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ANATOMY

ISSLS Prize Winner: Smudging the Motor Brain in Young Adults With Recurrent Low Back Pain

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Study Design. Cross-sectional design.

Objective. To investigate whether recurrent low back pain (LBP) is associated with changes in motor cortical representation of different paraspinal muscle fascicles.

Summary of Background Data. Fascicles of the lumbar paraspinal muscles are differentially activated during function. Human studies indicate this may be associated with a spatially separate array of neuronal networks at the motor cortex. Loss of discrete control of paraspinal muscle fascicles in LBP may be because of changes in cortical organization.

Methods. Data were collected from 9 individuals with recurrent unilateral LBP and compared with 11 healthy participants from an earlier study. Fine-wire electrodes selectively recorded myoelectric activity from short/deep fascicles of deep multifidus (DM) and long/superficial fascicles of *longissimus erector spinae* (LES), bilaterally. Motor cortical organization was investigated using transcranial magnetic stimulation at different scalp sites to evoke responses in paraspinal muscles. Location of cortical representation (center of gravity; CoG) and motor excitability (map volume) were compared between healthy and LBP groups.

Results. Individuals with LBP had a more posterior location of LES center of gravity, which overlapped with that for DM on both hemispheres. In healthy individuals, LES center of gravity was located separately at a more anterior location to that for DM. Map volume was reduced in LBP compared to healthy individual across muscles.

Conclusion. The findings highlight that LBP is associated with a loss of discrete cortical organization of inputs to back muscles. Increased overlap in motor cortical representation of DM and LES may underpin loss of differential activation in this group. The results further unravel the neurophysiological mechanisms of motor changes in recurrent LBP and suggest motor rehabilitation that includes training of differential activation of the paraspinal muscles may be required to restore optimal control in LBP.

Key words: low back pain, motor control, motor cortex organization, lumbar paraspinal muscles. **Spine 2011;36:1721–1727**

Low back pain (LBP) is associated with altered back muscle activation,[1–3] but the mechanisms are poorly understood. Although changes in muscle morphology,[4–6] denervation from nerve root lesions,[7] and reflex inhibition[6] are implicated, changes in descending control by supraspinal centers may contribute.[8–11] Unraveling the mechanisms that underpin motor changes with LBP has important implications for rehabilitation. For instance, interventions to reverse muscle atrophy[12] would differ from those to alter muscle recruitment.[13] Recent insights into motor cortex organization using noninvasive magnetic brain stimulation provide a unique opportunity to explore neural mechanisms associated with movement changes in pain.[8,11]

An observation that may provide insight into the mechanisms underlying motor control changes with pain is altered coordination between functionally distinct paraspinal muscle fascicles. Short/deep and long/superficial back muscle fascicles are differentially activated in healthy individuals in a range of tasks.[14–16] For instance, change in spinal curvature from flat to more lordosed postures increases activity of the medially positioned multifidus muscle, whereas, this change has limited effect on the iliocostalis muscle.[15,16] Furthermore, predictable challenges to the trunk are associated with earlier activation of the short/deep fascicles of deep multifidus (DM) than the long/superficial paraspinal muscles.[14,17] Such discrete activation argues for fine control of the complex array of back muscle fascicles in healthy individuals.

Differential control of paraspinal muscles is compromised in LBP. In this group, paraspinal muscle fascicles tend to be recruited *en masse* with similar activation of iliocostalis and DM with changes in spinal curvature in sitting,[18] and simultaneous activation of deep and superficial multifidus with trunk perturbation.[14] The mechanisms for loss of discrete control are unclear, but one possibility can be gleaned from another painful condition associated with loss of discrete control—focal hand dystonia. In that condition, loss of discrete cortical organization of somatosensory regions associated with each finger[19] (*i.e.*, cortical map "smudging") contributes to reduced

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ability to isolate finger movements. Changes in brain organization may underlie changes in paraspinal muscle activation.

Recent brain mapping studies using transcranial magnetic stimulation (TMS) suggest separate regions of the motor cortex underpin discrete activation in healthy individuals.[20] That work showed a more posterior region of the motor cortex associated with corticospinal input to DM compared to that for the long/superficial fascicles of the *longissimus erector spinae* (LES). Although organization of these cortical networks has not been studied in LBP, responsiveness (excitability) of corticomotor pathways to paraspinal muscles using TMS is reduced[11] and the motor cortical map for other trunk muscles is reorganized.[8] Those studies showed an association between cortical changes and changes in pain intensity,[11] or motor control.[8] Central mechanisms may contribute to deficits in differential control of paraspinal muscles.

This study aimed to investigate organization of areas of the motor cortex associated with inputs to different paraspinal muscle fascicles in individuals with recurring episodes of LBP. Identification of smudging between discrete cortical networks with outputs to paraspinal muscle fascicles would provide novel and convincing evidence of supraspinal mechanisms underlying motor adaptation with pain.

MATERIALS AND METHODS

Participants

Nine right-handed individuals with recurrent LBP were recruited and data were compared to that for 11 right-handed healthy participants reported previously.[20] Participants were included in the LBP group if they had nonspecific episodic unilateral LBP (± buttock pain) lasting longer than 3 months with periods of symptom aggravation and remission. Each episode was to last at least 1 week with sufficient intensity to limit function. LBP participants had minimal pain at time of testing and symptoms were not aggravated by experimental procedures. Pain-free participants were included in the earlier study if they had no history of back or lower limb pain or injury that limited their function and/or required treatment from a health profession. Participants were excluded from both groups if they had any major circulatory, neurological, respiratory disorders, recent or current pregnancies, previous spinal surgery, analgesic or anti-inflammatory medication in the past month, or participation in trunk muscle exercise in the past 12 months. Subject demographics are shown in Table 1. The institutional medical research ethics committee approved the study and procedures conformed to the Declaration of Helsinki.

TABLE 1. Subject Demographics (Mean ± SD)*

	Healthy (n = 11)	LBP (n = 9)
Age (yr)	24 ± 5	25 ± 3.4
Sex (female/male)	6/5	5/4
Weight (kg)	62 ± 10	56 ± 15
Height (cm)	170 ± 8	164 ± 12
Pain VAS (0–10 cm)	…	4.7 ± 1.1
Pain duration (Yr)	…	3.6 ± 2.4
Pain side (right/left)	…	5/4

**Note individual with low back pain (LBP) rated their average pain intensity over the last 6 months on a visual analog scale (VAS). Note pain duration is the total time that participants had experienced recurrent LBP and including periods of aggravation and remission. No difference was detected between the two groups in age, weight, and height (The Student t test, $P > 0.82$).*

Electromyography

Fine-wire intramuscular electrodes recorded electromyographic (EMG) activity from the paraspinal muscles (Teflon-coated stainless steel wires, 75 um diameter with 1 mm Teflon removed and tips bent back ~1 and ~2 mm to form hooks). Electrodes were inserted *via* hypodermic needles with ultrasound guidance into DM and LES bilaterally (Figure 1), adjacent to the L4 spinous process.[14] A ground electrode was placed over the ribcage. EMG data were amplified 2000 times, band-pass filtered between 20 and 1000 Hz and sampled at 2000 Hz using a Power1401 Data Acquisition System with Spike2 software (Cambridge Electronic Design, Cambridge, UK).

Motor Cortex Mapping

TMS *via* a single-pulse monophasic Magstim 200[2] (Magstim Company, UK) was used to map the motor cortex (Figure 1). As motor-evoked potentials (MEPs) were difficult to elicit at rest,[21–23] motor cortex stimulation was conducted during submaximal paraspinal muscle contractions. Subjects sat comfortably with their arms crossed and feet on the floor. Three maximum voluntary contractions of the paraspinal muscles against resistance for approximately 3 seconds were used to determine submaximal contraction intensity. The highest root-mean-square EMG for 1 second was identified from the DM bilaterally. The target DM activation during TMS was set at approximately 20% of this value (feedback provided on a monitor) and achieved by leaning forward with the back straight.[20,22]

Motor cortex mapping was conducted using a 7-cm figure-of-eight coil to excite neuronal networks associated with paraspinal muscles. The coil handle was oriented along the sagittal plane to induce currents in an anterior direction. This coil orientation provides consistent paraspinal MEPs with minimal concurrent excitation of the opposite hemisphere.[22] Subjects wore a tight-fitting bathing cap and the vertex was determined using the International 10/20 system.[24] Using a stimulator intensity of 100%, stimuli were delivered over premarked scalp sites on a 5 × 7-cm grid over each hemisphere (0–5 cm lateral, and from 5-cm anterior to 2-cm posterior to the vertex; Figure 1).[8] Five stimuli were delivered at each cross on the grid (interstimulus interval: ~5 s).[25] Participants rested for 1 minute after stimulation of each site to minimize fatigue.

Data Analysis

EMG was full-wave rectified and MEPs were averaged at each scalp site. This was plotted with individual traces for visual

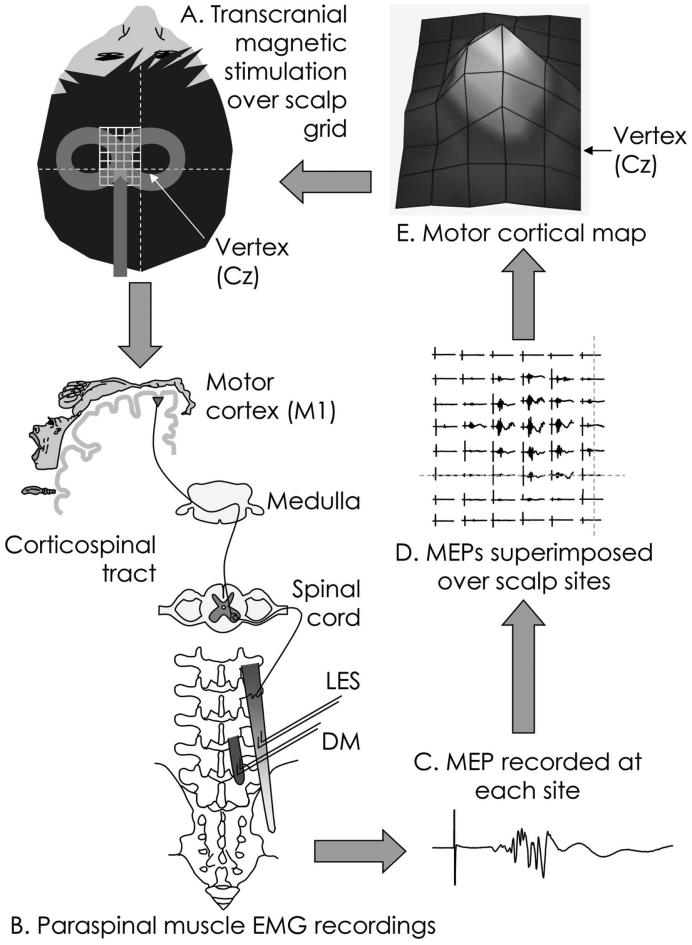

Figure 1. Mapping of the motor cortex using transcranial magnetic stimulation (TMS). **A,** Stimuli over the motor cortex excite intracortical neurons that provide synaptic input to corticospinal cells. **B,** Recordings were made from short/deep fascicles of multifidus (DM) and *longissimus erector spinae* (LES) at the L4 level. **C,** The descending volley excites the spinal motoneurons and results in a motor-evoked potential (MEP), mainly in the contralateral muscles. **D,** MEPs were recorded in both muscles from stimuli applied at each point on the grid placed over the scalp and aligned to the vertex (Cz). **E**, A three-dimensional map of MEP amplitude was created for each muscle from the individual and then group data.

identification of MEP onset and offset. MEP amplitude was calculated as the root-mean-square EMG amplitude between the onset and offset, and background root-mean-square EMG from 55 to 5 m prior to stimulation was subtracted. MEP amplitudes were averaged at each scalp site across subjects after normalization to the peak response to produce a topographical map (Figure 1). Normalized values less than 25% of peak values were removed and the remaining responses were rescaled from 0% to 100%. Removal of small amplitude MEPs minimally affects TMS maps.[26]

Two parameters were calculated from normalized maps. Map volume (measure of total motor cortical excitability) was calculated as the sum of normalized MEP amplitudes across scalp sites with responses. Center of gravity (CoG) was calculated using the formula CoG = $\sum z_i x_i / \sum z_i$; $\sum z_i y_i / \sum z_i$ (x_i – mediolateral location; y_i – anteroposterior location; z_i –normalized amplitude). This measure provides an amplitude-weighted indication of map position. Although map volume is sensitive to changes in corticomotor excitability, the CoG is a valid and repeatable measure of a muscle's motor cortex representation.[26,27]

Statistical Analysis

Statistica 7 was used for analysis (Statsoft, USA). Map volume, CoG location, and MEP onset at the optimal site were compared between muscles, group, and side using repeated measures analyses of variance. The Duncan test was used for *post hoc* analyses. A separate analysis compared CoG location, map volume, and MEP onset between the painful and nonpainful sides (Pain) for the LBP group. The relationship between pain intensity/duration and cortical measures (CoG coordinates and map volume) were investigated using the Pearson correlation. Significance was set at $P < 0.05$.

RESULTS

Data were rejected for left DM and right LES from one LBP participant each because of poor EMG quality. It was difficult to elicit MEPs ipsilateral to the stimulated hemisphere in all but one LBP participant. As MEPs contralateral to the stimulated cortex were consistently elicited, mapping was only undertaken using this data.

Figure 2 shows rescaled normalized TMS maps for the LBP group and the previous control data.[20] CoG location did not differ between left and right sides ($P = 0.69$), or between painful and nonpainful sides in the LBP group ($P = 0.15$). In healthy individuals, DM CoG was located posteriorly to that for LES (interaction – pain × muscle: $P < 0.001$, *post hoc*: $P < 0.001$; Figure 3). In contrast, for the LBP group, DM CoG was located similarly to that for LES (Figures 3, 4; post hoc: $P > 0.23$). This is explained by a more posterior location of the LES CoG in the LBP group compared to controls (post hoc: $P < 0.001$). There was no significant association between CoG

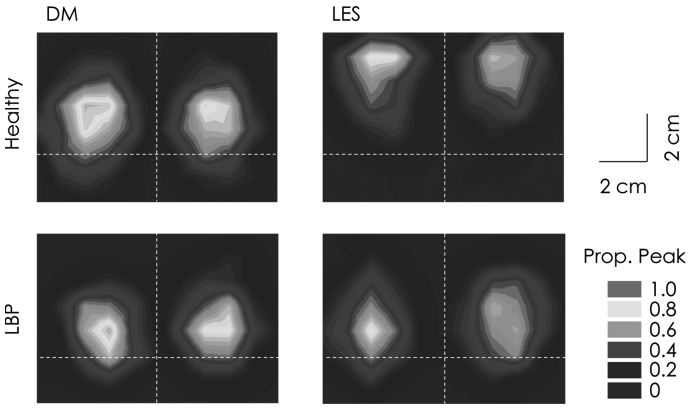

Figure 2. Normalized maps of the left and right motor cortex for short/deep fibres of multifidus (DM; left panel) and *longissimus erector spinae* (LES; right panel), for healthy (top panel) and low back pain (LBP) groups (bottom panel). Dotted lines denote sagittal and frontal planes, intersecting at the vertex. Note motor cortical maps for DM overlap that for LES in the LBP group, whereas DM is located posteriorly compared to LES in healthy group.

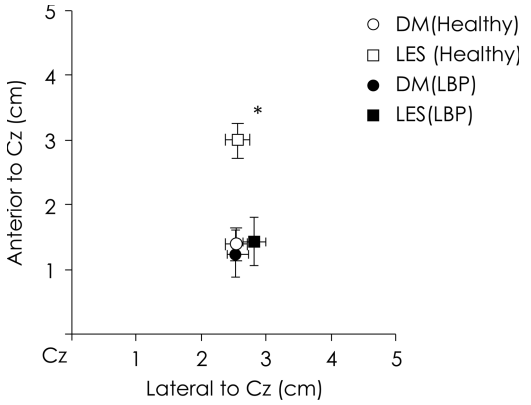

Figure 3. Group data for center of gravity (CoG) of short/deep fibres of multifidus (DM) and *longissimus erector spinae* (LES) in healthy and low back pain (LBP). Mean and 95% confidence interval (averaged across sides) are shown. *$P < 0.05$. Cz indicates vertex

location and pain intensity (all $r^2 < 0.15$; all $P > 0.30$) or duration (all $r^2 < 0.35$; all $P > 0.09$).

Figure 5 shows map volume for healthy and LBP groups. Map volume was reduced in LBP compared to controls (main effect – pain: $P < 0.001$; *post hoc*: $P < 0.021$), although this did not differ between muscles ($P = 0.26$) or sides ($P = 0.68$). Reduced map volume suggests reduced corticomotor excitability in LBP, but this was not correlated with pain intensity ($r^2 < 0.18$; $P > 0.25$) or duration ($r^2 < 0.23$; $P > 0.19$).

The latency to onset of MEPs was shorter for DM compared to LES in both groups (muscle: $P < 0.001$; *post hoc* $P < 0.001$), but no difference was observed between group ($P > 0.35$) and sides ($P > 0.39$). There was no difference in MEP onset between painful and nonpainful side in the LBP group ($P > 0.19$; Averaged across sides: healthy - DM = 15.8 ± 1.1 m, LES = 18.0 ± 3.0 m; LBP - DM = 15.2 ± 1.5 m, LES = 18.3 ± 4.0 m).

DISCUSSION

These results show for the first time loss of discrete organization of neuronal networks that control functionally distinct

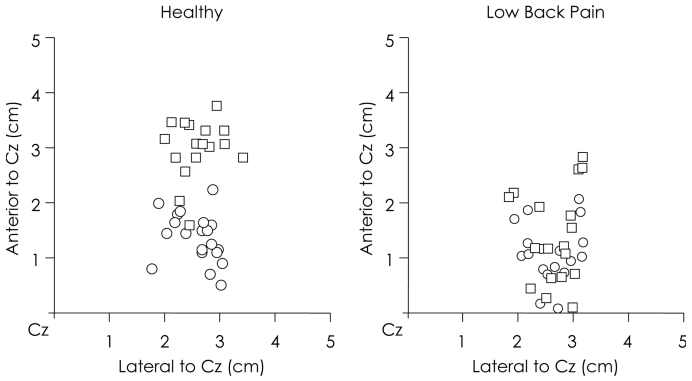

Figure 4. Individual data for center of gravity (CoG) of short/deep fibres of multifidus (DM; circles) and *longissimus erector spinae* (LES; squares) in healthy and low back pain (LBP). Data for left/right and pain/nonpainful sides were not different and are not separately identified in the figure. Cz indicates vertex.

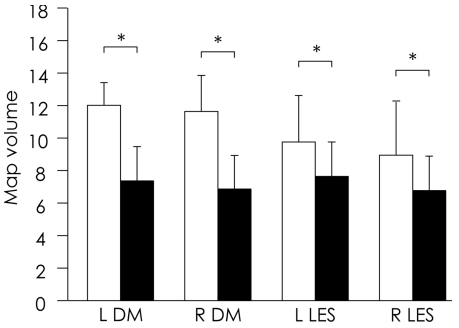

Figure 5. Map volume of short/deep fibres of multifidus (DM) and *longissimus erector spinae* (LES) for healthy (white) and low back pain (LBP; black) groups. Mean and 95% confidence intervals are shown. Note reduced map volume in LBP group compared with healthy individuals. *$P < 0.05$. R indicates right; L, left.

paraspinal muscle fascicles in LBP. Individuals with recurrent LBP had overlapped motor cortical areas for DM and LES, which contrasts recent evidence of discretely organized cortical inputs to these muscles in healthy individuals.[20] This provides novel evidence of a neural substrate that may underlie reduced potential to differentially activate parts of the paraspinal muscles during function in LBP. This work has implications for motor rehabilitation that aims to restore optimal control of paraspinal muscles for management of LBP.

Smudging of Motor Cortical Organization and Reduced Motor Excitability in LBP

Paraspinal muscles receive descending input from the motor cortex and corticospinal pathways.[23,28] These pathways not only contribute to voluntary activation, but also to postural control. For instance, motor cortical stimulation in cats elicits flexion of the contralateral forelimb and anticipatory postural adjustments in the supporting forelimb.[29] Human data demonstrate reduced trunk muscle postural activity with limb movements when motor cortex is inhibited with submotor-threshold TMS.[30] Furthermore, recent work confirms a relationship between organization of cortical neural networks and temporal parameters of postural activation of trunk muscles.[8,9] This suggests motor cortex contribution to a range of motor functions including postural activity of trunk muscles.

LBP is associated with adaptive changes in organization of cortical neuronal networks.[8,9,11] This shows these pain-related cortical changes include smudging of cortical representation of functionally distinct paraspinal muscles fascicles. The posterior shift in LES representation reflects motor cortex reorganization and cannot be accounted for by changes in excitability.[26,27]

Smudging of motor cortical organization has functional implications for neural control of paraspinal muscles. It may reduce the potential to independently control these muscles. The degree of differential motor control is related to discrete organization of motor cortical networks; for example, discrete representation of individual fingers on the primary motor and sensory cortices are related to independent fine control of finger movements.[31] Reduced differentiation of this discrete organization, such as smudging between areas

representing adjacent fingers in focal hand dystonia, reduces the isolation of finger movements.[19] Smudging of motor cortical representations of DM and LES may explain observations of reduced discrete activation of different paraspinal muscle fascicles in LBP.[14,18] We did not investigate paraspinal muscle activity in functional tasks. Thus, we cannot directly investigate the relationship between organization and function. Nevertheless, this work provides the basis for studies into the relationship between cortical remodeling and altered trunk muscle coordination.

Motor cortex reorganization supports the notion that the nervous system adopts a new strategy for movement/stability with LBP.[32] It has been hypothesized that in the presence of pain and/or injury, the nervous system implements new motor strategies to "protect the part" from further injury/pain.[33,34] This is often mediated by increased trunk muscle activity, particularly large superficial muscles, to splint the spine. This is supported by several observations. First, when pain is induced in healthy individuals, trunk muscle EMG increases in keeping with the goal to protect the spine, although the pattern varies between individuals.[35] Second, biomechanical modeling suggests various patterns of muscle activity observed in LBP increase trunk stability.[36] In this case, fine coordination between different fascicles of the paraspinal muscles may no longer be used *in lieu* of a simplified protective strategy. That is, nervous system remodeling (including smudging of motor cortex) may simplify the motor strategy to a more *en masse* paraspinal muscle recruitment. Reorganization of LES but not DM appears consistent with the proposal that reorganization of inputs to long/superficial fascicles underpins the adaptive search for a new motor strategy.[34,37]

Motor maps exhibited the same organization bilaterally in LBP, despite selection of patients on the basis of unilateral symptoms. The isolation of some motor control changes to the painful side[14] may question the association between motor control and symmetrical changes in cortical organization. However, many changes in motor control are not systematically related to the side of pain. For instance, unilateral experimental pain induces variation in motor coordination that is not exclusive to the pain side.[35] The nervous system appears to select a protective solution that is generic, regardless of the location/side of pain. Further investigation is required to examine cortex reorganization in other LBP subgroups (*e.g.*, bilateral LBP, specific spinal pathology).

DM and LES map volume was reduced in LBP. This suggests either reduced corticomotor excitability or that neural networks with input to these muscles occupy a smaller area of cortex. Reduced excitability has been interpreted from earlier data of increased paraspinal muscle motor threshold to TMS[11] (*i.e.*, greater stimulation intensity to evoke a response). However, it is not possible to determine whether those changes were because of effects at the cortex or spinal cord as excitability of both may affect the responsiveness to TMS.[38] Recent animal[39] and human[40] studies highlight opposite changes at the cortex and spinal cord, making interpretation impossible without techniques that isolate measures to a specific site. In animal studies, responsiveness to stimulation over the brain increased after a disc lesion, but responsiveness of spinal pathways reduced.[39] Thus, although reduced corticomotor excitability would seem to contradict the suggestion of enhanced activation of long/superficial fascicles (such as the LES) to protect the spine from further injury, the reduced responsiveness to TMS could simply reflect the complex interaction of effects of pain along the motor pathway. The possibility that reduced map volume reflects a more focal cortical network cannot be confirmed as reduced excitability of spinal motoneurons cannot be excluded. Fortunately, changes of map organization, the key observation in this study, do not reflect changes in excitability and interpretation is less problematic.

Although current data provide evidence of changes to the motor cortex and corticospinal tracts, the exact mechanisms of how pain induces such changes require further investigation. Changes in other parts of the nervous system could contribute to altered motor coordination (*e.g.*, basal ganglia, cerebellar, and spinal circuits).[41] Exactly how different networks of the sensorimotor system contribute to changes in motor coordination requires further work.

Relationship Between Cortical Changes and Pain

Previous work has identified a relationship between pain and cortical changes. Amplitudes of changes in the sensory representation of the back[42] and motor threshold[11] are related to pain duration. No relationship was identified in our data. This could be because of the small sample size or lesser pain intensity than previous studies.[11] However, pain report is subjective and influenced by a complex array of biological and psychological (*e.g.*, catastrophization) factors.[43,44] The lack of association between pain and motor cortical parameters may reflect this complex interaction.

Methodological Issues

The main limitation of the study is the small sample size. Yet, despite the small sample, findings were consistent across subjects and yielded significant results. For practical reasons, subjects were only required to match DM EMG during TMS. We did not control LES EMG. Although this may increase intersubject variability in motor cortical excitability for LES, this does not compromise our main finding as CoG measures are less affected by motor excitability.[26] Furthermore, this study involved young adults and studies of older adult population should be considered.

Clinical Implications

A loss of differential activation of paraspinal muscle fascicles is likely to compromise fine-tuned segmental control and alter loading of spinal structures,[34] potentially increasing the risk of LBP recurrence.[33,34] As our data show that such motor changes may be mediated by loss of discrete organization at the motor cortex, it could be argued that a goal of LBP rehabilitation, at least initially, should include motor training to restore cortical organization. Although it is beyond this study to speculate on how best to restore cortical organization, studies in focal hand dystonia suggest redifferentiation of discrete finger representation is possible with training of isolated finger

movements.[19] In addition, motor training with specific activation of deep abdominal muscles can reverse pain-associated reorganization at the motor cortex[9] and this is associated with improved motor behavior.[9,13,45] A recent study of a small group with LBP shows training that targets DM can improve coordination of the paraspinal muscle fascicles during functional tasks toward that observed in healthy individuals.[46] Although motor rehabilitation should not be exclusive to DM, changes in paraspinal muscle activation after motor training provide a marker for further investigations to study whether these improvements are associated with redifferentiation of muscle representation at the motor cortex (and improvement in clinical symptoms).

➢ Key Points

- ❑ Fascicles of the paraspinal muscles are differentially activated during function.
- ❑ Differential activation is associated with discrete organization of neuronal networks at the motor cortex.
- ❑ Loss of discrete organization of different fascicles of the paraspinal muscles at the motor cortex is observed in individuals with recurrent low back pain.
- ❑ Reorganization at the motor cortex may underpin this loss of differential activation in low back pain.

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