EUROPEAN JOURNAL OF PSYCHOTRAUMATOLOGY
2025, VOL. 16, NO. 1, 2479923
https://doi.org/10.1080/20008066.2025.2479923

CLINICAL RESEARCH ARTICLE

OPEN ACCESS Check for updates

# The pain of PTSD: integrating persistent or chronic pain within emotional processing theory of posttraumatic stress disorder

Natalie Hellman [a,b,c], Stephanie M. Haft [a,d], Anna Woodbury [d,e], Andrew M. Sherrill [a] and Sheila A. M. Rauch [a,d]

[a]Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta, GA, USA; [b]Prisma Health Upstate, Prisma Health, Greenville, SC, USA; [c]Department of Family Medicine, University of South Carolina School of Medicine at Greenville, Greenville, SC, USA; [d]Joseph Maxwell Cleland Atlanta Veteran Affairs Medical Center, Atlanta GA, USA; [e]Department of Anesthesiology, Emory University, Atlanta, GA, USA

## ABSTRACT

**Background:** Posttraumatic stress disorder (PTSD) and chronic pain are devastating conditions that often co-occur. Current understanding of comorbid PTSD and chronic pain is limited, and treatment options are undereffective.
**Objective:** This paper presents a theoretical basis for conceptualising chronic pain symptoms within Emotional Processing Theory (EPT), the foundation for Prolonged Exposure (PE), an effective treatment for PTSD. EPT conceptualises the development and treatment of PTSD using a trauma structure that strongly overlaps with pain's neurobiology.
**Method:** This paper proposes a model of shared aetiology and treatment of comorbid PTSD and chronic pain, emphasising these shared neurobiological underpinnings. Discussion details how the comorbidity is maintained through parallel avoidance processes focused on: (1) trauma memories and reminders in PTSD preventing reduction of negative affect (extinction) and inhibitory learning, and (2) physical pain in chronic pain fuelling increased pain and reduced function.
**Results:** A conceptualisation is presented on how PTSD and chronic pain symptomology can be addressed within the EPT framework, increasing the confidence of providers and patients while addressing an important gap in the literature. Finally, recommendations for providers using PE with patients with PTSD and pain are provided including a case example and treatment plan based on real patients.
**Conclusions:** This model provides a clinically useful understanding of the underlying neurobiology for the co-occurrence of PTSD and chronic pain and offers direction for future research.

## El dolor del TEPT: integrando el dolor persistente o crónico con la teoría del procesamiento emocional del trastorno de estrés postraumático

**Antecedentes:** El trastorno de estrés postraumático (TEPT) y el dolor crónico son condiciones devastadoras que frecuentemente ocurren juntas. El entendimiento actual de la comorbilidad del TEPT y el dolor crónico es limitado, y las opciones de tratamiento no son efectivas.
**Objetivo:** Este artículo presenta una base teórica para la conceptualización de los síntomas de dolor crónico dentro de la Teoría del Procesamiento Emocional (TPE), la fundación para la Exposición Prolongada (EP), un tratamiento efectivo para el tratamiento del TEPT. TPE conceptualiza el desarrollo y tratamiento del TEPT usando una estructura del trauma que se superpone fuertemente con la neurobiología del dolor.
**Método:** Este artículo propone un modelo de etiología compartida y de tratamiento de TEPT y dolor crónico comórbidos, enfatizando que estos comparten fundamentos neurobiológicos. La discusión detalla cómo la comorbilidad es mantenida a través de procesos de evitación paralelos centrados en: (1) las memorias de trauma y los recordatorios en el TEPT previniendo la reducción del afecto negativo (extinción) y el aprendizaje inhibitorio, y (2) el dolor físico en el dolor crónico alimentando el aumento del dolor y una reducida función.
**Resultados:** Se presenta una conceptualización en cómo la sintomatología del TEPT y el dolor crónico puede ser abordado dentro del marco de la TPE, aumentando la confianza de los proveedores y los pacientes, mientras se aborda una brecha importante en la literatura. Finalmente, las recomendaciones para los proveedores en usar EP con los pacientes con TEPT y dolor será discutida incluyendo el ejemplo de un caso y plan de tratamiento basado en pacientes reales.
**Conclusiones:** Este modelo proporciona un entendimiento clínico útil de la neurobiología a la base de la co-ocurrencia del TEPT y el dolor crónico y ofrece direcciones para investigaciones futuras.

## ARTICLE HISTORY
Received 22 July 2024
Revised 12 February 2025
Accepted 6 March 2025

## KEYWORDS
PTSD; chronic pain; emotional processing theory; treatment; case vignette; neurobiology; comorbidity

## PALABRAS CLAVE
TEPT; dolor crónico; tratamiento; teoría de procesamiento emocional; neurobiología; comorbilidad

## HIGHLIGHTS

- PTSD and chronic pain are highly comorbid may have shared neurological processes.
- Integrating chronic pain development and treatment within the emotional processing theory framework can support integrated treatment and increase access to care for those with PTSD and chronic pain.
- An integrated model for treatment of PTSD and chronic pain using emotional processing theory to conceptualise the interplay of distress is presented.

**CONTACT** Sheila A. M. Rauch sheila.a.m.rauch@emory.edu Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, 12 Executive Park, 3rd Floor, Atlanta GA, 30329, USA

## 1. Introduction

Posttraumatic stress disorder (PTSD) and chronic physical pain are damaging and costly. PTSD and pain (PTSD-Pain) symptomology are highly comorbid, as 10–20% of patients with chronic pain also suffer from PTSD (Siqveland et al., 2017). Patients with PTSD-Pain report greater opioid use, more severe depression, anxiety, and more disability than patients with only one condition (Kind & Otis, 2019). Concerningly, patients with PTSD-Pain are more likely to engage in suicidal behaviours than patients with chronic pain alone (Finley et al., 2015). PTSD-Pain interventions are needed.

The current article presents a novel theoretical basis for conceptualising chronic pain symptoms within Emotional Processing Theory (EPT) (Foa & Kozak, 1986), the foundation for Prolonged Exposure (PE), an effective treatment for PTSD. We propose a model of shared aetiology and treatment of comorbid PTSD-Pain emphasising neurobiological underpinnings, discussing how PTSD-Pain is maintained through parallel avoidance processes (i.e. PTSD symptoms preventing extinction and inhibitory learning, and pain increasing distressed and reduced function). We then present how PTSD-Pain symptomology could be addressed within the EPT framework, with the goal of increasing provider and patient confidence to address PTSD-Pain and contribute to the literature. Specific recommendations for providers using PE with patients with PTSD-Pain are provided in a case example and treatment plan. This novel theory expands access and may improve clinical outcomes as providers trained in PE can use it to address co-occurring pain.

PTSD-Pain develop in the context of the individual including personality factors, such as extraversion and neuroticism, that may be related to the incidence and persistence of chronic pain (Rouch et al., 2023). This individual context provides a backdrop to development. PTSD-Pain may be complicated by the fact that PTSD and chronic pain symptoms predict one another and overlap (Ravn et al., 2018). For example, PTSD's hyperarousal symptoms may reflect the pain experience itself or be related to catastrophic misinterpretations of somatic sensations from their PTSD symptomology (Ravn et al., 2018). This makes the assessment of PTSD in patients with chronic pain (or assessing pain in patients with PTSD) challenging, as symptoms may be conceptualised as chronic pain, PTSD, or both diagnoses (Ravn et al., 2018). Further, PTSD and pain symptoms emerge in various trajectories (pain from a trauma-related injury, pain predating trauma exposure, or unrelated) (Bosco et al., 2013). Despite this entanglement, our field tends to address these diagnoses with stand-alone treatments (Murphy, Driscoll, et al., 2022) that do not address their interdependence – for PTSD-Pain has no identified gold standard intervention.

### 1.1 Posttraumatic stress disorder

While most people experience at least one trauma during their lifetime (70%), a minority of survivors develop PTSD (4%) (Kessler et al., 2017), which includes persistent symptomology (re-experiencing, avoidance, negative alterations in cognition and mood, and hyperarousal and reactivity) (American Psychiatric Association, 2013).

#### 1.1.1 Emotional Processing Theory for PTSD

Emotional Processing Theory (EPT (Foa & Kozak, 1986)) describes the development of anxiety disorders and PTSD and is the basis for Prolonged Exposure (PE). PE is a gold-standard PTSD intervention implemented worldwide (Foa et al., 2019), helping trauma survivors successfully recover from PTSD by approaching trauma memories and reminders. EPT (Foa & Kozak, 1986) posits and Figure 1a illustrates that following a traumatic event, a 'trauma structure' is formed that includes trauma-related stimuli, response, and meaning elements. These elements are connected based on the experience at the time of the trauma and may be helpful or unhelpful. Additionally, other traumatic or different life experiences can become connected to the trauma structure. Activation of any part of the trauma structure can activate the entire structure, including the intense fear and anxiety experienced during the trauma. For example, a survivor who sustained a traumatic injury and is undergoing surgery may incorporate their entire medical treatment experience and the resulting pain/scar as elements of their trauma structure. Even current pain from the injury or the feeling of injury-related vulnerability can become part of the trauma structure and activate the whole structure.

A trauma structure is adaptive when it reflects the current threat and promotes adaptive escape behaviours. When a trauma structure does not reflect the current environment, activation of the structure can lead to excessive responses (i.e. fear and anxiety, re-experiencing symptoms) and avoidance. Importantly, avoidance reinforces the sense of danger and prevents opportunities for a trauma survivor to experience corrective learning experiences, such as learning a trauma-related reminder (e.g. certain activity) is objectively safe and tolerable. For a visualised schematic map of a trauma memory structure and how it is modified with effective treatment, see details in Figure 1 and the corresponding case vignette.

#### 1.1.2 Emotional processing theory applied: prolonged exposure therapy for PTSD

PE aims to address unhelpful associations in the trauma structure and facilitate the incorporation of corrective information by approaching, instead of avoiding, the trauma memory and reminders. For a more detailed review, see Rauch & Foa (Rauch & Foa, 2006a). PE approaches the memory using two exposure techniques: *imaginal* (repeated approach of the trauma memory in imagination) and *in vivo* (repeated approach of trauma-related people, places, situations) to activate the stimuli, response, and meaning elements within the trauma structure. As the trauma survivor approaches the memory and reminders and feels the emotions connected to the trauma structure, new information is incorporated into the structure that disconfirms the unhelpful associations and allows new learning of more helpful associations (i.e. 'I can handle negative affect') (Rauch & Foa, 2006a). For instance, as survivors experience extinction within and across sessions, this experience disconfirms the sense that they might go crazy if they approach the trauma memory.

### 1.2 Chronic pain

An estimated 20% of the US population currently suffers from chronic pain (Dahlhamer et al., 2018), and chronic pain is the most prevalent disease worldwide (Nijs et al., 2021). Chronic pain is persistent or reoccurring pain lasting longer than three months and is further classified by the ICD-11 (Treede et al., 2015) into seven non-mutually exclusive but independent groups based on aetiology, underlying pathophysiological mechanisms, and body site. In the short term, acute pain is considered adaptive. It alerts organisms to potential threats and motivates self-preserving behaviours. However, when acute pain does not resolve and instead becomes chronic, pain is no longer adaptive. Instead, chronic pain promotes dysfunction via producing an exaggerated response and hypersensitivity, resulting in nociplastic rather than nociceptive or neuropathic pain (Fitzcharles et al., 2021).

For many patients with chronic pain, pain is widespread and non-specific (meaning no identified pathology or tissue damage would adequately explain their experience). Several terms with various clinical criteria are used to explain this type of pain (such as primary pain (Treede et al., 2015), centralised pain (Fitzcharles et al., 2021), chronic overlapping pain conditions (Fitzcharles et al., 2021), and nociplastic pain (Fitzcharles et al., 2021)). For clarity, we use the term primary pain. Despite no known aetiology, many people with chronic primary pain conditions (e.g. fibromyalgia, chronic lower back pain, chronic migraine, endometriosis, and chronic tension-type headache) experience central sensitisation, a phenomenon of nociceptive neurones within the central nervous system that show increased responsiveness to normal or subthreshold afferent input and promote pain hypersensitivity (Nijs et al., 2021). Central sensitisation is considered a key mechanism for the aetiology and maintenance of primary pain conditions (Nijs et al., 2021). Focusing on this mechanism of chronic pain (rather than the overarching condition) is clinically useful, as central sensitisation can be addressed in pharmacological and psychotherapy treatments (Bazzari & Bazzari, 2022).

#### 1.2.1 Chronic pain theory: the gate control theory of chronic pain

The gate control theory (Melzack & Wall, 1965), developed in 1965, is the basis for cognitive behavioural therapy for chronic pain (CBT-CP) (Thorn, 2017). This seminal theory proposed a theoretical mechanism, confirmed by decades of research that has identified specific neurobiological processes (Treede, 2016), exists within the dorsal horn of the spinal cord that modulates the transmission of pain signals (i.e. 'nociception') towards the brain. This mechanism functions like a 'gate' and is influenced by various factors including the release of neurochemicals related to emotion, to be more 'open' or 'closed'. A 'more open gate' indicates increased transmission of pain signals, while a 'closed gate' indicates reduced transmission. When central sensitisation is present, more frequent and/or greater number of signals progress towards the brain through spontaneous activity from peripheral or central nociceptors (gain control) – and when this frequently occurs, the patient experiences nociplastic, chronic primary pain resulting from altered pain modulation (Fitzcharles et al., 2021).

We acknowledge that several effective interventions for chronic pain do not focus heavily on gate control theory (such as Acceptance and Commitment Therapy (Gloster et al., 2020), Mindfulness-Based Stress Reduction (Anheyer et al., 2017), and Emotion Awareness and Expression Therapy (Lumley et al., 2017)). As gate control theory is the foundation for CBT-CP, we provide a focused review to establish a foundational knowledge base for readers who may be unfamiliar with this literature.

Similar to how PTSD is a generalised reaction to the trauma memory, central sensitisation appears to be a more generalised biological reaction in those with chronic pain. This phenomenon may be especially relevant for persons experiencing PTSD, as PTSD symptoms partially explain the relationship between trauma exposure and clinical indicators of central sensitisation (McKernan et al., 2019). Indeed, experiential avoidance may amplify central sensitisation and increase pain intensity scores, suggesting nociplastic

processes in PTSD patients (McKernan et al., 2019). While this is speculation from cross-sectional data and thus limited in inferential ability, it is an intriguing consideration that may be addressed through psychological interventions like PE.

### 1.2.2 Chronic pain theory: the fear avoidance model and predictive coding

Conceptually, scholars propose psychological models for chronic pain maintenance which are built upon the gate control theory of pain. One of the most well-known is the fear-avoidance model (Vlaeyen & Linton, 2000), which proposes that a person's perception of their pain leads them to either confront (and thereby have a life not limited by pain) or avoid (which then leads to disability or avoidance in life) pain. Therefore, it is the person's *expectation of potential* pain, not their lived experience, that promotes avoidant behaviours and perpetuates pain interference and disability. For instance, when patients fear pain from a primary pain condition (believing it indicates serious harm), they will avoid any activity (even if medically safe) that *could* cause any pain. Addressing the maladaptive use of avoidance is a treatment target in CBT-CP, as patients learn to manage their pain without labelling all avoidance as problematic and engage in a fulfilling life (Goldstein et al., 2019). Indeed, a recent meta-analysis found that CBT was effective in reducing fear avoidance of pain in patients with a primary pain condition (Yang et al. 2022).

Many constructionist and active inference models of chronic pain build upon the gate control theory and the fear-avoidance model. These updated models (e.g. predictive coding model (Kiverstein et al., 2022)) propose that patients with primary pain view pain as a prediction of physical bodily damage, which, when coupled with fearful appraisals of pain, promotes repetitive avoidance. This learned response can trap patients in a vicious positive feedback cycle, such as: patient receives uncomfortable sensory input during a safe activity > thinks 'pain is coming' > feels fear > increases bodily arousal > the 'gate' opens and

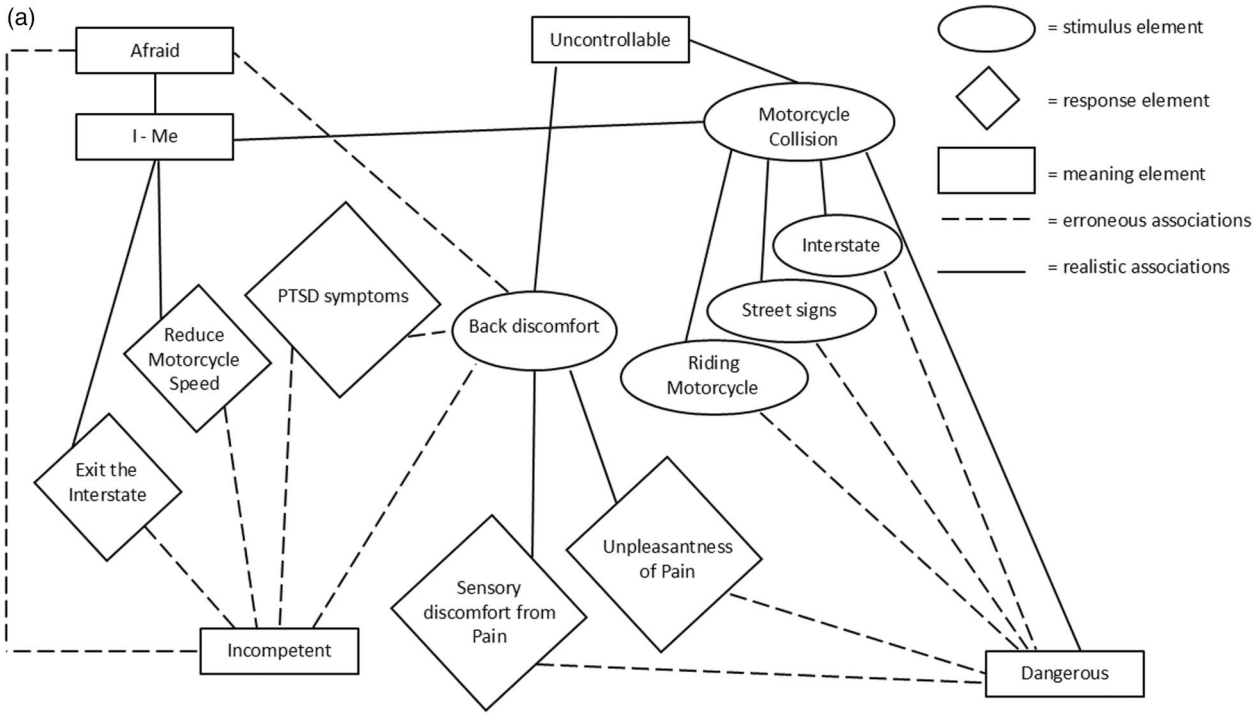



**Figure 1.** a. A schematic representation of a trauma memory network following a motor vehicle collision in a patient with PTSD-Pain prior to receiving treatment. This patient's memory holds a 'trauma structure,' linking stimuli, response, and meaning elements together. Stimulus elements may include people, places, or situations that were present at the time of the trauma. In this example, stimulus elements can be the interstate, street signs, experiencing back discomfort, or the act of riding a motorcycle. Response elements may include physiological reactions and behaviours that occurred at the time of the trauma. In this example, response elements can be PTSD symptoms, the sensory experience or unpleasantness of pain, and the behaviours to reduce motorcycle speed or exit the interstate. Meaning-making elements may include thoughts about what this traumatic event means to the survivor or the world. Meaning-making thoughts can include 'I can't control anything,' 'I am incompetent,' 'the crash is my fault', or 'the world is dangerous.' While some elements are connected via helpful associations (i.e. reflecting danger in the current environment), other elements are connected via unhelpful associations and reflect PTSD-pain symptomology (i.e. reflecting danger at the time of the trauma/pain, rather than their current danger risk). The solid line connections represent these helpful connections. For instance, the connection between danger and a motorcycle accident reflects a real threat in the current environment and is a helpful connection. The dotted line connections represent unhelpful connections or connections that overestimate the current risk in the environment. For example, the connection between incompetence and exiting the highway is not helpful, because exiting the highway is not related to incompetence.

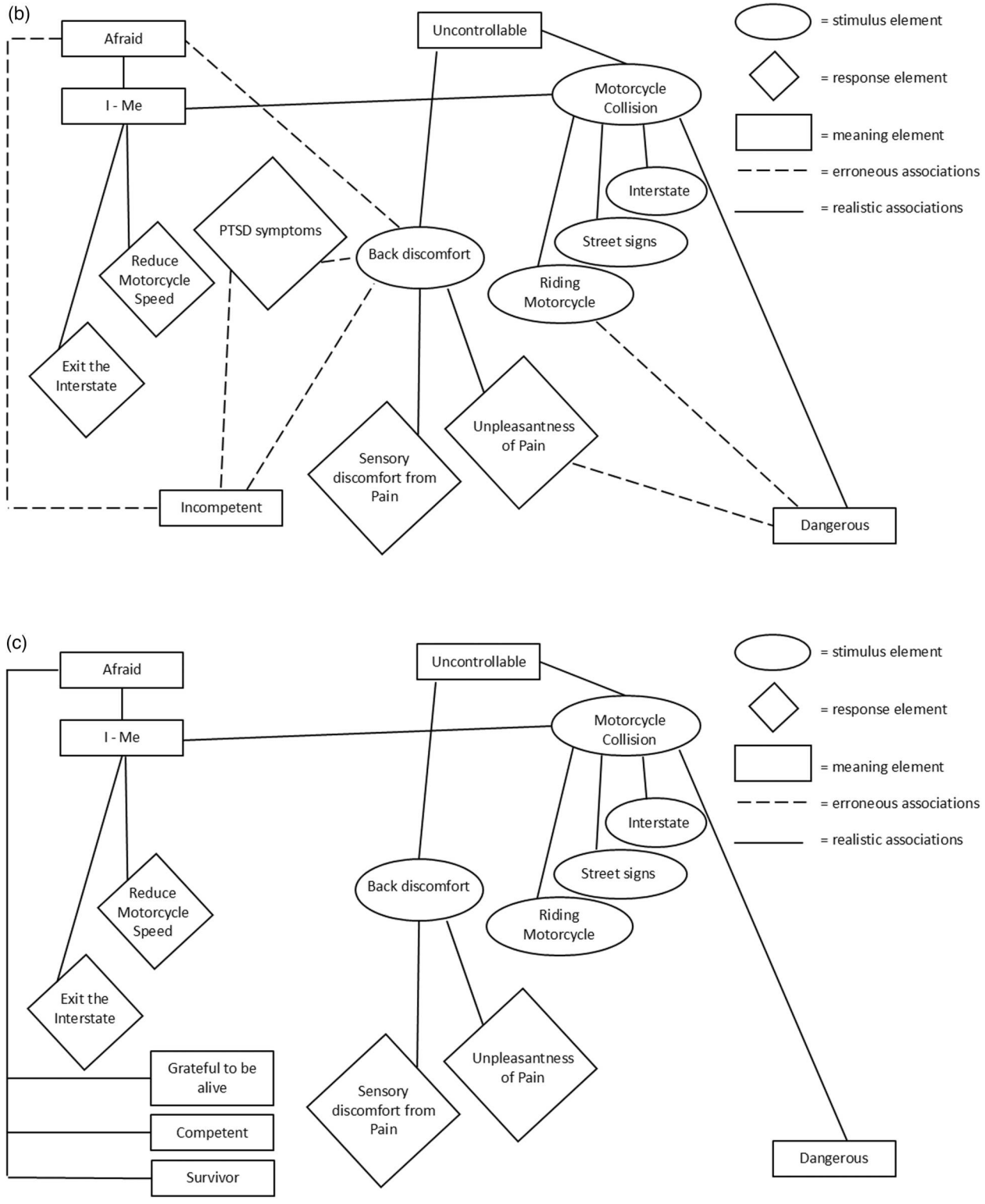



**Figure 1.** Continued b. A schematic representation of a trauma memory network of the same patient, during PE treatment. Through repeated *imaginal* and *in vivo* exposure exercises, which are individually tailored to the patient's unique presentation in each session, the patient is learning disconfirming information about the unhelpful associations that reduces or removes their connection in the trauma structure. For example, during PE, the patient may learn that street signs, by themselves, are not inherently dangerous and the dotted line connection is removed. c. A schematic representation of a trauma memory network of the same patient at the termination session of PE treatment. The unhelpful associations are no longer part of the trauma structure and the helpful associations remain (such as meaning-making elements like being grateful to be alive, believing they are competent as they acted quickly and were able to come out of the collision alive).

allows greater nociception to reach the brain > perceives more pain > thinks 'I am in danger' > increases bodily arousal > avoids activity to escape these sensations.

Taken together, these newer models suggest that patients with primary pain conditions could have their condition conceptualised as *originating* via gate control theory mechanisms (via greater central

sensitisation) and *maintained* via fear avoidance or predictive coding mechanisms (avoidant behaviour patterns and catastrophic misunderstandings of pain).

### 1.3 Approaches to comorbid PTSD and chronic pain

PTSD-Pain psychotherapy treatment approaches commonly take a sequential or 'stepped' approach and administer disorder-specific treatments sequentially (Murphy, Driscoll, et al., 2022). One example (Åkerblom et al., 2022) is patients completing two treatment courses (PE first, followed by CBT-CP) with providers using two distinct theoretical models (EPT and gate control theory) to address PTSD-Pain. While this sequential approach may address some symptoms, it may not simultaneously address both conditions (Åkerblom et al., 2022). Further, this isolation method is costly, burdensome for patients, and may promote attrition as it doubles treatment time and effort.

Another approach is parallel treatment, in which patients receive treatment for each condition from separate specialty care teams without explicit coordination (Murphy, Driscoll, et al., 2022). This model also has issues (Lumley et al., 2022; Murphy, Driscoll, et al., 2022), as patients may be overwhelmed by the number of appointments and providers and feel a sense of competing priorities (Murphy, Driscoll, et al., 2022). Concerningly, effective independent psychotherapies for PTSD and chronic pain each have high attrition rates (50% for PTSD (Schnurr et al., 2022); 20% for CBT-CP (Murphy, Cordova, et al., 2022)), making it difficult for patients to receive relief from their full symptomology.

#### 1.3.1 Research on integrated PTSD-pain treatment models

Integrated treatments synergistically address symptomology from both disorders, employing the overlapping symptomology to fuel change and reduce patient distress and healthcare utilisation. This is an emerging frontier of treatment research, and several combined models (fear-avoidance model (Vlaeyen & Linton, 2000); mutual maintenance model (Sharp & Harvey, 2001); shared vulnerability model (Asmundson et al., 2002); perpetual avoidance model (Liedl & Knaevelsrud, 2008); triple vulnerability model (Barlow, 2004) have garnered research support (Murphy, Driscoll, et al., 2022). These models offer useful insight and can enhance existing exposure-based treatments by extending their purview. However, much of this prior work offers distinct models that produce distinct interventions that are not ready for dissemination. A systematic review and meta-analysis on psychotherapies for comorbid PTSD and pain found cognitive–behavioural and mindfulness-based interventions show efficacy in using varying degrees of exposure-based strategies to reduce PTSD and pain severity (Goldstein et al., 2019). This emerging area is exciting and shows the promise of integrated approaches. Some approaches conceptualise the conditions as separate issues and develop techniques for each (The PATRIOT programme (Otis et al., 2024), and others take an integrated approach (Emotional Awareness and Expression Therapy (Lumley et al., 2022)).

Additionally, the overlap of avoidance processes in both PTSD and chronic pain development suggests that we may adapt PE to address pain. PE aligns with CBT-CP principles and PE-related exposures can be integrated into CBT-CP techniques, thereby tapping into the CBT-CP providers network to address PTSD-Pain (Bosco et al., 2013). While promising, this idea involves significant implementation and dissemination efforts and is sequential. Contrastingly, it is fruitful to explore if integrating CBT-CP elements into PE would be a productive integrated treatment approach for PTSD-Pain patients.

### 1.4 Theoretical basis for enveloping pain symptoms within emotional processing theory

#### 1.4.1 Emotional processing theory for PTSD-pain

PE is individualised, allowing robust pain intervention elements to be seamlessly integrated into a PE treatment course. Our perspective builds upon previous work (Bosco et al., 2013; Rauch & Foa, 2006b) by bringing together distinct literatures into one existing PTSD theory that explicitly discusses how these ideas apply to many pain conditions, allowing PE providers to address both PTSD and pain. Providing this integrated model using two disseminated and effective psychotherapies reduces the expense of developing new dissemination and implementation efforts. Instead, it builds upon the skills of PE providers already serving on the frontlines of treating PTSD-Pain and can be quickly rolled out to patients. This approach, while in need of evaluation through rigorous clinical trials and independent research groups, could provide an important contribution to the field.

Another strength of EPT is that it is a psychological theory, *not* a biological theory. In this way, EPT does not posit what biological underpinnings are involved. EPT focuses on identifying markers of treatment progress within sessions and guides providers to achieve the optimal outcome. However, many PTSD-related neurohormones, neurotransmitters, and inflammatory system factors that have been implicated in effective PE are also implicated in pain processing (Scioli et al., 2015). Within an EPT framework and our reconceptualisation of pain symptoms as a part of a trauma structure, changes in these biological processes during PE could be interpreted as reflections of change and treatment progress for PTSD pain-related concerns.

### 1.4.2 Neuroanatomical correlates PTSD and pain

Work examining dual-diagnosed PTSD-Pain is limited; however, both PTSD and pain have been studied independently. Reviewing these segregated literatures shows that the neural pathways implicated in PTSD strongly overlap with the 'pain matrix' – the neural pathways implicated in pain processes. Figure 2 provides a summary of the neural pathways that transmit neural signals to the brain (Cioffi, 2017), and Figure 3 depicts previously identified supraspinal structures for PTSD (3a (Rauch et al., 2015)), chronic pain (3b (Martucci & Mackey, 2018)), and how these regions may contribute to the interdependence of PTSD-Pain (3c).

The following is an oversimplified review of the neurobiological research on PTSD and pain. It follows the flow of neural signalling from a somatic region (peripheral) towards the central processing of pain (using pathways highlighted within the gate control theory of pain), ultimately culminating in the somatic and emotional experience of pain. This overview is intended to provide a foundational understanding and to highlight that PTSD and pain have many overlapping neurobiological processes.

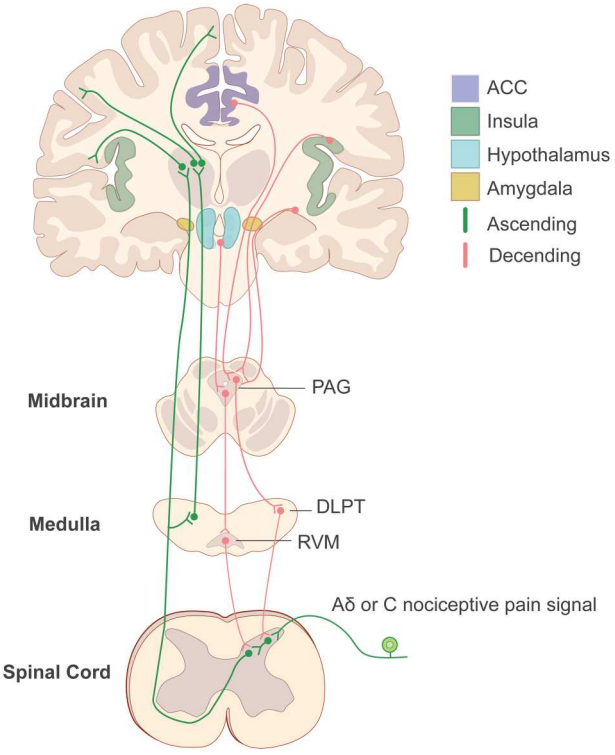



**Figure 2.** The descending pain modulation system (adapted from (Martucci et al., 2014)). The spinal pathway from a noxious stimulus (depicted by a green circle outside of the spinal cord) travelling via Aδ and C nociceptive fibres within the dorsal horn of the spinal cord and relayed via the subcortical structures to the cortical structures. This figure depicts the neural pathways that correspond to the gate control theory of chronic pain and are targeted in Cognitive Behavioral Therapy for Chronic Pain. Illustration by Bona Kim; used with permission from ©Emory University. Abbreviations: ACC – anterior cingulate cortex, DLPT – dorsolateral pontine tegmentum, RVM – rostroventral medulla.

Pain typically begins in the periphery with injury or inflammation resulting in nociceptive input, carried by Aδ and C fibres, which synapse in the spinal cord and are carried up the ascending pathway and pass through the 'gate' identified within gate control theory to the brain (Figure 2). The pain response is interpreted through the 'pain matrix', identified as the somatosensory cortex and pain processing centres such as the anterior cingulate cortex, insula, hypothalamus, and amygdala. These areas (also involved in processing emotion, memory, and other functions) interpret pain and may modulate the pain through descending pathways, including descending inhibitory controls. Notably, activation of non-nociceptive, wide dynamic range neurones in the periphery may serve to inhibit transmission of these painful sensations in normal, healthy individuals, which serves as a basis for many applications of the gate control theory. Although PTSD is no longer categorised as an anxiety disorder (American Psychiatric Association, 2013), PTSD has been conceptualised from a neurobiological perspective as a disorder of fear learning and extinction processes, with studies examining circuitry involved in both fear learning and extinction of fear learning to identify treatment targets to facilitate extinction of fear and re-engagement with trauma-related elements (Rauch et al., 2015) (Figure 2a).

Neuroimaging studies reveal areas of activation implicated in PTSD and pain processing (Figure 3). Several cortical regions overlap in chronic pain and PTSD, specifically the prefrontal cortex, the anterior cingulate cortex, the thalamus, the insula, the hippocampus, the locus coeruleus, and the periaqueductal grey (Figure 3c). Many of these areas have been implicated in nociplastic pain due to aberrant pain processing and central sensitisation (Martucci et al., 2014). The orbitofrontal cortex, well-studied in PTSD for its role in learning and re-learning, has also emerged as an area of potential importance in pain. This prominent overlap between PTSD and chronic pain suggests underlying neuroanatomical pathways that may be targeted to address both conditions through shared mechanisms. While neuroimaging literature specifically investigating PTSD-Pain processes simultaneously is limited, one fMRI study (Mickleborough et al., 2011) found that when patients with PTSD were presented with trauma-related stimuli (thus activating the trauma structure), it produced an adaptive and defensive pain response (responding as if a threat is present and requires a protective response). This interesting finding can guide future research.

Taken together, EPT provides a useful framework for understanding PTSD-Pain and showcases psychobiological pathways underlying symptoms. Therefore, using EPT to address pain concerns alongside PTSD symptoms may be useful, as pain may be a co-

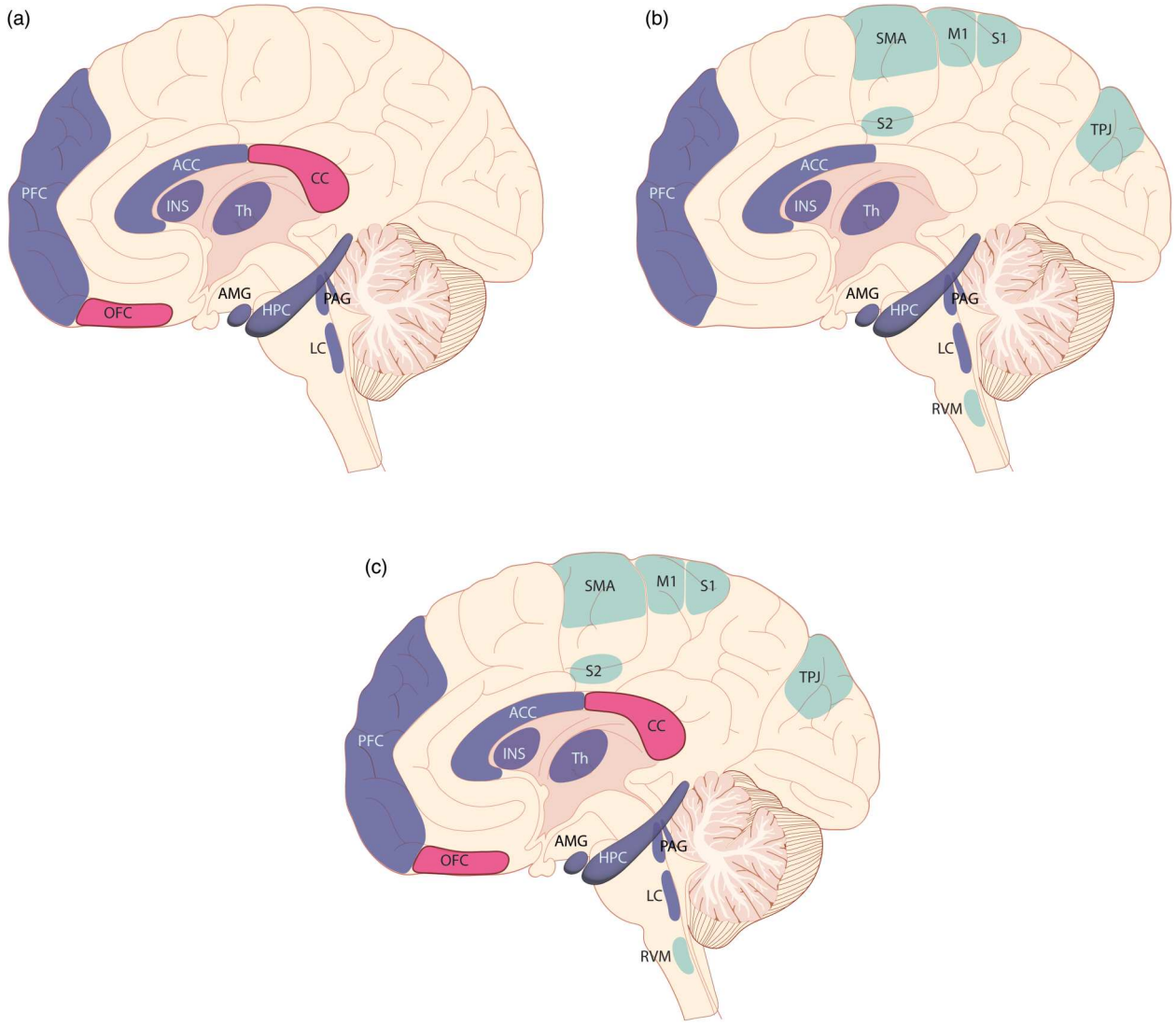



**Figure 3.** Summary of the main neural pathways implicated in both PTSD and pain processing based on prior literature. The multiple cortical and subcortical structures involved in PTSD are depicted in red (Figure 3a; adapted from (Rauch et al., 2015)), and pain is depicted in blue (Figure 3b; adapted from (Martucci & Mackey, 2018)). The neural regions depicted in purple showcases the overlapping regions implicated in both PTSD and pain (fully combined in Figure 3c). These figures combine findings from previously segregated literature into one clear overlapping figure and highlight the overlapping neuroanatomy within PTSD and chronic pain conditions. Illustration by Bona Kim; used with permission from ©Emory University. Abbreviations: ACC – anterior cingulate cortex, AMG – amygdala, CC – cingulate cortex, HPC – hippocampus, INS – insula, LC – locus coeruleus, M1 – primary motor cortex, OFC – orbitofrontal cortex, PAG – periaqueductal grey, PFC – prefrontal cortex; RVM – rostroventral medulla, SMA – supplementary motor area, S1 – primary somatosensory cortex, S2 – secondary somatosensory cortex; TPJ – temporal parietal junction; Th – thalamus.

manifested symptom of dysregulated emotional processing and reflective of PTSD symptoms.

### *1.4.3 Case example*

To illustrate how a patient diagnosed with PTSD-Pain may experience and benefit from PE that addresses pain concerns, we provide an anonymised case vignette and corresponding trauma structures (Figure 1) based on an amalgam of real PTSD-Pain patients who received PE by authors NH and AS. A schematic map of this patient's trauma structure showcases the linked stimulus, response, and meaning elements, how these are interconnected, and how the clinical presentation was conceptualised at PE initiation (Figure 1a), during PE (Figure 1b), and at treatment termination (Figure 1c).

Patient X is an African American man in his early 20s who experienced a motorcycle accident one year ago, when he was riding his motorcycle on the interstate within speed limits and crashed into a street sign and fractured three ribs. During PTSD-Pain assessment, Mr. X reported that he was diagnosed with a primary pain condition and that recurrent back discomfort resulting from the injury cued intrusive memories of the accident. To cope with his discomfort, Mr. X restricted gross motor movements (e.g. 'guarding' from pain) to prevent activating both physical pain and the trauma memory (i.e. passive avoidance). He reported attempting to ride his motorcycle on the interstate several times since the accident, but he decelerated and exited as soon as he felt back

discomfort and saw street signs. His avoidance coping strategies and unhelpful thoughts prevented him from learning that the trauma memory and trauma reminders are safe and that his pain, while uncomfortable, is not a signal of danger. Mr. X medically recovered from his nociceptive injuries and completed physical therapy, yet he still experienced frequent pain.

In Mr. X's course of outpatient PE, he identified *in vivo* exposures targeting both PTSD and pain-related avoidance simultaneously. Mr. X's *in vivo* hierarchy included, from least to most activating, the following behaviours: a gentle yoga class, riding a stationary cycle, weightlifting, playing basketball, riding his motorcycle in his neighbourhood, and riding his motorcycle on the interstate. These exposures aimed not to eliminate discomfort (including his pain) but to help Mr. X experientially learn that his affective responses to these trauma reminders are not dangerous and reduce as he engages in activities-thereby learning that body discomfort is safe and not a threat signal. During *imaginal* exposure to the trauma memory, Mr. X reported strong activation of back pain when revisiting the hot spot-specifically hearing his ribs break during the collision. During the first hot spot repetition, Mr. X positioned his body as if bracing for impact, and as he described the collision, he reached behind his back as if to soothe it. The provider praised Mr. X's strong effort and provided feedback to not physically act out the memory or provide any soothing touch (a safety behaviour). Over repeated *imaginal* exposures, Mr. X experienced the extinction of fear-based responses and back pain simultaneously. After one *imaginal* exposure near the end of treatment, Mr. X reported, 'It was the worst pain I ever felt, but that part is just a memory now – getting back my life makes everything worthwhile.' He returned to riding his motorcycle on the interstate and he learned that motorcycle riding is relatively safe, and that fear can be adaptive in unsafe environments. Mr. X and his therapist decided PE was complete after Mr. X reported reduction of negative affect to the trauma memory and returned to all previous activities.

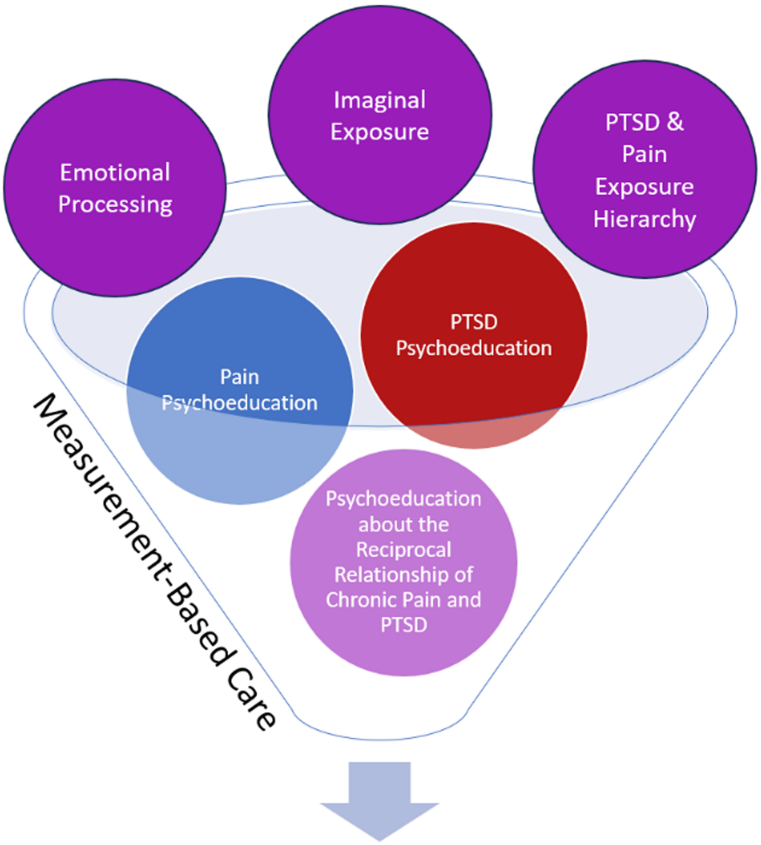
## 1.5 Therapeutic implications and clinical recommendations

### 1.5.1 Assessment of chronic pain and PTSD symptoms

PTSD-Pain assessment often occurs across multiple settings. Prior to identifying appropriate treatment goals, providers should take a thoughtful approach to assessment using evidence-based assessment tools for both PTSD (e.g. screening via PTSD Checklist for the DSM-5 (Bovin et al., 2016); diagnosis via the Clinician-Administered PTSD Scale for the DSM-5 (Weathers et al., 2018)) and chronic pain (e.g. Defense and Veterans Pain Rating Scale (Polomano et al., 2016), McGill Pain Questionnaire (Hawker et al., 2011), Numerical Rating Scale (Hawker et al., 2011), and Visual Analog Scale (Hawker et al., 2011)). Reviewing medical records (when possible) to examine PTSD-Pain symptoms provides a robust clinical picture prior to treatment initiation. Additionally, PTSD-Pain symptoms must be contextualised within cultural factors, across symptom experience, patient report, and assessment of symptoms. This is necessary, as individuals with marginalised racial and ethnic identities are at a greater risk of more prevalent and more severe PTSD (Kind & Otis, 2019) and pain symptoms. In the context of pain management and treatment, clinician implicit bias interferes with clinical decision-making, negatively impacting clinician-patient interactions, and also predicts poor management of pain (Burgess et al., 2006). Historically, biologically based explanations were used to explain pain disparities in Black, Indigenous, and other people of colour (i.e. the speculation that racial groups process pain differently) – an idea dating back to chattel slavery and colonisation (Owens & Fett, 2019). Patient-centred communication and practising measurement-based care can address this bias by centring and prioritising patient concerns (Burgess et al., 2006).

### 1.5.2 Clinical recommendations

The clinical considerations from this theoretical perspective and suggested possibilities expand upon previously proposed in the literature (Åkerblom et al., 2022; Bosco et al., 2013; Goldstein et al., 2019; Rauch & Foa, 2006b) by explicitly linking them to EPT. Figure 4 outlines how pain psychoeducation and exposures can be integrated into a PE treatment plan (based on Foa et al., 2019; Murphy, Cordova, et al., 2022; Thorn, 2017). Psychoeducation is a core feature of both PE and pain management and combining both into one psychoeducation element (facilitating approaching trauma-related and pain-related internal stimuli) may be beneficial. Providers can determine how trauma stimuli can evoke pain, educate patients that pain does not always indicate tissue damage, and instil hope that patients can handle their distress. As subjective units of distress (SUDs) ratings encompass all affective experiences, pain could be measured as part of SUDs ratings during *imaginal* and *in vivo* exposures. Providers can also explain how safety behaviours apply to pain experiences – and set behavioural expectations that any safety behaviour is not to be used during exposures. In addition to making efforts to identify PTSD-related avoidance strategies (pre-treatment and during treatment), providers may also identify pain-related avoidance strategies (e.g. physical guarding when pain is present) that may interfere with PTSD-Pain recovery.




| Proposed Integration Strategies. | |
|---|---|
| Prolonged Exposure for PTSD Elements | Exposure Therapy for Pain Elements |
| Treatment component: psychoeducation | |
| • PTSD psychoeducation<br>• Common reactions to trauma<br>• PTSD avoidance cycle<br>• PTSD-based exposure rationale | • Pain education (i.e., not all pain is dangerous)<br>• Pain avoidance cycle<br>• Pain-based exposure rationale |
| Treatment component: breathing retraining | |
| • Breathing retraining | • Relaxation training (deep breathing) |
| Treatment component: *in vivo* exposures | |
| • PTSD based fear and avoidance hierarchy<br>• Begin PTSD-exposures which align values and goals | • Pain based fear and avoidance hierarchy<br>• Begin pain exposures which align with values and goals<br>• Activity pacing (appropriate levels of physical activity without overexertion) |
| Treatment component: *imaginal* exposure | |
| • Imaginal exposure to index trauma<br>• Emotional processing<br>• Limit PTSD-related safety behaviors | • Provide psychoeducation regarding affective pain may be present during imaginal<br>• Limit pain-related safety behaviors |
| Treatment component: Discharge and Termination | |
| • Review treatment gains<br>• Anticipate future challenges<br>• Relapse prevention skills for PTSD concerns<br>• Encourage exposure as a lifestyle | • Review treatment gains<br>• Anticipate future challenges<br>• Relapse prevention skills for pain concerns<br>• Encourage exposure as a lifestyle |

**Figure 4.** A visual depiction of the components of Prolonged Exposure and Exposure Based CBT for Chronic Pain components that are complementary and a proposed integration strategy. PE content elements (Owens & Fett, 2019), exposure therapy for pain elements (Goldstein et al., 2019; Schnurr et al., 2022).

These examples of PTSD-Pain avoidance strategies can be addressed during PE exposures. *In vivo* exposures can encompass pain-evoking (yet medically safe) situations and allow the patient to connect to meaningful and enjoyable activities in the context of living with chronic pain. This would promote the reduction of negative affective responses to pain and trauma-related cues and inhibitory learning of PTSD and pain-related stimuli, ultimately achieving a reduction of PTSD-Pain. Further, this may provide additional opportunities for cognitive shifts related to incompetence (e.g. 'I cannot handle being in pain' to 'I can tolerate pain') and enhance corrective learning within PE. Although the efficacy of this proposed integrated PTSD-Pain treatment has not been formally evaluated, current findings appear to support this approach (Rauch & Foa, 2006b).

### *1.5.3 Future research*

It is imperative that additional research focuses on: (1) the efficacy of integrating CBT-CP elements into PE, (2) whether current and/or novel PTSD-Pain treatments need further development (3) the adaptation of other gold-standard interventions for PTSD-Pain, (4) developing a PTSD-Pain screening measure to further integrate PTSD-Pain care and reduce patient burden, and (5) delineation of robust treatments across pain conditions (including pain from traumatic injury) and individual differences (including race, gender, sex, ethnicity, pre-existing pain, and personality factors).

## 2. Conclusion

Many patients suffering from PTSD also suffer from chronic pain, and current treatments are undereffective. This paper proposes an extension of EPT to pain which allows providers trained in PE, a well-established treatment for PTSD, to accommodate pain into their case conceptualisation and treatment of those with PTSD-Pain. By integrating chronic pain symptoms into EPT, the shared neurobiological and psychological mechanisms that maintain PTSD-Pain symptoms are clarified. The case vignette provides an illustration of an integrated treatment approach. This paper offers a framework for PE clinicians treating PTSD-Pain patients to emulate and integrate pain-related treatment targets into their treatment plan for PTSD.

## Acknowledgements

We would like to thank Emory University's medical illustrator, Bona Kim, MA, CMI, RN, for creating our neuroanatomy figures.

## Author contributions

Natalie Hellman – conceptualisation, methodology, original draft, reviewing, and editing. Stephanie Haft, Andrew Sherrill, Anna Woodbury, and Sheila Rauch – conceptualisation, revisions of the original draft, with continued review and edits.

## Author note

N. Hellman transitioned institutions from Emory University to the University of South Carolina in Greenville in August 2024 and contributed to this project at both institutions. S. Haft transitioned institutions from Emory University to the VA in August 2024 and contributed to this project at both institutions.

## Disclosure statement

No potential conflict of interest was reported by the authors.

## Funding

Effort on this manuscript was partly supported by the Wounded Warrior Project (N. Hellman, S. Haft, A. Sherrill, and S. Rauch) and by the US Department of Veterans Affairs Rehabilitation Research and Development Service Career Development Awards RX003227 (A. Woodbury).

## Data availability statement

There is no dataset associated with this manuscript.

## Disclaimer

The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs or the US government.

## ORCID

*Natalie Hellman* http://orcid.org/0000-0003-4811-2346
*Stephanie M. Haft* http://orcid.org/0009-0002-7542-4708
*Anna Woodbury* http://orcid.org/0000-0003-0644-6049
*Andrew M. Sherrill* http://orcid.org/0000-0002-7743-745X
*Sheila A. M. Rauch* http://orcid.org/0000-0001-9686-4011

## References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Association.

Anheyer, D., Haller, H., Barth, J., Lauche, R., Dobos, G., & Cramer, H. (2017). Mindfulness-Based stress reduction for treating Low back pain. *Annals of Internal Medicine*, *166*(11), 799–807. https://doi.org/10.7326/M16-1997

Asmundson, G. J. G., Coons, M. J., Taylor, S., & Katz, J. (2002). PTSD and the experience of pain: Research and clinical implications of shared vulnerability and mutual maintenance models. *The Canadian Journal of Psychiatry*, *47*(10), 930–937. https://doi.org/10.1177/070674370204701004

Åkerblom, S., Perrin, S., Fischer, M. R., & McCracken, L. M. (2022). Prolonged exposure for pain and comorbid

PTSD: A single-case experimental study of a treatment supplement to multiprofessional pain rehabilitation. *Scandinavian Journal of Pain*, *22*(2), 305–316. https://doi.org/10.1515/sjpain-2021-0100

Barlow, D. H. (2004). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. Guilford Press.

Bazzari, A. H., & Bazzari, F. H. (2022). Advances in targeting central sensitization and brain plasticity in chronic pain. *The Egyptian Journal of Neurology, Psychiatry and Neurosurgery*, *58*(1), 38. https://doi.org/10.1186/s41983-022-00472-y

Bosco, M. A., Gallinati, J. L., & Clark, M. E. (2013). Conceptualizing and treating comorbid chronic pain and PTSD. *Pain Research and Treatment*, *2013*, 1–10. https://doi.org/10.1155/2013/174728

Bovin, M. J., Marx, B. P., Weathers, F. W., Gallagher, M. W., Rodriguez, P., Schnurr, P. P., & Keane, T. M. (2016). Psychometric properties of the PTSD checklist for diagnostic and statistical manual of mental disorders-fifth edition (PCL-5) in veterans. *Psychological Assessment*, *28*(11), 1379–1391. https://doi.org/10.1037/pas0000254

Burgess, D. J., Van Ryn, M., Crowley-Matoka, M., & Malat, J. (2006). Understanding the provider contribution to race/ethnicity disparities in pain treatment: Insights from dual process models of stereotyping. *Pain Medicine*, *7*(2), 119–134. doi:10.1111/j.1526-4637.2006.00105.x

Cioffi, C. L. (2017). Modulation of glycine-mediated spinal neurotransmission for the treatment of chronic pain. *Journal of Medicinal Chemistry*, *61*(7), 2652–2679.

Dahlhamer, J., Lucas, J., Zelaya, C., Nahin, R., Mackey, S., DeBar, L., Kerns, R., Von Korff, M., Porter, L., & Helmick, C. (2018). Prevalence of chronic pain and high-impact chronic pain Among adults - United States, 2016. *MMWR. Morbidity and Mortality Weekly Report*, *67*(36), 1001–1006. https://doi.org/10.15585/mmwr.mm6736a2

Finley, E. P., Bollinger, M., Noël, P. H., Amuan, M. E., Copeland, L. A., Pugh, J. A., Dassori, A., Palmer, R., Bryan, C., & Pugh, M. J. V. (2015). A national cohort study of the association between the polytrauma clinical triad and suicide-related behavior among US veterans who served in Iraq and Afghanistan. *American Journal of Public Health*, *105*(2), 380–387. https://doi.org/10.2105/ajph.2014.301957

Fitzcharles, M. A., Cohen, S. P., Clauw, D. J., Littlejohn, G., Usui, C., & Häuser, W. (2021). Nociplastic pain: Towards an understanding of prevalent pain conditions. *Lancet*, *397*(10289), 2098–2110. https://doi.org/10.1016/s0140-6736(21)00392-5

Foa, E. B., Hembree, E. A., Rothbaum, B. O., & Rauch, S. A. M. (2019). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences: Therapist guide* (2nd ed). Oxford University Press. xi, 181-xi, 181.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*(1), 20–35. https://doi.org/10.1037/0033-2909.99.1.20

Gloster, A. T., Walder, N., Levin, M. E., Twohig, M. P., & Karekla, M. (2020). The empirical status of acceptance and commitment therapy: A review of meta-analyses. *Journal of Contextual Behavioral Science*, *18*, 181–192. https://doi.org/10.1016/j.jcbs.2020.09.009

Goldstein, E., McDonnell, C., Atchley, R., Dorado, K., Bedford, C., Brown, R. L., & Zgierska, A. E. (2019). The impact of psychological interventions on posttraumatic stress disorder and pain symptoms: A systematic review and meta-analysis. *The Clinical Journal of Pain*, *35*(8), 703–712. doi:10.1097/AJP.0000000000000730

Hawker, G. A., Mian, S., Kendzerska, T., & French, M. (2011). Measures of adult pain: Visual analog scale for pain (VAS pain), numeric rating scale for pain (NRS pain), McGill pain questionnaire (MPQ), short-form McGill pain questionnaire (SF-MPQ), chronic pain grade scale (CPGS), short form-36 bodily pain scale (SF-36 BPS), and measure of intermittent and constant osteoarthritis pain (ICOAP). *Arthritis Care & Research*, *63*(Suppl 11), S240–S252. https://doi.org/10.1002/acr.20543

Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Benjet, C., Bromet, E. J., Cardoso, G., Degenhardt, L., Girolamo, G., Dinvolova, R. V., Ferry, F., Florescu, S., Gureje, O., Haro, J. M., Huang, Y., Karam, E. G., Kawakami, N., Lee, S., Lepine, J., Levinson, D., ... Koenen, K. C. (2017). Trauma and PTSD in the WHO world mental health surveys. *European Journal of Psychotraumatology*, *8*(sup5), 1–16. https://doi.org/10.1080/20008198.2017.1353383

Kind, S., & Otis, J. D. (2019). The interaction between chronic pain and PTSD. *Current Pain and Headache Reports*, *23*(12), 1–7. https://doi.org/10.1007/s11916-019-0828-3

Kiverstein, J., Kirchhoff, M. D., & Thacker, M. (2022). An embodied predictive processing theory of pain experience. *Review of Philosophy and Psychology*, *13*(4), 973–998. https://doi.org/10.1007/s13164-022-00616-2

Liedl, A., & Knaevelsrud, C. (2008). Chronic pain and PTSD: The perpetual avoidance model and its treatment implications. *Torture*, *18*(2), 69–76.

Lumley, M. A., Schubiner, H., Lockhart, N. A., Kidwell, K. M., Harte, S. E., Clauw, D. J., & Williams, D. A. (2017). Emotional awareness and expression therapy, cognitive behavioral therapy, and education for fibromyalgia: A cluster-randomized controlled trial. *Pain*, *158*(12), 2354–2363. https://doi.org/10.1097/j.pain.0000000000001036

Lumley, M. A., Yamin, J. B., Pester, B. D., Krohner, S., & Urbanik, C. P. (2022). Trauma matters: Psychological interventions for comorbid psychosocial trauma and chronic pain. *Pain*, *163*(4), 599–603.

Martucci, K. T., & Mackey, S. C. (2018). Neuroimaging of pain: Human evidence and clinical relevance of central nervous system processes and modulation. *Anesthesiology*, *128*(6), 1241–1254. https://doi.org/10.1097/aln.0000000000002137

Martucci, K. T., Ng, P., & Mackey, S. (2014). Neuroimaging chronic pain: What have we learned and where are we going? *Future Neurology*, *9*(6), 615–626. https://doi.org/10.2217/fnl.14.57

McKernan, L. C., Johnson, B. N., Crofford, L. J., Lumley, M. A., Bruehl, S., & Cheavens, J. S. (2019). Posttraumatic stress symptoms mediate the effects of trauma exposure on clinical indicators of central sensitization in patients with chronic pain. *The Clinical Journal of Pain*, *35*(5), 385–393. https://doi.org/10.1097/ajp.0000000000000689

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science*, *150*(3699), 971–979.

Mickleborough, M. J. S., Daniels, J. K., Coupland, N. J., Kao, R., Williamson, P. C., Lanius, U. F., Hegadoren, K., Schore, A., Densmore, M., Stevens, T., & Lanius, R. A. (2011). Effects of trauma-related cues on pain processing in posttraumatic stress disorder: An fMRI investigation. *Journal of Psychiatry & Neuroscience*, *36*(1), 6–14. https://doi.org/10.1503/jpn.080188

Murphy, J. L., Cordova, M. J., & Dedert, E. A. (2022). Cognitive behavioral therapy for chronic pain in veterans:

Evidence for clinical effectiveness in a model program. *Psychol Serv. Feb*, *19*(1), 95–102. https://doi.org/10.1037/ser0000506

Murphy, J. L., Driscoll, M. A., Odom, A. S., & Hadlandsmyth, K. (2022). Posttraumatic stress disorder and chronic pain. *PTSD Research Quatrerly*, *22*(2), 1050–1835.

Nijs, J., George, S. Z., Clauw, D. J., Fernández-Carnero, J., Polli, A., Kapreli, E., Huysmans, E., Cuesta-Vargas, A. I., Mani, R., Lundberg, M., Leysen, L., Rice, D., Sterling, M., & Curatolo, M. (2021). Central sensitisation in chronic pain conditions: Latest discoveries and their potential for precision medicine. *The Lancet Rheumatology*, *3*(5), e383–e392.

Otis, J. D., Comer, J. S., Keane, T. M., Checko, E., & DB, P. (2024). Intensive treatment of chronic pain and PTSD: The PATRIOT program. *Behavioral Sciences*, *14*(11), 1103–1117.

Owens, D. C., & Fett, S. M. (2019). Black maternal and infant health: Historical legacies of slavery. *American Journal of Public Health*, *109*(10), 1342–1345. https://doi.org/10.2105/ajph.2019.305243

Polomano, R. C., Galloway, K. T., Kent, M. L., Brandon-Edwards, H., Kwon, K., Morales, C., & Buckenmaier III, C. (2016). Psychometric testing of the defense and veterans pain rating scale (DVPRS): A new pain scale for military population. *Pain Medicine (Malden, Mass)*, *17*(8), 1505–1519. https://doi.org/10.1093/pm/pnw105

Rauch, S. A. M., Abelson, J. L., Javanbakht, A., & Liberzon, I. (2015). Neurobiology and translational approaches to posttraumatic stress disorder. In K. J. Ressler, D. J. Pine, & B. O. Rothbaum (Eds.), *Anxiety disorders: Translational perspectives on diagnosis and treatment* (pp. 121–134). Oxford University Press.

Rauch, S. A. M., & Foa, E. B. (2006a). Emotional processing theory (EPT) and exposure therapy for PTSD. *Journal of Contemporary Psychotherapy: On the Cutting Edge of Modern Developments in Psychotherapy*, *36*(2), 61–65. https://doi.org/10.1007/s10879-006-9008-y

Rauch, S. A. M., & Foa, E. B. (2006b). Moving from trauma to triumph: Posttraumatic stress disorder with chronic pain and alcohol use. In R. L. Spitzer (Ed.), *DSM-IV TR casebook volume 2: Experts tell how they treated their own patients* (1st ed., pp. 237—247). American Psychiatric Publishing.

Ravn, S. L., Hartvigsen, J., Hansen, M., Sterling, M., & Andersen, T. E. (2018). Do post-traumatic pain and post-traumatic stress symptomatology mutually maintain each other? A systematic review of cross-lagged studies. *Pain*, *159*(11), 2159–2169. https://doi.org/10.1097/j.pain.0000000000001331

Rouch, I., Strippoli, M. F., Dorey, J., Ranjbar, S., Laurent, B., Gunten, A., & Preisig, M. (2023). Psychiatric disorders, personality traits, and childhood traumatic events predicting incidence and persistence of chronic pain: Results from the CoLaus|PsyCoLaus study. *Pain*, *164*(9), 2084–2092. https://doi.org/10.1097/j.pain.0000000000002912

Schnurr, P. P., Chard, K. M., Ruzek, J. I., Chow, B. K., Resick, P. A., Foa, E. B., Marx, B. P., Friedman, M. J., Bovin, M. J., Caudle, K. L., Castillo, D., Curry, K. T., Hollifield, M., Huang, G. D., Chee, C. L., Astin, M. C., Dickstein, B., Renner, K., Clancy, C. P., … Shih, M. (2022). Comparison of prolonged exposure vs cognitive processing therapy for treatment of posttraumatic stress disorder Among US veterans: A randomized clinical trial. *JAMA Network Open*, *5*(1), e2136921–e2136921. https://doi.org/10.1001/jamanetworkopen.2021.36921

Scioli, E., Forman, D., Otis, J., Gregor, K., Valovski, I., & Rasmusson, A. (2015). The shared neuroanatomy and neurobiology of comorbid chronic pain & PTSD. *The Clinical Journal of Pain*, *31*(4), 363–374. doi:10.1097/AJP.0000000000000115

Sharp, T. J., & Harvey, A. G. (2001). Chronic pain and posttraumatic stress disorder: Mutual maintenance? *Clinical Psychology Review*, *21*(6), 857–877. https://doi.org/10.1016/S0272-7358(00)00071-4

Siqveland, J., Hussain, A., Lindstrøm, J. C., Ruud, T., & Hauff, E. (2017). Prevalence of posttraumatic stress disorder in persons with chronic pain: A meta-analysis. *Frontiers in Psychiatry*, *8*, 164. https://doi.org/10.3389/fpsyt.2017.00164

Thorn, B. E. (2017). *Cognitive therapy for chronic pain: A step-by-step guide*. Guilford Publications.

Treede, R. D. (2016). Gain control mechanisms in the nociceptive system. *Pain*, *157*(6), 1199–1204. https://doi.org/10.1097/j.pain.0000000000000499

Treede, R. D., Rief, W., Barke, A., Qasim, A., Bennett, M. I., Benoliel, R., Milton, C., Stefan, E., Finnerup, N. B., First, M. B., Giamberardino, M. A., Kaasa, St., Kosek, E., Lavand'homme, P., Nicholas, M., Perrot, S., Scholz, J., Schug, St., Smith, B. H., … Wang, S. (2015). A classification of chronic pain for ICD-11. *Pain*, *156*(6), 1003–1007. https://doi.org/10.1097/j.pain.0000000000000160

Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain*, *85*(3), 317–332. https://doi.org/10.1016/s0304-3959(99)00242-0

Weathers, F. W., Bovin, M. J., Lee, D. J., Sloan, D. M., Schnurr, P. P., Kaloupek, D. G., Keane, T. M., & Marx, B. P. (2018). The clinician-administered PTSD scale for DSM–5 (CAPS-5): development and initial psychometric evaluation in military veterans. *Psychological Assessment*, *30*(3), 383–395. https://doi.org/10.1037/pas0000486

Yang, J., Lo, W. L. A., Zheng, F., Cheng, X., Yu, Q., & Wang, C. (2022). Evaluation of cognitive behavioral therapy on improving pain, fear avoidance, and self-efficacy in patients with chronic low back pain: A systematic review and meta-analysis. *Pain Research & Management*, *2022*, 4276175. https://doi.org/10.1155/2022/4276175