



Regular Article

Impacts of early life adversity on the neurocircuitry of emotional memory in children

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Abstract

Similar to adults with posttraumatic stress disorder, children with early life adversity show bias in memory for negative emotional stimuli. However, it is not well understood how childhood adversity impacts mechanisms underlying emotional memory. $N = 56$ children (8–14 years, 48% female) reported on adverse experiences including potentially traumatic events and underwent fMRI while attending to emotionally pleasant, neutral, or negative images. Post-scan, participants completed a cued recall test to assess memory for these images. Emotional difference-in-memory (DM) scores were computed by subtracting negative or positive from neutral recall performance. All children showed enhancing effects of emotion on recall, with no effect of trauma load. However, children with less trauma showed a larger emotional DM for both positive and negative stimuli when amygdala or anterior hippocampal activity was higher. In contrast, highly trauma-exposed children demonstrated a lower emotional DM with greater amygdala or hippocampal activity. This suggested that alternative neural mechanisms might support emotional enhancement of encoding in children with greater trauma load. Whole-brain analyses revealed that right fusiform activity during encoding positively correlated with both trauma load and successful later recall of positive images. Therefore, highly trauma-exposed children may use alternative, potentially adaptive neural pathways via the ventral visual stream to encode positive emotional events.

Keywords: Childhood trauma; Emotional episodic memory; early life adversity; fMRI; ventral visual cortex

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Introduction

Trauma can exert both impairing and facilitating effects on memory for emotional events. Individuals with posttraumatic stress disorder (PTSD) may experience inability to recall specific details of traumatic experiences, which are memories imbued with high levels of negative emotion (American Psychiatric Association, 2013) as well as more general deficits in long-term memory (Petzold & Bunzeck, 2022; Zlomuzica et al., 2018). However, it appears that negatively valenced information is remembered better by adults with PTSD compared to otherwise healthy individuals (Durand et al., 2019; Imbriano et al., 2022). In both healthy adults and children (Dolcos et al., 2017; Massol et al., 2021; Stenson et al., 2019) it has been shown that emotion enhances memory, but few neuroimaging studies have investigated the impact of trauma on children's emotional memory systems. By understanding emotional memory encoding for children who experience psychological trauma during an important window of development, we may

begin to understand how childhood adversity is linked to persistent brain changes that can increase transdiagnostic risk for psychiatric disorders in adulthood (McKay et al., 2022; Vrijns et al., 2017).

Emotion plays an important role in the encoding of episodic memories, a type of hippocampal-dependent long-term memory for the conscious recollection of personal experiences. Emotional enhancement of memory has been well documented in both adults (for review, see Dolcos et al., 2017) and children (Hamann & Stevens, 2013; Massol et al., 2021; Stenson et al., 2019). Studies typically look at the difference-in-memory (DM) for emotional stimuli by examining the difference in either memory performance or neural responses, for negative or positive items versus neutral items (Wagner et al., 1998). Children typically remember negative and positive images better than neutral, with a larger effect seen for recall of negative images (Massol et al., 2021; Stenson et al., 2019). The encoding of emotionally arousing information relies on interactions between the amygdala and medial temporal lobe areas such as the hippocampus (Dolcos et al., 2004; Murty et al., 2010; Pinabiaux et al., 2013; Qasim et al., 2023), as well as aspects of the prefrontal cortex (PFC) and visual regions (Dahlgren et al., 2020; Kaneda et al., 2017; Krauel et al., 2007). These same regions are frequently implicated in the pathophysiology of PTSD (Pitman et al., 2012; Shin & Liberzon, 2010). Children exposed to early life adversity show similar hyperresponsiveness in the amygdala and

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other salience regions (Hein et al., 2020; McLaughlin et al., 2015; Suzuki et al., 2014; White et al., 2019; van Rooij et al., 2020), as well as altered engagement of regions associated with contextual encoding like the hippocampus and medial PFC (mPFC; Herringa et al., 2013; Krugers et al., 2017; van Rooij et al., 2020). Although these findings have primarily been observed in studies of emotional processing rather than memory, there is a notable potential functional relevance to emotional declarative memory.

Trauma exposure facilitates some components of memory while disrupting other components in children. One study of children and adolescents with PTSD showed that the PTSD group had poorer overall recall for both positive and neutral words compared to healthy children (Moradi et al., 2000). However, these same youths with PTSD performed similarly to the healthy control group for memory for negative images, suggesting a bias toward recalling negatively valenced words. It is worth noting that the authors did not see the typical pattern of emotional enhancement on memory within either the healthy or clinical groups of their sample, so further work may be needed to address differences in memory for emotional words among both healthy youth and those with PTSD. Violence-exposed children compared to unexposed children showed worse memory for context-specific learning paired with angry faces, despite greater engagement of a functional PFC-hippocampal circuit typically linked with successful memory encoding (Lambert et al., 2017). This suggested that violence-exposed children may be attending to threatening cues and not encoding broader contexts. Violence-exposed children also show disruption of associative learning for neutral objects paired with threatening cues, relative to unexposed children. This pattern was accompanied by reduced engagement of the intraparietal sulcus (IPS) but increased right middle frontal gyrus (rMFG) activity (Lambert et al., 2019). As previously mentioned, all children demonstrate a slight bias for negative emotional memory (Massol et al., 2021), but these studies suggest that children exposed to trauma show heightened bias toward encoding negative emotional information at the expense of neutral information, associated with activation of frontoparietal executive regions. However, this work used paired associative learning in which negative emotional stimuli typically distracted attention away from item encoding. It is important to understand how traumatic experiences might alter the simple encoding of negative and neutral stimuli specifically to help us further understand the development of trauma-related disorders.

Positive emotional memory may also be impacted by trauma exposure. In trauma-exposed adults, PTSD symptoms are negatively correlated with the ability to recall positive memories (Contractor et al., 2019; Dolan et al., 2020; McNally et al., 1995). Similarly, children with PTSD show a reduced ability to recall positive words (Moradi et al., 2000). The neural correlates of this effect are unknown. In healthy adolescents, successful recall of positive images is facilitated by greater amygdala activation during encoding (Vasa et al., 2011). However, as previously discussed, children exposed to trauma already demonstrate hyperactive amygdala responses to emotional stimuli. Therefore, it is not clear what neural mechanisms may impact positive emotional memory encoding.

We sought to examine the effects of childhood trauma on neural processes supporting emotional episodic memory. We recruited parent-child dyads from community samples at high risk for trauma exposure (Stevens et al., 2021; van Rooij et al., 2020). Children were between the ages of 8–14 years, a sensitive window for the development of emotional neurocircuitry (Herringa, 2017;

Stevens et al., 2018). Children underwent functional magnetic resonance imaging (fMRI) while attending to emotionally positive, neutral, or negative images. Following the scan, children completed a cued recall task testing for images seen in the scanner. We hypothesized that children with greater trauma load would show a greater DM for negative-neutral images but smaller DM for positive-neutral images and overall memory performance, similar to patterns observed in adults with PTSD (Durand et al., 2019; Imbriano et al., 2022). Based on neuroimaging studies of emotional reactivity in trauma-exposed children (van Rooij et al., 2020), we predicted greater amygdala and hippocampal activity during the encoding of negative emotional stimuli, as well as lower IPS and greater rMFG activity, following Lambert et al (2019). Based on findings in healthy adolescents (Vasa et al., 2011), we predicted that greater positive DM would be associated with heightened amygdala activation during encoding, but it was unclear how trauma would influence that relationship.

Methods

Participants

Sixty-eight children (33 girls, 35 boys) ages 8–14 years were recruited from an ongoing study of mother and child pairs approached at primary care clinics at Grady Memorial Hospital. This publicly-funded hospital serves primarily low-income patients in the Atlanta area with high rates of trauma exposure (Gluck et al., 2021). All participants were Black American, reflecting the underlying population of patients served in the Grady Healthcare System, and the surrounding Atlanta area. As discussed in previous studies, children in this sample reported high frequencies of exposure to violence and other types of trauma (Stevens et al., 2021; van Rooij et al., 2020). Participants were excluded if they met diagnosis for autism spectrum disorder, bipolar or psychotic disorders, neurological disorder, or cognitive disability. Children received a study t-shirt, a small toy, and a \$10 gift certificate, and mothers received \$50 for each study visit. Study procedures were approved by the Emory University Institutional Review Board and the Grady Hospital Research Oversight Committee. A parent or legal guardian provided written informed consent and children gave written assent prior to research participation.

Of the $n = 68$, 6 children were excluded from analysis due to excessive head motion during the scan [further detail in section 2.4.2], 4 had low behavioral response during the encoding task ($<68\%$ responsiveness likely indicating sleepiness), and 2 did not complete the recall task outside the scanner. The final sample for analysis included $n = 56$ ($M = 10.09$ years, $SD = 1.33$ years; 27 girls, 29 boys) (Table 1).

Trauma load and symptom assessment

Children's self-report of trauma load was measured using the Traumatic Events Screening Inventory for Children (TESI-C) version 8.4 (Ford et al., 2002). This questionnaire uses child-appropriate language to ask ("yes" vs "no") if the child has been exposed to 19 potentially traumatic or adverse experiences. These events include witnessing or exposure to serious accidents, hospitalization, natural disaster, loss of a relative or close friend, extended separation from caregivers, family or community conflict or violence, and kidnapping. Following Cross et al (2018), item number 5 regarding sexual molestation was omitted during the interview, resulting in 18 potential types of traumatic events.

Table 1. Demographic and clinical characteristics ($n = 56$)

	Descriptives	Correlation with Trauma Load (TESI-C)
	Mean (SD) or %	Chi-square test
Sex	48.2 % female	$\chi^2 = 7.26, p = 0.61$
Household Income	63.6% < 2013–19 federal poverty level	$\chi^2 = 38.2, p = 0.37$
		Spearman Correlation
Age (years)	10.09 (1.32)	0.178 ($p = 0.19$)
Pubertal Development Scale (PDS)	1.51 (0.71)	−0.115 ($p = 0.40$)
Traumatic events screening inventory for children (TESI-C)	4.26 (2.62)	–
At least 1 event (TESI-C > 0)	91.07%	–
PTSD symptoms (UCLA child report)	17.42 (13.17)	0.607 ($p < 0.001$)
Met for PTSD diagnosis (DSM)	19.64%	–
Anxiety symptoms T-score (BASC)	48.72 (10.3)	0.461 ($p < 0.001$)
Depression symptoms T-score (BASC)	48.39 (8.17)	0.241 ($p = 0.08$)

Trauma load was defined as the number of different traumatic events endorsed by the participant, which has been shown to have a dose-dependent impact on PTSD risk and symptom severity (Kolassa *et al.*, 2010; Neugebauer *et al.*, 2009).

In order to further characterize the sample of participants, we collected other psychiatric and demographic measures (Table 1). PTSD symptoms and diagnosis were determined using the child-report UCLA PTSD Reaction Index (UCLA-RI; range 0–41) (Steinberg *et al.*, 2004). Anxiety and depression symptoms were also evaluated using the Behavior Assessment System for Children (BASC) (Reynolds *et al.*, 2011). Children's pubertal status was measured using the Pubertal development scale (PDS; Petersen *et al.*, 1988).

Procedures

The day before the MRI scan, children completed a training session using a mock MRI scanner. During this training session, participants viewed a movie while wearing a head-mounted motion tracker (MoTrak® | Psychology Software Tools ([n.d.](https://www.psychologysoftwaretools.com/))). During a 15-minute session, the movie would pause if participants moved more than 2–22 mm, with the goal of getting head motion down to less than 2 mm. This training method has been shown to help minimize head motion during real fMRI data collection (Raschle *et al.*, 2012).

On the day of the actual MRI scan, children underwent an MRI-adapted emotional memory encoding and retrieval task modified from Leventon *et al.* (2014). During the emotional encoding phase, participants viewed emotional scene stimuli while undergoing a fMRI scan. Thirty minutes after the scan, participants completed a cued recall task to assess for memory of the images seen during encoding.

fMRI emotional encoding phase

90 child-appropriate scene stimuli were selected (30 positive, 30 neutral, 30 negative), using a version of the task from Leventon *et al.* (2014), with stimulus presentation timing modified for fMRI. The images originated from the International Affective Picture System (IAPS; Lang *et al.*, 2008) and were supplemented with similar

images identified from web searches of public image repositories. Images of weapons, mutilated bodies, or sexual content were not selected. Half of the images within each emotion condition (positive, negative, neutral) displayed scenes without humans to control the proportion of social stimuli across conditions (Proverbio *et al.*, 2009). Parents or guardians were sent thumbnails of all images via email for approval before all study visits and indicated any images they did not approve. Up to 6 images were replaced with a set of parent-approved replacement images within the same emotion category, on a participant-by-participant basis.

During fMRI recording, images were shown in a pseudo-randomized order so that no one condition appeared more than twice in a row (i.e. positive, positive, neutral). The images and condition order were counterbalanced across participants. At the start of each trial, images were surrounded by a blue border for 1500ms. Then the border turned green for an additional 1500ms, and children were asked to make a button selection to indicate whether the picture scene contained a person or part of a person. These button selections were used to ensure participant engagement during the encoding task. An intertrial interval of 1500 – 3500 ms was used. At the end of the encoding task, an additional five very happy scenes were shown to end the session on a positive note. These additional five trials were not included in any analyses and controlled for in the implicit baseline.

Post-scan cued recall assessment

30–45 minutes after the fMRI emotional encoding task, children completed a surprise cued recall assessment to test their memory for all 90 images seen during fMRI. Following Dolcos *et al.* (2004), participants were given a verbal recall cue (i.e. “Farmer”) and then asked to verbally describe any details they could remember about the corresponding image (i.e. “His hand was in the dirt”). Children were informed that each cue may or may not correspond to an image they saw during the fMRI scan, and they could say “pass” if they did not recall a corresponding image. Two researchers independently scored the participant descriptions to determine whether an image was correctly remembered (“yes” vs “no”). To be scored as remembered, participants had to describe at least 1 correct detail about the image which could not have been inferred

from the cue word alone. Discrepancies in scores were settled by a third researcher.

Recall scores for each emotional condition were calculated by summing the number of images correctly recalled for a particular condition and dividing it by 30, which was the total number of images in any condition. A higher score indicated better recall of a particular emotion category. Additionally, an overall recall score was calculated as the proportion of all items correctly recalled. Finally, emotion-related DM scores were created, to index the enhancing effects of positive or negative emotion on recall performance. The positive DM was calculated as the proportion of positive minus neutral items correctly recalled, and the negative DM was calculated as the proportion of negative minus neutral items correctly recalled.

Image acquisition and processing

Data acquisition

Brain images were collected using two 3T Siemens Magnetom TrioTim scanners with a 32-channel head coil at Emory University Facility for Education and Research in Neuroscience. 25 children were scanned with the first TrioTim scanner, and the remainder were scanned using the second scanner of the same type. Participants viewed a short movie while structural T1-weighted volumes were collected using a multi-echo MPRAGE sequence (TR: 2250ms, TE: 4.18 ms, flip angle: 9°, FOV: 256 mm, 176 slices, voxel size: 1x1x1 mm). During the memory encoding task, functional images were acquired using a gradient-echo T2*-weighted EPI sequence, with images gathered in a descending order of 44 slices, and a 0.5 mm slice gap (TR: 2330ms, TE: 30 ms, flip angle: 90°, FOV: 204 mm, voxel size: 3x3x2.5 mm).

Preprocessing and quality assurance

For full preprocessing details, see Supplementary Materials. EPI images were coregistered to T1w space, spatially realigned, slice-time corrected, and normalized to ICBM 152 Nonlinear Asymmetrical template using fMRIPrep standard procedures (version 2.2.0; Esteban et al., 2018). Automatic removal of motion artifacts using independent component analysis (ICA-AROMA, Pruim et al., 2015) was performed to correct for motion artifacts after spatial smoothing with a Gaussian kernel of 6 mm FWHM. In cases where excessive head motion was too high for ICA-AROMA correction, an overall motion threshold was used to exclude subjects exceeding 1.5 mm framewise displacement on more than 15% of volumes.

1st and 2nd level analyses were conducted in SPM12 (<https://www.fil.ion.ucl.ac.uk/spm/>). At the first level, image stimuli were modeled with an event-related design using onset times for negative, neutral, and positive images and resulting in 3 conditions of 30 trials each. Additionally, onset times for the extra 5 happy scenes shown at the end of the encoding task were modeled in order to remove their influence on the implicit baseline. A duration of 0 ms was used for each event, along with a 128-second high-pass filter, subject specific explicit masks, and autoregressive serial correlations. Linear contrasts compared negative > neutral trials and positive > neutral trials. Global signal, CSF, and white matter were included as covariates of no interest in 1st-level models.

The REX toolkit (<https://www.nitrc.org/projects/rex/>) was used to extract beta values for the following regions of interest (ROIs): bilateral amygdala defined by California Institute of Technology (CIT168) high-resolution in vivo amygdala atlas (Tyszka & Pauli, 2016), bilateral hippocampal regions based on the Harvard-Oxford

probabilistic subcortical atlas thresholded at 75% (<https://fsl.fmrib.ox.ac.uk/fsl/fslwiki/Atlases>), and left IPS and right MFG, for which 10 mm spheres around peak voxels (MNI coordinates: -18, -48, 60 for L IPS & 46, 30, 38 for R MFG) showed effects in a previous childhood trauma and memory study (Lambert et al., 2019). The hippocampus was divided along the long-axis, and the most anterior and posterior aspects were selected (Murty et al., 2010) to account the established gradient of affective involvement and item versus spatial memory toward the more anterior regions.

Statistical analyses

Recall assessment

In order to understand the magnitude of effects for differences between positive, negative, and neutral recall scores, one-way repeated measures ANOVA was used to evaluate whole group recall differences for each of the emotional conditions (positive, neutral, negative). Previous work has shown that emotional episodic memory performance may be impacted by age and sex (Pauls et al., 2013; Stenson et al., 2019), and we therefore included these terms as moderators to fully explore their effects on cued recall performance. To test for sex effects on recall performance we used repeated measures ANOVA with emotion condition as a within-subjects factor, with sex as a between-subjects factor, and using Benjamini-Hochberg correction. To explore age effects and test whether memory worsened with trauma load, we conducted Spearman correlations after confirming non-normality (Shapiro-Wilk $p = 0.001$ for age and $p = 0.03$ for TESI-C). Paired t-tests for the emotion comparisons with age and TESI-C were corrected using Bonferroni methods.

ROI analyses

To confirm the responsivity of the ROIs to affective scene content, we conducted random effects models for each ROI, with the primary predictor being emotion condition (positive, negative, neutral). For bilateral ROIs, hemisphere (left, right) was also included as a second term. For the hippocampus, the division along the longitudinal axis (anterior, posterior) was included as a third term. Next, to test the hypothesis that trauma load would be associated with differences in ROI activity, we performed mixed-effects ANOVAs for each of our ROIs that included TESI-C scores as a continuous, between-subjects factor and emotion condition, as well as hemisphere and anterior/posterior (A/P) when applicable, as within-subject factors.

To investigate the neural impacts of trauma on emotional memory, we looked at ROI activity during encoding associated with TESI-C scores and later emotional DM recall. Additionally, since sex was a significant predictor of recall performance, it was included as a covariate for all models evaluating interactions with memory. As previously demonstrated, test-retest reliability in subcortical BOLD signal is poor for remembered vs forgotten trials, especially in the hippocampus. Whereas encoding of all trials shows relatively reliable test-retest signal across most regions of interest (Tang et al., 2021). Therefore, to examine trauma-related differences in the enhancing effects of positive emotion on encoding, we focused on ROI activity extracted from positive and neutral trials, modeling regional activation associated with the positive DM. Models of the negative DM included ROI activity from negative and neutral trials. General linear models for each of the ROIs was conducted with TESI-C and recall performance scores as continuous, between-subjects factors and emotion

condition, as well as hemisphere and A/P when applicable, as within-subject factors.

In follow-up analyses, we wanted to test whether effects of adversity on neural responses during memory encoding in turn predicted symptoms of PTSD and internalizing. We performed post-hoc analyses using the negative > neutral or positive > neutral neural activity from ROIs that showed a significant emotional DM by trauma load interaction. We performed correlations with these regions and our symptom measures for PTSD, anxiety, and depression. Because the symptom scores were skewed (Shapiro-Wilk p s < 0.05 for PTSD symptoms (UCLA), anxiety symptoms (BASC), and depression symptoms (BASC)), Spearman correlation was used.

Whole-brain analysis

To test whether trauma load or recall performance were associated with differences in regions external to the a priori ROIs during emotional encoding, exploratory group-level multiple regression analyses were performed in SPM12. In models evaluating positive stimuli encoding and recall, neural activation for positive > neutral images were regressed with positive DM recall scores. Similarly, negative stimuli encoding and recall models used negative > neutral whole-brain contrasts and negative DM scores. Subsequent memory analyses were not conducted given prior evidence that test-retest reliability is restricted in developmental studies using a subtraction of recalled versus forgotten images, compared to analysis of all encoding trials (Tang *et al.*, 2021). All whole-brain analyses used an initial cluster-forming threshold of $p < 0.005$ and a cluster-wise false discovery rate correction at $p < 0.05$. Peak voxel activity from these regressions were used to create 10 mm spheres that were then correlated with trauma load with sex as a covariate of no interest.

Follow-up correlations tested whether whole-brain findings related to emotional memory and trauma load in turn predicted symptoms of PTSD and internalizing. We performed post-hoc Spearman correlations with PTSD, anxiety, and depression scores. Contrast estimates were extracted from a 10 mm sphere centered on peak coordinates for any cluster that significantly correlated with both emotional DM and trauma load.

Results

Trauma and symptom assessments

As reported in Table 1, the average PDS score was a 1.49 out of 5, indicating that most children in this sample were early in pubertal development, with most children (63.6%) belonging to low-income households.

Children in our sample had relatively high rates of exposure to adversity. On average, children reported 4.26 different types of adversity, with 51 out of 56 (~91%) having experienced at least one major adverse event in their lifetime (Table 1). However, only about 20% of the children met criteria for PTSD, and the average UCLA symptom score of 17.42 was well below the clinical cutoff of 35. Additionally, anxiety and depression symptoms were not elevated (t -scores ~48–49 percentile on a normative scale). As previously discussed in our other works, these relatively low levels of psychopathology in the face of high trauma exposure may indicate some resilience in our study sample (van Rooij *et al.*, 2020).

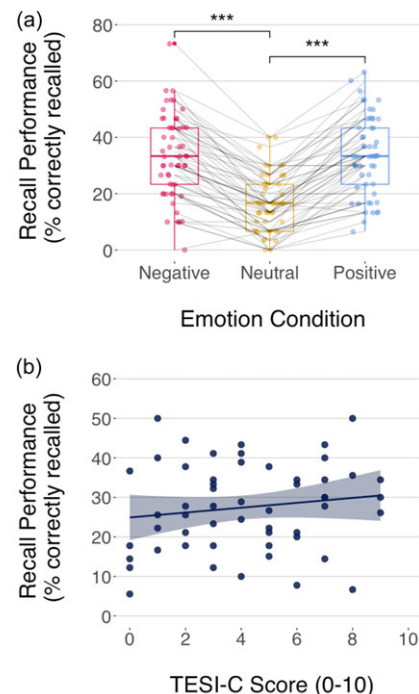


Figure 1. Recall performance in the whole sample. (a) Children showed greater recall for positive ($M = 0.34$, $SD = 0.14$) and negative ($M = 0.33$, $SD = 0.15$) images compared to neutral images ($M = 0.17$, $SD = 0.12$). Pairwise t -tests adjusted using Bonferroni, *** = $p < 0.001$. (b) Negative, neutral, positive, or overall recall performance did not vary by trauma load. Error bars depict standard error.

Cued recall

In the whole group, cued recall performance varied by emotion (Figure 1a; $F(2,106) = 91.29$, $p < 0.001$). There was an enhancing effect of emotion on memory, such that participants had better recall of positive or negative images when compared to neutral images (p 's < 0.001). There was no difference in recall of positive and negative images ($p = 0.91$). Interestingly, neither trauma load, age, nor their interaction (TESI*age) correlated with overall cued recall performance or recall for positive, neutral, or negative scenes specifically (p 's > 0.05, Figure 1b & S1A). There was a significant sex by emotion recall interaction in which girls typically showed greater cued recall performance than boys (Figure S1B; $F(1,52) = 8.69$, $p < 0.001$). Post-hoc tests showed that girls compared to boys had greater negative ($t = 3.23$, $p = 0.01$), neutral ($t = 2.46$, $p = 0.03$), and overall ($t(46.87) = 2.91$, $p = 0.02$), but not positive ($t(50.4) = 1.95$, $p = 0.08$) recall performance. Due to these significant effects, sex was used as a covariate in all neuroimaging analyses that evaluated interactions with memory performance.

Trauma load associations with regional brain activity and difference-in-memory recall performance

In the hippocampus, there was an interaction between trauma load*positive DM*hemisphere*A/P ($F(1,50) = 9.44$, $p = 0.004$). Post-hoc tests by hemisphere and A/P showed the trauma*positive DM interaction only within the left anterior hippocampus (Figure 2a; $F(1,50) = 6.77$, $p = 0.01$) indicating that greater left anterior hippocampal activation was associated with better positive DM memory performance in children with low trauma exposure, whereas lower activation was correlated with better memory in

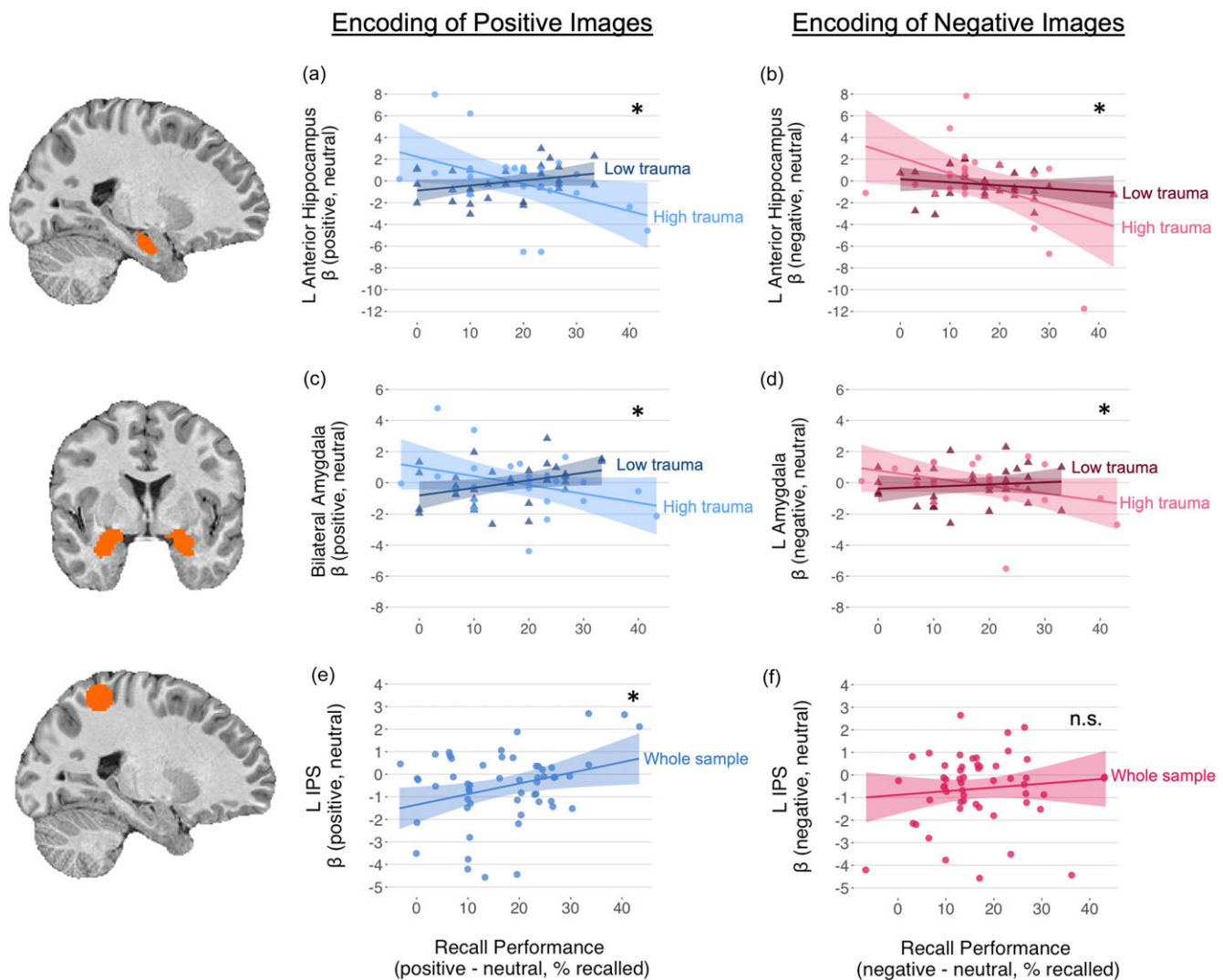


Figure 2. Regions of interest activation associated with emotional difference-in-memory recall. Left anterior hippocampal response during emotional encoding is associated with differences in later positive (a) and negative (b) recall performance for high vs low trauma load. (c) Bilateral amygdala and (d) left amygdala response was associated with differences in later recall performance for high vs low trauma load. Left IPS response during encoding of positive (e) or negative (f) and neutral images associated with recall performance in the whole group. CR/RM ANOVA evaluated interactions among emotion*hemisphere*trauma load*recall performance (*A/P in the hippocampus only). For visualization of these effects, a median split was used to visually divide the sample into low (TESI-C ≤ 4 , triangles) and high (TESI-C > 4 , circles) trauma groups (although trauma load was quantified as a dimension in statistical models). * $p < 0.05$.

children with higher exposure. Of note, trauma load was evaluated as a continuous variable (TESI-C scores), but figure graphs depict low vs high trauma groups for easier visual interpretation (Figure 2a–d).

Similarly, in negative emotion DM models with the hippocampus there was an interaction between trauma load*negative DM*hemisphere*A/P ($F(1,50) = 15.93$, $p < 0.001$). This effect was again significant in the left anterior hippocampus only (Figure 2b; trauma load*negative DM; $F(1,50) = 5.49$, $p = 0.02$). Children with higher trauma load showed a negative association between left anterior hippocampal engagement during encoding and the subsequent effect of negative emotion on recall performance. In contrast, children with lower trauma load did not show much of a relationship between hippocampal activation and negative DM memory.

In the amygdala, the omnibus test showed an interaction between trauma load and the positive DM (Figure 2c;

$F(1,50) = 4.12$, $p = 0.048$), and no interaction with hemisphere. Children reporting lower trauma showed enhanced post-scan recall for positive images when bilateral amygdala activity was higher during encoding. In contrast, children with higher trauma load demonstrated worse positive DM with greater amygdala activity during encoding. In the negative emotion model there was an interaction of trauma load*negative DM*hemisphere ($F(1,50) = 5.79$, $p = 0.02$). Further testing by hemisphere revealed that the interaction was primarily observed in the left amygdala (Figure 2d; left: $F = 6.46$, $p = 0.01$; right: $p = 0.86$). Children with higher trauma load demonstrated less of a DM for negative-neutral images when amygdala activity was greater during encoding.

There were no significant interactions between trauma and negative or positive DM effects on cued recall for the right MFG or left IPS ($p > 0.05$). However, within the whole sample, left IPS activity positively predicted later recall of positive-neutral images ($F(1,50) = 7.20$, $p = 0.01$; Figure 2e).

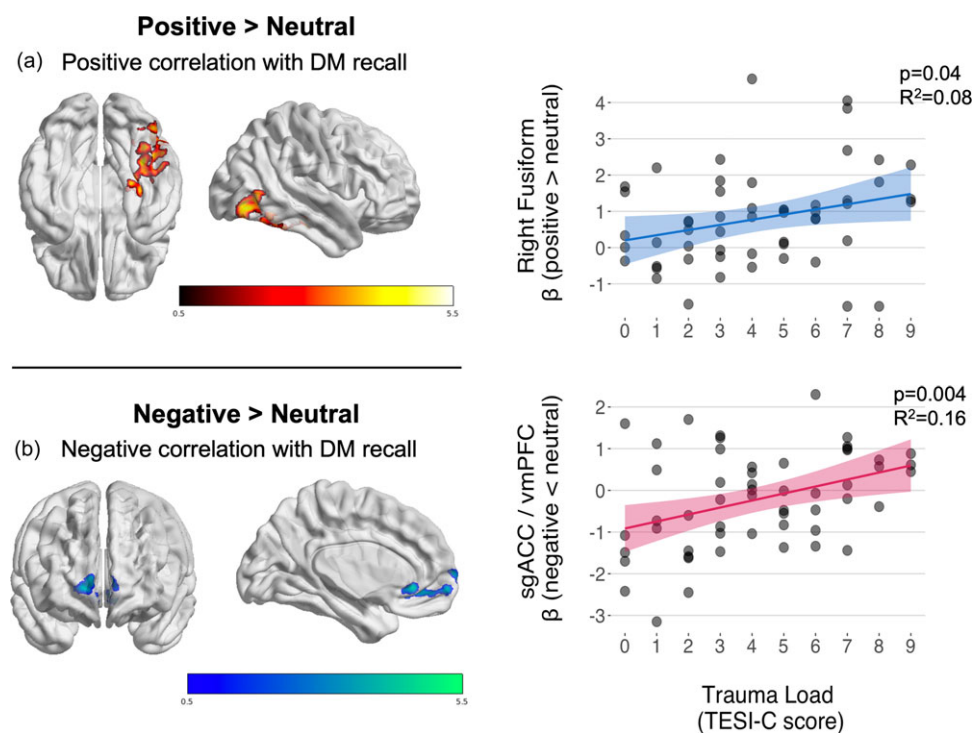


Figure 3. Whole-brain associations with emotional difference-in-memory (DM) effects, and relation to trauma exposure. (left) whole-brain activation along the right ventral visual stream for the association between the positive DM and positive > neutral encoding-related activation (a), as well as the negative DM and negative > neutral encoding-related activation (b). Peak voxel activity within these clusters were used to create 10 mm sphere ROIs for subsequent analyses. (right) right fusiform and sgACC/vmPFC activation in these clusters was significantly correlated with trauma load.

Effects of whole-brain activity during affective scene encoding on recall performance

We then explored additional brain regions whose engagement during encoding predicted later recall, and tested whether any of these regions' responses could explain the positive and negative DM effects on recall performance in children with higher trauma load. These whole-brain analyses showed four clusters of activation associated with a greater positive DM effect on cued recall performance (Figure 3). The positive DM was correlated with a greater response to positive > neutral images in the right and left fusiform and visual regions (Table 2; Figure S5). The positive DM was negatively correlated with activation to positive > neutral images in the dorsomedial prefrontal cortex (dmPFC) (Table 2; Figure S6). These regions' engagement predicted an enhancing effect of positive emotion on recall in the full sample. Using a 10 mm sphere, we extracted the contrast value for positive > neutral from each of these regions, in order to test whether children with higher trauma load engaged these regions preferentially. Linear regression revealed no significant correlations with trauma load in the left or right visual cortex (left: $R^2 = 0.07$, $p = 0.35$; right: $R^2 = 0.03$, $p = 0.40$), left fusiform gyrus ($R^2 = 0.01$, $p = 0.63$), or dmPFC ($R^2 = 0.01$, $p = 0.82$). However, trauma load did significantly correlate with activation in the right fusiform gyrus ($R^2 = 0.08$, $p = 0.04$). This suggested that children with greater trauma load show better positive DM recall when the right fusiform gyrus is more active during encoding.

The negative DM effect on recall performance was negatively correlated with negative > neutral encoding in the subgenual anterior cingulate cortex / ventromedial prefrontal cortex (sgACC / vmPFC) with some overlap in the left calcarine region and right (Table 2; Figure S7). Similar to the positive DM models, a 10 mm sphere was used to extract the contrast value for negative > neutral from these regions. ROI activity in the sgACC/vmPFC region significantly correlated with increasing trauma load ($R^2 = 0.16$,

$p = 0.004$), suggesting that children with higher trauma load showed greater functional activity for negative > neutral images in a region related to smaller negative DM effects on recall performance. There were no significant correlations for trauma load with the left calcarine ($R^2 = 0.04$, $p = 0.25$) or right PFC ($R^2 = 0.03$, $p = 0.63$).

Post-hoc associations between encoding-related brain activity and symptoms of PTSD, anxiety, and depression

Although children in our sample were shown to be somewhat resilient to the effects of their trauma and experiences of adversity (see Results 3.1), we wanted to test whether PTSD or internalizing symptoms might correlate with the neural patterns that were linked to emotional memory and children's exposure to adversity. Neither PTSD, depression, nor anxiety symptom severity were correlated with activation of the left anterior hippocampus, amygdala, or right fusiform gyrus ($ps > 0.05$). However, similar to the pattern of associations observed for trauma load, the engagement of the sgACC/vmPFC cluster to negative > neutral scenes was positively correlated with PTSD ($R^2 = 0.081$, $p = 0.043$) and anxiety ($R^2 = 0.079$, $p = 0.046$) symptoms. This suggested that children who were more symptomatic had greater engagement of this region during the encoding of negative > neutral images, but given that this region was negatively associated with the negative DM in recall performance, it may interfere with the encoding of negative stimuli in children who have experienced greater lifetime adversity and current symptoms of PTSD and anxiety. There was no significant correlation for depression with this region.

Discussion

This was one of the first neuroimaging studies to look at effects of childhood adversity, including experiencing potentially traumatic events, on children's emotional episodic learning. The findings

Table 2. Whole-brain regressions with positive and negative difference in memory (DM)

Anatomical Region	Correlation	x	y	z	voxels	z-max
Positive DM regressions						
Fusiform (R)	positive	38	−54	−14	1563	4.62
Fusiform (L)	positive	−36	−64	−10	1260	4.32
Visual (R)	positive	36	−84	32	485	3.98
Visual (L)	positive	−26	−84	24	213	3.63
Dorsomedial Prefrontal Cortex	negative	0	42	26	390	4.28
Negative DM regressions						
Subgenual anterior cingulate cortex / ventromedial prefrontal cortex	negative	4	48	−6	617	4.94
Calcarine (L)	negative	−8	−64	18	242	4.32
Prefrontal Cortex (R)	negative	28	58	22	243	3.95

highlight an important, potentially adaptive neural pathway facilitating memory for positive emotional stimuli. Although children in this sample had high rates of trauma exposure, symptoms and diagnoses of psychopathology were relatively low. Unexpectedly, children with greater exposure to adversity had similar recall and emotional DM scores as those with lower exposure, suggesting that there was no general impact of adversity on episodic recall ability or emotional learning processes. As we hypothesized, greater amygdala and hippocampal activity was associated with both negative and positive DM recall, but this effect was only observed for children with lower adversity. Furthermore, the hypothesis that adversity would associate with lower IPS and greater rMFG activity during negative scene encoding was not supported. Since children with higher exposure to adversity showed similar success in emotional recall tasks, this suggested to us that there might be alternative, potentially adaptive neural pathways that help support the preserved levels of emotional encoding that we observed in children who reported greater exposure to adverse experiences. This idea was partly confirmed; greater trauma load was correlated with activity in the right fusiform gyrus, an area along the ventral visual stream that showed greater activation during successful encoding of positive > neutral images. This suggested that children impacted by trauma may be using alternative, ventral visual stream circuitry to encode positive episodic events. Additionally, while this alternative neural pattern for positive episodic encoding was significantly linked with exposure to adversity, it was not correlated with any symptoms of psychopathology. Taken together, these findings suggest that childhood adversity may have an effect on memory encoding processes that allows children to preserve emotional memory, but without robust impacts on childhood symptoms of PTSD, depression, or anxiety. Instead, psychopathology due to impacts of childhood trauma and adversity may emerge in adulthood.

Emotional enhancement of memory and trauma effects on recall

Regardless of exposure to adversity, all children showed enhancing effects of emotion on memory, such that children correctly recalled

a greater proportion of positive or negative images than neutral. Enhancing effects of emotion on memory are consistently observed among healthy children and adults, primarily driven by emotional arousal-related hormones such as norepinephrine (Bahtiyar et al., 2020). We therefore anticipated that children who have experienced hyper-arousal due to traumatic experiences might show a greater effect of emotion on memory than trauma-unexposed children. However, this was not the case in our sample – trauma load did not predict any differences in recall performance. Trauma load had no effect on children's recall of emotional stimuli, suggesting that children who experience traumatic events may not experience general effects on declarative memory for either emotional or neutral stimuli.

Sex also had an interesting effect on recall performance; girls were better at recalling neutral and negative images but showed similar memory for positive images compared to boys. This is consistent with prior work investigating the impacts of stress and cortisol levels in healthy 6- and 7-year-old children, which found that girls showed greater emotional enhancement in memory as cortisol levels increased whereas boys typically did not show emotional memory enhancement with increasing cortisol (Raffington et al., 2020). Therefore, girls may be more sensitive than boys to emotional memory changes when exposed to stress and trauma. However, due to limitations in sample size, we did not have sufficient power to explore moderating effects of sex in our neuroimaging analyses.

Emotional DM x trauma interactions in hypothesized regions of interest

It is known that the amygdala and hippocampus play important roles in emotional learning and memory. During emotional arousal, noradrenergic activation of the amygdala enhances episodic encoding in the hippocampus (reviewed in Roesler et al., 2021). Adult human neuroimaging studies have found that greater amygdala activation predicts higher likelihood of later recall for emotionally arousing images (Canli et al., 2000). Intracranial EEG studies in humans show that high-frequency activity in the amygdala and hippocampus is correlated with successful encoding of emotional stimuli (Qasim et al., 2023), and direct stimulation of the amygdala have been shown to enhance hippocampal encoding for declarative memories (Inman et al., 2018; Sendi et al., 2021). Neuroimaging studies on healthy children likewise find that increased amygdala activation enhances emotional memory (Pinabiaux et al., 2013; Vasa et al., 2011). Supporting previous literature, our findings indicated that children with less trauma show improved emotional memory when amygdala or hippocampal activation was higher during encoding. However, and unexpectedly, as trauma load increased, activation in these regions actually appeared to hinder children's emotional learning. Children with greater trauma showed lower emotional DM when amygdala and hippocampal regions were more engaged by the emotional stimuli. Similarly, in trauma-exposed adults with high negative affect symptoms, lower amygdala activation during encoding predicted enhanced recall for negative, neutral, and positive scenes (Stevens et al., 2018). In a study of acute stress in adults, reduced hippocampal activation was likewise associated with improved memory performance (Henckens et al., 2009). As previously discussed, children with higher trauma load performed just as well as well on the cued recall task as the children with lower trauma load. Therefore, if amygdala and hippocampal activity were not facilitating emotional learning in highly trauma-exposed

children, it seemed likely that alternative neurocircuits might be responsible for the enhancing effects of emotion on memory.

Alternative neural pathways for emotional encoding in highly trauma-exposed children

Findings partially confirmed the idea that alternative pathways may facilitate emotional encoding after trauma. Multiple regions along the right ventral visual stream positively correlated with positive DM. Among these, the peak activation in the right fusiform gyrus was also associated with increasing trauma load, suggesting that children with higher trauma are successfully encoding positive details with engagement of this visual pathway. The fusiform gyrus is part of the ventral visual stream, which is responsible not only for the identification and recognition of objects and scenes (Ishai *et al.*, 1999; Long *et al.*, 2018; Ungerleider & Haxby, 1994), but also for the encoding of distributed representations across multiple memory systems, with greater complexity and context being integrated in a caudal to rostral direction (Bussey & Saksida, 2007; Kravitz *et al.*, 2013). In particular, the ventral visual stream helps process affective visual stimuli (McClellan France & Jovanovic, 2023; Pessoa & Adolphs, 2010) and has been shown to help with retrieval of emotionally threatening stimuli in adult populations (Kark & Kensinger, 2015). In large multimodal neuroimaging studies of adults with acute trauma load, increased ventral visual stream integrity was correlated with increased acute PTSD symptoms 2 weeks post-trauma and 12 months later (Harnett *et al.*, 2022a). Additionally, it was found that greater ventral visual stream integrity was correlated with negative connectivity between hippocampus/amygdala and the inferior temporal gyrus (Harnett *et al.*, 2022b). Harnett and colleagues suggested that these altered patterns of structure and neural activity may be impacting threat-relevant visual encoding and PTSD susceptibility. It is possible that affective encoding of positive stimuli in our sample of high-trauma-exposed children might also rely upon this ventral visual pathway and rely less on connections with the hippocampus and amygdala. The ventral visual stream is capable of undergoing rapid plasticity (Arbel *et al.*, 2023). A study using transcranial direct-current stimulation (tDCS) found that tDCS to the ventral visual stream improved memory encoding in adults (Zhao & Woodman, 2021). Therefore, perhaps in the face of increased stress and trauma, children may be able to utilize these alternative cortical networks via the ventral visual stream to encode emotionally positive events instead of relying on more subcortical amygdala/hippocampal regions.

However, it is unclear which regions supported the enhancing effect of negative emotion on encoding in high trauma-exposed children. Similar to hippocampal and amygdala results, activity in the sgACC/vmPFC appeared to hinder negative-DM recall for children with higher trauma load, as well as those with greater PTSD and anxiety symptoms. These findings contradict previous literature in which vmPFC activity supports episodic memory (Rolls, 2022). There has been a parallel in aging adult populations, among whom activation of the amygdala, hippocampus, and vmPFC likewise hinders negative emotional memory. This has been attributed to potential downregulation of negative affect during encoding (Corbett *et al.*, 2020). Perhaps children with high trauma load are engaging some unobserved mechanism or pathway for the successful encoding of negative events. Future work will be needed to identify these mechanisms supporting the encoding of negative events in children with high trauma load.

Limitations

There are several limitations worth mentioning. First, while the TESI-C provided an index of the number of different types of traumas, these are self-identified and retrospective reports, which suggests that there may be some bias or inaccuracy about the total number or severity of traumas children in this sample experience. Similarly, a child with a low TESI-C score might still be majorly burdened by their exposure to adversity if an event was especially impactful or if the particular trauma was repeated. However, it has been shown that the number of different traumatic event types self-reported has a dose-dependent impact on PTSD risk and symptom severity (Kolassa *et al.*, 2010; Neugebauer *et al.*, 2009), suggesting that evidence supports the current method of quantifying trauma load. Similarly, we did not query for sexual abuse when admitting the TESI-C measure due to concerns some parents and guardians may have had about the appropriateness of their younger children being exposed to topics about sex. It is therefore possible that we have underestimated the impact of sexual abuse within our current participant sample. Children who are sexually abused often develop PTSD and other psychiatric disorders (Trickett *et al.*, 2011). In general with fMRI studies, it can be difficult to recruit and retain large cohorts of participants, and these practical barriers are even greater among populations at high risk for trauma and multiple compounding forms of adversity. Therefore, our study focused on a relatively small sample of participants and used a cross-sectional design to evaluate children across the 8–14 year old age range. This limits our statistical power and ability to detect small but potentially meaningful effect sizes. Additionally, collecting fMRI and behavioral data with children can be difficult due to increased movement artifacts and a loss of attentional engagement with the tasks. Almost 18% of the original sample of participants had to be removed from analyses due to these challenges, which further reduced statistical power. The current findings shed light on an underexplored area of research but require future replication and validation efforts among larger sample sizes as well as longitudinal study designs to further explore the neural and behavioral outcomes within trauma-exposed children.

Conclusions

While initially surprising that trauma load did not predict emotional recall performances, school-age children with low vs high exposure to adversity showed different neural patterns of activity associated with successful encoding of emotional stimuli. Engagement of the amygdala and hippocampus supported emotional learning in children with less trauma. However, those participants with greater trauma load showed correlations in the right ventral visual stream with positive emotional recall. Age 8–14 years is an important developmental window for brain growth and plasticity. While childhood trauma can have lasting impacts on brain neural circuitry and emotional memory, children with higher trauma loads may be utilizing adaptive, right ventral visual pathways to maintain their positive emotional memory encoding. Follow-up studies should further investigate potential alternative neural pathways supporting negative emotional memory in children with high trauma exposure. Likewise, future research might examine the relationship of emotional memory and trauma among adolescent populations or in a longitudinal fashion in order to investigate dynamic changes of neural pathways involved with emotional learning that may be impacted by childhood trauma.

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