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Research paper

Developmental timing of trauma exposure and emotion dysregulation in adulthood: Are there sensitive periods when trauma is most harmful?

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ABSTRACT

Background: This study aimed to determine whether there were sensitive periods when a first exposure to trauma was most associated with emotion dysregulation symptoms in adulthood.
Methods: Adult participants came from a public urban hospital in Atlanta, GA (n = 1944). Lifetime trauma exposure was assessed using the Traumatic Events Inventory (TEI). Multiple linear regression models were used to assess the association between the developmental 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 emotion dysregulation symptoms, measured using the abbreviated Emotion Dysregulation Scale.
Results: Participants exposed to trauma at any age had higher emotion dysregulation scores than their unexposed peers. However, participants first exposed to child maltreatment or interpersonal violence during middle childhood had higher emotion dysregulation scores relative to those first exposed during other developmental stages; these developmental timing differences were detected even after controlling for sociodemographic factors, exposure to other trauma, and frequency of exposure to trauma. Further, after controlling for current psychiatric symptoms, the effect of other interpersonal trauma exposure in middle childhood was diminished and first exposure to other interpersonal violence in early childhood was associated with significantly lower emotion dysregulation symptoms.
Limitations: Limitations of this study include the use of retrospective reports and absence of complete information about trauma severity or duration.
Conclusion: These findings should be replicated in other population-based samples with prospective designs to confirm the importance of developmental timing of trauma on later emotion dysregulation.

1. Introduction

Emotion regulation, or the ability to effectively regulate one's emotions, is a critical component of healthy social functioning and mental health (Berking and Wupperman, 2012; Gross and Thompson, 2007; Hu et al., 2014). Conversely, deficits in emotion regulation, or emotion dysregulation, have been linked to many psychiatric disorders (Powers et al., 2015a; Sheppes et al., 2015), including borderline personality disorder (Carpenter and Trull, 2013), anxiety disorders (Jazaieri et al., 2015), eating disorders (Lavender et al., 2015), substance-use disorders (Berking et al., 2011; Fox et al., 2007), depression (Ehring and Quack, 2010) and non-suicidal self-injury (Andover and Morris, 2014). Emotion dysregulation is defined by deficits in several areas, including the ability to monitor and evaluate one's emotional experiences, modulate the intensity or duration of emotions, and/or to adaptively manage emotional reactions in order to meet situational demands (Cole et al., 1994; Gross and Thompson, 2007). Indeed, the importance of emotion dysregulation for psychopathology risk is reflected in the fact that emotion regulation is encompassed in the negative valence, cognition, and social processing domain of the Research Domain Criteria (Murdock et al., 1998) Initiative, a National Institutes of Mental Health (NIMH) effort to identify underlying transdiagnostic biobehavioral mechanisms responsible for psychopathology (Insel et al., 2010; Sanislow et al., 2010).

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Trauma exposure, particularly child maltreatment (e.g., neglect, emotional, physical and sexual abuse), is one of the primary determinants of emotion dysregulation and as is also a known risk factor for psychiatric disorders, especially depression and posttraumatic stress disorder (PTSD) (McLaughlin et al., 2012; McLaughlin et al., 2013). Prior studies have shown that trauma exposure is associated with deficits in emotion regulation across the lifespan, including during preschool (Langevin et al., 2016), adolescence (Shields and Cicchetti, 1997; Vettese et al., 2011) and adulthood (Briere and Rickards, 2007; Thompson et al., 2014). For example, preschool-age children exposed to sexual abuse have been shown to have emotion regulation scores that are, on average, one standard deviation below their unexposed peers (Langevin et al., 2016). These deficits appear patterned by frequency of trauma exposure, with children exposed to more chronic or frequent maltreatment having significantly worse outcomes relative to those who were never exposed or exposed to less chronic maltreatment (Thompson et al., 2014). Notably, relatively few studies have examined the relationship between other interpersonal or non-interpersonal trauma and emotion dysregulation. Efforts to understand the effects of trauma exposure on emotion dysregulation are needed, as epidemiological studies estimate that 70% of the world's population (Benjet et al., 2016), including 40% of children under age thirteen (Koenen et al., 2010), have experienced one or more traumatic events at some point in their lifetime.

Although these studies document the importance of trauma exposure, few studies have examined whether the effect of trauma exposure on emotion dysregulation varies based on the developmental timing of the trauma occurrence. Thus, it remains unclear whether there are “sensitive periods” (Bornstein, 1989; Dunn et al., 2013; Hensch, 2004) for the development of emotion dysregulation, or windows of time when the developing human brain is especially vulnerable or sensitive to trauma and when trauma exposure thus leads to greater levels of emotion dysregulation. Greater insights about the possible existence of sensitive periods for emotion dysregulation are needed to help guide the investment of limited public health dollars towards possible “high-risk” stages when trauma may be particularly harmful and thus the “high-reward” periods when interventions could be most efficacious in promoting emotion regulation abilities.

The developmental timing of trauma exposure may be important in shaping emotion regulation for several reasons. Emotion regulation is known to develop in conjunction with cognition, rapidly maturing during early childhood (Calkins, 1994) and through the influence of observational learning, modeling, and social referencing (Morris et al., 2007). Broadly, exposure to stressful stimuli, such as a chaotic home environment and childhood maltreatment, may reduce exposure to adaptive emotional labeling, expression, and regulation behaviors often modeled in families (Parke, 1994) and thus disrupt, delay, or impede normative emotion regulation development (Bradley et al., 2011a; Dvir et al., 2014; Kolk and Fisler, 1994). While parental responsiveness and encouragement of emotional expression promote emotional development (Roberts and Strayer, 1987), living in an invalidating environment where emotional expression is ignored, rejected, or punished may lead to emotion dysregulation (Linehan, 1993). Prior studies have shown that physical and emotional availability of the mother in infancy are critical to emotion regulation development. Thus, infancy and early childhood may be a developmental period when emotional development is particularly malleable and highly affected by trauma exposure (Field, 1994). Conversely, trauma exposure during adolescence may be more deleterious, as adolescence is a period characterized by increased executive functioning and heightened social sensitivity, which can interact with changing social environments to greatly influence behavior and emotion regulation (Blakemore and Mills, 2014).

It is also possible that the developmental timing of trauma exposure from infancy to adolescence is unrelated to emotion dysregulation or that it depends on the type of trauma. Indeed, social adversities have been shown to disrupt maturing mechanisms of emotional regulation at multiple phases of development (in preschoolers: differentiation of basic emotions; in school-age children: elaboration on emotional expression; in adolescence: understanding of the origins and consequences of negative emotions) (Pynoos et al., 1999). Collectively, these findings indicate there may be multiple sensitive periods to adversity in childhood and adolescence, perhaps depending on the type of trauma exposure, whereby the interaction between emotion regulation development and negative social exposures have differential, negative effects on future emotion dysregulation.

To our knowledge, only three studies have examined the effect of the developmental timing of trauma exposure on emotion dysregulation. In one prospective cohort study, Kim and colleagues found that children exposed to maltreatment between birth and 36 months (infancy and toddlerhood), but not those exposed after age 3 (preschool and school-age), had higher levels of emotion dysregulation at age 6–12 compared their unexposed peers (Kim and Cicchetti, 2010). A second study of children adopted from institutional care found that children adopted after age 15 months had higher levels of emotion dysregulation at age 8 compared to both those adopted before 15 months and a control group who was never institutionalized (Tottenham et al., 2010; Tottenham and Sheridan, 2010). A third retrospective study found that children exposed to interpersonal trauma exposure between birth and age 14 had higher emotion dysregulation in adulthood, compared to children exposed to interpersonal trauma after age 14 (Ehring and Quack, 2010). Taken together, these results suggest there may be differential effects of trauma on emotion dysregulation depending on timing of exposure as well as type of trauma.

The current study aimed to build upon prior work by assessing the impact of the developmental timing of trauma exposure on emotion dysregulation in adulthood using data from a cohort of urban African American adults with high trauma exposure. By studying adults, we could increase the likelihood that observed emotion regulation deficits are stable, and not potentially transient or confounded by variations in emotion regulation capabilities seen during their development (Calkins, 1994; McRae et al., 2012; Pynoos et al., 1999; Raffaelli et al., 2005). Our goals were to: 1) determine the extent to which different types of trauma exposure were associated with emotion dysregulation in adulthood; and 2) investigate whether timing of first trauma exposure was associated with emotion dysregulation in adulthood.

2. Methods

2.1. Sample and procedures

Data came from the Grady Trauma Project (GTP), an ongoing NIMH-funded study of the genetic and environmental risk and protective factors for PTSD and other psychiatric disorders (Binder et al., 2008; Bradley et al., 2008; Gillespie et al., 2009a, 2009b, 2009c). The GTP study recruited adults (ages 18–90) from general medical and obstetric/gynecological clinic waiting rooms at Grady Hospital in Atlanta, Georgia, a large public non-profit healthcare center. Grady Hospital primarily serves an African American, urban population from low socioeconomic backgrounds. This particular sample is beneficial to trauma research as there are high rates of trauma exposure, African Americans are an understudied population, and the sample is relatively homogeneous in terms of socioeconomic status. Eligibility criteria for participation included being at least 18 years old, not actively psychotic, and able to give written and verbal consent. Consenting individuals completed in-person interviews about their trauma history, current psychiatric symptoms, and general demographic information. Interviews were administered by trained research assistants and lasted approximately 45–75 min. Participants received $15 for their participation. All study procedures were approved by Emory University's Institutional Review Board and the Grady Health Care System Research Oversight Committee.

The current analysis included 1944 African American adults who

had complete data on all measures relevant to this analysis. This analytic sample represents 21.9% of the total GTP sample, who completed at least one item in the GTP study battery (N = 8886). Participants were excluded from the analytic sample if they had incomplete exposure, outcome, or covariate data. Interviews were conducted in clinic waiting rooms and continued until participants or their family members were seen by the clinic, thus the majority of participants did not complete all study measures. Therefore, due to this unique design, we suspect it is unlikely that those who were excluded were systematically different from those who were included. Indeed, the distribution of age, education, and income did not differ significantly between the analytic sample and the total GTP sample (all $p > 0.05$). However, relative to the analytic sample, the excluded sample did include slightly more females (74.2% in analytic sample; 71.3% in total sample; $p = 0.014$) and people who were unemployed (28.9% in analytic sample; 32.8% in total sample; $p = 0.0003$). All analyses were restricted to African Americans because individuals from other racial/ethnic groups comprised only 7.1% of the sample. By restricting to one racial/ethnic group, we more effectively controlled for confounding by eliminating variability associated with race, which was important as the distribution of trauma exposure, covariates, and outcome varied significantly by race. Stratification by race would have resulted in low power to detect associations due to small cell counts.

2.2. Measures

2.2.1. Exposure to trauma

Presence vs. absence of trauma exposure, age at first trauma exposure, and frequency of trauma occurrence were collected using the Traumatic Events Inventory (TEI), a 14-item screening measure that assessed lifetime history of trauma exposure (Gillespie et al., 2009a, 2009b, 2009c; Schwartz et al., 2006, 2005). We focused on 11 events that could plausibly occur in multiple developmental stages; the additional three events that were excluded from current analyses were military trauma, attacked by a romantic partner, and attacked by someone besides a romantic partner. These events were grouped into four trauma types, consistent with prior research (Breslau et al., 1998; McLaughlin et al., 2012): (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 being attacked with or without a weapon); (3) *non-interpersonal trauma* (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, etc).

If a participant reported being exposed to a traumatic event, their age at first exposure (in years) was recorded. We grouped these ages of first exposure for each traumatic event into four stages: early childhood (age 0–5 years), middle childhood (6–10 years), adolescence (11–18 years), and adulthood (19+ years). These categories were used to match previous research and minimize recall bias, relative to studying specific years of age (Dunn et al., 2013).

Participants also reported the number of times they were exposed to a given traumatic event, on a categorical scale ranging from 0 (unexposed) to 8 (greater than 20 times). We used this information to create a frequency indicator for each traumatic event, designating low versus high frequency of occurrence. High frequency of occurrence was defined as greater than or equal to the 75th percentile for that specific trauma event. These frequency indicators were used as covariates in models examining developmental timing of exposure to account for the possibility that people exposed at younger ages were more likely to have more occurrences of a given trauma and in acknowledgment that trauma exposure features (e.g., timing, frequency, and severity) may have independent effects on psychopathology risk (Manly et al., 1994, 2001).

2.2.2. Emotion dysregulation

Emotion dysregulation was assessed using a shortened version of the Emotion Dysregulation Scale (Powers et al., 2015b), a 12-item scale that captures emotional, cognitive, and behavioral components of emotion regulation. The 12-item scale was adapted from an original 24-item version based on a clinician-rated Affect Regulation and Experience Q-sort Questionnaire (Conklin et al., 2006; Westen et al., 1997). Participants indicated on a Likert-type scale (1 = not true; 7 = very true) the extent to which they thought the item describes them. In this sample, total emotion dysregulation scores were calculated by averaging all individual items (where at least 11 were completed) and then multiplying that mean by 12 (range = 12–84). Higher scores therefore indicated greater emotion dysregulation. The 12-item scale demonstrated excellent internal consistency reliability in this sample (standardized α coefficient = 0.94) and good construct validity relative to the original scale (Powers et al., 2015b). Sample items on this 12-item scale included: “When I'm upset, I have trouble thinking clearly,” “I have trouble soothing myself when I am upset,” and “When my emotions are strong, I often make bad decisions.”

2.2.3. Covariates

The following covariates were included in all analytic models: 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; $1,000+), and employment status (unemployed; unemployed receiving disability support; employed with or without disability support). We also adjusted for high (vs. low) levels of current depressive and posttraumatic symptoms based on responses to the Beck's Depression Inventory - Second Edition (Beck et al., 1988) and the Modified PTSD Symptom Scale to assess posttraumatic stress symptoms (American Psychiatric Association, 2000; Coffey et al., 1998). While emotion dysregulation is closely related to multiple forms of psychopathology, it is important to distinguish between the deficits in emotion regulation capacity and symptoms of mental illness. In particular, emotion dysregulation is found in individuals with high depressive and posttraumatic stress symptomatology (Aldao et al., 2010; Tull et al., 2007), two forms of psychopathology greatly impacted by early trauma exposure (Copeland et al., 2007; McLaughlin et al., 2010). Therefore, assessments of relationships between trauma exposure and later emotion dysregulation should control for depressive and posttraumatic stress symptoms.

2.3. Data analyses

First, we conducted univariate and bivariate analyses to examine the distribution of emotion dysregulation and trauma exposure in the sample and compare emotion dysregulation values by each covariate. Second, we conducted a series of linear regressions, separately for each traumatic event and trauma type, to determine the association between trauma exposure and emotion dysregulation after controlling for covariates. Model 1 assessed the effect of trauma exposure (1 = exposed vs. 0 = never exposed) on emotion dysregulation. Model 2 assessed the effect of age at first exposure (1 = early childhood; 2 = middle childhood; 3 = adolescence; 4 = adulthood vs. 0 = never exposed) on emotion dysregulation. In this second model, we adjusted for exposure to any other trauma (beyond the focal trauma examined), as trauma exposures were moderately correlated in this sample (tetrachoric correlation: $r = 0.06$ to $r = 0.63$ for individual traumatic events; average correlation for child maltreatment events: $r = 0.48$) and prior studies have shown that failure to account for co-occurring trauma exposure may overemphasize the impact of a single trauma (McLaughlin et al.,

2010). Model 3 expanded upon Model 2 by additionally adjusting for frequency of each trauma event (0 = low frequency; 1 = high frequency). Model 4 built upon Model 3 by additionally controlling for current level of depressive and posttraumatic symptoms as described above.

Third, we conducted tests of homogeneity for all models assessing the role of developmental timing (Models 2, 3, and 4) to determine whether the beta coefficients, which indicated the effect of age at first exposure relative to never exposed, were significantly different from each other. When significant omnibus differences were found in the homogeneity test, we then performed post hoc Tukey comparisons to determine whether individual beta coefficients indicating the effect of age at first trauma exposure were significantly different from each other. A significant Tukey value signifies differences in effect of exposure during different age periods after correcting for multiple testing. All analyses were performed using SAS Version 9.4 (SAS Institute, Inc, Cary, North Carolina).

3. Results

In the analytic sample of 1944 African American adults, 74.2% of the sample was female, the mean age was 40.2 years (SD = 13.6; range 18–78) and the mean emotion dysregulation score was 38.26 (SD = 21.63). Emotion dysregulation scores varied significantly across all covariates (Table 1), with women, middle aged individuals, and those with lower education, income, and unemployment with disability status having higher emotion dysregulation scores. Slightly less than half of the sample (40.7%; n = 791) had high depressive symptoms, posttraumatic stress symptoms or both. High depressive and posttraumatic symptoms were significantly associated with higher emotion dysregulation scores (tetrachoric correlation: r = 0.66, p < 0.0001).

3.1. Trauma exposure: distribution, age at first exposure, and frequency of exposure

Nearly all participants (94.9% of the sample) reported at least one trauma exposure; 84.3% reported at least two event exposures. The most commonly reported trauma was serious accident or injury.

The distribution of age at first trauma exposure varied by trauma type (Table 2). For example, about half (50.5%) of individuals exposed to child maltreatment were first exposed in middle childhood, whereas only 20.3% of individuals were first exposed to any type of other interpersonal violence in middle childhood. Non-interpersonal trauma was most often reported as first occurring during adulthood.

There was a gradient in the relationship between age at first exposure and frequency of exposure, specifically for child maltreatment and other interpersonal violence, suggesting that those first exposed in earlier developmental periods also tended to report more frequent occurrences of exposure (Fig. 1).

3.2. Trauma exposure and emotion dysregulation symptoms: the role of exposure type and timing of exposure

As shown in Table 3, where the results from Model 1 are reported in the row for each bolded trauma type, exposure to child maltreatment, interpersonal violence, and non-interpersonal trauma were all associated with increases in emotion dysregulation symptoms in adulthood. The largest observed effect was for child maltreatment, where we found that people exposed to maltreatment scored 9.94 points higher than their unexposed peers on the emotion dysregulation symptoms measure, even after adjusting for covariates and other trauma exposure (β = 9.94; 95% CI = 8.04, 11.83, p < 0.0001).

3.2.1. Child maltreatment

As shown in the columns of Table 3 and Fig. 2, which presents results from Models 2–4, the effect of the developmental timing of trauma

Table 1
Distribution of covariates and emotion dysregulation symptoms in the Grady Trauma Project (GTP) analytic sample (N = 1944).

Covariate	Total sample	Emotion dysregulation symptoms		
	N (%)	Mean (SD)	F value	*p value*
Age				
18–25	408 (21.0)	38.71 (20.3)	7.88	< 0.0001
26–35	366 (18.8)	39.22 (21.8)		
36–45	354 (18.2)	41.28 (22.9)		
46–55	535 (27.5)	38.52 (22.0)		
56+	281 (14.5)	32.05 (19.6)		
Sex				
Male	501 (25.8)	36.37 (21.3)	5.16	0.023
Female	1443 (74.2)	38.91 (21.7)		
Education				
Less than 12th grade	432 (22.2)	43.30 (22.9)	19.42	< 0.0001
High school graduate or GED	842 (43.3)	38.22 (21.6)		
Greater than high school or GED/College graduate	670 (34.5)	35.06 (20.2)		
Income				
$0–499	618 (31.8)	41.93 (22.5)	16.59	< 0.0001
$500–999	526 (27.1)	38.43 (21.9)		
$1000 or more	800 (41.1)	35.31 (20.2)		
Employment Status				
Unemployed	1047 (53.9)	39.33 (21.9)	14.78	< 0.0001
Unemployed (with disability)	336 (17.3)	41.55 (22.9)		
Employed (with or without disability)	561 (22.8)	34.30 (19.7)		
Depressive and posttraumatic stress symptoms				
Low symptoms	1153 (59.3)	28.65 (17.0)	785.01	< 0.0001
High symptoms	791 (40.7)	52.27 (20.0)		

Descriptive statistics are presented for the analytic sample. Linear regressions were performed by covariate, with F-statistics and corresponding p-values listed. High depressive and posttraumatic stress symptoms are based on measures of depression (Beck's Depression Inventory - Second Edition (BDI-II) and posttraumatic stress symptoms (Modified PTSD Symptom Scale (MPSS)). Participants were coded as having high depressive and posttraumatic stress symptoms (vs. low symptoms) if BDI scores were 20 or greater and/or if on the MPSS the participated reported the presence of at least one re-experiencing symptom (scored 1 or higher), two avoidance/numbing symptoms, and two hyperarousal symptoms.

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

Exposure	Exposed		Age at First Exposure (year)		Age at First Exposure (category)							
					Ages 0–5		Ages 6–10		Ages 11–18		Ages 19+	
	%	N	Mean	SD	%	N	%	N	%	N	%	N
Child Maltreatment												
Violence between Caregivers	30.1	585	8.10	3.3	24.6	144	53.8	315	21.6	126	–	–
Physical Abuse	19.1	371	8.19	3.3	23.7	88	55.0	204	21.3	79	–	–
Emotional aAbuse	24.4	474	9.99	3.8	13.9	66	43.9	208	42.2	200	–	–
Sexual Abuse	29.8	579	9.68	3.8	15.0	87	44.2	256	40.8	236	–	–
Any of the Above	54.4	1058	8.08	3.5	27.1	287	50.5	533	22.5	238	–	–
Other Interpersonal Violence												
Family/friend Murdered	54.0	1049	22.22	11.8	1.6	17	11.0	115	37.1	389	50.3	528
Witness Attack (Family or friend)	42.0	817	17.49	10.0	5.1	42	20.0	163	39.9	326	35.0	286
Witness Attack (non-family/friend)	43.4	843	19.16	10.3	2.7	23	14.9	126	42.8	361	39.5	333
Any of the Above	77.6	1509	17.81	10.4	4.6	70	20.3	307	40.9	616	34.2	516
Non-Interpersonal Trauma												
Natural Disaster	26.1	507	20.19	13.5	5.1	26	24.6	125	28.4	144	41.8	212
Serious Accident or Injury	64.2	1247	20.33	11.7	4.1	51	18.0	224	30.7	383	47.2	589
Life Threatening Illness	22.3	433	33.81	15.6	5.1	22	3.7	16	10.1	44	81.1	351
Any of the Above	74.4	1446	18.84	11.9	6.7	97	22.5	325	30.1	435	40.7	589
Any Other Trauma	29.1	566	27.42	13.9	2.7	15	6.2	35	21.0	119	70.1	397

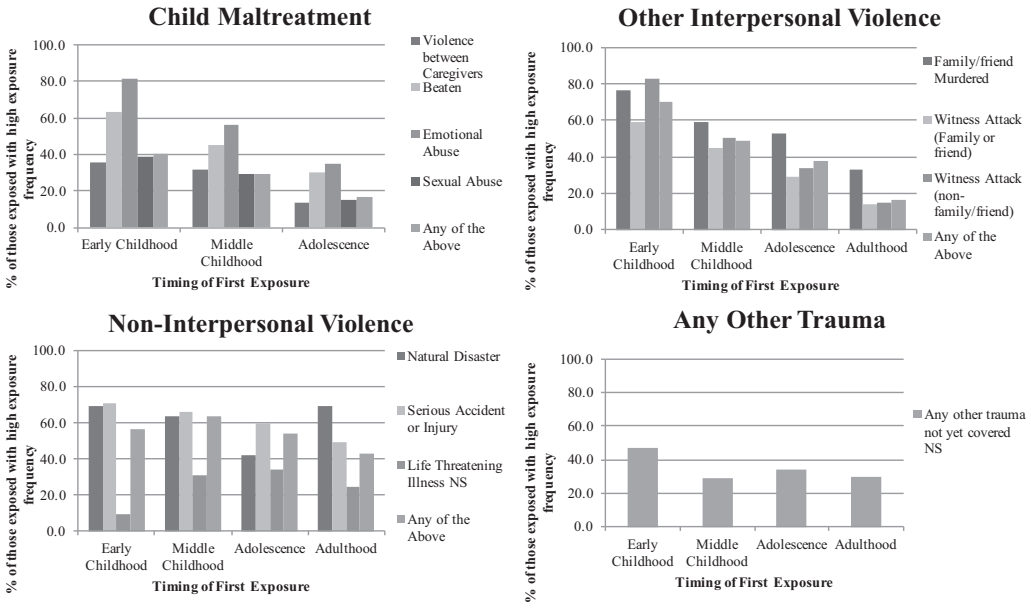

Fig. 1. Percent of respondents exposed to frequent trauma by age at first exposed to trauma. The figure presents the percentage of those exposed to frequent trauma, within each age category, among those exposed. All Chi Square Goodness of Fit models for each trauma event, which evaluated whether there 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 ($p > 0.05$). 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: natural disaster ≥ 2; serious accident or injury ≥ 2; and sudden life threatening illness ≥ 2; a friend or family member being murdered ≥ 2; a family member or friend being attacked ≥ 4; a non-family member or friend attacked ≥ 5; violence between parents or caregivers ≥ 8; beaten ≥ 8; emotional abuse ≥ 8; or 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.

on emotion dysregulation varied by trauma type. Differences in the magnitude of association were observed for both child maltreatment (homogeneity p-value = 0.004) and other interpersonal violence (homogeneity p-value = 0.008). Specifically, although child maltreatment beginning at any age was associated with increased emotion dysregulation symptoms relative to no exposure, people first exposed in early childhood (β = 11.868, 95% CI = 9.08, 14.65) or middle childhood (β = 10.543, 95% CI = 8.30, 12.79) had higher emotion dysregulation symptoms relative to people first exposed in adolescence (β = 6.389, 95% CI = 3.43, 9.35; Tukey post-hoc pairwise p-values < 0.05). These differences were no longer significant after adjustment for frequency of exposure (Model 3). After further controlling for depressive and posttraumatic stress symptoms (Model 4), effect estimates were attenuated, though the differences in magnitude of effect persisted in comparing those first exposed in middle childhood to those first exposed in adolescence (Tukey post-hoc pairwise p-value < 0.05).

3.2.2. Other interpersonal violence

For other interpersonal violence, only middle childhood was associated with increased emotion dysregulation symptoms in adulthood after controlling for covariates, other trauma exposure (Model 2: middle childhood β = 6.368, 95% CI = 3.26, 9.48), and frequency of the trauma occurrence (Model 3: middle childhood β = 5.807, 95% CI = 2.53, 9.08) (Table 3 and Fig. 2). The effect of first exposure in middle childhood was significantly higher than first exposure in adolescence or adulthood (Tukey post-hoc pairwise p-values < 0.05). After controlling for depressive and posttraumatic stress symptoms (Model 4), these differences were no longer observed. However, we did find that first exposure during early childhood was associated with lower emotion

Table 3
Results of linear regression analyses examining the effect of age at first trauma exposure on emotion dysregulation symptoms, adjusting for multiple covariates.

		Model 2 (adjustment for socio-demographic factors and other trauma exposure)			Model 3 (adjustment for Model 2 plus frequency of trauma)			Model 4 (adjustment for Model 3 plus depressive or posttraumatic stress symptoms)		
	N (%)	Beta	95% CI	Homogeneity F-statistic (p-value)	Beta	95% CI	Homogeneity F-statistic (p-value)	Beta	95% CI	Homogeneity F-statistic (p-value)
Child Maltreatment		**9.936**	**(8.04, 11.83)**	**5.45 (0.004)**	**8.421**	**(6.38, 10.47)**	**3.65 (0.026)**	**3.479**	**(1.65, 5.31)**	**3.36 (0.035)**
Early Childhood	287 (14.8)	**11.868**[b]	**(9.08, 14.65)**		**10.028**	**(7.04, 13.01)**		**3.698**	**(1.04, 6.36)**	
Middle Childhood	533 (27.4)	**10.543**[d]	**(8.30, 12.79)**		**9.201**	**(6.83, 11.58)**		**4.655**[d]	**(2.55, 6.76)**	
Adolescence	238 (12.2)	**6.389**	**(3.43, 9.35)**		**5.603**	**(2.62, 8.59)**		1.058	(−1.58, 3.69)	
Other Interpersonal Violence		**2.913**	**(0.65, 5.18)**	**3.99 (0.008)**	**2.499**	**(0.24, 4.66)**	**3.81 (0.010)**	0.326	(−1.74, 2.39)	**4.33 (0.005)**
Early Childhood	70 (3.6)	−0.751	(−6.02, 4.52)		−1.560	(−7.03, 3.91)		**−5.529**[a]	**(−10.32, −0.74)**	
Middle Childhood	307 (15.8)	**6.368**[d,e]	**(3.26, 9.48)**		**5.807**[d,e]	**(2.53, 9.08)**		2.647	(−0.23, 5.52)	
Adolescence	616 (31.7)	2.308	(−0.29, 4.91)		1.886	(−0.82, 4.60)		−0.046	(−2.42, 2.33)	
Adulthood	516 (26.5)	2.262	(−0.39, 4.91)		2.095	(−0.57, 4.76)		0.080	(−2.25, 2.42)	
Non-Interpersonal Trauma		**2.542**	**(0.34, 4.75)**	2.32 (0.074)	1.706	(−0.71, 4.12)	2.01 (0.111)	0.085	(−2.03, 2.20)	2.44 (0.063)
Early Childhood	97 (5.0)									
Middle Childhood	325 (16.7)									
Adolescence	435 (22.4)									
Adulthood	589 (30.3)									
Any Other Trauma		1.682	(−0.39, 3.75)	1.14 (0.330)	1.448	(−0.92, 3.81)	1.12 (0.342)	−0.463	(−2.53, 1.61)	1.90 (0.128)
Early Childhood	15 (0.8)									
Middle Childhood	35 (1.8)									
Adolescence	119 (6.0)									
Adulthood	397 (20.4)									

The table presents results from Models 1-4, which examined exposure to trauma (v. unexposed) and age at first exposure to trauma (early childhood = age 0–5, middle childhood = age 6–10, adolescence = age 11–18, adulthood = age 19+; v. unexposed) on emotion dysregulation symptoms. Model 1 (exposed v. unexposed) is presented in the first row of each trauma type. Models 1 and 2 controlled age, for sex, education, income, employment status and exposure to any other traumatic event. Model 2 (age at first exposure v. unexposed) controlled age, sex, education, income, employment status, and exposure to any other traumatic event. Model 3 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). Model 4 controlled for all covariates in Model 3 plus presence (vs. absence) of current high levels of either depressive or posttraumatic stress symptoms. Each trauma type was examined separately, thus the table includes results of twelve separate multiple regression models (three models for each exposure).

Cell entries are beta coefficients and 95% confidence intervals (CI) estimating the association between exposure to trauma type (exposed vs. unexposed) and timing of exposure to maltreatment (e.g., exposed first during early childhood vs. unexposed), 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 for homogeneity (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 beta = adult beta)

[c]refers to a significant difference ($p < 0.05$) between early childhood vs. adulthood, based on the Tukey post-hoc test.

[a] Refers to a significant difference ($p < 0.05$) between early childhood vs. middle childhood, based on the Tukey post-hoc test.

[b] Refers to a significant difference ($p < 0.05$) between early childhood vs. adolescence, based on the Tukey post-hoc test.

[d] Refers to a significant difference ($p < 0.05$) between middle childhood vs. adolescence, based on the Tukey post-hoc test.

[e] Refers to a significant difference ($p < 0.05$) between middle childhood vs. adulthood, based on the Tukey post-hoc test.

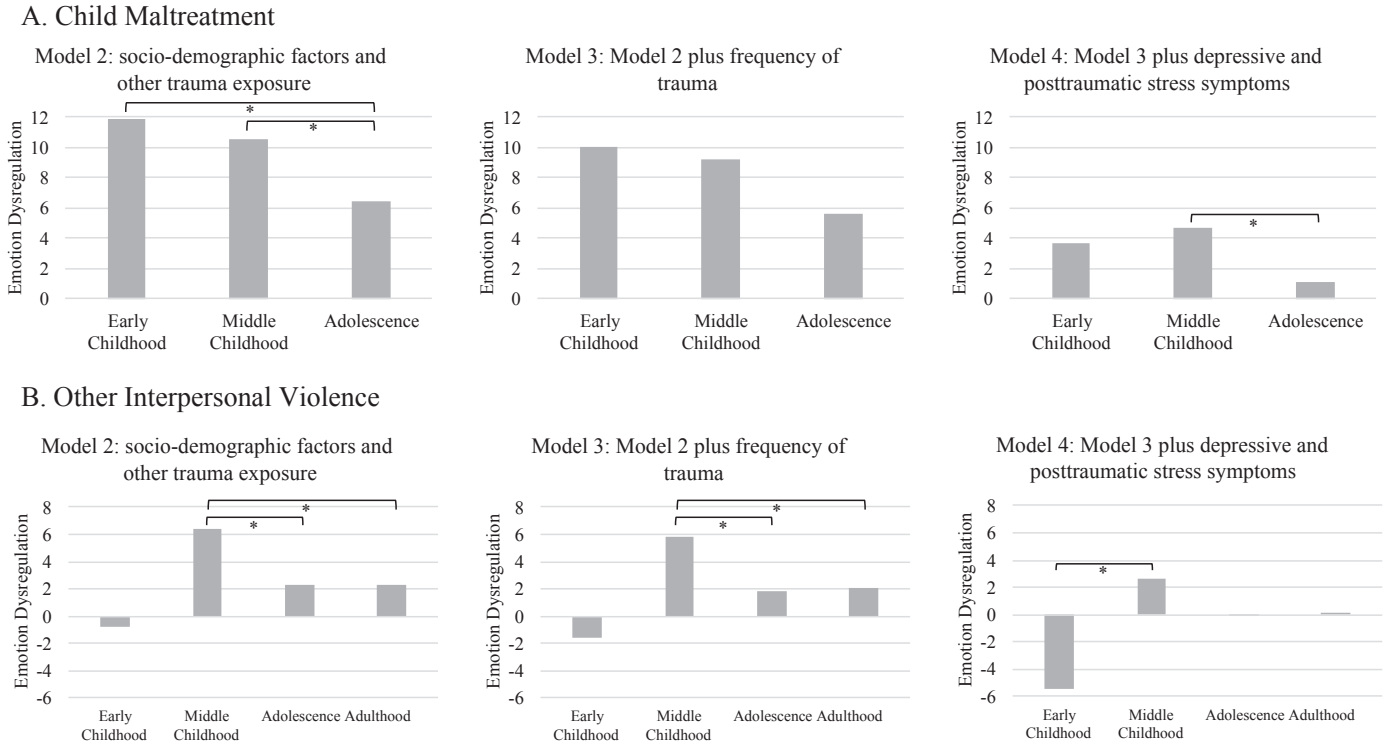

Fig. 2. Regression coefficients for the effect of age at first trauma exposure on emotion dysregulation symptoms, adjusting for multiple covariates. The figure presents results from Models 2, 3, and 4 which examined age at first exposure to trauma (early childhood = age 0–5, middle childhood = age 6–10, adolescence = age 11–18, adulthood = age 19+; v. unexposed) on emotion dysregulation symptoms. Model 2 controlled age, sex, education, income, employment status, and exposure to any other traumatic event. Model 3 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). Model 4 controlled for all covariates in Model 3 plus presence (vs. absence) of current high levels of either depressive or posttraumatic stress symptoms. The y-axis indicates the beta coefficient from the regression models, or the effect of exposure to trauma during an age group compared to those unexposed on emotion dysregulation symptoms. All omnibus tests for homogeneity were significant, indicating that the beta coefficients were not equivalent across all groups (i.e., the null hyopthesis is that $\beta^{\text{early childhood}} = \beta^{\text{middle childhood}} = \beta^{\text{adolescence}} = \beta^{\text{adulthood}}$). Brackets and * indicate a significant difference ($p < 0.05$) between age at first exposure group effect size, based on the Tukey post-hoc test (e.g., in A. Child Maltreatment, Model 2, the effect of early childhood was significantly different from the effect of adolescence, and the effect of middle childhood significantly different from the effect of adolescence).

dysregulation symptoms in adulthood relative to first exposure in middle childhood (Model 4: early childhood $\beta = -5.529$, 95% CI = $-10.32, -0.74$).

3.2.3. Non-interpersonal trauma

There were no significant developmental timing differences by age at first exposure to non-interpersonal trauma on emotion dysregulation (all effects $p > 0.05$).

3.2.4. Any trauma Not Yet Covered

No significant developmental timing differences were found for age at first exposure to other trauma and emotion dysregulation (all effects $p > 0.05$).

4. Discussion

In this study, we examined the association between different types of trauma and levels of emotion dysregulation in adulthood. Conducting these analyses in a sample with high trauma exposure allowed for both between group (exposed and unexposed individuals) and within group (exposed) comparisons. A particularly unique focus of this work was to investigate the role of the developmental timing of trauma exposure on emotion dysregulation scores before and after controlling for potentially confounding factors, including frequency of trauma occurrence as well as other trauma types, which facilitated the identification of potential sensitive periods when trauma was most likely to affect emotion dysregulation.

Our results suggest that people exposed to trauma were generally more likely than their unexposed peers to have higher emotion dysregulation. However, the effect of trauma varied as a function of not just trauma type, but also when in course of the lifespan the trauma first occurred. Specifically, three findings related to trauma type and developmental timing emerged from this study. First, we found that exposure to child maltreatment beginning in middle childhood was most associated with adult emotion dysregulation symptoms, compared to other developmental time periods, and that this relationship persisted after controlling for socioeconomic factors, frequency of trauma occurrence, and current depressive and posttraumatic stress symptoms. Second, other interpersonal trauma exposure beginning in middle childhood was also the developmental period most associated with emotion dysregulation, even after accounting for frequency of trauma occurrence. Thirdly, after controlling for current depressive and posttraumatic stress symptoms, the effect of other interpersonal violence exposure in middle childhood was diminished and first exposure to other interpersonal violence in early childhood was associated with significantly *lower* emotion dysregulation symptoms.

Collectively, these results are consistent with prior literature showing increased levels of emotion dysregulation in adulthood among adults exposed to child maltreatment or interpersonal trauma (Briere and Rickards, 2007; Thompson et al., 2014), particularly when the trauma was interpersonal in nature, chronic, and began prior to adolescence (Ehring and Quack, 2010). However, our findings differ from previous studies assessing the role of developmental timing of exposure to maltreatment and emotion dysregulation. For example, early maltreatment exposure (between birth to age 3), but not later maltreatment (between ages 3–5) (Kim and Cicchetti, 2010), and longer institutional care (after age 15 months), compared to shorter institutional care (adopted prior to 15 months) (Tottenham and Sheridan, 2010) have

been previously associated with higher emotion dysregulation in early adolescence. Our findings may differ from these two studies because both of them assessed emotion dysregulation in early adolescence, a time when emotion regulation capabilities are still developing; we, instead, focused on adulthood, when emotion regulation capabilities are likely more fixed. Furthermore, differences between our study and these two prior ones could be explained by differences in the measurement of trauma features (e.g., frequency, chronicity, severity, etc.) and methods to adjust for covariates (e.g., controlling for psychiatric symptoms, stratifying by psychiatric status, assessing emotion dysregulation as a mediator or moderator, etc.).

Child maltreatment during middle childhood could be especially harmful for emotion regulation capacities for several reasons. As noted previously, emotion regulation depends on an individual's cognitive capabilities as well as his or her positive interpersonal, familial, and other social relationships (Calkins, 1994; Morris et al., 2007). Emotion regulation skills rapidly develop during middle childhood and once children reach older childhood (about age 8–12), these skills have developed and children can effectively use multiple emotional coping strategies (Fields and Prinz, 1997). Therefore, disruptions of attachment with caregivers and other social relationships and the failure to receive appropriate modeling of behavior, at a time when the cognitive and emotional capabilities to navigate such relationships are developing (Bradley et al., 2011b; Dvir et al., 2014), may be therefore especially damaging. There is also evidence of a stress-hyporesponsive period characterized by lower stress reactivity and lower glucocorticoid activity that occurs approximately from ages one to six (Gunnar and Quevedo, 2007), which may contribute to the relative lack of negative impact of early childhood trauma. Further, memory is not fully developed in early childhood, thus individuals mostly remember and report traumatic exposures beginning around middle childhood. These observations may also explain our finding that after accounting for current depressive and posttraumatic stress symptoms, first exposure to other interpersonal violence during early childhood was associated with less emotion dysregulation as compared to first exposure during middle childhood.

Results from this study should be evaluated in light of several limitations. First, lifetime trauma exposure was assessed retrospectively in the context of a cross-sectional design. Retrospective reports of child maltreatment, in particular, may be less reliable and valid compared to prospective reports due to the influence of current psychiatric state, inaccurate memory, and reluctance to report sensitive personal information (Hardt and Rutter, 2004). However, recent work has found similar effect estimates for psychiatrically-relevant outcomes with both retrospective and prospective measures of trauma (Scott et al., 2012). Further, with retrospective reporting, adults have been shown to minimize their degree of exposure, suggesting that false negative reports may be more common than false positive reports (Brewin et al., 1993; Shaffer et al., 2008). Relatedly, due to recall bias, there may be potential inaccuracies in the reported age at first exposure to trauma. We aimed to minimize these inaccuracies by grouping exposure ages into developmental time periods. Prospective research would be ideal to replicate our cross-sectional findings and further assess the impact of developmental timing of trauma exposure.

Second, the characteristics of trauma assessed in this study were limited to exposure, age at first exposed, and frequency of trauma occurrence, thereby potentially overlooking other important aspects of trauma such as its severity or duration. However, the frequency of trauma occurrence information allows us to distinguish between individuals exposed only once or twice from those exposed multiple times, potentially yielding new insight into the role of these characteristics. Third, generalizability of results may be limited due to the nature of our sample. However, low-income African American women are generally underserved (Carrington, 2006) and research is needed to understand risks within this population due to their high burden of trauma exposure. Finally, the assessment of psychiatric symptoms was exclusively based on self-reported questionnaires, rather than interviewer-based measures or clinician-based diagnostic assessments.

In conclusion, results from this study suggest that the effects of child maltreatment and other interpersonal violence on emotion dysregulation in adulthood vary as a function of when in the lifecourse these traumas first occurred. Evidence in support of possible sensitive periods could inform policy and practice to guide implementation and increase effectiveness of early interventions for individuals exposed to trauma. Specifically, new knowledge about the timing of sensitive periods could lead clinicians to deliver interventions at specific age stages when these interventions would be more likely to yield stronger impacts in terms of offsetting the negative sequela of trauma. However, for these types of clinical possibilities to be realized, these findings should be replicated in other population-based samples with prospective designs to confirm the importance of developmental timing of trauma on later emotion dysregulation.

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