Factors that Influence the Effectiveness of Children's Trauma Treatment

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Thesis Abstract

Background. An understanding of treatment moderators and mediators allows treatment optimisation. Methodological limitations, however, exist in current literature. Evidence regarding how parental psychopathology affects child's trauma treatment response appears limited and mixed.

Methods. This thesis examined the factors that influence the effectiveness of child's trauma treatment. A systematic review identified the moderators and mediators of trauma-focused treatment for children and adolescents. An empirical study then examined the association between parental psychopathology and child's trauma symptomatology before treatment, and investigated the moderating effects of parental psychopathology on child's treatment outcome.

Results. The systematic review included 16 studies, 15 of which employed cognitive-behavioural approaches. Little to no evidence was found for age, gender, and race in moderating treatment outcomes. Support was found for the mediating effect of post-traumatic cognitive appraisal, trauma memory quality, and rumination on treatment outcomes. In the empirical study, parental depression modestly moderated treatment effectiveness; CT-PTSD demonstrated greater benefits than TAU at higher levels of parental depression. This moderation effect was not sustained at follow-up and was absent for parental anxiety and PTSD.

Conclusions. Findings suggest that trauma-focused interventions, particularly those based on cognitive-behavioural principles, could be beneficial across diverse child and adolescent populations. Cognitive processes like post-traumatic cognitive appraisal, trauma memory quality, and rumination appeared to explain treatment effects, highlighting the importance of targeting these components during interventions.

Chapter One: Introduction to Thesis Portfolio

Trauma and its prevalence

Traumatic events involve exposure to actual or threatened death, serious injury or sexual violence through direct experience, witnessing, learning that the event happened to a close person or repeated exposure to the related aversive details (DSM-V; American Psychiatric Association (APA), 2013). Individuals exposed to a traumatic event tend to experience intense fear, helplessness or horror (APA, 2013). Such events can include combat, sexual assault, robbery, natural disasters, accidents or life-threatening illnesses (APA, 2000). It was found that in the United Kingdom, nearly one third of young people experience a traumatic event in their childhood (Lewis et al., 2019). While many recover naturally, a substantial proportion develops Post-Traumatic Stress Disorder (PTSD) (12–25%; Alisic et al., 2014; Visser et al., 2025; Tamir et al., 2025). PTSD is a psychiatric disorder that can be distressing and disabling (Allen et al., 2021), and is characterised by four categories of symptoms: (1) intrusions (e.g., flashbacks, distressing dreams), (2) avoidance of reminders (e.g., people, places, activities), (3) alterations in cognition and mood (e.g., loss of interest in activities previously enjoyed, distrust in others, distorted beliefs about oneself that lead to guilt and shame), and (4) hyperarousal and reactivity (e.g., irritable, behave recklessly or self-destructively, hypervigilant, experience difficulties concentrating or sleeping) (APA, 2022). These symptoms may present differently in young children from adults, such as reenactment of trauma themes in play and aggressive behaviours (APA, 2013).

Impact of trauma on young people

Trauma has a far-reaching and long-lasting impact on young people's lives, understandably given that their neurobiological, emotional and social development is still under way (Gunnar, 2001; Shonkoff et al., 2000). Young people exposed to traumatic events are at an elevated risk of developing a range of mental health disorders compared to their peers (Kilpatrick et al., 2003; Copeland et al., 2007). They are more likely to have comorbidity in depression, anxiety, conduct disorder, substance use (e.g., Lewis et al., 2019; Khoury, 2010; Perkonigg et al., 2000; Hoven et al., 2005; Lansford et al., 2002; Cutajar et al., 2010a). Many of them also tend to carry out self-harm and suicide attempts (Rojas et al., 2017; Nock et al., 2013; Cutajar et al., 2010b). Beyond psychological impact, trauma is well-

documented to lead to physical health problems, such as cardiovascular diseases, diabetes and chronic pain (e.g., Mock & Arai, 2011; Kascakova et al., 2020; Almuneef et al., 2014). Relatedly, these result in poorer economic outcomes (Copeland et al., 2018; Metzler et al., 2017; Currie & Widom, 2010) and increased public healthcare expenditure (Kezelman et al., 2015; Loxton et al., 2019). It is crucial to provide timely and effective treatment early on given that the child's difficulties risk developing into more complex presentations which become more difficult to assess and treat (Su & Stone, 2020).

Treatment of PTSD

The National Institute for Health and Care Excellence (NICE) and international guidelines recommend trauma-focused Cognitive Behavioural Therapy (TF-CBT) (NICE, 2018; Forbes et al., 2020; Lethbridge & Australia, 2000), an empirically based treatment (e.g., Bastien et al., 2020; Bisson & Olff, 2021; Thielemann et al., 2022; Lenz & Hollenbaugh, 2015), to be offered to children and young people with PTSD. Other recommended psychotherapeutic interventions for the pediatric population include eye movement desensitisation and reprocessing (EMDR; Hoppen et al., 2025; Lewey et al., 2018), cognitive processing therapy (CPT; Xiang et al., 2021; Rosner et al., 2019) narrative exposure therapy (NET; Peltonen & Kangaslampi, 2019; Ruf et al., 2010), and prolonged exposure therapy (PET; Foa et al., 2013; Rossouw et al., 2022). This generally mirrors the adult literature, with TF-CBT and EMDR demonstrating strongest evidence of efficacy, though with considerable variation in reported effect sizes across trials (Kip et al., 2025; Cusack et al., 2016).

Factors influencing treatment outcomes

Factors that influence the effectiveness of trauma-focused interventions (i.e. treatment moderators) for children and young people have received growing interest. Understanding for whom and under what conditions a particular intervention works best, or less well, helps guide treatment decisions and optimise treatment outcomes. In both child and adult PTSD literature, evidence regarding treatment moderators largely appears limited and mixed. For example, age was found to be a significant treatment moderator in some studies (e.g., Danzi & La Greca, 2021; Gutermann et al., 2016; McLean et al., 2022) but non-significant in others (e.g., Somers et al., 2024; de Haan et al., 2024;

van Vliet et al., 2024). Inconclusive findings were also reported for baseline PTSD severity, with some studies finding that baseline PTSD symptom severity does not influence treatment outcome (e.g., Hoeboer et al., 2021; Horesh et al., 2017; van Vliet et al., 2024) while others reported a significant moderating effect (e.g., Li et al., 2023; Cloitre et al., 2016). Moreover, the direction of the moderating effect seems unclear; Li et al. (2023) showed that treatment gains are greater with higher baseline PTSD severity while Cloitre et al. (2016) found lower treatment gains for participants with higher baseline symptom severity when using a composite symptom score for the severity of PTSD, depression, dissociation, and interpersonal problems.

A small yet expanding body of research has looked at factors that explain why and how treatments are effective (i.e. treatment mediators). Understanding the mechanisms of change allows treatment optimisation as clinicians can target those treatment components to enhance treatment effectiveness. Supported by cognitive theories of PTSD (Dalgleish, 2004; Ehlers & Clark, 2000), negative post-traumatic cognitions appeared to be the most frequently studied treatment mediator in both child and adult populations and has received substantial support for its mediating effect (e.g., Meiser-Stedman et al., 2017; McLean et al., 2015; Alpert et al., 2023). In the adult literature, fear habituation is also commonly examined, with much support for its role as a mechanism of change in PTSD treatments. Such observation is in line with emotional processing theory (Foa & Kozak, 1986) which postulates that exposure allows emotional activation and replacement of maladaptive associations with realistic information (Rauch & Foa, 2006).

Current thesis portfolio

Broadly, this thesis aims to understand the factors that influence the effectiveness of children's trauma treatment. The systematic review in Chapter Two provides a narrative synthesis of findings on moderators and mediators of trauma treatment for children and young people aged 18 and below. The review focuses on randomised controlled trials evaluating the effectiveness of trauma-focused psychotherapeutic interventions for young people with PTSD symptoms (i.e. met diagnostic criteria or validated clinical cutoffs for PTSD). The empirical study in Chapter Four examines (i) the association

between parental psychopathology and child's trauma symptomatology before treatment, and (ii) the moderating effects of parental psychopathology on child's trauma treatment outcomes. Chapter Three serves as a bridge between the systematic review and the empirical study. Lastly, Chapter Five summarises the findings from both studies, evaluates the strengths and limitations of the work, discusses the implications of findings, and suggests directions for future work.

Chapter Two: Systematic Review

Prepared for Submission to Journal of Anxiety Disorders

(Author guidelines available in Appendix A)

Moderators and Mediators of PTSD Treatment for Children and Adolescents: A Narrative Synthesis

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Abstract

Background. Understanding the factors that influence the efficacy of trauma-focused psychotherapeutic interventions and the mechanism of change is essential for treatment optimisation. Methodological limitations, however, exist in current meta-analyses and systematic reviews.

Methods. This review aimed to provide a narrative synthesis of moderators and mediators of trauma-focused treatment for children and adolescents aged 18 and below. Participants were required to meet diagnostic criteria or validated clinical threshold for PTSD.

Results. 3296 articles were screened against the eligibility criteria, of which 16 studies were included in the review. Most studies (*k* = 15) employed cognitive-behavioural approaches. A large proportion of the moderating and mediating factors were studied by only a single study. Little to no evidence was found for age, gender, and race in moderating treatment outcomes. There is support for the mediating effect of post-traumatic cognitive appraisal, trauma memory quality, and rumination on treatment outcomes in the child and adolescent population.

Conclusions. Trauma-focused interventions, particularly those employing cognitive-behavioural techniques, appeared to benefit diverse child and adolescent populations. Addressing cognitive components like post-traumatic cognitions, trauma memory, and rumination would likely lead to trauma symptom improvement.

Keywords: moderator, mediator, mechanism, PTSD, trauma-focused treatment, child, adolescent

1. Introduction

1.1 Post-traumatic stress in children and adolescents

Trauma exposure is common in childhood, with a substantial proportion of children experiencing a traumatic event before age 18 (Copeland et al., 2007; Lewis et al., 2019). Among children and adolescents exposed to traumatic events, approximately 12–25% develop post-traumatic stress disorder (PTSD; Alisic et al., 2014; Visser et al., 2025; Tamir et al., 2025). The adverse long-term impact of trauma is well-established (e.g., Dams et al., 2020; Maschi et al., 2013; Măirean et al., 2024). Individuals exposed to childhood traumatic experiences are more likely to struggle with mental health problems (Lewis et al., 2019; McKay et al., 2021), social difficulties (McLean et al., 2013; Cloitre et al., 2005), and academic performance (Vilaplana-Pérez et al., 2020; Morissette et al., 2021). Given its prevalence and far-reaching impact on later life, there is a robustly expanding body of research on the efficacy of trauma-focused psychotherapies for the paediatric population.

1.2 Trauma-focused psychotherapies for children and adolescents

Trauma-focused cognitive behavioural therapy (TF-CBT) is postulated to be most effective intervention for post-traumatic stress symptoms in the paediatric population (Mavranezouli et al., 2020; Hoppen et al., 2025; Thielemann et al., 2022; Xiang et al., 2021). It has been shown to significantly reduce trauma symptoms and chance of a PTSD diagnosis post-treatment than control groups (e.g., Smith et al., 2007). Accordingly, TF-CBT is the recommended psychological intervention for the prevention and treatment of PTSD in the paediatric population by international practice guidelines (National Institute for Health and Care Excellence (NICE), 2018; Phoenix Australia, 2021; International Society for Traumatic Stress Studies (ISTSS), 2019). Recommended as the second-line intervention, eye movement desensitisation and reprocessing (EMDR) has received relatively strong support as an effective intervention for PTSD in this population (Hoppen et al., 2025; Lewey et al., 2018; Bastien et al., 2020). Other interventions that have revealed to be effective include cognitive processing therapy (CPT; e.g., Xiang et al., 2021; Rosner et al., 2019) narrative exposure therapy (NET;

Peltonen & Kangaslampi, 2019; Ruf et al., 2010), and prolonged exposure therapy (PET; Foa et al., 2013; Rossouw et al., 2022).

1.3 Understanding the factors influencing treatment effectiveness

There has been growing interest in understanding factors that influence the efficacy of those interventions (i.e. treatment moderators). An understanding of these factors allows tailoring of treatment to enhance its effects. Over the past decade, there have been several meta-analyses and systematic reviews on the treatment moderators of trauma-focused interventions for the younger population (e.g., Danzi & La Greca, 2021; Taylor et al., 2015; Yohannan et al., 2022; de Haan et al., 2024). Reviewed treatment moderators could largely fall under three categories (Somers et al., 2024): 1) individual characteristics (e.g., age, gender, baseline symptom severity, type of trauma); 2) treatment characteristics (e.g., caregiver involvement, number of sessions, treatment duration, delivery format; and 3) study characteristics (e.g., control condition, measurement instrument, informant, study quality). Generally, evidence behind most treatment moderators seems limited and inconclusive. In some meta-analyses (e.g., Somers et al., 2024; de Haan et al., 2024; Yohannan et al., 2022; Szota et al., 2023), age was not found to be a significant moderator of treatment effectiveness while the review by Danzi & La Greca (2021) and meta-analysis by Gutermann et al. (2016) concluded that treatments appear more effective for older children. The finding that older children benefit more from treatment could be explained by their higher cognitive ability (Gutermann et al., 2016). Effective treatments like TF-CBT rely heavily on cognitive components (e.g., narrate trauma, identify maladaptive appraisals, challenge unhelpful beliefs) which may not be developmentally suitable for younger children (Lenz & Hollenbaug, 2015). Moreover, measurement of trauma symptoms using the DSM criteria might not be able to adequately capture young children's trauma severity and their treatment progress (Scheeringa et al., 2006; Scheeringa et al., 2001).

Similarly, a mixed picture was found for caregiver involvement. Meta-analyses by Szota et al. (2023), Gutermann et al. (2016; 2017) and Corcoran and Pillai (2008) revealed that interventions that involved caregivers led to greater reduction of trauma symptoms. This is consistent with studies that

reported an association between enhanced perceived social support and symptom reduction (Birkeland et al., 2020; Fletcher et al., 2021), and the TF-CBT model which posits that caregivers can positively impact their child's trauma treatment by offering support, modelling healthy coping skills, and managing their own maladaptive trauma-related cognitions (Brown et al., 2020). They however reported that the moderation effects were not significant beyond 12 months post-treatment. These seem to suggest that caregiver-involvement might boost treatment effectiveness but not necessary in the long term (Szota et al., 2023). On the other hand, Morina et al. (2016) and de Haan et al. (2024) reported little evidence that caregiver involvement significantly affected treatment outcome.

1.4 Understanding the mechanisms through which treatments are effective

Relatively fewer studies have investigated factors that may account for the treatment effectiveness (i.e. treatment mediators). Understanding the mechanisms that facilitate therapeutic change is crucial for treatment optimisation (Taubner et al, 2024; Alpert et al, 2023). In a recent review by Protić et al. (2024), mediators were categorised into 1) cognitive (child's post-traumatic cognitions, over-accommodation, trauma-related memory, rumination); 2) parent/family-related (e.g., parental emotional reactions, parental depressive symptoms, parental dysfunctional post-traumatic cognitions); 3) mental-health-related (i.e. child's depression symptoms); 4) therapy-related (i.e. therapeutic alliance); and 5) behavioural (i.e. safety-seeking behaviours). Child's post-traumatic cognition was shown to be the most studied mediator and a significant mediator in four out of five papers reviewed by Protić et al. (2024) (e.g., Meiser-Stedman et al., 2017; McLean et al., 2015). This is in accordance with cognitive theories of PTSD that propose that dysfunctional appraisals of the trauma threaten one's appraisal of self and the world which lead to and maintain PTSD (Dalgleish, 2004; Ehlers & Clark, 2000). Other cognitive factors like trauma-related memory and rumination were tested in one study (i.e. Meiser-Stedman et al., 2017) and found to be significant treatment mediators.

1.5 Rationale for current review

Prior meta-analyses and systematic reviews have contributed to our understanding of factors that could influence PTSD treatment effectiveness for children and adolescents (e.g., Danzi & La Greca,

2021; Taylor et al., 2015; Yohannan et al., 2022; de Haan et al., 2024; Somers et al., 2024; Szota et al., 2023). While the methodological approaches adopted in those papers were valuable in addressing their research aims, they introduce limitations that the present review sought to complement. For instance, some reviews restricted inclusion to cognitive behavioral techniques (e.g., Yohannan et al., 2022) or specifically TF-CBT (e.g., de Haan et al., 2024), incorporated non-trauma-focused treatments (e.g., Yohannan et al., 2022) or non-randomised study designs (e.g., Yohannan et al., 2022; Szota et al., 2023), or focused exclusively on interventions with caregiver involvement (e.g., Somers et al., 2024; Szota et al., 2023). Danzi & La Greca (2021) offered a timely update to an earlier review by Taylor et al. (2015) by consolidating findings from meta-analyses and systematic reviews to examine the moderators of trauma treatment responses in children and adolescents. This approach, however, risks aggregation bias wherein heterogeneous results from primary studies may be averaged out, potentially obscuring certain moderator effects.

With respect to treatment mediators, Protić et al. (2024) conducted the first systematic review of treatment mediators for young people with trauma symptoms. Recognising the underrepresentation of transition age youths (TAY; 20 – 29 years) in the literature, the authors intended to include studies involving both adolescents (10 – 19 years) and TAY. However, as no studies focused exclusively on TAY, the included studies were ultimately limited to adolescent samples. Notably, participants were included on the basis of trauma-related symptoms, without the requirement of meeting formal diagnostic criteria or validated clinical threshold for PTSD (e.g., Allan et al., 2015; Kangaslampi et al., 2016). Further, they included studies that looked at mediators of pre- to post-treatment symptom change (e.g., McLean et al., 2015), and not specifically mediators of the relationship between treatment condition and post-treatment outcome (i.e. treatment mediators).

This review aimed to extend the literature by providing a developmentally targeted narrative synthesis of both moderators and mediators of trauma-focused psychotherapies for children and young people aged 18 years and below. To address the above-mentioned methodological gaps, the present review broadened the scope to encompass any trauma-focused psychotherapeutic

intervention, irrespective of caregiver involvement while imposing stricter inclusion criteria to ensure rigor and relevance. Specifically, participants must meet diagnostic criteria or validated clinical cutoffs for PTSD, and only RCTs would be included to enhance the robustness of the findings.

2. Method

2.1 Preregistration

This review adheres to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Page et al., 2021). We registered the review with PROSPERO (CRD420251014299; https://www.crd.york.ac.uk/ PROSPERO/view/CRD420251014299).

2.2 Search strategy

The searches were conducted in July 2025, then re-run on 6th January 2026 to update the review to include any publications since the initial search was run. To allow a comprehensive search, studies published since 1st January 1980 were considered. Databases used include: PsycInfo, PubMed, Scopus, PTSDpubs (formerly PILOTS), and ProQuest.

Search terms fall under three main areas: 1) child and young person (child* OR adolescen* OR boy* OR girl* OR teen* OR youth* OR student* OR pupil* OR minor* OR pediat* OR paediat*); 2) post-traumatic stress (ptsd OR ptss OR "posttraumatic stress" OR "post-traumatic stress"); 3) randomised controlled trial ("RCT" OR "randomised controlled trial" OR "randomized controlled trial" OR "clinical trial" OR "randomised trial" OR "randomized trial" OR "controlled trial"). The search terms were used within the title and abstract fields.

2.3 Eligibility criteria

Articles were screened against the following criteria:

2.3.1 Population

Mean age of participants was 18 years and below. Participants were reported to have PTSD symptoms following a traumatic event(s) (e.g., sexual assault, war). PTSD symptoms were operationalised as meeting diagnostic criteria (e.g., DSM-5, ICD-10) or validated clinical threshold for

PTSD (e.g., Child PTSD Symptom Scale for DSM-5 (CPSS-5) ≥ 31). There were no restrictions on PTSD severity and trauma type.

2.3.2 Intervention

Interventions were trauma-focused psychotherapeutic treatments. They are defined as interventions that facilitate the processing of traumatic memories and identify this process as the core component of the therapeutic work (Schnurr, 2017; Sin et al., 2017). Processing memories of the trauma could be addressed through identifying maladaptive appraisals of the experience, modifying unhelpful beliefs, and repeatedly recounting memories of the traumatic event (Furuta et al., 2018). Such interventions include TF-CBT, cognitive therapy (CT), EMDR, and NET. Interventions that only included psychoeducation and/or meditation were excluded.

We included interventions of any length, format (one-to-one or group, face-to-face or online). There was no restriction on the profession of the treatment facilitators (e.g., psychologist, teacher, social care worker), treatment setting, and language. Comorbidities were allowed, as long as the intervention focused on evaluating treatment effectiveness for PTSD symptoms. Eligible studies were required to evaluate a single trauma-focused psychotherapeutic treatment (i.e. the intervention under investigation). Comparison groups were limited to non-trauma-focused interventions, which could include active controls (e.g., client-centred therapy, treatment as usual) and wait list. Studies comparing two or more trauma-focused psychotherapeutic interventions were excluded.

2.3.3 Outcome measures

The outcome measure was the measure of post-traumatic stress symptoms following treatment (e.g., post-treatment, 6-month follow-up, 1-year follow-up). It could be assessed using child reports, parent reports, or clinical interviews.

2.3.4 Analysis

Studies were to have conducted moderation or mediation analyses of treatment effectiveness. For both types of analyses, the independent variable would be the treatment condition while the dependent variable would be a measure of post-traumatic stress symptoms following treatment.

2.4 Screening process

The screening process is outlined in a PRISMA diagram (Figure 1). After removing duplicates, the first author screened articles by title, abstract, and full text for eligibility. A second coder independently coded approximately 10% of the studies screened at the title and abstract stage (*n* = 330) and 20% of the studies (*n* = 56) at the full-text stage. The inter-rater agreement was substantial (κ= .648 and .791 respectively) (Landis & Koch, 1977).

2.5 Data extraction

The following data was extracted from the studies: sample size, country, studied interventions and comparison groups, results on each studied moderator and mediator, and information for assessment of study quality.

2.6 Quality assessment

Included studies were assessed for their methodological quality using assessment criteria proposed by Cuijpers et al. (2010), which were based on the Cochrane Collaboration criteria for assessing the methodological validity of studies (Higgins & Green, 2008) and an authoritative review of empirically supported psychological therapies (Chambless & Hollon, 1998). There were eight quality criteria: 1) participants met the diagnostic criteria for PTSD at baseline; 2) study used a treatment manual; 3) clinicians who delivered the intervention were trained; 4) treatment integrity was checked; 5) intention-to-treat analyses were conducted; 6) study had a minimal level of statistical power to find significant effects of the intervention and included at least 50 participants; 7) randomisation was conducted independently; and 8) assessors of outcome were blinded. Each criterion was coded with '1' if it was fulfilled or '0' if it was not met or reported. Studies were assigned a quality score between zero and eight, with higher scores reflecting higher study quality. The overall agreement for total quality scores was good (ICC(2,1) = 0.854, 95% CI [0.641, 0.946]) (Shrout & Fleiss, 1979).

2.7 Data analysis

Due to the substantial heterogeneity between studies (e.g., intervention, setting), we conducted a narrative synthesis to summarise the included studies. Study characteristics, like sample

size, country, and intervention type, were extracted. Reported moderators and mediators of treatment effectiveness were examined for patterns across studies.

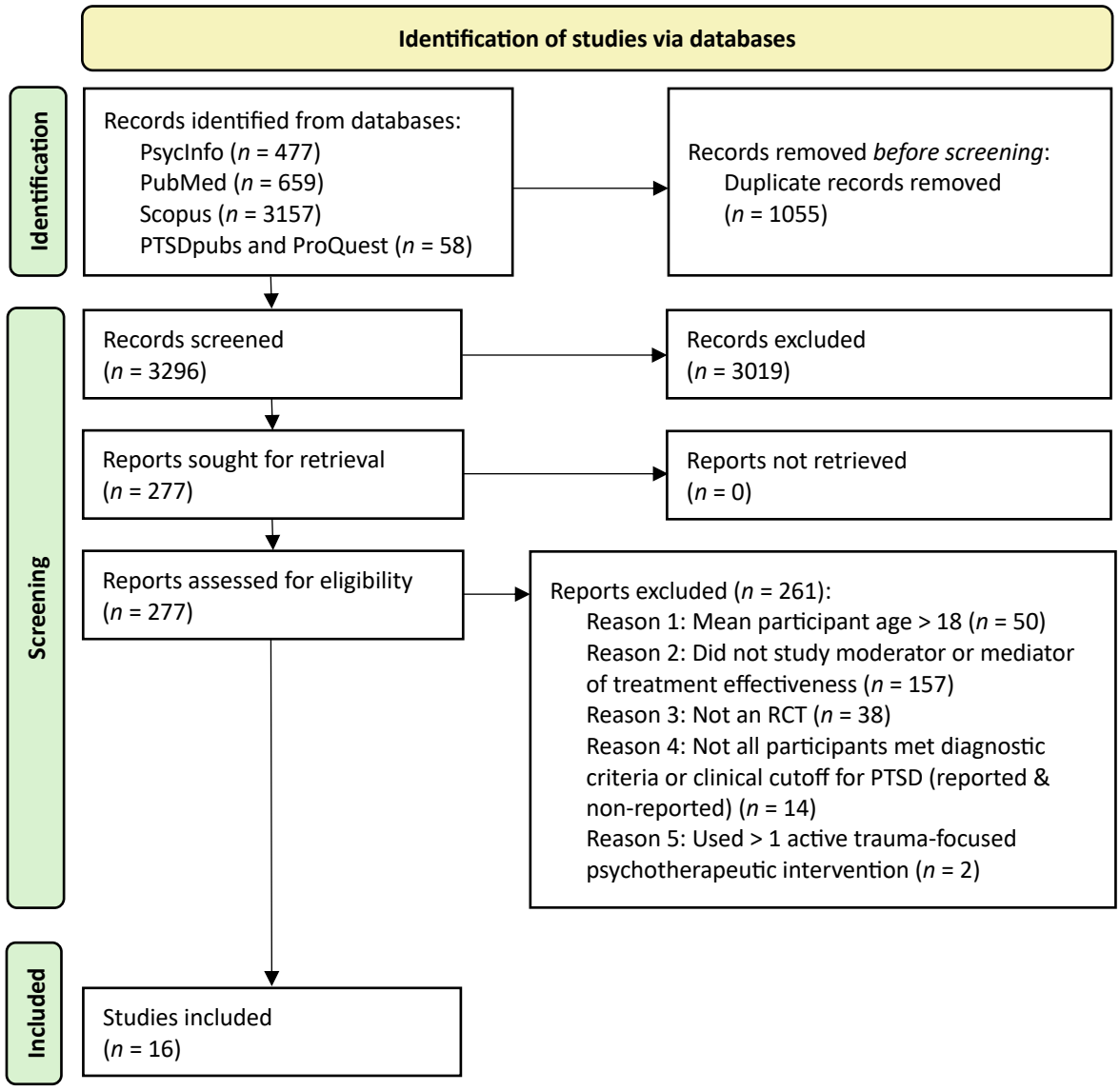
During data synthesis, it appeared that many identified moderators and mediators had been investigated in only a single study. Therefore, a post hoc decision was made to interpret only those moderators and mediators that had been examined in at least two studies. Factors investigated by a single study were reported descriptively (see Tables 2 and 3) but were not interpreted as having evidence of an effect, to minimise the risk of over-interpreting isolated findings.

3. Results

3.1 Study selection

The study selection process for the identification of studies is presented in Figure 1 in accordance with the PRISMA 2020 guidelines (Page et al., 2021).

Figure 1. *PRISMA Flowchart*



3.2 Study characteristics

Study characteristics are summarised in Table 1. The included 16 studies were published between 2004 and 2025. Study sample size ranged from 24 to 403. The mean sample age fell between 9.6 and 15.3. The studies came from China (*k* = 2), Germany (*k* = 2), Indonesia (*k* = 2), Israel (*k* = 1), Norway (*k* = 2), United Kingdom (UK) (*k* = 3), United States of America (USA) (*k* = 3), and Zambia (*k* = 1).

Psychotherapeutic interventions used in the studies predominantly adopted a cognitive-behavioural approach (*k* = 15) (i.e. TF-CBT (*k* = 9); classroom-based intervention (*k* = 2); cognitive therapy for PTSD (*k* = 2); prolonged exposure therapy for adolescents (*k* = 1); Power up Children's Psychological Immunity (*k* = 1). One exception was group intervention based on expressive-supportive model (*k* = 1).

Further details on the outcome measures for post-traumatic stress, study population, and whether a main treatment effect was found significant can be found in Appendix B.

3.3 Study quality

The quality of trials was high for 11 studies (sum score ≥ 7 out of 8), and the mean quality score was 6.50 (*SD* = 0.89). Total quality ratings are reported in Table 1, and quality ratings for each item per study are reported in Appendix C.

Table 1. *Study Characteristics*

Author (Year)	Country	Sample size	Mean age (SD)	% female	Study groups	Moderators studied	Mediators studied	Quality score
Capaldi et al. (2016)	USA	61	15.3 (1.5)	100	PE-A; CCT	Therapeutic alliance		5
Cohen et al. (2004)	USA	203	10.8 (NR)	78.8	TF-CBT; CCT	Age; Gender; Race; Ethnicity; Location (site); Number of sessions		6
Cohen et al. (2011)	USA	124	9.6 (2.5)	50.8	TF-CBT; CCT	Age; Gender; Race; Therapist		7
Holt et al. (2014)	Norway	135	14.8 (2.2)	80	TF-CBT; TAU		Parental emotional reactions to child's traumatic experiences; Parental depressive symptoms	7
Jensen et al. (2018)	Norway	156	15.1 (2.2)	79.5	TF-CBT; TAU		Cognitive appraisals	7
Kane et al. (2016)	Zambia	257	TF-CBT: 14.0 (2.8); TAU: 13.3 (3.0)	49.8	TF-CBT; TAU	Age; Gender; Orphan status; Current school status; Number of trauma types experienced; Caregiver participation in TF-CBT; Primary caregiver; Experience of sexual abuse		7
Li et al. (2023; 2024)	China	234	10.4 (0.9)	41.5	TF-CBT; TAU	Child baseline PTSD severity	Maladaptive emotion regulation strategies; Trauma memory quality; Resilience; Adaptive emotion regulation strategies	7
Meiser-Stedman et al. (2017)	UK	29	13.3 (2.5)	72.4	CT-PTSD; WL		Trauma-related misappraisals; Memory quality; Rumination; Safety-seeking behaviours	7
Pfeiffer et al. (2017)	Germany	123	13.0 (2.8)	73.2	TF-CBT; WL		Maladaptive post-traumatic cognitions	6

Shechtman & Mor (2010)	Israel	164	NR; 79 5th & 6th grade, 85 7th & 9th grade	NR	Group intervention; WL	Age; Gender; Type of trauma	4
Smith et al. (2007)	UK	24	13.9 (2.8)	50	TF-CBT; WL	Maladaptive post-traumatic cognitions	7
Smith et al. (2025)	UK	31	NR; Median: 15; IQR: (15, 16)	87.1	iCT-PTSD-YP; WL	Post-traumatic cognitions; Memory quality; Rumination	7
Tol et al. (2008; 2010)	Indonesia	403	9.9 (1.2)	48.6	CBI; WL	Age; Gender; Exposure to traumatic events; Family connectedness; Displacement status; Household size; Social support	Hope; Positive coping; Negative coping; Peer social support; Emotional social support; Play social support	2008: 7; 2010: 6
Tutus et al. (2019)	Germany	113	13.1 (2.8)	73.5	TF-CBT; WL		Parental maladaptive post-traumatic cognitions	7

Note. NR = not reported; WL = wait list; PE-A = prolonged exposure therapy for adolescents; CCT = client-centered therapy; TF-CBT = trauma-focused cognitive-behavioural therapy; TAU = treatment as usual; CT-PTSD = cognitive therapy for PTSD; iCT-PTSD-YP = internet-delivered cognitive therapy for PTSD in young people; CBI = classroom-based intervention.

3.4 Moderators

Moderators studied (*n* = 22) are summarised in Table 2. They were grouped into three categories: 1) individual factors (*n* = 15); 2) family factors (*n* = 4); and 3) treatment factors (*n* = 3). To caution against over-interpretation, only factors that were examined by at least two studies were interpreted for its potential effect (i.e. three out of 22 moderators).

Relatively well-studied (*k* = 6 each), age and gender were largely not significant moderators of treatment outcomes. In the two studies where gender emerged as a significant moderator, girls demonstrated greater treatment gains. Race, examined in two studies, was not found to significantly moderate outcomes.

Table 2. *Moderators studied*

Factor	Author (Year)	Finding (in the direction of greater treatment effects)
Individual factors		
Age	Cohen et al. (2004)	ns
	Kane et al. (2016)	ns
	Shechtman & Mor (2010)	ns
	Tol et al. (2008)	ns
	Tol et al. (2010)	ns
	Cohen et al. (2011)	Not reported
Gender	Tol et al. (2008)	* Girls
	Tol et al. (2010)	* Girls
	Cohen et al. (2004)	ns
	Kane et al. (2016)	ns
	Shechtman & Mor (2010)	ns
	Cohen et al. (2011)	Not reported
Race	Cohen et al. (2004)	ns
	Cohen et al. (2011)	ns
Ethnicity	Cohen et al. (2004)	ns
Education status	Kane et al. (2016)	In school – ns
Site	Cohen et al. (2004)	ns
Displacement status	Tol et al. (2010)	ns
Orphan status	Kane et al. (2016)	ns
Social support	Tol et al. (2010)	ns
Baseline PTSD	Li et al. (2023)	* Higher severity level
Exposure to traumatic events	Tol et al. (2010)	ns
History of sexual abuse	Kane et al. (2016)	*
Type of trauma	Shechtman & Mor (2010)	ns
Number of trauma types experienced	Kane et al. (2016)	ns

Family factors		
Caregiver's participation in treatment	Kane et al. (2016)	ns
Primary caregiver	Kane et al. (2016)	* Had mother as primary caregiver, followed by father, and someone else or "no one"
Family connectedness	Tol et al. (2010)	ns
Household size	Tol et al. (2010)	ns
Treatment factors		
Therapeutic alliance	Capaldi et al. (2016)	ns
Number of sessions	Cohen et al. (2004)	ns
Therapist	Cohen et al. (2011)	Not reported

Note. * indicates a significant moderating effect; ns indicates a non-significant moderating effect.

3.5 Mediators

Mediators studied (*n* = 16) are summarised in Table 3. The mediators reviewed were grouped into two categories: 1) individual factors (*n* = 13); and 2) family factors (*n* = 3). Only factors that were examined by at least two studies were interpreted for its potential effect (i.e. three out of 16 mediators).

The most studied mediator was post-traumatic cognitive appraisal (*k* = 5), followed by quality of traumatic memories (*k* = 3), rumination (*k* = 2). Of the five papers examining post-traumatic cognitive appraisal, four reported significant mediating effects. For quality of traumatic memories, two of the three studies found it to be a significant mediator. Evidence regarding rumination was mixed: one study did not observe a mediating effect, whereas the other found that changes in rumination from pre- to post-treatment, but not from pre- to mid-treatment, significantly mediated treatment effects.

Table 3. *Mediators studied*

Factor	Author (Year)	Finding
Individual factors		
Post-traumatic cognitive appraisal	Jensen et al. (2018)	Overall change * Pre–mid changes – ns Mid–post changes *
	Meiser-Stedman et al. (2017)	Pre–post changes * Pre–mid changes *
	Pfeiffer et al. (2017)	*
	Smith et al. (2007)	*

	Smith et al. (2025)	ns
Quality of traumatic memories	Li et al. (2024)	*
	Meiser-Stedman et al. (2017)	Pre–post changes * Pre–mid changes – ns
	Smith et al. (2025)	ns
Rumination	Meiser-Stedman et al. (2017)	Pre–post changes * Pre–mid changes – ns
	Smith et al. (2025)	ns
Safety-seeking behaviours	Meiser-Stedman et al. (2017)	Pre–post changes * Pre–mid changes *
Resilience	Li et al. (2024)	*
Maladaptive emotion regulation strategies	Li et al. (2024)	*
Adaptive emotion regulation strategies	Li et al. (2024)	ns
Hope	Tol et al. (2010)	ns
Positive coping	Tol et al. (2010)	ns
Negative coping	Tol et al. (2010)	ns
Peer social support	Tol et al. (2010)	ns
Emotional social support	Tol et al. (2010)	ns
Play social support	Tol et al. (2010) [#]	*
Family factors		
Parental depression	Holt et al. (2014)	ns
Parent emotional reaction	Holt et al. (2014)	ns
Parental dysfunctional post-traumatic cognitions	Tutus et al. (2019)	Child's PTSS assessed using caregiver report * Child's PTSS assessed using interview and self-report – ns

Note. * indicates a significant mediating effect; ns indicates a non-significant mediating effect; [#] indicates that the effect was in an unexpected direction; PTSS = post-traumatic stress symptoms.

4. Discussion

This systematic review looked at the moderators and mediators of child PTSD treatment outcome. The search strategy resulted in 3296 articles of which 16 were included in the review. Although this review adopted broad eligibility criteria encompassing any trauma-focused psychotherapeutic intervention in an attempt to address methodological gaps in previous work, the resulting evidence base was highly homogeneous, with 15 of 16 studies employing cognitive-behavioural approaches. Only one study adopted a non-cognitive-behavioural intervention (i.e. expressive-supportive group therapy; Shechtman & Mor, 2010). This pattern reflects the current state of the literature, in which cognitive-behavioural interventions dominate the evidence base for

paediatric PTSD. Findings of this review, as a result, primarily pertain to cognitive-behavioural trauma-focused interventions.

Across the 16 studies, 22 moderators and 16 mediators were analysed. A large proportion of the identified moderating and mediating factors were studied by only a single study, limiting the extent to which consistent conclusions could be drawn. Consequently, the discussion focuses on the three moderators and three mediators that were examined in at least two studies to reduce the risk of over-interpreting findings reported in only one study.

4.1 Treatment moderators

Treatment effects were generally consistent across demographic groups: age, gender, and race. Age and gender, each examined in six studies, largely did not moderate treatment outcomes, suggesting that interventions were broadly effective across developmental stages and between boys and girls. This is in line with de Haan et al.'s (2024) review findings. Notably, in the two studies where gender emerged as a significant moderator, girls demonstrated greater treatment gains, indicating the potential for gender-specific responsiveness in certain contexts. Both studies (i.e. Tol et al., 2008; 2010) integrated cognitive behavioural therapy (CBT) techniques with expressive exercises, such as drama, dance and music, distinguishing them from the other four studies, which employed therapeutic approaches like writing, drawing, or game-based activities. Race, examined in two studies, was not observed to significantly influence treatment outcomes, although the small number of studies limits the ability to draw definitive conclusions.

Several variables were each examined and found to be significant in only one study; greater treatment effects were reported for children with more severe baseline PTSD (Li et al., 2023), a history of sexual abuse (Kane et al., 2016), and those whose primary caregiver was their mother, followed by father (Kane et al., 2016).

The robustness of the moderator findings was considered in relation to Shechtman & Mor (2010), the only study utilising a non-cognitive-behavioural intervention, which reported non-significant moderating effects for age, gender, and type of trauma. Its inclusion did not meaningfully

influence the overall pattern of findings for age and gender, which were largely reported as non-significant treatment moderators across multiple cognitive-behavioural trials. The moderating effect of type of trauma was assessed only in this study and was therefore not interpreted in line with the review's approach of limiting interpretation to factors examined in multiple studies.

4.2 Treatment mediators

Investigated in five studies, post-traumatic cognitive appraisal emerged as the most frequently examined mediator. It was found to significantly mediate treatment effects in four of those studies (i.e. Jensen et al., 2018; Meiser-Stedman et al., 2017; Pfeiffer et al., 2017; Smith et al., 2007). This pattern aligns with cognitive theories of PTSD (Dalgleish, 2004; Ehlers & Clark, 2000), which posit that maladaptive trauma-related cognitions maintain post-traumatic symptoms and symptom reduction occurs through changes in the dysfunctional cognitions.

Quality of traumatic memories was identified as a significant mediator in two of the three studies that examined it (i.e. Li et al., 2024; Meiser-Stedman et al., 2017). The role of trauma memory quality as a mechanism of change is supported by the dual representation theory (Brewin & Burgess, 2014). Trauma memories typically involve strong sensory representations that are weakly associated with contextual details, which can trigger involuntary uncontextualised fearful responses. By strengthening the association between sensory and contextual representations (i.e. improving trauma memory quality), trauma symptoms like intrusions and heightened arousal can be alleviated.

Rumination was examined as a potential mediator in two studies. One study found that changes in rumination from pre- to post-treatment, but not pre- to mid-treatment, significantly mediated treatment effects (i.e. Meiser-Stedman et al., 2017) while the other study did not detect a significant mediating effect. Conceptualised as a dysfunctional cognitive strategy, rumination involves dwelling on the traumatic event without processing the traumatic memory and its meaning (Ehlers & Clark, 2000; Ehlers & Steil, 1995; Michael et al., 2007). It is thought to maintain PTSD by triggering intrusions and negative emotions while interfering with emotional processing and cognitive restructuring of the trauma (Birrer & Michael, 2011; Michael et al., 2007; Moulds et al., 2020).

It is noteworthy that the study which did not find significant mediation effects for any of the three putative treatment mediators (i.e. post-traumatic cognitive appraisal, trauma memory quality, and rumination) was an early-phase trial (i.e. Smith et al., 2025). The trial focused on evaluating the feasibility, acceptability, and clinical efficacy of an online cognitive therapy programme for young people with PTSD. Given that the mediation analyses were conducted on an exploratory basis, the analyses might have been underpowered to detect indirect (i.e. mediation) effects.

Each examined in only one study, the following variables were identified as significant treatment mediators: safety-seeking behaviours (Meiser-Stedman et al., 2017), resilience (Li et al., 2024), maladaptive emotion regulation strategies (Li et al., 2024), play social support (Tol et al., 2010), and parental dysfunctional post-traumatic cognitions (Tutus et al., 2019). Of note, parental dysfunctional post-traumatic cognitions significantly mediated treatment effects only when children's post-traumatic stress symptoms were assessed via caregiver report, and not when measured by clinical interview or child self-report.

4.3 Strengths and limitations

This review offered an updated consolidation of factors that influence treatment effectiveness (i.e. treatment moderators) and mechanisms through which the treatment is effective (i.e. treatment mediators). It sought to address methodological limitations identified in previous meta-analyses and systematic reviews by including any trauma-focused psychotherapeutic intervention while applying stricter eligibility criteria (e.g., studies were required to be RCTs) to enhance the rigour of the evidence. Following study protocol registration, the eligibility criteria were refined to improve the internal validity and interpretability of the review. Specifically, included studies were restricted to participants who met diagnostic criteria or validated clinical cutoff for PTSD, thereby reducing clinical heterogeneity and improving the relevance of findings to young people with clinically significant PTSD. Additionally, studies were limited to RCTs evaluating a single trauma-focused psychotherapeutic intervention against a non-trauma-focused intervention (e.g., wait list, treatment as usual). This refinement was made to improve the interpretability of moderator and mediator findings by allowing treatment effects

to be more confidently attributed to the intervention under investigation rather than to differences between multiple active trauma-focused interventions. The review followed recommended practice of PROSPERO registration and PRISMA guidance. An extensive search was undertaken with broad search terms and wide date range, and an independent rater was used to confirm the reliability of the screening process.

A post hoc methodological decision was made during data synthesis to interpret only moderators and mediators examined in at least two studies, as most identified factors had been investigated in only a single study. This approach was adopted to minimise the risk of over-interpreting isolated findings and to focus the synthesis on factors with some degree of replication.

In assessing the study quality, one of the criteria was that participants were to have met the diagnostic criteria of PTSD. Given the challenges in diagnosing PTSD in the paediatric population (Cohen & Scheeringa, 2009), it could be developmentally inappropriate to apply such a rigid criterion for the purpose of this review. Developmentally sensitive approaches such as the alternative algorithm for PTSD diagnosis in young children (PTSD-AA; Scheeringa et al., 2003) and the use of a CPSS cutoff score of 15 (Foa et al., 2001; Kassam-Adams et al., 2015) could be considered as an adaptation to the standard study quality assessment criterion.

Included studies were assessed for their methodological quality using the checklist proposed by Cuijpers et al. (2010) rather than their risk of bias, for example, using the Cochrane Risk of Bias 2 (RoB 2) tool (Sterne et al., 2019). While the Cuijpers checklist overlaps with RoB 2 in assessing methodological procedures such as randomisation, allocation concealment, and intention-to-treat analyses, it does not evaluate some bias domains included in RoB 2 (e.g., bias in outcome measurement, selective reporting). Conversely, it includes psychotherapy-specific indicators of methodological quality not considered by RoB 2, such as therapist training, use of treatment manuals, and treatment integrity.

The generalisability of these findings requires consideration. Although the review included studies from a range of countries, most were conducted in Western settings, with relatively few from

China, Indonesia, Israel, and Zambia. Moreover, these non-Western studies represent diverse sociocultural contexts rather than a single cultural group, limiting cross-cultural applicability of findings. Half of the studies included predominantly female participants while the rest reported more balanced gender distributions. As culture and gender may influence trauma experiences, trauma symptom expression, and engagement with psychological interventions (Shrestha et al., 2019; Hinton & Lewis-Fernández, 2011; Ascienzo et al., 2024), these sample characteristics should be considered when interpreting the findings.

4.4 Future research

Future RCTs could focus on examining treatment moderators and mediators for specific interventions. It was noted in this review that existing moderation and mediation analyses were conducted over a diverse range of interventions. This limits our ability to conclude the effects of the studied moderators and mediators as they may have a significant effect for certain interventions but not for some. Given the inconsistent results on the moderating effect of gender, a meta-analysis could be helpful in teasing out the effect of gender that might otherwise go undetected within trials.

Currently, as most factors were studied by only a single study, conclusions regarding their influence remain preliminary. This review identified several understudied variables that warrant further investigation. It could be helpful for future trials to adopt a theory-driven approach when investigating treatment moderators and mediators. This would minimise risks associated with multiple testing and strengthen the evidence base for each proposed factor.

4.5 Clinical implications

Given that a couple of studies, out of six, found that girls benefit more than boys from treatment, it could be worthwhile monitoring treatment gains across genders under clinical services. Nevertheless, evidence broadly suggests that the trauma-focused interventions could be beneficial across diverse child and adolescent populations.

Post-traumatic cognitive appraisal and trauma memory quality received promising support for their mediating effects on treatment outcomes. These findings underscore the importance of

addressing maladaptive trauma-related cognitions and facilitating the integration of sensory and contextual details of trauma memories into a coherent narrative (e.g., through imaginal reliving and cognitive restructuring).

The role of rumination as a mechanism of change is less clear. The sparse and mixed evidence regarding its impact on treatment outcomes nonetheless points to its potential as a treatment mechanism. It could hence be helpful for interventions to target this maladaptive cognitive strategy (e.g., through behavioural activation and cognitive restructuring). However, given the limited evidence, clinicians should be cautious in assuming that targeting rumination alone will drive symptom reduction in this population.

4.6 Conclusion

This systematic review consolidated the moderators and mediators of trauma-focused psychotherapies for children and adolescents. It included 16 studies which analysed 22 moderators and 16 mediators in total. Of the 16 included studies, 15 employed cognitive-behavioural approaches. A large proportion of the factors were studied by only a single study. To prevent over-interpretation of findings, only factors that were studied by at least two trials were examined (i.e. three moderators and three mediators). Findings suggested that age, gender, and race did not significantly moderate the treatment effects, with limited evidence indicating a differential response based on gender. On the other hand, there is support for the mediating effects of post-traumatic cognitive appraisals, quality of traumatic memories, and rumination on treatment outcomes.

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Chapter Three: Bridging Chapter

Summary of the systematic review

The systematic review in the previous chapter examined the current evidence on moderators and mediators of trauma-focused treatment outcomes for children and adolescents. Overall, the review found little to no evidence to support demographic characteristics like age, gender, and race as moderators of treatment effectiveness. On the other hand, several cognitive processes, including post-traumatic cognitive appraisal, trauma memory quality, and rumination, emerged as potential mediators of treatment outcomes. These findings reinforce cognitive models of PTSD (Dalgleish, 2004; Ehlers & Clark, 2000) and highlight the importance of targeting cognitive processes during intervention.

Limited research into parental factors

The review also identified an important gap within the literature regarding parental factors as potential moderators and mediators of treatment outcomes. Fewer than a handful of studies examined parental influences (i.e. Kane et al., 2016; Holt et al., 2014; Tutus et al., 2019), with research limited to factors such as caregiver participation in treatment, parental depression, parent emotional reaction, and parental dysfunctional post-traumatic cognitions. The small number of studies and the diversity of parental factors investigated precluded firm conclusions regarding their influence on child's treatment outcomes.

The empirical study

The empirical study presented in the following chapter builds on the findings of the systematic review by addressing this gap in the literature. It examines the association between parental psychopathology and child's pre-treatment trauma symptom severity, and investigates whether parental depression, anxiety, and PTSD moderate child's response to cognitive therapy for PTSD (CT-PTSD), a specific trauma-focused intervention. The study aims to clarify the role of parental psychopathology in child's trauma treatment outcomes and inform more targeted, evidence-based interventions for children and adolescents.

Chapter Four: Empirical Research Paper

Prepared for Submission to Journal of Anxiety Disorders

(Author guidelines available in Appendix A)

Relationship between Parental Psychopathology and Young People's Trauma Symptomatology and Treatment: Correlational and Moderation Analyses

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Abstract

Background. Parents play an important role in child's trauma recovery. They influence the recovery outcomes through modelling coping strategies, shaping appraisals of events, and the quality of parent-child interactions. Further, parental psychopathology appears to be a risk factor for child's trauma symptoms and influence their response to trauma treatment.

Aims. This study aimed to examine the relationship between parental and child symptomatology pre-treatment, and clarify the moderating effects of parental psychopathology on child's trauma treatment outcome.

Methods. This study was a secondary analysis of the DECRYPT randomised controlled trial. The sample comprises 111 young people aged 8–17 years old (mean age = 14.4 years; *SD* = 2.5) with clinically relevant post-traumatic stress disorder (PTSD). Participants were randomised to either intervention group (cognitive therapy for PTSD; CT-PTSD) or treatment-as-usual (TAU) group. Participants' symptomatology was assessed using Child Revised Impact of Event Scale, Revised Child Anxiety and Depression Scale, and Children's Global Assessment Scale. Parent psychopathology was assessed using the Patient Health Questionnaire, Generalised Anxiety Disorder Assessment, and Post-traumatic Stress Diagnostic Scale.

Results. Parental baseline psychopathology was weakly associated with aspects of child's baseline symptomatology, including PTSD severity, depression, separation anxiety, and overall functioning. Parental depression moderated treatment effectiveness post-treatment; CT-PTSD yielded modestly greater benefits relative to TAU at higher levels of parental depression. This moderation effect was absent at follow-up, and for parental anxiety and PTSD.

Conclusions. The moderation findings tentatively suggested that CT-PTSD can be effective for children regardless of the severity of their parents' mental health needs.

Keywords: moderator, parental psychopathology, PTSD, child, CT-PTSD, depression, anxiety

1. Introduction

1.1 Prevalence and impact of trauma on young people

While natural recovery happens post-trauma, a significant minority of young people – around 25% of them – go on to develop post-traumatic stress disorder (PTSD) (Lewis et al., 2019). Young people exposed to traumatic events experience a higher risk of developing mental health disorders (Kilpatrick et al., 2003; Copeland et al., 2007), self-harm and suicide attempts (Rojas et al., 2017; Nock et al., 2013; Cutajar et al., 2010), and having physical health problems (e.g., Mock & Arai, 2011; Kascakova et al., 2020; Almuneef et al., 2014), relative to their non-trauma-exposed peers.

1.2 Parental influence on child's trauma recovery

Parents play a crucial role in young people's recovery from traumatic experiences. Cognitive appraisals, coping styles and adjustment to stress are often transmitted from parents to children through modelling and social learning (Schwartz et al., 1994). Parent-child interactions influence the way in which the young person remembers and appraises events (Williamson et al., 2016), and maladaptive coping displayed by parents are picked up by children (Marsac et al., 2014; Nugent et al., 2007). For instance, children were more likely to use distraction to cope when their parents used that as a coping strategy (Marsac et al., 2014). Further, parental psychological well-being impacts on their ability to parent which increases the risk for poorer child outcomes (Hiller et al., 2018; Lambert et al., 2014; Schwerdtfeger & Geoff, 2007). Parental PTSD is related to greater parenting stress (Christie et al., 2019), poorer relationship with their child (Christie et al., 2019), and lower parental functioning (e.g., involvement in raising the child, meeting the physical and emotional needs of the child) (Solomon et al., 2011). PTSD symptoms in parents were also associated with more coercive interactions and less positive engagement with their children, which were linked to the children's internalising and externalising behaviours (Snyder et al., 2016). Parent depression was found to be associated with parenting stress (McDonald et al., 2011) and neglect of child (Lee et al., 2012; Chemtob et al., 2013). Understandably, trauma treatments with parental involvement showed better outcomes than those

that did not involve parents (Gutermann et al., 2016; Lenz & Hollenbaugh, 2015; Harvey & Taylor, 2010; Corcoran & Pillai, 2008).

1.3 Relationship between parental psychopathology and child's trauma symptomatology

Parental psychopathology has largely been found to be negatively associated with children's trauma symptoms (e.g., Weems & Scheeringa, 2013). Trickey et al. (2012) reported in their meta-analysis a small to medium-sized effect for parental psychopathology as a risk factor for the development of PTSD in youths. Children have more serious post-traumatic stress (PTS) symptoms when their caregivers suffer from more severe PTS symptoms and depression (Morris et al., 2012; Scheeringa et al., 2015). A meta-analysis by Lambert et al. (2014) revealed a moderate correlation between parents' PTSD symptom severity and children's psychological distress and behavioural problems. Thakar et al. (2013) found that after controlling for child's trauma history, maternal depression still accounted for variance in children's behavioural and emotional adjustment following trauma exposure. In a clinical sample of children with PTSD, around 50% of the parents presented with at least moderate PTS symptoms while one third of them reported clinically significant depressive symptoms (Tutus & Goldbeck, 2016). Parents with depressive and PTS symptoms tended to have dysfunctional thoughts related to their child's traumatic experiences, and the authors hypothesised that they could hinder the child's recovery from PTSD.

1.4 Impact of parental psychopathology on child's treatment outcome

Parental psychopathology appears to influence child's treatment outcomes. Nixon et al. (2012) found that children's PTSD severity post-treatment was moderated by maternal depression. When maternal depression is high, children with more severe PTSD symptoms are likely to still have those symptoms beyond clinical threshold post-treatment and at 6-month follow-up. Maternal PTS symptoms, on the other hand, was not found to moderate treatment outcome (Nixon et al., 2012; Weems & Scheeringa, 2013). Likewise, Martin et al. (2019) reported that more severe caregiver depression, but not caregiver PTSD or emotional distress related to the child's trauma, was associated with worse child treatment outcomes. More severe maternal depression also seemed to be linked to

potential PTSD symptom relapse in children (Weems & Scheeringa, 2013). A possible explanation for this preliminary observation where parental depression, but not parent PTS symptoms, impacts child's response to trauma treatment could be that parental depression is more reflective of a general impaired functioning which may affect parental engagement in the child' treatment and interfere with parental ability to offer support for their children (Nixon et al., 2012).

On the other hand, a randomised controlled trial by de Roos et al. (2021) demonstrated that the child's PTSD treatment outcome was significantly moderated by the severity of the parental PTSD, depression, anxiety, and overall psychopathology. Parental psychopathology, an aggregate for parental post-traumatic stress, anxiety and depression symptoms, was also found to be a significant treatment moderator by Overbeek et al. (2014).

1.5 Research gaps

Current literature highlights the need to consider parental adjustment when treating trauma symptoms in young people. While there is broad consensus that parental psychopathology contributes to children's PTSD treatment outcome (e.g., Scheeringa et al., 2015; Trickey et al., 2012), evidence on whether parental PTSD symptoms affect children's treatment response appears mixed (de Roos et al., 2021; Nixon et al., 2012). Further, the moderating influence of parental anxiety on treatment response seems to only have been investigated by de Roos et al. (2021). As Danzi and La Greca (2021) suggested, it is important to understand treatment moderators as they will guide clinicians' selection of treatment for young people based on client characteristics and environmental factors.

Using a sample of children and young people who have significant trauma symptoms (i.e. met the PTSD diagnosis, exposed to multiple traumas), the present study sought to provide more clarity on the relationship parents'[1] psychopathology has with children trauma symptomatology and recovery to optimise trauma recovery of young people. Firstly, we aimed to establish the association between parental psychopathology and child symptomatology pre-treatment. We then studied how parental

[1] In this paper, *parents* referred to parents and caregivers of the young person.

psychopathology affects child's trauma treatment outcome in terms of moderation effects. The moderating effects of parental psychopathology on treatment outcome were examined through two conceptualisations as studied in existing literature: (i) as a moderator of the relationship between child baseline and post-treatment PTSD (e.g., Nixon et al., 2012; Weems & Scheeringa, 2013), and (ii) as a moderator of the relationship between treatment condition and child's post-treatment PTSD (i.e. treatment effectiveness) (e.g., de Roos et al., 2021).

We hypothesised that:

H1. Parental baseline psychopathology (i.e. depression, anxiety, and PTSD) will be positively associated with the child's symptomatology before treatment.

H2a. Parental baseline psychopathology (i.e. depression, anxiety, and PTSD) will moderate the relationship between the child's pre-treatment and post-treatment PTSD symptomatology (post-treatment and 11 months post-randomisation).

H2b. Parental baseline psychopathology (i.e. depression, anxiety, and PTSD) will moderate the relationship between the treatment condition (i.e. CT-PTSD vs. TAU) and the child's post-treatment PTSD symptomatology (post-treatment and 11 months post-randomisation).

2. Method

2.1 Design

This study was a secondary quantitative analysis of the data collected for the Delivery of Cognitive Therapy for Young People after Trauma (DECRYPT) trial by Allen et al. (2021). It was a two-arm, patient-level, single blind, superiority randomised controlled trial that aimed to compare the effectiveness of cognitive therapy for PTSD (CT-PTSD) and treatment-as-usual (TAU) in treating PTSD in young people with multiple trauma exposure. The DECRYPT trial was approved by a UK Health Research Authority Research Ethics Committee (16/EE/0233). Full details of the trial methods (e.g., procedure, treatment adherence, randomization, blinding protocols) can be found in Meiser-Stedman et al. (2025).

2.2 Participants

Participants were young people aged 8–17 years old recruited from National Health Service (NHS) child and adolescent and youth mental health services in England and Wales.

The inclusion criteria for the study were: (1) meeting the criteria for a diagnosis of PTSD, as defined by Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), scoring 17 and above on the Child Revised Impact of Events Scale (8-item version; CRIES-8; Perrin et al., 2005); and (2) exposure to multiple traumatic stressors (e.g., domestic violence, sexual abuse, accident, witness of violence).

Exclusion criteria for the study comprises: (1) change of prescribed psychiatric medication within the previous two months; (2) pervasive developmental disorder or neurodevelopmental disorder (e.g., autism, but not attention deficit hyperactivity disorder); (3) intellectual disability; (4) another primary psychiatric diagnosis or clinical need that warrants treatment ahead of PTSD (e.g., psychosis, suicidal behaviour, conduct disorder); (5) inability to speak English; (6) ongoing exposure to threat (e.g., living with an abuser; regularly placing self in danger) or safeguarding issues; (7) strong likelihood of being unable to complete treatment (e.g., imminent house or foster placement move); or (8) history of organic brain damage (Allen et al., 2021).

2.3 Procedure

Participants were referred to the DECRYPT trial team by clinicians in participating NHS Trusts teams. They were provided with the study information and 48 hours to decide whether to participate. A baseline assessment was conducted for consenting families to determine if they meet the study eligibility criteria. For participants under age 16, their parents provided informed consent while the child or young person provided their assent. Participants above age 16 provided their own informed consent.

Through a web-based randomisation service, participants were randomised to either CT-PTSD group or TAU group, stratified by CRIES-8 score (17–28 vs. 29-–40) and recruitment site (i.e. NHS Trust).

Participants completed a variety of outcome measures at five time points: baseline, mid-treatment, post-treatment, 11-month post-randomisation (approximately 6-month follow-up) and 29-month post-randomisation (approximately 2-year follow-up). Parents completed questionnaires about their child and their mental health at baseline, post-treatment, and 11-month post-randomisation. Participants and their parents were remunerated fairly for completing the questionnaires. Post-treatment and follow-up data was collected by trained assessors who were not involved in the trial and were blind to the group allocation.

2.4 Interventions

2.4.1 CT-PTSD

Based on the cognitive model of PTSD (Ehlers & Clark, 2000), CT-PTSD is a structured and manualised psychological intervention adapted for children and adolescents. Its efficacy in children and young people has been largely supported (Meiser-Stedman et al., 2017, 2025).

Up to 15 sessions, lasting 60–90 minutes each, were offered. A maximum of three sessions were allowed for managing other comorbid conditions and difficulties (e.g., depression, self-harm).

Key components of CT-PTSD treatment include: (1) psychoeducation (e.g., how the appraisal of event maintains PTSD symptoms), (2) narrative work and imaginal reliving to identify worst moments and help establish a coherent trauma narrative, (3) cognitive restructuring to modify threatening interpretations related to the trauma, and (4) coping management (e.g., address safety-seeking behaviours, drop rumination) (Smith et al., 2010).

Trial therapists delivering CT-PTSD have a professional qualification (e.g., nurse, occupational therapist, clinical psychologist) and completed a CT-PTSD training by the trial team. They were supervised by a clinical psychologist from the trial team. To assess treatment fidelity and quality of the CT-PTSD intervention, clinical psychologists experienced in the intervention rated the session recordings with a scale adapted from Allen et al. (2021).

2.4.2 TAU

For the TAU group, trial therapists were encouraged to provide any active treatment they considered necessary (e.g., supportive counselling, family therapy, medication, TF-CBT). Placement on a waiting list was not allowed for TAU. Trial therapists delivering TAU were not trained by the trial team in CT-PTSD and they received supervision according to their usual practice.

2.5 Measures

Being a secondary analysis of the DECRYPT trial, this study looked at a subset of measures for the research questions of interest. The primary clinical outcome was the child's PTSD severity which was measured using the Child Revised Impact of Event Scale (8-item version; Perrin et al., 2005). Child's baseline symptomatology was further assessed by Revised Child Anxiety and Depression Scale (Chorpita et al., 2000) and Children's Global Assessment Scale (Shaffer et al., 1983). Parental psychopathology was assessed using the Patient Health Questionnaire (Kroenke et al., 2001a), Generalised Anxiety Disorder Assessment (Spitzer et al., 2006), and Post-traumatic Stress Diagnostic Scale, DSM-5 version (Foa et al., 2016).

2.5.1 Child Revised Impact of Event Scale (8-item version; CRIES-8)

CRIES-8 is a screening tool designed to measure PTSD severity (Perrin et al., 2005). There are two subscales: Intrusion (e.g., think about the event when you don't mean to, have waves of strong feelings about it) and Avoidance (e.g., try to remove it from memory, stay away from reminders). The brief self-reported measure is suitable for use with young people aged eight to 18. Assessed to have good face and construct validity (Perrin et al., 2005), CRIES is endorsed by the UK Children and Young People's programme – Talking Therapies and is considered the most widely used screening tool for PTSD in children and young people (Leigh et al., 2016). It is pragmatic of the DECRYPT trial and this study to select this measure as the primary outcome measure since study findings can be made sense of with reference to clinicians' routine outcome measure.

2.5.2 Revised Child Anxiety and Depression Scale (47-item version; RCADS)

The RCADS (Chorpita et al., 2000) is a self-report questionnaire that assesses anxiety and depression symptoms in children and adolescents. It yields a depression score, a total anxiety score,

and anxiety subscale scores for separation anxiety disorder, social phobia, generalised anxiety disorder, panic disorder, obsessive-compulsive disorder. Commonly used in child population and clinical settings in the UK (Child Outcomes Research Consortium, 2014), the measure has shown favourable reliability, convergent and discriminant validity (Chorpita et al., 2000; Chorpita et al., 2005; Baron et al., 2021).

2.5.3 Children's Global Assessment Scale (CGAS)

An adaptation of the Global Assessment Scale for adults, the CGAS (Shaffer et al., 1983) is a clinician-rated measure of a child or adolescent's general functioning across psychological, social, and academic domains. Scores range from 1 to 100, with higher scores indicating better overall functioning. It has demonstrated discriminant validity, concurrent validity, inter-rater reliability, and test-retest reliability (Shaffer et al., 1983).

2.5.4 Patient Health Questionnaire (9-item version; PHQ-9)

The PHQ-9 is a brief self-report measure of depression severity in the past two weeks (Kroenke et al., 2001a). Total scores range from 0 to 27, with scores of 0–4 indicating no or minimal depressive symptoms, 5–9 mild depression, 10–14 moderate depression, 15–19 moderately severe depression, and 20–27 severe depression. Despite being a subset of the full PHQ (28–58 items, depending on number of items that are skipped), it achieved criterion validity, construct validity, and external validity (Kroenke et al., 2001b).

2.5.5 General Anxiety Disorder (7-item version; GAD-7)

The GAD-7 is a brief self-report measure of anxiety severity experienced over the past two weeks (Spitzer et al., 2006). Total scores range from 0 to 21, with scores of 0–4 indicating minimal anxiety, 5–9 mild anxiety, 10–14 moderate anxiety, and scores of 15 or greater indicating severe anxiety. Originally developed as a screening tool, it has demonstrated strong criterion validity, construct validity, internal consistency, and test-retest reliability (Spitzer et al., 2006).

2.5.6 Post-traumatic Stress Diagnostic Scale for DSM-5 (PDS-5)

The PDS-5 is a 24-item self-report measure of PTSD symptomatology and severity in the last month based on DSM-5 diagnostic criteria (Foa et al., 2016). The scale includes one item for each of

the 20 DSM-5 PTSD symptoms, with four additional items assessing trauma-related distress, functional interference, symptom onset, and symptom duration. Total scores range from 0 to 80. Scores of 1–10 indicate mild PTSD symptom severity, 11–20 moderate, 21–35 moderate to severe, and 36 or higher severe (Corran, n.d.). A total score of 28 or higher is often used to indicate probable PTSD (Foa et al., 2016). Demonstrating high internal consistency, test-retest reliability and discriminant validity, it is recognised as a valid and reliable measure of PTSD (Foa et al., 2016).

2.6 Data analysis

As recommended for research questions relating to treatment efficacy and mechanisms of efficacy (Silverman et al., 2024), the per-protocol (PP) approach was adopted for the moderation analyses which are the primary analyses in this paper (research question 2). Though research question 1 did not depend on treatment adherence, analyses were conducted in the PP sample to ensure consistency across research questions. To assess robustness of findings, corresponding intention-to-treat (ITT) analyses were conducted and reported in Appendices F and G. Nine participants from the TAU group were assessed to have received CT-PTSD or TF-CBT so they were removed from the PP analyses to avoid dilution of genuine moderating effects. All analyses were exploratory given that this study was a secondary analysis of the DECRYPT dataset. As part of the initial data analysis, descriptive statistics of each outcome variable were generated.

To account for possible missing data within a participant, participants who responded to a set minimum number of items on each outcome measure were included. For example, for CRIES-8, participants were included if they responded to at least seven out of the eight items. For these participants, the mean of the seven items was calculated and multiplied by eight to generate an average CRIES-8 score. See Appendix D for more details on the calculation of the outcome measure scores.

To test H1, correlational analyses were conducted to examine the univariate relationship between parental baseline psychopathology (i.e. depression, anxiety and PTSD scores) and child's baseline symptomatology (e.g., CRIES-8, RCADS, overall functioning).

To answer H2a and H2b, moderation analyses were conducted in R using the Lavaan package (Rosseel, 2012). Age and gender were included as covariates. The use of psychotropic medication was not controlled for. For moderation analyses associated with H2a (i.e. child's baseline CRIES-8 score as the independent variable), treatment condition was not controlled for given that the analyses aimed to study the moderating effects of parental baseline psychopathology regardless of treatment allocation. For moderation analyses associated with H2b (i.e. treatment condition as the independent variable), the child's baseline CRIES-8 score was controlled for as a covariate. After conducting Little's (1988) Missing Completely at Random (MCAR) test to assess if data was missing completely at random, full information maximum likelihood (FIML) was used to manage missing data. To reduce multicollinearity, continuous predictors (i.e. child baseline PTSD, parental depression, parental anxiety, parental PTSD) were mean-centered prior to the computation of interaction terms.

3. Results

3.1 Sample

The per-protocol sample comprised 111 young people aged 8–17 years old. The mean age was 14.4 years (*SD* = 2.5). 72.1% were female (*n* = 80). The mean CRIES-8 score at baseline was 31.6 (*SD* = 5.9), with 100% of the sample meeting the suggested clinical cutoff score of 17 and above (Perrin et al., 2005). More than half of the sample fell within the clinical range (T score > 70) on the depression and total anxiety subscales (see Appendix E for more details of the RCADS scores). The completion rate for the CRIES-8 questionnaire was 100% (*n* = 111) at baseline, 79.3% (*n* = 88) post-treatment, and 64.9% (*n* = 72) at 11-month post-randomisation. Participant flow and sample baseline characteristics (e.g., demographics, trauma history, use of psychotropic medication) are detailed in Meiser-Stedman et al. (2025).

For parents, the number of PHQ-9, GAD-7 and PDS-5 responses collected at baseline was 84, 85 and 86 respectively. A large proportion of the parents in this study experienced moderate to severe levels of depression, anxiety and PTSD (see Table 1).

Table 1. *Parent Baseline Psychopathology*

	Parental depression *n* (%)	Parental anxiety *n* (%)	Parental PTSD *n* (%)
None – mild	40 (47.6)	43 (50.6)	16 (18.6)
Moderate^	34 (40.5)	21 (24.7)	34 (39.5)
Severe	10 (11.9)	21 (24.7)	36 (41.9)

Note. ^ Moderate here covers Moderate and Moderately severe for PHQ-9; Moderate for GAD-7; Moderate and Moderate to Severe for PDS-5.

3.2 Baseline group comparisons

As reflected in Table 2, at baseline, the CT-PTSD and TAU groups were significantly different in terms of their age and gender. Participants in the CT-PTSD group were significantly more likely to be older and female. No between-group differences were found in participants' baseline symptomatology and parental psychopathology.

Table 2. *Group Comparisons at Baseline*

	Mean/Freq	SD/%	Mean/Freq	SD/%	Test statistic
Child					
	CT-PTSD (*n* = 58)		TAU (*n* = 53)		
Age (years)	14.8	2.1	13.8	2.9	$t_{109} = -2.12$*
Gender (female)	47	81.0	33	62.3	$\chi(1) = 4.85$*
PTSD (CRIES-8)	31.7	5.9	31.6	5.9	$t_{109} = -0.08$
RCADS (clinical range; T score ≥ 70)					
Total	32	55.2	34	51.5	$\chi(1) = 0.93$
Depression	41	70.7	40	75.5	$\chi(1) = 0.32$
Overall anxiety	30	51.7	28	52.8	$\chi(1) = 0.01$
Social phobia	17	29.3	16	30.2	$\chi(1) = 0.01$
Panic disorder	38	65.5	27	50.9	$\chi(1) = 2.42$
Separation anxiety	34	58.6	28	52.8	$\chi(1) = 0.38$
Generalised anxiety	20	34.5	17	32.1	$\chi(1) = 0.07$
Obsessive-compulsive	17	29.3	15	28.3	$\chi(1) = 0.01$
CGAS	48.0	10.8	51.2	10.7	$t_{109} = 1.58$
Parent					
	CT-PTSD (*n* = 43)		TAU (*n* = 41)		
Depression (PHQ-9)	10.9	7.4	10.2	7.7	$t_{82} = -0.40$
	CT-PTSD (*n* = 43)		TAU (*n* = 42)		
Anxiety (GAD-7)	9.9	6.4	9.6	6.5	$t_{83} = -0.24$
	CT-PTSD (*n* = 44)		TAU (*n* = 42)		

PTSD (PDS-5)	32.0	20.9	30.6	21.2	$t_{84} = -0.31$

Note. *p<0.05; **p<0.01; ***p<0.001.

3.3 Hypothesis 1: Parental baseline psychopathology will be positively associated with the child's symptomatology before treatment.

There was a significant positive correlation between parental depression and child baseline PTSD severity ($r = .25$, $n = 84$, $p = .023$) and depression ($r = .24$, $n = 84$, $p = .026$). Parental anxiety was correlated to child's depression ($r = .28$, $n = 85$, $p = .010$) and overall functioning ($r = -.25$, $n = 85$, $p = .020$). Parental PTSD was correlated with child's separation anxiety ($r = .24$, $n = 86$, $p = .028$) and overall functioning ($r = -.22$, $n = 86$, $p = .047$).

Table 3. *Correlation Matrix for Parent Psychopathology and Child Symptomatology*

	Parental depression (PHQ-9)	Parental anxiety (GAD-7)	Parental PTSD (PDS-5)
CRIES-8 (pre)	0.25*	0.18	0.14
CRIES-8 (post)	−0.04	0.08	0.08
CRIES-8 (11mo)	0.05	0.08	0.27
RCADS			
Depression	0.24*	0.28**	0.18
Overall anxiety	0.15	0.16	0.10
Social phobia	0.06	0.06	0.02
Panic disorder	0.09	0.08	−0.01
Separation anxiety	0.18	0.17	0.24*
Generalised anxiety	0.16	0.15	0.10
Obsessive-compulsive	0.10	0.20	0.12
CGAS	−0.17	−0.25*	−0.22*

Note. *p<0.05; **p<0.01; ***p<0.001; pre = pre-treatment; post = post-treatment; 11mo = 11-month post-randomisation.

3.4 Hypothesis 2a: Parental baseline psychopathology will moderate the relationship between the child's baseline and post-treatment PTSD symptomatology.

Little's (1988) MCAR test was non-significant, $\chi^2(37) = 46.4$, $p = .139$, indicating that data was missing completely at random. Therefore, FIML was employed to handle missing data for the moderation analyses.

Moderation analyses were conducted to examine whether parental baseline psychopathology (i.e. depression, anxiety, PTSD) moderated the relationship between child's baseline and post-treatment PTSD, controlling for child's age and gender. None of the parental baseline psychopathology variables was found to be significant moderators (see Table 4). To examine the robustness of the findings, sensitivity analyses were conducted in which (i) age was removed, (ii) gender was removed, and (iii) both age and gender were removed. Across all models, the interaction term remained non-significant. See Appendix H for more details on the moderation results.

Table 4. *Moderating Effects of Parental Baseline Psychopathology (IV: Child Baseline PTSD)*

Variable	*b*	SE	*z*	*p*	R^2
Post-treatment					
Parental depression (PHQ-9)					
Age	0.18	0.50	0.35	0.724	0.217
Gender	-5.05	2.94	-1.72	0.085	
Child baseline PTSD	0.75	0.34	1.74	0.000	
Parental depression	-0.15	0.17	-0.93	0.354	
Interaction (child baseline PTSD * parental depression)	0.02	0.03	0.758	0.560	
Parental anxiety (GAD-7)					
Age	0.20	0.49	0.40	0.686	0.223
Gender	-4.95	2.84	-1.74	0.082	
Child baseline PTSD	0.71	0.20	3.48	0.000	
Parental anxiety	0.06	0.20	0.28	0.777	
Interaction (child baseline PTSD * parental anxiety)	0.03	0.04	0.80	0.423	
Parental PTSD (PDS-5)					
Age	0.17	0.48	0.35	0.723	0.216
Gender	-4.94	2.86	-1.73	0.084	
Child baseline PTSD	0.70	0.20	3.51	0.000	
Parental PTSD	0.02	0.06	0.29	0.771	
Interaction (child baseline PTSD * parental PTSD)	0.01	0.01	0.64	0.523	
11-month post-randomisation					
Parental depression (PHQ-9)					
Age	-0.62	0.45	-1.38	0.168	0.340
Gender	-6.21	2.97	-2.10	0.036	

Child baseline PTSD	1.11	0.18	6.08	0.000	
Parental depression	-0.15	0.20	-0.76	0.445	
Interaction (child baseline PTSD * parental depression)	0.03	0.04	0.69	0.489	
Parental anxiety (GAD-7)					
Age	-0.56	0.46	-1.22	0.223	0.330
Gender	-5.43	2.81	-1.93	0.053	
Child baseline PTSD	0.97	0.20	4.98	0.000	
Parental anxiety	0.08	0.23	0.33	0.745	
Interaction (child baseline PTSD * parental anxiety)	-0.04	0.06	-0.66	0.512	
Parental PTSD (PDS-5)					
Age	-0.55	0.44	-1.26	0.206	0.346
Gender	-5.23	2.70	-1.94	0.053	
Child baseline PTSD	0.94	0.20	4.74	0.000	
Parental PTSD	0.11	0.07	1.41	0.159	
Interaction (child baseline PTSD * parental PTSD)	-0.00	0.02	-0.22	0.829	

3.5 Hypothesis 2b: Parental baseline psychopathology will moderate the relationship between the treatment condition and child's post-treatment PTSD symptomatology.

Moderation analyses were conducted to examine whether parental baseline psychopathology (i.e. depression, anxiety, PTSD) moderated the relationship between treatment condition and child's post-treatment PTSD, controlling for child's age, gender, and baseline PTSD scores. Sensitivity analyses (i.e. removing age only, removing gender only, removing both age and gender) were run.

Results revealed a significant interaction between treatment condition and parental baseline depression (b = −0.59, SE = 0.29, z = −2.07, p = .039) (see Table 5). This indicates that the effect of treatment on post-treatment PTSD scores varied depending on the level of parental depression at baseline. In particular, at higher levels of parental baseline depression, CT-PTSD was associated with greater reductions in children's PTSD symptoms compared to TAU. Figure 1 shows the moderating effect. A significant main effect was observed for treatment condition (b = −5.93, SE = 2.02, z = −2.93, p = .003). The model accounted for 31.1% of the variance in child's post-treatment PTSD scores. The interaction term, however, became non-significant when age was removed, (b = −0.50, SE = 0.27, z = −1.83, p = .067, R^2 = .292), and when age and gender were removed from the model (b = −0.53, SE = 0.30, z = −1.77, p = .078, R^2 = .237). This moderating effect of parental baseline depression was not

found significant at 11-month post-randomisation (*b* = −0.28, *SE* = 0.41, *z* = −0.69, *p* = .492). It was also non-significant when ITT sample was used (*b* = −0.50, *SE* = 0.30, *z* = −1.66, *p* = .097) (see Table G.2 in Appendix G).

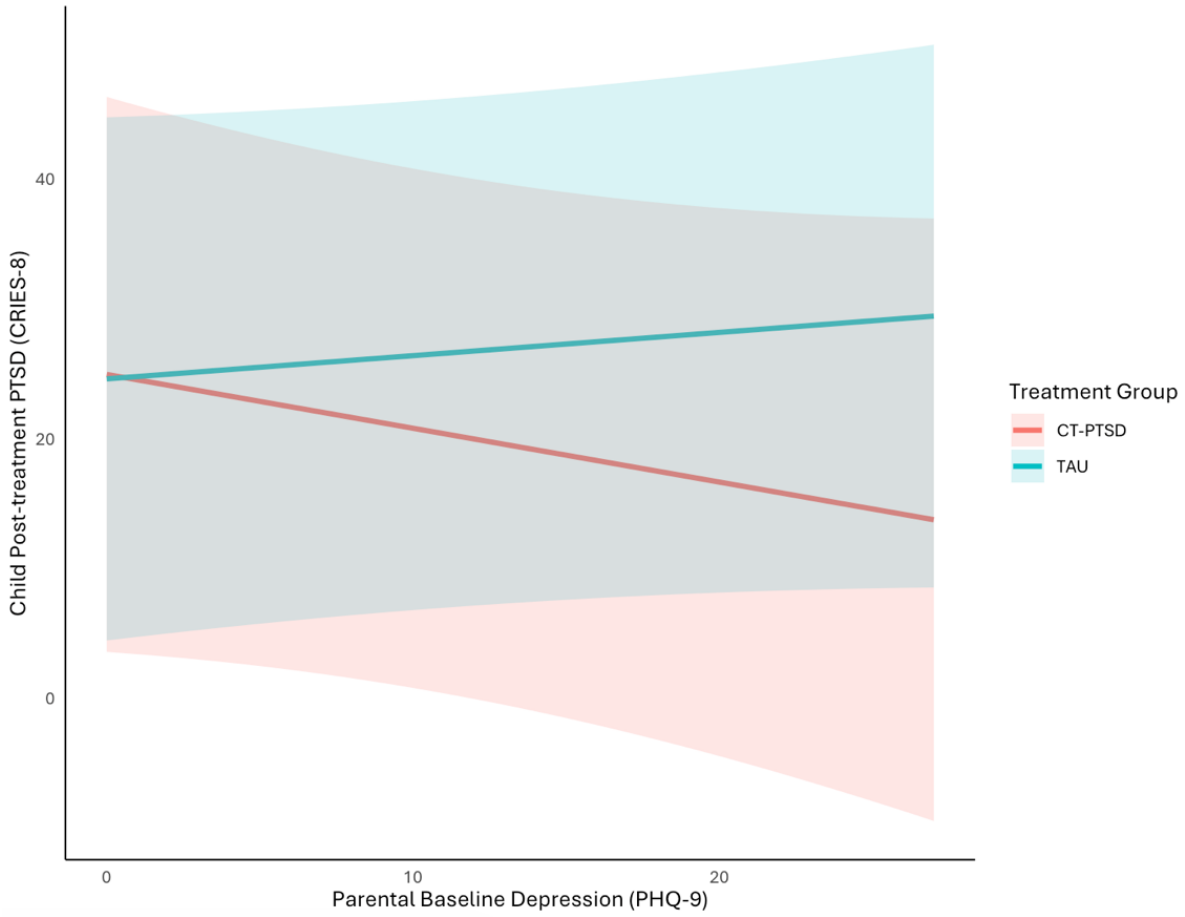
Parental anxiety and PTSD at baseline were not found to moderate the relationship between the treatment condition and child's post-treatment PTSD, both at post-treatment and 11-month post-randomisation (see Table 5). See Appendix H for more details on the moderation results.

Table 5. *Moderating Effects of Parental Baseline Psychopathology (IV: Treatment Condition)*

Variable	*b*	*SE*	*z*	*p*	R^2
Post-treatment					
Parental depression (PHQ-9)					
Age	0.57	0.48	1.19	0.233	0.311
Gender	-5.33	2.74	-1.95	0.052	
Child baseline PTSD	0.68	0.23	3.01	0.003	
Treatment condition	-5.93	2.02	-2.93	0.003	
Parental depression	0.18	0.23	0.79	0.427	
Interaction (treatment condition * parental depression)	-0.59	0.29	-2.07	0.039	
Parental anxiety (GAD-7)					
Age	0.43	0.46	0.93	0.351	0.292
Gender	-5.90	2.78	-2.12	0.034	
Child baseline PTSD	0.63	0.22	2.89	0.004	
Treatment condition	-5.90	2.05	-2.88	0.004	
Parental anxiety	0.34	0.27	1.25	0.213	
Interaction (treatment condition * parental anxiety)	-0.51	0.39	-1.32	0.187	
Parental PTSD (PDS-5)					
Age	0.44	0.43	1.02	0.308	0.293
Gender	-5.45	2.68	-2.03	0.042	
Child baseline PTSD	0.62	0.23	2.73	0.006	
Treatment condition	-5.70	2.04	-2.79	0.005	
Parental PTSD	0.11	0.08	1.36	0.175	
Interaction (treatment condition * parental PTSD)	-0.17	0.11	-1.55	0.121	
11-month post-randomisation					
Parental depression (PHQ-9)					
Age	-0.25	0.45	-0.55	0.583	0.394
Gender	-6.60	2.92	-2.26	0.024	
Child baseline PTSD	1.01	0.18	5.48	0.000	
Treatment condition	-5.94	2.27	-2.62	0.009	
Parental depression	0.01	0.31	0.03	0.978	
Interaction (treatment condition * parental depression)	-0.28	0.41	-0.69	0.492	

Parental anxiety (GAD-7)					
Age	-0.24	0.44	-0.54	0.590	0.401
Gender	-7.00	3.01	-2.32	0.020	
Child baseline PTSD	0.95	0.19	5.06	0.000	
Treatment condition	-5.95	2.23	-2.67	0.008	
Parental anxiety	0.28	0.37	0.75	0.456	
Interaction (treatment condition * parental anxiety)	-0.56	0.49	-1.14	0.255	
Parental PTSD (PDS-5)					
Age	-0.24	0.43	-0.56	0.574	0.414
Gender	-6.41	2.82	-2.28	0.023	
Child baseline PTSD	0.87	0.20	4.38	0.000	
Treatment condition	-5.74	2.19	-2.62	0.009	
Parental PTSD	0.17	0.11	1.49	0.136	
Interaction (treatment condition * parental PTSD)	-0.14	0.15	-0.91	0.363	

Figure 1. *Moderating Effect of Parental Baseline Depression on Treatment Effectiveness*



Note. Solid lines show predicted post-treatment CRIES-8 scores, and shaded regions represent the 95% confidence interval around the predicted values.

4. Discussion

This study examined the relationship between parental psychopathology and child's trauma symptomatology and treatment outcomes. Specifically, it investigated the association between parental baseline psychopathology and child's baseline symptoms, as well as the moderating role of parental psychopathology in child's treatment response.

4.1 Relationship between parental psychopathology and child's symptomatology at baseline

In relation to H1, parental baseline psychopathology was weakly and sporadically associated with aspects of child's baseline symptomatology, including PTSD severity, depression, separation anxiety, and overall functioning. As both child and parent samples showed a high degree of psychopathology at baseline, the sparse correlations could possibly reflect limited variability within a high-risk sample. While the associations were modest, they are broadly consistent with existing literature.

The positive relationship between parental depression and child's PTSD symptoms has received robust support (Morris et al., 2012; Naeem et al., 2025). Depressed parents may experience diminished emotional responsiveness or increased withdrawal, which can interfere with the child's sense of safety and emotion regulation following traumatic exposure, thereby exacerbating child's PTSD symptomatology (Cobham et al., 2016; Goodman et al., 2011; Morris et al., 2012). Supported by previous meta-analyses (e.g., Micco et al., 2009; Lawrence et al., 2019; Goodman et al., 2011; Morris et al., 2012), higher levels of parental depression and anxiety were linked to greater child depressive symptoms. Parental depression could lead to elevated internalising problems in children through mechanisms such as negative parenting behaviors, impaired emotional co-regulation, and heightened family stress (Goodman & Tully, 2006; McLeod et al., 2007). More severe parental anxiety and PTSD was associated with poorer overall functioning in the child. Creech & Misca (2017) postulated that behavioural avoidance and maladaptive threat perception, which are symptomatic of PTSD and anxiety, by parents can result in family overaccommodation and low self-efficacy in children, impairing their

functioning. The association between parental PTSD and child's separation anxiety echoed Lester et al.'s (2016) findings in the military population.

4.2 Parental psychopathology as a moderator of child PTSD treatment outcomes

Moderation analyses addressed two conceptualisations of how parental psychopathology influences treatment response: (i) as a moderator of the relationship between child baseline and post-treatment PTSD (H2a), and (ii) as a moderator of the relationship between treatment condition and child's post-treatment PTSD (i.e. treatment effectiveness) (H2b).

With reference to H2a, parental psychopathology did not moderate the relationship between child's baseline and post-treatment PTSD symptoms. This is inconsistent with findings from Nixon et al. (2012) and Weems & Scheeringa (2013), who reported maternal depression as a significant treatment moderator. It however aligns with their findings that parental anxiety and PTSD are not significant treatment moderators.

In contrast, tentative support was found for H2b in the case of parental depression. A significant interaction emerged between treatment condition and parental baseline depression, such that CT-PTSD was more effective relative to TAU for children whose parents had higher levels of depressive symptoms. In other words, children whose parents reported elevated depression at baseline showed greater reductions in PTSD symptoms following CT-PTSD than TAU. Although statistically significant, the moderation effect was small in magnitude (β = −0.27). Importantly, this moderating effect was observed post-treatment but not at follow-up (i.e. 11-month post-randomisation), suggesting that parental depression has a more immediate, rather than sustained, impact on child treatment response. Parental anxiety and PTSD were not significant moderators of treatment efficacy at either post-treatment or 11-month post-randomisation.

The direction of the moderating effect contrasts with studies which revealed that parental depression hinders child treatment gains, possibly by reducing parental involvement or modeling maladaptive coping (Nixon et al., 2012; Weems & Scheeringa, 2013). Nonetheless, it may be important to point out that Nixon et al.'s (2012) and Weems & Scheeringa's (2013) findings were investigated in

the context of H2a instead of H2b (i.e. parental depression as a moderator of the relation between child's baseline and post-treatment PTSD symptoms).

Encouragingly, our preliminary finding that CT-PTSD is particularly effective for young people with parents with more severe depression supports evidence that CT-PTSD can be effective for young people with more complex trauma presentations (e.g., multiple traumas and comorbidities) (e.g., Meiser-Stedman et al., 2025; Smith et al., 2025). Given emerging evidence that CT-PTSD delivered to the child improves parental depression (Hammond et al., 2021), components of the child's trauma treatment could have mitigated the potentially disruptive effects of parental depression on child's engagement in treatment and parent's ability to offer emotional support (Cohen et al., 2016; Nixon et al., 2012).

4.3 Strengths and limitations

The pragmatic trial design enhances ecological validity where the findings provide clinically meaningful insights into the effectiveness of CT-PTSD under routine clinical practice and are directly applicable to real-world clinical settings. Another strength of this study is the clinical severity of the sample at baseline. All participants met the recommended clinical cutoff for PTSD on the CPSS-I-5 (Foa et al., 2013), and the majority reported depressive and anxiety symptoms within the clinical range. This study characteristic enhances the clinical relevance and external validity of the findings, as the sample mirrors real-world populations seeking specialised trauma treatment in mental health services. The elevated baseline severity further supports the treatment efficacy of CT-PTSD, demonstrating that CT-PTSD can be effective even among young people with substantial and complex mental health needs. Lastly, to the authors' knowledge, this is the first study that examined the moderating effect of parental psychopathology on the relationship between treatment condition and child's post-treatment PTSD using an active intervention group (CT-PTSD) and a control group (TAU). In a similar trial by de Roos et al. (2021), two active interventions groups were compared (i.e. Cognitive Behavioural Writing Therapy (CBWT) vs. Eye Movement Desensitisation and Reprocessing (EMDR)).

A few study limitations were noted. Positioned as a pragmatic trial, participants in the TAU group received interventions that were available in routine mental health services. It could be argued that the control group received active psychotherapeutic treatment components which might have reduced the observed differences between CT-PTSD over TAU and potentially attenuated moderation effects. Consequently, the significant moderating role of parental baseline depression observed in the present study may represent a robust effect that emerged despite an active comparison condition. Sample size was modest when considering complete cases with parent responses. This might have limited the statistical power to detect small moderation effects. Given the sparse literature on how parental psychopathology could moderate trauma-focused treatments for young people, existing evidence may be vulnerable to publication bias. This highlights the need for replication in larger, well-powered, and methodologically rigorous samples. It was beyond the scope of this study to examine whether parents accessed any form of support for their mental health or how their mental health changed over the course of the child's treatment. It therefore remains unclear how child's PTSD treatment may have influenced, or been influenced by, broader systemic factors such as parental mental health.

4.4 Future research

Future research should replicate these findings in larger, more heterogeneous samples and explore mechanisms through which parental depression enhances CT-PTSD effectiveness, such as parental engagement or modeling of adaptive coping. It should also look at treatment mediators like parental response to child's trauma or parent-child interaction quality. Additionally, future work could examine parental mental health at multiple time points during child's PTSD treatment to characterise trajectories of parental psychopathology. It could also investigate whether changes in parental mental health are associated with child's treatment outcomes and identify factors that contribute to improvements or persistence of parental psychopathology.

4.5 Clinical implications

An assessment of parental depression at treatment outset could help identify families for whom CT-PTSD could yield greater benefit. Practically, mental health services could preferentially allocate families with elevated depressive symptoms to CT-PTSD, or at minimum, not rule out CT-PTSD as a treatment option for these families. The absence of long-term moderation effects at 11-month post-randomisation suggests that CT-PTSD can produce comparable treatment effects across children with varying parental depressive symptoms in the longer term. Additional parent-focused interventions may not be imperative to ensure child's treatment gains.

4.6 Conclusion

Overall, the findings provided partial support for our hypotheses. Parental psychopathology was weakly associated with certain aspects of child's symptomatology pre-treatment. Parental depression, but not anxiety or PTSD, moderated the effect of CT-PTSD on child PTSD symptoms post-treatment but not at 11-month post-randomisation. The small and short-term moderating effect should be interpreted cautiously. Nonetheless, the results appeared to suggest that CT-PTSD could benefit young people whose parents present with more severe psychopathology.

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Chapter Five: Discussion and Critical Evaluation

Summary of findings

Systematic review

The review included 16 studies published between 2004 and 2025. Across the studies, 22 treatment moderators and 16 treatment mediators were examined. Although the review adopted broad inclusion criteria encompassing any trauma-focused psychotherapeutic intervention, the resulting evidence base was predominantly composed of cognitive-behavioural interventions, with only one study employing a non-cognitive-behavioural approach (i.e. Shechtman & Mor, 2010). Findings of this review hence primarily reflect outcomes associated with cognitive-behavioural trauma-focused interventions. A large proportion of the factors were studied by one study (i.e. 19 moderators and 13 mediators). Of the remaining moderators, age and gender were each examined by six studies and largely did not appear to moderate treatment outcomes. The two papers that revealed a significant moderating effect of gender reported greater treatment gains for girls. Studied in two trials, race was not observed to significantly influence treatment outcomes. On the other hand, considerable empirical support was found for the mediating effect of post-traumatic cognitive appraisal which is in line with cognitive theories of PTSD (Dalgleish, 2004; Ehlers & Clark, 2000). Preliminary support was established for quality of traumatic memories and rumination as treatment mediators. Of note, the one study that did not report significant mediating effects for the three above-mentioned mediators was an early-phase trial which conducted the mediation analyses on an exploratory basis and could be underpowered.

Empirical research paper

Modest and sparse correlations were demonstrated between parental baseline psychopathology (i.e. depression, anxiety, and PTSD) and child's baseline symptomatology, which were both of substantial severity pre-treatment. Consistent with existing literature, some of the significant associations included: parental depression and child PTSD, parental depression and child depressive symptoms, parental anxiety and poorer child overall functioning. Parental psychopathology did not appear to moderate the relationship between child's baseline and post-treatment PTSD symptoms.

Tentative support was found for the moderating effect of parental depression on the relationship between treatment condition and child's post-treatment PTSD symptoms. Specifically, children whose parents had higher levels of depressive symptoms enjoyed greater treatment gains from CT-PTSD than TAU. This small moderation effect was noted post-treatment but not at follow-up. Parental anxiety and PTSD were not significant treatment moderators at post-treatment and follow-up.

Strengths and limitations

Systematic review

While acknowledging the value of the methodological approaches employed by existing meta-analyses and systematic reviews, the current review sought to complement the literature by addressing the methodological limitations introduced by those approaches. For example, some reviews on treatment moderators were restricted to cognitive behavioural treatment (e.g., Yohannan et al., 2022), TF-CBT (e.g., de Haan et al., 2024), or cognitive behavioural trauma interventions with caregiver involvement (e.g., Somers et al., 2024). Some required participants to present with trauma-related symptoms or have experienced traumatic events but not necessarily have clinically relevant post-traumatic stress symptoms (e.g., Yohannan et al., 2022; Protić et al., 2024). To ensure robustness and comprehensiveness of evidence, this review included any trauma-focused psychotherapeutic intervention, regardless of caregiver involvement or theoretical approach, while tightening inclusion criteria, such as requiring studies to be of RCT design and participants to have PTSD symptoms of clinical severity. The latter requirement strengthens the clinical relevance and external validity of the review findings as the participant characteristic is likely representative of individuals seeking specialised trauma treatment in mental health services.

Despite the deliberately broad inclusion criteria to capture the full range of trauma-focused psychotherapeutic interventions and address limitations in prior syntheses, the available evidence was overwhelmingly dominated by cognitive-behavioural approaches, with only one included study adopting a non-cognitive-behavioural intervention (i.e. Shechtman & Mor, 2010). Consequently, the review findings primarily reflect the moderators and mediators of cognitive-behavioural trauma-

focused interventions rather than trauma-focused interventions more broadly, highlighting a gap in the literature rather than a limitation of the review methodology.

The Cuijpers et al. (2010) checklist was used to assess study quality. Specifically, it evaluated psychotherapy-specific indicators of methodological quality, including blinded assessors, independent randomisation, therapist training, use of treatment manuals, and treatment integrity. Designed to assess methodological quality rather than risk of bias, the checklist does not fully capture all potential sources of bias addressed by contemporary risk-of-bias assessment tools (e.g., the Cochrane RoB 2 tool; Sterne et al., 2019), such as bias arising from outcome measurement and selective reporting.

The inclusion criteria inevitably precluded potentially informative universal trials. Many of these trials looked at young people living in war-affected areas (e.g., Diab et al., 2014; Jobson et al., 2025; Jordans et al., 2010; Kangaslampi & Peltonen, 2020; Qouta et al., 2012; Tol et al., 2012; 2014) while some of the others focused on children who experienced parental death (e.g., Dorsey et al., 2020) or marital violence (e.g., Ippen et al., 2011). This restriction limits the generalisability of findings to broader trauma-exposed populations. A larger and more diverse sample could also allow stronger inferences of patterns.

The generalisability of the review findings should be considered in light of the characteristics of the included samples. Although the review included studies from multiple countries, most of the studies were conducted in Western populations, with limited representation from non-Western populations. Furthermore, the non-Western studies represented diverse and distinct sociocultural contexts rather than a single comparable population, limiting the cross-cultural applicability of findings. Additionally, several studies recruited predominantly female samples. These may influence the generalisability of the findings.

A number of studies included in this review only employed self-report measures to assess child's PTSD symptoms. As discrepancies between caregiver and child reports had been noted in the literature (e.g., Oransky et al., 2013), the assessment of child's PTSD symptomatology and symptom

improvement could be more accurate and complete with the use of parallel assessment (e.g., caregiver and child, child and clinician) (Skar et al., 2021).

Empirical research paper

Similar to the inclusion criteria for the systematic review, participants in the empirical study met the criteria for a diagnosis of PTSD. This ensures relevance of findings to real-life mental health services. The elevated baseline severity of the sample and their parents reinforced the notion that CT-PTSD can be effective for high-risk families. Perhaps more importantly, to the best of the author's knowledge, this is the first study that investigated how parental psychopathology moderated treatment effectiveness in children and adolescents; a similar trial by de Roos et al. (2021) studied how parental psychopathology moderated the effectiveness of two trauma-focused interventions: Cognitive Behavioural Writing Therapy (CBWT) vs. Eye Movement Desensitisation and Reprocessing (EMDR) in relation to each other. The use of a control group allows insights into how the effectiveness of a particular treatment (CT-PTSD in this case) is affected by the studied moderators (i.e. parental psychopathology in this study).

An inherent limitation of correlational analyses meant that a causal or temporal relationship between parental psychopathology and child's symptomatology could not be established. The observed relationships may reflect confounding variables (e.g., socioeconomic stressors, shared prior trauma exposure), or reciprocal influences between parent and child functioning. Accordingly, the correlational findings should be interpreted cautiously, and longitudinal research is required to clarify the temporal ordering and potential mechanisms underlying the association between parental psychopathology and child symptomatology. Keeping in mind that the sparse literature on treatment moderators could be vulnerable to publication bias, current finding about the moderating effect of parental depression on treatment outcomes should be interpreted tentatively. Moreover, the risk of Type I error needs to be considered. For H2b, six moderation tests were conducted (i.e. parental psychopathology (three factors: depression, anxiety & PTSD) x outcome measure (two factors: post-treatment & 11-month post-randomisation)). With Holm-Bonferroni correction (Holm, 1979), the

moderation effect of parental depression would not have reached significance (α = 0.05/6 ≈0.0083, which is smaller than the observed *p*-value of 0.039). While this study examined parental depression, anxiety and PTSD, other parental factors, such as parenting stress, parental coping, parental response to child's trauma, may also influence children treatment response.

Clinical and theoretical implications

Taking the findings of the review and empirical study together, there is preliminary evidence that CT-PTSD is an effective treatment for diverse child and adolescent populations, specifically in terms of their age, gender, race or parents' mental health (i.e. depression, anxiety, PTSD). This converges with adult findings which generally found an absence of moderating effects by age, gender, and race for PTSD treatments (Murad et al., 2024; Lester et al., 2010). The lack of moderating effects of parental psychopathology on treatment outcome at follow-up tentatively suggests that CT-PTSD can produce comparable treatment effects in young people across varying severity levels of parental psychopathology even in the longer term. Given the modest sample and effect size, the significant moderation results must be interpreted cautiously. The bottom line, nevertheless, appears to be that clinicians should not withhold CT-PTSD from young people with parents of greater mental health needs. This aligns with emerging evidence that CT-PTSD can benefit young people with more complex presentations (e.g., multiple traumas and comorbidities) (e.g., Meiser-Stedman et al., 2025; Smith et al., 2025).

Considering the empirical support for the three treatment mediators as highlighted in the review, results suggest the need to focus on cognitive processes (e.g., cognitive restructuring, contextualisation of memory) and challenge behavioural approaches that rely primarily on exposure. Encouragingly, addressing those cognitive components in PTSD treatments could lead to transdiagnostic improvement, in particular the alleviation of depressive and anxiety symptoms which are common comorbidities of PTSD (Moulds & McEvoy, 2025; Zhang et al., 2025; A-Tjak et al., 2021; Quilty et al., 2008; Barry et al., 2019). This is in support of the transdiagnostic view that mental health difficulties should be understood in terms of shared underlying mechanisms (e.g., repetitive negative

thinking, selective attention, negative valence systems) (Dalgleish et al., 2020). Clinically, this has the potential of lowering costs in often resource-limited mental health services.

Although parental psychopathology did not appear to moderate treatment outcome at follow-up in the present study, this should not be interpreted as indicating that parental mental health is clinically irrelevant. Given the bidirectional relationship between child and parental psychological functioning (Greene et al., 2023), monitoring parental mental health throughout a child's treatment may support a systemic approach to care by identifying parents who require additional support, facilitating timely referral to appropriate mental health services, and informing treatment adaptations to optimise child treatment outcomes.

Findings from the empirical study can be considered within systemic frameworks of child trauma, such as the Pyramid of Need (Golding, 2013), which emphasise the role of relational environments and parental support in child's emotional development and recovery. The observed associations between parental psychopathology and child's symptomatology at baseline are consistent with the proposition that parental emotional availability and ability to co-regulate can influence child's adjustment following trauma. On the other hand, the moderation findings extend this perspective by suggesting that although parental psychological difficulties may compromise the lower tiers of the pyramid by reducing caregivers' capacity to provide consistent emotional availability and support, potentially contributing to greater child symptomatology at presentation, these vulnerabilities do not necessarily impede child's response to CT-PTSD.

Future research

This paper recommends future work to adopt a theory-driven approach when investigating treatment moderators and mediators (e.g., cognitive mechanisms, social support including parental response) and pursue further investigation of the understudied variables identified in the review. Currently, the area of work is arguably in its infancy stage where a large proportion of studied variables were examined by one study. This greatly limits the interpretation of findings. Future research could also explore treatment moderators and mediators for specific interventions (e.g., TF-CBT, classroom-

based intervention, prolonged exposure therapy). This would allow further tailoring of the interventions to the client population in question. For instance, in adult studies, McLean et al. (2022) investigated moderators of exposure treatments and reported population type (e.g., refugee vs. civilian vs. military), trauma types (e.g., sexual assault, combat, natural disaster, interpersonal), and complexity of trauma as significant treatment moderators whereas Wright et al. (2025) reported marital status and psychotropic medication as potential moderators for TF-CBT treatments. Future systematic reviews of psychotherapeutic interventions may benefit from assessing both risk of bias and methodological validity of studies. This approach would allow comprehensive assessment of potential sources of bias while retaining important information about intervention quality, such as therapist competence, treatment fidelity, and adherence to treatment protocols, which are particularly relevant in psychological intervention research.

Conclusion

A narrative synthesis of treatment moderators and mediators for children and young people with PTSD symptoms was conducted. Across the 16 studies reviewed, treatment effects generally appeared to be consistent across demographics: age, gender, and race. Moderation results from the empirical study offered tentative evidence that treatment gains from CT-PTSD were larger for children whose parents are more depressed. Considering the small effect size and absence of this effect at follow-up, the moderating effect of parental depression should be interpreted cautiously. Nonetheless, the key takeaway would be that young people could benefit from CT-PTSD regardless of their parents' mental health needs. Evidence for three treatment mediators was found through the review: post-traumatic cognitive appraisal, trauma memory quality, and rumination.

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Appendices

Appendix A. Author guidelines for Journal of Anxiety Disorders

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About the journal

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Appendix B. Additional study characteristics

Table B.1. *Additional study characteristics*

Author (Year)	Study population/setting	Outcome measures for PTSS	Significant main effect of treatment on PTSS
Capaldi et al. (2016)	Have diagnosis of chronic or subthreshold PTSD related to sexual assault	CPSS	Yes
Cohen et al. (2004)	Experienced sexual abuse	K-SADS-PL	Yes
Cohen et al. (2011)	Experience PTSS related to intimate partner violence	K-SADS-PL; UCLA PTSD RI	Yes
Holt et al. (2014)	Experienced at least one potentially traumatising event and have CPSS cutoff score of 15	TESI-C	Not reported
Jensen et al. (2018)	Experienced at least one potentially traumatising event and have CPSS cutoff score of 15	CAPS-CA	Yes
Kane et al. (2016)	Orphans and vulnerable children	UCLA PTSD RI	Yes
Li et al. (2023)	Experienced at least one traumatic event and have diagnosis of full or subthreshold PTSD	UCLA PTSD RI	Yes*
Li et al. (2024)	Experienced at least one traumatic event and have diagnosis of full or subthreshold PTSD	UCLA PTSD RI	Yes
Meiser-Stedman et al. (2017)	Have PTSD relating to a single trauma	CPTSDI	Yes
Pfeiffer et al. (2017)	Exposure to one or more traumatic events	CAPS-CA	Yes
Shechtman & Mor (2010)	Affected by war	CPTS-RI	Yes
Smith et al. (2007)	Have PTSD symptoms related to a single trauma	CAPS-CA; CPSS; CRIES-13	Yes

Smith et al. (2025)	Have PTSD symptoms related to a single trauma	CAPS-CA; CPSS; CRIES-13	Yes
Tol et al. (2008)	Exposed to political violence	CPSS	Yes
Tol et al. (2010)	Exposed to political violence	CPSS	Yes
Tutus et al. (2019)	Have clinically relevant PTSS	CAPS-CA; UCLA PTSD RI	Yes

Note. CPSS = Child Post-traumatic Stress Disorder Symptom Scale; K-SADS-PL = Kiddie-Schedule for Affective Disorders and Schizophrenia Present and Lifetime Version; UCLA PTSD RI = University of California at Los Angeles Post-traumatic Stress Disorder Reaction Index; TESI-C = Traumatic Events Screening Inventory for Children; CAPS-CA = Clinician-Administered PTSD Scale for Children and Adolescents; CPTSDI = Children's PTSD Inventory; CPTS-RI = Child Post-Traumatic Stress-Reaction Index; CRIES-13 = Children Revised Impact of Event Scale; * main effect of treatment was significant at post-treatment, not 3-month follow-up.

Appendix C. Quality ratings

Table C.1. *Quality ratings for included studies*

Author (Year)	Met diagnostic criteria	Use of treatment manual	Trained therapists	Treatment integrity checked	Intention-to-treat analyses	Sample size ≥ 50	Randomisation by independent party	Blinded assessors	Total quality score
Capaldi et al. (2016)	0	1	1	1	1	0	0	1	5
Cohen et al. (2004)	0	1	1	1	1	1	0	1	6
Cohen et al. (2011)	0	1	1	1	1	1	1	1	7
Holt et al. (2014)	0	1	1	1	1	1	1	1	7
Jensen et al. (2018)	0	1	1	1	1	1	1	1	7
Kane et al. (2016)	0	1	1	1	1	1	1	1	7
Li et al. (2023)	0	1	1	1	1	1	1	1	7
Li et al. (2024)	0	1	1	1	1	1	1	1	7
Meiser-Stedman et al. (2017)	1	1	1	1	1	0	1	1	7
Pfeiffer et al. (2017)	0	1	1	1	0	1	1	1	6
Shechtman & Mor (2010)	0	1	1	1	0	1	0	0	4
Smith et al. (2007)	1	1	1	1	1	0	1	1	7
Smith et al. (2025)	1	1	1	1	1	0	1	1	7
Tol et al. (2008)	0	1	1	1	1	1	1	1	7
Tol et al. (2010)	0	1	1	1	0	1	1	1	6
Tutus et al. (2019)	0	1	1	1	1	1	1	1	7

Appendix D. Calculation of outcome measure scores

Child Revised Impact of Event Scale (8-item version; CRIES-8)

The total score across the 8 items at each timepoint was calculated. To account for possible missing data within a participant, participants who responded to at least 7 out of the 8 CRIES-8 items were included. For participants who only responded to 7 out of the 8 items, the mean of the 7 items was calculated and multiplied by 8 to generate an average CRIES-8 score.

Revised Child Anxiety and Depression Scale (47-item version; RCADS)

Guidance on management of missing data was taken from (Revised Children's Anxiety and Depression Scale (RCADS), n.d.).

For Depression score, participants who responded to at least 8 out of the 10 RCADS-Depression items were included. For participants who only responded to 8 or 9 out of the 10 items, the mean of the 8 or 9 items was calculated and multiplied by 10 to generate an average RCADS-Depression score.

For Total Anxiety score, participants who responded to at least 27 out of the 37 RCADS-Total Anxiety items were included, but only if they do not have more than 2 missing items per anxiety subscale. For these participants, the mean of the items they had responded to was calculated and multiplied by 37 to generate an average RCADS-Total Anxiety score.

For Social Phobia score, participants who responded to at least 7 out of the 9 RCADS-Social Phobia items were included. For these participants, the mean of the 7 or 8 items was calculated and multiplied by 9 to generate an average RCADS-Social Phobia score.

For Panic Disorder score, participants who responded to at least 7 out of the 9 RCADS-Panic Disorder items were included. For these participants, the mean of the 7 or 8 items was calculated and multiplied by 9 to generate an average RCADS-Panic Disorder score.

For Separation Anxiety score, participants who responded to at least 5 out of the 7 RCADS-Separation Anxiety items were included. For these participants, the mean of the 5 or 6 items was calculated and multiplied by 7 to generate an average RCADS-Separation Anxiety score.

For Generalised Anxiety score, participants who responded to at least 4 out of the 6 RCADS-Generalised Anxiety items were included. For these participants, the mean of the 4 or 5 items was calculated and multiplied by 6 to generate an average RCADS-Generalised Anxiety score.

For Obsessive-Compulsive score, participants who responded to at least 4 out of the 6 RCADS-Obsessive-Compulsive items were included. For these participants, the mean of the 4 or 5 items was calculated and multiplied by 6 to generate an average RCADS-Obsessive-Compulsive score.

Patient Health Questionnaire (9-item version; PHQ-9)

The total score across the 9 items was calculated. To account for possible missing data within a participant, participants who responded to at least 8 out of the 9 PHQ-9 items were included. For participants who only responded to 8 out of the 9 items, the mean of the 8 items was calculated and multiplied by 9 to generate an average PHQ-9 score.

General Anxiety Disorder (7-item version; GAD-7)

The total score across the 7 items was calculated. The total score across the 7 items will be calculated. To account for possible missing data within a participant, participants who responded to at least 6 out of the 7 GAD-7 items were included. For participants who only responded to 6 out of the 7 items, the mean of the 6 items was calculated and multiplied by 7 to generate an average GAD-7 score.

Post-traumatic Stress Diagnostic Scale for DSM-5 (PDS-5)

The total score across the 20 items was calculated. To account for possible missing data within a participant, participants who responded to at least 18 out of the 20 PDS-5 items were included. For participants who only responded to 18 or 19 out of the 20 items, the mean of the 18 or 19 items was calculated and multiplied by 20 to generate an average PDS-5 score.

Appendix E. Participants' RCADS scores at baseline (per-protocol sample)

Table E.1. *Participants' RCADS scores at baseline (n = 111)*

	n (%)		
	Normal	Borderline	Clinical
Total	30 (27.0)	15 (13.5)	66 (59.5)
Depression	23 (20.7)	7 (6.3)	81 (73.0)
Overall anxiety	43 (38.7)	10 (9.0)	58 (52.3)
Social phobia	60 (54.1)	18 (16.2)	33 (29.7)
Panic disorder	31 (27.9)	15 (13.5)	65 (58.6)
Separation anxiety	33 (29.7)	16 (14.4)	62 (55.9)
Generalised anxiety	58 (52.3)	16 (14.4)	37 (33.3)
Obsessive-compulsive	70 (63.1)	9 (8.1)	32 (28.8)

Appendix F. Baseline descriptives (intention-to-treat sample)

Baseline characteristics

The intention-to-treat sample comprised 120 young people aged 8–17 years old. The mean CRIES-8 score at baseline was 31.6 (SD = 6.0), with 100% of the sample meeting the suggested clinical cutoff score of 17 and above (Perrin et al., 2005). The completion rate for the CRIES-8 questionnaire was 100% (*n* = 120) at baseline, 80.0% (*n* = 96) post-treatment, and 67.5% (n = 81) at 11-month post-randomisation. Please refer to Meiser-Stedman et al. (2025) for additional baseline characteristics of the ITT sample (e.g., mean age, proportion of females).

For parents, the number of PHQ-9, GAD-7 and PDS-5 responses collected at baseline was 92, 93 and 94 respectively. A large proportion of the parents in this study experienced moderate to severe levels of depression, anxiety and PTSD (see Table 1).

Table F.1. *Parent Baseline Psychopathology*

	Parental depression n (%)	Parental anxiety n (%)	Parental PTSD n (%)
None – mild	45 (48.9)	48 (51.6)	19 (20.2)
Moderate^	35 (38.0)	21 (22.6)	36 (38.3)
Severe	12 (13.0)	24 (25.8)	39 (41.5)

Note. ^ Moderate here covers Moderate and Moderately severe for PHQ-9; Moderate for GAD-7; Moderate and Moderate to Severe for PDS-5.

Baseline group comparisons

No between-group differences were found in participants' age, baseline symptomatology and parental psychopathology. There were significantly more females in the CT-PTSD group (*n* = 47; 81.0%) than in the TAU group (*n* = 40; 64.5%), $\chi(1) = 4.10$, $p = .043$. Please refer to Meiser-Stedman et al. (2025) for more details of the baseline group comparisons of the ITT sample (e.g., mean age, proportion of females, RCADS scores).

Table F.2. *Group Comparisons at Baseline*

	Mean/Freq	SD/%	Mean/Freq	SD/%	Test statistic
Child					
	CT-PTSD (*n* = 58)		TAU (*n* = 62)		
CRIES-8	31.7	5.9	31.5	6.1	$t_{109} = -0.22$
CGAS	48.0	10.8	51.1	10.5	$t_{109} = 1.62$
Parent					
	CT-PTSD (*n* = 43)		TAU (*n* = 49)		
Depression (PHQ-9)	10.9	7.4	10.0	8.0	$t_{82} = -0.56$
	CT-PTSD (*n* = 43)		TAU (*n* = 50)		
Anxiety (GAD-7)	9.9	6.4	9.2	6.7	$t_{83} = -0.52$
	CT-PTSD (*n* = 44)		TAU (*n* = 50)		
PTSD (PDS-5)	32.0	20.9	30.1	21.9	$t_{84} = -0.41$

Note. *p<0.05; **p<0.01; ***p<0.001.

Table F.3. *Correlation Matrix for Parent Psychopathology and Child Symptomatology*

	Parental depression (PHQ-9)	Parental anxiety (GAD-7)	Parental PTSD (PDS-5)
CRIES-8 (pre)	0.23*	0.18	0.13
CRIES-8 (post)	−0.04	0.06	0.06
CRIES-8 (11mo)	0.06	0.08	0.25
RCADS			
Depression	0.29**	0.33**	0.24*
Overall anxiety	0.16	0.18	0.11
Social phobia	0.05	0.05	0.00
Panic disorder	0.10	0.09	−0.00
Separation anxiety	0.23*	0.22*	0.27**
Generalised anxiety	0.19	0.18	0.11
Obsessive-compulsive	0.08	0.19	0.09
CGAS	−0.16	−0.24*	−0.19

Note. *p<0.05; **p<0.01; ***p<0.001; pre = pre-treatment; post = post-treatment; 11mo = 11-month post-randomisation.

Table F.4. *Participants' RCADS scores at baseline (n = 120)*

	n (%)		
	Normal	Borderline	Clinical
Total	33 (27.5)	16 (13.3)	71 (59.2)
Depression	25 (20.8)	8 (6.7)	87 (72.5)
Overall anxiety	47 (39.2)	12 (10.0)	61 (50.8)
Social phobia	65 (54.2)	20 (16.7)	35 (29.2)
Panic disorder	34 (28.3)	17 (14.2)	69 (57.5)
Separation anxiety	39 (32.5)	16 (13.3)	65 (54.2)

Generalised anxiety	63 (52.5)	17 (14.2)	40 (33.3)
Obsessive-compulsive	77 (64.2)	11 (9.2)	32 (26.7)

Appendix G. Moderating effects of parental baseline psychopathology (intention-to-treat sample)

Little's (1988) MCAR test was non-significant, $\chi^2(37) = 48.0$, $p = .106$, indicating that data was missing completely at random. Therefore, FIML was employed to handle missing data for the moderation analyses. No significant moderating effects of parental psychopathology was observed across all models. Sensitivity analyses were conducted in which (i) age was removed, (ii) gender was removed, and (iii) both age and gender were removed. Across all models, the interaction term remained non-significant.

Table G.1. *Moderating Effects of Parental Baseline Psychopathology (IV: Child Baseline PTSD)*

	Variable	*b*	SE	*z*	*p*	R^2
Post-treatment						
Parental depression (PHQ-9)						
Model 1	Age	0.05	0.46	0.12	0.907	0.192
	Gender	-6.78	2.78	-2.44	0.015	
	Child baseline PTSD	0.60	0.20	2.99	0.003	
	Parental depression	-0.12	0.16	-0.72	0.472	
Model 2	Age	0.07	0.45	0.16	0.870	0.196
	Gender	-6.49	2.82	-2.30	0.021	
	Child baseline PTSD	0.60	0.20	2.93	0.003	
	Parental depression	-0.12	0.17	-0.71	0.481	
	Interaction (child baseline PTSD * parental depression)	-0.02	0.03	-0.49	0.626	
Parental anxiety (GAD-7)						
Model 1	Age	0.03	0.45	0.08	0.939	0.192
	Gender	-6.75	2.76	-2.45	0.014	
	Child baseline PTSD	0.57	0.20	2.84	0.005	
	Parental anxiety	0.06	0.19	0.33	0.744	
Model 2	Age	0.04	0.44	0.08	0.935	0.192
	Gender	-6.66	2.74	-2.43	0.015	
	Child baseline PTSD	0.56	0.20	2.77	0.006	
	Parental anxiety	0.07	0.19	0.34	0.732	
	Interaction (child baseline PTSD * parental anxiety)	-0.01	0.04	-0.20	0.843	
Parental PTSD (PDS-5)						
Model 1	Age	0.03	0.45	0.08	0.939	0.190
	Gender	-6.73	2.76	-2.44	0.015	
	Child baseline PTSD	0.57	0.20	2.91	0.004	
	Parental PTSD	0.01	0.06	0.24	0.813	
Model 2	Age	0.05	0.44	0.12	0.904	0.195
	Gender	-6.40	2.73	-2.35	0.019	
	Child baseline PTSD	0.57	0.20	2.91	0.004	
	Parental PTSD	0.02	0.06	0.27	0.789	

	Interaction (child baseline PTSD * parental PTSD)	-0.01	0.01	-0.50	0.615	
11-month post randomisation						
Parental depression (PHQ-9)						
Model 1	Age	-0.68	0.39	-1.76	0.078	0.316
	Gender	-7.08	2.54	-2.78	0.005	
	Child baseline PTSD	0.95	0.17	5.68	0.000	
	Parental depression	-0.04	0.19	-0.20	0.843	
Model 2	Age	-0.68	0.39	-1.73	0.084	0.316
	Gender	-7.04	2.81	-2.50	0.012	
	Child baseline PTSD	0.95	0.17	5.63	0.000	
	Parental depression	-0.04	0.19	-0.19	0.846	
	Interaction (child baseline PTSD * parental depression)	-0.00	0.04	-0.04	0.965	
Parental anxiety (GAD-7)						
Model 1	Age	-0.68	0.40	-1.70	0.090	0.317
	Gender	-7.08	2.54	-2.79	0.005	
	Child baseline PTSD	0.93	0.17	5.60	0.000	
	Parental anxiety	0.05	0.21	0.25	0.805	
Model 2	Age	-0.65	0.40	-1.64	0.101	0.323
	Gender	-6.68	2.70	-2.47	0.014	
	Child baseline PTSD	0.91	0.16	5.75	0.000	
	Parental anxiety	0.08	0.21	0.38	0.707	
	Interaction (child baseline PTSD * parental anxiety)	-0.03	0.05	-0.69	0.493	
Parental PTSD (PDS-5)						
Model 1	Age	-0.73	0.39	-1.85	0.065	0.343
	Gender	-6.93	2.39	-2.89	0.004	
	Child baseline PTSD	0.87	0.16	5.49	0.000	
	Parental PTSD	0.10	0.07	1.52	0.130	
Model 2	Age	-0.72	0.39	-1.85	0.064	0.344
	Gender	-6.64	2.62	-2.53	0.011	
	Child baseline PTSD	0.87	0.16	5.60	0.000	
	Parental PTSD	0.11	0.07	1.57	0.116	
	Interaction (child baseline PTSD * parental PTSD)	-0.00	0.01	-0.40	0.687	

Table G.2. *Moderating Effects of Parental Baseline Psychopathology (IV: Treatment Condition)*

	Variable	*b*	*SE*	*z*	*p*	R^2
Post-treatment						
Parental depression (PHQ-9)						
Model 1	Age	0.14	0.44	0.31	0.758	0.225
	Gender	-7.65	2.77	-2.76	0.006	
	Child baseline PTSD	0.56	0.20	2.79	0.005	
	Treatment condition	-4.78	2.04	-2.35	0.019	
	Parental depression	-0.10	0.16	-0.64	0.525	
Model 2	Age	0.32	0.46	0.69	0.491	0.260
	Gender	-7.21	2.77	-2.61	0.009	
	Child baseline PTSD	0.59	0.20	2.90	0.004	

	Treatment condition	-4.87	2.04	-2.39	0.017	
	Parental depression	0.12	0.22	0.55	0.582	
	Interaction (treatment condition * parental depression)	-0.50	0.30	-1.66	0.097	
Parental anxiety (GAD-7)						
Model 1	Age	0.12	0.43	0.28	0.782	0.227
	Gender	-7.64	2.77	-2.76	0.006	
	Child baseline PTSD	0.52	0.20	2.60	0.009	
	Treatment condition	-4.89	2.05	-2.39	0.017	
	Parental anxiety	0.08	0.20	0.40	0.687	
Model 2	Age	0.21	0.45	0.47	0.636	0.244
	Gender	-7.60	2.75	-2.76	0.006	
	Child baseline PTSD	0.54	0.20	2.69	0.007	
	Treatment condition	-4.92	2.06	-2.39	0.017	
	Parental anxiety	0.25	0.25	0.99	0.323	
	Interaction (treatment condition * parental anxiety)	-0.39	0.39	-1.00	0.318	
Parental PTSD (PDS-5)						
Model 1	Age	0.12	0.43	0.28	0.781	0.224
	Gender	-7.61	2.76	-2.76	0.006	
	Child baseline PTSD	0.53	0.20	2.69	0.007	
	Treatment condition	-4.85	2.04	-2.38	0.018	
	Parental PTSD	0.01	0.06	0.23	0.820	
Model 2	Age	0.19	0.43	0.45	0.653	0.242
	Gender	-7.35	2.75	-2.67	0.008	
	Child baseline PTSD	0.53	0.20	2.66	0.008	
	Treatment condition	-4.75	2.04	-2.33	0.020	
	Parental PTSD	0.08	0.09	0.88	0.378	
	Interaction (treatment condition * parental PTSD)	-0.13	0.12	-1.08	0.282	
11-month post randomisation						
Parental depression (PHQ-9)						
Model 1	Age	-0.45	0.39	-1.16	0.246	0.379
	Gender	-7.83	2.61	-3.00	0.003	
	Child baseline PTSD	0.93	0.16	5.89	0.000	
	Treatment condition	-5.91	2.08	-2.84	0.004	
	Parental depression	-0.05	0.18	-0.29	0.769	
Model 2	Age	-0.39	0.39	-1.00	0.316	0.395
	Gender	-7.93	2.67	-2.97	0.003	
	Child baseline PTSD	0.92	0.16	5.79	0.000	
	Treatment condition	-6.11	2.07	-2.96	0.003	
	Parental depression	0.10	0.22	0.47	0.637	
	Interaction (treatment condition * parental depression)	-0.36	0.35	-1.05	0.296	
Parental anxiety (GAD-7)						
Model 1	Age	-0.44	0.40	-1.12	0.262	0.379
	Gender	-7.80	2.58	-3.02	0.003	
	Child baseline PTSD	0.90	0.16	5.78	0.000	
	Treatment condition	-5.89	2.06	-2.85	0.004	
	Parental anxiety	0.05	0.21	0.25	0.802	
Model 2	Age	-0.39	0.40	-0.98	0.327	0.406

	Gender	-8.41	2.69	-3.13	0.002	
	Child baseline PTSD	0.88	0.16	5.48	0.000	
	Treatment condition	-6.21	2.03	-3.06	0.002	
	Parental anxiety	0.29	0.27	1.07	0.287	
	Interaction (treatment condition * parental anxiety)	-0.57	0.41	-1.39	0.166	
Parental PTSD (PDS-5)						
Model 1	Age	-0.49	0.39	-1.26	0.208	0.400
	Gender	-7.63	2.46	-3.10	0.002	
	Child baseline PTSD	0.85	0.15	5.61	0.000	
	Treatment condition	-5.74	2.04	-2.82	0.005	
	Parental PTSD	0.10	0.06	1.53	0.125	
Model 2	Age	-0.47	0.40	-1.19	0.234	0.415
	Gender	-7.97	2.53	-3.16	0.002	
	Child baseline PTSD	0.82	0.16	5.13	0.000	
	Treatment condition	-5.99	1.99	-3.01	0.003	
	Parental PTSD	0.15	0.08	1.79	0.074	
	Interaction (treatment condition * parental PTSD)	-0.12	0.13	-0.95	0.344	

Appendix H. Moderating effects of parental baseline psychopathology (per-protocol sample)

Table H.1. *Moderating Effects of Parental Baseline Psychopathology (IV: Child Baseline PTSD)*

	Variable	*b*	SE	*z*	*p*	R^2
Post-treatment						
Parental depression (PHQ-9)						
Model 1	Age	0.19	0.50	0.38	0.706	0.214
	Gender	-4.89	2.92	-1.68	0.094	
	Child baseline PTSD	0.73	0.21	3.45	0.001	
	Parental depression	-0.15	0.17	-0.84	0.400	
Model 2	Age	0.18	0.50	0.35	0.724	0.217
	Gender	-5.05	2.94	-1.72	0.085	
	Child baseline PTSD	0.75	0.34	1.74	0.000	
	Parental depression	-0.15	0.17	-0.93	0.354	
	Interaction (child baseline PTSD * parental depression)	0.02	0.03	0.758	0.560	
Parental anxiety (GAD-7)						
Model 1	Age	0.17	0.48	0.36	0.720	0.212
	Gender	-4.86	2.88	-1.69	0.092	
	Child baseline PTSD	0.68	0.21	3.21	0.001	
	Parental anxiety	0.07	0.20	0.36	0.719	
Model 2	Age	0.20	0.49	0.40	0.686	0.223
	Gender	-4.95	2.84	-1.74	0.082	
	Child baseline PTSD	0.71	0.20	3.48	0.000	
	Parental anxiety	0.06	0.20	0.28	0.777	
	Interaction (child baseline PTSD * parental anxiety)	0.03	0.04	0.80	0.423	
Parental PTSD (PDS-5)						
Model 1	Age	0.18	0.48	0.37	0.713	0.211
	Gender	-4.81	2.88	-1.67	0.094	
	Child baseline PTSD	0.68	0.21	3.29	0.001	
	Parental PTSD	0.02	0.06	0.31	0.755	
Model 2	Age	0.17	0.48	0.35	0.723	0.216
	Gender	-4.94	2.86	-1.73	0.084	
	Child baseline PTSD	0.70	0.20	3.51	0.000	
	Parental PTSD	0.02	0.06	0.29	0.771	
	Interaction (child baseline PTSD * parental PTSD)	0.01	0.01	0.64	0.523	
11-month post randomisation						
Parental depression (PHQ-9)						
Model 1	Age	-0.57	0.45	-1.26	0.208	0.326
	Gender	-5.72	2.76	-2.07	0.038	
	Child baseline PTSD	1.05	0.17	6.24	0.000	
	Parental depression	-0.10	0.22	-0.47	0.638	
Model 2	Age	-0.62	0.45	-1.38	0.168	0.340
	Gender	-6.21	2.97	-2.10	0.036	
	Child baseline PTSD	1.11	0.18	6.08	0.000	
	Parental depression	-0.15	0.20	-0.76	0.445	

	Interaction (child baseline PTSD * parental depression)	0.03	0.04	0.69	0.489	
Parental anxiety (GAD-7)						
Model 1	Age	-0.55	0.46	-1.22	0.224	0.323
	Gender	-5.60	2.76	-2.03	0.043	
	Child baseline PTSD	1.02	0.17	5.93	0.000	
	Parental anxiety	0.02	0.24	0.09	0.932	
Model 2	Age	-0.56	0.46	-1.22	0.223	0.330
	Gender	-5.43	2.81	-1.93	0.053	
	Child baseline PTSD	0.97	0.20	4.98	0.000	
	Parental anxiety	0.08	0.23	0.33	0.745	
	Interaction (child baseline PTSD * parental anxiety)	-0.04	0.06	-0.66	0.512	
Parental PTSD (PDS-5)						
Model 1	Age	-0.55	0.44	-1.26	0.208	0.347
	Gender	-5.31	2.65	-2.00	0.045	
	Child baseline PTSD	0.96	0.17	5.54	0.000	
	Parental PTSD	0.10	0.08	1.31	0.189	
Model 2	Age	-0.55	0.44	-1.26	0.206	0.346
	Gender	-5.23	2.70	-1.94	0.053	
	Child baseline PTSD	0.94	0.20	4.74	0.000	
	Parental PTSD	0.11	0.07	1.41	0.159	
	Interaction (child baseline PTSD * parental PTSD)	-0.00	0.02	-0.22	0.829	

Table H.2. *Moderating Effects of Parental Baseline Psychopathology (IV: Treatment Condition)*

	Variable	*b*	*SE*	*z*	*p*	R^2
Post-treatment						
Parental depression (PHQ-9)						
Model 1	Age	0.31	0.46	0.69	0.494	0.261
	Gender	-6.04	2.88	-2.10	0.036	
	Child baseline PTSD	0.66	0.22	2.98	0.003	
	Treatment condition	-5.70	1.98	-2.88	0.004	
	Parental depression	-0.13	0.17	-0.74	0.462	
Model 2	Age	0.57	0.48	1.19	0.233	0.311
	Gender	-5.33	2.74	-1.95	0.052	
	Child baseline PTSD	0.68	0.23	3.01	0.003	
	Treatment condition	-5.93	2.02	-2.93	0.003	
	Parental depression	0.18	0.23	0.79	0.427	
	Interaction (treatment condition * parental depression)	-0.59	0.29	-2.07	0.039	
Parental anxiety (GAD-7)						
Model 1	Age	0.30	0.45	0.68	0.499	0.262
	Gender	-6.04	2.85	-2.12	0.034	
	Child baseline PTSD	0.61	0.22	2.74	0.006	
	Treatment condition	-5.80	2.01	-2.89	0.004	
	Parental anxiety	0.08	0.21	0.40	0.691	
Model 2	Age	0.43	0.46	0.93	0.351	0.292
	Gender	-5.90	2.78	-2.12	0.034	

	Child baseline PTSD	0.63	0.22	2.89	0.004	
	Treatment condition	-5.90	2.05	-2.88	0.004	
	Parental anxiety	0.34	0.27	1.25	0.213	
	Interaction (treatment condition * parental anxiety)	-0.51	0.39	-1.32	0.187	
Parental PTSD (PDS-5)						
Model 1	Age	0.31	0.45	0.68	0.494	0.260
	Gender	-5.99	2.84	-2.11	0.034	
	Child baseline PTSD	0.62	0.22	2.83	0.005	
	Treatment condition	-5.79	2.01	-2.88	0.004	
	Parental PTSD	0.02	0.06	0.30	0.762	
Model 2	Age	0.44	0.43	1.02	0.308	0.293
	Gender	-5.45	2.68	-2.03	0.042	
	Child baseline PTSD	0.62	0.23	2.73	0.006	
	Treatment condition	-5.70	2.04	-2.79	0.005	
	Parental PTSD	0.11	0.08	1.36	0.175	
	Interaction (treatment condition * parental PTSD)	-0.17	0.11	-1.55	0.121	
11-month post randomisation						
Parental depression (PHQ-9)						
Model 1	Age	-0.31	0.45	-0.70	0.485	0.388
	Gender	-6.64	2.87	-2.32	0.021	
	Child baseline PTSD	1.02	0.17	5.88	0.000	
	Treatment condition	-5.80	2.27	-2.56	0.010	
	Parental depression	-0.15	0.21	-0.70	0.486	
Model 2	Age	-0.25	0.45	-0.55	0.583	0.394
	Gender	-6.60	2.92	-2.26	0.024	
	Child baseline PTSD	1.01	0.18	5.48	0.000	
	Treatment condition	-5.94	2.27	-2.62	0.009	
	Parental depression	0.01	0.31	0.03	0.978	
	Interaction (treatment condition * parental depression)	-0.28	0.41	-0.69	0.492	
Parental anxiety (GAD-7)						
Model 1	Age	-0.32	0.46	-0.68	0.494	0.378
	Gender	-6.43	2.85	-2.25	0.024	
	Child baseline PTSD	0.99	0.18	5.62	0.000	
	Treatment condition	-5.68	2.26	-2.52	0.012	
	Parental anxiety	-0.03	0.25	-0.11	0.912	
Model 2	Age	-0.24	0.44	-0.54	0.590	0.401
	Gender	-7.00	3.01	-2.32	0.020	
	Child baseline PTSD	0.95	0.19	5.06	0.000	
	Treatment condition	-5.95	2.23	-2.67	0.008	
	Parental anxiety	0.28	0.37	0.75	0.456	
	Interaction (treatment condition * parental anxiety)	-0.56	0.49	-1.14	0.255	
Parental PTSD (PDS-5)						
Model 1	Age	-0.30	0.45	-0.68	0.497	0.395
	Gender	-6.13	2.76	-2.22	0.026	
	Child baseline PTSD	0.92	0.18	5.26	0.000	
	Treatment condition	-5.46	2.22	-2.46	0.014	
	Parental PTSD	0.09	0.07	1.25	0.213	

Model 2	Age	-0.24	0.43	-0.56	0.574	0.414
	Gender	-6.41	2.82	-2.28	0.023	
	Child baseline PTSD	0.87	0.20	4.38	0.000	
	Treatment condition	-5.74	2.19	-2.62	0.009	
	Parental PTSD	0.17	0.11	1.49	0.136	
	Interaction (treatment condition * parental PTSD)	-0.14	0.15	-0.91	0.363	