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TITLE PAGE

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Regional distribution of myoelectric median frequency in the erector spinae muscles under the influence of delayed-onset muscle soreness.

Alvaro Pano-Rodriguez^{1,3-4}, Julien Ducas^{2,3}, Guillaume Vadez^{1,3}, Jacques Abboud^{1,3}

¹Department of Human Kinetics, Université du Québec à Trois-Rivières, Trois-Rivières, QC, Canada.

²Department of Anatomy, Université du Québec à Trois-Rivières, Trois-Rivières, QC, Canada.

³Groupe de Recherche sur les Affections Neuromusculosquelettiques (GRAN), Université du Québec à Trois-Rivières, Trois-Rivières, QC, Canada.

⁴ Faculty of Education, Psychology and Social Work, Department of Specific Didactics, University of Lleida, Lleida, Spain

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Correspondence Address:

Jacques Abboud,

Department of Human Kinetics

3351, boul. des Forges, Trois-Rivières,

Qc, Canada, G8Z 4M3.

Telephone number: +1 (819) 376-5011. Email: jacques.abboud@uqtr.ca

32 **Abstract**

33 Delayed-onset muscle soreness (DOMS) is a non-invasive pain model offering a unique
34 opportunity to study trunk neuromuscular adaptations. While prior research has examined
35 regional muscle activation in the lumbar region, the spatial distribution of median
36 frequencies (MF) under DOMS has not been explored. This study investigated the effect
37 of DOMS-induced pain on the spatial distribution of MF in the lumbar erector spinae
38 muscles and its association with trunk force variability during submaximal contractions.
39 Twenty healthy adults completed two laboratory sessions: one pain-free and one under
40 low back DOMS. High-density surface EMG was recorded bilaterally on the erector
41 spinae during submaximal isometric trunk extensions. MF distribution was analyzed
42 using centroid coordinates with and without DOMS, and force variability was assessed.
43 DOMS significantly increased perceived muscle pain and soreness in the lumbar region.
44 It also caused a cranial and medial shift of the MF centroid, significant on one side of the
45 trunk. However, force variability remained stable between conditions. These results
46 suggest that DOMS induces regional adaptations in lumbar muscle MF. The spatial
47 distribution of MF may serve as a novel and sensitive marker of neuromuscular adaptation
48 to pain, highlighting the trunk system's ability to maintain force steadiness despite pain
49 and soreness.

50 **Keywords:** DOMS, high-density EMG, lumbar spine, pain, median frequency

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52 **Word Count:** 3998

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55 Clinical low back pain (LBP) poses a significant challenge due to its multifactorial nature
56 ¹. Experimental pain models, such as intramuscular injections of hypertonic saline or
57 thermal cutaneous pain ^{2,3} have provided valuable insights into neuromuscular
58 adaptations to pain. However, these methods present inherent limitations: hypertonic
59 saline can excite motor axons, influencing lumbar sensorimotor control independent of
60 pain processes ^{4,5}, limiting the interpretability of results. In contrast, delayed onset muscle
61 soreness (DOMS) offers a more representative model of LBP ⁶. Unlike other pain models,
62 DOMS does not involve invasive procedures or external stimuli that could confound the
63 interpretation of neuromuscular adaptations. It enables the study of pain-induced changes
64 in muscle recruitment pattern, proprioception, and functional performance over a
65 prolonged period, providing insights into pain neuromuscular adaptations ^{7,8}. Moreover,
66 the non-invasive nature of DOMS facilitates repeated measures and pre–post intervention
67 studies, enhancing the ability to track the progression of pain and its effects on the
68 musculoskeletal system ⁹. This makes DOMS a unique pain model in understanding the
69 mechanisms underlying LBP.

70 Recent research by Arvanitidis et al. (2024) showed that DOMS in the lumbar muscles
71 lead to changes in lumbar electromyographic (EMG) patterns. Specifically, their research
72 highlighted altered high-density surface EMG (HDsEMG) torque relationships and
73 improvements in torque steadiness, despite muscle soreness, suggesting compensatory
74 motor adaptations that maintain performance ¹⁰. The authors focused on analyzing the
75 HDsEMG signal amplitude. By generating topographical maps of EMG amplitude values,
76 they explored the regional distribution of lumbar muscle activity under the influence of
77 DOMS. In contrast, the present study uses another approach by focusing on the spatial
78 distribution of median frequencies (MF) under the influence of DOMS. Specifically, how

79 pain and muscle soreness induced by DOMS influence the spatial recruitment of motor
80 units, given that nociceptive input can alter motor unit discharge rate differently across
81 the motor unit pool ¹⁰. This is particularly relevant for the lumbar extensor muscles, which
82 have a large and regionally distributed pool of motor units, allowing for flexible and
83 spatially differentiated recruitment strategies ¹¹. This approach has previously been used
84 for assessing how different muscle regions contribute to force production and how fatigue
85 can lead to localized changes in muscle activity. Gallina et al. (2011) demonstrated that
86 the myoelectric manifestations of fatigue are regionally distributed within the medial
87 gastrocnemius muscle, with some areas showing more pronounced MF decreases during
88 a fatigue task ¹².

89 The physiological changes associated with DOMS, primarily muscle fiber damage and
90 inflammation, affect excitation-contraction coupling, influencing muscle electrical
91 activity ¹³. Mechanical damage to type II fibers during eccentric contractions triggers an
92 inflammatory response ⁶, leading to increased muscle stiffness, and the recruitment of
93 additional motor units to compensate for the loss of force production from fatigued or
94 damaged fibers ¹⁴. Given the possible localized nature of muscle damage and
95 inflammation in DOMS ¹⁰(Arvanitidis et al., 2024), changes in motor unit recruitment
96 patterns and muscle activation strategies may result in regional differences in EMG
97 frequencies ¹⁵.

98 In addition, a recent systematic review has reported significant impairments in force
99 steadiness during both clinical and experimentally induced pain conditions ¹⁶. However,
100 minimal attention has been given to the effects of DOMS on trunk force steadiness ¹⁰.

101 The primary objective of this study is to investigate the impact of DOMS-induced pain
102 on the recruitment strategies of the lumbar muscles. In particular, we aim to assess the
103 spatial distribution of EMG frequencies with and without the influence of low back

104 DOMS. We hypothesize that DOMS-induced pain will trigger specific adaptations in
105 muscle activation strategies, which may vary between low back regions. These
106 adaptations are expected to manifest as shifts in the spatial distribution of MF. The
107 secondary objective of this study is to explore how these strategies in response to DOMS
108 influence force production.

109

Methods

110 Participants:

111 Participants were recruited via online advertisements (e.g., social media platforms) and
112 word-of-mouth within the local community of Trois-Rivières (Québec, Canada). All
113 experimental procedures and data collection were carried out between February and July
114 2023. Twenty adult participants, including 11 men and 9 women, with an average age of
115 26.05 ± 5.09 years, height of 1.72 ± 0.08 m, weight of 72.15 ± 16.54 kg, and BMI of
116 24.14 ± 4.65 kg/m², were enrolled in this study. Exclusion criteria included a history of
117 LBP within the past year, spinal surgery, inflammatory arthritis affecting the axial
118 skeleton, advanced osteoporosis, pregnancy, and severe incapacitating pain in regions
119 other than the lower back that would hinder the ability to undergo the evaluation protocol
120 in the laboratory. Thus, all participants were pain-free at the time of the study. Sample
121 size estimation was based on a previous study using a similar protocol involving trunk
122 extension contractions in the same position and experimental pain induced by electrical
123 stimulation ¹⁷. In that study, a significant spatial shift in lumbar muscle activity was
124 observed under the influence of pain, with reported effect sizes ranging from moderate to
125 large ($\eta^2 = 0.066$ to 0.232). Accordingly, an a priori power analysis was conducted using
126 G*Power 3.1 software ¹⁸, assuming a moderate effect size (Cohen's $d = 0.6$), a power of

127 80%, an alpha level of 0.05, and the primary outcome variable the centroid location of
128 the MF distribution.

129 Design:

130 This study adopts a within-subject design with repeated measures. Approval for the
131 research protocol was obtained from the Research Ethics Board for Human Research at
132 the Université du Québec à Trois-Rivières (CER-22-290-07.11). Participants gave written
133 informed consent, acknowledging their right to withdraw from the experiment without
134 prejudice, which align with the Declaration of Helsinki (2013)¹⁹. The study design and
135 reporting followed recommendations from the STROBE guidelines for observational
136 research.

137 Experimental Protocol:

138 The experimental protocol comprised two distinct sessions, with each session involving
139 a voluntary isometric contraction protocol to assess the neuromuscular strategies of the
140 lumbar muscles. In the initial session (no DOMS condition), participants completed a
141 voluntary isometric contraction protocol. This was followed by a validated DOMS-
142 inducing protocol to ensure the occurrence of experimental LBP in the second session²⁰.
143 The second session took place 24-36 hours later because it coincides with the peak
144 manifestation of low back DOMS effects.

145 Participants were positioned on a custom-designed apparatus to perform isometric
146 contractions at 45° of trunk flexion in a semi-seated posture (Fig. 1). Real-time
147 measurement of the target trunk flexion position was facilitated using a digital
148 inclinometer (precision of $\pm 0.1^\circ$, model 40-6067, Johnson Level & Tool Mfg. Co., Inc.
149 Mequon, WI) positioned on the L3 vertebra. Once the target 45° trunk flexion position
150 was reached, a belt was placed on the participant's shoulders and adjusted to the correct

151 length. This belt was connected to a load cell (Model LSB350; Futek Advanced Sensor
152 Technology Inc, Irvine, CA, USA) to record the force exerted during the isometric
153 contractions.

154 Myoelectric activity of the lumbar muscles on both the right and left sides was recorded
155 using two HDsEMG arrays, each consisting of 64 electrodes arranged in an 8x8 matrix
156 with a 10 mm space between electrodes (semidisposable adhesive matrix; model
157 ELSCH064, OTBioelettronica, Torino, Italy). Before electrode placement, the skin was
158 prepared by shaving, gently abrading with fine-grade sandpaper (Red DotTrace Prep; 3M,
159 St. Paul, MN), and cleaning with rubbing alcohol (ethyl alcohol 70%). The electrode grids
160 were aligned with the orientation of the muscle fibers and consistently positioned across
161 participants to minimize variability. The center of the grid was positioned at L3, with the
162 medial edge about 1 cm from spinous process of this vertebra. A reference electrode was
163 positioned over the right iliac crest, and hypoallergenic ink marks were used to ensure
164 identical electrode placements across both sessions. Additionally, the same investigator
165 performed all electrode placements across sessions ensuring test-retest reliability ²¹.

166 *(***Figure 1 near here***)*

167 Participants performed five-second maximal voluntary isometric contractions (MVIC) of
168 trunk extension in the previously explained position, with one minute of rest between
169 repetitions. The highest MVIC value was used to standardize the subsequent submaximal
170 isometric contractions, ensuring the target torque exerted was performed based on each
171 individual's MVIC.

172 Participants executed a 20-second submaximal isometric contraction of trunk extension,
173 maintaining 20% of their MVIC without a specific ramp-up phase. This force level
174 reflects the typical intensity of ES contractions during daily activities ²². Participants

175 received real-time visual feedback displayed on a monitor at eye level. The visual
176 feedback consisted of a line representing the target force level (20% MVC). Participants
177 were instructed to maintain their force as steadily as possible.

178 A validated DOMS-inducing protocol was used to induce experimental pain in the lumbar
179 muscles²⁰. Participants first familiarized themselves with the apparatus and then
180 performed three MVICs of trunk extension in a prone position on a 45° inclined Roman
181 chair, exerting maximal force against a belt connected to a load cell (Model LSB350;
182 Futek Advanced Sensor Technology Inc., Irvine, CA, USA). A one-minute rest interval
183 minimized muscle fatigue between MVICs, and the highest MVIC value was used to set
184 the DOMS-inducing load. The DOMS-inducing protocol involved four sets of 25 trunk
185 flexion–extension movements with one-minute rest intervals, executed in the same
186 configuration as the MVIC protocol, with an additional load equivalent to 10% of MVIC
187 applied. Straps at the hip level were used to minimize pelvic tilt. Participants followed a
188 3-3-1 tempo (three seconds eccentric contraction, three-second static hold, one second
189 concentric contraction) with auditory feedback and verbal encouragement throughout.

190 Data Collection and Analysis:

191 Sociodemographic information, including age, sex, weight, and height, was collected
192 from all participants. Low back pain and muscle soreness levels were collected on the
193 second session using a numerical rating scale, anchored between 0 (no pain; no soreness)
194 and 10 (worst possible pain; worst possible soreness).

195 Lumbar pressure pain sensitivity was assessed at the beginning of both sessions (DOMS
196 and no DOMS). A handheld dynamometer (MicroFet 2, Hoggan Industries Inc., West
197 Jordan, UT, USA) was used to apply constant pressure at specific sites on the lumbar
198 muscles (bilaterally at L2 and L4) and on the vastus medialis (control site). Participants

199 reported when they first perceived pain. Pressure pain thresholds (PPT) were used to
200 identify the presence and localization of DOMS-induced muscle soreness and mechanical
201 hyperalgesia ²³.

202 HDsEMG data were analyzed separately for the left and right lumbar muscles. The signals
203 were digitally band-pass filtered using a 4th-order Butterworth filter (30–400 Hz) to
204 remove noise and artifacts, and notch filters were applied to eliminate 60 Hz power line
205 interference. The signals were imported and analyzed using Matlab (version R2023b; The
206 MathWorks, Natick, MA). A detailed visual examination of the raw HDsEMG signals
207 was conducted to identify electrodes with contact issues. Problematic electrodes were
208 addressed by replacing them through an interpolation technique that used data from
209 neighboring electrodes. Specifically, the interpolation was conducted by averaging the
210 values from the electrodes located directly above and below the faulty one along the
211 craniocaudal axis (aligned with the muscle fibers). In cases where there were no
212 neighboring electrodes above or below, such as in the first and last rows of the grid, these
213 problematic electrodes were excluded from the analysis to maintain data integrity.
214 Following the recommendations of Gallina et al. (2022) ²⁴, recordings were excluded
215 from the analysis if the number of removed electrodes exceeded 10% of the total
216 electrodes. The central 10 seconds of each signal during the isometric contraction were
217 analyzed and segmented into 0.5-second windows, following previous methodology²⁵.

218 To assess the impact of DOMS-induced pain on neuromuscular recruitment strategies, we
219 analyzed the spatial distribution of MF, with lower frequencies often associated with
220 changes in action potential propagation ^{12,24}. By calculating the MF across all channels,
221 we were able to generate topographical maps showing how MF were distributed across
222 the ES muscles during the isometric contractions. The mean of MF on each side and both
223 conditions was also computed.

224 To compute the spatial distribution of MF, we first performed a Fast Fourier Transform
225 on the HDsEMG signals to obtain the power spectrum for each channel. We analyzed the
226 medio-lateral and cranio-caudal coordinates of the centroid of MF, defined as the average
227 position of the channels with MF values exceeding 70% of the maximum observed across
228 all channels ^{12,24}. Right-side data were mirrored along the x-axis for clarity, ensuring that
229 higher x-coordinates indicated a more medial location of the MF centroid on both sides.

230 Force signals were low-pass filtered at 10 Hz using a 2nd-order Butterworth filter to
231 smooth the data and remove high-frequency noise. Force variability was computed using
232 the coefficient of variation (CV) to account for variations in force levels, using the
233 formula: $CV = (SD / \text{Mean}) \times 100$, where SD is the standard deviation of the filtered force
234 signal and Mean is the corresponding average force during the analyzed data.

235 Statistical Analysis:

236 All statistical analyses were conducted using SPSS, version 28 (SPSS Inc., IBM Corp.,
237 Armonk, NY, USA). Parametric tests were chosen based on the normal distribution of the
238 data, as determined through the Kolmogorov-Smirnov test and visual inspection. T-tests
239 for dependent sample were performed to assess the impact of experimental pain (no pain
240 vs. DOMS-induced pain) on mean of MF, MF centroid coordinates (mediolateral and
241 craniocaudal axes), force variability, and pressure pain threshold. To identify potential
242 side differences in pain sensitivity, paired-sample t-tests were conducted to compare
243 DOMS PPTs between both sides. A significance threshold of $p < 0.05$ was used for all
244 statistical analyses. Effect sizes were indicated using Cohen's d with classifications as
245 follows: small ($d = 0.2$), medium ($d = 0.5$), and large ($d = 0.8$) ²⁶. The presentation of
246 results includes mean and standard deviation (SD).

247 **RESULTS**

248 At the beginning of the second post-test session (DOMS condition), the average level of
249 muscle soreness was 4.35 (SD = 1.91) and the average mean pain in the lumbar region
250 was 1.4 (SD = 1.90), indicating that participants perceived low level of pain.

251 Results showed significant reductions in PPTs under pain conditions compared to no-pain
252 conditions in all the pressure points except the control region (Table 1). No significant
253 differences were observed between sides (L2 left vs. L2 right: $p = 0.613$; L4 left vs. L4
254 right: $p = 0.162$) following the DOMS protocol.

255 *(***Table 1 near here***)*
256

257 Results showed no significant difference of the MF mean between conditions on both
258 sides. More precisely, on the left side, MF means were 87.72 (SD = 10.01) without DOMS
259 and 86.37 (SD = 10.26) with DOMS ($p = 0.511$). On the right side, MF means were 89.49
260 (SD = 11.30) without DOMS and 86.47 (SD = 9.74) with DOMS ($p = 0.168$).

261 Centroid coordinates of the MF along the mediolateral axis (Figures 2 and 3) showed no
262 significant differences between DOMS and no-DOMS conditions on the left side ($p =$
263 0.764 ; Cohen's $d = 0.068$) while a significant difference was observed on the mediolateral
264 axis on the right side between pain conditions ($p = 0.020$; Cohen's $d = 0.566$). Results
265 indicate that the MF centroid shifted medially under pain conditions (~ 35 mm).

266 For the craniocaudal axis (Figures 2 and 3), a significant difference was found ($p = 0.013$;
267 Cohen's $d = -0.609$) on the left side, showing that the MF centroid shifted cranially under
268 pain conditions (~ 27 mm). No significant difference was observed on the right side ($p =$
269 0.241 ; Cohen's $d = -0.270$).

270 *(***Figure 2 near here***)*

271 Force variability, assessed via CV, was 3.40 (\pm 1.69) in the no-DOMS condition and 3.01
272 (\pm 1.24) in the DOMS condition. This difference was not statistically significant (95%
273 CI: -0.31 to 1.09, $t(19) = 1.17$, $p = 0.256$; Cohen's $d = 0.27$).

274 (**Figure 3 near here**)

275 **DISCUSSION**

276 This study investigated the impact of DOMS on the recruitment strategies of the lumbar
277 muscles, emphasizing spatial adaptations in muscle fiber activation and their effects on
278 force variability. Using HDsEMG, this study provides insights into how DOMS-induced
279 pain influence trunk neuromuscular control. While a spatial shift of MF was observed
280 under the influence of DOMS, lumbar muscle force variability remained unaffected.

281 Significant reductions in PPTs across all pressure points, except for the control region,
282 confirm the sensitivity of lumbar muscles to DOMS-induced pain. The lack of changes
283 in the control region suggests that the pain response remains localized to the lumbar area,
284 minimizing systemic nociceptive effects. This observation reinforces the specificity of
285 DOMS as a model for studying localized muscle pain and its implications for
286 neuromechanical adaptations. These results align with prior research indicating that
287 DOMS heighten pressure sensitivity through localized inflammation^{6,14}. Additionally, no
288 PPT differences between the left and right lumbar sides were observed, suggesting that
289 DOMS-induced hyperalgesia was similar across sides. Participants reported an increase
290 in lumbar muscle soreness and muscle pain in the second session (DOMS condition). This
291 increase highlights the development of DOMS, characterized by localized discomfort and
292 stiffness commonly associated with intense or unfamiliar physical activity. The average
293 pain level of approximately 2/10 aligns with reports from other studies in which delayed
294 onset muscle soreness (DOMS) was used to induce low back pain^{27 28 29}. **However, the**

295 average pain score is below the typical pain intensity scores of people with acute clinical
296 pain. Further research is needed to confirm our results in people with higher pain scores.

297 Our study revealed a cranial and medial migration of the centroid of MF under DOMS-
298 induced pain conditions, only significant on one side. It is well known that acute or
299 experimental muscle pain induces motor adaptations, such as a redistribution of muscle
300 activity between or within the muscles ³¹. For example, Tucker et al. demonstrated that
301 deep-tissue pain in the quadriceps and flexor pollicis longus induces specific shifts in
302 motor unit recruitment patterns as a compensatory response to discomfort, enabling the
303 preservation of function ³². In the lumbar region, similar adaptations have been observed,
304 where pain induces spatial redistribution of muscle activity ¹⁷. Notably, these changes
305 appear to vary between individuals, suggesting distinct neuromuscular adaptation
306 strategies in response to pain ¹⁷.

307 Unlike prior studies that assessed muscle activity patterns, such as spatial redistribution
308 of muscle activity amplitude, the present study's focus on spatial changes in MF which
309 provides a novel perspective on how the lumbar muscles adapt to DOMS-induced pain.
310 Another group of researchers has used this approach to explore the specific roles of
311 muscle subregions in generating force and to understand how fatigue impacts muscle
312 activity in a localized manner. For instance, Gallina et al. (2011) found that fatigue-related
313 myoelectric signals vary across the medial gastrocnemius, with certain areas experiencing
314 greater reductions in MF during sustained exertion ¹². The current study suggests that
315 muscle fibers with higher frequency discharge were mainly recruited in the cranial and
316 medial region of the lumbar muscle under the influence of DOMS. Before the onset of
317 DOMS, participants may have predominantly recruited the lumbar muscle region that was
318 biomechanically most favorable for the task, as evidenced by a relatively localized MF
319 centroid. In the lumbar region, where multiple muscle segments contribute to trunk

320 stabilization and movement, the presence of DOMS may disrupt typical motor patterns,
321 leading to a redistribution of fiber recruitment across different lumbar regions. Yet it is
322 important to note that this adaptation was only significant on one side of the trunk.
323 Previous studies have reported distinct trunk neuromuscular adaptations between the left
324 and right sides of the lumbar muscles during symmetrical tasks ^{11,17,33}. These asymmetries
325 may be explained by side dominance in the lumbar musculature, which has been observed
326 in response to muscle fatigue and perturbation tasks ^{34,35}. Our findings support the idea
327 that the lumbar muscles can modulate their activation strategy independently on each
328 side, potentially as a functional adaptation to pain. This highlights the relevance of
329 analyzing each side separately when investigating trunk neuromuscular responses.
330 Further research is warranted to better understand the origin and functional implications
331 of these asymmetrical adaptations.

332 The cranial shift in MF centroid observed in this study may reflect an adaptive motor
333 strategy to redistribute load and reduce discomfort during DOMS. Falla et al. (2014)
334 reported that reduced centroid migration during a repetitive lifting task in low back pain
335 individuals ³⁶. These findings suggest that spatial adaptations in muscle activation may
336 play a protective role. The absence of a shift in patients with chronic low back pain may
337 either be caused by their pain or result from it. Determining which occurs first remains
338 challenging. Arvanitidis et al. (2022) identified cranial shifts in lumbar ES activity in
339 individuals with chronic low back pain during submaximal contractions, highlighting
340 region-specific neuromuscular strategies to redistribute load and avoid exacerbating pain
341 in caudal regions ³⁷. Although the present study focuses on acute DOMS-induced pain,
342 the MF spatial redistribution observed may represent a shared neuromuscular adaptation
343 to maintain functionality under DOMS conditions.

344 The present study found that force variability during submaximal isometric contractions
345 showed negligible changes under DOMS conditions. The non-significant reduction in
346 force variability suggests that compensatory neuromuscular adaptations effectively
347 mitigate the impact of DOMS pain on force variability. These findings reinforce the
348 robustness of the trunk neuromuscular system in maintaining task performance despite
349 low back discomfort and pain. Supporting this idea, Mista et al. (2015) analyzed the
350 modulation of motor variability related to experimental muscle pain during elbow flexion
351 contractions and observed that, despite the presence of pain, the neuromuscular system
352 adjusted motor control strategies to maintain force stability ³⁸. Similarly, Cleary et al.
353 found that acute experimental pain in the tibialis anterior muscle alters the recruitment
354 strategies in the vastus lateralis and vastus medialis, but participants were still able to
355 maintain the force target ³⁹. However, a systematic review by Arvanitidis et al. (2024),
356 which examined the effects of experimental pain on force steadiness across various
357 muscle groups and tasks, concluded that pain generally increases force variability,
358 particularly in clinical contexts ¹⁶. Nonetheless, the review also highlighted substantial
359 variability among tasks and individuals, suggesting that the specific characteristics of
360 DOMS and the nature of the submaximal tasks assessed in the present study may
361 contribute to the observed stability in force variability.

362 The findings of this study have important implications for understanding trunk
363 neuromuscular control in the presence of experimental pain. The observed spatial
364 adaptations in MF distribution and the maintenance of force production stability
365 underscore the adaptability of the neuromuscular system under pain conditions. The
366 spatial redistribution of muscle activity observed under DOMS suggests that the central
367 nervous system may selectively recruit new muscle regions to preserve force control. The

368 next step will be to identify whether these strategies differ in individuals with chronic low
369 back pain, as the long-term effects of chronic pain may lead to distinct trunk adaptations.

370 While this study provides valuable insights, a few limitations should be acknowledged.
371 First, the sample size was relatively small and comprised mainly of young adults,
372 potentially limiting the generalizability of the findings. Second, the focus on a single
373 muscle group (lumbar muscles) may not fully capture the systemic effects of DOMS on
374 the trunk musculoskeletal system. Third, the assessors were not blinded to the
375 experimental condition during EMG analysis. However, automated algorithms
376 procedures were implemented to minimize the influence of subjective interpretation.
377 Another limitation is the timing of the post-intervention assessment, which was conducted
378 24 to 36 hours after the DOMS-inducing protocol. While this timeframe corresponds to
379 the typical peak of muscle soreness²⁰, it is possible that different temporal windows (e.g.,
380 earlier or later phases of recovery) might produce distinct neuromuscular responses.
381 Additionally, the observed neuromuscular adaptations may not be generalizable to
382 conditions involving higher levels of perceived pain. Furthermore, the study focused
383 exclusively on EMG activity of the lumbar muscles, without including complementary
384 measurements such EMG recordings from lower limb or abdominal muscles. This limits
385 our ability to fully characterize the trunk neuromuscular adaptations under DOMS
386 condition. Finally, while previous studies have established the reliability of traditional
387 EMG measures, such as amplitude or amplitude spatial distribution using HDsEMG^{21,40}
388 in the thoracolumbar erector spinae, the test-retest reliability of the spatial distribution of
389 the MF remains to be established.

390 This study highlights the impact of DOMS-induced pain on neuromuscular control,
391 revealing spatial adaptations in MF distribution of the ES and the maintenance of force
392 production stability. These findings underscore the importance of understanding localized

393 pain mechanisms to develop targeted interventions that optimize functional performance
394 and recovery following intense exercises.

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398 **Data Statement**

399

400 **Citation Diversity Statement**

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524 **Tables**

525 **Table 1. Pressure Pain Thresholds Results**

Pressure Point	No Pain (Mean ± SD)	Pain (Mean ± SD)	Change (Mean ± SD)	95% CI Lower	95% CI Upper	t	p	Effect Size (Cohen's d)
L2 left	10.99 ± 5.88	7.21 ± 3.84	3.79 ± 3.24	2.28	5.31	5.24	<0.001	1.17
L2 Right	10.49 ± 6.02	7.40 ± 4.50	3.09 ± 3.19	1.60	4.58	4.33	<0.001	1.09
L4 Left	10.39 ± 6.07	7.44 ± 3.83	2.95 ± 3.37	1.37	4.53	3.92	0.001	0.98
L4 Right	9.96 ± 5.48	7.05 ± 3.63	2.91 ± 2.45	1.76	4.05	5.31	<0.001	1.20
Control Region	10.70 ± 5.23	10.36 ± 4.55	0.34 ± 2.48	-0.82	1.50	0.62	0.544	0.08

526 Table 1. Pressure pain threshold at different points and control region. Units are expressed in kilograms-force (kgf).