

Editorial

Updating our model of pain: low back pain through the lens of active inference

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Introduction

Despite numerous available treatment options, many people who experience an episode of low back pain progress to chronic low back pain.^{1,2} In most circumstances, this condition is recognised as chronic non-specific low back pain (CNSLBP) – persisting pain in the absence of any identifiable specific or serious underlying pathology.³ For people with CNSLBP seeking care, various treatments may produce modest short-term effects on pain and disability,^{4–8} although a long-term solution remains elusive.^{9,10} In this population, ‘high-value’ care appears hard to find. There is an urgent need to better understand CNSLBP and to develop and test treatments based on this understanding. One potential solution may be to reconsider pain in ways that align with contemporary neuroscience. Active inference is an emerging framework within neuroscience that suggests that conscious experiences, like pain, involve the interaction of predictions based on internal models of the world and ourselves, sensory information from the body, information from the external world, and a recalibration of our interactions with the environment.^{11–13} Active inference unifies planning, attention, cognition, memory, action and perception to serve the biological imperative of minimising prediction error – the difference between what is expected (predicted) and what is realised (sensory events).^{12,14}

This paper aims to introduce the concept of active inference for clinicians working with people experiencing pain. In Part 1, we explore how our percepts are inferred based on the integration of precision-weighted, top-down predictions and bottom-up sensory evidence. We also outline how we act and use our bodies to shape our experiences. In Part 2, we consider the problem of CNSLBP through the lens of active inference^a, although it is likely that the proposed concepts could equally apply to other persistent pain states. Key technical terms used throughout the paper are defined in [Box 1](#). By synthesising concepts from the broader active inference literature and evidence from low back pain research, we aim to help clinicians develop a new understanding of pain that aligns with the active inference framework and to outline key implications for clinical practice.

Part 1: The inferred nature of our conscious experiences and the power of predictions

Contemporary explanations of perception, including predictive processing, emphasise that we do not operate as passive recipients

of sensory information.¹⁵ Rather, we try to make sense of ourselves and the world by actively (though largely unconsciously) predicting the sensory information impacting the system. The role of perception is to infer the cause of sensory input. What we perceive represents the best-fitting model based on both what was expected and the sensory information that was subsequently received.^{15–17}

Numerous observations clearly substantiate the idea that our conscious experiences are not a direct read-out of sensory inputs. For example, when taste-testing identically flavoured chocolate, participants rated the samples labelled *dark chocolate* as having a more intense chocolate flavour than the samples labelled *milk chocolate*.¹⁸ In this example, sensory information is integrated with predictions (based on internal models, such as ‘dark chocolate has a more intense chocolate taste’) and the emergent experience reflects an entangled combination of the two.¹⁸ Similarly, it has been demonstrated that manipulating the colour of wine with an odourless dye alters people’s descriptions of how the wine smells.¹⁹ That our percepts of smell and taste can be influenced by information available prior to the interaction with the apparatus of smell or taste demonstrates that sensory evidence is not processed at *face value*, but according to the expectations of an ‘active, predicting, perception maker’.^{b 16,17,21–23}

Integrating top-down predictions and bottom-up nociception – experimental pain examples

Pain experiments show that predictions directly influence the pain experience. Healthy people using a sham ‘head-stimulator’ reported pain, and their pain intensity corresponded to the device’s visible intensity setting.²⁴ In this example, the experience of pain was realised by manipulating the individual’s prediction of pain in the absence of any additional noxious stimulation.

When pain-free participants were touched on the neck with an extremely cold metal rod, those who were told that the bar was ‘hot’ tended to rate the painfulness as higher than those who were told that the bar was ‘cold’.²⁵ Similarly, when pain-free participants received repeated thermal stimulation corresponding to low-intensity, medium-intensity, and high-intensity noxious inputs preceded by auditory cues that signalled the forthcoming noxious stimulus, medium-temperature trials were rated as substantially more painful when preceded by high-pain cues compared with when preceded by low-pain cues.²⁶ These examples suggest that pain may be shaped by

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^a We use active inference to mean combined perceptual and action strategies.

^b We confine our discussion to sensory apparatus; however, this process is considered to apply to the wider person including the neuroendocrine, neuro-immune and autonomic nervous systems. (For a more detailed discussion see²⁰).

Box 1. Glossary of technical terms.

Active inference is an emerging framework that unifies planning, attention, cognition, memory, action and perception. According to active inference, our percepts (what we experience) are never solely reconstructions of what is happening in ourselves or in the world. Rather, they reflect the action-perception loops that enable co-ordinated behaviours that act with reference to and on our environments. According to this framework, we have a biological imperative to minimise discrepancies between (unconsciously) predicted sensory information and incoming sensory information.¹⁵

Generative (internal) model is the implicit model used by a person to predict and explain incoming sensory information. Furthermore, it also represents beliefs about how the world and the body should be. Accordingly, the generative model guides both a person's perception and their actions.¹⁶

Prediction error refers to the discrepancy between predictions (based on the generative model) and incoming sensory information.¹⁵

Prediction error minimisation is the process of minimising prediction error. This can be achieved through model updating (analogous to learning in so far as the model changes to fit the sensory information). We can also act in ways that make the sensory information more closely align with predicted sensory information — in this way we are seeking to confirm the prediction of our generative model. Thus, action and perception serve a singular purpose — prediction error minimisation. Acting in ways that minimise prediction error and maximise model evidence; this **self-evidencing** is intended to satisfy biological needs and our chances of survival.¹⁷

Precision weighting is the process of assigning more weight to sensory information and predictions that are considered more precise, trustworthy or reliable. Accordingly, we assign greater importance to prediction errors or sensory information that are more predictable, based on factors like signal versus noise and other environmental cues.¹⁸

explicit expectations rather than simply by the sensory evidence from the externally applied stimulus.

The variable interaction between predictions and sensory information also occurs when the resulting percept pertains to our inner physical (interoceptive) selves, rather than being confined to externally applied (exteroceptive) noxious stimuli.²⁷ Participants with delayed onset muscle soreness affecting the quadriceps reported higher pain intensity during a standardised muscle contraction when viewing their thigh under magnification than when viewing it without magnification.²⁸ Here, the same movement produced two different pain experiences, dependent on the visual information available. The authors propose that an enlarged view of the thigh suggests a swollen, more severely affected leg, in which an enhanced protective response to loading is indicated.

Integrating top-down and bottom-up — examples from people living with pain

Similar findings emerge from research conducted on people living with pain. Non-noceptive sources of information that influence generative models about the health of the body part (and therefore the prediction of pain on loading) have also been shown to influence clinical pain. In a proof-of-concept study of augmented reality, participants were provided with real-time visual input showing the back with enhanced muscularity while they performed a lifting task. Embodying the illusion of a strong, fit back lessened pain intensity with lifting.²⁹ Additionally, virtual reality has been used to induce a mismatch between visual field rotation and true head rotation in people with neck pain evoked by rotation. When the visual field moved more than the true head rotation, pain occurred earlier in the movement; when the visual field moved less than the true head rotation, pain occurred later in the movement.³⁰ Therefore, the onset of pain was influenced by the range of motion

predicted or estimated to have occurred in the neck based on visual models and not the actual range achieved. Similar results have been shown for lumbar spine flexion³¹ and extension.³²

The content of diagnostic information about the cause of pain can also influence pain. Pain intensity during movement was higher in people with Achilles tendinopathy who had just received a traditional diagnostic explanation that explicitly referenced tissue pathology than in participants whose diagnostic explanation did not reference tissue pathology.³³ In this study, potential sources of bias were rigorously controlled for, providing robust evidence that the prior information was a key determinant of the clinical pain experience.

Prediction error minimisation, model updating and self-evidencing

Predictive processing makes clear the interaction of predictions and incoming sensory evidence in generating and modulating our percepts, including pain.¹⁵ However, it is unlikely that predictive processing occurs in isolation, and it is perhaps better understood when integrated into active inference. When trying to make sense of and experience the world, there is an inseparable connection between the internal and the external worlds; both physically and socioculturally.^{20,34,35} Accordingly, action and perception are thought to be inseparably linked because we actively impinge upon and selectively sample our sensory modalities to confirm our models.¹² Current theory and accumulating evidence suggest this is enacted through a hierarchically arranged nervous system, in which each level generates cascades of top-down predictions about 'what is coming next'.^{13,15–17} It is posited that at each level of the hierarchy, top-down predictions are continually compared to the bottom-up information, to calculate a prediction error, and it is this error that is the essential currency of message passage in the central nervous system.^{13,36}

Making sense of the body and the world is fundamentally enacted by processes that seek to minimise prediction error.¹² According to active inference, perception and action are two complementary ways to achieve the same objective of minimising prediction error by maximising model evidence (or self-evidencing).³⁷ This is a key tenet of active inference — we, as active agents, constantly seek to maximise model evidence by preferentially garnering sensory information that is consistent with our model.¹² In this way, we (attempt to) stay within 'preferred states' that optimise survival.³⁸

Conceptually, we minimise prediction error at every level in the hierarchy by: updating internal generative models and revising predictions to better explain incoming sensory input; effecting bodily actions to modify sensory input to better interrogate predictions; and modulating the likelihood of detecting error through differential weighting of incoming sensory streams. Although, for simplicity, these three mechanisms are outlined in isolation below, they are more typically employed en masse.

Updating internal models

Updating generative or internal models can be considered synonymous with knowledge acquisition or 'learning'. For example, you are listening to a song you love, but there are a few lyrics that you cannot decipher. A friend tells you what the lyrics are. This new information allows you to form updated predictions. The next time you hear the song, you can clearly discern the previously 'hidden' words. In this example, the incoming sensory information has not changed, but your updated generative model makes more precise predictions, allowing a credible percept to emerge. Here, the model has changed to fit the evidence. That the generative model can be updated so quickly and easily points to the extraordinary capacity for change within the system. Notably, if the lyrics provided by your friend are not correct, you will likely hear what they suggested and not the true lyrics.^c It appears that what we experience is based on

^c See <https://www.facebook.com/thepeterkay/videos/peter-kay-misheard-lyrics/508276933203663/> for amusing examples.

the best available evidence, not some 'essential sensory truth'; this is also the basis for optical and other forms of sensory illusion.

Although model updating is an important component of the active inference framework, it is important to clarify that we are not suggesting that someone in pain can merely update their model and 'decide' that they are not in pain. We are suggesting that the framing one applies to pain can matter — for an interesting example, see³⁹ where a physically identical noxious stimulus was found to hurt more or less, depending on the participant's (manipulated) perception of whether the stimulus was potentially dangerous or entirely safe.

Effecting bodily actions to adjust sensory input to better match predictions

Under the active inference framework, perception and action are inextricably linked. We predict a given set of sensory inputs and act to confirm or generate the expected sensory consequences. Under normal conditions, we selectively sample evidence consistent with our predictions with the aim of confirming our model or, at least, minimising uncertainty regarding the model.⁴⁰ To that end, bodily actions are deployed to sharpen sensory information and to resolve the incongruence between prediction and sensory evidence.^{15,17,41–43} For example, you are sitting in the lounge room at home, staring at the computer, and call out to ask your daughter to come through and help you with today's Wordle. As you ponder what five-letter word could contain the letter 'm' twice, you notice in your peripheral vision someone entering the room who seems to be of a different size and shape from your daughter. The sensory evidence you have available does not match the sensory stream predicted by your daughter entering the room. To resolve this prediction error, you turn your head to focus more light on the fovea and obtain more high-quality visual data; the error is now resolved because the additional data allow you to infer that it was, in fact, your son who entered the room.

Modulating the likelihood of detecting error through differential weighting of incoming sensory inputs

We are constantly faced with a barrage of sensory information from multiple proprioceptive, interoceptive and exteroceptive apparatus. So, the system must prioritise, or weight, what information is deemed reliable in any given circumstance or moment.⁴⁴ The weighting of incoming prediction errors is paramount in determining whether a given prediction error signal is attenuated or prioritised within the system. This process, referred to as precision weighting, means that sensory information from varying sources, both intrinsic and extrinsic to the body, has variable potential 'influence' on the final percept.⁴⁴ There is a clear interaction between prediction and weighting, whereby sensory information more aligned with predictions is given greater priority.¹² Additionally, the precision (the inverse of variance) of a sensory modality itself, otherwise known as its reliability or trustworthiness, also determines the weighting given to the information.^{45,46}

More confidence is placed on predicted information and on precise information, with the result that some sensory channels are afforded a greater capacity to: generate an error signal; drive the shaping of perception and action; and influence future predictions. The apparent power of visual input to shape our predictions (as demonstrated in several of the experiments described previously) is not surprising, considering that visual input is normally highly precise and therefore heavily weighted under most conditions. Precision weighting provides a mechanism by which predictions and sensory input coalesce to influence perceptions.^{13,47} Weighting allows us to rely more on sensory evidence when it is deemed highly trustworthy and to rely more on top-down predictions when the sensory evidence is noisy and imprecise.^{15,48}

The processes by which prediction error is resolved indicate that action and perception are intimately entwined, such that the

nervous system cannot be operationally separated into discrete motor and sensory systems, nor separated from the wider person or, indeed, their world (Figure 1).^{20,35} Thus, attention, learning, perception, cognition and action, all of which have a reported impact on pain, are intrinsically and dynamically linked because we selectively sample and adjust sensory evidence.^{12,13,15,48,49}

Part 2: Reinterpreting low back pain through the lens of active inference

Internal models of the back (thoughts as prediction)

Although internal models are normally dynamically formed and in a constant state of flux, rigidly held internal models of our physical and psychological self are considered relevant to persistent back pain.⁵⁰ We suggest that much of the health-related information available to people with CNSLBP likely contributes to the formulation of a pain-expectant model, and such beliefs may be held rigidly and be hard to shift.⁵⁰ For example, elements from back care education and messaging, particularly within the workplace, often present the back as weak, vulnerable and easily injured.^{51–56} Additionally, spinal imaging is frequently obtained in people with CNSLBP (often against guideline recommendations) and is very likely to provide evidence of structural changes within the spine.^{57,58} In clinical contexts, these findings are often described in terms of damage and structural fragility.^{59,60} Indeed, lay terms commonly used by health professionals to describe often incidental findings — most notably 'slipped disc' — conjure thoughts of catastrophic risk to the body.⁶¹ Recent attempts to invert the impact of the information provided by imaging show promise; for example, the 'Green light imaging interpretation to enhance recovery' ('GLITtER') intervention⁶² and including epidemiological information on radiology reports.^{63–65} However, patients often still receive explanations of their problem that would reasonably reinforce internal models that the back is vulnerable and that pain with activity is to be expected.⁶⁶ Indeed, a recent experiment demonstrated that when encountering pathoanatomical labels such as 'degeneration' or 'slipped disc', people with low back pain expressed higher perceived seriousness of the problem and lower expectations of recovery than when terms such as 'lumbar sprain' or 'non-specific low back pain'³ were presented.⁶⁷ Worse still, the negative impact of the pathoanatomical labels was highest in those at risk of poor outcome.⁶⁷ Although not focused on the back, we now have causal data showing that information about the state of the body provided to people with a musculoskeletal pain problem has an immediate effect on activity-related pain intensity.³³ Finally, it is worth reflecting that asking open questions and giving targeted questionnaires probing psychological constructs thought to be relevant to a person's CNSLBP will only give access to the conscious and explicit parts of a person's internal model, leaving even the most inquisitive clinicians with an incomplete appreciation of their patients' internal models. Nonetheless, we encourage clinicians to be deeply curious and to embark on a journey with their patients to explore the person's understanding of pain both from their journey to date and their expectations for the future.

Differential weighting of sensory information and model updating in back pain

People with CNSLBP appear to perceive that 'all is not well' with the back. They have been shown to demonstrate decreased connectedness of the painful area⁶⁸ and describe a loss of ownership of the back, as well as feelings of foreignness, peculiarity and diminished control.^{69–72} Evidence suggests that CNSLBP is associated with a decrease in sensitivity to non-noxious sensory streams that help a person be comfortable with and connected to the back. People with CNSLBP: have larger movement detection thresholds⁷³ and mechanical detection thresholds;^{74–77} are less able to detect postural drift;⁷⁸ and are less responsive to back muscle vibration.^{79,80} They

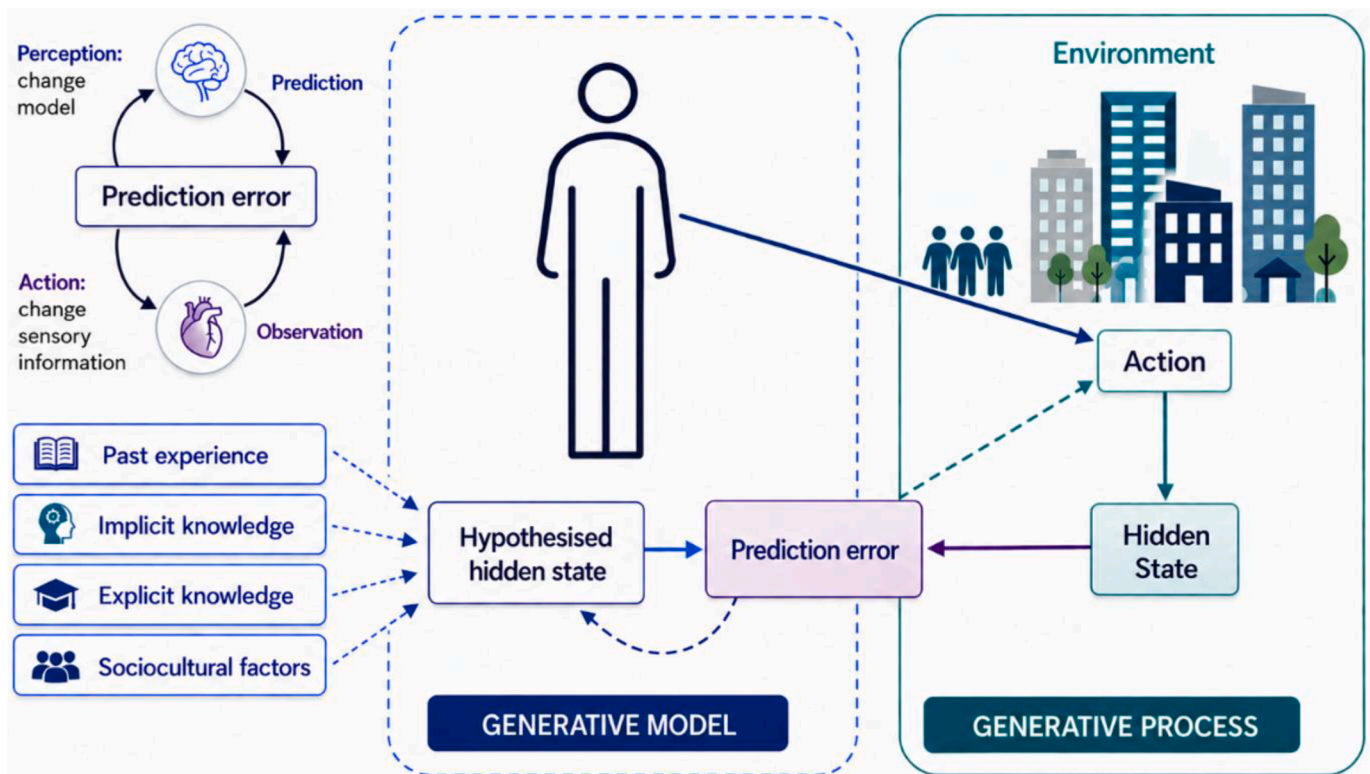


Figure 1. Action-perception loop of active inference. The figure outlines the delineation between the generative process (the causal structure of the world that generates observations for the person) and the person's generative model (how the person expects the observations to be generated). Predictions ('Hypothesised hidden states') are informed by a multitude of factors — here, we depict past experiences, implicit knowledge and sociocultural factors to illustrate the individual nature of this process. The degree of prediction error is measured by comparing the sensory states predicted by the generative model ('Hypothesised hidden state') and the actual sensory states generated by the true causal structure of the environment ('Hidden state' — referred to as hidden because the person is always one step removed from the sensory observations). We have a biological imperative to minimise the discrepancy between predictions and actual sensory states. This occurs with respect to interoceptive and exteroceptive sensory streams (top left). The image depicts complementary ways to minimise prediction error (white squares and dotted arrows in the main image): by updating the generative model to more closely match realised sensory states (ie, perception-as-inference or predictive coding) and/or by acting to change the environment in such a way that the next sensations will match the predictions (ie, action-as-inference or active inference) (see Chapter 2 of¹²). The role of precision weighting is not depicted in this image.

have decreased tactile acuity^{81,82} and a reduced ability to localise tactile inputs delivered to the back.^{83,84} Furthermore, these alterations in sensitivity to non-noxious stimuli may manifest throughout the neural axis (eg, low somatosensory cortex excitability in the acute stage of low back pain has been causally linked with the development of persisting symptoms).⁸⁵ This constellation of symptoms may be considered to be an alteration in the normal weighting relationship between non-noxious and noxious sensory information, with down-weighting of non-noxious information from the body. Conversely, it appears that there is greater sensitivity (weighting) to sensory information that reinforces 'all is not well'. Although individual variation clearly exists, on average people with CNSLBP have lower pain thresholds associated with noxious thermal,^{86,87} electrical,⁸⁸ pressure^{89–91} and punctate stimulation^{74,92} and reported pain intensities are higher for the same noxious stimulus intensity.⁸⁹

Down-weighting non-noxious information and/or up-weighting noxious information will reinforce the view of a vulnerable and load-intolerant back and may strengthen generative models that predict pain with activity.^{71,93–97} Simultaneously, the stronger the prediction of pain, the more the system will be driven to down-weight non-noxious information and up-weight noxious information, creating a self-sustaining cycle. This is consistent with the active inference position that these changes in modulation of different sensory streams are attempts by the system to minimise prediction error because they serve to better match sensory evidence to predictions. Thus, a 'pain-expectant internal model' likely predicts a dominance of noxious over non-noxious information from the body with loading, and differential weighting of these two sensory streams decreases the likelihood of creating error. In this way, sensitisation — so often regarded as a fundamental contributor

to CNSLBP — emerges as a mechanism of self-evidencing a pain-expectant internal model.

Moving and self-evidencing with back pain

Active inference emphasises the need to consider the individual and the world with which they currently interact, both now and in the past³⁵; emerging evidence suggests that future events are also considered. We are agents who are situated in the world and act upon the world to test and refine our predictions. Resulting percepts are never solely reconstructions of what is happening in ourselves or in the world. Rather, they are a reflection of the action-perception loops that enable co-ordinated behaviours that act with reference to and on our environments.⁴⁸ The suggestion is that movement strategies reflect efforts to influence information about the world and oneself in order to refine and/or sharpen internal models.⁹⁸ Consider the 'maladaptive' behaviours, such as breath-holding or co-activating the trunk muscles, often seen when a person with CNSLBP performs a feared and provocative task, such as bending or lifting.^{99,100} Generally, these behaviours have been thought of as preparatory for an expected perceptual outcome of pain and labelled maladaptive when they appear paradoxically to increase noxious loading of the back during movement;^{101–103} however, it is possible to reinterpret these strategies through the lens of active inference. They may be viewed as adaptive actions taken by the individual to better match sensory evidence with prediction. According to this theory, maximising model evidence could be enacted by enhancing nociceptive sensory evidence, or it is possible that such action is intended to reduce incoming proprioceptive information relating to the task (by limiting movement), thus reducing accompanying and competing non-noxious information, and making incoming

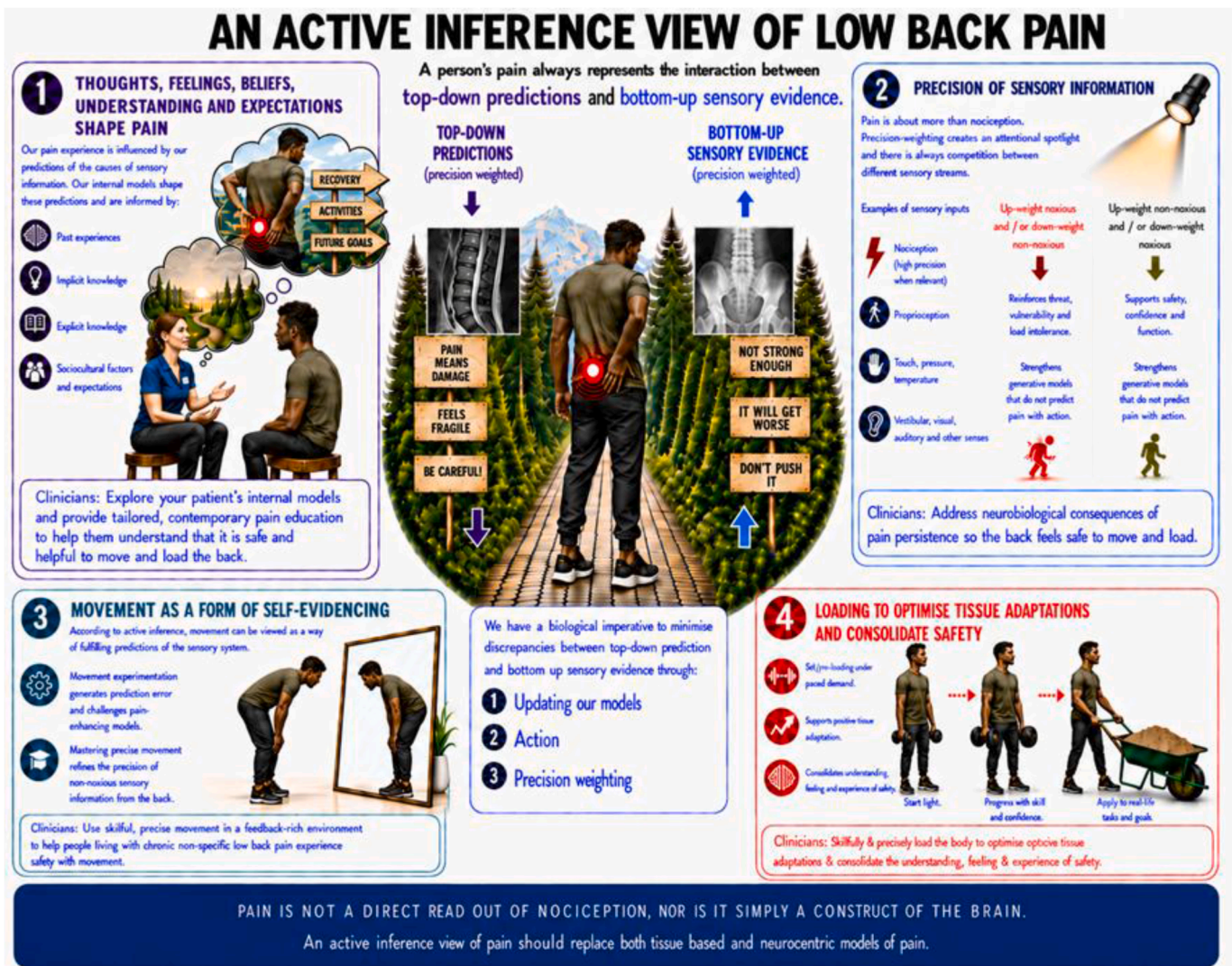


Figure 2. Key implications for clinicians.

nociceptive input 'stand out' and, therefore, more noticeable. Whether the outcome is increased noxious information or diminished non-noxious information reaching the system, it may be that such movement patterns subserve the strengthening of internal models by altering the sensory information to match top-down predictions. Repetition of this cycle may represent powerful experiential learning pathways that render internal models highly resistant to change, making them 'sticky'¹⁰⁴ or highly precise.¹⁰⁵

Reframing our understanding of movement as a way of sampling information from the body and the world may be transformative to understanding pain and its management. Movement patterns viewed by the clinician as 'maladaptive' may be viewed by the active inference-informed community as highly adaptive, insofar as they fulfil the homeostatic imperative of minimising prediction error.^{d,12} Considered in this way, exercise-based therapies are likely to be more successful if steps are first taken to positively influence generative models about the health and load tolerance of the back. Furthermore, movement and loading employed in ways that optimise prior and ongoing safety information present an opportunity to provide positive input to unhelpful internal models of the body and invoke desired prediction errors that produce adaptive model formation. This may explain why exposure therapy for pain-related fear,^{107,108} cognitive functional therapy (CFT),^{109–112} pain

reprocessing therapy (PRT),¹¹³ and education-focused active interventions¹¹⁴ have shown some promise for people in pain. In each case, cognitive strategies to reinforce the structural integrity of the body and the meaning of pain are integrated with movement and loading that is consistent with that supposition.

Recently, a large, well-conducted clinical trial randomised 276 adults with CNLBP to receive a 12-week program of pain education and sensorimotor retraining or a complex, credible sham.¹¹⁵ The intervention group reported clinically meaningful¹¹⁶ and sustained differences in pain and disability in favour of the intervention, although follow-up beyond the 1-year time point and external validation in a multicentre trial are as yet unpublished. Importantly, the graded sensorimotor retraining aligns closely with, and can be interpreted and described through, the lens of active inference. First, the intervention aimed to help participants understand that it is safe and helpful to move and load the back. This represents an attempt to generate a less pain-expectant state by addressing internal *cognitive* models (a very small subset of one's internal model) of the meaning of pain, the health of the back, and the risk to the back from movement and loading. The next step was to refine sensorimotor processing, so that the back felt safe to move and load. This could be interpreted as minimising pain expectancy through updating internal *perceptual* models of the back, as well as optimising the precision of ongoing non-noxious sensory information as the back is moved and loaded. Next, participants undertook precision-focused, feedback-enriched, graded functional movement training to experience safety with movements of increasing threat and complexity. This

^d For an entertaining account of the so-called Bogeyman and error minimisation, see¹⁰⁶.

could be viewed as movement experimentation to generate error and challenge existing pain-enhancing models of the back and further refine the precision of non-noxious sensory information from the back during more functionally relevant tasks. The final step was to consolidate the concepts of understanding, feeling and experiencing safety through the prescription of general strengthening exercises orientated towards patient-derived goals. Through the lens of active inference, this last step could be viewed as repeatedly self-evidencing the capability of the body through loaded movement and sharpening internal models that the body is capable, robust and fit-for-purpose.

A recent trial of CFT for people with CNSLBP had favourable effects on disability and pain¹¹² and these outcomes appear to be sustained at 3-year follow-up.¹¹⁷ The CFT approach appears to align, in part, with the active inference view of low back pain, as the aim of 'making sense of pain' can be interpreted as aiming to update internal models regarding pain and the body, and the threat value of pain. Furthermore, the CFT focus on 'exposure with control' can be interpreted as helping the person experience safety with movements when viewed through the lens of active inference. A favourable long-term result has been observed for CFT plus a placebo movement sensor compared to usual care¹¹² and a shorter-term benefit for pain and disability has been observed when compared with sham laser therapy;¹¹⁸ however, long-term follow-up data comparing CFT only to a complex and credible sham would strengthen the evidence for this promising treatment.

A third treatment, PRT, has demonstrated a remarkable effect on pain for people living with CNSLBP when compared to an open-label placebo and usual care.¹¹³ This result appears to be sustained at 5-year follow-up.¹¹⁹ A PRT approach to managing CNSLBP also seems to align, in part, with the active inference perspective of pain outlined in this editorial by taking a 'top-down' approach to understanding and managing pain; however, larger multicentre trials using a complex and credible sham as a comparator would give greater confidence in the efficacy of PRT. The landscape of treatment may be changing, with these three interventions^{112,113,115} offering considerable hope in the fight against CNSLBP and demonstrating the promise of interventions that align with a contemporary understanding of pain.

How should this be interpreted by the clinician scientist?

The purpose of this paper was to explore and discuss active inference in the context of pain and clinical practice. Key implications for clinicians are summarised in Figure 2. This perspective recognises that pain is always influenced by both sensory information and predictions based on existing multilevel models and their relative weighting. Accordingly, this framework should offer an alternative to brain-focused and peripheralist perspectives of pain by making clear the entangled and inseparable roles of the whole person and their world in the emergence and maintenance of pain. We have specifically sought to translate these ideas to the context of pain and CNSLBP. Such translation is largely based on the interpretation of theoretical and fundamental research, and observational data from people in pain. It is our opinion that much of what is observed in the clinical pain literature and clinical practice aligns well with this framework. We contend that by understanding these principles, clinicians and researchers may be better placed to identify and target key factors relevant to the perception and modulation of pain and engage in robust discussions that seek to align treatment targets and develop strategies consistent with contemporary neuroscience theory.

Footnotes: Nil

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