Falla et al. [4] present an elegant study that provides critical support for the predictions of recent theories for adaptation in motor control in pain [9,16]. By simultaneously recording activity over an area of back muscles and movement across multiple body segments, the authors have addressed key predictions regarding how people in pain move and control their body differently from those without pain. The authors provide a rich dataset that enables consideration of the interplay between kinematics and muscle recruitment to advance thinking in this field. The challenge is to consider possible mechanisms and consequences of the differences in behaviour between individuals with and without back pain. A point worthy of further reflection is the potential importance of individual variation in the adaptation to pain, which receives limited attention with the selected design.

What are the potential mechanisms for adaptation?

The authors distil 3 observations where participants with back pain differ from pain-free controls: (i) increased electromyographic (EMG) activity; (ii) lack of systematic caudal shift of EMG centroid with repetition; and (iii) differences in spine-hip motion. These observations are not straightforward. Increased EMG activity may compensate for muscle fatigue (greater muscle activity for same force output) or greater antagonist activity (greater spine load). Furthermore, the caudal shift was small (~4 mm—half the inter-electrode distance) and the mechanical effect is unclear. If the muscle is fatigued, as implied by changes in median frequency, activation would increase and load sharing between muscles is reduced at higher activation levels [1]. Falla et al. [4] present a range of potential mechanisms that might explain the changes in muscle activation and movement; some peripheral and some central. The challenge in human research is to consider the multiple, potentially interacting influences on motor control strategy ultimately reflected in function.

In terms of EMG, one element of complexity for interpretation is how to disentangle the effects of pain and fatigue. Both may affect EMG and, as identified by Falla et al. [4], nociceptive input has been argued to underpin some adaptations in fatigue. Although impossible to resolve from this study, nociceptive input from a sensitized system (as indicated by hyperalgesia) might modify drive to motorneurons. This has been contentious, with inconsistent effects observed when motoneuron excitability is studied during nociceptive excitation [13,18]. Several studies report changes in the discharge rate of individual motor units with pain [5,17,19], but inconsistent changes when activity is recorded over a larger muscle area. This can be accounted for by redistribution of activity between motor units rather than a complete change in regional activity [19]. Whether nociceptive input changes fibre properties by local or spinal effects remains unresolved.

Alternatively, difference in muscle recruitment between groups might be explained by descending drive from higher centres. This could be a purposeful adaptation to achieve some goal, such as protection of the painful region from further pain/injury. Although it may be mechanically efficient for the nervous system to shift activity to caudal regions to share load and avoid fatigue in painfree individuals, in pain there may be some benefit to maintain the initial strategy for “protection”, and the nervous system may limit the shift in activity [9]. Of note, movement adaptation does not depend on nociceptive input, and can be replicated by anticipation of pain [15,21]. An interesting observation of Falla et al. [4] was that the patients adopted greater hip extension and less back extension. Perhaps the difference in strategy reflects an attempt to rely less on the back, preferring to adapt at other regions (ie, hip) such that the back remains “protected”. Modification of spine posture might also alter load on the painful region, without changing muscle activation as is proposed for other body regions (Hug et al., unpublished data).

An alternative view is that it may be impossible to shift activity to caudal regions. Changes in muscle structure could mean the new solution (relative increase in caudal muscle activity) is not possible for the pain group. Changes in the muscle fibre composition of the multifidus muscles (major bulk of the caudal back muscles) towards a lower proportion of slow-twitch (fatigue-resistant) fibres in pain/injury have been suggested in cross-sectional human studies [11] and confirmed in longitudinal animal studies [8]. This may render caudal muscles less able to accommodate to increased demand and preclude the “usual” shift of activity to this region.

The failure of the pain group to adopt the same solution as painfree participants (caudal shift in muscle activity to avoid fatigue and maintain performance) could simply represent a deficit in the motor system secondary to nociceptive input. However, it may also represent a purposeful “selection” of strategy on the basis of balancing competing demands of protection of the painful region, energy demand, muscle capacity, etc. Recent data imply the nervous system does not always adopt the simplest solution to prevent pain/injury, as a consequence of balancing these demands (Bergin, Hug, Tucker and Hodges, unpublished data).

What are the consequences of adaptation?

The 3 features of adapted motor control identified by Falla et al. [4] are consistent with typical adaptations in earlier studies [9]. As
predicted by theory, each has potential short-term benefit—to reduce the risk of injury/pain—but may also have long-term consequences. Increased muscle activity, although potentially protective of the region, will increase tissue load [12] and if maintained could underlie further effects at the tissue level. Reduced movement (reduced range of spine motion) impairs shock absorption/damping, with resultant greater tissue load [14]. Reduced variability (reduced EMG centroid shift) compromises load sharing between structures [6]. Evidence for a role of these features in recurrence/persistence of pain is emerging [2].

**Do all patients with back pain adopt a similar solution?**

Participants studied by Falla et al. [4] were those with “nonspecific” low back pain, selected by pain duration and its effect on function. The implication is that this is a homogeneous group who adapt similarly. The analysis involved data averaging across the group, apart from one analysis that considered the proportion of participants with a caudal EMG centroid shift. Current theories predict that pain is associated with individual variation in motor control adaptation [9]. This cannot be reflected in the present analysis.

Although some changes may be apparent from group averages, such as increased EMG [4], when data from individual patients are considered the response is generally variable. For instance, when pain is provoked experimentally in healthy individuals during slow trunk movements, there is an overall EMG increase for most participants, which translates to augmented spine stability (estimated trunk movements), there is an overall EMG increase for most participants, which translates to augmented spine stability (estimated range/benefit) remains elusive, but this study provides convincing evidence to increase the net EMG. Within that variation some muscle responses are considered, no 2 participants adopt the same solution to increase the net EMG. Within that variation some clustering of patterns emerges, which coincides with clinical data pointing to subgroups or phenotypes in the apparently heterogeneous nonspecific low back pain group [3,10]. Methods to account for individual variation in pattern require consideration. Recent data from measures of muscle stress (ultrasound elastography) imply nonsystematic changes in strategy, even with an identical pain stimulus between individuals [20].

**Conclusion**

The elegant data of Falla et al. [4] highlight differences in motor strategy in back pain. This adds substance to interpretations of movement changes in pain. Resolution of the mechanism or consequence/benefit remains elusive, but this study provides convincing and multifaceted data upon which to found further investigation. This could be aided by techniques that directly measure tissue loading in individuals.

**Conflict of interest statement**

There are no conflicts of interest.

**Acknowledgments**

The author thanks Dr Francois Hug for comments on the manuscript. The author is supported by a Senior Principal Research Fellowship (APP1002190) from the National Health and Medical Research Council (NHMRC) of Australia.

**References**


Paul W. Hodges *
The University of Queensland, Centre of Clinical Research Excellence in Spinal Pain, Injury and Health, School of Health and Rehabilitation Sciences, Brisbane, Qld 4072, Australia.
* Tel.: +61 7 3365 2008.

E-mail address: p.hodges@uq.edu.au