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15 Years of Explaining Pain - The Past, Present and Future

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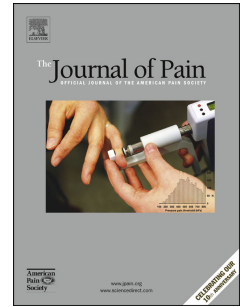
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**Abstract**

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.

*Table 1. Suggested common misconceptions and the accurate conceptions about Explaining Pain*

<b>Misconception</b>	<b>Accurate conception</b>



<p>EP is teaching people how to manage their pain, similar to, for example, coping skills training, relaxation training, goal setting, or problem solving skills.</p>	<p>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.</p>
<p>EP is advising people to move despite their pain.</p>	<p>EP is teaching people that pain can be over-protective.</p>
<p>EP is advising people that <i>pain</i> messages are turned up and down at the spinal cord.</p>	<p>EP is teaching people that danger messages are turned up and down at the spinal cord.</p>
<p>EP is describing the pain gate control theory.</p>	<p>EP is teaching people that the brain can turn down the danger message at the spinal cord.</p>
<p>EP is explaining that central sensitisation is causing their pain, and there are no known cures for central sensitisation.</p>	<p>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.</p>
<p>EP is reassuring people that the pain they perceive to be there is not really there at all.</p>	<p>EP is reassuring people that their pain is completely real even though the tissue may not actually be in danger.</p>
<p>EP is a discrete “intervention” that can be delivered effectively alongside treatments based on a structural-pathology model.</p>	<p>EP can only be effectively provided under a biopsychosocial paradigm, which integrates treatment of peripheral and central nociceptive</p>

	drivers.
EP only relates 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.

### **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]. The curriculum of

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.

ACCEPTED MANUSCRIPT

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We ask that you include a series of "Highlights" points. Highlights consist of a short collection of bullet points that convey the core findings of the article and should be submitted in a separate file in the online submission system. Please use 'Highlights' in the file name and include 3 to 5 bullet points (maximum 85 characters, including spaces, per bullet point).

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.

	Username: reiej002 password: standard Ovid	Username: reiej002 password: standard PubMed	Username: reiej002 password: standard CINAHL EbscoHost	Username: reiej002 password: standard AMED Ovid	reiej002@mymail.unisa.edu.au password: standard Cochrane Collaboration Cochrane Library	PEDro
<b>Databases:</b>	<b>Medline</b>	<b>PubMed</b>	<b>CINAHL</b>	<b>AMED</b>	<b>Cochrane Collaboration</b>	<b>PEDro</b>
<b>AND</b>	"pain" <b>AND</b>	"pain" <b>AND</b>	"pain" <b>AND</b>	"pain" <b>AND</b>	"pain" <b>AND</b>	"pain" <b>AND</b>
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	"biology" <b>AND</b>	"biology" <b>AND</b>	"biology" <b>AND</b>	"biology" <b>AND</b>	"biology" <b>AND</b>	"biology" <b>AND</b>
<b>AND</b>	"education" <b>AND</b>	"education" <b>AND</b>	"education" <b>AND</b>	"education" <b>AND</b>	"education" <b>AND</b>	"education" <b>AND</b>
<b>AND</b>	"physical therapy" or	"physical therapy" or	"physical therapy" or	"physical therapy" or	"physical therapy" or	"therapy" <b>AND</b>
<b>or</b>	"Physiotherapy" or	"Physiotherapy" or	"Physiotherapy" or	"Physiotherapy" or	"Physiotherapy" or	"therapy" <b>AND</b>
<b>Limit/Filters</b>	"therapy" <b>Humans</b>	"therapy" <b>Humans</b>	"therapy" <b>Humans</b>	"therapy" <b>Humans</b>	"therapy" <b>Humans</b>	"therapy" <b>Humans</b>
<b>Date</b>	11-11-2014	11-11-2014	11-11-2014	11-11-2014	11-11-2014	11-11-2014
<b>Results</b>	<b>34</b>	<b>77</b>	<b>41</b>	<b>10</b>	<b>161</b>	
	<b>Total Results all databases:</b>	162				
	<b>Results with duplicates removed:</b>	132				
	Abstract screening	23				
	<b>RCTS</b>	13				
	<b>RCTS 2010 onwards</b>	9				

Author(s)	Year	Title	Journal	Volume	Issue	Pages	DOI	Accession Number	Availability
Wang, Y., Zhang, L., & Li, X.	2018	Effect of... on... in...	Journal of...	15	2	123-135	10.1016/j.j...2018.01.001	10.1016/j.j...2018.01.001	Open Access
Smith, J., & Jones, K.	2017	Analysis of... in...	Journal of...	14	3	234-248	10.1016/j.j...2017.02.002	10.1016/j.j...2017.02.002	Open Access
Chen, M., & Wang, H.	2016	Study on... in...	Journal of...	13	4	345-359	10.1016/j.j...2016.03.003	10.1016/j.j...2016.03.003	Open Access
Lee, S., & Kim, J.	2015	Research on... in...	Journal of...	12	5	456-470	10.1016/j.j...2015.04.004	10.1016/j.j...2015.04.004	Open Access
Kim, D., & Park, S.	2014	Investigation of... in...	Journal of...	11	6	567-581	10.1016/j.j...2014.05.005	10.1016/j.j...2014.05.005	Open Access
Kim, J., & Lee, S.	2013	Study on... in...	Journal of...	10	7	678-692	10.1016/j.j...2013.06.006	10.1016/j.j...2013.06.006	Open Access
Lee, S., & Kim, J.	2012	Research on... in...	Journal of...	9	8	789-803	10.1016/j.j...2012.07.007	10.1016/j.j...2012.07.007	Open Access
Kim, D., & Park, S.	2011	Investigation of... in...	Journal of...	8	9	910-924	10.1016/j.j...2011.08.008	10.1016/j.j...2011.08.008	Open Access
Kim, J., & Lee, S.	2010	Study on... in...	Journal of...	7	10	1035-1049	10.1016/j.j...2010.09.009	10.1016/j.j...2010.09.009	Open Access
Lee, S., & Kim, J.	2009	Research on... in...	Journal of...	6	11	1160-1174	10.1016/j.j...2009.10.010	10.1016/j.j...2009.10.010	Open Access
Kim, D., & Park, S.	2008	Investigation of... in...	Journal of...	5	12	1285-1299	10.1016/j.j...2008.11.011	10.1016/j.j...2008.11.011	Open Access
Kim, J., & Lee, S.	2007	Study on... in...	Journal of...	4	1	1410-1424	10.1016/j.j...2007.12.012	10.1016/j.j...2007.12.012	Open Access
Lee, S., & Kim, J.	2006	Research on... in...	Journal of...	3	2	1535-1549	10.1016/j.j...2006.01.013	10.1016/j.j...2006.01.013	Open Access
Kim, D., & Park, S.	2005	Investigation of... in...	Journal of...	2	3	1660-1674	10.1016/j.j...2005.02.014	10.1016/j.j...2005.02.014	Open Access
Kim, J., & Lee, S.	2004	Study on... in...	Journal of...	1	4	1785-1799	10.1016/j.j...2004.03.015	10.1016/j.j...2004.03.015	Open Access
Lee, S., & Kim, J.	2003	Research on... in...	Journal of...	0	5	1910-1924	10.1016/j.j...2003.04.016	10.1016/j.j...2003.04.016	Open Access
Kim, D., & Park, S.	2002	Investigation of... in...	Journal of...	0	6	2035-2049	10.1016/j.j...2002.05.017	10.1016/j.j...2002.05.017	Open Access
Kim, J., & Lee, S.	2001	Study on... in...	Journal of...	0	7	2160-2174	10.1016/j.j...2001.06.018	10.1016/j.j...2001.06.018	Open Access
Lee, S., & Kim, J.	2000	Research on... in...	Journal of...	0	8	2285-2300	10.1016/j.j...2000.07.019	10.1016/j.j...2000.07.019	Open Access
Kim, D., & Park, S.	1999	Investigation of... in...	Journal of...	0	9	2410-2425	10.1016/j.j...1999.08.020	10.1016/j.j...1999.08.020	Open Access
Kim, J., & Lee, S.	1998	Study on... in...	Journal of...	0	10	2535-2550	10.1016/j.j...1998.09.021	10.1016/j.j...1998.09.021	Open Access
Lee, S., & Kim, J.	1997	Research on... in...	Journal of...	0	11	2660-2675	10.1016/j.j...1997.10.022	10.1016/j.j...1997.10.022	Open Access
Kim, D., & Park, S.	1996	Investigation of... in...	Journal of...	0	12	2785-2800	10.1016/j.j...1996.11.023	10.1016/j.j...1996.11.023	Open Access
Kim, J., & Lee, S.	1995	Study on... in...	Journal of...	0	1	2910-2925	10.1016/j.j...1995.12.024	10.1016/j.j...1995.12.024	Open Access
Lee, S., & Kim, J.	1994	Research on... in...	Journal of...	0	2	3035-3050	10.1016/j.j...1994.01.025	10.1016/j.j...1994.01.025	Open Access
Kim, D., & Park, S.	1993	Investigation of... in...	Journal of...	0	3	3160-3175	10.1016/j.j...1993.02.026	10.1016/j.j...1993.02.026	Open Access
Kim, J., & Lee, S.	1992	Study on... in...	Journal of...	0	4	3285-3300	10.1016/j.j...1992.03.027	10.1016/j.j...1992.03.027	Open Access
Lee, S., & Kim, J.	1991	Research on... in...	Journal of...	0	5	3410-3425	10.1016/j.j...1991.04.028	10.1016/j.j...1991.04.028	Open Access
Kim, D., & Park, S.	1990	Investigation of... in...	Journal of...	0	6	3535-3550	10.1016/j.j...1990.05.029	10.1016/j.j...1990.05.029	Open Access
Kim, J., & Lee, S.	1989	Study on... in...	Journal of...	0	7	3660-3675	10.1016/j.j...1989.06.030	10.1016/j.j...1989.06.030	Open Access
Lee, S., & Kim, J.	1988	Research on... in...	Journal of...	0	8	3785-3800	10.1016/j.j...1988.07.031	10.1016/j.j...1988.07.031	Open Access
Kim, D., & Park, S.	1987	Investigation of... in...	Journal of...	0	9	3910-3925	10.1016/j.j...1987.08.032	10.1016/j.j...1987.08.032	Open Access
Kim, J., & Lee, S.	1986	Study on... in...	Journal of...	0	10	4035-4050	10.1016/j.j...1986.09.033	10.1016/j.j...1986.09.033	Open Access
Lee, S., & Kim, J.	1985	Research on... in...	Journal of...	0	11	4160-4175	10.1016/j.j...1985.10.034	10.1016/j.j...1985.10.034	Open Access
Kim, D., & Park, S.	1984	Investigation of... in...	Journal of...	0	12	4285-4300	10.1016/j.j...1984.11.035	10.1016/j.j...1984.11.035	Open Access
Kim, J., & Lee, S.	1983	Study on... in...	Journal of...	0	1	4410-4425	10.1016/j.j...1983.12.036	10.1016/j.j...1983.12.036	Open Access
Lee, S., & Kim, J.	1982	Research on... in...	Journal of...	0	2	4535-4550	10.1016/j.j...1982.01.037	10.1016/j.j...1982.01.037	Open Access
Kim, D., & Park, S.	1981	Investigation of... in...	Journal of...	0	3	4660-4675	10.1016/j.j...1981.02.038	10.1016/j.j...1981.02.038	Open Access
Kim, J., & Lee, S.	1980	Study on... in...	Journal of...	0	4	4785-4800	10.1016/j.j...1980.03.039	10.1016/j.j...1980.03.039	Open Access
Lee, S., & Kim, J.	1979	Research on... in...	Journal of...	0	5	4910-4925	10.1016/j.j...1979.04.040	10.1016/j.j...1979.04.040	Open Access
Kim, D., & Park, S.	1978	Investigation of... in...	Journal of...	0	6	5035-5050	10.1016/j.j...1978.05.041	10.1016/j.j...1978.05.041	Open Access
Kim, J., & Lee, S.	1977	Study on... in...	Journal of...	0	7	5160-5175	10.1016/j.j...1977.06.042	10.1016/j.j...1977.06.042	Open Access
Lee, S., & Kim, J.	1976	Research on... in...	Journal of...	0	8	5285-5300	10.1016/j.j...1976.07.043	10.1016/j.j...1976.07.043	Open Access
Kim, D., & Park, S.	1975	Investigation of... in...	Journal of...	0	9	5410-5425	10.1016/j.j...1975.08.044	10.1016/j.j...1975.08.044	Open Access
Kim, J., & Lee, S.	1974	Study on... in...	Journal of...	0	10	5535-5550	10.1016/j.j...1974.09.045	10.1016/j.j...1974.09.045	Open Access
Lee, S., & Kim, J.	1973	Research on... in...	Journal of...	0	11	5660-5675	10.1016/j.j...1973.10.046	10.1016/j.j...1973.10.046	Open Access
Kim, D., & Park, S.	1972	Investigation of... in...	Journal of...	0	12	5785-5800	10.1016/j.j...1972.11.047	10.1016/j.j...1972.11.047	Open Access
Kim, J., & Lee, S.	1971	Study on... in...	Journal of...	0	1	5910-5925	10.1016/j.j...1971.12.048	10.1016/j.j...1971.12.048	Open Access
Lee, S., & Kim, J.	1970	Research on... in...	Journal of...	0	2	6035-6050	10.1016/j.j...1970.01.049	10.1016/j.j...1970.01.049	Open Access
Kim, D., & Park, S.	1969	Investigation of... in...	Journal of...	0	3	6160-6175	10.1016/j.j...1969.02.050	10.1016/j.j...1969.02.050	Open Access
Kim, J., & Lee, S.	1968	Study on... in...	Journal of...	0	4	6285-6300	10.1016/j.j...1968.03.051	10.1016/j.j...1968.03.051	Open Access
Lee, S., & Kim, J.	1967	Research on... in...	Journal of...	0	5	6410-6425	10.1016/j.j...1967.04.052	10.1016/j.j...1967.04.052	Open Access
Kim, D., & Park, S.	1966	Investigation of... in...	Journal of...	0	6	6535-6550	10.1016/j.j...1966.05.053	10.1016/j.j...1966.05.053	Open Access
Kim, J., & Lee, S.	1965	Study on... in...	Journal of...	0	7	6660-6675	10.1016/j.j...1965.06.054	10.1016/j.j...1965.06.054	Open Access
Lee, S., & Kim, J.	1964	Research on... in...	Journal of...	0	8	6785-6800	10.1016/j.j...1964.07.055	10.1016/j.j...1964.07.055	Open Access
Kim, D., & Park, S.	1963	Investigation of... in...	Journal of...	0	9	6910-6925	10.1016/j.j...1963.08.056	10.1016/j.j...1963.08.056	Open Access
Kim, J., & Lee, S.	1962	Study on... in...	Journal of...	0	10	7035-7050	10.1016/j.j...1962.09.057	10.1016/j.j...1962.09.057	Open Access
Lee, S., & Kim, J.	1961	Research on... in...	Journal of...	0	11	7160-7175	10.1016/j.j...1961.10.058	10.1016/j.j...1961.10.058	Open Access
Kim, D., & Park, S.	1960	Investigation of... in...	Journal of...	0	12	7285-7300	10.1016/j.j...1960.11.059	10.1016/j.j...1960.11.059	Open Access
Kim, J., & Lee, S.	1959	Study on... in...	Journal of...	0	1	7410-7425	10.1016/j.j...1959.12.060	10.1016/j.j...1959.12.060	Open Access
Lee, S., & Kim, J.	1958	Research on... in...	Journal of...	0	2	7535-7550	10.1016/j.j...1958.01.061	10.1016/j.j...1958.01.061	Open Access
Kim, D., & Park, S.	1957	Investigation of... in...	Journal of...	0	3	7660-7675	10.1016/j.j...1957.02.062	10.1016/j.j...1957.02.062	Open Access
Kim, J., & Lee, S.	1956	Study on... in...	Journal of...	0	4	7785-7800	10.1016/j.j...1956.03.063	10.1016/j.j...1956.03.063	Open Access
Lee, S., & Kim, J.	1955	Research on... in...	Journal of...	0	5	7910-7925	10.1016/j.j...1955.04.064	10.1016/j.j...1955.04.064	Open Access
Kim, D., & Park, S.	1954	Investigation of... in...	Journal of...	0	6	8035-8050	10.1016/j.j...1954.05.065	10.1016/j.j...1954.05.065	Open Access
Kim, J., & Lee, S.	1953	Study on... in...	Journal of...	0	7	8160-8175	10.1016/j.j...1953.06.066	10.1016/j.j...1953.06.066	Open Access
Lee, S., & Kim, J.	1952	Research on... in...	Journal of...	0	8	8285-8300	10.1016/j.j...1952.07.067	10.1016/j.j...1952.07.067	Open Access
Kim, D., & Park, S.	1951	Investigation of... in...	Journal of...	0	9	8410-8425	10.1016/j.j...1951.08.068	10.1016/j.j...1951.08.068	Open Access
Kim, J., & Lee, S.	1950	Study on... in...	Journal of...	0	10	8535-8550	10.1016/j.j...1950.09.069	10.1016/j.j...1950.09.069	Open Access
Lee, S., & Kim, J.	1949	Research on... in...	Journal of...	0	11	8660-8675	10.1016/j.j...1949.10.070	10.1016/j.j...1949.10.070	Open Access
Kim, D., & Park, S.	1948	Investigation of... in...	Journal of...	0	12	8785-8800	10.1016/j.j...1948.11.071	10.1016/j.j...1948.11.071	Open Access
Kim, J., & Lee, S.	1947	Study on... in...	Journal of...	0	1	8910-8925	10.1016/j.j...1947.12.072	10.1016/j.j...1947.12.072	Open Access
Lee, S., & Kim, J.	1946	Research on... in...	Journal of...	0	2	9035-9050	10.1016/j.j...1946.01.073	10.1016/j.j...1946.01.073	Open Access
Kim, D., & Park, S.	1945	Investigation of... in...	Journal of...	0	3	9160-9175	10.1016/j.j...1945.02.074	10.1016/j.j...1945.02.074	Open Access
Kim, J., & Lee, S.	1944	Study on... in...	Journal of...	0	4	9285-9300	10.1016/j.j...1944.03.075	10.1016/j.j...1944.03.075	Open Access
Lee, S., & Kim, J.	1943	Research on... in...	Journal of...	0	5	9410-9425	10.1016/j.j...1943.04.076	10.1016/j.j...1943.04.076	Open Access
Kim, D., & Park, S.	1942	Investigation of... in...	Journal of...	0	6	9535-9550	10.1016/j.j...1942.05.077	10.1016/j.j...1942.05.077	Open Access
Kim, J., & Lee, S.	1941	Study on... in...	Journal of...	0	7	9660-9675	10.1016/j.j...1941.06.078	10.1016/j.j...1941.06.078	Open Access
Lee, S., & Kim, J.	1940	Research on... in...	Journal of...	0	8	9785-9800	10.1016/j.j...1940.07.079	10.1016/j.j...1940.07.079	Open Access
Kim, D., & Park, S.	1939	Investigation of... in...	Journal of...	0	9	9910-9925	10.1016/j.j...1939.08.080	10.1016/j.j...1939.08.080	Open Access
Kim, J., & Lee, S.	1938	Study on... in...	Journal of...	0	10	10035-10050	10.1016/j.j...1938.09.081	10.1016/j.j...1938.09.081	Open Access
Lee, S., & Kim, J.	1937	Research on... in...	Journal of...	0	11	10160-10175	10.1016/j.j...1937.10.082	10.1016/j.j...1937.10.082	Open Access
Kim, D., & Park, S.	1936	Investigation of... in...	Journal of...	0	12	10285-10300	10.1016/j.j...1936.11.083	10.1016/j.j...1936.11.083	Open Access
Kim, J., & Lee, S.	1935	Study on... in...	Journal of...	0	1	10410-10425	10.1016/j.j...1935.12.084	10.1016/j.j...1935.12.084	Open Access
Lee, S., & Kim, J.	1934	Research on... in...	Journal of...	0	2	10535-10550	10.1016/j.j...1934.01.085	10.1016/j.j...1934.01.085	Open Access
Kim, D., & Park, S.	1933	Investigation of... in...	Journal of...	0	3	10660-10675	10.1016/j.j...1933.02		



Authors	Year	Title	Abstract	What	Outcome measures	Main findings	REFERENCE LIST SCANNED	Journal	Pages	
D. Pires, E. B. Cruz and C. Cairo	2014	Aquatic exercise and pain neurophysiology education versus aquatic exercise alone for patients with chronic low back pain: a randomized controlled trial	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. DESIGN: Single-blind randomized controlled trial. SETTING: Outpatient clinic. SUBJECTS: Sixty-two chronic low back pain patients were randomly allocated to receive aquatic exercise and pain neurophysiology education (n = 30) or aquatic exercise alone (n = 32). INTERVENTIONS: Twelve sessions of a 6-week aquatic exercise programme preceded by 2 sessions of pain neurophysiology education. Controls received only 12 sessions of the 6-week aquatic exercise programme. MAIN MEASURES: The primary outcomes were pain intensity (Visual Analogue Scale) and functional disability (Quebec Back Pain Disability Scale) at the baseline, 6 weeks after the beginning of the aquatic exercise programme and at the 3 months follow-up. Secondary outcome was kinesiophobia (Tampa Scale of Kinesiophobia). RESULTS: Fifty-five participants completed the study. Analysis using mixed-model ANOVA revealed a significant treatment condition interaction on pain intensity at the 3 months follow-up, favoring the education group (mean SD change: -25.4/-26.7 vs -6.6/-7.30.7, P < 0.005). Although participants in the education group were more likely to report perceived functional benefits from treatment at 3 months follow-up (RR=1.63, 95%CI: 1.01-2.63), no significant differences were found in functional disability and kinesiophobia between groups at any time. CONCLUSIONS: This study's findings support the provision of pain neurophysiology education as a clinically effective addition to aquatic exercise.	RCT: Aquatic exercise + 2 x PNE sessions n = 30 VS. Aquatic exercise n = 23 (NB. 12 sessions aquatic exercise over 6/52)	Pain + disability + kinesiophobia	Pain reduced in PNE group	Yes	Clin Rehabil		
* Picked up on google scholar (not database search)	Traeger AC, Moseley GL, Hubscher M, Lee H, Skinner IW, M. Dolphens, J. Nijs, B. Cagnie, M. Meeus, N. Roussel, J. Kregel, A. Malfliet, G. Vanderstraeten and L. Danneels	2014	Pain education to prevent chronic low back pain: a study protocol for a randomised controlled trial	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 double-blind (participant/outcome assessor) randomised controlled trial will investigate the efficacy of a brief educational approach to prevent chronic LBP in 'at-risk' individuals.	PROTOCOL ONLY		Yes			
							Yes	TRIAL REGISTRATION: ClinicalTrials.gov Identifier: NCT02098005. BMC Musculoskeletal Disorders	15 ##	
	S. Anandkumar and M. Manivasagam	2014	Multimodal physical therapy management of a 48-year-old female with post-stroke complex regional pain syndrome	BACKGROUND: Among the multiple conservative modalities, physiotherapy is a commonly utilized treatment modality in managing chronic non-specific spinal pain. Despite the scientific progresses with regard to pain and motor control neuroscience, treatment of chronic spinal pain (CSP) often tends to stick to a peripheral biomechanical model, without targeting brain mechanisms. With a view to enhance clinical efficacy of existing physiotherapeutic treatments for CSP, the development of clinical strategies targeted at 'training the brain' is to be pursued. Promising proof-of-principle results have been reported for the effectiveness of a modern neuroscience approach to CSP when compared to usual care, but confirmation is required in a larger, multi-center trial with appropriate evidence-based control intervention and long-term follow-up. The aim of this study is to assess the effectiveness of a modern neuroscience approach, compared to usual care evidence-based physiotherapy, for reducing pain and improving functioning in patients with CSP. A secondary objective entails examining the effectiveness of the modern neuroscience approach versus usual care physiotherapy for normalizing brain gray matter in patients with CSP. METHODS/DESIGN: The study is a multi-center, triple-blind, two-arm (1:1) randomized clinical trial with 1-year follow-up. 120 CSP patients will be randomly allocated to either the experimental (receiving pain neuroscience education followed by cognition-targeted motor control training) or the control group (receiving usual care physiotherapy), each comprising of 3 months treatment. The main outcome measures are pain (including symptoms and indices of central sensitization) and self-reported disability. Secondary outcome This case report describes a 48-year-old female who presented with complaints of right shoulder pain, hyperesthesia and swelling of the hand along with added symptoms of pain centralization following a cerebrovascular accident. On clinical evaluation, the patient satisfied the Budapest diagnostic criteria for Complex Regional Pain Syndrome (CRPS) type-1. Physical therapy management (1st three sessions) was initially focused on pain neurophysiology education with an aim to reduce kinesiophobia and reconceptualise her pain perception. The patient had an immediate significant improvement in her pain and functional status. Following this, pain modulation in the form of transcutaneous electrical nerve stimulation, kinesio tape application, "pain exposure" physical therapy and exercise therapy was carried out for a period of 7 weeks. The patient had complete resolution of her symptoms which was maintained at a six-month follow-up.	Case study, 48 y F with CRPS	3 x PNE then other therapies	Significant improvement in pain & functional status following PNE	Yes	Physiotherapy Theory & Practice	30 1 38-48
	K. Zimney, A. Louw and E. J. Puentudura	2014	Use of Therapeutic Neuroscience Education to address psychosocial factors associated with acute low back pain: a case report	Acute low back pain (LBP) from injuries is prevalent in the work place. It has been shown that patients with psychosocial factors often progress with persistent pain and lead to significant workers compensation costs. Therapeutic Neuroscience Education (TNE) has been shown to be beneficial in changing a patient's cognition regarding their pain state, which may result in decrease fear, anxiety and catastrophization. A 19-year-old female who developed LBP from a work injury was the patient for this case report. A physical examination, Numeric Pain Rating Scale (NRS), Oswestry Disability Index (ODI), Fear-Avoidance Beliefs Questionnaire (FABQ), Keele StarT Back Screening Tool (Keele SBST) and Acute Low Back Pain Screening (ALBPS) Questionnaires were assessed during initial physical therapy visit and discharge. Treatment consisted of use of TNE, manual therapy and exercises. She attended five total visits over a 2-week period prior to full discharge. During the initial visit the patient reported NRS = 3/10, ODI = 36%, FABQ-PA = 23, FABQ-W = 30, Keele SBST = 4/9, ALBPS = 101. At discharge the patient reported a 0 on all outcome questionnaires with ability to return to full work and no pain complaints.	Case study, 19yo F with LBP	PNE and other modalities x 5 visits over 2 weeks	Reduction in pain, disability, fear avoidance, and other questionnaires	YES -	Physiother Theory Pract	30 3 202-9
							Puentudura EJ, Louw A 2012 A neuroscience approach to managing athletes with low back pain. Physical Therapy in Sport 13: 123-133			
	A. Louw, I. Diener, M. R. Landers and E. J. Puentudura	2014	Preoperative pain neuroscience education for lumbar radiculopathy: a multicenter randomized controlled trial with 1-year follow-up	STUDY DESIGN: Multicenter, randomized, controlled trial on preoperative pain neuroscience education (NE) for lumbar radiculopathy. OBJECTIVE: To determine if the addition of NE to usual preoperative education would result in superior outcomes with regard to pain, function, surgical experience, and health care utilization posturgery. SUMMARY OF BACKGROUND DATA: One in 4 patients after lumbar surgery (LS) for radiculopathy experience persistent pain and disability, which is nonresponsive to preoperative treatments. NE focusing on the neurophysiology of pain has been shown to decrease pain and disability in populations with chronic low back pain. METHODS: Eligible patients scheduled for LS for radiculopathy were randomized to receive either preoperative usual care (UC) or a combination of UC plus 1 session of NE delivered by a physical therapist (verbal one-on-one format) and a NE booklet. Sixty-seven patients completed the following outcomes prior to LS (baseline), and 1, 3, 6, and 12 months after LS: low back pain (numeric rating scale), leg pain (numeric rating scale), function (Oswestry Disability Index), various beliefs and experiences related to LS (10-item survey with Likert scale responses), and postoperative utilization of health care (utilization of health care questionnaire). RESULTS: At 1-year follow-up, there were no statistical differences between the experimental and control groups with regard to primary outcome measure of low back pain (P = 0.183), leg pain (P = 0.075), and function (P = 0.365). In a majority of the categories regarding surgical experience, the NE group scored significantly better: better prepared for LS (P = 0.001), preoperative session preparing them for LS (P < 0.001) and LS meeting their expectations (P = 0.021). Health care utilization post-LS also favored the NE group (P = 0.007) resulting in 45% less health care expenditure compared with the control group in the 1-year follow-up period. CONCLUSION: NE resulted in significant behavior change. Despite a similar pain and functional trajectory during the 1-year trial, patients with LS who received NE viewed their surgical experience more favorably and used less health care facility in the form of medical tests and treatments. LEVEL OF EVIDENCE: 2.	RCT for preop PNE in lumbar radiculopathy - usual care VS. PNE with PT and booklet	1, 3, 6 and 12 m f/u on low back pain, leg pain, function and beliefs	No diff in pain btw groups - PNE group was better prepared with behavioural change	yes	Spine (Phila Pa 1976)	39 18 1449-57
	A. Louw	2014	Therapeutic neuroscience education via e-mail: a case report	Abstract Therapeutic neuroscience education (TNE) aims to alter a patient's thoughts and beliefs about pain and has shown efficacy in treating chronic pain. To date, TNE sessions mainly consist of one-on-one verbal communication. This approach limits availability of TNE to pain patients in remote areas. A 32-year-old patient with chronic low back pain (CLBP) who underwent surgery for thoracic outlet syndrome (TOS) attended a single clinic one-on-one TNE session followed by TNE via electronic mail (e-mail), pacing and graded exposure over a 4-month period. A physical examination, Numeric Rating Scale (NRS), Oswestry Disability Index (ODI), the Disabilities of Arm, Shoulder and Hand (DASH), and Fear-Avoidance Beliefs Questionnaire (FABQ) were assessed during her initial physical therapy visit as well as 1 and 4 months later. Pre-TNE, the patient reported: NRS (arm) = 7/10; NRS (leg) = 4/10; ODI = 10.0%; DASH = 36.7%; FABQ-W = 24; and FABQ-PA = 17. After 5 e-mail sessions all outcome measures improved, most notably NRS (arm) = 2/10; NRS (leg) = 0/10; DASH = 16.7%; FABQ-W = 8; and FABQ-PA = 7. TNE can potentially be delivered to suffering pain patients in remote areas or to individuals who have time and financial constraints, and likely at a significant reduced cost via e-mail.	Case study PNE 32yo CLP with one on one PNE session then 5 x email PNE and pacing/graded exposure therapy over 4m	Pain, Disability, Fear at 1 and 4 months	Reduction in pain and upperlimb disability and fear		Physiother Theory Pract	30 8 588-96
* Picked up on google scholar (not database search) and REF list	Ittersum MW, Wilgen CP,	2013	Written pain neuroscience education in fibromyalgia: a multicenter randomised controlled trial	Mounting evidence supports the use of face-to-face pain neuroscience education for the treatment of chronic pain patients. This study aimed at examining whether written education about pain neuroscience improves illness perceptions, catastrophizing, and health status in patients with fibromyalgia. A double-blind, multicenter randomized controlled clinical trial with 6-month follow-up was conducted. Patients with FM (n = 114) that consented to participate were randomly allocated to receive either written pain neuroscience education or written relaxation training. Written pain neuroscience education comprised of a booklet with pain neuroscience education plus a telephone call to clarify any difficulties; the relaxation group received a booklet with relaxation education and a telephone call. The revised illness perception questionnaire, Pain Catastrophizing Scale, and fibromyalgia impact questionnaire were used as outcome measures. Both patients and assessors were blinded. Repeated-measures analysis with last observation carried forward principle were performed. Cohen's d effect sizes (ES) were calculated for all within-group changes and between-group differences. The results reveal that written pain neuroscience education does not change the impact of FM on daily life, catastrophizing, or perceived symptoms of patients with FM. Compared with written relaxation training, written pain neuroscience education improved beliefs in a chronic timeline of FM (P = 0.03; ES = 0.50), but it does not impact upon other domains of illness perceptions. Compared with written relaxation training, written pain neuroscience education slightly improved illness perceptions of patients with FM, but it did not impact clinically meaningful effects on pain, catastrophizing, or the impact of FM on daily life. Face-to-face sessions of pain neuroscience education are required to change inappropriate cognitions and perceived health in patients with FM.	RCT with FM patients - PNE booklet and phone call VS. relaxation booklet and phone.	Illness perception, catastrophising, FM impact - at 6m follow up	PNE did not change impact of FM, catastrophising or perceived symptoms. No clinically meaningful effects on pain, catastrophising or impact -> need face to face session for change			
	L. Gallagher, I. McAuley and G. L. Moseley	2013	A randomized-controlled trial of using a book of metaphors to reconceptualize pain and decrease catastrophizing in people with chronic pain	OBJECTIVES: Reconceptualization of pain and reduction of pain-related catastrophizing are primary objectives in chronic pain rehabilitation. Teaching people about the underlying biology of pain has been shown to facilitate these objectives. The objective of this study was to investigate whether written metaphor and story can be used to increase knowledge of the biology of pain and reduce pain-related catastrophizing. METHODS: In this randomized single-blind partial cross-over controlled trial, 79 people with chronic pain received either a booklet of metaphors and stories conveying key pain biology concepts or a booklet containing advice on how to manage chronic pain according to established cognitive-behavioral principles. The primary outcome variables, pain biology knowledge and catastrophizing, were measured before randomization, at 3 weeks and at 3 months, at which time the control group was crossed over to receive the metaphors and stories booklet. Pain and disability were secondary outcome variables. RESULTS: The Metaphors group showed larger changes in both variables (time x group interactions: P < 0.01, effect size Cohen d = 0.7 for catastrophizing and 1.7 for pain biology knowledge). Gains were maintained for at least 3 months. Changes were replicated in the Advice group when crossed over. There was no change in pain or self-reported disability in either group. DISCUSSION: We conclude that providing educational material through metaphor and story can assist patients to reconceptualize pain and reduce catastrophizing. Metaphor and story could be used as a precursor to other interventions that target functional capacity.	RCT - n = 79 either PNE via metaphors and stories OR CBT style education	PNE knowledge, catastrophizing at 3 weeks and 3 months, Pain and disability.	Change in knowledge and catastrophizing in PNE group but no diff in pain or disability btw groups.	yes	Clinical Journal of Pain	29 1



J. Van Oosterwijk, M. Meeus, L. Paul, M. De Schryver, A. Paschal, L. Lambrecht and J. Nijns	2013	<b>Pain physiology education improves health status and endogenous pain inhibition in fibromyalgia: a double-blind randomized controlled trial</b>	OBJECTIVES: There is evidence that education on pain physiology can have positive effects on pain, disability, and catastrophization in patients with chronic musculoskeletal pain disorders. A double-blind randomized controlled trial (RCT) was performed to examine whether intensive pain physiology education is also effective in fibromyalgia (FM) patients, and whether it is able to influence the impaired endogenous pain inhibition of these patients. METHODS: Thirty FM patients were randomly allocated to either the experimental (receiving pain physiology education) or the control group (receiving pacing self-management education). The primary outcome was the efficacy of the pain inhibitory mechanisms, which was evaluated by spatially accumulating thermal nociceptive stimuli. Secondary outcome measures included pressure pain threshold measurements and questionnaires assessing pain cognitions, behavior, and health status. Assessments were performed at baseline, 2 weeks and 3 months follow-up. Repeated measures ANOVAs were used to reveal possible therapy effects and effect sizes were calculated. RESULTS: After the intervention the experimental group had improved knowledge of pain neurophysiology (P<0.001). Patients from this group worried less about their pain in the short term (P=0.004). Long-term improvements in physical functioning (P=0.046), vitality (P=0.047), mental health (P<0.001), and general health perceptions (P<0.001) were observed. In addition, the intervention group reported lower pain scores and showed improved endogenous pain inhibition (P=0.041) compared with the control group. DISCUSSION: These results suggest that FM patients are able to understand and remember the complex material about pain physiology. 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.	RCT intensive PNE n = 30 FM patients either PNE or control (pacing education)	2 weeks & 3 months /u- efficacy of pain inhibit mechanisms, pressure pain threshold, pain cognition, behaviour and health status	PNE group improved knowledge of pain, less worried, less disability, improved mental health etc, lower pain and improved endogenous pain inhibit	Clinical Journal of Pain	29	10	873-882
* Picked up on google scholar (not database search)	Robinson, Victoria; King, Richard; Ryan, Cornac G	2013	<b>Pain Neurophysiology Education' as Part of a Pain Management Service Decreases Fear Avoidance and Improves Patient's Understanding of the Neurophysiology of Chronic Pain at Four Months Follow Up.</b>	The aim of this service evaluation was to investigate whether the <i>Pain Neurophysiology Education (PNE)</i> service provided at a pain clinic in a northern hospital in the UK increases patients understanding of the neurophysiology of chronic pain and reduces fear avoidance beliefs and pain catastrophizing. Data was collected using the Neurophysiology of Pain Questionnaire (NPPQ), the Tampa Scale of Kinesiophobia (TSK) and the Pain Catastrophising Scale (PCS). Patient data (n=18) was collected pre-intervention, post-intervention and at the four month follow up point. The results demonstrated a mean improvement of 22.5% from pre to post intervention on the NPPQ and a maintained improvement of 14% from post to follow up. This result was shown to be statistically significant. There was a mean improvement of 4 points on the TSK which was also shown to be statistically significant. There was a small, but not statistically significant, improvement of 2 points on the PCS. This service evaluation provides some basic evidence that PNE delivered by our physiotherapy team can improve and maintain patients understanding of their pain and start to address some of their negative beliefs associated with complex persistent pain.	Clinical study, no control group, PNE delivered by PT in chronic pain	neurophys pain questionnaire, kinesiophobia, catastrophising at pre/post and 4 month followup	neurophys knowledge increase, kinesiophobia improved and non sign. Catastrophising improvement.	Pain and Rehabilitation - The Journal of the Physiotherapy Pain Association		
A. Louw, E. L. Puentudura and P. Mintken	2012	<b>Use of an abbreviated neuroscience education approach in the treatment of chronic low back pain: a case report</b>	Chronic low back pain (CLBP) remains prevalent in society, and conservative treatment strategies appear to have little effect. It is proposed that patients with CLBP may have altered cognition and increased fear, which impacts their ability to move, perform exercise, and partake in activities of daily living. Neuroscience education (NE) aims to change a patient's cognition regarding their pain state, which may result in decreased fear, ultimately resulting in confrontation of pain barriers and a resumption of normal activities. A 64-year-old female with history of CLBP was the patient for this case report. A physical examination, the Numeric Pain Rating Scale (NPRS), Oswestry Disability Index (ODI), Fear-Avoidance Beliefs Questionnaire (FABQ), and Zung Depression Scale were assessed during her initial physical therapy visit, immediately after her first physical therapy session, and at 7-month follow-up. Treatment consisted of an abbreviated NE approach, exercises (range of motion, stretches, and cardiovascular), and aquatic therapy. The attended twice a week for 4 weeks, or 8 visits total. Pre-NE, the patient reported NPRS = 9/10; ODI = 54%; FABQ-W = 25/42; FABQ-PA = 20/24, and Zung = 58. Immediately following the 75-minute evaluation and NE session, the patient reported improvement in all four outcome measures, most notably a reduction in the FABQ-W score to 2/42 and the FABQ-PA to 1/24. At a 7-month follow-up, all outcome measures continued to be improved. NE aimed at decreasing fear associated with movement may be a valuable adjunct to movement-based therapy, such as exercise, for patients with CLBP.	case study 64 yo female with CLBP, treatment PNE, exercises and aquatic therapy - 2 x week for month	pain, disability, fear, depression with 7m follow up	improvement in all outcome measures	Physiother Theory Pract	28	1	50-62
A. Louw, I. Diener, D. S. Butler and E. J. Puentudura	2011	<b>The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain</b>	OBJECTIVE: To evaluate the evidence for the effectiveness of neuroscience education (NE) for pain, disability, anxiety, and stress in chronic musculoskeletal (MSK) pain. DATA SOURCES: Systematic searches were conducted on Biomed Central, BMJ.com, CINAHL, the Cochrane Library, NLM Central Gateway, OVID, ProQuest (Digital Dissertations), Psycinfo, PubMed/Medline, ScienceDirect, and Web of Science. Secondary searching (PEARLing) was undertaken, whereby reference lists of the selected articles were reviewed for additional references not identified in the primary search. STUDY SELECTION: All experimental studies including randomized controlled trials (RCTs), nonrandomized clinical trials, and case series evaluating the effect of NE on pain, disability, anxiety, and stress for chronic MSK pain were considered for inclusion. Additional limitations: studies published in English, published within the last 10 years, and patients older than 18 years. No limitations were set on specific outcome measures of pain, disability, anxiety, and stress. DATA EXTRACTION: Data were extracted using the participants, interventions, comparison, and outcomes (PICCO) approach. DATA SYNTHESIS: Methodological quality was assessed by 2 reviewers using the Critical Review Form-Quantitative Studies. This review includes 8 studies, consisting of 6 high-quality RCTs, 1 pseudo-RCT, and 1 comparative study involving 401 subjects. Most articles were of good quality, with no studies rated as poor or fair. Heterogeneity across the studies with respect to participants, interventions evaluated, and outcome measures used	SR 2011 - studies included - 8 included	pain, disability, catastrophising, and physical performance	"compelling evidence that PNE can have positive effect"	Arch Phys Med Rehabil	92	12	2041-56
C. L. Clarke, C. G. Ryan and D. J. Martin	2011	<b>Pain neurophysiology education for the management of individuals with chronic low back pain: systematic review and meta-analysis</b>	Pain neurophysiology education (PNE) is a form of education for patients with chronic low back pain (CLBP). The purpose of this systematic review was to investigate the evidence for PNE in the management of patients with CLBP. A literature search of MEDLINE, CINAHL and AMED was performed from 1996(01)-2010(09). RCT appraisal and synthesis was assessed using the Cochrane Back Review Group (CBRG) guidelines. The main outcome measures were pain, physical-function, psychological-function, and social-function. Two moderate quality RCTs (n=122) were included in the final review. According to the CBRG criteria there was very low quality evidence that PNE is beneficial for pain, physical-function, psychological-function, and social-function. Meta-analysis found PNE produced statistically significant but clinically small improvements in short-term pain of 5mm (0, 10.0mm) [mean difference (95%CI)] on the 100mm VAS. This review was limited by the small number of studies (n=2) that met the inclusion criteria and by the fact that both studies were produced by the same group that published the PNE manual. These factors contributed to the relatively low quality of the evidence. There is a need for more studies investigating PNE for chronic whiplash as a debilitating condition characterized by increased sensitivity to pain stimuli and increased movement dysfunction. Previous work in people with chronic low back pain and chronic fatigue syndrome indicates that pain neurophysiology education is able to improve illness beliefs and attitudes as well as movement performance. This single case study (A-B-C design) with six patients with chronic whiplash associated disorders (WAD) was aimed at examining whether education about the neurophysiology of pain is successful by changes in symptoms, daily functioning, pain beliefs, and behavior. Periods A and C represented assessment periods, while period B consisted of the intervention (pain neurophysiology education). Results showed a significant decrease in kinesiophobia (Tampa Scale for Kinesiophobia), the passive coping strategy of resting (Pain Coping Inventory), self-rated disability (Neck Disability Index), and photophobia (WAD Symptom List). At the same time, significantly increased pain pressure thresholds and improved pain-free movement performance (visual analog scale on Neck Extension Test and Brachial Plexus Provocation Test) were established. Although the current results need to be verified in a randomized controlled trial, they suggest that education about the neurophysiology of pain is able to increase pain thresholds and improve pain behavior. The following paper is a brief overview of an audit carried out by the pain management service at James Cook Hospital. The Educational approach "Explain Pain" has recently been added to our pain management service. The aim of the audit was to investigate if the Explain Pain service that we provide increases patients understanding of chronic pain. Forty patients completed the audit process and completed a 19 item questionnaire evaluating pain knowledge pre and post the education. The mean pre-test score was 7.8/19 (41%) and the mean post test score was 12.9/19 (68%). This showed a statistically significant mean improvement of 5.2 (SD 2.6) (p<0.01). This provides some basic evidence that Explain Pain as delivered by our team can improve patient's understanding of their pain. Qualitative feedback from the patients was also recorded and was generally positive in nature. We are now undertaking follow up work to investigate the effect of Explain Pain on clinical outcomes as well as getting more in-depth	SR - only included moseley 2004 and 2009 Moseley conference proceedings?	pain, function, psychological function and social function	low evidence for small clinical improvement in short term pain and function	Man Ther	16	6	544-9
J. Van Oosterwijk, J. Nijns, M. Meeus, S. Truijien, J. Craps, N. Van den Keybus and L. Paul	2011	<b>Pain neurophysiology education improves cognitions, pain thresholds, and movement performance in people with chronic whiplash: a pilot study</b>	Chronic whiplash is a debilitating condition characterized by increased sensitivity to pain stimuli and increased movement dysfunction. Previous work in people with chronic low back pain and chronic fatigue syndrome indicates that pain neurophysiology education is able to improve illness beliefs and attitudes as well as movement performance. This single case study (A-B-C design) with six patients with chronic whiplash associated disorders (WAD) was aimed at examining whether education about the neurophysiology of pain is successful by changes in symptoms, daily functioning, pain beliefs, and behavior. Periods A and C represented assessment periods, while period B consisted of the intervention (pain neurophysiology education). Results showed a significant decrease in kinesiophobia (Tampa Scale for Kinesiophobia), the passive coping strategy of resting (Pain Coping Inventory), self-rated disability (Neck Disability Index), and photophobia (WAD Symptom List). At the same time, significantly increased pain pressure thresholds and improved pain-free movement performance (visual analog scale on Neck Extension Test and Brachial Plexus Provocation Test) were established. Although the current results need to be verified in a randomized controlled trial, they suggest that education about the neurophysiology of pain is able to increase pain thresholds and improve pain behavior. The following paper is a brief overview of an audit carried out by the pain management service at James Cook Hospital. The Educational approach "Explain Pain" has recently been added to our pain management service. The aim of the audit was to investigate if the Explain Pain service that we provide increases patients understanding of chronic pain. Forty patients completed the audit process and completed a 19 item questionnaire evaluating pain knowledge pre and post the education. The mean pre-test score was 7.8/19 (41%) and the mean post test score was 12.9/19 (68%). This showed a statistically significant mean improvement of 5.2 (SD 2.6) (p<0.01). This provides some basic evidence that Explain Pain as delivered by our team can improve patient's understanding of their pain. Qualitative feedback from the patients was also recorded and was generally positive in nature. We are now undertaking follow up work to investigate the effect of Explain Pain on clinical outcomes as well as getting more in-depth	case study n = 6 in whiplash - PNE education	change in symptoms, function, pain beliefs and behaviour - kinesiophobia, coping, disability and photophobia	increased pressure pain thresholds and improved pain free movement, decrease in kinesiophobia, disability etc.	J Rehabil Res Dev	48	1	43-58
* Picked up on google scholar (not database search)	Robinson, Victoria	2011	<b>'Explain Pain' as part of a pain management service improves patient's understanding of the neurophysiology of Chronic Pain.</b>	Clinical study, no control, 40 chronic pain patients given explain pain	pre and post 19 item questionnaire	Significant improvement in patient understanding of knowledge	Pain and Rehabilitation - The Journal of the Physiotherapy Pain Association	32	27-30	No control group - clinical setting - 40 pain patients - Outcome = pain knowledge
* Picked up on google scholar (not database search)	M.W. van Ittersum, C.P. van Wilgen, J.W. Groothoff, C.P. van der Schans	2011	<b>Is appreciation of written education about pain neurophysiology related to changes in illness perceptions and health status in patients with fibromyalgia?</b>	No control group, written PNE only to FM patients	Appreciation of pain knowledge, reassurance, illness perceptions, catastrophising, health status, impact at 2 weeks and 6 weeks	illness coherence, emotional representations, pain and fatigue changed - but no clinical differences in long term -> written info inadequate	Patient education and counselling	85	269-274	
C. G. Ryan, H. G. Gray, M. Newton and M. H. Granat	2010	<b>Pain biology education and exercise classes compared to pain biology education alone for individuals with chronic low back pain: a pilot randomised controlled trial</b>	Should be incorporated into a broader multidisciplinary self-management program. The aim of this single-blind pilot RCT was to investigate the effect of pain biology education and group exercise classes compared to pain biology education alone for individuals with chronic low back pain (CLBP). Participants with CLBP were randomised to a pain biology education and group exercise classes group (EDX) (n = 20) or a pain biology education only group (ED) (n = 18). The primary outcome was pain (0-100 numerical rating scale), and self-reported function assessed using the Roland Morris Disability Questionnaire, measured at pre-intervention, post-intervention and three month follow up. Secondary outcome measures were pain self-efficacy, pain related fear, physical performance testing and free-living activity monitoring. Using a linear mixed model analysis, there was a statistically significant interaction effect between time and intervention for both pain (F[2,49] = 3.975, p < 0.05) and pain self-efficacy (F[2,51] = 4.011, p < 0.05) with more favourable results for the ED group. The effects levelled off at the three month follow up point. In the short term, pain biology education alone was more effective for pain and pain self-efficacy than a combination of pain biology education and group exercise classes. This pilot study highlights the OBJECTIVE: To examine whether pain physiology education was capable of changing pain cognitions and pain thresholds in patients with chronic fatigue syndrome (CFS) and chronic widespread pain. DESIGN: Double-blind randomized controlled trial. SETTING: Specialized chronic fatigue clinic in university hospital. PARTICIPANTS: A random sample of patients (N=48) with CFS patients (8 men, 40 women) experiencing chronic pain, randomly allocated to the control group (n=24) or experimental group (n=24). Two women in the experimental group did not complete the study because of practical issues (lack of time and restricted mobility). INTERVENTIONS: One individual pain physiology education session (experimental) or 1 pacing and self-management education session (control). MAIN OUTCOME MEASURES: Algometry, the Neurophysiology of Pain Test, and questionnaires evaluating pain cognitions-the Pain Coping Inventory, the Pain Catastrophizing Scale, and the Tampa Scale for Kinesiophobia-version CFS-were completed immediately before and immediately after the intervention. RESULTS: After the intervention, the experimental group demonstrated a significantly better understanding of the neurophysiology of pain (P<.001) and a reduction of the Pain Catastrophizing Scale subscale "ruminating" (P=.009) compared with controls. For these variables, moderate to large Cohen d effect sizes were revealed (.79-2.53). CONCLUSIONS: A 30-minute educational session on pain physiology imparts a better understanding of pain and brings about less rumination in the short term. Pain physiology education can be an important therapeutic modality in the approach of patients with CFS and chronic pain, given the clinical relevance of inappropriate pain.	RCT on CLBP with PNE/exercise and PNE only	pain, disability self-efficacy, fear, activity at post intervention and 3 months	post intervention PNE more effective for pain and self efficacy than PNE with exercise... not maintained 3m	Man Ther	15	4	382-7
M. Meeus, J. Nijns, J. Van Oosterwijk, V. Van Alsenoy and S. Truijien	2010	<b>Pain physiology education improves pain beliefs in patients with chronic fatigue syndrome compared with pacing and self-management education: a double-blind randomized controlled trial</b>	OBJECTIVE: To examine whether pain physiology education was capable of changing pain cognitions and pain thresholds in patients with chronic fatigue syndrome (CFS) and chronic widespread pain. DESIGN: Double-blind randomized controlled trial. SETTING: Specialized chronic fatigue clinic in university hospital. PARTICIPANTS: A random sample of patients (N=48) with CFS patients (8 men, 40 women) experiencing chronic pain, randomly allocated to the control group (n=24) or experimental group (n=24). Two women in the experimental group did not complete the study because of practical issues (lack of time and restricted mobility). INTERVENTIONS: One individual pain physiology education session (experimental) or 1 pacing and self-management education session (control). MAIN OUTCOME MEASURES: Algometry, the Neurophysiology of Pain Test, and questionnaires evaluating pain cognitions-the Pain Coping Inventory, the Pain Catastrophizing Scale, and the Tampa Scale for Kinesiophobia-version CFS-were completed immediately before and immediately after the intervention. RESULTS: After the intervention, the experimental group demonstrated a significantly better understanding of the neurophysiology of pain (P<.001) and a reduction of the Pain Catastrophizing Scale subscale "ruminating" (P=.009) compared with controls. For these variables, moderate to large Cohen d effect sizes were revealed (.79-2.53). CONCLUSIONS: A 30-minute educational session on pain physiology imparts a better understanding of pain and brings about less rumination in the short term. Pain physiology education can be an important therapeutic modality in the approach of patients with CFS and chronic pain, given the clinical relevance of inappropriate pain.	RCT with chronic fatigue syndrome with pain, N = 24 control group - pacing, self MX VS. n=24 PNE x 1 session indiv.	Algometry, knowledge, pain cognitions, coping, catastrophising, kinesiophobia - pre and immediately post 30 minute intervention	PNE -> better understanding of pain and reduced catastrophising.	Arch Phys Med Rehabil	91	8	1153-9
P. Brem	2010	<b>Can pain neurophysiology education contribute to improving the functional abilities of chronic pain patients from a physiotherapeutic perspective? [German]</b>	Physiotherapists employ one-to-one pain physiology education as a method of improving functional capacity in chronic pain population. This strategy is often used in chronic non-specific low back pain (CNLBP) in combination with active functional physiotherapy. The mechanism underpinning the effect of pain-physiology education is not definitively clarified. There is limited evidence concerning how specific pain physiology education influences different brain processes involved in the pain matrix and how patients may benefit from this approach. This case study discusses aspects of pain physiology education in clinical practice in a young man with CNLBP following a snowboard accident.	Clinical case study for chronic LBP young male			Manuelle Therapie	14	1	22-28

R. E. Johnson, G. T. Jones, N. J. G. L. Moseley	2007	<b>Active exercise, education, and cognitive behavioral therapy for persistent disabling low back pain</b>	STUDY DESIGN: A randomized controlled trial. OBJECTIVES: To determine 1) whether, among patients with persistent disabling low back pain (LBP), a group program of exercise and education using a cognitive behavioral therapy (CBT) approach, reduces pain and disability over a subsequent 12-month period; 2) the cost-effectiveness of the intervention; and 3) 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 who underwent functional magnetic resonance imaging scans during performance of a voluntary trunk muscle task under three conditions: directly after training in the task and, after one week of practice, before and after a 2.5-hour pain physiology education session. Before education there was widespread brain activity during performance of the task, including activity in cortical regions known to be involved in pain, although the task was not painful. After education widespread activity was absent so that there was no brain activation outside of the primary somatosensory cortex. The results suggest that pain physiology education markedly altered brain activity during performance of the task. The data offer a possible mechanism for difficulty in acquisition of trunk muscle training in people with pain and suggest that the change in activity associated with education may reflect reduced threat value.	Not PNE	Spine	32	15	1578-1585
G. L. Moseley	2005	<b>Widespread brain activity during an abdominal task markedly reduced after pain physiology education: fMRI evaluation of a single patient with chronic low back pain</b>	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 who underwent functional magnetic resonance imaging scans during performance of a voluntary trunk muscle task under three conditions: directly after training in the task and, after one week of practice, before and after a 2.5-hour pain physiology education session. Before education there was widespread brain activity during performance of the task, including activity in cortical regions known to be involved in pain, although the task was not painful. After education widespread activity was absent so that there was no brain activation outside of the primary somatosensory cortex. The results suggest that pain physiology education markedly altered brain activity during performance of the task. The data offer a possible mechanism for difficulty in acquisition of trunk muscle training in people with pain and suggest that the change in activity associated with education may reflect reduced threat value.	Case study CLBP Brain activity pre/post PNE Reduced brain activity in areas associated with pain experience after PNE	Aust J Physiother	51	1	49-52
G. Moseley, M. Nicholas and P. Hodges	2004	<b>A randomized controlled trial of intensive neurophysiology education in chronic low back pain</b>	OBJECTIVES: Cognitive-behavioral pain management programs typically achieve improvements in pain cognitions, disability, and physical performance. However, it is not known whether the neurophysiology education component of such programs contributes to these outcomes. In chronic low back pain patients, we investigated the effect of neurophysiology education on cognitions, disability, and physical performance. METHODS: This study was a blinded randomized controlled trial. Individual education sessions on neurophysiology of pain (experimental group) and back anatomy and physiology (control group) were conducted by trained physical therapist educators. Cognitions were evaluated using the Survey of Pain Attitudes (revised) (SOPAR(R)), and the Pain Catastrophizing Scale (PCS). Behavioral measures included the Roland Morris Disability Questionnaire (RMDQ), and 3 physical performance tasks; (1) straight leg raise (SLR), (2) forward bending range, and (3) an abdominal 'drawing-in' task, which provides a measure of voluntary activation of the deep abdominal muscles. Methodological checks evaluated non-specific effects of intervention. RESULTS: There was a significant treatment effect on the SOPAR(R), PCS, SLR, and forward bending. There was a statistically significant effect on RMDQ; however, the size of this effect was small and probably not clinically meaningful. DISCUSSION: Education about pain	RCT - PNE individual vs. back physiology in chronic low back pain Pain cognitions - catastrophizing, disability and physical performance PNE improved pain attitudes, catastrophizing and physical performance. Small but not clinically meaningful improvement in disability. Back school education not helpful.	Clinical Journal of Pain	20	5	324-30.
L. Moseley	2003	<b>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</b>	To identify why reconceptualization of the problem is difficult in chronic pain, this study aimed to evaluate whether (1) health professionals and patients can understand currently accurate information about the neurophysiology of pain and (2) health professionals accurately estimate the ability of patients to understand the neurophysiology of pain. Knowledge tests were completed by 276 patients with chronic pain and 288 professionals either before (untrained) or after (trained) education about the neurophysiology of pain. Professionals estimated typical patient performance on the test. Untrained participants performed poorly (mean +/- standard deviation, 55% +/- 19% and 29% +/- 12% for professionals and patients, respectively), compared to their trained counterparts (78% +/- 21% and 61% +/- 19%, respectively). The estimated patient score (46% +/- 18%) was less than the actual patient score (P < .005). The results suggest that professionals and patients can understand the neurophysiology of pain but professionals underestimate patients' ability to understand. The implications are that (1) a poor knowledge of currently accurate information about pain and (2) the underestimation of patients' ability to understand currently	Knowledge tests by patients and health professionals before and after PNE estimate patient performance PNE training improved knowledge in both groups, professionals underestimated patients ability to understand.	J Pain	4	4	184-9
G. L. Moseley	2004	<b>Evidence for a direct relationship between cognitive and physical change during an education intervention in people with chronic low back pain</b>	BACKGROUND: Unhelpful pain cognitions of patients with chronic low back pain (LBP) may limit physical performance and undermine physical assessment. It is not known whether a direct relationship exists between pain cognitions and physical performance. AIMS: To determine if a relationship exists between change in pain cognitions and change in physical performance when chronic LBP patients participate in a single one-to-one education intervention during which they have no opportunity to be active. METHODS: In a quasi-experiment using a convenience sample, moderately disabled chronic LBP patients (n=121) participated in a one-to-one education session about either lumbar spine physiology or pain physiology. Multiple regression analysis evaluated the relationship between change in pain cognitions measured by the survey of pain attitudes (SOPA) and the pain catastrophizing scale (PCS) and change in physical performance, measured by the straight leg raise (SLR) and standing forward bending range. RESULTS: There was a strong relationship between cognitive change and change in straight leg raise (SLR) and forward bending range (P < .001, respectively). P < .001, respectively. P < .001, respectively.	CLBP n = 121 - Individual session on PNE vs. Lumbar physiology education pain cognitions, catastrophizing, physical performance change in pain cognitions associated with change in physical performance	Eur J Pain	8	1	39-45
Moseley GL	2003	<b>Joining forces—combining cognition-targeted motor control training with group or individual pain physiology education: a successful treatment for chronic low back pain</b>	Patients: Direct lecture from a specifically trained PT. Hand-drawn images. Neurophysiology of pain -> Professionals: Seminar on neurophysiology of pain 3 hours, AV format. Chronic unremitting low back pain (LBP) is characterised by cognitive barriers to treatment. Combining a motor control training approach with individualised education about pain physiology is effective in this group of patients. This randomized comparative trial (I) evaluates an approach to motor control acquisition and training that considers the complexities of the relationship between pain and motor output, and (II) compares the efficacy and cost of individualized and group pain physiology education. After an "ongoing usual treatment"	RCT - 4w motor control and PNE program - 4x indiv session VS. 1 x 4hr group lecture Pain and disability individual PNE bigger decreases in pain and disability maintained 12m. Improvement in both groups - with combined motor control.	J Man Manip Therap	2003;11:88-94		
Moseley GL	2002	<b>Combined physiotherapy and education is efficacious for chronic low back pain</b>	Manual therapy, exercise and education target distinct aspects of chronic low back pain and probably have distinct effects. This study aimed to determine the efficacy of a combined physiotherapy treatment that comprised all of these strategies. By concealed randomisation, 57 chronic low back pain patients were allocated to either the four-week physiotherapy program or management as directed by their general practitioners. The dependent variables of interest were pain and disability. Assessors were blind to treatment group. Outcome data from 49 subjects (86%) showed a significant treatment effect. The physiotherapy program reduced pain and disability by a mean of 1.5/10 points on a numerical rating scale (95% CI 0.7 to 2.3) and 3.9 points on the 18-point Roland Morris Disability Questionnaire (95% CI 2 to 5.8), respectively. The number needed to treat in order to gain a clinically meaningful change was 3 (95% CI 3 to 8) for pain, and 2 (95% CI 2 to 5) for disability. A treatment effect was maintained at one-year follow-up. The findings support the efficacy of	8 PT sessions with manual therapy, exercise and PNE VS medical care for LBP Pain and disability Clinically meaningful improvement in combined PT approach	Aust J Physiother	2002;48:297-302		
Pruettnera EJ, Linn A	2012	<b>A neuroscience approach to managing athletes with low back pain</b>	Low back pain (LBP) is a common complaint within the athletic population and is commonly managed through a biomedical approach. The injured or damaged structure causing the LBP is identified and treated, and complete recovery from the episode is expected. Clinical experience shows us that often, athletes with LBP will not recover from their episode and may continue their sports participation despite persistent pain, or they may limit participation. Recent neuroscience research into the biology of pain suggests that clinicians involved in the management of athletes with LBP should embrace a biopsychosocial approach by engaging the brain and nervous system. This manuscript provides an overview of such a biopsychosocial approach, and presents information on the neurobiology of the athlete's pain experience.	NOT intervention based	Physical Therapy in Sport	13: 123-133		

Study	Type	Outcome measures	Results
Louw A, Diener I, Butler DS, Puenteadura EJ. <b>The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain.</b> 2011	<b>SR - 8 studies included</b>	Pain, disability, catastrophizing, and physical performance	↓ pain, increasing physical performance, ↓ perceived disability and ↓ catastrophisation in chronic LBP, chronic fatigue syndrome, widespread pain and chronic whiplash-associated disorders. <i>Very heterogenous sample</i>
Clarke CL, Ryan CG, Martin DJ. <b>Pain neurophysiology education for the management of individuals with chronic low back pain: systematic review and meta-analysis.</b> 2011	<b>SR - 2 studies included</b>	Pain, function, psychological function and social function	"Low evidence for small clinical improvement in short term pain and function" LBP ONLY
Pires D, Cruz EB & Caeiro C. <b>Aquatic exercise and pain neurophysiology education versus aquatic exercise alone for patients with chronic low back pain: a randomized controlled trial.</b> 2014	<b>RCT (CLBP)</b> Aquatic exercise + 2xPNE sessions n = 30 VS. Aquatic exercise n = 23 (12 sessions aquatic exercise over 6/52)	Pain + disability + kinesiophobia	Pain ↓ in PNE group
Louw A, Diener I, Landers MR & Puenteadura EJ. <b>Preoperative pain neuroscience education for lumbar radiculopathy: a multicenter randomized controlled trial with 1-year follow-up.</b> 2014	<b>RCT (Preop lumbar radiculopathy)</b> Usual care n = 34 VS. PNE with PT & booklet n = 31	1, 3, 6 and 12 m f/u on low back pain, leg pain, function and beliefs	No diff in pain between groups - PNE group was better prepared with behavioural change seen.
Ittersum MW, Wilgen CP, Schans CP, Lambrecht L, Groothoff JW, Nijs J. <b>Written pain</b>	<b>RCT (FM)</b> PNE booklet and phone call n = 53 VS. relaxation booklet and	Illness perception, catastrophizing, FM impact 0 at	PNE did not change impact of FM, catastrophizing or perceived symptoms
Gallagher L, McAuley J & Moseley GL. <b>A randomized-controlled trial of using a book of metaphors to reconceptualize pain and decrease catastrophizing in people with chronic pain.</b> 2013	<b>RCT (Chronic pain)</b> PNE via metaphors and stories n = 40 Vs. CBT style education	Pain and disability, PNE knowledge, catastrophizing at 3 weeks and 3 months.	Change in knowledge and catastrophizing in PNE group but no difference in pain or disability between groups.
Oosterwijck J, Meeus M, Paul L, Schryver M, Pascal A, Lambrecht L & Nijs J. <b>Pain physiology education improves health status and endogenous pain inhibition in fibromyalgia: a double-blind randomized controlled trial.</b> 2013	<b>RCT (FM)</b> Intensive PNE n = 15 VS. Control (pacing education) n = 15	2 weeks & 3 months f/u - efficacy of pain inhibition mechanisms, pressure pain threshold, pain cognition, behaviour and health status	↓ disability, catastrophizing, pain ↑ pain knowledge, mental health improved endogenous pain inhibition
Ryan CG, Gray HG, Newton M, Granat MH. <b>Pain biology education and exercise classes compared to pain biology education alone for individuals with chronic low back pain: a pilot randomised controlled trial.</b> 2010	<b>'RCT' (CLBP)</b> PNE + exercise n = 20 Vs. PNE only n = 18	Pain, disability, self-efficacy, fear, activity post intervention and 3 months f/u	Post intervention PNE more effective for pain and self-efficacy than PNE with exercise, but not maintained at 3m
Meeus M, Nijs J, Oosterwijck J, Alsenoy V & Truijten S. <b>Pain physiology education improves pain beliefs in patients with chronic fatigue syndrome compared with pacing and self-management education: a double-blind randomized controlled trial.</b> 2010	<b>RCT (Chronic fatigue syndrome with pain)</b> Control group: pacing, self MX n = 24 Vs. PNE x 1 session 30min individual n = 24.	Algometry, knowledge, pain cognitions, coping, catastrophizing, kinesiophobia - pre and immediately post intervention	PNE ↑ understanding of pain & ↓ catastrophizing.