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Research Article

The Effects of Childhood Exposure to Violence on Neurocognitive Performance in Adult Offenders with Pediatric Brain Injury: A Comparative Study Using Hierarchical Linear Modeling

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ABSTRACT

Introduction: Research has illustrated the long–term risks associated with pediatric traumatic brain injury (TBI) and exposure to violence in childhood, including cognitive dysfunction in the areas of executive function and memory. Many individuals in the criminal justice system present with histories of pediatric TBI and childhood violence exposure.

Objective: The present study investigated differences in neurocognitive performance between justice–involved individuals with a reported history of pediatric TBI who were exposed to violence during childhood and justice-involved individuals with a reported history of pediatric TBI who were not exposed to violence during childhood. This study's aim was to further explore the hazards of early childhood events on cognitive functioning in adulthood.

Method: The study used retrospective Automated Neuropsychological Assessment Metrics data. The sample included persons who were on probation or incarcerated (n=280) with a history of reported TBI sustained before age 15. Hierarchical linear modeling was used to examine the relationship between childhood violence exposure and cognitive performance on measures of executive function and memory.

Results: Results indicated statistically significant associations between exposure to violence in childhood and poor memory functioning among persons who had a history of pediatric brain injury. That is, individuals who were exposed to violence during childhood and who also sustained a TBI during that time performed worse on measures of memory function than individuals who sustained a TBI during childhood but were not exposed to violence.

Conclusion: These findings emphasize the importance of primary prevention efforts by highlighting the additive impact of childhood violence exposure and pediatric TBI on adult cognition in a vulnerable population. Secondary prevention efforts aimed at designing more supportive intervention and support programming after exposure to violence or pediatric brain injury may help minimize the risk for the worst of outcomes.

Keywords: Traumatic brain injury; Childhood violence; Cognitive flexibility; Brain development

INTRODUCTION

Childhood is understood to be a critical period for brain development for cognitive, emotional, and social aptitudes. Events taking place during this stage of growth are shown to impact functioning in adulthood so; there is a great deal of research on traumatic exposures that occur during childhood and adolescence [1]. Adverse Childhood Experience (ACE) is a collective term for exposures to traumatic or stressful events. This includes various forms of maltreatment, including abuse, household challenges, and deprivation or neglect [2]. More than half of the child population in the United States experience at least one ACE by the time they reach adulthood [3].

ACEs are identified as critical contributors to negative health outcomes throughout the lifespan, including delays and deficits in neurocognitive functioning. For example, one study reported young children with adverse experiences had a moderate to high risk of developmental delay; specifically, ACEs increased the risk of neurocognitive delays by 17% [1]. Further, ACEs have been found

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to inhibit brain development in children and increase the risk of poor cognitive outcomes into adulthood [4].

Childhood violence exposure

Childhood violence is defined as a personal exposure to physical acts of intentional harm occurring in the first 18 years of life [5]. An estimated three in five children are exposed to violence each year in the United States [6]. Exposure to violence in childhood has detrimental consequences for brain development that are observed in both the short and long term [7,8].

In the short term, exposure to violence in childhood has been shown to negatively impact cognitive functioning through adolescence [5]. Although deficits are observed in many areas of functioning, executive functions appear particularly vulnerable to the effects of violence exposure during periods of brain development [9]. Executive functions refer to a set of processes involving cognitive flexibility, organization, planning, initiation, and inhibitory control [10]. These complex mental skills are incredibly important to complete daily living tasks, cognitive duties, and social functioning. Therefore, deficits in executive functioning have widespread consequences on an individual's life, interrupting abilities in attention, processing, emotional management, reflection, and self-regulation [11]. Self-regulation, or inhibitory control, involves the ability to actively inhibit or override behavioral responses. This process allows an individual to stop and think before acting impulsively [12]. The vulnerability of executive functions to adverse events is demonstrated in how quickly these deficits occur. For example, one study observed impairments in various executive function abilities among preschoolers one week after the violence exposure [13].

Exposure to childhood violence can also have long–lasting effects on cognition, often observed for decades after the initial exposure [14]. Longitudinal studies have reported persistent impairments in executive function among adults who were exposed to violence in childhood [15]. For example, one study examined the cognitive function of adults who had been exposed to violence during childhood. The researchers suggested exposure to childhood violence predicted poorer executive functioning in adulthood [15]. A more recent study using a computerized assessment battery, ANAM, to examine differences in executive function between adults who had been exposed to violence in childhood, and controls, reported poorer executive function performance in the childhood violence group [16]. These results indicated specific impairments on measures of cognitive flexibility and inhibitory control. Inhibitory control was measured using a Go/No-Go test, a task that assesses monitoring and impulsivity. Further, more violence exposures in childhood were significantly associated with worse performance on the Go/No-Go test [16]. The report of poor performance on measures of inhibitory control is replicated in other research. For example, results of another study reflected a correlation between childhood violence exposure and impaired performance on the Go/No-Go task [17].

Like executive functioning, childhood violence exposure also has a pronounced and long–term detrimental effect on memory [18]. Memory includes the ability to encode, store, retain, and recall information in the immediate, short, and long term [19]. Working memory is an ability that involves temporarily retaining small amounts of information to complete cognitive tasks, including learning, reasoning, and comprehension [19]. Persistent deficits in working memory functioning have been observed in studies of adults who were exposed to violence during childhood [7,16]. Longitudinal studies have reported poorer working memory for adults who were exposed to violence in childhood. For example, results from a 25 year longitudinal study of approximately 3,000 adults between the ages of 18-38 who were exposed to childhood violence reported persistent impairments in working, auditory, and verbal memory [20].

The cognitive effects of being exposed to violence in childhood can also limit opportunities in adulthood. Research has demonstrated exposure to violence in childhood puts adults at risk for poorer educational qualifications, unemployment, and lower earnings [21]. One of the worst outcomes of exposure to violence during childhood is involvement in the criminal justice system. Criminal behavior has been highlighted as a problematic outcome for individuals with childhood violence exposure [22]. Specifically, exposure to violence in childhood increases the likelihood of incarceration and involvement in the criminal justice system in adulthood. One study examined the association between exposure to violence in childhood and criminal behavior among adults and reported persons with a history of exposure to violence in childhood were more likely to exhibit externalizing behavior problems, including aggression and hostility as adults [23]. In that study of nearly 400 adults, results reflected significant associations between records of childhood violence and more lifetime arrests, convictions, and incarcerations Another study reported childhood maltreatment doubles the risk for criminal behavior in adulthood [24]. This study also suggested individuals who were exposed to violence as children were more likely to commit criminal offenses as adults than those who were not.

Pediatric traumatic brain injury

Another traumatic childhood experience that can result in short– and long–term cognitive consequences is traumatic brain injury [25]. TBI is defined as a form of nondegenerative acquired brain injury caused by the application of an external force. Each year in the United States, approximately half a million children are admitted to emergency rooms for TBI [26]. TBI is recognized as one of the leading causes of death and disability across the world [27]. The consequences and recovery trajectory of a TBI depend on the injury severity, type of injury, and areas of the brain affected [25]. TBIs are classified as either mild, moderate, or severe. Research has shown moderate and severe TBI are more likely to impart long-lasting cognitive consequences. For moderate and severe TBI, the typical sequelae of injury include short and long term impairments in executive functions and memory [28,29].

Specific disruptions in inhibitory control are characteristic of children with TBI [30]. For example, when compared to a non-injured control group, the pediatric TBI group exhibited substantial deficits in executive functioning [28]. Notably, problems related to self-regulation have been linked to broader difficulties with social and behavioral adjustment [31]. Children with TBI were shown to demonstrate difficulty inhibiting learned responses on an executive function measure, despite receiving feedback that their responses were incorrect [32]. Poor memory function is also commonly reported as a short–term consequence of pediatric TBI [33]. For example, when compared to non-injured children, children with moderate and severe TBI demonstrated deficits in information storage, retention, and retrieval for both immediate and delayed memory.

Research has suggested these deficits persist over time. A 2009 meta-analysis also reported significant and persistent neurocognitive impairments in executive function for years after severe pediatric TBI [28]. That body of research suggested a group of people injured as children not only fails to catch up to their peers but also appears to fall further behind over time [28]. One longitudinal study of individuals with pediatric TBI measured executive function, specifically inhibitory control and cognitive flexibility six years after injury [34]. Cognitive flexibility is a component of executive function involving intentionally switching behavioral responses depending on the situation or context. In that study, the TBI group demonstrated poorer inhibitory control than a comparison group without injury, reflecting the vulnerability of executive functions to long–term impairment after childhood injury [34].

Planning and organization, two components of executive function involving higher order thinking, are also shown to be impaired among adults with a history of brain injury in childhood. For example, two separate studies assessed cognitive functioning 6-10 years after injury among adults and found deficits in planning, organization, problem solving, and reasoning abilities as measured by a complex drawing task [35,36].

Memory deficits also persist over time after pediatric TBI. One study reported a decline in working memory across an eight-year evaluation period among adults who had sustained a brain injury during childhood [37].

Similar to the worst outcomes related to exposure to violence during childhood, one of the worst outcomes for pediatric TBI is involvement in the criminal justice system [38]. Current research has suggested a history of TBI is overrepresented in the criminal justice system (55%) relative to the general public [39]. There is a relationship between deficits in executive functioning and an increase in externalizing behaviors, including aggression and impulsivity. Externalizing behaviors are commonly reported to increase an individual's likelihood of becoming involved in the criminal justice system. Specifically, this relationship appears related to impairments in inhibition, planning, and decision making. One study of 12,000 people examined the risk of criminal involvement after brain injury [40]. Results suggested that, among individuals who sustained brain injuries in childhood, the risk of criminal offenses in adulthood was four times higher. A more recent study of 1,265 adults reported, at 25 years of age, individuals with a history of pediatric brain injury were more likely to have been arrested for both property and violent offenses [41]. The poor long–term outcomes for both individuals with histories of pediatric TBI and those exposed to violence during childhood warrant further investigation, especially in the context of criminal justice where the prevalence of that history is very high.

Childhood violence exposure and pediatric traumatic brain injury

Exposure to violence in childhood is the leading cause of TBI in early childhood and many children will experience both adverse events with very poor outcomes [42]. The co-occurrence of TBI and childhood violence has been relatively well researched. One study of 3,500 adults reported individuals who experienced three to four ACEs were at significantly greater odds of sustaining a TBI [43], and a more recent study reported the same relationship between adverse experiences and TBI [42].

Researchers have suggested the worst of ACEs, exposure to violence and TBI, both have an impact on cognitive functioning in adults [43]. Sometimes childhood violence is the cause of a TBI and there is a growing consensus that persons with violence-based ACEs are also more likely to have a history of brain injury [42]. Research has shown that executive function and memory are commonly impaired in adults with either childhood violence exposures or childhood TBI, thus suggesting these cognitive domains are especially vulnerable to adversity or insult. Further, the cognitive sequelae of violence exposure and TBI each appear to impart a risk for involvement in the criminal justice system. That is, the incidence of exposure to either childhood violence or childhood TBI is higher for individuals in the criminal justice system compared to the general population [44]. However, there is very little research on cognitive outcomes for persons who were exposed to both pediatric TBI and childhood violence.

The aim of this study was to investigate group differences in cognitive performance on tasks of executive function and memory between justice involved individuals with a reported history of pediatric TBI who were exposed to violence during childhood and those with a reported history of pediatric TBI who were not exposed to violence during childhood. This research is necessary to emphasize the importance of primary prevention efforts by quantifying the cumulative risks of childhood violence exposure and pediatric TBI on adult cognition in the most vulnerable population.

Through independent group analyses, group differences on measures of executive function and memory were investigated, specifically between justice-involved individuals with reported childhood violence exposure and reported history of pediatric TBI and justice involved individuals with a reported history of pediatric TBI and no reported childhood violence exposure. It was expected that executive function and memory subtest scores for participants who have a reported history of childhood TBI and were exposed to violence during childhood would be significantly lower than executive function and memory subtest scores for participants who have a reported history of childhood TBI who were not exposed to violence during childhood.

Therefore, it was hypothesized that differences in ANAM test scores would be lower for participants exposed to childhood violence than those who were not.

METHODOLOGY

This study used archival data from the TBI Implementation Grant database. This study was reviewed and approved by the University of Denver Institutional Review Board (674894-16). The TBI Implementation Grant database was developed as a research and program following collaboration between the University of Denver, The Colorado Department of Human Services Brain Injury Program, and multiple county jails and probation systems in the Front Range area of Colorado.

In the original study, a total of 4,002 adults were screened for reported TBI history. Of those screened, 1,818 reported significant TBI history. Individuals were included if they reported significant TBI history with a reported TBI before the age of 15 years, participated in a clinical interview and neuropsychological screen evaluation, were over 18 years of age, and consented to have their de-identified data used for research purposes between 2012 and 2020. The total sample consists of 425 individuals. Records with missing data on at least two Memory subtests or three Inhibition subtests or no childhood violence were removed (n=145) leaving a

total of 280 individuals (206 men and 74 women). Demographic information, including sex, race, age, years of education, reported youngest age of TBI, number of reported TBIs, mental and physical health, and childhood exposure to violence, is presented in Table 1.

A statistical power analysis was performed for HLM using the R code Shiny app at jakewestfall.org/two_factor_power/with an NCC design, 280 participants, and nine targets (β=.797; d=.68). Adequate sample size was indicated (Table 1).

Table 1: Violence exposure characteristics and demographics.

Variable	n	%
Sex		
Male	206	73.6
Female	74	26.4
Race/Ethnicity White	164	58.6
Hispanic	58	20.7
Black or African American	27	9.6
American Indian/Alaskan Native	8	2.9
Asian	2	0.7
Race/Ethnicity White	164	58.6
Hispanic	58	20.7
Black or African American	27	9.6
American Indian/Alaskan Native	8	2.9
Asian	2	0.7
Native Hawaiian or other Pacific Islander	1	0.4
More than one race	19	6.7
Unknown/Not Reported	1	0.4
Age, years 0-19	10	3.6
20-29	71	25.4
30-39	106	37.9
40-49	55	19.6
50-59	34	12.1
60-69	4	1.4
Education, years 12 years or less	174	62.1
13-15 years	83	29.6
16 or more years	23	8.2
Childhood victim of violence Yes	174	62.1
No	106	37.9
History of substance abuse Yes	261	93.2
No	19	6.8
Physical health diagnoses Yes	141	50.4
No	136	48.6
Unknown/Not reported	3	1.1
Mental health diagnoses Yes	228	81.4
No	52	18.6
Youngest age of TBI, years 0-5	82	29.3
06-Oct	107	38.2
Nov-21	84	30
Unknown/Not reported	7	2.5
Number of TBIs 0-2	60	21.4
03-May	136	48.6
6+	74	26.4
Unknown/Not reported	10	3.6
0,3	0,3	0,3

Measures

Unstructured clinical interviews were conducted to obtain pertinent information regarding participant's history, including age, years of education, history of criminality, and mental and physical health. Participants were also asked if they had been exposed to violence in childhood during the clinical interview [44]. A modified version of the Ohio State University Traumatic Brain Injury Identification Method [45] was administered to identify reported brain injury history. The OSU TBI-ID is a structured interview designed to elicit the report of lifetime history of TBI. This measure was developed to offer a brief and retrospective tool for the identification of TBI history in settings that do not allow for full neuropsychological evaluation or medical record review. The OSU TBI-ID has been widely implemented in clinical and criminal justice settings. Bogner

and Corrigan found the reliability of the OSU TBI-ID has been demonstrated by both inter-rater and test/re-test reliability, with reliability scores ranging from acceptable to high (>.60). For this study, the scoring was modified to reduce the risk of false positives and includes First, Worst, and Multiple TBI [46].

During the interview, persons are asked a series of questions related to injury of the head and neck. If the individual describes an injury to the head or neck, the interviewer asks additional questions to elicit specific information related to age, mechanism of injury, and immediate sequelae. Individuals who report a history of TBI with loss of consciousness occurring before the age of 15 meet the criteria for First.

Individuals who report a TBI with loss of consciousness greater than 30 minutes meet the criteria for Worst. The criteria for Multiple TBI includes three or more injuries with either altered consciousness (e.g., dazed or memory gaps) or loss of consciousness, or two or more TBIs with loss of consciousness within a three–month period [46].

The ANAM Version 4 is an automated, computerized neurocognitive measure that assesses gross cognitive functioning [47]. Subtests include Code Substitution, Matching to Sample, Mathematical Processing, Procedural Reaction Time, Simple Reaction Time, Code Substitution Delayed, and Simple Reaction Time Repeated. Higher scores indicate higher levels of cognitive functioning. To measure executive function on the ANAM, the Go/No-Go subtest was used [48]. This task requires the participant to respond as quickly as possible to a stimulus and inhibit their responses when presented with another stimulus. To measure memory, two subtests were used: Code Substitution and Matching to Sample [49,50]. Code Substitution has three components: learning, immediate, and delayed. The learning and immediate components evaluated visual scanning, visual perception, attention, associative learning, and information processing speed. The participant pushes a button to indicate if the digit-symbol pair is correct or incorrect relative to the key. Code Substitution Delayed is administered after a delay and measures delayed visual recognition memory. The participant is asked to recognize digit-pair symbols presented in the learning trial. Matching to Sample is a visual-spatial processing, working memory, and visual short term recognition memory task. Participants are asked to remember a pattern that is presented and correctly identify the matching pattern.

Procedure

In the original study, participants were administered the OSU TBI-ID by a trained criminal justice professional. Persons with a reported history of TBI completed a neuropsychological screening battery that included an unstructured clinical interview and a cognitive screening battery [51]. Specific subtests from the ANAM that measure executive function and memory were selected for this study to assess performance. Memory function was measured using the Code Substitution subtest, and this study used the learning throughput score, working memory throughput score, and delayed memory throughput score as variables of interest for this study, and Matching to Sample subtest (spatial working memory throughput score). Executive function is measured using scores from the Go/No-Go subtest, a task of inhibitory control. The Go/No-Go subtest does not yield a composite score, and therefore mean response time for correct responses (standard score), percent correct (standard score), number of hits (standard score), number of omission errors (standard score), and the number of commission errors (standard score) were used as composite scores as has been reported in previous studies.

Study data were collected and managed using the Research Electronic Data Capture (REDCap) electronic data capture tools hosted at the University of Denver. REDCap is a secure, web-based application designed to support data capture for research studies, providing (a) an intuitive interface for validated data entry, (b) audit trails for tracking data manipulation and export procedures, (c) automated export procedures for seamless data downloads to standard statistical packages, and (d) procedures for importing data from external sources [52]. Data were organized in the REDCap Clinical Registry and exported to a Microsoft Excel spreadsheet to be entered into IBM SPSS (Version 26.0) for data analyses.

Statistical analyses

All data were converted to z scores for the ANAM subtests (Go/No-Go, Code Substitution Delayed, and Matching to Sample). Multiple linear regression models with simultaneous predictor entry for the ANAM subtest scores were performed using childhood violence as the predictor. Regression-derived collinearity diagnostics were run to ensure the relative performance of each predictor contributed to reliable executive function and memory assessment, and precise values were obtained. Variable Inflation Factors (VIF) for each predictor were used to evaluate multicollinearity. VIF >4 indicated significant multicollinearity, whereas serious multicollinearity was designated by VIF close to 10. To assess normality for the multilevel regression, plots of standardized residuals against observed scores were conducted. Results indicated violations of univariate normality for predictor and outcome variables. Therefore, a sequence of Kendall's tau-b tests was used to assess differences in subtests between participant ANAM scores and childhood violence. An a priori significance level of α=.05 was declared.

Previous research on childhood TBI and exposure to violence during childhood highlights the following confounding variables: age, gender, race, ethnicity, mental and physical health, educational attainment, length of incarceration, history of substance abuse, self-reported mental health diagnoses, socioeconomic status, TBI severity, youngest age of TBI, number of TBIs, handedness, family history, and trauma history [53-56]. The following covariates were used in this study: Gender, race, ethnicity, and self-reported substance abuse history, mental health history, and physical health history. All covariates were dichotomized except for age and years of education. No confounding effects were identified for any covariate following evaluation of covariate influence.

Hierarchical Linear Models. The use of Hierarchical Linear Models (HLMs), (also known as multilevel modeling, linear mixed models, or linear mixed-effect models) to investigate relationships among variables pertaining to both individuals and groups are being used in psychological research with increasing frequency [57].

Psychological experimental data analysis traditionally seeks generalizability from methods relying on the assumption of one random factor (typically participants); however, two or more random factors are typically involved (participants and stimulus, i.e., persons, images, words). This could potentially lead to serious testing effects bias with the use of conventional analytics [58]. By modeling interdependence in the data specifically, these mixed effects models avoid complications related to within-participant or within-stimulus mean scores analysis [59].

HLM is valuable because it explicitly allows for the estimation of

multiple sources of error variation developing from multiple random factors. This permits the inclusion of individuals with incomplete data, and it considers multiple assessments simultaneously, thereby accounting for multiple components of variance at once within a single model [60]. Additionally, data can be extended to nonlinear models and can be organized in nested levels.

Smith and Schatz used HLM to test the extent to which the Processing Speed score on the Executive Abilities: Methods and Instruments for Neurobehavioral Evaluation Research (EXAMINER) or the EXAMINER Cognitive Control Composite score functioned as mediators of the deficit in working memory found in the SCD group. They conducted two separate MLM analyses to determine the strength of the relationship between the independent variable (SCD vs. non-SCD group membership) and each mediator (EXAMINER Processing Speed and Cognitive Control) followed by two separate hierarchical linear regression analyses to determine the extent to which group differences in the mediator accounted for variance in working memory.

Hawks et al. also used HLM to assess the effects of age and group on word production (# words generated) across multiple timepoints that were nested within individuals to account for longitudinal dependencies in the data. This approach has the advantage of modeling linearity (age) separately for each individual (i.e., as random effects). Then moderation was examined for moderation by group. HLM uses the entire sample to inform prediction at a given age, unlike univariate analyses. As such, the wide age range of the study sample was a major strength, providing information about cognitive development across childhood without sacrificing statistical power. They then used the same approach to evaluate the relationship between verbal fluency performance, phenylalanine (Phe) control, and age in children with Phenylketonuria (PKU).

HLM was used to examine the nesting of participants within violence exposure comparison groups, while accurately accounting for variance. This design allowed for the investigation of the relationships among the variables of interest pertaining to both individuals and groups. The two random factors (participants and ANAM scores) and one fixed factor (childhood violence) design allowed for dependency of executive function and memory within childhood violence, and to examine the extent of between-childhood violence variation in ANAM scores [61]. The outcome variables were memory composite scores and executive function MeanRT. The ANAM MeanRT is an executive function composite score and was converted to z scores [51]. To maintain consistency and continuity, memory subtest raw scores were averaged to create memory composite scores and then were converted to z scores.

Covariates were controlled to measure latent influence of preexisting participant attributes and to isolate the relationship between the outcome variables. These data have a hierarchical structure as youngest age of TBI is nested within participants (Level 1) and participants are nested in childhood violence exposure (Level 2).

A series of nonlinear two-level models were run to examine ANAM subtest and composite memory scores and childhood violence groups separately using HLM8 (α=.05). First, to ensure a significant proportion of variance attributed to differences between and within participants, a null model was specified using only memory subtests and composite scores to ensure a significant proportion of variance attributed to differences between and within participants. Next, a second null model was specified using memory composite scores, gender, race, ethnicity, substance abuse history, mental health diagnosis, and physical health diagnosis to confirm prior covariate results.

Results revealed a significant effect for physical illness (t_{262}=-2.70, p<.01); therefore, it was included as a Level 1 explanatory variable.

At Level 1, the youngest age of TBI variable was entered as a grand-mean centered predictor of within-participant variability in memory controlling for physical illness (grand-mean centered), allowing for the examination of the effect of CV on TBI participant's memory (Hox, 2010). Reported youngest age of TBI data was sorted into three balanced groups and coded as nominal-level data (Age 0-5=1, Age 6-1=2, and Age 11-21=3). CV, substance abuse history, mental health diagnosis, physical health diagnosis, and gender were dummy coded (yes=1, no=0; male=0, female=1). At Level 2, CV was entered as an uncentered predictor of variability in participants' scores because the primary purpose of this study investigated CV differences in memory, and to evaluate whether CV uniquely influenced memory.

This process was then repeated using executive function MeanRT scores. In support of the multiple regression results, HLM null model results indicated no significant effect for any covariates, thus no covariates were included in the executive function HLM.

RESULTS

Descriptive statistics

Table 2 provides descriptive statistics of childhood violence exposure and the ANAM memory and executive function subtest data (n=280). These subtests yielded a throughput score in which higher scores indicated better performance (Table 2).

Table 2: Descriptive statistics for study variables by childhood violence exposure.

Variables	Childhood violence exposure					
	Yesa		Nob		Overall	
	M	SD	M	SD	M	SD
Youngest age TBI	8.25	3.92	8.57	3.81	8.37	3.87
Memory mean composite	60.62	10.37	56.2	10.65	57.87	10.75
Code substitution learning	92.75	15.46	85.45	16.66	88.21	16.57
Code substitution working memory	87.7	12.37	83.83	13.27	85.29	13.05
Code substitution delayed memory	33.06	14.55	29.83	14.45	31.05	14.55
Matching to sample spatial working	28.96	11.61	25.68	11.01	26.93	11.33
Go/No-Go Mean RT	88.69	20.55	84.29	22.93	85.96	22.12
Go/No-Go percent correct	90.84	23.67	92.87	18.27	92.1	20.47

Go/No-Go hits	86.44	28.26	89.02	22.76	88.05	24.97
Go/No-Go omissions	84.99	32.18	86.45	25.88	85.9	28.39
Go/No-Go commissions	98.87	14.99	98.33	14.36	98.54	14.58

Note: [a] Yes (n=174); [b] No (n=106); Overall (n=280).

Table 3 provides mean response time (Mean RT) score classifications for performance on the ANAM Go/No-Go subtest. Results showed individuals who were exposed to violence during childhood and individuals who were not exposed to violence during childhood performed similarly on the Mean RT measure of the Go/No-Go subtest. Overall, nearly half of the participants in each group performed in the below average and clearly below ranges (Table 3).

Table 3: Score classification for performance on ANAM Go/NO Mean RT between groups.

Classification	Childhood violence exposure			
	Yesa		Nob	
	N	%	N	%
Average or above	90	52	57	54
Below average	44	25	26	25
Clearly below	40	23	23	22

Kendall's tau-b

Associations between childhood violence exposure, youngest age TBI, and ANAM memory (Code Substitution; Match-to-Sample) and executive function (Go/No Go) results can be found in Tables 4 and 5, respectively. The following effect size cutoffs were used: |τb|=0.07 indicates a weak association |τb|=0.21 indicates a medium association, |τb|=0.35 indicates a strong association.

All memory subtests were significantly associated with childhood violence, with estimates ranging from -.11 (p=.026) to -.19 (p=.000). This indicated medium to weak, negative associations between childhood violence exposure and performance on measures of memory functioning, including Code Substitution Learning (τb=-.19; p=.000), Code Substitution Working Memory (τb=-.13; p=.008), Code Substitution Delayed (τb=-.11; p=.026) and Matching to Sample (τb=-.13; p=.012). These negative associations suggest a relationship between childhood violence and poor memory function (Table 4).

Table 4: Associations between childhood violence exposure, youngest age TBI, and ANAM memory scores.

Measure\	CV[a]	TB[b]	MnMEM[c]	LRN[d]	WM[d]	DM[d]	SWM[e]
Childhood violence exposure	1						
Youngest age TBI	-.04 ns	1					
Mean memory composite	-.19**	.06 ns	1				
Code substitution learning	-.19**	.07 ns	.66**	1			
Code substitution working memory	-.13**	.02 ns	.50**	.29**	1		
Code substitution delayed memory	-.11*	.05 ns	.62**	.48**	.23**	1	
Match to sample spatial working memory	-.13*	.05 ns	.53**	.31**	.29**	.33**	1

[a] Childhood violence.
[b] Youngest age TBI.
[c] Mean memory composite.
[d] Code Substitution abbreviations: LRN=Learning; WM=Working Memory; DM=Delayed Memory.
[e] Match to Sample abbreviation: SWM=Spatial Working Memory.
Note: Significant differences between variables using Kendall's tau-b. *p<.05 (two-tailed). **p<.01 (two-tailed).

Kendall's tau-b estimates indicated no statistically significant associations between childhood violence estimates and measures of executive functioning or youngest age TBI and executive function (Table 5).

Table 5: Associations between childhood violence exposure, youngest age TBI, and ANAM executive function scores.

Measure	CV[a]	TBI[b]	MRT[c]	PC[c]	H[c]	O[c]	C[c]
Childhood violence exposure	1						
Youngest age TBI	-.04 ns	1					
Go/No-Go mean RT	-.07 ns	-.03 ns	1				
Go/No-Go percent correct	-.01 ns	-.02 ns	0.05	1			
Go/No-Go hits	-.02 ns	.02 ns	.22**	.60**	1		
Go/No-Go omissions	-.04 ns	.02 ns	.32**	.48**	.70**	1	
Go/No-Go commissions	-.01 ns	-.03 ns	-.14**	.64**	.25**	.14**	1

[a] Childhood violence
[b] Youngest age TBI
[c] Go/No-Go abbreviations: MRT=Mean RT; PC=Percent Correct; H=Hits; O=Omissions; C=Commissions.
Note: Significant differences between variables using Kendall's tau-b. *p<.05 (two-tailed). **p<.01 (two-tailed).

Table 6 provides results of the hierarchical linear regression models. The Level 1 model results for memory indicated significant baseline memory scores for learning and memory composite scores across all participants. Physical health diagnosis was negatively associated with working memory, delayed memory, and memory composite scores, meaning a physical health diagnosis would predict a 0.33 to 0.34 unit decrease in memory function in these domains. Associations between youngest age TBI and all memory scores were nonsignificant.

The Level 2 model was used to evaluate whether childhood exposure to violence uniquely influenced memory function. Results revealed childhood violence exposure was inversely related with performance on measures of composite memory functioning and learning specifically. Notably, results revealed no significant differences in scores for working memory, delayed memory, and spatial working memory.

Remarkably, no statistically significant results were indicated for

any executive function measure across participants regardless of childhood violence exposure or youngest age TBI (Table 6).

Table 6: HLM results for childhood violence exposure, youngest age TBI, and ANAM scores.

ANAM code substitution and match to sample memory results					
Characteristic	**Learning**	**Working memory**	**Delayed memory**	**Spatial working memory**	**Memory composite score**
Intercept (Baseline) γ_{00}	0.28 (.09)**	0.13 (.09) ns	0.16 (.10)ns	0.17 (.10)ns	0.24 (.09)**
Childhood violence γ_{01} youngest age TBI γ_{10}	-0.45 (.12)***	-0.22 (.12) ns	-0.24 (.12) ns	-0.27 (.12)ns	-0.39 (.12)**
Intercept (Baseline) γ_{00}	0.10 (.08) ns	-0.01 (.08) ns	0.11 (.08) ns	0.08 (.07)ns	0.09 (.07) ns
Physical health diagnosis γ_{20}	-0.18 (.12) ns	-0.34 (.12)**	-0.34 (.12)**	-0.18 (.12)ns	-0.33 (.12)**
Deviance	755.78	756.35	760.28	756.97	748.37
ANAM Go/No-Go executive function results					
Characteristic	**Percent correct**	**Hits**	**Omissions**	**Commissions**	**Mean RT**
	Coefficient (SE)	Coefficient (SE)	Coefficient (SE)	Coefficient (SE)	Coefficient (SE)
Intercept (Baseline)	-0.05 (.12) ns	-0.06 (.12) ns	-0.03 (.11) ns	-0.03 (.10)ns	0.10 (.09) ns
Childhood violence γ_{01}	0.10 (.13) ns	0.11 (.13)ns	0.05 (.13) ns	-0.03 (.12)ns	-0.17 (.12) ns
Youngest age TBI γ_{10}	-0.05 (.08) ns	0.03 (.08) ns	-0.01 (.08) ns	-0.08 (.08)ns	-0.07 (.07) ns
Deviance	777.06	770.25	778.7	773.09	768.53

Note: n=271 due to missing data for youngest age TBI.
SE=Standard Error.
*p<.05. **p<.01. ***p<.001. ns=non-significant.

DISCUSSION

Results from this study confirmed the hypothesis that performance on measures of memory function was lower among participants in criminal justice who were exposed to childhood violence relative to persons who sustained a TBI during childhood who were not also exposed to violence. Specifically, exposure to childhood violence was predictive of impaired performance on measures of learning, delayed memory, and spatial working memory. Among the memory subtests, individuals exposed to violence during childhood demonstrated the poorest performance on the learning memory task, which involved visual scanning, processing speed, and attention, relative to persons who sustained a TBI during childhood and were not exposed to violence. Overall, these findings highlight persistent and ongoing neurocognitive complaints among justice ninvolved individuals with two ACEs: exposure to violence and TBI. In this way, these results add to a growing body of literature on the lasting memory consequences of ACEs, and this study extends that inquiry into criminal justice.

Interestingly, individuals with pediatric TBI who were exposed to violence during childhood did not perform worse on measures on executive function relative to persons who sustained a TBI during childhood who were not also exposed to violence. That is, these individuals did not demonstrate poorer performance on measures of reaction time, response inhibition, and impulsivity when compared to the group of individuals who sustained a TBI in childhood who were not exposed to violence, and that hypothesis was not supported. This is a departure from previous literature suggesting long term deficits in executive function after exposure to violence in childhood and after exposure to pediatric TBI.

Previous studies reported executive function deficits among adults who were exposed to violence in childhood or who sustained a TBI during childhood. For example, a 2009 meta-analysis reported significant deficits in executive function for years following brain injuries sustained during childhood [28]. Research also has reported a dose response relationship between childhood violence and executive function deficits. Individuals who reported a higher number of adverse experiences in childhood had poorer performance on measures of executive function. In one study, a higher number of ACEs was significantly associated with poorer performance on the ANAM Go/No-Go task used in this study [16].

In the current study, there were no significant differences in executive function between individuals who were exposed to violence during childhood and who also sustained a TBI in childhood and individuals who sustained a TBI during childhood who were not exposed to violence during that time. It is possible the association was not apparent because both groups were categorized by executive dysfunction, one no more than the other. Results suggest both groups were characterized by executive dysfunction (i.e., below average and clearly below average scores). Specifically, more than half of the individuals in both groups scored in either the below average or clearly below average ranges on mean response time for the ANAM Go/No-Go task, which is consistent with the literature on TBI and childhood violence. Another possibility may be attributed to a moderation effect between memory and executive function which was a limitation of this study.

As reported in previous research, this study included the following confounding variables: age, gender, race, ethnicity, and self-reported substance abuse history, mental health history, and physical health history. Physical illness, most commonly chronic pain, hypertension, and headaches were negatively associated with working memory, delayed memory, and memory composite scores. This finding contributes to a growing body of literature suggesting a reciprocal relationship between memory and physical health, notably declines in physical health, are associated with lower memory function [62].

Results of this study did not reflect an association between an earlier age of TBI and poorer memory function. That is, individuals who sustained a TBI earlier in childhood did not appear to perform worse on measures of memory functioning when compared to individuals who sustained a TBI at a later stage of childhood. This is less consistent with existing literature that reports a greater cognitive impact after TBI among individuals who sustained a TBI at a younger age [63].

There were also no significant associations between performance on measures of executive function and any demographic data, including childhood violence exposure, history of TBI, youngest age of TBI, or physical illness. This result is a departure from previous studies that have identified executive functioning as a cognitive domain vulnerable to ACEs, including exposure to violence and TBI.

Implications

The current study contributes to existing research by emphasizing the role of childhood experiences on adult functioning and extending this body of work to include justice– involved individuals. This

study evaluated persons who were on probation or in jail, a group of people more likely to have both a history of TBI and to have been exposed to childhood violence. These findings highlight the vulnerability of this population and the need for trauma–informed treatments modified to accommodate cognitive deficits.

Trauma–informed treatments prioritize the development of therapeutic rapport to improve emotional regulation [64-66]. When working with this particular population, the use of compensatory memory strategies to moderate the effects of cognitive complaints is advised. In this study, justice–involved individuals who experienced two adverse events during childhood demonstrated poorer memory function, so this suggestion includes encouraging the use of external memory aids like calendars and notebooks and inviting clients to record important information during meetings [67].

These results also highlight the importance of prevention efforts. Secondary prevention includes efforts aimed at tempering the cognitive consequences of ACEs. Secondary prevention programming, in this case, includes interventions focused on improving cognitive outcomes for children after exposure to violence or brain injury. That includes advocacy for educational needs and linkage with statewide resources to optimize recovery [68]. In the school system, return-to-learn models support a child's needs after a brain injury by making accommodations and decreasing the overall workload to prioritize recovery [69].

Special education services, including Individualized Pducation Plans (IEP) and Section 504 plans, ensure individualized accommodations are delivered to promote successful outcomes, including higher levels of educational attainment. For children exposed to violence, research supports modifying parenting approaches yields positive outcomes for families [70]. For example, the Breaking the Cycle campaign, which promotes appropriate discipline strategies, changed parenting attitudes and reduced violence in families with histories of abuse [71].

Research to outline the long–term consequences of adverse childhood experiences, including potential risks of criminal justice involvement, is imperative. Primary prevention in childhood focuses on preventing children from being exposed to violence or sustaining brain injuries by minimizing the risks for those experiences.

Those risk factors include high levels of familial conflicts, inadequate supervision, poor parent child relationships, contact sports, and other risky behaviors [72-74]. Public health education and laws focused on safety, including the use of seatbelts, car seats, and helmets, have yielded significant reductions in the incidence of childhood TBI [75]. For example, prevention programs for childhood brain injury that incorporate awareness, educational, and policy changes, like WalkSafe and BikeSafe, led to a 78% decrease in pediatric pedestrian motor-vehicle accidents and a 30% decrease in bicycle accidents [76].

Importantly, given exposure to violence in childhood is the leading cause of TBI in early childhood, programs that improve parent-child relationships have the potential to decrease the incidence of both violence and TBI [73]. One parenting program, Adults and Children Together against violence: Parents Raising Safe Kids (ACT), has been associated with reductions in physical and emotional abuse and an increase in nurturing behavior [77]. This program focuses on teaching positive parenting skills, developing the parent-child relationship, and recognizing hazards in the home environment to reduce risk factors for abuse and injury [71].

LIMITATIONS

Despite results reflecting poorer memory performance among justice–involved individuals exposed to both violence and brain injury during childhood, this study did not confirm the hypothesis that individuals who experienced two ACEs would perform worse on measures of executive function. In this case, executive function deficits were observed among both groups, with scores in the below and clearly below range, which may have obscured score differences [78-84].

With respect to methodology, there are obvious limitations to self–report data. In this study, information about personal history of violence exposure and TBI was collected from structured and clinical interviews. The accuracy of self-report can be jeopardized by poor memory and this population of justice involved persons who reported exposure to violence in childhood and childhood TBI is characterized by memory impairment. Some participants may also have been reluctant to report their history of childhood violence [85-90].

The current study did not control for some of the confounding variables identified in previous research, including premorbid cognitive functioning and socioeconomic status. Future research should address the contribution made by these variables to cognitive function after adverse experiences. Specifically, future studies should control for additional variables that are associated with childhood violence exposure and may correlate with factors that impact cognitive development, including socioeconomic status and exposure to toxins. Finally, the generalizability of this study is limited to persons in criminal justice. In this study, all participants were under supervision in a mountain state, so there is no way to know whether these results apply to persons in other parts of the country [91-93].

CONCLUSION

Overall, these results add to a growing body of research suggesting justice involved individuals exposed to both violence and brain injury in childhood demonstrate the poorest cognitive function; here they exhibited poorer memory performance. The findings also highlight the importance of implementing trauma informed treatment that accommodates memory deficits in forensic settings. Finally, the current study emphasized the importance of primary and secondary prevention efforts for two harmful ACEs exposure to violence and brain injury to allay the potential for these poor long term criminal justice outcomes. Increased awareness, education, and programming aimed at improving cognitive development in childhood can promote the development of protective factors and minimize psychosocial risk factors.

FUTURE RESEARCH

Moving forward, TBI should be defined more clearly as an ACE to integrate the bodies of literature on TBI and exposure to violence during childhood. Future studies should consider the impact of these two adverse events on long–term cognitive outcomes to encourage the development of appropriate screening and customized treatment protocols. Future research should examine whether childhood violence exposure makes people more vulnerable to adverse outcomes after TBI and whether TBI makes people more vulnerable to adverse outcomes after violence

exposure during childhood.

Research on the degree to which these two adverse events are predictive of incarceration risk is also warranted. Additionally, further examination of the potential moderation of memory on executive function is indicated (interaction effect), particularly for participants who perform in the below average and clearly below classifications for the ANAM Go/No-Go Mean RT subtest.

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DECLARATION OF INTERESTS

The authors report no conflict of interest.

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