Increased activation of the fear neurocircuitry in children exposed to violence

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Abstract
Most studies investigating the effect of childhood trauma on the brain are retrospective and mainly focus on maltreatment, whereas different types of trauma exposure such as growing up in a violent neighborhood, as well as developmental stage, could have differential effects on brain structure and function. The current magnetic resonance imaging study assessed the effect of trauma exposure broadly and violence exposure more specifically, as well as developmental stage on the fear neurocircuitry in 8- to 14-year-old children and adolescents (N = 69). We observed reduced hippocampal and increased amygdala volume with increasing levels of trauma exposure. Second, higher levels of violence exposure were associated with increased activation in the amygdala, hippocampus, and ventromedial prefrontal cortex during emotional response inhibition. This association was specifically observed in children younger than 10 years. Finally, increased functional connectivity between the amygdala and brainstem was associated with higher levels of violence exposure. Based on the current findings, it could be hypothesized that trauma exposure during childhood results in structural changes that are associated with later risk for psychiatric disorders. At the same time, it could be postulated that growing up in an unsafe environment leads the brain to functionally adapt to this situation in a way that promotes survival, where the long-term costs or consequences of these adaptations are largely unknown and an area for future investigations.

KEYWORDS
amygdala, brain structure, childhood trauma, functional magnetic resonance imaging, hippocampus, response inhibition, ventromedial prefrontal cortex

1 | INTRODUCTION

Childhood adversity has been shown to increase risk for psychiatric disorders across the lifespan, including posttraumatic stress disorder (PTSD; Dunn, Nishimi, Powers, & Bradley, 2016; McLaughlin et al., 2010, 2012; Norman et al., 2012; Widom, DuMont, & Czaja, 2007) and depression (Dunn et al., 2016). One of the primary mechanisms by which childhood trauma is theorized to contribute to PTSD and similar pathologies is through alterations to the fear neural circuitry (Jovanovic & Ressler, 2010); however, the influence of trauma exposure on this circuitry during development is not fully understood.

The primary brain regions involved in the fear inhibition circuit are the amygdala, hippocampus, and ventromedial prefrontal cortex (vmPFC). The amygdala serves as an essential locus for fear acquisition, memory, and expression (Kim & Jung, 2006). Increased amygdala activation has been demonstrated in adults reporting childhood maltreatment (Dannlowski et al., 2012, 2013; Grant, Cannistraci, Hollon, Gore, & Shelton, 2011; van Harmelen et al., 2013) as well as in maltreated or neglected children (Maheu et al., 2010; McCrory et al., 2013; Suzuki et al., 2014; Tottenham et al., 2011). Most structural magnetic resonance imaging (MRI) studies in children or adolescents with adverse childhood
In inhibition also takes place on a cognitive level via response inhibition, where a learned response must be suppressed. A Go/NoGo task is frequently used to measure response inhibition, and trauma exposure and PTSD have been associated with impaired brain responses to this task (Falconer et al., 2008; Jovanovic et al., 2013; Stevens et al., 2016; van Rooij et al., 2016, 2018). Interestingly, impairments were found in fear inhibition regions such as the vmPFC (Jovanovic et al., 2013; Stevens et al., 2016) and hippocampus (van Rooij et al., 2016, 2018), and several prior studies used (emotional) response inhibition tasks to show alterations in the fear neurocircuitry in maltreated children (Carrion et al., 2008; Mueller et al., 2010; Tottenham et al., 2011). The majority of prior studies have focused on childhood maltreatment or neglect, whereas much less is known about the effects of exposure to other types of trauma, such as violence exposure. The Grady Trauma Project is an ongoing study of PTSD risk factors in a low-income population in Atlanta, GA. Most participants live in unsafe neighborhoods in inner-city Atlanta and report high levels of trauma exposure, and PTSD and depression symptoms (Gillespie et al., 2009). While trauma research has focused extensively on the effects of continuous exposure to stress and trauma in an unsafe environment as part of military deployment, little is known about the neurobiological consequences of growing up in a dangerous environment and being exposed to violence on a regular basis as a normal part of life. The current study focused on the children of our adult participants to assess the effect of trauma and violence exposure during development on the fear neurocircuitry.

2 | MATERIALS AND METHODS

2.1 | Participants

African American children and adolescents aged 8–14 years (N = 69, 36 female) were recruited through the Grady Trauma Project (Gillespie et al., 2009). Exclusion criteria for children were a history of bipolar disorder or schizophrenia, active psychotic symptoms, or cognitive disability, previous head injury with loss of consciousness, history of stroke, epilepsy, neurological disorder, autism spectrum
disorder, or brain tumor, metal in the body, or hearing or vision impairment unable to be corrected by glasses.

Testing took place at Grady Memorial Hospital and the scan at Facility for Education and Research in Neuroscience at Emory University. The protocol was approved by the Institutional Review Boards of Emory University and Research Oversight Committee at Grady Memorial Hospital. Written consent was obtained from a legal guardian of the child participant and oral (younger than 11) or written (ages 11 and older) consent was obtained from child participants.

2.2 Trauma and violence assessment

Interviews were conducted with each child to assess for trauma exposure broadly, violence exposure more specifically, as well as PTSD, anxiety, and depression symptoms. Child-reported trauma exposure was assessed with the Traumatic Events Screening Inventory (TESI) for children (Ribbe, 1996). This 19-item questionnaire assessed a variety of potentially traumatic events, such as disasters, accidents, injuries, violence, and abuse, and was answered with Yes or No to each item. The total score was used in the analyses as a measure for trauma exposure broadly. Exposure to violence specifically was measured using the Violence Exposure Scale for Children-Revised (VEX-R; Fox & Leavitt, 1995). The 25-item questionnaire has a male and female version of drawings that accompany questions and a frequency rating scale to inquire about exposure to violence in the home, school, and community. This assessment has been previously used with children from the Grady Trauma Project demonstrating high rates of violence exposure (Cross et al., 2018). Current PTSD symptoms were assessed using the child-report UCLA PTSD Reaction Index (UCLA-RI; Steinberg, Brymer, Decker, & Pynoos, 2004). The Behavioral Assessment System for Children (BASC) was used to assess anxiety and depression symptoms (Reynolds, Kamphuis, & Vannest, 2011). The BASC score is a gender- and age-corrected t value with 50 indicating the mean.

2.3 Emotional Go/NoGo functional MRI (fMRI) task

The emotional Go/NoGo (eGNG) task has been previously used by Tottenham et al. (2011) in a study with children with early-life stress. Participants were instructed to press a button for fearful faces (Go trial) and withhold responses for neutral faces (NoGo trial), or vice versa, and the order of runs was counterbalanced among participants. Each stimulus was followed by a varied intertrial interval ranging from 2,500 to 15,000 ms. There were two runs with 36 Go trials and 12 NoGo trials in each run.

Overall reaction time, % correct Go’s, % correct NoGo’s, and accuracy (correct Go’s – incorrect NoGo’s)/total number of trials were calculated for behavioral analyses. The contrast for correct NoGo trials larger than Go trials was used for the fMRI analyses.

Only accurate trials were included in the imaging analyses to ensure proper engagement of participants with the task.

2.4 MRI procedures and analyses

Participants completed a mock scan protocol at a visit before their actual scan during which they were acclimated to the scanner. Participants completed practice sessions of the study tasks both inside and outside of the mock scanner to ensure that they understood how to complete the task.

Functional and structural MRI scans were acquired on a 3.0-T Siemens Trio (whole-body) MR scanner using a 32-channel head coil. A T1-weighted image (176 slices, repetition time [TR] = 2,250 ms, echo time [TE] = 4.18 ms, and voxel size $1 \times 1 \times 1 \text{ mm}$) was used for within-subject registration and to measure left and right hippocampal and amygdala volumes. Structural T1-weighted MRI scans were analyzed using Freesurfer v6.0. Quality control and processing were performed in conjunction with standardized ENIGMA protocols (http://enigma.ini.usc.edu). Left and right hippocampal and amygdala volumes, and intracranial volumes (ICV) were extracted and exported to SPSS 26.0 (IBM SPSS Statistics 26.0).

Two runs of 131 echo-planar imaging blood oxygen level-dependent images (total of 262) were acquired during which the participants performed the emotional Go/NoGo task. Volumes contained 44 slices of 2.5 mm thickness acquired in a descending sequential slice order parallel to the anterior–posterior commissure line, with a 0.5 mm slice gap. GRAPPA parallel imaging with an acceleration factor of 1 was used to facilitate speed of acquisition. The following parameters were used: TR = 2,330 ms, TE = 30 ms, flip angle = 90°, and voxel size $3 \times 3 \times 3 \text{ mm}$.

Functional images were analyzed (file conversion, image preprocessing, and statistical analyses) using Statistical Parametric Mapping, version 8 (http://www.fil.ion.ucl.ac.uk/spm/). ArtRepair was used to detect and repair bad slices. Functional images were slice-time corrected and realigned to the first image in the session to correct for motion. Next, ArtRepair was used to detect and repair bad volumes and to test if participants exceeded the motion threshold set for exclusion (>2 mm/TR). The average maximum (framewise) motion was 1.06 mm/TR. Bad volumes were interpolated, a maximum of 10% per participant with an average of 3.9% for included participants. The structural T1 volume was coregistered to the mean of the realigned functional images and spatially normalized to standardized Montreal Neurological Institute (MNI) space. The normalization parameters were then applied to the functional volumes and the images smoothed with a 6 mm full-width at half maximum Gaussian kernel.

Subject-level statistical maps were created for the correct NoGo > Go contrast. Data were extracted for a priori regions of interest (ROIs) and as there were no specific hypotheses for unilateral analyses, bilateral ROIs were extracted in accordance with prior studies (Stevens et al., 2014; van Rooij et al., 2018): bilateral amygdala based on the Anatomical Automatic Labeling atlas.
(http://www.gin.cnrs.fr/AAL), bilateral hippocampus based on the Hammers atlas (Rodionov et al., 2009), and the vmPFC, for which a 6 mm sphere around a peak voxel (MNI coordinates: 4, 44, −4) from a previous response inhibition study showing vmPFC activation (Jovanovic et al., 2013). ROI data were exported to SPSS.

2.5 | Group analyses

For both trauma exposure broadly (TESI) and violence exposure specifically (VEX-R frequency) we performed correlation analyses with behavioral data, structural measures of the hippocampus and amygdala, and functional measures of bilateral hippocampal, amygdala, and vmPFC activation. Second, partial correlations correcting for age, sex, and ICV for structural measures were performed.

Exploratory regression analyses were performed for significant correlations to assess the effect of age group (<10 and ≥10) by creating interaction terms for trauma or violence exposure × age group and adding them to the model along with main effects.

Whole brain analyses and functional connectivity analyses were performed, and methods, results, and discussion of the findings are presented in the Supporting Information. Additional correlation analyses with PTSD symptoms and regression analyses assessing the differential effect of sex are also presented in the Supporting Information.

3 | RESULTS

3.1 | Participants

Sixty-nine African American children and adolescents (8–14 years) were scanned (Table 1) and data from these participants were included in the structural analyses, functional analyses, or both, resulting in different analytical datasets. Structural data of six participants was unusable for analyses due to motion, resulting in N = 63 for the structural analyses. Functional eGNG data were collected on 66 participants, however, 15 participants exceeded the motion threshold of 2 mm/TR, two participants fell asleep during the scan, and two participants did not (correctly) press any buttons, resulting in a sample of N = 47. Behavioral response inhibition data were available for 62 participants. No significant differences were observed in demographics for participants included in the structural and functional analyses. Age (in months) and sex were included as covariates in secondary analyses.

3.2 | Behavioral findings

The group means for accuracy, % correct Go’s, % correct NoGo’s, and overall Go reaction time are presented in Table 2. The means for the N = 62 participants did not significantly differ from means of the N = 47 included in the functional MRI analyses.

There were no significant correlations between trauma exposure and behavioral measures. There was a significant negative correlation of violence exposure with reaction time (r = −0.34, p = .008), however, after correcting for age and sex, this effect was no longer significant (r = −0.14, p = .263). Follow-up analyses showed a positive correlation between age and violence exposure (r = 0.32, p < .01).

| TABLE 2 | Behavioral data |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| N = 62 (behavioral data available) | N = 47 (included in fMRI analyses) |
| Mean | SD | Mean | SD |
| Accuracy (%) | 77.3 | 15.8 | 77.0 | 17.4 |
| Correct Go’s (%) | 85.4 | 16.2 | 84.0 | 17.5 |
| Correct NoGo’s (%) | 75.7 | 19.2 | 78.8 | 15.4 |
| Go reaction time (ms) | 830.4 | 204.6 | 832.9 | 212.5 |

Abbreviation: fMRI, functional magnetic resonance imaging.

| TABLE 1 | Demographics and clinical data (N = 69) |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Age (in months) | 130.0 | 19.4 | 99–177 |
| Sex (% females) | 52.2 |
| Household income (% <2019 federal poverty level) | 76.9 |
| Global trauma exposure (TESI) | 5.5 | 3.4 | 0–18 |
| Violence exposure (VEX-R) | 14.1 | 8.3 | 1–44 |
| PTSD symptoms | 14.7 | 11.5 | 0–41 |
| Meet for PTSD (DSM) | 15.9 |
| Anxiety symptoms T score (BASC) | 48.4 | 12.0 | 29–77 |
| >1SD above mean | 14.5 |
| Depression symptoms T score (BASC) | 50.5 | 10.15 | 37–80 |
| >1SD above mean | 16.1 |

Abbreviations: BASC, Behavioral Assessment System for Children; PTSD, posttraumatic stress disorder; TESI, Traumatic Events Screening Inventory; VEX-R, Violence Exposure Scale for Children-Revised.
3.3 | Trauma exposure

More trauma exposure correlated with smaller left and right hippocampal volume (Table 3a and Figure 1). After correcting for ICV, age, and sex, the negative correlation with the left hippocampus remained significant, and a positive correlation with the left amygdala was observed. Only the correlations with the left hippocampus survived correction for multiple comparisons. There was no effect of trauma exposure on functional measures.

### TABLE 3  Correlation analyses for trauma and violence exposure with structural and functional magnetic resonance imaging measures

<table>
<thead>
<tr>
<th>Brain volumes</th>
<th>Inhibition-related activation</th>
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<tr>
<td></td>
<td>L HPC</td>
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<tr>
<td>(a) Trauma exposure</td>
<td>( r ) = -0.33, -0.26, 0.07, -0.05</td>
</tr>
<tr>
<td>corrected for age, sex (and ICV for volumes)</td>
<td>( p ) = 0.008, 0.042, 0.607, 0.672</td>
</tr>
<tr>
<td>(b) Violence exposure</td>
<td>( r ) = 0.04, -0.01, 0.20, 0.15</td>
</tr>
<tr>
<td>corrected for age, sex (and ICV for volumes)</td>
<td>( p ) = 0.732, 0.965, 0.118, 0.23</td>
</tr>
</tbody>
</table>

Abbreviations: AMYG, left/right amygdala; L/R HPC, left/right hippocampus; ICV, intracranial volume; vmPFC, ventromedial prefrontal cortex.

Note: Bold values indicate a \( p < .05 \).

3.4 | Violence exposure

More violence exposure correlated with more activation in the bilateral hippocampus and amygdala, and marginally with the vmPFC (Table 3b and Figure 2). After correcting for age and sex, all three correlations were significant. Only the correlation with bilateral hippocampal activation survived correction for multiple comparisons. No correlations were observed between violence exposure and structural measures.

Exploratory regression analyses were performed to assess the effect of age group (<10, \( N = 15 \) vs. \( \geq 10, N = 32 \)) on the relation between violence exposure and functional outcomes, because significant correlations were observed in the initial analyses.
A significant interaction between age group and violence exposure was observed for hippocampal activation ($F_{(3, 46)} = 4.63, p = .007$; interaction, $t = -2.08, p = .043$) and amygdala activation ($F_{(3, 46)} = 3.59, p = .021$; interaction, $t = -2.42, p = .020$), such that significant correlations were only observed in younger children (hippocampus, $r = 0.59, p = .037$; amygdala, $r = 0.75, p = .003$).

**DISCUSSION**

The current study in 69 children and adolescents showed that more trauma exposure in general (including multiple types of trauma) was associated with structural changes in the hippocampus and amygdala, whereas more violence exposure specifically correlated with more functional changes in the amygdala, hippocampus, and vmPFC, particularly in children younger than 10 years of age. Furthermore, more violence exposure was related to stronger functional connectivity between the left amygdala and the brainstem (in the Supporting Information).

As hypothesized, more trauma exposure was associated with smaller (left) hippocampal volume. This effect has been demonstrated repeatedly in prior adult retrospective studies and a few pediatric studies, but here we build on prior work by showing this effect can already be observed during development in a nonclinical sample of children and adolescents. This is important given that reduced hippocampal volume, in turn, has been shown to be a risk factor for the development of PTSD (Gilbertson et al., 2002) and depression (Rao et al., 2010). We also showed that only trauma exposure more...
broadly, but not violence exposure specifically, was related to reduced hippocampal volume. Amygdala volume was found to be positively correlated with trauma exposure, which parallels studies in previously institutionalized children (Mehta et al., 2009; Tottenham et al., 2010), but contradicts studies on maltreatment showing reduced amygdala volume (Edmiston et al., 2011; Hanson et al., 2015; Luby et al., 2019; McLaughlin et al., 2016). However, as our amygdala finding did not survive correction for multiple comparisons, replication is warranted before further interpretation.

Further, increased levels of violence exposure were associated with more activation in the amygdala, hippocampus, and vmPFC during emotional response inhibition. These findings parallel studies in maltreated or neglected children who demonstrated more amygdala activation compared with controls (Maheu et al., 2010; McCrory et al., 2013; Tottenham et al., 2011), and increased hippocampal activation (Maheu et al., 2010) to emotional faces. Our findings also correspond with increased mPFC activation observed on a Go/NoGo task in children with trauma exposure and PTSS (Carrion et al., 2008) and adolescents with early-life stress (Mueller et al., 2010). However, these previous studies only demonstrated group differences and did not show a continuous association between trauma exposure and brain function as we have demonstrated. Only Suzuki et al. (2014) showed a dose-response relation between number of cumulative stressful and/or traumatic life events and amygdala, subgenual anterior cingulate cortex, and hippocampal activation in response to emotional faces. Furthermore, in a retrospective study in adults, more childhood trauma was found to positively correlate with hippocampal activation during a Go/NoGo task, but only in individuals with the COMT Val/Val genotype (van Rooij et al., 2016), suggesting the need for further assessment of genetic influences.

A possible explanation for the positive correlation between violence exposure and amygdala, hippocampus, and vmPFC activation is that children with high levels of violence exposure show an appropriate, increased attention-directing response. Following prior work by Tottenham et al. (2011), it could be suggested that increased amygdala activation is a manifestation of increased vigilance to emotional stimuli, provoked by exposure to violence in our population. Based on prior studies on the role of the hippocampus and vmPFC in fear regulation, it can be hypothesized that increased hippocampal recruitment could help children contextualize experiences, and augmented prefrontal control could regulate fear accordingly. Therefore, increased fear neurocircuitry activation with higher levels of exposure to violence may reflect an adaptive brain response to growing up in a dangerous, violent environment, especially since this association in our nonclinical population is only observed for violence exposure specifically, and not trauma exposure in general.

Levels of psychopathology in our population were relatively low considering the high levels of trauma exposure. Moreover, there was no relation between PTSD symptoms and brain structure or function (in the Supporting Information). Importantly, while heightened neural activation may be an adaptive mechanism during childhood in an adverse environment, the long-term potentially excitotoxic effects of this over-engagement of the fear neurocircuitry and its risk for later psychopathology are not clear. It is possible that chronic, adaptive vmPFC and hippocampal overactivation in childhood lead to maladaptive vmPFC and hippocampal underactivation in adulthood, patterns observed in adults with PTSD (Jovanovic et al., 2013; van Rooij et al., 2016). This pattern was suggested by Tarullo and Gunnar (2006) as the explanation for why maltreated children over-secrete cortisol while adults with childhood maltreatment under-secrete cortisol. Yet, as our study population is not a clinical population, many of the participants may become resilient adults and these brain alterations may promote this as suggested in van Rooij et al. (2016). Therefore, based on this study we cannot conclude whether the observed correlation between violence exposure and brain activation is a marker of future resilience or psychiatric risk.

As hypothesized, we observed an effect of developmental stage on our functional outcomes. Variability in the amygdala, hippocampus, and vmPFC activation was tightly linked with violence exposure specifically in younger children. This could be explained by a variety of maturation processes, such as critical periods for brain development (Knudsen, 2004), prefrontal maturation during adolescence (Caballero, Granberg, & Tseng, 2016), changes in amygdala-vmPFC functional and structural connectivity (Gee et al., 2013; Jalbrzikowski et al., 2017), improvements in safety signal processing in older children (Jovanovic et al., 2014), and changes in social functioning, though these hypotheses require further exploration. Notably, the sample size of the younger children was relatively small (N = 15) and replication in a larger sample is warranted. However, this is a very difficult sample to collect and therefore largely understudied, and much needed data to be added to the literature.

Other limitations of this study included that it is a cross-sectional study, and therefore no directional conclusions can be drawn from this data, and it is unclear if the associations we observed between brain function and violence exposure indicate a long-term protective or harmful effect. Second, the participants have a somewhat wide age range (8–14) across pre- and postpubertal developmental stages. Though we assessed the effects of age by stratifying participants by age group (<10 and ≥10) and included age in months as a covariate in our secondary analyses, it would be informative to more carefully control for age and pubertal stage in future studies. Third, the findings in our high-risk African American population from inner-city Atlanta may not generalize to other populations. Finally, many children who grow up in an unsafe environment live below the poverty line. Research has shown pervasive effects of poverty on brain structure (Hair, Hanson, Wolfe, & Pollak, 2015), including the hippocampus and amygdala (Luby et al., 2013). In this study, we did not separately assess the effects of poverty, because the majority of our participants were from low-income families with little variation to include in the analyses. On the other hand, this population therefore better allowed us to examine the effects of trauma and violence exposure than in studies with a wide income range where poverty effects can confound the effects of trauma/violence.
5 | CONCLUSION

In this neuroimaging study in an at-risk pediatric population ages 8–14, we observed (a) an association between structural brain alterations and childhood trauma more generally, and (b) functional changes which correlated with violence exposure specifically. Based on the current findings, it could be hypothesized that general trauma exposure during childhood results in structural changes in the hippocampus (and amygdala) that are associated with later risk for psychiatric disorders. At the same time, it can be postulated that growing up in an unsafe environment with high levels of violence exposure leads the brain to functionally adapt to this situation in a way that promotes survival, where the long-term costs or consequences of these adaptations are largely unknown and an area for future investigations. Given the importance of the fear neurocircuitry for psychiatric disorders, increased understanding of the effects of trauma exposure on the developing brain is essential for early detection of individuals at risk for developing psychiatric disorders.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

DATA AVAILABILITY STATEMENT

Part of the data is shared through RDoCdb (NIH data archive). The other data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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