University of Southampton

Faculty of Environmental and Life Sciences

School of Psychology

Exploring the Impact of Psychological Trauma: Associations Between Childhood Trauma, PTSD, Pain Management Strategies, and Neuroticism in Adulthood

by

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Abstract

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Childhood trauma exerts enduring influence on adult psychological functioning, shaping emotional regulation, personality development, and stress responses. This thesis presents two complementary investigations into the long-term consequences of trauma. Chapter 2 offers the most comprehensive and large-scale synthesis to date on the relationship between childhood trauma and adult neuroticism, a transdiagnostic trait linked to a range of mental health vulnerabilities. Drawing on data from over 436,000 participants across 127 studies, the meta-analysis found a significant and robust association between trauma and neuroticism (g = 0.48), with the strongest effects observed for emotional abuse. These findings highlight the consistency of this association across trauma subtypes and underscore the psychological and neurobiological mechanisms, such as attachment disruptions and stress system dysregulation through which trauma may shape transdiagnostic traits. Chapter 3 builds on this by examining how trauma-related adaptations play out in the context of pain. Using survey data from 159 adults with chronic or acute pain, this empirical study explores the interplay between childhood trauma, posttraumatic stress disorder (PTSD) symptoms, and pain coping strategies. Emotional and physical abuse were positively associated with pain intensity and PTSD symptoms, while coping strategies like distraction and coping self-statements showed protective associations, particularly in the chronic pain group. These results suggest that trauma-related traits may influence pain perception and coping differently depending on pain chronicity, supporting models such as shared vulnerability and mutual maintenance. Implications span multiple levels: at the micro level, increasing individual awareness and coping support; at the meso level, informing trauma-sensitive clinical interventions and interdisciplinary care; and at the macro level, reinforcing the need for systemic preventative measures and public health strategies that address the long-term psychological consequences of childhood trauma. The thesis also reflects a commitment to inclusive research practices, including the adoption of more compassionate language around coping. These insights inform both theoretical understanding and practical approaches to supporting individuals affected by the enduring consequences of trauma.

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Research Thesis: Declaration of Authorship

Print name: Norma Rosenek

Title of thesis: Exploring the Impact of Psychological Trauma: Associations between Childhood Trauma, PTSD, Pain Management and Neuroticism

I declare that this thesis and the work presented in it are my own and has been generated by me as the result of my own original research.

I confirm that:

1. This work was done wholly or mainly while in candidature for a research degree at this University;
2. Where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;
3. Where I have consulted the published work of others, this is always clearly attributed;
4. Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
5. I have acknowledged all main sources of help;
6. Where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
7. None of this work has been published before submission (Chapter 3 has been submitted to *Cognitive Therapy and Research* and is currently under review).

Signature: ..... Date: 16.05.2025 .....

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Definitions and Abbreviations

ACT.....Acceptance and Commitment Therapy

ACEs.....Adverse Childhood Experiences

ANOVA.....Analysis of Variance

CBT.....Cognitive Behaviour Therapy

CFT.....Compassion Focused Therapy

DBT.....Dialectical Behaviour Therapy

EMDR.....Eye Movement Desensitisation and Reprocessing

EPHPP.....Effective Public Health Practice Project

HPA axis.....Hypothalamic-Pituitary-Adrenal Axis

PRISMA.....Preferred Reporting Items for Systematic Reviews and Meta-Analyses

PTSD.....Posttraumatic Stress Disorder

tf-CBT.....Trauma-focused Cognitive Behaviour Therapy

Chapter 1 An Overall Introduction and Bridging Chapter

1.1 Overview: The lasting impact of childhood trauma on adult health

The effects of childhood trauma on adult psychological and physiological well-being are profound and far-reaching. Over the past few decades, research has increasingly highlighted the long-term consequences of adverse childhood experiences (ACEs) on various aspects of adult life, from emotional regulation to poorer mental health outcomes and heightened pain responses (Dalechek et al., 2024; Hughes et al., 2017; Tzouvara et al., 2023). Understanding the intricate relationships between childhood trauma and adult outcomes is essential for both theoretical exploration and clinical practice. This introduction sets the stage for two chapters that examine these relationships from distinct yet interconnected perspectives. Chapter 2 presents a systematic review and meta-analysis exploring the association between childhood trauma and adult neuroticism. Chapter 2 investigates the relationships between childhood trauma, adult acute post-traumatic stress disorder (PTSD) symptoms and pain coping strategies, with a particular focus on comparing these relationships for individuals with chronic versus acute pain conditions.

Both chapters explore the broad psychological impact of childhood trauma, yet they focus on different dimensions of its impact; one from the perspective of transdiagnostic traits, and the other from the standpoint of pain perception and coping. Together, these studies provide a holistic view of how childhood trauma shapes the emotional and psychological landscape of adulthood. In this bridging chapter, I outline the empirical and theoretical foundations that inform each study, highlighting their respective novelties and research aims. Additionally, I clarify the use of key terminology to ensure consistency and conceptual clarity across the two chapters that follow.

1.2 Childhood trauma and its influence on adult neuroticism

Chapter 2 explores the relationship between childhood trauma and adult neuroticism through a systematic review and meta-analysis of existing studies. Neuroticism is a personality trait that is characterised by a predisposition toward negative emotional states such as anxiety, depression, and irritability (Barlow et al., 2021; Barlow, Ellard, et al., 2014; Barlow, Sauer-Zavala, et al., 2014). Neurotic personality traits have been consistently linked to early-life stress and trauma, whereby adults with higher levels of neuroticism are often more sensitive to stress, prone to experiencing negative emotions, and vulnerable to developing a range of psychological disorders, including anxiety and mood disorders (Chen et al., 2023; He et al., 2024; Ogle et al., 2014; Schwandt et al., 2018). These traits seem not to be reflective of current emotional states but seem to represent

enduring patterns of heightened emotional reactivity that are deeply rooted in early experiences (Dvir et al., 2014; Moskvina et al., 2007; Wrzus et al., 2021).

1.2.1 Theoretical underpinnings

Attachment theory provides an essential framework for understanding these findings. Proposed by John Bowlby (1969), attachment theory emphasises the role of early caregiving experiences in shaping emotional regulation and self-concept (Bowlby, 1998). When a child experiences trauma, particularly neglect or abuse, the attachment system becomes dysregulated, leading to insecure or disorganised attachment styles (Finzi et al., 2001; Oshri et al., 2015). These attachment disturbances are thought to predispose individuals to higher levels of neuroticism by making them more sensitive to stress, less capable of managing negative emotions, and more likely to experience difficulties in interpersonal relationships (Crawford et al., 2007). Consequently, early trauma might contribute to difficulties with forming healthy emotional regulation strategies, which contributes to the heightened emotional reactivity seen in neuroticism.

Furthermore, neurobiological mechanisms help to explain the link between childhood trauma and neuroticism. Early-life stress has been shown to alter critical stress response systems in the brain, such as the hypothalamic-pituitary-adrenal (HPA) axis, which governs the body's response to stress (Van Bodegom et al., 2017). Dysregulation of these systems results in heightened emotional reactivity and an increased sensitivity to stressors (Heim & Nemeroff, 2001). Additionally, childhood trauma is associated with structural and functional changes in key brain regions involved in emotion regulation, such as the amygdala, hippocampus, and prefrontal cortex (Teicher et al., 2016). These changes, especially when occurring during critical developmental windows, may create lasting vulnerabilities that manifest as neuroticism in adulthood (Maniam et al., 2014; Robayo, 2024). This chapter presents evidence for these mechanisms, helping to shed light on how early traumatic experiences influence the emotional and neurobiological systems that underlie neuroticism.

1.2.2 Novelty and aims of the study

Given previous empirical evidence suggesting that childhood trauma is a significant predictor of emotional dysregulation and personality development, Chapter 2 primarily focuses on examining the relationship between childhood trauma and neuroticism in adulthood. Neuroticism, a trait closely linked to emotional instability and increased vulnerability to mental health difficulties, is particularly relevant in the context of early adversity. In addition to this core focus, the study also explores how different subtypes of childhood trauma, such as emotional, physical, and sexual

abuse, as well as emotional and physical neglect, may differentially contribute to elevated levels of neuroticism. Notably, some forms of trauma, particularly emotional abuse (e.g., r = .38), appear to have stronger associations with higher neuroticism than others (e.g., physical abuse: r = .13; sexual abuse r = .16; Martín-Blanco et al., 2014; Peng et al., 2025; Ponder et al., 2024).

To systematically examine these relationships and quantify their effects, this chapter presents a large-scale systematic review and meta-analysis, incorporating data from 127 studies and a combined sample size of 436,834 participants. While the primary focus of this study is on examining the overall relationship between childhood trauma and adult neuroticism, we also adopted an exploratory approach to further investigate how specific trauma subtypes may differentially relate to neuroticism, providing additional insight into the unique contributions of various forms of early adversity. While the link between early trauma and adult psychological functioning has been widely studied (e.g., Crede et al., 2023), this chapter provides the to-date most comprehensive and large-scale synthesis that maps the strength and consistency of the associations across adult neuroticism and distinct childhood trauma types. As such, these findings are impactful as they not only align with prior work but significantly extend it: this review drew on a larger dataset, employed a more inclusive search strategy across multiple and broader databases, and ultimately revealed stronger effect sizes. Together, these enhancements offer a more comprehensive and detailed synthesis than has previously been available in this area of research.

This work holds important implications for both research and clinical practice. From a research perspective, the nuanced approach to trauma subtypes opens new avenues for theory-building around personality development and emotional vulnerability. Clinically, the findings have the potential to inform more targeted and trauma-sensitive approaches to assessment, formulation and intervention, particularly in populations where transdiagnostic traits, such as neuroticism are known to heighten risk for affective disorders and chronic health conditions. By highlighting which forms of early adversity are most strongly linked to emotional instability, this chapter supports the development of more individualised prevention and treatment strategies rooted in a deeper understanding of the long-term psychological effects of trauma.

1.3 Childhood trauma, PTSD and pain coping strategies

Chapter 3 shifts focus to another important impact of childhood trauma: its relationship to PTSD symptoms and pain coping strategies. The specific focus of this chapter will lie in understanding differences in these relationships between individuals with chronic versus acute pain conditions. Acute pain is typically short-term and acts as a warning signal of tissue injury, while chronic pain persists beyond normal tissue healing time, generally defined as pain lasting longer than three

months (Merskey, 1986). PTSD is a psychiatric disorder that often arises in response to traumatic experiences, leading to symptoms such as re-experiencing the trauma, for example through intrusive memories, nightmares or flashbacks, an increased sense of threat or hyperarousal, and avoidance behaviours (Kessler et al., 2005). The relationship between childhood trauma and PTSD is well established, with those who have experienced early adverse events being at higher risk for developing PTSD later in life (Cloitre et al., 2005; Cloitre et al., 2009; Pratchett & Yehuda, 2011; Zlotnick et al., 2008). Furthermore, early life adversities are known to impact mental and physical health in later life, including increased levels of reported pain and a greater likelihood of developing chronic pain conditions (Davis et al., 2005; Lampe et al., 2003).

Research shows that up to 75–84% of individuals with chronic pain report a history of ACEs, compared to around 62% in the general population (Davis et al., 2005; Felitti et al., 1998). PTSD is also highly prevalent among those with chronic pain, with meta-analyses estimating that approximately 11.7% meet diagnostic criteria (Siqveland et al., 2017), with even higher rates observed among individuals with chronic pain (Afari et al., 2014). These findings highlight the significant overlap between trauma exposure, PTSD, and chronic pain.

1.3.1 Understanding pain coping strategies

Pain coping strategies refer to the cognitive and behavioural efforts individuals use to manage the sensory and emotional dimensions of pain (Lazarus & Folkman, 1984). In this study, we focus on six key coping approaches commonly used: catastrophising, distraction, distancing, ignoring, self-coping statements, and praying or hoping (Peres & Lucchetti, 2010; Sullivan et al., 2001). These strategies reflect a range of responses, from efforts to reduce the emotional impact of pain (e.g., distraction or self-coping statements), to those involving a reframing of the experience (e.g., distancing), and those that reflect a spiritual or reflective approach (e.g., praying). Our aim is to investigate how these strategies are employed across chronic and acute pain groups, and how they may be influenced by the presence of trauma-related symptoms. Understanding the distinctions between acute and chronic pain is crucial, as the mechanisms underlying each may interact differently with trauma-related symptoms. Given the high prevalence of trauma histories among individuals with pain, particularly in chronic pain populations, investigating how coping strategies are employed across these groups can offer valuable insights into underlying psychological processes. Moreover, this knowledge has direct clinical relevance, as it may inform more tailored interventions to address both pain and trauma symptoms concurrently.

1.3.2 Theoretical underpinnings

The chapter explores these relationships through the lens of two theoretical models: the Shared Vulnerability Model and the mutual maintenance model. These models are particularly relevant as they offer frameworks for understanding how childhood trauma can predispose individuals to both PTSD symptoms and chronic pain through overlapping psychological risk factors and coping styles. These models help explain not only the high co-occurrence of these conditions but also how specific strategies, like avoidance or catastrophising, may reinforce and sustain both symptom profiles over time.

The Shared Vulnerability Model (Asmundson et al., 2002) proposes that individuals with a history of trauma share underlying neurobiological and psychological vulnerabilities that predispose them to both PTSD and chronic pain. This shared vulnerability results from alterations in brain regions responsible for emotion regulation and pain perception, such as the amygdala, hippocampus, and prefrontal cortex. These changes in neural processing create a heightened sensitivity to both emotional and physical stressors, increasing the likelihood of developing both PTSD and chronic pain in response to early trauma. This model provides a valuable framework for interpreting the findings of Chapter 3, which examines how trauma-related symptoms and pain coping strategies manifest across individuals with chronic and acute pain presentations.

The Mutual Maintenance Model (Sharp & Harvey, 2001) builds on this idea by suggesting that chronic pain and PTSD symptoms mutually reinforce each other in a cyclical process. In this model, the distress and hypervigilance associated with chronic pain can exacerbate PTSD symptoms, while PTSD symptoms can increase pain perception and emotional distress. This feedback loop creates a self-perpetuating cycle of pain and psychological suffering, which is particularly pronounced in individuals with chronic pain who have experienced childhood trauma (Felitti et al., 1998). Chapter 3 draws on this model to explore how the interplay between trauma symptoms and pain coping strategies may differ between individuals with chronic and acute pain, shedding light on potential mechanisms underlying this reciprocal relationship.

Both models provide a useful framework for understanding the complex interplay between trauma, PTSD, and chronic pain. By highlighting the neurobiological and emotional factors that contribute to this comorbidity, these models underscore the importance of integrated treatment approaches that address both the psychological and physical aspects of trauma-related conditions.

1.3.3 Novelty and aims of the study

The empirical study presented in Chapter 3 explores the relationship between childhood trauma, current PTSD symptoms, and pain coping strategies across individuals experiencing either chronic or acute pain. While previous research has explored how some of these variables may be associated within chronic (Burke et al., 2017; Kisiel et al., 2009) or acute (Keene et al., 2011; Pacella et al., 2013; Reed & Schurr, 2020) pain populations separately, to our knowledge, this is the first study to directly compare chronic and acute pain groups in how they utilise specific pain coping strategies in context of their post-traumatic stress symptoms and experiences of childhood trauma. This comparison allows for a more nuanced understanding of how trauma history and current psychological distress may influence the ways in which individuals manage pain across differing pain trajectories. From a research perspective, it offers important insights into the complex interplay between psychological factors and pain coping processes. Clinically, given the high prevalence and frequent overlap of trauma-related conditions such as PTSD and chronic pain, these findings have the potential to inform more tailored, trauma-sensitive approaches to pain management and psychological support.

1.3.4 Language matters: A compassionate perspective

An important consideration in this research is the language used to describe pain-related experiences and coping, specifically in the context of trauma. During the recruitment phase of this study, we engaged in conversations with individuals with lived experience of complex PTSD and chronic pain. A recurring theme in these discussions was that certain terms commonly used in the literature, particularly "catastrophising", are perceived as invalidating or stigmatising. While "pain catastrophising" remains a widely recognised and validated construct within pain research (Pedler, 2010; Sullivan, 2012), we are mindful of the potential impact of language on participants and readers alike.

As such, we are intentionally adopting a more compassionate and strength-based lens in our writing. For example, in this context, we propose using the term pain-driven worry as a more person-centred alternative to catastrophising. Although individuals with lived experience encouraged us to re-evaluate the discourse surrounding so-called “maladaptive coping” particularly in relation to pain catastrophising, we were unable to obtain feedback on the final terminology selected for this project. We view this shift as part of an ongoing process, and we are committed to continuing these conversations in future work. We strongly value open dialogue and hope to create space for those affected by trauma and chronic pain to shape the language used to describe their experiences.

This shift in terminology is not intended to dismiss existing measures or theoretical frameworks, but rather to integrate community perspectives and support a gradual, thoughtful evolution in how we talk about psychological responses to pain. We believe these changes can foster a more inclusive research environment and hope this work may serve as an early step in that direction. Importantly, this shift in language is part of a broader decision to move away from pathologising or labelling coping strategies as maladaptive. We recognise that such terms, while clinically familiar, can inadvertently reinforce shame or deficit-based narratives for people already managing complex and distressing experiences. Instead, we aim to acknowledge the function and context of these responses, often adaptive under earlier conditions, even if they become less helpful over time. We hope that this approach resonates with individuals who have lived experience and contributes to a more validating and empowering discourse in trauma and pain research.

1.3.5 Dissemination of findings

The dissemination of this research has been carefully considered to ensure it reaches both academic and clinical audiences most likely to benefit from the findings. The meta-analytic component of the thesis is being prepared for submission to *Clinical Psychology Review*, a leading journal that publishes high-impact, integrative reviews relevant to the field of clinical psychology. This journal is particularly well suited to the aims of the meta-analysis, which synthesises evidence across studies to address an important and clinically relevant question. *Clinical Psychology Review* is widely read by researchers, clinicians, and policymakers, and its focus on rigorous methodological standards aligns well with the comprehensive and systematic nature of the current meta-analysis. Publication in this outlet would maximise the visibility of the findings and facilitate translation into clinical practice, particularly for professionals interested in evidence-based interventions and transdiagnostic approaches.

In addition, the empirical study from this thesis has been submitted to and is currently under review by *Cognitive Therapy and Research*, a journal that specialises in the evaluation and development of cognitive-behavioural theories and treatments. This journal offers an ideal platform for Chapter 2, which directly tests theoretical mechanisms within a cognitive framework. Its audience consists of clinical researchers, practitioners, and academics who are specifically interested in advancing cognitive therapy through empirical evidence. By targeting *Cognitive Therapy and Research*, the aim is to contribute to ongoing theoretical refinement and to inform future clinical applications, especially in the context of improving psychological treatment outcomes.

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Chapter 2 The Relationship Between Childhood Trauma and Adult Neuroticism: A Systematic Review and Meta-Analysis

2.1 Abstract

Childhood trauma has been consistently associated with elevated levels of neuroticism in adulthood, a transdiagnostic trait marked by emotional instability, heightened negative affect, and stress sensitivity. This systematic review and meta-analysis aimed to synthesise evidence examining the association between childhood trauma and adult neuroticism, both overall and by specific trauma subtypes. A comprehensive search of four electronic databases identified 127 eligible studies, encompassing a total of 436,834 individuals. Using a random-effects meta-analysis, results revealed a significant positive association between childhood trauma and adult neuroticism (g = 0.48). Separate meta-analyses showed that this association was consistent across all trauma subtypes, including emotional abuse (g = 0.52), emotional neglect (g = 0.40), physical abuse (g = 0.15), physical neglect (g = 0.12), sexual abuse (g = 0.15), unspecified abuse (g = 0.13), and victimisation (g = 0.21), with the exception of unspecified neglect, which showed no significant association. These findings demonstrate a robust relationship between early adversity and neuroticism, regardless of trauma type. Childhood trauma may lead to adaptions that give rise to neuroticism through several psychological mechanisms such as disruptions in attachment and the formation of negative self-beliefs, and neurobiological alterations in stress regulation systems. These results underscore the importance of systemic preventative measures and early intervention strategies that may alleviate the psychological and neurobiological consequences of trauma, with the potential to increase awareness of adaptions such as neuroticism in trauma-exposed populations.

2.2 Introduction

Childhood trauma is the exposure to adverse experiences during formative years and has been widely recognised as a critical factor influencing psychological development (Crede et al., 2023; Fletcher & Schurer, 2017; van der Kolk et al., 2009). These adverse experiences, which include emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse, have been consistently linked to long-term consequences for mental health and well-being (Teicher et al., 2016). A growing body of research suggests that such early-life adversities may be associated with the development of transdiagnostic traits, particularly in relation to neuroticism (also known as negative emotionality), which is associated with increased vulnerability to mental health conditions (Lahey, 2009; McLaughlin et al., 2020; Ogle et al., 2014; Ormel et al., 2013; Rossiter et al., 2015).

Given the potentially lasting effects of early adversity, understanding its influence on transdiagnostic traits is crucial. Transdiagnostic traits are shaped by a complex interplay of genetic and environmental factors, including early childhood experiences (Costa & McCrae, 2008). Among the Big Five personality traits, neuroticism has been extensively studied in relation to adverse childhood experiences. Neuroticism is characterised by heightened emotional instability, susceptibility to stress, and a tendency toward negative emotional states such as anxiety and negative mood, such as depression (Widiger & Mullins-Sweatt, 2009). Research indicates that individuals with higher levels of childhood trauma often exhibit elevated neuroticism in adulthood, suggesting that early adversity may lead to trait-like adaptions in emotional reactivity and regulation tendencies (Shackman et al., 2016).

The connection between childhood trauma and neuroticism may be understood through multiple psychological and neurobiological mechanisms. For instance, early adverse experiences are thought to shape the development of a person's sense of self and core beliefs about the world. Attachment theory posits that early caregiving experiences play a fundamental role in shaping self-concept and emotional regulation (Bowlby, 1998). Children who experience trauma may develop schemas that result in negative self-perceptions and shame, which persist into adulthood and contribute to increased neuroticism (Pilkington et al., 2021). These negative self-appraisals may increase tendencies toward rumination, emotional lability, and a pervasive sense of threat, all of which are hallmarks of high neuroticism (Bowlby, 1998; Ormel et al., 2013; Pilkington et al., 2021). Moreover, exposure to early-life stress has been shown to alter stress response systems, including the hypothalamic-pituitary-adrenal (HPA) axis, leading to greater emotional reactivity and sensitivity to stress (Heim & Nemeroff, 2001). Additionally, childhood trauma is associated with structural and functional changes in brain regions involved in emotion regulation, such as the amygdala, hippocampus, and prefrontal cortex (Teicher et al., 2016).

Since these brain regions undergo critical periods of development during childhood, exposure to trauma at an early age may lead to longer-term changes to the neural circuitry supporting emotion regulation and impulse control (Cremers et al., 2010; Forbes et al., 2014; Silverman et al., 2019; Yang et al., 2020). In particular, such adaptions may result in heightened susceptibility to stress and negative emotionality in adulthood (Chia & Tan, 2024; Kolassa & Elbert, 2007).

Since trauma can influence self-concept and neurobiological systems in different ways, it follows that different subtypes of childhood trauma may impact specific mechanisms that support the development of neuroticism. Emotional abuse and neglect may influence a child's self-worth and emotional security (Glaser, 2002). In contrast, physical and sexual abuse may contribute to the development of heightened stress sensitivity and altered emotion regulation tendencies (Infurna et al., 2016). A recent meta-analysis examined the relationship between adverse childhood experiences (r = .20) and adult neuroticism (Crede et al., 2023). These findings also highlighted a robust, positive relationship between exposure to subtypes childhood trauma and higher levels of neuroticism in adulthood, with emotional abuse emerging as the most strongly associated subtype (r = .25), whereas physical abuse (r = .14) and physical neglect (r = .14) showed weaker, though still significant, associations, whereas sexual abuse had the weakest association (r = .10). While these findings represent an important contribution, several limitations highlight the need for a more comprehensive synthesis. For example, Crede et al. did not specify the full extent of their search period, with the most recent study included having been published in 2021. Given the likely growth of literature in the intervening years, a more up-to-date synthesis is warranted. Furthermore, the current review aims to expand the scope by applying broader search terms across a wider range of databases, allowing for the inclusion of additional relevant studies that may have been missed in previous reviews. The rationale for an updated review lies in the opportunity to build on and broaden the scope of Crede et al.'s previous work. Their review, while valuable, included only the term "emotional stability" in its search criteria, omitting "neuroticism", a concept that, although conceptually opposite, represents the same psychological trait. Additionally, they focused exclusively on non-clinical populations, whereas the present review aims to be more inclusive by including both clinical and non-clinical populations, thereby capturing a broader spectrum of relevant studies. These decisions likely resulted in the exclusion of studies that used alternative but equivalent terminology. In contrast, the present study will include both "neuroticism" and "emotional instability" in its search terms, thereby capturing a wider and more conceptually inclusive body of literature. Furthermore, whereas Crede et al. utilised databases such as *PsycINFO, ERIC, Dissertations and Theses Global, PTSDpubs*, and *Google Scholar*, which incorporate grey literature and education-specific sources, the present study will draw on *EBSCOhost, Web of Science, PsycINFO*, and *Scopus*. These databases prioritise peer-reviewed, published research with broad international and

multidisciplinary coverage. The exclusion of grey literature in the present study will be an intentional decision to ensure academic rigour and consistency in source quality. Together, these methodological refinements extend the scope of the original review and provide a clearer, more comprehensive understanding of the literature, thereby justifying the need for an updated review.

This systematic review and meta-analysis aimed to synthesise the literature on childhood trauma and neuroticism in adulthood. Specifically, we examined whether

(1) there is an association between childhood trauma and neuroticism in adult life

and

(2) different subtypes of childhood trauma (emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse) are associated with adult neuroticism.

2.3 Methods

This systematic review was conducted following the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The study was registered in the International Prospective Register of Systematic Reviews (PROSPERO) in August 2024 (CRD42024580278).

2.3.1 Eligibility criteria and study selection

Studies included in this review met specific eligibility criteria designed to ensure methodological rigor and relevance to the research question. Inclusion criteria were as follows: studies had to be published in English in peer-reviewed journals, with no restrictions on publication date. Eligible studies were required to report a statistical association between childhood trauma and neuroticism (or negative emotionality) using quantitative methods, including correlation or regression analyses, group comparisons, structural equation modelling, or path analyses. To ensure measurement quality, included studies had to assess both constructs using well-validated instruments, defined as questionnaires or indices that demonstrated at least adequate psychometric properties (e.g., Cronbach's alpha ≥ .70) and reported evidence of reliability and validity in line with established standards. Neuroticism had to be measured in adulthood (18 years or older) to ensure that personality traits were assessed post-developmentally. Exclusion criteria included qualitative studies, case studies, reviews, book chapters, conference abstracts, theses and dissertations, and other forms of grey literature. Studies that used proxy indicators without psychometric validation or that measured neuroticism during adolescence or childhood were also excluded (Table 2.4).

Study selection adhered to PRISMA guidelines (Liberati et al., 2009, see Figure 2.1). First, a literature search was conducted across six digital databases (EBSCO, Web of Science, PsycINFO and Scopus) using the following search terms: " ("childhood trauma" OR "early life stress" OR "early trauma" OR "childhood adversity" OR "childhood maltreatment" OR "childhood abuse" OR "childhood neglect" OR "adverse childhood experience*" OR "ACEs") AND ("neurotic*" OR "neurotic traits" OR "emotional instability" OR "negative affectivity" OR "negative emotionality") AND ("childhood trauma" OR "early life stress" OR "early trauma" OR "childhood advers*" OR "childhood maltreatment" OR "childhood abuse" OR "childhood neglect" OR "adverse childhood experience*" OR "ACEs") AND ("neurotic*" OR "neurotic traits" OR "emotional instability" OR "negative affectivity" OR "negative emotionality") AND ("impact" OR "effect" OR "consequences" OR "relationship")". Searches were conducted between July 25, 2024, and August 4, 2024. An updated search was completed in April 2025. Search results were uploaded to the software 'Rayyan' (Ouzzani et al., 2016) where authors screened them. After removing

duplicate results, abstracts from all sources were screened against the eligibility criteria. Full-text review was conducted by at least two members of the research team (NR plus at least one other researcher). Any discrepancies were resolved through discussion; however, there were no disagreements in the final inclusion decisions, resulting in 100% agreement.

Table 2.4

Presentation of inclusion and exclusion criteria.

Inclusion Criteria	Exclusion Criteria
• Published in English	• Not published in English
• Empirical study, published in a peer-reviewed journal	• Reviews, book chapters, conference abstracts, theses, dissertations, and other grey literature
• Report a statistical association between childhood trauma and neuroticism (or negative emotionality) • Use quantitative methods (e.g., correlation, regression, group comparisons, structural equation modelling, path analysis) • Use well-validated instruments to assess both childhood trauma and neuroticism (e.g., Cronbach's alpha ≥ .70, with reported reliability and validity)	• Do not report a statistical association between childhood trauma and neuroticism (or negative emotionality) • Use qualitative methods or case studies • Use proxy indicators or instruments without psychometric validation
• Measure neuroticism in adulthood (18 years or older)	• Measure neuroticism during adolescence or childhood
• No restrictions on publication date	

2.3.2 Risk of bias assessment

The risk of bias in the included studies was assessed using the Effective Public Health Practice Project (EPHPP) Quality Assessment Tool for Quantitative Studies (EPPHP, 2009). This tool evaluates studies across eight key domains: selection bias, study design, confounders, blinding, data collection methods, withdrawals and dropouts, intervention integrity, and analyses. Each component is rated as strong, moderate, or weak, allowing for an overall assessment of study quality. The EPHPP tool was developed to provide a structured framework

for appraising the methodological quality of a wide range of quantitative research designs, explicitly including observational studies, such as longitudinal or cross-sectional studies (Deeks et al., 2012; EPHPP, 2009; Jackson & Waters, 2005; Thomas et al., 2004). In contrast to tools that are restricted to randomised controlled trials, the EPHPP includes domains that are equally relevant to non-experimental research, such as selection bias, data collection methods and withdrawals or dropouts, which allows for consistent and comparable evaluation across diverse study types (EPHPP, 2009). Inter-rater reliability and construct validity testing showed acceptable agreement among reviewers when the tool was applied to cohort and cross-sectional designs (Armijo-Olivo et al., 2012; Thomas et al., 2004). In addition, comparative methodological research demonstrated that the EPHPP is more flexible than instruments such as the Cochrane Risk of Bias tool, since it captures important threats to validity that are specific to non-randomised designs (Armijo-Olivo et al., 2012). Assessments were conducted by the lead researcher (NR) and spot-checked by the senior author (JM) to ensure reliability and consistency. JM independently reviewed 20% of the included studies, and there was complete agreement between raters, yielding a Cohen's kappa of $\kappa = 1.00$ The results of the risk of bias assessment provide insight into the methodological rigor of the included studies and inform the interpretation of the findings in this review.

To tailor the tool to the specific aims and characteristics of the included studies, a selective approach was adopted regarding the EPHPP domains. From Section A (Selection Bias), both questions were retained. In Section B (Study Design), all questions were kept, although questions three and four, which pertain specifically to randomised controlled trials, were not relevant, as none of the included studies employed a randomised design. Sections C (Confounders) and D (Blinding) were excluded entirely, as they were not applicable to the predominantly observational study designs in this review. Section E (Data Collection Methods) was fully included, given its relevance to assessing the validity and reliability of measurement tools used across studies. In Section F (Withdrawals and Dropouts), both questions were retained to capture issues related to participant attrition. Section G (Intervention Integrity) was excluded, as it pertains to the consistency and delivery of interventions, which was not relevant to the studies assessed. For Section H (Analyses), questions two and three were retained. As part of the screening process, it was ensured that all studies were conducted at the individual level, as studies not meeting this criterion would have automatically been assigned a weak rating in this section. As described, three domains were omitted from the quality assessment. The decision to modify this tool is reported transparently and was made in order to avoid introducing potential bias into the overall quality ratings, since retaining subscores from domains that were not applicable to the included study designs may distort the global risk of bias assessment. It is important to note that the authors of the EPHPP tool do not recommend removing domains and advise that all eight

domains should be applied consistently when deriving the global rating (EPHPP, 2009). Nonetheless, several published systematic reviews and meta-analyses have also chosen to omit or modify domains for similar reasons, demonstrating precedent for this approach (Barbek et al., 2022; Buccini et al., 2024; Chew et al., 2023; Conklin et al., 2018; De Souza et al., 2021; Dunn & Sicouri, 2022; Hill et al., 2022; Li et al., 2014; Madana Civi et al., 2024; Malfliet et al., 2017; Mulligan et al., 2024; Newman et al., 2018; Shah et al., 2020; Silveira et al., 2011; Yang et al., 2023). While this may reduce the risk of artificially inflating or deflating quality scores, it should be recognised that omitting domains can itself affect the global bias rating by narrowing the range of criteria on which a study is judged.

Following the domain-level assessments, a global quality rating was assigned to each study: strong (no weak ratings across included domains), moderate (one weak rating), or weak (two or more weak ratings). Overall, of the 127 papers included, 88 received a strong rating, 42 received a moderate rating, and 0 were rated as weak (Appendix 4A, Table 2.4). After screening all full texts, data extraction included (1) sample characteristics, (2) methodology, (3) statistical analyses performed, (4) outcome measures pertinent to the review's objectives, (5) resulting effect sizes, and (6) a brief description of study outcome. This information was then used to conduct a narrative synthesis of the findings of the included experiments in alignment with the study's aims.

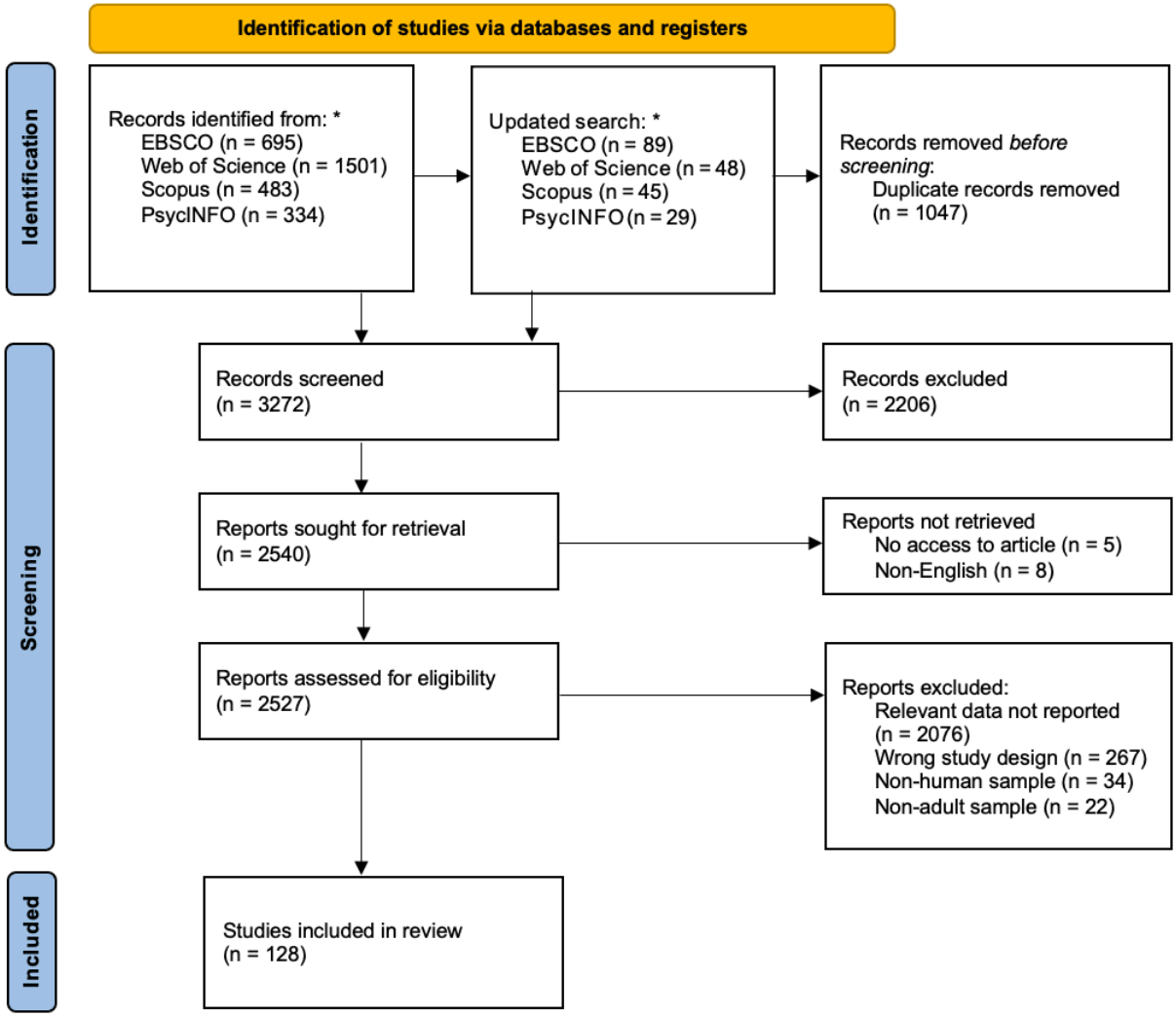
2.3.3 Data and meta-analysis model

Effect sizes that were extracted included *r*, *beta* and *d* and were transformed to Hedges' *g* effect size values. Thus, the only effect size index used to quantity effects for the relationship between trauma (and its subtypes) and neuroticism was Hedges' *g*. A positive Hedges' *g* value represents a positive relationship between trauma and neuroticism. In line with conventional guidelines, Hedges' *g* values of 0.20, 0.50, and 0.80 were interpreted as small, medium, and large effects, respectively (Cohen, 1988; Hedges & Olkin, 2014). Random-effect meta-analyses were carried out in RStudio (RStudio, Inc., Boston, MA). Effect size outcomes were modelled for overall trauma and the subtypes with a random-effects model due to its tolerance of heterogeneous effect sizes and conservative nature of estimation (Schmidt et al., 2009). Heterogeneity across effects sizes were measured by I^2 statistic. To evaluate the presence of publication bias, funnel plots were visually examined and Egger's test was performed (Egger et al., 1997). Given the small number of studies included in some trauma sub-type meta-analyses (i.e., unspecified neglect and abuse, and victimization; $n < 5$), the ability to detect asymmetry in funnel plots is limited. Consequently, a more lenient significance threshold ($p = 0.10$) was applied instead of the conventional 0.05 (Fleiss, 1993). When relevant, the Duval and Tweedie 'Trim and Fill' procedure was utilised to adjust for the potential influence of such bias (Duval & Tweedie,

2000). We reported Egger's test for all outcomes but did not conduct trim-and-fill analyses in cases of significant Egger's outcomes when fewer than 10 studies were included, as the test lacks reliability in such cases and follow-up adjustments like trim-and-fill are not recommended with small k (Mavridis & Salanti, 2014).

2.4 Results

Figure 2.1 - Flowchart to show the process of inclusion eligibility for meta-analysis



*Initial search conducted in August 2024; Updated search conducted April 2025

2.4.1 Study characteristics

A total of 127 studies were included in the meta-analysis, comprising a combined sample of 436,834 participants (see Table 2.1; Appendix 1A due to size). Analyses were conducted for overall childhood trauma and separately for different trauma subtypes. We extracted 97 effect sizes for overall trauma (k = 90; n = 411,407). For emotional abuse, we extracted 45 effect sizes (k = 44; n = 34,081), and for emotional neglect, 34 effect sizes (k = 33; n = 19,928). Unspecified neglect was examined in 3 effect sizes (k = 3; n = 326). Physical abuse yielded 37 effect sizes (k = 36; n = 24,037), while physical neglect yielded 25 effect sizes (k = 24; n = 12,285). Unspecified abuse was represented by 4 effect sizes (k = 3; n = 1,265), and sexual abuse by 43 effect sizes (k = 42; n = 21,456). Three effect sizes were extracted for victimisation experiences (k = 3; n = 1,441).

The studies were conducted predominantly in North America, which accounted for 47 studies, with the United States alone contributing 43 studies. Asia followed closely with 36 studies, driven largely by a high number of studies from China (22 studies). Europe represented about 34, with significant contributions from the Netherlands, Germany, and the United Kingdom. Seven studies collected data in Oceania (New Zealand and Australia) and 2 studies were conducted in South America (Brazil and Colombia). Africa was represented in one study (Togo) as part of a multi-country analysis, and one study was classified as international or online.

Across the 127 studies, female participants substantially outnumbered males, with female-majority studies (k = 83) far exceeding male-majority studies (k = 16). Single-gender studies included 11 female-only studies and 6 male-only studies. Gender data were not reported in two studies, with an additional 7 studies providing incomplete or unclear gender breakdowns for separate participant subgroups. Participants across studies ranged in age from 18 to 93 years, with sample mean ages ranging from 19 to 72 years. Specific participant cohorts included student cohorts (k = 8), birth cohorts (k = 6), or older adults (aged 60 years and older) that were initially recruited as part of a longitudinal study (k = 4). Ethnicity data were reported in 49 studies, with White/Caucasian participants comprising the majority in 44 of these studies. Three studies included only White participants and two studies included no White/Caucasian participants. Three studies reported participant nationality but did not provide a details about ethnicity. Information about distribution of minority ethnicities were reported in 36 studies and included Black/African American, Asian (including South Asian, East Asian, Southeast Asian), Hispanic/Latino, Mixed/Multiracial/Biracial ethnicity, Native American/American Indian/Alaska Native, Middle Eastern/Arab/North African, Pacific Islander/Hawaiian, Aboriginal/Torres Strait Islander, Māori, and Indian participants (see Appendix 5A; Table 2.5 for specific percentages of ethnicity distributions). However, 78 studies did not report ethnicity data, limiting comprehensive assessment of representativeness. The majority of studies were cross-sectional, accounting for

85% of studies (*k* = 108), while longitudinal or follow-up designs made up 15% (*k* = 19; Appendix 4A, Table 2.4).

2.4.2 Childhood trauma measures

Childhood trauma was assessed using a variety of retrospective measures across the included studies. The most frequently used instrument was the Childhood Trauma Questionnaire – Short Form (CTQ-SF; *k* = 63), which included translated versions in Korean, Thai, and Chinese. The full version of the CTQ was also used in a smaller subset (*k* = 10). Other widely employed measures included the Adverse Childhood Experiences scale (ACEs) or adaptations thereof (*k* = 16), incorporating both Thai and Portuguese versions, as well as the Child Abuse and Trauma Scale (CATS; *k* = 5). Less commonly used tools were the Early Trauma Inventory Self Report – Short Form (ETISR-SF; *k* = 4), the Childhood Trauma Screener (CTS; *k* = 2), the Childhood Psychological Maltreatment Scale (CPMS; *k* = 2), and the NEMESIS Childhood Trauma Interview (*k* = 2), which included a Dutch adaptation. A wide range of other trauma instruments were used only once each across the studies. These included: the Conflict Tactics Scale (CTS); Juvenile Victimization Questionnaire – Adults Retrospective Version; Childhood Sexual Trauma Questionnaire (CSTQ); adaptations of the Childhood Trauma Interview from ACE-IQ and national mental health surveys (e.g., MHQ); Childhood Adversity score from the Christchurch Health and Development Study (CHDS); Abuse-Perpetration Inventory (API); Assessment Scale of Victimization in Childhood; Childhood Experiences of Violence Questionnaire (CEVQ); Family and Sexual History Questionnaire; Childhood Victimization Rating Scale; Traumatic Life Events Questionnaire (TLEQ); Childhood Threat Inventory (PTI); Early Life Stress (ELS) scale; the Daily Inventory of Stressful Events (DISE) with trauma-relevant adaptations; short mistreatment and abuse scales (e.g., items adapted from Bryer et al., 1987, and Finkelhor, 1979); trauma subscales from the Health and Retirement Study (HRS); the Traumatic Events Screening Inventory – Youth/Self-Report (TESI-Y/SR); the MIDUS childhood trauma subscale; and data from the Violent Experiences Questionnaire (VEQ-R) and LONGSCAN consortium. See Table 2.2 in Appendix 2A for an overview.

2.4.3 Neuroticism measures

Neuroticism was measured using a range of validated personality instruments across the included studies. The most commonly used measure was the NEO Five-Factor Inventory (NEO-FFI/NEO-FFI-3; *k* = 49), which included translated versions such as Dutch. The Eysenck Personality Questionnaire and its variants (EPQ, EPQ-R, EPQ-RSC, EPQR-AF, EPQR-N, EPQR-S) were also widely used (*k* = 19). Other frequently applied instruments included the Revised NEO

Personality Inventory (NEO PI-R; *k* = 9), the Big Five Inventory and short forms (BFI, BFI-S; *k* = 11), which included Chinese and Thai versions, the International Personality Item Pool (IPIP; *k* = 7), and the Ten-Item Personality Inventory (TIPI; *k* = 5), including a Korean version. Less frequently used measures were the Multidimensional Personality Questionnaire (MPQ, MPQ-BF; *k* = 3), the Emotionality Personality Inventory (EPI; *k* = 3), and the PANAS or its international short form (I-PANAS-SF; *k* = 4). A variety of instruments were used only once across studies, including the Temperament and Character Inventory short forms (TSDI, S5), the Minnesota Multiphasic Personality Inventory – 2 (MMPI-2; Korean version), the Affective Intensity Measure (AIM), Defense Style Questionnaire (DSQ-40), the Five-Factor Narcissism Inventory – Short Form (FFNI-SF), the Personality Inventory for DSM-5 (PID-5), a six-item negative affect scale, the HADS neuroticism/worry subscale, the Type D personality scale (DS14), the 16 Personality Factor Questionnaire (16PF), the Psychological Distress Scale from the Mental Health Index, and the trait version of the State–Trait Anxiety Inventory (STAI-T). In some studies, only the neuroticism subscale of broader instruments was used. For full details, see Table 2.3 in Appendix 3A.

2.4.4 Meta-analytic results

2.4.4.1 The relationship between overall trauma and neuroticism

Effect sizes for the relationship between an overall measure of trauma and neuroticism were taken from 97 samples (*k* = 90; *n* = 411,407). The random-effects model was significant and estimated a moderate positive effect, Hedges' *g* = 0.48 (95% CI = 0.47; 0.49), *p* < .001 (Figure 2.2A). There was considerable heterogeneity across studies, I^2 = 97.8%. An examination of the funnel plots and the outcome of Egger's test (*p* = 0.26) indicated no evidence of publication or other selection bias.

2.4.4.2 The relationship between distinct subtypes of trauma and neuroticism

Emotional Abuse

Effect sizes for the relationship between emotional abuse and neuroticism were taken from 45 samples (*k* = 44; *n* = 34,081). The random-effects model was significant and estimated a moderate positive effect, Hedges' *g* = 0.52 (95% CI = 0.43; 0.60), *p* < .001 (Figure 2.3A). There was considerable heterogeneity across studies, I^2 = 92.7%. An examination of the funnel plots and the outcome of Egger's test (*p* = 0.15) indicated no evidence of publication or other selection bias.

Emotional Neglect

Effect sizes for the relationship between emotional neglect and neuroticism were taken from 34 (k = 33; n = 19,928). The random-effects model was significant and estimated a moderate positive effect, Hedges' g = 0.40 (95% CI = 0.33; 0.47), p < .001 (Figure 2.2C). There was considerable heterogeneity across studies, I^2 = 78.7%. An examination of the funnel plots and the outcome of Egger's test (p = 0.15) indicated no evidence of publication or other selection bias.

Unspecified Neglect

Effect sizes for the relationship between unspecified neglect and neuroticism were taken from 3 samples (k = 3; n = 326). The random-effects model estimated a moderate but not statistically significant positive effect, Hedges' g = 0.39 (95% CI = -0.04; 0.83), p = .08 (Figure 2.2D). There was considerable heterogeneity across studies, I^2 = 71.2%. An examination of the funnel plots and the outcome of Egger's test (p = 0.42) indicated no evidence of publication or other selection bias.

Physical Abuse

Effect sizes for the relationship between physical abuse and neuroticism were taken from 36 studies. These studies contributed 37 effect sizes (k = 36; n = 24,037). The random-effects model estimated a small but statistically significant positive effect, Hedges' g = 0.27 (95% CI = 0.21; 0.33), p < .001 (Figure 2.3C). There was considerable heterogeneity across studies, I^2 = 80.5%. An examination of the funnel plots and the outcome of Egger's test (p = 0.01) indicated evidence of publication or other selection bias. The Trim and Fill procedure suggested an adjusted effect size of g = .15 (95% CI = .10; .23; Figure 2.5A).

Physical Neglect

Effect sizes for the relationship between physical neglect and neuroticism were taken from 24 samples (*k* = 24; *n* = 12,285). These studies contributed 25 samples, which comprised of 12,285 individuals. The random-effects model was significant and estimated a moderate positive effect, Hedges' *g* = 0.32 (95% CI = 0.22; 0.42), *p* < .001 (Figure 2.4B). There was heterogeneity across studies, I^2 = 86.0%. An examination of the funnel plots and the outcome of Egger's test (*p* = 0.02) indicated evidence of publication or other selection bias. The Trim and Fill procedure suggested an adjusted effect size of *g* = .12 (95% CI = .03; .23; Figure 2.5B).

Unspecified Abuse

Effect sizes for the relationship between unspecified abuse and neuroticism were taken from 4 samples (*k* = 3; *n* = 1,265). The random-effects model estimated a small but statistically significant positive effect, Hedges' *g* = 0.13 (95% CI = 0.02; 0.24), *p* = .02 (Figure 2.3B). There was low heterogeneity across studies, I^2 = 26.1%. An examination of the funnel plots and the outcome of Egger's test (*p* = 0.04; Figure 2.5C) indicated evidence of publication or other selection bias. However, due to small number of studies included in this analysis, use of the Trim and Fill method was unnecessary in this instance (Mavridis & Salanti, 2014).

Sexual Abuse

Effect sizes for the relationship between sexual abuse and neuroticism were taken from 43 samples (*k* = 42; *n* = 21,456). The random-effects model estimated a small but statistically significant positive effect, Hedges' *g* = 0.23 (95% CI = 0.17; 0.28), *p* < .001 (Figure 2.4A). There was considerable heterogeneity across studies, I^2 = 75.2%. An examination of the funnel plots and the outcome of Egger's test (*p* = 0.005) indicated evidence of publication or other selection bias. The Trim and Fill procedure suggested an adjusted effect size of *g* = .15 (95% CI = .10; .21; Figure 2.5D).

Victimisation

Effect sizes for the relationship between victimisation and neuroticism were taken from 3 samples (*k* = 3; *n* = 1,441). These studies contributed 3 samples, which comprised of 1,441 individuals. The random-effects model estimated a small but statistically significant positive effect, Hedges' *g* = 0.21 (95% CI = 0.14; 0.29), *p* < .001 (Figure 2.2B). There was extremely low heterogeneity across studies, I^2 = 0.0%. An examination of the funnel plots and the outcome of Egger's test (*p* = 0.56) indicated no evidence of publication or other selection bias.

Figure 2.2 - Forest plot demonstrating a small effect size across studies for the relationship between neuroticism and (A) overall childhood trauma, (B) victimisation, (C) emotional neglect and (D) unspecified neglect.

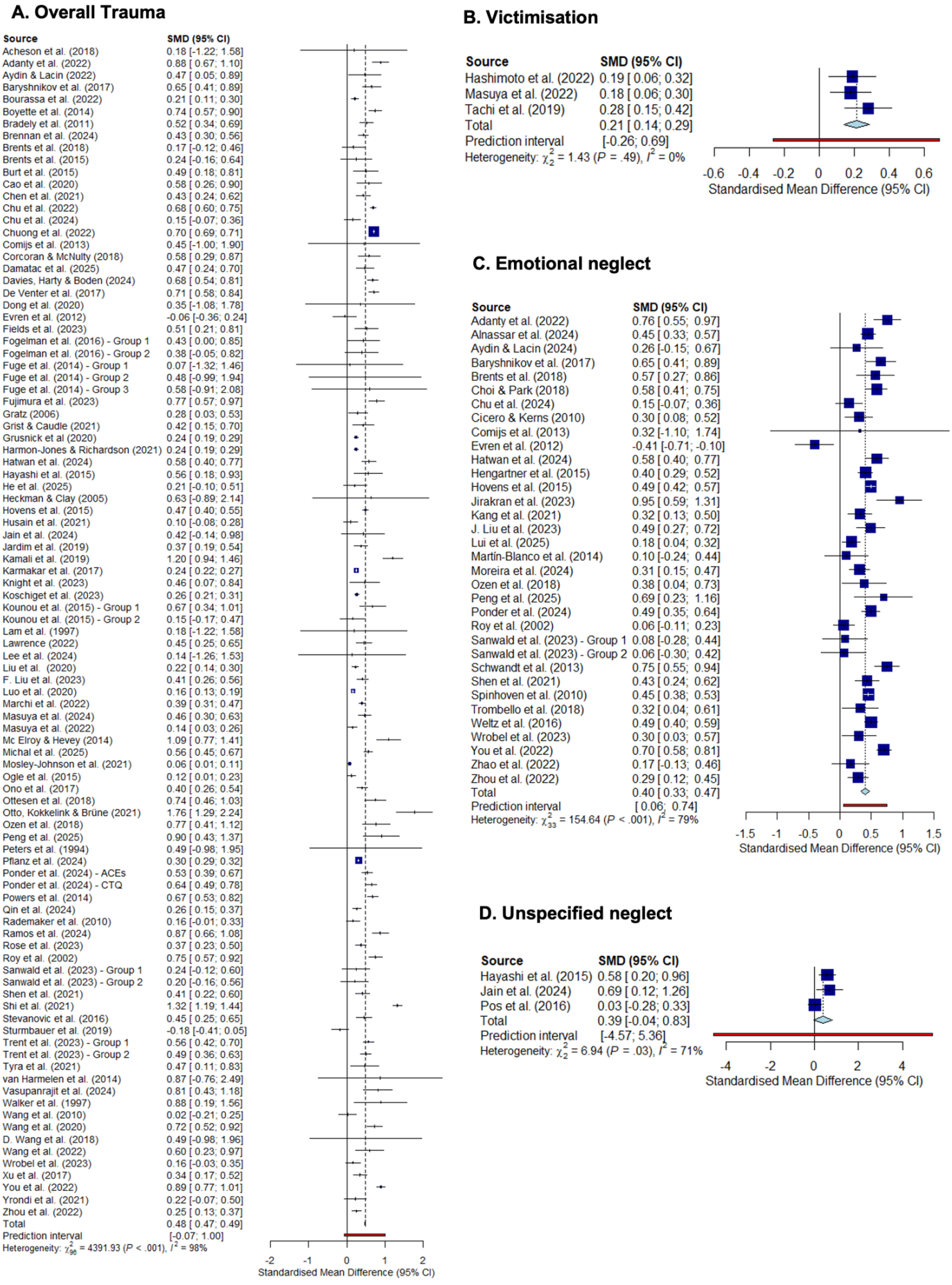


Figure 2.3 - Forest plot demonstrating a medium effect size across studies for the relationship between neuroticism and (A) emotional abuse, and a small effect size for (B) unspecified abuse and (C) physical abuse.

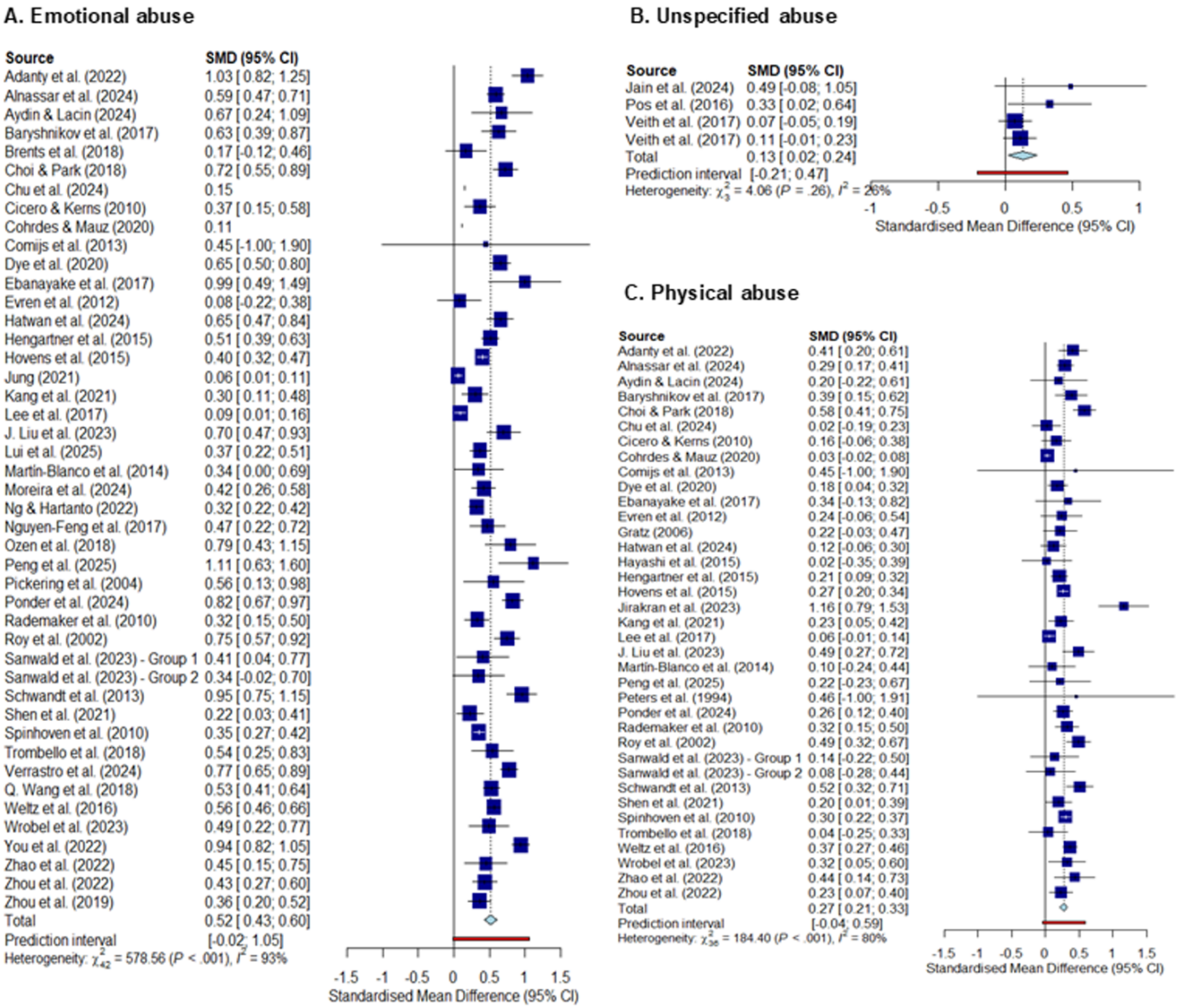


Figure 2.4 - Forest plot demonstrating a small effect size across studies for the relationship between neuroticism and (A) sexual abuse and (B) physical neglect.

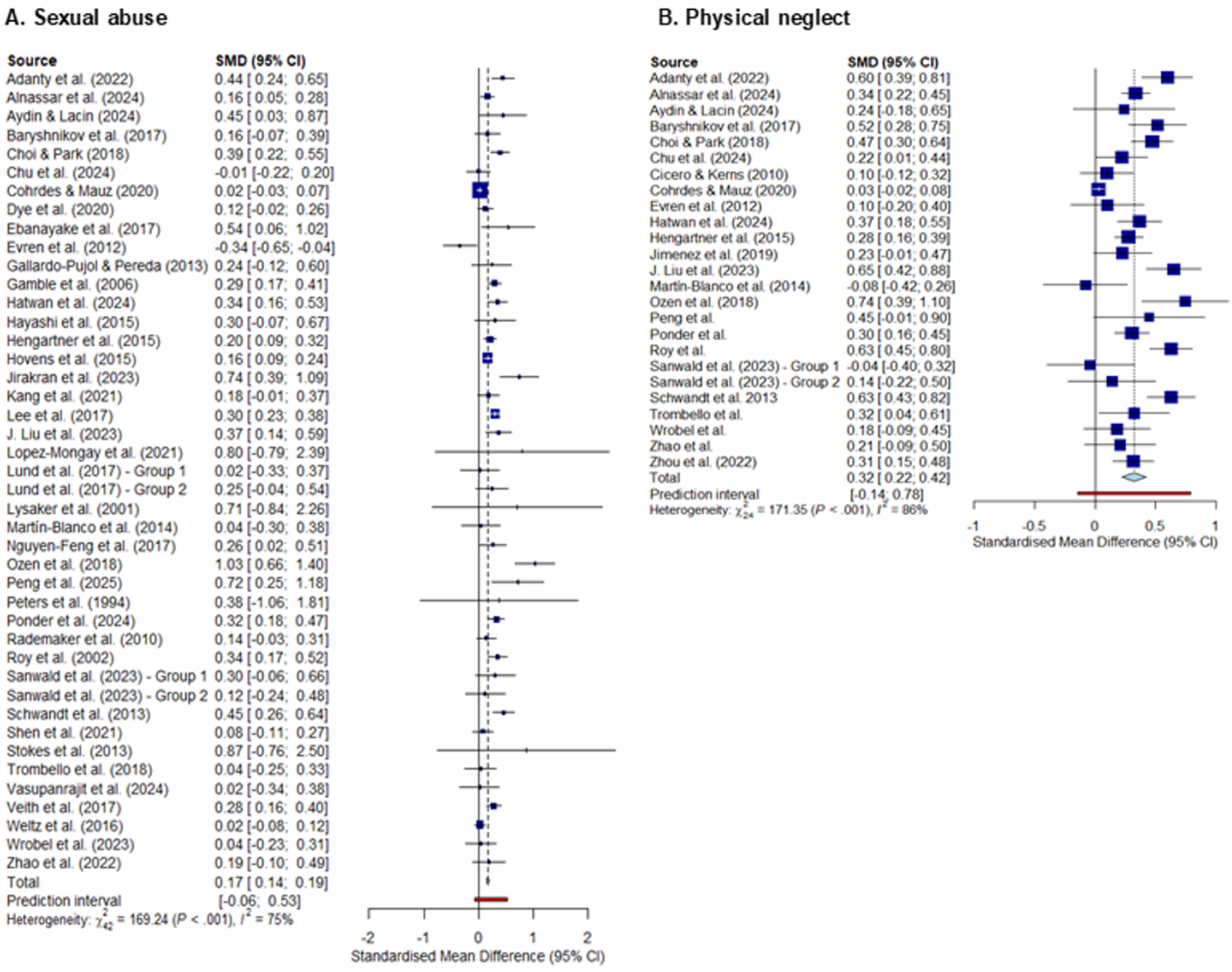
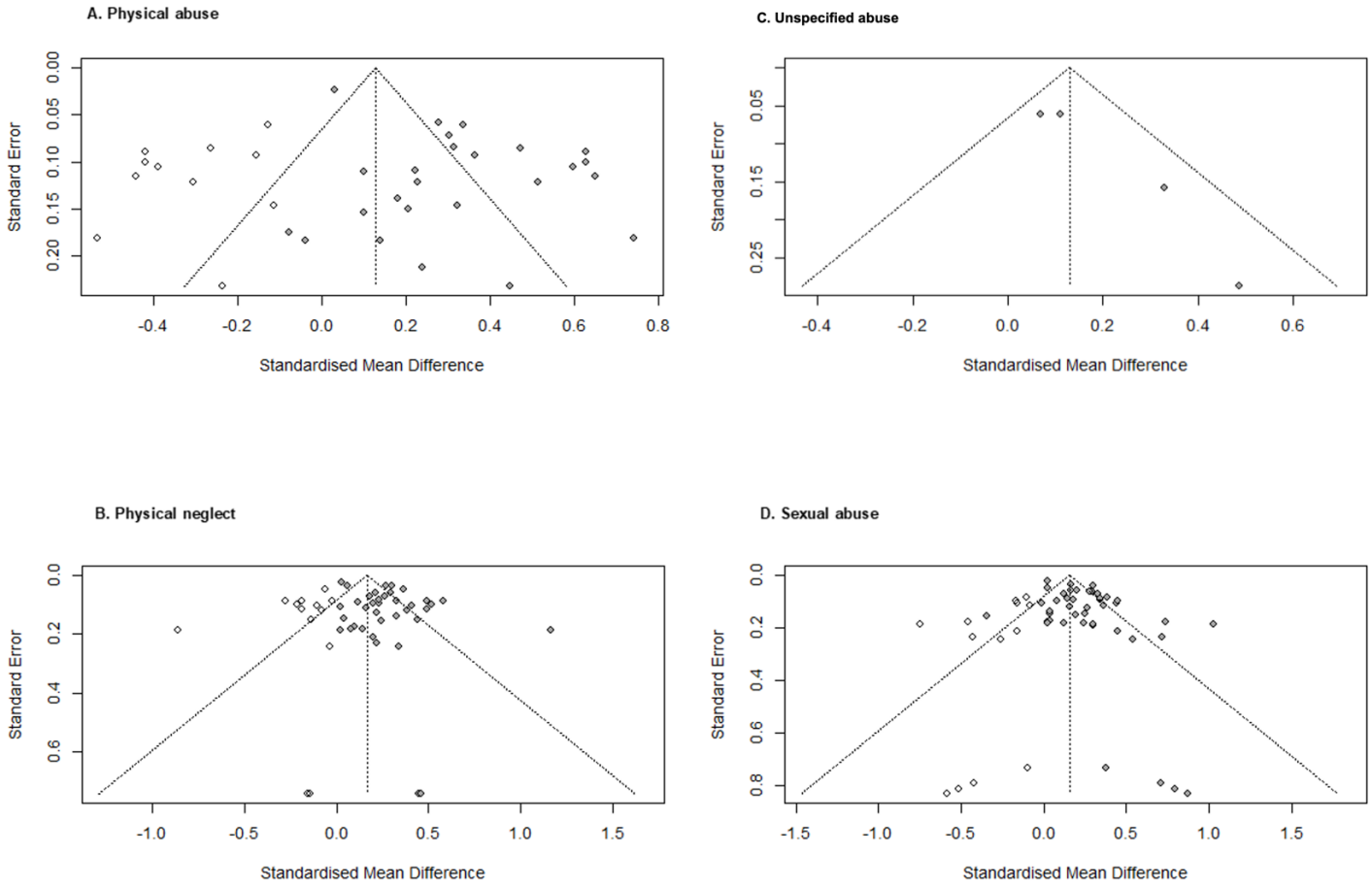


Figure 2.5 - Funnel plots assessing publication bias for studies included that were examining the relationship between neuroticism and (A) physical abuse, (B) physical neglect, (C) unspecified abuse and (D) sexual abuse.



2.5 Discussion

The present meta-analysis is the largest and most comprehensive synthesis of the literature examining the association between childhood trauma (including its subtypes) and adult neuroticism to date. The findings provide robust evidence for an association between early-life adversity and the development of neuroticism in adulthood, underscoring the potential long-term influence of childhood trauma on neuroticism. Overall, childhood trauma was associated with a small-medium effect size, indicating a modest but consistent relationship with adult neuroticism. Similarly, all specific trauma subtypes, including physical abuse, sexual abuse, emotional and physical neglect, and unspecified abuse, were positively associated with neuroticism, each demonstrating small effect sizes. These findings suggest that childhood trauma may lead to adaptions that give rise to neuroticism through several psychological mechanisms such as disruptions in attachment and the formation of negative self-beliefs, and neurobiological alterations in stress regulation systems. These results underscore the importance of systemic preventative measures and early intervention strategies that may alleviate the psychological and neurobiological consequences of trauma, with the potential to increase awareness of adaptions such as neuroticism in trauma-exposed populations.

Building on a growing body of evidence, the present meta-analysis offers robust support for a small but consistent positive association between overall childhood trauma and adult neuroticism. These findings align with foundational theories in developmental psychology, particularly attachment theory, which emphasises the importance of secure early relationships on emotional reactivity and regulation tendencies (Bowlby, 1969, 1998; Bowlby & Solomon, 1989; Mikulincer & Shaver, 2019). Disruptions in caregiving, such as neglect, inconsistency, or maltreatment, can impair the formation of internal working models that foster emotional security. This impairment may heighten susceptibility to psychological processes that are often associated with neuroticism, including emotional reactivity, persistent worry, and vulnerability to stress. In addition to these psychological pathways, early exposure to trauma can lead to dysregulation of the HPA axis, the body's central stress response system. Chronic activation of the HPA axis in response to early adversity has been linked to long-term alterations in cortisol secretion and heightened stress sensitivity, both of which are implicated in the development of neurotic traits (Lupien et al., 2009; McEwen, 2017). By synthesising data from a large and diverse sample, this meta-analysis extends previous findings (Anda et al., 2007; McLaughlin, 2018) and suggests that childhood trauma, irrespective of its specific form, likely constitutes a generalised risk factor for adaptions such as neurotic traits across the lifespan.

Notably, emotional abuse emerged as having one of the strongest associations with neuroticism, showing a medium effect size and suggesting a particularly potent impact on long-

term emotional functioning. Theoretical models, particularly those grounded in attachment theory, may offer valuable insights into these patterns (Bowlby, 1969, 1998; Bowlby & Solomon, 1989; Mikulincer & Shaver, 2019). Attachment theory (Bowlby, 1969; Bowlby & Solomon, 1989) posits that early interactions with caregivers are critical for the development of emotional regulation. Experiences of trauma, especially emotional abuse or neglect, can disrupt the formation of secure attachment bonds (Bifulco et al., 2006; Finzi et al., 2000), which are essential for fostering a stable sense of self and trust in others. The absence of these secure bonds may leave individuals with fewer social resources for safety-seeking and emotion regulation, both in childhood and later life (Hengartner et al., 2015; Hovens et al., 2010; Ponder et al., 2024). This may increase susceptibility to internalising negative self-beliefs, heighten perceived threat in social contexts, and ultimately contribute to elevated neuroticism (Mikulincer, 1998; Mikulincer & Shaver, 2019). Future research is needed to directly test these pathways, ideally using longitudinal or prospective designs that can examine whether disruptions in attachment-related processes mediate the link between specific types of childhood trauma, particularly emotional abuse, and the development of neurotic traits over time.

Given the observed strength of the association for emotional abuse in particular, it is important to consider the neurobiological mechanisms that may underlie this link. For example, early exposure to adversity has been shown to alter the functioning of the HPA axis and shape the development of brain regions involved in emotional regulation, such as the amygdala and prefrontal cortex (McEwen, 2017; Teicher et al., 2016). These changes are often associated with heightened and prolonged physiological responses to stress (Juster et al., 2010; Lupien et al., 2009; McEwen, 2017). Neurobiological adaptations of this kind align closely with core features of neuroticism and negative emotionality (Faravelli et al., 2012; Laird et al., 2019; Teicher et al., 2016; Twardosz & Lutzker, 2010), supporting the idea that trauma-related physiological changes may play a key role in both the emergence and persistence of adaptions such as neuroticism. Individuals high in neuroticism also tend to exhibit altered cortisol reactivity, including blunted responses to acute stress (e.g., during the Trier Social Stress Test; (Kirschbaum et al., 1993)) and disrupted diurnal patterns, with elevated cortisol levels in the morning and evening (Montoliu et al., 2020; Xin et al., 2017). The particularly strong association observed for emotional abuse in our findings may reflect the chronic, interpersonal nature of this trauma subtype, which could exert disruptive effects on stress-regulatory systems and emotional processing circuits in the brain. Future research should aim to examine these neurobiological pathways directly, using longitudinal designs that integrate biological measures (e.g., cortisol reactivity, neuroimaging biomarkers) with detailed assessments of trauma exposure and personality development. Additionally, experimental studies employing psychophysiological methods, such as stress reactivity paradigms, salivary cortisol sampling, or heart rate variability, could offer valuable

insights into how trauma-related disruptions in stress physiology and emotional regulation unfold in real time and underscore neurotic traits. Collectively, these approaches are essential for identifying causal mechanisms, sensitive developmental windows, and trauma subtypes associated with elevated risk, ultimately providing empirical evidence to further refine psychological models of early adversity and informing the development of early interventions

While previous meta-analytic work (Crede et al., 2023) examined the relationship between neuroticism and a range of childhood trauma subtypes, their review was limited to studies published up to 2021, and relied on a more restricted set of databases, potentially omitting relevant research. Thus, 63 peer-reviewed articles have been published since Crede and colleagues had completed their searches, which were included in the present study (15 published in 2021; 15 published in 2022; 11 published in 2023; 17 published in 2024; 5 published up to April 2025). In contrast, the present review extended the search window to April 2025 and employed broader search terms across a wider selection of databases. By addressing these limitations, we were able to identify a larger and more diverse dataset, building on the foundation laid by Crede et al. and offering a more comprehensive synthesis. Our findings were broadly consistent with Crede et al.'s, demonstrating positive associations between childhood trauma and adult neuroticism. Overall, the association between childhood trauma and neuroticism was medium in magnitude (g = 0.48). Emotional abuse and neglect were associated with medium and small-to-medium effects, respectively (g = 0.52 and g = 0.40), whereas physical abuse, physical neglect, and sexual abuse showed small effects (g = 0.15; g = 0.12; g = 0.15). These results replicated Crede et al.'s findings and further suggest that emotional forms of maltreatment exert relatively stronger influences on neuroticism. By quantifying these effects, our study facilitates cross-study comparisons and reinforces the broader literature on the pervasive impact of multiple forms of early adversity on personality development (Boillat et al., 2017; Gamble et al., 2006; Lee & Song, 2017; Pickering et al., 2004; Talbot et al., 2000). These findings align with and further substantiate existing psychological theories of early relational development (e.g., attachment theory, as well as neurobiological research on the lasting effects of early stress exposure and HPA axis dysregulation). They also contribute to a growing body of evidence linking childhood trauma to mental health vulnerabilities later in life, including anxiety (De Venter et al., 2017; He et al., 2024) and mood disorders (Hayashi et al., 2015; Heim et al., 2008), alcohol dependency (Cloninger et al., 1988; Davies et al., 2024; Schwandt et al., 2015; Schwandt et al., 2013), and higher rates of suicidality (Jirakran et al., 2023; Roy, 2002; Zhou et al., 2022). Taken together, these findings emphasise that experiences of interpersonal trauma, particularly those involving betrayal, violation, or neglect by caregivers or other trusted figures during sensitive developmental periods continue to confer meaningful risk for the development of neuroticism in adulthood (D'Andrea et al., 2012; Huh et al., 2014; Van Assche et al., 2020).

When recognising the potentially enduring influence of childhood trauma on the development of neuroticism, it becomes essential to consider how targeted interventions at the micro level, as well as policy change at the meso and macro levels, can help prevent and mitigate these long-term effects (Bronfenbrenner, 1979). From both clinical and public health perspectives, our findings highlight the long-term psychological consequences of childhood trauma and the need for preventive strategies that target early-life risk factors. Interventions such as parenting programmes (Chang et al., 2024; Chen & Chan, 2016; Coore Desai et al., 2017), family-based support services (Goodrum & Prinz, 2022; Kimber et al., 2019), and larger scale policy-level efforts (Bowen & Murshid, 2016; Murphey & Bartlett, 2019) to ensure safe, stable, and nurturing environments for children are essential not only to prevent immediate psychosocial harm but also to reduce the likelihood of neuroticism becoming a longer-term adaption . These approaches have the potential to confer significant downstream benefits in mental health, given the well-established links between neuroticism and increased vulnerability to anxiety, mood disorders, suicidality, and physical health problems (Heim et al., 2008; Lahey, 2009; Roy, 2002; Zhou et al., 2022). Clinically, the findings underscore the relevance of trauma-informed assessment and treatment strategies, particularly for individuals high in neuroticism, who may experience heightened emotional reactivity and stress (Barlow et al., 2014; Widiger & Mullins-Sweatt, 2009). Although neuroticism has been viewed as a stable transdiagnostic trait, growing evidence suggests it can be meaningfully altered through psychological intervention. Evidence supports the use of therapies, such as trauma-focused Cognitive Behavioural Therapy (tf-CBT), Dialectical Behaviour Therapy (DBT), Compassion Focused Therapy (CFT), and Eye Movement Desensitisation and Reprocessing (EMDR) in helping individuals build more adaptive emotion regulation strategies, reshape core beliefs, and cultivate secure relational patterns (Amari & Mahoney, 2022; Bohus et al., 2019; Chen et al., 2018; Ford, 2021; Herman & van der Kolk, 2020; Lewey et al., 2018; Rolling et al., 2024; Sachser et al., 2017; Smith et al., 2024; Whalley & Lee, 2019). Additionally, interventions explicitly targeting neuroticism, such as mindfulness-based cognitive therapy (MBCT; Armstrong & Rimes, 2016; Sauer-Zavala et al., 2017) and neuroticism-focused CBT have shown promise in addressing cognitive and emotional processes linked to high neuroticism, including rumination, emotional avoidance, and internalised self-criticism (Kolesnichenko et al., 2021; Sauer-Zavala et al., 2021). Emerging research also supports the value of Acceptance and Commitment Therapy (ACT) which enhances psychological flexibility and has shown promising outcomes in reducing neurotic perfectionism and internalised self-criticism, core features often aligned with high neuroticism (Khadem Dezfuli et al., 2023). These findings collectively suggest that various therapeutic approaches can modify neurotic traits, offering meaningful clinical benefits and underscore the value of integrating trauma-informed and personality-focused approaches in both prevention and intervention efforts. The evidence provided by this meta-analysis offers a robust empirical foundation for informing clinical practice

and shaping public health strategies aimed at reducing the psychological effects of early adversity and promoting compassion and empowerment for those who have experienced childhood trauma.

There are several limitations of the present review to acknowledge. The EPHPP tool is designed to assess the methodological quality of a broad range of quantitative studies, including, but not limited to, intervention studies (EHPP, 2009; Thomas et al., 2004). Although the adaptations to the EPHPP tool were applied consistently across all included studies, the tool does not formally permit the exclusion of domains. In this review, exclusions were considered necessary due to the inapplicability of certain sections (e.g., blinding and intervention integrity) to the study designs. While these adaptations were applied consistently, and the decision to omit domains was made transparently with clear justification, it nevertheless represents a deviation from the standard application of the tool. This is recognised as a limitation, since modifying the tool in this way may have led to the appraisal results to be biased, which in turn could have influenced the global risk of bias ratings by narrowing the criteria on which studies were assessed due to concerns around the suitability of the chosen appraisal tool. Consequently, the appraisal results should be interpreted with caution, acknowledging the potential for bias introduced by the adapted use of the tool. While this may reduce the risk of artificially inflating or deflating quality scores, it should be recognised that omitting domains can itself affect the global bias rating by narrowing the range of criteria on which a study is judged. Nonetheless, multiple published systematic reviews and meta-analyses have also chosen to omit or modify domains for similar reasons, demonstrating precedent for this approach (e.g., Barbek et al., 2022; Buccini et al., 2024; Chew et al., 2023; Conklin et al., 2018; De Souza et al., 2021; Dunn & Sicouri, 2022; Hill et al., 2022; Li et al., 2014; Madana Civi et al., 2024; Malfliet et al., 2017; Mulligan et al., 2024; Newman et al., 2018; Shah et al., 2020; Silveira et al., 2011; Yang et al., 2023).We further acknowledge that tools specifically developed for observational research may have provided a closer methodological fit for the cross-sectional designs included in this review. In particular, the National Institute of Health (NIH) Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies (National Institutes of Health, 2015) places greater emphasis on aspects such as exposure measurement, temporality, and control of confounding, which are especially relevant in non-interventional designs.

Significant heterogeneity across studies suggests that contextual factors, such as differences in the assessment methods for trauma and neuroticism, study design, and population demographics, may influence the observed effect sizes. The predominance of cross-sectional designs (over 80% of studies) limits the ability to draw conclusions about causality or developmental pathways, particularly in relation to the timing and progression of trauma exposure and the emergence of neurotic traits. Longitudinal studies, which were comparatively

underrepresented, are needed to clarify these temporal dynamics and better identify developmental trajectories. Additionally, variation in clinical presentations (e.g., general population samples vs. clinical groups with PTSD, depression, or bipolar disorder) may moderate the trauma–neuroticism relationship, yet few studies conducted subgroup or moderator analyses to systematically test these effects (He et al., 2024; Moskvina et al., 2007; Zhou et al., 2019). Future research should prioritise subgroup and moderation analyses to examine how associations may differ across gender, age, cultural background, trauma subtype, or clinical status. Such analyses are critical for identifying vulnerable populations and for developing more targeted interventions. Lastly, several trauma subtypes, particularly less commonly studied forms such as emotional neglect or non-interpersonal trauma, were often represented by small sample sizes, reducing statistical power and potentially attenuating effect sizes. Increased attention to understudied trauma types and larger, more diverse samples will be crucial for refining our understanding of the nuanced ways in which early adversity shapes personality development.

Furthermore, while self-report measures are common and often necessary in trauma research, their dominance in the reviewed studies raises concerns about shared method variance and the reliability of retrospective reporting. Memory for traumatic events is subject to distortion over time and can be shaped by current psychological states, emotional salience, and social context (Boskovic et al., 2024; Merckelbach & Muris, 2001). Future work would benefit from multimethod assessment approaches, such as clinical interviews, informant reports, and biological or behavioural markers, to enhance construct validity and triangulate findings. Importantly, potential confounding variables may further complicate interpretation of associations observed in correlational designs. For example, samples were often female-dominated, with a higher number of studies including proportionately more women than men, despite evidence that gender may moderate the relationship between trauma exposure and personality outcomes (Cohen et al., 2024; Cyniak-Cieciura et al., 2022). Thus, the high variability in participant gender distributions, with a strong skew toward female samples in many studies, raises questions about the generalisability of findings across sexes. Likewise, the wide age range across studies (from young adulthood to older adults) and the limited attention to developmental stages may obscure age-specific patterns in how trauma relates to neuroticism.

A further limitation concerns the nature of the samples from which conclusions are drawn. The majority of studies reviewed were conducted in Western populations, particularly in North America and Europe, with relatively few studies based in non-Western or culturally diverse settings. This geographical skew limits the generalisability of findings, as both the expression of neuroticism and the interpretation and reporting of childhood trauma may vary significantly across cultural context which may shape both the expression of neuroticism and the experience

and interpretation of trauma (Boudouda & Gana, 2020; Park et al., 2013; Viola et al., 2016). Moreover, ethnicity was infrequently reported, making it difficult to assess how racial, ethnic, or cultural identity might intersect with trauma and neuroticism. Future studies should aim to include more culturally diverse, gender-balanced, and age-heterogeneous samples, and should consistently report and analyse ethnicity to better understand how cultural background may shape the experience of trauma and its psychological consequences.

In conclusion, this meta-analysis offers robust evidence for a consistent association between overall childhood trauma and adult neuroticism, reinforcing the notion that early adversity may exert a lasting influence on the development of neurotic traits. Importantly, the strength of this association varied by trauma subtype, with emotional abuse showing the strongest link to neuroticism, followed by emotional and physical neglect. These findings underscore the particularly detrimental impact of relational forms of trauma, those involving violations or absences of care from trusted figures, on the development of emotional regulation and self-concept. While associations with sexual abuse and other trauma types were smaller, they remained statistically significant, supporting the view that a wide range of early adverse experiences can increase vulnerability to heightened emotional reactivity and stress sensitivity in adulthood (Alnassar et al., 2024; Schwandt et al., 2018). By integrating a large and diverse body of research, this review not only extends prior meta-analytic work but also provides a clearer, more differentiated understanding of how specific forms of childhood trauma contribute to the development of adaptions such as neuroticism. Promisingly, there are ample opportunities to further research how early adversity leads to adaptions such as neuroticism via longitudinal and multi-method approaches within diverse populations. Such research has the potential to support and inform initiatives at all levels (e.g. micro, meso and macro) that aim to prevent and mitigate the effects of early adversity.

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Chapter 3 The Role of Childhood Trauma, PTSD Symptoms and Pain Coping Strategies in Individuals with Chronic and Acute Pain: A Strength-Based Approach

3.1 Abstract

Associations between chronic pain and childhood trauma or post-traumatic stress disorder (PTSD) symptoms are well-documented, but mechanisms underlying these relationships remain unclear. This study explores the interplay between childhood trauma, PTSD symptoms, and pain coping strategies in individuals with chronic and acute pain. A total of 159 participants (chronic and acute pain groups) completed an online survey assessing childhood trauma, pain coping strategies and current PTSD symptoms. Correlations between variables within each group, and differences between chronic and acute pain groups were evaluated using *z*-tests. Significant positive correlations emerged between pain intensity and childhood trauma (particularly emotional (r = .32) and physical abuse (r = .24)). PTSD symptoms, including re-experiencing (r = .29) and hyperarousal (r = .28), were linked to higher pain intensity. Coping strategies such as distraction and coping self-statements were negatively associated with pain intensity, suggesting potential buffering effects. However, the chronic pain group exhibited stronger negative correlations between certain coping strategies (e.g., distraction, self-statements) and PTSD, as well as childhood trauma-related variables (effect size ranges: z = -2.57 to z = 5.43), indicating more complex coping dynamics. Trauma-related outcomes and PTSD symptoms showed more pronounced associations in the chronic pain group, highlighting the role of pain chronicity. Childhood trauma and PTSD symptoms significantly influence pain perception and coping. Chronic pain sufferers exhibit more complex patterns of coping and trauma-related responses. These results have important clinical implications, emphasising the need for trauma-informed care in pain management interventions. Further research should explore effective strategies for managing the intersection of pain and trauma, particularly in chronic pain populations.

3.2 Introduction

Over the past two decades, a strong link between chronic pain (persistent pain lasting over three months) and post-traumatic stress disorder (PTSD) has been well established (Fishbain et al., 2017). PTSD, characterised by symptoms such as flashbacks, hypervigilance, and avoidance, is significantly more prevalent in individuals with chronic pain (up to 57%) compared to the general population (2–9%; Siqveland et al., 2017). This comorbidity poses significant challenges in pain management, being associated with higher pain severity and greater disability. Additionally, individuals with both conditions often experience heightened PTSD severity (Morasco et al., 2013), emotional distress, and report additional psychiatric comorbidities (Outcalt et al., 2015). In contrast to chronic pain, the International Association for the Study of Pain (IASP) describes acute pain defined as a sudden-onset, time-limited pain typically linked to tissue injury or a specific event, and it serves a protective function, unlike chronic pain, which persists beyond normal healing time and often lacks a clear protective or biological purpose (Nicholas et al., 2019; Treede et al., 2019). Conceptual models, such as shared vulnerability and mutual maintenance, suggest that neurobiological, emotional, and cognitive factors interplay in this comorbidity (Asmundson et al., 2002; Sharp & Harvey, 2001). However, the precise nature of the relationship remains uncertain, with evidence suggesting that pain may contribute to and sustain PTSD, while PTSD can also increase the risk of developing chronic pain. In fact, several longitudinal studies support a bidirectional relationship: in burn survivors and military veterans, PTSD symptoms have been shown to predict later increases in pain intensity, and vice versa (Bair et al., 2020; Benedict et al., 2020; Giannoni-Pastor et al., 2016; Ravn et al., 2018; Stratton et al., 2014; Sveen et al., 2011; Van Loey et al., 2003). Experimental and longitudinal data also indicate that intrusive symptoms in particular, may play a key role in the maintenance of pain (Peter et al., 2011). These findings also align with the fear-avoidance model of pain, which proposes that the interpretation of pain as threat leads to catastrophic thinking, fear, and avoidance behaviours, mechanisms that can reinforce both chronic pain and PTSD symptoms (Vlaeyen & Linton, 2000).

Additionally, history of childhood trauma is a common factor observed alongside PTSD symptoms and chronic pain (Karimov-Zwienenberg et al., 2024). More specifically, experiencing trauma in childhood has been shown to increase the risk of developing symptoms associated with PTSD (Nishith et al., 2000). For example, hyper-arousal is a common symptom present in individuals with PTSD and is often the result of being exposed to early adverse experiences (Kendall-Tackett, 2000). In particular, emotional and sexual abuse in childhood have been strongly linked to high levels of baseline stress (Lemieux & Coe, 1995; Nicolson et al., 2010), which are known to exacerbate pain and physical disability (Hannibal & Bishop, 2014; Pratchett & Yehuda, 2011; Stephens & Wand, 2012).

Repeated exposure to ongoing trauma during childhood is linked to complex symptomatology, encompassing not only post-traumatic stress symptoms but multiple alterations in affective and interpersonal functioning (Cloitre et al., 2005; Kisiel et al., 2009). Moreover, it is well recognised that survivors of childhood adversity may develop more intricate and multifaceted reactions that go beyond those typically observed in PTSD. These reactions are often classified as complex trauma or complex PTSD (CPTSD; Van der Kolk et al., 2005). Furthermore, early life adversities impact mental and physical health in later life, including increased levels of reported pain and a greater likelihood of developing chronic pain conditions (Davis et al., 2005; Lampe et al., 2003).

Pain coping is a critical factor in understanding the complex interplay between chronic pain and PTSD, particularly in individuals with a history of childhood trauma. Models of stress and coping (Zautra & Manne, 1992) highlight chronic pain as a stressor eliciting diverse adaptive responses, influenced by cognitive appraisals of pain and the coping strategies employed. The mutual maintenance model (Asmundson et al., 2002) and theories of shared vulnerability (Geisser et al., 1996; Kuch et al., 1994; Sharp & Harvey, 2001) suggest overlapping psychological mechanisms, such as re-experiencing, avoidance, negative cognitions, and heightened arousal, contribute to the persistence and intensification of both pain and PTSD symptoms. While coping strategies are central to managing pain, identifying those consistently linked to better outcomes has been challenging, with constructs like catastrophising often conflating appraisal and coping (Jensen et al., 1991). Meta-analytic findings (Goldstein et al., 2019) indicate that psychological interventions are more effective in reducing PTSD symptoms than pain outcomes, underscoring a gap in understanding whether improving pain coping can simultaneously alleviate both PTSD and pain conditions. Investigating the relationships between childhood adversity, current PTSD symptoms, and pain coping strategies could inform more integrated, patient-centred interventions. A strength-based approach that focuses on individual coping mechanisms may enhance treatment efficacy for those experiencing co-occurring pain and trauma, emphasising the need for further research to address these interconnected factors.

Chronic pain patients tend to use a wider range of coping strategies compared to those presenting with acute pain (Baastrup et al., 2016). While individuals without pain are more likely to rely on problem-focused and active coping strategies, such as exercise, relaxation techniques, problem-solving, positive self-statements, and distraction, those with chronic pain often use emotion-focused and passive coping strategies (Jensen et al., 1991). It is important to emphasise that in this work, we intentionally move away from pathologising or labelling these strategies as "maladaptive." While some strategies have been associated with greater distress or disability in the literature, they often reflect attempts to manage unpredictable and overwhelming symptoms, particularly in contexts where individuals may feel a lack of control over their pain (Sullivan et al.,

2001). These responses can serve meaningful and functional purposes, such as conserving energy, seeking safety, or attempting to emotionally regulate in the face of persistent discomfort. However, when such responses are relied upon rigidly or without support for alternative approaches, they may inadvertently contribute to a cycle of distress and reduced functioning. In contrast, individuals without chronic pain may use similar strategies (e.g., rest or distraction), but typically in the context of short-term discomfort or for general well-being, rather than as necessary tools for ongoing pain management (Jensen et al., 1991; López-López et al., 2023) (López-López et al., 2023; Jensen et al., 1991).

To our knowledge, there is no empirical research examining the use of pain coping strategies that directly compares pain coping mechanisms between acute and chronic pain populations within the context of trauma. To extend this literature, we aim to investigate the intricate relationships between trauma exposure, pain coping strategies, and pain chronicity by conducting an online survey collecting the following variables: childhood trauma (emotional abuse, emotional neglect, sexual abuse, physical abuse, physical neglect), PTSD symptoms (re-experiencing, avoidance, hyperarousal, and negative alterations in cognition and mood), and pain coping strategies (catastrophising, distraction, ignoring, distancing, coping self-statements, and praying). Pain coping strategies were assessed in relation to pain experienced within the last month, with participants indicating whether the pain was chronic or acute. Understanding how individuals with trauma histories cope with acute versus chronic pain is important not only for advancing theoretical models, such as the mutual maintenance model and fear-avoidance model, but also for informing psychological interventions. In particular, identifying how coping strategies may differ across pain types and trauma exposure can guide more targeted, trauma-informed approaches in therapies such as Cognitive Behavioural Therapy (CBT), Acceptance and Commitment Therapy (ACT), and Compassion-Focused Therapy (CFT), where addressing unhelpful coping patterns and fostering psychological flexibility can support improved outcomes for individuals living with pain and trauma (Åkerblom et al., 2024; Birdsey, 2020; Lumley et al., 2022).

We hypothesised that:

(H1) individuals with chronic pain will report significantly greater exposure to trauma compared to those with acute pain, reflecting the potential role of trauma in the development and maintenance of chronic pain conditions (Davis et al., 2005; Karimov-Zwienenberg et al., 2024).

(H2) pain coping strategies will differ between acute and chronic pain groups, with individuals experiencing chronic pain more likely to engage in coping responses oriented toward managing emotional distress or perceived threat, such as

heightened pain-related worry and activity reduction compared to those with acute pain (Baastrup et al., 2016; Jensen et al., 1991; Vlaeyen & Linton, 2000).

(H3) the type of childhood trauma experienced will be correlated with the severity of both pain and current PTSD symptoms (Karimov-Zwienenberg et al., 2024).

(H4) the association between trauma exposure and pain coping strategies will vary depending on whether an individual experiences acute or chronic pain (Siqveland et al., 2017).

3.3 Methods

3.3.1 Participants

Participants in the analyses were recruited internationally to complete a cross-sectional online survey. Eligibility criteria included being at least 18 years old and able to read and understand English. The survey, hosted on Qualtrics, was distributed via various online platforms to ensure a diverse sample.

Based on previous research (Cohen, 2013; Hruschak et al., 2021; Neville et al., 2018) we used a small effect size to determine this study's sample size. In G*power (Faul et al., 2009), we ran a power analysis for a bivariate correlation (r = 0.2 with α = .05 , β = .8). The power calculation suggested recruiting a minimum of 153 participants, consistent with previous studies that used a cross-sectional survey data design (Hirsh et al., 2011; Lee et al., 2019). We ran two additional power analyses: one for the planned one-way ANOVAs (f = 0.25 with α = .95, β = .8), which suggested a sample size of at least 128, and one to determine the appropriate sample size for comparing two correlation coefficients (q = 0.5, α = .05 , β = .8), which showed that a sample size of 132 would be sufficient. Given these analyses, we oversampled to increase power and to account for potential dropout for online data.

A total of 213 participants were initially recruited for this study. Participants were recruited through online forums focused on trauma-related and pain-related content. These forums advertised our study to their members, providing information about the research objectives and participation details. The advertisements reached a diverse audience, including individuals with lived experiences relevant to the study's focus. The forums who advertised the study details included MyPTSD, Sexual Violence Research Initiative, Blue Knot Foundation, PainConcern, CRPS UK, Pain Relief Foundation, Guts, UK. Given the nature of online data collection, responses were screened for potential bot activity to ensure data quality. The following variables were measured: childhood trauma (emotional neglect, emotional abuse, sexual abuse, physical neglect, physical abuse), current post-traumatic stress symptoms (re-experiencing, avoidance, hyperarousal, negative alterations in cognition and mood), pain coping strategies (catastrophising, distraction, ignoring, distancing, self-coping statements, praying). After excluding participants who did not complete the key questionnaires (CTQ-SF, PCL-5, CSQ-R) and those identified as bot responses, 159 participants remained (117 females; 40 males; 2 preferred not to disclose their sex). The mean age of participants was 37.36 years (SD = 12.22), ranging from 20 to 78 years. The sample was internationally diverse, with participants representing multiple continents, including Europe (n = 121), North America (n = 47), Australia (n = 18), Africa (n = 6), Asia (n = 5), and South America (n = 3), while 13 participants did not provide nationality data. In terms of ethnicity, the sample comprised individuals identifying as White (n =

173), Mixed (*n* = 12), Asian (*n* = 6), Other (*n* = 6) and Black (*n* = 4), with 13 participants not reporting their ethnicity.

3.3.2 Questionnaires

3.3.2.1 Idiosyncratic questions about pain

To assess the presence of chronic and acute pain, participants were asked to confirm their diagnosis, describe the nature of their pain, and indicate how long they had been experiencing it, ensuring the condition met the diagnostic criterion of more than three months (Merskey, 1986). This approach allowed us to distinguish between individuals with chronic pain, who rated their coping strategies in relation to their ongoing condition as experienced within the past month, and those with acute pain, who rated their coping strategies in response to an episode of acute pain experienced during the same period. This reference pain was used to guide responses to the Coping Strategies Questionnaire – Revised (CSQ-R), which evaluates the coping strategies participants use during pain episodes.

3.3.2.2 Coping Strategies Questionnaire-Revised – CSQ-R

The CSQ-R is a pain coping assessment tool that was designed to evaluate the extent to which patients employ six distinct cognitive coping strategies and two behavioural coping strategies (Riley & Robinson, 1997). The CSQ contains 27 items that load onto six subscales (distraction, catastrophising, ignoring pain, distancing from the pain, coping self-statements, praying). Participants rate responses on a 7-point Likert scale, ranging from 0 (never do that) to 6 (always do that), to indicate how often they employ particular coping activities when they experience pain. The pain catastrophising subscale consists of six items. Higher scores indicate more pain catastrophising (scores range from 0 to 36). All subscales demonstrated acceptable to excellent internal consistency, with the following Cronbach's alpha values: catastrophising (α = .91), distraction (α = .89), ignoring (α = .86), distancing (α = .91), coping self-statements (α = .84), and praying (α = .86).

Although the CSQ-R includes a subscale traditionally labelled "pain catastrophising," we made a deliberate decision not to use this term in the present manuscript. This choice was informed by discussions with individuals with lived experience of complex trauma and chronic pain, who shared that such language does not reflect the nuance of their experiences and may feel pathologising or invalidating. While we acknowledge that "catastrophising" is a well-established and psychometrically valid construct within the pain literature, we also recognise the importance of using inclusive and compassionate language, particularly given that this work is intended for both academic and clinical audiences, as well as individuals with lived experience.

Therefore, in alignment with trauma-informed and person-centred values, we refer to this construct using more neutral descriptors such as “pain-related worry”, which we believe better capture the function of this coping response without imposing deficit-based assumptions.

3.3.2.3 Childhood Trauma Questionnaire (Short Form) – CTQ-SF

The Childhood Trauma Questionnaire-Short Form (CTQ-SF) is a 28-item self-report questionnaire designed to evaluate a history of childhood maltreatment. It was developed through exploratory and confirmatory factor analyses of the original 70-item version (Bernstein et al., 2003). The CTQ-SF uses a five-point Likert scale for respondents to rate the items, ranging from 0 ("never") to 5 ("very often"). This questionnaire comprises five clinical subscales: Sexual, Physical, and Emotional Abuse, as well as Physical and Emotional Neglect. Scores from each subscale (5 to 25) can be summed to a total score (5-125). Our data showed that all subscales showed excellent to acceptable internal consistency ($\alpha > .70$), with the following values: emotional abuse ($\alpha = .89$), physical abuse ($\alpha = .85$), sexual abuse ($\alpha = .95$), emotional neglect ($\alpha = .90$), and physical neglect ($\alpha = .75$).

3.3.2.4 Posttraumatic Stress Disorder Checklist - PCL-5

The PCL-5 is a self-report questionnaire consisting of 20 items designed to assess the extent to which an individual has experienced distress in the past month due to PTSD symptoms as defined by the Diagnostic and Statistical Manual of mental Disorders Fifth Edition (DSM-5), related to their most currently distressing event (Weathers et al., 2013). Respondents rate items on a 5-point Likert scale from 0 (“not at all”) to 4 (“extremely”), which is added up to a *total severity* score (0 to 80). The four subscales represent the DSM-5 PTSD symptom clusters: re-experiencing, avoidance, negative alterations in cognitions and mood and hyper-arousal. The current study had internal consistency (Cronbach’s alpha) of $\alpha = .92$ for **B** symptoms (Re-experiencing), $\alpha = .88$ for **C** symptoms (Avoidance), $\alpha = .93$ for **D** symptoms (Alterations in Cognition and Mood), and $\alpha = .90$ for **E** symptoms (Hyperarousal).

3.3.3 Procedure

Informed consent was obtained electronically before participation, and all responses were anonymised to maintain confidentiality. Ethical approval for this study was obtained from the Ethics Committee at the University of Southampton [ERGO: 92746], and all procedures adhered to the ethical guidelines outlined in the Declaration of Helsinki.

Participants were recruited through the Qualtrics recruitment panel and completed the study online via the Qualtrics survey platform. Upon accessing the survey, participants first

provided informed consent before proceeding to the questionnaire. They were informed that they could opt in to participate in a random prize draw at the end of data collection. To ensure data completeness, participants were required to respond to all questions on each page before progressing further (see description of questionnaires above). In addition to the above questionnaires, participants were asked to complete measures on dissociation (Dissociative Experiences Scale - DES-II (Carlson & Putnam, 2000)), paranoia (The Revised Green et al. Paranoid Thoughts Scale – R-GPTS (Freeman et al., 2021)) and intolerance of uncertainty (Intolerance of Uncertainty – IUS-12 (Carleton et al., 2007)). However, data from these additional measures were not included in the present analysis as they were beyond the scope of the current study's research questions. Only participants who consented to their data being used and who completed all core measures relevant to the study's aims were included in the final sample.

3.3.4 Data analysis

Statistical analyses were conducted using SPSS version 29.0 (SPSS, Inc., Chicago, Illinois). Initially, descriptive statistics were calculated to assess the distributions of childhood trauma, current PTSD symptoms, and pain-related variables among individuals with chronic and acute pain. To determine whether there were significant differences in trauma severity between individuals with chronic and acute pain, independent samples t-tests were conducted. Subsequently, independent t-tests was performed to examine whether individuals with chronic pain utilised different pain coping strategies compared to those with acute pain. Due to non-parametric data (see Appendix 1), non-parametric correlation analyses (Spearman's rho) were conducted to investigate the relationships between childhood trauma, current PTSD symptoms, pain unpleasantness, pain intensity, and pain coping strategies across the full sample. Additionally, correlation analyses were conducted separately for two subsamples: (a) individuals with chronic pain and (b) individuals with acute pain, to explore potential differences in these associations within each group. Finally, *r*-to-*z* transformations were employed to compare the strength of correlations between trauma and pain coping strategies across the chronic and acute pain groups, testing for significant differences in these relationships based on pain chronicity. Statistical significance was set at $p < .05$ for all analyses. Effect sizes were interpreted following conventional guidelines, with correlation coefficients (r) of .10, .30, and .50 representing small, medium, and large effects, respectively (Cohen, 1988). Similarly, for Cohen's d, values of .2, .5, and .8 were considered small, medium, and large effects, respectively (Cohen, 1988). Interpreting the magnitude of r followed Cohen's conventional benchmarks, with small, medium, and large effects reflecting increasingly meaningful associations. When using r-to-z transformation to compare correlations, the z-scores were evaluated for statistical significance, with $|z| \geq 1.96$ considered indicative of a meaningful difference at $p < .05$.

3.4 Results

3.4.1 Descriptive statistics

3.4.1.1 Pain-related characteristics

Out of a total sample of 159 participants, 74 (46.5%) were in the chronic pain group and 85 in the acute pain group. When completing the CSQ-R, participants were asked to rate the intensity and unpleasantness of the pain they had experienced in the past month, as identified at the beginning of the questionnaire (i.e., their specific chronic pain or a particular episode of acute pain). Participants with chronic pain reported significantly higher levels of pain intensity (M = 63.11; SD = 23.44; $t(156) = 3.41$, $p < .01$, $d = .54$) and pain unpleasantness (M = 65.68; SD = 25.85; $t(156) = 3.88$, $p < .01$, $d = .62$) compared to those experiencing acute pain (intensity: M = 49.88; SD = 25.15; unpleasantness: M = 49.45; SD = 26.54). These findings highlight the greater severity of pain-related experiences in the chronic pain group. Data was normally distributed (see Appendix 1B).

3.4.1.2 Trauma-related characteristics

Childhood trauma

A substantial proportion of participants reported experiences of childhood abuse and neglect, with varying levels of severity (Table 3.1). Emotional abuse was reported at severe levels by 20.2% of the sample, while 13.6% reported severe physical abuse and 15% reported severe sexual abuse. Emotional neglect was the most commonly reported form of maltreatment, with 30% of participants endorsing at least low levels of exposure.

Current PTSD symptoms

Based on the PCL-5 a total symptom severity score was calculated, with a cutoff score of 33 used to indicate moderate to severe PTSD symptoms (Weathers et al., 2013). Based on this criterion, 82.1% of participants met the threshold for moderate-severe PTSD symptoms, while 17.9% scored below the cutoff. This distribution suggests a high prevalence of moderate to severe PTSD symptoms within the sample.

Table 3.1 - Severity of Childhood Trauma Questionnaire (CTQ) Subscales

CTQ Subscale	None (%)	Low (%)	Moderate (%)	Severe (%)
Emotional Abuse	23.5	18.8	10.3	20.2
Physical Abuse	39.9	7.5	11.7	13.6
Sexual Abuse	38.0	4.2	15.5	15.0
Emotional Neglect	18.8	30.0	12.2	13.6
Physical Neglect	29.6	12.7	18.3	14.1

Note. Percentages reflect the proportion of participants reporting different levels of childhood maltreatment severity on each CTQ subscale.

3.4.2 Prevalence of trauma in chronic pain

One-way ANOVAs were conducted to examine differences in childhood trauma and PTSD symptoms between individuals reporting chronic and acute pain. Individuals with chronic pain reported significantly more childhood trauma (CTQ-SF total score; Table 3.2): $t(1,153) = 3.14$, $p = .002$, $d = .51$) across most domains, including emotional abuse $t(1,153) = 2.23$, $p = .03$, $d = .36$), physical abuse $t(1,153) = 3.93$, $p < .001$,$d = .63$), sexual abuse $t(1,153) = 3.59$, $p = .005$, $d = .20$), and physical neglect $t(1,153) = 2.18$, $p = .03$, $d = .35$), compared to those with acute pain. However, there was no significant difference between the groups in emotional neglect $t(1,153) = 1.21$, $p = .23$, $d = .16$). In addition to greater childhood trauma exposure, individuals with chronic pain also reported more severe PTSD symptoms (PCL-5 total score: $t(149) = 5.74$, $p < .001$, $d = .94$), including higher levels of re-experiencing ($t(149)= 5.11$, $p < .001$, $d = .83$), avoidance ($t(149) = 2.55$, $p = .01$, $d = .42$), hyperarousal ($t(149) = 6.12$, $p < .001$, $d = 1.00$), and negative alterations in cognition and mood ($t(149) = 5.28$, $p < .001$, $d = .86$). These findings suggest that chronic pain is associated with both a history of more extensive childhood trauma and more pronounced PTSD symptomatology.

Table 3.2 - Descriptive Statistics for Childhood Trauma Questionnaire (CTQ) by pain chronicity

Scale	Chronic Pain (*n* = 75)		Acute Pain (*n* = 84)	
	Mean	**SD**	**Mean**	**SD**
CTQ - total score	56.68	17.41	47.66	18.21
Emotional abuse	13.18	5.33	11.22	5.57
Physical abuse	9.92	4.32	7.45	3.50
Sexual abuse	10.17	5.84	7.82	5.44
Emotional neglect	13.50	4.66	12.53	5.21
Physical neglect	9.92	3.51	8.65	3.70
PCL - total score	63.20	17.63	45.71	19.59
Re-experiencing	15.41	4.88	11.08	5.47
Avoidance	6.62	2.30	5.56	2.74
Hyperarousal	18.81	5.61	13.00	6.02
Negative alterations in cognition and mood	22.35	6.92	16.08	7.60

3.4.3 Use of Pain Coping Strategies for Chronic and Acute Pain

One-way ANOVAs were conducted to examine differences in pain coping strategies between individuals with chronic pain and those with acute pain. Individuals with chronic pain were significantly more likely to use a range of coping strategies, including pain-related worry ($t(156) = 3.75$, $p < .001$, $d = .60$; Table 3.3), distraction ($t(156) = 5.31$ $p < .001$, $d = .85$),), distancing ($t(156) = 3.40$, $p < .001$, $d = .54$), coping self-statements ($t(156) = 3.28$, $p < .001$, $d = .52$) and praying ($t(156) = 3.02$, $p = .001$, $d = .48$), when reflecting on their most recent pain experience within the past month and related to their chronic pain. However, there was no significant difference between the groups in the use of ignoring as a coping strategy ($t(156) = .56$, $p = .58$, $d = .09$).

Table 3.3 - Descriptive Statistics for Pain Coping Strategies (CSQ-R) by pain group

CSQ-R subscale	Chronic Pain (*n* = 75)		Acute Pain (*n* = 84)	
	Mean	**SD**	**Mean**	**SD**
Pain-related worry	16.41	9.40	10.95	8.88
Distraction	15.91	7.63	9.77	6.88
Ignoring	12.27	7.64	11.63	6.82
Distancing	9.43	7.24	5.70	6.50
Coping Self-statements	16.10	7.12	13.30	5.41
Praying	7.36	5.53	4.60	5.65

3.4.4 Relationship between childhood trauma history, current PTSD symptoms and pain coping strategies

In the full sample, non-parametric correlations revealed that pain intensity and pain unpleasantness were strongly associated ($r = 0.89$ $p < 0.001$), reflecting their conceptual overlap. Childhood trauma, particularly emotional abuse ($r = 0.32$, $p < 0.001$) and physical abuse ($r = 0.24$, $p = 0.003$), showed moderate positive correlations with pain intensity, while emotional and physical neglect were similarly associated with both pain and PTSD symptoms (Table 3.4). PTSD symptoms, especially re-experiencing and avoidance, were significantly correlated with higher pain intensity and unpleasantness (e.g., PCL total score, $r = 0.32$, $p < 0.001$). Pain-related worry emerged as a key coping strategy linked to heightened pain ($r = 0.362$, $p < 0.001$) and PTSD symptoms, whereas strategies like distraction and distancing showed weaker associations (Table 3.4).

Table 3.4 - Correlations between pain ratings, childhood trauma and PTSD symptoms (n = 159)

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	Pain Intensity	1.000																	
2	Pain Unpleasantness	.888**	1.000																
3	CTQ - Total Score	.257**	.227**	1.000															
4	CTQ - Emotional Abuse	.316**	.264**	.878**	1.000														
5	CTQ - Physical Abuse	.241**	.235**	.757**	.614**	1.000													
6	CTQ - Sexual Abuse	.168*	.169*	.584**	.406**	.424**	1.000												
7	CTQ - Emotional Neglect	.185*	.172*	.786**	.716**	.495**	.263**	1.000											
8	CTQ - Physical Neglect	.030	.031	.732**	.518**	.528**	.293**	.575**	1.000										
9	PCL - Total Score	.322**	.343**	.541**	.506**	.433**	.404**	.357**	.415**	1.000									
10	PCL - Re-experiencing	.293**	.292**	.473**	.427**	.403**	.359**	.302**	.402**	.908**	1.000								
11	PCL - Avoidance	.281**	.274**	.466**	.486**	.344**	.301**	.315**	.314**	.743**	.660**	1.000							
12	PCL - Negative Alterations in Cognition and Mood	.296**	.325**	.530**	.515**	.394**	.440**	.361**	.356**	.944**	.787**	.640**	1.000						
13	PCL - Hyperarousal	.279**	.315**	.469**	.420**	.390**	.329**	.302**	.405**	.935**	.792**	.626**	.844**	1.000					
14	CSQ - Pain Catastrophising	.362**	.392**	.273**	.260**	.268**	.131	.246**	.226**	.546**	.487**	.449**	.524**	.505**	1.000				
15	CSQ - Distraction	-.011	.016	-.014	-.056	.106	.033	-.103	.118	.145	.170*	.061	.089	.157	.283**	1.000			
16	CSQ - Ignoring	-.152	-.191*	.009	.014	-.018	.027	.031	-.108	.088	.053	.099	.091	.070	-.151	.270**	1.000		
17	CSQ - Distancing	-.065	-.059	.123	.106	.156	.162*	.024	.126	.349**	.346**	.278**	.296**	.319**	.249**	.538**	.439**	1.000	
18	CSQ - Coping Self-statements	.072	.109	.076	.084	.123	.068	.021	.006	.172*	.195*	.167*	.125	.148	.012	.421**	.527**	.369**	1.000
19	CSQ - Praying	-.003	.044	-.011	-.087	.127	.046	-.073	.149	.209**	.279**	.074	.145	.204*	.402**	.467**	-.080	.300**	.198*

*$p < .01$; **$p < .001$

Within the chronic pain sample, the correlations showed several significant associations between pain, childhood trauma, current PTSD symptoms, and coping strategies. Pain intensity was strongly correlated with pain unpleasantness (r = .84, p < .001) and showed significant positive relationships with childhood trauma, particularly emotional abuse (r = .32, p < .001) and physical abuse (r = .74, p < .001). Additionally, pain intensity was positively associated with PTSD symptoms, including re-experiencing (r = .33, p < .001) and hyperarousal (r = .34, p < .001). Notably, coping strategies such as distraction (r = -.29, p < .05) and coping self-statements (r = -.42, p < .001) were negatively correlated with pain intensity, suggesting that these strategies may buffer against pain severity. Emotional neglect was significantly associated with higher PTSD symptoms (PCL total score, r = .39, p < .001), while praying (r = -.24, p < .05) was negatively correlated with pain intensity, indicating potential protective effects (see appendix 2B, Table 3.5).

Within the acute pain group, significant correlations revealed meaningful relationships between psychological and pain-related variables. Pain intensity was highly correlated with pain unpleasantness (r = 0.92, p < .001), underscoring their close association. Childhood trauma, particularly emotional abuse, showed a strong correlation with the total CTQ score (r = 0.87, p < .001), indicating consistency within trauma subscales. Current PTSD symptoms were also interrelated, as shown by the strong association between the PCL total score and re-experiencing symptoms (r = 0.85, p < .001). Importantly, pain-related worry was significantly associated with both pain intensity (r = 0.26, p = .02) and PTSD re-experiencing symptoms (r = 0.50, p < .001), suggesting its role in amplifying both physical and psychological distress. Conversely, non-significant correlations, such as between pain intensity and distraction (r = 0.02, p = .87), indicate that some coping strategies may have limited direct influence on pain perception (see appendix 3B, Table 3.6) for individuals who reported managing acute pain.

3.4.5 Difference in relationship between trauma and pain coping by pain chronicity

3.4.5.1 Relationship between childhood trauma and pain coping

In comparing the chronic pain and acute pain groups, significant differences emerged in the correlations between pain coping strategies and both childhood trauma and current post-traumatic stress symptoms (Figure 3.1). For childhood trauma, although no significant group differences were observed in the correlation between coping strategies and the CTQ total score for pain-related worry (z = -0.78, p = 0.435), ignoring (z = -1.57, p = 0.116), or praying (z = -0.97, p = 0.322), significant differences were found for distraction (z = -2.57, p = 0.010), distancing (z = -

3.17, $p < 0.001$), and coping self-statements ($z = -3.30$, $p < 0.001$), with the chronic pain group showing stronger negative associations.

At the subscale level, emotional neglect was more strongly negatively correlated with distraction ($z = -3.59$, $p < 0.001$), distancing ($z = -3.78$, $p < 0.001$), and self-statements ($z = -2.79$, $p = 0.005$). Physical neglect also showed stronger negative correlations with ignoring ($z = -2.84$, $p = 0.004$), distancing ($z = -4.02$, $p < 0.001$), and self-statements ($z = -3.53$, $p < 0.001$). Emotional abuse was more negatively associated with distraction ($z = -2.78$, $p = 0.005$), distancing ($z = -2.53$, $p = 0.011$), and self-statements ($z = -2.13$, $p = 0.033$). Additionally, physical abuse and sexual abuse each showed one significant group difference: self-statements were more negatively correlated with physical abuse ($z = -2.90$, $p = 0.003$) and sexual abuse ($z = -2.08$, $p = 0.038$).

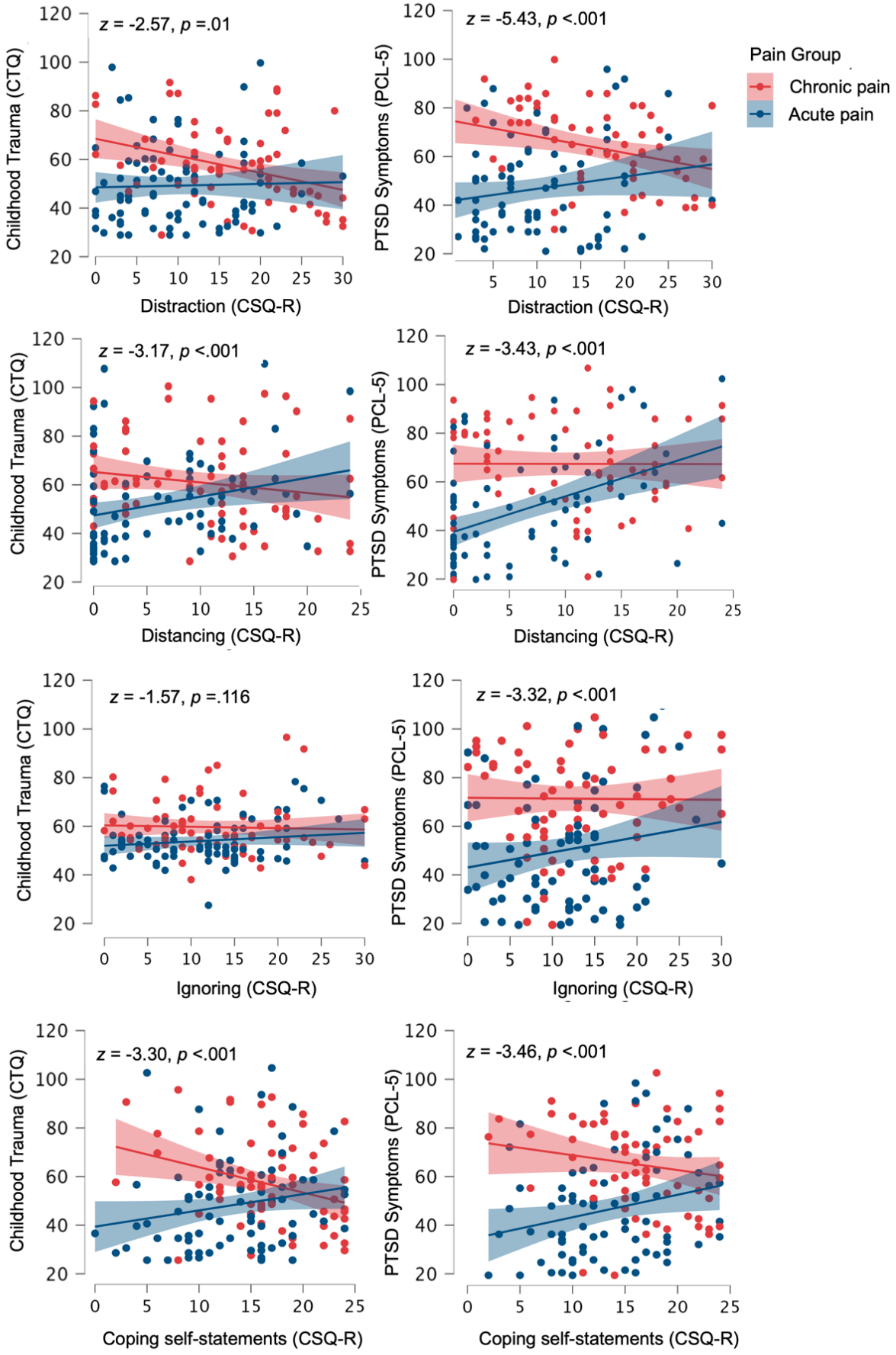
3.4.5.2 Relationship between current PTSD symptoms and pain coping

In relation to PTSD symptoms, although no significant group differences were found in the correlations between PCL total scores and pain-related worry ($z = 0.08$, $p = 0.936$) or praying ($z = -1.14$, $p = 0.25$), stronger negative associations were observed for distraction ($z = -5.43$, $p < 0.001$), ignoring ($z = -3.32$, $p < 0.001$), distancing ($z = -3.43$, $p < 0.001$), and self-statements ($z = -3.46$, $p < 0.001$) in the chronic pain group.

At the subscale level, re-experiencing symptoms were more negatively correlated with distraction ($z = -3.12$, $p = 0.001$) and self-statements ($z = -2.45$, $p = 0.014$). Avoidance was more negatively associated with distraction ($z = -2.02$, $p = 0.043$) and self-statements ($z = -2.43$, $p = 0.015$). Similarly, negative alterations in cognition and mood showed stronger negative correlations with distraction ($z = -2.72$, $p = 0.006$), distancing ($z = -2.79$, $p = 0.005$), and self-statements ($z = -3.64$, $p < 0.001$). Hyperarousal was also more negatively correlated with distraction ($z = -4.55$, $p < 0.001$), distancing ($z = -2.20$, $p = 0.028$), and self-statements ($z = -4.51$, $p < 0.001$).

In sum, individuals with chronic pain exhibit distinct patterns of coping in relation to both childhood trauma and PTSD symptoms, with more pronounced negative associations between certain adaptive coping strategies, particularly distraction, distancing, and self-statements and specific forms of childhood adversity and post-traumatic symptomatology (see full table of significant differences between correlation in appendix 4B; Table 3.7).

Figure 3.1 – Significant differences in correlations of pain coping strategies (CSQ-R) and childhood trauma (CTQ-SF) as well as current PTSD symptoms (PCL-5) between individuals who reported acute and chronic pain in the last month. R-to-z transformations performed. Confidence intervals of 95% displayed.



3.5 Discussion

We examined the relationship between childhood trauma, current PTSD symptoms and pain coping strategies and compared these between individuals with chronic and acute pain. We observed that individuals with chronic pain reported significantly greater exposure to childhood trauma compared to those with acute pain, particularly in domains such as emotional, physical, and sexual abuse, as well as physical neglect (H1). Additionally, we found that individuals with chronic pain were more likely to use a range of coping strategies, including pain-related worry, distraction, distancing, coping self-statements, and praying, whereas no significant differences emerged in the use of ignoring as a coping strategy (H2). Furthermore, pain intensity was strongly associated with childhood trauma history, PTSD symptoms, and several coping strategies, with emotional and physical abuse correlating with greater pain severity and PTSD symptoms such as re-experiencing and hyperarousal (H3). Finally, the association between trauma exposure and pain coping strategies varied between chronic and acute pain groups, with chronic pain individuals demonstrating stronger negative correlations between coping self-statements and PTSD symptoms, as well as more pronounced associations between certain trauma subtypes and specific coping strategies (H4). These findings suggest that the interplay between trauma history, PTSD symptoms, and pain coping mechanisms is more complex in chronic pain populations, highlighting the need for tailored interventions targeting both trauma and pain management strategies.

Our findings are in line with the well-established link between childhood trauma (Davis et al., 2005; Lampe et al., 2003), post-traumatic stress symptoms (Karimov-Zwienenberg et al., 2024), and pain experiences, supporting theoretical models such as the mutual maintenance model of chronic pain and PTSD (Asmundson et al., 2002). This model suggests that the interaction between trauma-related distress and pain perception creates a reinforcing cycle, wherein physiological hyperarousal, heightened threat sensitivity, and cognitive-emotional processes contribute to the persistence of both pain and psychological distress. The strong associations observed between pain intensity and both childhood emotional and physical abuse, as well as PTSD symptoms such as re-experiencing and hyperarousal, align with this perspective. These findings suggest that traumatic experiences may shape pain perception and responses, which is characterised by heightened sensitivity of the nervous system and changes in central pain processing pathways (Moeller-Bertram et al., 2014).

Individuals with chronic pain versus those with acute pain were significantly more likely to engage in strategies such as pain-related worry, distraction, distancing, coping self-statements, and praying. This pattern highlights that chronic pain may elicit a broader or more complex repertoire of coping responses, potentially due to the enduring and pervasive nature of chronic

pain and its psychological burden over time. Interestingly, the use of "ignoring" as a coping strategy did not significantly differ between the two groups in relation to childhood trauma, suggesting that this particular response may be a more general or reflexive attempt to disengage from pain, irrespective of early adverse experiences. One possibility is that ignoring pain represents a relatively automatic or low-effort strategy that is accessible to individuals regardless of their trauma history. Prior studies have found that avoidance-based strategies can occur across pain types and may reflect a short-term attempt to maintain functioning (Eccleston & Crombez, 1999; McCracken & Eccleston, 2003). However, the long-term effectiveness of ignoring remains questionable, especially in chronic pain populations, where persistent avoidance can lead to greater disability and emotional distress. These findings build on existing research that links chronic pain with heightened emotional and cognitive engagement with pain-related cues (Baastrup et al., 2016; Vlaeyen & Linton, 2000). However, a notable finding emerged with respect to PTSD: ignoring was significantly more negatively associated with PTSD symptoms in the chronic pain group. This suggests that individuals with chronic pain who report higher levels of current post-traumatic stress may be less likely to engage in ignoring as a coping strategy. One possible explanation is that chronic pain co-occurring with PTSD leads to heightened threat sensitivity and increased hypervigilance, making cognitive disengagement more difficult to sustain. This aligns with evidence that trauma can impair attentional control and increase emotional reactivity (Blair et al., 2013; Clauss et al., 2021), potentially interfering with avoidant strategies like ignoring. Clinically, this finding may highlight the need to assess for trauma-related symptoms in chronic pain populations, as individuals with elevated PTSD may require alternative strategies to manage pain-related distress, beyond those based on cognitive avoidance. By demonstrating distinct patterns of coping between acute and chronic pain groups, this study adds new empirical evidence that extends cognitive-behavioural models of pain (Turk, 2003; Turk et al., 2008), particularly by highlighting the relevance of meaning-making and emotional regulation strategies in chronic pain adaptation. Furthermore, the observed negative correlations between distraction, coping self-statements, and pain-related outcomes in the chronic pain group underscore the potential therapeutic value of these strategies for reducing both physical and psychological distress. Additionally, the negative correlation between praying and pain intensity suggests that meaning-making strategies, such as religious or spiritual coping, may serve as a source of resilience, providing emotional support in the face of persistent pain (Pargament et al., 2001).

The type of childhood trauma experienced was correlated with the severity of both pain and current PTSD symptoms. Emotional and physical abuse were moderately associated with pain intensity, while emotional and physical neglect were linked to both pain severity and PTSD symptoms. PTSD symptoms, particularly re-experiencing and avoidance, were significantly

correlated with higher pain intensity and unpleasantness, highlighting the complex interplay between psychological distress and pain perception. Pain-related worry emerged as a key factor exacerbating both pain and PTSD symptoms, whereas coping strategies including distraction and distancing showed weaker associations. These findings support the fear-avoidance model (Cook et al., 2006; Vlaeyen & Linton, 2000), which suggests that worry and fear related to pain can contribute to avoidance behaviours and maintain both physical and emotional distress. They also align with the mutual maintenance model (Sharp & Harvey, 2001), which proposes that PTSD and chronic pain reinforce each other through shared processes such as attentional focus on threat, catastrophic interpretations, and avoidance. Similarly, the shared vulnerability model (Asmundson et al., 2002) explains how pre-existing sensitivities, such as heightened anxiety awareness, may increase the likelihood of developing both conditions following trauma. The conceptual overlap across these models is notable, particularly between the mutual maintenance and fear-avoidance frameworks, both of which emphasise how cognitive-emotional responses to pain and trauma can sustain and intensify symptoms. However, while these models offer valuable theoretical foundations, they may not fully capture the influence of developmental trauma, which can impact emotional regulation, self-perception, and trust in others (Schimmenti & Caretti, 2016; Van der Kolk, 2003; Villalta et al., 2018), factors central to the capacity to engage with support and apply coping strategies effectively. These findings are further supported by experimental and longitudinal studies demonstrating bidirectional influences between PTSD and pain (e.g., Bair et al., 2020; Benedict et al., 2020; Giannoni-Pastor et al., 2016; Ravn et al., 2018; Stratton et al., 2014; Sveen et al., 2011; Van Loey et al., 2003), highlighting the cyclical nature of the relationship between pain and trauma. Notably, in the chronic pain group, coping strategies such as distraction and coping self-statements were more strongly associated with lower pain and trauma symptoms, suggesting potential protective effects. However, individuals with more severe trauma histories were less likely to report using these strategies, suggesting that trauma-related barriers, such as shame, emotional numbing, or fear of vulnerability may make it more difficult to access or benefit from them (DeCou et al., 2019; Harman & Lee, 2010; Lee et al., 2001; Saraiya & Lopez-Castro, 2016). This is a novel and meaningful finding, as it offers early evidence that trauma influences the types of coping strategies people tend to use.

The association between trauma exposure and pain coping strategies varied depending on whether an individual experienced acute or chronic pain, suggesting that the interplay between these factors shifts based on pain chronicity. The chronic pain group exhibited stronger negative correlations between distraction and emotional neglect, as well as between coping self-statements and emotional neglect. Additionally, praying showed a stronger negative correlation with emotional abuse in individuals with chronic pain. PTSD symptoms also exhibited different

patterns, with stronger negative correlations between coping self-statements and both re-experiencing and hyperarousal in the chronic pain group. These findings indicate that individuals with chronic pain may develop distinct coping patterns in relation to their trauma history and PTSD symptoms, with certain coping mechanisms showing more pronounced associations with childhood adversity and trauma-related distress. This suggests that these coping strategies are not generally helpful for individuals with chronic pain in addition to symptoms of PTSD and histories of childhood trauma. Interestingly, pain-related worrying was the only pain coping strategy that did not show a significant difference between the chronic and acute pain groups. Clinically, this indicates that while individuals with chronic pain may attempt to use coping strategies to manage their pain, these approaches may not address the complex relationship between pain and trauma. One possible explanation is that reframing pain-related thoughts may be particularly challenging when pain is a constant and overwhelming presence in daily life.

The findings from this study suggest that approaches such as CBT (Morley, 2011), Acceptance and Commitment Therapy (ACT; McCracken et al., 2022), mindfulness-based interventions (Chiesa & Serretti, 2011), and trauma-focused therapies (De Roos et al., 2010; Lumley et al., 2022) may help individuals develop coping strategies that reduce distress without invalidating the ways they have previously managed pain. ACT, for instance, encourages acceptance of pain and focuses on helping individuals engage in actions aligned with their values, despite the presence of pain (Hughes et al., 2017; McCracken & Vowles, 2014; Vowles & McCracken, 2008). By shifting the focus from trying to control pain to living a meaningful life based on personal values, these therapies may offer a more effective alternative for individuals with chronic pain, as they provide a way forward when traditional coping strategies appear less helpful. Another promising approach is Compassion-Focused Therapy (CFT), which aims to reduce self-criticism and shame. These are potential barriers that may prevent trauma-exposed individuals from accessing helpful coping strategies and developing emotional safety and self-compassion (Au et al., 2017; Lee, 2022; Lee, 2010; Luoma & Platt, 2015). By supporting individuals in feeling more able to engage with other resourceful or empowering strategies such as distraction or coping self-statements, CFT may enhance the effectiveness of pain management interventions, particularly for those with complex trauma histories. Furthermore, examining how different types of childhood trauma, such as emotional versus physical neglect or abuse, affect pain perception and coping could refine trauma-informed treatment approaches, tailoring interventions to the specific nature of an individual's trauma history (Sveen et al., 2011). Moreover, integrating compassion-focused approaches into pain management interventions could be particularly beneficial (Hadley & Novitch, 2021; Marelli et al., 2025) and a compassion focused therapy group aimed as pain management intervention for individuals with persistent

pain has shown improvements for self-compassion, pain-related disability, pain-related anxiety and pain self-efficacy (Malpus et al., 2023).

A key strength of this study is the novelty of its findings. The present study appears to be the first direct comparison of coping strategies between individuals with acute and chronic pain, in relation to trauma history and PTSD symptoms. As such, this research brings new and valuable insights for understanding the experiences of people living with trauma and chronic pain that may inform more responsive and supportive clinical approaches. Additional strengths include the use of validated measures and the integration of multiple psychological constructs, allowing for a more holistic understanding of the interplay between coping, pain, and trauma. The inclusion of trauma history and PTSD symptomatology further enhances the depth of analysis, supporting an integrated, person-centred perspective on pain.

However, several limitations should be noted. As per IASP definition of pain, pain perception is inherently subjective (IASP, 1994), and individual interpretations of pain rating scales (e.g., 0–10) may be interpreted idiosyncratically by participants, impacting comparability across individuals (Bakshi, Rathod & Salunkhe, 2021). Moreover, recall bias may affect recalling pain-related memory (Schoth et al., 2020), as well as self-reported childhood adversity (Maughan & Rutter, 1997), particularly for distant (Ottenstein & Lischetzke, 2020; Thomas & Diener, 1990) or traumatic events (Krayem et al., 2021), while social desirability bias might result in underreporting due to stigma including the report of PTSD symptoms (Krzemieniecki, & Gabriel, 2021; Henderson et al., 2012). Additionally, the cross-sectional design limits the ability to draw conclusions about causality or the direction of effects. The present approach also did not test for potential interactions between coping and demographic or clinical variables. Using moderated regression in future studies could facilitate a better understanding of the interplay of these variables by exploring the extent to which this relationship varies across groups. Future research would benefit from using longitudinal designs to track changes in coping, pain, and psychological symptoms over time, particularly in relation to trauma history and the transition from acute to chronic pain (Bair et al., 2020; Giannoni-Pastor et al., 2016). Additionally, the sample was predominantly female and white. While this reflects broader trends, such as the higher likelihood of women receiving a chronic pain diagnosis (Fillingim et al., 2009), it also highlights an important imbalance. Cultural and gender-related factors are known to influence pain expression, access to care, and coping, and there is a need to better understand the experiences of individuals from underrepresented backgrounds (Samulowitz et al., 2018). Future studies should aim to recruit more diverse and inclusive samples, particularly given known barriers to diagnosis and treatment among racially minoritised and marginalised communities. Addressing these disparities is vital for ensuring that findings and interventions are both equitable and representative (Lee et al., 2001). Finally, while there was an initial effort during the early stages of this study to explore the

development of a more compassionate alternative to the term “pain catastrophising”, which many individuals with lived experience of trauma and chronic pain felt to be deficit-focused, this work could not be sustained due to practical constraints for this project. Nonetheless, these early conversations were meaningful and underscored the importance of language in shaping both research and clinical engagement. In future, we hope to build on this work by fostering more open and sustained dialogue with people with lived experience through Patient and Public Involvement (PPI). Creating space for their voices to be genuinely heard is essential for research that aims to reflect, respect, and support the communities it seeks to serve.

In conclusion, this study provides novel and valuable insights into the complex relationship between childhood trauma, PTSD, and pain coping strategies, with important clinical implications. The key finding that the associations between pain coping strategies, childhood trauma, and PTSD symptoms differ significantly between acute and chronic pain groups underscores the dynamic interplay between trauma history, pain perception, and coping mechanisms. Specifically, individuals with chronic pain demonstrated stronger negative correlations between certain coping strategies (e.g., distraction, coping self-statements) and trauma-related symptoms, suggesting that these strategies may not be as effective in managing pain and trauma in this population. This highlights the need for tailored interventions that account for both the chronicity of pain and the impact of trauma history on coping. Furthermore, the study’s novel focus on the role of trauma in shaping pain coping strategies provides crucial insights into how childhood adversity may complicate pain management, particularly in individuals with complex trauma histories. The findings suggest that traditional coping strategies may be less effective for those with chronic pain and PTSD, pointing to the importance of exploring alternative therapeutic approaches, such as CBT, ACT, and CFT, that aim to modify the emotional and cognitive responses to pain, rather than directly altering the pain experience itself. In sum, this study contributes to a deeper understanding of the challenges faced by individuals with both chronic pain and trauma histories, offering important directions for future research and intervention development aimed at improving pain management outcomes in this population.

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Appendix A - Chapter 2

Appendix 1A:

Table 2.1 - Overview of included studies, childhood trauma and neuroticism measures, study location and overall study outcome

Author (Year)	N	Childhood Trauma Measure	Neuroticism Measure	Study Location	Outcome: Correlation with Neuroticism
Acheson et al. (2018)	1031	C-DIS-IV	EPI; TDSI	USA	OT ↑
Adanty et al. (2022)	374	CTQ-SF	NEO-FFI	Canada	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Alnassar et al. (2024)	1116	CTQ	TSDI	United Kingdom	EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Aydin & Lacin (2022)	90	CTQ-SF	EPQR-AF	Turkey	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Baryshnikov et al. (2017)	282	TADS	S5	Finland	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Bourassa et al. (2022)	859	ACEs scale	MPQ	New Zealand	OT ↑
Boyette et al. (2014)	327	CTQ-SF	NEO-FFI	Netherlands	OT ↑
Bradley et al. (2011)	530	CTQ-SF	PANAS	USA	OT ↑
Brennan et al. (2024)	920	ACEs scale	MPQ-BF	New Zealand	OT ↑
Brents et al. (2015)	92	CTQ-SF	NEO-FFI	USA	OT ↑; EA ↑; EN ↑
Brents et al. (2018)	94	CTQ-SF	NEO-FFI	USA	OT ↑
Burt et al. (2015)	160	CTQ-SF	ATQ	USA	OT ↑
Cao et al. (2020)	159	CTQ	NEO-FFI	USA	OT ↑
Chen et al. (2021)	433	CTQ-SF	EPQ	USA	OT ↑
Choi & Park (2018)	557	CTQ (Korean)	MMPI-2 (Korean)	Republic of Korea	EA ↑; EN ↑; PA ↑; PN ↑; SA ↑

Chu et al. (2022)	3009	CTQ-SF	EPQ	China	OT ↑
Chu et al. (2024)	171	CTQ-SF	NEO-FFI	China	OT ↑; EA ↑; EN ↑; PA =; PN ↑; SA =
Chuong et al. (2022)	148129	MHQ	EPQ-R	United Kingdom	OT ↑
Cicero & Kerns (2010)	325	CTQ-SF	IPIP	United States	EA ↑; EN ↑; PA =; PN =
Cohrdes & Mauz (2020)	3,704	ACE-IQ	BFI	Germany	EA ↑; EN ↑; PA =; PN =; SA =
Comijs et al. (2013)	510	CIDI	NEO-FFI	Netherlands	EA ↑; EN ↑; PA ↑
Corcoran & McNulty (2018)	190	ACE	I-PANAS-SF	Ireland	OT ↑
Damatac et al. (2025)	300	CTQ-SF	NEO-FFI-3	Netherlands	OT ↑
Davies, Harty & Boden (2024)	911	CAS	EPI	New Zealand	OT ↑
De Venter et al. (2017)	539	CTI	NEO-FFI	Netherlands	OT ↑
Dong et al. (2020)	170	CTQ	NEO-PI-R	China	OT ↑
Dye et al. (2020)	748	CTQ-SF	NEO-FFI	USA	EA ↑; PA =; SA =
Ebanayake et al. (2017)	69	CTQ-SF	NEO-PIR	USA	EA ↑; PA =; SA ↑
Evren et al. (2012)	169	CTQ-SF	DSQ-40	Turkey	OT =; EA =; EN ↓; PA ↑; PN =; SA ↓
Fields et al. (2023)	177	ACEs scale	TIPI	USA	OT ↑
Fogelman et al. (2016)	170	CTQ-SF	NEO-FFI	USA	Sample 1: OT ↑ Sample 2: OT ↑
Fuge et al. (2014)	541	CTQ-SF	NEO-FFI	Germany	OT ↑
Fujimura et al. (2023)	404	CATS	EPQ-R	China	OT ↑
Gallardo-Pujol & Pereda (2013)	119	JVC	NEO-FFI	Spain	V ↑
Gamble et al. (2006)	549	CTQ-SF	NEO-PI-R	USA	V ↑
Gratz (2006)	249	API	AIM	USA	OT ↑; EN ↑; PA =

Grist & Caudle (2021)	207	ACEs	M5-50	USA	OT ↑
Grusnick et al. (2020)	6323	ACEs scale	MIDI Personality Scale	USA	OT ↑
Harmon-Jones & Richardson (2021)	134	CATS	TIPI	Australia	OT ↑
Hashimoto et al. (2022)	433	ASVC	EPQ-R	Japan	V ↑
Hatwan et al. (2024)	475	CTQ-SF	TIPI	USA	OT ↑; EA ↑; EN ↑; PA =; PN ↑; SA ↑
Hayashi et al. (2015)	113	CATS	NEO-FFI	Japan	OT ↑; UnspN ↑; PA =; SA ↑
He et al. (2025)	84	CTQ-SF	BFI (Chinese)	China	OT ↑
Heckman & Clay (2005)	201	ACEs scale	Psychological Distress Scale of the Mental Health Index	USA	OT ↑
Hengartner et al. (2015)	1173	CTQ	BFI-S	Switzerland	EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Hovens et al. (2015)	2981	NEMESIS (CTI)	NEO-FFI	Netherlands	OT ↑; EA ↑; EN ↑; PA ↑; SA ↑
Husain et al. (2021)	455	CTQ-SF	NEO PI-R	Pakistan	OT =
Jain et al. (2024)	50	ACES scale	TIPI	USA	OT ↑
Jardim et al. (2019)	260	CTQ-SF	NEO-FFI	Brazil	OT ↑
Jimenez et al. (2019)	272	CTQ-SF	BFI-S	Colombia	OT ↑; EA ↑; EN ↑; PA =; PN ↑; SA ↑
Jirakran et al. (2023)	133	ACEs scale (Thai)	BFI (Thai)	Thailand	EN ↑; PA ↑; SA ↑
Jung (2021)	3,034	CATS	I-PANAS-SF	USA	EA ↑
Kamali et al. (2019)	270	CTQ-SF	NEO-PI-R	USA	OT ↑
Kang et al. (2021)	444	CTQ-SF	EPQ	China	EA ↑; EN ↑; PA ↑; SA =

Karmakar et al. (2017)	13493	ACEs scale	IPIP	USA	OT ↑
Knight et al. (2023)	105	ETISR-SF	BFI	USA	OT ↑
Koschiget et al. (2023)	3176	CTS	BFI-S	Germany	OT ↑
Kounou et al. (2015)	150	CTQ-SF	IPIP	France and Togo	Sample 1: OT ↑ Sample 2: OT ↑
Lam et al. (1997)	264	CTQ-SF	NEO-FFI	USA	OT ↑
Lawrence (2022)	398	ACEs scale	IPIP	USA	OT ↑
Lee et al. (2024)	111,931	CTQ-SF	EPQ-N	United Kingdom	OT ↑
Lee et al. (2017)	1396	ETISR-SF (Korean)	TIPI (Korean)	Republic of Korea	EA ↑; PA ↑; SA ↑
J. Liu et al. (2023)	314	CTQ-SF	EPI	China	EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Liu et al. (2020)	1169	CPMS	NEO-FFI	China	OT ↑
F. Liu et al. (2023)	717	CPMS	NEO-FFI	China	OT ↑
Lopez-Mongay et al. (2021)	50	CTQ-SF	NEO-FFI	Spain	SA ↑
Lui et al. (2025)	773	CTQ-SF (EA/EN)	FFNI-SF	China	OT ↑
Lund et al. (2017)	155	CEVQ	EPQ-R	Canada	SA ↑
Luo et al. (2020)	20,000	HRS	MIDUS Big Five Adjectival scale	USA	OT ↑
Lysaker et al. (2001)	44	CSTQ	NEO-FFI	USA	SA ↑
Marchi et al. (2022)	1262	CTQ-SF	NEO-FFI	Netherlands	OT ↑
Martín-Blanco et al. (2014)	130	CTQ-SF	ZKPQ	Spain	EA ↑; EN =; PA =; PN =; SA =
Masuya et al. (2022)	576	CATS	EPQ-R	Japan	OT ↑
Masuya et al. (2024)	584	CATS; ASVC	EPQ-N	Japan	OT ↑; SA ↑
Mc Elroy & Hevey (2014)	176	CTQ-SF	NEO-FFI	Ireland	OT ↑

Michal et al. (2025)	1255	CTQ-SF; ACEs scale	The Composite International Diagnostic	USA	OT ↑
Moreira et al. (2024)	609	ACEs scale (Portugese)	TIPI	Portugal	EA ↑; EN ↑
Mosley-Johnson et al. (2021)	3234	ACEs scale (Portugese)	NEO-FFI	Portugal	OT =
Ng & Hartanto (2022)	1553	ACEs scale	DISE	US	EA ↑
Nguyen-Feng et al. (2017)	260	CTS	BFI	United States	EA ↑
Ogle et al. (2015)	1,186	CTQ-SF	NEO-FFI	United States	OT =
Ono et al. (2017)	413	TLEQ	NEO-PI	United States	OT ↑
Ottesen et al. (2018)	209	CATS	EPQ-R	Japan	OT ↑
Otto et al. (2021)	95	CTQ-SF	EPQ	Denmark	OT ↑
Ozen et al. (2018)	130	CTQ-SF	NEO-FFI	Turkey	OT ↑; EN ↑; PN ↑; SA ↑
Peng et al. (2025)	76	CTQ-SF	EPQ	Turkey	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Peters et al. (1994)	136	CTQ-SF	16PF	China	OT ↑; PA ↑; SA ↑
Pflanz et al. (2024)	63,360	Family and Sexual History Questionnaire	EPQ	United States	OT ↑
Pickering et al. (2004)	90	CTS-5	EPQ	United Kingdom	EA ↑
Ponder et al. (2023)	768	CTQ-SF	EPQ	United Kingdom	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Pos et al. (2016)	163	ACEs scale; CTQ-SF	EPQ-N	USA	UnspN ↑; UnspA ↑
Powers et al. (2014)	814	CTQ-SF	NEO-FFI (Dutch)	Netherlands	OT ↑
Qin et al. (2024)	1272	CTQ-SF	PANAS	United States	OT ↑
Rademaker et al. (2010)	522	ETISR-SF	DS14	Netherlands	OT =; EA ↑; PA ; SA =
Ramos et al. (2024)	380	CTQ-SF	NEO-FFI	International	OT ↑

Rose et al. (2023)	822	CTES; ACEs scale	NEO-PI-R	USA	OT ↑
Roy et al. (2002)	532	CTQ	EPQ	USA	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Sanwald et al. (2023)	238	CTQ-SF	NEO-FFI	Germany	Sample 1: OT ↑; EA ↑; EN =; PA =; PN =; SA =
					Sample 2: OT ↑; EA ↑; EN =; PA =; PN =; SA =
Schwandt et al. (2013)	417	CTQ-SF	NEO PI-R	USA	EA ↑; EN ↑; PA ↑; PN ↑; SA ↑
Shen et al. (2021)	433	CTQ-SF	EPI	China	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑; SA =
Shi et al. (2021)	1266	CTQ-SF	BFI	China	OT ↑
Spinhoven et al. (2010)	2786	MIDI	NEO-FFI	Netherlands	EA ↑; EN ↑; PA ↑
Stevanovic et al. (2016)	394	ETIS-R-SF	NEO PI-R	Croatia, Serbia, Italy, and the Netherlands	OT ↑
Stokes et al. (2013)	36	CAQ	M5-50	USA	V ↑
Sturmbauer et al. (2019)	298	ACEs scale; CTQ-SF	TIPI	Germany	OT =
Tachi et al. (2019)	432	ASVC	EPQ-R	Japan	V ↑
Trent et al. (2023)	855	PTI	STAI-T	USA	OT ↑
Trombello et al. (2018)	188	CTQ-SF	HADS	USA	EA ↑; EN ↑; PA =; PN ↑; SA ↑
Tyra et al. (2021)	119	ACEs scale	BFI-S	USA	OT ↑
van Harmelen et al. (2014)	194	NEMESIS (CTI)	NEO-FFI	Netherlands	OT ↑
Vasupanrajit et al. (2024)	118	ACEs scale	IPIP	Thailand	OT ↑; SA ↑

Veith et al. (2017)	526	VEQ-R	PID-5	USA	Sample 1: UnspA ↑; Sample 1: UnspA =;
Verrastro et al. (2024)	1176	CEA; CTQ-SF	BFI-N	Italy	EA ↑
Walker et al. (1997)	36	CTQ-SF	NEO-FFI	USA	OT ↑
D. Wang et al. (2018)	555	CTQ-SF	EPQ	China	OT ↑
Q. Wang et al. (2018)	1253	CTQ-SF	EPQ	China	EA ↑
Wang et al. (2010)	289	ETIS-R-SF	NEO-FFI	USA	OT ↑
Wang et al. (2020)	404	CTQ-SF	NEO-FFI	China	OT ↑
Wang et al. (2022)	120	CTQ-SF	NEO-FFI	Netherlands	OT ↑
Weltz et al. (2016)	1634	TESI-Y/SR	NEO-PI	USA	EA ↑; EN ↑; PA ↑; SA ↑
Wrobel et al. (2023)	209	CTQ-SF	NEO PI-R	Australia	OT ↑; EA ↑; EN ↑; PA ↑; PN =; SA =
Xu et al. (2017)	523	ACEs scale	EPQ (Chinese)	China	OT ↑
You et al. (2022)	1222	CTQ-SF	BFI	Korea	OT ↑; EA ↑; EN ↑;
Yrondi et al. (2021)	96	CTQ-SF	BFI	France	OT ↑
Zhao et al. (2022)	179	CTQ-SF	NEO-FFI	China	EA ↑; EN ↑; PA ↑; PN ↑; SA =
Zhou et al. (2019)	312	CTQ-SF	EPQR-S	China	EA ↑
Zhou et al. (2022)	565	CTQ-SF	EPQ	China	OT ↑; EA ↑; EN ↑; PA ↑; PN ↑

Note:

Childhood trauma measures – CTQ-SF – Childhood Trauma Questionnaire – Short Form; CTQ – Childhood Trauma Questionnaire (full version); ACEs – Adverse Childhood Experiences scale; CATS – Child Abuse and Trauma Scale; ETISR-SF – Early Trauma Inventory Self Report – Short Form; CTS – Childhood Trauma Screener; CPMS – Childhood Psychological Maltreatment Scale; NEMESIS – Netherlands Mental Health Survey and Incidence Study – Childhood Trauma Interview; CSTQ – Childhood Sexual Trauma Questionnaire; API – Abuse-Perpetration Inventory; CEVQ – Childhood Experiences of Violence Questionnaire; CHDS – Christchurch Health and Development Study – Childhood Adversity Score; PTI – Childhood Threat Inventory; ELS – Early Life Stress scale; DISE – Daily Inventory of Stressful Events; TESI-Y/SR – Traumatic Events Screening Inventory – Youth/Self Report; VEQ-R – Violent Experiences Questionnaire – Revised; LONGSCAN – Longitudinal Studies of Child Abuse and Neglect; HRS – Health and Retirement Study.

Neuroticism measures – NEO-FFI – NEO Five-Factor Inventory; NEO-PI-R – Revised NEO Personality Inventory; EPQ – Eysenck Personality Questionnaire; EPQ-R – Eysenck Personality Questionnaire – Revised; EPQR-S – Eysenck Personality Questionnaire – Revised Short Form; EPQR-N – Eysenck Personality Questionnaire – Neuroticism subscale; EPQ-RSC – Eysenck Personality Questionnaire – Revised Short Form for Children; EPQR-AF – Eysenck Personality Questionnaire – Afrikaans version; BFI – Big Five Inventory; BFI-S – Big Five Inventory – Short Form; BFI-N – Big Five Inventory – Neuroticism subscale; SOEP-BFI – Socio-Economic

Panel Big Five Inventory; TIPI – Ten-Item Personality Inventory; TIPI-G – Ten-Item Personality Inventory – German version; IPIP – International Personality Item Pool; IPIP-NEO – International Personality Item Pool – NEO version; mini-IPIP – Mini International Personality Item Pool; IPIP-50 – International Personality Item Pool – 50-item version; MPQ – Multidimensional Personality Questionnaire; MPQ-BF – Multidimensional Personality Questionnaire – Brief Form; PANAS – Positive and Negative Affect Schedule; I-PANAS-SF – International Positive and Negative Affect Schedule – Short Form; TSDI – Trait Self-Description Inventory; S5 – Short Five; DSQ-40 – Defense Style Questionnaire – 40 items; AIM – Affective Intensity Measure; ATQ – Automatic Thoughts Questionnaire; MIDI – Midlife Development Inventory Personality Scale; 16PF – Sixteen Personality Factor Questionnaire – Emotional Stability subscale; Korean MMPI-2 – Korean Minnesota Multiphasic Personality Inventory – Neuroticism subset; HADS – Hospital Anxiety and Depression Scale – Neuroticism/Worry subscale; PID-5 – Personality Inventory for DSM-5; STAI-T – State–Trait Anxiety Inventory – Trait subscale; FFNI-SF – Five-Factor Narcissism Inventory – Short Form.

Outcome abbreviations – OT – overall trauma; EA – emotional abuse; EN – emotional neglect; PA – physical abuse; PN – physical neglect; SA – sexual abuse.

Appendix 2A

Table 2.2 - Trauma measures breakdown by version and frequency used in meta-analysis

Instrument	Frequency (k)	Notes
Instrument	**Frequency (k)**	**Notes**
Childhood Trauma Questionnaire – Short Form (CTQ-SF)	63	Includes Korean, Thai, Chinese versions; emotional abuse/neglect subscales used in some
Adverse Childhood Experiences scale (ACEs)	16	Includes Thai, Portuguese versions; includes adaptations and studies combining with CTQ-SF
Childhood Trauma Questionnaire – Full version (CTQ)	10	Includes Korean version
Child Abuse and Trauma Scale (CATS)	5	Used in combination with Childhood Victimization Rating Scale in some studies
Early Trauma Inventory Self Report – Short Form (ETISR-SF)	4	Includes Korean version
Childhood Trauma Screener (CTS)	2	–
Childhood Psychological Maltreatment Scale (CPMS)	2	–
NEMESIS Childhood Trauma Interview (CTI)	2	Includes Dutch version
Structured interviews adapted from national mental health surveys (e.g., MHQ)	1	–
Juvenile Victimization Questionnaire – Adults Retrospective Version	1	–
Traumatic Life Events Questionnaire (TLEQ)	1	–
Assessment Scale of Victimization in Childhood	1	–
Diagnostic Interview Schedule for DSM-IV (C-DIS-IV) – Early life adversity scale	1	–
Childhood Experiences of Violence Questionnaire (CEVQ)	1	–
Conflict Tactics Scale (CTS)	1	CTS-5 used in some studies
Childhood Sexual Trauma Questionnaire (CSTQ)	1	–
Daily Inventory of Stressful Events (DISE)	1	Included additional questions related to trauma and mood
Family and Sexual History Questionnaire	1	Focused on childhood family/sexual experiences
Childhood sexual abuse questionnaire – Finkelhor adaptation	1	Based on Finkelhor's Sexual Experience Questionnaire

Appendix 3A

Table 2.3 - Neuroticism measures breakdown by version and frequency used in meta-analysis

Instrument	Frequency (k)	Notes
NEO Five-Factor Inventory (NEO-FFI / NEO-FFI-3)	49	Includes Dutch version
Eysenck Personality Questionnaire (EPQ, EPQ-R, EPQ-N, EPQR-AF, EPQR-S)	19	Includes multiple forms (revised, neuroticism-focused, short forms)
Big Five Inventory (BFI, BFI-S)	11	Includes Chinese and Thai versions; BFI-S is short form
NEO Personality Inventory – Revised (NEO PI-R)	9	–
International Personality Item Pool (IPIP)	7	
Ten-Item Personality Inventory (TIPI)	5	Includes Korean version
PANAS / I-PANAS-SF (Positive and Negative Affect Schedule)	4	PANAS and its international short form (I-PANAS-SF)
Emotionality Personality Inventory (EPI)	3	–
Multidimensional Personality Questionnaire (MPQ / MPQ-BF)	3	–
Temperament and Character Inventory – Short Forms (TSDI, S5)	2	–
Minnesota Multiphasic Personality Inventory – 2 (MMPI-2)	1	Korean version used
Affective Intensity Measure (AIM)	1	–
Descriptive Personality Scales (DSQ-40)	1	–
FFNI-SF (Five-Factor Narcissism Inventory – Short Form)	1	Focused on maladaptive neurotic traits
Personality Inventory for DSM-5 (PID-5)	1	–
Six-item negative affect scale (mental health indices)	1	Includes “hopeless,” “nervous,” “worthless,” etc.
HADS (Hospital Anxiety and Depression Scale – neuroticism/worry subscale)	1	–
16 Personality Factor Questionnaire (16PF)	1	–
Psychological Distress Scale (Mental Health Index)	1	–
STAI-T (Trait scale of State–Trait Anxiety Inventory)	1	Measures trait anxiety linked to neuroticism
Personality Assessment Inventory – Borderline Features Scale	1	Neuroticism-related traits assessed through borderline features scale

Appendix 4A

Table 2.4 – Risk of bias assessment – quality assessment too

Author	Year	Section A Selection bias rating	Target population	% agreed to participate	Section B Study design Strong, moderate, weak	Study design	Appropriate Method	Section E Data collection methods	Valid data collection tools	Reliable data collection tools	Section F Withdrawal and Drop-outs	Withdrawal / Dropout reported	% percentage of comoletion	Section H Analyses	Unit of Analysis	Appropriate statistical method	Global rating*
Acheson et al.	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	77%	Strong	Individual	Yes	Strong
Adanty et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Alnassar et al	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Aydin & Lacin	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Baryshnikov	2017	Strong	Very likely	58.8%%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	31.40%	Strong	Individual	Yes	Strong
Bourassa et al.	2022	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	94.10%	Strong	Individual	Yes	Strong
Boyette et al.	2014	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Bradely et al.	2011	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Brennan et al.	2024	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	88.70%	Strong	Individual	Yes	Strong
Brents et al.	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	84%	Strong	Individual	Yes	Strong
Brents et al.	2015	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Burt et al.	2015	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Cao et al.	2020	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Chen et al.	2021	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Choi & Park	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Chu et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Chu et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Chuong et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Cicero & Kerns	2010	Strong	Very likely	96.9%%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	85.30%	Strong	Individual	Yes	Strong

Cohrdes & Mauz	2020	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	62%	Strong	Individual	Yes	Strong
Comijs et al.	2013	Moderate	Very likely	Can't tell	Strong	Cross-sectional / follow up	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Corcoran & McNulty	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
D. Wang et al.	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Damatac et al	2025	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Davies, Harty & Boden	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional / follow up	Yes	Strong	Yes	Yes	Strong	Yes	72%	Strong	Individual	Yes	Strong
De Venter et al.	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional / follow up	Yes	Strong	Yes	Yes	Strong	Yes	80.70%	Strong	Individual	Yes	Strong
Dong et al.	2020	Moderate	Very likely	Can't tell	Strong	Cross-sectional / follow up	Yes	Strong	Yes	Yes	Strong	Yes	n/a	Strong	Individual	Yes	Strong
Dye et al.	2020	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Ebanayake et al.	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Evren et al.	2012	Moderate	Very likely	Can't tell	Strong	Cross-sectional / follow up	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
F. Liu et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	95.60%	Strong	Individual	Yes	Strong
Fields et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Fogelman et al.	2016	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	87.20%	Strong	Individual	Yes	Strong
Fuge et al,	2014	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	38.60%	Strong	Individual	Yes	Strong
Fujimura et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Gallardo-Pujol & Pereda	2013	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Gamble et al.	2006	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	60.30%	Strong	Individual	Yes	Strong
Gratz	2006	Strong	Very likely	78%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	78%	Strong	Individual	Yes	Strong
Grist & Caudle	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	Yes	n/a	Strong	Individual	Yes	Moderate
Grusnick et al	2022	Strong	Very likely	99.90%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	99.90%	Strong	Individual	Yes	Strong

Harmon-Jones & Richardson	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	46.20%	Strong	Individual	Yes	Strong
Hashimoto et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Hatwan et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	63.50%	Strong	Individual	Yes	Strong
Hayashi et al	2015	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
He et al.	2025	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	93.30%	Strong	Individual	Yes	Strong
Heckman & Clay	2005	Strong	Very likely	84%%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	64%	Strong	Individual	Yes	Strong
Hengartner et al.	2015	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Hovens et al.	2015	Strong	Very likely	75.9%%	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	75.9%%	Strong	Individual	Yes	Strong
Husain et al.	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
J. Liu et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Jain et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Jardim et al.	2019	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Jimenez et al.	2019	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Jirakran et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Jung	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	86.80%	Strong	Individual	Yes	Strong
Kamali et al.	2019	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	77.50%	Strong	Individual	Yes	Strong
Kang et al.	2021	Strong	Very likely	100%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	100%	Strong	Individual	Yes	Strong
Karmakar et al.	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Knight et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	No	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Koschig et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	97%	Strong	Individual	Yes	Strong
Kounou et al.	2015	Strong	Very likely	81.50%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	81.50%	Strong	Individual	Yes	Strong
Lam et al.,	1997	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Lawrence	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Lee et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Lee et al.,	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Liu et al.	2020	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	97.40%	Strong	Individual	Yes	Strong
Lopez-Mongay et al.	2021	Strong	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Lui et al	2025	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Lund et al.	2017	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Luo et al.	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Lysaker et al.	2001	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Marchi et al.	2022	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Martín-Blanco et al.	2014	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Masuya et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Masuya et al.	2022	Strong	Very likely	48.30%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	48.30%	Strong	Individual	Yes	Strong
Mc Elroy & Hevey	2014	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Michal et al	2025	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Moreira et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Mosley-Johnson et al.	2021	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Ng &Hartanto	2022	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Nguyen-Feng	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Ogle et al.,	2015	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	76.42%	Strong	Individual	Yes	Strong
Ono et al.	2017	Strong	Very likely	48.40%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	48.40%	Strong	Individual	Yes	Strong
Ottesen et al.	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Otto, Kokkelink & Brüne	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Ozen et al.	2018	Strong	Very likely	73.00%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	73.00%	Strong	Individual	Yes	Strong
Peng et al.	2025	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Peters et al.	1994	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Pflanz et al	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Pickering et al.	2004	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Ponder et al.	2024	Strong	Very likely	100.00%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	86.70%	Strong	Individual	Yes	Strong
Pos et al.	2016	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Powers et al.	2014	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Q. Wang et al.	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	98.90%	Strong	Individual	Yes	Strong
Qin et al	2024	Strong	Very likely	100.00%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	97.8%%	Strong	Individual	Yes	Strong
Rademaker et al.	2010	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Ramos et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Rose et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	No	n/a	Strong	Individual	Yes	Moderate
Roy et al.	2002	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Sanwald et al.	2023	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Schwandt et al.	2013	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Shen et al.	2021	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	85.70%	Strong	Individual	Yes	Strong
Shi et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Spinhoven et al	2010	Strong	Very likely	100.00%	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	76.80%	Strong	Individual	Yes	Strong
Stevanovic et al	2016	Strong	Very likely	82.40%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	82.40%	Strong	Individual	Yes	Strong
Stokes et al.	2013	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	n/a	Strong	Individual	Yes	Strong
Sturmbauer et al	2019	Strong	Very likely	100%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	100%	Strong	Individual	Yes	Strong
Tachi et al.	2019	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Trent et al.	2023	Strong	Very likely	98.80%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	98.80%	Strong	Individual	Yes	Strong
Trombello et al.	2018	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Tyra et al.	2021	Strong	Very likely	27.10%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	26.03%	Strong	Individual	Yes	Strong
van Harmelen et al.	2014	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	7.50%	Strong	Individual	Yes	Strong

Vasupanrajit et al.	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Veith et al.	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Verrastro et al	2024	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Walker et al.	1997	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Wang et al.	2022	Strong	Very likely	100.00%	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	96.70%	Strong	Individual	Yes	Strong
Wang et al.	2020	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Wang et al.	2010	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	41.50%	Strong	Individual	Yes	Strong
Weltz et al.	2016	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Strong	Yes	89.90%	Strong	Individual	Yes	Strong
Wrobel et al.	2023	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Xu et al.	2017	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
You et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Yrondi et al.	2021	Moderate	Very likely	Can't tell	Strong	Longitudinal	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate

Zhao et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Zhou et al	2019	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Weak	No	n/a	Strong	Individual	Yes	Moderate
Zhou et al.	2022	Moderate	Very likely	Can't tell	Strong	Cross-sectional	Yes	Strong	Yes	Yes	Strong	Yes	n/a	Strong	Individual	Yes	Strong

***Strong - no Weak ratings; Moderate - one Weak rating; Weak - two or more Weak rating**

Appendix 5A:

Table 2.5

Demographic information by study

Author (Year)	Age (Mean in years)	Ethnicity	Gender	Diagnostic Information	Study location
Acheson et al. (2018)	23.7 years; SD = 3.3 years	White (n = 806, 78.2%); Black (n = 104, 10.1%); Native American (n = 34, 3.3%); Biracial (n = 32, 3.1%); Asian (n = 10, 1.0%); Hawaiian/Pacific Islander (n = 4, 0.4%); Other (n = 41, 4.0%)	Females (n = 617, 59.8%); Males (n = 414, 40.2%)	Non-clinical group	USA
Adanty et al. (2022)	40.54 years; SD = 13.27 years	White (n = 192, 51.3%); Black (n = 61, 16.3%); South Asian (n = 32, 8.6%); East Asian (n = 21, 5.6%); Mixed Ethnicity (n = 20, 5.3%); Middle Eastern (n = 17, 4.5%); Latino (n = 14, 3.7%); Southeast Asian (n = 12,	Males (n = 247, 66.0%); Females (n = 127, 34.0%)	Non-clinical group	Canada

		3.2%); Aboriginal (n = 5, 1.3%)			
Alnassar et al. (2024)	18–24 (387 participants, 34.7 % of sample); 25–34 (297 participants, 26.6 % of sample); 35–44 (144 participants, 12.9 % of sample); 45–54 (112 participants, 10 % of sample); 55–64 (125 participants, 11.2 % of sample) 65 and over (51 participants, 4.6 % of sample)- mean and SD Not reported	Not reported	Males (n = 232, 20.8%); [Females not specified]	Non-clinical group	United Kingdom
Aydin & Lacin (2022)	Bipolar disorder- 1 (Med = 35; IQR = 19.3); Controls (Med = 35.5; IQR = 24.3)	Not reported	Bipolar disorder-1: Females (n = 46, 51.1%); Males (n = 44, 48.9%); Controls: Females (n = 49, 54.4%); Males (n = 41, 45.6%)	Bipolar disorder- 1 at least 8 weeks of remission (n = 90); Controls (n = 90)	Turkey
Baryshnikov et al. (2017)	42.2 years; SD = 13.1 years	Not reported	Females (n = 209, 74.1%); Males (n = 73, 25.9%)	Unipolar Depression (n = 183; comorbid Borderline Personality D disorder n = 39);	Finland

				Bipolar Disorder (n = 99; (type 2 n = 55; type 1 n = 36; not otherwise specified n = 8; comorbid Borderline Personality D disorder n =17))	
Bourassa et al. (2022)	longitudinal birth- 45	White (93%)	Females (49.6%); [Males not specified]	Non-clinical group	New Zealand
Boyette et al. (2014)	Patients with traumatic events (31.2 years; SD = 7.9 years); Patients without traumatic events (29.4 years; SD = 6.3 years); Controls (29.9 years; SD = 9.2 years)	Patients with traumatic events (Caucasian (81.3%)); Patients without traumatic events (Caucasian (81.9%)); Controls (Caucasian (84.1%))	Patients with traumatic events: Males (79.5%); Patients without traumatic events: Males (83.1%); Controls: Males (55.3%)	Psychotic disorders (n = 192); Healthy controls (n= 132)	Netherlands
Bradley et al. (2011)	42.3 years; SD = 12.6 years	African American (88%); White (5%); Mixed or other (3%); Latino (1%)	Females (62%); [Males not specified]	Non-clinical group	USA
Brennan et al. (2024)	longitudinal 38 years and 45 years	White (93%)	Males (51.6%); [Females not specified]	Non-clinical group	New Zealand

Brents et al. (2015)	37.8 years; SD = 8.8 years	White (non-Hispanic origin) (n = 71, 77.2%); Black (n = 22, 23.9%); Asian/Pacific Islander (n = 1, 1.1%)	Males (n = 57, 62.0%); Females (n = 37, 40.2%)	Non-clinical group	USA
Brents et al. (2018)	38 years; SD = 8.8 years	Not reported	Females (n = 37, 39.4%); [Males not specified]	Non-clinical group	USA
Burt et al. (2015)	19.74 years; SD = 1.69 years	Not reported	Females (n = 83, 51.9%); Males (n = 77, 48.1%)	Non-clinical group	USA
Cao et al. (2020)	26.23 years; SD = 5.66 years	European American (59.1%); African American (40.9%)	Females (100%)	Non-clinical group	USA
Chen et al. (2021)	18.92 years; SD = 1.41 years	Not reported	Males (n = 389, 89.8%); Females (n = 44, 10.2%)	Non-clinical group	China
Choi & Park (2018)	37.69 years; SD = 13.92 years	Not reported	Females (n = 294, 52.8%); [Males not specified]	Non-clinical group	Republic of Korea
Chu et al. (2022)	18.00 years; SD = 0.772 years	Not reported	Females (n = 1995, 66.3%); Males (n = 1014, 33.7%)	Non-clinical group	China
Chu et al. (2024)	39.91 years; SD = 12.33 years	Not reported	Females (n = 92, 53.8%); Males (n = 79, 46.2%)	Non-clinical group	China

Chuong et al. (2022)	56.00 years; SD = 7 years	White (100%)	Females (n = 76995, 52.0%); [Males not specified]	Non-clinical group	England
Cicero & Kerns (2010)	18.69 years; SD = 51.3 years	White (85.4%); African American (7.3%); Asian American (2.1%); Mixed ethnicity (4.0%)	Females (51.1%); [Males not specified]	Non-clinical group	USA
Cohrdes & Mauz (2020)	25.0 years	Not reported	Males (44.9%); [Females not specified]	Non-clinical group	Germany
Comijs et al. (2013)	60-93 years	Not reported	Not reported	378 depressed (major depression, dysthymia, or minor depression), 132 non-depressed	Netherlands
Corcoran & McNulty (2018)	22.02 years; SD = 4.24 years	Not reported	Females (n = 145, 76.3%); Males (n = 45, 23.7%)	Non-clinical group	Ireland
Damatac et al. (2025)	33.89 years; SD = 2.84 years	Not reported	Females (n = 139, 46.3%); [Males not specified]	Non-clinical group	Netherlands
Davies, Harty & Boden (2024)	Longitudinal: birth-40 years	New Zealand European/'other' (85.9%); Māori (11.2%); Pacific (2.9%)	Males (n = 635, 69.7%); Females (n = 630, 69.2%)*	Non-clinical group	New Zealand

De Venter et al. (2017)	41.2 years; SD = 11.9 years	Not reported	Females (69.8%); [Males not specified]	Panic disorder with agoraphobia (62.9%), Major depression (57.3%), social phobia (44.9%), Panic disorder without agoraphobia (37.1%), Generalised anxiety disorder (28.2%), dysthymia (17.1%)	Netherlands
Dong et al. (2020)	21.34 years; SD = 1.64 years	Not reported	Females (n = 95, 55.9%); Males (n = 75, 44.1%)	Non-clinical group	China
Dye et al. (2020)	Not reported; 99% undergraduates and 1% graduate	White/Caucasian (n = 586, 78.3%); Black/African-American (n = 58, 7.8%); Hispanic/Latino (n = 23, 3.1%); Other (n = 65, 8.7%)	Females (n = 466, 62.3%); Males (n = 271, 36.2%)	Healthy controls; some with high impulsivity	USA

Ebanayake et al. (2017)	PMD=46.7 years (SD=12.7); PNES=40.5years (SD=11.6); Healthy controls=45.9 years (SD=13.5)	Not reported	PMD: 73% females; PNES: 86% females; Healthy Controls: 65% females	PMD=59; PNES=43; Healthy controls=26	USA
Evren et al. (2012)	Heroin dependents=29.5 or 29.5 years (SD=8.7); Healthy controls=35.3 years (11.8)	Not reported	Males (n = 169, 100%)	Heroin dependence=109; Healthy controls=60	Turkey
Fields et al. (2023)	25.2 years; SD = 5.5 years	White (39.5%); Black (13.6%); Hispanic (13.6%); Native American (17.5%)	Females (n = 177, 100%)	Non-clinical group	USA
Fogelman et al. (2016)	Initial cohort = 63.46 years (7.15); Second cohort=24.07years (7.74)	Initial cohort: Caucasian (91.5%); Asian (3.7%); Hispanic (3.7%); African American (1.2%); Second cohort: All Caucasian except 1.1% Asian; 3.4% multiple races	Initial cohort: Females (n = 46, 27.1%); Males (n = 36, 21.2%); Second cohort: Males only (n = 88, 51.8%)	Non-clinical group	USA
Fuge et al. (2014)	No ELS=36.0years (SD=17); Low ELS=41.6years (SD=19.4); Moderate ELS=41.3 years(SD=19.1); Severe ELS=43.5 years(SD=17.2)	Not reported	Males (n = 230, 42.5%); Females (n = 221, 40.9%)	Non-clinical group	Germany

Fujimura et al. (2023)	42.3 years; SD = 11.9 years	Not reported	Males (n = 220, 54.5%); Females (n = 184, 45.5%)	Physical disease=81; first-degree relative with psychiatric disease=40	China
Gallardo-Pujol & Pereda (2013)	23.31 years; SD = 7.48 years	Caucasian (100%)	Males (20%); [Females not specified]	Non-clinical group	Spain
Gamble et al. (2006)	50+ years	Not reported	Females (n = 61, 11.1%); [Males not specified]	Major Depressive Disorder (n = 105)	United Sates
Gratz (2006)	23.30 years; SD = 5.96 years	White (66%); Asian (16%); Black/African American (8%); Hispanic (5%); Other (5%)	Females (n = 249, 100%)	Non-clinical group	USA
Grist & Caudle (2021)	35.58 years; SD = 9.88 years	White (n = 157, 75.8%); Black (n = 36, 17.4%); Hispanic (n = 7, 3.4%); Native American (n = 4, 1.9%); Other (n = 3, 1.4%)	Females (n = 205, 99.0%); Males (n = 2, 1.0%)	Non-clinical group	USA
Grusnick et al. (2020)	Med= 46 years (36-57)	White (n = 5651, 89.4%); Black (n = 336, 5.3%); Other (n = 266, 4.2%); Missing data (n = 70, 1.1%)	Females (n = 3320, 52.5%); Males (n = 3003, 47.5%)	Non-clinical group	USA

Harmon-Jones & Richardson (2021)	19.29 years; SD = 1.92 years	Asian (47.0%); White/European (33.6%); Other (9.7%); Indian (6.7%); Middle Eastern (2.2%); Aboriginal/Torres Strait Islander (0.7%)	Females (n = 79, 59.0%); Males (n = 55, 41.0%)	Non-clinical group	Australia
Hashimoto et al. (2022)	40.9 years; SD = 11.8 years	Not reported	Females (n = 248, 57.3%); Males (n = 195, 45.0%)	Non-clinical group	Japan
Hatwan et al. (2024)	22.26 years; SD = 2.09 years	Not reported	Males (n = 256, 53.9%); Females (n = 254, 53.5%)	Non-clinical group	USA
Hayashi et al. (2015)	41.91 years; SD = 11.20 years	Not reported	Females (n = 58, 51.3%); Males (n = 55, 48.7%)	Major Depressive Disorder (113)	Japan
He et al. (2025)	33 years; SD = 9 years	Not reported	Females (n = 44, 52.4%); Males (n = 40, 47.6%)	Panic Disorder (84)	China
Heckman & Clay (2005)	42.35 years; SD = 13.80 years	White/Caucasian (94%); Other (2%, n = 4); Asian/Pacific Islander (1.5%, n = 3); Hispanic (1%, n = 2); Black/African-American (1%, n = 2); Native American (1%, n = 2)	Females (n = 201, 100%)	Non-clinical group	USA

Hengartner et al. (2015)	29.17 years; SD = 6.84 years	Not reported	Females (n = 750, 63.9%); Males (n = 750, 63.9%)*	Non-clinical group	Switzerland
Hovens et al. (2015)	18- 65 years	Not reported	Females (67%); [Males not specified]	Current depression or anxiety disorder (n = 1701); life-time diagnoses or at risk or subthreshold symptoms (n = 907); healthy controls (n = 373)	Netherlands
Husain et al. (2021)	Depressed group (36.27 years; SD = 10.63 years); Non depressed group (33.24 years; SD = 11.18 years)	Not reported	Females (n = 455, 100%)	Major depressive disorder (n = 246); Non-depressed controls (n = 209)	Pakistan
Jain et al. (2024)	64 years; SD = 9 years	White (79%); Asian/Pacific Islander (14%); Black/African American (5%); Hispanic (4%); Other (2%)	Females (80%); [Males not specified]	Non-clinical group	USA
Jardim et al. (2019)	72.2 years; SD = 7.11 years	Not reported	Females (n = 200, 76.9%); [Males not specified]	Non-clinical group	Brazil

Jimenez et al. (2019)	21.3 years; SD = 3.8 years	Not reported	Females (75%); [Males not specified]	Non-clinical group	Colombia
Jirakran et al. (2023)	Healthy control (37.9 years; SD = 9.2 years); Major depressive disorder (37.0 years; SD = 11.5 years)	Not reported	Healthy control: Females (n = 58, 43.6%); Males (n = 9, 6.8%); Major depressive disorder: Females (n = 48, 36.1%); Males (n = 18, 13.5%)	Healthy controls (n = 67); Major Depressive Disorder (n = 66)	Thailand
Jung (2021)	Aged 20 to 74 years (M=47.056, SD=13.119)	Not reported	Females (n = 1563, 51.5%); Males (n = 1471, 48.5%)	Non-clinical group	USA
Kamali et al. (2019)	Bipolar (40.6 years; SD = 12.2); control (31.6 years; 13.9 years)	Not reported	Bipolar: Females (n = 99, 36.7%); Control: Females (n = 63, 23.3%)	Bipolar Type 1 (n = 151); control (n = 119)	USA
Kang et al. (2021)	21.36 years; SD = 20.22 years	Not reported	Females (n = 334, 75.2%); Males (n = 110, 24.8%)	Major Depressive Disorder (n = 444)	China
Karmakar et al. (2017)	29.3 years; SD = 1.86 years	White/Caucasian (60.8%); Black/African American (19.9%); Asian (5.9%); Hispanic (estimated 15.8%)	Females (n = 7230, 53.6%); [Males not specified]	(Self-reported provider diagnosis) Migraine (n = 1927)	United Sates
Knight et al. (2023)	19.63 years: SD = 2.33 years	Mexican American (n = 105, 100%)	Males (44%); [Females not specified]	Non-clinical group	USA

Koschiget et al. (2023)	23.31 years; SD = 1.40 years	Not reported	Females (59.0%); [Males not specified]	Non-clinical group	Germany
Kounou et al. (2015)	France (41.0 years; SD = 12.0); Togo (38.9 years; SD = 9.2 years)	Not reported	France: Females (n = 53, 35.3%); Togo: Females (n = 47, 31.3%)	Major Depressive Disorder (n = 150)	France and Togo
Lam et al. (1997)	18.8 years; SD = 2.3 years	Caucasian (n = 172, 65.2%); Asian American (n = 59, 22.3%); Hispanic (n = 15, 5.7%); African American (n = 9, 3.4%); Other (n = 8, 3.0%); Native American (n = 1, 0.4%)	Females (n = 264, 100%)	Non-clinical group	USA
Lawrence (2022)		White/Caucasian (46.5%); Black/African American (33.1%); Hispanic (18.7%); Other (1.7%)	Females (n = 398, 100%)	Non-clinical group	USA
Lee et al. (2024)	62.43 years; SD = 7.69 years	White (n = 109630, 97.9%); Asian/British Asian (n = 791, 0.7%); Black/British Black (n = 716, 0.6%); Mixed (n =	Females (n = 60262, 53.8%); Males (n = 51669, 46.2%)	Non-clinical group	United Kingdom

		568, 0.5%); Chinese (n = 226, 0.2%)			
Lee et al. (2017)	50.616 years; SD = 18.203 years	Not reported	Females (n = 769, 55.1%); Males (n = 628, 45.0%)	Non-clinical group	Republic of Korea
J. Liu et al. (2023)	19.93 years; SD = 1.35 years	Not reported	Males (n = 387, 123.2%); Females (n = 330, 105.1%)*	Non-clinical group	China
Liu et al. (2020)		Not reported	Not reported	Non-clinical group	China
F. Liu et al. (2023)	19.89 years; SD = 1.25 years	Not reported	Males (n = 657, 91.6%); Females (n = 512, 71.4%)*	Non-clinical group	China
Lopez-Mongay et al. (2021)	Without childhood sexual abuse (40.48 years; SD = 9.1 years); With childhood sexual abuse (38.98 years; SD = 10.3 years)	Not reported	Males (n = 31, 62.0%); Females (n = 19, 38.0%)	Schizophrenia (n = 33); Schozoaffective disorder (n= 17)	Spain
Lui et al. (2025)	Community (24.91 years; SD = 10.19 years); Offender (37.04 years; SD = 10.56)	Not reported	Community: Females (60.2%); Offenders: Males (100%)	Non-clinical group	China
Lund et al. (2017)	22-26 years; 30-35 years	Not reported	22-26 years: Females (n = 35, 22.6%); Males (n = 28, 18.1%); 30-35 years:	Non-clinical group	Canada

			Females (n = 56, 36.1%); Males (n = 36, 23.2%)		
Luo et al. (2020)	Sample 1 (65.04 years; SD = 8.89 years); Sample 2 (55.64 years; SD = 12.42)	Sample 1: White/Caucasian (85.9%); African American (10.6%); Other (3.5%)	Sample 1: Females (61.8%); Sample 2: Females (53.8%)	Non-clinical group	USA
Lysaker et al. (2001)	44 years; SD = 9.32 years	Caucasian (n = 38, 86.4%); African American (n = 16, 36.4%)*	Males (n = 52, 118.2%); Females (n = 2, 4.5%)*	Schizophrenia (n = 36); Schizoaffective disorder (n =18)	USA
Marchi et al. (2022)	20.5 years; SD = 2.5 years	Not reported	Males (n = 606, 48.0%); [Females not specified]	Non-clinical group	Netherlands
Martín-Blanco et al. (2014)	30.4 years; SD = 6.9 years	Not reported	Females (n = 111, 85.4%); [Males not specified]	Borderline Personality Disorder (n = 130)	Spain
Masuya et al. (2022)	41.7 years; SD = 12.1 years	Not reported	Females (n = 335, 58.2%); Males (n = 249, 43.2%)	Non-clinical group	Japan
Masuya et al. (2024)	41.6 years; SD = 12.0 years	Not reported	Females (n = 327, 56.0%); Males (n = 249, 42.6%)	Non-clinical group	Japan

Mc Elroy & Hevey (2014)	18-68 years	White (n = 176, 100%)	Females (n = 90, 51.1%); Males (n = 86, 48.9%)	Mood disorder (n = 50); Dual diagnosis (n = 50); No disorder (n = 35); Substance dependence (n = 26); Substance induced mood disorder (n = 15)	Ireland
Michal et al. (2025)	57.32 years; SD = 11.55 years	White (n = 985, 78.5%); African American/Black (n = 215, 17.1%); Other (n = 31, 2.5%); Native American (n = 17, 1.4%); Refused (n = 4, 0.3%); Asian (n = 3, 0.2%)	Females (n = 713, 56.8%); [Males not specified]	Non-clinical group	USA
Moreira et al. (2024)	32.90 years; SD = 15.12	Not reported	Females (n = 420, 69.0%); [Males not specified]	Non-clinical group	Portugal
Mosley-Johnson et al. (2021)	Wave 1 (20-74 years); Wave 2 (20-75+ years)	Wave 1: White (n = 1354, 41.9%); Black (n = 86, 2.7%); Other (n = 47, 1.5%); Wave 2: White (n = 1615, 49.9%); Black (n =	Wave 1: Females (n = 802, 24.8%); Males (n = 694, 21.5%); Wave 2: Females (n = 985, 30.5%); Males (n = 753, 23.3%)	Non-clinical group	USA

		61, 1.9%); Other (n = 62, 1.9%)			
Ng & Hartanto (2022)	54.43 years; SD = 11.30 years	Not reported	Males (53.7%); [Females not specified]	Non-clinical group	USA
Nguyen-Feng et al. (2017)	21 years; SD = 3.49 years	White/European American (71%); Asian American/Asian (21%); Other (8%)	Females (74%); [Males not specified]	Non-clinical group	USA
Ogle et al. (2015)	63.43 years; SD = 2.78	Not reported	Males (61.3%); Females (38.7%)	Non-clinical group	USA
Ono et al. (2017)	42.31 years; SD = 11.99	Not reported	Males (n = 221, 53.5%); Females (n = 192, 46.5%)	Non-clinical group	Japan
Ottesen et al. (2018)	36.6 years	Not reported	Females (n = 153, 73.2%); [Males not specified]	Unipolar disorder (n = 83); Other non-afective disorders (n = 61); Bipolars disorder (n = 31)	Denmark
Otto et al. (2021)	25.9 years; SD = 4.6 years	Not reported	Females (n = 95, 100%)	Control (n = 51); Borderline Personality Disorder (n = 44)	Germany

Ozen et al. (2018)	27.39 years; SD= 10.56 years	Not reported	Males (n = 66, 50.8%); Females (n = 64, 49.2%)	PTSD (n = 21)	Turkey
Peng et al. (2025)	Major depressive disorder without childhood maltreatment (35.22 years; SD = 10.04 years); Major depressive disorder with childhood maltreatment (35.18 years; SD = 9.38 year); Healthy controls without childhood maltreatmemt (30.97 years; SD = 7.50 years); Healthy controls with childhood maltreatment (33.73 years; SD = 7.89 years)	Not reported	MDD without childhood maltreatment: Males (n = 41, 53.9%); Females (n = 35, 46.1%); MDD with childhood maltreatment: Females (n = 68, 89.5%); Males (n = 44, 57.9%); Healthy controls without childhood maltreatment: Males (n = 44, 57.9%); Females (n = 29, 38.2%); Healthy controls with childhood maltreatment: Males (n = 30, 39.5%); Females (n = 29, 38.2%)*	Major Depressive Disorder (n = 188); Healthy controls (n = 132)	China
Peters et al. (1994)	Control parents (47.6 years); Patients parents (45.5 years)	Caucasian (90%); Asian/African American/Hispanic (10%)	Not reported	Non-clinical group	USA
Pflanz et al. (2024)	55.63 years; SD = 7.61 years	Not reported	Females (54.8%); [Males not specified]	Non-clinical group	United Kingdom

Pickering et al. (2004)	47 years	Caucasian (n = 90, 100%)	Females (73%); Males (27%)	2 or more episodes of unipolar depression of at least mederate severity (n = 90)	United Kingdom
Ponder et al. (2023)	18.87 years; SD = 1.28 years	Black/African American (37.0%); White (28.9%); Hispanic/Latino/Spanish Origin (15.5%); Multiple ethnicities (9.6%); Asian/Asian American (5.7%); Middle Eastern/Arab/North African (1.4%); Other (0.9%); Native Hawaiian/Other Pacific Islander (0.5%); American Indian/Alaskan Native (0.4%)	Females (n = 563, 73.3%); [Males not specified]	Non-clinical group	USA

Pos et al. (2016)	34.7 years; SD = 7.4 years	15.6% non-white ethnic minority, no other information given	Males (n = 133, 81.6%); Females (n = 34, 20.9%)	55.2% Schizophrenia, paranoid type, 14.1% schizoaffective disorder, 14.1% psychotic disorder NOS or spectrum disorder, 10.4% schizophrenia, residual type or undifferenciated, 6.1% schizophrenia, disorganised type	Netherlands
Powers et al. (2014)	Med= 41 years	African American (92.5%); White (4.3%); Hispanic/Latino (3.7%); Mixed/other (2.3%)	Females (65%); [Males not specified]	Non-clinical group	USA
Qin et al. (2024)	19.71 years; SD = 11.93 years	Not reported	Females (n = 774, 60.8%); Males (n = 498, 39.2%)	Depression (n = 544)	China
Rademaker et al. (2010)	31.10 years; SD = 8.98 years	Not reported	Males (n = 369, 70.7%); [Females not specified]	Not reported-measures PTSD symptoms but	Netherlands

				does not mention diagnoses	
Ramos et al. (2024)	23.46 years; SD = 2.84 years	Not reported	Females (n = 201, 52.9%); Males (n = 161, 42.4%); Non-binary/Queer/Gender fluid (n = 12, 3.2%); Two-spirit (n = 1, 0.3%); Agender (n = 1, 0.3%); Prefer not to answer (n = 1, 0.3%)	Non-clinical group	international, online
Rose et al. (2023)	24.92 years; SD = 4.50 years	White (64.5%); Black/African American (12%); Latinx (11.4%); Asian American (8.4%); Other/Prefer not to say (3.8%)	Female (n = 481, 58.5%); Male (n = 330, 40.1%); Non-Binary (n = 11, 1.3%); Prefer not to say (n = 12, 1.5%)	Non-clinical group	USA
Roy et al. (2002)	Not reported	Not reported	Males (n = 516, 97.0%); Females (n = 16, 3.0%)	Cocaine and/or Opiate dependent (n = 448); Alcohol dependence (n = 84)	USA
Sanwald et al. (2023)	Healthy control (32.45 years; SD = 11.97 years); Patients (32.42 years; SD = 12.40 years)	Not reported	Females (n = 172, 72.3%); [Males not specified]	Major Depressive Disorder (n = 119); Healthy control (n = 119)	Germany

Schwandt et al. (2013)	Alcohol- dependent (41.4 years; SD = 10.0 years); Control (28.7 years; SD = 9.7 years)	Alcohol-dependent: White (n = 158, 37.9%); Black/African American (n = 98, 23.5%); Unknown (n = 13, 3.1%); Mixed (n = 5, 1.2%); Asian (n = 4, 1.0%); American Indian/Alaskan (n = 2, 0.5%); Control: White (n = 88, 21.1%); Black/African American (n = 30, 7.2%); Asian (n = 11, 2.6%); Mixed (n = 3, 0.7%); Unknown (n = 2, 0.5%); American Indian/Alaskan (n = 0, 0%)	Alcohol-dependent: Males (n = 190, 45.6%); Females (n = 90, 21.6%); Control: Males (n = 85, 20.4%); Females (n = 52, 12.5%)	Alcohol dependence (n = 280); Control (n = 137)	USA
Shen et al. (2021)	(range= 17-22 years) 18.94 years (±1.44)- at time 1	Not reported	Females (n = 389, 89.8%); Males (n = 44, 10.2%)	Not reported- measures depression symptoms but does not mention diagnoses	China
Shi et al. (2021)	Not reported	Not reported	Females (n = 726, 57.3%); Males (n = 540, 42.7%)	Non-clinical group	China

Spinhoven et al. (2010)	18- 65 years	Not reported	Not reported	Anxiety or Depressive Disorder (n = 2288); Control (n = 498)	Netherlands
Stevanovic et al. (2016)	Mean age= 41.13 (SD= 11.71)	Not reported	Sample consisted of only female participants (100%)	20.7% currently met the criteria for PTSD	Croatia, Serbia, Italy, and the Netherlands
Stokes et al. (2013)	Mean age= 21.03 (SD Not reported)	86% Caucasian; 2.8% Hawaiian or Pacific Islander; 5.6% Hispanic/Latino; 2.8% mixed race	86% female; 14% male	Not reported- measures various symptoms using scales but no mention of formal diagnoses	USA
Sturmbauer et al. (2019)	Mean age= 30.3 (SD= 19.9)	93% White - information about the remaining 7% is not reported	Women (n = 217, 72.8%); Men (n = 81, 27.2%)	No mention of diagnoses	Germany
Tachi et al. (2019)	Healthy controls: (Mean age= 44.6, SD= 11.2) Major depressive disorder (MDD) patients: (Mean age= 46.0, SD= 12.0)	Not reported	Healthy controls: Men (n = 206, 47.7%); Women (n = 144, 33.3%); MDD patients: Men (n = 46, 10.6%); Women (n = 36, 8.3%)	MDD= 82	Japan

Trent et al. (2023)	Mean age= 18.75 (SD= 1.05)	83.2% non-Hispanic white; 7.0% Asian American; 4.7% African American; 4.2% Hispanic/Latino; 0.7% Pacific Islander; 0.2% American Indian	Women (70.8%); Men (29.2%)	No mention of diagnoses	USA
Trombello et al. (2018)	Mean age= 37.16 (SD= 13.03)	White (n = 124, 66.0%); Black/African American (n = 45, 23.9%); Asian (n = 14, 7.4%); Native American/Alaska Native (n = 1, 0.5%); Other (n = 11, 5.9%)	Female (n = 129, 68.6%); Male (n = 66, 35.1%)*	Any anxiety disorder= 85, Anxiety not otherwise specified= 10, Generalised anxiety disorder= 24, Obsessive compulsive disorder= 5, Panic disorder= 17, Specific phobia= 35, PTSD= 16	USA
Tyra et al. (2021)	Mean age= 19.40 (SD= .95)	68.9% Caucasian; 22.7% Hispanic/Latino (does not specify the rest)	Female (71.4%); [Male not specified]	No mention of diagnoses	USA

van Harmelen et al. (2014)	Mean age= 41.9 (SD= 13.0)	97% Dutch (rest not specified)	Women (n = 1979, 1020.1%); Men (n = 1002, 516.5%)*	1701 participants with current diagnosis of depression or anxiety disorder	Netherlands
Vasupanrajit et al. (2024)	Healthy controls mean age= 23.48 (SD= 3.18), Low ACEs mean age= 22.79 (SD= 3.45), High ACEs mean age= 21.89 (SD= 2.42)	Not mentioned	Healthy controls: Female (n = 37, 31.4%); Male (n = 7, 5.9%); Low ACEs: Female (n = 32, 27.1%); Male (n = 5, 4.2%); High ACEs: Female (n = 30, 25.4%); Male (n = 7, 5.9%)	74 participants with major depressive disorder	Thailand
Veith et al. (2017)	Mean and SD not specified	77.3% Caucasian; 6.9% African American; 4.4% Hispanic; 4.6% biracial/multi-racial; 3.4% Asian American; 1.5% Native American; 1.9% other	Women (n = 353, 67.1%); Men (n = 173, 32.9%)	No mention of diagnoses	USA
Verrastro et al. (2024)	Age range= 18-25 years (mean and SD not specified)	Not reported	Women (50%); [Men not specified]	No mention of diagnoses	Italy
Walker et al. (1997)	Not reported	Not reported	Sample consisted of only female participants (100%)	Some participants with Rheumatoid arthiritis and some	USA

				with Fibromyalgia (frequencies not specified)	
D. Wang et al. (2018)	Mean age (female participants)= 19.0 (SD= 2.0), Mean age (male participants)= 19.2 (SD= 1.75)	Not reported	Female (n = 454, 81.8%); Male (n = 101, 18.2%)	No mention of diagnoses	China
Q. Wang et al. (2018)	Mean age= 26.7 (SD= 4.1)	Not reported	Male (n = 103, 8.2%); [Female not specified]	Major depression= 7, PTSD= 2, Alcohol abuse= 6, Alcohol dependence= 3	USA
Wang et al. (2010)	Mean age= 20.1 (SD= 139)	Not reported	Female (n = 215, 74.4%); Male (n = 189, 65.4%)*	No mention of diagnoses	China
Wang et al. (2020)	Mean age= 20.1 (SD= 1.2)	Not reported	Females (n = 764, 189.1%); Males (n = 489, 121.0%)*	No mention of diagnoses	China
Wang et al. (2022)	Mean age= 22 (SD= 2.6)	No mention of ethnicity	Male only sample (n = 120, 100%)	No mention of diagnoses	Netherlands
Weltz et al. (2016)	Mean age= 19.23 (SD= 1.41)	79.6% European American (the rest is not specified)	Women (53.7%); [Men not specified]	No mention of diagnoses	USA

Wrobel et al. (2023)	Mean age= 51.5 (SD= 14.0)	Caucasian (n = 188, 90.0%); African American (n = 10, 4.8%); Asian (n = 3, 1.4%); Multiracial (n = 9, 4.3%); Unknown/not reported (n = 2, 1.0%)	Women (n = 140, 67.0%); Men (n = 68, 32.5%)	Bipolar disorder 1= 138 (66.0%), Bipolar disorder 2= 44 (21.1%), Bipolar disorder not otherwise specificied= 17 (8.1%), Schizoaffective disorder (bipolar type)= 10 (4.8%)	Australia
Xu et al. (2017)	Unable to access	Unable to access	Unable to access	Unable to access	China
You et al. (2022)	Mean age= 36.6 (SD= 11.64)	Not reported	Patient group: Females (n = 330, 27.0%); Males (n = 158, 12.9%); Comparison condition: Females (n = 389, 31.8%); Males (n = 345, 28.2%)	Major depressive disorder= 130, Bipolar disorder 1= 79, Bipolar disorder 2= 279	Republic of Korea
Yrondi et al. (2021)	Mean age= 67.2 (SD= 5.7)	Not reported	Female (n = 60, 62.5%); [Male not specified]	Unipolar Treatment-Resistant depression	France
Zhao et al. (2022)	Mean age= 9.93 (SD= .73)	Not reported	Boys (55.1%); Girls (44.9%)	N/A	China

Zhou et al. (2019)	34.76 years; SD = 10.90 years	Not reported	Female (n = 182, 58.3%); Male (n = 130, 41.7%)	Depression (n = 145); Control (n = 101); Bipolar disorder (n = 21); High risk for depression (n = 45)	China
Zhou et al. (2022)	22.29 years; SD = 1.54 years	Not reported	Female (n = 425, 75.2%); Male (n = 140, 24.8%)	Suicidal idealisation (n = 373)	China

Appendix B - Chapter 3

Appendix 1B: Histograms depicting the distribution of variables of interest

Figure 3.2 – Histogram: Coping Strategies Questionnaire (CSQ-R)

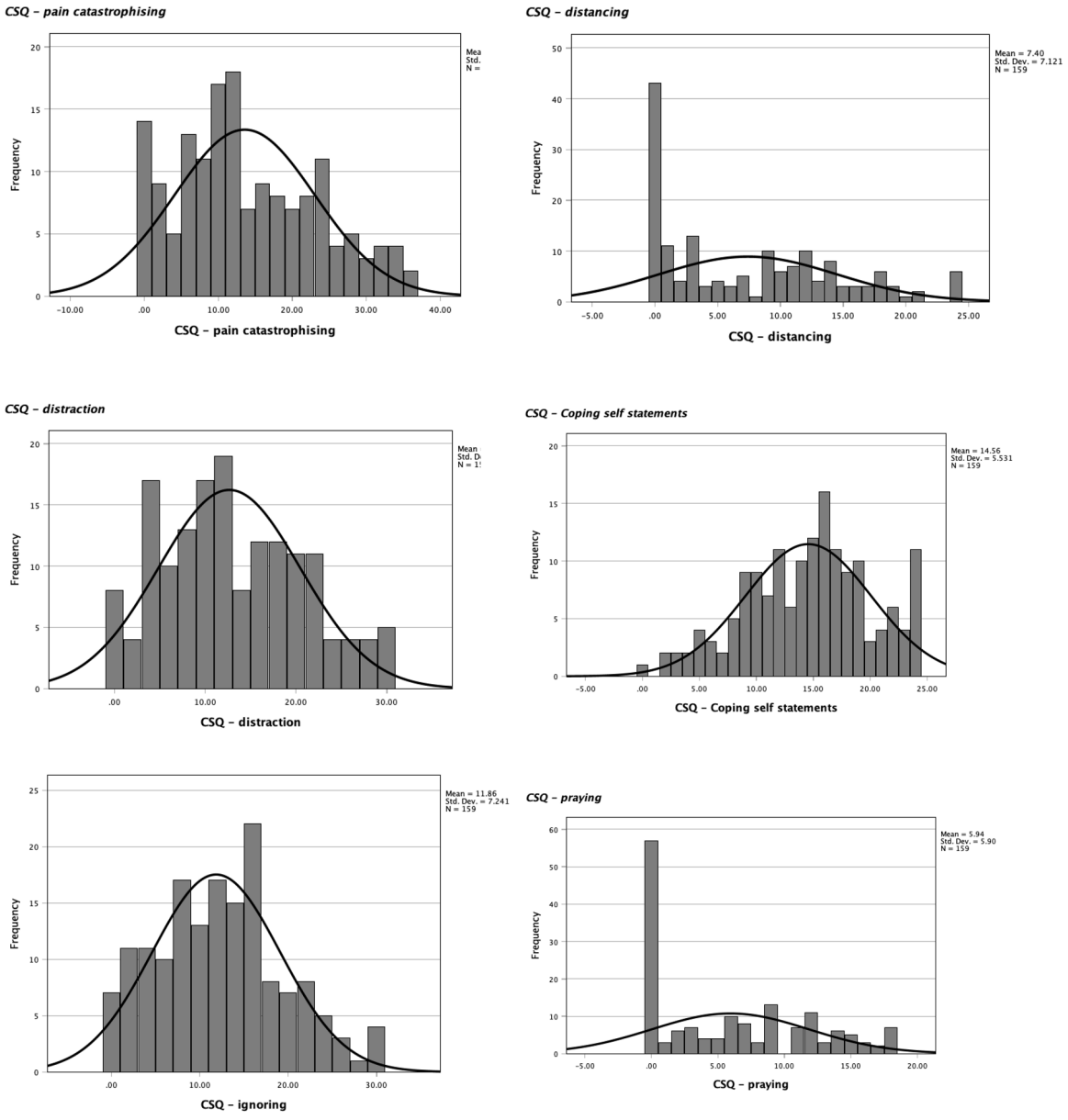


Figure 3.3 – Histogram: Childhood Trauma Questionnaire (CTQ-SF):

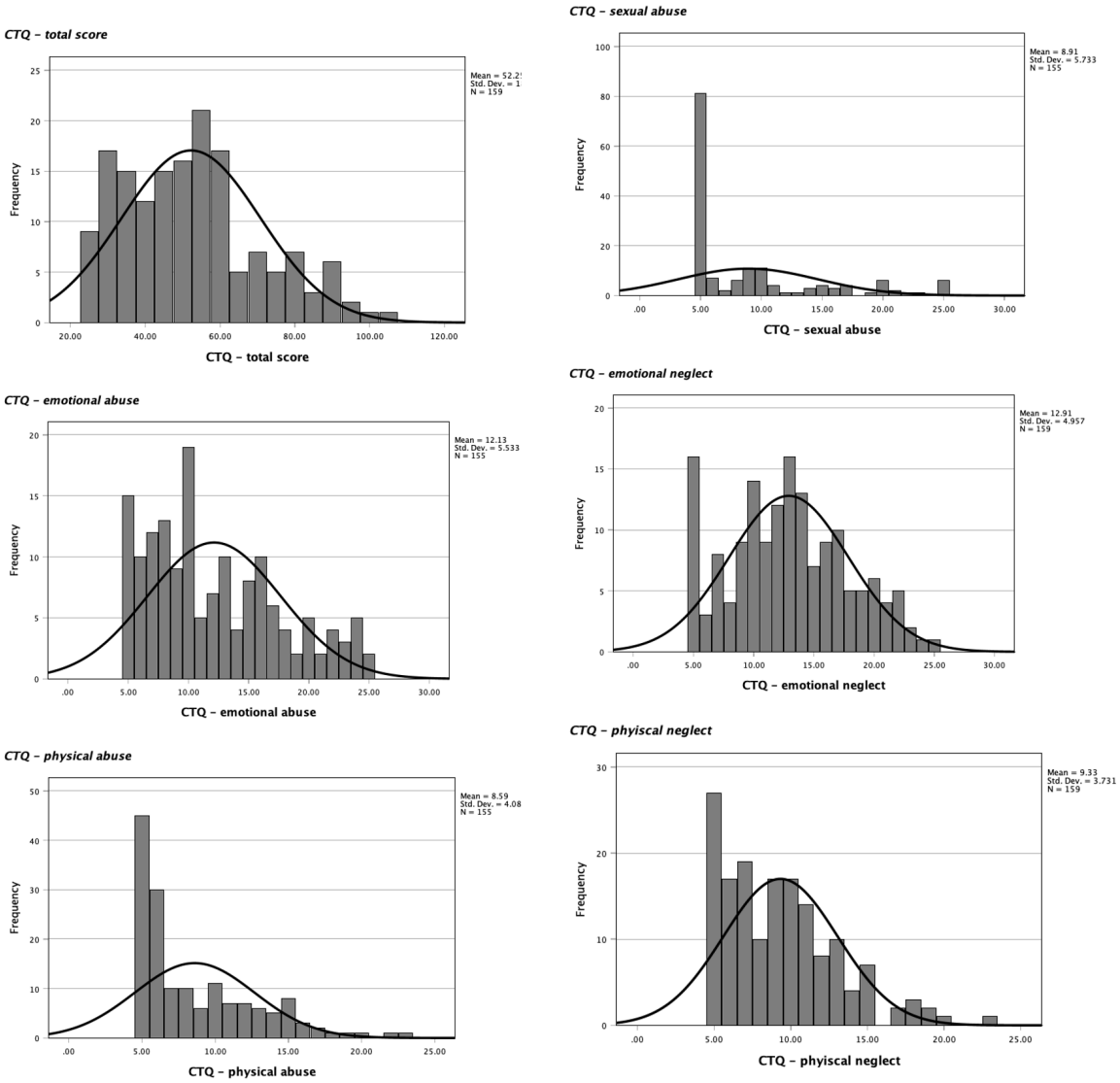
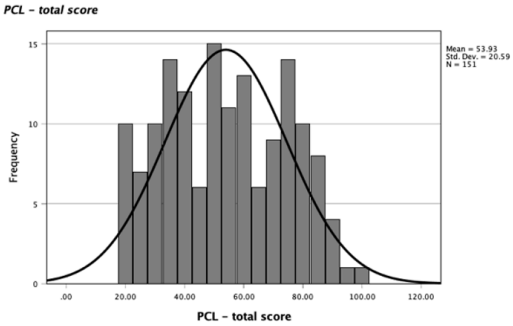
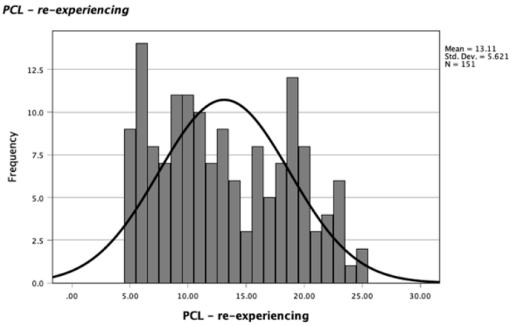
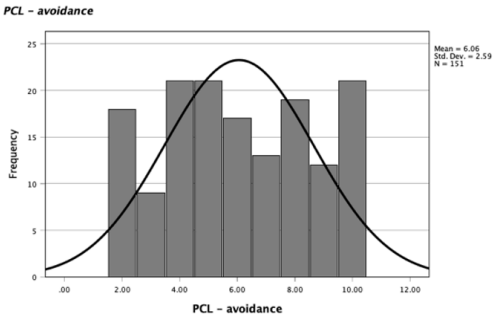
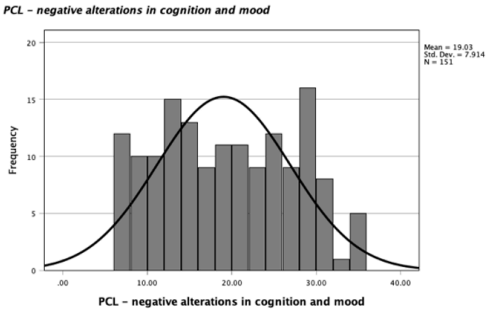


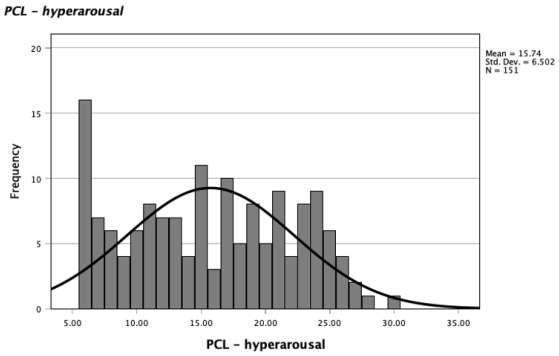
Figure 3.4 – Histogram: Posttraumatic Stress Disorder Checklist (PCL-5)











Appendix 2B: Correlational tables for all variables

Table 3.5 - Correlations between pain ratings, childhood trauma and PTSD symptoms for individuals with chronic pain (*n* = 75)

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	Pain Intensity	1																	
2	Pain Unpleasantness	.837**	1																
3	CTQ - Total Score	.307**	.237*	1															
4	CTQ - Emotional Abuse	.319**	.216	.878**	1														
5	CTQ - Physical Abuse	.136	.137	.738**	.537**	1													
6	CTQ - Sexual Abuse	.040	.041	.571**	.374**	.302*	1												
7	CTQ - Emotional Neglect	.319**	.265*	.716**	.682**	.437**	.130	1											
8	CTQ - Physical Neglect	.016	.019	.633**	.408**	.527**	.325**	.392**	1										
9	PCL - Total Score	.367**	.406**	.211	.192	.114	.271*	.038	.036	1									
10	PCL - Re-experiencing	.330**	.334**	.187	.147	.122	.229	.034	.088	.889**	1								
11	PCL - Avoidance	.278*	.294*	.132	.198	-.062	.323**	-.038	-.178	.638**	.502**	1							
12	PCL - Negative Alterations in Cognition and Mood	.325**	.371**	.229	.221	.095	.330**	.098	.017	.936**	.775**	.550**	1						
13	PCL - Hyperarousal	.337**	.394**	.165	.109	.119	.183	-.017	.087	.903**	.737**	.475**	.789**	1					
14	CSQ - Pain Catastrophising	.421**	.427**	.182	.079	.174	.126	.106	.106	.505**	.415**	.408**	.450**	.508**	1				
15	CSQ - Distraction	-.293*	-.280*	-.355**	-.376**	-.120	-.192	-.313**	-.130	-.355**	-.317**	-.192	-.355**	-.324**	-.054	1			
16	CSQ - Ignoring	-.216	-.286*	-.132	-.003	-.141	-.098	-.009	-.372**	-.060	-.112	.119	-.015	-.129	-.066	.457**	1		
17	CSQ - Distancing	-.276*	-.312**	-.205	-.150	-.072	-.024	-.261*	-.292*	-.024	-.005	.226	-.049	-.105	.059	.529**	.606**	1	
18	CSQ - Coping Self-statements	-.088	-.065	-.278*	-.143	-.218	-.186	-.228	-.377**	-.166	-.240*	.008	-.106	-.201	-.155	.481**	.647**	.419**	1
19	CSQ - Praying	-.243*	-.180	-.181	-.299*	.021	.031	-.230	.098	-.023	.003	-.095	-.014	-.019	.220	.461**	.078	.315**	.159

Note: *p < .01; ** p < .001

Appendix 3B: Correlational tables for all variables

Table 3.6 - Correlations between pain ratings, childhood trauma and PTSD symptoms for individuals with acute pain (*n* = 84)

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	Pain Intensity	1																	
2	Pain Unpleasantness	.917**	1																
3	CTQ - Total Score	.156	.139	1															
4	CTQ - Emotional Abuse	.240*	.228*	.874**	1														
5	CTQ - Physical Abuse	.224*	.191	.754**	.673**	1													
6	CTQ - Sexual Abuse	.196	.178	.482**	.356**	.399**	1												
7	CTQ - Emotional Neglect	.060	.070	.884**	.737**	.539**	.289**	1											
8	CTQ - Physical Neglect	-.040	-.044	.754**	.564**	.483**	.179**	.706**	1										
9	PCL - Total Score	.161	.146	.751**	.738**	.559**	.308**	.617**	.634**	1									
10	PCL - Re-experiencing	.182	.138	.627**	.590**	.488**	.247*	.529**	.559**	.851**	1								
11	PCL - Avoidance	.242*	.216	.689**	.666**	.585**	.212	.563**	.605**	.800**	.722**	1							
12	PCL - Negative Alterations in Cognition and Mood	.143	.144	.681**	.715**	.486**	.350**	.558**	.520**	.913**	.677**	.636**	1						
13	PCL - Hyperarousal	.074	.071	.659**	.651**	.459**	.251*	.545**	.574**	.915**	.703**	.670**	.785**	1					
14	CSQ - Pain Catastrophising	.260*	.312**	.302**	.346**	.267*	.013	.343**	.259*	.496**	.477**	.449**	.492**	.380**	1				
15	CSQ - Distraction	.018	.036	.050	.061	.064	-.021	-.027	.159**	.129	.181	.126	.074	.110	.345**	1			
16	CSQ - Ignoring	-.080	-.083	.116	.041	.085	.096	.073	.074	.161	.123	.063	.134	.208	-.255*	.172	1		
17	CSQ - Distancing	-.001	.035	.301**	.254*	.176	.202	.222**	.343**	.438**	.377**	.244*	.387**	.436**	.292**	.451**	.313**	1	
18	CSQ - Coping Self-statements	.144	.182	.250*	.185	.249*	.152	.178*	.179**	.257*	.338**	.226*	.156	.253*	.056	.286**	.466**	.247*	1
19	CSQ - Praying	.061	.091	-.024	-.041	.023	-.137	-.036	.083	.163	.290**	.107	.082	.145	.423**	.374**	-.176	.237*	.165

Note: * $p < .01$; ** $p < .001$

Appendix 4B:

Table 3.7 - Table showing *z*-tests comparing correlations between trauma variables and pain coping strategies between pain groups

Trauma Variable	Pain-related worry		Distraction		Ignoring		Distancing		Self-Statements		Praying	
	z	***p***	***z***	***p***	***z***	***p***	***z***	***p***	***z***	***p***	***z***	***p***
CTQ – Total Score	-0.78	.435	-2.57	.010	-1.57	.116	-3.17	<.001	-3.30	<.001	-0.97	.322
Emotional Abuse	-1.78	.075	-2.78	.005	-0.28	.780	-2.53	.011	-2.13	.033	-1.65	.099
Physical Abuse	-0.60	.549	-1.15	.250	-1.42	.156	-1.56	.119	-2.90	.003	-0.01	.992
Sexual Abuse	0.71	.478	-1.82	.069	-1.08	.280	-1.42	.156	-2.08	.038	1.06	.289
Emotional Neglect	-1.57	.116	-3.59	<.001	-1.06	.289	-3.78	<.001	-2.79	.005	-1.23	.219
Physical Neglect	-0.97	.332	-1.82	.069	-2.84	.004	-4.02	<.001	-3.53	<.001	0.09	.928
PCL – Total Score	0.08	.936	-5.43	<.001	-3.32	<.001	-3.43	<.001	-3.46	<.001	-1.14	.250
Re-experiencing	-0.47	.638	-3.12	.001	-1.48	.139	-0.43	.667	-2.45	.014	-1.82	.069
Avoidance	-0.30	.764	-2.02	.043	0.35	.726	-0.12	.904	-2.43	.015	-1.28	.200
Negative Alterations in Cognition and Mood	-0.35	.726	-2.72	.006	-0.94	.347	-2.79	.005	-3.64	<.001	-0.60	.549
Hyperarousal	1.01	.312	-4.55	<.001	-1.91	.056	-2.20	.028	-4.51	<.001	-1.04	.298

Note. *z*-values after r-to-z transformation are comparing correlation strengths between acute and chronic pain groups.

CTQ = Childhood Trauma Questionnaire; PCL = PTSD Checklist for DSM-5. Bolded rows indicate summary scores.

The End.