ARTICLE

Low-Back Pain Patients Learn to Adapt Motor Behavior With Adverse Secondary Consequences

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VAN DIEËN, J.H., H. FLOR, and P.W. HODGES. Low-Back pain patients learn to adapt motor behavior with adverse secondary consequences. *Exerc. Sport Sci. Rev.*, Vol. 45, No. 4, pp. 223–229, 2017. *We hypothesize that changes in motor behavior in individuals with low-back pain are adaptations aimed at minimizing the real or perceived risk of further pain. Through reinforcement learning, pain and subsequent adaptations result in less dynamic motor behavior, leading to increased loading and impoverished sensory feedback, which contribute to cortical reorganization and proprioceptive impairments that reduce the ability to control lumbar movement in a robust manner.*
Key Words: reinforcement learning, nociception, postural control, motor control, sensory feedback, perceived risk of pain

Key Points

- Low-back pain tends to change motor behavior toward increased coactivation, reduced deep muscle activity, and less within-subject variance.
- Minimizing a weighted sum of costs, including the risk of losing control and nociceptive input, predicts these pain-related changes.
- In addition to actual benefits with respect to risk and nociception, changes may be driven by maximization of perceived benefits.
- Originally adaptive changes have negative consequences that may outweigh benefits in the long term.

INTRODUCTION

Low-back pain (LBP) is widely prevalent and, globally, is the leading cause of years lived with disability due to the functional limitations, limited benefits of treatment, and frequent recurrence (15). Motor control exercise is a commonly used treatment for LBP. Although systematic reviews support its effectiveness, like other treatments for LBP, the effect size is not large (33). A better understanding of the nature of and the mechanisms behind motor control changes in LBP should lead to more effective treatments.

Motor control in LBP is changed at many levels of the nervous system. Studies of individuals with and without LBP have reported differences in voluntary trunk muscle activation (45), trunk muscle reflexes (32), and trunk kinematics (20) and in cortical mapping of sensory inputs from (5), and motor outputs to (36), the trunk. However, the literature is far from consistent regarding the nature of these differences. For example, according to a systematic review, there is support of both an increase and a decrease of trunk muscle activation in individuals with LBP (45).

Here, we propose a hypothesis on how LBP affects motor control, which may account for these inconsistencies. In reinforcement learning, a reward (positive reinforcement) or the absence or reduction of a cost (negative reinforcement) increases the likelihood that a performed behavior will be repeated and thus learned. In this context, movement-related pain may function as a negative reinforcement and the sense of being able to prevent pain provocation as a positive reinforcement. Motor control can be considered as the outcome of a learning process aimed at optimizing a combination of costs and rewards. For clarity, either costs or rewards usually are inverted, such that a sum of weighted costs can be minimized or a sum of weighted rewards can be maximized.

In pain-free conditions, costs associated with control efforts or muscle activation are considered most important. We assume that with LBP, weighting of costs associated with potential loss of control over posture and movement and costs associated with potential pain provocation increase, and one learns to minimize this new weighted sum of costs. Although the resulting muscle

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activation and movement patterns may differ between individuals, patients with LBP will tend to control posture and movement more rigidly, causing more stereotypical muscle activation and kinematics. Such adaptations entail greater muscle activity, greater compressive loads on the spine, and impoverished sensory feedback in the form of less variability of the afferent signals from the lumbar area. The latter would have the potential to contribute to neuroplastic changes in sensory and motor regions of the cortex and proprioceptive impairments reducing the ability to control lumbar movement in a robust manner. These secondary, long-term effects may contribute to recurrence and chronicity of back pain. We hypothesize that changes in motor control with LBP reflect functional adaptations acquired through reinforcement learning (Fig. 1).

The proposed hypothesis consists of two parts: 1) LBP causes adaptive motor control changes through reinforcement learning and 2) these adaptations potentially result in negative long-term consequences. Although an adaptive nature of motor control changes with LBP has been proposed previously (*e.g.*, (14,24,43,45)), resulting predictions mainly regarded levels of muscle activation. Furthermore, these previous publications either assumed hardwired reflexive changes in motor behavior with pain (24) or did not address the processes underlying these changes (14,43,45). The notion that adaptations arise through reinforcement learning is important from a clinical perspective. For the second part of the hypothesis, previous publications have alluded to adverse effects of adaptations (14,45), but that this may contribute to reorganization of the sensory and motor cortex has not been discussed previously. The ideas proposed in this article likely are applicable to other movement-related pain syndromes, but we will limit ourselves here to LBP because this allows more specific testing of the hypotheses.

The aims of the present article were to describe this hypothesis in more detail, to derive testable hypotheses, and to confront these hypotheses with empirical findings from previously published studies. In the first part of the article, we describe how LBP might induce learning that adapts motor behavior. We provide a formalization of this part of the hypothesis in a simple optimization model of trunk motor control, which yields predictions on motor behavior in patients with LBP that we compare with empirical findings. In the second part of the article, we describe how these changes on motor control may lead to adverse physiological, neurological, and mechanical effects and how these secondary effects might contribute to the persistence of LBP.

BY WHAT MECHANISM DOES LBP AFFECT MOTOR CONTROL?

Many studies have shown that normal motor control can be approximated by models in which the central nervous system (CNS) is assumed to use some form of optimal control, *i.e.*, it achieves the task goal at a minimal cost. Examples of costs proposed in the literature are neural drive or muscle activation, required muscle force or associated metabolic costs, and path length or jerk. In line with this assumption, pain-free subjects recruit trunk muscles in a manner that is consistent across individuals and compatible with minimization of dynamic costs (42).

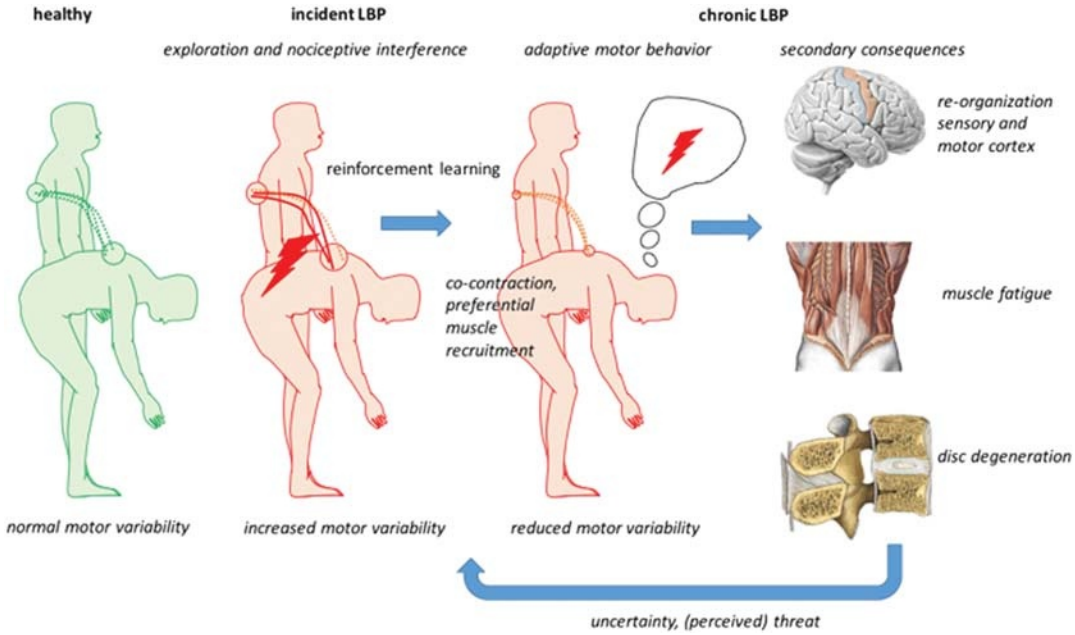

Figure 1. Schematic illustration of the hypothesis on motor control changes with low-back pain (LBP). In normal conditions, motor behavior is somewhat variable because many near optimal solutions can be used to achieve the task goal. This is illustrated on the left, with the dotted lines representing near optimal movement trajectories of a marker on the trunk and the circles representing the area of end point positions of this marker over a series of repeated movements. With incidence of LBP, variability will increase because nociception interferes with motor control. The subject will then learn an adapted motor pattern with negative reinforcement by nociception, pain perception, and positive reinforcement because the sense of having control over movement. Once the new optimal pattern (dashed line) is found, motor variability is reduced because costs of movement (pain, potential loss of control) may rapidly increase with changes in behavior. The adapted motor behavior furthermore consists of increased levels of antagonistic contraction and preferential recruitment of muscles with large moment arms. This adaptive behavior may lead to secondary consequences in joint structures and muscles due to increased and more sustained muscle activity and to reorganization of the sensory and motor cortex because of the more stereotypical sensory feedback and selective muscle recruitment. These changes may increase uncertainty about motor control and hence, perceived movement-related threats, which may further reinforce the adaptations in motor behavior.

Experimentally induced nociception from back muscles, or the resulting pain perception, changes control of trunk muscles in a manner resembling that observed in clinical LBP (9,45). Nociception may modify motor control online by providing feedback regarding the consequence of the exerted control, possibly even in pain-free conditions. In addition, pain experience is likely to cause anticipatory changes in motor control (38), *i.e.*, an individual would choose motor strategies to avoid pain provocation. We propose that changes in control arising from nociception and pain can be understood in the context of optimal control.

Optimal control generally is thought to be the outcome of reinforcement learning. Efference copies of the motor commands and feedback on the consequences of the motor actions provide the individual with information on performance and associated costs, allowing adaptation of motor commands to achieve the task goal with minimal costs in terms of neural drive (control effort) or metabolic or mechanical costs of muscle force production. It has been suggested that movement planning occurs sequentially at two hierarchical levels: initially to plan the kinematic trajectory and subsequently to plan a muscle recruitment pattern that fits the planned kinematic trajectory (18).

In the present context, adaptation of motor control to changing conditions (the presence of nociception from the spine and the perception of LBP) is of particular interest. Many reinforcement learning studies have addressed adaptation in the control of goal-directed arm movements to mechanical perturbations. Such studies show that subjects gradually but consistently adapt to the mechanical perturbations to converge on a near straight-line hand trajectory that closely resembles the unperturbed trajectory (18). Babic *et al.* (1) recently applied a similar paradigm to study adaptation of a squat movement to mechanical perturbations that could potentially lead to balance loss. The results differed in important ways from those in goal-directed arm movements. First, participants converged on an adapted center of mass trajectory after relatively few trials. Second, although individual participants converged on a consistent trajectory, between-subject variance in the adapted trajectories was substantial. These differences were interpreted as indicating that the CNS takes other costs into account in addition to costs of movement. Specifically, the risk of losing balance is significant. If preventing balance loss is an objective of the CNS, then rapid convergence on an adapted trajectory (consistent with experimental results) is of great importance. However, this may represent a local minimum because further exploration to adapt would involve a steep increase in costs that may actually increase the risk of balance loss.

More generally, we suggest that motor control is the outcome of a learning process in which a weighted sum of costs is minimized. The weighted sum comprises costs related to control effort and muscle activity, metabolic costs, and fatigue development, as well as costs that reflect more indirect effects of the selected motor pattern such as the risk of losing control over posture or movement and the possibility of pain provocation. In an individual who had pain, the latter two costs would be expected to receive higher weighting for an individual with LBP than one without pain.

A Model of Motor Control Changes With LBP

Here, we use the selection of a muscle recruitment strategy given a specific, static kinematic situation of the trunk as a simplified model of motor control changes with LBP. We simulate a simple muscle-joint system, representing the trunk, with one degree of freedom, controlled by two extensor muscles and one antagonistic flexor muscle (Fig. 2). The task requires a given net moment (20 N·m) to be produced by these three muscles, which is comparable to standing with a slightly forward-inclined trunk.

We assumed that three costs govern the selection of a muscle recruitment pattern. The first cost is to minimize the neural drive, modeled as

$$C_1 = \sum_{i=1}^{n} A_i^p \qquad \text{[Eq. 1]}$$

with A a vector of n (here 3) levels of muscle activation. The value of the exponent p was set to 3 to minimize the loss of muscle force due to sustained activity, which has been shown to accurately predict muscle activity of trunk muscles (42) (reasonable predictions were generated with any $p > 1$). The second cost is trunk admittance, in which the objective is to maximize the resistance against perturbations. In reality, this is achieved by combining coactivation and increases in feedback gains (47). In this simplified model, this effect can be achieved only through increasing muscle stiffness by means of coactivation. Effects of muscle contraction on joint stiffness can be modeled as a function of muscle force, muscle length and muscle moment arm (31), which yields the cost function

$$C_2 = 1/\sum_{i=1}^{n} \left(F_i d_i^2 / l_i\right) \qquad \text{[Eq. 2]}$$

with F the vector of muscle forces calculated as $A * F_{max}'$, d the vector of muscle moment arms and l the vector of muscle lengths. The third objective function is to minimize nociceptive afference. We used the simple assumption that nociception increases with the total muscle force produced, yielding

$$C_3 = \left(\sum_{i=1}^{n} F_i\right)^p \qquad \text{[Eq. 3]}$$

We arbitrarily set p to 1. However, results presented in the next sections held over a range of values for p from 0.5 to 10.

The three cost functions were normalized to their maximum values over the range from zero to full activation for all muscles to assure equal scaling over the range of muscle activation from 0 to 1. The costs as functions of the forces in the two extensor muscles are illustrated in the top panels of Figure 2. Costs 1 and 3 penalize high muscle forces, whereas cost 2 penalizes low muscle forces. The resulting cost function is a weighted sum of all three scaled-cost functions

$$\sum_{j=1}^{3} w_j C_j \qquad \text{[Eq. 4]}$$

with w the vector of weight factors and by definition $\sum_{j=1}^{3} w_j = 1$. This weighted cost function is minimized subject to the constraint

$$M_{net} = -\sum_{i=1}^{n} F_i d_i \qquad \text{[Eq. 5]}$$

ensuring that the three muscles together produce a net moment (M_{net}) of 20 N·m.

We explored the effects of changing the vector w to simulate the effects of LBP. The initial setting for the pain-free condition

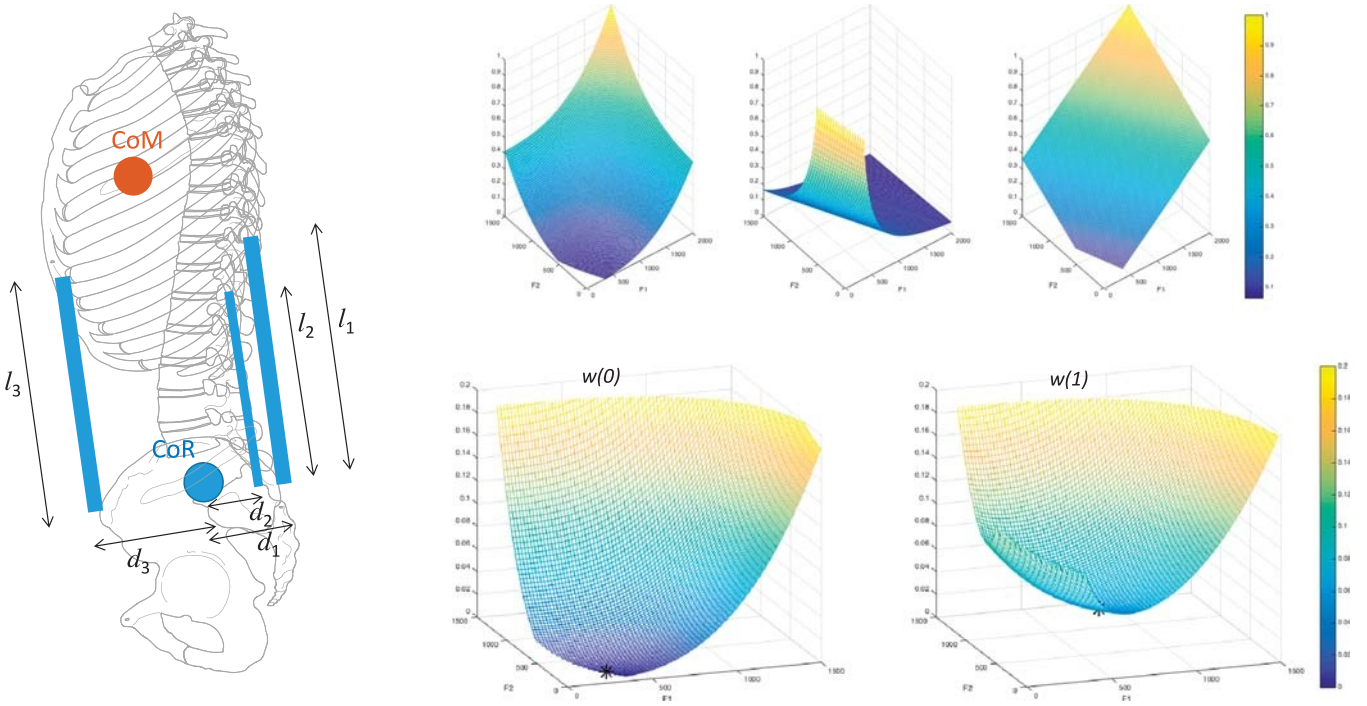

Figure 2. The left figure illustrates the model, with muscles represented by thick blue lines and muscle lengths (l_i) and moment arms (d_i) indicated by arrows. The upper body center of mass (CoM) is indicated by the red circle, the joint center of rotation (CoR) by the blue circle, with the inclination of the upper body leading to an external moment around the CoR of 20 N·m. The right graphs show in the upper panels the three objectives outlined in the text as a function of forces produced by muscles 1 and 2. For any combination of muscle forces in these diagrams, *i.e.,* any position on the x-y plane, the force in muscle 3 is determined given the constraint function, which requires the three muscles combined to produce an internal moment of −20 N·m. The lower panels illustrate the weighted sum of the three objectives for weight vectors specified in the text, with the pain-free condition (*w(0)*) on the left and the pain condition (*w(1)*) on the right. The * indicates the optimum combination of muscle forces for each objective function.

was $w(0)$ = [0.9975, 0.0025, 0], *i.e.*, no objective to minimize nociception and only a low weighting of admittance, which was selected to obtain a realistic level of antagonistic muscle activity. For the pain condition, weighting factors were set to $w(1)$ = [0.8, 0.1, 0.1], implying a substantial penalty for admittance and nociception.

The lower panels in Figure 2 illustrate the cost function relative to the load sharing between the two synergistic extensor muscles. Reinforcement learning takes place through variation of muscle forces and observing changes in the costs. This is accomplished by exploring the surface of the weighted cost function and converging on an optimal recruitment pattern that combines the muscle forces with the lowest cost (indicated by an asterisk).

Model Predictions

As can be seen in Figure 2, changing the weights in the cost function from $w(0)$ to $w(1)$ shifts the optimal muscle recruitment pattern toward higher activation of the larger of the two extensor muscles, because of the higher weighting of admittance, which favors the muscle with the larger moment arm. In addition, the level of antagonistic coactivation increases. Furthermore, the curvature of the surface of the weighted cost function around the optimum solution is steeper for $w(1)$ than for $w(0)$. The model results predict higher coactivation, preferential recruitment of large superficial muscles, and less exploration with pain than in the pain-free condition. Of note is that the same model would predict less obvious qualitative changes in muscle activity when higher net moments are produced. This is because the second cost function, limiting admittance, affects muscle recruitment mainly at low levels of muscle activity, whereas the third cost function, to limit nociception, penalizes high muscle activity. It should be noted that the present cost function for pain is quite simplistic. In reality, it could be that activity of one muscle provokes pain and that of another muscle does not. This would require another formalization of the cost function to minimize nociception. However, such a cost function is unlikely to be universal as the painful muscle may be different in different individuals. Therefore, we choose this simple cost function.

An important aspect of the proposed hypothesis is that minimization is based on reinforcement learning. This implies that costs taken into account can reflect perceptions rather than direct information from sensory feedback or efference copies. Although nociceptive afference may lead to the perception of pain, the presence, intensity, and nature of this perception is shaped by cognitive factors, which include the expectation of pain (4). The objective may be to minimize the perception of pain instead of the nociceptive input or to maximize perceived safety rather than actual robustness of the motor strategy. This introduces the possibility for a feedback mechanism, where one change to the motor strategy leads to a perception of increased safety or decreased pain. This may reinforce the strategy choice despite the possibility that the perception could be unrelated to effects of the movement strategy on actual safety or nociception. This may explain why increasing muscle activity can reduce perceived pain under a constant nociceptive input (19). We recently have provided more specific data to this effect. When

participants were provided with a noxious stimulus during wrist movement, adaptation of the motor strategy led to reduced pain perception regardless of whether the noxious input was reduced by the adaptation (Bergin MJ, Tucker K, Hodges PW. Unpublished data, 2016).

It is important to note that the proposed model does not predict local minima of the weighted sum of costs. Nevertheless, the curvature of the objective function for the condition with pain suggests that individuals might end up using suboptimal solutions because exploration starting from any near optimal point is heavily penalized with changes of the recruitment pattern in most directions. Local minima will occur when one of the objective functions is nonconvex. This may occur for pain-related objective functions in kinematic planning because pain often changes nonmonotonically with trunk angle, a so-called painful arc (2). It also may occur when a cost function varies with activity of certain muscles only, which as discussed could be the case for nociception.

CHANGES IN MOTOR BEHAVIOR WITH LBP

The proposed reinforcement learning model predicts that individuals with LBP will display 1) greater coactivation, 2) more activity of large superficial muscles, and 3) lower variability in muscle recruitment than individuals without pain. Pain-related changes will be driven not only by nociception but also by pain-related cognitions, such as fear or anticipation of pain, which would change weighting of the objectives similarly to nociception. Between-subject variability in motor strategies may be larger among individuals with LBP than among those without pain. The following sections review the evidence that clinical LBP and experimental noxious stimuli to the low back are associated with changes in motor behavior in line with these predictions.

Coactivation

The model predicts that coactivation increases during pain to reduce admittance. Such a response has been observed in healthy subjects when task conditions are perceived to threaten control of trunk movement (44). A pain-related increase in coactivation also would be expected in low-demanding tasks, such as maintenance of unloaded upright posture because intrinsic stiffness of the trunk is minimal in such conditions (3). No feedback control was implemented in the model, but a high weighting of an objective to minimize admittance with pain also would predict an increase in feedback gains (47). These changes in muscle recruitment would be associated with small displacements after mechanical perturbations. In line with the model predictions, experiments using experimental noxious stimuli to the low back have shown increased activity of trunk muscles and decreased displacements after trunk perturbations (8). In addition, studies comparing participants with and without LBP have commonly reported increased activity of trunk muscles, particularly in low-intensity activities (45), as proposed by the model. Moreover, smaller displacements after perturbations have been found in participants with LBP (7,17).

Preferential Recruitment of Superficial Muscles

The model predicts that pain biases recruitment toward superficial muscles with large moment arms relative to deep muscles with smaller moment arms. This prediction is supported by studies on anticipatory activation of trunk muscles in association with perturbations of trunk posture caused by rapid movements of the upper and lower limbs. These studies have reported reduced and delayed activation of deep muscles relative to superficial muscles after administration of a noxious stimulus to the low back (10). In addition, studies comparing participants with and without LBP have consistently reported delayed recruitment of deep muscles relative to superficial muscles (9,11–13,25,28) and earlier activation of the superficial abdominal muscles in participants with LBP (30).

Within-Subject Variability

The higher weighting of objectives related to admittance and nociception caused the surface of the weighted cost function to be more curved, which would suggest that exploration is limited by pain. This prediction was supported in a recent study, where we applied a force field to the ankle during gait and provided noxious input to the tibialis anterior muscle. Participants with and without pain adapted quickly to the force field to continue walking without loss of balance, but only the participants without pain were able to continue to refine their adaptation; participants with pain continued to use their initially adopted solution (Bouffard J, Salomoni SE, Mercier C, Tucker K, Roy JS, van den Hoorn W, Hodges PW, Bouyer LJ. Unpublished data, 2016.). The limited exploration would lead to a reduced within-subject variance of trunk muscle recruitment and trunk kinematics. Both reduced variance of muscle recruitment patterns after administration of a noxious stimulus to the low back (*e.g.*, (40)) and increased variance have been reported (21). Note that our hypothesis would predict an initial increase in variability in response to incident pain because this acutely changes movement-related costs followed by a reduced variability as a result of the steeper increase in the weighted sum of costs with pain. This is in line with differences observed between effects of experimentally induced acute pain and chronic pain in the neck and shoulder region on movement variability (26). Individuals with chronic LBP have been reported to display lower variability of muscle recruitment (6,16) and lower variability of trunk kinematics (22,29) than individuals without pain. However, opposite findings also have been reported (22,49).

Effect of Pain-Related Cognitions

In line with our hypothesis, the effects of LBP on trunk muscle activity (41) and trunk displacement after perturbations (17) are more pronounced in participants with high scores on pain catastrophizing or fear of movement. Similarly, delayed deep muscle activation was more pronounced in participants with a high fear of pain (39), and variability was decreased in participants with negative pain-related perceptions (6), which persisted after pain had resolved in these participants (30).

Between-Subject Variability

Although not a prediction of our model, the general hypothesis suggests that different individuals may end up using different motor strategies after the development of LBP because individuals do not explore to find the optimum. Formal comparisons of between-subject variability in groups of individuals with and without LBP are lacking, but one systematic review concluded that between-subject variability of trunk kinematics is indeed larger among study populations with LBP than among those without LBP (20).

Conclusions Regarding Motor Behavior in LBP

The model used to illustrate the hypothesis that changes in motor behavior with LBP are determined by reinforcement learning, which minimizes uncertainty and pain, is simplified. Most importantly, nociceptive inputs or pain experiences may shape the objective function for avoiding pain in a much more complicated way than represented here. Despite this limitation, empirical observations are consistent with predictions overall. Inconsistency of findings with respect to within-subject variability may be related to the fact that LBP also interferes with motor control through factors such as impaired proprioception (50), or pain-related inhibition (*e.g.*, (25)). Consequently, the observed variance may be the net effect of opposing effects of pain interference and adaptations resulting from reinforcement learning. Moreover, pain-related perceptions moderate the effects of LBP on motor behavior (as proposed by the model), which leads to variance in effects between individuals and between conditions. For instance, adaptive motor behavior may be more obvious in tasks that impose a substantial threat of pain.

CONSEQUENCES OF CHANGES IN MOTOR BEHAVIOR WITH LBP

Although adaptive and with short-term or immediate benefits, changes in trunk motor control due to LBP may have adverse mechanical, physiological, and neurological consequences that, in the long term, may outweigh the benefits. Increased and more sustained trunk muscle activity would come at a cost in terms of muscle and spine loading. Trunk extensor contractions at intensities below 5% of maximum activation cause a decline in muscle force capacity within 30 min if variability of activation is low (46). Patients maintaining sustained trunk muscle activity, even at a low level, may incur decreased muscle capabilities and related discomfort, or even pain of muscular origin (48), especially in the presence of sensitization. Increased trunk muscle activation to reduce admittance also comes at the cost of increased spinal loading. Individuals with LBP have been shown to expose their spine to higher forces during lifting than individuals without LBP due to coactivation (*e.g.*, (27)). Furthermore, low-level coactivation of trunk muscles may occur in LBP patients even at rest (45), implying sustained compression of the spine, which animal models implicate as a cause of intervertebral disc degeneration (23). There also may be adverse mechanical consequences of the decreased variation that would be expected to accompany LBP. It is increasingly recognized that some degree of variation in tissue mechanical loading is essential for tissue health (34).

In addition to mechanical consequences, adaptations in trunk control due to LBP can be expected to have neurological consequences. Reduced variability in posture and movement likely yields impoverished afferent feedback. We hypothesize that this may contribute to the changes in representations of the trunk in the somatosensory (37) cortex observed with LBP. In turn, these changes in the sensory cortex could contribute to the impaired proprioception (35) and the related imprecision in control of trunk movement in individuals with chronic LBP (50). Furthermore, conventional muscle recruitment patterns, with relative inhibition of deep muscle activity, are associated with changes in representations of trunk muscles on the motor cortex in individuals with LBP (5). These brain changes, in addition to the reduced motor variability (30), may limit the ability to relearn normal motor control even after LBP has receded. It should be noted that reduced proprioceptive information might strengthen the reinforcement learning process induced by LBP as a consequence of increased uncertainty of the outcomes of motor commands and consequently increasing perceived threat.

CONCLUSIONS

To further test the hypothesis that motor behavior changes in individuals with LBP are the result of a reinforcement learning process — in which potential loss of control over posture and movement and provocation of pain would be minimized — several avenues of research could be explored. Manipulation of nociception and pain as a function of motor behavior could be used to shape objective functions and, when used as such, test the hypothesis that strategies to avoid the painful stimulus develop through exploration and reinforcement. Furthermore, the predictions of our model that within-subject variance of trunk motor behavior is smaller in individuals with LBP than in individuals without pain needs further study, as does the prediction that between-subject variance is higher among individuals with LBP.

The proposed hypothesis, specifically the potential for changes in motor behavior to underpin adverse secondary effects, indicates that interventions targeting these changes may be needed despite their initially adaptive nature. Furthermore, the hypothesis suggests that motor control exercises should be aimed at increasing admittance, inhibition of superficial muscles and stimulation of deep muscles, training of proprioception, or instilling confidence in the ability to control lumbar motion. Finally, when designing interventions, the potential for a reduced ability to relearn normal motor control because of the presence of pain and the secondary effects thereof needs to be considered.

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