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Motor Control Changes in Low Back Pain: Divergence in Presentations and Mechanisms

There is no question that many people with low back pain (LBP) move differently than do those without pain, but the mechanism of and reason for these motor control changes are poorly understood. There are several major challenges with interpreting current literature, particularly regarding how to reconcile the enormous interindividual variation in presentation. Motor control is defined here as the way in which the nervous system controls posture and movement to perform a specific motor task, and includes consideration of all the associated motor, sensory, and integrative processes. Given the redundancy in the musculoskeletal system, the

nervous system has flexibility in how different muscles and joints are recruited to achieve a motor task.

The quality of the control process is reflected in how well a posture is main-

tained or a movement is achieved in response to specific demands. Trunk posture and movement are continuously perturbed by neuromuscular noise (ie, the imprecision in our control system), concurrent motor tasks such as breathing,³⁴ and external mechanical perturbations such as the impact forces at ground contact in walking.⁴⁶ These perturbations are dealt with by modulating trunk stiffness through tonic muscle activity,^{8,33,121} anticipatory/feedforward control,^{39,118} and feedback based on proprioceptive, visual, tactile, and vestibular information.^{1,18,62,79} Since the early observations of differences in muscle activation in individuals with LBP, it has been generally considered that many, if not all, of those with LBP present with some change in motor control.

In section 1 of this article, *Is Motor Control Different Between Individuals With and Without LBP?*, we consider the current state of the evidence regarding changes in motor control in individuals with LBP and conclude that findings on motor control in LBP are largely inconsistent. This illustrates the danger of basing interpretations on a limited number of studies. Published data support a specific

• **SYNOPSIS:** Compared to healthy individuals, patients with low back pain demonstrate differences in all aspects of trunk motor control that are most often studied as differences in muscle activity and kinematics. However, differences in these aspects of motor control are largely inconsistent. We propose that this may reflect the existence of 2 phenotypes or possibly the ends of a spectrum, with “tight control” over trunk movement at one end and “loose control” at the other. Both may have beneficial effects, with tight control protecting against large tissue strains from uncontrolled movement and loose control protecting against high muscle forces and resulting spinal compression. Both may also have long-term negative consequences. For example, whereas tight control may cause high compressive loading on the spine

and sustained muscle activity, loose control may cause excessive tensile strains of tissues. Moreover, both phenotypes could be the result of either an adaptation process aimed at protecting the low back or direct interference of low back pain and related changes with trunk motor control. The existence of such phenotypes would suggest different motor control exercise interventions. Although some promising data supporting these phenotypes have been reported, it remains to be shown whether these phenotypes are valid, how treatment can be targeted to these phenotypes, and whether this targeting yields superior clinical outcomes. *J Orthop Sports Phys Ther* 2019;49(6):370-379. Epub 12 Jun 2018. doi:10.2519/jospt.2019.7917

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interpretation of motor control changes in those with LBP, but a similar number of studies contradict this interpretation.

In section 2, Divergence of Motor Control Features in LBP, we propose an interpretation of the large individual variation in motor control changes in those with LBP. We suggest that it may reflect the existence of 2 different phenotypes resulting from adaptations in motor control to LBP and interference of LBP with motor control. Furthermore, we discuss the relevance that the existence of such phenotypes would have for LBP.

Finally, in section 3, Implications for Clinical Approaches to Address Motor Control Adaptation, we present clinical implications and considerations for future development in this field. The interpretation of the literature on motor control in individuals with LBP proposed here requires further validation and, hence, cannot be translated directly into guidelines for clinical practice; however, if correct, this interpretation provides a framework for further research and clinical reasoning.

Is Motor Control Different Between Individuals With and Without LBP?

In relation to LBP, motor control has been studied at the level of the neural structures and processes involved,^{47,107,108,132} but more commonly at the level of patterns of trunk muscle activity and trunk movements, which represent the outcomes of these processes. Evaluation of the sensory elements of motor control has largely been limited to conscious repositioning tasks and responses to muscle vibration. The following sections present a brief overview of the evidence for motor control changes in individuals with LBP.

Is Trunk Muscle Activity Different Between Individuals With and Without LBP? In general, investigations of motor control in people with LBP have separately considered 3 main classes of motor tasks: control of the trunk in steady-state posture and movement, control of trunk posture and movement when challenged by predictable perturbations (anticipatory/

feedforward control), and control of trunk posture and movement when challenged by unpredictable perturbations (reactive/feedback control).

Theoretical models and empirical observations indicate that both excitatory and inhibitory effects on muscle activity may result from injury and nociception,⁴³ as well as from anticipation or fear of pain.^{80,116} In line with this divergence of effects, a review on differences in lumbar extensor muscle activity during steady-state tasks between individuals with non-specific LBP and pain-free participants showed that findings are highly variable when patients are considered as a single homogeneous group. Some studies reported higher lumbar extensor muscle activity in patients, other studies reported no differences, and still other studies reported lower activity in patients.¹²³

Anticipatory activation of trunk muscles has commonly been investigated in association with perturbations of trunk posture caused by rapid movements of the upper and lower limbs, which are inherently predictable with respect to direction, timing, and amplitude of related forces.⁵ Some studies have reported late activation of the transversus abdominis and multifidus muscles in participants with recurrent LBP^{38,40,41,63,72} and in response to an experimental noxious stimulus to the low back.³⁷ In contrast, another study showed no difference in onset of activation of the abdominal muscles between patients with LBP and controls,⁷¹ and 2 other studies showed earlier activation of the oblique abdominal muscles in people with LBP.^{20,78}

A systematic review on reactive trunk motor control in response to mechanical perturbations concluded that delayed onset or offset of muscle activity in patients with LBP compared to healthy participants was found in all but 1 of the included studies, while amplitudes of these responses were highly variable between patients and studies.⁸⁷ Delayed offset of activity of the abdominal muscles following release of a load into trunk extension has been associated with greater risk for

a subsequent episode of LBP in varsity athletes.⁹ This highlights the possible role of motor control changes in the development or recurrence of pain.

Although not directly indicative of motor control deficits, the ability of the muscle to enact the commands from the motor system will determine the ultimate efficacy of motor control, and there is evidence of structural/morphological changes in the trunk muscles with LBP. Specifically, there are substantial data from human imaging^{13,26,27,131} and biopsy^{49,67} studies that show changes in muscle fiber types (transition from fatigue-resistant type I to fatigable type II muscle fibers,^{49,67} muscle atrophy,^{26,27,131} and fatty infiltration¹³) of the multifidus muscle in acute, recurrent, and persistent LBP. Animal models, which allow a more detailed analysis of structural changes, indicate that the muscle not only shows changes in adipose tissue content, but also undergoes a process of fibrosis.^{35,36}

In summary, there is considerable evidence for changes in muscle activation and muscle morphology in individuals with a history of LBP, but the observations vary. Several features may account for this variation in findings. First, the trunk system is highly redundant, with many options available to achieve a similar objective, and different individuals may adopt different solutions for the same outcome.³¹ Second, changes may depend on the specific muscles investigated; deeper muscles, such as the transversus abdominis and multifidus, appear more consistently inhibited,^{38,40,41,63,72} whereas changes in the larger, more superficial muscles are more variable, though activity is often increased.¹²³ Third, differences in motor control may depend on the tasks and contexts investigated. For instance, in anticipation of a perturbation, an individual in pain may be more likely to adopt a strategy of trunk stiffening^{7,121}; consequently, studies that include threatening perturbations may yield different results from those of studies with a less threatening paradigm. Finally, differences in measurement techniques, such

as the use of surface versus intramuscular electromyography, may account for some differences in results between studies.⁶⁶ How to reconcile the individual differences is a major issue, and new hypotheses are presented in section 2.

Are Trunk Alignment, Trunk Posture, and Trunk Movement Different Between Patients and Healthy Individuals? Spine and pelvis alignment have often been considered in relation to LBP. Although many studies failed to find differences between individuals with and without LBP,^{53,127} differences such as greater lumbar flexion/posterior pelvic tilt, lumbar extension, or flattening of the lumbar spine have been identified when specific subgroups within the heterogeneous LBP population were studied.^{10,45,81}

Low back pain is commonly expected to be associated with compromised quality of control of trunk posture and the contribution of the trunk to overall whole-body postural control. Quality of postural control has been studied in several ways, but most frequently as postural sway in standing. These studies have largely identified that individuals with LBP tend to display larger postural sway, but this finding is not universal,⁷³ and interpretation is complicated by the potential capacity to compensate for changes in spine function with increased reliance on postural adjustments from the lower limbs.¹⁰⁰ A limited number of studies focused more specifically on postural control of the trunk in tasks that reduce the contribution of the lower limbs to balance control, such as seated balancing and standing on a narrow beam. Some studies showed worse balance performance in patients with LBP,^{76,89,114} but others did not find a difference between participants with and without LBP.^{59,122,134}

In dynamic movement tasks, trunk movements are usually performed more slowly by participants with LBP than by those without LBP.⁵³ In addition, some studies reported a stronger coupling of pelvis and thorax movements and reduced variability of trunk movements in gait^{56,57,115} and in repetitive trunk bend-

ing.¹⁴ The opposite observation has also been made, with higher variability of trunk movements in individuals with LBP than in pain-free individuals during gait,^{55,130} reaching movements,⁹⁷ and repetitive trunk bending.²

Inconsistency between studies regarding variability of trunk movement requires further reflection, and it is important to distinguish intraindividual variation (variation between repetitions) from interindividual variation (different strategies adopted by different individuals), as well as between variability that negatively affects movement outcomes and variability that does not, as its effect on movement outcomes is compensated at other degrees of freedom in the motor system.⁹⁶ High intraindividual variability may reflect poor control, but may also reflect the ability of individuals to be variable because they can adequately limit variability if needed.¹²⁶ It may also be beneficial to share load between structures¹⁰³ or to provide exposure to new options of movement to aid learning and adaptation.¹⁰² Ambiguity can be avoided by using tasks that require participants to position or move their trunk as precisely as possible. Although only investigated in a limited number of studies, there are indications that patients with LBP are less able to precisely control trunk posture,¹³⁵ trunk movement,^{12,133} and force production by trunk muscles.^{3,11,19,21,86}

When using mechanical perturbations of posture and movement to probe trunk motor control, inconsistent results were found, with smaller initial displacements after perturbations in patients, no significant differences between patients and healthy participants, and even larger initial displacements in patients.⁸⁷

Another paradigm to study movement control has focused on the interaction between adjacent body segments. This work has identified greater and earlier motion of the pelvis and spine during movement of the hip in patients with LBP,^{17,95} but, again, this was specific to some individuals.

As concluded for muscle activation, discussed above, the literature on

changes in trunk alignment, posture, and movement in LBP clearly indicates that differences in trunk motor control are present between participants with and without a history of LBP. However, the literature is also characterized by inconsistency in findings. Methodological differences between studies may account for some of the inconsistency, but the disparities may also be related to variance between patients with LBP, which will be discussed below.

Divergence of Motor Control Features in Individuals With LBP

Overall, the literature regarding motor control in patients with LBP shows inconsistent results. Some methodological explanations for this were addressed above. In addition, many studies included only a small number of participants. As variance in parameters used to characterize motor control has generally been large, the differences between studies may simply be due to chance, which could be addressed by larger studies. Although the literature confirms that motor control may differ between individuals with and without LBP, it also shows that motor control changes are not observed in all patients and not in the same manner. This is no surprise, as heterogeneity in the presentation of individuals with LBP, across all domains from symptoms to response to treatment, is well known. In general, where group means have indicated different control in patients, the variance within groups (between-participant variation) has been substantial, and the range of observations in patient groups has partially overlapped the range of observations in the group of healthy participants.^{56,58,117,133} Furthermore, between various studies, patients have sometimes differed from healthy participants in opposite directions.

Beyond methodological differences between studies, there are possible explanations for the variation between studies and the apparent variation between participants with LBP that have clinical relevance. The clinical literature has pop-

ularized the hypothesis that variation in motor control changes is a consequence of patient subgroups.^{10,94} The foundation for this argument lies in a body of work that has proposed and tested divergence in mechanisms, presentation, and outcomes in patients with LBP.¹²⁵ According to this suggestion, variation between study participants with LBP would directly reflect the presence of subgroups within the heterogeneous LBP population, who present with different characteristic muscle activation, alignment, and movement changes.

Furthermore, differences between studies might be explained by intentional or unintentional biases in patient inclusion (ie, populations may have differed with respect to severity of LBP, psychological factors, or presumed pathology). Finally, such differences may be the consequence of an interaction between the differences between patient subgroups and study context. For instance, individuals with high fear of pain are more likely to stiffen their trunk in anticipation of a perturbation.⁵¹ Consequently, differences between patients with LBP and controls may be more pronounced in patients who are more afraid of pain, especially in somewhat threatening paradigms.

As a starting point to understanding the variation between individual patients, it is important to consider that divergence in motor control presentation may not be explained by a single factor. Differences in presentation might be explained by divergence of the underlying mechanisms for the response to injury/nociceptive input/pain; for instance, the changed motor control may represent a purposeful strategy for protection, or, alternatively, it may be a consequence of interference by pain/nociception and injury.³⁰ From another perspective, the divergence of changes in motor control may be considered with respect to different mechanical consequences of adaptations; for example, in some individuals/contexts, the net outcome of the adaptation may be increased stiffness of the trunk, whereas in others it may be decreased stiffness. Both

proposed models of understanding the divergence in motor control changes (ie, based on underlying mechanisms versus mechanical consequences) can help reconcile some observations and are worthy of further discussion.

Divergence of Mechanisms Underlying Motor Control Changes in Individuals With LBP The literature summarized in the preceding sections is largely based on cross-sectional studies, which do not allow inferences on the direction of causality, if existent, between motor control changes and LBP. Studies that introduced experimental nociceptive input and lesions suggest that many of the differences between patients and healthy individuals can be the direct or indirect effects of pain and/or injury. On the other hand, while, for example, delayed trunk muscle responses after mechanical perturbations can be elicited by experimentally induced pain,⁴ similar changes have been observed to precede LBP and increase LBP risk.⁹ Thus, motor control changes can likely be both a cause and an effect of pain and injury, but we will consider them as effects here.

Injury/nociceptive input and pain are potent stimuli to change motor control, and several mechanism-based theories have been developed to reconcile the diversity of observed changes. These can be distilled into 2 main categories: those that consider the change as a consequence of motivation of the system to adapt as a purposeful strategy to protect the body region from further pain/nociception and injury, and those that consider changes to result from interference by pain/nociception and injury with motor control.

In theories considering motor control changes as purposeful adaptations to avoid pain, it was initially assumed that reflex-like changes induced by nociception cause higher activation of antagonistic muscles and lower activation of agonistic muscles, leading to higher stiffness and slower movement.⁶¹ This view has been criticized based on the variability of empirical findings¹²³ that we have also highlighted. More contemporary

views imply that learning processes play a role in adaptation of motor control to LBP.¹¹⁹ Such learning processes could result in different responses to a seemingly identical stimulus and in association with anticipation or fear of pain and/or (re)injury in the absence of injury or nociceptive input, or in response to pain-related distress.^{25,54,78,80,113,116}

Injury or nociception can directly interfere with motor control, as it can change excitability of motor pathways at different levels of the nervous system. Importantly, it can cause either an increase or decrease of excitability,³² which may account for some of the changes and variability in changes in muscle activation observed in patients with LBP. In addition, nociception may affect proprioceptive afference¹⁰⁵ and, consequently, interfere with motor control. This would be in line with findings of impaired proprioception in patients with LBP,¹⁰⁶ which appears to cause reduced precision in the control of trunk movement.¹³³ Changes observed in LBP in the sensory cortex¹⁶ and in the motor cortex¹⁰⁸ and reduced corticomotor excitability¹⁰⁴ may also interfere with motor control. Finally, structural changes, such as loss of segmental stiffness,^{74,88,137} muscle atrophy,²⁶⁻²⁸ and connective tissue changes,³⁵ will change the relation between motor commands and motor output and may interfere with motor control as a result.

Divergence of Mechanical Consequences of Motor Control Changes in Individuals With LBP The literature on motor control changes with LBP suggests that patterns of change observed can be divided based on their mechanical consequences. One pattern of change, which involves increased excitability of trunk muscles, may provide tight control over lumbar movements at the cost of higher tissue loading.¹¹⁷ This could be the result of increased cocontraction, reflex gains, and/or attention to movement control. The opposite pattern, which involves reduced muscle excitability, might avoid high tissue loading, at the cost of a loose control over movement. These 2 patterns, which are

referred to as “tight control” and “loose control” in the following discussion, may be adaptations to LBP, as suggested by their positive consequences (enhanced control, reduced tissue loading), but may also be caused by interference.

Although plausible, the existence of different phenotypes of patients based on these mechanical consequences of divergent presentations has largely been inferred by data from separate studies. A single study by Reeves et al⁹¹ provides evidence for 2 identifiable subgroups in line with this distinction. In this study, participants with LBP fell into 1 of 2 groups: those who showed preferential activation of lumbar extensors over thoracic extensors, and those who showed the opposite activation pattern. Biomechanical modeling predicts that preferential activation of the lumbar extensor muscles enhances control over lumbar movement, while causing higher tissue loads, and vice versa for preferential recruitment of thoracic extensors.¹¹⁷ This study¹¹⁷ thus provides an indication of the existence of tight control and loose control subgroups with high and low tissue loading, respectively. These subgroups are likely part of a continuum, as a middle group with normal trunk extensor activation was also present. The long-term consequences of, and clinical strategies to address, these responses are likely to be different for such subgroups.

In summary, individuals with LBP may show a spectrum of deviations in motor control, and this will affect mechanical loading on lumbar tissues. In some cases, these changes may be beneficial to the health of the tissue (at least in the short term); in others, the resultant loading may be or become the source of nociceptive input. Tissue loading may not be relevant in all individuals with LBP and is likely to be most important for those who continue to have a contribution of nociceptive input to their ongoing pain. Tissue loading may have enhanced relevance in the presence of peripheral and central sensitization, where lower load magnitudes may be sufficient to

excite sensitized afferents. The potential consequences of tissue loading resulting from motor control changes at the divergent ends of the spectrum require more detailed consideration.

Consequences of Tight Control Tight control implies augmented constraint of movement, presumably with the objective to avoid nociceptive excitation, pain, or injury, or in anticipation of such threats. In the short term, tight control would tend to increase the “safety margin” for control of movement and resulting tissue strains. For example, increased cocontraction and reflex gains would increase trunk stiffness such that greater force would be required to perturb the spine from its position or trajectory. An advantage would be a reduction in the need to intricately control the sequences of muscle activation matched to the task demands, thus reducing the potential for error that may arise when sensory feedback is inaccurate or the force-generating capacity of the muscle has been modified. This strategy would also be expected to reduce variation in movement and the need for finely controlled anticipatory actions and feedback responses to counteract perturbations.

Tight control could be subtle, with slight modifications of activation within a region of a muscle,¹⁰⁹ or more extreme, such as bracing of the body region.^{29,117} Complete avoidance of a task/function that is characteristic of some people with LBP might also be considered as an extreme example of a tightly controlled protection solution.⁴² Although tight control appears logical and beneficial, at least in the short term, it could also have negative consequences. Data showing an association between pain relief after spinal manipulation and a reduction in lumbar stiffness suggest that stiffening of the trunk may even be directly linked to pain.¹³⁶

Increased trunk muscle activation to tighten control comes at the cost of increased spinal loading. Patients with LBP have been shown to expose their spine to higher forces than healthy participants

after perturbations⁶⁵ and during lifting.⁶⁸⁻⁷⁰ Because the most pronounced differences in loading were found during the least heavy tasks,⁶⁹ the risk of acute overloading of the spine is probably limited, but increased cumulative loading may elevate the risk. Further, low-level cocontraction of trunk muscles has been found in patients with LBP even at rest,¹²³ implying that compression of the spine is sustained during recovery periods. Animal models implicate sustained low-level compression as a cause of intervertebral disc degeneration, allegedly due to disrupted fluid flow into and out of the disc.^{60,85} Recovery of body height during rest after exercise, an indication of reuptake of water in intervertebral discs, was reduced in patients with LBP, and the lack of recovery was correlated to trunk muscle activity during rest.^{23,24} This suggests that fluid inflow into the disc may be impaired by sustained muscle contractions in patients with LBP, with possible adverse effects on disc health.

Sustained low-level muscle activity, as was found in some patients,¹²³ may also have noxious effects in the muscles.¹²⁹ Trunk extensor contractions at intensities as low as 2% of maximum activation do cause fatigue manifestations within half an hour.¹²⁴ Patients who show sustained trunk muscle activity may thus incur muscle fatigue and related discomfort,^{50,98,99} or even LBP of muscular origin,¹²⁹ especially if peripheral sensitization is present.

There may also be consequences of the decreased motor variability that is associated with tight control.¹¹⁵ It is increasingly recognized that some degree of variation is essential for tissue health.¹⁰³ Although too much variation may reflect uncontrolled motion, some variation is beneficial, as it allows sharing the load between different structures across repetitions.^{22,103} In addition, motor variability appears essential to provide an opportunity to learn through exposure to alternative ways of performing the same movement task.^{6,48,102,111} Participants who showed a change in trunk muscle recruitment in

fast arm movements during which pain was experimentally induced also showed a strong decrease in variability of muscle recruitment, and maintained these changes over the course of 70 arm movements when pain stimuli were no longer presented.⁷⁸ This clearly suggests that decreased variability hampers relearning of “normal” motor behavior, even after pain has subsided.

Finally, high trunk stiffness in patients with LBP appears to be related to a reduced use of trunk movement to counteract anticipated perturbations, which coincides with larger involuntary trunk displacement due to the perturbation.⁷⁵ Further, although enhanced trunk stiffness may be an effective strategy to counteract small disturbances, it may compromise an individual’s capacity to maintain balance on unstable or restricted surfaces,^{76,92} or when encountering larger disturbances.⁷⁷

Consequences of Loose Control At the loose end of the spectrum, patients have less control over trunk posture and movement. This might be the result of a protective adaptation to prevent pain provocation and reduce tissue loading related to large muscle forces or resulting compressive spine loading.

It is well accepted that the lumbar spine is an unstable structure whose configuration requires control by the surrounding musculature. Given the large number of degrees of freedom in the spine and given the fact that loads imposed on this system can be high and unpredictable, this poses a substantial control problem.^{83,120} Muscular control over spine movement would be reduced by inhibition of muscle activity and associated increases in delays in response to perturbations. This would be associated with faster and larger amplitude movements, with more variability between repeated performances of the same task. If muscular control over the spine fails, mid-range alignment of the lumbar vertebral segments may be compromised, resulting in large tissue strains.^{83,84,120} Also, sustained end-range alignment may, through

creep loading of spinal tissues, cause tissue responses and, potentially, pain.^{82,101} Whether modified or uncontrolled motion constitutes instability or is simply less robust control of motion with greater potential for abnormal tissue loading has been debated.⁹³ Cholewicki et al⁹ showed that large displacements after trunk perturbations were predictively associated with LBP, providing support for the notion that loose control can cause LBP.

Implications for Clinical Approaches to Address Motor Control Adaptation

Given the mechanical consequences and loading outcomes of the divergent presentations of motor control changes in people with LBP, it follows that different interventions are likely to be required to address different patient phenotypes. From the perspective of tight control linked to protection, in the early acute phase, the response may seem reasonable; however, if persistent, the negative consequences (increased loading, reduced movement) would likely become problematic for spine health. Thus, clinical strategies in later stages could be reasonably targeted to reduce excitability and cocontraction and to increase movement and potentially movement variation.⁴⁴

For loose control, strategies to augment control may be required.⁴⁴ The notion that loose control has a negative impact on clinical outcome in LBP forms the foundation for many exercise approaches. This has been targeted in some trials of interventions tailored to specific phenotypes of patients with LBP.^{15,52,64} In support of this approach, 2 clinical trials have shown greater clinical efficacy in patients identified to have deficient control of deep trunk muscles at baseline, and better clinical outcomes in those with improved function of these muscles after motor control intervention.^{15,112} Complicating treatment choices, there is a potential for overlap between effects of adaptation to and interference by LBP (eg, lower activation of the multifidus muscle might occur due to reflex inhibition with a concurrent protective strategy

of increased activation of the erector spinae muscle).

Despite promising data, there are significant challenges before validity of the existence of the proposed subgroups or phenotypes can be supported. It is critical to have valid assessments that can identify the pattern to change, therapeutic methods (eg, exercise approaches) to enact the change, and evidence that treatment targeted to the individual presentation leads to better outcomes than treatment that is not targeted. Some data are available,¹²⁸ but are far from complete.

CONCLUSION

ALTHOUGH MOTOR CONTROL ADAPTATIONS to pain present across a spectrum, 2 broad phenotypes of patients with LBP have been tentatively defined at the extremes of a spectrum, based on changes in trunk motor control observed from many studies. One phenotype shows tight control over trunk posture and movement due to increased excitability, at the cost of increased tissue loading secondary to increased muscle contraction. The other group shows loose control due to reduced excitability, with the potential cost of increased tissue loading from excessive spinal movements. Both groups involve abnormal loading of tissues in the low back, but with different mechanisms.

For both groups, there may also be an adaptive value of changes in motor control, at least in the short term: the first group may avoid excessive movement, and the second group may avoid high muscle forces. For both, it remains unclear whether the adaptive value outweighs the negative consequences, and this may differ between individuals, depending on the motor tasks to be performed and the integrity of the tissues in the low back. It is, in this context, important to note that nonspecific triggers, such as fear, can cause changes in motor control similar to those identified with pain.¹¹⁰ In case of unwarranted fear, there would be no benefit of the

adaptation, as no additional protection is required. Differences between these different phenotypes of motor control changes in individuals with LBP and the different consequences for mechanical loading support the notion that targets in motor control intervention should be different, and possibly even opposite, for these groups. This supports the plausibility of phenotyping and treatment targeting based on motor control presentation for the management of LBP. ●

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