

The Effects of Trauma Type and Executive Dysfunction on Symptom Expression of Polyvictimized Youth in Residential Care

Hilary B. Hodgdon,^{1,3} Rachel Liebman,¹ Lia Martin,² Michael Suvak,³ Kari Beserra,² Wendy Rosenblum,⁴ and Joseph Spinazzola¹

¹The Trauma Center at Justice Resource Institute, Brookline, Massachusetts, USA

²Justice Resource Institute, Needham, Massachusetts, USA

³Suffolk University, Department of Psychology, Boston, Massachusetts, USA

⁴The Victor School, Justice Resource Institute, Acton, Massachusetts, USA

Risk for traumatic sequelae is conveyed directly by risk factors (i.e., exposure to trauma), and via the disruption of developmental competencies. Exposure to caregiver trauma is an especially salient risk factor, as its early and pervasive nature is likely to undermine multiple facets of development, most notably the emergence of cognitive controls (i.e., executive function [EF]). Deficits in EF have been observed among youth exposed to multiple types of trauma and are associated with a range of functional impairments, posttraumatic stress symptoms (PTSS), and behavioral disorders; they represent a mechanism by which the negative impact of caregiver trauma is conveyed. This study included 672 youth in residential placement, and examined the associations between both caregiver and noncaregiver trauma, measured by the Trauma History Profile (THP); executive dysfunction, measured by the Behavioral Inventory of Executive Function (BRIEF); PTSS, measured by the UCLA Posttraumatic Stress Disorder (PTSD) Reaction Index (PTSD-RI); and externalizing and internalizing problems, measured by the Child Behavior Checklist (CBCL). A structural equation model demonstrated direct associations between caregiver trauma and PTSS, $\beta = .15$; noncaregiver trauma and externalizing problems, $\beta = .14$; gender and PTSS, $\beta = .26$, externalizing problems, $\beta = .12$, and internalizing problems, $\beta = .26$; and age and externalizing problems, $\beta = -.11$. We observed indirect effects via deficits in EF between caregiver trauma and PTSS, $\beta = .04$ and externalizing problems, $\beta = .19$. Results indicate for screening for executive dysfunction among trauma-impacted youth is needed, as it represents a critical therapeutic target.

Exposure to multiple forms of trauma, termed “polyvictimization,” has myriad deleterious effects on youth, and predicts the presence and severity of traumatic sequelae above and beyond specific forms of trauma or repeated forms of the same trauma (Finkelhor, Ormrod, & Turner, 2007; Finkelhor, Turner, Hamby, & Ormrod, 2011). Youth who live in “dangerous families,” where child maltreatment and domestic violence occur, and/or in families suffering from additional adversities (i.e., poverty, substance abuse) that disrupt caregiving, are more likely to experience polyvictimization (Finkelhor et al., 2011). This “caregiver trauma” has severe and lasting effects and is associated with an increased risk for a range of clinical problems (Kisiel et al., 2014). The neurobiological impact of caregiver trauma and resulting disruptions to cognitive development

appear to be pervasive, and are hypothesized to be influenced by the early and chronic nature of interpersonal childhood trauma (see Cross, Fani, Powers, & Bradley, 2017, for a review).

Although caregiver trauma represents an important risk factor for psychopathology, youth display heterogeneity in outcomes, necessitating examination of the processes that underlie the association between exposure to caregiver trauma and later dysfunction. Developmental psychopathology (DP; Cicchetti & Toth, 1995) emphasizes the disruption of salient developmental tasks as individual-level mechanisms conferring risk for psychopathology. The multilevel analytic approach of DP posits that individual and contextual risk factors place children at risk for negative outcomes, both through direct (main) effects and via the interaction of risk factors over time (mediation and moderation).

The disruption of cognitive development represents one potential mechanism that may link exposure to caregiver trauma and resulting traumatic sequelae. Trauma that occurs within the caregiving system represents extreme failures in the expectable environment. These failures in the early attachment system interfere with the development of the child’s nascent stress-coping system (Schore, 2001). As a result, the development of higher-order cognitive controls, such as executive function (EF), may

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Correspondence concerning this article should be addressed to Hilary B. Hodgdon, The Trauma Center at Justice Resource Institute, Brookline, MA, 02446. E-mail: hhodgdon@jri.org

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be disrupted due to the prioritization of limbic responses that support survival (Perry, 2009). Disruption of cognitive control, including EF, represents one mechanism by which youth may adapt to early failures in the attachment system.

Executive function refers to deliberate, top-down neurocognitive processes such as cognitive flexibility, working memory, and inhibitory control (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000) that are central in navigating almost all facets of functioning. Two primary functional domains are encompassed by EF: metacognition, which includes cognitive processes necessary for initiating, planning, and executing activities; and behavioral regulation, which includes cognitive and emotional processes involved in managing behavioral reactions (Gioia, Isquith, Guy, & Kenworthy, 2000). A person's EF develops rapidly across childhood (Zelazo, 2004), and trauma-impacted children display deficits in many indices of EF, including working memory (Augusti & Melinder, 2013), attention (Buckner et al., 2012), abstract reasoning, planning, inhibition, problem solving, and cognitive flexibility (Beers & De Bellis, 2002; DeBellis, Hooper, Spratt, & Woolley, 2009).

Symptoms of posttraumatic stress (PTS), as well as internalizing and externalizing problems, have been linked to EF deficits in trauma-impacted children (Beers & De Bellis, 2002; DeBellis et al., 2009), suggesting that deficits in EF may represent a "transdiagnostic" factor underlying a multitude of clinical presentations (Synder, Mikaye, & Hankin, 2015). Children with maltreatment-related posttraumatic stress disorder (PTSD) display deficits in a range of higher-order cognitive processes, and greater cognitive deficits are correlated with PTSD symptom severity (see Carrion, Wong, & Kletter, 2013 for a review). Deficits in EF heighten risk for externalizing problems, including aggression and delinquency, through increased impulsivity, reactivity, and risk-taking behaviors (Fite, Goodnight, Bates, Dodge, & Pettit, 2008; Seguin, Pihl, Harden, Tremblay, & Boulerice, 1995; Vigil-Colet & Codorniu-Raga, 2004). Internalizing problems, on the other hand, are associated with low attentional regulation and low levels of impulsivity, suggesting a distinct profile of regulation deficits reflective of *over* control rather than *under* control (Eisenberg et al., 2001). Despite the evidence that disruptions in cognitive controls are commonly observed, and linked to clinical and behavioral problems, among maltreated children, few studies have examined EF as a mediator of the association between trauma exposure and traumatic sequelae.

Youth in residential settings have been shown to have high rates of trauma exposure and severe impairments in cognitive control, exhibiting poor decision-making skills and impulsivity, which lead to high risk behaviors (e.g., self-harm, running away, aggression, delinquency; Layne et al., 2014). Exposure to multiple types of traumatic events is the rule rather than the exception among youth in residential care (Spinazzola et al., 2005), with an average of 5.6 types of traumatic events reported, compared to an average of 3.6 types for youth in lower levels of care (Briggs et al., 2012). Youth in residential care also have higher rates of caregiver trauma (including physical, sexual,

and emotional maltreatment), exposure to domestic violence, and impaired caregiving (Briggs et al., 2012; Singer, 2007). The combination of exposure to caregiver trauma and deficits in cognitive control makes youth in residential treatment the ideal population in which to examine the mechanistic role of EF in the association between trauma exposure and traumatic sequelae.

Finally, from a DP perspective, EF provides a useful exemplification of the continuity between adaptation and maladaptation, and the importance of treatment interventions that realign youths' behavior with their current "normal" (Cicchetti & Toth, 1995). Executive processes are critical during adolescence, as youth become more independent and autonomous during that time. Few studies have examined the link between EF and trauma exposure among adolescents, despite the obvious implications this association has for their development (DeBellis et al., 2009). Understanding the role of impaired EF in the emergence of psychopathology in adolescence could inform interventions that target the impaired judgment and decision-making skills often seen in this population.

The primary aim of our study was to test an indirect effects model of EF to explain the association between exposure to two categories of trauma (caregiver and noncaregiver trauma) and mental health symptoms, among youth in residential care. Specifically, we hypothesized: (a) caregiver and noncaregiver trauma exposure would be associated with PTS symptom severity, and severity of internalizing and externalizing problems; (b) exposure to caregiver trauma specifically would be associated with greater deficits in EF; and (c) executive dysfunction would indirectly link caregiver trauma exposure and internalizing problems, externalizing problems, and PTS symptom severity.

Method

Participants

Data for this study were collected from a deidentified quality improvement database from a large behavioral health provider in the New England region of the United States. The sample consisted of 672 youth across 14 residential facilities (group home, residential treatment, and secure treatment and juvenile justice settings) who were between the ages of 11 and 18 years ($M_{\text{age}} = 15.32$ years, $SD = 1.62$) and 52.4% female. The racial and ethnic composition of the sample included 0.6% American Indian or Alaskan Native, 1.5% Asian, 12.8% Black/African American, 0.2% Native Hawaiian/Other Pacific Islander, 67.6% White, 16.9% biracial or multiracial, and 18.9% Latino/Hispanic individuals. Youth had involvement with several types of official systems. Over 50% of participants experienced out-of-home placement in their lifetime (in addition to the residential placement in which they were living at the time of participation), with a mean number of 3.69 ($SD = 5.45$) out-of-home placements; additionally, 33.9% of youth were in the custody of the Department of Children and Families and 3.2% were in the custody of the Department of Youth Services.

About one third of the sample had an arrest history, with a mean number of 1.43 arrests ($SD = 1.81$).

Procedure

Upon their intake to the residential facility, participants consented to taking part in quality improvement activities as part of their clinical care, and were informed that deidentified data would be utilized in research. The Justice Resource Institute Institutional Review Board provided approval for this research. Participants were informed that they could decline participation, or could refuse to complete any or all measures, without impact to their treatment. Measures were administered within 30 days of program intake and included a combination of self-report, clinician-report, and teacher-report measures. Participants who completed measures were given a \$10 gift card.

Measures

Lifetime trauma exposure. We measured lifetime exposure to trauma using the Trauma History Profile (THP) of the UCLA Posttraumatic Stress Disorder Reaction Index (Steinberg, Brymer, Decker, & Pynoos, 2004). The THP assesses exposure to 20 trauma types, including both interpersonal and noninterpersonal traumas. Lifetime exposure, indicated by one of four response options (“yes,” “no,” “suspected,” and “unknown”), is rated by the participant’s clinician, using multiple sources of information (e.g., client- and caregiver-report, record review). In the current study, trauma exposure items on the THP endorsed as “yes” or “suspected” were considered a positive endorsement of event exposure. We then coded all trauma events dichotomously (yes or no) for lifetime exposure. Exposure to the number of caregiver-perpetrated trauma types (i.e., neglect; physical, sexual, and psychological abuse; impaired caregiving; exposure to domestic violence; and separation from caregiver) and noncaregiver-perpetrated trauma types (i.e., physical and sexual assault, community and school violence, natural disaster, illness and/or medical, injury and/or accident, war, kidnapping, trafficking, bullying, bereavement, and extreme interpersonal violence) were then summed to create two trauma total variables: caregiver trauma and noncaregiver trauma. A log transformation was performed on the noncaregiver trauma variable to correct skew. In the current study, the THP demonstrated acceptable reliability, with a Cronbach’s alpha value of .72.

PTS symptoms. We used the checklist portion the UCLA PTSD-RI to evaluate the presence and frequency of *DSM-5* PTS symptoms during the past month. In this assessment, items map directly onto PTSD diagnostic criteria (i.e., symptom clusters of intrusions, avoidance, arousal, and cognition). In the current study, all items were weighted by frequency, and we used cluster score totals as observed indicators to create a PTS symptoms latent construct. Convergent validity of the symptom checklist portion of the PTSD-RI has been established for the previous version of the measure (UCLA PTSD-RI for *DSM-IV*) but are yet to be established for the current version of the measure

(Steinberg, Brymer, Decker, & Pynoos, 2004). In the current study, the PTSD-RI symptom checklist portion demonstrated internal consistency values ranging from acceptable to excellent, with a Cronbach’s alpha value of .93 for the full scale, and Cronbach’s alpha values ranging from .70 to .87 for reexperiencing, avoidance, arousal, and cognition symptoms.

Child behavior problems. We used the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), a 120-item caretaker report of behavior problems for youth aged 6 to 18 years, to assess children’s behavior problems. The CBCL yields two broad brand scales: Externalizing behavior problems, composed of the Rule-Breaking and Aggression subscales; and Internalizing behavior problems, composed of the Somatic Complaints, Withdrawn-Depressed, and Anxious-Depressed subscales. The CBCL also has adequate test-retest reliability ($r \geq .80$) and interrater reliability ($r \geq .57$). In the current study, the CBCL had excellent internal consistency, with a Cronbach’s alpha value of .88 for the total score, .86 for the Internalizing scale, and .89 for the Externalizing scale.

Executive dysfunction. We assessed executive dysfunction using the Behavior Rating Inventory of Executive Functioning–Teacher Report Form (Gioia et al., 2000), an 86-item observer report of youth executive functioning in the school setting, which yields two theoretically and statistically derived indexes of executive functioning: (a) Behavioral Regulation (BR), composed of Inhibition, Shifting, and Emotional Control; and (b) Metacognition (MC), composed of Initiation, Working Memory, Planning/Organizing, Organization of Materials, and Self-Monitoring. The measure has strong test-retest reliability, with r values ranging from .76 to .85 (Baron, 2000). Cronbach’s alpha values range from .80 to .98 in both normative and clinical samples. In the current study, the BRIEF demonstrated excellent internal consistency, with Cronbach’s alpha values of .97 for the Behavioral Regulation index, and .98 for the Metacognition index.

Data Analysis

Analyses were conducted using SPSS version 22.0, and we utilized IBM AMOS (Arbuckle, 2012) to conduct structural equation modeling (SEM). The maximum likelihood estimation was used to estimate the model using all available data. Four latent constructs were created: (1) executive function (EF), with the BRIEF’s Behavioral Regulation and Metacognition subscales as indicators; (2) externalizing (EXT), with the Rule-Breaking and Aggression subscales of the CBCL as indicators; (3) internalizing (INT), with the Somatic Complaints, Withdrawn-Depressed, and Anxious-Depressed CBCL subscales as indicators; and (4) PTS, with the Arousal, Intrusion, Avoidance, and Cognition symptom cluster totals from the PTSD-RI as indicators.

We tested the primary study hypotheses in three phases. In the first phase, we tested a direct-effects model using SEM, and

examined paths from caregiver trauma, noncaregiver trauma, two control variables (gender and age), and EF to PTS, internalizing, and externalizing symptoms. In the second phase, we tested the hypothesized structural model, and examined: (a) the direct paths from caregiver trauma and noncaregiver trauma to EF and clinical outcome variables; (b) the indirect paths from caregiver and noncaregiver trauma to the clinical outcome variables via EF; and (c) the direct paths from the two control variables to EF and the clinical outcome variables. In the final phase of the analysis, we tested a null model with the regression weights of the indirect paths in the hypothesized model set to zero. The fit of the three models (direct effects, hypothesized, and null models) were compared in order to determine which model had the best fit to the data. The use of SEM in AMOS allowed for the examination of both direct and indirect effects, while accounting for intercorrelations among variables (Kline, 2010). As data were cross-sectional, a theoretically informed order of entry was used, as trauma exposure occurred prior to entry into residential care, and disruptions in EF are developmental, resulting from exposure to early adversity (Cross et al., 2017).

Overall model fit was assessed using the following benchmarks: (a) a comparative fit index (CFI; Bentler, 1990) of .90 or higher; (b) a Tucker-Lewis Index (TLI) of .90 or higher (Hu & Bentler, 1999); and (c) a root mean square error of approximation (RMSEA; Steiger, 1990) value of .08 or less. These benchmarks have been identified as indicators of good model fit (Hu & Bentler, 1999). We compared models by examining the Akaike Information Criterion (AIC) for each model, and conducting chi-square difference tests. Conclusions about model fit were derived from consideration of all indicators; no single measure was considered a “gold standard” (Brown, 2006). We further assessed indirect pathways in the model for statistical significance by using the Monte Carlo bootstrapping procedure in the online software program RMediation (Tofighi & MacKinnon, 2011) to construct 95% confidence intervals. When zero is not within the confidence interval, the indirect effect is considered significant. As estimates of the strength of the indirect paths, we computed the ratio of the total effect from each predictor to the outcome ($c' + ab$) that was accounted for by the indirect effect (ab ; MacKinnon, 2008).

Results

Trauma types and exposure rates are presented in Table 1. Means, standard deviations, and correlations between all study variables are presented in Table 2. Youth were exposed to an average of over five different trauma types over the life course ($M = 5.21$, $SD = 2.70$).

Direct Effects Analysis

A SEM testing of the direct paths from caregiver and noncaregiver trauma, EF, age, and gender to each of the clinical indicators demonstrated adequate model fit, $\chi^2(71, n = 672) = 356.89$, $CFI = .87$, $RMSEA = .08$, $TLI = .79$, $AIC = 484.89$,

Table 1
Rates of Exposure to Each Trauma Type for Residential Youth

Trauma Type	<i>n</i>	%
Neglect	434	64.6
Psychological maltreatment	412	61.3
Physical abuse	370	55.1
Impaired caregiver	329	49.0
Sexual abuse	318	47.3
Domestic violence	270	40.2
Bereavement	226	33.6
Sexual assault	213	31.7
Physical assault	159	23.7
Separation	123	18.3
Illness/medical trauma	93	13.8
Community violence	88	13.1
Serious injury/accident	55	8.2
School violence	47	7.0
Extreme interpersonal violence	34	5.1
Bullying	33	4.9
Kidnapping	19	2.8
Trafficking	14	2.1
War/terrorism outside U.S.	14	2.1
War/terrorism in the U.S.	3	0.4
Natural disaster	2	0.3

Note. $N = 672$.

and explained 12% of the variance for internalizing, 10% of the variance for externalizing, and 11% of the variance for PTS symptoms. A review of the path coefficients indicated statistically significant associations between: (a) caregiver trauma and PTS symptoms, $B = 0.41$, $\beta = .15$, $p = .007$; (b) noncaregiver trauma and externalizing problems, $B = 0.28$, $\beta = .14$, $p = .003$; (c) executive functioning and externalizing problems, $B = 0.20$, $\beta = .19$, $p < .001$, and internalizing problems, $B = -0.01$, $\beta = -.14$, $p = .011$; (d) gender and internalizing problems, $B = 0.02$, $\beta = .27$, $p < .001$, externalizing problems, $B = 0.23$, $\beta = .11$, $p = .021$, and PTS symptoms, $B = 2.81$, $\beta = .26$, $p < .001$; and (e) age and externalizing problems, $B = -0.08$, $\beta = -.12$, $p = .009$.

Indirect Effects Analysis

The primary hypothesis of the study was that impairment in EF would indirectly influence the association between childhood trauma exposure and clinical indicators, including internalizing, externalizing, and PTS symptoms. A model testing both the direct and indirect paths from caregiver and noncaregiver trauma to internalizing, externalizing, and PTS symptoms via EF, with age and gender entered as control variables, was tested next, and demonstrated good fit, $\chi^2(67, n = 672) = 290.71$, $CFI = .90$, $RMSEA = .07$, $TLI = .82$, $AIC = 426.71$, which explained 13% of the variance in internalizing, 10% of the variance in externalizing, and 11% of the variance in PTS

Table 2
Means, Standard Deviations, and Bivariate Correlations among Study Variables

Variable	M	SD	1	2	3	4	5	6	7	8	9
1. Caregiver trauma	3.36	1.99	-	.18**	-.09*	.11**	.15**	.16**	.21**	.22**	.19**
2. Noncaregiver trauma	1.49	1.31		-	.11**	.08*	.13**	.13*	-.00	-.00	.00
3. Age (years)	15.32	1.62			-	.01	-.16**	.02	-.25**	-.24**	-.24**
4. CBCL Internalizing	58.27	9.60				-	.18**	.23**	-.09*	-.04	-.10*
5. CBCL Externalizing	60.95	9.38					-	.09	.24**	.28**	.20**
6. UCLA PTSD-RI	30.91	19.88						-	-.01	.08	-.05
7. BRIEF Executive Composite	78.71	19.16							-	.91**	.96**
8. BRIEF Behavior Regulation	80.41	20.68								-	.78**
9. BRIEF Metacognition	73.49	16.99									-

Note. CBCL = Child Behavior Checklist; UCLA-PTSD-RI = UCLA PTSD Reaction Index; BRIEF = Behavior Rating Inventory of Executive Function. * $p < .05$. ** $p < .01$.

symptoms. In this model (see Figure 1 for standardized estimates and p values; only significant paths and correlations are displayed), the direct path from caregiver trauma to PTS symptoms, $B = 0.40$, $\beta = .15$, $p = .009$, remained significant, as did the path from noncaregiver trauma to externalizing symptoms, $B = 0.28$, $\beta = .14$, $p = .003$. The direct path between gender and internalizing problems, $B = 0.02$, $\beta = .26$, $p < .001$; externalizing problems, $B = 0.25$, $\beta = .12$, $p = .013$; and PTS problems, $B = 2.84$, $\beta = .26$, $p < .001$ remained significant, as did the path from age to externalizing problems, $B = -0.07$,

$\beta = -.11$, $p = .022$. In addition, age, $B = -0.15$, $\beta = -.23$, $p < .001$, and gender, $B = -0.37$, $\beta = -.17$, $p < .001$, were negatively associated with EF. Path analysis demonstrated that caregiver trauma, $B = 0.11$, $\beta = .20$, $p < .001$, was positively associated with EF, which in turn was negatively associated with internalizing problems, $B = -0.01$, $\beta = -.14$, $p = .011$, and positively associated with externalizing problems, $B = 0.19$, $\beta = .19$, $p < .001$.

A test of the indirect effects using RMediation indicated that the path from caregiver trauma to externalizing symptoms

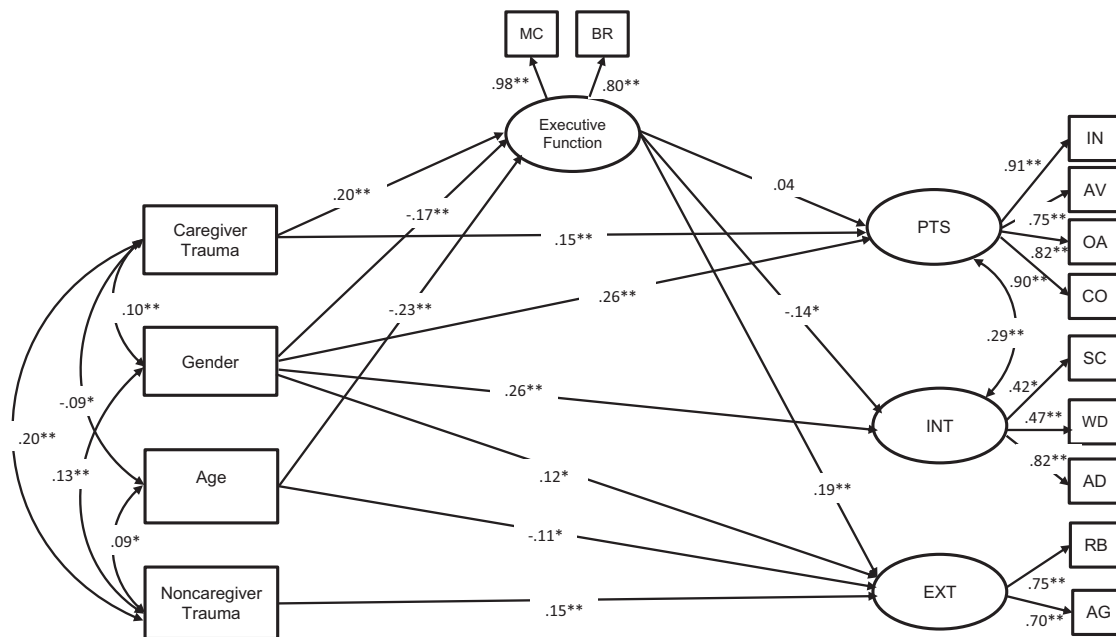


Figure 1. Significant direct and indirect paths and correlations of hypothesized structural equation model. Standardized regression weights and significance indicators displayed. Only significant paths and correlations are displayed. Correlation between endogenous variables represents the association between the residual (error) terms. MC = BRIEF Metacognition; BR = BRIEF Behavioral Regulation; EXT = Externalizing; RB = CBCL Rule Breaking; AG = CBCL Aggression; INT = Internalizing; SC = CBCL Somatic Complaints; WD = CBCL Withdrawn-Depressed; AD = CBCL Anxious-Depressed; PTS = Posttraumatic Stress; IN = PTSD-RI Intrusion; AV = PTSD-RI Avoidance; OA = PTSD-RI Overarousal; CO = PTSD-RI Cognition.

* $p < .05$. ** $p < .01$.

through EF was significant, $\beta = .19$, 95% Monte Carlo CI [.006, .031], and accounted for 38% of the association between caregiver trauma and externalizing problems. Although the path from EF to PTS problems was not significant, $B = 0.20$, $\beta = .04$, $p = .541$, when we used RMediation to examine the indirect association between caregiver trauma and PTS symptoms via EF, a significant indirect path was indicated, $\beta = .04$, 95% Monte Carlo CI [.009, .033], which accounted for 5% of the association between caregiver trauma and PTS symptoms. The path from caregiver trauma to internalizing symptoms through EF was not significant, $\beta = -.14$, 95% Monte Carlo CI [-.013, .012].

As a last step, we tested a null model, with the regression weights of the indirect paths (i.e., paths from EF to externalizing problems, internalizing problems, and PTS symptoms) set to zero. This model demonstrated adequate fit, $\chi^2(70, n = 672) = 311.31$, CFI = .89, RMSEA = .07, TLI = .82. Comparison of the three models using the AIC demonstrated that the hypothesized model had superior fit, AIC = 426.71, in comparison with the direct effects model, AIC = 484.89, and the null model, AIC = 441.31, as indicated by a difference of more than 10 points in the AIC (Burnham & Anderson, 2003). Finally, model comparison using the chi-square difference test indicated that the hypothesized model was significantly different from the direct effects model, $\chi^2(4, n = 672) = 66.18$, $p < .001$, and the null model, $\chi^2(3, n = 672) = 20.61$, $p < .001$.

Discussion

Study results partially supported our primary hypotheses that exposure to caregiver trauma negatively impacts EF, which in turn is associated with a higher level of clinical symptoms. Among a sample of youth in residential care, SEM demonstrated that the total number of caregiver trauma types was indirectly associated with severity of externalizing problems through EF, an effect that is consistent with full mediation. Caregiver trauma was directly and indirectly associated with PTS symptom severity through EF, an effect that is consistent with partial mediation. These findings point to a potential mechanism underlying the association between trauma perpetrated by a caregiver and psychopathology during adolescence.

The link between caregiver trauma, EF, and PTS symptoms among youth is a novel finding. Executive function as assessed in this study is a reflection of youths' ability to modulate emotions, inhibit prepotent responses, shift attention, utilize working memory, and organize thinking and behavior in a goal-directed manner. Disrupted emotional modulation is a core feature of PTSD (van der Kolk, 2014), and incrementally related to greater PTS symptom severity, even when accounting for cumulative trauma exposure (Medina, et al., 2011). In addition, difficulties with inhibition may lead to increased intrusions (i.e., traumatic memories) and physiological arousal, thus increasing PTS symptoms, whereas deficits in cognitive flexibility (i.e., ability to shift attention) may underlie symptoms of avoidance

and the behavioral component of hypervigilance, which represent rigid strategies for coping with stressful stimuli.

Caregiver trauma, but not noncaregiver trauma, was also directly associated with PTS symptoms. This finding is surprising considering that acute forms of trauma subsumed under the non-caregiver trauma category (e.g., sexual and physical assault), fit *DSM-5* Criterion A1 for PTSD. However, it may be that when accounting for the effect of caregiver trauma, notably sexual, physical, and psychological maltreatment (the individual and combined contributions of which have been linked to greater severity of PTS symptoms; Spinazzola et al., 2014), the association between later-occurring and acute forms of trauma and PTS is reduced. This finding requires further exploration and replication to be fully understood.

Caregiver trauma also indirectly predicted externalizing problems, an effect consistent with full mediation. While separate lines of research have shown links between child maltreatment and externalizing problems (VanZomeren-Dohm, Xu, Thibodeau, & Cicchetti, 2016), exposure to familial trauma and EF deficits (DePrince et al., 2009) and EF deficits and behavioral disorders and increased rates of aggression (Seguin et al., 1995; Vigil-Colet & Codorniu-Raga, 2004), no previous studies have examined EF as a mediator of the link between trauma and externalizing problems. The current study findings capture these associations within one model, suggesting that trauma exposure may lead to deficits in EF, which then acts as a mechanism that confers risk for externalizing symptoms.

Noncaregiver trauma was directly associated with externalizing problems, a finding that replicates past research indicating a direct link between trauma exposure and externalizing behaviors such as delinquency (Ford, Elhai, Connor, & Frueh, 2010). Exposure to some of the trauma types subsumed under noncaregiver trauma (physical assault; community, school, and extreme interpersonal violence; and war and/or terrorism) may lead to modeling and internalization of maladaptive interpersonal strategies that contribute to children utilizing aggressive or hostile approaches in their interactions with others (Crick & Dodge, 1994). A complementary explanation for this finding is that engaging in aggressive and/or rule-breaking behaviors represents a survival strategy adopted by youth who may be confronted with injustice and violence on a regular basis in their broader environment.

Due to the cross-sectional nature of the study data and the overlap between some of the core features of PTS symptoms, externalizing problems and EF, namely disrupted emotional modulation, definitive conclusions about the directionality of effects observed in this study cannot be drawn. However, the finding that trauma that occurs within the caregiving system, as opposed to acute forms of trauma (i.e., natural disaster, accidents) and forms of trauma that occur in the broader environment (i.e., community and school violence), was predictive of clinical symptoms via EF, supports our conclusion that EF serves as a mediator of effects. Notably, the developmental timing of exposure to caregiver trauma precedes the emergence of EF, as shown in a study by Pynoos et al. (2014), which examined

a large, clinic-referred population of trauma-exposed children and youth and demonstrated that caregiver trauma (as defined in this study) had an earlier age of onset (i.e., before 5 years of age), while noncaregiver trauma types were more likely to occur after age 6. The early maltreating environment necessitates prioritization of survival-based responses, which over time become ingrained, and take precedence over the development of high-order cognitive capacities that would normally support self-regulation, inhibition and impulse control, problem-solving ability, and attention. Indeed, trauma that occurs early in childhood is more likely to result in neurobiological disruptions that in turn undermine the development of core competencies such as EF (Cowell, Cicchetti, Rogosch, & Toth, 2015), and recent findings from the neuroscience literature demonstrate that the underlying brain regions associated with EF are dysfunctional among youth with maltreatment histories (Teicher & Sampson, 2016).

Neither form of trauma exposure directly nor indirectly predicted internalizing symptoms in the current study, although EF demonstrated a negative association with internalizing symptoms. It is notable that base rates of clinically significant internalizing problems (i.e., a *t*-test score of 65 or higher) were low among participants in this sample (i.e., under 30% of the total sample). Clinicians may have been better able to reliably report on the overt behaviors that comprise externalizing problems, while having difficulty identifying the covert behaviors represented by internalizing behaviors. Additionally, youth in residential care are often referred due to the problematic “acting out” behaviors represented by externalizing problems, while youth who display an internalizing profile may be less likely to receive this level of care. Moreover, internalizing problems may reflect a tendency toward overcontrol of behavior and emotion (i.e., emotional constriction, numbing, etc.), rather than the undercontrol that is associated with deficits in EF. This interpretation is supported by the finding that there was a negative association between EF and internalizing problems. Overmodulation of affect among traumatized individuals has been associated with a distinct neurological profile that reflects increased activation of some of the brain areas involved in EF (i.e., the medial prefrontal cortex; Lanius et al., 2010).

Findings from our study highlight the need to assess and screen for disruptions in child development that result from trauma exposure, such as deficits in EF. Deficits in EF represent a transdiagnostic factor linking trauma to many types of clinical presentations (Snyder et al., 2015), and thus are a critical “driver” of multiple forms of psychopathology, including behavioral problems and PTS symptoms. Assessment of EF should not be limited to performance-based methods, such as those used most routinely in neuropsychological testing, but should also include rating measures that utilize multiple reporters, in order to capture a youth’s EF capacity as it plays out in the real world. Indeed, such rating measures may capture a distinct but complementary underlying mental construct that is reflective of behavior that serves to foster goal achievement, as

opposed to the efficiency of cognition under highly controlled conditions (Toplak, West, & Stanovich, 2013).

The results of this study underscored the need to target developmental capacities, such as EF, as a core component of trauma treatment. Strengths-based interventions that look beyond target symptoms (i.e., PTS symptoms) are sorely needed and should be informed by research on the developmental impact of trauma. The developmental-ecological approach to trauma treatment, as proposed by Pynoos and Steinberg (2006), delineates multiple levels of the child’s ecology as potential intervention points, including areas of “developmental compromise.” Our study’s findings point to a need to target EF directly as part of the treatment process, an approach that may be especially effective during adolescence, a time during which there are significant developmental gains in EF (Steinberg, 2008). Underlying shifts in neurobiology, including development of the prefrontal cortex (PFC) and myelination of pathways between the PFC and the brain regions that support increased cognitive control, create an opportunity for intervention, as “adolescence may be a sensitive or critical developmental period, much like early development in its ability to shape future trajectories” (Keating, 2004, p. 49). Youth may be especially amenable to interventions that support EF during adolescence, as their brains are “primed” to develop these capacities.

Strengthening EF may not only reduce a variety of clinical symptoms and bolster daily functioning, but it might also increase the capacity to tolerate and benefit from trauma-informed treatments. By design, trauma-focused cognitive behavioral therapy (Cohen, Mannarino, & Deblinger, 2006), a widely utilized treatment for pediatric PTSD, requires a baseline degree of EF, as children must be able to think about, express, and shift cognitions and feeling states experienced during a traumatic event. Moreover, EF is also a critical capacity used to support processing of overwhelming emotions and thoughts associated with traumatic memories. In line with this, it is imperative that trauma informed interventions address deficits in EF, as it may be an important first phase of treatment, and serve to increase ability to successfully engage in and fully benefit from treatment.

Targeting deficits in EF is especially salient for youth in residential care. Impaired EF undermines risk evaluation, decision making, consideration of consequences, and future planning, and negatively impacts functioning across a range of settings and situations. These are especially problematic impairments for youth in residential placement, who prototypically show elevated rates of high-risk behaviors, such as substance and alcohol use and/or abuse, sexual promiscuity, running away, and self-harm and suicidal behaviors (Briggs, et al., 2012; Connor, Doerfler, Toscano, Volungis, & Steingard, 2004; Lyons, Libman-Mintzer, Kisiel, & Shallcross, 1998). In fact, it is these very behaviors that result in youth placement in residential care. The confluence of attempts to adapt to the early traumatizing environment (i.e., using substances to numb painful emotions), traumatic reenactment (i.e., youth acting out sexually in an attempt to gain emotional closeness), and deficits in EF (i.e., an

inability to control impulses, generate alternatives, and follow an adaptive course of action) place youth on a trajectory of dysfunction that results in residential placement. If unaddressed, these formative deficits in EF are likely to persist into adulthood and continue to disrupt functioning throughout the life course. It is therefore of the utmost importance to both assess and treat these underlying drivers of high-risk behavior by aiding youth in residential treatment settings in the acquisition and practice of problem-solving skills, versus utilization of overly restrictive and even punitive approaches.

Finally, EF is critical in the ability to respond to changing environmental demands. Children who display deficits in EF experience difficulty in navigating life transitions, including discharging to a lower level of care or back to the family of origin. Indeed, while many youth make substantial gains during residential treatment, those improvements are often lost when they return to the community (Leichtman & Leichtman, 2001). The factors that contribute to recidivism among residential youth are inherently complex; youth are often returning to environments that are less structured and supportive, and exposure to ongoing stress and trauma is often an inevitable occurrence. Therefore, a needed component of the residential treatment process is more intensive step-down services for youth who display deficits in EF, in order to facilitate the use of skills they learned during their course of treatment in “real world” contexts.

The current study had several strengths and limitations. We utilized multiple reporters (youth, clinicians, teachers), which provided a comprehensive assessment of youth symptoms and functioning. The nature of the study sample (youth in residential care) is both a strength and a limitation. This population of youth is understudied, but represents a distinct subpopulation of youth who experience an extreme degree of adversity and psychopathology, thereby limiting the generalizability of the study findings. Due to the nature of the data set, we were unable to account for the impact of trauma-specific characteristics (e.g., chronicity, age of onset, perpetrator type) that may also have contributed to or accounted for some of the observed associations in the model (i.e., caregiver trauma may be longer in duration than noncaregiver trauma). This study utilized cross-sectional data as opposed to longitudinal data, which limited our ability to draw definitive conclusions regarding mediation. The use of cross-sectional data to test mediation may have introduced bias (Maxwell, Cole, & Mitchell, 2011), and therefore the study results require replication with a longitudinal sample. The lack of performance-based methods (i.e., computer paradigms, neuropsychological testing) to assess EF, which would serve as a complement to ratings measurement, represents an additional limitation. Finally, we did not assess premorbid EF, and children with preexisting EF deficits may have been more susceptible to the impact of traumatic stress and in turn more likely to develop symptoms of PTSD and externalizing problems.

Future studies could expand upon findings from the current study by examining the association between trauma exposure, EF, and additional risk and resiliency outcomes, using longitudinal methodologies to more conclusively examine mediation.

Examination of the impact of caregiver and noncaregiver trauma on performance-based paradigms of EF would further expand understanding of how trauma impacts EF more broadly. Including measurement and analysis of trauma characteristics (i.e., age of onset, duration of exposure) and constellations of trauma exposure (i.e., co-occurrence of trauma types) on EF would enhance understanding of the importance of developmental timing and chronicity of exposure in these associations. In particular, examination of the trajectories of interaction between developmental competencies and mental health problems over time would be especially informative for intervention targeting.

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