

Pain and motor control of the lumbopelvic region: effect and possible mechanisms

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Abstract

Many authors report changes in the control of the trunk muscles in people with low back pain (LBP). Although there is considerable disagreement regarding the nature of these changes, we have consistently found differential effects on the deep intrinsic and superficial muscles of the lumbopelvic region. Two issues require consideration; first, the potential mechanisms for these changes in control, and secondly, the effect or outcome of changes in control for lumbopelvic function. Recent data indicate that experimentally induced pain may replicate some of the changes identified in people with LBP. While this does not exclude the possibility that changes in control of the trunk muscles may lead to pain, it does argue that, at least in some cases, pain may cause the changes in control. There are many possible mechanisms, including changes in excitability in the motor pathway, changes in the sensory system, and factors associated with the attention demanding, stressful and fearful aspects of pain. A new hypothesis is presented regarding the outcome from differential effects of pain on the elements of the motor system. Taken together these data argue for strategies of prevention and rehabilitation of LBP

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Contents

1. Introduction	361
2. Changes in motor control of the lumbopelvic region	362
3. Possible mechanisms for pain to affect motor control of the trunk muscles	363
4. Possible outcomes of motor control changes	366

1. Introduction

Changes in motor control and function of the trunk muscles have been reported frequently in the literature. These changes range from changes in recruitment to reduced strength and endurance of the trunk muscles. Notably, patterns of hyperactivity and hypoactivity have been reported and a variety of hypotheses have been developed to explain the effects and mechanisms of the

changes. The majority of available hypotheses are broadly consistent with two main theories that propose; (i) that changes in muscle activity cause spinal pain (muscle–tension or pain–spasm–pain model), or (ii) changes in muscle activity serve to restrict spinal motion (pain adaptation model). Experimental (e.g. [11,70,95,113]), and clinical [7,50] data suggest that the muscle tension model is too simplistic, and offer support for the pain adaptation model [63], however considerable debate exists [114]. The purpose of this paper is to review the evidence for changes in motor control, discuss possible mechanisms for these changes and the effect of these changes on function of the lumbopelvic region.

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2. Changes in motor control of the lumbopelvic region

Although early studies of trunk muscle function focused on the strength and endurance of the trunk muscles in patients with LBP (e.g. [94,101]), more recently the focus has shifted to issues of motor control. The challenge of motor control of the lumbopelvic region is immense and must serve to move and control the spine in a range of environments and with a complex interaction between internal and external forces. The challenge is further complicated by the fact that without muscle the spine and pelvis are inherently unstable [62,77]. Trunk muscles must have sufficient strength and endurance to satisfy the demands of control, but the efficacy of the muscle system is dependent on its controller, the central nervous system (CNS) [77]. The CNS must continually interpret the status of stability and movement, plan mechanisms to overcome predictable challenges and rapidly initiate activity in response to unexpected challenges. It must interpret the afferent input from peripheral mechanoreceptors and other sensory systems, consider this input and the impending requirements against an “internal model of body dynamics” and then generate a coordinated response of the trunk muscles so that the muscle activity occurs at the correct time, with the correct amplitude, and so on. Further, muscle activity must be coordinated to maintain control of the spine within a hierarchy of interdependent levels; control of intervertebral translation and rotation, control of spinal posture/orientation, control of body with respect to the environment [44,45,77]. Finally, unlike the muscles of the limb, trunk muscles perform a variety of homeostatic functions in addition to movement and control of the trunk (e.g. respiration and continence) [39]. In view of the complex requirements of trunk muscle control, it is not surprising that aspects of control are altered in people with LBP.

Many studies report changes in motor control in people with acute and chronic LBP (e.g. [45,81,89]). While there is considerable variability in results, we have consistently found differential changes in activity between the deep and superficial trunk muscles. In terms of deep intrinsic trunk muscle activity, there is evidence of delayed activity of transversus abdominis (TrA), the deepest of the abdominal muscles (recorded with intramuscular EMG electrodes), in association with rapid limb movements in people with chronic LBP [47]. It is well accepted that the CNS initiates a sequence of muscle activity involving the limb and trunk muscles in advance of limb movements to prepare the body for the predictable disturbance to stability from the reactive forces caused by movement [3,4,8,46]. This sequence of responses is feedforward, that is, it is preplanned by the CNS and occurs in advance of the movement. Therefore, these responses precede any afferent input from the

movement [4]. While changes have been identified in these tasks in feedforward activity of both deep and superficial muscles, the most consistent change (between subjects and movements) occurs in TrA [45]. The changes in TrA have been replicated when pain is induced by intramuscular injection of hypertonic saline into the longissimus at L4 [41]. Notably, the changes observed in patients were identified in people who had a history of LBP but were in remission from their symptoms. Although these studies have reported a delay in activation, it is likely that the change is not confined to this parameter, but instead may be reflective of a change in control. For example tonic activity of TrA, which is normally observed during repetitive trunk [17] and limb movements [40], is reduced during experimentally-induced pain [41]; relative EMG activity of rectus abdominis and EMG activity recorded with electrodes over the inferiolateral abdominal wall is altered in people with chronic LBP during a novel task to move the abdominal wall inwards [75].

There is preliminary evidence that the deep paraspinal muscles show similar changes in activity. During functional tasks, there is reduced amplitude of activity of multifidus, the deepest back muscle in the lumbar region, in people with LBP [60,89] and altered responses have been observed during loading of the trunk. For example, when a load is unexpectedly dropped into the hands, there is normally a short-latency response of the paraspinal muscles [59,73]. In healthy control subjects, studies have reported earlier activity of the deep [73] and superficial [59] fibres of multifidus when the loading can be anticipated compared to trials when the load cannot be anticipated [73]. However, when people with sciatica catch a load that is predictable, the response of the paraspinal muscles (recorded with surface electrodes) does not occur earlier than the unpredictable trials [59]. Others, using an unexpected loading paradigm, report both delayed [67,109] and no change [113] in activity of the paraspinal muscles. The apparent greater specificity of the delay to predictable tasks suggests that the change is dependent on input from higher centres of the CNS. The reported changes in activity of multifidus are consistent with changes in its morphology and fatigability, which in turn could be explained by altered use of the muscle. For example, studies report changes in muscle fibre composition [83] and increased fatigability [6,87], and reduced cross-sectional area of multifidus has been identified as little as 24 hours after the onset of acute, unilateral LBP [37], although it is not clear as to whether this is a premorbid phenomenon. In summary, the evidence seems to suggest that, with LBP, there is an alteration in control of the deep intrinsic spinal muscles that consistently manifests as hypoactivity. The possible implications of these changes are discussed below. Although others argue that paraspinal muscles react to

pain and injury with hyperactivity (e.g. [49,90,116]) this may vary between components of the paraspinal group.

Due to the ease of accessibility of the superficial trunk muscles to surface EMG recordings, there is a large literature that investigates changes in these muscles in LBP. Despite the popularity of the muscle tension model, there is considerable debate about the presence of augmented activity of the paraspinal muscles. Studies have had variable results, some reporting increased activity [1,111], others reporting decreased [89] or asymmetrical activity [16] and others reporting no change in activity [15], for a review see [114]. One finding that has been consistently observed in people with LBP is sustained activity of the erector spinae muscles at the end of range of spinal flexion, a point at which the erector spinae muscles are normally inactive (the so-called flexion-relaxation response) [88]. Importantly, this normal response is only lost in a subset of patients, suggesting that factors other than the presence of pain influence this change in activity (e.g. fear of pain, see below). Nonetheless, this finding has been replicated by experimental pain [113] and has been shown to limit intervertebral motion [56]. In this regard, changes in paraspinal muscle activity during gait may serve a similar purpose. The normal periods of silence in erector spinae activity between heel contacts are reduced in LBP patients and in otherwise symptomatic participants given experimentally induced LBP [2], which may serve to splint the area during this period.

Variability in superficial trunk muscle activity associated with pain has been observed in other tasks. In a study by Radebold and colleagues [81] in which a load was removed from the trunk, augmentation of superficial trunk muscles was observed, but only in a subset of patients. Experimentally elicited pain caused variable responses of the superficial trunk muscles in association with rapid limb movements [41]. However, importantly, although there was considerable inter-subject variability in the pattern of superficial trunk muscle activity, at least one superficial muscle was augmented during pain in every subject. Notably the hypoactivity of the intrinsic spinal muscle, TrA, was a consistent finding across the group.

In addition to changes in muscle recruitment, impairment of other elements of motor control has been identified in people with LBP. For example changes in balance control and sensory aspects. Balance has been shown to be impaired in people with LBP when standing on one [64] or two legs [10] or sitting [82], and people with poor performance in a test of standing balance have an increased risk of LBP [100]. Because both feedforward and feedback-mediated components of motor control are dependent on sensory input, any change in sensory input is likely to be important. Numerous studies have reported reduced acuity [32] and impaired ability to perform repositioning tasks [9] in people with LBP.

Other more complex elements of control have also been found to be altered in LBP. For instance people with LBP have a slower reaction time [65], and slow reaction time has been associated with musculoskeletal injuries (including LBP) in a variety of sports [99]. Although there is marked variability between individuals and studies, the relationship between pain and motor control of the spine appears complex. Importantly, there is increasing evidence for differential changes in activity of the deep and superficial trunk muscles with pain. In this regard, two issues require consideration. What are the possible mechanisms for this change and what are the potential outcomes in terms of spinal function?

3. Possible mechanisms for pain to affect motor control of the trunk muscles

It is not certain whether pain causes changes in motor control or whether motor control changes lead to pain, or both. Farfan [25] and Panjabi [77], amongst others, have presented models that suggest that deficits in motor control lead to poor control of joint movement, repeated microtrauma and pain. Consistent with this model, Janda [53] has argued that people who have mild neurological signs (e.g. minor coordination difficulties) are more likely to have pain as adults. Furthermore slow reaction times have been linked to increased risk of musculoskeletal injury [99]. However, the converse may also be true. Perhaps pain leads to changes in motor control. Numerous studies using experimental models of pain have provided support for this hypothesis by reproduction of changes in control that have been identified in clinical populations [2,41,113]. Consequently, a number of mechanisms have been proposed to explain the effect of pain on motor control (Fig. 1). These include changes in excitability at the spinal or cortical level, changes in proprioception or afferent mediated control, or specific cortical effects imparted by aspects of pain, such as its demand on CNS resources, stress or fear. The following sections will review each of these possible mechanisms.

Widespread changes in excitability have been identified at many levels of the motor system during pain. Acute experimental pain has been shown to cause changes in spinal motoneuron activity [70,96,97]. For instance, increased stretch reflex amplitude of the soleus muscle has been reported after intramuscular injection of hypertonic saline [70]. Others report reduced amplitude of motor potential evoked by transcranial magnetic stimulation over the motor cortex in response to experimental pain [102]. However, these responses may be task or muscle specific as other studies have reported no changes in excitability of the motoneuron or motor cortex [29,112]. Those authors argued that changes in motor drive may occur 'upstream' of the motor cortex, for

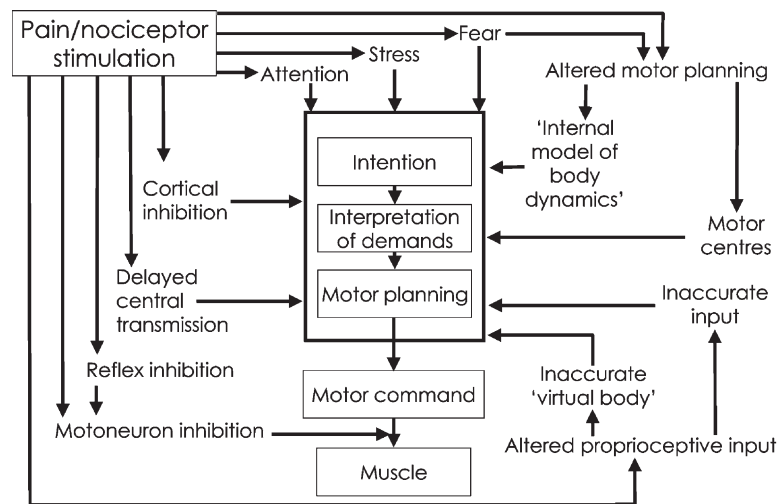


Fig. 1. Possible mechanisms for pain to affect motor control. Multiple mechanisms have been proposed for pain to affect motor control. It is unlikely that the simple inhibitory pathways (left) can mediate the complex changes in motor control of the trunk muscles. The most likely candidates are changes in motor planning via a direct influence of pain on the motor centres, fear-avoidance, or due to changes in the sensory system.

instance, involving areas associated with motor planning. Reflex inhibition of motoneuron excitability has also been suggested to occur in association with swelling [92] and injury to joint structures [24], which has been argued to indicate polysynaptic inhibition at a spinal level [93]. While this may be a factor in clinical populations, it cannot explain the findings of studies of experimental pain that are not associated with oedema and injury, and similar effects cannot be produced by injection of similar volumes of isotonic saline [33].

Evidence from several groups argues that changes in trunk muscle activity in LBP may not be mediated by simple changes in excitability. Zedka et al [113] were unable to identify changes in the short latency response of the paraspinal muscles to a mechanical tap to the muscle following pain induced by injection of hypertonic saline into the muscle (changes in this component would be consistent with changes in motoneuron excitability). These authors did find changes in later components of the response that can be influenced by input from higher centres. We have shown several changes in coordination of the trunk muscles in association with pain that are inconsistent with a change in excitability or delayed transmission of the motor command. For example, when people move an arm rapidly, normally the response of TrA is independent of the direction of arm movement [46]. If the delay in response observed during pain was due to a change in excitability it may be predicted that the response would remain consistent between movement directions, although delayed. However, this is not the case. The response of TrA in people with LBP is earlier with shoulder extension than the other movements, which is similar to the response of the superficial trunk muscles, normally under differential control [45,73]. Also, in a healthy population, when the preparation for movement is reduced, despite slowing of the

response of the prime mover of the arm and the oblique abdominal muscles, the response of TrA is not affected [48]. However, in people with LBP, the response is delayed along with the increased reaction time of the movement in the reduced preparation trials [42]. Taken together, these findings are likely to represent a change in motor planning.

Consistent with the identification of changes in motor planning there is compelling evidence that pain has strong effects at the supraspinal level [20,38,57,61,64,66,104]. Both short- and long-term changes are thought to occur in activity of the supraspinal structures including the cortex with pain. Many studies have reported changes during experimental pain in activity of regions of the brain involved in movement planning and performance (see [20]). One area that has been consistently found to be affected is the anterior cingulate cortex (ACC) [79]. The ACC has also been reported to be chronically active in people with chronic LBP [51]. The ACC has long been thought to be important in motor responses and directly projects to motor and supplementary motor areas [80]. Hypothetically at least, activation of these cortical regions during pain may influence movement control directly and mediate the changes reported above. However, confirmation of this hypothesis is difficult because movement is not permitted in many imaging studies. Other authors have identified increased activity in areas of somatosensory cortex activated by noxious cutaneous stimulation of the finger and back in people with LBP [27]. Furthermore, the area activated increased as a function of the duration of their pain. These changes may contribute to the perpetuation of pain in the absence of peripheral nociception, but may also contribute to the motor changes. Further work is required to clarify these findings as they relate to motor control.

Although nociceptive stimulation and pain may disrupt motor output directly, it is also possible that an effect is caused by aspects of pain, such as its attention demanding requirements, stress or fear. In terms of attention demand, it is widely considered that pain utilises attentional resources, probably by virtue of its direct relevance for survival (see [80] for review). Several studies support this hypothesis. For example, recordings of event-related potentials in the cortex [86], brain imaging studies [20], cognitive performance tasks [18,19,22] and a combination of these methods [61] indicate increased latencies and/or error rates in the presence of pain. Thus pain may lead to changes in movement coordination as a result of the increased demand placed on information processing resources. While several authors have identified slower reaction times in people with LBP, which may be attributable to this mechanism [66], we have recently shown that performance of an attention-demanding task does not replicate the changes in trunk muscle activity seen in people with LBP [72]. In this study subjects rapidly moved an arm in response to a visual stimulus while performing an attention-demanding task. Although the reaction time of the arm movement was delayed, the response of the deep trunk muscles (TrA and deep MF) occurred earlier relative to the deltoid response (i.e. opposite to the changes seen in LBP). There was no change in the activity of the superficial abdominal or paraspinal muscles.

A further possibility is that the stress associated with pain produces the change in control of the trunk muscles. Numerous studies have shown that stress (i.e. perception of threat) may affect motor control [54,103,108]. Notably, trunk muscle activity during a lifting task is altered when the task is performed in the presence of psychosocial stressors [69] and shoulder muscle activity during a keyboard task is altered by work-related stress [23]. Furthermore, changes in paraspinal muscle activity in chronic pain patients have been linked to subjective measures of distress and anxiety, rather than just the intensity of pain [26,28,106]. We have tested the effect of stress on the postural response of the trunk muscles during rapid arm movements by repeating the attention-demanding task described above, but with negative feedback of performance and other negative psychosocial cues [72]. Although the addition of stress did not replicate the changes that we had identified with experimentally induced pain, there was a delay in the response of the deep trunk muscles relative to tasks when the attention demand was non-stressful, indicating some effect of stress.

Another alternative argues that the changes in control may relate to the fear associated with pain. The notion that fear is important in behavioural and motor output associated with pain is not new, with the fear-avoidance model gaining considerable support in the literature (see [105] for review). In brief, the fear avoidance model

argues that fear of pain and (re)injury prevents normal return to activity, which leads to deconditioning and disability [105]. Although the primary application of the fear avoidance model has been in consideration of behavioural response to pain and injury, corresponding findings have been reported in the pattern of motor control [68]. Several studies have reported differences in trunk muscle activity between fearful and non-fearful back pain patients. For instance fearful patients have a greater reduction in endurance of the paraspinal muscles [6] and less relaxation of the paraspinal muscles at the end of trunk flexion [107] than non-fearful patients and controls. Furthermore, it has been suggested that chronic LBP patients have increased paraspinal muscle activity when they are exposed to personally relevant stressors but not when they are exposed to general stressors [26]. Finally, when pain-free subjects rapidly move an arm, but are subjected to moderately painful electrical shocks to the back that are unpredictable in time and amplitude, the response of TrA and deep MF is delayed in a manner that is similar to that seen with experimentally induced LBP [72]. While the latter finding does not confirm that fear of pain causes the changes seen in people with LBP it does suggest that fear may at least replicate the changes. Moreover, it is possible that both pain and fear of pain act directly on the motor centres through a common mechanism. It is important to consider that fear of pain may explain why people who have a history of pain have delayed activity of TrA. Furthermore, if fear of pain can disrupt the normal control of the trunk muscles, this may provide a link between psychosocial factors and physiological changes that lead to recurrence of pain. It could also be interpreted that these changes in motor control are an adaptation to limit loading and prevent recurrence. However, we propose that these adaptive strategies may provide a short term solution with long term sequelae (see below).

If pain or other supraspinal mechanisms such as fear can disrupt motor control, why does this lead to the relatively consistent finding of reduced activity of deep intrinsic spinal muscles and increased activity of the large superficial muscles? The explanation may lie in the pain-adaptation model of Lund and colleagues [63]. This model stipulates that in the event of pain, the alteration in motor control serves to limit movement. During movement, this involves a decrease in agonist muscle activity and an increase in antagonist activity so as to limit the velocity, force and range of movement [95]. This pattern of response has been observed in clinical and experimental pain studies for many regions of the body including the jaw [95] and trunk [113]. In terms of control of a segment such as the trunk, the response may also involve general stiffening of the body segment(s) by muscle co-activation. Panjabi [77] and Cholewicki [13] predicted that such a response would

increase vertebral control and is consistent with the augmented activity of the large, superficial trunk muscles.

Consistent with this, there is evidence of relative stiffening of the spine in pain. Moe-Nilssen et al. [71] reported reduced trunk movement during gait during experimentally induced pain, and [35] showed that trunk movement following a support surface translation is reduced during pain. Hypothetically, if the general stiffness of the spine is increased, the CNS may perceive the demand for ‘fine-tuning’ to be diminished, leading to reduced activity of the deep intrinsic spinal muscles despite the potential long-term sequelae of this strategy (see below). After resolution of the pain, this adapted strategy may also resolve, or, in the presence of ongoing fear of pain or other reinforcement, persist to chronicity. This hypothesis requires investigation.

An additional factor to consider is that accurate control of movement is dependent on the sensory element of the motor system. Inaccurate afferent input would affect all aspects of motor control from simple reflex responses (e.g. those arising from stimulation of mechanoreceptors in the muscles [113] or other elements of the spine [52,91,116]) to complex movements that are dependent on an accurate ‘internal model of body dynamics’ (see [34]), which allows the CNS to predict the interaction between internal and external forces. Several studies have reported decreased acuity to spinal motion in LBP [98] and impaired ability to accurately reposition with LBP [9,32]. In addition, muscle spindle sensitivity is altered by pain (e.g. [78] and muscle activity [30], thus any change in activation may adversely affect perception of movement. Finally, several studies have argued that sensory acuity may be reduced by fatigue [12], thus decreased muscle endurance with injury or pain may lead to impaired sensory acuity via increased fatigability.

4. Possible outcomes of motor control changes

In view of the differential changes in the deep intrinsic muscles and the superficial muscles in the presence of pain, it is critical to consider possible sequelae of these changes. All trunk muscles are required for control and stability of the spine [77] and it is clear that stability is dependent on the interplay between an array of muscles, both intrinsic and superficial [5,14,115]. Yet, there is considerable redundancy in the motor system with many muscles potentially able to perform similar functions. A change in strategy of trunk muscle control, toward increased stiffening of the spine via increased activity of large superficial muscles, which has been predicted [13,77] and shown to occur (see above), would seem to satisfy the demands for spinal function. However, we propose several side effects of this strategy that may compromise lumbopelvic health and potentially lead to

long-term sequelae. The basis for this hypothesis is that the contribution of the deep intrinsic spinal muscles to trunk control is that of ‘fine-tuning’ of intervertebral motion. Although it is unlikely that differentiation in muscle function can be described in a dichotomous manner, in general it has been suggested that, in contrast to intrinsic muscles, the large and superficial trunk muscles that transcend the lumbar spine and pelvis have a more significant contribution to prevention of buckling of the spine [5,14,115] and to balance external loads [5]. These are also the muscles that have the greatest potential to generate torque to move the trunk.

In contrast, in vivo [43,55,85], in vitro [110] and modelling studies [110] argue that the deep intrinsic muscles, such as TrA and the deep fibres of multifidus, are critical for the control of intervertebral motion. Thus, data suggests that the deep muscles might provide the ‘fine-tuning’ as a component of the complex interdependent activity of the trunk muscles to stabilise the spine. We suggest that in the pain adaptation model, the response to pain of stiffening the spine with increased activity of the large muscles may be at the ‘cost’ of a loss of this ‘fine tuning’. Other factors require consideration. First, movement is an important element of spinal function. It is known that in healthy subjects the CNS uses movement rather than simple stiffening of the spine to overcome challenges to stability [44] and reduce energy expenditure [58]. A strategy of trunk stiffening, although requiring less complex neural control, may compromise optimal spinal function. Second, co-activation of the superficial muscles may have a loading cost. The superficial trunk muscles generate torque at the trunk. This torque must be overcome by antagonist activation in order to keep the spine upright, and this co-activation results in a compressive load on the spine [31]. Excessive compression, which results in increased intradiscal pressure and loading through the posterior elements of the spine has long been considered to be a risk factor for spinal degeneration and pain [74]. If greater demand is placed on the superficial muscle system, the loading may be increased. Third, trunk muscles are involved in functions other than spinal control and movement. As the superficial abdominal muscles depress the rib cage and are involved in forced expiration [21], increased activity of these muscles in people with pain may lead to compromised respiratory function, for example restricted movement of the chest wall. In contrast, TrA has a limited effect on rib cage motion due to its horizontal fibre orientation and contributes to expiratory airflow via rostral displacement of the abdominal contents [21]. In a recent study we have shown that of the abdominal muscles only TrA can coordinate respiratory and postural functions [40]. Thus, changes in trunk muscle activity may be problematic from a systemic point of view. While each of these hypotheses requires further investigation, additional support comes from the litera-

ture which suggests that reorganisation of the control of the deep and superficial trunk muscles through motor learning strategies [84] leads to reduced pain and disability associated with LBP [76] and reduced recurrence of pain [36].

In summary, relatively consistent patterns of change in activity have been identified in the literature, although there are considerable inter-individual differences. While the mechanisms of these changes are not completely understood, there is compelling evidence to suggest that pain may be responsible for the change, at least in some individuals. The consequence of these changes may potentially be a factor in the recurrence of LBP. Taken together these data are consistent with contemporary strategies for rehabilitation of patients with LBP.

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