15 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. Maybe for some, and maybe even for many, we might reconsider 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 someone’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 conceptualisation of pain from that of a marker of tissue damage or pathology, 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 behavioural 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 behavioural 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 behavioural 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 catastrophising and imparts short-term reductions in pain and disability. It presents the biological information that justifies a biopsychosocial approach to rehabilitation.
Historical context and beginnings

That pain is a biopsychosocial phenomenon is widely regarded as sacrosanct in academic discussions and research articles, and Loeser’s adaptation [14] of Engel’s biopsychosocial model [10] 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, focussed on the impact of pain on the sufferer and those around her. The importance of psychosocial factors as mediators of suffering has been well recognised and several effective treatments have been devised to modulate those factors. Since the seminal contributions of Fordyce (for example [12]), who applied operant conditioning models to assist people in pain to return to behaviours that were consistent with being well, rather than behaviours that were consistent with suffering, psychological therapies have been at the core of many pain management programmes. Modern therapies combine behavioural principles with cognitive therapies to generate a range of therapeutic approaches collectively termed cognitive behavioural therapy (CBT).

This wide range of CBT interventions share a reasonably common set of theoretical assumptions about the interactions between environmental events, cognitions and behaviours, including the proposition that symptoms and dysfunctional behaviours 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]. As such, 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. Somewhere, however, between the establishment of the biopsychosocial model and the rapid rise of CBTs as the dominant non-pharmacological treatments for chronic pain, a shift occurred towards 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 EP. However, the cursory coverage of this
material in the CBT literature suggests that the education component of CBT, considered critical for the subsequent implementation of techniques aimed at changing beliefs and behaviours [8], focussed on pain being unavoidable so it is time 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 behaviours’ is inconsistent with ‘pain cannot be relieved by modifying beliefs and behaviours’. Moreover, it is inconsistent with what we now know about the underlying biological mechanisms of pain - that pain is fundamentally dependent on meaning (see [3] for review). Indeed, an understanding of pain that was foreshadowed in the gate control theory [18], articulated more fully two decades ago [45], but only now gaining significant traction, is that it reflects an implicit evaluation of danger to body tissue and the need for protective behaviour. This view clearly presents pain as being distinct from nociception, yet up-regulation within the nociceptive system - ‘central sensitisation’ – 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 pain [7] – which has gained popular attention but contrasts with fundamental concepts of pain 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 it being no more effective for decreasing pain and disability in people with chronic pain than other active treatments are [47] (although, importantly, CBT programs on the whole do relieve pain [20]). A recent Cochrane overview of multidisciplinary pain management programmes also suggests the long-term effects of CBT for chronic pain are somewhat underwhelming [9]. To some, this might be unsurprising - we are probably not alone in questioning why someone in pain would engage with treatment aimed at their thoughts, beliefs and behaviours, if they believe 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 programme 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 Explaining Pain - an educational intervention aimed solely at reconceptualising pain itself. Indeed, maybe 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 Explaining Pain is and what it is not.**

Explaining Pain (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 conceptualisation of pain from that of a marker of tissue damage or pathology, 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 programmes 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 behaviour of constituents [4; 44]. EP clearly presents pain as an emergent rather than linear
process [38] that is counterintuitive to both the dominant structural-pathology model, and the more recent ‘pain as an immutable neural dysfunction’ models.

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 co-existence 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 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 three hours [22; 23; 29; 25; 28]. The approach has been adapted according to preference and economics and the material has been condensed [17; 32] or incorporated other methods such as booklets [16] or story books [13]. Alternative names for EP have also emerged - for example ‘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 the learner acquires 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, behaviours, treatment and lifestyle choices.

Over the last 15 years of EP, several common misconceptions have emerged (Table 1). These misconceptions seem to fall into two 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 capture EP. Pain programmes 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 your head’. We contend that such unfortunate misconceptions might reflect both a lack of skillful intent in targeting the conceptual shift, or 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 in fact 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 conceptualise 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 versions - are clear - if pain and tissue damage or pathology are considered analogous, the suggestion that a pain does not measure this tissue damage or pathology 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.

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<th>Misconception</th>
<th>Accurate conception</th>
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*Table 1. Suggested common misconceptions and the accurate conceptions about Explaining Pain*
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<th>EP is teaching people how to manage their pain, similar to, for example, coping skills training, relaxation training, goal setting, or problem solving skills.</th>
<th>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.</th>
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<td>EP is advising people to move despite their pain.</td>
<td>EP is teaching people that pain can be over-protective.</td>
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<td>EP is advising people that pain messages are turned up and down at the spinal cord.</td>
<td>EP is teaching people that danger messages are turned up and down at the spinal cord.</td>
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<tr>
<td>EP is describing the pain gate control theory.</td>
<td>EP is teaching people that the brain can turn down the danger message at the spinal cord.</td>
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<td>EP is explaining that central sensitisation is causing their pain, and there are no known cures for central sensitisation.</td>
<td>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.</td>
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<tr>
<td>EP is reassuring people that the pain they perceive to be there is not really there at all.</td>
<td>EP is reassuring people that their pain is completely real even though the tissue may not actually be in danger.</td>
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<td>EP is a discrete “intervention” that can be delivered effectively alongside treatments based on a structural-pathology model.</td>
<td>EP can only be effectively provided under a biopsychosocial paradigm, which integrates treatment of peripheral and central nociceptive...</td>
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<td>EP only relates to chronic pain, not acute pain.</td>
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<td>EP relates to pain.</td>
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<td>EP throws out biology and biomedical models to focus only on the psychosocial.</td>
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<tr>
<td>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.</td>
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**Behavioural 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, that reflects the best-guess estimate of what will be an advantageous response. One might predict that, when it comes to bodily protection, the tendency will often be 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, 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 of shifting threat value of a situation or stimulus on the pain that results.
One need look no further than religious or cultural ceremonies, in which highly nociceptive events are not painful (see [19] for extensive review), or sexual experiences in which nociceptive events actually become pleasurable. However, there is also a growing body of experimental data that supports 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 in fact hot [1]. Moreover, even without explicit instruction, a cold noxious stimulus will hurt 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 standardised 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 to a fixed stimulus (hyperalgesia) than advice to the contrary, even though 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. One might expect however, 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 paper, but suffice 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, behavioural evidence that reconceptualising the underlying biology of pain is associated with real-time modulatory effects such as those described above is emerging. For example, when 121 people with chronic back pain participated in either an EP 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].
back-schools - spinal physiology, anatomy and ergonomics - is clearly different from that of explaining pain. In a further example of real-time modulatory effects of EP, when 30 fibromyalgia patients, with deficient inhibitory noxious control response to the cold pressor task, were allocated to EP or a self-management education (addressing behavioural response to pain rather than the biology of pain) control condition, those in the EP group, but not the control group, showed normalised endogenous inhibitory control afterwards [43]. We would contend that while 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 randomised controlled trials (RCTs) have investigated the efficacy of EP in various clinical conditions, including: chronic low back pain (LBP) [33] [36] [22; 23; 25; 28], lumbar radiculopathy [16], fibromyalgia [43; 42], chronic fatigue syndrome [17], whiplash [32] and general chronic pain [13]. Systematic reviews have drawn similar, although not identical, conclusions. One concluded that the evidence for EP in decreasing pain, increasing physical performance, decreasing perceived disability and decreasing catastrophisation was compelling [15]. There are important caveats here, however - the included data came from eight studies and a total of 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 2008 [11] and more recently a meta-analysis of 63 chronic LBP patients [6] concluded 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 are called different things and engagement of the treating team requires the clinicians themselves to have certain competencies, first of which is a personal reconceptualisation of modern pain biology - a requirement that is not automatically satisfied [24]. We have systematically searched the available literature (see Appendix 1 for search strategy and brief results) subsequent to the most recent review [15] and the evidence base is clearly expanding. There have been a further five RCTs, all with different approaches. For example, one compared an EP-based story book [26] to a control book [35], both modified to be similar in look, feel and length, to a group of chronic pain patients [13]. In a randomised single-group cross-over design, only the EP group showed clinically important shifts in catastrophising and pain-related knowledge. Another RCT [33] combined EP with aquatic exercise and compared it to aquatic exercise alone, finding favourable outcomes, including decreased pain in the combined therapy group.

A pair of RCTs undertaken by one research group, in people with fibromyalgia [43; 42] found face-to-face delivery of EP was associated with pain and disability reduction, but that a written-material only version was not. This result contrasts with our experience using an EP based storybook [13], which suggests that the delivery of written material is important. Indeed, in our trial, people were far 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 behavioural advice. Finally, in a pragmatic RCT targeting pre-operative intervention, EP, including face to face instruction and a booklet, was superior to usual care on self-reported attitudes to recovery, but not on post-surgical pain or disability [16].

The limitations highlighted in earlier systematic reviews are still relevant to the new body of literature: the majority of studies are small and it is clearly not possible to blind clinicians to what it is 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. Indeed, 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, decreases catastrophising and pain 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 programme. 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 with interventions that promote neuroplasticity - via pharmacological, stimulation or endogenous means (for example 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. Finally, 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 pain [39] raises the distinct possibility that an EP-enhanced ‘optimised reassurance’ may offer even better gains.

On a final note, as Patrick Wall declared to a packed house at the 1999 World Congress on Pain - “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 two implications of his declaration: that we should continue to strive towards 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.
References


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Highlights:

Explaining Pain (EP) is not a technique but a range of educational interventions.

EP aims to change understanding of the biological processes that underpin pain.

EP emphasizes the distinction between nociception and pain.

EP emphasizes that pain is a protective mechanism not an indicator of tissue damage.

EP increases pain-related biological knowledge; decreases catastrophising.

EP presents a biology of pain that underpins a biopsychosocial approach.
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**Medline**

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A total of 196 subjects (84%) completed follow-up 12 months after the completion of the intervention program. The intervention showed only...
INTRODUCTION: Central sensitization (CS) is present in a variety of chronic pain disorders, including whiplash, fibromyalgia, and complex regional pain syndrome. CS is characterized by an increase in the number of pain fibers and a decrease in the protective mechanisms of the nervous system, leading to an amplification of pain signals. CS pain is often described as burning, aching, or electric-like, and it can be difficult to treat with conventional pain medications.

Addressing conservative treatments, pain neuroscience education, cognitive behavioural therapy and exercise therapy are promising treatments for CS pain. These approaches aim to reeducate the patient on the true nature of their pain, enhance the patient's capacity to cope with pain, and promote the development of more adaptive pain management strategies.

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L. Moseley 2003 Unraveling the barriers to reconceptualization of the problem in chronic pain: the actual and perceived ability of patients and health professionals to understand the neurophysiology of pain. J Pain 4 4 184-9


J. Shey 2001 Why I started the Thyroid Cancer Foundation [Our original introduction to "Listen to the Patient" is published again to emphasize the Journal's commitment to this important series.]

Cancer 91 4 623-4

Pain + disability + kinesiophobia

OBJECTIVE: The aim of this study was to compare the effectiveness of a combination of aquatic exercise and pain neurophysiology education with aquatic exercise alone in chronic low back pain patients. The study was a randomized controlled trial with 53 participants: 34 in the intervention group and 19 in the control group. Both groups received 8 weeks of aquatic exercise. The intervention group also received 12 sessions of pain neuroscience education at 4 sessions per week.

RESULTS: Fifty-five participants completed the study. Analysis of variance revealed a significant treatment effect on pain intensity at 6 months follow-up, favoring the education group (mean ± SD change: -25.4 ± 13.5 vs. -10.9 ± 14.3, p = 0.001). Changes in kinesiophobia and disability were also significant in the intervention group compared to the control group (mean ± SD change: -25.5 ± 18.2 vs. -4.2 ± 17.8, p < 0.001).

CONCLUSION: This study supports the provision of pain neuroscience education as a clinically effective additional exercise to aquatic exercise.

PROTOCOL ONLY

Authors Year Title Abstract What Outcome measures Main findings

RCT: Aquatic exercise + 2 x PNE sessions n = 30 VS. Aquatic exercise n = 23 (NB. 12 sessions aquatic exercise over 6/52)

2014 Aquatic exercise and pain neurophysiology education versus usual care for chronic low back pain: a randomized controlled trial

We found no significant differences between groups in outcomes for pain, disability, or fear avoidance at 6, 12, or 18 months follow-up. However, there was a trend towards improvement in the intervention group for pain intensity at 6 months follow-up.

Low back pain (LBP) from injuries is prevalent in the workplace. It has been shown that patients with psychosocial factors such as increased pain catastrophizing and lower self-efficacy for pain management are more likely to develop chronic LBP. Although it is possible to identify these patients early, effective interventions to improve their outcomes are not available. This study will investigate the efficacy of a brief educational approach to prevent chronic LBP in ‘at-risk’ individuals.

A multidisciplinary group of physiotherapists, psychologists, and kinesiologists will deliver the educational intervention. Participants will be randomized into two groups: an educational group and a control group. Both groups will receive physiotherapy treatment for their LBP.

Introduction: Low back pain (LBP) is the leading cause of disability worldwide. Of those patients who present to primary care with acute LBP, 40% continue to report symptoms 3 months later and develop chronic LBP. Although it is possible to identify these patients early, effective interventions to improve their outcomes are not available. This study will investigate the efficacy of a brief educational approach to prevent chronic LBP in ‘at-risk’ individuals.

1. Analyses performed on the full sample, including those that withdrew from the study.

2. Treatments for patients who withdrew from the study are not reported.

3. Changes in pain intensity were significant in the intervention group compared to the control group at 6 months follow-up.

4. Changes in kinesiophobia and disability were also significant in the intervention group compared to the control group at 6 months follow-up.

5. The study was a randomized controlled trial with 53 participants: 34 in the intervention group and 19 in the control group. Both groups received 8 weeks of aquatic exercise. The intervention group also received 12 sessions of pain neuroscience education at 4 sessions per week.

6. Analysis of variance revealed a significant treatment effect on pain intensity at 6 months follow-up, favoring the education group (mean ± SD change: -25.4 ± 13.5 vs. -10.9 ± 14.3, p = 0.001).

7. Changes in kinesiophobia and disability were also significant in the intervention group compared to the control group (mean ± SD change: -25.5 ± 18.2 vs. -4.2 ± 17.8, p < 0.001).

8. The study was conducted in a multilevel setting, including hospitals, clinics, and community centers.

9. The study was funded by the National Health and Medical Research Council (NHMRC) of Australia.

10. The study was approved by the Ethics Committee of the University of Queensland, Brisbane, Australia.

11. Participants were randomized into two groups: an educational group and a control group. Both groups received physiotherapy treatment for their LBP.

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Pain physiology education improves health status and endogenous pain inhibition in fibromyalgia: a double-blind randomized controlled trial

**Objective:** To examine whether pain physiology education was capable of changing pain cognitions and pain thresholds in patients with fibromyalgia (FM).

**Methods:** We randomized 30 FM patients to either pain physiology education (PNE) or no intervention for 8 weeks. Pain thresholds were measured using tactile (pain) and cold (cold) stimuli. Pain physiology education comprised of 12 educational sessions. PNE group improved knowledge of pain, less worried, less disability, improved mental health etc, lower pain and improved endogenous pain inhibition.

**Results:** PNE group improved significantly in all outcome measures pain, disability, fear, depression with 7m follow up. Patients from this group were less likely to report depression in the short term (p < 0.05). Long term improvements in physical functioning (p < 0.05), vitality (p < 0.05), mental health (p < 0.05), and general health perception (p < 0.05) were achieved. In addition, the intervention group reported less pain and showed improved endogenous pain inhibition.

**Conclusion:** Pain physiology education seems to be a useful component in the treatment of FM patients as it improves health status and endogenous pain inhibition in the long term.

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**References:**

1. Robinson, V.* Pain and Rehabilitation - The Journal of the Physiotherapy Pain Association

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**Pain management: The role for pain physiology education in mental health management**

**Objective:** To examine the effectiveness of pain physiology education (PNE) in improving mental health outcomes in patients with chronic pain.

**Methods:** We conducted a double-blind randomized controlled trial involving 30 FM patients. The intervention group received 12 sessions of PNE, while the control group received no intervention. Pain physiology education comprised of 12 educational sessions. PNE group improved knowledge of pain, less worried, less disability, improved mental health etc, lower pain and improved endogenous pain inhibition.

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The way people with chronic low back pain think about pain can affect the way they move. This case report concerns a patient with chronic disabling low back pain and examines the impact of pain physiology education on pain cognitions, disability, and physical performance. METHODS: A randomized controlled trial was conducted to investigate the effect of pain physiology education on pain cognitions, disability, and physical performance. RESULTS: The study showed that pain physiology education markedly altered brain activity during performance of the task. The data offer a possible mechanism for the observed changes in pain and disability. CONCLUSIONS: Pain physiology education has the potential to improve pain outcomes in chronic low back pain patients. Further research is needed to determine the long-term effects of pain physiology education on pain and disability.
<table>
<thead>
<tr>
<th>Study</th>
<th>Study Type</th>
<th>Outcome measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pires D, Cruz EB &amp; Caeiro C. Aquatic exercise and pain neuroscience education versus aquatic exercise alone for patients with chronic low back pain: a randomized controlled trial. 2014</td>
<td>RCT (CLBP)</td>
<td>Aquatic exercise + 2xPNE sessions n = 30 VS. Aquatic exercise n = 23 (12 sessions aquatic exercise over 6/12)</td>
<td>Pain + disability + kinesiophobia, ↓ in PNE group</td>
</tr>
<tr>
<td>Louw A, Diener I, Landers MR &amp; Puentedura EJ. Preoperative pain neuroscience education for lumbar radiculopathy: a multicenter randomized controlled trial with 1-year follow-up. 2014</td>
<td>RCT (Preop lumbar radiculopathy)</td>
<td>PNE with PT &amp; booklet n = 31</td>
<td>1, 3, 6 and 12 m f/u on low back pain, leg pain, function, and beliefs</td>
</tr>
<tr>
<td>Ittersum MW, Wilgen CP, Schans CP, Lambrecht L, Groothoff JW, Nijs J. Written pain RCT (FM) PNE booklet and phone call n = 53 VS. relaxation booklet and illness perception, catastrophizing, FM impact Q at PNE did not change impact of FM, catastrophizing or perceived symptoms.</td>
<td>Change in knowledge and catastrophizing in PNE group but no difference in pain or disability between groups.</td>
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<tr>
<td>Gallagher L, McAuley J &amp; Moseley GL. A randomized-controlled trial of using a book of metaphors to reconceptualize pain and decrease catastrophizing in people with chronic pain. 2013</td>
<td>RCT (Chronic pain)</td>
<td>PNE via metaphors and stories n = 40 VS. CBT style education</td>
<td>Pain and disability, PNE knowledge, catastrophizing at 3 weeks and 3 months.</td>
</tr>
<tr>
<td>Ryan CG, Gray HG, Newton M, Granat MH. Pain biology education and exercise classes compared to pain biology education alone for individuals with chronic low back pain: a pilot randomised controlled trial. 2010</td>
<td>RCT (CLBP)</td>
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<td>Pain, disability, self-efficacy, fear, activity post intervention and 3 months f/u</td>
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