

Critical Review

Fifteen Years of Explaining Pain: The Past, Present, and Future

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Abstract: The pain field has been advocating for some time for the importance of teaching people how to live well with pain. Perhaps some, and maybe even for many, we might again consider the possibility that we can help people live well without pain. Explaining Pain (EP) refers to a range of educational interventions that aim to change one's understanding of the biological processes that are thought to underpin pain as a mechanism to reduce pain itself. It draws on educational psychology, in particular conceptual change strategies, to help patients understand current thought in pain biology. The core objective of the EP approach to treatment is to shift one's conceptualization of pain from that of a marker of tissue damage or disease to that of a marker of the perceived need to protect body tissue. Here, we describe the historical context and beginnings of EP, suggesting that it is a pragmatic application of the biopsychosocial model of pain, but differentiating it from cognitive behavioral therapy and educational components of early multidisciplinary pain management programs. We attempt to address common misconceptions of EP that have emerged over the last 15 years, highlighting that EP is not behavioral or cognitive advice, nor does it deny the potential contribution of peripheral nociceptive signals to pain. We contend that EP is grounded in strong theoretical frameworks, that its targeted effects are biologically plausible, and that available behavioral evidence is supportive. We update available meta-analyses with results of a systematic review of recent contributions to the field and propose future directions by which we might enhance the effects of EP as part of multimodal pain rehabilitation.

Perspective: *EP is a range of educational interventions. EP is grounded in conceptual change and instructional design theory. It increases knowledge of pain-related biology, decreases catastrophizing, and imparts short-term reductions in pain and disability. It presents the biological information that justifies a biopsychosocial approach to rehabilitation.*

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Key words: Pain education, cognitive intervention, chronic pain, therapeutic neuroscience education, pain biology education.

Historical Context and Beginnings

That pain is a biopsychosocial phenomenon is widely regarded as sacrosanct in academic discussions and research articles, and Loeser's adaptation¹⁴ of

Engel's biopsychosocial model¹⁰ is rightly considered a landmark contribution to the pain field. The dominant application of the biopsychosocial model has been, and to a large extent remains, focused on the impact of pain on sufferers and those around them. The importance of psychosocial factors as mediators of suffering has been well recognized, and several effective treatments have been devised to modulate those factors. Since the seminal contributions of Fordyce,¹² for example, who applied operant conditioning models to assist people in pain to return to behaviors that were consistent with being well, rather than behaviors that were consistent with suffering, psychological therapies have been at the core of many pain management programs. Modern therapies combine behavioral principles

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with cognitive therapies to generate a range of therapeutic approaches collectively termed cognitive-behavioral therapy (CBT).

This wide range of CBT interventions share a reasonably common set of theoretical assumptions about the interactions among environmental events, cognitions, and behaviors, including the proposition that symptoms and dysfunctional behaviors are often cognitively mediated and can therefore be improved by modifying problematic thinking and inaccurate beliefs.² That pain itself is modulated by beliefs appears fundamental to the idea that pain is a biopsychosocial phenomenon.⁴¹ The proposition follows that pain is in part cognitively mediated and can therefore be improved by modifying inaccurate beliefs. This CBT-driven work led the way in advocating for the importance of teaching people how to live well with pain. However, somewhere between the establishment of the biopsychosocial model and the rapid rise of CBTs as the dominant nonpharmacological treatments for chronic pain, a shift occurred toward a *modus operandus* more consistent with "pain is unavoidable—suffering is optional." That is, CBT aimed to manage pain, rather than to treat it. Of course, many well-trained and effective CBT practitioners almost certainly provide credible explanations that include aspects of Explaining Pain (EP). However, the cursory coverage of this material in the CBT literature suggests that the educational component of CBT, considered critical for the subsequent implementation of techniques aimed at changing beliefs and behaviors,⁸ focused on pain's being unavoidable and therefore on the need to learn how to cope with it: "It is important to remember that because the pain is chronic the [pain management program's] approach will not cure or relieve the pain..."³¹ Exactly when or why this shift occurred is not clear; "pain can be modified by our beliefs and behaviors" is inconsistent with "pain cannot be relieved by modifying beliefs and behaviors." Moreover, it is inconsistent with what we now know about the underlying biological mechanisms of pain: that pain is fundamentally dependent on meaning (see Butler et al³ for review). An understanding of pain that was foreshadowed in the gate control theory,¹⁸ articulated more fully 2 decades ago⁴⁵ but only now gaining significant traction, is that it reflects an implicit evaluation of danger to body tissue and the need for protective behavior. This view clearly presents pain as being distinct from nociception, yet upregulation within the nociceptive system (central sensitization) may underpin the idea that pain relief is not a viable target of intervention. Such a perspective is central to the proposal that chronic pain is a disease of the brain (an "immutable neural disruption" model of pain⁷), which has gained popular attention but contrasts with fundamental concepts of pain's being something one feels and the inconsistent link between brain changes and clinical presentation.³⁷

We contend that the absence of strong biological justification for CBT has contributed to its being no more effective for decreasing pain and disability in people with chronic pain than other active treatments⁴⁷

(although, importantly, CBT programs on the whole do relieve pain²⁰). A recent Cochrane overview of multidisciplinary pain management programs also suggests that the long-term effects of CBT for chronic pain are underwhelming.⁹ To some, this suggestion might be unsurprising; we are probably not alone in questioning why someone in pain would engage with treatment aimed at their thoughts, beliefs, and behaviors if they believe that their pain is an accurate marker of tissue damage or of another disease process afflicting their spinal cord and brain. Patients capture this apparent nonsense eloquently: "I *understand* that hurt doesn't always equal harm, but my pain *really* hurts," or "This program is really excellent for those who think they have pain, but it is not for me—I have *real* pain." Such comments provided the impetus for EP: an educational intervention aimed at reconceptualizing pain itself. Perhaps for some, and maybe even for many, it is time to extend the idea of helping people live well with pain to the possibility that we can help people live well without pain.

What EP Is and What It Is Not

EP refers to a range of educational interventions that aim to change someone's understanding of what pain actually is, what function it serves, and what biological processes are thought to underpin it. It refers to both a theoretical framework from which to approach pain treatment and also the approach itself. EP is not a specific set of procedures or techniques. It takes its key tenets from educational psychology, in particular conceptual change strategies, health psychology, and pain-related neuroimmune sciences. The core objective of the EP approach to treatment is to shift one's conceptualization of pain from that of a marker of tissue damage or disease to that of a marker of the perceived need to protect body tissue. This new conceptualization is a pragmatic application of the biopsychosocial model to pain itself rather than to pain-related disability per se.

An explicit grounding in conceptual change theory is one way in which EP is clearly differentiated from previous educational components of pain programs and CBTs. Conceptual change learning is specifically shaped around challenging existing knowledge and knowledge structures, rather than simply learning new information, and refining learning strategies that engage new and potentially challenging concepts.⁴⁴ The conceptual change field was borne from increasing evidence of difficulties that students have in understanding counterintuitive concepts in science phenomena (such as diffusion) that rely on collective or emergent behavior of constituents, as distinct from linear behavior of constituents.^{4,44} EP clearly presents pain as an emergent rather than a linear process³⁸ that is counterintuitive to both the dominant structural pathology model and the more recent model of pain as an immutable neural dysfunction.

EP emphasizes that any credible evidence of danger to body tissue can increase pain and any credible evidence of safety to body tissue can decrease pain.²¹ Key learning

targets in EP include the variable relationship between danger messages (nociception) and pain; the potent influence of context on pain; upregulation in the danger transmission (nociceptive) system as pain persists; the coexistence of several potential protective systems, of which pain is one, but the only one that the sufferer necessarily knows has been engaged; the potential influence of these other protective systems on pain; the adaptability, and therefore trainability, of our biology (including but not limited to the concept of neuroplasticity) and the knowledge that this adaptation back to normality is likely to be slow.

EP has thus far taken several different formats. Early investigations of EP involved intensive one-on-one, small group tutorial-type sessions, or large group seminars lasting up to 3 hours.^{22,23,25,28,29} The approach has been adapted according to preference and economics, and the material has been condensed^{17,32} or has incorporated other methods such as booklets¹⁶ or storybooks.¹³ Alternative names for EP have also emerged (eg, therapeutic neuroscience education, pain biology education, pain neuroscience education), perhaps each aiming to commercially brand a subtle variation on the original concepts. The unifying aspect of all of these modifications is that the core objective is to explain to the learner the key biological concepts that underpin pain, with a proficiency and effect such that learners acquire a functional pain literacy. That is, they understand how their pain is produced (at least to the extent that science currently allows) and they are able to integrate this new understanding into their wider pain and function-related beliefs, attitudes, behaviors, treatment, and lifestyle choices.

Over the last 15 years of EP, several common misconceptions have emerged (Table 1). These misconceptions seem to fall into 2 categories: those that mistake EP for conventional CBT or aspects of it, and those that misunderstand the material itself. For example, EP has been mistaken for advice to move despite pain, or advice on how to manage the demands of daily life around a pain problem, both of which are important in most CBT programs for chronic pain,³⁰ but neither of which captures EP. Pain programs also often present the gate control theory or the idea that the cause of pain has shifted from the tissues to a pain signal-generating disease process in their spinal cord and brain,³⁰ neither of which is EP. Perhaps most tragically, EP has been mistaken for advice that chronic pain is not real pain but is instead all in the head. We contend that such unfortunate misconceptions might reflect both a lack of skillful intent in targeting the conceptual shift, and a perspective of the beholder that is firmly grounded in a structural pathology model of pain and the erroneous assumption that pain and nociception are one and the same. This is important because the conceptual shifts that are targeted by EP in patients have at times not yet occurred in the clinicians who treat them or are considered beyond the capacity of patients to understand.²⁹ We do not make these contentions lightly; we expect them to meet resistance from several corners, not least those who rely only on finding the peripheral pain driver and those who see that approach as futile but nonetheless conceptualize the problem as one in which the pain driver has moved into the spinal cord or brain. The implications of both versions of the structural pathology model (the peripheral and central

Table 1. Suggested Common Misconceptions and the Accurate Conceptions About EP

MISCONCEPTION	ACCURATE CONCEPTION
EP is teaching people how to manage their pain, similar to, for example, coping skills training, relaxation training, goal setting, or problem solving skills	EP is teaching people about the biological processes underpinning pain. EP does not include instruction on strategies or skills with which to reduce the impact of pain on one's life. EP draws on instructional design and multimedia principles to present pain biology information
EP is advising people to move despite their pain	EP is teaching people that pain can be overprotective
EP is advising people that pain messages are turned up and down at the spinal cord	EP is teaching people that danger messages are turned up and down at the spinal cord
EP is describing the pain gate control theory	EP is teaching people that the brain can turn down the danger message at the spinal cord
EP is explaining that central sensitization is causing their pain, and there are no known cures for central sensitization	EP is teaching people that their danger transmission system can become very sensitive, which can lead to more danger messages, but it is always the brain that decides whether or not to produce pain
EP is reassuring people that the pain they perceive to be there is not really there at all	EP is reassuring people that their pain is completely real even although the tissue may not be in danger
EP is a discrete intervention that can be delivered effectively alongside treatments based on a structural pathology model	EP can be effectively provided only under a biopsychosocial paradigm, which integrates treatment of peripheral and central nociceptive drivers
EP relates only to chronic pain, not acute pain	EP relates to pain
EP throws out biology and biomedical models to focus only on the psychosocial	EP is a pragmatic application of the biopsychosocial model of pain, which integrates treatment of peripheral and central nociceptive drivers alongside other contributions to pain

versions) are clear; if pain and tissue damage or disease are considered analogous, the suggestion that a pain does not measure this tissue damage or disease implies necessarily that pain is not really pain. The conundrum that faces anyone who holds onto the idea that pain and nociception are the same is clear. That this perspective still persists suggests that it is not just the lay community who are naive to modern thought on the biology of pain (such naivety is understandable) but that this naivety extends to at least some of the clinical and scientific communities, who, one might provocatively suggest, should know better by now.

Behavioral Evidence and Biological Plausibility

As mentioned earlier, a core principle of EP is that pain is a truly biopsychosocial phenomenon. Considering this issue from a Bayesian perspective, pain can be considered a perceptual inference, whereby the experience is considered an output into consciousness, which reflects the best-guess estimate of what will be an advantageous response. One might predict that when it comes to bodily protection, the tendency is often to err on the side of protection. Considering perception, therefore, as the construction of "what is most likely to be reality,"⁵ one can readily appreciate that credible evidence of danger should modulate the perception regardless of the modality of that evidence, be it nociceptive, somatosensory, somatic, visual, auditory, cognitive, or social. In this sense, the working hypothesis of the mechanism of EP is that it changes the threat value that is associated with a given suite of sensory inputs, such that the construction of "what is most likely to be reality" is shifted from that which requires protection to that which does not. That is, the threat value shifts from that which results in pain to that which does not.

How effective, then, is a cognitively mediated shift in threat value in modifying the perceptual response to a given sensory stimulus? There is clearly a large body of anecdotes that suggest potentially powerful effects on pain of shifting the threat value of a situation or stimulus. One need look no further than religious or cultural ceremonies in which highly nociceptive events are not painful (see Moerman¹⁹ for extensive review), or sexual experiences, in which nociceptive events become pleasurable. However, there is also a growing body of experimental data that support the idea as well. For example, when a very cold noxious stimulus is applied to the skin of healthy volunteers, it hurts more if accompanied by advice that the stimulus being applied is hot.¹ Moreover, even without explicit instruction, a cold noxious stimulus hurts more if it is simply accompanied by a red visual cue, which implies heat, than if it is accompanied by an otherwise identical light blue cue, which implies cool.²⁷ Similarly, when healthy volunteers received standardized noxious laser stimuli to their foot, the prior (and deceitful) advice that a particular stimulus site was thin skinned and vulnerable

resulted in a higher likelihood of pain (allodynia) and more intense pain (hyperalgesia) to fixed stimuli than advice to the contrary, even although skin thickness did not really vary at all.⁴⁶ The functional neurology of such immediate effects has been investigated, and several cortical areas, for example anterior insular cortex, and their connections to the periaqueductal gray^{34,46} have been implicated in mediating the effect. However, one might expect that a range of brain areas are involved in the cognitive modulation of pain, with the exact areas dependent on the individual and the type of modulation. Exhaustive review is beyond the scope of this article, but suffice it to suggest that what evidence there is from neuroimaging studies clearly points to the biological plausibility of cognitive modulation of pain.

At this stage, brain imaging data that elucidate the effects of EP are lacking; there are clear methodological and conceptual barriers to capturing such complex mechanisms in terms of their underlying neural substrate. However, there is emerging behavioral evidence that reconceptualization of the underlying biology of pain is associated with real-time modulatory effects such as those described earlier. For example, when 121 people with chronic back pain participated in either an EP-based or a back school-based education session, those in the EP group demonstrated an immediate increase in pain-free straight leg raise, whereas those in the back school group did not.²⁵ The curriculum of back schools (spinal physiology, anatomy, and ergonomics) is clearly different from that of EP. In a further example of real-time modulatory effects of EP, when 30 patients with fibromyalgia who had a deficient inhibitory noxious control response to the cold pressor task were allocated to EP or a self-management education (addressing behavioral response to pain rather than the biology of pain) control condition, those in the EP group, but not the control group, showed normalized endogenous inhibitory control afterward.⁴³ We contend that although the precise biological mechanisms and locations within the nervous system, by and at which EP modulates pain, remain to be discovered, there is compelling evidence that the effect itself is biologically plausible.

Clinical Effects of EP

The bottom line, when it comes to any intervention, is efficacy. Several randomized controlled trials (RCTs) have investigated the efficacy of EP in various clinical conditions, including chronic low back pain (LBP),^{22,23,25,28,33,36} lumbar radiculopathy,¹⁶ fibromyalgia,^{42,43} chronic fatigue syndrome,¹⁷ whiplash,³² and general chronic pain.¹³ Systematic reviews have drawn similar, although not identical, conclusions. One¹⁵ concluded that the evidence for EP in decreasing pain, increasing physical performance, decreasing perceived disability, and decreasing catastrophization was compelling. However, there are important caveats here: the included data came from 8 studies and 401 patients (including patients with chronic LBP, chronic fatigue

syndrome, widespread pain, and chronic whiplash-associated disorders); the heterogeneity in outcome measures and in the frequency and duration of the EP sessions restricted meta-analysis.¹⁵ Other reviews were more measured; for chronic LBP specifically, a Cochrane review in 2008¹¹ and more recently a meta-analysis of 63 patients with chronic LBP⁶ concluded that there was only low-level evidence for EP in improving short-term pain and function.

When considered in light of the wider field of chronic pain, the evidence base is clearly growing quickly, but it is not yet mature: there are diverse delivery methods; EP is often investigated in isolation rather than as part of a multimodal approach, as it is clinically intended; similar approaches have different names, and engagement of the treating team requires the clinicians themselves to have certain competencies, first of which is a personal reconceptualization of modern pain biology, a requirement that is not automatically satisfied.²⁴ We have systematically searched the available literature (see [Supplemental Appendix 1](#) for search strategy and brief results) since the most recent review,¹⁵ and the evidence base is clearly expanding. There have been a further 5 RCTs, all with different approaches. For example, one compared an EP-based storybook²⁶ with a control book,³⁵ both modified to be similar in look, feel, and length, presented to a group of patients with chronic pain.¹³ In a randomized single-group crossover design, only the EP group showed clinically important shifts in catastrophizing and pain-related knowledge. Another RCT³³ combined EP with aquatic exercise and compared it with aquatic exercise alone, finding favorable outcomes, including decreased pain, in the combined therapy group.

A pair of RCTs undertaken by one research group in people with fibromyalgia^{42,43} found that face-to-face delivery of EP was associated with pain and disability reduction but that a version using only written material was not. This result contrasts with our experience using an EP-based storybook,¹³ which suggests that the delivery of written material is important. In our trial, people were more likely to read the book of stories and metaphors used to explain fundamental concepts in pain biology than they were to read an equivalent-looking book containing behavioral advice. In a pragmatic RCT targeting preoperative intervention, EP, including face-to-face instruction and a booklet, was superior to usual care on self-reported attitudes to recovery but not on postsurgical pain or disability.¹⁶

The limitations highlighted in earlier systematic reviews are still relevant to the new body of literature: most studies are small, and it is clearly not possible to blind clinicians to what it is that they are delivering. Critically, the state of the evidence does not suggest EP alone as a viable intervention to induce long-lasting improvements in pain and disability. However, this is not the intent of EP. Rather, EP exploits a range of strategies to present a compelling case for a biology of pain that underpins management according to a

biopsychosocial approach, including but not limited to multimodal CBT-based reactivation. The most parsimonious interpretation of the wider body of evidence concerning EP appears to be that as a stand-alone treatment for a wide range of chronic pain states, EP changes knowledge of pain biology, improves participation in subsequent biopsychosocially based rehabilitation, and decreases catastrophizing and pain-related and activity-related fear. When combined with other treatments that are also consistent with a biopsychosocial framework, EP seems to offer clinically important improvements in pain and disability.

Conclusions and Future Directions

EP is a biologically plausible approach to treatment that seems to offer clear benefits when tested in isolation or as part of a wider rehabilitation program. Delivering EP both requires and targets a shift in one's understanding of pain, from that of a biomedical or structural pathology paradigm to that of a truly biopsychosocial paradigm. Larger and more pragmatic clinical trials are clearly required, and the possibility of enhancing the effects of EP by combining it with other promising interventions is enticing. For example, exploration of the combined effect of EP and brain-training strategies, or EP combined with interventions that promote neuroplasticity (via pharmacological, stimulation, or endogenous means, eg, hypnosis, exercise, or meditation), is worth pursuing. Future directions should also explore the notion of individual and group curricula; the term itself is a call for quality in what is taught, how it is taught, competencies of the teacher, management of outliers, and measurement. We suspect that EP may have an important role to play to prevent chronicity after an acute episode of pain.⁴⁰ A recent meta-analysis showing that targeted reassurance is an important management strategy in management of acute back pain³⁹ raises the distinct possibility that an EP-enhanced optimized reassurance may offer even better gains.

As Patrick Wall declared to a packed house at the 1999 World Congress on Pain in Vienna, Austria: "Considering pain not as a marker of injury but as a human experience should not be an alternative or niche therapy, but the very thing that unites us." We wholeheartedly and unreservedly endorse his view and suggest 2 implications of his declaration: that we should continue to strive toward understanding this experience of pain, in all its complexity, and that we should explain what we know to those in pain. The manner in which we seek to explain pain should be as grounded in scientific process and discovery as the material itself.

Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.jpain.2015.05.005>.

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