships between measures, D_{max} was seen to have the most correlations to the other measures of injury (Table 3). In addition, it was the only MMG variable to temporally follow the decline and recovery of the subject's pain scores. Here, it was shown that D_{max} had moderate correlations to MVC and VAS (negatively), whilst having a negatively low correlation for myoglobin. Furthermore, D_{max} had the highest R^2 value from the stepwise linear regression with MVC, VAS, and myoglobin shown to have an effect with D_{max} (Table 4). These results suggest that D_{max} could be the best MMG contractile variable to act as a marker of acute muscle damage—a result that has considerable implications for the clinical applications of the MMG technique. This has previously been suggested by Hunter et al. (2012) in regards to eccentric induced muscle damage of the elbow flexors, and Macgregor et al. (2016) for detecting local muscle fatigue in the gastrocnemius medialis.

Both Hunter et al. (2012) and Macgregor et al. (2016) employed a contact sensor within their studies, whilst the current study employed a laser sensor. The contact sensor has been found to be more sensitive to D_{max} as it can account for muscle contractions that do not displace the skin whilst laser sensors cannot (Seidl et al. 2017). Considering the current study's findings, monitoring eccentric muscle damage may be better characterised using a contact sensor, rather than laser. However, Seidl et al. (2017) explicitly state that, from a clinical standpoint, differences between contact and laser sensors may not be considered significant. Thus, findings with one type of sensor may be readily applicable to the other. Nevertheless, further investigations into the differences between MMG sensors may be warranted.

Conclusion

This study is the first to show that MMG can detect changes in lumbosacral muscle contractile properties associated with acute injury following a strenuous eccentric exercise protocol. D_{max} and V_c at multiple spinal levels were MMG variables sensitive to acute injury. The recovery of V_c 24 h (day 2) after the exercise stimulus suggests the fatigue resistant nature of the muscular spine. However, the significantly later resolution of D_{max}, along with the subject's perception of pain scores, suggested that D_{max} might be the best MMG contractile variable to act as a marker of acute muscle damage. This is supported by D_{max} having moderate correlations to strength deficits (MVC) and negatively moderate correlations to pain scores (VAS), with low negative correlations to myoglobin. This result has considerable implications for the clinical application of the MMG technique. Thus, MMG appears useful in detecting acute injury in lower back muscles and so may be beneficial in aiding diagnostic and rehabilitative protocols for back injury in athletic, industrial, and the general populations.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

Allegri M, Montella S, Salici F, Valente A, Marchesini M, Compagnone C, Baciarello M, Manferdini ME, Fanelli G (2016) Mechanisms of low back pain: a guide for diagnosis and therapy. F1000Res 5:F1000 Faculty Rev-1530. https://doi.org/10.12688/f1000research.8105.2

Amirdelfan K, Mcroberts P, Deer TR (2014) The differential diagnosis of low back pain: a primer on the evolving paradigm. Neuromodulation 17(Suppl 2):11–17

Belavý DL, Albracht K, Bruggemann G-P, Vergroesen P-PA, Van Dieën JH (2016) Can exercise positively influence the intervertebral disc? Sports Med 46:473–485

Beneck GJ, Kulig K (2012) Multifidus atrophy is localized and bilateral in active persons with chronic unilateral low back pain. Arch Phys Med Rehabil 93:300–306

Bishop MD, Horn ME, George SZ, Robinson ME (2011) Self-reported pain and disability outcomes from an endogenous model of muscular back pain. BMC Musculoskelet Disord 12:35

Brown SJ, Child RB, Day SH, Donnelly AE (1997) Indices of skeletal muscle damage and connective tissue breakdown following eccentric muscle contractions. Eur J Appl Physiol Occup Physiol 75:369–374

Cheung K, Hume P, Maxwell L (2003) Delayed onset muscle soreness: treatment strategies and performance factors. Sports Med 33:145–164

Clarkson PM, Newham DJ (1995) Associations between muscle soreness, damage, and fatigue. Adv Exp Med Biol 384:457–469

Creze M, Nyangoh Timoh K, Gagey O, Rocher L, Bellin MF, Soubeyrand M (2017) Feasibility assessment of shear wave elastography to lumbar back muscles: a radioanatomic study. Clin Anat 30:774–780

Dickx N, Cagnie B, Achten E, Vandemaele P, Parlevliet T, Danneels L (2010) Differentiation between deep and superficial fibers of the lumbar multifidus by magnetic resonance imaging. Eur Spine J 19:122–128

Essendrop M, Schibye B, Hansen K (2001) Reliability of isometric muscle strength tests for the trunk, hands and shoulders. Int J Ind Ergon 28:379–387

Farina D, Kallenberg LAC, Merletti R, Hermens HJ (2003) Effect of side dominance on myoelectric manifestations of muscle fatigue in the human upper trapezius muscle. Eur J Appl Physiol 90:480–488

Fett D, Trompeter K, Platen P (2017) Back pain in elite sports: a cross-sectional study on 1114 athletes. PLoS ONE 12:e0180130

Friden J, Lieber RL (1992) Structural and mechanical basis of exercise-induced muscle injury. Med Sci Sports Exerc 24:521–530

Golob AL, Wipf JE (2014) Low back pain. Med Clin N Am 98:405–428

Gueorguieva R, Krystal JH (2004) Move over ANOVA: progress in analyzing repeated-measures data and its reflection in papers published in the Archives of General Psychiatry. Arch Gen Psychiatry 61:310–317

Harrison DE, Colloca CJ, Harrison DD, Janik TJ, Haas JW, Keller TS (2005) Anterior thoracic posture increases thoracolumbar disc loading. Eur Spine J 14:234–242

Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH (1994) Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine (Phila Pa 1976) 19:165–172

Hjortskov N, Essendrop M, Skotte J, Fallentin N (2005) The effect of delayed-onset muscle soreness on stretch reflexes in human low back muscles. Scand J Med Sci Sports 15:409–415

Hodges P, Holm AK, Hansson T, Holm S (2006) Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. Spine (Phila Pa 1976) 31:2926–2933

Howell DW (1984) Musculoskeletal profile and incidence of musculoskeletal injuries in lightweight women rowers. Am J Sports Med 12:278–282

Hunter AM, Galloway SDR, Smith IJ, Tallent J, Ditroilo M, Fairweather MM, Howatson G (2012) Assessment of eccentric exercise-induced muscle damage of the elbow flexors by tensiomyography. J Electromyogr Kinesiol 22:334–341

Kanda K, Sugama K, Hayashida H, Sakuma J, Kawakami Y, Miura S, Yoshioka H, Mori Y, Suzuki K (2013) Eccentric exercise-induced delayed-onset muscle soreness and changes in markers of muscle damage and inflammation. Exerc Immunol Rev 19:72–85

Kawczynski A, Mroczek D, Andersen RE, Stefaniak T, Arendt-Nielsen L, Madeleine P (2018) Trapezius viscoelastic properties are heterogeneously affected by eccentric exercise. J Sci Med Sport 21:864–869

Larsen LH, Hirata RP, Graven-Nielsen T (2017) Pain-evoked trunk muscle activity changes during fatigue and DOMS. Eur J Pain 21:907–917

Lau WY, Muthalib M, Nosaka K (2013) Visual analog scale and pressure pain threshold for delayed onset muscle soreness assessment. J Musculoskelet Pain 21:320–326

Lee J, Clarkson PM (2003) Plasma creatine kinase activity and glutathione after eccentric exercise. Med Sci Sports Exerc 35:930–936

Macgregor LJ, Ditroilo M, Smith IJ, Fairweather MM, Hunter AM (2016) Reduced radial displacement of the gastrocnemius medialis muscle after electrically elicited fatigue. J Sport Rehabil 25:241–247

Mannion AF (1999) Fibre type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain. J Electromyogr Kinesiol 9:363–377

Mukaka MM (2012) A guide to appropriate use of correlation coefficient in medical research. Malawi Med J J Med Assoc Malawi 24:69–71

Nogueira Ade C, Vale RG, Gomes AL, Dantas EH (2011) The effect of muscle actions on the level of connective tissue damage. Res Sports Med 19:259–270

Panjabi MM (2006) A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. Eur Spine J 15:668–676

Pisot R, Narici MV, Simunic B, De Boer M, Seynnes O, Jurdana M, Biolo G, Mekjavic IB (2008) Whole muscle contractile parameters and thickness loss during 35-day bed rest. Eur J Appl Physiol 104:409–414

Roy SH, De Luca CJ, Casavant DA (1989) Lumbar muscle fatigue and chronic lower back pain. Spine (Phila Pa 1976) 14:992–1001

Sayers SP, Clarkson PM (2003) Short-term immobilization after eccentric exercise. Part II: creatine kinase and myoglobin. Med Sci Sports Exerc 35:762–768

Seidl L, Tosovic D, Brown JM (2017) Test–retest reliability and reproducibility of laser- versus contact-displacement sensors in mechanomyography: implications for musculoskeletal research. J Appl Biomech 33:130–136

Sward L, Hellstrom M, Jacobsson B, Peterson L (1990) Back pain and radiologic changes in the thoraco-lumbar spine of athletes. Spine (Phila Pa 1976) 15:124–129

Than C, Tosovic D, Seidl L, Mark Brown J (2016) The effect of exercise hypertrophy and disuse atrophy on muscle contractile properties: a mechanomyographic analysis. Eur J Appl Physiol 116:2155–2165

Than C, Seidl L, Tosovic D, Brown JM (2018) Test–retest reliability of laser displacement mechanomyography in paraspinal muscles while in lumbar extension or flexion. J Electromyogr Kinesiol 41:60–65

Tosovic D, Than C, Brown JM (2016) The effects of accumulated muscle fatigue on the mechanomyographic waveform: implications for injury prediction. Eur J Appl Physiol 116:1485–1494

Trainor TJ, Trainor MA (2004) Etiology of low back pain in athletes. Curr Sports Med Rep 3:41–46

Trost Z, France CR, Thomas JS (2011) Pain-related fear and avoidance of physical exertion following delayed-onset muscle soreness. Pain 152:1540–1547

Wan Q, Lin C, Li X, Zeng W, Ma C (2015) MRI assessment of paraspinal muscles in patients with acute and chronic unilateral low back pain. Br J Radiol 88:20140546

Williams DM, Sharma S, Bilodeau M (2002) Neuromuscular fatigue of elbow flexor muscles of dominant and non-dominant arms in healthy humans. J Electromyogr Kinesiol 12:287–294