HHS Public Access

Author manuscript

J Orthop Sports Phys Ther. Author manuscript; available in PMC 2020 July 31.

Published in final edited form as:

J Orthop Sports Phys Ther. 2019 June ; 49(6): 370–379. doi:10.2519/jospt.2019.7917.

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

JAAP H. VAN DIEËN, PhD[1], **N. PETER REEVES, PhD**[2,3,4], **GREG KAWCHUK, PhD**[5], **LINDA R. VAN DILLEN, PT, PhD**[6], **PAUL W. HODGES, PT, PhD, DSc, MedDr, BPhty (Hons)**[7]

[1]Department of Human Movement Sciences, Vrije Universiteit Amsterdam and Amsterdam Movement Sciences, Amsterdam, the Netherlands. [2]Center for Orthopedic Research, Michigan State University, Lansing, MI. [3]Department of Osteopathic Surgical Specialties, Michigan State University, East Lansing, MI. [4]Sumaq Life LLC, East Lansing, MI. [5]Department of Physical Therapy, Faculty of Rehabilitation Medicine, University of Alberta, Edmonton, Canada. [6]Program in Physical Therapy and Department of Orthopaedic Surgery, Washington University School of Medicine, St Louis, MO. [7]Clinical Centre for Research Excellence in Spinal Pain, Injury and Health, School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane, Australia.

Keywords

back pain; exercise; postural control; spine; subgrouping

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 maintained 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

Address correspondence to Dr Jaap H. van Dieën, Department of Human Movement Sciences, Vrije Universiteit Amsterdam, van der Boechorststraat 9, NL-1081 BT Amsterdam, the Netherlands. j.van.dieen@vu.nl.

The authors certify that they have no affiliations with or financial involvement in any organization or entity with a direct financial interest in the subject matter or materials discussed in the article.

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 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 nonspecific 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 infiltration[13]) 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 bending.[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 structures[103] 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 popularized 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 findings[123] 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.[25,54,78,80,90,113,116]

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 afference[105] 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 cortex[16] and in the motor cortex[108] and reduced corticomotor excitability[104] 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 al[91] 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 study[117] 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 perturbations[65] 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 reup-take 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, midrange 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 al[9] 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.

ACKNOWLEDGMENTS:

The authors would like to thank Dr Peter O'Sullivan and Dr Julie Fritz for reviewing a previous version of this paper. The forum on which this body of research was based, "State-of-the-Art in Motor Control and Low Back Pain: International Clinical and Research Expert Forum," was supported by the National Health and Medical Research Council of Australia, in collaboration with the North American Spine Society. The forum was chaired by Dr Paul Hodges.

Dr van Dillen was supported, in part, by funding from the National Center for Medical Rehabilitation Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development, US National Institutes of Health (grant R01 HD047709). Dr Reeves is the founder and president of Sumaq Life LLC. Dr Hodges receives book royalties from Elsevier. Professional and scientific bodies have reimbursed him for travel costs related to presentation of research on pain, motor control, and exercise therapy at scientific conferences/symposia. He has received fees for teaching practical courses on motor control training. He is also supported by a Senior Principal Research Fellowship from the National Health and Medical Research Council of Australia (APP1102905).

REFERENCES

1. Andreopoulou G, Maaswinkel E, Cofré Lizama LE, van Dieën JH. Effects of support surface stability on feedback control of trunk posture. Exp Brain Res. 2015;233:1079–1087. 10.1007/s00221-014-4185-5 [PubMed: 25537472]
2. Bauer CM, Rast FM, Ernst MJ, et al. Pain intensity attenuates movement control of the lumbar spine in low back pain. J Electromyogr Kinesiol. 2015;25:919–927. 10.1016/j.jelekin.2015.10.004 [PubMed: 26524940]
3. Boucher JA, Abboud J, Nougarou F, Normand MC, Descarreaux M. The effects of vibration and muscle fatigue on trunk sensorimotor control in low back pain patients. PLoS One. 2015;10:e0135838 10.1371/journal.pone.0135838 [PubMed: 26308725]
4. Boudreau S, Farina D, Kongstad L, et al. The relative timing of trunk muscle activation is retained in response to unanticipated postural-perturbations during acute low back pain. Exp Brain Res. 2011;210:259–267. 10.1007/s00221-011-2629-8 [PubMed: 21442223]
5. Bouisset S, Zattara M. Biomechanical study of the programming of anticipatory postural adjustments associated with voluntary movement. J Biomech. 1987;20:735–742. 10.1016/0021-9290(87)90052-2 [PubMed: 3654672]
6. Braun DA, Aertsen A, Wolpert DM, Mehring C. Motor task variation induces structural learning. Curr Biol. 2009;19:352–357. 10.1016/j.cub.2009.01.036 [PubMed: 19217296]
7. Carpenter MG, Frank JS, Silcher CP, Peysar GW. The influence of postural threat on the control of upright stance. Exp Brain Res. 2001;138:210–218. 10.1007/s002210100681 [PubMed: 11417462]
8. Cholewicki J, Panjabi MM, Khachatryan A. Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. Spine (Phila Pa 1976). 1997;22:2207–2212. [PubMed: 9346140]
9. Cholewicki J, Silfies SP, Shah RA, et al. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine (Phila Pa 1976). 2005;30:2614–2620. [PubMed: 16319747]
10. Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine (Phila Pa 1976). 2006;31:698–704. 10.1097/01.brs.0000202532.76925.d2 [PubMed: 16540876]

11. Descarreaux M, Blouin JS, Teasdale N. Force production parameters in patients with low back pain and healthy control study participants. Spine (Phila Pa 1976). 2004;29:311–317. [PubMed: 14752355]
12. Descarreaux M, Blouin JS, Teasdale N. Repositioning accuracy and movement parameters in low back pain subjects and healthy control subjects. Eur Spine J. 2005;14:185–191. 10.1007/s00586-004-0833-y [PubMed: 15759173]
13. D'hooge R, Cagnie B, Crombez G, Vanderstraeten G, Dolphens M, Danneels L. Increased intramuscular fatty infiltration without differences in lumbar muscle cross-sectional area during remission of unilateral recurrent low back pain. Man Ther. 2012;17:584–588. 10.1016/j.math.2012.06.007 [PubMed: 22784801]
14. Dideriksen JL, Gizzi L, Petzke F, Falla D. Deterministic accessory spinal movement in functional tasks characterizes individuals with low back pain. Clin Neurophysiol. 2014;125:1663–1668. 10.1016/j.clinph.2013.11.037 [PubMed: 24418221]
15. Ferreira ML, Ferreira PH, Latimer J, et al. Comparison of general exercise, motor control exercise and spinal manipulative therapy for chronic low back pain: a randomized trial. Pain. 2007;131:31–37. 10.1016/j.pain.2006.12.008 [PubMed: 17250965]
16. Flor H, Braun C, Elbert T, Birbaumer N. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. Neurosci Lett. 1997;224:5–8. 10.1016/S0304-3940(97)13441-3 [PubMed: 9132689]
17. Gombatto SP, Collins DR, Sahrmann SA, Engsberg JR, Van Dillen LR. Patterns of lumbar region movement during trunk lateral bending in 2 subgroups of people with low back pain. Phys Ther. 2007;87:441–454. 10.2522/ptj.20050370 [PubMed: 17374634]
18. Goodworth AD, Peterka RJ. Contribution of sensorimotor integration to spinal stabilization in humans. J Neurophysiol. 2009;102:496–512. 10.1152/jn.00118.2009 [PubMed: 19403751]
19. Grabiner MD, Koh TJ, el Ghazawi A. Decoupling of bilateral paraspinal excitation in subjects with low back pain. Spine (Phila Pa 1976). 1992;17:1219–1223. [PubMed: 1440012]
20. Gubler D, Mannion AF, Schenk P, et al. Ultra-sound tissue Doppler imaging reveals no delay in abdominal muscle feed-forward activity during rapid arm movements in patients with chronic low back pain. Spine (Phila Pa 1976). 2010;35:1506–1513. 10.1097/BRS.0b013e3181c3ed41 [PubMed: 20431436]
21. Hadizadeh M, Mousavi SJ, Sedaghatnejad E, Tale-bian S, Parnianpour M. The effect of chronic low back pain on trunk accuracy in a multidirectional isometric tracking task. Spine (Phila Pa 1976). 2014;39:E1608–E1615. 10.1097/BRS.0000000000000628 [PubMed: 25271509]
22. Hamill J, van Emmerik RE, Heiderscheit BC,Li L. A dynamical systems approach to lower extremity running injuries. Clin Biomech (Bristol, Avon). 1999;14:297–308. 10.1016/S0268-0033(98)90092-4
23. Healey EL, Fowler NE, Burden AM, McEwan IM. The influence of different unloading positions upon stature recovery and paraspinal muscle activity. Clin Biomech (Bristol, Avon). 2005;20:365–371. 10.1016/j.clinbiomech.2004.11.003
24. Healey EL, Fowler NE, Burden AM, McEwanIM. Raised paraspinal muscle activity reduces rate of stature recovery after loaded exercisein individuals with chronic low back pain. Arch Phys Med Rehabil. 2005;86:710–715. 10.1016/j.apmr.2004.10.026 [PubMed: 15827922]
25. Hebert JJ, Koppenhaver SL, Magel JS, Fritz JM. The relationship of transversus abdominis and lumbar multifidus activation and prognostic factors for clinical success with a stabilization exercise program: a cross-sectional study. Arch Phys Med Rehabil. 2010;91:78–85. 10.1016/j.apmr.2009.08.146 [PubMed: 20103400]
26. Hides J, Gilmore C, Stanton W, Bohlscheid E. Multifidus size and symmetry among chronic LBP and healthy asymptomatic subjects. Man Ther. 2008;13:43–49. 10.1016/j.math.2006.07.017 [PubMed: 17070721]
27. Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine (Phila Pa 1976). 1994;19:165–172. [PubMed: 8153825]

28. Hodges P, Holm AK, Hansson T, Holm S. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. Spine (Phila Pa 1976). 2006;31:2926–2933. 10.1097/01.brs.0000248453.51165.0b [PubMed: 17139223]
29. Hodges P, van den Hoorn W, Dawson A, Cholewicki J. Changes in the mechanical properties of the trunk in low back pain may be associated with recurrence. J Biomech. 2009;42:61–66. 10.1016/j.jbiomech.2008.10.001 [PubMed: 19062020]
30. Hodges PW. Motor control and pain In: Sluka KA, ed. Mechanisms and Management of Pain for the Physical Therapist. 2nd ed. Philadelphia, PA: Wolters Kluwer/IASP Press; 2016:67–81.
31. Hodges PW, Coppieters MW, MacDonaldD, Cholewicki J. New insight into motor adaptation to pain revealed by a combination of modelling and empirical approaches. Eur J Pain. 2013;17:1138–1146. 10.1002/j.1532-2149.2013.00286.x [PubMed: 23349066]
32. Hodges PW, Galea MP, Holm S, Holm AK. Corticomotor excitability of back muscles is affected by intervertebral disc lesion in pigs. Eur J Neurosci. 2009;29:1490–1500. 10.1111/j.1460-9568.2009.06670.x [PubMed: 19519631]
33. Hodges PW, Gandevia SC. Changes in intra-abdominal pressure during postural and respiratory activation of the human diaphragm. J Appl Physiol (1985). 2000;89:967–976. 10.1152/jappl.2000.89.3.967 [PubMed: 10956340]
34. Hodges PW, Gandevia SC, Richardson CA. Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. J Appl Physiol (1985). 1997;83:753–760. 10.1152/jappl.1997.83.3.753 [PubMed: 9292460]
35. Hodges PW, James G, Blomster L, et al. Multifidus muscle changes after back injury are characterized by structural remodeling of muscle, adipose and connective tissue, but not muscle atrophy: molecular and morphological evidence. Spine (Phila Pa 1976). 2015;40:1057–1071. 10.1097/BRS.0000000000000972 [PubMed: 25943090]
36. Hodges PW, James G, Blomster L, et al. Can proinflammatory cytokine gene expression explain multifidus muscle fiber changes after an intervertebral disc lesion? Spine (Phila Pa 1976). 2014;39:1010–1017. 10.1097/BRS.0000000000000318 [PubMed: 24718080]
37. Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC. Experimental muscle pain changes feed-forward postural responses of the trunk muscles. Exp Brain Res. 2003;151:262–271. 10.1007/s00221-003-1457-x [PubMed: 12783146]
38. Hodges PW, Richardson CA. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Arch Phys Med Rehabil. 1999;80:1005–1012. 10.1016/S0003-9993(99)90052-7 [PubMed: 10489000]
39. Hodges PW, Richardson CA. Contraction of the abdominal muscles associated with movement of the lower limb. Phys Ther. 1997;77:132–142; discussion 142–144. 10.1093/ptj/77.2.132 [PubMed: 9037214]
40. Hodges PW, Richardson CA. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. J Spinal Disord. 1998;11:46–56. [PubMed: 9493770]
41. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine (Phila Pa 1976). 1996;21:2640–2650. [PubMed: 8961451]
42. Hodges PW, Smeets RJ. Interaction between pain, movement, and physical activity: short-term benefits, long-term consequences, and targets for treatment. Clin J Pain. 2015;31:97–107. 10.1097/AJP.0000000000000098 [PubMed: 24709625]
43. Hodges PW, Tucker K. Moving differently in pain: a new theory to explain the adaptation to pain. Pain. 2011;152:S90–S98. 10.1016/j.pain.2010.10.020 [PubMed: 21087823]
44. Hodges PW, van Dillen L, McGill S, Brumagne S, Hides JA, Moseley GL. Integrated clinical approach to motor control interventions in low back and pelvic pain In: Hodges PW, Cholewicki J, van Dieën JH, eds. Spinal Control: The Rehabilitation of Back Pain. State of the Art and Science Edinburgh, UK: Elsevier/Churchill Livingstone; 2013:243–310.
45. Hoffman SL, Johnson MB, Zou D, Van Dillen LR. Differences in end-range lumbar flexion during slumped sitting and forward bending between low back pain subgroups and genders. Man Ther. 2012;17:157–163. 10.1016/j.math.2011.12.007 [PubMed: 22261650]

46. Hu H, Meijer OG, Hodges PW, et al. Control of the lateral abdominal muscles during walking. Hum Mov Sci. 2012;31:880–896. 10.1016/j.humov.2011.09.002 [PubMed: 22119422]
47. Jacobs JV, Henry SM, Nagle KJ. Low backpain associates with altered activity of the cerebral cortex prior to arm movements that require postural adjustment. Clin Neurophysiol. 2010;121:431–440. 10.1016/j.clinph.2009.11.076 [PubMed: 20071225]
48. James EG. Short-term differential training decreases postural sway. Gait Posture. 2014;39:172–176. 10.1016/j.gaitpost.2013.06.020 [PubMed: 23877033]
49. Jørgensen K Human trunk extensor muscles physiology and ergonomics. Acta Physiol Scand Suppl. 1997;637:1–58. [PubMed: 9246395]
50. Jørgensen K, Fallentin N, Krogh-Lund C, Jensen B. Electromyography and fatigue during prolonged, low-level static contractions. Eur J Appl Physiol Occup Physiol. 1988;57:316–321. 10.1007/BF00635990 [PubMed: 3371340]
51. Karayannis NV, Smeets RJ, van den Hoorn W, Hodges PW. Fear of movement is related to trunk stiffness in low back pain. PLoS One. 2013;8:e67779 10.1371/journal.pone.0067779 [PubMed: 23826339]
52. Kim TH, Kim EH, Cho HY. The effects of the CORE programme on pain at rest, movement-induced and secondary pain, active range of motion, and proprioception in female office workers with chronic low back pain: a randomized controlled trial. Clin Rehabil. 2015;29:653–662. 10.1177/0269215514552075 [PubMed: 25269569]
53. Laird RA, Gilbert J, Kent P, Keating JL. Comparing lumbo-pelvic kinematics in people with and without back pain: a systematic review and meta-analysis. BMC Musculoskelet Disord. 2014;15:229 10.1186/1471-2474-15-229 [PubMed: 25012528]
54. Lamoth CJ, Daffertshofer A, Meijer OG, Moseley GL, Wuisman PI, Beek PJ. Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking. Clin Biomech (Bristol, Avon). 2004;19:551–563. 10.1016/j.clinbiomech.2003.10.006
55. Lamoth CJ, Meijer OG, Daffertshofer A, Wuisman PI, Beek PJ. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. Eur Spine J. 2006;15:23–40. 10.1007/s00586-004-0825-y [PubMed: 15864670]
56. Lamoth CJ, Meijer OG, Wuisman PI, van Dieën JH, Levin MF, Beek PJ. Pelvis-thorax coordination in the transverse plane during walking in persons with nonspecific low back pain. Spine (Phila Pa 1976). 2002;27:E92–E99. [PubMed: 11840116]
57. Lamoth CJ, Stins JF, Pont M, Kerckhoff F, Beek PJ. Effects of attention on the control of locomotion in individuals with chronic low back pain. J Neuroeng Rehabil. 2008;5:13 10.1186/1743-0003-5-13 [PubMed: 18439264]
58. Larivière C, Forget R, Vadeboncoeur R, Bilodeau M, Mecheri H. The effect of sex and chronic low back pain on back muscle reflex responses. Eur J Appl Physiol. 2010;109:577–590. 10.1007/s00421-010-1389-7 [PubMed: 20174929]
59. Larivière C, Gagnon DH, Mecheri H. Trunk postural control in unstable sitting: effect of sex and low back pain status. Clin Biomech (Bristol, Avon). 2015;30:933–939. 10.1016/j.clinbiomech.2015.07.006
60. Lotz JC, Chin JR. Intervertebral disc cell death is dependent on the magnitude and duration of spinal loading. Spine (Phila Pa 1976). 2000;25:1477–1483. [PubMed: 10851095]
61. Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol. 1991;69:683–694. 10.1139/y91-102 [PubMed: 1863921]
62. Maaswinkel E, Veeger HE, van Dieen JH. Interactions of touch feedback with muscle vibration and galvanic vestibular stimulation in the control of trunk posture. Gait Posture. 2014;39:745–749. 10.1016/j.gaitpost.2013.10.011 [PubMed: 24192277]
63. MacDonald D, Moseley GL, Hodges PW. Whydo some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain. 2009;142:183–188. 10.1016/j.pain.2008.12.002 [PubMed: 19186001]

64. Macedo LG, Maher CG, Hancock MJ, et al. Predicting response to motor control exercises and graded activity for patients with low back pain: preplanned secondary analysis of a randomized controlled trial. Phys Ther. 2014;94:1543–1554. 10.2522/ptj.20140014 [PubMed: 25013000]

65. Mannion AF, Adams MA, Dolan P. Sudden and unexpected loading generates high forceson the lumbar spine. Spine (Phila Pa 1976). 2000;25:842–852. [PubMed: 10751296]

66. Mannion AF, Pulkovski N, Schenk P, et al. A new method for the noninvasive determination of abdominal muscle feedforward activity based on tissue velocity information from tissue Doppler imaging. J Appl Physiol (1985). 2008;104:1192–1201. 10.1152/japplphysiol.00794.2007 [PubMed: 18187614]

67. Mannion AF, Weber BR, Dvorak J, Grob D, Müntener M. Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. J Orthop Res. 1997;15:881–887. 10.1002/jor.1100150614 [PubMed: 9497814]

68. Marras WS, Davis KG, Ferguson SA, Lucas BR, Gupta P. Spine loading characteristics of patients with low back pain compared with asymptomatic individuals. Spine (Phila Pa 1976). 2001;26:2566–2574. [PubMed: 11725237]

69. Marras WS, Ferguson SA, Burr D, Davis KG, Gupta P. Functional impairment as a predictor of spine loading. Spine (Phila Pa 1976). 2005;30:729–737. [PubMed: 15803073]

70. Marras WS, Ferguson SA, Burr D, Davis KG, Gupta P. Spine loading in patients with low back pain during asymmetric lifting exertions. Spine J. 2004;4:64–75. 10.1016/S1529-9430(03)00424-8 [PubMed: 14749195]

71. Massé-Alarie H, Beaulieu LD, Preuss R, Schneider C. Task-specificity of bilateral anticipatory activation of the deep abdominal muscles in healthy and chronic low back pain populations. Gait Posture. 2015;41:440–447. 10.1016/j.gaitpost.2014.11.006 [PubMed: 25482033]

72. Massé-Alarie H, Flamand VH, Moffet H, Schneider C. Corticomotor control of deep abdominal muscles in chronic low back pain and anticipatory postural adjustments. Exp Brain Res. 2012;218:99–109. 10.1007/s00221-012-3008-9 [PubMed: 22311467]

73. Mazaheri M, Coenen P, Parnianpour M, KiersH, van Dieën JH. Low back pain and postural sway during quiet standing with and without sensory manipulation: a systematic review. Gait Posture. 2013;37:12–22. 10.1016/j.gaitpost.2012.06.013 [PubMed: 22796243]

74. Mimura M, Panjabi MM, Oxland TR, Crisco JJ, Yamamoto I, Vasavada A. Disc degeneration affects the multidirectional flexibility of the lumbar spine. Spine (Phila Pa 1976). 1994;19:1371–1380. [PubMed: 8066518]

75. Mok NW, Brauer SG, Hodges PW. Failure touse movement in postural strategies leads to increased spinal displacement in low back pain. Spine (Phila Pa 1976). 2007;32:E537–E543. 10.1097/BRS.0b013e31814541a2 [PubMed: 17762795]

76. Mok NW, Brauer SG, Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine (Phila Pa 1976). 2004;29:E107–E112. [PubMed: 15014284]

77. Mok NW, Hodges PW. Movement of the lumbar spine is critical for maintenance of postural recovery following support surface perturbation. Exp Brain Res. 2013;231:305–313. 10.1007/s00221-013-3692-0 [PubMed: 24036601]

78. Moseley GL, Hodges PW. Reduced variability of postural strategy prevents normalization of motor changes induced by back pain:a risk factor for chronic trouble? Behav Neurosci. 2006;120:474–476. 10.1037/0735-7044.120.2.474 [PubMed: 16719709]

79. Moseley GL, Hodges PW, Gandevia SC. External perturbation of the trunk in standing humans differentially activates components of the medial back muscles. J Physiol. 2003;547:581–587. 10.1113/jphysiol.2002.024950 [PubMed: 12562944]

80. Moseley GL, Nicholas MK, Hodges PW. Does anticipation of back pain predispose to back trouble? Brain. 2004;127:2339–2347. 10.1093/brain/awh248 [PubMed: 15282214]

81. Norton BJ, Sahrmann SA, Van Dillen LR. Differences in measurements of lumbar curvature related to gender and low back pain. J Orthop Sports Phys Ther. 2004;34:524–534. 10.2519/jospt.2004.34.9.524 [PubMed: 15493520]

82. Panjabi MM. A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. Eur Spine J. 2006;15:668–676. 10.1007/s00586-005-0925-3 [PubMed: 16047209]

83. Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J Spinal Disord. 1992;5:383–389; discussion 397. [PubMed: 1490034]
84. Panjabi MM. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. J Spinal Disord. 1992;5:390–396; discussion 397. [PubMed: 1490035]
85. Paul CP, Schoorl T, Zuiderbaan HA, et al. Dynamic and static overloading induce early degenerative processes in caprine lumbar intervertebral discs. PLoS One. 2013;8:e62411 10.1371/journal.pone.0062411 [PubMed: 23638074]
86. Pranata A, Perraton L, El-Ansary D, et al. Lumbar extensor muscle force control is associated with disability in people with chronic low back pain. Clin Biomech (Bristol, Avon). 2017;46:46–51. 10.1016/j.clinbiomech.2017.05.004
87. Prins MR, Griffioen M, Veeger TTJ, et al. Evidence of splinting in low back pain? A systematic review of perturbation studies. Eur Spine J. 2018;27:40–59. 10.1007/s00586-017-5287-0 [PubMed: 28900711]
88. Quint U, Wilke HJ. Grading of degenerative disk disease and functional impairment: imaging versus patho-anatomical findings. Eur SpineJ. 2008;17:1705–1713. 10.1007/s00586-008-0787-6 [PubMed: 18839226]
89. Radebold A, Cholewicki J, Polzhofer GK, Greene HS. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine (Phila Pa 1976). 2001;26:724–730. [PubMed: 11295888]
90. Ramprasad M, Shenoy DS, Sandhu JS, Sankara N. The influence of kinesiophobia on trunk muscle voluntary responses with pre-programmed reactions during perturbation in patients with chronic low back pain. J Bodyw Mov Ther. 2011;15:485–495. 10.1016/j.jbmt.2010.12.002 [PubMed: 21943622]
91. Reeves NP, Cholewicki J, Silfies SP. Muscle activation imbalance and low-back injury in varsity athletes. J Electromyogr Kinesiol. 2006;16:264–272. 10.1016/j.jelekin.2005.07.008 [PubMed: 16129623]
92. Reeves NP, Everding VQ, Cholewicki J, Morrisette DC. The effects of trunk stiffness on postural control during unstable seated balance. Exp Brain Res. 2006;174:694–700. 10.1007/s00221-006-0516-5 [PubMed: 16724177]
93. Reeves NP, Narendra KS, Cholewicki J. Spine stability: the six blind men and the elephant. Clin Biomech (Bristol, Avon). 2007;22:266–274. 10.1016/j.clinbiomech.2006.11.011
94. Sahrmann SA. Diagnosis and Treatment of Movement Impairment Syndromes. St Louis, MO: Mosby; 2001.
95. Scholtes SA, Gombatto SP, Van Dillen LR. Differences in lumbopelvic motion between people with and people without low back pain during two lower limb movement tests. Clin Biomech (Bristol, Avon). 2009;24:7–12. 10.1016/j.clinbiomech.2008.09.008
96. Scholz JP, Schöner G. The uncontrolled manifold concept: identifying control variables for a functional task. Exp Brain Res. 1999;126:289–306. 10.1007/s002210050738 [PubMed: 10382616]
97. Silfies SP, Bhattacharya A, Biely S, Smith SS, Giszter S. Trunk control during standing reach: a dynamical system analysis of movement strategies in patients with mechanical low back pain. Gait Posture. 2009;29:370–376. 10.1016/j.gaitpost.2008.10.053 [PubMed: 19046882]
98. Sjøgaard G Muscle energy metabolismand electrolyte shifts during low-level prolonged static contraction in man. Acta Physiol Scand. 1988;134:181–187. 10.1111/j.1748-1716.1988.tb08478.x [PubMed: 3227943]
99. Sjøgaard G, Kiens B, Jørgensen K, Saltin B. Intramuscular pressure, EMG and blood flow during low-level prolonged static contraction in man. Acta Physiol Scand. 1986;128:475–484. 10.1111/j.1748-1716.1986.tb08002.x [PubMed: 3788624]
100. Smith M, Coppieters MW, Hodges PW. Effect of experimentally induced low back pain on postural sway with breathing. Exp Brain Res. 2005;166:109–117. 10.1007/s00221-005-2352-4 [PubMed: 16032406]
101. Solomonow M, Baratta RV, Zhou BH, Burger E, Zieske A, Gedalia A. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. J Electromyogr Kinesiol. 2003;13:381–396. 10.1016/S1050-6411(03)00045-2 [PubMed: 12832168]

102. Sporns O, Edelman GM. Solving Bernstein's problem: a proposal for the developmentof coordinated movement by selection. Child Dev. 1993;64:960–981. 10.2307/1131321 [PubMed: 8404271]
103. Srinivasan D, Mathiassen SE. Motor variability in occupational health and performance. Clin Biomech (Bristol, Avon). 2012;27:979–993. 10.1016/j.clinbiomech.2012.08.007
104. Strutton PH, Theodorou S, Catley M, McGregor AH, Davey NJ. Corticospinal excitability in patients with chronic low back pain. J Spinal Disord Tech. 2005;18:420–424. 10.1097/01.bsd.0000169063.84628.fe [PubMed: 16189454]
105. Thunberg J, Ljubisavljevic M, Djupsjöbacka M, Johansson H. Effects on the fusimotor-muscle spindle system induced by intramuscular injections of hypertonic saline. Exp Brain Res. 2002;142:319–326. 10.1007/s00221-001-0941-4 [PubMed: 11819039]
106. Tong MH, Mousavi SJ, Kiers H, Ferreira P, Refshauge K, van Dieën J. Is there a relationship between lumbar proprioception and low back pain? A systematic review with meta-analysis. Arch Phys Med Rehabil. 2017;98:120–136.e2. 10.1016/j.apmr.2016.05.016 [PubMed: 27317866]
107. Tsao H, Danneels LA, Hodges PW. ISSLS Prize winner: smudging the motor brain in young adults with recurrent low back pain. Spine (Phila Pa 1976). 2011;36:1721–1727. 10.1097/BRS.0b013e31821c4267 [PubMed: 21508892]
108. Tsao H, Galea MP, Hodges PW. Reorganizationof the motor cortex is associated with postural control deficits in recurrent low back pain. Brain. 2008;131:2161–2171. 10.1093/brain/awn154 [PubMed: 18669505]
109. Tucker K, Butler J, Graven-Nielsen T, Riek S, Hodges P. Motor unit recruitment strategies are altered during deep-tissue pain. J Neurosci. 2009;29:10820–10826. 10.1523/JNEUROSCI.5211-08.2009 [PubMed: 19726639]
110. Tucker K, Larsson AK, Oknelid S, Hodges P. Similar alteration of motor unit recruitment strategies during the anticipation and experience of pain. Pain. 2012;153:636–643. 10.1016/j.pain.2011.11.024 [PubMed: 22209423]
111. Turnham EJ, Braun DA, Wolpert DM. Facilitation of learning induced by both random and gradual visuomotor task variation. J Neurophysiol. 2012;107:1111–1122. 10.1152/jn.00635.2011 [PubMed: 22131385]
112. Unsgaard-Tøndel M, Lund Nilsen TI, Magnussen J, Vasseljen O. Is activation of transversus abdominis and obliquus internus abdominis associated with long-term changes in chronic low back pain? A prospective study with 1-year follow-up. Br J Sports Med. 2012;46:729–734. 10.1136/bjsm.2011.085506 [PubMed: 21791459]
113. Unsgaard-Tøndel M, Nilsen TI, Magnussen J, Vasseljen O. Are fear avoidance beliefs associated with abdominal muscle activation outcome for patients with low back pain? Physiother Res Int. 2013;18:131–139. 10.1002/pri.1539 [PubMed: 23147914]
114. Van Daele U, Hagman F, Truijen S, Vorlat P, Van Gheluwe B, Vaes P. Differences in balance strategies between nonspecific chronic low back pain patients and healthy control subjects during unstable sitting. Spine (Phila Pa 1976). 2009;34:1233–1238. 10.1097/BRS.0b013e31819ca3ee [PubMed: 19444072]
115. van den Hoorn W, Bruijn SM, Meijer OG, Hodges PW, van Dieën JH. Mechanical coupling between transverse plane pelvis and thorax rotations during gait is higher in people with low back pain. J Biomech. 2012;45:342–347. 10.1016/j.jbiomech.2011.10.024 [PubMed: 22078275]
116. van der Hulst M, Vollenbroek-Hutten MM, Schreurs KM, Rietman JS, Hermens HJ. Relationships between coping strategies and lumbar muscle activity in subjects with chronic low back pain. Eur J Pain. 2010;14:640–647. 10.1016/j.ejpain.2009.10.011 [PubMed: 19962923]
117. van Dieën JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine (Phila Pa 1976). 2003;28:834–841. [PubMed: 12698129]
118. van Dieën JH, de Looze MP. Directionality of anticipatory activation of trunk muscles in a lifting task depends on load knowledge. Exp Brain Res. 1999;128:397–404. 10.1007/s002210050860 [PubMed: 10501812]

119. van Dieën JH, Flor H, Hodges PW. Low-back pain patients learn to adapt motor behavior with adverse secondary consequences. Exerc Sport Sci Rev. 2017;45:223–229. 10.1249/JES.0000000000000121 [PubMed: 28704216]
120. van Dieën JH, Kingma I. Spine function and low back pain: interactions of active and passive structures In: Hodges PW, Cholewicki J, van Dieën JH, eds. Spinal Control: The Rehabilitation of Back Pain. State of the Art and Science Edinburgh, UK: Elsevier/Churchill Livingstone; 2013:41–58.
121. van Dieën JH, Kingma I, van der Bug P. Evidence for a role of antagonistic cocontraction in controlling trunk stiffness during lifting. J Biomech. 2003;36:1829–1836. 10.1016/S0021-9290(03)00227-6 [PubMed: 14614936]
122. van Dieën JH, Koppes LL, Twisk JW. Low back pain history and postural sway in unstable sitting. Spine (Phila Pa 1976). 2010;35:812–817. 10.1097/BRS.0b013e3181bb81a8 [PubMed: 20195213]
123. van Dieën JH, Selen LP, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. J Electromyogr Kinesiol. 2003;13:333–351. 10.1016/S1050-6411(03)00041-5 [PubMed: 12832164]
124. van Dieën JH, Westebring-van der Putten EP, Kingma I, de Looze MP. Low-level activity of the trunk extensor muscles causes electromyo-graphic manifestations of fatigue in absence of decreased oxygenation. J Electromyogr Kinesiol. 2009;19:398–406. 10.1016/j.jelekin.2007.11.010 [PubMed: 18178450]
125. Van Dillen LR, Sahrmann SA, Norton BJ, Caldwell CA, McDonnell MK, Bloom N. The effect of modifying patient-preferred spinal movement and alignment during symptom testing in patients with low back pain: a preliminary report. Arch Phys Med Rehabil. 2003;84:313–322. 10.1053/apmr.2003.50010 [PubMed: 12638097]
126. van Wegen EE, van Emmerik RE, Riccio GE. Postural orientation: age-related changes in variability and time-to-boundary. Hum MovSci. 2002;21:61–84. 10.1016/S0167-9457(02)00077-5
127. Vaucher M, Isner-Horobeti ME, Demattei C, et al. Effect of a kneeling chair on lumbar curvature in patients with low back pain and healthy controls: a pilot study. Ann Phys Rehabil Med. 2015;58:151–156. 10.1016/j.rehab.2015.01.003 [PubMed: 25956202]
128. Vibe Fersum K, O'Sullivan P, Skouen JS, Smith A, Kvåle A. Efficacy of classification-based cognitive functional therapy in patients with non-specific chronic low back pain: a randomized controlled trial. Eur J Pain. 2013;17:916–928. 10.1002/j.1532-2149.2012.00252.x [PubMed: 23208945]
129. Visser B, van Dieën JH. Pathophysiology of upper extremity muscle disorders. J Electromyogr Kinesiol. 2006;16:1–16. 10.1016/j.jelekin.2005.06.005 [PubMed: 16099676]
130. Vogt L, Pfeifer K, Portscher M, Banzer W. Influences of nonspecific low back pain on three-dimensional lumbar spine kinematics in locomotion. Spine (Phila Pa 1976). 2001;26:1910–1919. 10.1097/00007632-200109010-00019 [PubMed: 11568705]
131. Wallwork TL, Stanton WR, Freke M, Hides JA. The effect of chronic low back pain on size and contraction of the lumbar multifidus muscle. Man Ther. 2009;14:496–500. 10.1016/j.math.2008.09.006 [PubMed: 19027343]
132. Wand BM, Parkitny L, O'Connell NE, et al. Cortical changes in chronic low back pain: current state of the art and implications for clinical practice. Man Ther. 2011;16:15–20. 10.1016/j.math.2010.06.008 [PubMed: 20655796]
133. Willigenburg NW, Kingma I, Hoozemans MJ,van Dieën JH. Precision control of trunk movement in low back pain patients. Hum Mov Sci. 2013;32:228–239. 10.1016/j.humov.2012.12.007 [PubMed: 23427936]
134. Willigenburg NW, Kingma I, van Dieën JH. Center of pressure trajectories, trunk kinematicsand trunk muscle activation during unstable sitting in low back pain patients. Gait Posture. 2013;38:625–630. 10.1016/j.gaitpost.2013.02.010 [PubMed: 23473809]
135. Willigenburg NW, Kingma I, van Dieën JH. Precision control of an upright trunk posture in low back pain patients. Clin Biomech (Bristol, Avon). 2012;27:866–871. 10.1016/j.clinbiomech.2012.06.002

136. Wong AY, Parent EC, Dhillon SS, Prasad N, Kawchuk GN. Do participants with low back pain who respond to spinal manipulative therapy differ biomechanically from nonresponders, untreated controls or asymptomatic controls? Spine(Phila Pa 1976). 2015;40:1329–1337. 10.1097/BRS.0000000000000981 [PubMed: 26020851]

137. Zhao F, Pollintine P, Hole BD, Dolan P, Adams MA. Discogenic origins of spinal instability. Spine (Phila Pa 1976). 2005;30:2621–2630. [PubMed: 16319748]

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.