



Is developmental timing of trauma exposure associated with depressive and post-traumatic stress disorder symptoms in adulthood?



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ABSTRACT

Background: Trauma exposure is a known risk factor for psychopathology. However, the impact of the developmental timing of exposure remains unclear. This study examined the effect of age at first trauma exposure on levels of adult depressive and posttraumatic stress disorder (PTSD) symptoms.

Methods: Lifetime trauma exposure (including age at first exposure and frequency), current psychiatric symptoms, and sociodemographic information were collected during interviews with adults participating in a study at a public urban hospital in Atlanta, GA. Multiple linear regression models assessed the association between timing of first trauma exposure, classified as early childhood (ages 0–5), middle childhood (ages 6–10), adolescence (ages 11–18), and adulthood (ages 19+), on adult psychopathology in 2892 individuals.

Results: Participants exposed to trauma (i.e., child maltreatment, other interpersonal violence, non-interpersonal violence, and other events) at any age had higher depressive and PTSD symptoms compared to their unexposed peers. However, participants first exposed to child maltreatment during early childhood had depression and PTSD symptoms that were about twice as high as those exposed during later developmental stages. This association was detected even after controlling for sociodemographic characteristics, exposure to other trauma types, and frequency of exposure. Participants first exposed during middle childhood to other interpersonal violence also had depressive symptoms scores that were about twice as high as those first exposed during adulthood.

Conclusions: Trauma exposure at different ages may differentially impact depressive and PTSD symptoms in adulthood. More detailed examination of timing of trauma exposure is warranted to aid in identifying sensitive periods in development.

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1. Introduction

Exposure to traumatic life events, including child maltreatment or natural disasters, is increasingly recognized as one of the major social determinants of psychiatric disorders. Trauma exposure has

been shown to about double the risk for major depressive disorder (McLaughlin et al., 2010a; Norman et al., 2012; Widom et al., 2007), which is currently estimated to affect 11.7% of adolescents (Merikangas et al., 2011) and 16.6% of adults (Kessler et al., 2005). Trauma exposure (or more precisely, exposure to events involving

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perceived or threatened loss of life, serious injury, or loss of physical integrity), is also a requirement to meet DSM-IV criteria for post-traumatic stress disorder (PTSD) (American Psychiatric Association, 2013), which has a lifetime prevalence of 4.7% among adolescents (McLaughlin et al., 2013) and 7.8% among adults (Kessler et al., 1995). As trauma exposure is common in the population, with six out of every 10 children (McLaughlin et al., 2013) and one out of two adults in the United States reporting a lifetime trauma exposure (Kessler et al., 1995), greater insight into the role of trauma exposure on both depression and PTSD etiology is needed.

One understudied facet of trauma exposure that may be linked to future psychopathology risk is the developmental period of trauma occurrence. Researchers studying child abuse and neglect, in particular, sometimes consider developmental timing as an important dimension of defining maltreatment, finding that age at onset to maltreatment may influence the etiology of mental health problems (Barnett et al., 1993; English et al., 2005). However, few attempts have been made in the broader trauma literature to examine ways in which age at onset to trauma exposure correlates with risk for psychopathology. As a result, we currently lack knowledge about the existence of “sensitive periods” (Knudsen, 2004; Bailey et al., 2001; Bornstein, 1989) for psychopathology risk, meaning windows of time in the course of development when the brain is especially “plastic” and therefore when trauma exposure substantially elevates risk for the onset of depression or PTSD. Determining whether, and when, sensitive periods exist across the lifespan will be important for understanding developmentally-relevant biological pathways implicated in the etiology of psychopathology and guiding the investment of limited public health resources to the “high-risk” stages when deleterious exposures are most harmful and the “high-reward” stages when enriching exposures and interventions could offer their greatest benefit.

Thus far, only a small number of studies have examined possible sensitive periods corresponding to risk for either depression or PTSD. Among these studies, no consensus has emerged regarding whether earlier or later exposure is predictive of elevated risk for either outcome. With respect to depression, three prospective studies observed that individuals with maltreatment prior to age 5 had higher levels of teacher-reported internalizing symptoms in early childhood (Keiley et al., 2001) and self-reported depressive symptoms in early (Thornberry et al., 2010) and early to mid-adulthood (Kaplow and Widom, 2007) compared to those who were either never exposed or exposed during later stages. Retrospective studies have also found earlier abuse - before age 5 (Dunn et al., 2013), before age 12 (Schoedl et al., 2010; Maercker et al., 2004; McCutcheon et al., 2009), or before age 17 (Chu et al., 2013) or trauma - between 4 and 6 (Rusby and Tasker, 2009) and before age 12 (McCutcheon et al., 2009; Maercker et al., 2004) particularly elevates risk for depressive symptoms and major depressive disorder. In two of these studies (McCutcheon et al., 2009; Maercker et al., 2004), early exposure to interpersonal trauma, such as witnessing trauma, physical attacks, and sexual molestation, conferred the largest harm relative to other traumas. However, prospective studies have also found exposure to maltreatment during adolescence (between 10 and 12 (Harpur et al., 2015) or 12–17 (Thornberry et al., 2001)) was more strongly associated with adolescent depressive symptoms than earlier maltreatment. A recent retrospective study also found emotional abuse specifically at age 14 was most predictive of depression during young adulthood (Khan et al., 2015). Three prospective studies (English et al., 2005; Jaffee and Maikovich-Fong, 2011; Oldehinkel et al., 2014) and two retrospective studies (Manly et al., 2001; Pietrek et al., 2013) found no effect of developmental timing of maltreatment in relation to internalizing

symptoms and adolescent or adult depression.

For PTSD symptoms or PTSD diagnoses, similarly mixed findings have been observed. For instance with respect to child abuse, retrospective studies have found that children with PTSD tended to report a lower age at first exposure (between 3 and 5 (Glod and Teicher, 1996)), and that sexual assault or physical abuse before age 11, but not childhood neglect, conferred the highest risk for PTSD (McCutcheon et al., 2010). Retrospective studies have also found older children (ages 13–18 (Schoedl et al., 2010)) had higher risk for PTSD relative to their peers exposed at other ages. One prospective study of childhood sexual abuse found no association between age at abuse onset and PTSD symptoms (Kaplow and Widom, 2007). Evidence regarding the effects of age at onset to natural disasters appears more consistent, with both retrospective (Demir et al., 2010) and prospective studies (Green et al., 1991; Copeland et al., 2007) observing higher levels of PTSD symptoms or PTSD diagnoses among older children (around age 7 and above) compared to younger children (those younger than about age 7). However, results for other traumas is less conclusive, with retrospective studies suggesting there are no differences (Mueller-Pfeiffer et al., 2013; Maercker et al., 2004) in risk for PTSD based on age at onset of trauma, that early-life trauma is more harmful (Ogle et al., 2013, 2015), or that middle childhood (ages 6–11) is more strongly associated with PTSD (Schrag et al., 2008).

Although these studies suggest the developmental timing of trauma exposure may be associated with subsequent risk for depression or PTSD, these studies are limited by a focus on a small subset of adversities, reliance on small clinical or convenience samples, and failure to account for the correlated nature of adversities (McLaughlin et al., 2012a). Moreover, relatively few have examined the time-dependent effects of specific trauma types. Instead, most prior studies have focused generally on “early life adversity,” meaning adversities occurring over a broad span of ages (typically birth to age 14). In addition, even fewer studies have accounted for the frequency of exposure to adversity, leaving open the possibility earlier trauma exposure may be confounded by the number of times exposed.

The current study aimed to address these limitations by investigating whether developmentally-sensitive measures of trauma exposure were associated with depressive and PTSD symptoms in a sample of highly-trauma exposed adults. We conducted these analyses using a low income, urban-sample of African American adults with both high rates of trauma exposure (>90% exposed to at least one traumatic event) and high rates of depressive and PTSD symptoms (Gillespie et al., 2009), which provided an opportunity to examine the differential effects of age at onset to trauma that would not be possible in samples where the prevalence of trauma was lower. More specifically, we examined the effect of timing of first exposure to trauma, coded as: *early childhood* (age 0–5 years), *middle childhood* (6–10 years), *adolescence* (11–18 years), and *adulthood* (19+ years), on self-reported depressive and PTSD symptoms in adulthood. Traumatic event types were separated into child maltreatment, other interpersonal trauma, non-interpersonal trauma, and other events in order to determine if the effects of developmental timing of exposure differed based on the type of trauma exposure.

2. Methods

2.1. Sample and procedures

Data came from the Grady Trauma Project (GTP), an ongoing National Institute of Mental Health (NIMH)-funded study examining genetic and environmental risk and resilience factors for the development of PTSD and other psychiatric disorders (Gillespie

et al., 2009; Binder et al., 2008; Bradley et al., 2008). The GTP sample consists of 8886 adults (ages 18–90) who were recruited from general medical clinics and obstetric/gynecological clinics at Grady Hospital in Atlanta, Georgia. The clinics are part of a publicly funded, non-profit healthcare center serving a primarily African American, urban population from low socioeconomic backgrounds. The benefits of this particular sample include identification of an understudied population, a group with a high rate of trauma, and a relatively homogeneous socioeconomic status distribution. Participants were recruited from clinic waiting areas. Eligible participants were at least 18 years old, not actively psychotic, and able to give formal written and verbal consent. Consenting individuals participated in verbal interviews administered by trained research assistants lasting approximately 45–75 min, depending on participant's trauma history and symptoms. Participants received \$15 for participation. Emory University's Institutional Review Board and the Grady Health Care System Research Oversight Committee approved all study procedures.

In the current analysis, we analyzed data from 2892 African American adults whose data were collected between 2005 and 2013 (74.6% female; mean age = 41.0, SD = 13.8). These adults had complete data on all measures relevant to the current analyses. We restricted analyses to African Americans, as significant differences were observed in the distribution of trauma exposure, covariates, and both outcomes by race/ethnicity; restriction to one racial/ethnic group eliminated the variability associated with race, allowing us to more effectively control for confounding. Adjustment or stratification by race was not possible as individuals from other racial/ethnic groups comprised only 7.7% of the sample (3.6% were White and 4.1% identified as other), resulting in low power to detect associations due to small cell counts. Of note, our analytic sample was smaller than the larger study sample due to the fact that participants completed study questionnaires in clinic waiting rooms until the clinic was ready to see them/their family member, thus the majority of participants did not complete all measures.

2.2. Measures

2.2.1. Exposure to trauma

Presence or absence of trauma exposure, age at first exposure to trauma, and trauma frequency were ascertained using the Traumatic Events Inventory (TEI), a 14-item screening measure assessing lifetime history of traumatic events (Gillespie et al., 2009; Schwartz et al., 2005, 2006). We focused on 11 different traumatic events, which had information about age at first onset and could plausibly occur in multiple developmental stages. These events were grouped into four types, consistent with prior research (Breslau et al., 1998; McLaughlin et al., 2012b): (1) *child maltreatment* (i.e., witnessing violence between parents or caregivers; being beaten; experiencing emotional abuse; or experiencing sexual abuse); (2) *other interpersonal violence* (i.e., witnessing or being confronted with a friend or family member being murdered; witnessing a family member or friend being attacked with or without a weapon; witnessing a non-family member or friend attacked with or without a weapon); (3) *non-interpersonal violence* (i.e., experiencing a natural disaster; witnessing or experiencing a serious accident or injury; experiencing a sudden life threatening illness); and (4) *other trauma* (i.e., any other event or experience not covered by the previously stated categories that participants self-identified as a traumatic experience, including witnessing a death or suicide, bereavement, divorce or familial disruption, extended caregiving, job loss, etc).

For each traumatic event, participants reported their age (in year) of their first experience. We used this data to develop age categories for age at first exposure: *early childhood* (age 0–5 years),

middle childhood (6–10 years), *adolescence* (11–18 years), and *adulthood* (19+ years). These age categories were chosen to match previous research and minimize recall bias (relative to studying specific years of age) (Kaplow and Widom, 2007; Dunn et al., 2013).

Participants also reported the frequency of each trauma event occurrence on a scale ranging from 0 (unexposed) to 8 (greater than 20 times). Using this data, we generated a set of indicator variables (one for each traumatic event) to denote low versus high frequency of trauma exposure, with high being at or above the top quartile of frequency for a specific event. Details regarding cut-points used for each trauma event are denoted in the footnote of Fig. 1. These frequency indicators were used in models examining timing of exposure to account for the possibility that people exposed at younger ages would be more likely have a higher number of occurrences of a given trauma exposure.

2.2.2. Depressive symptoms

Depressive symptoms were measured using the Beck Depression Inventory, Second Edition (BDI-II), a 21-item psychometrically validated and widely-used inventory of current depressive symptoms (Beck et al., 1996; Wang and Gorenstein, 2013; Arnau et al., 2001; Beck et al., 1988). The BDI contains items assessing the presence and severity of depressive symptoms over the past two weeks rated on a scale of 0 (not at all/never) to 3 (extremely/every day). Total BDI score were calculated by averaging all individual items (where at least 19 items were completed) and multiplying that mean by 21; this approach to deriving a summary score enabled us to incorporate data from individuals with small amounts of missing data. In this sample, the BDI demonstrated excellent internal consistency reliability ($\alpha = 0.93$).

2.2.3. Post-traumatic stress disorder symptoms

Post-traumatic stress disorder symptoms were captured using the modified Posttraumatic Symptom Scale (MPSS), a psychometrically validated self-report measure of the frequency and severity of PTSD symptoms (Coffey et al., 1998). The 17 items on the MPSS correspond to symptom criteria to diagnose PTSD as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV-TR) (Association. AP, 2000). These symptoms encompassed re-experiencing, avoidance, emotional numbing, as well as hyperarousal and reactivity categories. Participants were asked about these symptoms generally, and therefore the symptoms do not reference a specific index trauma but can reflect symptoms related to any of the traumas reported on the TEI. All items were scored on a scale of frequency from 0 (not at all) to 3 (5 or more times a week) as experienced by the individual in the past two weeks from their point of assessment. MPSS total scores were calculated by averaging all individual items (where at least 15 values were completed) and multiplying the average by 17. In this sample, the MPSS also showed excellent internal consistency reliability ($\alpha = 0.92$). Of note, the PTSD scale was not administered to individuals that did not report exposure to at least one type of trauma.

2.2.4. Covariates

All linear regression models adjusted for the following covariates: sex; age (continuous); highest level of education (less than 12th grade; high school graduate or GED; greater than high school graduate or GED/college graduate); household monthly income (\$0–499; \$500–999; \$1000+), and employment status (unemployed; unemployed receiving disability support; and employed, with or without disability support).



Fig. 1. Percent of respondents exposed to frequent trauma by age at first onset to trauma. Frequency of each trauma event occurrence was ascertained on a scale ranging from 0 (unexposed) to 8 (greater than 20 times). Using this data, we generated indicator variables for each traumatic event denoting low versus high frequency of trauma exposure, with high being at or above the top quartile of frequency for a specific event. High frequency was defined as greater than or equal to the following values for each individual trauma event: experiencing a natural disaster ≥ 2 ; witnessing or experiencing a serious accident or injury ≥ 2 ; and experiencing a sudden life threatening illness ≥ 2 ; witnessing or being confronted with a friend or family member being murdered ≥ 2 ; witnessing a family member or friend being attacked with or without a weapon ≥ 4 ; witnessing a non-family member or friend attacked with or without a weapon ≥ 5 ; witnessing violence between parents or caregivers ≥ 8 ; being beaten ≥ 8 ; experiencing emotional abuse ≥ 8 ; or experiencing sexual abuse ≥ 5 . For the trauma categories, the 75th percentile for reported frequency of exposure was taken for all events included in the category and high frequency was defined as greater than or equal to the following values for trauma categories: child maltreatment ≥ 7 , interpersonal trauma ≥ 3 , non-interpersonal trauma ≥ 2 ; and any other trauma not reported ≥ 2 . The figure presents the % of those exposed to frequent trauma within each age category among those exposed. All Chi Square Goodness of Fit models for each trauma event, which evaluated whether there were significant differences between the frequencies of exposure to each trauma by age at first exposure, are significant ($p < 0.0001$) unless indicated with NS ($p > 0.05$).

2.3. Data analyses

To facilitate interpretation and comparison, both outcomes were standardized (mean = 0; SD = 1) prior to analyses. After standardization, we conducted basic univariate and bivariate analyses to compare outcome values by each covariate. We also examined the distribution of exposure to each traumatic event in the total sample and by age at first exposure among those who were exposed. To determine whether respondents exposed at earlier ages also had more frequent trauma exposure, we examined the percent of respondents exposed to frequent trauma by reported age at first exposure. Following these analyses, we fit a series of linear regression models that examined, separately for each traumatic event and type, the association between trauma exposure and each outcome, after adjusting for covariates. Model 1 examined the effect of being exposed (vs. non-exposed) at any age to trauma. Model 2 (partial adjustment) examined the effect of timing of exposure (compared to the referent group of non-exposed during any period), after adjusting for covariates as well as exposure to all other traumatic events, given that some exposures, particularly child maltreatment events, were highly correlated (tetrachoric correlation values ranged from $r = 0.07$ to $r = 0.63$). Model 3 (full adjustment) expanded upon Model 2 by additionally including the indicator for frequency of each trauma event occurrence (0 = low frequency; 1 = high reported number of occurrences of that trauma exposure). For Models 2 and 3, we conducted a test of homogeneity to evaluate whether the beta coefficients (indicating the effect of timing of exposure relative to never exposed) were significantly

different from each other. In cases where the null hypothesis was rejected for the test for homogeneity ($p < 0.05$), indicating that the effects of developmental timing differed across groups, we then conducted post-hoc Tukey tests to evaluate, after adjustment for multiple testing, which specific age at exposure groups differed from one another. All analyses were conducted using SAS Version 9.4 (SAS Institute, Inc, Cary, North Carolina).

3. Results

The analytic sample comprised mostly women (74.6%) and middle-age adults (mean age = 41.0; SD = 13.8). Depressive and PTSD symptoms significantly differed by age, education level, household monthly income, and employment, but not sex (Table 1). Specifically, both depressive symptoms and PTSD symptoms were higher among middle-age adults, those with lower education and income, and those who were unemployed or on disability.

3.1. Distribution of trauma exposure and trauma timing

Nearly three-quarters of the sample reported being exposed to some type of interpersonal or non-interpersonal violence event (Table 2). Slightly more than half of the sample had been exposed to some type of child maltreatment, with violence between caregivers and sexual abuse being the two most common sub-types.

The mean age at first exposure to any trauma was 11.4 (SD = 8.8), but ranged between before one years of age through age 62. Overall, middle childhood (ages 6–10) was the most often

Table 1
Distribution of depressive and posttraumatic stress disorder symptoms in the Grady Trauma Project (GTP) analytic sample (N = 2892).

Covariate	Total sample	Depressive symptoms			PTSD symptoms		
	N (%)	Mean (SD)	F Value	p value	Mean (SD)	F Value	p value
Age							
18–25	579 (20.0)	11.78 (9.7)	21.17	<0.0001	10.23 (10.9)	23.45	<0.0001
26–35	527 (18.2)	14.59 (12.0)			12.39 (11.9)		
36–45	499 (17.3)	16.26 (13.3)			14.28 (13.6)		
46–55	817 (28.3)	16.24 (12.8)			14.60 (13.0)		
56+	470 (16.2)	11.63 (11.1)			9.00 (10.5)		
Sex							
Male	733 (25.4)	14.03 (12.4)	0.50	0.500	12.14 (12.7)	0.30	0.585
Female	2159 (74.6)	14.39 (12.0)			12.43 (12.2)		
Education							
Less than 12th grade	658 (22.7)	16.89 (13.4)	20.35	<0.0001	14.39 (13.2)	11.69	<0.0001
High school graduate or GED	1202 (41.6)	13.79 (11.8)			11.68 (12.0)		
Greater than GED/College graduate	1023 (35.7)	13.24 (11.3)			11.84 (12.1)		
Income							
\$0–499	879 (30.4)	16.88 (13.5)	36.18	<0.0001	13.83 (13.2)	14.29	<0.0001
\$500–999	779 (26.9)	14.40 (11.8)			12.83 (12.5)		
\$1000 or more	1234 (42.7)	12.40 (10.8)			11.01 (11.5)		
Employment Status							
Unemployed	1468 (50.8)	15.34 (12.7)	31.06	<0.0001	12.99 (12.7)	17.57	<0.0001
Unemployed (disability)	549 (19.0)	15.75 (12.5)			13.84 (13.0)		
Employed (with or without disability)	875 (30.2)	11.65 (10.2)			10.36 (11.0)		

Descriptive statistics are presented for the analytic sample. Linear regressions were performed for each outcome by covariate, with F-statistics and corresponding p-values listed. Analyses were performed on the non-standardized outcomes to facilitate interpretation.

reported time period for first exposure to child maltreatment (Table 2). In contrast, adolescence (ages 11–18) and adulthood (ages 19 and above) were the most often reported time periods for first exposure to other interpersonal violence and non-interpersonal violence, respectively.

For most traumas, there was also an age-frequency gradient, suggesting that those first exposed earlier in the lifespan also tended to report experiencing more frequent exposure (Fig. 1).

3.2. Trauma exposure and depressive and PTSD symptoms

As shown in Table 3, respondents reporting exposure to trauma at any age had higher depressive and PTSD symptoms, on average, relative to their non-exposed peers. For example, participants exposed to child maltreatment had, on average, depressive and PTSD symptoms scores that were half of a standard-deviation unit greater than their unexposed peers.

3.3. Timing of trauma exposure and depressive symptoms

Depressive symptoms varied as a function age at first exposure to child maltreatment and other interpersonal violence, but not non-interpersonal violence or any other trauma (Table 4). Specifically, participants first exposed to child maltreatment during early childhood ($\beta = 0.739$) had depressive symptoms scores that were about 1.5 times as high as those first exposed during middle childhood ($\beta = 0.519$) and almost twice as high as those first exposed during adolescence ($\beta = 0.397$), even after adjusting for sociodemographic covariates and exposure to other trauma types (Tukey p-value <0.05 for both comparisons). In other words, the predicted depressive symptom score for those first exposed during early childhood would be 0.470 (SD = 0.27), whereas the predicted depressive symptom score for those first exposed during middle childhood would be 0.245 (SD = 0.27). These differences persisted after additionally adjusting for frequency of trauma exposure

Table 2
Distribution of exposure to each traumatic event in total sample and by age at first exposure to trauma among those who were exposed.

Trauma exposure	Exposed		Age at first exposure (year)		Age at first exposure (category)							
	%	N	Mean	SD	Ages 0-5		Ages 6-10		Ages 11-18		Ages 19+	
					%	N	%	N	%	N	%	N
Child Maltreatment												
Violence between Caregivers	31.0	897	8.06	3.3	8.1	235	16.2	469	6.7	193	–	–
Physical Abuse	19.2	555	8.10	3.3	4.6	133	10.8	311	3.8	111	–	–
Emotional Abuse	25.9	750	9.91	3.8	3.9	112	11.2	325	10.8	313	–	–
Sexual Abuse	30.4	878	9.70	3.7	4.4	128	13.4	387	12.6	363	–	–
Any of the Above	56.9	1624	8.07	3.5	15.5	449	28.0	809	12.7	366	–	–
Other Interpersonal Violence												
Family/friend Murdered	53.9	1560	22.19	12.0	0.9	26	6.4	186	19.5	564	27.1	784
Witness Attack (Family or friend)	42.2	1220	17.37	10.3	2.7	77	8.7	252	16.5	476	14.4	415
Witness Attack (non-family/friend)	44.0	1271	18.99	10.6	1.4	40	7.0	201	18.9	547	16.7	483
Any of the Above	77.4	2238	17.52	10.5	4.3	123	16.5	476	31.1	900	25.6	739
Non-Interpersonal Violence												
Natural Disaster	25.7	744	19.68	13.4	1.2	35	6.9	200	7.3	210	10.3	299
Serious Accident or Injury	64.5	1865	20.37	11.9	2.8	81	11.6	334	20.0	572	30.4	878
Life Threatening Illness	23.1	669	34.59	15.5	1.0	30	0.8	24	2.3	67	19.0	548
Any of the Above	74.6	2157	18.91	12.2	4.8	140	17.1	495	22.5	651	30.1	871
Any other trauma	31.5	911	27.73	14.2	0.9	26	2.0	59	6.5	188	22.1	638

Table 3
Results of linear regression analyses examining trauma exposure (exposed vs. unexposed) on depressive and PTSD symptoms, adjusting for covariates.

	N (%)	Depressive symptoms		PTSD symptoms	
		Beta	95% CI	Beta	95% CI
Child Maltreatment	1624 (56.9)	0.549	(0.48, 0.62)	0.575	(0.51, 0.64)
Other Interpersonal Violence	2238 (77.4)	0.211	(0.13, 0.29)	0.297	(0.21, 0.38)
Non-Interpersonal Violence	2157 (74.6)	0.146	(0.06, 0.23)	0.256	(0.18, 0.34)
Any trauma not yet covered	911 (31.5)	0.152	(0.08, 0.22)	0.260	(0.19, 0.33)

Cell entries are sample size (n and %), beta coefficients, and 95% confidence intervals (CI). The table presents results from Model 1 examining exposure to trauma (coded as 0 = never exposed; 1 = exposed) on depressive and PTSD symptoms. Both outcomes were standardized to z-scores. Each trauma type was examined separately, thus the table includes results of eight separate multiple regression models (two for each exposure). All models controlled for age, sex, education, income, employment status, and exposure to any other traumatic event type.

(Model 3 results) and appeared driven primarily by exposure to sexual abuse (Supplemental Table 1).

Similarly, after adjusting for sociodemographic characteristics and frequency of trauma exposure, participants first exposed to other interpersonal violence during middle childhood ($\beta = 0.334$) had depressive symptoms scores that were about twice as large as those first exposed during adulthood ($\beta = 0.169$; Tukey p-value < 0.05). These results did not appear driven by exposure to a specific trauma type (Supplemental Table 1).

3.4. Timing of trauma exposure and PTSD symptoms

PTSD symptoms also varied as a function age at first exposure to child maltreatment and to some extent other interpersonal violence, but not non-interpersonal violence or any other trauma (Table 5). After adjusting for all covariates, those exposed to child

maltreatment during early childhood ($\beta = 0.681$) had significantly higher levels of PTSD symptoms relative to those first exposed during middle childhood ($\beta = 0.468$) or adolescence ($\beta = 0.342$; Tukey p-value < 0.05 for both comparisons). These differences were explained by exposure to child sexual abuse (Supplemental Table 2).

Although exposure to other interpersonal violence appeared initially most damaging in increasing levels of PTSD symptoms (Table 5), no significant differences were found across those exposed at different ages (relative to those never exposed) after adjusting for frequency of exposure (Tukey p-value < 0.05).

4. Discussion

This study examined the association between developmental timing of exposure to trauma and levels of depression and PTSD

Table 4
Results of linear regression analyses examining the effect of timing of first exposure to trauma on depressive symptoms, adjusting for covariates and frequency of exposure.

	N (%)	Model 2 (partial adjustment)			Model 3 (full adjustment)		
		Beta	95% CI	F-statistic (p-value)	Beta	95% CI	F-statistic (p-value)
Child Maltreatment				15.59 (<0.0001)			10.44 (<0.0001)
Early Childhood	449 (15.5)	0.739 ^{a,b}	(0.64, 0.84)		0.633 ^{a,b}	(0.52, 0.74)	
Middle Childhood	809 (28.0)	0.519	(0.44, 0.60)		0.444	(0.36, 0.53)	
Adolescence	366 (12.7)	0.397	(0.29, 0.51)		0.356	(0.25, 0.46)	
Other Interpersonal Violence				4.28 (0.005)			3.84 (0.009)
Early Childhood	123 (4.3)	0.304	(0.12, 0.49)		0.291	(0.10, 0.48)	
Middle Childhood	476 (16.5)	0.344 ^{c,d}	(0.23, 0.46)		0.334 ^d	(0.21, 0.46)	
Adolescence	900 (31.1)	0.177	(0.08, 0.27)		0.170	(0.07, 0.27)	
Adulthood	739 (25.6)	0.172	(0.07, 0.27)		0.169	(0.07, 0.27)	
Non-Interpersonal Violence				0.95 (0.414)			0.50 (0.684)
Early Childhood	140 (4.8)	0.099	(-0.07, 0.27)		-0.009	(-0.18, 0.17)	
Middle Childhood	495 (17.1)	0.179	(0.07, 0.29)		0.065	(-0.05, 0.18)	
Adolescence	651 (22.5)	0.172	(0.07, 0.27)		0.077	(-0.03, 0.18)	
Adulthood	871 (30.1)	0.113	(0.02, 0.21)		0.041	(-0.06, 0.14)	
Any trauma not yet covered				1.26 (0.287)			1.22 (0.302)
Early Childhood	26 (0.9)	0.195	(-0.16, 0.55)		0.210	(-0.15, 0.57)	
Middle Childhood	59 (2.04)	0.153	(-0.09, 0.39)		0.167	(-0.08, 0.41)	
Adolescence	188 (6.5)	0.056	(-0.08, 0.19)		0.070	(-0.08, 0.22)	
Adulthood	638 (22.1)	0.210	(0.12, 0.29)		0.221	(0.13, 0.31)	

The table presents results from Models 2 and 3, which examined age at first exposure (early childhood = age 0–5, middle childhood = age 6–10, adolescence = age 11–18, adulthood = age 19+) on depressive symptoms (z-score standardized). Model 2 (partial adjustment) controlled for age, sex, education, income, employment status, and exposure to any other traumatic event. Model 3 (full adjustment) controlled for all covariates included in Model 2 plus frequency of each trauma event occurrence (0 = low frequency; 1 = high reported number of occurrences of that trauma exposure). Each trauma type was examined separately, thus the table includes results of eight separate multiple regression models (two for each exposure). Beta coefficients and 95% confidence intervals (CI) indicate effects for developmental timing of exposure relative to the reference of never exposed in any developmental period. Effects are significant at $p < 0.05$ when CI does not include 0. Omnibus tests (F-tests and p-values) indicate the overall main effect of age at first exposure categories; these test the null hypothesis that the beta coefficients were equivalent across all groups (i.e., early childhood $\beta =$ middle childhood $\beta =$ adolescence β).

^a Refers to a significant difference ($p < 0.05$) between early childhood versus middle childhood.

^b Refers to a significant difference ($p < 0.05$) between early childhood versus adolescence.

^c Refers to a significant difference ($p < 0.05$) between middle childhood versus adolescence.

^d Refers to a significant difference ($p < 0.05$) between middle childhood versus adulthood.

symptoms within a sample of highly-trauma exposed adults. By studying a racially and socioeconomically homogeneous sample, where trauma exposure was common, we were able to examine the effects of age at first exposure to trauma. Such analyses may not have been possible in a heterogeneous sample where trauma exposure was more rare.

In line with previous research (McLaughlin et al., 2010a, 2012a; Norman et al., 2012; Widom et al., 2007), we found that those who were trauma exposed (regardless of age at first exposure) had significantly higher levels of depressive and PTSD symptoms relative to those who were unexposed to trauma. These findings add further support to the well-documented finding that trauma exposure elevates risk for psychopathology across the lifecourse.

However, a more novel observation from this study was that there appeared to be two developmental stages when trauma exposure was associated with elevated levels of subsequent psychopathology. First, participants exposed to child maltreatment during early childhood (ages 0–5) had both depression and PTSD symptoms that were up to twice as high as those exposed during later developmental stages. These effects were detected even after controlling for sociodemographic characteristics, exposure to other types of traumas, and the number of occurrences of child maltreatment (i.e., the frequency of exposure). Such findings are consistent with several prospective (Keiley et al., 2001; Thornberry et al., 2010; Kaplow and Widom, 2007) and retrospective studies (Dunn et al., 2013; Glod and Teicher, 1996) also showing an elevated risk for subsequent depression, in particular, among those first exposed in the first five years of life. Although the mechanisms linking early trauma exposure to subsequent psychopathology risk

are not well known (McLaughlin, 2016), early trauma exposure may be more damaging than later trauma exposure because it compromises a child's ability to successfully master stage-salient developmental tasks (e.g., self-regulation, secure attachments) (Cicchetti and Toth, 1995) and damages the foundation of brain architecture and neurobiological systems involved in regulating arousal, emotion, stress responses, and reward processing (McLaughlin et al., 2010b, 2011; Fox et al., 2010; Sheridan et al., 2012), which are all implicated in the onset and persistence of stress-related disorders like depression and PTSD.

Second, we also found that participants first exposed during middle childhood (ages 6–10) to other types of interpersonal violence, including having a friend or family member murdered or witnessing a friend or family member being attacked with or without a weapon, had depressive symptoms scores that were about twice as high as those first exposed during adulthood. Similar results were also detected for PTSD, but were not statistically significant after adjusting the frequency or number of occurrences of child exposure. These results are consistent with at least some prior studies suggesting that trauma during middle childhood, including severe illness (Schrage et al., 2008), or natural disasters (Demir et al., 2010; Green et al., 1991; Copeland et al., 2007) is associated with an elevated risk for PTSD relative to exposure in other periods. Although the mechanisms driving this association are unclear, exposure to interpersonal violence events during middle childhood may be more harmful than exposure in adulthood for a number of reasons: school-age children may be in a unique developmental stages where they do not benefit as much from parental buffering (Garbarino, 2001), when demands from the social environment

Table 5

Results of linear regression analyses examining the effect of timing of first exposure to trauma on PTSD symptoms, adjusting for covariates and frequency of exposure.

	N (%)	Model 2 (partial adjustment)			Model 3 (full adjustment)		
		Beta	95% CI	F-statistic (p-value)	Beta	95% CI	F-statistic (p-value)
Child Maltreatment				21.94 (p < 0.0001)			15.16 (<0.0001)
Early Childhood	449 (15.5)	0.792 ^{a,b}	(0.69, 0.89)		0.681 ^{a,b}	(0.57, 0.79)	
Middle Childhood	809 (28.0)	0.547 ^c	(0.47, 0.63)		0.468	(0.38, 0.55)	
Adolescence	366 (12.7)	0.386	(0.28, 0.49)		0.342	(0.24, 0.45)	
Other Interpersonal Violence				2.94 (0.032)			1.67 (0.171)
Early Childhood	123 (4.3)	0.315	(0.14, 0.49)		0.229	(0.04, 0.42)	
Middle Childhood	476 (16.5)	0.410 ^d	(0.30, 0.52)		0.345	(0.23, 0.46)	
Adolescence	900 (31.1)	0.288	(0.19, 0.38)		0.241	(0.14, 0.34)	
Adulthood	739 (25.6)	0.250	(0.15, 0.35)		0.231	(0.13, 0.33)	
Non-Interpersonal Violence				2.02 (0.109)			1.22 (0.302)
Early Childhood	140 (4.8)	0.224	(0.06, 0.39)		0.135	(-0.04, 0.31)	
Middle Childhood	495 (17.1)	0.305	(0.20, 0.41)		0.211	(0.09, 0.33)	
Adolescence	651 (22.5)	0.296	(0.20, 0.39)		0.218	(0.11, 0.32)	
Adulthood	871 (30.1)	0.204	(0.11, 0.30)		0.144	(0.05, 0.24)	
Any trauma not yet covered				1.08 (0.356)			1.12 (0.340)
Early Childhood	26 (0.9)	0.363	(0.01, 0.71)		0.341	(-0.01, 0.69)	
Middle Childhood	59 (2.04)	0.170	(-0.07, 0.41)		0.152	(-0.09, 0.39)	
Adolescence	188 (6.5)	0.206	(0.07, 0.34)		0.187	(0.04, 0.33)	
Adulthood	638 (22.1)	0.319	(0.24, 0.40)		0.303	(0.21, 0.39)	

The table presents results from Models 2 and 3, which examined age at first exposure (early childhood = age 0–5, middle childhood = age 6–10, adolescence = age 11–18, adulthood = age 19+) on PTSD symptoms (z-score standardized). Model 2 (partial adjustment) controlled for age, sex, education, income, employment status, and exposure to any other traumatic event. Model 3 (full adjustment) controlled for all covariates included in Model 2 plus frequency of each trauma event occurrence (0 = low frequency; 1 = high reported number of occurrences of that trauma exposure). Each trauma type was examined separately, thus the table includes results of eight separate multiple regression models (two for each exposure). Beta coefficients and 95% confidence intervals (CI) indicate effects for developmental timing of exposure relative to the reference of never exposed in any developmental period. Effects are significant at $p < 0.05$ when the CI does not include 0. Omnibus tests (F-tests and p-values) indicate the overall main effect of age at first exposure categories; these test the null hypothesis that the beta coefficients were equivalent across all groups (i.e., early childhood β = middle childhood β = adolescence β).

^a Refers to a significant difference ($p < 0.05$) between early childhood versus middle childhood.

^b Refers to a significant difference ($p < 0.05$) between early childhood versus adolescence.

^c Refers to a significant difference ($p < 0.05$) between middle childhood versus adolescence.

^d Refers to a significant difference ($p < 0.05$) between middle childhood versus adulthood.

increase (i.e., relating to peers, participating in school activities) (Margolin and Gordis, 2000), and when adaptive coping capacities are still developing (Zimmer-Gembeck and Skinner, 2011).

It is important to emphasize that these developmental timing differences would have been missed had we not considered the effect of timing of trauma exposure. Our findings therefore underscore that a basic comparison of those “exposed” to those who are “unexposed” may potentially mask important within-group differences that are only revealed when examining developmental timing of exposure to trauma. These findings also emphasize the need to adjust, where possible, for frequency of exposure to trauma, as some developmental timing differences may be attenuated after considering this information.

Several limitations of the current study must be noted. First, the measure of trauma exposure included in this study did not capture other characteristics of the trauma, including its severity, chronicity, or duration. Our frequency indicator variable may have captured some of these domains, though this remains unclear, as the frequency measure was not specific to a given year. In the case of abuse, the relationship of the perpetrator to the victim was also not examined and could have impacted the specific results for abuse types. Indeed, there is evidence suggesting more negative psychological outcomes among those experiencing abuse perpetrated by a family member rather than non-family member (Canton-Cortes and Canton, 2010; Ullman, 2007). Moving forward, larger scales studies are needed to examine the extent to which chronicity, duration, and perpetrator of the trauma varies as a function of developmental timing. Second, trauma exposure and age at first exposure to trauma were assessed retrospectively in adulthood. Retrospective reports of child maltreatment, in particular, have been shown in some cases to be less reliable and valid than prospective reports, because of memory inaccuracies, a reluctance to disclose personal matters, or current mood states (Hardt and Rutter, 2004). However, recent studies have found retrospective and prospective measures produce similar estimates of effect for mental disorders (Scott et al., 2012), suggesting that trauma exposure is harmful regardless of ascertainment strategy and that even if recall bias is present, effect estimates are unlikely altered. Some studies have documented differential recall bias across the lifespan, showing increasing problems in recall and disclosure of early trauma events as age increases (see for example (Yoshihama and Gillespie, 2002)) as well as an association between earlier age at onset of child abuse with greater amnesia in adult memory recovery of those events (Chu et al., 1999). However, accurate recall of memories as early as age 2–3 years old has been documented (Chu et al., 1999; Usher and Neisser, 1993), older individuals show no autobiographical memory recall difference for memories from any point in their lives (Howes and Katz, 1992), and underreporting is more likely than falsely positively reporting early abuse (Hardt and Rutter, 2004). Moreover, by using developmental periods, rather than specific ages, we were able to maintain consistency with prior studies and reduce recall bias as compared to reports focused on single ages. Participants unsure of their age at first trauma exposure were also excluded. Prospective research is needed to replicate these findings; this work would ideally incorporate repeated measures of trauma exposure and mental health in order to differentiate short-versus long-term effects of trauma timing on psychopathology. Finally, our sample was largely comprised of participants who were low income, female, and African Americans. Our analyses were also restricted to African Americans, due to the small number of respondents in other racial/ethnic groups. However, as noted above, a number of the findings from this study are consistent with those found in studies using samples with different demographic profiles. Further, this study focuses on a largely under-studied population with high levels of

mental health problems; studying this unique population is key to help determine that factors that might impact the development of psychiatric conditions and the developmental stages when interventions or supports may be most beneficial.

In conclusion, our study underscores the need to consider the developmental period of trauma exposure, as the effect of some traumas varied as a function of when in the course of development the trauma first occurred. Identification of these developmental stages of heightened vulnerability will aid in determining sensitive periods and guiding the investment of limited public health dollars towards the life stages when age-tailored interventions can be delivered and possibly yield their greatest returns.

Conflict of interest

The authors do not report any conflicts of interest.

Contributions

All authors contributed substantially to the work presented in this paper. ECD and KN wrote the main paper and analyzed data. AP and BB designed and ran the study and contributed to writing the paper. All authors contributed to conceptualizing the analysis, interpreting the results and editing the paper.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.jpsychires.2016.09.004>.

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