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Pathways from Child Maltreatment to Proactive and Reactive Aggression: The Role of Post-Traumatic Stress Symptom Clusters

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Abstract

Objective: Childhood aggression is related to a myriad of negative concurrent and long-term outcomes. To mitigate the risks associated with childhood aggression, it is important to understand risk factors that might predispose one to aggressive behaviors. One risk factor commonly associated with aggression is the experience of child maltreatment. A common outcome associated with child maltreatment is the development of post-traumatic stress symptoms (PTSS). Several prevailing theoretical models of both post-traumatic stress and aggression indicate that these constructs have similar underlying cognitive, behavioral, and emotional mechanisms. Therefore, the present study examined the relations between and among child maltreatment, PTSS clusters, and proactive and reactive aggression in children. **Method:** Children between the ages of 6 and 14 who were enrolled in a residential treatment program completed self-report measures to evaluate variables of interest. These variables were included as multiple outcomes in a path analysis model in which individual PTSS clusters were examined as potential multiple mediators of the relations between child maltreatment and proactive and reactive aggression. **Results:** Direct effects of child maltreatment and PTSS clusters on aggression were observed. Significant indirect effects of the intrusion PTSS cluster on the relation between child maltreatment and reactive aggression was found. **Conclusions:** Findings suggest that symptoms associated with these specific PTSS clusters might help explain the relation between child maltreatment and reactive aggression and therefore present important implications for clinical practice and future research.

Keywords: Adverse Childhood Experiences, Child Maltreatment, Aggression, PTSD

Clinical Impact Statement

The current study sheds light on the need to potentially broaden our conceptualization of trauma-related responses in children and adolescents and evaluate the need for a trauma-informed treatment approaches even when diagnostic criteria for PTSD is not clearly met. It may be that children with a history of maltreatment engage in more impulsive and externalizing behaviors secondary to experiencing PTSS symptoms and to properly address underlying processes, a treatment model supported for PTSD may be appropriate.

Childhood aggression has been associated with adverse outcomes across the lifespan including concurrent academic, social, and emotional functioning (e.g., Bierman et al., 2014; Card & Little, 2006; Walker & Bright, 2009) and later mental health problems, poor occupational attainment, interpersonal difficulties, and involvement with the criminal justice system in adulthood (e.g., Althoff et al., 2010; Huesmann et al., 2009; Mumford et al., 2019). Given the extent of its impact, the high prevalence of clinically significant levels of childhood aggression (2-16%; Merikangas et al., 2009), and the heterogeneous nature of aggression, researchers have sought to understand distinctions between the etiological and risk factors associated with different types of aggression. While many factors influence the development and maintenance of aggression, the impact of early Adverse Childhood Experiences (ACEs) and subsequent mental health processes have recently emerged in the literature as important areas of study. Thus, the purpose of the present study was to evaluate the relations between a specific type of ACEs, child maltreatment, and childhood subtypes of aggression, proactive and reactive aggression. Additionally, we sought to evaluate the impact of post-traumatic stress symptoms (PTSS) on these relations.

The differentiation between proactive and reactive aggression is commonly examined in the literature (Dodge & Coie, 1987). Proactive aggression is described as instrumental, offensive, and manipulative, often requiring no provocation or anger whereas reactive aggression is affective and defensive and often occurs in response to some real or perceived threat (Brendgen et al., 2006; Dodge & Coie, 1987). Although proactive and reactive aggression have overlapping features and often co-occur (Vitaro & Brendgen, 2005), they are also marked by distinct sets of associated features and underlying mechanisms (e.g., autonomic nervous system arousal; Card & Little, 2006; Fite et al., 2009; Hubbard et al., 2001; Wagner & Abaied, 2015).

Although many factors are thought to influence the presence of childhood aggression, the experience of traumatic events early in development has emerged in the literature as a significant contributor to aggression in children (Erwin et al., 2000; Ford et al., 2012). Traumatic stress responses and aggression have been shown to have multiple shared etiological mechanisms. For example, early stressful experiences have been shown to negatively impact a child's ability to regulate and express their emotions effectively, which has also been implicated as a potential etiological factor in childhood aggression (Card & Little, 2006; Erwin et al., 2000). Consequences of ACEs and the presence of aggressive behaviors are associated with similar changes in the neuroendocrine and immune systems (e.g., Metzler et al., 2017), and these perturbations in neuroendocrine functioning often overlap with the processes implicated in the predisposition for high levels of aggression, especially among children (e.g., Stoppelbein et al., 2014). Similarly, both

aggression and trauma experiences have been found to show similar changes in brain development/structure in areas responsible for executive function (e.g., emotional and behavioral regulation, attention, etc.; Cozolino, 2014).

The negative effects of traumatic stress are especially evident when these events are perceived as outside of the child's control and/or involve disruptions in their relationships with primary caregiver(s) or environment (Roosa et al., 2010; Nagin &Tremblay, 2001). Thus, it is not surprising that the experience of child abuse and/or neglect has emerged as a particularly salient set of predictors for many detrimental outcomes in children, including aggressive behavior. Although many types of ACEs have been associated with higher levels of aggressive behaviors in youth (Felitti et al., 1998; Ford et al., 2012), ACEs specifically related to child maltreatment (i.e., physical, sexual, and emotional abuse and physical and emotional neglect; CM-ACEs) appear more strongly linked to aggression, particularly reactive aggression, than other types of adverse events (Kolla et al., 2013; Rehan et al., 2017). While strong support exists for the reactive aggression/CM-ACEs relation, the findings for proactive aggression are mixed (Fite et al., 2012; Murray-Close & Rellini, 2012). Collectively, these studies help elucidate the ways in which CM-ACEs might be related to subtypes of aggression, but the equivocal nature of the results regarding proactive aggression highlight the importance of continuing to examine these relations.

Not everyone who experiences maltreatment in childhood engages in aggressive behaviors, suggesting the presence of other mechanisms that might help to explain the relation between CM-ACEs and aggression. For some children and adolescents with a history of CM-ACEs, the presence of protective factors can help attenuate negative outcomes. Individual factors (e.g., intellect, social competence) can help reduce the effects of CM-ACEs (Afifi & MacMillan, 2011; Kim et al., 2009), including aggressive behavior. The role of these protective factors is supported by research examining genetic resilience, as well. Findings from studies examining genotypes in youth with a history of CM-ACEs have found that certain genes involved in brain development, behavioral and emotional regulation (especially stress), and attachment are associated with higher levels of resilience (Cicchetti & Rogosch, 2012).

However, the effects of CM-ACEs on cognitive, neurodevelopmental, and biological functioning (De Bellis & Zisk, 2014) can make these protective factors difficult to access. Therefore, it is also important to consider risk factors that might explain a predisposition towards aggressive behavior in children who have experienced maltreatment. Several mental health factors have been identified as potential mechanisms of influence on the relation between CM-ACEs and aggression. Foremost in this discussion is Post-Traumatic Stress Disorder (PTSD). According to the *Diagnostic and Statistical Manual, Fifth Edition* (*DSM-5*; American Psychological Association [APA], 2013), PTSD is

marked by alterations in cognitive, emotional, behavioral, and physiological processes following a traumatic event or series of events. Children who experience traumatic events that include interpersonal violence, such as physical or sexual abuse, have the highest risk of developing PTSD when compared to other types of trauma exposure (Breslau et al., 2004; Lewis et al., 2019; McLaughlin et al., 2015), and it is estimated that between 30 and 70% of children who experience maltreatment will develop PTSD (Lewis et al., 2019; McLaughlin et al., 2015).

PTSD related to CM-ACEs has also been associated with aggression. For example, PTSD has emerged as a mediator between child abuse/neglect and a range of adverse outcomes in adulthood, including aggressive behaviors, self-harm, and substance abuse (Cross et al., 2015; Dyer et al., 2009). Other studies including child and adolescent populations have observed association between PTSD and proactive/ reactive aggression (Bubier & Drabick, 2009; Stimmel et al., 2014), albeit through different sets of mechanisms. That is, the link between reactive aggression and CM-ACEs and/or PTSD is typically explained through difficulties or changes in the emotion regulation systems, impulsivity, and heightened autonomic nervous system arousal (e.g., Cicchetti & Toth, 1995; van der Kolk, 2005), while the link between CM-ACEs and proactive aggression is typically explained by alterations in decision-making processes, reward response style, and emotional/behavioral regulation systems (Sailer et al., 2008). These findings are consistent with one of the prevailing theoretical models of aggression – social information processing theory. This theory suggests proactive and reactive aggression are related to errors in the ability to process social information, a skill that is influenced by the aforementioned executive functions and many of the neurobiological, behavioral, cognitive, and emotional correlates of PTSD (Dodge & Crick, 1990; Weber, 2008).

Despite what appears to be consistent links between PTSD and aggressive behaviors, most of the existing research examining this relation among those with a history of CM-ACEs has only examined PTSD as a homogenous construct. Nevertheless, some attempts to examine differential relations between the four distinct symptom clusters of PTSD (APA, 2013), potential etiological factors, and behavioral outcomes have been made. For example, sexual but not physical abuse has been associated with experiencing more intrusion and hyperarousal symptoms (Deblinger et al., 1990) while the experience of emotional abuse in childhood has been suggested as a strong predictor of all symptom clusters (Sullivan et al., 2006). However, results of these studies are equivocal, possibly because they fail to account for the high rates of co-occurrence between different types of abuse and neglect (Edwards et al., 2007). An emerging literature has begun to document the differential relations between post-traumatic stress symptom (PTSS) clusters and aggression with re-experiencing, hyper-arousal, and negative cognition/mood symptoms emerging as significant

predictors of later aggression among adults with a history of CM-ACEs (e.g., Aebi et al., 2017; Dyer et al., 2009). Taken together, these findings lay the foundation for potential differences in etiological underpinnings and associated outcomes for each PTSS cluster. However, this body of research is limited to retrospective studies with adult samples and findings across studies are often inconsistent.

THE PRESENT STUDY

Broad associations between CM-ACEs, PTSD, and aggression have been well-established in the literature (e.g., Auslander et al., 2016) and some studies support differential relations between CM-ACEs, PTSS clusters, and aggression subtypes. Although the existing body of literature provides a strong foundation for understanding the relations between these variables, there are several gaps in the literature. Despite the extensive research supporting a distinction between subtypes of aggression, there remain discrepancies in the literature regarding the association between aggression subtypes and the experience of CM-ACEs that should be further evaluated. Furthermore, PTSD as it relates to CM-ACEs and aggression has largely been studied as a homogenous construct and retrospectively in adult samples, many of which have been limited to samples of participants who already had a diagnosis of PTSD. Because PTSD is thought to be underdiagnosed in youth (Grasso et al., 2009), it is important to investigate the role of these symptoms outside the context of a clinical diagnosis. Ultimately, examining differential associations between individual PTSS clusters, ACEs related to CM-ACEs, and subtypes of aggression is warranted, especially earlier in development when pathology is emerging, and children are more sensitive to the effects of stress and the effects of intervention.

The overarching goal of the present study was to examine relations between the experience of CM-ACEs, PTSS clusters, and proactive and reactive aggression in a clinical sample of male youth. More specifically, we expected that CM-ACEs would be significantly related to proactive and reactive aggression; however, the underlying mechanisms to explain these relations would differ across PTSS clusters. Consistent with the social information processing theory of aggression, it was expected that intrusive, negative mood/cognitions and altered arousal symptoms would explain the indirect effect of CM-ACEs to reactive aggression while the indirect effect to proactive aggression would be explained by the avoidance cluster of PTSS symptoms. Aggression and rates of PTSD diagnoses differ across gender; however, the present study focuses on male children and adolescents for several reasons. First, boys are exposed to more ACEs, including certain types of CM-ACEs (Blum et al., 2019), than girls, yet they are less likely to be diagnosed with PTSD (Breslau et al., 2004; Mueser & Taub, 2008). Research suggests that boys often exhibit clinically significant levels of post-traumatic stress symptoms that are commensurate with those observed in their female

counterparts yet are more likely to be given diagnoses of ADHD, Conduct Disorder, and/or ODD than PTSD (Lahey et al., 2012; Mueser & Taub, 2008). This pattern of findings appears to be particularly true in inpatient treatment settings (Mueser & Taub, 2008) where PTSS might be overshadowed by externalizing behavior that often precipitate the need for higher levels of care in male youth.

Method

Participants

Participants included male youth receiving educational and clinical services in a residential treatment setting (N = 86). Most participants were in state custody and had a documented history of abuse and/or neglect. Inclusion criteria for participation included being enrolled in the residential treatment and school services at the treatment facility, being between 6 and 14 years of age, having a verbal IQ standard score of 75 or higher, and no psychosis-related diagnoses at the time of data collection.

Procedure

Data collected for this study was completed as part of a quality assurance program in the school setting imbedded within the residential treatment facility. The quality assurance program was designed to evaluate the use of positive behavior supports on child outcomes in the classroom environment. Participants were asked if they would be willing to complete a packet of assessment measures that could be used for later research. If the child assented, he was given the option of completing the self-reports independently or with the help of a research assistant. Data were collected within two weeks of the child's admission to the treatment facility to minimize the effects the treatment (e.g., positive behavior supports, medication management, therapy services, etc.) and/or the impact that living in a safe, structured setting might have on the variables of interest.

Measures

Reactive and Proactive Aggression. Aggression was assessed using child self-report responses on the Dodge and Coie (1987) measure of proactive and reactive aggression. The measure includes 6 items, 3 assessing proactive aggression, and 3 assessing reactive aggression. Participants reported on items using a 5-point Likert scale (1 = Never to 5 = Almost Always), indicating how often they engage in a particular behavior. Previous research suggests good psychometric properties for the measure when used as a self-report instrument (Fite et al., 2009; Waschbusch et al., 1998). Internal consistency in the present study was commensurate with that reported in the literature (α = .76).

Child Maltreatment. The Adverse Childhood Experiences – Short Form (ACE-SF; Dube et al., 2003; Felitti et al., 1998) is a retrospective, self-report questionnaire that measures lifetime exposure to trauma-related events, including experiences of neglect, physical, sexual, and emotional abuse, and several types of household dysfunction (e.g., witnessing violence, having a family member incarcerated, etc.). The CM-ACEs variable for the present study was comprised of the 4 child maltreatment ACE items. Items are phrased as questions to be answered in a "yes" or "no" format, and the measure is scored as a total sum of items endorsed as "yes". For the purposes of the present study, some of the language was modified from the original ACE-SF to be more age-appropriate for the sample while maintaining the content of the original questions. Internal consistency for the ACE-SF has been found to be "good to excellent" (Dube et al., 2003).

Post-Traumatic Stress Symptoms (PTSS). The UCLA PTSD Reaction Index for DSM-5 (PTSD-RI-5; Elhai et al., 2013; Steinberg et al., 2004, 2013) is a measure designed to assess the presence of PTSS, including disturbances in thoughts, behaviors, and functioning. Self-report of symptoms was used in the present study. The current version of the UCLA PTSD Reaction Index reflects the diagnostic criteria for PTSD in the DSM-5 and includes statements that assess the presence of re-experiencing, avoidance/numbing, negative or distorted cognitions, hyperarousal, and dissociative symptoms (APA, 2013). Respondents rate these statements on a 5-point scale (0 = None of the time to 4 = Most of the time) to indicate how often they have experienced symptoms in the past month. Psychometric properties of the UCLA PTSD Reaction Index for *DSM-5* are considered "good to excellent" (Elhai et al., 2013; Steinberg et al., 2013). In the present study, the PTSD-RI-5 had high internal consistency (α = .93).

Data Analysis Plan

Using SPSS software (IBM Version 26, 2019), stepwise regression analyses were performed to examine relations between PTSS clusters and aggression. Proactive and reactive aggression were entered as dependent variables to determine which PTSS clusters were significantly related to each aggression subtype. Based on the results of the regression equation and preliminary bivariate correlations, a path analysis model using maximum likelihood estimation was performed to examine direct and indirect paths from CM-ACEs and PTSS clusters while simultaneously examining proactive and reactive aggression as multiple outcome variables. Covariances between a) proactive and reactive aggression and b) the included PTSS clusters were specified in the model, resulting in a fully saturated model. Consequently, fit indices are not reported; however, a post-hoc analysis to examine overall model fit was conducted using Wald Test of Parameter. Path analyses were performed using MPLUS 8.4 software (Muthen & Muthen, 2017).

Results

Sample Characteristics

The average age of participants was 10.44 (*SD* = 2.29), and the sample ranged from 6 to 14 years of age (see Table 1). Data were collected from a treatment facility in the Southeastern United States and the racial and ethnic distribution of the sample is representative of the area in which data were collected, with nearly 60% of the sample identifying as Caucasian, 35% as African-American, and 5% identifying as another racial or ethnic group . Most participants had a documented history of more than one type of CM-ACEs (*M* = 1.80; *SD* = 0.91), with participants endorsing an average of 2.16 (*SD* = 1.06) CM-ACEs items. Upon admission to the treatment program, the average number of diagnoses was 3.14 (SD = 0.99). Notably, only 23% of participants had a diagnosis of a Trauma- or Stress-Related Disorder at the time of admission. As seen in Table 1, participants endorsed high levels of proactive aggression, reactive aggression, and PTSS compared to other clinical/high-risk youth populations. This was true both in terms of overall PTSS and within each PTSS cluster (Fite et al., 2009; Kaplow et al., 2020).

Stepwise Linear Regressions

Separate stepwise linear regressions were performed in which proactive and reactive aggression were each entered as the dependent variables (DV) and regressed on all four PTSS clusters. Neither age nor race, which were entered as control variables in both analyses, were associated with either outcome variable. The overall model for proactive aggression entered as the DV was significant [F (1, 84) = 21.30, $p < .01$, $R^2 = .20$], but only Negative Mood/Cognitions emerged as a significant independent variable (IV; $\beta = .45$; $t = 4.62$, $p < .01$). Similarly, the overall model for the regression analysis in which reactive aggression was regressed on all PTSS clusters was significant, as well (F (2, 83) = 19.15, $p < .01$, $R^2 = .30$]. In this model, two PTSS clusters – Negative Mood/Cognitions ($\beta = .30$; $t = 2.37$, $p = .02$) and Intrusion ($\beta = .31$; $t = 2.49$, $p = .02$) emerged as significant IV's.

Path Analysis

Bivariate correlations established significant associations between CM-ACEs, PTSS clusters, and both subtypes of aggression. Stepwise regression analyses then established which PTSS clusters were significantly related to the aggression subtypes. Based on these preliminary findings, a path analysis model was performed to simultaneously evaluate relations between CM-ACEs, the Intrusion and Negative Mood/Cognitions PTSS clusters, proactive aggression, and reactive aggression (Figure 1).

Results indicated that CM-ACEs were significantly related to Intrusion (b = 2.38, SE = .53, β = .40, p < .01) and Negative Mood/Cognitions (b = 5.34, SE = 1.05, β = .47, p < .01). CM-ACEs were marginally associated with reactive (b = .16 SE = .13, β = .15, p = .06) but not proactive aggression. Similar to the results of the regression analyses, Negative Mood/Cognitions was marginally significantly related to both proactive (b = .02, SE = .01, β = .13, p = .06) and reactive aggression (b = .02, SE = .01, β = .11, p = .08) while the Intrusion symptom cluster maintained a significant association with reactive aggression (b = .03, SE = .03, β = .31, p = .03). Indirect effects of PTSS symptom clusters on relations between CM-ACEs and aggression were also examined. Results yielded a significant indirect effect of Intrusion on the relation between CM-ACEs and reactive aggression (b = .14, SE = .07, β = .13, p < .05). Marginally significant indirect effects of Negative Mood/Cognitions on the relations between CM-ACEs and both proactive (b = .12, SE = .07, β = .11, p = .09) and reactive aggression (b = .11, SE = .06, β = .10, p = .08) emerged, as well. The total indirect effects within the model were significant for both proactive (b = .18, SE = .06, β = .17, p < .01) and reactive aggression (b = .25, SE = .06, β = .23, p < .01). Results of a Wald Chi-Squared test [χ^2 (8, 86) = 52.23, p < .01] indicated that the combined direct and indirect effects in the overall model significantly predicted the outcome variables, thus supporting the fit of the model to the data.

DISCUSSION

The goal of the present study was to examine relations between CM-ACEs, PTSS clusters, and subtypes of aggression in a clinical high-risk sample of male youth. We sought to examine potential indirect effects from CM-ACEs to the proactive and reactive subtypes of aggression through PTSS clusters. To date, most research examining relations among these variables have examined both PTSD and aggression as unitary constructs. Although a few studies have examined differential relations between PTSS clusters and overall aggression, most of these studies have been limited to adults in military settings (Watkins et al., 2017). As such, existing research on these issues might have limited generalizability to children and adolescents. In light of these gaps in the literature, the present study contributes to this body of research in meaningful ways by parsing the broad constructs of post-traumatic stress and aggression to examine differential effects of child maltreatment-related PTSS clusters on aggression subtypes during childhood.

Results suggested significant relations among CM-ACEs and proactive and reactive aggression as well as relations between and among PTSS clusters and these variables. Our findings are consistent with existing literature that identifies significant associations between CM-ACEs and PTSD (Moore et al., 2013), proactive aggression (Augsburger et al., 2017), and reactive aggression (Richey et al., 2016). It was hypothesized that the Intrusion, Altered

Arousal/Reactivity, and Negative Mood/Cognitions symptom clusters would be related to reactive aggression. However, only the Intrusion and Negative Mood/Cognitions PTSS clusters emerged as significantly related to reactive aggression, and in contrast to our hypothesis that avoidance would explain the relation between CM-ACEs and proactive aggression, only negative mood/cognitions symptom cluster was found to have a marginally significant indirect effect.

Although most findings were consistent with our predictions and with existing literature, results indicating a non-significant relation between Altered Arousal/Reactivity and reactive aggression despite elevated levels of PTSS endorsed (Makin-Byrd et al., 2012; Taft et al., 2007). However, previous research has included adult populations and did not distinguish between subtypes of aggression. This finding might also reflect noted differences in the presentation of PTSD between adults and children, further emphasizing the importance of conceptualizing PTSS through a developmental psychopathology lens (Cicchetti & Toth, 1995; Margolin, 2005; McLaughlin & Lambert, 2017). Most developmental models of PTSD acknowledge the impact of altered arousal and reactivity on negative outcomes that occur in part because of the effects of traumatic stress on early brain development (Ford, 2005; van der Kolk, 2005). The neural changes associated with traumatic experiences early in development may impact the cognitive, linguistic, and regulatory capacities required to regulate the experience of other types of symptoms (e.g., intrusive thoughts) or the ability to challenge negative cognitions associated with PTSD. Consequently, these PTSS clusters might emerge as more strongly related to aggression than hyperarousal in children and adolescents despite theoretical support that hyperarousal could also contribute to aggressive behavior.

The Intrusion and Negative Mood/Cognitions PTSS clusters were included in a path analysis model with CM-ACEs, proactive aggression, and reactive aggression. As expected, CM-ACEs were significantly related to the both the Intrusion and Negative Mood/Cognitions PTSS clusters. This finding is consistent with existing literature that identifies PTSD in general as an outcome associated with child abuse and neglect (Moore et al., 2013). CM-ACEs were also significantly related to reactive but not proactive aggression in the model. Although most literature suggests that CM-ACEs has stronger effects on reactive than proactive aggression (Kolla et al., 2013; Richey et al., 2016), this body of literature remains somewhat equivocal as more research has begun to suggest links between CM-ACEs and proactive aggression. The clear overlap between the types of emotional and behavioral dysregulation associated with CM-ACEs and reactive aggression (Shackman & Pollack, 2014; Thibodeau et al., 2015) might make this relation more robust. However, possible links between CM-ACEs and proactive aggression should not be ignored, especially given that the pattern of relations in the present study appears to suggest conceptual links between these constructs. Similar conceptual

links have been established in the literature, with research supporting decreased empathy, the desire to regain feelings of power and control, and a propensity to rationalize aggressive behaviors as possible outcomes associated with CM-ACEs (Kreps & Gonzalez, 2010). These outcomes represent clear overlaps with characteristics of proactive aggression. Thus, the link between CM-ACEs and reactive aggression may be more robust, the associations between CM-ACEs and proactive aggression should be not be disregarded.

The significant relations observed between the Intrusion and Negative Mood/Cognitions PTSS clusters and reactive aggression were consistent with our hypotheses. These findings might be best understood through the social information processing theory of aggression in which reactive aggression is thought to be driven by the inaccurate encoding and interpretation of social cues (Crick & Dodge, 1996). The presence of Intrusion and Negative Mood/Cognitions might increase the likelihood of these errors. Similarly, symptoms within the Negative Mood/Cognitions cluster could also influence the way social cues are interpreted. For example, making hostile attributions towards ambiguous social cues is a common interpretation error related to reactive aggression (Crick & Dodge, 1994). Several cognitive distortions associated with PTSS, such as beliefs that the world is unsafe, might predispose one to making such hostile attributions. These errors in social information processing and propensity towards reactive aggression might be further exacerbated by the emotion regulation deficits commonly associated with both of these PTSS clusters, as well (Chan et al., 2010). Common negative alterations in mood associated with PTSS are persistent feelings of anger and fear (Fani et al., 2011). These emotional states might increase the likelihood of attending more to cues that confirm these feelings (attentional bias), thereby further predisposing a child to reactive aggression.

We hypothesized that the Avoidance cluster would be related to proactive aggression. However, Negative Mood/Cognitions emerged as the only PTSS cluster related to proactive aggression. This finding is of particular interest in light of equivocal research on PTSS and proactive aggression. As with reactive aggression, it might be that the Negative Mood/Cognitions cluster is related to proactive aggression through the ways in which symptoms within this cluster influence errors in social information processing. For proactive aggression, these errors are related to the goal-clarification and response-decision stages of social information processing (Crick & Dodge, 1996). For example, individuals who exhibit high levels of proactive aggression often demonstrate a propensity towards agentic goals (Crick & Dodge, 1996; Salmivalli, 2005). This propensity might be driven by a cognitive bias related to power orientation and the need to protect oneself from negative appraisal, which are commonly associated with PTSS (Cuadra et al., 2014; Oostermeijer et al., 2017). The effects of such cognitive biases on goal-clarification (and thus proactive aggression)

might also be exacerbated by negative alterations in mood that are commonly associated with PTSS such as blunted affect and emotional detachment (Hoeve et al., 2012; van der Vegt et al., 2009) which could lead one to respond in more callous or non-empathic ways, resulting in a proactive aggressive response style.

In addition to significant direct effects, the significant indirect effects of the PTSS clusters that emerged for the relation between CM-ACEs and proactive and reactive aggression are important to note. Results revealed that the Intrusion symptom cluster emerged as a part of the significant indirect effect of CM-ACEs and reactive aggression, even after accounting for the Negative Mood/Cognitions symptom cluster and proactive aggression. Essentially, symptoms within the Intrusion symptom cluster appear to help explain how the experience of maltreatment might predispose children to reactive aggression. In further accordance with the social information processing theory of aggression, it might be that children who experienced maltreatment are further predisposed to reactive aggression through the effects of Intrusive PTSS on the encoding and interpretation of social information.

The Negative Mood/Cognitions PTSS cluster emerged alongside the Intrusion PTSS cluster as having a marginally significant indirect effect on the relation between CM-ACEs and both subtypes of aggression. Although this marginal effect should be interpreted with caution, the role of negative mood/cognitions of PTSS in this model are not negligible. Existing research suggests that the Intrusion and Negative Mood/Cognitions PTSS clusters are highly related. Specifically, some studies have shown that maladaptive cognitions and cognitive processes (e.g., rumination, minimization, etc.) exacerbate the risk of experiencing intrusive symptoms; furthermore, the bidirectional nature of the interactions between symptoms within these clusters appear to hinder the effective processing of trauma (Elwood et al., 2009; Kearney et al., 2010) which increases the risk of negative outcomes such as aggression. The potential role of the Intrusion and Negative Mood/Cognitions PTSS clusters as multiple mediators between CM-ACEs and reactive aggression should be considered for future research.

Clinical Implications

Findings from the present study have several implications for clinical practice. First, results suggest that many youths presenting with externalizing behavior concerns may need to be considered for trauma-informed treatments. In the present study, relatively few participants met full diagnostic criteria for PTSD, yet most endorsed significant levels of symptoms in several PTSS clusters. Clinicians should be aware of the importance of broadening conceptualizations of post-traumatic stress beyond the constraints of diagnostic criterion sets. The present study also highlights the differential effects of PTSS clusters on aggression in children who have experienced maltreatment. Implications for

clinical practice include the importance of treatment approaches that specifically target intrusive symptoms and negative alterations in mood and cognition in developmentally appropriate ways. For example, interventions aimed at integrating fragmented memories, which are most commonly related to intrusive symptoms, might be particularly beneficial. One evidenced-based approach to integrating traumatic experiences is the development of a trauma narrative, a core component of trauma-specific interventions such as Trauma-Focused Cognitive Behavior Therapy (TF-CBT; Cohen et al., 2012) and KID Narrative Exposure Therapy (KIDNET; Ruf et al., 2012). Trauma narratives promote the integration of traumatic memories and the development of more adaptive appraisals and schemas (Neuner et al., 2004) which, in light of the present study, seems especially important for children and adolescents. To account for developmental factors, the use of adapted approaches to narrative therapy such as the use of a play, drawing, and/or storytelling instead of writing to develop a trauma narrative might be beneficial. Other alternate methods that place fewer cognitive and linguistic demands on children should also be considered. For older children and adolescents, a focus on skill-building in areas such as self-regulation and mindfulness could be incorporated with narrative therapy to strengthen the child's ability to process and integrate painful memories and events without experiencing re-traumatization (Ford & Cloitre, 2009).

Despite having statistically marginal effects, the role of the Negative Mood/Cognitions cluster suggests other potential implications for clinical practice. Specifically, these findings underscore the importance of the cognitive processing and restructuring components of most evidence-based treatments for PTSD (Strand et al., 2013). However, developmental factors should also be considered in cognitive-based therapies, such as children's tendencies towards concrete thought patterns and inductive reasoning (i.e., making broad generalizations from specific information; Piaget, 1971). These cognitive patterns might limit the effectiveness of trauma-focused interventions on changing cognitive biases and thought distortions for children; instead, interventions aimed at targeting this PTSS cluster might focus on skills-building approaches to emotion regulation and executive functioning to address negative alterations in mood and cognitive processes, respectively. Finally, the results of the present study have important implications for future applied clinical research. Although the indirect effect of altered mood and cognitions were only marginally significant, existing theory and research support the idea of negative mood/cognitions and intrusive symptoms as multiple mediators of the relation between CM-ACEs and reactive aggression. The social information processing theory provides a strong theoretical foundation for the connection between these two symptom clusters as well as their significant association

with aggression. Future research should continue to examine these symptom clusters as potentially inter-related mechanisms of influence on negative outcomes of CM-ACEs.

Limitations

Some limitations should be considered when interpreting these results. First, although the sample size met guidelines regarding the number of observations per model parameter and variable (e.g., Bentler & Chou, 1987), the sample size was still relatively small given the complexity of the model. Nevertheless, the direct and indirect effects that did emerge might speak to the strength of the model. Future research should replicate these analyses within a larger sample and using other informants/direct observations of behaviors rather than relying on child self-report measures. Furthermore, this sample represents clinical male youth with significant exposure to CM-ACEs and reportedly high levels of both PTSS and aggressive behaviors. As such, findings have important clinical implications for youth with this high level of need; however, results might be less applicable to children and adolescents across a wider range of trauma exposure and/or clinical need. Additionally, future research should examine possible differential effects of sex on these study variables. Finally, results of the present study are consistent with existing research that examines the mediating role of PTSS clusters – especially intrusion and negative alterations in mood and cognitions – in several longitudinal studies (e.g., Zhou & Wu, 2016). However, the data in the present study are cross-sectional, thus mediated effects should not be interpreted as causal relations. Longitudinal examinations of these variables within child and adolescent populations should be a target for future research.

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Table 1

Demographic, Sample Means, and Frequencies on Study Variables

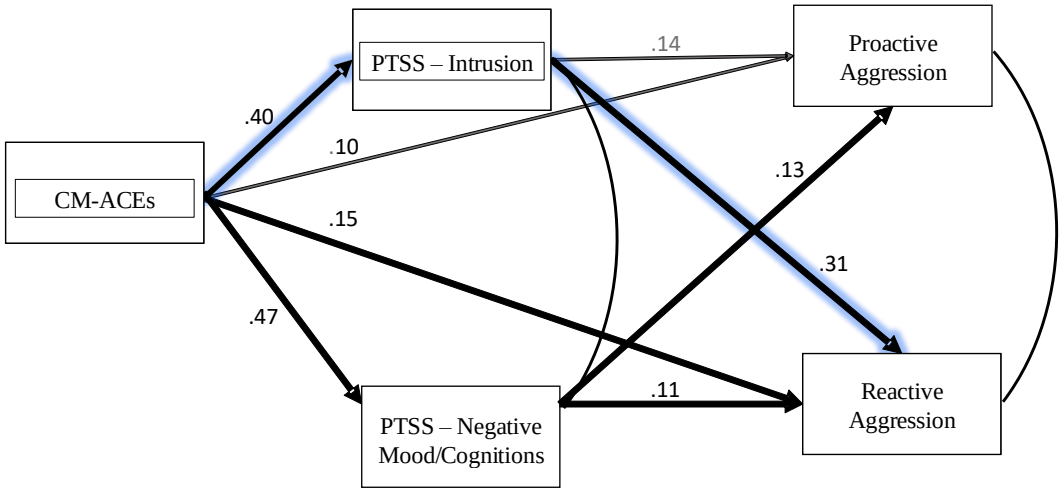
Variables	*M (SD)*	Percent
Age[+]	10.44 (2.99)	
Race/ethnicity		
Caucasian	–	59.3
African American	–	34.9
Other	–	5.8
Documented history of abuse and/or neglect		68.6
Physical abuse	–	46.6
Psychological abuse	–	11.0
Sexual abuse	–	20.1
Neglect	–	57.5
State custody at time of admission	–	69.0
Diagnoses at time of admission		
ADHD	–	81.2
Other neurodevelopmental disorder	–	41.2
Mood disorder	–	42.4
Anxiety disorder	–	12.9
Trauma- or stress-related disorder	–	27.4
Disruptive, impulse-control, and conduct disorder	–	69.4
CM-ACEs score	2.16 (1.06)	–
PTSD Reaction Index for DSM-5*	–	–
Total PTSS score	39.20 (18.39)	46.5
PTSS – Intrusion	10.08 (6.18)	66.3
PTSS – Avoidance	4.15 (2.60)	65.1
PTSS – Negative Mood/Cognitions	12.62 (6.49)	64.0
PTSS – Altered Arousal/Reactivity	12.12 (6.02)	61.6
Proactive aggression	2.20 (1.15)	–
Reactive aggression	3.27 (1.16)	–

**Percent of the sample considered to have endorsed clinically significant levels of these symptoms based on *PTSD Reaction Index for DSM-5* scoring criteria

Notes: CM-ACEs = Child maltreatment – Adverse Childhood Experiences; PTSS = Post-Traumatic Stress Symptoms

[+]Four of the children were 6 years of age with 40% of the sample falling between 6 and 9 years of age, 36% falling between 10 and 12 years of age and 24% were ages 13 to 14.

Figure 1. Path analysis model with standardized path coefficients



Notes: CM-ACEs = Child maltreatment – Adverse Childhood Experiences; PTSS = Post-Traumatic Stress Symptoms