PTJ: Physical Therapy & Rehabilitation Journal | *Physical Therapy*, 2023;103:1–13
https://doi.org/10.1093/ptj/pzac151
Advance access publication date November 1, 2022
Perspective

The Fit-for-Purpose Model: Conceptualizing and Managing Chronic Nonspecific Low Back Pain as an Information Problem

Benedict M. Wand, PhD[1,*], Aidan G. Cashin, PhD[2,3], James H. McAuley, PhD[2,3], Matthew K. Bagg, PhD[2,4,5], Gemma M. Orange, MSc[1], G. Lorimer Moseley, PhD[6]

[1]Faculty of Medicine, Nursing and Midwifery and Health Sciences, The University of Notre Dame Australia, Fremantle, Australia
[2]Centre for Pain IMPACT, Neuroscience Research Australia, Sydney, Australia
[3]School of Health Sciences, Faculty of Medicine and Health, University of New South Wales, Sydney, Australia
[4]Curtin Health Innovation Research Institute, Faculty of Health Sciences, Curtin University, Perth, Australia
[5]Perron Institute for Neurological and Translational Science, Perth, Australia
[6]IIMPACT in Health, University of South Australia, Kaurna Country, Adelaide, Australia

*Address all correspondence to Dr Wand at: benedict.wand@nd.edu.au; or Dr Moseley at lorimer.moseley@unisa.edu.au

Abstract

Chronic nonspecific low back pain (LBP) is a complex and multifaceted problem. The following Perspective piece tries to help make sense of this complexity by describing a model for the development and maintenance of persistent LBP that integrates modifiable factors across the biopsychosocial spectrum. The Fit-for-Purpose model posits the view that chronic nonspecific LBP represents a state in which the person in pain holds strong and relatively intransient internal models of an immutably damaged, fragile, and unhealthy back, and information that supports these models is more available and trustworthy than information that counters them. This Perspective proposes a corresponding treatment framework for persistent pain that aims to shift internal models of a fragile, damaged, unhealthy, and unchangeable self toward the formulation of the back as healthy, strong, adaptable, and fit for purpose and to provide the system with precise and trustworthy evidence that supports this supposition while minimizing information that works against it.

Keywords: Back Pain, Biopsychosocial, Chronic Pain, Predictive Processing, Rehabilitation

Received: February 2, 2022. **Revised:** May 6, 2022. **Accepted:** August 20, 2022

Introduction

Chronic nonspecific low back pain (CNSLBP) is a common and complex problem with considerable personal and societal consequences.[1–3] Prognosis is unfavorable, with recovery occurring in less than one-half of people with CNSLBP over a 1-year period.[4] Despite a vast range of potential treatment approaches,[5] an optimal form of care has not yet been identified. A large number of cross-sectional studies have detected countless potential targets for management across the biopsychosocial spectrum,[2] and it is commonly suggested that effective care needs to target multiple drivers to the clinical condition.[6] Combining interventions from different specialties into a multimodal treatment plan has been shown to be more effective than single therapies or usual care,[7] and multimodal care is an approach endorsed by clinical practice guidelines,[8] though improvements in outcome remain modest. Optimization of multimodal care and further improvements in treatment outcomes may come from a richer understanding of the interaction between modifiable contributing factors that exist across the biopsychosocial spectrum and how these issues coalesce to shape the chronic pain experience and trajectory.

Here we present the Fit-for-Purpose Model (FFPM),[9] which shares some characteristics with other approaches to understanding and managing CNSLBP but extends them by integrating cognitive and behavioral factors with modifiable neuroimmune processes. Similar to the Fear-Avoidance,[10,11] Health Belief,[12] and Common Sense[13] models, the FFPM recognizes the importance of interactions between beliefs and behaviors in persistent pain. Also similar to those models, the FFPM has not suddenly come into being at a "single, defined moment" (eg, Leventhal et al.[13p937]) but has undergone iterative development as new discoveries have been made and new interventions tested. What sets the FFPM apart from other models is the integration of and weighting given to foundational cognitions about "how pain and healing work"; the impact on information processing, cognitions, and clinical symptoms of a plastic and adaptive neuroimmune system[14–16]; and incorporation of contemporary models of learning, not least predictive processing,[17–19] into explaining the persistence of pain and disability. Finally, we have developed a coherent, staged, complex treatment approach aimed at secondary and tertiary prevention for people with CNSLBP based on our model, which is currently being tested in a clinical trial.[20] This Perspective Piece describes the theoretical underpinnings of the FFPM and the rationale behind each component of the complex care package. Ultimately, our aim is to provide a reasoning-based framework that will help clinicians make sense of the complexity of CNSLBP and thus provide contemporary, coherent, biopsychosocial informed management.

Theoretical Underpinnings

Noxious input from the body is likely an important part of many peoples' pain experience, but pain does not reflect a simple readout of nociceptor activity.[21] Current theory suggests that pain might best be considered an actively constructed experience based on multiple sources of information and reflects both conscious and non-conscious assessment that one's body is under threat and in need of protection.[19,22]

This standpoint is consistent with current concepts of perception more generally. Where once the brain was viewed as a passive processor of sensory information, it is now considered a dynamic organ of inference that makes sense of the world by actively generating hypotheses regarding the sensory inputs the individual encounters and then tests these hypotheses against sensory evidence.[23] What is perceived is the system's final estimate of the most likely cause of sensory input derived from both prior beliefs about the body and the world and current sensory information from the body and the world.[17] With regard to pain, the individual builds models of the body based on prior information from and about the body and uses these models to predict the causes of ongoing sensory information from the body, contingent on the context in which the individual is situated. Under this paradigm, pain then represents the conscious expression of a final estimate, from the integration of these information sources, that the sensory stream represents an abnormal somatic event in which there is a threat to bodily integrity and protective behavior would mitigate that threat.[18,19] In this way psychological and social factors are stitched into the fundamental neurobiological processes that underpin the emergence of a conscious experience we recognize as pain.

The FFPM leans heavily on the notion of pain emerging as the result of a dynamic interplay between stored information and information transformed by, and encoded within, sensory processing systems. Particularly, the FFPM posits the view that CNSLBP represents a state in which the individual holds relatively intransient internal models of an immutably damaged, fragile, and unhealthy back and information from the body (and the world) that supports these models is more available and trustworthy than information that counters them. The following section explores how this self-reinforcing state could arise and lead to maintenance of an ongoing pain state.

Development of CNSLBP

A Cognitive Model of an Immutably Damaged, Fragile, and Unhealthy Self Begins to Emerge

Every persistent pain state has an onset, and there are characteristics of the experiences of the individual with acute low back pain (LBP) that are particularly relevant to the FFPM. Most important are those factors that start to shape beliefs that the back is damaged, fragile, and unhealthy and that this state is resistant to change. At a societal level, it seems that LBP is viewed primarily as a result of injury to somatic structures within the back,[24,25] generally as a response to mechanical overload.[26] We contend, alongside others, that much of the societal messaging about back care, particularly in the work place, reinforces ideas that "backs are fragile and easily injured"[24] and that back pain is a particularly intransient problem.[27] This has implications for the individual trying to make sense of their problem based on their own internal resources but also shapes the information they would obtain from their family, their social networks, and their world more broadly. That acute LBP is commonly characterized by movement-evoked pain and often eased by rest and inactivity makes this model of pain as a marker of damage intuitively sensible as well. So both the lived experience of the person in pain and their social and informational environment contribute to the formulation of a damaged, unhealthy, and load-intolerant self.

Seeking professional help for the problem may also enhance this narrative. A number of diagnostic models for spinal pain reference tissue injury and damage as primary contributors to the pain state[28,29] and reinforce this through the provision of treatment strategies that promote unloading and volitional protection of the spine,[30–32] including messaging that protecting "your fragile back" may be required long term.[33] Furthermore, though not recommended,[34,35] imaging of the spine is still common for people receiving care for acute nonspecific LBP.[36] The high rate of positive imaging findings in the general population[37] makes it highly likely that people exposed to spinal imaging will be provided with information that appears consistent with stored impressions of damage or structural insufficiency. Moreover, explanatory models for symptoms that reference imaging findings not only reinforce damage but they also augment this impression with implied irreversibility and a pathway of progressive deterioration.[38]

Specific clinical characteristics are also likely to drive a strong formulation of an unalterable, unhealthy self. There has been considerable research into identifying factors associated with the transition to chronicity in those with recent-onset LBP.[39–42] A number of clinical characteristics consistently identified by this research effort are likely to reinforce beliefs that the back is damaged, fragile, and unhealthy. Known indicators of poor prognosis such as high pain intensity,[43–45] symptom persistence and/or recurrence,[46,47] and the presence of other health issues[39,48–50] potentially impede recovery as they serve as reinforcers of the notion that the individual is significantly injured and unhealthy. Psychological factors that hinder recovery such as negative affect,[39,41,51–53] pessimism about future outcome,[43,54] and health-related anxiety[51,55] are also plausible reinforcers of this account, particularly with reference to capacity for reversibility and positive change.

The majority of people experiencing an episode of acute LBP recover reasonably quickly.[4] The FFPM contends that those who do not are those in whom social, psychological, experiential, and clinical factors shape particularly strong meta-cognitive and cognitive models of the back as being immutably damaged, fragile, and unhealthy. For these people, the process of trying to make sense of their LBP experience early in an episode leads to an understanding of the problem in which the back is appraised as being fragile and not fit for purpose and viewed as being under threat and in need of protection.

Initial Responses to the View That the Back Is Not Fit for Purpose and in Need of Protection

The model we are proposing brings 3 learning-related mechanisms into play. First, from a predictive processing perspective, as the back is moved and loaded multiple streams of sensory information from the back are generated, and, even in normal circumstances, this likely includes nociceptive input.[56] Information that is expected, based on predictive models of the response of the back to moving and loading, is given more weighting and is therefore more likely to contribute to perception.[57] In the pain-expectant individual, nociceptive input will be particularly weighted and will therefore have more influence on determining if the sensory stream accompanying movement is perceived as representing a harmful, abnormal somatic event. Critically, this increased weighting of nociceptive input is potentially accompanied by decreased weighting of non-nociceptive somatosensory information from the back, the various proprioceptive streams that are associated with back movement, and loading. Second, at a meta-cognitive level, viewing the back as easily injured and pain as a marker of tissue damage will impact the various complex and intertwined processes that are involved in making sense of what is perceived. In the individual who believes the back is not fit for purpose, pain with action is not only more likely, but pain is potentially interpreted in a more catastrophic way. The abnormal somatic event that pain signifies is not seen as minor or transient but taken to represent significant harm or damage to the body[11,58] and an experience more likely to foster distress and disablement. Third, as implicated in the fear avoidance model, concerns about the decreased capacity of the back to cope safely with movement and loading, and viewing pain as a marker of tissue damage, may drive avoidance of some tasks[11] and contribute to changing the way the spine is moved and controlled during other tasks, particularly the promotion of control strategies that increase rigidity[59–62] and decrease movement variability.[63–66] Actions that promote poverty of movement and inflexibility of the movement repertoire potentially change how the back is represented cortically[67] and the information available from the back with movement and loading.

These Responses Induce Functional Changes That Drive Perceptual Models of a Damaged Fragile and Unhealthy Self

Ongoing changes in action, attention, and appraisal of sensory information have potential functional consequences for the back and brain of people in pain. The negative effects of inactivity and unloading on the musculoskeletal system are well documented[68,69] and are recognized as important contributors to ongoing pain in approaches such as the fear-avoidance model.[11] More recently, substantial evidence has also emerged highlighting significant changes within the central nervous system in people with persistent pain. Multiple studies have documented neurochemical,[70] morphological,[16,71] and organisational[14,16,71,72] changes in the brains of people with CNSLBP, and many of these changes are evident in networks involved in attention, sensory processing, and motor planning and function.[73]

Functional changes effecting the back and brain may impact the person with LBP in a number of ways. The factors that we think are most important in maintaining the chronic pain state and that are supported by the current literature are (1) changes in musculoskeletal health such that the back is less fit and load tolerant,[74–76] (2) increased efficiency within nociceptive networks such that there is augmentation of noxious information from the back,[77,78] (3) decreased efficiency within proprioceptive and tactile networks such that there is diminution of non-noxious somatosensory information,[79–88] and (4) disruption of brain grounded sensory and motor neural representations of the back.[67,89–95]

Disruption of neurally encoded representations of the back and diminution of non-noxious somatosensory information from the back will degrade motor control of the back and likely impacts self-perception of the back such that the back starts to *feel* foreign, peculiar, disconnected, and unfit. Qualitative investigations support this, noting that people with LBP perceive the back as fragile and vulnerable[25,27,96] and feel a sense of alienation and rejection of the back.[97–99] When quantitatively evaluated, people with LBP represent the back differently when asked to draw how the back feels to

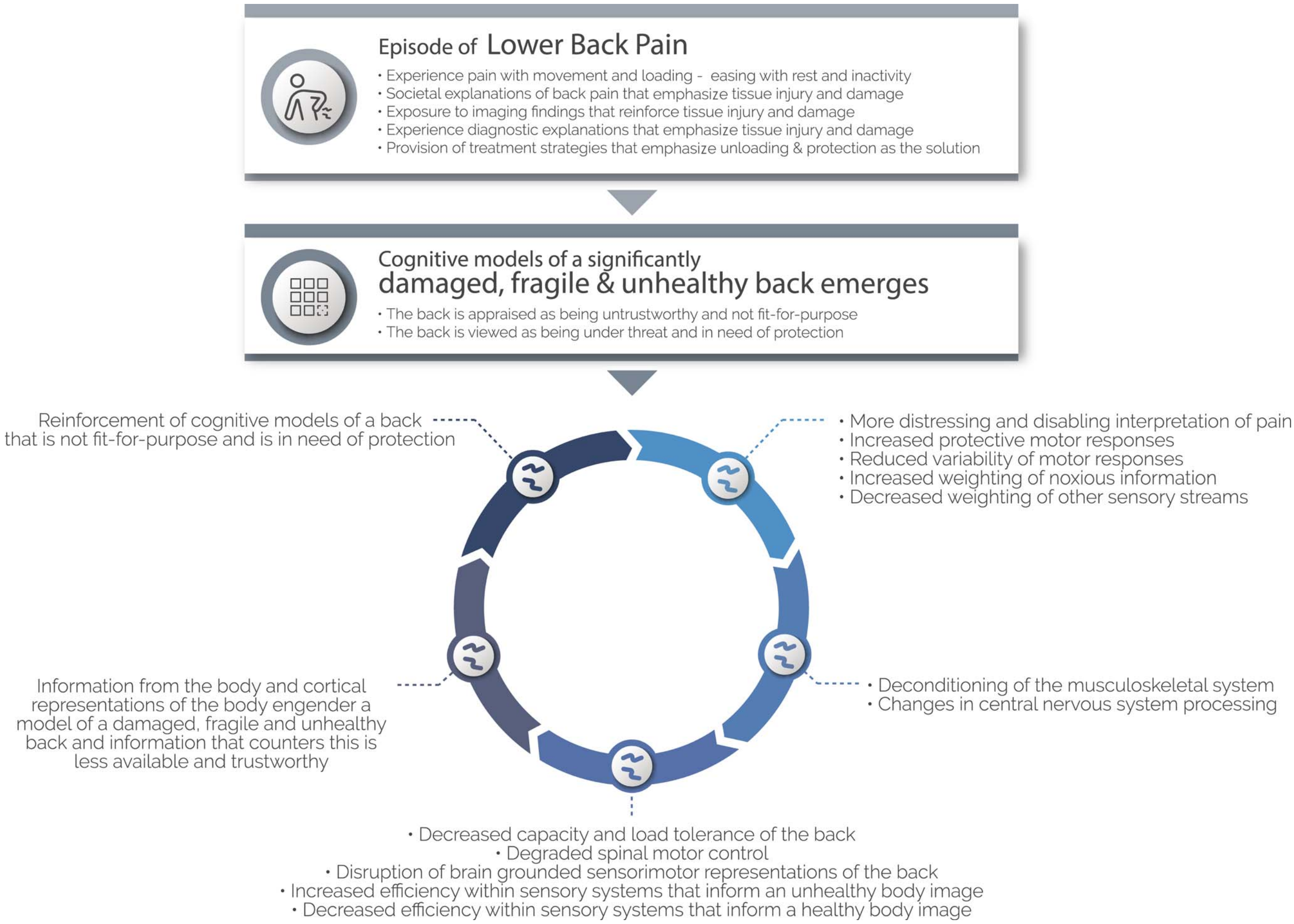

Figure 1. The Fit-For-Purpose Model (FFPM) of chronic non-specific low back pain (CNSLBP).

them[100,101] and endorse questionnaire items associated with altered perceptual awareness of the back.[101–104]

Emerging Perceptual Models Reinforce Cognitive Models of a Damaged Fragile and Unhealthy Self

Central to our approach is the potential for self-sustaining interactions between cognitive models, or how we think about the body in pain, and perceptual models, how the body in pain feels to the individual. A body initially conceptualized as damaged, fragile, and unhealthy increasingly feels damaged, fragile, and unhealthy. The view that the back is not fit for purpose and in need of protection is thus reinforced by information from the back, and we contend that this state can become somewhat self-sustaining. The back is more strongly conceptualized as vulnerable and damaged and the associated behavioral responses further facilitated as the back deconditions, spinal motor control is degraded, self-perception is disrupted, and information that supports the supposition of a damaged, fragile, and unhealthy self is facilitated and more strongly weighted while information that counters this becomes less available and considered less trustworthy (see Fig. 1).

Relevance of the FFPM to Treatment of People With CNSLBP

The FFPM not only provides a novel perspective to help patients and clinicians understand CNSLBP, it also offers a framework to guide treatment and help integrate various common and contemporary practices into a coherent, graded, rehabilitation framework. Management strategies aligned with the FFPM aim to shift meta-cognitive, cognitive, and bodily related internal models that are consistent with a fragile, damaged, unhealthy, and unchangeable self toward the formulation of the back as healthy, strong, fit for purpose, and able to adaptively respond to progressive movement and loading. Information sources from the body as well as information sources external to the person can be used to drive change. A coherent treatment approach that aims to foster fitness for purpose can be organized according to 4 therapeutic targets that seek to help the person understand it is safe and helpful to move, refine neural representations so the back feels safe to move, load the back to promote positive tissue adaptations, and allow the person the experience safety with movement and consolidate safety under load through targeted self-management strategies.

Understand It Is Safe and Helpful to Move

To foster fitness for purpose, we contend that it is important that people with CNSLBP are provided with a less threatening and more hopeful understanding of their problem and a conceptualization of the drivers to the condition that make explicit both the safety and value of movement and loading. This may encompass the benefits that movement and activity impart on the health and load tolerance of the musculoskeletal system and the dynamic sensitivity of the body's protective systems as well as the opportunity that movement and loading provide for learning about the back, exploring its capabilities, and influencing how it is represented cortically. Pain science education, which accommodates multiple "ways of knowing" and multiple types of knowledge,[105] is a key intervention in promoting this understanding. This type of education emerged 2 decades ago in response to the failure of conventional back pain education and cognitive therapies to impact problematic and change-resistant conceptualizations of a damaged, fragile, and unhealthy self.[22,106,107] Modern pain science education is based primarily on a constructivist model[108] and encompasses a wide range of educational interventions aimed to improve an individual's understanding of "how pain works," including the distinction between nociception and pain and the clinical implications of this distinction; the dynamic sensitivity of nociceptors and other components of a wider "pain system"; the multifactorial processes that underpin the experience of pain and its persistence; and the ever-present tendency of biological systems to adapt to the demands placed on them, a concept recently coined as "bioplasticity."[109,110]

A central objective of pain science education is to shift an individual's conceptualization of pain from being simply a marker of tissue damage or disease to being a dynamic protective feeling that is influenced by multiple, largely reversible factors from across the biopsychosocial spectrum that vary across temporal scales ranging from moments to years.[22] Pain science education has both generic learning objectives (eg, Moseley and Butler[105]) and learning objectives tailored to the individual. Individual learning objectives reflect specific misconceptions about the condition and individual factors that might shape the view that the back is not fit for purpose. Generic learning objectives, or "target concepts,"[105] are based on contemporary pain science and consumer perspectives on recovery (eg, Leake et al[111]), so they change over time as new discoveries are made.[112] Pain science education is foundational to our intervention because of the imperative for the individual to have the required procedural and conditional knowledge[113] to make optimal collaborative decisions throughout the program and beyond. Inherent in this procedural and conditional knowledge is the above-mentioned concept of bioplasticity as it applies to both positive adaptation of body tissue and the development and reversibility of "pain system hypersensitivity." Therefore, individually specific evidence of positive adaptation of tissue—for example via available radiological reports[114] or experiences of soft tissue healing—helps to promote deep understanding of why movement and loading are critical for full recovery. The FFPM considers scientifically based beliefs about pain, loading, movement, and functional reversibility of the biological systems that underpin pain are potentially critical if one is to engage in subsequent aspects of the treatment package.

Refine Neural Representations of the Body So the Back Feels Safe to Move

There is a growing body of evidence to show that chronic pain is associated with progressive disruptions in bodily awareness and processing of bodily information.[104,115–117] Disruptions of bodily awareness can be distressing and provide clear evidence to the individual experiencing them that the body is not fit for purpose and contribute to degraded motor control of the spine. Clinical investigation of these disruptions relies on measures such as tactile acuity[118–120] and motor imagery performance,[121] and laboratory investigation relies on brain imaging[90,122] and electromyography.[123] Performance in both clinical and laboratory tests seems to relate to perceptual and motor disruptions,[100] and neural representation training normalizes performance (eg, Moseley and Flor[120]). The impact of these interventions when applied in isolation on outcomes such as pain and disability is small. However, normalizing these disruptions through neural representation training,[124] such that the body begins to feel safe to move, may serve to reinforce the central educational message that the back is safe to move and help facilitate engagement in subsequent functional activities that move and load the back. Our inclusion of neural representation training to normalize the processes that subserve "how our body feels to us" is grounded in an interpretation of embodied cognition that emphasizes that cognitive processes are influenced by the body,[125] particularly that information from the body shapes our views about the capacity of the body (eg, Zadra et al[126]). With respect to training, we propose that having a back that feels safe to move encompasses both how the back feels before and during movement. The aims, then, of neural representation training are to influence bodily-related neural representations that mold the internal models of health and load tolerance of the back—factors that shape the prediction of pain with movement and loading—and to utilize strategies that seek to increase the availability and precision of ongoing non-nociceptive somatosensory information from the back while the back is moved and loaded.

To meet these aims, neural representation training should involve both precision focused sensory and motor strategies. Sensory precision training requires a combination of repeated stimuli delivered to the back along with goal-orientated attention to each stimulus using techniques such as asking the patient to describe the location of stimulation or discern different types of stimulation.[120] There is some evidence of an analgesic effect of sensory precision training in phantom limb pain[127] and in complex regional pain syndrome (CRPS).[128] In CRPS, that sensory stimulation alone has no effect[128] and that performance and analgesia are augmented by visual enhancement of touch and spatial attention[129] point to cortical adaptations underpinning the effect. Preliminary data from participants with CNSLBP suggest sensory acuity improves with sensory discrimination training,[130,131] and these improvements are associated with small reductions in pain intensity and disability.[132] Moreover, data from other persistent pain conditions suggest that sensory precision training not only improves pain and sensory acuity but is also associated with normalization of cortical stimulus response profiles assessed using brain imaging approaches.[127,133]

A possible starting point for motor precision training is motor imagery training, which typically progresses from implicit to explicit motor imagery and involves a graded engagement of neural pathways that subserve movement planning and execution without moving the physical body.[134,135] Implicit motor imagery commences with left/right judgement training in which a decision is made on whether a model viewed in an image has their back turned to the left or the right.[136] Performance on this type of task is thought to offer insight into the integrity of proprioceptive representations of the pictured body part[137] (but see Alazmi et al[138] for 1 contrasting result), so it is plausible that training targeted at improving speed and accuracy in left/right judgements might update and improve cortical representations of the back.[139] Preliminary data from people with CRPS offer some support for this idea,[140–143] and there is a strong theoretical basis for proposing that implicit motor imagery induces rapid changes in cortical processing through subthreshold inhibitory pathways between pre-motor and primary motor areas (see Moseley et al[135] for an expanded discussion). Movement observation, closely watching others move, is another approach to implicit motor imagery that has both theoretical and empirical support[144] and has the advantage of being able to integrate contextual and functional characteristics that are individually tailored to a patient's fears and goals.

To further drive activation of cortical pathways involved in movement, implicit motor imagery can be progressed to explicit motor imagery, where the individual imagines performing a movement or task in the first person. This can be augmented by having the individual simultaneously observe and mentally embody videos of people performing tasks of increasing complexity and personal relevance. Clinical trials in CRPS and phantom limb pain patients demonstrate the importance of the order of components (implicit followed by explicit) in progressive motor imagery training[145] as well as possible clinical benefits.[134]

Progression from pre-movement strategies to movement of the physical body aims to further impact on cortical representations of the back and facilitate the availability and trustworthiness of non-noxious somatosensory information associated with back movement. Exercises that focus on mindful body mastery, such as learning how to perform precise and localized movements of the lumbar spine and hips, present an opportunity to achieve this aim. For example, learning to delineate the lumbar spine from the thorax and hips and precisely demarcate movement between these adjacent areas[146,147] or learning to independently activate anatomically adjacent trunk muscles[148] satisfies the requirements of a task that, theoretically, should improve precision of non-nociceptive information and sharpen cortical representations. Empirical support is sparse, but preliminary data suggest that specific trunk muscle training may shift motor cortex processing in people with back pain.[149] Making goal attainment contingent on attention to specific movement characteristics, which will increase the weighting given to non-nociceptive data and improve the precision of cortical processing of that data, may be an important element of such approaches. Relevant here are the many common movement strategies employed by clinicians in the treatment of LBP. For example, performing a "hip stretch" could be reframed as a useful way of learning to delineate movement in the hip from movement in the back. Movement in this context, both active and passive, is seen not as a way of modifying tissue mechanics but as a way of sampling information from the body, learning about the body, updating internal models of the body, and manipulating the weighting given to sensory inputs within the context of potentially powerful cues of safety and health.

Load the Back to Promote Positive Tissue Adaptation and Experience Safety With Movement

As stated earlier, the experience of pain with movement is a very valid reason for concluding that the back is damaged, fragile, and unhealthy, and it is important to provide information to counter this view. Once the person understands that it is safe to move, observes both the disruption in somatosensory and motor processing and the resolution of these disruptions with training, and potentially starts to feel safe to move, then the treatment program can progress to functional movement training and loading. Here, the objectives are to (1) load the body and thus trigger positive adaptation towards greater load-bearing capacity of the tissues; and (2) through skilled movement coaching, provide movement and loading experiences that involve less pain than predicted and thus update internal models associated with how fit for purpose the back is. Experiencing that the back is safe to move and load builds on exposure based approaches to managing CNSLBP informed by the fear-avoidance model[150–154] such that task selection is a shared decision-making process based on the person's goals and employs behavioral experiments designed to violate prior expectations of the effect of the task on the back. In addition to these concepts, we would advocate skillful movement coaching and task modification in which the therapist and patient work together to identify strategies that minimize symptom provocation with action and thereby provide clear evidence of a healthy, load-tolerant self and safety with function, as well as demonstrate the potential for reversibility of movement sensitivity.

The epistemic nature of movement is at the forefront of this type of exercise prescription with a focus on the individual learning about their body, how to move and complete functional tasks with decreased provocation, the impact of context on sensitivity, and the reversible nature of their sensitivity state–their so-called "protect by pain zone."[109] We think this is best facilitated by a precision-focused and feedback-enriched approach to movement training and coaching in which participants progress from part practice to whole task practice of functionally relevant tasks while attention is directed towards non-nociceptive aspects of task performance through visual,[155] tactile, proprioceptive, and even auditory sources of information. Several treatment models have been proposed that seek to decrease movement sensitivity through reducing unhelpful protective behaviors such as breath holding and muscle co-activation[156] or provocative lumbar movement patterns such as disrupted load sharing.[157–159] These approaches provide valuable skills to help the clinician enact the ideas presented here. Strategies to consider could also extend beyond movement quality or loading patterns and seek to utilize cues from across psychological and social domains that infer safety (see, eg, "the Protectometer" tool[109]). Emerging methods such as virtual reality-based training may also facilitate this step.[160,161]

Consolidate Safety Under Load

The long-term goal of treating CNSLBP according to the FFPM is resilience under the movement and loading conditions to which the individual is likely to be exposed. Thus, progression may continue to work-hardening contexts, traditional aerobic-based exercises, or general strength training according to the individual patient's personal goals and values. That movement and loading of tissues triggers adaptations in those tissues and their functional engagement is well established,[162] but there seems to be only weak relationships between peripheral musculoskeletal changes and clinical status.[163] What little evidence is available suggests that traditional physical training may benefit people with back pain by decreasing fear and increasing confidence.[164] Our approach would support a synergistic relationship between changes in

RESOLVE: A progressive complex care package based on the Fit-for-Purpose model		
UNDERSTAND that it is safe and helpful to move	Tailored contemporary pain science education	
REFINE neural representations of the body so the back FEELS safe to move	Sensory Precision Training	Graded sensory localization training
		Graded sensory localization and discrimination training
		Graded graphaesthesia training
	Motor Imagery Training	Graded left/right judgement training
		Graded motor empathy training
		Graded imagined movement training
	Motor Precision Training	Low load, precision focused and feedback enriched independent spinal movements
		Low load, precision focused and feedback enriched independent hip movements
LOAD the back to promote positive tissue adaptation and experience safety with movement	Precision focused and feedback enriched graded exercises Part practice of functional tasks relevant to the individuals goals	
	Precision focused and feedback enriched graded exercises Whole practice of functional tasks relevant to the individuals goals	
CONSOLIDATE safety under load	Ongoing general exercise prescription of increasing intensity that integrates skills learned above and is orientated towards patient-derived goals.	

Figure 2. RESOLVE: a progressive complex care package based on the Fit-For-Purpose Model (FFPM).

the body and changes in beliefs. Positive adaptations within the tissues of the back may well contribute information that updates internal models toward a strong, healthy, and load-tolerant back. Simultaneously, the experience of undertaking intense physical training without significant symptom exacerbation provides strong evidence in support of this view and that the back is fit for purpose, even under large loads. That is, strength and fitness training provide countless potential opportunities to update meta-cognitive, cognitive, and proprioceptive internal models about the structural integrity and resilience of the back under given demands. This calls for expertly prescribed and appropriately dosed exercise in which priority is given to optimizing movement confidence rather than dosing regimens designed to simply optimize peripheral tissue adaptations.[165] In this way, the FFPM represents a clear shift from previous models that recognize only peripheral adaptations in response to training[166] or only meta-cognitive adaptations.[154] A final therapeutic target of the FFPM is empowering the individual with the understanding and skills to self-manage and maintain optimal physical and psychological health under future challenges.

Treatment Summary

Figure 2 outlines a complex care package based on the FFPM currently being tested in a large sham-controlled explanatory clinical trial ("RESOLVE"[20]). The key aim of care is to reinforce fitness for purpose of the back by helping the individual: UNDERSTAND that it is safe and helpful to move; REFINE the neural representations of the body so the back FEELS safe to move; LOAD the back to promote positive tissue adaptation and experience safety with movement; and CONSOLIDATE the concept of safety under load through general exercise prescription and work-hardening programs orientated toward patient-derived goals. Each stage reinforces previous stages. Our, as yet untested, prediction is that optimal outcomes will be realized by treatment approaches that integrate these features in a synergistic manner. The FFPM has been the key theoretical guide to develop this complex care package, and we hope the model facilitates other health care professionals to make sense of those treatments they perceive as helpful and to implement them in synergistic ways that align with contemporary understanding of how pain and recovery work. We do not see this complex care package as a one-size-fits-all approach but an overarching blueprint for care in which there is significant scope for individualization in terms of the weighting of attention devoted to each of the steps, what is emphasized in each step, and how the aims of each step are best achieved for the individual. Note that all phases are individualized toward patient-derived fears, misconceptions, behaviors, and goals. Finally, tailoring of care should be optimized by assessing knowledge about "how pain works,"[167] beliefs about the fitness for purpose of the back,[168] back-specific body representations and awareness,[104] motor imagery performance,[121] sensory precision,[169] and movement precision.[146] These assessments can help shape and refine care as well as be used to evaluate the process of care.[170]

CNSLBP is a complex problem that is best understood through a biopsychosocial lens, particularly one that incorporates a contemporary understanding of the neurobiology of the pain experience. The FFPM regards CNSLBP as a problem of internal meta-cognitive, cognitive, and proprioceptive models that hold that the back is not fit for purpose. Based on this premise, we propose a theoretically justified, coherent, progressive complex care approach that targets modifiable factors to help shift internal models of a fragile, damaged, unhealthy, and unchangeable self towards the formulation of the back as healthy, strong, adaptable, and fit for purpose.

Key Points

- CNSLBP represents a dynamic interplay between beliefs about the body in pain, neural representations that impact on how the painful area feels and moves, the information available as the back is moved and loaded, and the capacity of the musculoskeletal system to tolerate load.
- Multimodal care for the person with back pain should seek to foster fitness for purpose by helping the person reconceptualize pain so they understand it is safe and helpful to move, targeting neural representations so the back feels safe to move, employing graded movement

coaching to promote positive tissue adaptations and the experience of safety with movement and consolidate these through targeted self-management.

Author Contributions

Concept/idea/research design: B.M. Wand, A.G. Cashin, J.H. McAuley, M.K. Bagg, G.L. Moseley
Writing: B.M. Wand, A.G. Cashin, J.H. McAuley, M.K. Bagg, G.L. Moseley
Project management: B.M. Wand, A.G. Cashin
Consultation (including review of manuscript before submitting): B.M. Wand, M.K. Bagg, G.M. Orange, G.L. Moseley

Acknowledgments

The authors thank Dr Neil O'Connell from Brunel University for his comments on the manuscript and Associate Professor William Gibson and Dr Mervyn Travers from the University of Notre Dame Australia for their contribution and insightful observations on predictive processing and pain.

Funding

No specific grant was received for this study from any funding agency in the public, commercial, or not-for-profit sectors.

Disclosures

The authors completed the ICMJE Form for Disclosure of Potential Conflicts of Interest and reported no conflicts of interest.

B.M. Wand has received fees to speak about pain neuroscience and low back pain rehabilitation and is an investigator on the RESOLVE trial, which was funded by the National Health and Medical Research Council (NHMRC) of Australia, ID1087045. A.G. Cashin was supported by the University of New South Wales Prince of Wales Clinical School Postgraduate Research Scholarship and a NeuRA PhD Candidature Supplementary Scholarship. M.K. Bagg was supported by a NeuRA PhD Candidature Scholarship and was supported during this work by an Australian Research Training Program Scholarship and a UNSW Research Excellence Award. M.K. Bagg received conference travel support from the Chiropractor's Association of Australia to speak about pain neuroscience and rehabilitation and Memorial University of Newfoundland to speak about engagement with research evidence. J.H. McAuley is supported by a Leadership Investigator Grant from the NHMRC of Australia; has received other research funding from the Australian Commonwealth National Health and Medical Research Council (NHMRC) and the Australian Medical Research Future Fund (MRFF); and is an investigator on the RESOLVE trial. G.L. Moseley is supported by a Leadership Investigator grant from the NHMRC of Australia (ID 1178444), has received other research funding from NHMRC, the Australian Medical Research Future Fund (MRFF), and is an investigator on the RESOLVE trial (NHMRC ID1087045), other Australian and overseas governments, and not for profit agencies. G.L. Moseley also has received support from Reality Health; ConnectHealth UK; Seqirus; Kaiser Permanente; Workers' Compensation Boards in Australia, Europe, and North America; AIA Australia; the International Olympic Committee; Western Australia Cricket Association; Arsenal Football Club; Brighton and Hove Albion Football Club; and Melbourne Football Club. Professional and scientific bodies have reimbursed G.L. Moseley for travel costs related to presentation of research on pain at scientific conferences/symposia. G.L. Moseley has received speaker fees for lectures on pain and rehabilitation and receives book royalties from NOIgroup Publications and Dancing Giraffe Press, including key texts involved in the education component of the FFPM.

References

1. Cholewicki J, Popovich JM, Aminpour P, Gray SA, Lee AS, Hodges PW. Development of a collaborative model of low back pain: report from the 2017 NASS consensus meeting. *Spine J*. 2019;19:1029–1040. https://doi.org/10.1016/j.spinee.2018.11.014.
2. Hartvigsen J, Hancock MJ, Kongsted A et al. What low back pain is and why we need to pay attention. *Lancet*. 2018;391: 2356–2367. https://doi.org/10.1016/S0140-6736(18)30480-X.
3. Maher C, Underwood M, Buchbinder R. Non-specific low back pain. *Lancet*. 2017;389:736–747. https://doi.org/10.1016/S0140-6736(16)30970-9.
4. Menezes Costa LDC, Maher CG, Hancock MJ, McAuley JH, Herbert RD, Costa LOP. The prognosis of acute and persistent low-back pain: a meta-analysis. *Can Med Assoc J*. 2012;184:E613–E624. https://doi.org/10.1503/cmaj.111271.
5. Haldeman S, Dagenais S. A supermarket approach to the evidence-informed management of chronic low back pain. *Spine J*. 2008;8:1–7. https://doi.org/10.1016/j.spinee.2007.10.009.
6. Bagg MK, McAuley JH, Moseley GL, Wand BM. Recent data from radiofrequency denervation trials further emphasise that treating nociception is not the same as treating pain. *Br J Sports Med*. 2018;53:841–842. https://doi.org/10.1136/bjsports-2017-098510.
7. Kamper SJ, Apeldoorn AT, Chiarotto A et al. Multidisciplinary biopsychosocial rehabilitation for chronic low back pain. *Cochrane Database Syst Rev*. 2014;9:CD000963. https://doi.org/10.1002/14651858.CD000963.pub3.
8. Bernstein IA, Malik Q, Carville S, Ward S. Low back pain and sciatica: summary of NICE guidance. *BMJ*. 2017;356:i6748. https://doi.org/10.1136/bmj.i6748.
9. Wand, B.M. Chronic lower back pain: a maladaptive perceptions model. In: *NOI Neurodynamics and the Neuromatrix Conference*. Adelaide, Australia: April, 2012.
10. Leeuw M, Goossens MEJB, Linton SJ, Crombez G, Boersma K, Vlaeyen JWS. The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *J Behav Med*. 2007;30:77–94. https://doi.org/10.1007/s10865-006-9085-0.
11. Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain*. 2000;85: 317–332. https://doi.org/10.1016/S0304-3959(99)00242-0.
12. Jones CL, Jensen JD, Scherr CL, Brown NR, Christy K, Weaver J. The health belief model as an explanatory framework in communication research: exploring parallel, serial, and moderated mediation. *Health Commun*. 2015;30:566–576. https://doi.org/10.1080/10410236.2013.873363.
13. Leventhal H, Phillips LA, Burns E. The common-sense model of self-regulation (CSM): a dynamic framework for understanding illness self-management. *J Behav Med*. 2016;39:935–946. https://doi.org/10.1007/s10865-016-9782-2.
14. Goossens N, Rummens S, Janssens L, Caeyenberghs K, Brumagne S. Association between sensorimotor impairments and functional brain changes in patients with low back pain. *Am J Phys Med Rehabil*. 2018;97:200–211. https://doi.org/10.1097/PHM.0000000000000859.
15. Malfliet A, Coppieters I, Van Wilgen P et al. Brain changes associated with cognitive and emotional factors in chronic pain: a systematic review. *Eur J Pain (United Kingdom)*. 2017;21: 769–786. https://doi.org/10.1002/ejp.1003.
16. Ng SK, Urquhart DM, Fitzgerald PB, Cicuttini FM, Hussain SM, Fitzgibbon BM. The relationship between structural and functional brain changes and altered emotion and cognition in chronic low back pain. *Clin J Pain*. 2017;34:237–261. https://doi.org/10.1097/AJP.0000000000000534.
17. Edwards MJ, Adams RA, Brown H, Pareés I, Friston KJ. A Bayesian account of "hysteria." *Brain*. 2012;135:3495–3512. https://doi.org/10.1093/brain/aws129.

18. Ongaro G, Kaptchuk TJ. Symptom perception, placebo effects, and the Bayesian brain. *Pain*. 2019;160:1–4. https://doi.org/10.1097/j.pain.0000000000001367.
19. Tabor A, Thacker MA, Moseley GL, Körding KP. Pain: a statistical account. *PLoS Comput Biol*. 2017;13:e1005142. https://doi.org/10.1371/journal.pcbi.1005142.
20. Bagg MK, Hübscher M, Rabey M et al. The RESOLVE Trial for people with chronic low back pain: protocol for a randomised clinical trial. *J Physiother*. 2017;63:47–48. https://doi.org/10.1016/j.jphys.2016.11.001.
21. Wall PD, McMahon SB. The relationship of perceived pain to afferent nerve impulses. *Trends Neurosci*. 1986;9:254–255. https://doi.org/10.1016/0166-2236(86)90070-6.
22. Moseley GL, Butler DS. Fifteen years of explaining pain: the past, present, and future. *J Pain*. 2015a;16:807–813. https://doi.org/10.1016/j.jpain.2015.05.005.
23. Picard F, Friston K. Predictions, perception, and a sense of self. *Neurology*. 2014;83:1112–1118. https://doi.org/10.1212/WNL.0000000000000798.
24. Darlow B. Beliefs about back pain: the confluence of client, clinician and community. *Int J Osteopath Med*. 2016;20:53–61. https://doi.org/10.1016/j.ijosm.2016.01.005.
25. Darlow B, Dean S, Perry M, Mathieson F, Baxter GD, Dowell A. Easy to harm, hard to heal. *Spine (Phila Pa 1976)*. 2015;40: 842–850. https://doi.org/10.1097/BRS.0000000000000901.
26. Setchell J, Costa N, Ferreira M, Makovey J, Nielsen M, Hodges PW. Individuals' explanations for their persistent or recurrent low back pain: a cross-sectional survey. *BMC Musculoskelet Disord*. 2017;18:466. https://doi.org/10.1186/s12891-017-1831-7.
27. Bunzli S, Watkins R, Smith A, Schütze R, O'Sullivan P. Lives on hold: a qualitative synthesis exploring the experience of chronic low-back pain. *Clin J Pain*. 2013;29:907–916. https://doi.org/10.1097/AJP.0b013e31827a6dd8.
28. Ford JJ, Hahne AJ, Surkitt LD et al. Individualised physiotherapy as an adjunct to guideline-based advice for low back disorders in primary care: a randomised controlled trial. *Br J Sports Med*. 2016;50:237–245. https://doi.org/10.1136/bjsports-2015-095058.
29. Petersen T, Olsen S, Laslett M et al. Inter-tester reliability of a new diagnostic classification system for patients with non-specific low back pain. *Aust J Physiother*. 2004;50:85–94. https://doi.org/10.1016/s0004-9514(14)60100-8.
30. May S, Donelson R. Evidence-informed management of chronic low back pain with the McKenzie method. *Spine J*. 2008;8: 134–141. https://doi.org/10.1016/j.spinee.2007.10.017.
31. Parreira P, Heymans MW, van Tulder MW et al. Back schools for chronic non-specific low back pain. *Cochrane Database Syst Rev*. 2017;8:CD011674. https://doi.org/10.1002/14651858.CD011674.pub2.
32. Tariq RA, George JS, Ampat G, Toney-Butler TJ. *Back Safety*. Tampa, FL: StatPearls Publishing; 2021.
33. Verbeek JH, Martimo KP, Kuijer PPFM, Karppinen J, Viikari-Juntura E, Takala EP. Proper manual handling techniques to prevent low back pain, a Cochrane systematic review. *Work*. 2012;41:2299–2301. https://doi.org/10.3233/WOR-2012-0455-2299.
34. Hall A, Aubrey-Bassler K, Thorne B, Maher CG. Do not routinely offer imaging for uncomplicated low back pain. *BMJ*. 2021;372:n291. https://doi.org/10.1136/bmj.n291.
35. Oliveira CB, Maher CG, Pinto RZ et al. Clinical practice guidelines for the management of non-specific low back pain in primary care: an updated overview. *Eur Spine J*. 2018;27:2791–2803. https://doi.org/10.1007/s00586-018-5673-2.
36. Kamper SJ, Logan G, Copsey B et al. What is usual care for low back pain? A systematic review of health care provided to patients with low back pain in family practice and emergency departments. *Pain*. 2020;161:694–702. https://doi.org/10.1097/j.pain.0000000000001751.
37. Brinjikji W, Luetmer PH, Comstock B et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. *Am J Neuroradiol*. 2015;36:811–816. https://doi.org/10.3174/ajnr.A4173.
38. Karran EL, Medalian Y, Hillier SL, Moseley GL. The impact of choosing words carefully: an online investigation into imaging reporting strategies and best practice care for low back pain. *PeerJ*. 2017;5:e4151. https://doi.org/10.7717/peerj.4151.
39. Chou R, Shekelle P. Will this patient develop persistent disabling low back pain? *JAMA-J Am Med Assoc*. 2010;303:1295–1302. https://doi.org/10.1001/jama.2010.344.
40. Kent PM, Keating JL. Can we predict poor recovery from recent-onset nonspecific low back pain? A systematic review. *Man Ther*. 2008;13:12–28. https://doi.org/10.1016/j.math.2007.05.009.
41. Pincus T, Burton AK, Vogel S, Field AP. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine (Phila Pa 1976)*. 2002;27:E109. https://doi.org/10.1097/00007632-200203010-00017.
42. Steenstra IA, Verbeek JH, Heymans MW, Bongers PM. Prognostic factors for duration of sick leave in patients sick listed with acute low back pain: a systematic review of the literature. *Occup Environ Med*. 2005;62:851–860. https://doi.org/10.1136/oem.2004.015842.
43. Campbell P, Foster NE, Thomas E, Dunn KM. Prognostic indicators of low back pain in primary care: five-year prospective study. *J Pain*. 2013;14:873–883. https://doi.org/10.1016/j.jpain.2013.03.013.
44. Green DJ, Lewis M, Mansell G et al. Clinical course and prognostic factors across different musculoskeletal pain sites: a secondary analysis of individual patient data from randomised clinical trials. *Eur J Pain*. 2018;22:1057–1070. https://doi.org/10.1002/ejp.1190.
45. Grotle M, Foster NE, Dunn KM, Croft P. Are prognostic indicators for poor outcome different for acute and chronic low back pain consulters in primary care? *Pain*. 2010;151:790–797. https://doi.org/10.1016/j.pain.2010.09.014.
46. da Silva T, Mills K, Brown BT et al. Recurrence of low back pain is common: a prospective inception cohort study. *J Physiother*. 2019;65:159–165. https://doi.org/10.1016/j.jphys.2019.04.010.
47. da Silva T, Mills K, Brown BT, Herbert RD, Maher CG, Hancock MJ. Risk of recurrence of low back pain: a systematic review. *J Orthop Sport Phys Ther*. 2017;47:305–313. https://doi.org/10.2519/jospt.2017.7415.
48. Gore M, Sadosky A, Stacey BR, Tai KS, Leslie D. The burden of chronic low back pain: clinical comorbidities, treatment patterns, and health care costs in usual care settings. *Spine (Phila Pa 1976)*. 2012;37:E668–E677. https://doi.org/10.1097/BRS.0b013e318241e5de.
49. Ramanathan S, Hibbert P, Wiles L, Maher CG, Runciman W. What is the association between the presence of comorbidities and the appropriateness of care for low back pain? A population-based medical record review study 11 Medical and Health Sciences 1117 Public Health and Health Services. *BMC Musculoskelet Disord*. 2018;19:391. https://doi.org/10.1186/s12891-018-2316-z.
50. van der Zee-Neuen A, Putrik P, Ramiro S et al. Impact of chronic diseases and multimorbidity on health and health care costs: the additional role of musculoskeletal disorders. *Arthritis Care Res*. 2016;68:1823–1831. https://doi.org/10.1002/acr.22913.
51. Linton SJ. A review of psychological risk factors in back and neck pain. *Spine (Phila Pa 1976)*. 2000;25:1148–1156. https://doi.org/10.1097/00007632-200005010-00017.
52. Nicholas MK, Linton SJ, Watson PJ, Main CJ. Early identification and management of psychological risk factors ("yellow flags") in patients with low back pain: a reappraisal. *Phys Ther.* 2011;91: 737–753. doi: https://doi.org/10.2522/ptj.20100224.

53. Pinheiro MB, Ferreira ML, Refshauge K et al. Symptoms of depression as a prognostic factor for low back pain: a systematic review. *Spine J*. 2016;16:105–116. https://doi.org/10.1016/j.spinee.2015.10.037.
54. Myers SS, Phillips RS, Davis RB et al. Patient expectations as predictors of outcome in patients with acute low back pain. *J Gen Intern Med*. 2007;23:148–153. https://doi.org/10.1007/s11606-007-0460-5.
55. Vowles KE, Zvolensky MJ, Gross RT, Sperry JA. Pain-related anxiety in the prediction of chronic low-back pain distress. *J Behav Med*. 2004;27:77–89. https://doi.org/10.1023/B:JOBM.0000013645.40613.42.
56. Baliki MN, Apkarian AV. Nociception, pain, negative moods, and behavior selection. *Neuron*. 2015;87:474–491. https://doi.org/10.1016/j.neuron.2015.06.005.
57. Friston K. The free-energy principle: a unified brain theory? *Nat Rev Neurosci*. 2010;11:127–138. https://doi.org/10.1038/nrn2787.
58. Den Hollander M, Goossens M, De Jong J et al. Expose or protect? A randomized controlled trial of exposure in vivo vs pain-contingent treatment as usual in patients with complex regional pain syndrome type 1. *Pain*. 2016;157:2318–2329. https://doi.org/10.1097/j.pain.0000000000000651.
59. Geisser ME, Haig AJ, Wallbom AS, Wiggert EA. Pain-related fear, lumbar flexion, and dynamic emg among persons with chronic musculoskeletal low back pain. *Clin J Pain*. 2004;20:61–69. https://doi.org/10.1097/00002508-200403000-00001.
60. Karayannis NV, Smeets RJEM, van den Hoorn W, Hodges PW. Fear of movement is related to trunk stiffness in low back pain. *PLoS One*. 2013;8:e67779. https://doi.org/10.1371/journal.pone.0067779.
61. Lamoth CJC, Meijer OG, Daffertshofer A, Wuisman PIJM, 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. https://doi.org/10.1007/s00586-004-0825-y.
62. Lamoth CJC, Meijer OG, Wuisman PIJM, 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. https://doi.org/10.1097/00007632-200202150-00016.
63. Abboud J, Nougarou F, Pagé I, Cantin V, Massicotte D, Descarreaux M. Trunk motor variability in patients with non-specific chronic low back pain. *Eur J Appl Physiol*. 2014;114:2645–2654. https://doi.org/10.1007/s00421-014-2985-8.
64. Ebrahimi S, Kamali F, Razeghi M, Haghpanah SA. Comparison of the trunk-pelvis and lower extremities sagittal plane inter-segmental coordination and variability during walking in persons with and without chronic low back pain. *Hum Mov Sci*. 2017;52:55–66. https://doi.org/10.1016/j.humov.2017.01.004.
65. Falla D, Gizzi L, Tschapek M, Erlenwein J, Petzke F. Reduced task-induced variations in the distribution of activity across back muscle regions in individuals with low back pain. *Pain*. 2014;155:944–953. https://doi.org/10.1016/j.pain.2014.01.027.
66. Gizzi L, Röhrle O, Petzke F, Falla D. People with low back pain show reduced movement complexity during their most active daily tasks. *Eur J Pain (United Kingdom)*. 2019;23:410–418. https://doi.org/10.1002/ejp.1318.
67. 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. https://doi.org/10.1097/BRS.0b013e31821c4267.
68. Kehler DS, Theou O, Rockwood K. Bed rest and accelerated aging in relation to the musculoskeletal and cardiovascular systems and frailty biomarkers: a review. *Exp Gerontol*. 2019;124:110643. https://doi.org/10.1016/j.exger.2019.110643.
69. Ramsey KA, Rojer AGM, D'Andrea L et al. The association of objectively measured physical activity and sedentary behavior with skeletal muscle strength and muscle power in older adults: a systematic review and meta-analysis. *Ageing Res Rev*. 2021;67:101266. https://doi.org/10.1016/j.arr.2021.101266.
70. Zhao X, Xu M, Jorgenson K, Kong J. Neurochemical changes in patients with chronic low back pain detected by proton magnetic resonance spectroscopy: a systematic review. *Neuroimage Clin*. 2017;13:33–38. https://doi.org/10.1016/j.nicl.2016.11.006.
71. Kregel J, Meeus M, Malfliet A et al. Structural and functional brain abnormalities in chronic low back pain: a systematic review. *Semin Arthritis Rheum*. 2015;45:29–237. https://doi.org/10.1016/j.semarthrit.2015.05.002.
72. Schouppe S, Van Oosterwijck S, Danneels L, Van Damme S, Van Oosterwijck J. Are functional brain alterations present in low back pain? A systematic review of EEG studies. *J Pain*. 2020;21:25–43. https://doi.org/10.1016/j.jpain.2019.06.010.
73. 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. https://doi.org/10.1016/j.math.2010.06.008.
74. Briggs AM, Straker LM, Burnett AF, Wark JD. Chronic low back pain is associated with reduced vertebral bone mineral measures in community-dwelling adults. *BMC Musculoskelet Disord*. 2012;13:49. https://doi.org/10.1186/1471-2474-13-49.
75. Noonan AM, Brown SHM. Paraspinal muscle pathophysiology associated with low back pain and spine degenerative disorders. *JOR Spine*. 2021;4:e1171. https://doi.org/10.1002/jsp2.1171.
76. Steele J, Bruce-Low S, Smith D. A reappraisal of the deconditioning hypothesis in low back pain: review of evidence from a triumvirate of research methods on specific lumbar extensor deconditioning. *Curr Med Res Opin*. 2014;30:865–911. https://doi.org/10.1185/03007995.2013.875465.
77. Den Bandt HL, Paulis WD, Beckweé D, Ickmans K, Nijs J, Voogt L. Pain mechanisms in low back pain: a systematic review with meta-analysis of mechanical quantitative sensory testing outcomes in people with nonspecific low back pain. *J Orthop Sports Phys Ther*. 2019;49:698–715. https://doi.org/10.2519/jospt.2019.8876.
78. McPhee ME, Vaegter HB, Graven-Nielsen T. Alterations in pronociceptive and antinociceptive mechanisms in patients with low back pain: a systematic review with meta-analysis. *Pain*. 2020;161:464–475. https://doi.org/10.1097/j.pain.0000000000001737.
79. Adamczyk W, Luedtke K, Saulicz E. Lumbar tactile acuity in patients with low back pain and healthy controls. *Clin J Pain*. 2017;34:1. https://doi.org/10.1097/AJP.0000000000000499.
80. Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. *Neurosci Lett*. 2004;366:63–66. https://doi.org/10.1016/j.neulet.2004.05.013.
81. Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. *Eur J Appl Physiol*. 2011;111:115–123. https://doi.org/10.1007/s00421-010-1637-x.
82. Moseley GL, Gallagher L, Gallace A. Neglect-like tactile dysfunction in chronic back pain. *Neurology*. 2012c;79:327–332. https://doi.org/10.1212/WNL.0b013e318260cba2.
83. Puta C, Schulz B, Schoeler S et al. Somatosensory abnormalities for painful and innocuous stimuli at the back and at a site distinct from the region of pain in chronic back pain patients. *PLoS One*. 2013;8:e58885. https://doi.org/10.1371/journal.pone.0058885.
84. Rausch Osthoff AK, Ernst MJ, Rast FM et al. Measuring lumbar reposition accuracy in patients with unspecific low back pain: systematic review and meta-analysis. *Spine (Phila Pa 1976)*. 2015;40:E97–E111. https://doi.org/10.1097/BRS.0000000000000677.
85. 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. doi:https://doi.org/10.1016/j.apmr.2016.05.016.
86. Wand BM, Keeves J, Bourgoin C et al. Mislocalization of sensory information in people with chronic low back pain: a preliminary investigation. *Clin J Pain*. 2013;29:737–743. https://doi.org/10.1097/AJP.0b013e318274b320.
87. Wang H, Fischer C, Chen G, Weinsheimer N, Gantz S, Schiltenwolf M. Does long-term opioid therapy reduce pain sensitivity of patients with chronic low back pain? Evidence from quantitative sensory testing. *Pain Physician*. 2012;15:ES135–ES143. https://doi.org/10.1111/j.1526-4637.2011.01276.x.
88. Willigenburg NW, Kingma I, Hoozemans MJM, van Dieën JH. Precision control of trunk movement in low back pain patients. *Hum Mov Sci*. 2013;32:228–239. https://doi.org/10.1016/j.humov.2012.12.007.
89. Elgueta-Cancino E, Schabrun S, Hodges P. Is the organization of the primary motor cortex in low back pain related to pain, movement, and/or sensation? *Clin J Pain*. 2018;34:207–216. https://doi.org/10.1097/AJP.0000000000000535.
90. 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. https://doi.org/10.1016/S0304-3940(97)13441-3.
91. Goossens N, Janssens L, Brumagne S. Changes in the organization of the secondary somatosensory cortex while processing lumbar proprioception and the relationship with sensorimotor control in low back pain. *Clin J Pain*. 2019;35:394–406. https://doi.org/10.1097/AJP.0000000000000692.
92. Hotz-Boendermaker S, Marcar VL, Meier ML, Boendermaker B, Humphreys BK. Reorganization in secondary somatosensory cortex in chronic low back pain patients. *Spine (Phila Pa 1976)*. 2016;41:E667–E673. https://doi.org/10.1097/BRS.0000000000001348.
93. Lloyd D, Findlay G, Roberts N, Nurmikko T. Differences in low back pain behavior are reflected in the cerebral response to tactile stimulation of the lower back. *Spine (Phila Pa 1976)*. 2008;33:1372–1377. https://doi.org/10.1097/BRS.0b013e3181734a8a.
94. Schabrun SM, Elgueta-Cancino EL, Hodges PW. Smudging of the motor cortex is related to the severity of low back pain. *Spine (Phila Pa 1976)*. 2017;42:1172–1178. https://doi.org/10.1097/BRS.0000000000000938.
95. Tsao H, Galea MP, Hodges PW. Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. *Brain*. 2008;131:2161–2171. https://doi.org/10.1093/brain/awn154.
96. Stenberg G, Fjellman-Wiklund A, Ahlgren C. "I am afraid to make the damage worse" - fear of engaging in physical activity among patients with neck or back pain - a gender perspective. *Scand J Caring Sci*. 2014;28:146–154. https://doi.org/10.1111/scs.12043.
97. Afrell M, Biguet G, Rudebeck CE. Living with a body in pain—between acceptance and denial. *Scand J Caring Sci*. 2007;21:291–296. https://doi.org/10.1111/j.1471-6712.2007.00475.x.
98. Crowe M, Whitehead L, Gagan MJ, Baxter GD, Pankhurst A, Valledor V. Listening to the body and talking to myself - the impact of chronic lower back pain: a qualitative study. *Int J Nurs Stud*. 2010;47:586–592. https://doi.org/10.1016/j.ijnurstu.2009.09.012.
99. Osborn M, Smith JA. Living with a body separate from the self. The experience of the body in chronic benign low back pain: an interpretative phenomenological analysis. *Scand J Caring Sci*. 2006;20:216–222. https://doi.org/10.1111/j.1471-6712.2006.00399.x.
100. Moseley LG. I can't find it! Distorted body image and tactile dysfunction in patients with chronic back pain. *Pain*. 2008;140:239–243. https://doi.org/10.1016/j.pain.2008.08.001.
101. Nishigami T, Mibu A, Osumi M et al. Are tactile acuity and clinical symptoms related to differences in perceived body image in patients with chronic nonspecific lower back pain? *Man Ther*. 2015;20:63–67. https://doi.org/10.1016/j.math.2014.06.010.
102. Ehrenbrusthoff K, Ryan CG, Grüneberg C, Martin DJ. A systematic review and meta-analysis of the reliability and validity of sensorimotor measurement instruments in people with chronic low back pain. *Musculoskelet Sci Pract*. 2018;35:73–83. https://doi.org/10.1016/j.msksp.2018.02.007.
103. Janssens L, Goossens N, Wand BM, Pijnenburg M, Thys T, Brumagne S. The development of the Dutch version of the Fremantle Back Awareness Questionnaire. *Musculoskelet Sci Pract*. 2017;32:84–91. https://doi.org/10.1016/j.msksp.2017.09.003.
104. Wand BM, Catley MJ, Rabey MI, O'Sullivan PB, O'Connell NE, Smith AJ. Disrupted self-perception in people with chronic low back pain. Further evaluation of the Fremantle Back Awareness Questionnaire. *J Pain*. 2016;17:1001–1012. https://doi.org/10.1016/j.jpain.2016.06.003.
105. Moseley G, Butler D. *Explain Pain Supercharged*. Adelaide, Australia: Noi Group Publications; 2017.
106. Moseley GL, Nicholas MK, Hodges PW. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. *Clin J Pain*. 2004;20:324–330. https://doi.org/10.1097/00002508-200409000-00007.
107. Moseley L. Combined physiotherapy and education is efficacious for chronic low back pain. *Aust J Physiother*. 2002;48:297–302. https://doi.org/10.1016/S0004-9514(14)60169-0.
108. Yager RE. The constructivist learning model. *Sci Teach*. 1991;58:52–57.
109. Moseley GL, Butler DS. *The Explain Pain Handbook: Protectometer*. Adelaide, Australia: Noigroup Publications; 2015.
110. Moseley LG. It is not just the brain that changes itself—time to embrace bioplasticity? *Interational Association for the Study of Pain*. 2022. Accessed April 19, 2022. https://www.iasp-pain.org/publications/relief-news/article/time-to-embrace-bioplasticity/.
111. Leake HB, Moseley GL, Stanton TR, O'Hagan ET, Heathcote LC. What do patients value learning about pain? A mixed-methods survey on the relevance of target concepts after pain science education. *Pain Publish Ah*. 2021;162:2558–2568. https://doi.org/10.1097/j.pain.0000000000002244.
112. Cashin AG, Lee H, Traeger AC et al. Producing clinically meaningful reductions in disability: a causal mediation analysis of a patient education intervention. *J Pain*. 2021;23:236–247. https://doi.org/10.1016/j.jpain.2021.07.007.
113. de Jong T, Ferguson-Hessler MGM. Types and qualities of knowledge. *Educ Psychol*. 1996;31:105–113. https://doi.org/10.1207/s15326985ep3102_2.
114. Karran EL, Hillier SL, Yau Y-H, McAuley JH, Moseley GL. A quasi-randomised, controlled, feasibility trial of GLITtER (Green Light Imaging Interpretation to Enhance Recovery)—a psychoeducational intervention for adults with low back pain attending secondary care. *PeerJ*. 2018;6:e 4301. https://doi.org/10.7717/peerj.4301.
115. Moseley GL, Gallace A, Spence C. Bodily illusions in health and disease: physiological and clinical perspectives and the concept of a cortical 'body matrix. *Neurosci Biobehav Rev*. 2012b;36:34–46. https://doi.org/10.1016/j.neubiorev.2011.03.013.
116. Nishigami T, Watanabe A, Maitani T et al. Development and validation of a shoulder-specific body-perception questionnaire in people with persistent shoulder pain. *BMC Musculoskelet Disord*. 2021;22:98. https://doi.org/10.1186/s12891-021-03944-z.
117. Nishigami T, Mibu A, Tanaka K et al. Development and psychometric properties of knee-specific body-perception questionnaire in people with knee osteoarthritis: The Fremantle Knee Awareness Questionnaire. *PLoS One*. 2017;12:e0179225. https://doi.org/10.1371/journal.pone.0179225.
118. Catley MJ, O'Connell NE, Berryman C, Ayhan FF, Moseley GL. *Is tactile acuity altered in people with chronic pain?* A systematic review and meta-analysis. *J Pain*. 2014;15:985–1000 doi:https://doi.org/10.1016/j.jpain.2014.06.009.

119. Luomajoki H, Moseley GL. Tactile acuity and lumbopelvic motor control in patients with back pain and healthy controls. *Br J Sports Med*. 2011;45:437–440. https://doi.org/10.1136/bjsm.2009.060731.
120. Moseley GL, Flor H. Targeting cortical representations in the treatment of chronic pain. *Neurorehabil Neural Repair*. 2012;26: 646–652. https://doi.org/10.1177/1545968311433209.
121. Bowering KJ, Butler DS, Fulton IJ, Moseley GL. Motor imagery in people with a history of back pain, current back pain, both, or neither. *Clin J Pain*. 2014;30:1–19. https://doi.org/10.1097/AJP.0000000000000066.
122. Chang W-J, O'Connell NE, Beckenkamp PR, Alhassani G, Liston MB, Schabrun SM. Altered primary motor cortex structure, organization, and function in chronic pain: a systematic review and meta-analysis. *J Pain*. 2018;19:341–359. https://doi.org/10.1016/j.jpain.2017.10.007.
123. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. *Spine (Phila Pa 1976)*. 1996;21:2640–2650. https://doi.org/10.1097/00007632-199611150-00014.
124. Wallwork SB, Bellan V, Catley MJ, Moseley GL. Neural representations and the cortical body matrix: implications for sports medicine and future directions. *Br J Sports Med*. 2015;50: 990–996. https://doi.org/10.1136/bjsports-2015-095356.
125. Goldinger SD, Papesh MH, Barnhart AS, Hansen WA, Hout MC. The poverty of embodied cognition. *Psychon Bull Rev*. 2016;23: 959–978. https://doi.org/10.3758/s13423-015-0860-1.
126. Zadra JR, Weltman AL, Proffitt DR. Walkable distances are bioenergetically scaled. *J Exp Psychol Hum Percept Perform*. 2016;42:39–51. https://doi.org/10.1037/xhp0000107.
127. Flor H, Denke C, Schaefer M, Grüsser S. Effect of sensory discrimination training on cortical reorganisation and phantom limb pain. *Lancet*. 2001;357:1763–1764. https://doi.org/10.1016/S0140-6736(00)04890-X.
128. Moseley GL, Zalucki NM, Wiech K. Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain. *Pain*. 2008;137:600–608. https://doi.org/10.1016/j.pain.2007.10.021.
129. Moseley GL, Wiech K. The effect of tactile discrimination training is enhanced when patients watch the reflected image of their unaffected limb during training. *Pain*. 2009;144:314–319. https://doi.org/10.1016/j.pain.2009.04.030.
130. Trapp W, Weinberger M, Erk S et al. A brief intervention utilising visual feedback reduces pain and enhances tactile acuity in CLBP patients. *J Back Musculoskelet Rehabil*. 2015;28:651–660. https://doi.org/10.3233/BMR-140561.
131. Wälti P, Kool J, Luomajoki H. Short-term effect on pain and function of neurophysiological education and sensorimotor retraining compared to usual physiotherapy in patients with chronic or recurrent non-specific low back pain, a pilot randomized controlled trial rehabilitation, physic. *BMC Musculoskelet Disord*. 2015;16:83. https://doi.org/10.1186/s12891-015-0533-2.
132. Kälin S, Rausch-Osthoff A-K, Bauer CM. What is the effect of sensory discrimination training on chronic losw back pain? A systematic review. *BMC Musculoskelet Disord*. 2016;17:143. https://doi.org/10.1186/s12891-016-0997-8.
133. Pleger B, Tegenthoff M, Ragert P et al. Sensorimotor returning in complex regional pain syndrome parallels pain reduction. *Ann Neurol*. 2005;57:425–429. https://doi.org/10.1002/ana.20394.
134. Bowering KJ, O'Connell NE, Tabor A et al. The effects of graded motor imagery and its components on chronic pain: a systematic review and meta-analysis. *J Pain*. 2013;14:3–13. https://doi.org/10.1016/j.jpain.2012.09.007.
135. Moseley GL, Butler DS, Beames TB, Giles TJ. *The Graded Motor Imagery Handbook*. Adelaide, Australia: Noigroup Publications; 2012.
136. Bray H, Moseley GL. Disrupted working body schema of the trunk in people with back pain. *Br J Sports Med*. 2011;45: 168–173. https://doi.org/10.1136/bjsm.2009.061978.
137. Parsons LM, Fox PT, Downs JH et al. Use of implicit motor imagery for visual shape discrimination as revealed by PET. *Nature*. 1995;375:54–58. https://doi.org/10.1038/375054a0.
138. Alazmi L, Gadsby GE, Heneghan NR, Punt TD. Do trunk-based left/right judgment tasks elicit motor imagery? *Musculoskelet Sci Pract*. 2018;35:55–60. https://doi.org/10.1016/j.msksp.2018.03.002.
139. Priganc VW, Stralka SW. Graded motor imagery. *J Hand Ther*. 2011;24:164–169. https://doi.org/10.1016/j.jht.2010.11.002.
140. Lotze M, Moseley GL. Clinical and neurophysiological effects of progressive movement imagery training for pathological pain. *J Pain*. 2022;23:1480–1491. https://doi.org/10.1016/j.jpain.2022.04.008.
141. Strauss S, Barby S, Härtner J et al. Graded motor imagery modifies movement pain, cortical excitability and sensorimotor function in complex regional pain syndrome. *Brain Commun*. 2021;3:fcab216. https://doi.org/10.1093/BRAINCOMMS/FCAB216.
142. Strauss S, Barby S, Härtner J, Neumann N, Moseley GL, Lotze M. Modifications in fMRI representation of mental rotation following a 6 week graded motor imagery training in chronic CRPS patients. *J Pain*. 2021a;22:680–691. https://doi.org/10.1016/j.jpain.2020.12.003.
143. Walz AD, Usichenko T, Moseley GL, Lotze M. Graded motor imagery and the impact on pain processing in a case of CRPS. *Clin J Pain*. 2013;29:276–279. https://doi.org/10.1097/AJP.0b013e318250f4e8.
144. Zhang X, de Beukelaar TT, Possel J et al. Movement observation improves early consolidation of motor memory. *J Neurosci*. 2011;31:11515–11520. https://doi.org/10.1523/JNEUROSCI.6759-10.2011.
145. Moseley GL. Is successful rehabilitation of complex regional pain syndrome due to sustained attention to the affected limb? A randomised clinical trial. *Pain*. 2005;114:54–61. https://doi.org/10.1016/j.pain.2004.11.024.
146. Elgueta-Cancino E, Schabrun S, Danneels L, Hodges P. A clinical test of lumbopelvic control: development and reliability of a clinical test of dissociation of lumbopelvic and thoracolumbar motion. *Man Ther*. 2014;19:418–424. https://doi.org/10.1016/j.math.2014.03.009.
147. Sahrmann S. *Diagnosis and Treatment of Movement Impairment Syndromes*. St Louis: Mosby Inc; 2021.
148. Macedo LG, Latimer J, Maher CG et al. Effect of motor control exercises versus graded activity in patients with chronic nonspecific low back pain: a randomized controlled trial. *Phys Ther*. 2012;92:363–377. https://doi.org/10.2522/ptj.20110290.
149. Tsao H, Galea MP, Hodges PW. Driving plasticity in the motor cortex in recurrent low back pain. *Eur J Pain*. 2010;14:832–839. https://doi.org/10.1016/j.ejpain.2010.01.001.
150. Boersma K, Linton S, Overmeer T, Jansson M, Vlaeyen J, De Jong J. Lowering fear-avoidance and enhancing function through exposure in vivo: a multiple baseline study across six patients with back pain. *Pain*. 2004;108:8–16. https://doi.org/10.1016/j.pain.2003.03.001.
151. George SZ, Zeppieri G, Cere AL et al. A randomized trial of behavioral physical therapy interventions for acute and sub-acute low back pain (NCT00373867). *Pain*. 2008;140:145–157. https://doi.org/10.1016/j.pain.2008.07.029.
152. Leeuw M, Goossens MEJB, van Breukelen GJP et al. Exposure in vivo versus operant graded activity in chronic low back pain patients: results of a randomized controlled trial. *Pain*. 2008;138: 192–207. https://doi.org/10.1016/j.pain.2007.12.009.
153. Linton SJ, Boersma K, Jansson M, Overmeer T, Lindblom K, Vlaeyen JWS. A randomized controlled trial of exposure in vivo for patients with spinal pain reporting fear of work-related activities. *Eur J Pain*. 2008;12:722–730. https://doi.org/10.1016/j.ejpain.2007.11.001.
154. Vlaeyen JWS, De Jong J, Geilen M, Heuts PHTG, Van Breukelen G. Graded exposure in vivo in the treatment of pain-related

fear: a replicated single-case experimental design in four patients with chronic low back pain. *Behav Res Ther*. 2001;39:151–166. https://doi.org/10.1016/S0005-7967(99)00174-6.

155. Wand BM, Tulloch VM, George PJ et al. Seeing it helps: movement-related back pain is reduced by visualization of the back during movement. *Clin J Pain*. 2012;28:602–608. https://doi.org/10.1097/AJP.0b013e31823d480c.
156. O'Sullivan PB, Caneiro JP, O'Keeffe M et al. Cognitive functional therapy: an integrated behavioral approach for the targeted management of disabling low back pain. *Phys Ther*. 2018;98:408–423. https://doi.org/10.1093/ptj/pzy022.
157. Marich AV, Lanier VM, Salsich GB, Lang CE, Van Dillen LR. Immediate effects of a single session of motor skill training on the lumbar movement pattern during a functional activity in people with low back pain: a repeated-measures study. *Phys Ther*. 2018;98:605–615. https://doi.org/10.1093/ptj/pzy044.
158. Sahrmann S, Azevedo DC, Van Dillen L. Diagnosis and treatment of movement system impairment syndromes. *Brazilian J Phys Ther*. 2017;21:391–399. https://doi.org/10.1016/j.bjpt.2017.08.001.
159. Van Dillen LR, Lanier VM, Steger-May K et al. Effect of motor skill training in functional activities vs strength and flexibility exercise on function in people with chronic low back pain: a randomized clinical trial. *JAMA Neurol*. 2021;78:385–395. https://doi.org/10.1001/jamaneurol.2020.4821.
160. Alemanno F, Houdayer E, Emedoli D et al. Efficacy of virtual reality to reduce chronic low back pain: proof-of-concept of a non-pharmacological approach on pain, quality of life, neuropsychological and functional outcome. *PLoS One*. 2019;14:e0216858. https://doi.org/10.1371/journal.pone.0216858.
161. Harvie DS. Immersive education for chronic condition self-management. *Front Virtual Real*. 2021;2:657761. https://doi.org/10.3389/frvir.2021.657761.
162. Khan KM, Scott A. Mechanotherapy: how physical therapists' prescription of exercise promotes tissue repair. *Br J Sports Med*. 2009;43:247–252. https://doi.org/10.1136/bjsm.2008.054239.
163. Steiger F, Wirth B, De Bruin ED, Mannion AF. Is a positive clinical outcome after exercise therapy for chronic non-specific low back pain contingent upon a corresponding improvement in the targeted aspect (s) of performance? A systematic review. *Eur Spine J*. 2012;21:575–598. https://doi.org/10.1007/s00586-011-2045-6.
164. Lee Mansell G, McAuley JH, Kamper SJ et al. Causal mechanisms in the clinical course and treatment of back pain. *Best Pract Res Clin Rheumatol*. 2016;30:1074–1083. https://doi.org/10.1016/j.berh.2017.04.001.
165. Cashin AG, Booth J, McAuley JH et al. Making exercise count: considerations for the role of exercise in back pain treatment. *Musculoskeletal Care*. 2021;20:259–270. https://doi.org/10.1002/msc.1597.
166. McGill S. *Low Back Disorders. Evidence-Based Prevention and Rehabilitation*. 3rd ed. Champaign, IL: Human Kinetics; 2015.
167. Catley MJ, O'Connell NE, Moseley GL. How good is the neurophysiology of Pain Questionnaire? A Rasch analysis of psychometric properties. *J Pain*. 2013;14:818–827. https://doi.org/10.1016/j.jpain.2013.02.008.
168. Travers M, Gibson W, Darlow B, et al. My back is fragile and it is never getting better (The fremantle danger in Me Questionnaire). In: *Australian Physiotherapy Association Conference: Transform*. Adelaide, Australia: Australian Physiotherapy Association; 2019.
169. Cashin AG, McAuley JH. Measuring two-point discrimination threshold with a caliper. *J. Physiother.* 2017;63:186. https://doi.org/10.1016/j.jphys.2017.04.005.
170. Cashin AG, Lee H, Bagg MK et al. Investigating the mechanisms of graded sensorimotor precision training in adults with chronic nonspecific low back pain: protocol for a causal mediation analysis of the RESOLVE Trial. *JMIR Res Protoc*. 2021;10:e26053. https://doi.org/10.2196/26053.