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BRAIN ACTIVATION TO FACIAL EXPRESSIONS IN YOUTH WITH PTSD SYMPTOMS

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Abstract

Objective: This study examined activation to facial expressions in youth with a history of interpersonal trauma and current posttraumatic stress symptoms (PTSS) compared to healthy controls (HC).

Design and analysis: Twenty-three medication-naive youth with PTSS and 23 age- and gender-matched HC underwent functional magnetic resonance imaging (fMRI) while viewing fearful, angry, sad, happy, and neutral faces. Data were analyzed for group differences in location of activation, as well as timing of activation during the early versus late phase of the block. Using SPM5, significant activation ($P < .05$ FWE [Family-Wise Error] corrected, extent = 10 voxels) associated with the main effect of group was identified. Activation from selected clusters was extracted to SPSS software for further analysis of specific facial expressions and temporal patterns of activation.

Results: The PTSS group showed significantly greater activation than controls in several regions, including the amygdala/hippocampus, medial prefrontal cortex, insula, and ventrolateral prefrontal cortex, and less activation than controls in the dorsolateral prefrontal cortex (DLPFC). These group differences in activation were greatest during angry, happy, and neutral faces, and predominantly during the early phase of the block. Post hoc analyses showed significant Group \times Phase interactions in the right amygdala and left hippocampus.

Conclusions: Traumatic stress may impact development of brain regions important for emotion processing. Timing of activation may be altered in youth with PTSS. *Depression and Anxiety* 29:449–459, 2012.

Keywords

fMRI; PTSD; pediatric; emotion; faces

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INTRODUCTION

Traumatic stress is a common experience in youth. A recent study found that more than 68% of youth had experienced a traumatic event by 16 years of age and more than 20% of these children subsequently suffered from problems in school, emotional difficulties, and physical ailments.^[1] Other studies have also found adverse effects of trauma on youth, including elevated cortisol levels,^[2] emotional distress, and impaired daily function.^[3] Posttraumatic stress symptoms (PTSS) refer to the psychiatric condition suffered by those who have experienced a significant trauma and are experiencing a severe and prolonged stress response, but do not meet full criteria for Posttraumatic Stress Disorder (PTSD). Our previous research indicates significant functional impairment in children with PTSS^[3] and research by others underscores the importance of studying these children.^[4]

Neuroimaging studies of youth with PTSS provide a valuable opportunity to better understand the effects of traumatic stress on neural function during development, and may lead to improved treatments for this age group. Structural MRI studies of brain volume in youth with PTSD have indicated reduced corpus callosum area, whereas adult studies of PTSD have reported deficits in the hippocampus and anterior cingulate cortex, although not consistently (see reviews of brain structure in pediatric PTSD^[5] and adult PTSD.^[6]) There are few neuroimaging studies in pediatric PTSD that use functional magnetic resonance imaging (fMRI) data. An early study of five adolescents with earthquake-related PTSD reported increased parahippocampal gyrus and decreased anterior cingulate activation to trauma-related tasks compared to six trauma-exposed youth who did not develop PTSD.^[7] Previous fMRI studies from our lab have shown aberrant activation of ventral prefrontal regions during a response inhibition task^[8] and reduced hippocampal activation during a verbal memory task^[9] in youths with PTSS. fMRI studies in adults with PTSD may suggest hypotheses about pediatric PTSS. Recent reviews of adult PTSD report relatively consistent findings of increased amygdala activation and decreased medial prefrontal activation to emotion-related tasks (recent reviews include^[10,11]). Amygdala activation is associated with perceiving the emotional saliency of stimuli, and has a central role in fear conditioning and memory,^[12] thus it has been studied extensively in the context of PTSD^[13] as well as other psychiatric disorders. Medial prefrontal cortex activation may be related to self-reflection,^[14] and is strongly associated with fear extinction paradigms.^[15] More fMRI studies are clearly needed in youths to determine the effects of trauma on the developing brain, particularly in emotion processing regions such as the amygdala and medial prefrontal cortex.

In the study described here, emotional facial expressions were presented to youth with PTSS, as faces reliably activate emotion-related brain regions in both psychiatric and healthy control (HC) populations.^[16] Some facial expressions may be trauma-mimetic stimuli for youth with a history of interpersonal trauma. Behavioral studies have found that children of hostile parents recognize expressions of anger more quickly (using less perceptual information) than children of parents with low hostility.^[17] This suggests a priming effect, such that neural systems become more efficient at recognizing salient information in order to avoid harm. Similarly, several behavioral studies have indicated that traumatized children

process threat-related stimuli more quickly, indicating attention bias toward threatening stimuli,^[18,19] whereas others respond more slowly, suggesting attention bias away from threatening stimuli, which can be interpreted as avoidance or difficulty disengaging from trauma-related stimuli.^[20,21] Based on these behavioral observations, we predicted that children with PTSS would demonstrate aberrant timing in addition to aberrant magnitude of activation to emotional face expressions. Aberrant timing may be observed as greater early-phase activation, suggesting priming, greater late-phase activation, suggesting failure to habituate, or greater increase from the early to late phase, indicating increased sensitization. Adults with PTSD have greater early-phase activation of the amygdala to trauma-related words, and lack of habituation to panic-related words^[22] and facial expressions.^[13] Understanding the chronometry of activation in youth with PTSS would contribute to our understanding of the effects of trauma on brain function during development.

METHODS

PARTICIPANTS

Participants included 56 medication-naive youth between 10 and 16 years of age. All subjects reported no neurological disorders, major medical illness, or contraindications for the MRI procedure. This study was approved by Stanford University IRB and parent consent and child assent were obtained for all participants.

For the PTSS group, 30 youths (19 females) with a history of interpersonal trauma (>6 months prior) and posttraumatic symptoms were recruited from local social service departments and mental health clinics. Inclusion in the PTSS group required at least one exposure to inter-personal trauma as defined by DSM-IV, including sexual abuse, physical abuse, and/or witnessing violence. The participants were required to currently live in a safe environment (confirmed during Clinician-Administered PTSD Scale for Children and Adolescents [CAPS-CA] interview) and to have no history of alcohol or drug abuse or dependence. A protocol was in place to contact Child Protective Services if necessary, but no contacts were needed. Twenty-six HC youth (13 females) with no history of trauma (as reported by child and caregiver) were recruited by advertisement in the community.

CLINICAL ASSESSMENT

PTSS subjects were required to score at least 13 on the CAPS-CA^[23]. A PTSD symptom was considered present if it received scores of at least 1 in frequency and 2 in intensity, totalling 3 in severity.^[3,4] The Schedule for Affective Disorders and Schizophrenia for School-Age Youth-Present and Lifetime Version (K-SADS-PL) was used to identify comorbid Axis I DSM-IV disorders.^[24] A child psychiatrist (VC) or psychologist (CW) administered all interviews. Our previous publication provides additional information on standardization of clinical interviews.^[8]

The caregivers of H C participants confirmed the absence of psychiatric symptoms by completing the Child Behavior Checklist.^[25] The Childhood Trauma Questionnaire^[26] was used to rule out a history of trauma. For all subjects, the Wechsler Abbreviated Scale of Intelligence^[27] and the Edinburgh Inventory determined handedness.^[28] Ethnic

composition, family income, and other demographic information were assessed by self-report questionnaire.

fMRI PROCEDURES

IMPLICIT EMOTIONAL FACIAL EXPRESSIONS TASK

Validation of these affective faces stimuli has been reported in a previous publication.^[29] Briefly, a set of 96 pictures were taken from several sources and edited to be monochromatic and of the same size. The faces displayed either a happy, sad, fearful, angry, or neutral expression. Blocks of scrambled pictures, created by randomly rearranging the voxels into an unrecognizable pattern, were included as a baseline comparison condition. Half of the pictures showed male models, and half showed female models. Pictures were matched (across types of emotion) for intensity of emotional expression and gender of model. Pictures were rated by an independent group of subjects to verify that they were perceived as intended.

During fMRI, the faces were presented in a block design, each containing eight pictures having the same expression. None of the stimuli were repeated. Each individual picture was shown for 3 s with no interstimulus interval. Therefore, each block lasted 24 s. The blocks were ordered such that a nonaffective block (neutral or scrambled pictures or rest) always preceded and followed each affective block to avoid carryover effects.^[30] To lessen the probability of head movement during the scan, the task was presented in two runs with approximately 1 min of rest between runs. Happy, sad, neutral, and scrambled blocks were presented in one run, and angry, fearful, neutral, and scrambled blocks in the other run. Each condition was presented for four (nonsequential) blocks total. In order to minimize order effects, there were four different sequences of blocks presented in the study. To assure attention to the stimuli, subjects judged the gender of the model in each picture and pressed button 1 with the right index finger to indicate female and button 2 with the right second digit to indicate male pictures. During scrambled blocks, subjects alternated pushing buttons 1 and 2. Correct and incorrect responses and response times were recorded. The task was presented using Psycscope software (<http://poppy.psy.cmu.edu/psyscope>). Onset of scanner and task were synchronized using a trigger pulse delivered to the scanner. Stimuli were projected onto a screen attached to the headcoil, and viewed using a mirror.

IMAGE ACQUISITION

Images were acquired on a 3T GE Signa scanner using a standard GE whole head coil (General Electric, Milwaukee, WI). A custom-built head stabilization system prevented head movement. The entire brain was imaged in 28 axial slices (4-mm thick, 0.5 mm skip) parallel to the Anterior Commissure-Posterior Commissure (AC-PC) line. Functional images were acquired using a T2*-weighted gradient echo spiral pulse sequence (Relaxation Time [TR] = 2,000 ms, Echo Time [TE] = 30 ms, flip angle = 89°, 1 interleave; Field of View (FOV) = 20 cm²; matrix = 64 × 64; in-plane resolution = 3.125 mm; slice thickness = 4 mm, 0.5 mm skip; 28 slices). Spiral fMRI optimizes signal to noise and minimizes susceptibility effects, therefore improving Blood Oxygenation Level Dependent (BOLD) signal in regions such as the ventral prefrontal cortex and the medial temporal lobe.^[31] A high-resolution T1-

weighted Spoiled Grass Gradient Recalled (SPGR) 3D anatomical image was acquired during the same session (TR = 35 ms; TE = 6 ms; flip angle = 45°; FOV = 24 cm²; 124 slices; 256 × 192 matrix; acquired resolution = 1.5 × 0.9 × 1.2 mm³).

DATA ANALYSIS

fMRI DATA ANALYSIS

Images were processed using SPM5 (Wellcome Department of Imaging Neuroscience, London, UK). The images were spatially realigned, and motion was corrected using ArtRepair toolbox for SPM (<http://cibsr.stanford.edu/tools/ArtRepair/ArtRepair.htm>). The functional images were spatially normalized to an age-appropriate stereotactic template (CCHMC: <http://irc.cchmc.org/software/pedbrain.php>) and smoothed with an 8-mm full-width half maximum isotropic Gaussian filter. Low-frequency noise was removed from the time series using a high-pass filter set to 0.5 cycles per minute and applied to the fMRI time series at each voxel.

ANALYSIS OF EARLY AND LATE PHASES

A fixed-effects model was used to identify activation associated with each facial expression compared to scrambled images. Each block of faces was divided into an early and late phase, so that the first 12 s of the faces block was modeled as the early phase, and the last 12 s was modeled as the late phase. The early phase of each facial expression block was contrasted with the early phase of the scrambled images block, and the late phase was contrasted with the late phase of the scrambled block, collapsing across all blocks within each facial expression condition. Voxel-wise *t*-statistics were normalized to *Z* scores.

The group analysis involved two steps. First, a random-effects voxel-wise whole brain analysis was conducted in SPM5 using a multivariate ANOVA. The ANOVA included the factors group (PTSS and HC), and face (angry, fearful, happy, sad, neutral) and phase (early and late). Variance associated with IQ was removed by modeling IQ as a nuisance covariate. Voxels significantly activated for the main effect of group and the interaction of Group × Phase were identified, using a stringent threshold of $P = .05$ FWE corrected, and minimum cluster extent = 10. Regions of activation were localized with reference to the stereotaxic atlas of Talairach and Tournoux.^[32] The software program Rfxplot (rfxplot.sourceforge.net) was used to plot time-courses to verify that group differences were not attributed to the 'scrambled' baseline condition.

The second step of the group analysis determined the relative contributions of each facial expression and phase to the results obtained from the SPM analysis of the main effect of group. Only clusters showing significant activation in step 1 and having prominence in the PTSD literature were further analyzed, including the amygdala, insula, ventral medial prefrontal, ventrolateral prefrontal, and dorsolateral prefrontal cortices (DLPFC). The MARSBAR (<http://marsbar.sourceforge.net/>) toolbox in SPM5 was used to extract mean activation for export to SPSS (<http://www.spss.com/>) for further analysis. In SPSS, independent group *t*-tests were conducted for each brain region, comparing group activation to each facial expression and phase, for example PTSS versus HC in the amygdala/

hippocampus for the early phase of angry faces, etc. We also assessed habituation and sensitization of activation (significant decreases or increases in activation from the early to late phase of the block, respectively). Group differences surpassing a corrected α of .01 were flagged as contributing substantially to the main effect of group.

Because no significant clusters were identified by the interaction of Group \times Phase at this threshold, we also investigated the interaction of Group \times Phase using a small volume correction confined to the bilateral amygdalae and hippocampi (using masks from the AAL Atlas^[33]), as these regions have been strongly implicated in the literature. Clusters of activation passing threshold ($P = .05$ FWE corrected) were plotted for qualitative analysis of timing of activation.

RESULTS

DESCRIPTIVE GROUP DIFFERENCES

Six of the PTSS and three of the HC subjects were rejected for excessive movement during the scan (for both groups, this was defined as greater than 25% of the time points having motion exceeding a signal threshold of 3% from the mean, or 0.5 mm per temporal frame). For the remaining subjects, the number of time points corrected for motion did not differ between groups ($P > .05$). One of the PTSS subjects was removed for missing IQ data, as all fMRI analyses were covaried for group differences in IQ. This left 23 PTSS and 23 HC subjects.

Table 1 shows the demographic descriptors of the groups. There were no group differences in age, gender distribution, or handedness. The PTSS group had significantly lower IQ scores than the HC group, so all fMRI analyses were covaried for IQ. Although 17 of the 23 PTSS subjects had no comorbid diagnoses, six subjects had depression, anxiety, and behavioral disorders characteristic of youth with PTSS.

There were no significant group differences in task accuracy or response time, although there was a trend for the PTSS group to identify gender less accurately than the HC group for the fearful faces only ($P = .06$).

BRAIN ACTIVATION GROUP DIFFERENCES

Significant clusters of activation from the main effect of group are given in Table 2. Results of follow-up analyses of these significant clusters are summarized in Figure 3: Group differences in amygdala/hippocampal activation were greatest during the late phase of angry faces ($P = .003$) and the early ($P = .002$) and late ($P = .007$) phases of neutral faces; activation to neutral faces increased significantly from the early to late phase of the block for all subjects together ($P = .003$) and for control subjects alone ($P = .005$). Group differences in the medial prefrontal cortex cluster were greatest during the early phase of the angry faces ($P = .006$). Group differences in insula activation were greatest during the early phase of angry faces ($P = .002$) and the early phase of neutral faces ($P = .005$). The HC group, but not PTSS group, shows sensitization of activation to happy ($P = .008$) and neutral faces ($P = .003$) in the insula. Group differences in left ventrolateral prefrontal cortex activation were greatest during the early phase of happy faces ($P = .002$) and the early phase of neutral faces ($P = .$

006), and both groups together show sensitization of activation to fear faces ($P = .008$). Group differences in the DLPFC were greatest during the late phase of happy faces ($P = .002$), and sensitized to neutral faces ($P = .009$) in the PTSS group only. Post hoc analyses of group differences in timing of activation using a small volume corrected voxel-wise analysis of the Group \times Phase interaction found significant activation in the right amygdala and left hippocampus. The average time-courses in these regions are shown in Figure 2. The graphs suggest that the PTSS group showed greater right amygdala activation predominantly in the early (but not late) phase of the block of angry and fearful faces. For the left hippocampus, the PTSS group appears to show greater activation for angry and fear faces in the early phase of the block, but decreased activation compared to controls in response to happy and sad faces in the late phase of the block. The group \times face and the Group \times Face \times Phase interactions were not significant.

CORRELATIONS WITH CAPS SCORES IN THE PTSS GROUP

A Spearman's nonparametric correlation (threshold of $P = .01$ corrected for multiple comparisons) showed that increasing activation in the medial prefrontal cortex during the late phase of the fear block was associated with both total CAPS score ($\rho = .55$, $P = .008$) and the B (re-experiencing) subscale ($\rho = .54$, $P = .008$). Scatter plots of these associations are given in Figure 3. No other regions were significantly correlated with symptom severity.

We did not collect detailed information about severity of trauma exposure that would allow correlations with brain activation. However, we attempted to examine associations with trauma severity by correlating activation with number of types of trauma (e.g., physical abuse + sexual abuse + witnessing violence = 3). No correlations reached significance ($P = .05/5$ regions = .01).

COMORBID DIAGNOSES

To investigate effects of comorbid diagnoses on our findings, we removed the six subjects from the PTSS group who had a comorbid diagnosis. Between-group differences in extracted values were reduced along with the reduced power, but α remained less than .03.

EFFECTS OF A DIAGNOSIS OF PTSD

We directly compared Region of Interest (ROI) activation in subjects with PTSS ($N = 12$) to those with a full diagnosis of PTSD ($N = 11$). The only difference was greater activation in the left ventrolateral prefrontal cortex to happy faces in subjects with PTSD compared to those with PTSS ($P = .028$).

DISCUSSION

This study examined brain activation to emotional facial expressions in youth with a history of interpersonal trauma and PTSS. Individuals in the PTSS group showed a significant degree of subsyndromal symptoms, and our previous studies of this sample have reported significant abnormalities in brain structure^[34] and function.^[8,9] The current study adds to the previous reports by finding abnormalities in emotion processing brain circuitry. Regions showing abnormal activation are consistent with previous neuroimaging studies of adults

with PTSD, including the amygdala and ventromedial prefrontal cortex. Notably, youth with PTSS show aberrant activation to neutral faces as well as emotional expressions, and show altered timing of activation in the amygdala and hippocampus. Therefore, traumatic stress in youth may impact the development of brain regions important for emotion perception.

Group differences in activation of the amygdala/hippocampus, medial prefrontal cortex, and insula were associated with angry face blocks but not fearful or sad. It is possible that angry facial expressions have particular relevance to this group of children who have been exposed to interpersonal trauma. If so, a previous trauma and/or current PTSD symptoms (such as hypervigilance) may prime the amygdala to respond to trauma-related stimuli more quickly. Magnetoencephalography shows that priming enhances amygdala theta activity as soon as 150–400 ms after presentation of the subsequent target word.^[35] Therefore we would expect that priming would enhance early-phase amygdala activation, and we did observe exaggerated early-phase activation of the right amygdala in the PTSS group to angry and fearful faces. It is also possible that individuals with PTSS displayed hypersensitivity to threat even before experiencing trauma.

Greater activation in the PTSS group in response to neutral faces was noted in the amygdala, along with reduced activation in the DLPFC. The reactivity of the amygdala to neutral faces may reflect the ambiguity or a negative interpretation of these stimuli for this young age group^[36] and has been reported in neuroimaging studies of children with anxiety and depression,^[37] as well as adults with mild PTSD.^[38]

Activation to happy faces shows an interesting pattern. For the PTSS group compared to HC, activation to happy faces is increased in the ventrolateral prefrontal cortex but decreased in the DLPFC, suggesting altered emotion regulation in response to these stimuli.^[39–41] Interestingly, happy faces are the only stimuli that elicit marked sensitization of activation from early to late phase in the insula in the HC group, but elicit no change from early to late phase in the PTSS group. In light of the somewhat consistent trend for sensitization of activation to the other stimuli, lack of sensitization to happy faces may be related to anhedonia or numbing symptoms reported in PTSS.

Severity of PTSD symptoms, as measured by the CAPS-CA score, was significantly correlated with medial prefrontal cortex activation during the late phase of the fearful faces block. In general, the PTSS group showed greater activation in this region compared to the HC group. This region has been consistently implicated in the PTSD neuroimaging literature^[10,10] and is important for emotion regulation^[42,43] and extinction of conditioned fear.^[15] Interestingly, we observed a trend ($P = .06$) for decreased task accuracy during fearful faces trials only, even though the gender identification task was easy and proficiently performed for all other faces. Taken together, these data may suggest subtle avoidance of processing fearful faces in the PTSS group. This interpretation is consistent with previous studies finding attention bias away from threat-related stimuli in PTSD.^[21] Thus, increased medial prefrontal activation during the fearful faces may reflect attention regulation to avoid processing fearful faces.

We did not observe habituation (a decrease in activation from early to late phase) in any of the regions in either group. This may have occurred because we did not repeat any of the faces, which is typically the method used in studies reporting rapid habituation of amygdala response.^[44,45] The previous study of adult PTSD that examined early and late phases of activation^[22] found increased early-phase activation to trauma-related words in the left amygdala in PTSD compared to HC, and a lack of habituation to panic-related words. However, that study defined the early and late phases as the first two and last two epochs of the experiment, whereas our late and early phases were the first and last 12 s of a block.

This study was strengthened by including a large sample of unmedicated subjects with few comorbid diagnoses. Limitations of the study include group differences in IQ, which we controlled by covarying statistically. Both adults and children with PTSD symptoms tend to have lower IQ, particularly lower verbal IQ, but not visual IQ. It is possible that symptoms of PTSD interfere with the cognitive functions that contribute to verbal IQ skills, such as memory and attention. If so, we would expect the IQ of these children to increase when symptoms are relieved. It is also possible that lower verbal IQ exists before trauma, and places children at risk for PTSD following a trauma, because it makes them less likely to process the experience verbally. A third possibility is that early trauma and symptoms of PTSD interfere with the ability of children to acquire verbal skills.

As we did not require a PTSD diagnosis, our findings may not generalize to studies of more severe PTSD symptoms in youth. However, the similarity of our findings to studies in adults suggests that our sample shows neurofunctional abnormalities representative of PTSD.

Acknowledgments

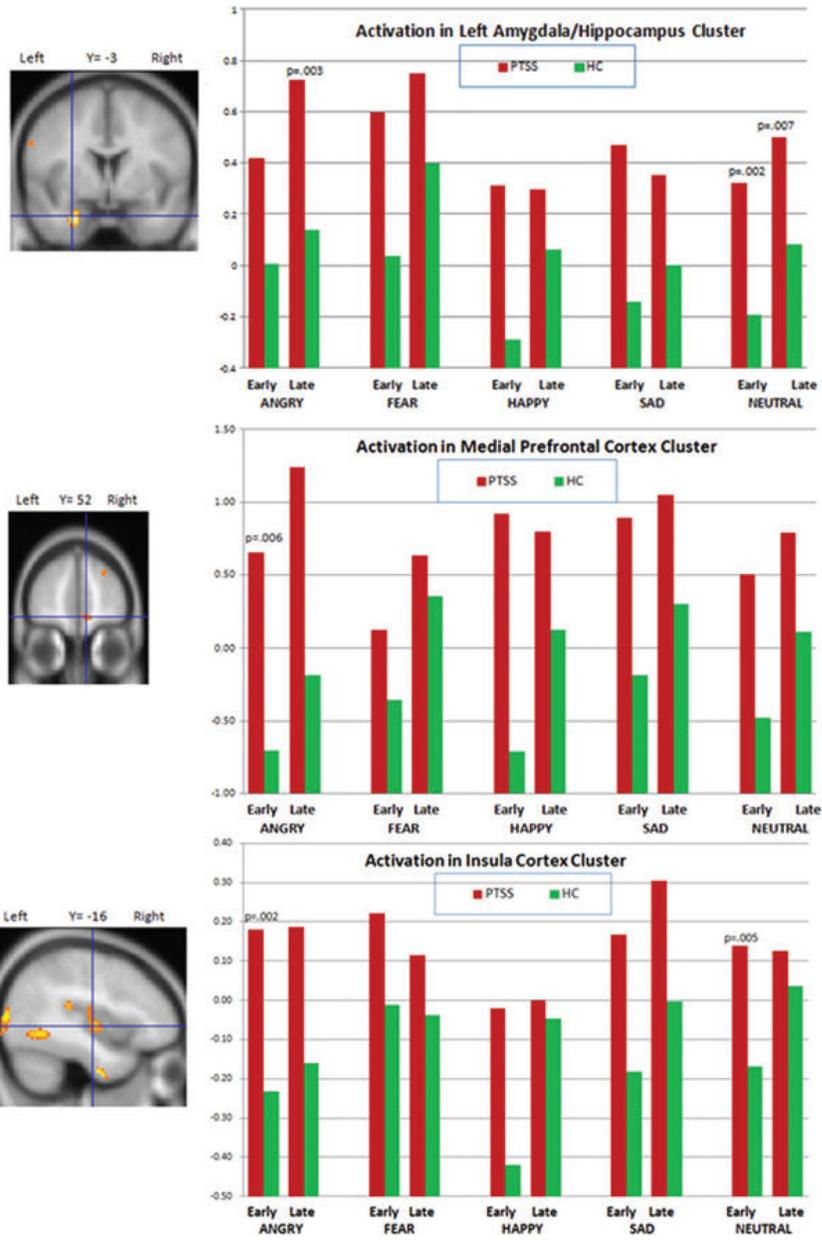
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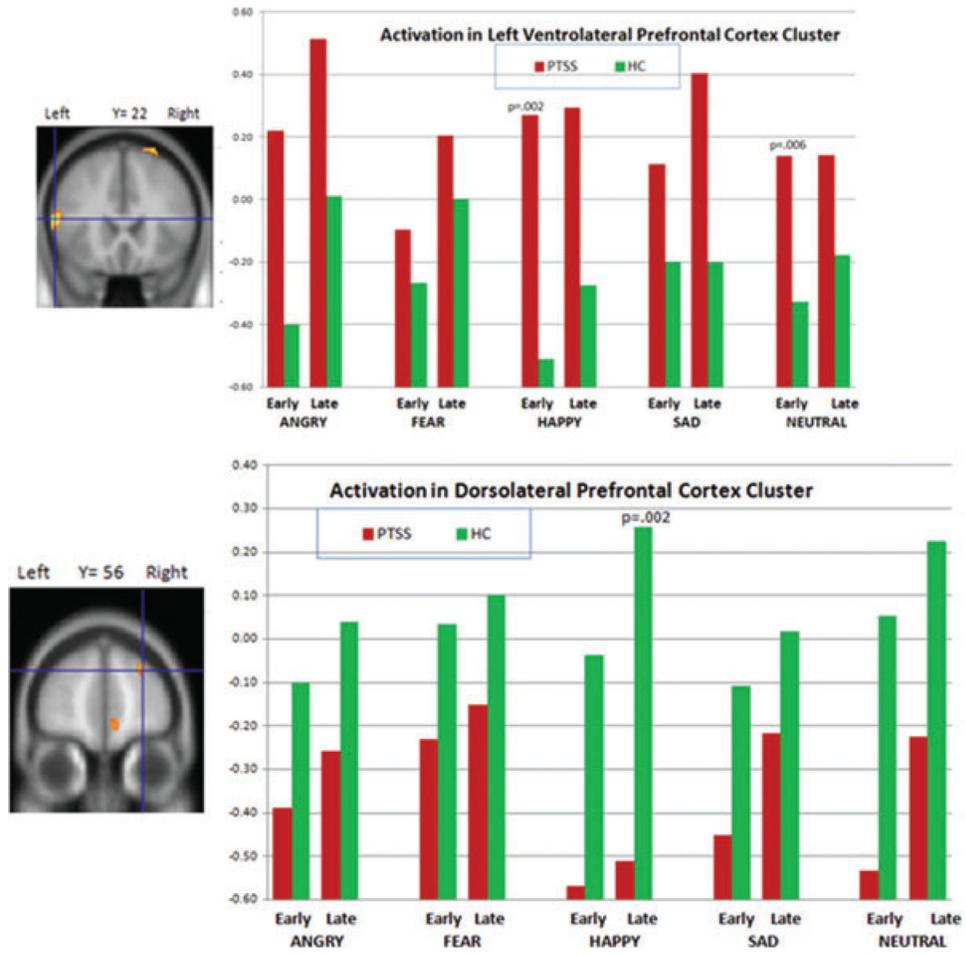


Figure 1. Main effect of group broken down by facial expression and phase. Significant group differences in left amygdala/hippocampus activation (at crosshairs) were greatest in the late phase of angry faces, and early and late phases of neutral faces; group differences in activation of the medial prefrontal cluster (at crosshairs) were greatest in the early phase of angry faces; group differences in activation in the insula were greatest during the early phase of angry faces, and early phase of neutral faces; group differences in activation in the left ventrolateral cluster (at crosshairs) were greatest during the early phase of happy faces, and the early phase of neutral faces; group differences in activation in the dorsolateral prefrontal cortex (DLPFC) were greatest in the late phase of happy faces.

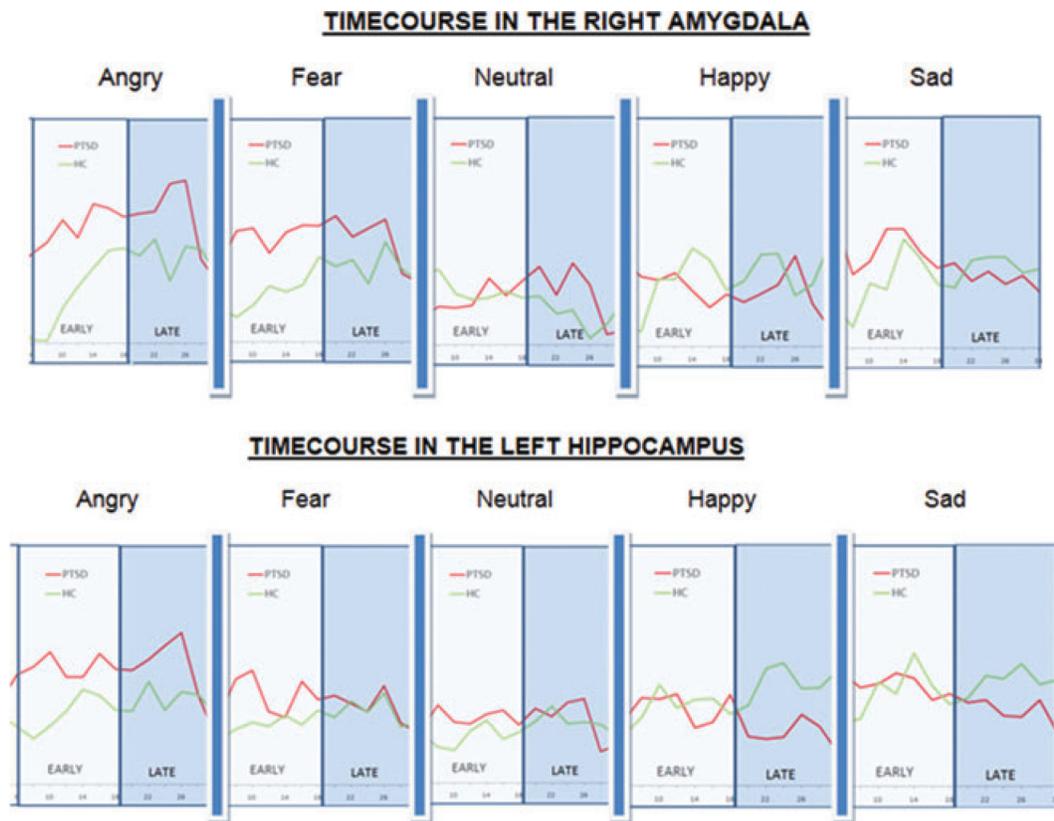


Figure 2.

Time-courses of activation in the right amygdala (top) and left hippocampus (bottom) displaying a significant Group \times Phase interaction. The posttraumatic stress symptoms (PTSS) group time-course is shown in red and the HC group is shown in green. The x -axis is seconds, and the early phase (0–12 s) and late phase (12–24 s) are indicated (both shifted 6 s forward to account for the hemodynamic lag). For the right amygdala, the PTSS activation is greater than the HC particularly in the early phases of the angry and fear blocks. For the left hippocampus, PTSS activation is greater during early and late phase of the angry blocks, but less during the late phase of the happy and sad blocks.

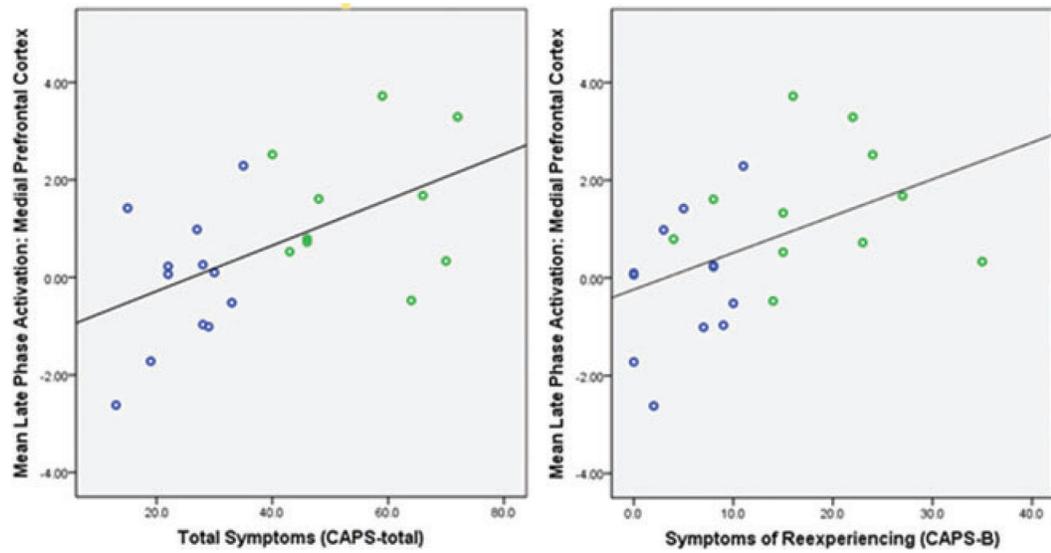


Figure 3.

Significant correlation between CAPS score and activation in the ventral medial prefrontal cortex during the late phase of the fearful faces block: for total CAPS score, $\rho = .55$, $P = .008$; for the CAPS-B (re-experiencing) subscale ($\rho = .54$, $P = .008$). Green datapoints indicate subjects with a full PTSD diagnosis, whereas blue are subthreshold.

TABLE 1.

Clinical, demographic, and behavioral descriptors of the participants

Descriptor	Youth with PTSS (PTSS: N = 23)				Healthy control (HC: N= 23)			
	Mean	SD	Range	Range	Mean	SD	Range	Range
Age	14.1	2.0	14-17	14-17	14.6	1.8	14-17	14-17
WASIQ**	104.7	15.6	71-136	71-136	115.1	11.2	92-133	92-133
CAPS total	39.4	17.8	13-72	13-72	NA			
Left handed	3	-	-	-	1			
Gender	13 F/10M	-	-	-	12F/11M	-	-	-

Description	Description
Trauma	5 SA SPA 4WV 4SA/PA 4PA/WV 1SA/PA/WV
Comorbid diagnoses	2 MDD only 1 MDD + panic 1 sep anxiety 1 enuresis + MDD 1 MDD, ADHD, CD 17 no comorbid Dx
Ethnic group	13 White 3 Hispanic 3 mixed 2 Black 1 Pacific Islander 1 native American
Family education level*	16 White 2 Hispanic 2 mixed 1 Black 1 Pacific Islander 1 Asian
	1 junior high 5 high school 6 partial college 3 partial college

	Description	Description
	8 college grad	3 college grad
		8 grad degree
	2 missing	9 missing
Family income *	10 (10–50K)	
	3 (50–75K)	5 (50–75K)
	2 (75–100K)	1 (75–100K)
	3 (100–125K)	2 (100–125K)
	2 (125–200K)	
		5 over 200K
	3 missing	10 missing

	Mean	SD	Mean	SD
Percent correct happy face	89	6	88	8
Response time happy face (ms)	916.4	184.7	929.2	170.3
Percent correct sad face	86	9	87	7
Response time sad face (ms)	932.9	194.6	954.7	136.6
Percent correct fear face	80	9	85	9
Response time fear face (ms)	960.8	161.1	976.8	162.8
Percent correct angry face	81	11	85	12
Response time angry face (ms)	992.2	176.4	1,017.4	177.8
Percent correct neutral face	86	9	88	8
Response time neutral face (ms)	931.7	172.6	948.7	157.0

* The family education level and income of the posttraumatic stress symptoms (PTSS) subjects corresponds to the current caretaker, in most cases this was not the biological parents; CAPS, Clinician-Administered PTSD Scale for Children and Adolescents; SA, sexual abuse; PA, physical abuse; WV, witnessing violence; meds, medication; MDD, major depressive disorder; sep anxiety, separation anxiety; ADHD, attention-deficit hyperactivity disorder; CD, conduct disorder; Dx, diagnosis.

** $P = .014$.

*** Trend for decreased percent correct in PTSS group for fear faces; $P = .06$.

TABLE 2.

Significantly activated clusters from the main effect of group

No.	Region	BA	k	Peak Z score	TAL:X	TAL:Y	TAL:Z
1	L cuneus	17/18	719	7.32	-2	-73	18
	L precuneus	23/31		6.70	-2	-75	11
2*	L amygdala	NA	252	6.01	-26	-3	-22
	L hippocampus	NA		5.36	-26	-10	-8
	L globus pallidus	NA		5.95	-24	-8	-6
	L putamen	NA		5.93	-26	-8	-6
	L entorhinal cortex	28		5.52	-30	5	-22
3	R inferior parietal cortex	40	241	6.15	61	-33	42
	R postcentral gyrus	2/3		6.03	61	-27	42
	R precentral gyrus	6/4		5.35	63	-12	36
4	L superior temporal gyrus	22	214	6.92	-63	-42	11
	L middle temporal gyrus	21		6.60	-61	-48	8
5	L precentral gyrus	6/4	140	6.81	-61	-8	34
	L inferior frontal gyrus	9		5.62	-61	9	25
6*	L inferior frontal gyrus	45/44	149	6.55	-57	22	12
7	L inferior temporal gyrus	37/20/21	88	6.46	-59	-53	-9
	L middle temporal gyrus	37		5.83	-57	-51	-9
8	R inferior temporal gyrus	20	71	6.46	40	-11	-33
	R middle temporal gyrus	21		5.82	40	-7	-33
9	R middle occipital gyrus	19/18	266	6.45	44	-83	13
10	L inferior parietal lobule	40	164	6.12	-57	-39	42
	L postcentral gyrus	2		4.94	-61	-25	36
11*	R insula(1)	13	111	6.02	46	-34	18
	R superior temporal gyrus	41		5.78	44	-34	20
12	L inferior temporal gyrus (2)	20	76	5.71	-61	-22	-21
	L middle temporal gyrus (2)	21		4.99	-63	-22	-14
13	R insula (2)	13	159	5.82	44	-16	-1
	R superior temporal gyrus (2)	22		5.35	46	-17	1

No.	Region	BA	k	Peak Z score	TAL:X	TAL:Y	TAL:Z
	R claustrum	na		4.93	38	-11	4
	R superior temporal gyrus	38	37	4.94	32	10	-26
14	L paracentral lobule	6	52	5.34	-12	-23	49
15	R superior temporal gyrus	22	42	5.61	57	-48	12
	R middle temporal gyrus	39		4.67	57	-52	10
16*	R DLPFC (superior frontal)	9	27	5.26	24	56	25
	R middle frontal gyrus	10		4.84	24	55	21
17	L precuneus	7	29	5.21	-14	-66	33
18*	R medial frontal gyrus	10/11	21	5.02	6	52	-11

Threshold of $P = .05$ FWE corrected, and minimum cluster size of 10 voxels. Clusters with “*” were extracted for follow-up analyses (shown in the table).

R, right hemisphere; L, left hemisphere; k, number of voxels in cluster; BA, Brodmann area; p, corrected cluster-wise p; TAL, Talairach Atlas Coordinate; NA, not applicable.