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Timing of Trauma Exposure and Criminal Involvement on PTSD and Mediation by Emotion Dysregulation

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2014

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Abstract

Timing of Trauma Exposure and Criminal Involvement on PTSD
and Mediation by Emotion Dysregulation
By Ye Ji Kim

Identifying sensitive periods in development and the timing of trauma and criminal involvement are critical in understanding the biological etiology of post-traumatic stress disorder (PTSD). In defining sensitive periods and mechanistic pathways, emotion dysregulation (ED) may be a potential trans-diagnostic risk factor for PTSD. We conducted secondary analyses of a cross-sectional study of highly trauma-exposed, civilian adults in Atlanta, GA (n=10,503). The current study investigated whether developmentally sensitive timing of trauma and justice system involvement are associated with PTSD diagnosis as well as PTSD severity among non-Hispanic, AA adults (n=4,000). In addition, ED was examined as a potential mediator of the relationship between both age of onset of trauma and PTSD symptom severity and age of onset of justice system involvement and PTSD severity. All analyses adjusted for sociodemographic characteristics, depressive symptoms, and frequency of exposure to trauma. Participants exposed to trauma in early childhood had greater odds of a PTSD diagnoses compared to those not exposed to trauma (aOR=1.5, 95% CI=0.5, 4.3). Participants exposed to trauma in middle childhood had greater PTSD symptom severity than those exposed during later developmental stages and to those not exposed (adjusted beta=0.7, 95% CI=-2.9, 4.3). Participants with juvenile age of onset of justice system involvement had greater odds (arrest: aOR=1.3, 95% CI=1.0, 1.7; jail: aOR=1.3, 95% CI=1.0, 1.8) and greater symptom severity than those whose age of onset of justice system involvement were in adulthood (arrest: adjusted beta=1.5, 95% CI=0.7, 2.3; jail: adjusted beta=1.5, 95% CI=0.5, 2.5). ED mediated the relationship between age of onset of trauma in middle childhood and PTSD symptom severity, and between juvenile age of onset of justice system involvement and PTSD symptom severity, but not for adulthood age of onset of justice system involvement. The current study findings provide new information that may guide further research in identifying critical developmental periods where trauma exposures and involvement with the justice systems may have detrimental effects in mental health of an already highly vulnerable populations.

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Background

1.1 Introduction

In the general population, the lifetime exposure to a traumatic event ranges with prevalence rates between 51% to 74% (1–3). Traumatic events include, but are not limited to, crime victimization, sexual assaults, exposure to natural disasters, and combat exposure. Experiencing a traumatic event has been suggested as a risk factor to a wide range of both mental and physical health problems (4–8). In addition, exposure to a traumatic event has also been associated with an increase in risky health behaviors, such as substance use and engaging in activities associated with HIV transmission risk (6,9–11).

Only a subsample of those who experience or witness a traumatic event develops posttraumatic stress disorder (PTSD; 3). PTSD is a disorder defined in the DSM-5 with fear-based emotional and behavioral symptoms after an exposure to a traumatic event and the DSM-5 criteria for PTSD includes criterion A through H (12). PTSD is prevalent among 6.8% of all adult Americans during their lifetime (13). Along with symptoms that debilitate functioning, such as hypervigilance, emotional numbing, and negative cognition, traumatic exposure and subsequent PTSD have been associated with worse physical health status than those with a traumatic exposure but without PTSD (7). In particular, PTSD criterion B (re-experiencing) symptoms have been related to reduced physical health functioning and higher experience of bodily pain, whereas criterion D (hypervigilance) symptoms have been related to poorer perceptions of emotional health (14).

With understanding of the detrimental health outcomes due to traumatic exposure and PTSD, recent research has focused on the various individual- and neighborhood-level risk factors of PTSD for prevention. Research findings indicate that living in high-crime neighborhoods may increase childhood trauma survivors' risk for both major depression and PTSD symptoms (15). Individuals of racial and ethnic minority subgroups from urban and economically disadvantaged backgrounds are at greater likelihood of poor mental health, and specifically PTSD. The reported national average prevalence of PTSD is likely an underestimate for individuals from urban and economically disadvantaged areas, due to the higher risk of exposure to traumatic events than the general population. Despite the potentially high rate of PTSD, trauma is under-recognized and PTSD is under-diagnosed in the urban population (16,17), particularly for the African-American (AA) community (18,19).

1.2 Criminal involvement, overall mental health, and PTSD

Individuals in prisons or jails are at greater odds of having a more extensive trauma history that also place them at a higher risk for developing PTSD (20,21). A study of individuals in the Iowa's state prison population indicate that those who are involved with the justice system experience an inordinate burden of mental disorders with 35.9% of AA men and 62.1% of AA women having at least one mental illness diagnosis (22). In addition, AA individuals with a history of incarceration are twice more likely to have a diagnosis of current or lifetime PTSD compared to those without a history of incarceration (23). Despite such high rates of mental health disorders, particularly PTSD, among AAs with history of justice system involvement (JSI), there has been little

research to understand the mechanism in which JSI, early trauma exposure, mental health, and PTSD intersect.

Furthermore, the experience of incarceration and involvement with the justice system is unevenly distributed in the United States (US). AA comprise 13.3% of the general US population, yet they comprise 41% in males and 24% in females of the state or federal correctional institutional population (24). Individuals involved in the justice system are also from disadvantaged neighborhoods (15,25) and once they re-enter the community, individuals with mental illness suffer from difficulties in accessing mental health resources that are sensitive to their clinical needs and economic status. Thus, research of AA population with a previous history of involvement with correctional facilities may be particularly relevant when understanding mental health disorders.

Despite substantial evidence of psychopathology and high lifetime trauma exposure among incarcerated populations, limited research has been conducted on PTSD in the AA population. In one nationally representative sample of Black Americans, previously incarcerated individuals had higher rates of exposure to potentially traumatic experiences and of a diagnosis of both 12-month and lifetime PTSD (23). In a sample of majority AAs, both trauma history and PTSD was associated with incarceration history (26). While these studies provide support for the greater prevalence of PTSD in AA and previously incarcerated populations, further research is needed to understand the impact of timing of trauma and JSI, and the mechanisms by which PTSD develops in AA individuals with a history of involvement with the justice system.

1.3 Interpersonal trauma, non-interpersonal trauma, and PTSD

Epidemiologic studies report higher rates of PTSD following interpersonal, potentially traumatic events than non-interpersonal traumatic events (2,27,28). Interpersonal trauma may include intimate partner violence and childhood physical, sexual, or emotional abuse, where perpetrators are in caregiving or intimate relationships with the survivors. Furthermore, interpersonal trauma seem to have lasting impact; in addition to the direct threat it poses, interpersonal trauma has been associated with emotional and behavioral dysregulation and difficulties with relationships (29), and limits survivors' assumptions about the safety and predictability of the world (30,31). When comparing survivors of interpersonal trauma to survivors of non-interpersonal trauma, survivors of interpersonal trauma have greater PTSD symptoms, specifically greater intrusive memories and reminders of past trauma, and greater suppression of emotional responsivity (32). In addition, survivors of interpersonal trauma, compared to those of non-interpersonal trauma, demonstrate more severe PTSD symptoms in the early aftermath of trauma with persistent symptoms mainly involving fear-based symptoms (30). These studies indicate a need for early treatment of PTSD symptoms particularly for those who have experienced interpersonal trauma. Examining the differential impact of age of onset of trauma by interpersonal versus non-interpersonal traumas may provide further understanding of the developmental mechanisms and etiology of PTSD.

1.4 Developmental timing of trauma and criminal justice system involvement exposure on PTSD

An under-studied area of trauma exposure that may lead to future psychopathology is the developmental timing of trauma exposure. Sensitive periods for

psychopathology risk are specific periods of time during development when the plasticity of the brain is particularly high (33–35). Trauma and JSI during these times may especially heighten the risk for developing PTSD. Identifying such sensitive periods of risk factors for PTSD will be crucial in understanding the biological etiology of the development of PTSD. Such identification will also guide resources for psychological and public health interventions to target at stages in development with elevated risk.

Research that considers such developmental timing while defining maltreatment found that age of onset of maltreatment influence the etiology of mental health disorders (36,37). Only few research studies focused on the developmental timing of trauma exposure and the development of PTSD symptomatology or PTSD diagnosis and no consensus has yet emerged regarding whether earlier or later exposure is predictive of elevated risk of PTSD. In one study examining the relationship between developmental timing of trauma exposure with PTSD and major depression, findings indicated that individuals first exposed to trauma in early childhood had both depression and PTSD symptoms twice as high as those exposed to trauma in later developmental stages (38). Other retrospective studies found that children with PTSD report a lower age (ages 3 – 5) of first exposure to child abuse (39). Those who experience sexual assault or physical abuse prior to age 11 are at higher risk for PTSD (40). In contrast, one study found that children first exposed to trauma during adolescence of ages 13 through 18 are at greater risk for PTSD than their peers exposed at earlier ages (41). Multiple studies found no differences in risk for PTSD based on age at onset of first trauma (42–44). As indicated by the inconsistent research findings, there is no clear consensus of the developmental stage that most greatly impacts PTSD severity.

Among a nationally representative youth population who were recently involved in the justice system, early age of onset of trauma exposure was associated with higher PTSD symptoms in females than in males (45). This finding is consistent with findings in the general population. For sensitive periods of exposure to JSI, there is limited extant literature exists for the developmental timing of JSI as a risk factor for PTSD. Such mixed findings on relatively small subset of traumatic exposures, small clinical and convenience samples on the developmentally sensitive period of trauma exposure, and limited research on the timing of JSI provide only minimal, preliminary evidence for the sensitive periods for development of PTSD.

1.5 Emotion dysregulation as a mediator of trauma, incarceration, and PTSD

Emotion dysregulation (ED) may be defined as the inability to regulate intense, negative, shifting emotional states and their physiological responses, and it is studied as a trans-diagnostic risk factor for a number of psychiatric conditions, including PTSD (46–48). There is an interplay between emotional and cognitive operations that are involved in emotion regulation, which include selectively modifying situations with emotional significance, integrating information, and making decisions, and selecting behavioral responses (49). Emotional awareness and social cognition, key features of emotion regulation, are generally impaired in a number of psychiatric conditions (46,47).

Accumulation of research focuses on defining mechanisms of the development of PTSD from exposures to a traumatic event through one's emotion dysregulation. In a non-clinical sample, ED significantly mediated the relationship between exposure to traumas and the development of PTSD symptoms, especially among those associated with higher levels of betrayal (i.e trauma perpetrated by a close friend or family member)

than those with low levels of betrayal (50). In another sample of socio-economically disadvantaged and largely AA women, along with low social support and high exposure to adult interpersonal violence, ED mediated the direct effect between childhood abuse and PTSD symptoms (51). Furthermore, ED has been studied as a potential mediator between PTSD symptoms and dissociative symptoms (52), which involves disruptions in memory, identity, and perceptions, and occur in more symptomatic cases of PTSD.

There is limited research on the mechanism of ED and PTSD among incarcerated populations. In one sample of incarcerated women, findings indicated that women who had histories of childhood sexual abuse and sexual re-victimization in adulthood had significantly greater emotion regulation difficulties when compared to singly victimized and non-victimized women (53). From this, we can see that there may be an association between cumulative effect of trauma, incarceration, and emotion dysregulation. In another study of male incarcerated methamphetamine users, among those with greater PTSD symptom severity, those with lesser access to emotion regulation strategies reported more aggressive behaviors (54). This is consistent with theories of aggression where there may be potential mechanisms that connect violent recidivism, emotion regulation, and PTSD symptoms. Despite these studies, it is evident that there is minimal research to fully understand the mechanism of the timing dimensions of incarceration, emotion dysregulation, and PTSD symptom severity.

In consideration of the extant literature, emotion dysregulation may be a crucial construct to consider when implementing interventions with a focus on developing adaptive emotion regulation strategies to address PTSD symptoms. Difficulties with emotion regulation may be a key component in understanding the mechanism between

the age of onset of traumatic exposure, age of onset of involvement with the justice systems, and PTSD symptom severity.

1.6 Current study

The current study investigated whether developmentally sensitive timing of trauma exposure and JSI exposure are associated with PTSD diagnosis. We conducted secondary analyses of a cross-sectional study of highly trauma-exposed, civilian, AA adults in Atlanta, GA. The current study included an urban sample of non-Hispanic, AA adults with high rates of trauma exposure, JSI, PTSD symptoms, and ED symptoms, and addressed four main hypotheses. First, this study examined the differential odds of PTSD diagnosis by the age at onset of trauma exposure and by the age of onset of JSI (arrest, jail, and prison). Effect modification in the relationships between age of onset of trauma exposure with PTSD diagnosis and age of onset of JSI with PTSD diagnosis was examined by frequency of total lifetime trauma exposure. Second, the age at onset of trauma exposure was categorized as interpersonal and non-interpersonal trauma exposure and the relationship to PTSD diagnosis was examined. Third, the relationships between age at onset of trauma exposure and age at onset of JSI was examined with PTSD symptom severity. Finally, ED was examined as a potential mediator of the relationship between both age of onset of trauma and PTSD symptom severity and age of onset of JSI and PTSD severity.

Methods

2.1 Procedure

Participants were recruited from the Grady Trauma Project (GTP), an ongoing National Institute of Mental Health funded study of risk factors for the development of PTSD in a low socioeconomic and primarily AA urban population (19,48,55–57). The GTP sample consisted of 10,503 adults (ages 18 – 65). Participants were randomly approached in the waiting rooms of gynecology and primary care medical (non-psychiatric) clinics at Grady Memorial Hospital, a non-profit hospital that provides care to the socioeconomically disadvantaged populations in metropolitan Atlanta, Georgia. To be eligible to participate, potential participants needed to be between 18 and 65 years of age, not actively psychotic, and able and willing to provide informed consent. After signing the informed consent approved by Grady Health Care System Research Oversight Committee and the Institutional Review Board of Emory University, an initial interview was administered with questionnaires regarding trauma history, incarceration history, PTSD symptoms, and psychological variables. Trained research assistants administered this interview, which took 45 to 75 minutes to complete, in the clinics. Participants received $15 for participation. PTSD symptoms, trauma history, JSI history, and emotion dysregulation were measured during this initial interview.

2.2 Participants

Our analytic subsample was smaller than the larger study sample (N=10,503) because we selected only individuals with complete trauma and incarceration history data, and only individuals who self-reported as non-Hispanic, AA, which reduced the

heterogeneity associated with race, allowing us to more effectively control for confounding, and resulted in a sample of N=6,319 individuals. This group is under-represented in psychiatric research studies despite high rates of incarceration, trauma, and PTSD symptoms being previously observed in this population (58,57). Finally, individuals were excluded from analyses if they had discrepancies in responses between trauma exposure, age of onset of trauma, and frequency of trauma, as well as JSI exposure and age of onset of JSI.

The final analytic sample consisted of N=4,000 non-Hispanic, AA individuals, with 84.6% females and whose data were collected between 2006 and 2017. The participants had a mean age of 41 (SD = 13.9). The sample generally had low or low-to-medium income, with 50.3% coming from households with a monthly income of less than $1,000. Participants included hospital patients and those who were present in the waiting rooms for other reasons, such as accompanying a family member or friend that had an appointment. Almost all participants in the sample reported experiencing or witnessing at least one criterion A traumatic event (96.7% reported at least 1 criterion A trauma; n=131 reported no exposure to trauma; n=475 reported 1 criterion A trauma) and 89.6% of participants reported experiencing at least two types of traumatic events in their lifetime (n=3,394). A subset of participants reported having involvement with the justice system in their lifetime. Based on self-report data, 44.8% reported a lifetime history of arrest.

2.3 Measures

Participants' self-report of sociodemographic information used in the analyses included their sex, age, household monthly income, highest education obtained, and history of JSI. Clinical measurements used in the analyses included the Traumatic Events Inventory (TEI), the Modified PTSD Symptom Scale (MPSS), Emotion Dysregulation Scale (EDS) and Beck Depression Inventory (BDI).

2.3.1 *Traumatic Events Inventory* – The TEI is a 14-item interview for history of lifetime traumatic events, assessing both directly experiencing and witnessing traumatic events.(19) The frequency of being confronted with traumatic events was also assessed, where appropriate. Frequency of event occurrence on a scale ranging from 0 (unexposed) to 8 (greater than 20 times) and age of onset of exposure to an event (in years) was assessed for each traumatic event. Consistent with prior research (19,55), total level of trauma exposure was measured by a sum score reflecting the total number of different types of trauma. Eleven different traumatic events were measured and we had information about age at first onset. As done in previous research (38), we developed age categories for age of onset of trauma exposure: unexposed, early childhood (age 0 – 5 years), middle childhood (6 – 10 years), adolescence (11– 18 years), and adulthood (19+ years).

2.3.2 *History of justice system involvement* – The participant history of JSI was collected through a locally developed and previously published self-report measure (55). It assesses arrest and detention history of participants. Presence or absence of JSI in an individual's lifetime of having been arrested, been in jail, and been in prison were ascertained. For each JSI type, participants reported their age

(in years) at first experience. We used this data to develop three categories for age of onset of JSI: unexposed, juvenile (age 0 – 18 years), and adulthood (19+ years).

2.3.3 *Modified PTSD Symptom Scale* – PTSD symptoms over the prior two weeks were assessed with the 17-item MPSS, a psychometrically reliable scale with a range from 0 to 51 (59–62). Symptoms were rated on a 4-point scale that ranged from 0 ("Not at all") to 3 ("5 or more times per week"). This scale was designed to assess DSM-IV-TR criteria for PTSD (63). These symptoms included re-experiencing, avoidance, emotional numbing, and hyper-arousal. Participants were asked about these symptoms in general. Therefore, they do not reference a specific index trauma, yet reflect symptoms related to any of the traumas reported on the TEI. Symptoms were also summed to reflect overall symptom severity (19,58).

2.3.4 *Emotion Dysregulation Scale* – Domains of emotional experiencing (e.g., "Emotions overwhelm me"), cognition (e.g., "When I'm upset, everything feels like a disaster or crisis"), and behavior (e.g., "When my emotions are strong, I often make bad decisions") were assessed with the EDS, a 12-item, self-report scale (64) adapted from the clinician-rated Affect Regulation and Experience Q-Sort Questionnaire (65–67). The EDS items are scored on a 7-point Likert scale that ranged from 1 ("Not true at all") to 7 ("Very true"). Symptoms were totaled to reflect overall ED severity.

2.3.5 *Covariates* – All logistic regression models, linear regression models, and mediation analyses were adjusted for the following covariates: sex, age (categorical: 18–25; 26–35; 36–45; 46–55; 56–65), 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+), depressive symptoms. Depressive symptoms were measured using the BDI, a 21-item psychometrically validated and widely-used inventory of current depressive symptoms (68–71). The total BDI score was measured by a sum score to reflect overall symptom severity.

2.4 Statistical analysis

All analyses were conducted using SAS Version 9.4 (SAS Institute Inc., Cary, North Carolina). Covariates in all models were included on a priori reasoning rather than using stepwise selection and included previously listed variables, and total frequency of traumatic events. Interactions by the total frequency of traumatic events in all models were considered and included on a priori hypotheses that participants exposed at earlier ages also would have more frequent trauma exposure. We conducted collinearity assessment of all predictor variables and we found no evidence of collinearity, where all models had condition indices below 30 and variance decomposition proportions below 0.5. Baseline differences in characteristics between participants with and without PTSD were compared using t tests and χ^2 tests. We also examined the distribution of exposure to total, interpersonal, and non-interpersonal traumatic event and the distribution of exposure to JSI in the total sample and by age at onset to exposure. To determine whether participants exposed at earlier ages also had more frequent trauma exposure, we examined the percent of participants exposed to frequent trauma by reported age of onset of exposure.

Following these analyses, crude and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were obtained from a series of logistic regression models that estimated the likelihood of the age of onset to trauma (overall, interpersonal, non-interpersonal) or age of onset to JSI (been arrested, been in jail, been in prison) with PTSD diagnosis. Linear regression models were used to estimate the age of onset of trauma and the age of onset to JSI with current PTSD symptom severity (betas and 95% CIs were obtained).

To examine the role of emotion dysregulation as a potential mediator of the relationship between age of onset to trauma and age of onset to JSI with current PTSD symptom severity, we planned a mediation analysis with bootstrapping techniques using a macro developed by Preacher and Hayes (PROCESS; 71). This allowed us to generate 5,000 bootstrap samples to generate a 95% bias-corrected confidence interval of the indirect effect *a* x *b*. In our analysis, the *a* path represented the path from age of onset to trauma or from age of onset to a type of JSI to ED, and the *b* path represented the impact of the mediator, ED, on PTSD symptoms. Output from the macro also included path *c*, the direct impact of age of onset to trauma or age of onset to JSI on current PTSD symptoms, and *c*', the impact of age of onset to trauma or age of onset to JSI on PTSD symptoms when accounting for ED. The product *a* x *b* represents the indirect effect, and if the confidence interval for this indirect effect using bootstrapping methods does not include zero, the indirect effect is significant and mediation is established. This method does not assume normally distributed indirect effects, and for this and other reasons, it is more powerful and more accurate in testing mediation than the commonly used Sobel test and causal steps approach (73,74). Our hypothesis required a mediation analysis using

current PTSD symptoms as the target dependent variable and ED as the potential mediator. Of note, this mediation analysis cannot confirm causality because of the cross-sectional nature of the data (75).

Results

3.1 Sample

The analytic sample was comprised mostly of women (84.6%) and middle-age adults (mean age = 40.9; SD = 13.9). The prevalence of any involvement with the justice system was 39.3% among women and 75.0% among men. PTSD diagnosis differed significantly by categorical age, education, household monthly income, been arrested, been in jail, childhood trauma history, lifetime trauma history, depressive symptoms, and emotion dysregulation symptoms, but not by continuous age nor sex (Table 1). Specifically, PTSD diagnosis was more prevalent among middle-age adults, those with lower income, lower education, and with a history of being arrested and in jail. Furthermore, those with a PTSD diagnosis had greater childhood and lifetime trauma history, and greater depressive and emotion dysregulation symptoms than those without a PTSD diagnosis.

3.2 Prevalence of trauma exposure and trauma timing

On average, the sample reported having at least one childhood traumatic event (SD = 1.2) and 11.3 traumas in their lifetime (SD = 9.8). The mean age of onset of trauma exposure was 11.3 (SD = 8.9), but ranged between before one years of age through age 62. Overall, middle childhood (ages 6-10) was the most often reported time period for age of onset of trauma exposure, adolescence (ages 11-18) and adulthood (ages 19+) were the most often reported time periods for age of onset of interpersonal and non-interpersonal trauma, respectively.

3.3 Prevalence of criminal involvement exposure and criminal involvement timing

Overall, 44.8% of the sample had a history of being arrested. Among those who answered whether or not they had a history of being in jail (n = 2,510), nearly two-thirds reported having been in jail in their lifetime (63.3%), while of those who answered whether or not they had a history of being in prison (n = 2,503), 11.8% reported having been in prison in their lifetime. Age of onset of arrest, of incarceration, and of imprisonment, occurred, on average, during early adulthood (Table 1). However, the range of age of onset of arrest and incarceration was between ages 10 and 64 with 34.4% had an age of onset before or at age 18. The range of age of onset of imprisonment was between ages 13 and 65 with 14.2% had an age of onset before or at age 18.

3.4 Relationship between age of onset of trauma exposure and age of onset of criminal involvement with PTSD diagnosis

The adjusted odds of a PTSD diagnosis were greatest for individuals whose age of onset of a traumatic event was in their early childhood (ages 0 – 5) compared to those without any exposure (adjusted OR = 1.46, 95% CI = 0.50, 4.31). However, the odds of a PTSD diagnosis were greatest for individuals whose age of onset of a non-interpersonal trauma was in adolescence (ages 11-18) whereas the odds of a PTSD diagnosis was greatest for those whose age of onset of a interpersonal trauma in adulthood (ages 19+, Table 2). Further, the odds of having a PTSD diagnosis were increased for individuals whose age of onset of non-interpersonal trauma or to age of onset of interpersonal trauma was in adolescence and adulthood, respectively, however the odds were relatively similar and had largely overlapping confidence intervals (Table 2). The interaction between age

of onset of trauma and frequency of lifetime traumatic events on PTSD diagnosis was not statistically significant for overall trauma, non-interpersonal trauma, or interpersonal trauma.

When examining the relationship between age of onset of having been arrested, the adjusted odds of having a PTSD diagnosis were 1.3 times for those with a juvenile exposure relative to those without any exposure, whereas this relationship attenuated for those with adulthood age of onset (OR_{juv} (95% CI) = 1.3 (1.0, 1.6) vs. OR_{adult} (95% CI) = 1.1 (0.9, 1.4)). This relationship was similar for the relationship between age of onset of having been in jail and PTSD diagnosis (Table 2). Lastly, there was no statistically meaningful relationship between age of onset of having been in prison and PTSD diagnosis. The interaction between age of onset of JSI and frequency of lifetime traumatic events on PTSD diagnosis was not statistically significant for having been arrested, been in jail, or been in prison.

3.5 Relationship between age of onset of trauma and age of onset of criminal involvement with PTSD symptom severity

After adjusting for covariates and frequency of lifetime trauma, those whose age of onset to overall trauma was in middle childhood (ages 6 -10), relative to other onset age categories, had the highest PTSD symptoms. Also, they had PTSD symptoms 0.72 higher than those without any exposure to trauma (β (95% CI) = 0.72 (-2.88, 4.32), Table 3). However, this result was not statistically significant at the alpha level of 0.05. In addition, for non-interpersonal trauma, those whose age of onset was in early childhood (ages 0-5) relative to other onset age categories had the greatest increase in PTSD

symptoms of 1.15 (95% CI = -0.94, 3.24; Table 3). Whereas for interpersonal trauma, those whose age of onset was in adulthood relative to other onset age categories had greatest increase in symptoms of 1.09 (95% CI = 0.30, 1.88). The interaction between age of onset of trauma and frequency of lifetime traumatic events on PTSD symptom severity was not statistically significant for overall trauma or for interpersonal trauma, but was statistically significant for non-interpersonal trauma, suggesting that the relationship in PTSD symptom severity and age of onset of non-interpersonal trauma may differ by the number of traumatic events an individual has experienced.

The PTSD symptom severity score was 1.47 higher among those whose age of onset of been arrested or been in jail was juvenile than adulthood exposures in comparison to those without any history of such JSI (Table 3). PTSD symptom severity did not differ between the juvenile and adulthood onset age groups for those who served time in prison. The interaction between age of onset of JSI and frequency of lifetime traumatic events on PTSD symptom severity was not statistically significant for having been arrested, been in jail, or been in prison.

3.6 Mediation by emotion dysregulation

Bivariate correlations confirmed the relationship of emotion dysregulation with age of onset to overall trauma, age of onset to interpersonal trauma, age of onset of having been arrested, age of onset of having been in jail, and current PTSD symptoms, but not for age of onset of non-interpersonal trauma and age of onset of having been in prison. Thus, further hypothesized mediation analyses were continued only for age of

onset of overall trauma, of interpersonal trauma, of having been arrested, and of having been in jail.

The mediation models provided evidence supporting a mediation effect by emotion dysregulation on the relationship between age of onset of trauma and current PTSD symptoms for those whose age of onset to trauma was in middle childhood (ages 6 -10; Figure 1a). We found the mean indirect effect from the bootstrap analysis was positive and significant (a_2 x b = 0.47) for those with age of onset in middle childhood, (95% CI = 0.09, 0.94). The direct effect c (2.06) is not significant whereas the indirect effect c' (1.59) is statistically significant. These relationships did not hold at the alpha level of 0.05 for interpersonal trauma onset age categories.

In addition, mediation models provided evidence supporting a mediation effect by emotion dysregulation on the relationship between age of onset of JSI and current PTSD symptoms for those with juvenile first exposure (ages 0-18) to having been arrested and been in jail (Figure 1b & 1c). We found the mean indirect effects from the bootstrap analysis were positive and significant (arrest: a_1 x b = 0.20, jail: a_1 x b = 0.26) for those with juvenile age of onset, (95% CI: arrest = 0.05, 0.39; jail = 0.04, 0.52). The direct effects c (arrest = 1.34, jail = 1.15) are not significant whereas the indirect effects c' (arrest = 1.14, jail = 0.89) are statistically significant.

Discussion

The current study examined the association between the developmental timing of exposure to trauma and JSI with both PTSD diagnosis and PTSD symptom severity among a sample of highly trauma-exposed adults. Conducting this research among a racially and socio-economically homogeneous sample, where prevalence of trauma exposure and JSI were high, we were able to examine the consequences of age of onset of trauma and age of onset of JSI. Such analyses may not have been possible in a heterogeneous sample where trauma exposure and JSI were rare, or confounded by sociodemographic factors.

In concordance with previous research (76–79), we found that those who were exposed to trauma, regardless of age of onset of exposure, had significantly higher levels of PTSD diagnoses and symptoms relative to those never exposed to trauma. In addition, we found that those who had a history of JSI (also regardless of age of onset) had significantly higher levels of PTSD diagnoses and symptoms relative to those with no JSI, in line with previous research findings (20,21,23). These findings add further support to the literature that trauma exposure and JSI increase the risk for lifetime psychopathology.

There appeared to be variations in the developmental stages in which trauma affects PTSD diagnoses and PTSD symptom severity from our study findings. Overall, participants exposed to trauma in early childhood (ages 0–5) had greater odds of a PTSD diagnoses and participants exposed to trauma in middle childhood (ages 6-10) had greater PTSD symptom severity than those exposed during later developmental stages and to those non-exposed to trauma, after adjusting for sociodemographic characteristics,

depressive symptoms, and frequency of exposure to trauma. Such findings are consistent with findings that childhood trauma exposure may be more damaging than later trauma exposure because it hinders a child's ability to master skills pertinent to their developmental stages (47), and damages neurodevelopment involved in regulating arousal, emotion, stress responses, and reward processing (80–83). Such early impairments in development may serve as implications for the onset and persistence of stress-related psychopathology.

In addition, when separating trauma exposure types by either non-interpersonal and interpersonal traumas, participants first exposed to interpersonal trauma during adulthood (ages 19+) had 65% greater odds of PTSD diagnoses and greater PTSD symptom severity, after adjusting for sociodemographic characteristics, depressive symptoms, and frequency of exposure to trauma. Participants whose age of onset of non-interpersonal trauma was during adolescence (age 11-18) had greater odds of PTSD diagnoses, whereas, those whose age of onset of non-interpersonal trauma was during early childhood (ages 0-5) had slightly greater PTSD symptom severity, but was not statistically significant after adjusting for covariates. Although mechanisms driving these associations are unclear, exposure to interpersonal traumatic events during adulthood may be more relevant due to greater temporal proximity when considering current PTSD diagnoses and symptom severity.

Across analyses of all age of onset to JSI, juvenile age of onset of exposure (ages 0–18) to JSI had greater odds and greater symptom severity than those who were first criminally involved in adulthood (ages 19+). Our findings are relevant to the literature about the relationships of trauma and PTSD of justice-involved youth, where an earlier

age of the onset of trauma exposure are associated with greater PTSD symptoms, particularly for female justice-involved youth (45). Furthermore, both increased types and number of victimizations during childhood have been associated with greater prevalence of anxiety, depression, post-traumatic stress, suicide attempts and self-harm (84). Particularly among juvenile justice-involved youths, those who experience multiple types of childhood trauma victimizations had more severe emotional and behavioral problems, PTSD, and alcohol and drug use problems (85). Even among those on juvenile probations, who represent the majority of juveniles in the justice system, history of trauma and PTSD have been associated with further internalizing and externalizing problems, affective symptoms, anxiety, and conduct problems (86). Research findings all indicate that those with early trauma victimizations worsen psychopathology, especially for those who are involved with the juvenile justice systems. Our present findings, in addition to the extant literature, provide critical new information that early juvenile exposure than adulthood exposure to justice systems may worsen PTSD symptoms and increase the odds of a PTSD diagnosis.

To understand the mechanistic pathway between age of onset of trauma exposure and of JSI with PTSD, we examined emotion dysregulation as a potential mediator. Our findings that ED mediated the relationship between age of onset of trauma in middle childhood and PTSD symptom severity adds to a growing literature on ED as a mechanistic, trans-diagnostic risk factor to psychopathology from early trauma exposures (50–52). As previously mentioned, middle childhood (ages 6-10) may be a crucial stage in development where serious damages in neurodevelopment hinder regulation of arousal, emotion, stress responses, and reward processing (80–83) and may have potential

damages in lifetime psychopathology. Moreover, ED was a significant mediator between the relationship between juvenile age of onset of exposure to justice system justice systems and PTSD symptom severity, but not for adulthood age of onset of JSI. To our knowledge, our study is the first to examine a mechanistic pathway through ED between the age of onset of JSI and PTSD symptom severity. The current study provides new information that may guide further research in identifying critical developmental periods where trauma exposures and involvement with the justice systems may have detrimental effects in mental health of an already highly vulnerable populations.

4.1 Strengths and Limitations

It is important to emphasize that our findings on the developmental timing differences would have been missed had we not considered the effect of timing of trauma exposure and JSI. The basic comparison of those exposed to those who are unexposed may not identify the crucial within-group differences when examining developmental timing of exposure to trauma and JSI. Additionally, our findings also indicate the importance to co-vary for the frequency of exposure to trauma, as some developmental timing difference may be attenuated after considering this information.

Several limitations of the current study are worth noting. First, due to the cross-sectional nature of this study and the use of retrospective reports of trauma, JSI, PTSD symptoms and ED, we cannot make assertions about causality or time of onset for PTSD symptoms and ED. Future prospective, longitudinal studies are required to examine the timing dimension and to identify sensitive periods of trauma and JSI for prevention of ED and PTSD. In addition, it is possible that PTSD symptoms preceded the first exposure to

JSI, and therefore should serve as a predictor rather than an outcome variable. However, our measure of PTSD reflects general symptoms, not specifically related to JSI, and in this study, we were interested in understanding how reports of age of onset to JSI and ED symptoms may explain varying levels of PTSD symptoms and diagnoses in our population. This is preliminary evidence that needs to be followed up with longitudinal research, and we are currently conducting a longitudinal study of children and their mothers in this population to potentially answer such temporal and developmental questions.

Furthermore, due to using retrospective, self-reports for our exposures, mediator, and outcome variables, correlation of classification errors may have induced spurious, but significant relationships due to bias away from the null, towards the null, or even cross-over bias of our estimates (87). In addition, prison involvement among juveniles is rare in the US and due to use of self-reports, there is potential for misclassification of the types of juvenile JSI. To reduce such systematic errors in our estimates, use of separate sources of information on exposure and outcome is necessary. Due to the study being conducted as secondary analyses from a larger study of PTSD risk factors, such methods were not readily available. Replication of our study using public records for JSI and clinician-administered PTSD scales would provide non-correlated classifications and likely more accurate estimates.

This study's sample was largely low-income, female, and African American. Our analyses were also restricted to non-Hispanic, African Americans, due to the small number of respondents in other racial/ethnic groups. This weakness due to homogeneity of the sample and non-generalizability is counterbalanced by the public health

importance of studying these variables in an often under-researched and under-served population with high rates of trauma exposure, mass incarceration, and mental health problems. In particular, the Bureau of Justice Statistics reported that states in the South region of the US held the greatest number of individuals under the age of 18 in State prisons at midyear 2006 (88). Despite the high number of young adults held in the justice systems, there are minimal mental health resources available for these individuals, irrespective of the clear need for treatment options given the high rates of trauma, JSI, and trauma-related psychopathology. Thus, greater efforts are needed to understand and study factors that influence mental health in this population. This would allow us to recognize potential differences in the effectiveness of current evidence-based treatments in such groups.

4.2 Future Directions and Conclusion

Future longitudinal studies examining children over time are necessary to understand the early childhood trauma exposures, early JSI, and the critical periods in developmental stages that having lasting impacts on PTSD symptomatology and on trans-diagnostic risk factors, like ED. Furthermore, future studies examining the potential synergistic or protective effect of the combined exposure to trauma and involvement with the justice systems would provide a comprehensive understanding of the etiology of PTSD in this population. In conclusion, our study provides preliminary evidence that support the need for further investigation of developmental period of trauma exposure and JSI, as the effect of types of traumas varied by the onset age of trauma exposure and juvenile exposures to JSI increased PTSD severity. Identification of sensitive

developmental stages of heightened vulnerability will indicate specific target populations and guide the investment of limited and scarcely funded public health interventions, particularly in schools and juvenile justice systems.

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Tables

Table 1. Sociodemographic factors, history of justice system involvement, trauma history, and clinical symptoms overall and by PTSD status

	Overall (n = 4000)	No PTSD (n = 2760)	PTSD (n = 1240)	*P*-value
Sociodemographic factors				
Age (yrs)	40.9 ± 13.9	40.8 ± 14.4	41.1 ± 12.8	0.55
18 – 25 (%)	19.7	21.1	16.6	<.001
26 – 35	20.0	20.0	20.1	
36 – 45	16.3	15.3	18.6	
46 – 55	26.9	24.8	31.5	
56 – 65	17.1	18.8	13.2	
Female (%)	84.6	84.4	84.9	0.69
Education attainment (%)				<.001
Less than high school graduate	18.9	17.2	22.5	
High school graduate or GED	40.1	41.2	37.8	
More than high school	41.0	41.6	29.7	
Household monthly income (%)				<.001
$0 – 499	24.4	22.3	29.2	
$500 – 999	25.9	25.1	27.6	
$1000 or more	49.7	52.6	43.2	
History of justice system involvement				
Been arrested (n = 4000; %)	44.8	40.8	53.6	<.001
Age of onset of arrest (yrs)	23.8 ± 9.1	24.0 ± 9.1	23.5 ± 9.0	0.34
Been in jail (n = 2510; %)	63.3	59.7	70.5	<.001
Age of onset of incarceration (yrs)	24.4 ± 9.0	24.7 ± 9.1	24.1 ± 8.8	0.21
Been in prison (n = 2503; %)	11.8	11.6	12.3	0.60
Age of onset of imprisonment (yrs)	27.5 ± 9.0	27.7 ± 9.2	27.1 ± 8.5	0.58
Trauma history				
No. of childhood traumatic events	1.1 ± 1.2	0.9 ± 1.1	1.7 ± 1.3	<.001
No. of lifetime traumatic events	11.3 ± 9.8	9.2 ± 8.3	16.5 ± 11.0	<.001
Age of onset of traumatic event (yrs)	11.3 ± 8.9	12.3 ± 9.3	9.2 ± 7.3	<.001
Age of onset of interpersonal trauma (yrs)	17.5 ± 10.7	18.1 ± 11.0	16.4 ± 10.1	<.001
Age of onset of non-interpersonal trauma (yrs)	18.8 ± 12.2	19.3 ± 12.6	17.8 ± 11.5	<.001
Clinical symptoms				
Depression symptoms	14.4 ± 11.6	10.3 ± 9.1	23.6 ± 11.5	<.001
Emotion dysregulation (n = 2201)	38.1 ± 21.1	32.6 ± 18.8	50.9 ± 20.7	<.001
PTSD symptoms	12.8 ± 12.1	6.5 ± 6.8	26.9 ± 9.2	<.001

Values are mean ± SD or %.
Yrs = years, GED = general educational development, PTSD = post-traumatic stress disorder, and No. = number.

Table 2. Relationship between onset age of trauma and the onset age of justice system involvement with PTSD diagnosis, crude and adjusted for covariates and frequency of trauma

	Unadjusted OR (95% CI)	Adjusted OR* (95% CI)
Overall trauma		
Early childhood	1.10 (0.46, 2.59)	1.46 (0.50, 4.31)
Middle childhood	0.74 (0.32, 1.75)	1.39 (0.47, 4.10)
Adolescence	0.43 (0.18, 1.01)	1.37 (0.46, 4.07)
Adulthood	0.28 (0.67, 8.06)	1.36 (0.45, 4.16)
Non-interpersonal trauma		
Early childhood	2.41 (1.76, 3.31)	1.40 (0.94, 2.06)
Middle childhood	2.14 (1.70, 2.70)	1.21 (0.91, 1.61)
Adolescence	2.34 (1.88, 2.90)	1.60 (1.23, 2.08)
Adulthood	1.86 (1.51, 2.29)	1.53 (1.19, 1.97)
Interpersonal trauma		
Early childhood	3.80 (2.71, 5.32)	1.24 (0.80, 1.92)
Middle childhood	3.50 (2.78, 4.40)	1.48 (1.09, 1.99)
Adolescence	2.56 (2.09, 3.15)	1.50 (1.16, 1.95)
Adulthood	2.38 (1.92, 2.95)	1.65 (1.27, 2.15)
Justice system involvement		
Been arrested (n = 4000)		
Juvenile	1.87 (1.55, 2.26)	1.30 (1.02, 1.65)
Adulthood	1.59 (1.36, 1.85)	1.12 (0.94, 1.36)
Been in jail (n = 2510)		
Juvenile	1.77 (1.40, 2.23)	1.34 (1.00, 1.79)
Adulthood	1.55 (1.28, 1.88)	1.22 (0.97, 1.55)
Been in prison (n = 2503)		
Juvenile	1.01 (0.53, 1.93)	0.82 (0.37, 1.83)
Adulthood	1.08 (0.82, 1.42)	0.96 (0.67, 1.37)

* Adjusted for sex, categorical age, highest education attained, household monthly income, depression symptom severity, and lifetime total trauma frequency.

OR = odds ratio. CI = confidence interval.

Table 3. Relationship between onset age of trauma and the effect of onset age of justice system involvement with PTSD symptom severity, adjusted beta estimates

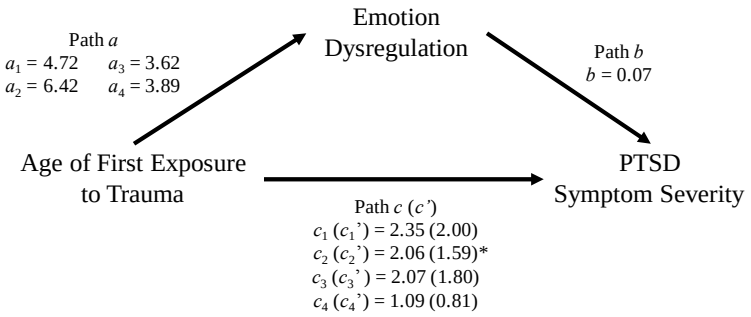
	Adjusted beta* (95% CI)	*P*-value of exposure x frequency of trauma interaction
Overall trauma		0.34
Early Childhood	0.67 (-2.94, 4.28)	
Middle Childhood	0.72 (-2.88, 4.32)	
Adolescence	0.30 (-3.33, 3.93)	
Adulthood	-0.33 (-4.01, 3.36)	
Non-interpersonal trauma		0.03
Early Childhood	1.15 (-0.94, 3.24)	
Middle Childhood	0.51 (-0.85, 1.87)	
Adolescence	0.26 (-0.95, 1.46)	
Adulthood	0.49 (-0.62, 1.58)	
Interpersonal trauma		0.75
Early Childhood	-0.61 (-2.06, 0.85)	
Middle Childhood	0.80 (-0.15, 1.74)	
Adolescence	1.00 (0.22, 1.78)	
Adulthood	1.09 (0.30, 1.88)	
Justice system involvement		
Been arrested (n = 4000)		0.15
Juvenile	1.47 (0.66, 2.28)	
Adulthood	0.78 (0.15, 1.41)	
Been in jail (n = 2510)		0.35
Juvenile	1.47 (0.45, 2.49)	
Adulthood	0.96 (0.16, 1.77)	
Been in prison (n = 2503)		0.46
Juvenile	0.01 (-2.76, 2.79)	
Adulthood	0.24 (-0.99, 1.47)	

* Adjusted for sex, categorical age, highest education attained, household monthly income, depression symptom severity, and lifetime total trauma frequency.

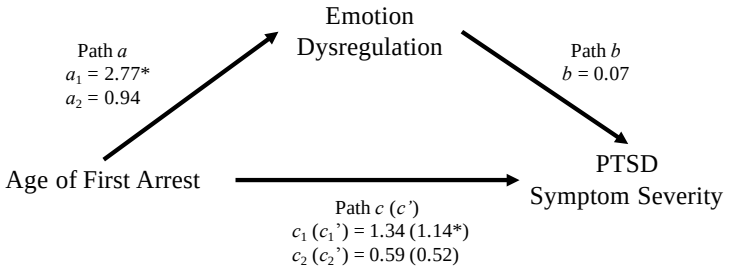
CI = confidence interval.

Figures

a.



b.



c.

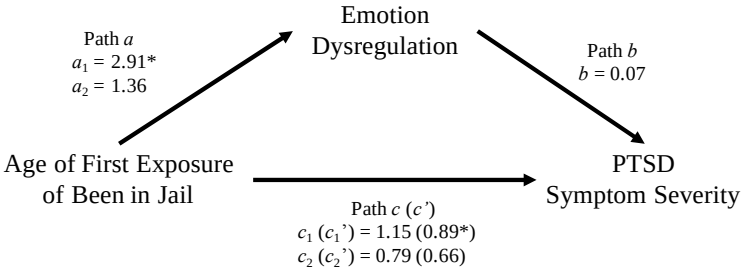


Figure 1. Mediation of relationship between onset age of trauma and justice system involvement with PTSD symptom severity through emotion dysregulation. Unstandardized coefficients are reported for each path. Path c' is the effect of onset age of exposure on current PTSD symptom severity while accounting for emotion dysregulation. (a.) Mediation through emotion dysregulation was statistically significant only for those who had age of onset of trauma during middle childhood (ages 6-10). Subscripts in estimates indicate age of onset of trauma categories: 1 = early childhood, 2 = middle childhood, 3 = adolescence, and 4 = adulthood. (b. & c.) Mediation through emotion dysregulation was statistically significant only for those whose age of onset of been arrested and been in jail were juvenile exposures (ages 0-18). Subscripts in estimates indicate age of onset of justice system involvement categories: 1 = juvenile, and 2 = adulthood.