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CHANGE IN BLAME ATTRIBUTIONS AS A MEDIATOR OF SYMPTOM REDUCTION IN TRAUMA-FOCUSED COGNITIVE BEHAVIORAL THERAPY

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CHANGE IN BLAME ATTRIBUTIONS AS A MEDIATOR OF SYMPTOM REDUCTION IN TRAUMA-FOCUSED COGNITIVE BEHAVIORAL THERAPY

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ABSTRACT

CHANGE IN BLAME ATTRIBUTIONS AS A MEDIATOR OF SYMPTOM REDUCTION IN TRAUMA-FOCUSED COGNITIVE BEHAVIORAL THERAPY

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Youth exposed to interpersonal violence often develop maladaptive posttraumatic cognitions about the event(s), the world, and themselves. These maladaptive cognitions have robust associations with onset and maintenance of posttraumatic stress disorder (PTSD) and internalizing symptoms (Mitchell et al., 2017). Growing literature suggests changes in cognitions, particularly appraisals about control, vulnerability, and personal change following trauma exposure, serve as a mechanism of change in trauma-specific psychotherapy interventions for youth (Meiser-Stedman et al., 2017; Smith et al., 2007). Little research has examined change in blame attributions as a mediator of treatment outcome, despite their importance in understanding how youth process their trauma and are their robust associations with trauma-related symptoms (McGee et al., 2001). The current study sought to assess whether changes in self-blame and perpetrator blame mediate changes in PTSD, anxiety, and depression mid- and post-Trauma-Focused Cognitive Behavior Therapy (TF-CBT; Cohen, Mannarino, & Deblinger, 2006), a phase-based evidence-based intervention that addresses maladaptive cognitions through a variety of treatment components. Path models were analyzed to examine indirect effects

of changes in self-blame (N = 164) and perpetrator blame (N = 420) on symptom change in a diverse sample of youth ages 4 to 17. Results indicated that reductions in self-blame mediated decreases in anxiety and depressive symptoms during Phase I of TF-CBT, which focuses on stabilization, psychoeducation, and coping skills. Path models demonstrated that perpetrator blame mediated decreases in PTSD and anxiety following imaginal exposure and cognitive processing of the traumatic events in Phases II and III of TF-CBT. Findings highlight the importance of assessing and addressing attributions of blame toward the self and the perpetrator(s) of interpersonal violence throughout treatment.

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INTRODUCTION

Each year, more than 25% of children and adolescents in the United States are exposed to interpersonal violence including sexual abuse, physical abuse, witnessing domestic violence, and peer sexual assault (Finkelhor et al., 2009), often leading to impairing posttraumatic stress, internalizing, and externalizing symptoms (Alisic et al., 2015; Vachon et al., 2015). Following exposure to trauma, youth often develop maladaptive cognitions about the event(s), themselves, and the world which have shown to be associated with trauma-related psychopathology (Mitchell et al., 2017). Growing literature demonstrates that change in youths' posttraumatic cognitions, particularly appraisals about control, vulnerability, and personal change since the event, serves as a mediator of trauma-specific treatment outcomes (Meiser-Stedman et al., 2017; Smith et al., 2007). However, this literature has failed to examine blame attributions as a mechanism of change, despite robust associations between self-blame and a range of trauma-related psychopathology (Feiring et al., 2002). The current study aims to examine whether changes in self- and perpetrator blame attributions during the course of trauma-focused treatment mediate decreases in PTSD, anxiety, and depression symptom severity in a diverse sample of youth exposed to interpersonal trauma.

Maladaptive Posttraumatic Cognitions and Trauma-Related Psychopathology

Research has supported the role of cognitive theories in understanding the development and maintenance of trauma sequalae in children and adolescents (Meiser-Stedman et al., 2009; Mitchell et al., 2017; Trickey et al., 2012). Ehlers and Clark (2000) proposed that negative trauma appraisals contribute to an overgeneralized sense of current threat, which are further maintained by maladaptive cognitive and behavioral

coping strategies. This theory also posits that thoughts about one's reaction during the traumatic event may lead to generalized negative beliefs about the self and sense of autonomy, and that specific appraisals are associated with specific emotions such as fear, guilt, and sadness (Ehlers & Clark, 2000; Beck et al., 2014). Similarly, Resick and Schnicke's (1992) information processing model of trauma response patterns theorizes that the ways individuals integrate traumatic experiences into their schematic beliefs explains the development of PTSD symptoms. A review of theoretical models of PTSD etiology and maintenance found commonalities in processes considered to be significant in understanding PTSD development: negative thoughts about the self, negative thoughts about the world, negative beliefs about the meaning of PTSD symptoms, and perceptions of loss of control and autonomy (Beck et al., 2014).

Studies have demonstrated the links between post-traumatic cognitions and trauma-related symptoms in youth. Meta-analyses have demonstrated large effect sizes for the association between negative appraisals about trauma and PTSD symptoms in youth (Gómez de La Cuesta et al., 2019; Mitchell et al., 2017). More specifically, attributions of blame, perceived threat, and thought suppression (i.e., attempts to stop unwanted thoughts) have shown medium to large effect sizes in the prediction of posttraumatic stress disorder (Trickey et al., 2012). Maladaptive cognitive appraisals have also been shown to be a critical mechanism in the development and maintenance of depressive symptoms following child maltreatment (Bassani et al., 2013). Understanding the etiology and maintenance of PTSD and internalizing symptoms following trauma exposure informs intervention design and helps to discern why specific interventions are effective.

Cognitive Change as a Mechanism of Change in Treatment

Growing evidence suggests that cognitive change is a mediator of PTSD treatment outcomes for youth. Smith and colleagues (2007) found that the effects of a 10-week course of cognitive behavioral therapy (CBT) were partially mediated by changes in maladaptive cognitions in a sample of 24 children and adolescents who experienced a single-incident traumatic event. Additionally, a study comparing prolonged exposure and client-centered therapy for adolescent sexual assault survivors demonstrated that change in maladaptive posttraumatic cognitions mediated change in PTSD and depressive symptoms in both interventions, whereas change in PTSD and depressive symptoms did not mediate change in cognitions (McLean et al., 2015). Furthermore, Meiser-Stedman and colleagues (2017) showed that decreases in PTSD, anxiety, and depression following cognitive therapy for PTSD were mediated by changes in maladaptive posttraumatic cognitions in children exposed to accidental trauma. A mixed method study showed that impact statements written following cognitive processing therapy adapted for adolescents contained fewer overgeneralized and inaccurate statements at post-treatment compared to pre-treatment, and that increases in adaptive thoughts about the trauma were associated with decreased PTSD and depressive symptoms (König et al., 2019).

Additional research suggests that behavioral interventions may be mediated by cognitive changes as well. Research with adults has demonstrated that trauma-specific cognitive behavioral interventions that include exposure elements effectively reduce posttraumatic cognitions regardless of whether they include cognitive restructuring elements (Diehle et al., 2014). This finding suggests that behavioral components such as exposure may be effective through changes in learning and cognition. This is further

explained by the inhibitory learning model, which suggests that through exposure to feared stimuli, the original feared association remains but is inhibited by a newly learned association representing safety and a reduction in overgeneralization of threat (Craske et al., 2012). Furthermore, researchers have suggested a cognitive mechanism of action in the efficacy of relaxation training, positing that the decrease in anxiety symptoms may occur through distraction or development of a sense of control (Garssen et al., 1992).

Change in Blame Attributions as a Mediator of Treatment Outcome

Attributions of blame, a specific type of posttraumatic cognition, are key components in understanding how youth process their trauma and are predictors of subsequent psychopathology (Feiring et al., 2002; McGee et al., 2001). Following interpersonal violence, youth most often tend to blame themselves and the individual(s) who perpetrated the abuse (Feiring & Cleland, 2007). Self-blame is the most commonly studied blame attribution following child trauma. The extant literature on self-blame demonstrates robust positive associations with higher PTSD and internalizing symptoms in maltreated youth (Brown & Kolko, 1999; Feiring & Cleland, 2007; Feiring et al., 2002, Mannarino & Cohen, 1996; McGee, et al., 2001). Furthermore, general self-blame attributions have been shown to mediate the relation between abuse-specific self-blame and posttraumatic stress, internalizing symptoms, and anger in sexually abused adolescents (Daigneault et al., 2006). This finding suggests that those who blame themselves for their abuse exhibit poorer outcomes through the generalization of self-blame to other life events. Sharma-Patel and Brown (2016) found that decreases in self-blame between pre- and post-TF-CBT mediated decreases in caregiver-reported externalizing behavior problems in youth exposed to interpersonal violence,

demonstrating the necessity of investigating blame attributions and their relation to symptoms throughout treatment.

The reformulation of the learned helplessness theory (Abramson et al., 1978; Shapiro et al., 1995) suggests that internal, temporally stable, and global (i.e., affecting a variety of outcomes) attributions are more associated with negative outcomes than the opposite dimensions (i.e., external, unstable, and specific). Given that self-blame is robustly predictive of maladjustment, it is critical to investigate whether external blame attributions, such as blame directed at the perpetrator of the interpersonal violence, are adaptive in processing traumatic events. This is especially important given research suggesting clinicians often encourage victims to attribute blame for abuse to the offender (Celano et al., 2002; Shapiro, 1995). The few studies that have examined the relation between perpetrator blame and trauma-related psychopathology have yielded inconsistent results. Feiring et al. (2002) assessed children and adolescents at the time of child sexual abuse discovery, prior to them receiving treatment. Results indicated that higher perpetrator blame was significantly associated with lower internalizing symptoms, suggesting a protective relationship. In a cross-sectional study, Canton-Cortes, Canton, and Cortes (2012) recruited a sample of college students to complete a retrospective measure of child sexual abuse history, the Attributions of Responsibility and Blame Scale (McMillen & Zuravin, 1997), and the Severity of Symptoms of PTSD Scale (Echeburua et al., 1997). No significant relation between perpetrator blame and PTSD was found. The authors suggest that encouraging perpetrator blame during treatment may not be effective for symptom reduction. Notably, these two studies were specific to child sexual abuse, and did not assess history of or blame attributions for other types of maltreatment.

A study with youth exposed to physical abuse demonstrated that blame attributions toward the perpetrator or others involved in the trauma showed a trend toward an association with externalizing symptoms (Brown & Kolko, 1999). Sharma-Patel and colleagues (2014) performed a cluster analysis of blame attributions in multiply maltreated youth. Results revealed that those with a high self-blame and high perpetrator blame attribution pattern reported poorer treatment outcomes compared to youth with other blame profiles. Furthermore, a recent mixed method study collected responses from adult interpersonal trauma survivors by asking open-ended questions about why they believe their traumatic event occurred (Reich et al., 2023). The authors found that the degree of attributions of blame toward the self and perpetrator was not reliably associated with posttraumatic distress. These inconsistent findings may be explained by the varying samples, methodology, and measures used. Due to the lack of literature and theory in this area, it is currently unclear whether perpetrator blame can be considered an accurate and adaptive belief, or an unhelpful cognitive process associated with symptoms. Additional research is needed to understand how perpetrator blame may influence or serve as a protective factor against trauma-related symptoms in multiply maltreated youth throughout treatment.

Change in Maladaptive Posttraumatic Cognitions as a Mediator of TF-CBT Outcome

Trauma-focused cognitive behavioral therapy (Cohen, Mannarino, & Deblinger, 2006), is an evidence-based intervention for children and adolescents exposed to trauma that has been shown to effectively reduce internalizing, externalizing, and posttraumatic stress disorder (PTSD) symptoms in diverse samples of children and adolescents (Dorsey

et al., 2020; Hoogsteder et al., 2021; Lenz & Hollenbaugh et al., 2015; McGuire et al., 2021; Peters et al., 2021; Silverman et al., 2008). TF-CBT is a phase-based treatment in which treatment components are summarized with the acronym PRACTICE. Phase I (PRAC) focuses on safety and stabilization and includes Psychoeducation about trauma exposure, trauma reminders, and treatment, Parenting skills, Relaxation skills, Affect modulation, and Cognitive coping (Cohen, Mannarino, & Deblinger, 2017). In this treatment phase, provision of corrective psychoeducation about exposure to trauma and typical trauma reactions, as well as implementation of cognitive coping strategies may be used to directly address youths' general maladaptive thinking patterns (Cohen, Mannarino, & Deblinger, 2017). Phase II (TI) includes gradual exposure through Trauma narration, an extensive iterative exposure process in which youth describe their trauma experiences with increasing detail, cognitive restructuring of maladaptive trauma-specific cognitions, and In-vivo exposure of trauma reminders. Phase III (CE) highlights consolidation and integration through Conjoint parent-child sessions and reviewing strategies to Enhance future safety (Cohen, Mannarino, & Deblinger, 2017). In a dismantling study comparing length of TF-CBT, as well inclusion versus exclusion of exposure components, Deblinger and colleagues (2011) found that youth across all conditions exhibited significant decreases in symptoms. The authors suggest that children's cognitive processing of their traumatic experiences, which occurs throughout several cognitive and behavioral elements of both phases of treatment, is a critical mechanism for improved outcomes. It is unclear from extant literature which phase of treatment and which specific set of treatment components contribute to the greatest change in cognitions and subsequent posttraumatic stress symptoms.

Despite the empirical support for the importance of cognitive change in trauma-specific treatment, few studies have examined whether changes in dysfunctional posttraumatic cognitions mediated TF-CBT treatment outcome. Pfeiffer et al. (2017) assessed 123 children and adolescents at pre- and post-treatment using the Child Posttraumatic Cognition Inventory (CPTCI; Meiser-Stedman et al., 2009), which consists of 25 questions that produce two subscales: Permanent and Disturbing Change and Fragile Person in a Scary World. The items on the CPTCI assess perceived coping efficacy, sense of threat, and negative and permanent change in individuals following the trauma. Data from youth who attended at least 8 sessions of TF-CBT were included in the analysis and compared to youth on the waitlist. Bootstrap mediation analyses indicated that changes in posttraumatic misappraisals mediated the relation between treatment condition and posttraumatic stress symptoms (PTSS) at post-treatment, demonstrating that change in dysfunctional cognitions is a critical mechanism of action in TF-CBT. Similarly, Jensen et al. (2018) examined whether change in maladaptive posttraumatic cognitions mediated the relation between treatment condition and PTSS, depression, and general mental health. Participants in this study were randomized to either TF-CBT or treatment as usual in one of eight community clinics. Unlike Pfeiffer et al. (2017), Jensen and colleagues collected data at pre-treatment (Time 1), mid-treatment (Time 2), and post-treatment (Time 3). Mid-treatment assessments were given after 6 sessions and post-treatment assessments were given after 15 sessions. Results indicated that decreases in posttraumatic cognitions, as measured by the CPTCI, mediated the effect of TF-CBT on PTSS, depressive symptoms, and general mental health. Results also showed that the mediation effect occurred late in treatment, during sessions 7-15 (i.e., between Time 2

and Time 3), when trauma-specific cognitions are more likely to be restructured. However, because the mid-treatment assessment timepoint was based on session number rather than treatment phase, it is unclear which treatment components contributed to the mediation effect. Although both Pfeiffer et al. (2017) and Jensen et al. (2018) demonstrate the critical mediating role of maladaptive cognitions in treatment outcome, they fail to assess how blame attributions relate to change in symptoms in diverse samples of multiply maltreated youth. Only one known study has examined change in blame attributions as a mediator of symptom change in TF-CBT. Sharma-Patel and Brown (2016) examined how self-blame and emotion dysregulation mediated and moderated changes in PTSD and conduct problems following completion of trauma-specific CBTs for youth. Results showed that decreases in self-blame both mediated and moderated caregiver-reported conduct problems in a sample of 118 children and adolescents.

Current Study

Despite the evidence that cognitive changes mediate TF-CBT outcomes, there is limited extant literature examining how blame attributions explain symptom reductions throughout treatment. Both Pfeiffer et al. (2017) and Jensen et al. (2018) highlight the significance of addressing posttraumatic cognitions in TF-CBT. However, neither study examined cognitions related to who youth believe are responsible for their exposure to violence and trauma sequalae. The current study will build upon the results of Sharma-Patel and Brown (2016) by including a larger sample, adding a mid-treatment assessment timepoint based on treatment phase, and examining the potential mediation effects of change in perpetrator blame in addition to self-blame. Although Jensen et al., (2018)

demonstrated that mediation effects occurred late in treatment, it is unclear which TF-CBT components were administered before or after the mid-treatment assessment, given that the date of assessment was based on number of sessions rather than treatment component. It is critical to assess which treatment components in TF-CBT contribute significantly to changes in blame attributions, and whether these changes mediate changes in psychopathology. In addition to adding to our understanding of mechanisms of TF-CBT efficacy, this knowledge may help guide psychoeducation to increase treatment engagement and inform clinical decisions. Finally, studies have largely excluded ethnically and socio-economically diverse samples of youth with complex trauma histories. To address the gaps in the literature, the current study will examine whether changes in self-blame and/or perpetrator blame mediate changes in PTSD, anxiety, and depression symptom severity in a diverse sample of multiply traumatized youth. Specifically, analyses will examine mediation effects between pre-treatment (Time 1) and mid-treatment (Time 2), and between mid-treatment (Time 2) and post-treatment (Time 3). These three timepoints separate the phases of TF-CBT, providing insight into which treatment components contribute to the potential mediation effect. The current study will address the following aims:

Aim 1: to examine change in self-blame and perpetrator blame throughout the trajectory of TF-CBT.

Hypothesis 1: Self-blame will decrease minimally between Time 1 and Time 2 and will decrease significantly between Time 2 and Time 3. The exploratory hypothesis that perpetrator blame will increase minimally

between Time 1 and Time 2 and will significantly increase between Time 2 and Time 3 will be tested.

Aim 2: to examine whether changes in self-blame and perpetrator blame from Time 1 to Time 2 (Phase I) mediate changes in trauma-related symptom severity at Time 2.

Hypothesis 2: Changes in self-blame and perpetrator blame between Time 1 and Time 2 will not significantly mediate symptom change at Time 2.

Aim 3: to examine whether changes in self-blame and perpetrator blame from Time 2 to Time 3 (Phases II and III) mediate changes in trauma-related symptom severity at Time 3.

Hypothesis 3: Although several treatment components from all phases of the intervention may contribute to change in blame attributions and subsequent change in symptoms, given that trauma-specific cognitive restructuring occurs during Phase II (between Time 2 and Time 3), there will be significant changes in blame attributions, which will significantly mediate change in trauma-specific symptoms at Time 3.

METHODS

Power Analysis

Given the current study used data from an ongoing effectiveness study, post hoc power analyses were conducted using R studio Version 4.0.2 following procedures detailed by Moshagen and Erdfelder (2016). Calculations for all included models were based on $\alpha = .05$, RMSEA = .05, power = .80, and df = 8. Given the varied sample size used for each model, this study is between 11% and 50% powered. Thus, this study is underpowered to detect small effects.

Participants

Participants were drawn from an ongoing study examining the effectiveness of TF-CBT for youth ages 4 through 17 with a history of interpersonal trauma and their caregivers. Participants were included in the research study if they endorsed child physical abuse, child sexual abuse, witnessing domestic violence, peer sexual assault, or traumatic bereavement. Additional inclusion criteria included self- or caregiver-report of sub-threshold or clinically elevated internalizing, externalizing, or PTSD symptoms as well as the presence of a caregiver willing to participate in treatment with the child or adolescent. Exclusion criteria included presence of significant cognitive impairment that would prevent participation in the treatment components or acute symptoms that require stabilization at a higher level of care.

In the overall sample ($N = 420$) youth ranged from 4 to 17 years of age at pre-treatment (M = 12.00, SD = 3.69) and were 72% female. The sample was racially and ethnically diverse, with 79.5% of youth identifying as Hispanic/Latine, African American/Black, Multiracial, Guyanese, and Asian, 8.2% identifying as “Other,” and

7.9% identifying as Caucasian. The majority of youth (63%) endorsed histories of multiple interpersonal traumas (M = 1.88, SD = 0.88). Table 1 details the demographic characteristics of study participants.

Measures

Child and Family Demographics. Caregivers provided demographic information at the pre-treatment evaluation. Adolescents reported their gender identity at pre-treatment.

Blame Attributions. Blame attributions regarding interpersonal trauma were assessed using the PERceptions of Children Exposed to Interpersonal ViolencE-Short Form (PERCEIVE; Brown, 2006), which includes three open-ended questions followed by 38 quantitative items. Youth were asked which of the traumatic events endorsed they found most upsetting. Youth then indicated how true each statement is for them based on a 3-point scale (0 = *not at all*, 1 = *a little bit*, 2 = *a lot*). Sample items include "This happened because I'm not a good kid," "This happened because [perpetrator] is a bad person," "This happened because [perpetrator] had a bad day," and "This happened because sometimes accidents happen."

An exploratory factor analysis on the PERCEIVE revealed a 5-factor solution for children and adolescents. Items with factor loadings below 0.4 were removed from subscales. Factors included Self-Blame, Perpetrator Dispositional Blame, Perpetrator Situational Blame, General Accident/Situational Blame, and Family/Neighborhood Blame. Perpetrator dispositional blame refers to attributes of the perpetrator (e.g., "This happened because [perpetrator] is mean,") whereas perpetrator situational blame refers to the situation that led the perpetrator to inflict the abuse (e.g., "This happened because

[perpetrator] had a stressful day at work.") For the purposes of the current study, the Self-Blame and Perpetrator Dispositional Blame (8 items) factors were used as blame attribution subscales. The Self-Blame subscale contains nine items that include both global self-blame ("This happened because I am a bad kid,") and situation-specific self-blame ("This happened because I did something wrong") items. The Perpetrator Blame scale contains seven items related to stable traits of the perpetrator. Sample items include ("This happened because [perpetrator] likes to hurt people," and "This happened because [perpetrator] is a bad person." Each item was summed to obtain a total score for each of the two subscales. Internal reliability was good for both the self-blame and perpetrator blame subscales (α = .87 and α = .86, respectively).

PTSD Symptom Severity. The Child PTSD Symptom Scale (CPSS; Foa et al., 2001; Foa et al., 2018) is a self-report measure of PTSD symptom severity for children ages 8 to 18. The CPSS-4 includes 17 items which form re-experiencing, avoidance, and hyperarousal subscales consistent with DSM-IV PTSD criteria. Children and adolescents rated how frequently they experienced each symptom in the past two weeks on a 4-point scale (0 = *Not at all or only one time* to 3 = *5 or more times a week/almost always*). The CPSS-4 demonstrates excellent internal consistency (α = .87), and test-retest reliability (r = .86; Foa et al., 2001). The revised CPSS-5 includes 20 items and includes the additional cognition and mood subscale consistent with DSM-5 PTSD symptom clusters. Children and adolescents rated how frequently they experienced each symptom in the past month on a 5-point scale (0 = *Not at all* to 4 = *6 or more times a week or almost always*). The CPSS-5 demonstrates excellent internal consistency (α = .92) and good test-retest reliability (r = .90; Foa et al., 2018). Total scores were calculated by summing all

response items. Approximately seventy-two percent of study participants completed the CPSS-4, and the remaining percentage completed the CPSS-5. Due to this discrepancy, the total CPSS-4 and CPSS-5 scores were converted to z-scores to yield PTSD symptom severity scores for use in the current study. Internal consistency for the CPSS-4 (17 items) and CPSS-5 (20 items) subscales were acceptable (.90 and .91, respectively). Research assistants attempted administration of the CPSS with 68 youth under eight years of age and determined on an individual basis if the youth could understand and answer the questions in a meaningful way. CPSS data was kept for 47% of these individuals, with the youngest being five years old.

Depression and Anxiety Symptom Severity. The Behavioral Assessment System for Children (BASC-3; Reynolds & Kamphaus, 2015) is a comprehensive measure of emotional and behavioral symptoms in youth. The BASC-3 Self-Report of Personality (SRP) Child Form contains 137 items and is administered to children ages 8 to 11. The BASC-3 SRP Adolescent Form contains 189 items and is administered to adolescents ages 12 to 21. The Depression and Anxiety Subscale scores were used to measure depression and anxiety symptom severity. The Anxiety Subscale on the child form is made up of 11 items whereas the Anxiety Subscale on the adolescent form is made up of 13 items. The Depression Subscale consists of 10 items on the child form and 12 items on the adolescent form. The BASC-3 Self-Report demonstrates adequate to excellent psychometric properties (Konold & Medway, 2017). Estimates of internal consistency for the self-report English form ranged between form ranged between .70 and .80, with higher values above .90 for composite scores such as the Internalizing Composite (Konold & Medway, 2017). Test-retest reliability for subscales on the SRP

ranged from .59 to .87, with lower scores for the youngest group of respondents (Konold & Medway, 2017). Research assistants attempted administration of the BASC-3 Self-Report Child Form with 68 youth under eight years of age and determined on an individual basis if the youth could understand and answer the questions in a meaningful way. For both subscales, data was kept for 53% of these individuals, with the youngest being five years old.

Trauma-Focused Cognitive Behavioral Therapy

Youth participated in trauma-focused CBT, an evidence-based intervention for youth ages 3 to 18 (and their non-offending caregivers) that targets PTSD and other trauma-related symptoms (Cohen, Mannarino, & Deblinger, 2017). Treatment components include psychoeducation, parenting skills, relaxation skills, affect modulation, cognitive coping, trauma narration, trauma-specific cognitive restructuring and processing, conjoint parent-child sessions, in-vivo exposure of trauma reminders, and strategies to enhance future safety (Cohen, Mannarino, & Deblinger, 2017). TF-CBT is typically administered in 12-20 sessions for children and adolescents with a range of traumatic experiences including accidental trauma and interpersonal violence (Cohen, Mannarino, & Deblinger, 2017).

Postdoctoral fellows and doctoral level clinical and school psychology students served as TF-CBT clinicians in a community clinic under the supervision of licensed clinical psychologists. All clinicians received training in the intervention by a TF-CBT national trainer and participated in weekly supervision to ensure treatment fidelity.

Procedure

The St. John's University Institutional Review Board approved all study procedures. Referral sources included local child advocacy centers, mental health agencies, schools, and community organizations. Each referral was administered a phone screen by the intake coordinator to assess eligibility. Families deemed ineligible for the study were given referrals to appropriate mental health agencies. If deemed eligible for the study, families were scheduled for a pre-treatment assessment.

Masters and doctoral-level research assistants conducted pre- (Time 1), mid- (Time 2), and post-treatment (Time 3) assessments. Pre-treatment assessments were given prior to the first psychotherapy session. Mid-treatment assessments occurred after the completion of Phase I of TF-CBT, following stabilization and prior to beginning trauma narration. Post-treatment assessments occurred after both Phase II and Phase III were completed. At the pre-treatment assessment, informed consent was collected from caregivers and assent was obtained from youth. Research assistants administered measures to youth and caregivers separately through interviews at all time points. Demographic information and data regarding lifetime trauma history was collected at pre-treatment. Measures assessing child symptomatology and posttraumatic cognitions were administered at pre-, mid-, and post-treatment. Youth received $10 gift cards for completion of each assessment and caregivers received between $15 and $20 depending on the type of assessment.

Data Analytic Plan

Preliminary analyses including descriptive statistics, data transformations, and missing data analyses were conducted in R Studio Version 4.0.2. All symptom severity

variables were transformed into Z-scores to match scaling of the CPSS Z-scores. In order to retain as many participants as possible given rates of missing data, paired samples *t*-tests were used to examine change in blame attributions throughout the phases of treatment. Path models were analyzed to examine mediation effects of changes in self-blame on changes in PTSD (Model 1), anxiety (Model 2), and depression (Model 3). Additional path models were analyzed to assess whether changes in perpetrator blame mediated changes in PTSD (Model 4), anxiety (Model 5), and depression (Model 6; See Figures 1-3). These models contain all intent-to-treat (ITT) participants who completed baseline (i.e., Time 1) measures. Each model assessed mediation effects between Time 1 and Time 2, and between Time 2 and Time 3. Fit indices (i.e., CFI, TLI and RMSEA) and parameter estimates (i.e., factor loadings) were used to assess model fit. The same analyses were conducted with s of participants who completed treatment (i.e., study completers; see Appendices E and F).

Descriptive statistics demonstrated that only 50.3% of the sample endorsed self-blame at pre-treatment. Thus, models including self-blame scores as the mediator included only the subsample of participants who endorsed self-blame at pre-treatment. The sample that endorsed self-blame had significantly higher anxiety, depression, and PTSD scores, indicating a more indicating a more clinical sample, consistent with previous research (Feiring & Cleland, 2007). The sample was also significantly older, suggesting that adolescents may be more likely to disclose self-blame prior to treatment. The groups did not differ in their experience of trauma characteristics.

Age, gender, and trauma type have been identified in previous literature as being associated with improvement in PTSD and internalizing symptoms during trauma-

specific treatment (Danzi & LaGreca, 2020; Kane et al., 2016). Child age at pre-treatment, gender identity, and trauma type identified as the most upsetting to the child (i.e., reference trauma) were selected and analyzed as potential covariates. Despite previous research, none of the covariates were significantly related to change in PTSD, anxiety, or depression at Time 2 or Time 3 ($p = .053 – .805$). Given the path models with covariates had poor fit and most covariates were not associated with change in any outcome variables, the covariates were excluded from analyses in favor of a more parsimonious model (see Appendix H).

Among the total number of participants ($n = 420$), 176 completed treatment, 159 dropped out after attending at least one therapy session, 31 never initiated treatment after completing the pre-treatment assessment, 49 did not complete the pre-treatment assessment, and 5 did not meet criteria for the study after completing pre-treatment. Missing data percentages on the BASC-3 anxiety and depression severity subscales ranged from 50.2% to 82.1% between Time 1 and Time 3. Missing data percentages on the CPSS-4 and CPSS-5 PTSD severity subscales ranged between 17.6% and 65.2% between Time 1 and Time 3. Missing data percentages on the PERCEIVE subscales ranged between 22.6% and 69.0%. Although Little's Missing Completely at Random test (Little, 1988) suggested that these data were likely missing completely at random, $\chi^2 (515, N = 420) = 520.15$, $p = .313$, older participants and females had greater levels of missingness on symptom and blame attribution scales at pre-treatment than younger participants and males, respectively ($p < .001$). These results suggest that results are more generalizable to younger clinical samples. Although females had more attrition, the sample is disproportionately female. Missingness at Time 2 and Time 3 was not

significantly associated with child-reported PTSD, anxiety, or depression symptom severity at Time 1 (p values ranged from .13 to .93). Maximum likelihood with robust standard errors was used to estimate missing data in R Studio (Maydeu-Olivares, 2017). Path analyses were conducted with all participants who completed baseline measures (self-blame model $N = 164$; perpetrator blame model $N = 420$). See Appendices E and F for analyses conducted with treatment completers (self-blame model $N = 80$; perpetrator blame model $N = 176$).

RESULTS

Correlations Among Study Variables

Descriptive statistics of study variables are displayed in Table 2. Table 4 details relations between blame attributions and symptom variables. All symptom severity scores from each timepoint were significantly positively correlated with all other symptom severity scores at all other timepoints (p = .039 – < .001). Self-blame scores were significantly positively correlated with self-blame scores at all other timepoints (p < .001), and perpetrator blame scores were significantly positively correlated with perpetrator blame scores at all other timepoints (p < .001). Self-blame and perpetrator blame scores were not significantly correlated with each other at any timepoint (p = .257 – .888).

Change in Blame Attributions

Results of paired samples t-tests (see Table 3) showed self-blame decreased significantly from Time 1 to Time 2 $t(115) = -2.05$, $p < .05$ and from Time 2 to Time 3 $t(83) = -2.44$, $p < .05$, both with small effect sizes (d = -.19 and -.26, respectively). Perpetrator blame increased slightly from Time 1 to Time 2 $t(112) = 0.68$, $p = .50$ and decreased from Time 2 to Time 3 $t(78) = -0.64$, $p = .52$. Despite the increase at Time 2, perpetrator blame decreased slightly from Time 1 to Time 3 $t(122) = -1.06$, $p = .29$. Specifically, of the 77 individuals who had complete perpetrator blame attribution data for all three timepoints, 26% demonstrated the pattern of a decrease in perpetrator blame between Time 1 and Time 2 and then an increase between Time 2 and Time 3. Contrastingly, twenty-six percent demonstrated an increase between Time 1 and Time 2 and then a decrease between Time 2 and Time 3. Additionally, 9.1% of individuals

showed a steady increase in perpetrator blame over the three timepoints, whereas 7.8% showed a steady decrease. For 5.2% of individuals with complete data, perpetrator blame remained the same from Time 1 to Time 2, and then decreased from Time 2 to Time 3. Conversely, perpetrator blame decreased from Time 1 to Time 2 and then remained the same from Time 2 to Time 3 for 11.7% of youth who reported perpetrator blame at all timepoints. Additionally, perpetrator blame remained the same between Time 1 and Time 2 and then increased between Time 2 and Time 3 for 6.5% of individuals. Perpetrator blame increased from Time 1 to Time 2 and then remained the same from Time 2 to Time 3 for 6.5% of youth. Finally, 1.3% of the sample with complete attribution data reported no change. Despite the lack of significant change in perpetrator blame, the path models examining indirect effects of perpetrator blame were analyzed due to the heterogeneity in patterns of scores throughout treatment.

Direct and Indirect Effects of Self-Blame Models

Path analyses were analyzed to test for indirect effects of change in self-blame between Time 1 and Time 2 on symptom change at Time 2, and indirect effects of change in self-blame between Time 2 and Time 3 on symptom change at Time 3. See Table 6 for summary of indices of fit and parameter estimates. Across all self-blame models, self-blame at Time 1 predicted self-blame at Time 2, which predicted self-blame at Time 3. As expected, the same pattern was demonstrated with perpetrator blame. Additionally, in all models, the symptom type at Time 1 predicted the same symptom type at Time 2, which predicted the symptom at Time 3.

Model 1, which analyzed change in self-blame as a mediator of change in PTSD symptoms, demonstrated acceptable fit (CFI = .98, TLI = .96, RMSEA = .05; see Figure

1). Notably, Model 1 also showed a significant direct pathway from self-blame at Time 3 to PTSD at Time 3 (B = .26, SE = .09, β = .83, p < .001). The pathway between self-blame at Time 2 and PTSD Time 2 was not significant. The indirect effect of the change in self-blame between Time 2 and Time 3 on PTSD change at Time 3 approached significance (p = .07). No other indirect effects were significant.

Fit indices for Model 2, (see Figure 2) which examined direct and indirect effects of change in self-blame on anxiety symptom change, were inconsistent (CFI = .93, TLI = .87, RMSEA = .09; see Figure 2). Significant direct effects included self-blame at Time 2 to anxiety at Time 2 (B = .18, SE = .08, β = .19, p < .05). The pathway between self-blame at Time 3 and anxiety at Time 3 was not significant. The mediation effect between Time 1 and at Time 2 was significant (B = .13, SE = .06, β = .13, p < .05) as was the total indirect effect (B = .20, SE = .07, β = .20, p < .05).

Model 3 examined direct and indirect effects of change in self-blame on change in depression. Fit indices demonstrated acceptable fit (CFI = .96, TLI = .93, RMSEA = .06; See Figure 3). All direct pathways were significant including self-blame at Time 2 to depression at Time 2 (B = .20, SE = .10, β = .55, p < .05) and self-blame at Time 3 to depression at Time 3 (B = .37, SE = .14, β = .38, p < .05). The mediation effect between at Time 1 and at Time 2 was significant (B = .15, SE = .07, β = .14, p < .05), as was the total indirect effect (B = .32, SE = .09, β = .31, p < .05).

Due to heterogeneity in patterns of change in perpetrator blame, path models were analyzed to test for indirect effects of perpetrator blame between Time 1 and Time 2 on symptom change at Time 2, and indirect effects of perpetrator blame between Time 2 and Time 3 on symptom change at Time 3 (See Table 6 for fit indices and estimates.) Model

4 examined perpetrator blame (PB) as a mediator of change in PTSD symptoms. Fit indices were acceptable (CFI = .95, TLI = .91, RMSEA = .08; see Figure 1). Significant pathways included PB at Time 3 to PTSD at Time 3 (B = .22, SE = .07, β = .21, p < .05). The pathway between PB at Time 2 to PTSD at Time 2 was not significant. The mediation effect between at Time 2 and at Time 3 was significant (B = .16, SE = .05, β = .16, p < .05), as was the total indirect effect (B = .21, SE = .07, β = .20, p < .05).

Model 5 examined the direct and indirect effects of perpetrator blame on changes in anxiety. Fit indices for this model were inconsistent (CFI = .94, TLI = .89, RMSEA = .10; see Figure 2). Significant direct effects included PB at Time 3 to anxiety at Time 3 (B = .14, SE = .07, β = .15, p < .050. The pathway from PB at Time 2 to anxiety at Time 2 was not significant. The mediation effect between at Time 2 and at Time 3 was significant (B = .11, SE = .05, β = .11, p < .05), as was the total indirect effect (B = .15, SE = .07, β = .15, p < .05).

Model 6 examined PB as a mediator of change in depression symptoms. Fit indices were acceptable (CFI = .96, TLI = .93, RMSEA = .07; see Figure 3). The pathways from PB at Time 2 to depression at Time 2 and PB at Time 3 to depression at Time 3 approached significance (B = .13, SE = .07, β = .13, p =.05; B = .14, SE = .07, β = .15, p = .05, respectively). The overall indirect effect was significant (B = .20, SE = .07, β = .21, p < .05). Both mediation effects approached significance (at Time 1 to at Time 2 B = .10, SE = .05, β = .09, p = .05; at Time 2 to at Time 3 B = .10, SE = .05, β = .11, p = .06).

Additional models (Models 1B-6B) were fit with study completers only to assess differences between the total sample versus those who completed treatment. The overall

pattern of results were mostly consistent with the exception of some findings that fell out of significance and fit indices that changed slightly from acceptable to poor, likely due to loss of power and lower sample size in an already underpowered study. The estimates had the same sign but no longer statistically significant.

DISCUSSION

The aims of the current study were to examine the change in blame attributions throughout the phases of TF-CBT and assess whether these changes mediate treatment outcomes at mid- (i.e., Time 2) and post-treatment (i.e., Time 3). Path models were analyzed to assess whether changes in self- and perpetrator blame attributions served as a mechanism of change in symptoms of PTSD, anxiety, and depression in a diverse sample of multiply maltreated youth. Despite much research on cognitive mechanisms of change in trauma-focused treatment, the current study is the first to examine the timing of mediation effects for both self- and perpetrator blame in relation to change in a number of trauma-related symptoms. The complex findings of this study have significant implications for treatment engagement and implementation.

Change in Blame Attributions

At baseline, about half of the sample did not endorse self-blame. One potential explanation of this finding is that youth entering treatment may have already been exposed to corrective education about who is responsible for perpetrating maltreatment from family members, child welfare caseworkers, teachers, or other adults in the community. Furthermore, the format of the blame attribution measure, which starts with open-ended questions followed by scaled items, may have influenced the endorsement of self-blame, as research has shown that youth are less likely to endorse self-blame on open-ended measure (Feiring & Cleland, 2007). Previous studies have found inconsistent results regarding age differences in endorsement of self-blame following traumatic events (Hazzard et al., 1995; Hunter et al., 1992; Sharma-Patel et al., 2014). The current study demonstrated that older youth were more likely to endorse self-blame at baseline than

younger youth, suggesting that adolescents may have a higher capacity to develop and express complex cognitions and attributions about interpersonal violence (Cicchetti & Rogosch, 2002).

Contrary to hypotheses, self-blame decreased significantly following Phase I and following Phases II and III of TF-CBT. This finding suggests that in addition to trauma-specific cognitive restructuring, other elements of treatment such as psychoeducation about trauma (e.g., who is responsible for maltreatment, common reactions to trauma, etc.), or general cognitive coping strategies may be effective at reducing self-blame (Shapiro, 1995). Kletter and colleagues (2009) found that children and adolescents exposed to interpersonal violence often question their behaviors following the traumatic events and how their responses may have led to or prolonged the violence. A lack of understanding of common complex reactions to trauma, including arousal, flight, fight, freeze, appeasement behavior, or tonic immobility, can result in self-blame, shame, and guilt (Gilbert, 2019). Thus, normalization of typical trauma reactions occurring early in treatment is likely to reduce self-blame (Knipschild et al., 2024). Knipschild and colleagues (2024) have begun to conduct an RCT evaluating the effects of an online psychoeducation intervention for adolescents exposed to interpersonal violence, but results are not yet available. Furthermore, research has shown that youth who blame themselves for traumatic events generalize self-blame to other negative events (Daigneault et al., 2006). Thus, participation in restructuring automatic thoughts and cognitive coping strategies throughout Phase I may reduce self-blame about other general stressors as well as the traumatic events. This finding demonstrates the need for

additional research to examine more nuanced cognitive outcomes of treatment components such as psychoeducation.

In contrast with exploratory hypotheses, perpetrator blame did not change significantly at either time point. Although perpetrator blame decreased marginally overall from Time 2 to Time 3, perpetrator blame scores remained consistently high compared to self-blame. This finding demonstrates that the intervention elements of TF-CBT decrease perpetrator disposition blame overall without eliminating the belief, which has been shown to be adaptive in longitudinal research with child sexual abuse survivors (Feiring & Cleland, 2007). Furthermore, individuals who exhibit external explanations for negative events are less likely to show reductions in self-esteem that often accompany internalizing symptoms, as evidenced by the attributional reformulation of the learned helplessness theory (Abramson et al., 1978).

Blame Attributions as a Mechanism of Change Throughout Phases of TF-CBT

Several models demonstrated inconsistencies in fit indices. More specifically, Models 2 and 6 showed acceptable CFI and poor TLI and RMSEA. Research suggests that inconsistences in indices of fit may arise because they evaluate fit using different methods and information (Lai & Green, 2016). The high RMSEA and low TLI displayed in several models may be partially due to their reliance on sample size (Hu and Bentler, 1999; Peugh & Feldon, 2020). Furthermore, RMSEA tends to penalize simpler model structures (Peugh & Feldon, 2020). Thus, all model results will still be interpreted given acceptable CFI.

Model 1 examined change in self-blame as a mediator of PTSD symptom change. Despite being a well-fitting model, the mediation effect between Time 2 and Time 3 only

approached significance. This finding may be due to the analysis being underpowered to detect small effects. Alternatively, this finding may suggest that decreases in self-blame explain change in certain PTSD clusters more than others. The results of Models 2 and 3 provide more evidence that reductions in self-blame may be more likely to mediate changes in general anxiety and persistent negative emotional state than symptoms of intrusion and hyperarousal, which may be more affected by changes in beliefs about the safety of their environment or the ability of trusted adults to help maintain safety. Previous research has demonstrated that different types of posttraumatic cognitions and blame attributions differentially predicted specific PTSD clusters (Blain et al., 2013). This research is based on the theories of both Foa and Rothbaum (1998) and Ehlers and Clark (2000) which proposed that individual's negative beliefs about their abilities to tolerate recalling and processing traumatic memories leads to intrusion symptoms, whereas negative beliefs about safety in the world and an overgeneralized sense of threat lead to the development of avoidance and hyperarousal clusters of PTSD (Blain et al., 2013). Specifically, Blain and colleagues (2013) found that in a sample of adults with PTSD, self-blame was predictive of numbing symptoms, whereas negative beliefs about their safety in the world (e.g.., "Nowhere is safe") was related to avoidance and hyperarousal, and negative self-cognitions (e.g., "I will lose control if I think about the trauma") was related to both re-experiencing and numbing symptoms. Future studies should examine whether changes in self-blame and dangerous world beliefs mediate change in the specific clusters of PTSD throughout trauma-specific treatment, after accounting for or removing items related to cognitive disturbances.

Results demonstrated acceptable CFI for Model 2 which examined self-blame and general anxiety. Reductions in self-blame mediated reductions in anxiety during Phase I, when psychoeducation, relaxation, and cognitive coping skills are offered. This finding may signal the efficacy of corrective psychoeducation in reducing self-blame and increasing youths' self-efficacy and beliefs about their abilities to keep themselves safe from danger. An additional explanation, consistent with previous research (Daigneault et al., 2006), is that reductions in general attributions of self-blame via cognitive coping strategies in Phase I may mediate reductions in trauma-specific self-blame attributions, leading to subsequent symptom amelioration. These findings are consistent with the attributional reformulation of the learned helplessness theory, which posits that internal, global, and stable attributions ("I can never do anything right") are more associated with symptomatology than specific and unstable attributions, which highlight more situational and transient factors (Abramson et al., 1978). Thus, a reduction in this attributional style may explain symptom reduction.

Model 3, which exhibited acceptable fit indices, demonstrated that decreases in self-blame mediated decreases in symptoms of depression during Phase I of TF-CBT. This mediation effect was expected given much research demonstrating the link between self-blame and internalizing symptoms (Tanzer et al., 2021). Given that the self-blame scale of the attribution measure used in this study included a combination of internal-unstable-specific attributions (e.g., "This happened because I misbehaved.") and internal-stable-global attributions (e.g., "This happened because I am a bad kid"), these findings also offer some support for the links between reductions in internal, stable, and global attributional style throughout Phase I interventions and subsequent decreases in

depressive symptoms (Peterson & Seligman, 1984). However, more research is needed to examine whether there are differential mediation effects for internal attributions that are stable and global versus unstable and specific. Furthermore, the timing of the effect (i.e., Phase I) is in contrast with hypotheses. Consistent with prior research demonstrating the efficacy of TF-CBT in increasing self-efficacy in abused youth (Farina et al., 2018), it is also possible that psychoeducation coupled with a myriad of relaxation, behavioral, and cognitive coping skills improve individuals' beliefs about themselves, their worth, and their perception of their ability to change their emotions and behaviors, in turn improving symptoms of depression.

Model 4 investigated perpetrator blame as a mediator of PTSD symptom change. In this well-fitting model, the decrease in perpetrator blame significantly mediated reductions in PTSD between Time 2 and Time 3. This finding contrasts with the hypothesis that increases in external perpetrator blame would mediate PTSD reductions following trauma-specific cognitive restructuring, which would have suggested a protective relationship. However, this finding is consistent with previous research demonstrating that youth with high levels of both self-blame and perpetrator blame exhibit poorer treatment outcomes (Sharma-Patel et al., 2014). These authors suggest that the combination of multiple blame attributions may create a constant sense of threat given beliefs that they are not safe around others and cannot keep themselves safe (Sharma-Patel et al., 2014). Therefore, a decrease in perpetrator blame may represent a decrease in a general sense of threat and subsequently reduce symptoms. Another explanation may be related to the high levels of anger that may accompany perpetrator blame. Research with youth and adults has shown that higher levels of anger are

associated with higher levels of PTSD (Orth & Maercker, 2009; Saigh et al., 2006). Additionally, higher levels of anger at baseline were associated with less improvement in PTSD symptoms in a sample of female adolescents (Kaczkurkin et al., 2016). Thus, lowering perpetrator blame, rumination, and anger toward the perpetrator may facilitate greater symptom reductions. The finding that reductions in perpetrator blame mediate PTSD symptom reduction contrasts with aspects of the attributional model of the learned helplessness theory, which suggests that external attributions are more associated with adjustment than internal attributions. Perhaps in the case of youth exposure to interpersonal violence, a high level of perpetrator blame is still adaptive, with lowered emphasis on the stable, dispositional traits of the perpetrator. In other words, shifting blame away from the stability of perpetrator's dispositional characteristics (i.e., perpetrator likes to hurt people) to other specific and transient ideas about why individuals perpetrate abuse (e.g., effects of intergenerational trauma, situational factors), may also reduce the sense of helplessness, persistent threat, and danger. Future research should examine whether changes in situational factors ("e.g., "perpetrator was under the influence," "perpetrator had a bad day,") mediate changes in symptoms.

Model 5 indicated acceptable indices and showed that decreases in perpetrator blame mediated decreases in anxiety between Time 2 and Time 3. Consistent with the results from the PTSD model, following cognitive restructuring and processing of trauma, youth may have more nuanced attributions of blame that are more specific and less global in nature. Thus, these reductions in perpetrator disposition blame may indicate reductions in sense of threat and danger, resulting in lowered anxiety.

Despite good fit in Model 6, which examined the mediation effect of change in perpetrator blame on change in depression, mediation effects at both timepoints approached significance ($p = .05 – .06$). Given the pattern of mediation results with perpetrator blame and other trauma-related symptomology, these results may be a product of the study being underpowered to detect small effects.

Clinical Implications

The varied and intricate results of this study have significant implications for treatment implementation and outcomes. The results show that reductions in both internal and external blame attributions serve as a mechanism of change at different timepoints throughout TF-CBT. Thus, clinicians and researchers should continue to assess attributions of blame related to the exposure to the traumatic event, the maintenance of the abuse, and behavioral responses to trauma throughout the intervention. Given that reductions in self-blame mediate reductions in anxiety and depression symptoms during Phase I of TF-CBT, if high levels of PTSD persist, it may be beneficial to assess youths' attributions toward the perpetrator or dangerous world beliefs during Phase II. Furthermore, despite hypotheses postulating that increases in perpetrator blame (i.e., shifting self-blame to perpetrator blame) would be adaptive, this study showed that slight decreases in perpetrator blame effectively reduced the severity of PTSD and anxiety symptoms following trauma narration. This finding suggests that throughout trauma-specific cognitive restructuring, it may be more beneficial to incorporate developmentally appropriate explorations of other more specific explanations of why individuals perpetrate abuse (e.g., intergenerational trauma or mental health challenges of the perpetrator that have the potential to be addressed in their own treatment). However,

mixed method research is needed to better understand which attributions are most adaptive and helpful in processing interpersonal violence. Moreover, despite previous research that individuals with high self-blame and or perpetrator blame exhibit poorer trauma-specific treatment outcomes (Øktedalen et al., 2015; Sharma-Patel et al., 2014), the current study demonstrated that several TF-CBT components are effective at addressing maladaptive attributions and reducing PTSD, anxiety, and depression symptom severity. These findings may be used an as engagement tool for families, especially those hesitant to engage in trauma narration and processing, as the results demonstrate the importance of participation in all phases of TF-CBT.

Limitations and Research Recommendations

The findings of the current study should be interpreted in the context of several limitations. First, the analyses were conducted with relatively small sample sizes and were underpowered to detect small effects, increasing the likelihood of Type II errors. The sample size may have also skewed indices of fit. Replicating the study with a larger sample size may facilitate interpretation of more generalizable and robust results. Attrition rates in the current study are consistent with attrition in other community-based samples of maltreated youth (Koverola et al., 2007; Lau & Weisz, 2003). However, missingness in the data was related to older age, suggesting the results of this study may be more generalizable to younger clinical samples. Additionally, age was related to both missingness and self-blame. Therefore, the current study was unable to control for one potential cause of missingness in the sample, which may have led to biased results. Furthermore, the lack of a control group in the study limits the ability to make causal conclusions about the effects of TF-CBT components on blame attributions and

subsequent symptom change. The use of an active control group (e.g., treatment as usual) would more definitively demonstrate the effects of treatment components in TF-CBT. Relatedly, given the results of this study, future research should also examine the amount of exposure to each specific component of treatment (e.g., psychoeducation, parenting skills, general cognitive coping, trauma-specific cognitive restructuring) to better understand which interventions lead to change in blame attributions and subsequent symptoms. Although blame attributions in the current study did not differ depending on trauma type endorsed as the most upsetting, research should continue to assess whether changes in attributions throughout treatment are related to specific types of interpersonal violence.

An additional limitation of the current study is that the measure of blame attributions used, the PERCEIVE (Brown, 2000), has not been externally validated with a large sample. However, this measure exhibits several psychometric strengths (e.g., good internal reliability, factor analysis derived subscales) and is uniquely broad in measuring several kinds of blame attributions. This is particularly important given recent research calling for more methodologically sound measures of trauma-specific attributions (Reich et al., 2023; Seah et al., 2023). Given the nuance of posttraumatic blame cognitions, measure developers should consider not only who the individual believes is to blame for the occurrence of the trauma, but the percent belief in the thoughts and the level of distress the thoughts elicit for the individual (Reich et al., 2023). Future studies should examine the potential differential mediation effects of internal-global-stable, internal-specific-unstable, external-global-stable, and external-specific-unstable cognitions on changes in symptoms. Furthermore, additional research is needed on the mediation

effects of other attributions such as blame toward non-offending caregivers, and cultural or religious practices, as well as combinations of attributions. Future research should also explore whether other beliefs about why individuals perpetrate abuse, such as intergenerational trauma, situational factors (e.g., perpetrator was under a lot of stress, perpetrator was under the influence of alcohol or drugs, etc.), or symptoms of mental illness are more protective and adaptive than focusing on dispositional traits of the perpetrator. Given the heterogeneity of patterns of perpetrator blame throughout treatment timepoints, future studies should examine whether symptom trajectory throughout treatment is moderated by baseline levels of perpetrator blame.

Finally, researchers should consider the integral role that caregivers play in their children's treatment outcomes. Non-supportive responses from caregivers are directly linked to abuse-specific self-blame attributions in adolescents (Jouriles et al., 2022). Research has also shown higher levels of blame and criticism from caregivers were associated with higher in-session child distress during TF-CBT (Canale et al., 2022). An additional study demonstrated that caregiver blame expressed during trauma narration was associated with greater maladaptive trauma-specific cognitions and fewer balanced and adaptive cognitions in youth (Yasinski et al., 2016). Given research that shows that TF-CBT can effectively change caregivers' posttraumatic cognitions (Tutus et al., 2019), it is critical to assess and target attributions of blame in both the child and caregiver throughout the course of treatment.

Table 1

Descriptive Statistics of the Study Sample

Variable	*M*	*SD*	(*N* = 420)
Child age (years)	12.00	3.69	
Number of traumas endorsed	1.88	0.88	
	n	%	
Reference Trauma*			
Traumatic bereavement	3	1.0	
Witnessing domestic violence	51	15.8	
Physical abuse	56	17.3	
Sexual abuse	195	60.4	
Peer sexual assault	17	5.3	
Child gender (female)	286	72.2	
Child race/ethnicity			
Hispanic	156	41.2	
African American / Black	69	18.2	
Multiracial	62	16.4	
Caucasian	30	7.9	
Guyanese	16	4.2	
Asian	14	3.7	
Other	31	8.2	

*Note. Reference trauma is the trauma rated by the child as the most upsetting traumatic event.

Table 2

Descriptive Statistics of Study Variables

Scale	Time 1		Time 2		Time 3	
	M	SD	M	SD	M	SD
Self-blame	2.26	3.31	1.86	2.96	1.04	2.08
Self-blame Endorsed at Time (n = 164)	4.49	3.43	2.78	3.41	1.82	2.68
Perpetrator blame	7.25	4.73	7.48	4.71	7.15	4.64
PTSD symptom score	0.0014	1.00	-0.0009	1.00	-0.0047	1.00
Anxiety symptom score	56.92	12.60	51.59	11.01	47.81	9.57
Depression symptom score	56.97	13.93	50.87	11.02	47.69	8.89

*Note. Self-blame and perpetrator blame were measured by the *Perceptions of Children Exposed to Interpersonal ViolencE (PERCEIVE) Short Form.* Scales were computed by summing ratings rated from 0 to 2. The second row displays the means and standard deviations for the subsample of participants who endorsed self-blame at Time 1. PTSD symptom scores are represented as Z-scores due to combining CPSS-4 and CPSS-5 scores in which items were coded 0-3 and 0-4, respectively. Anxiety, depression, and somatization subscale scores from the BASC-3 Self-Report of Personality (SRP) Child and Adolescent Forms. The descriptive statistics presented represent the full sample (N = 420).

Table 3

Results of Analyses Examining Change in Blame Attributions

	Time 1 to Time 2				Time 2 to Time 3			
	t	df	*p*	*d*	*t*	df	*p*	*d*
Self-Blame	-2.05*	115	.04	-.19	-2.44*	83	.02	-.26
Perpetrator Blame	0.68	112	.50	.06	-0.64	78	.52	.07

**Note. * indicates significance at the .05 level.*

Table 4

Correlations Among Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Self-blame T1	-														
2. Self-blame T2	.59**														
3. Self-blame T3	.44**	.52**													
4. Perpetrator blame T1	.08	-.13	-.03												
5. Perpetrator blame T2	.05	-.001	-.09	.72**											
6. Perpetrator blame T3	.06	-.02	-.02	.67**	.73**										
7. PTSD T1	.19**	.08	-.08	.33**	.31**	.27**									
8. PTSD T2	.16*	.17	.02	.15*	.15	.21*	.56**								
9. PTSD T3	.12	.12	.17*	.14	.13	.30**	.41**	.60**							
10. Anxiety T1	.33**	.19	-.03	.27**	.22*	.30**	.62**	.45**	.42**						
11. Anxiety T2	.36**	.31**	.02	.19*	.19	.19*	.47**	.64**	.46**	.63**					
12. Anxiety T3	.26**	.25*	.12	.16	.15	.27**	.44**	.57**	.51**	.58**	.71**				
13. Depression T1	.34**	.21*	.14	.25**	.23*	.20*	.53**	.40**	.36**	.70**	.45**	.48**			
14. Depression T2	.33**	.41**	.09	.20**	.20**	.16	.44**	.56**	.44**	.43**	.61**	.52**	.60**		
15. Depression T3	.40**	.33**	.34**	.18*	.11	.24**	.30**	.51**	.47**	.33**	.35**	.58**	.55**	.66**	

**Note. * indicates significance at the .05 level. **indicates significance at the .001 level.* Correlation analyses conducted on full sample (N = 420)

Table 5

Fit Indices and Estimates of Path Analysis Models Examining Change in Self-Blame as a Mediator of Symptom Change

Criterion Variable	CFI	TLI	RMSEA	Estimate	Standard Error	*p*	Standardized Estimate
PSTD (Model 1)	.98	.96	.05				
Phase I Mediation				.03	.06	.65	.03
Phase II-III Mediation				.11	.06	.07	.10
Total Indirect Effect				.14	.10	.16	.13
Anxiety (Model 2)	.93	.87	.09				
Phase I Mediation				.13	.06	.03*	.13
Phase II-III Mediation				.07	.05	.15	.07
Total Indirect Effect				.20	.07	.01*	.20
Depression (Model 3)	.96	.93	.06				
Phase I Mediation				.15	.07	.04*	.14
Phase II-III Mediation				.17	.10	.08	.18
Total Indirect Effect				.32	.09	.001**	.31

**Note. * indicates significance at the .05 level. **indicates significance at the .001 level.*

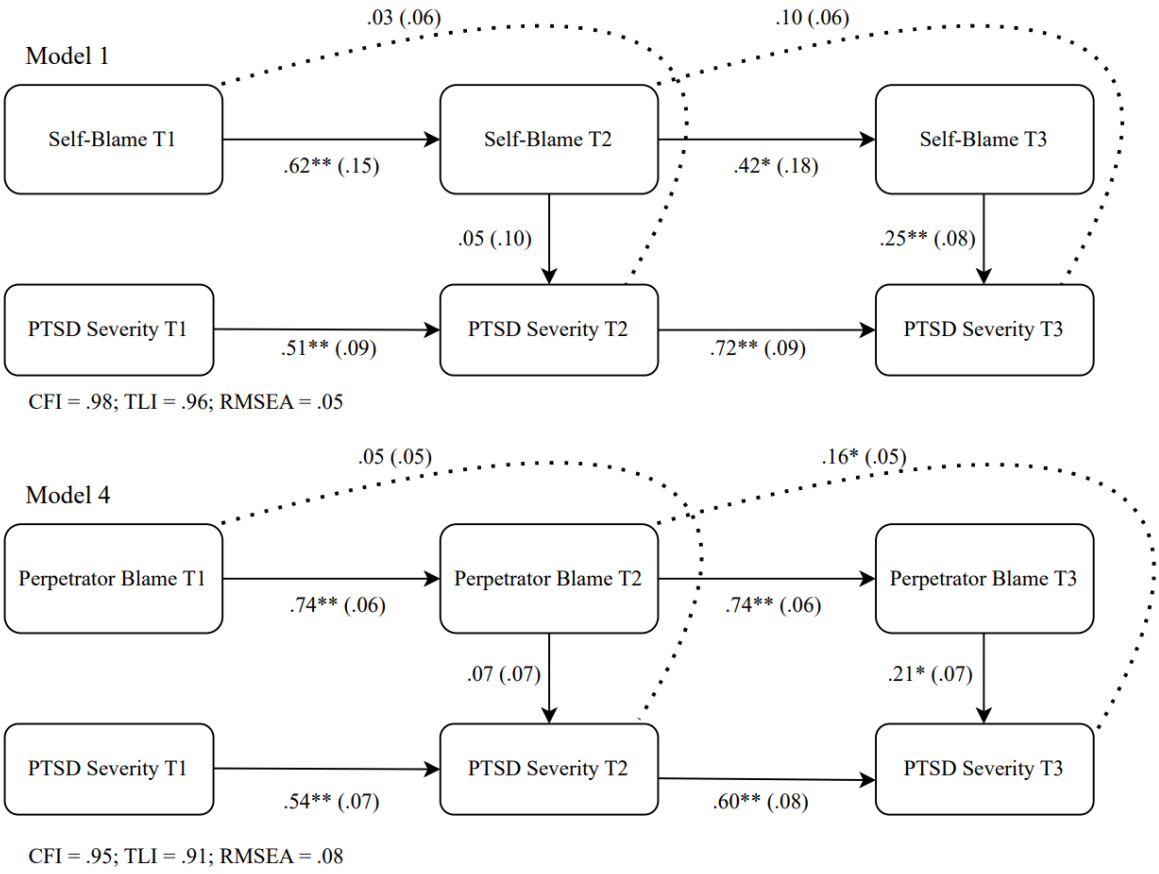
Table 6

Fit Indices and Estimates of Path Analysis Models Examining Change in Perpetrator Blame as a Mediator of Symptom Change

Criterion Variable	CFI	TLI	RMSEA	Estimate	Standard Error	*p*	Standardized Estimate
PSTD (Model 4)	.95	.91	.08				
Phase I Mediation				.05	.05	.33	.05
Phase II-III Mediation				.16	.05	.002*	.16
Total Indirect Effect				.21	.07	.01*	.20
Anxiety (Model 5)	.94	.89	.10				
Phase I Mediation				.04	.05	.39	.04
Phase II-III Mediation				.11	.05	.03*	.11
Total Indirect Effect				.15	.07	.03*	.15
Depression (Model 6)	.96	.93	.07				
Phase I Mediation				.10	.05	.05	.09
Phase II-III Mediation				.10	.05	.06	.11
Total Indirect Effect				.20	.07	.01*	.21

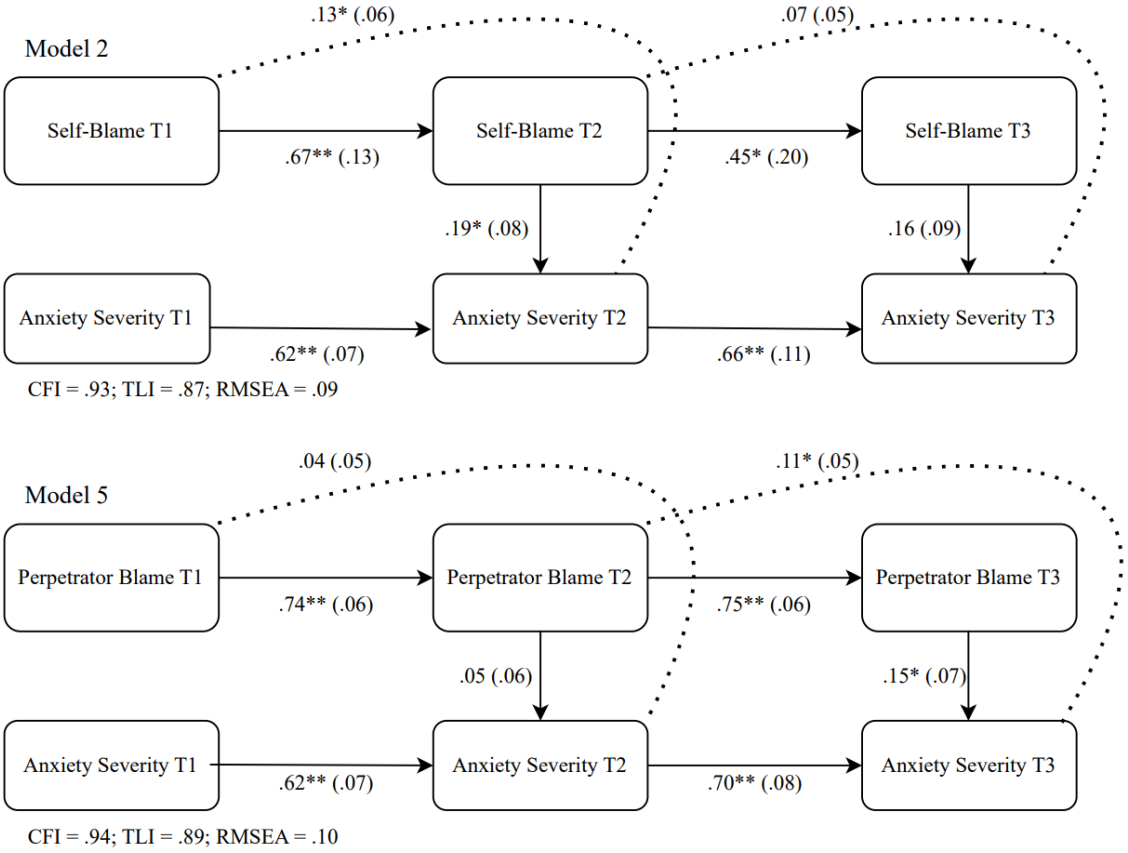
**Note. * indicates significance at the .05 level. **indicates significance at the .001 level.*

Figure 1. Path Models 1 and 4 depicting Direct and Indirect Effects of Blame Attributions on PTSD Severity Scores



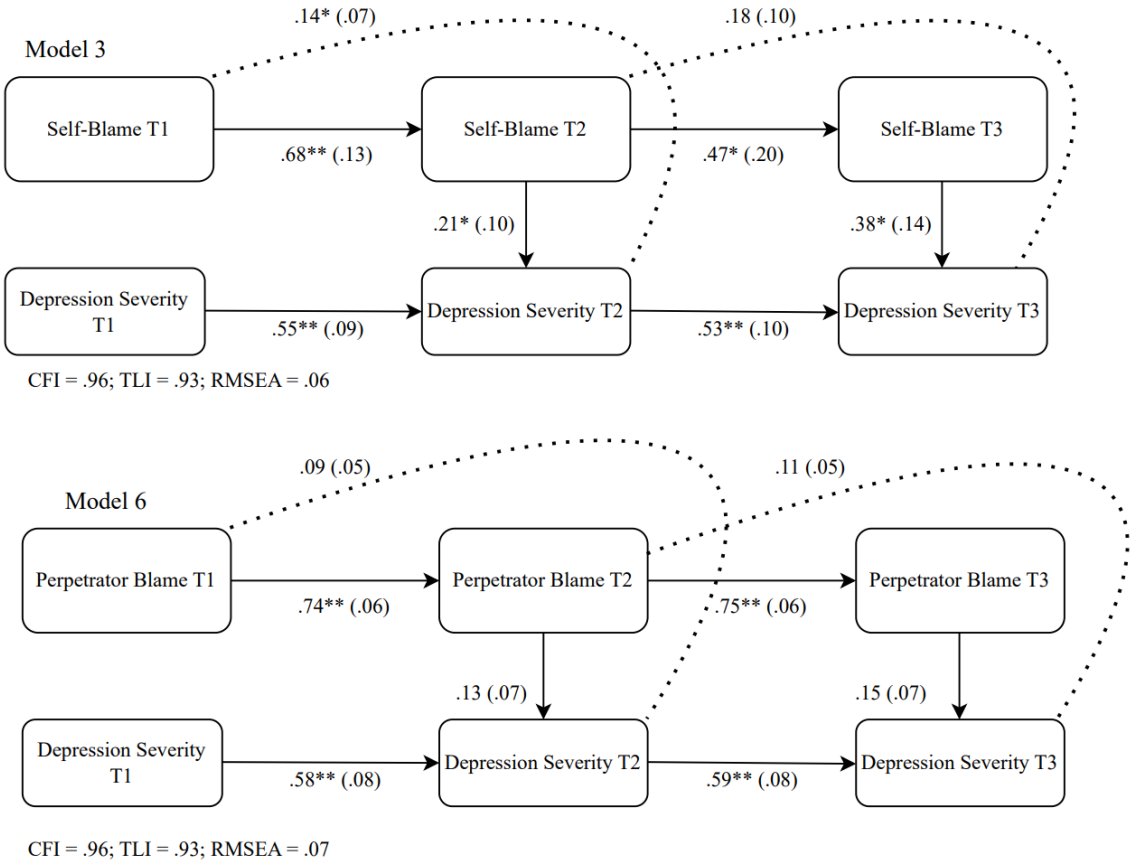
Note. Dotted lines indicate indirect effects whereas solid lines indicate direct effects. Standardized estimates are listed first followed by standard errors (in parentheses). ** Indicates significance at the .001 level and *indicates significance at the .05 level.

Figure 2. Path Models 2 and 5 depicting Direct and Indirect Effects of Blame Attributions on Anxiety Severity Scores



Note. Dotted lines indicate indirect effects whereas solid lines indicate direct effects. Standardized estimates are listed first followed by standard errors (in parentheses). ** Indicates significance at the .001 level and * indicates significance at the .05 level.

Figure 3. Path Models 3 and 6 depicting Direct and Indirect Effects of Blame Attributions on Depression Severity Scores



Note. Dotted lines indicate indirect effects whereas solid lines indicate direct effects. Standardized estimates are listed first followed by standard errors (in parentheses). ** Indicates significance at the .001 level and * indicates significance at the .05 level.

APPENDIX A

PERceptions of Children Exposed to Interpersonal ViolencE (PERCEIVE) Short Form

Now I am going to ask you some questions about the time that _____ (use a brief statement from their words on the TECS summarizing the incident).

(a) What do you remember about what happened?
(b) Why do you think this happened?
(c) Whose fault do you think it was?

For each statement I read, I want you to think about the time that _____ (child's terms) and why it happened. Using this scale, you'll tell me how true each sentence is for you.

0 = I don't think this at all,
1 = I think this a little bit
2 = I think this a lot

Before each item: *This happened because….*

1. I am a bad kid*
2. it was my fault*
3. the world is unfair
4. I cause trouble a lot*
5. (perpetrator) is a bad person+
6. my family doesn't care about children in our house
7. it's okay for family members to hurt each other
8. I did something wrong*
9. (perpetrator) didn't mean to do it
10. bad things happen sometimes
11. (perpetrator) likes to hurt people+
12. people in my neighborhood hurt each other
13. (perpetrator) had a bad day
14. (perpetrator) did it by accident
15. I'm too small to stop it
16. my family wasn't getting along
17. (perpetrator) didn't know what s/he was doing
18. I'm not a good kid*
19. (perpetrator) is mean+
20. people are bad+
21. my neighborhood isn't safe
22. people trusted (perpetrator) when they shouldn't have+
23. family members make mistakes
24. (perpetrator) doesn't care about other people+
25. people in my family hurt each other

26. I didn't behave*
27. (perpetrator) lost control
28. sometimes family members hurt one another by accident
29. (perpetrator) loses control
30. I make life bad for my family*
31. (perpetrator) gets upset easily
32. (perpetrator) enjoyed it^{+}
33. other grown-ups didn't stop it
34. the people in my family didn't keep each other safe
35. I do things wrong*
36. (perpetrator) has no patience
37. sometimes accidents happen
38. I misbehaved*

Note. *Denotes items on the self-blame scale whereas $^{+}$ denotes items on the perpetrator disposition blame scale.

APPENDIX B

Behavioral Assessment System for Children, third edition, Self-Report of Personality Rating Scales (BASC-3-SRP) Anxiety, Depression, and Somatization Subscales

Subscale	Child	Adolescent
Anxiety	Item 27	Item 32
	Item 49	Item 44
	Item 65	Item 58
	Item 76	Item 65
	Item 83	Item 75
	Item 85	Item 83
	Item 91	Item 100
	Item 104	Item 121
	Item 118	Item 138
	Item 125	Item 146
	Item 136	Item 153
		Item 161
		Item 183
Depression	Item 7	Item 25
	Item 14	Item 40
	Item 25	Item 46
	Item 38	Item 50
	Item 55	Item 55
	Item 62	Item 70
	Item 81	Item 96
	Item 94	Item 124
	Item 114	Item 134
	Item 129	Item 167
		Item 173
		Item 179

APPENDIX C

The Child PTSD Symptom Scale (CPSS-4)

Below is a list of problems kids sometimes have after experiencing an upsetting event. Read each one carefully and indicate the number (0-3) that best describes how often that problem has bothered you IN THE LAST 2 WEEKS.

0 = Not at all or only one time
1 = Once a week or less/once in a while
2 = 2 to 4 times a week/ half the time
3 = 5 or more times a week/almost always

1. Having upsetting thoughts or images about the event that came into your head when you didn't want them to
2. Having bad dreams or nightmares
3. Acting or feeling as if the event was happening again (hearing something or seeing a picture about it and feeling as if I am there again)
4. Feeling upset when you think about or hear about the event (for example, feeling scared, angry, sad, guilty, etc.)
5. Having feelings in your body when you think about or hear about the event (for example, breaking out in a sweat, heart beating fast)
6. Trying not to think about, talk about, or have feelings about the event
7. Trying to avoid activities, people, or places that remind you of the traumatic event
8. Not being able to remember important parts of the upsetting event
9. Having much less interest or not doing things you used to
10. Not feeling close to people around you
11. Not being able to have strong feelings (for example, being unable to cry or unable to feel very happy)
12. Feeling as if your future plans or hopes will not come true (for example, you will not have a job or get married, or have kids)
13. Having trouble falling or staying asleep
14. Feeling irritable or having fits of anger
15. Having trouble concentrating (for example, losing track of a story on television, forgetting what you read, not paying attention in class)
16. Being overly careful (for example, checking to see who is around you and what is around you)
17. Being jumpy or easily startled (for example, when someone walks up behind you)

Indicate YES or NO below if the problems you rated above have gotten in the way with any of the following areas of your life DURING THE PAST 2 WEEKS.

1. Doing your prayers
2. Chores and duties at home
3. Relationships with friends
4. Fun and hobby activities
5. Schoolwork

6. Relationships with your family
7. General happiness with your life

APPENDIX D

The Child PTSD Symptom Scale (CPSS-5)

Sometimes scary or upsetting things happen to kids. It might be something like a car accident, getting beaten up, living through an earthquake, being robbed, being touched in a way you didn't like, having a parent get hurt or killed, or some other very upsetting event. These questions ask about how you feel about the upsetting thing you wrote down. Read each question carefully. Then indicate the number (0-4) that best describes how often that problem has bothered you IN THE LAST MONTH.

0 = Not at all
1 = Once a week or less/a little
2 = 2 to 3 times a week/somewhat
3 = 4 to 5 times a week/a lot
4 = 6 or more times a week/almost always

1. Having upsetting thoughts or pictures about it that came into your head when you didn't want them to
2. Having bad dreams or nightmares
3. Acting or feeling as if it was happening again (seeing or hearing something and feeling as if you are there again)
4. Feeling upset when you remember what happened (for example, feeling scared, angry, sad, guilty, confused)
5. Having feelings in your body when you remember what happened (for example, sweating, heart beating fast, stomach or head hurting)
6. Trying not to think about it or have feelings about it
7. Trying to stay away from anything that reminds you of what happened (for example, people, places, or conversations about it)
8. Not being able to remember an important part of what happened
9. Having bad thoughts about yourself, other people, or the world (for example, "I can't do anything right", "All people are bad", "The world is a scary place")
10. Thinking that what happened is your fault (for example, "I should have known better", "I shouldn't have done that", "I deserved it")
11. Having strong bad feelings (like fear, anger, guilt, or shame)
12. Having much less interest in doing things you used to do
13. Not feeling close to your friends or family or not wanting to be around them
14. Trouble having good feelings (like happiness or love) or trouble having any feelings at all
15. Getting angry easily (for example, yelling, hitting others, throwing things)
16. Doing things that might hurt yourself (for example, taking drugs, drinking alcohol, running away, cutting yourself)
17. Being very careful or on the lookout for danger (for example, checking to see who is around you and what is around you)
18. Being jumpy or easily scared (for example, when someone walks up behind you, when you hear a loud noise)

19. Having trouble paying attention (for example, losing track of a story on TV, forgetting what you read, unable to pay attention in class)
20. Having trouble falling or staying asleep

Have the problems above been getting in the way of these parts of your life IN THE PAST MONTH? (Yes/No)

1. Fun things you want to do
2. Doing your chores
3. Relationships with your friends
4. Praying
5. Schoolwork
6. Relationships with your family
7. Being happy with your life

APPENDIX E

Fit Indices and Estimates of Path Models Examining Change in Self-blame as a Mediator of Symptom Change in Treatment Completers

Criterion Variable	CFI	TLI	RMSEA	Estimate	Standard Error	*p*	Standardized Estimate
PSTD (Model 1B)	.96	.93	.08				
Phase I Mediation				.02	.06	.71	.02
Phase II-III Mediation				.12	.06	.06	.10
Total Indirect Effect				.14	.10	.17	.12
Anxiety (Model 2B)	.88	.78	.15				
Phase I Mediation				.15	.07	.04*	.13
Phase II-III Mediation				.08	.05	.14	.07
Total Indirect Effect				.23	.09	.01*	.20
Depression (Model 3B)	.94	.90	.10				
Phase I Mediation				.17	.10	.08	.13
Phase II-III Mediation				.17	.10	.08	.16
Total Indirect Effect				.34	.10	.001**	.29

**Note. * indicates significance at the .05 level. **indicates significance at the .001 level*

APPENDIX F

Fit Indices and Estimates of Path Models Examining Change in Perpetrator blame as a Mediator of Symptom Change in Treatment Completers

Criterion Variable	CFI	TLI	RMSEA	Estimate	Standard Error	*p*	Standardized Estimate
PSTD (Model 5B)	.96	.94	.10				
Phase I Mediation				-.02	.06	.78	-.02
Phase II-III Mediation				.12	.06	.06	.10
Total Indirect Effect				.14	.10	.17	.12
Anxiety (Model 6B)	.93	.88	.14				
Phase I Mediation				.03	.06	.66	.03
Phase II-III Mediation				.10	.05	.04	.11
Total Indirect Effect				.14	.08	.10	.14
Depression (Model 7B)	.95	.91	.12				
Phase I Mediation				.08	.06	.21	.07
Phase II-III Mediation				.10	.06	.06	.11
Total Indirect Effect				.18	.08	.03*	.19

**Note. * indicates significance at the .05 level. **indicates significance at the .001 level.*

APPENDIX G

Baseline Differences Between Youth Who Endorsed or Denied Self-Blame at Time 1

	t	*df*	*p*
Age	2.16	312	.03
Anxiety Severity	5.52	306	< .001
Depression Severity	5.12	306	< .001
PTSD Severity	4.27	311	< .001
	χ^2	*df*	*p*
Gender	.78	1	.38

**Note. * indicates significance at the .05 level. **indicates significance at the .001 level.*

APPENDIX H

Fit Indices and Estimates of Path Models with Covariates Examining Change in Self and Perpetrator Blame as Mediators of Symptom Change

Self-Blame

Criterion Variable	CFI	TLI	RMSEA	Estimate	Standard Error	p	Standardized Estimate
PSTD (Model 4)	.89	.84	.06				
Phase I Mediation				.05	.06	.39	.05
Phase II-III Mediation				.11	.06	.06	.11
Total Indirect Effect				.16	.09	.08	.16
Anxiety (Model 5)	.88	.82	.07				
Phase I Mediation				.13	.06	.03*	.13
Phase II-III Mediation				.09	.06	.12	.08
Total Indirect Effect				.21	.08	.007*	.21
Depression (Model 6)	.93	.89	.05				
Phase I Mediation				.15	.07	.03*	.14
Phase II-III Mediation				.17	.10	.08	.17
Total Indirect Effect				.32	.09	.001*	.31

Perpetrator Blame

Criterion Variable	CFI	TLI	RMSEA	Estimate	Standard Error	p	Standardized Estimate
PSTD (Model 4)	.81	.72	.10				
Phase I Mediation				.07	.05	.18	.07
Phase II-III Mediation				.20	.06	<.001**	.19
Total Indirect Effect				.27	.07	<.001**	.26
Anxiety (Model 5)	.82	.73	.10				
Phase I Mediation				.05	.05	.05	.05
Phase II-III Mediation				.09	.05	.09	.09
Total Indirect Effect				.14	.07	.14	.14
Depression (Model 6)	.83	.74	.09				
Phase I Mediation				.11	.05	.04*	.10
Phase II-III Mediation				.12	.06	.12	.13
Total Indirect Effect				.22	.08	.22	.23

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