Superficial lumbar muscle recruitment strategies to control the trunk with delayed-onset muscle soreness

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Abstract

Purpose: The lumbar region offers various muscle recruitment strategies to achieve a task goal under varying conditions. For instance, trunk movement control can be reorganized under the influence of low back pain. How such task-modulation is obtained is not fully understood. The objective of this study was to characterize superficial lumbar muscles recruitment strategies under the influence of delayed-onset muscle soreness (DOMS) during unexpected trunk perturbations.

Methods: Healthy participants experienced a series of 15 sudden external perturbations with and without the influence of low back DOMS. During these perturbations, high-density surface electromyography was used to characterize recruitment strategies of superficial lumbar muscles, while kinematics sensors were used to characterize movements of the trunk. Lumbar muscle recruitment strategies, characterized by the amplitude of muscle activity amplitude, the latencies of the reflex activity and the spatial distribution of muscle activity, were compared across perturbations trials and with and without DOMS.

Results: An attenuation of lumbar muscle activity amplitude was observed across perturbation trials without DOMS, but not with DOMS. The spatial distribution of muscle activity was similar with and without DOMS. No significant changes in reflex activity latency and trunk flexion movement were observed.

Conclusions: Following an unexpected trunk perturbation under DOMS effects, trunk movement are controlled using two different superficial lumbar muscles control strategies: keeping a constant level of their overall muscle activity and using a variable muscle recruitment pattern.

Keywords: experimental pain; high-density EMG; erector spinae, postural perturbation

List of abbreviations

ANOVA: Analysis of variance

DOMS: Delayed-onset muscle soreness

EMG: Electromyography

HDsEMG: High-density surface electromyography

MVIC: Maximal voluntary isometric contraction

PPT: Pressure pain threshold

RMS: Root mean square

Introduction

The trunk system complexity offers various muscle recruitment strategies to control spine movement and stabilization. For example, a non-uniform spatial distribution of the lumbar muscle activity seems to play an important role in everyday life activities such as preventing the development of muscle fatigue (Tucker et al. 2009) or performing a repeated lifting task (Falla et al. 2014). Recently, it has been shown that motor units within the erector spinae (longissimus muscle) have fibers grouped in two distinct regions (upper and lower lumbar parts) during isometric contractions and voluntary movements (Abboud et al. 2020). These observations suggest that the regional control of superficial lumbar muscles could play an important role in the trunk movements. On the other hand, a decreased motor variability, measured by the spatial distribution of lumbar muscle activity, to perform a motor task, has been observed in chronic low back pain patients (Falla and Gallina 2020), while no change or an increased motor variability seems to occur under the influence of acute experimental pain (Meier et al. 2019; van Dieën et al. 2017). Overall, the central nervous system seems to able to find a new strategy to achieve the task goal under the influence of chronic or experimental low back pain, but how such task-modulation is obtained is not fully understood.

Over the past two decades, there has been a growing interest in the use of experimental pain models to better understand motor adaptations in individuals suffering from low back pain. While clinical low back pain still represents a major challenge because of the complex interactions between the numerous contributors related to its cause (Hartvigsen et al. 2018), experimental pain models can provide insights into neuromuscular adaptations to pain that are difficult to obtain in low back pain patients (Hodges et al. 2003). Delayed-onset muscle soreness (DOMS) is a painful condition that occurs due to different physiological alterations, such as muscle fiber damage, disruption of the connective tissue, and is usually induced by repetitive intense physical activity (Cleak and Eston 1992), where pain is mainly reproduced during active muscle contraction and stretching as well as muscle palpation. More importantly, pain associated with DOMS is intrinsically modulated by movement, a feature that cannot be obtained as efficiently with other common experimental pain models (e.g. injections of hypertonic saline solution). Therefore, DOMS represents a relevant pain models to investigate low back muscle activation patterns while performing challenging motor tasks.

When the task is to control the movement and stabilize the trunk under cutaneous acute low back pain, the central nervous system uses a variable lumbar muscle recruitment strategy to control the trunk, while it fails to do so under the influence of lumbar muscle fatigue (Abboud et al. 2018; Abboud et al. 2016). It was also reported that a smaller overall muscle response was necessary to

control the trunk using variable muscle recruitment patterns and vice versa, suggesting a lower functional cost while using a higher motor variability. The first objective of this study was to characterize the recruitment strategies of the superficial lumbar muscles to control the trunk under the influence of DOMS. The second objective was to identify the lumbar muscle response adaptations to control movement and stabilize the trunk under a series of sudden perturbations. We hypothesized that the recruitment strategies for lumbar muscles will be different under the influence of low back DOMS.

Methods

Participants

Twenty healthy adult participants (10 men and 10 women) without low back pain took part in this study. Participants mean for age, weight, height, and BMI were respectively: 25.5 ± 5.9 years, 69.9 ± 12.7 kg, 1.70 ± 0.11 m and 23.9 ± 2.4 kg/m^2. Participants were recruited among the university community. The project received approval from the Research Ethics Board for human research of the "Université du Québec à Trois-Rivières" (CER-19-256-07.06). All participants gave written informed consent, acknowledging their right to withdraw from the experiment without consequences.

Study design

The experimental protocol was conducted over two sessions separated by 24 to 36 hours because muscle soreness and pain usually peak during this period following an exercise-induced muscle damage protocol (Cheung et al. 2003; Clarkson and Hubal 2002). In the first session, participants completed the short version of International Physical Activity Questionnaire (Craig et al. 2003) about their physical activity level. Session 1 assessed the neuromuscular adaptations across a series of trunk perturbations before the exercise-induced muscle damage protocol. At the end of the session 1, participants were submitted to an exercise-induced muscle damage protocol targeting low back muscles. Session 2 assessed the neuromuscular adaptations across a series of trunk perturbations under the influence of low back DOMS. Figure 1 represents the experiment timeline.

Trunk perturbation protocol

In both sessions, participants were submitted to a series of 15 sudden trunk perturbations. To induce trunk perturbation, participants were installed in a semi-seated position on a custom-made apparatus as previously described (Figure 2a; (Abboud et al. 2016)). In this position, participant's knees were flexed at ~75°, while trunk was flexed at 20° from the neutral posture. The trunk

position was standardized using the kinematic sensors (see section kinematic data collection) to make sure that each participant started all perturbation trials with 20° of trunk flexion. This trunk flexion position was chosen based on pilot data to increase lumbar muscle stretching and consequently increase pain and/or soreness perceptions in the low back muscles during session 2. A cable attached at T9 vertebra level was fixed to a harness installed over the upper body. The perturbation trigger was connected to a small motor by a cable. Once the motor started, it was able to pull the trigger and initiate a posterior to anterior perturbation of the trunk by releasing the tension in the cable. To standardize the magnitude of the trunk perturbation across a series of perturbations, participants were asked to maintain a 20% of their trunk flexion maximal voluntary isometric contraction (MVIC; see trunk MVIC protocol using the perturbation apparatus for further details). The same trunk flexion MVIC was used in both sessions for the same participant. Participants were provided with visual feedback indicating the targeted force. Once the targeted force was achieved and maintained, the motor was activated and pulled the trigger with a random delay varied among 1, 3 and 5s to avoid any anticipation of the trunk perturbation onset. Moreover, participants wore headphones to mute the sound of the perturbation trigger. Participants were asked to return to their initial position after each perturbation.

Trunk MVIC protocol using the perturbation apparatus

Trunk flexion and extension MVICs were assessed using the same apparatus as the perturbation protocol (Figure 2a). For the trunk flexion, participants had to pull the cable anteriorly as hard as they can, while the trunk was flexed at 20° from the neutral posture. For the trunk extension MVIC, a belt was positioned on the upper thoracic (T4 vertebra) and in a neutral posture, participants were asked to pull posteriorly against this belt. Three trials were completed for each trunk MVIC. The highest MVIC in trunk flexion was used for the perturbation protocol while the highest MVIC in trunk extension was used for electromyography (EMG) normalization.

Exercise-induced muscle damage protocol

This protocol was performed after the first series of trunk perturbations during session 1. Participants were installed on a 45-degrees inclined Roman chair in a prone position with the participant's trunk parallel to the floor (Figure 2b). A strap was installed over the shoulders of the participants and was connected to a load cell (Model IPM250; Futek Advanced Sensor Technology Inc, Irvine, CA, USA). In this position, participants were asked to perform three trunk extension MVICs using the Roman chair. Then, the strap was removed. Ten percent of the highest trunk extension MVIC value was considered for the extra load used in the exercise-induced muscle

damage protocol. Extra load was added by asking the participant to hold a weight plate at their chest level. The protocol consisted in 5 series of 20 repetitions of trunk flexion-extension. Starting from an initial position, corresponded to the neutral alignment of the trunk, participants performed a trunk flexion of 30° in 3s, maintained this position for 3 more seconds and go back to the initial position in 1s. During the protocol, two belts were installed on the participant's hips and ankles to stabilize the participant's position and to isolate low back muscles. Visual and auditory feedbacks were provided, as well as strong verbal encouragements from the assessors to help the participants during the protocol. The validation of the exercise-induced muscle damage protocol was performed in a previous study (Abboud et al. 2019).

During session 2, participants were asked to perform three trunk extension MVICs using the Roman chair similarly to session 1. Lumbar pain and soreness levels were collected verbally after each perturbation trial using a numerical rating scale, anchored between 0 (no pain; no soreness) and 10 (worst possible pain; worst possible soreness). Moreover, these two scores were obtained over a three-day period following the exercise-induced muscle damage protocol via text message three time a day (9h, 15h and 21h).

Pressure pain threshold protocol

To evaluate the muscle sensitivity with and without DOMS, pressure pain threshold (PPT) were assessed at four different specific sites using a hand-held algometer (Model 01163; Lafayette Instrument Company, Lafayette IN USA) with a 12 mm diameter circular tip probe. Muscle sensitivity was defined as the moment at which pain first reported by the participant. As a control site, the assessment was made on the right rectus femoris. Then, PPT were assessed at both right and left biceps femoris, gluteus maximus and erector spinae muscle (L3). For each site, PPT were obtained using a force applied perpendicularly to the muscle belly. For biceps femoris, the PPT was taken in the middle of the hamstring; for the gluteus muscle, it was done on the posterolateral face of the muscle; for lumbar muscles, PPT was taken approximately at three centimeters from the vertebral column and for the rectus femoris, PPT was done on the vastus medialis. Participants were asked to report the moment when they felt the smallest pain (1/10). Participants were laying on a table in a prone position for every measure, except for the measure of the rectus femoris which was taken in a sitting position with knees flexed. Three assessment were made per site. The three PPT mean value was considered at each site for the analysis. The same order of assessment was used for all participants. To avoid inter-variability, the same assessor took all PPT measurements.

EMG data collection

Myoelectric activity was recorded from the lumbar erector spinae muscles (Figure 2a). The skin over the erector spinae was cleaned with fine-grade sandpaper (Red DotTrace Prep; 3 M, St. Paul, MN), and shaved. Muscle activity was recorded with two high-density surface EMG (HDsEMG) of 64 electrodes arranged in 8 columns and 8 rows spaced by 10 mm (semidisposable adhesive matrix; model ELSCH064, OTBioelettronica, Torino, Italy). The electrode grids were placed orienting the columns along the approximate fiber orientation of the erector spinae muscle (Kalimo et al. 1989). The center of each grid was located at L3 level, and the medial edge of the grid was at ~1 cm from the L3 spinous process. Manual palpation of the vertebrae was performed by the same assessor to localize the lumbar region. Reference electrodes were placed over the right iliac crest. Signals from the bipolar HDsEMG were amplified (128-channel EMG-USB; OTBioelettronica; –3 dB, bandwidths 10–500 Hz) by a factor of 5,000 and digitized at 2048 Hz using a 12-bit A/D converter. The same assessor was responsible for the placement of the grids and the skin was marked to minimize variability between days. Moderate to good between-day reliability has been established for amplitude distribution parameters measured with HDsEMG (Abboud et al. 2015; Afsharipour et al. 2016).

Kinematic data collection

Trunk movements during perturbation trials were collected with a three-dimensional motion analysis system (Optotrak Certus; Northern Digital, Waterloo, ON, Canada). Kinematic sensors were placed by the same assessor for each participant on the left side of participants' trunk over two anatomical landmarks: 1) T1 and 2) T11. Data from kinematic sensors were sampled at 100 Hz and low-pass filtered with a dual-pass, fourth-order Butterworth filter with a cutoff frequency of 5 Hz. HDsEMG and kinematic data were synchronized through a signal triggered by OTBioelettronica software and MATLAB (MathWorks).

Data analysis

Data from the HDsEMG were extracted during the trunk perturbation protocol to create four dependant variables: [1] Baseline muscle activity corresponded to the mean EMG amplitude of the root mean square (RMS) with a 500-ms window before the perturbation onset. The RMS mean of all electrodes from each grid (right and left) was computed and used for statistical analysis. [2] Reflex latency corresponded to the time (in millisecond) between the onset of the trunk perturbation and the EMG reflex onset. To compute the onset of the reflex, HDsEMG signals were filtered (Butterworth 6th order, 50 Hz cutoff frequency) and assessed using a sliding window of 25-ms (Larivière et al. 2010). The reflex onset was identified when the myoelectric signals exceeded three

SDs above the baseline muscle activity (Hodges and Bui 1996). Reflex latency exceeding 200-ms were removed from the analysis to avoid inclusion of voluntary responses (Abboud et al. 2017). The mean of all included reflex latency from each grid (right and left) was computed and used for statistical analysis. [3] HDsEMG reflex amplitude was defined as the mean RMS values computed in a 100-ms window divided equally (50-ms) on either side of the highest RMS value after the perturbation onset (reflex peak). The reflex peak had to be present in a 200-ms window following the perturbation onset to be included in the analysis. The mean of all included HDsEMG reflex amplitude from each grid (right and left) was computed and used for statistical analysis. [4] Spatial distribution of muscle activity corresponded to the shift of the centroid across trunk perturbation trials (Abboud et al. 2016). The medio-lateral and cranio-caudal coordinates of the centroid of the electrodes exhibiting RMS reflex values higher than 70% of the mean of all electrodes were considered for each perturbation trial (Vieira et al. 2010). The range of the centroid shift was computed based on the difference between the maximal and minimal medio-lateral and cranio-caudal coordinates from the 15 perturbations trials for both sessions. Electrodes with contact problems were identified through visual inspection of the raw HDsEMG signals and were reconstructed by interpolation of the neighbouring electrodes.

Data from kinematics sensors were analyzed to create a vector between T1-T11. Trunk flexion angle was obtained in the sagittal plane by calculating the angle between the T1-T11 vector and a horizontal vector relative to the ground. The trunk angle corresponded to the range of motion between the initial position before the trunk perturbation and the maximal trunk flexion after perturbation onset.

For dependent variables 1, 2, 3 and trunk angle, the mean of the first and last five perturbation trials of the first and second series of the 15 trunk perturbation trials were considered for the statistical analysis.

Statistical analysis

Parametric tests were chosen based on the normality of data distribution (Kolmogorov-Smirnov test and visual inspection). Student's t tests for dependant samples were used to compare PPT values and trunk extension MVICs using the Roman chair with and without DOMS. Range of centroid shift were compared for both sides using the same statistical analysis. A within repeated-measures ANOVA was conducted to assess the trial-to-trial adaptation effect across perturbations, the condition effect (without DOMS versus with DOMS), and the interaction effect (condition x adaptation) for each dependent variable. When necessary, the Tukey post hoc test was performed for pairwise comparisons. For all statistical analyses, $p < 0.05$ was considered to be significant.

Statistical analyses were performed with Statistica statistical package version 10 (Statsoft, Tulsa, OK). Data are reported as mean and SD.

Results

Participants were considered as minimally active (N=9) and active (N=11) and the mean MET-min per week was 3395 ± 1566. All participants were able to complete the entire exercise-induced muscle damage protocol. The day following the exercise-induced muscle damage protocol (mean of 28 h ± 2), participants' mean of the highest perceived low back pain was 2/10 (±1.9) while highest perceived soreness was and 2.9/10 (±1.6). All pain and soreness values for the three-day period following the exercise-induced muscle damage protocol are presented in Table 1. A decreased trunk extension MVICs using the Roman chair (90.6±35.8 lbs vs 83.8±30.5 lbs, p=0.03) was observed during session 2. A significant decrease in erector spinae PPT (left side: 4.5±1.5kg vs 3.6±1.6kg, p=0.002; right side: 4.6±1.7kg vs 3.7±1.3kg, p=0.005) were also observed during session 2. No change was observed in the lower limb PPT sites with and without DOMS (all p values > 0.10). One participant was excluded from the trunk extension MVIC using the Roman chair analysis because he was not able to perform the task appropriately and yielding scores that were considered as outliers (superior to the mean±3*SD).

Two participants were excluded from the EMG analysis due to technical issues with the load cell during the session 2 perturbation protocol. All main and interaction effect results from the repeated-measure ANOVAs are presented in Table 2. A significant effect of trial-to-trial adaptation was found for the baseline activity on the left side ($F(1,17)=4.46$, $p=0.049$), showing a decrease of the muscle activity amplitude in the last perturbation trials. No significant effect of DOMS was observed regarding reflex latency. A significant condition x adaptation interaction was observed regarding the HDsEMG reflex amplitude on the left side ($F(1,17)=7.07$, $p=0.02$; Figures 3 and 4). Post hoc results revealed a significant difference between the first and last perturbation trials only without DOMS ($p=0.004$). A similar observation was found on the right side but did not reach the significance threshold (condition x adaptation, $F(1,17)=3.83$, $p=0.07$; Figure 3). Finally, no significant difference was observed for the range of the centroid shift in the medio-lateral and cranio-caudal directions (all p values > 0.26; Figure 5).

In addition to the two excluded participants from the EMG analysis, one more participant was excluded from the kinematic due to data loss of one kinematic sensor during the perturbation protocol. No main effect of trial-to-trial adaptation and DOMS condition was found regarding the trunk flexion angle ($F(1,16)=0.05$, $p=0.82$; $F(1,16)=0.28$, $p=0.60$, respectively). The condition x

adaptation interaction was also not found significant ($F(1,16)=3.62$, $p=0.08$). Trunk flexion angle mean and SD are presented in Table 2.

Discussion

We investigated the recruitment strategies of the superficial lumbar muscles to control the trunk under the influence of low back DOMS. The results of the current study show that superficial lumbar muscles are able to control movement and stabilize the trunk by keeping a constant level of their overall muscle activity while using a variable muscle recruitment strategy.

Observations of a decreased maximal trunk extension MVIC, an increased lumbar sensitivity, as well as an increased subjective experienced pain and soreness strongly suggest the occurrence of low back DOMS following our repeated contraction protocol. Despite all these changes, similar trunk flexion angles following an external unexpected trunk perturbation were observed under DOMS effects. It should be noted that only two kinematic sensors were used to assess the movement of the trunk following the unexpected perturbations. While this does not reflect the complexity of segmental trunk movements, the main focus of this study was to investigate the low back muscle recruitment strategies. Further research is needed to determine whether different segmental movement strategies are used under DOMS effects.

The results of the current study have revealed the failure of the motor system to attenuate the reflex muscle activity response through the repetition of the same unexpected perturbation under the influence of low back DOMS. Without DOMS, a clear attenuation of the EMG reflex amplitude was reported, which has been commonly observed in studies using similar experimental designs (Blouin et al. 2003; Nashner 1976; Skotte et al. 2004). In the current study, a slight trunk flexion (20°) was maintained by participants prior to trunk perturbations. In an erect posture, the erector spinae fibers are oriented posteriorly and caudally (Christophy et al. 2012; Kalimo et al. 1989), generating posterior shear forces resisting anterior reaction shear forces (McGill et al. 2000). Interestingly, this study showed that when this mechanical advantage is reduced by trunk flexion, the central nervous system is able to attenuate reflex muscle activity responses necessary to control the trunk movement. Future studies should assess lumbar muscle fiber orientations in order to confirm such hypothesis. With low back DOMS, the amplitude of muscle reflex activity returned to baseline values indicating that prior adaptations are lost under DOMS. Following this process, the lumbar muscle EMG reflex amplitude remained constant across repetitions of the same unexpected trunk perturbation. This phenomenon has been previously reported with acute lumbar muscle fatigue, but not under the influence of experimental low back pain induced by cutaneous heat stimulation (Abboud et al. 2018; Abboud et al. 2016). In addition, a trunk perturbation trial-

to-trial decrease of the spatial distribution of the lumbar muscle activity has been observed under the influence of muscle fatigue while no change is observed under the influence of cutaneous heat pain or low back DOMS as observed in the current study. Such differences could be explained by the alteration of the physiological properties (motor units available) under the influence of muscle fatigue while no such alteration occurred during cutaneous heat pain. The effect of low back DOMS on trunk control provides new insights on how DOMS-associated pain modulates trunk system responses to an external perturbation. The current findings suggest that individuals experiencing low back DOMS take action by using an alternative motor strategy to reduce potential threats to surrounding tissues.

This study also showed no modification of the overall low back reflex muscle activity amplitude and muscle reflex latency before and after perturbation onset. These findings are supported by a previous study showing no reflex latency difference in the presence of low back DOMS in response to a sudden trunk perturbation (Hjortskov et al. 2005). It can be argued that trunk muscle co-contraction (not evaluated in the current study) might have occurred with DOMS explaining the lack of low back muscle activity amplitude changes. While trunk muscle co-contraction is usually present in low back pain population (van Dieën et al. 2017), it has been reported that during experimental pain the trunk flexor muscles may play a negligible role in the control of the trunk when facing a sudden perturbation (Larsen et al. 2017). Therefore, a more probable explanation of the absence of muscle activity changes could be the observation of a similar spatial distribution of the erector spinae muscle activity observed with and without the DOMS. Either way, the lumbar muscle recruitment strategies seemed variable indicating that a wide range of motor solutions are still available to perform a motor task (Srinivasan and Mathiassen 2012). Others experimental pain modalities have been associated to an increased motor variability, probably to explore new pain-free motor strategies. This relation has now been reported in different body regions (Madeleine 2010; Srinivasan and Mathiassen 2012), including the low back muscles (van Dieën et al. 2017). Based on these findings, it could be hypothesized that the preferred strategy to maintain an appropriate control of the trunk when it is challenged by DOMS is to use a variable recruitment strategy of the superficial lumbar muscles, while maintaining a similar overall level of muscle activity. Such adaptation could have the purpose of minimizing the functional cost of the task (Todorov and Jordan 2002) while adopting a safety mode to prevent the low back region from further injury (Sonkodi et al. 2020).

Although this study reports new findings regarding trunk motor adaptations to DOMS, some limitations should be considered. The small sample size in this study could limit the generalization of the results. Future studies should consider having more participants to confirm the tendencies

observed in the results, such as the significant EMG reflex amplitude adaptation observed in one side but not the other ($p = 0.07$). As our exercise-induced muscle damage protocol was able to only induce light low back pain (~2/10), we recognize that the current findings cannot be generalized to higher low back pain perception conditions, such as pain perception induced by hypertonic saline solution. However, it has been proposed that for patients with chronic low back pain, the minimal clinically important difference is 2/10 (Mannion et al. 2007). While our exercise-induced muscle damage protocol triggered a painful sensation in the lumbar region, this experimental pain model is limited to muscle pain and cannot reproduce other possible alterations present in patients with low back pain. Finally, with the absence of a control group we cannot exclude a possible learning effect between both sessions. However, in a previous study using the same perturbation protocol (Abboud et al. 2018) and a control group, the authors showed that a learning effect was observed in the control group with a clear attenuation of EMG reflex responses across perturbation trials and the two sessions separated by a 5-minute rest period. After the rest period, the EMG reflex response was even lower than the last one observed before the rest period. In the current study, the second session (with DOMS) took place more than 24 hours later and EMG reflex responses in the second session were higher than the last perturbations trials in the first session and very close to the first perturbation trials in the first session. Based on these findings, we believe that learning effect did not influence our results.

In conclusion DOMS have provided new information regarding how the central nervous system deals with experimental low back pain when the trunk control is challenged. The current study revealed that following an unexpected perturbation under DOMS effects, superficial lumbar muscles control the trunk movement using two strategies: keeping a constant level of their overall muscle activity and using a variable muscle recruitment strategy. The next step will be to assess whether these strategies are different with chronic low back pain patients since long-term consequence of chronic pain may trigger different trunk adaptations.

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Legends for illustrations and tables

Table 1. Mean and SD values of pain and soreness values for the three-day period following the DOMS-protocol.

Table 2. Mean and SD values of dependent variables before and after the exercise-induced muscle damage protocol

Figure 1. Timeline of the experimental protocol.

Figure 2. (a) Experimental set up to induce external sudden trunk perturbation. (b) Exercise-induce muscle damage set up.

Figure 3. HDsEMG reflex amplitude trial-to-trial adaptations.
Means of the first and last 5 perturbations trial are represented without and with DOMS. Vertical bars represent the standard deviations. RMS, root mean square; HDsEMG, high-density surface electromyography; EIMD, exercise-induce muscle damage.

Figure 4. Representation of the mean EMG reflex activity traces for the left erector spinae muscle without and with DOMS.
Means of the first and last 5 perturbations trial are represented for both conditions, without and with DOMS. The black dashed line represents the perturbation onset. (A.U. Arbitrary Unit).

Figure 5. Spatial distribution of muscle activity across trunk perturbation trials.
Four typical participants are represented in this figure during pre- (top panel) and post-DOMS (bottom panel) conditions. White crosses represent the position of the centroid. Note that raw data are illustrated and therefore EMG amplitude (color coding) cannot be compared across participants and conditions.

Figure1

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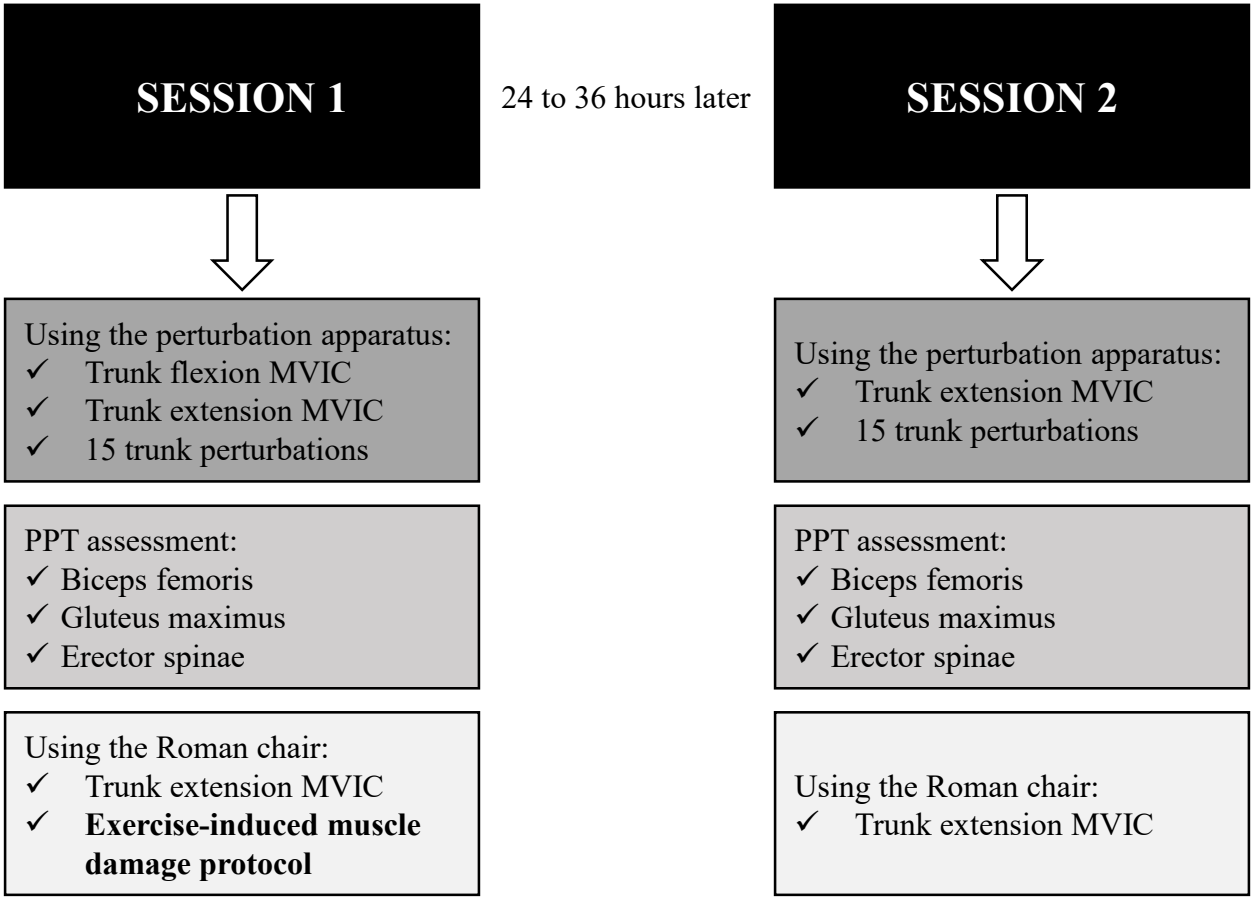

Figure2

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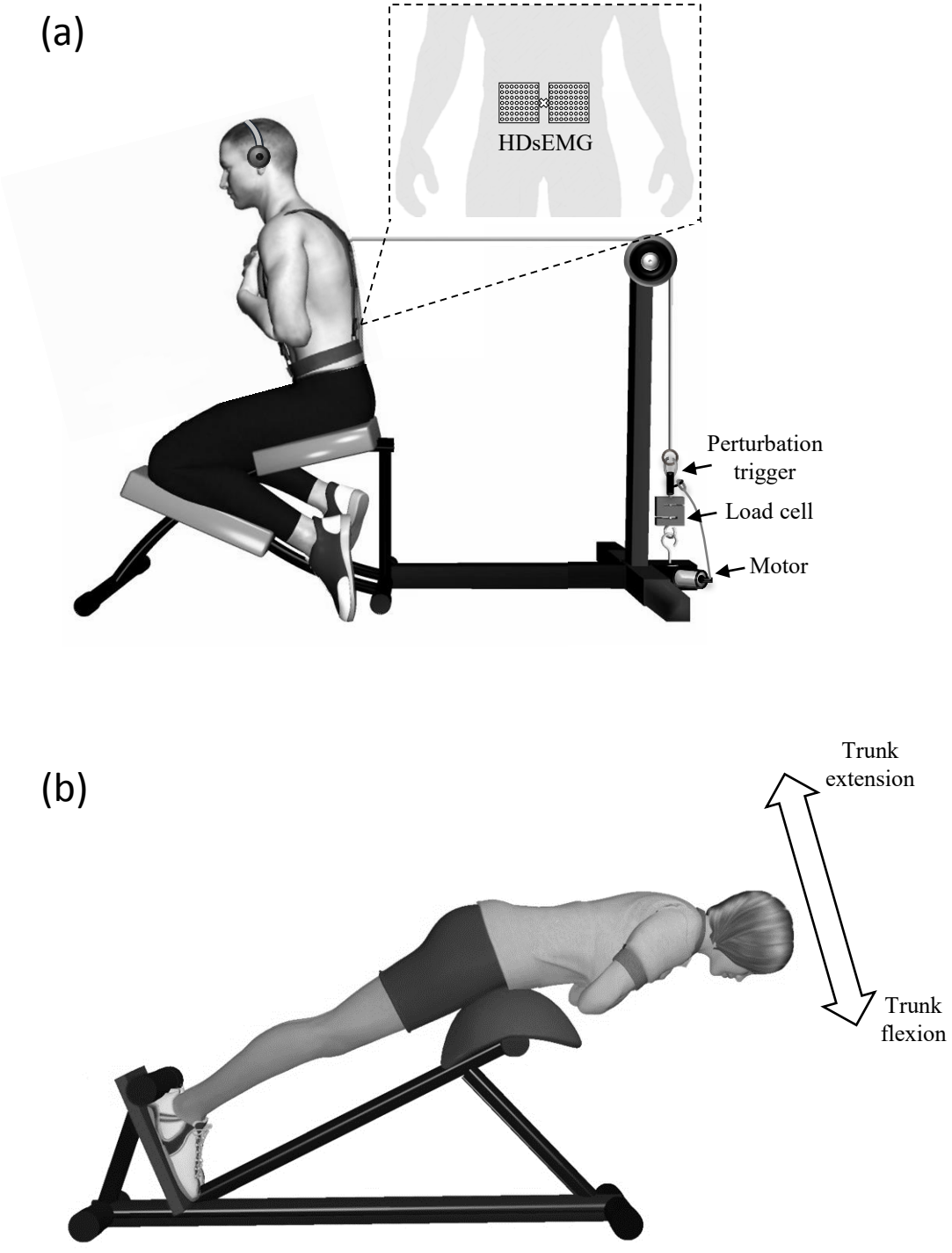

Figure3

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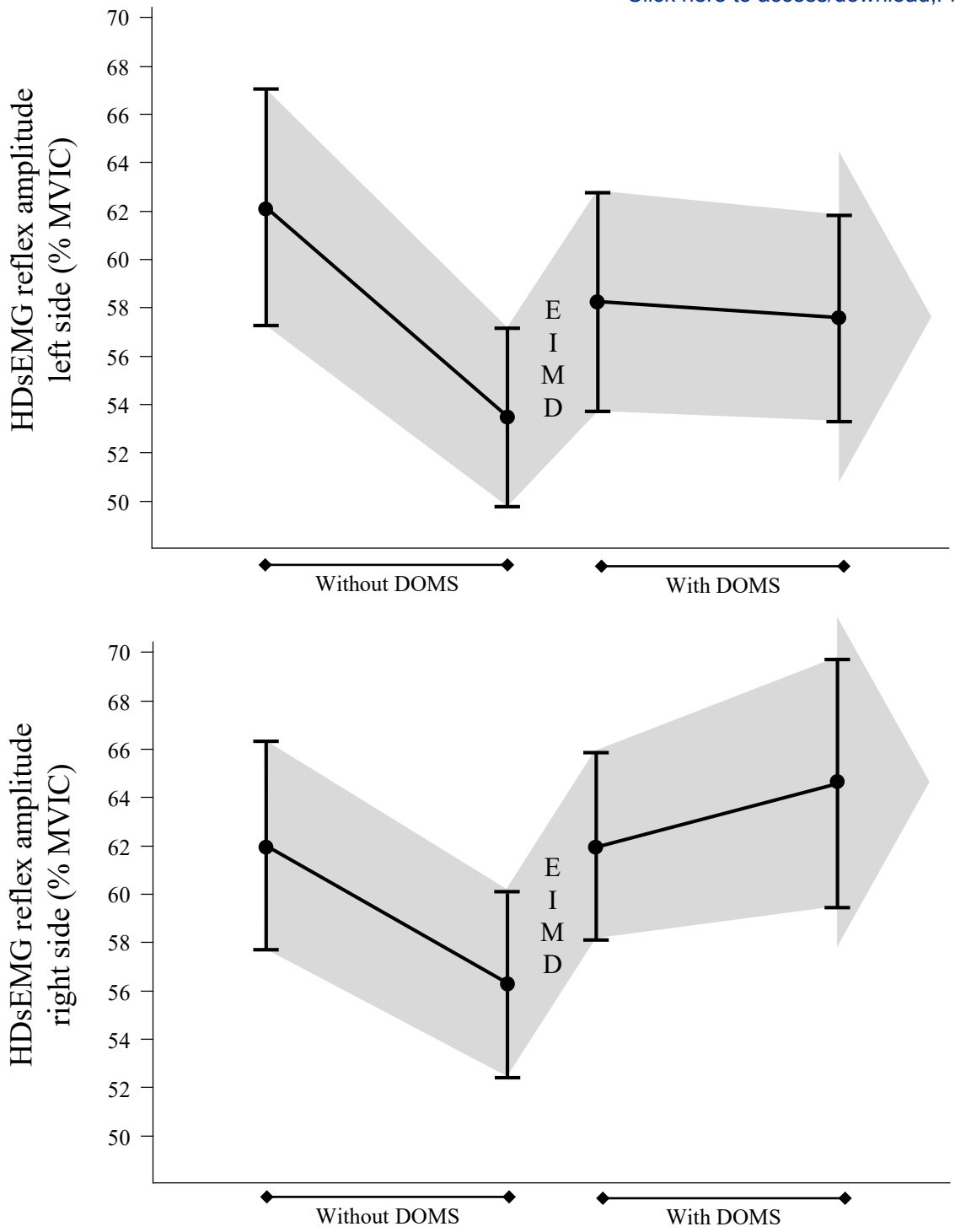

Figure4

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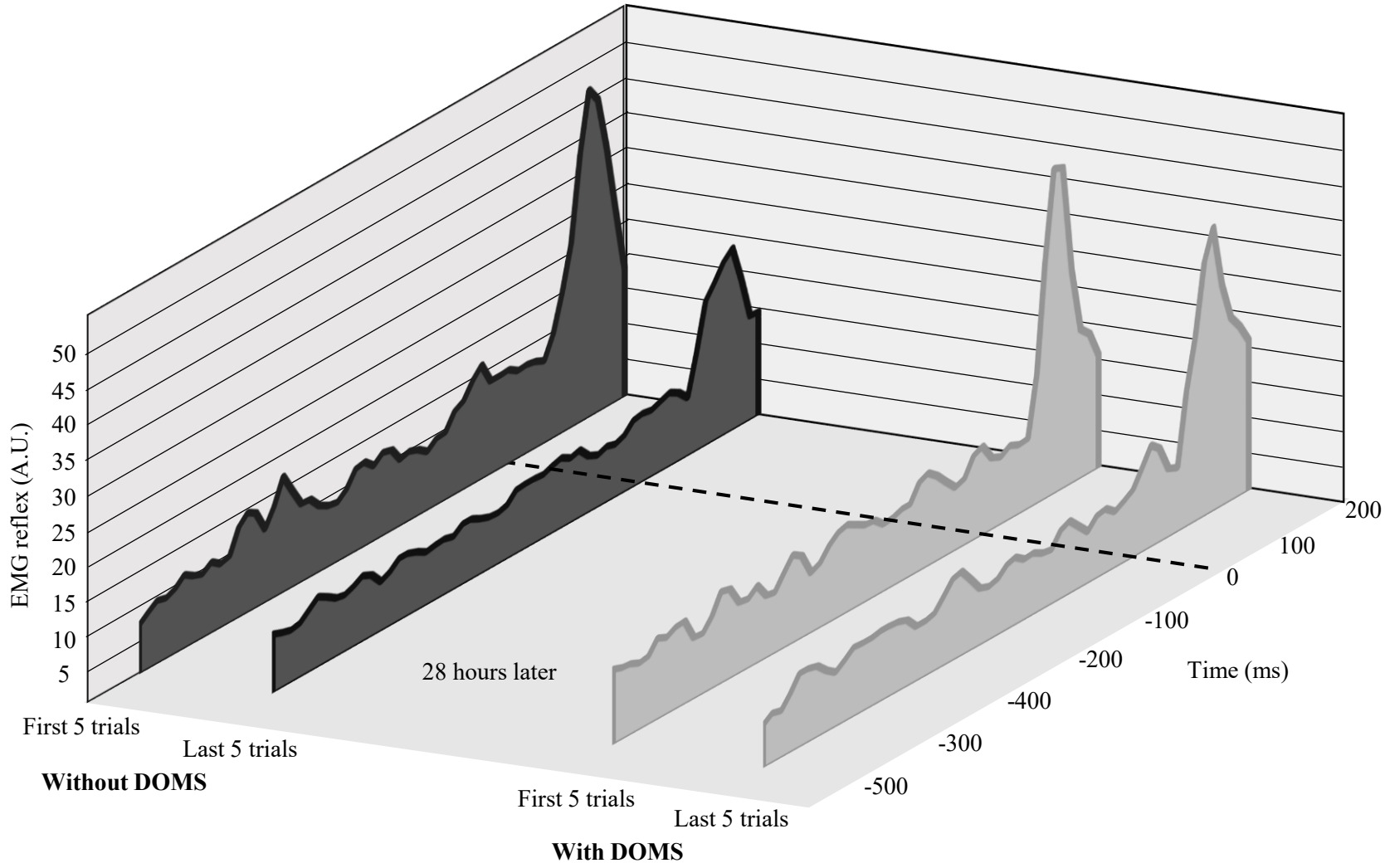

Figure5

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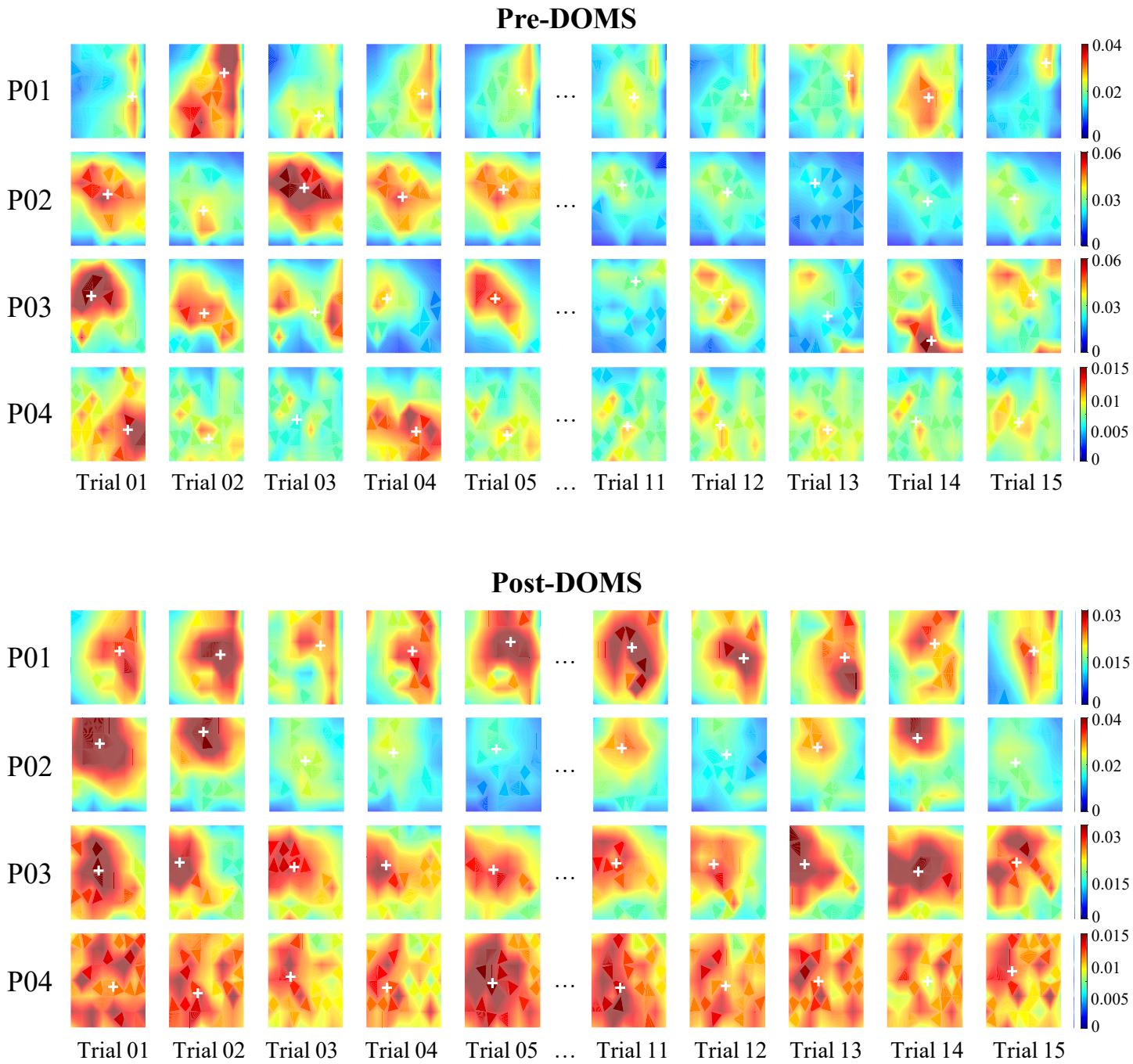

Table1

Day	Hour	Pain (/10)	Soreness (/10)
1	9h	1.9±1.9	2.9±1.6
	15h	2.0±1.9	2.6±1.6
	21h	1.9±2.0	2.7±1.8
2	9h	1.3±1.6	2.0±1.6
	15h	1.0±1.5	1.4±1.5
	21h	0.7±1.0	0.9±1.0
3	9h	0.4±0.6	0.4±0.7
	15h	0.3±0.4	0.5±0.8
	21h	0.3±0.6	0.3±0.7

Table2

			First 5 trials mean (SD)	Last 5 trials mean (SD)	*p**		
					Condition	Adaptation	Interaction
Baseline activity (% MVIC)	L	Pre-DOMS	24 (10)	22 (9)	0.30	**0.049**	0.30
		Post-DOMS	26 (13)	22 (9)			
	R	Pre-DOMS	29 (12)	28 (10)	0.42	0.44	0.39
		Post-DOMS	31 (16)	31 (20)			
Reflex latency (ms)	L	Pre-DOMS	95 (23)	94 (17)	0.47	0.57	0.31
		Post-DOMS	91 (14)	94 (16)			
	R	Pre-DOMS	102 (21)	103 (15)	0.13	0.69	0.90
		Post-DOMS	95 (18)	97 (19)			
HDsEMG reflex amplitude (% MVIC)	L	Pre-DOMS	62 (21)	53 (16)	0.98	0.05	**0.02**
		Post-DOMS	58 (19)	62 (16)			
	R	Pre-DOMS	62 (18)	56 (16)	0.32	0.50	0.07
		Post-DOMS	58 (18)	65 (22)			
Trunk angle (°)		Pre-DOMS	6.8 (4.9)	6.1 (4.3)	0.60	0.82	0.08
		Post-DOMS	6.4 (4.2)	7.2 (5.1)			

*Based on repeated-measures ANOVA. L: left side of the erector spinae; R: right side of the erector spinae)

Author contribution statement: J.A. and M.D. conceived and designed the experiment. J.A. and A.L. undertook the experimental work and analysis. All authors drafted the manuscript or reviewed it critically for important intellectual content. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.