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Grace McMillen April 7, 2020

Distinctive impacts of sexual trauma versus non-sexual trauma on PTSD profiles in highly trauma-exposed, African American women

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Abstract

Distinctive impacts of sexual trauma versus non-sexual trauma on PTSD profiles in highly trauma-exposed, African American women

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Prior findings suggest that posttraumatic psychopathology following assaultive and interpersonal violence like sexual assault and abuse may be more severe and present differently from prototypical PTSD. We predicted that sexual trauma (ST) exposure may result in higher symptoms of numbing and avoidance and blunted fear responses, relative to individuals who experienced non-sexual trauma (NST). We also predicted that childhood sexual abuse (CSA) would result in a more blunted affective profile than adulthood sexual assault (ASA). Participants were n=5134 African American women recruited through the Grady Trauma Project from an urban hospital serving a primarily lower-income population in Atlanta, GA. We investigated the unique effects of ST on symptoms assessed using the modified PTSD Symptom Scale, fear-potentiated startle (FPS) response in a subset of n=286, and amygdala reactivity and habituation to social threat cues using fMRI in n=95. ST was associated with greater PTSD symptoms (F(5159)=90.6, p=2.64e–21), but this was observed across all symptom clusters. Both CSA and ASA, but not CSA alone, was associated with greater PTSD symptoms (F(2,2220)=6.989, p=9.43e–04) and greater avoidance and numbing symptoms (F(2,2220)=8.530, p=2.04e–04). In the FPS paradigm, the ST group startled more on all blocks and stimulus types than the NST group (F(1,1684)=7.19, p=0.007). Timing of sexual trauma did not have a significant effect on startle magnitude (F(2,964)=1.99, p=0.138). ST was not associated with any unique patterns of amygdala habituation or reactivity to social threat cues. We conclude that while survivors of sexual traumas may present with more severe PTSD symptoms, their profiles are not characteristically different from similar NST controls. Experiencing CSA alone did not result in a symptom profile unique from experiencing ASA alone. The pattern of symptoms, psychophysiology and neuroimaging responses observed do not suggest an affectively-blunted subtype of PTSD in survivors of sexual trauma. Implications for treatment and suggestions for future studies are discussed.

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Introduction

It is estimated that 1 in 4 women in the U.S. will experience rape or sexual assault in her lifetime (Elliott et al., 2004). Assaultive violence is associated with the highest rates of posttraumatic stress disorder (PTSD) of all types of traumatic events (Gillespie et al., 2009; Kessler et al., 1995). Despite this high incidence rate, there remain many questions regarding the underlying neurophysiological effects and resulting symptomology of sexual trauma. Prior research suggests that PTSD arising from an interpersonal trauma (including sexual assault or abuse) may result in an increase in dysphoria-related symptoms such as avoidance and numbing. In contrast to this, the more traditional conceptualization of PTSD is of hyperarousal and re-experiencing, especially as experienced by combat veterans or as a result of serious traumatic injury (Admon et al., 2013). However, the current literature on sexual assault has typically either grouped it with other forms of interpersonal trauma or assaultive violence (e.g., Breslau et al., 2005; Forbes et al., 2011) or has looked at sexual trauma on its own, without comparing it to other types of traumas at all (e.g., Bennice et al., 2003). This lack of adequate comparison leaves unanswered the question of the extent to which survivors of sexual traumas versus non-sexual traumas differ in their symptom patterns as well as underlying neurobiology and psychophysiology. It is also unclear whether these differences depend on developmental timing of exposure to trauma, e.g., experiencing sexual abuse in childhood versus sexual assault in adulthood. The current study aims to further the literature on sexual trauma and its effect on posttraumatic stress psychopathology by comparing survivors of adulthood-only sexual assault, childhood-only sexual abuse, and of both to survivors of non-sexual traumas with similar overall trauma loads.

Sexual assault and abuse can be defined in several ways, but it is especially helpful to consider how survivors and their networks define them, as this working definition may be more pertinent to those survivors than a legal one. RAINN (Rape, Abuse & Incest National Network), the country's largest anti-sexual violence organization, defines sexual assault as "sexual contact or behavior that occurs without explicit consent of the victim." RAINN defines child sexual abuse as any sexual activity with a minor, who cannot consent to any form of sexual activity. Notably, there does not need to be direct physical contact between the perpetrator and the child to be considered sexual abuse. As previously stated, survivors of sexual assault or abuse often experience psychopathology. Commonly experienced is PTSD, an anxiety disorder resulting from exposure to a traumatic event including actual or threatened death, serious injury, or sexual violence. It is characterized by symptoms of intrusion and re-experiencing the traumatic event, purposeful avoidance of distressing reminders of the event, negative alterations in cognition or mood, and marked alterations in arousal and reactivity. PTSD can be diagnosed as soon as three months after a trauma, and symptoms must last for at least one month and cause significant distress and impairment in daily functioning to be diagnosed (American Psychiatric Association, 2013). The disorder is often chronic and persists for many years after the trauma occurs; a subset of people with PTSD never recover, even after receiving treatment (Kessler et al., 1995).

PTSD following interpersonal traumas like sexual assault and abuse typically results in higher rates of symptoms in general and across all symptom clusters (Forbes et al., 2011; Norris, 1992a). In addition, survivors of assaultive violence versus non-assaultive traumas experience greater numbing and avoidance symptoms in particular (Chung & Breslau, 2008). These include avoidance of distressing memories, thoughts, or feelings about or closely related to the traumatic event, avoidance of external reminders (people, places, conversations, activities, objects,

situations) that arouse distressing memories, markedly diminished interest or participation in significant activities, feelings of detachment from others, and persistent inability to experience positive emotions (American Psychiatric Association, 2013). The separation between numbing and avoidance symptoms, new to this edition of the DSM, is supported by evidence that numbing and effortful avoidance are separate, distinct phenomena that are underlaid by different mechanisms. However, they often appear together and are functionally similar in that they provide an escape from painful thoughts or memories (Foa et al., 1995), so recent research has often grouped the two together.

Recent literature supports the existence of a subpopulation of individuals with PTSD who display markedly higher symptoms of numbing and avoidance than other individuals with PTSD (Chung & Breslau, 2008; Forbes et al., 2011). This profile may be the result of the individual's attempt to cope with the persistent fear and arousal experienced in chronic PTSD, as well as repeated traumas or a perpetually dangerous environment (Foa et al., 1989; Forbes et al., 2011). Survivors of interpersonal traumas appear especially likely to present with these increased symptoms of numbing and avoidance (Breslau et al., 2005; Chung & Breslau, 2008). For example, in one study, at 24 months post-trauma, restriction of affect was the only dysphoria-related symptom experienced at significantly higher rates in survivors of interpersonal traumas than in survivors of non-interpersonal traumas (Forbes et al., 2011). In a study that distinguished between three discrete classes of PTSD that varied by severity as well as qualitatively, Breslau and colleagues (2005) found that the group with the most severe PTSD was differentiated by emotional numbing symptoms. In their follow-up study, survivors of assaultive violence were more likely to report emotional numbing symptoms than survivors of non-assaultive violence, with the largest differences in detachment and restriction of affect (Chung & Breslau, 2008).

These findings are especially important as numbing may represent an early robust predictor of chronic PTSD. Among women who were sexually or physically assaulted, one study found that initial emotional numbing remained predictive of PTSD severity at three months post-trauma even after controlling for dissociation and depression, which are closely related phenomena. Furthermore, numbing was even more predictive of chronic PTSD than depression (Feeny et al., 2000). Together, symptoms of numbing and avoidance may sustain PTSD by preventing survivors from sufficiently processing the trauma and therefore inhibiting the processes that lead to recovery (Foa et al., 1995). It remains vitally important to understand the mechanisms of numbing and avoidance symptoms and how they sustain PTSD in survivors of interpersonal traumas.

Some of the mechanisms underlying these dysphoria-related symptoms are brain regions involved in emotional regulation. The amygdala, a key component in fear circuitry and for regulating emotion and arousal (Phelps & LeDoux, 2005; Shin & Liberzon, 2010), is typically hyperactive in response to emotional stimuli in individuals with PTSD (Etkin & Wager, 2007; Stevens et al., 2017). This hyperactivation is accompanied by heightened responsivity in the dorsal Anterior Cingulate Cortex (dACC) and decreased activity in the prefrontal cortex, especially the ventromedial prefrontal cortex (vmPFC) (Admon et al., 2013). However, in correspondence with certain symptom presentations, there appears to be a subset of individuals with PTSD who show hypoactivity rather than hyperactivity in the amygdala. In particular, recent literature has been interested with a dissociative subtype of PTSD, in which individuals overmodulate their emotions and dissociate, becoming emotionally and physiologically numb, in response to reminders of traumatic events or other emotional stimuli unrelated to the traumatic event (Hopper et al., 2007; Lanius et al., 2002; Seligowski et al., 2019). These individuals have

decreased activity in the amygdala in response to script-driven imagery and increased activity in the medial prefrontal cortex (Lanius et al., 2002, 2010). This pattern of low amygdala activity and high prefrontal activity is present in individuals with PTSD and high numbing and avoidance symptoms, even in the absence of dissociation (Felmingham et al., 2014). This hyper-inhibition of the emotional regulation networks may be a response to cope with extreme arousal by dissociating, becoming numb, or using passive avoidant strategies (Felmingham et al., 2008). Importantly, among PTSD patients without dissociation but with high emotional numbing symptoms, a similar pattern of decreased amygdala and ventral striatal activation was observed when they viewed happy faces, suggesting an overall blunted emotional response to many different types of stimuli that is not limited to reminders of traumatic events (Felmingham et al., 2014). The existence of a subset of individuals with anxiety disorders who show hypoactivity in the amygdala appears to be unique to PTSD; this was not found in social anxiety or specific phobia disorders (Etkin & Wager, 2007).

Typically appearing alongside heightened amygdala activity in PTSD is an exaggerated startle response and decreased ability to extinguish fear in response to safety cues in a fear-potentiated startle (FPS) paradigm, especially in individuals with more severe PTSD (Jovanovic et al., 2009). However, this exaggerated startle response is not found in all individuals with PTSD, and the emotionally numb group of individuals with the disorder may represent a population whose physiological reactivity is blunted rather than exaggerated (McTeague et al., 2010). Furthermore, magnitude of startle response may be distinguished by trauma history. In one study, PTSD patients who had experienced multiple traumas rather than a single, discrete trauma showed defensive reactivity with lower startle potentiation than the single-trauma participants. Those with multiple traumas also reported greater overall distress and lengthier

chronicity of pathology, all of which seem to contribute to or be underlaid by the blunted psychophysiological response (McTeague et al., 2010). Similarly, a review found that patients with more severe anxiety disorders showed a more blunted fear-potentiated startle response than those with less severe disorders, and that this less robust startle corresponded with lengthier chronicity (Lang & McTeague, 2009). Another reported that PTSD patients had hyporeactive physiology to fear imagery, paired with more severe pathology overall (Cuthbert et al., 2003). Together, these findings suggest a kind of learned helplessness response in which participants with ongoing or repeated and severe traumas learn to blunt their physiological reactions to fear and employ defensive physiological reflexes.

Developmental timing of trauma may also impact symptom presentation and neurophysiological responses. However, there is insufficient understanding about the specific ramifications of experiencing either childhood sexual abuse (CSA) or adulthood sexual assault (ASA) or a combination of the two in terms of later PTSD presentation. What is well-documented in research are the long-term impacts of CSA, both in terms of psychological distress and subsequent assault experiences as adults. A study in which one-third of women reported CSA found that it was associated with elevated rates on all ten clinical scales of the Trauma Symptom Inventory, even after controlling for interpersonal revictimization as an adult and physical (as opposed to sexual) abuse as a child (Briere & Elliott, 2003). In terms of revictimization, Filipas & Ullman (2006) found that maladaptive coping strategies were reliable predictors of revictimization in adulthood above and beyond CSA severity, PTSD symptoms, and self-blame, suggesting that the ways in which individuals cope with abuse experiences have important consequences later in life. Rosenthal and colleagues (2005) found that women who experienced sexual abuse before the age of 14 and reported avoidance as their main method of

coping with unpleasant thoughts reported higher levels of trauma-related distress later in life. In addition, a study of ethnic minority women found that more severe CSA was associated with greatest symptoms in the avoidance and numbing cluster (Glover et al., 2010). It may be that victims of severe CSA are more likely to employ avoidant coping strategies in order to escape distressing and repeated experiences and memories, which in turn leads to more psychological distress that continues into adulthood and sustains posttraumatic stress pathology.

In evaluating later psychopathology of childhood sexual abuse survivors, research is generally conclusive that family environment is an important factor to consider; however, findings regarding the effects of different family environments are mixed. For example, Nash and colleagues (1993) conclude that perceived family environment is an important mediator of adult psychopathology in child abuse victims, and that subsequent impairment in these adults is likely due in part to the context in which the abuse occurred, i.e., in a generally pathogenic household. On the other hand, Molnar and colleagues (2001) suggest that CSA occurring in otherwise healthily functioning families may result in more profound difficulties for the abuse survivors, as it often represents a significant betrayal by trusted adults. However, it is more generally found that unstable, high-conflict, and controlling family environments are associated with greater distress and more severe psychopathology in survivors of CSA (Koverola et al., 1996; Fassler et al., 2005). This conclusion also has important implications for resilience in survivors of childhood sexual abuse; if dysfunctional family environments are associated with worse outcomes, then functional, supportive, and stable family environments may act as a buffer against the adverse effects of CSA. In particular, a recent meta-analysis of 37 studies of adult CSA survivors found that support from family, along with the wider social environment, was the strongest protective factor associated with resilience following childhood sexual abuse

experiences (Domhardt et al., 2015). Therefore, in order to understand the full picture of how CSA may contribute to later psychopathology, it is important to consider the context in which abuse occurred.

There are certain populations that are more vulnerable both to experiencing traumatic events like sexual victimization and to developing psychopathology as a result. Prior research suggests that minority populations, especially African Americans, are at increased risk for both trauma exposure and posttraumatic stress pathology. These rates are particularly high in populations of African Americans who are economically disadvantaged and living in urban settings (Breslau et al., 1998). In general, rates of sexual assault in African Americans are disproportionately high: 2.5 per 1,000 versus 0.5 per 1,000 in White populations (Alim et al., 2006). In our prior work in a publicly funded hospital serving primarily lower-income African American families, we found that nearly 90% of all respondents experienced some form of significant trauma in their lives, with a lifetime prevalence of PTSD being 46% (Gillespie et al., 2009). This is significantly higher than the U.S. general population's lifetime prevalence of PTSD, which is between 5 and 10% (Kessler et al., 1995). Concerning socioeconomic status, Gillespie and colleagues (2009) reported a mean monthly household income of $2,000 for 88% of the sample, an income that is just above the national poverty level for a family of four in that year. 88% of the population in this sample endorsed some form of significant trauma in their lifetime, including adult sexual assault in 13% of females and childhood sexual abuse in 24%. Interpersonal traumas (including ASA) had the largest impact on PTSD symptom severity, which is consistent with previous research (Kessler et al., 1995; Rothbaum et al., 1992). Known historical and systemic factors also contribute to these high rates of trauma, leading to mental health consequences that are disproportionate, relative to other populations.

The current study aimed to parse apart symptom profiles as well as psychophysiological and neurological differences in survivors of sexual trauma(s) from survivors with similar trauma histories but without sexual trauma. We investigated the relationship between trauma type and PTSD symptom severity and cluster patterns using a large sample of African American females in a highly trauma-exposed, low socioeconomic-status urban hospital setting. We also investigated associations between presence of sexual assault and neuroimaging data of amygdala reactivity, as well as startle response in a fear-potentiated startle paradigm. Based on previous findings of increased emotional numbing, avoidance, and dissociation symptoms in victims of interpersonal trauma (Forbes et al., 2011; Chung & Breslau, 2008), we hypothesized that survivors of sexual trauma would experience symptoms of avoidance and numbing at higher rates and with greater severity than their non-sexually assaulted counterparts. Secondly, building upon research relating overmodulation of arousal responses and defensive reactivity to numbing symptoms in survivors of repeated trauma (Etkin & Wager, 2007; Lanius et al., 2010; McTeague et al., 2010), we hypothesized that among survivors of sexual trauma, we would observe primarily hypoactivity rather than hyperactivity in the amygdala in response to fearful and neutral face stimuli as well as blunted rather than exaggerated startle response in a fear-potentiated startle paradigm. A related exploratory hypothesis is similar: we hypothesized that survivors of childhood sexual abuse would experience numbing and avoidance symptoms at even higher rates than survivors of adulthood-only sexual assault. We also predicted even more blunted reactivity in the amygdala and even more blunted startle responses in survivors of CSA.

Methods

Participants

6,535 African American women between the ages of 18 and 65 were recruited from a larger ongoing study of risk factors for PTSD. Participants were approached by study staff in outpatient clinics of Grady Memorial Hospital, a public inner-city hospital that serves a primarily African American, low-income population in Atlanta, GA. Only individuals who self-reported as African American or Black were included in the study, both because of the existing racial makeup of the hospital (>90% Black or African American; Gillespie et al., 2009) and to improve data homogeneity. Participants were excluded if any of the following criteria were present, based on self-report: history of psychiatric or neurological disorder, psychosis or current psychotropic medication, metal clips or implants, history of head injury, and occurrence of loss of consciousness exceeding five minutes. Participants provided a urine sample 24 hours before the MRI scan to screen for pregnancy and use of illegal drugs. Participants were English-speaking and completed written informed consent forms before enrolling in the study; compensation was provided. All study procedures were approved by the institutional review boards of Emory University and Grady Memorial Hospital.

From the initial dataset including 6,535 women, 5,134 remained after cleaning the dataset for participants missing critical data on childhood or adulthood sexual trauma exposure or PTSD symptom severity. The final sample for fear-potentiated startle (FPS) analysis included 286 women and for fMRI analysis included 95 women. The mean age of the women was 39 years (*SD*=14); demographics and clinical characteristics are shown in **Table 1**. *N*=1,572 women met criteria for PTSD diagnosis, representing 30% of our overall sample. The mean number of total traumas experienced (indexed by the modified PTSD Symptom Scale) was 31.1 (*SD*=21), and the mean number of types of trauma experienced was 4.1 (*SD*=3).

Measures

Trained interviewers collected demographic information and administered an extensive array of psychological measures. This study included the demographic information on sex, age, race, ethnicity, education, and household monthly income. Though more psychological assessments than what is included here were administered, this study used only the data from the following:

The modified PTSD Symptom Scale (PSS) (Foa & Tolin, 2000) was used to assess the frequency and severity of PTSD symptoms. This measure has 17 items that ask participants to indicate the frequency of symptoms experienced over the past two weeks, from 0 ("not at all/only once") to 3 ("5 or more times a week/almost always"). Symptoms are divided into four clusters in correspondence with DSM-5 criteria: intrusion/re-experiencing (e.g., *experience flashbacks*), avoidance (e.g., *avoid activities/situations/places related to the event*), numbing (e.g., *impaired range of emotions*), and hyperarousal (e.g., *jumpier, easily startled*). The current study combined the avoidance and numbing cluster in our analyses. A final question asks participants to rate the length of their symptoms, from 0 ("less than one month") to 3 ("greater than 1 year"). The PSS has shown strong internal consistency reliability (Cronbach's α=0.92) in this sample (Dunn et al., 2017). The total score is calculated by averaging individual items and multiplying the average by 17, yielding a max score of 51. Total number of avoidance symptoms and numbing symptoms were the primary variables of interest in this study.

The Childhood Trauma Questionnaire (CTQ) (Bernstein et al., 2003) is a 28-item screen that assesses five categories of maltreatment occurring in childhood: physical abuse, physical neglect, emotional abuse, emotional neglect, and sexual abuse, occurring from birth until the age of 17 years. In a previous study with this sample, Cronbach's αs were 0.83, 0.86, and 0.95 for physical abuse, emotional abuse, and sexual abuse, respectively (Mandavia et al., 2016). Rather

than endorsing the presence or absence of items, participants rank the extent to which they believe the statements to be true about their experiences as a child and teenager, ranging from 1 (“never true”) to 5 (“always true”). The measure yields a total score for childhood trauma load as well as subscores for each of the five categories of maltreatment. In this study, scores were used to identify participants who had experienced childhood sexual abuse (CSA) alone or in combination with adulthood sexual assault.

Lifetime trauma history was assessed using the Traumatic Events Inventory (TEI) (Schwartz et al., 2005). This 15-item screen asks about any traumatic or stressful events that the participants may have experienced, witnessed, or been confronted with in their lifetime, as well as age of first exposure and frequency of exposure. Trauma types queried include natural disasters, serious accidents or injury, military combat, being confronted with the murder of a close friend or family member, being attacked with and without a weapon or witnessing a close friend or family member being attacked with and without a weapon, witnessing violence between parents or caregivers as a child, being beaten or physically punished, verbally abused, or sexually abused as a child, and being raped or sexually assaulted as an adult. An open-ended question also queries any other events that may have been traumatic or particularly stressful for the participant. The total number of different types of traumas experienced or witnessed in adulthood and childhood was used as an index of total trauma load in the current study. Questions related to sexual trauma exposure were used to divide participants into those who had experienced sexual abuse before the age of 18 (CSA) and those who first experienced sexual assault after the age of 18 (ASA).

The Beck Depression Inventory (BDI) (Beck et al., 1996) is a common and well-validated 21-item instrument that evaluates current depressive symptoms and severity. In this

sample, Cronbach's αs for internal consistency have ranged from .89 to .93 (Booker et al., 2018). Participants endorse the extent to which they feel statements describe how they have been feeling during the past two weeks on a 4-point scale (sample item: 0="I get as much pleasure as I ever did from the things I enjoy" to 3="I can't get any pleasure from the things I used to enjoy"). Scores are totaled and depression symptom severity is considered minimal (scores of 0 to 9), mild (10 to 18), moderate (19 to 29), or severe (30 to 63).

The Clinical Data Form (CDF) is a 17-item measure that assesses a wide range of information from childhood family experiences to having been placed in a juvenile corrections facility (Harris et al., 2019). To pursue the link between childhood family environment and symptomology following childhood sexual abuse, the present study included in our analysis the answer to the questions "How stable was your family environment growing up?" and "How warm and nurturing was your family when you were growing up?" both of which range on a 5-point Likert scale from 1 (very unstable or cold/unpleasant) to 5 (very stable or warm/nurturing).

The State Trait Anxiety Inventory Form Y (STAI) is a widely-used and well-validated measure that was used to assess state (current; dynamic) and trait (over time; stable) anxiety in participants prior to the fMRI scan in this study (Spielberger et al., 1983; Spielberger, 1989). The trait anxiety portion of the questionnaire was administered once, prior to the scan, and includes items like "I feel that difficulties are piling up so that I cannot overcome them" and "I am a steady person," ranked on a 4-point Likert scale from "Almost never" to "Almost always." The state anxiety portion was administered once prior to the scan and again after the scan; items include "I feel frightened," "I am jittery," and "I feel calm." State anxiety items are scored on a 4-point Likert scale from "Not at all" to "Very much so."

MRI Procedures

The description of the neuroimaging procedure is extracted directly from Stevens et al., 2013 (p 1470). Eight fearful and eight neutral (4 male and 4 female) faces were selected from the stimulus set of Ekman and Friesen (1976). Stimuli were projected onto a 24-inch screen at a resolution of 1280 x 1024 using EPrime 2.0 software (Psychology Software Tools, Pittsburgh, PA). Blocks of fearful and neutral stimuli (15 blocks each) were presented in a pseudorandom order. Each block was composed of all eight faces presented in a random order. Each face stimulus was presented for 500 ms, followed by a 500 ms presentation of a fixation cross. After every 10th block, a 10,000 ms rest period with the instruction "relax and look at the screen" was presented. Face stimuli were presented at a size of 4.3 x 6.7" on a black background, and the fixation cross and instructions were presented in white 18-point Courier New font on a black background. Participants were instructed to pay attention to the faces, but not to make any behavioral response, in order to minimize motion artifacts and neural activation unrelated to processing the visual stimulus.

MRI Acquisition and Analysis

The description of the brain imaging acquisition is extracted directly from Kim et al., 2019 (p 649) and also follows that of Stevens et al. (2013). Preprocessing was conducted in SPM8 software (IBM Corp. Armonk, NY) and followed methods detailed in Kilaru et al. (2016). In SPM8, correction for slice timing and spatial realignment was applied to the images. Then the images were normalized with unified segmentation, and smoothed with an 8 mm Gaussian kernel. The six realignment parameters were included in the subject-level design matrix as covariates to correct for head motion. Amygdala responses were extracted from a bilateral region of interest (ROI) created using the probability mask from the SPM Anatomy Toolbox, at a threshold of 50% (Eickhoff et al., 2005).

Fear-Potentiated Startle Paradigm

The description of the fear-potentiated startle assessment is extracted directly from Jovanovic et al., 2013 (p 3). Startle response data were acquired at a 1000 Hz sampling frequency using the electromyography (EMG) module of the BIOPAC MP150 for Windows (Biopac Systems, Inc., Aero Camino, CA). The acquired data were filtered, rectified, and smoothed using the MindWare software suite (MindWare Technologies, Ltd., Gahanna, OH) and exported for statistical analyses. The EMG signal was filtered with low- and high-frequency cutoffs at 28 and 500 Hz, respectively. The maximum amplitude of the eyeblink muscle contraction 20e200 msec after presentation of the startle probe was used as a measure of the acoustic startle response.

The eyeblink component of the acoustic startle response was measured by EMG recordings of the right orbicularis oculi muscle using two 5-mm Ag/AgCl electrodes filled with electrolyte gel. One electrode was positioned 1 cm below the pupil of the right eye and the other was placed 1 cm below the lateral canthus. Impedance levels were less than 6 kilo-ohms for each participant. The startle probe was a 108-dB (A) sound pressure level (SPL), 40-msec burst of broadband noise with near instantaneous rise time, delivered binaurally through headphones.

The fear-potentiated startle paradigm was comprised of a Fear Acquisition and a Fear Extinction phase. In the Fear Acquisition phase, three blocks with four trials of each type were presented. The three blocks were a reinforced conditioned stimulus (CS+), a nonreinforced conditioned stimulus (CS–), and the 40 msec noise probe by itself (NA). The unconditioned stimulus (US) was a 250 msec burst of air with 140 psi. intensity directed at the participant's throat; in our previous research, this US has generated a strong fear-potentiated startle (Norrholm et al., 2011). Both conditioned stimuli were colored shapes that were shown for 6 seconds on the

computer screen. Inter-trial intervals were randomized to be between 9 and 22 seconds. The Fear Extinction phase began 10 minutes after the Fear Acquisition phase and involved six blocks of the four trials (both CSs and the NA from the Fear Acquisition phase). During this phase, the CSs were never presented with the US air blast.

Assessment of fear-potentiated startle followed that of our earlier work (Glover et al., 2012; Jovanovic et al., 2013). Presence of startle was calculated by comparing startle magnitude on the CS+/– trials to the noise alone (NA) trial. We assessed fear acquisition as a difference score by subtracting startle magnitude on the NA trials to startle magnitude to the CSs in each block.

Statistical Analyses

All statistical analyses were run in R version 3.5.1 and R Studio version 1.3.1073. Participants were categorized as either having experienced sexual trauma or not having experienced sexual trauma based on whether they endorsed sexual abuse on the CTQ and/or adulthood sexual assault on the TEI. *N*=2376 participants reported sexual assault or abuse (ST group), and *n*=2758 reported other traumas but not sexual traumas (NST group). The participants in the ST group were further divided into three groups regarding timing of sexual trauma: those who only reported childhood sexual abuse (CSA, occurring before the age of 18, *n*=1562), those who only reported adulthood sexual assault (ASA, occurring at age 18 or later, *n*=175), and those who reported both (*n*=488). Data on timing of sexual assault or abuse experience was missing for *n*=151 women in the ST group, and these were excluded from analyses related to timing of trauma exposure.

In order to control for the possible confounding effects of age and overall trauma loud (indexed by types of trauma, TEI), these two variables were included as covariates in all of our

analyses. Hypotheses regarding symptom severity were assessed in two stages using one-way analyses of covariance (ANCOVA): first, by comparing the ST group to the NST group, and second, by comparing the three different groups of sexual trauma timing (childhood only, adulthood only, or both). ANCOVAs were performed to assess the effect of ST on total PTSD symptom scores (indexed by the PSS), and total number of hyperarousal, avoidance and numbing, and re-experiencing cluster symptom scores. Additionally, we standardized symptom cluster scores and performed a multivariate analysis of covariance (MANCOVA) to assess whether there was a main effect of sexual trauma across the three categories of symptoms. A binomial logistic regression was performed to determine the effect of sexual trauma on the prevalence of PTSD diagnosis in the ST and NST groups.

To assess our hypothesis that experiencing ST in childhood (whether only in childhood or in both childhood and adulthood) would result in greater symptoms of numbing and avoidance, ANCOVAs were performed with sexual trauma timing as our between-groups factor and age and total trauma load as our covariates. We also assessed total symptom scores and symptom scores in each of the three clusters across the three groups of sexual trauma timing using ANCOVAs. We performed a logistic regression to determine the effect of timing of sexual trauma on prevalence of PTSD diagnosis and performed a MANCOVA to assess the effect of timing of sexual trauma across the standardized symptom categories.

In order to pursue our questions about early family environment and its effect on resiliency in childhood sexual abuse survivors, we investigated the relationship between CSA and survivors' self-rated early family environment warmth and stability (indexed with the CDF) using separate one-way ANCOVAs for each trait. We created a subset of our sample to assess these relationships among the ST group who endorsed sexual abuse only in childhood. Within

this subset, ANCOVAs were performed with age and total trauma load as covariates to assess the effects of family stability and of family warmth on total PTSD symptom scores and on each of the three symptom clusters separately. Binomial logistic regressions were performed to assess the effect of family warmth and family stability on prevalence of PTSD diagnosis among CSA survivors.

One-way ANCOVAs were performed with neuroimaging data to assess left and right amygdala habituation (a comparison of early and late blocks of stimuli) and left and right amygdala reactivity (a comparison of fearful and neutral face stimuli). One-way ANCOVAs were performed to analyze trait and state anxiety scores (both before and after the fMRI scan) using the STAI questionnaire in the two groups. We did not perform analyses of neuroimaging data across the three groups of sexual trauma timing due to the inadequate number of participants with fMRI data who endorsed adulthood-only trauma (n=4).

To assess fear-potentiated startle, we used mixed-measures ANCOVAs with Trial Type (i.e., CS+ or CS–) and block (i.e., early, mid, and late) as our within-subjects factors, and between-subjects factors for sexual trauma (yes/no) and timing of trauma (childhood only, adult only, both). Again, total trauma load and age were included as covariates.

Results

Sample characteristics

Table 1 shows demographic and clinical characteristics of our sample. Participants endorsed notably high levels of childhood maltreatment (CTQ total M=41.3, SD=18.2) and repeated traumatic events in adulthood (TEI adult traumas M=34.7, SD=19.9), but the group who endorsed sexual trauma exposure (ST) had significantly more traumas in both childhood and

adulthood than the group who endorsed no sexual trauma (NST). 31% of the overall sample met criteria for probable diagnosis of Major Depressive Disorder, but the ST group had a significantly higher prevalence of MDD diagnosis in their subset. They also had significantly higher depressive symptoms (indexed by the BDI).

The ST group had significantly greater educational achievement than the NST group. 55% of the overall sample had a household monthly income of $999 or less. All subsequent comparisons of the ST and NST groups therefore controlled for covariates including total trauma load (number of different traumas experienced in either childhood or adulthood) and age. We did not include depression symptoms as a covariate given the high known collinearity between PTSD and depression symptoms.

PTSD symptoms and diagnosis: Sexual trauma vs. non-sexual trauma survivors

First, we examined the influence of sexual trauma on PTSD diagnosis and symptom severity. As shown in **Fig. 1a**, ST had higher symptoms of PTSD on the PSS scale (n=2376; M=17.1, SD=12.8) than NST (n=2758; M=8.6, SD=9.8), F(5159=90.6, p=2.64e–21). Notably, this effect was observed above and beyond the independent effect of total trauma load, which was also positively associated with total symptoms, F(1,5159)=835.9, p=1.80e–170. Individuals in the ST group were significantly more likely to have a diagnosis of PTSD than the NST group, χ^2(3)=840.72, p<0.001 (**Fig. 1b**).

Next, we examined the effect of sexual trauma on the individual PTSD symptom clusters: hyperarousal (Criterion E), avoidance and numbing (Criterion C and D), and intrusion/re-experiencing (Criterion B). To assess the difference in symptom profiles between the ST and NST groups, scores were standardized within each symptom cluster, with the vector of standardized scores representing the outcome in a MANCOVA analysis. After adjusting for

covariates, there was a significant main effect of sexual trauma on the multivariate profile of symptoms across the three categories, $F(1,5127)=34.65$, $p<2.2e{-}16$ (**Fig. 2a**). The ST group showed greater symptom severity across all types, with prominence of both avoidance and hyperarousal, while the NST group had proportionately more re-experiencing symptoms. As shown in **Figs. 2b-d**, univariate follow-up tests indicated that ST was associated with greater hyperarousal ($F(1,5149)=91.3$, $p=1.88e{-}21$), avoidance and numbing ($F(1,5159)=72.7$, $p=1.95e{-}17$), and re-experiencing ($F(1,5135)=43.7$, $p=4.25e{-}11$), relative to NST. In sum, ST was associated with greater PTSD prevalence and symptom severity, with a symptom profile characterized primarily by hyperarousal rather than avoidance and numbing.

PTSD symptoms and diagnosis: Timing of sexual trauma

We then tested the hypothesis that sexual abuse in childhood would have a larger impact on later symptoms than adult sexual assault within the ST group. There was a significant effect of timing on total symptom count after adjusting for covariates ($F(2,2220)=6.989$, $p=9.43e{-}04$), such that experiencing sexual trauma in both timeframes was positively associated with higher symptom counts. Post hoc pairwise comparisons of estimated marginal means using the Bonferroni test indicated that the mean score for the group with ST experienced in both childhood and adulthood was associated with greater PTSD symptom severity ($M=22.6$, $SD=12.9$) than in the childhood-only group ($M=15.9$, $SD=12.5$) and the adulthood-only group ($M=14.5$, $SD=11.3$), while the childhood-only and adulthood-only groups did not differ significantly from each other (**Fig. 3a**). We also investigated the effect of sexual trauma timing on prevalence of PTSD diagnosis, finding that those who had experienced sexual trauma in both childhood and adulthood were significantly more likely to have a diagnosis of PTSD than those

who experienced it in only childhood or only adulthood, $\chi^2(4)=277.26$, $p<0.001$ (**Fig. 3b**). There was a main effect of trauma timing on the multivariate profile of PTSD symptom subtypes, $F(2,4436)=3.56$, $p=0.002$. As shown in **Fig. 4a**, the group with childhood trauma only had proportionately more avoidance/numbing and re-experiencing symptoms than the adulthood-only group. The group with traumas in both time periods had greater symptoms in each cluster and showed a particular prominence in avoidance and numbing symptoms. ANCOVAs performed on the total scores for each of the three clusters indicated significant effects of trauma timing for each cluster. After adjusting for covariates, there was a statistically significant difference in hyperarousal symptoms ($F(2,2220)=4.676$, $p=0.009$) in the three groups, such that the childhood-only group had significantly fewer symptoms in this cluster than group with both (**Fig. 4b**). Timing differences were more pronounced in the avoidance and numbing cluster, where the significant effect of timing ($F(2,2220)=8.530$, $p=2.04e{-}04$) was observed between the childhood-only and both group, as well as between the adulthood-only and both group (**Fig. 4c**). Finally, there was a group difference in re-experiencing symptoms ($F(2,2220)=3.507$, $p=0.03$), but the only significant between-groups difference was observed between the adulthood-only and both groups (**Fig. 4d**). Overall, experiencing sexual trauma in both childhood and adulthood was associated with the greatest PTSD prevalence and symptom severity and a profile characterized by avoidance and numbing symptoms, but experiencing CSA alone was not necessarily worse than experiencing ASA alone.

PTSD symptoms and diagnosis: Early family environment among CSA survivors

Given the severity of symptoms observed in childhood sexual abuse survivors, we considered aspects of early family environment that might serve as protective factors against

developing posttraumatic stress psychopathology. In a subset of ST participants who endorsed childhood sexual abuse only (n=1562), one-way ANCOVAs were conducted with covariates of total trauma load and age to analyze the effects of early family stability and early family warmth (indexed by the CDF) on total PTSD symptoms. Self-rated family stability (F(4,1123)=8.932, p=4.22e–07) and family warmth (F(4,1123)=9.351, p=1.96e–07) in childhood each had a significant effect on total symptoms. Results of a linear contrast indicated a negative association such that greater stability (F(6,1123)=26.73, p<2.2e–16) and warmth (F(6,1123)=27.04, p<2.2e–16) predicted fewer total symptoms (**Fig. 5a-b**). Similarly, there was a negative association between PTSD prevalence among childhood ST survivors and family stability (χ^2(6)=113.24, p<0.001; **Fig. 5c**) and family warmth (χ^2(6)=116.42, p<0.001; **Fig. 5d**). Analyses of each of the symptom clusters showed that self-rated family stability and family warmth were each negatively associated with hyperarousal symptoms (**Table 2**). The strongest negative association between either of the early family environment variables and symptom clusters was observed between family warmth and avoidance and numbing symptoms. In sum, family stability and warmth in childhood among CSA survivors were protective factors for PTSD prevalence and symptom severity, and were most protective for the avoidance and numbing cluster. There were no differences in the strengths of protective effects for stability versus warmth.

Fear-Potentiated Startle Paradigm

Potential unique fear learning patterns among ST survivors (n=118) versus NST survivors (n=168) were assessed with data from a fear-potentiated startle paradigm that included a subset of participants from our larger sample who returned for a separate visit. There was a main effect of sexual trauma on the magnitude of the startle response, such that the ST group startled more across all blocks and CS types than the NST group (F(1,1684)=7.19, p=0.007; **Fig.**

6). Main effects of trial type (i.e., CS+ or CS–) (F(1,1684)=12.75, p=0.0004) and block (i.e., early, mid, or late) (F(2,1684)=54.52, p=1.14e–21) indicated greater startle to the CS+ (threat) than to the CS– (safety) stimuli, and habituation to CS cues over the course of the experiment, as expected for the fear-potentiated startle paradigm. The interaction between block and type was also significant (F(2,1684)=7.03, p=9.12e–04), indicating the expected pattern of faster habituation to the CS– stimulus than the CS+. Younger age was also associated with greater startle (F(1,1684)=42.14, p=1.12e–10). We subsequently tested whether the developmental timing of sexual trauma was associated with different responses to threat and safety cues during fear-potentiated startle. Timing data was available in this subset for n=112 survivors of childhood-only ST, n=13 survivors of adulthood-only ST, and n=42 survivors of both. The effect of sexual trauma timing did not meet the threshold for statistical significance, F(2,964)=1.99, p=0.138 (**Fig. 7**). Overall, sexual trauma was associated with exaggerated startle magnitude, and in looking closer at timing of sexual trauma, experiencing both CSA and ASA was associated with a more blunted startle response, though not at the level of statistical significance.

Neuroimaging Paradigm

To test the extent to which sexual trauma influences affetive response to social threat cues, we examined amygdala reactivity and habituation to fearful and neutral faces in a neuroimaging paradigm. A subset of participants returned for a separate neuroimaging visit (n=53 ST, n=42 NST). Pre-fMRI state anxiety scores were significantly higher in the ST group than the NST group, F(1,91)=5.330, p=0.023 (**Fig. 8**). This variable was therefore included as a covariate in all subsequent fMRI analyses. There were no group differences in trait anxiety (F(1,91)=3.050, p=0.084) or post-fMRI state anxiety scores (F(1,84)=0.017, p=0.895).

There was no association of ST and right amygdala reactivity ($F(1,90)=0.470$, $p=0.495$) or left amygdala reactivity ($F(1,90)=0.225$, $p=0.636$). Additionally, sexual trauma showed no effect on right ($F(1,90)=1.875$, $p=0.174$) or left amygdala habituation ($F(1,90)=1.043$, $p=0.310$). However, total trauma load predicted greater left amygdala habituation to fearful faces ($F(1,90)=4.907$, $p=0.029$). Therefore, sexual trauma did not appear to be associated with unique patterns of habituation or reactivity in the amygdala to social threat cues.

Discussion

The current study examined the distinctive effects of sexual trauma on PTSD symptoms, fear-potentiated startle response, and amygdala reactivity and habituation. Overall, we did not find support for our hypotheses regarding emotional numbing in sexual trauma survivors. Findings that sexual trauma was related to more severe PTSD symptomology overall were consistent with previous literature (e.g., Breslau et al., 1991; Kelley et al., 2009; Kessler et al., 1995; Norris, 1992), but our hypothesis that avoidance and numbing symptoms would be most prominent in sexual assault/abuse survivors was not supported. When considering all sexual trauma survivors, hyperarousal symptoms predominated the symptom profile. However, when examining the timing of sexual trauma, the hyperarousal profile was only observed among individuals who experienced sexual assault in adulthood. Individuals exposed to sexual trauma in both childhood and adulthood exhibited a symptom profile predominated by avoidance and numbing symptoms, more consistent with our hypothesis. Our hypotheses that sexual trauma survivors (and CSA survivors in particular) would demonstrate affective overmodulation and defensive reactivity in physiological and neurofunctional responses were not supported.

Additionally, we observed no unique patterns of amygdala habituation or reactivity to a fearful vs. neutral faces paradigm in survivors of sexual trauma.

Our work concludes that survivors of sexual traumas (e.g., rape, sexual assault, and childhood sexual abuse) are more likely to develop PTSD and have more severe presentations of PTSD than survivors of other types of traumas, including non-sexual assaultive violence, childhood physical and emotional abuse and neglect, serious accident or injury, military combat, and natural disasters. In addition, our hypothesis that ST survivors would experience greater symptoms of avoidance and numbing than their non-sexually assaulted counterparts was supported, but this was also true for the re-experiencing and hyperarousal symptom clusters. In fact, the most prominent symptom cluster of the ST group was the hyperarousal cluster, followed by the avoidance and numbing cluster and lastly the re-experiencing cluster. This is somewhat surprising, given that previous studies have found restriction of affect and emotional numbing to be distinguishing features in survivors of assaultive violence (Chung & Breslau, 2008; Forbes et al., 2011) and in survivors of sexual traumas in particular (Graham et al., 2016; Kelley et al., 2009). In addition, there is evidence that emotional numbing distinguishes the class of survivors with the most severe PTSD from other classes, and that this class is more likely to be comprised of female assaultive violence survivors (Breslau et al., 2005). The fact that our large sample of female ST survivors was not distinguished by emotional numbing suggests that it may not be the most robust predictor of posttraumatic stress pathology severity in all samples. Furthermore, our sample may be more representative of the general population than other studies, which often use undergraduate students or veterans as their sample (e.g., Graham et al., 2016; Kelley et al., 2009). Given the importance of emotional numbing in predicting the development of future PTSD, future studies should continue to compare sexual assault/abuse to other types of traumas

and attempt to identify unique symptom patterns in different types as survivors, as well as the mechanisms through which these may occur.

In looking closer at the timing of sexual traumas, we found that women who experienced both sexual abuse in childhood and sexual assault/rape as adults reported symptoms more severe than women who experienced either childhood sexual abuse or adulthood sexual assault alone, even after controlling for total trauma load. Our hypothesis that CSA-only survivors would show the greatest prominence in avoidance and numbing symptoms was not supported; instead, the group with both CSA and ASA was distinguished by the avoidance and numbing cluster, while the ASA and CSA group did not differ from each other in this symptom cluster. This was unexpected, given that previous literature has found that CSA survivors experience more severe psychopathology than adult sexual assault survivors (Briere & Elliott, 2003), that severe CSA predicts more avoidance and numbing symptoms (Glover et al., 2010), and that the relationship between severity of CSA and trauma-related psychological distress in adulthood is mediated by avoidance (Rosenthal et al., 2005). The finding that the group with CSA and subsequent revictimization as adults had the greatest number of symptoms is less surprising, given that there is a large body of evidence that supports more severe psychopathology in rape survivors who were previously sexually abused in childhood than those who were not, beyond what is attributable to the effects of repeated traumas (e.g. Bolstad & Zinbarg, 1997). In particular, our results match those of a large study in a similar sample of African American women that found no difference in PTSD severity between women who reported child abuse but not rape and women who reported rape but not child abuse (i.e. CSA only and ASA only; Schumm et al., 2006). It appears that cumulative sexual trauma, rather than trauma in a specific developmental timeframe, may be a better predictor of later avoidance and numbing symptoms, even after

controlling for lifetime trauma experiences. In light of this, our findings do not support the existence of an emotionally numb profile unique to CSA survivors; more likely, this profile is found in survivors of both CSA and ASA, accompanied by overall higher symptoms. Future studies should seek to confirm this finding.

Among survivors of childhood sexual abuse, a robust relationship was observed between symptom severity and certain resiliency factors at play in early family environment. Both early family warmth and early family stability were significant protective factors for later severity of PTSD in those who experienced CSA. This was expected and aligns with previous research that has shown family stability (Chandy et al., 1996; Hyman & Williams, 2001; Katerndahl et al., 2005), and warmth/support (Feiring et al., 1998; Rosenthal et al., 2003) are protective factors for later psychopathology. We did not examine early family environment in survivors of both childhood and adulthood sexual traumas, nor did we assess the effects of family warmth and stability on FPS or fMRI results given the lack of significant effects of trauma timing in these analyses. Future studies should consider these resiliency factors as they contribute to psychopathology in survivors of childhood sexual abuse versus other types of childhood maltreatment.

Our hypothesis that ST survivors would show a decreased startle response in comparison to NST survivors was not supported. Rather than exhibiting a defensive physiological reaction and associated blunted startle response, ST survivors startled more for all trial types and blocks than NST survivors. This is somewhat surprising given that a number of studies have observed decreased startle potentiation in survivors of multiple and more severe traumas (McTeague et al., 2010), in correspondence to lengthier chronicity (Lang & McTeague, 2009), and in PTSD patients compared to other anxiety disorder patients (Cuthbert et al., 2003). However, several of

these studies paired startle paradigms with fear memory or aversive imagery, which may encourage participants prone to emotional numbness to suppress physiological responses as a defense mechanism against the aversive imagery, while our study did not incorporate aversive or script-driven imagery in conjunction with fear learning. Our findings are consistent with previous literature that has found that more severe PTSD symptomology corresponds to greater startle, greater fear potentiation to danger, and poorer discrimination between threat and safety cues (Glover et al., 2011; Griffin, 2008; Jovanovic et al., 2009). Additionally, several studies have found that victims of assaultive violence are distinguished by exaggerated startle, which our data support (Chung & Breslau, 2008). Among the three timing groups of sexual trauma, there was no significant difference in startle magnitude, which did not support our hypothesis that CSA survivors would show a decreased startle response in accordance with a general blunted affective profile. Instead, CSA survivors demonstrated a greater, albeit nonsignificant, startle than their ASA and both CSA and ASA counterparts. The hypothesized decreased startle response was observed in survivors of both CSA and ASA, but the fact that this observation did not meet the level of statistical significance may be the result of an underpowered analysis due to our smaller sample size in this subset. Fear potentiation to startle is an important indication of how individuals with PTSD are able to inhibit and extinguish fear, so further study is needed to better understand the differences in fear learning between these different types of sexual trauma survivors.

Finally, we did not observe any significant difference in amygdala habituation or reactivity in ST survivors. Our hypothesis that ST survivors would exhibit hypoactivity in the amygdala was not supported; non-significant results suggested increased reactivity rather than decreased reactivity in the amygdala in ST compared to NST survivors. These null findings are

particularly unexpected given the significant effects of sexual trauma on all other aspects examined in the study. Additionally, the fact that ST survivors had higher symptom counts, were more likely to be diagnosed with PTSD, and demonstrated more robust startle than NST survivors suggests a corresponding amygdala hyperactivation pattern widely supported by previous literature (Admon et al., 2013; Davis et al., 1993; Hayes et al., 2012). However, the fact that total trauma load was positively associated with amygdala habituation suggests that sequential or chronic trauma over a lifetime may cause women to quickly down-regulate affective responses to social threat cues encountered repeatedly, but sexual trauma does not seem to have a bearing on this area above and beyond total trauma.

This study has a number of strengths. To date, it is one of the largest studies to examine the impacts of sexual trauma in women, allowing more powerful effect sizes and more definitive results. We were also able to carefully control for confounding variables like lifetime trauma load while retaining well-powered analyses. Our work evaluating sexual trauma on three levels (psychopathology, psychophysiology, and neurobiology) allowed us to gain a clearer picture of the effects of sexual trauma in many facets of PTSD. Furthermore, our study also adds to the limited but growing body of literature regarding PTSD in an underrepresented sample of urban minority women, who are at higher risk for experiencing trauma and stress-related psychopathology but who remain understudied in research literature.

The current study should be viewed in light of several limitations. First, our sample was comprised of only African American women. This decision was made because of the increased prevalence of PTSD in urban minority women (Breslau et al., 1998) and because the makeup of the urban hospital setting is majority African American (Gillespie et al., 2009). Even so, generalizability of these findings is necessarily limited due to the homogeneity of the sample,

and results should be replicated in more diverse samples, including both men and women of a variety of races and ethnicities. Additionally, as with all cross-sectional studies, we are limited in making definitive statements about the impacts of sexual trauma within the context of chronic trauma over a lifetime. Longitudinal studies like the ABCD (Adolescent Brain Cognitive Development) study will reveal more definitive mechanistic understanding of trauma types over a lifetime.

Overall, our findings do not provide support for the existence of a blunted affective profile in survivors of sexual trauma. Instead, we found that ST survivors had profiles characteristically similar to their NST counterparts that differed primarily in overall severity of PTSD symptoms. The results of this study have implications for the treatment of PTSD in sexual assault and abuse victims. In particular, if ST survivors do not differ in their qualitative symptom presentation from survivors of other types of traumas, they are equally likely to benefit from treatments that target typical PTSD symptoms like impaired extinction learning. If ST survivors were chronically avoidant, emotionally numb, and hyporeactive to fear cues, these types of treatments may not be effective, but our study suggests the opposite. In particular, prolonged exposure (PE) therapy is a well-validated and widely-used treatment for PTSD that delivers extinction training through repeated engagement with traumatic memories and cues (Rothbaum & Davis, 2003). A study that implemented modified PE as an early intervention in the immediate aftermath of trauma found the greatest effect sizes of the intervention for rape victims (Rothbaum et al., 2012), suggesting that PE is a highly effective treatment for ST survivors. Cognitive processing therapy (CPT), which aims to identify distorted and overgeneralized beliefs about oneself and the world in the context of one's trauma, often referred to as "stuck points," has also proved to be highly efficacious as a treatment for rape survivors (Resick et al., 2002).

These findings are particularly promising for samples such as ours, which are highly symptomatic and trauma-exposed.

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Table 1. Sample Characteristics

	Sexual trauma (*n*=2376)	**Non-sexual trauma (*n*=2758)**	**Comparison (p-value)**
Characteristic			
Age, years (mean, SD*)*	39.9 (13.0)	37.8 (14.7)	< 0.001
Educational attainment, %			< 0.001
Some high school	23.5	19.8	
High school graduate or GED	35.0	45.3	
Some college or tech degree	31.2	25.8	
College graduate	8.5	7.3	
Graduate school	1.7	1.8	
Household monthly income, %			0.06
$0 – 999	56.9	54.0	
$1000 – 1,999	27.4	29.9	
$2000 or more	15.6	16.2	
Trauma history (mean + SD*)*			
Childhood traumas (CTQ total)	51.8 (20.1)	32.2 (9.6)	< 0.001
Adulthood traumas (TEI total)	37.9 (19.8)	31.5 (19.5)	< 0.001
Clinical symptoms (mean + SD *or %)*			
Depressive symptoms (BDI)	18.2 (12.7)	11.0 (10.1)	< 0.001
Probable MDD (BDI, %)	44.7	19.5	< 0.001

Table 2. Family warmth and stability and symptom clusters

	***F*-value**	***p*-value**	***pes***	**Linear contrast (*F*, *p*-values)**
Hyperarousal				
Family stability	6.351	4.71e–05	0.022	21.44 (*p*< 2.2e–16)
Family warmth	6.130	7.03e–05	0.021	21.28 (*p*< 2.2e–16)
Avoidance and Numbing				
Family stability	10.026	5.66e–08	0.034	25.96 (*p*<2.2e–16)
Family warmth	10.884	1.17e–08	0.037	26.59 (*p*<2.2e–16)
Re-experiencing				
Family stability	4.558	1.00e–03	0.016	14.2 (*p*=1.218e–15)
Family warmth	4.888	6.55e–04	0.017	14.43 (*p*=6.555e–16)

Figures

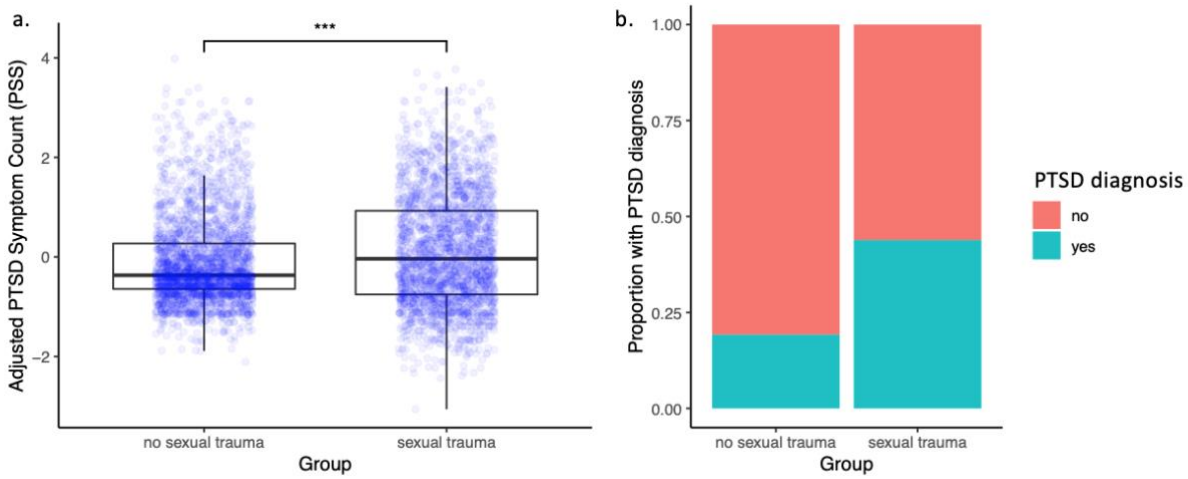

Figure 1. Effects of sexual trauma on PTSD symptoms and diagnosis. (a) Total PTSD symptom count, measured by the modified PTSD Symptom Scale, among the group with sexual trauma and without sexual trauma. Results indicated a significant difference (p<0.005) between the groups, with the ST group endorsing nearly twice as many total symptoms (M=17.1). (b) Prevalence of PTSD diagnosis between the ST group and NST group. A binomial logistic regression revealed a significant difference (p<0.005), such that 44% of the ST group had a PTSD diagnosis, versus just 19% in the NST group.

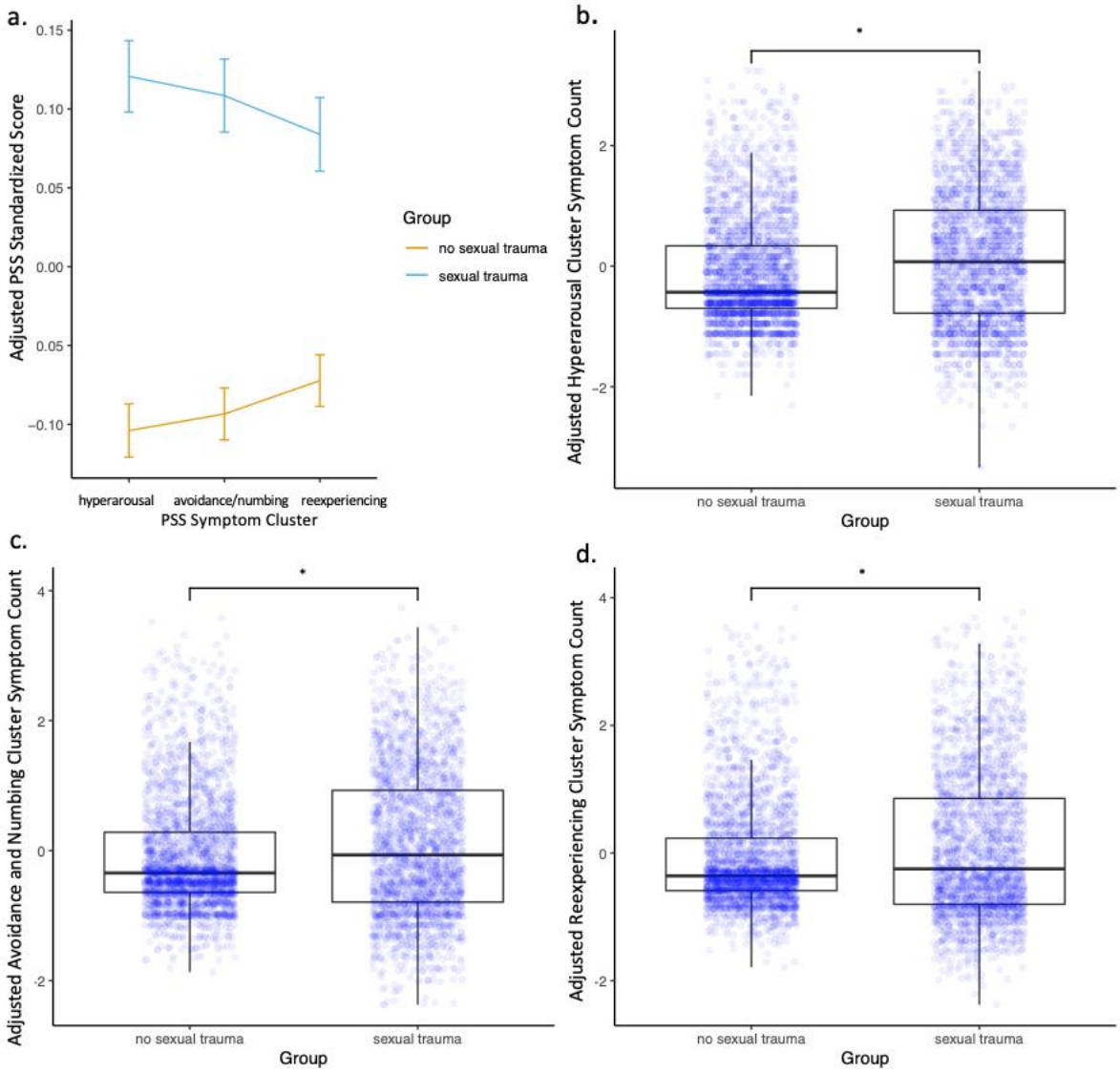

Figure 2. PTSD symptom clusters scores for ST and NST groups. (a) MANCOVA analysis results for standardized symptom cluster scores. Lines indicate mean scores and error bars indicate +/– 1 standard error (SE). Greater prominence of avoidance and hyperarousal was observed in the profile of ST survivors. (b) Hyperarousal symptom cluster scores for ST and NST groups. (c) Avoidance and numbing symptom cluster scores for ST and NST groups. (d) Re-experiencing/intrusion symptom clusters scores for ST and NST groups.

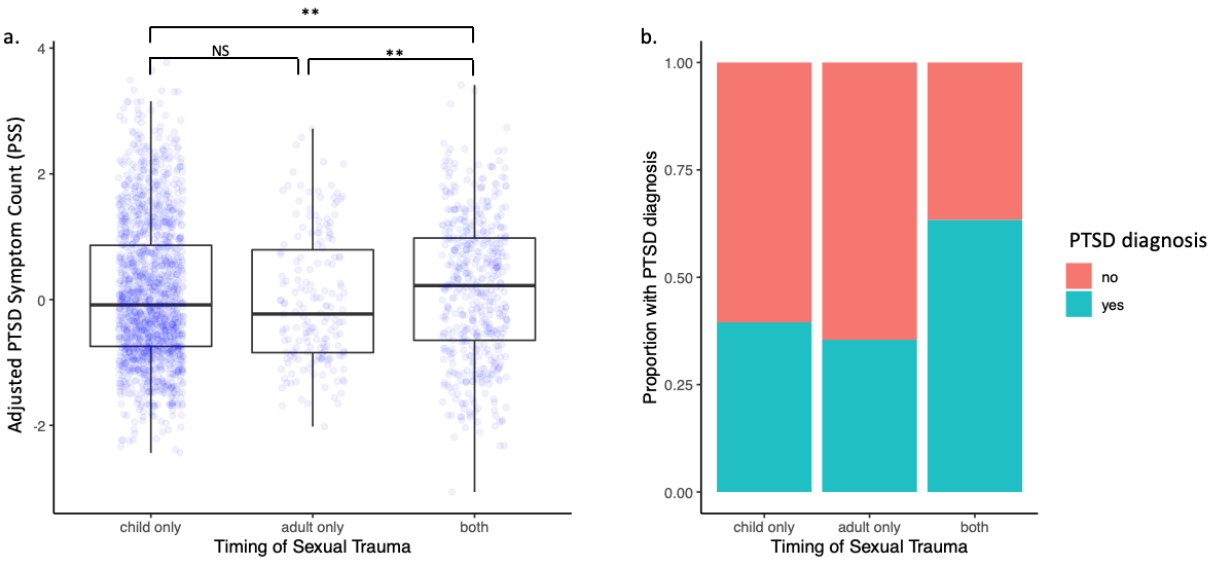

Figure 3. Effects of sexual trauma timing on PTSD symptoms and diagnosis. (a) Total PTSD symptom count, measured by the modified PTSD Symptom Scale, among the three different timing groups of sexual trauma. Results indicated a significant difference (p<0.005) between the groups, with the group that had sexual traumas in both childhood and adulthood having the highest scores (M=22.6). (b) Prevalence of PTSD in the three groups of sexual trauma timing. A binomial logistic regression revealed a significant difference (p<0.005) between the childhood-only group and the group with both, as well as the adult only group and group with both, but not between the childhood-only and adulthood-only group. 63% of the group with sexual trauma in both childhood and adulthood had a PTSD diagnosis, compared to just 40% of the childhood-only group and 35% of the adulthood-only group.

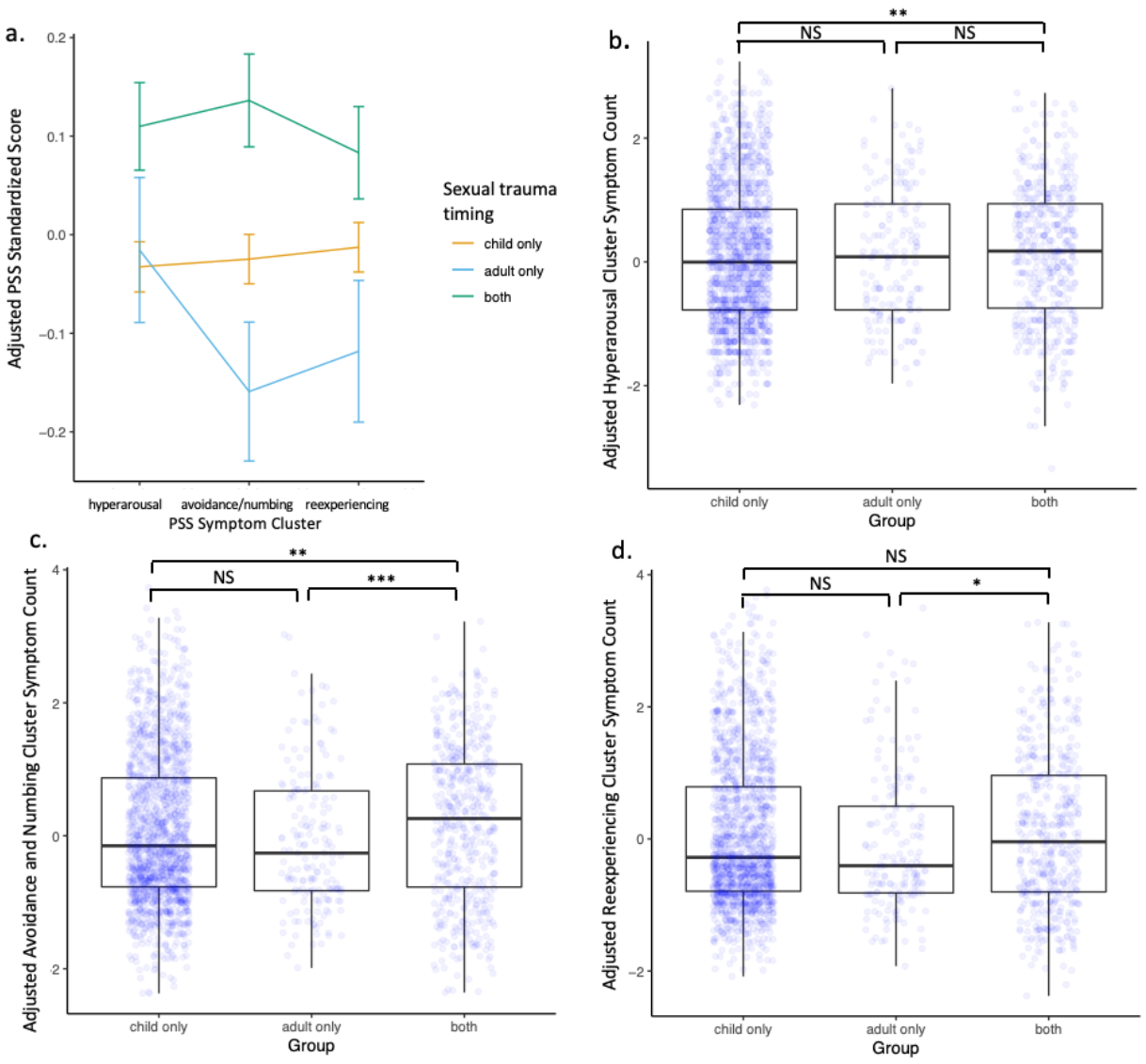

Figure 4. PTSD symptom clusters scores for the three groups of timing: childhood only sexual abuse, adulthood only sexual assault, and both. (a) MANCOVA analysis results for standardized symptom cluster scores. Lines indicate mean scores and error bars indicate +/– 1 SE. Survivors of both childhood and adulthood sexual traumas showed greater prominence across all symptom types. (b) Hyperarousal symptom cluster scores for the timing groups. (c) Avoidance and numbing symptom cluster scores for the timing groups. (d) Re-experiencing/intrusion symptom clusters scores for the timing groups.

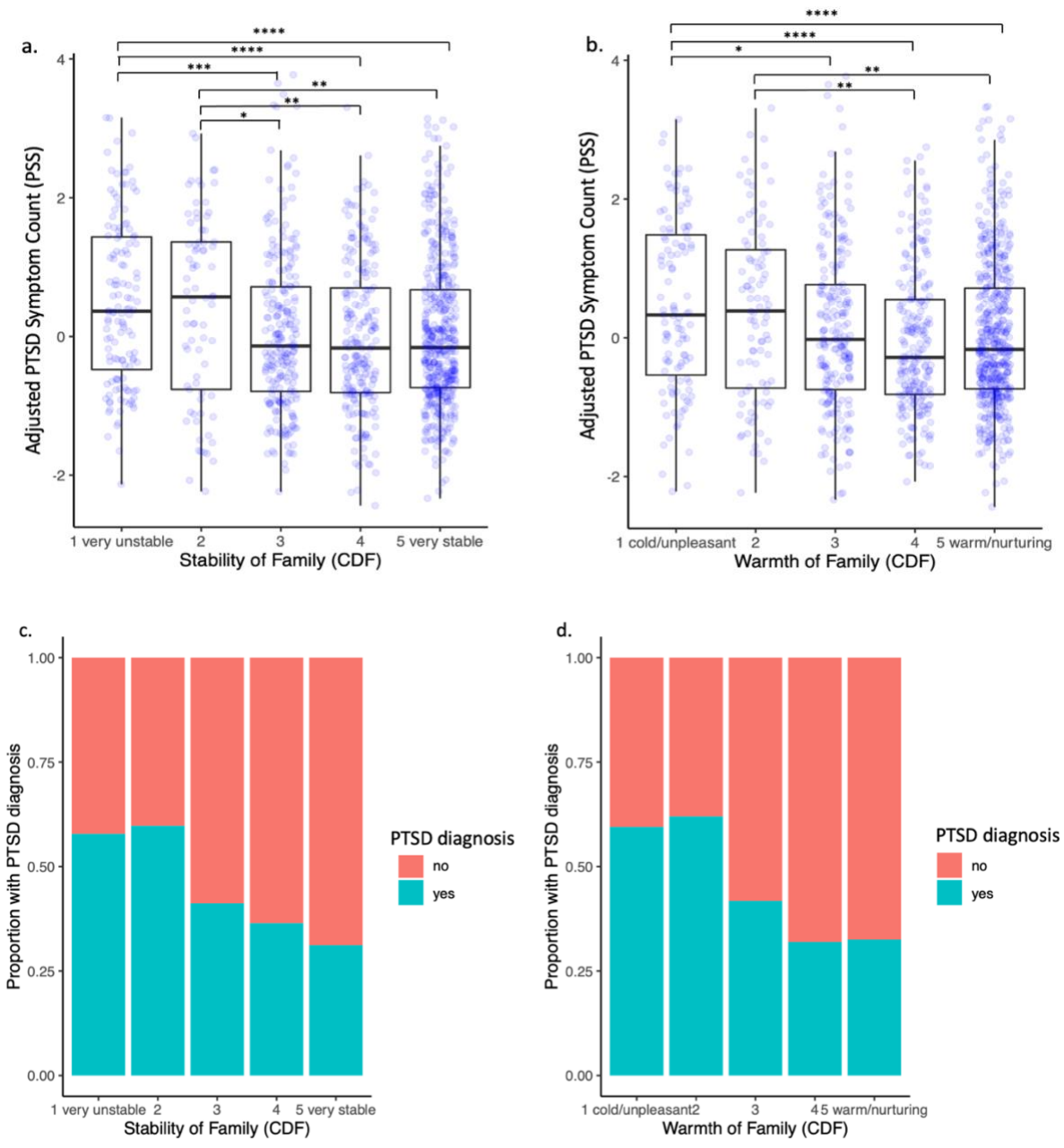

Figure 5. Relationships between early family warmth/stability and PTSD symptoms and diagnosis among survivors of CSA. (a) ANCOVA analysis results for symptoms in terms of early family stability. (b) ANCOVA analysis results for symptoms in terms of early family warmth. (c) Binomial logistic regression results for prevalence of PTSD diagnosis by early family stability. (d) Binomial logistic regression results for prevalence of PTSD diagnosis by early family warmth.

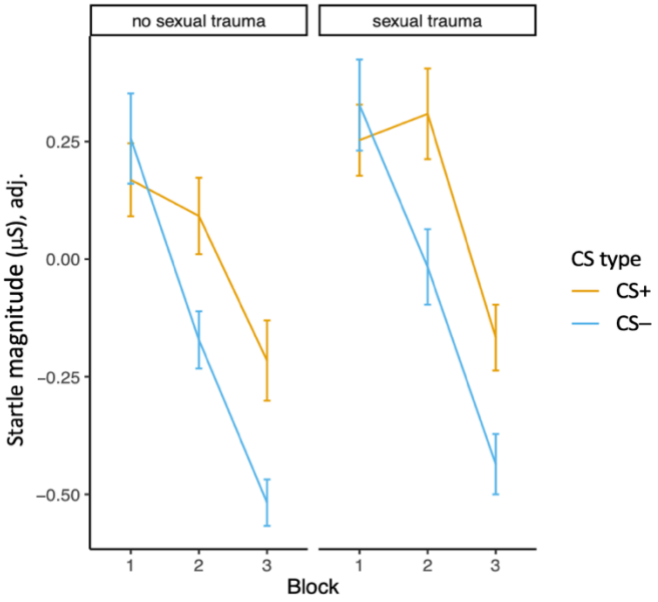

Figure 6. Mixed measures ANCOVA of fear-potentiated startle paradigm among sexual trauma and other trauma group. Task effects of block (early, middle, and late; indicates habituation to conditioned stimuli), type (CS+ vs. CS–), and block*type (faster habituation to CS– than to CS+) were significant. Main effect of sexual trauma was also significant, as the ST group had greater startle than the NST group in all phases.

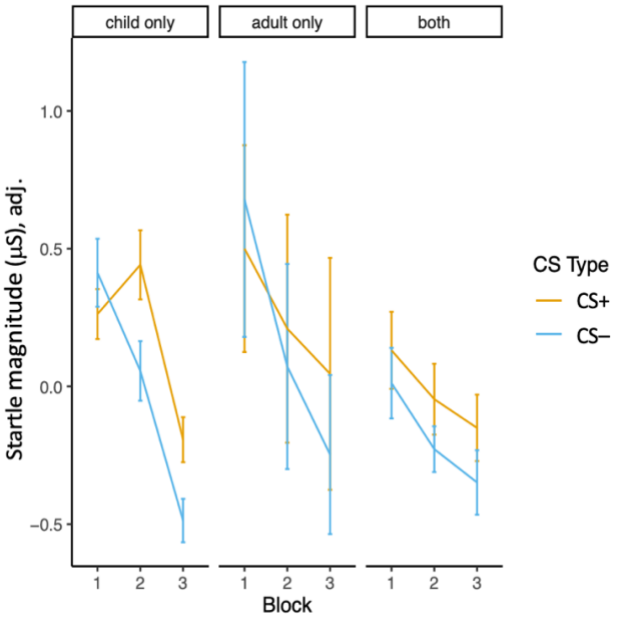

Figure 7. Mixed measures ANCOVA of fear-potentiated startle paradigm among survivors of sexual trauma in three phases of developmental timing. Main effects of trial type, block, and block*type were significant, indicating appropriate startle and habituation in all three groups. Between-subjects effect of timing of sexual trauma was not significant.

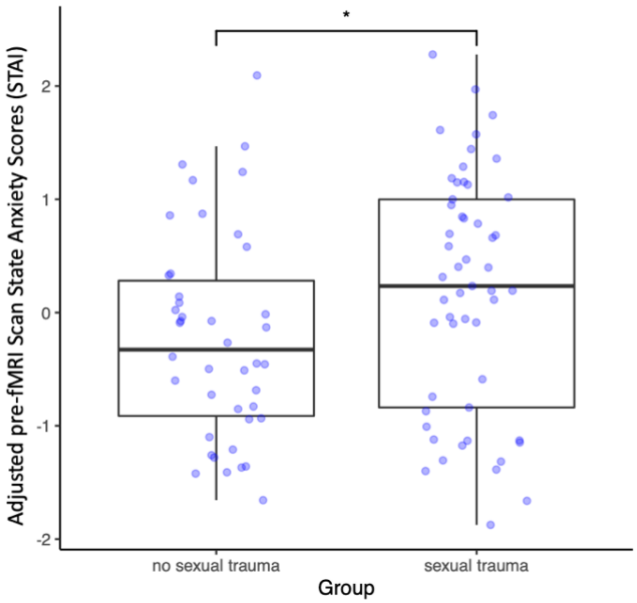

Figure 8. State anxiety scores evaluated prior to fMRI scan in the sexual trauma vs. non-sexual trauma groups. The sexual trauma group had higher state anxiety scores than the non-sexual trauma group.