# **Archival Report**

## Atypical Neural Activation During Emotional but Not Nonemotional Response Inhibition in Healthy Young People Exposed to Childhood Maltreatment and Peer Victimization

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#### **ABSTRACT**

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**BACKGROUND:** Early-life interpersonal stress, particularly childhood maltreatment (CM), is associated with neurobiological abnormalities and atypical emotion regulation. However, few studies have investigated the neural effects of peer victimization (PV). We examined neural alterations in emotional and nonemotional response inhibition in carefully matched healthy CM and PV groups.

METHODS: Functional magnetic resonance imaging data were collected from 113 age- and sex-matched nonclinical/community youths (38 CM, 39 PV, and 36 control) during an emotional (fearful/happy) and nonemotional (letter) Go/NoGo task.

RESULTS: There were no significant group differences in behavioral performance. However, during fearful face inhibition, the CM group exhibited hyperactivation compared with the PV group in a cluster comprising the bilateral calcarine, cuneus, and lingual gyri, which was related to higher parental antipathy in the CM group. Hyperactivation also occurred in limbic-striatal, middle temporal, and cerebellar regions, although at a more liberal threshold. Additionally, there was a trend of PV-specific underactivation in the left middle temporal gyrus during happy inhibition. Despite no significant group differences in nonemotional response inhibition, both the CM and PV groups exhibited greater activation than the control group in default mode network regions during the cognitively low-load LetterGo condition.

CONCLUSIONS: These findings suggest that early-life interpersonal stress is associated with atypical neural activation during emotionally driven decision making but not during nonemotional response inhibition, underscoring the importance of examining both "hot" and "cold" decision-making processes. The atypical activation of key emotion-visual processing regions may be a potential mechanism to cope with aversive experiences and may reflect the brain's attempt to facilitate emotional inhibitory control, particularly in resilient maltreated youths.

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There is increasing interest in understanding the effects of early-life stress on the developing brain. Childhood maltreatment (CM), which includes physical, sexual, and emotional abuse and neglect, is common worldwide, with a pediatric prevalence of 13% to 36% (1). It has been associated with a host of adverse neural consequences including abnormal error processing (2,3) and impaired attention, emotion, and reward processing (4-7). Parental antipathy, including coldness and hostility (8), is commonly experienced in CM and has been linked to aberrant social reward processing (9). Outcomes of CM include poor social development (10), psychological distress, and reduced emotional well-being (11). These psychopathological outcomes may be mediated by disruption of neural systems (12); therefore, elucidating the brain mechanisms of impaired cognitive and emotional processes may inform preventive treatments (13) and promote resiliency (14,15).

Research investigating response inhibition in CM has focused predominantly on the cognitive-motor domain using Go/NoGo (GNG) or Stop tasks, although evidence remains inconclusive. Prior studies indicate that CM is associated with increased left inferior frontal gyrus (IFG) activation in patients with psychosis (16) and reduced rostral anterior cingulate cortex (ACC) activation (17), increased medial prefrontal cortex (mPFC) activation, and reduced middle frontal activation in patients with posttraumatic stress disorder (18). However, other studies have found no significant differences in brain activation between healthy control and CM-exposed individuals (2,19). Therefore, there is a need to further examine response inhibition in maltreated individuals, particularly in the absence of psychopathology.

Healthy social connections are essential for human survival (20), and successful interactions require the inhibition of

inappropriate responses using emotional information conveyed through facial expressions (21). Deficits in socially appropriate response inhibition could result from impaired neural responses to emotional stimuli and/or regulatory mechanisms controlling emotional responses (22). For example, early-life stress has been associated with decreased left IFG activation and increased right anterior insula (AI) activation during response inhibition to fearful faces (23), while violence exposure in children has been associated with increased activation of fear neurocircuitry during fearful response inhibition (24).

Parents and peers play critical roles in children's socioemotional development, and aversive parental caregiving and maladaptive peer interactions create stressful experiences during early development. Similar to CM, peer victimization (PV) is a serious global issue, with a pediatric prevalence rate of 30% (25). It is characterized by repetitive aggressive behavior with the intension to cause harm to the victim (26) and can include overt confrontation, relational aggression, reputational aggression, and ostracism.

Most functional magnetic resonance imaging (fMRI) studies in PV have focused on social exclusion and reported enhanced activation of limbic and cortical regions including the amygdala, insula, ACC, and mPFC among bullied adolescents and young adults compared with control participants (27). Notably, few fMRI studies involving emotional face processing in this population exist. One study revealed that greater amygdala reactivity to angry and fearful faces predicted higher levels of relational victimization (28). Similarly, a history of victimization among female adolescents with high rejection sensitivity was associated with higher amygdala-ventrolateral PFC connectivity when viewing emotional faces, indicating lower effectiveness of emotional regulation (29). Therefore, similar to CM, PV may be associated with atypical limbic reactivity to negative faces.

Despite the widespread occurrence of CM and PV, research directly comparing their neurobiological underpinnings has been relatively limited. CM and PV may have both shared and differential effects on the brain. For example, a recent fMRI study found that CM and PV were associated with shared underactivation of limbic-thalamic-striatal and somatosensory regions during disgust processing (30). Anatomically, both CM and PV groups exhibited reduced gray matter volume (GMV) in inferior frontal-limbic and visual regions together with a CM-specific reduced Al GMV and a PV-specific increased cortical thickness in the left mPFC-ACC (31). Another study reported CM-specific increased fractional anisotropy in the limbic and corpus callosal pathways associated with enhanced affective theory of mind but a PV-specific reduced fractional anisotropy in the inferior fronto-occipital fasciculus (32).

Notably, not all individuals exposed to early-life stress develop psychiatric disorders. Healthy trauma-exposed individuals may show neural alterations, particularly in processing and inhibiting emotions, perhaps reflecting the brain's adaption of alternative strategies to achieve specific tasks (23). Indeed, prior studies have indicated that trauma-exposed individuals exhibit alterations at the neural but not the behavioral level (16,18,19,23) because they may use different (compensatory) neural mechanisms to facilitate inhibitory control. While differences in inhibitory functioning may increase later risk for psychopathology (33), preserved cognitive control may

indicate psychological resilience in psychologically healthy trauma-exposed individuals.

Prior work has focused on elucidating the neurocircuitry involved in the processing of negative emotions such as fear and anger. However, a core feature of posttrauma psychopathology is diminished positive affect, which has been relatively understudied (34). Individuals exposed to traumatic stress may have an impaired ability to experience positive emotions and exhibit a lack of reactivity to positive stimuli (34). For example, children exposed to harsh parenting showed reduced activity in the amygdala and cerebellum while processing positive emotional stimuli (35), and high-CM-exposed youths had lower cerebellar activation than their low-CM-exposed counterparts in response to relaxing cues (36).

Maltreated children are at increased risk of subsequent bullying by peers, possibly via altered neurocognitive functioning (37). Therefore, it is imperative to examine PV in the absence of CM and vice versa in a single study to elucidate their shared and/or distinct effects. In the current study, we examined the neural basis of emotional and nonemotional inhibitory control in nonclinical/community youths exposed to CM versus PV compared with nonstressed control participants. Importantly, our youths were free from psychopathology, drug abuse, and medications. We also controlled for the timing and duration of CM/PV exposure and the number of recent stressors experienced. Because resilient traumaexposed individuals may adopt different neural mechanisms to facilitate inhibitory control, particularly in emotional contexts, we hypothesized that the CM and PV groups would exhibit atypical frontolimbic activation during emotional inhibition (23) but would not differ from their nonstressed counterparts during nonemotional response inhibition.

#### **METHODS AND MATERIALS**

#### **Participants**

Participants were 117 healthy youths (age range: 17–21 years) from the community. Exclusion criteria were a history of both CM and PV, current and/or past psychiatric diagnoses, psychotropic medications, childhood sexual abuse, drug abuse, bullying perpetration, CM or PV exposure after the age of 16, neurological abnormalities, brain injuries, and learning disabilities. First, we conducted a thorough prescreening interview to assess the exclusion and inclusion criteria. Suitable individuals were invited to participate in the study, while those found unsuitable were notified, and their information was deleted at this stage.

CM was assessed using the Childhood Trauma Questionnaire (CTQ) (38). PV was assessed using the Revised Peer Experiences Questionnaire (rPEQ) (39) and European Cyberbullying Intervention Project Questionnaire (ECIPQ) (40). Information on the age of onset and duration of CM/PV were collected with the following 2 questions: "How old were you when you first experienced the harsh treatment from the caregiver(s) or peer(s)?"

Inclusion criteria for the CM group were exposure to nonsexual maltreatment from caregivers, with a score above the cutoff for moderate severity on at least one of the CTQ subscales, but not experiencing bullying from peers (scoring "never"/"once or twice" on all the rPEQ/ECIPQ items). Inclusion criteria for the PV group were being frequently bullied by peers (indicating at least "a few times" on all the rPEQ/ECIPQ items) but not experiencing maltreatment from caregivers (scoring below the cutoffs for none/low severity on all the CTQ subscales). The control group did not experience maltreatment from caregivers and peers (scoring below the same criteria as above). There were 39 participants in each group.

All participants and their guardians provided written informed consent and were reimbursed \$80 for their time. The study was approved by the institutional review board, and all MRI scans were reviewed by a neuroradiologist.

## **Study Design and Procedure**

The study consisted of a face-to-face interview followed by an MRI session within 1 week. During the interview session, all participants completed the following: the DSM-5 Level-1 Cross-Cutting Symptom Measure, Strengths and Difficulties Questionnaire (SDQ) (41), Beck Depression Inventory (BDI) (42), Beck Anxiety Inventory (BAI) (43), and Negative and Positive Affect Scale (NAPAS) (44). The Childhood Experience of Care and Abuse (CECA) interview (8) was used to corroborate the CTQ and provide additional information on parental antipathy. IQ was assessed using the Wechsler Abbreviated Scale of Intelligence (45). Socioeconomic status (SES) was measured with 6 items (on parental educational level, housing size and type) from the Family Affluence Scale (46). Lastly, recent stressful life events (RSLEs) were assessed using common stressors adapted from the Life Event Questionnaire for Adolescents (47), where participants rated the 12-month incidence and distress level of each stressor. A total RSLEs score was calculated by summing the number of items rated as quite or very stressful. In the current study, the internal consistency of the questionnaires ranged from 0.88 to 0.93.

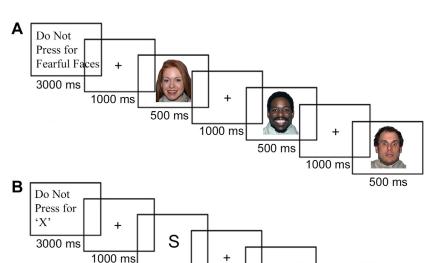
### **fMRI Paradigm: GNG Tasks**

The block-design fMRI task used in the current study was adapted from a previous GNG task (48) (see the Supplement). All stimuli were presented for 500 ms, followed by a 1000-ms interstimulus interval with a central fixation cross. Go blocks consisted of 100% Go trials, and NoGo blocks consisted of 50% Go and 50% NoGo trials presented randomly. For the emotional GNG task, participants viewed happy or fearful faces selected from the NimStim face set (49) and were instructed to either "press for all faces" or withhold responses specifically for happy or fearful faces (Figure 1A). Emotional task data were acquired over 3 runs. In the letter GNG task, participants were instructed to either "press for all letters" or "do not press for X" (Figure 1B). Letter task data were acquired over 2 runs.

## **MRI Acquisition and Analysis**

MRI data were acquired on a 3T Siemens MAGNETOM Prisma scanner at Nanyang Technological University with a 64-channel head coil. Blood oxygen level-dependent (BOLD) data preprocessing followed a conventional approach using SPM12 (http://www.fil.ion.ucl.ac.uk/spm/software/spm12/): realignment, coregistration, segmentation, normalization, and smoothing. Participants were removed from further analysis if movement away from the first collected volume exceeded 3 mm of displacement or 3° of rotation in any direction (see the Supplement).

Individual subject task contrast maps were created by specifying a general linear model for each participant. Data from each of the 6 task conditions (HappyGo, FearNoGo,



1000 ms

500 ms

1000 ms

500 ms

500 ms

Figure 1. Go/NoGo (GNG) tasks. Sample GNG task design for (A) a fear NoGo block during the emotion task and (B) a NoGo block during the letter task. In the emotion task, participants viewed happy or fearful faces presented in rapid succession and were instructed to press a response button for all faces (Go blocks) or inhibit responses specifically for the happy faces or fearful faces (NoGo blocks). In the letter task, subjects viewed a series of English consonant letters presented in rapid succession and were instructed to press for all letters (Go blocks) or to press for all letters except for "X" (NoGo blocks).

FearGo, HappyNoGo, LetterGo, and XNoGo) plus fixation were concatenated from each run and included as separate regressors. Motion parameters were included as covariates. Contrasts of interest were FearNoGo-HappyGo, HappyNoGo-FearGo, HappyGo-FearGo, XNoGo-LetterGo, and LetterGo-XNoGo. Contrasts comparing activation from FearNoGo with FearGo (or HappyNoGo with HappyGo) were not used because they would treat the task as a reversal task and not a GNG task. As with prior work using block design emotional GNG tasks (21,22,48), we compared NoGo to Go activation using contrasts that corresponded with the GNG block progression, together with contrasts that directly compared emotional valence during both Go and NoGo conditions. Hence, we examined neural activation corresponding to emotional response inhibition in the context of ongoing behavior (NoGo to Go), neural activation for each emotion when no executive decision was required (Go to Go), and the influence of emotion on the neural circuitry engaged by response inhibition (NoGo to NoGo).

Second-level contrast maps were then analyzed using analysis of covariance (ANCOVA) with RSLEs and age of onset and duration of early-life stress as covariates to avoid confounding the effect of early-life stress with recent stressors and the impact of timing and duration of early-life stress exposure. Cluster-level BOLD responses were deemed significant at p < .05, familywise error rate corrected. Next, regions showing significant group differences in the whole-brain analysis were extracted using MarsBaR (http://marsbar.sourceforge.net/) for subsequent planned group comparisons and correlational analyses with early-life stress measures (CTQ/CECA, rPEQ/ECIPQ) within each early-stress group.

## Statistical Analyses of Demographic and Performance Data

All nonimaging analyses were carried out using SPSS Statistics 26. Demographic and clinical data were analyzed with analysis of variance and Bonferroni-corrected post hoc t tests. Categorical demographic variables were analyzed using  $\chi^2$  and Fisher's exact tests. For the GNG task, ANCOVA with Bonferroni-corrected post hoc t tests was used to compare group differences in mean reaction time to Go trials and accuracy of Go and NoGo trials, controlling for RSLEs and age of onset and duration of early-life stress.

## **RESULTS**

## **Participant Characteristics**

All participants reported no current and/or past psychiatric disorders, corroborated with the DSM-5 measure. They also reported no head injuries, learning disabilities, history of drug and childhood sexual abuse, and bullying perpetration. After exclusions due to excessive movement (see the Supplement), the final sample consisted of 113 participants (38 CM, 39 PV, and 36 control) for the emotional GNG task and 109 participants (35 CM, 38 PV, and 36 control) for the nonemotional GNG task.

The groups did not differ significantly on age, sex, IQ, and SES (Table 1). As expected, participants in the CM and PV groups scored significantly higher than control participants on the BDI, BAI, NAPAS negative affect, RSLEs, and SDQ emotional and

total difficulties (p < .01) but scored lower than control participants on NAPAS positive affect (p < .001); nevertheless, their depression and anxiety scores were still within the normative range and below the cutoffs for moderate severity on the BDI and BAI, respectively. The CM and PV groups did not differ from one another except that the PV group scored higher on SDQ peer problems, and the CM group had a lower age of onset and a longer duration of early stress (p < .05) (Table 1).

#### **Task Performance**

There were no significant group differences on mean reaction times and performance accuracy for either GNG task. However, for inhibition to happy faces, participants in the PV group had marginally more false alarms (18%) than control participants (9%) (Table S1).

#### **Brain Activation**

#### Emotional GNG Task: FearNoGo-HappyGo Contrast.

During fearful face inhibition, the CM group exhibited greater activation than the PV group in a cluster comprising the bilateral calcarine, cuneus, and lingual gyri. In the CM group, BOLD response in this cluster was positively associated with parental antipathy (r = 0.54, p = .001, 95% CI [0.26 to 0.74]) (Figure S1). Additional clusters were observed in right limbic-striatal, left middle temporal gyrus (MTG), and bilateral cerebellum, although at a more liberal uncorrected threshold. There were no significant differences between the control group and the 2 stress-exposed groups (Table 2 and Figure 2A).

## Emotional GNG Task: HappyNoGo-FearGo Contrast.

During happy face inhibition, the CM group exhibited greater activation than the PV group in a left MTG cluster only at a more liberal uncorrected threshold. A planned comparison showed that the PV group also had lower activation than the control group in this cluster ( $F_{2,107} = 8.31$ , p = .009). No significant differences were observed between the CM and control groups, suggesting that MTG underactivation may be related to bullying exposure (Table 2 and Figure 2B).

**Emotional GNG Task: HappyGo-FearGo Contrast.** For happy face processing, the CM group had lower activation than the PV group in 4 clusters: the right orbitofrontal cortex, AI, and inferior, middle, and superior frontal gyri (cluster 1); the right inferior parietal lobule (IPL) and supramarginal gyrus, pre-/postcentral gyri, insula, putamen, and pallidum (cluster 2); the left IPL, superior parietal lobule (SPL), and precuneus (cluster 3); and the bilateral cuneus and precuneus (cluster 4). A planned comparison showed that the CM group had marginally lower activation in the right frontal cortex (cluster 1) than the control group (p = .08), but there was no significant difference between PV and control groups, suggesting that prefrontal underactivation may be related to aversive caregiving experiences (Table 2 and Figure 2C).

In the CM group, greater parental antipathy was significantly associated with lower activation in the right frontal cluster 1 (r=-0.40, p=.016, 95% CI [-0.64 to -0.08]) (Figure S2A), right supramarginal/parietal cluster 2 (r=-0.39, p=.017, 95% CI [-0.64 to -0.08]) (Figure S2B), and left parietal cluster 3 (r=-0.38, p=.022, 95% CI [-0.63 to -0.06]) (Figure S2C).

**Table 1. Sample Demographic Characteristics** 

	Childhood	D 16 11 1 11		Analysis <sup>a,b</sup>			
Characteristic	Maltreatment Group, $n = 38$	Peer Victimization Group, $n = 39$	Control Group, $n = 36$	$\chi^2$ or $F$	р	Group Comparisons	
Age, Years <sup>c</sup>	20.0 (1.64)	20.0 (1.85)	20.0 (1.69)	$F_{2,105} = 0.01$	n.s.	-	
IQ	104 (9.92)	103 (7.87)	102 (7.31)	$F_{2,105} = 0.91$	n.s.	-	
SES <sup>d</sup>	15.8 (4.18)	16.7 (3.77)	16.2 (3.22)	$F_{2,105} = 1.29$	n.s.	-	
Recent Stressful Life Events Scale	1.21 (1.04)	1.40 (1.33)	0.37 (0.71)	$F_{2.105} = 10.0$	<.001	CM, PV > C	
BDI	9.08 (7.05)	10.7 (9.25)	3.03 (3.49)	$F_{2.105} = 11.8$	<.001	CM, PV > C	
BAI	7.78 (8.21)	9.73 (9.96)	2.31 (3.45)	$F_{2,105} = 8.35$	<.001	CM, PV > C	
NAPAS	, ,	,	,			<u> </u>	
Negative affect	11.9 (3.90)	12.7 (5.83)	8.46 (2.95)	$F_{2.105} = 9.32$	<.001	CM, PV > C	
Positive affect	17.7 (4.72)	17.7 (4.49)	23.1 (3.18)	$F_{2.105} = 19.7$	<.001	CM, PV < C	
SDQ	, ,	,	,			<u> </u>	
Emotional problems	3.94 (2.14)	4.05 (2.48)	2.46 (1.67)	$F_{2.105} = 6.23$	.003	CM, PV > C	
Conduct problems	1.75 (1.50)	1.95 (1.60)	1.20 (1.08)	$F_{2.105} = 2.67$	(.07)	(PV > C)	
Hyperactivity	4.11 (2.44)	3.70 (2.41)	2.83 (2.24)	$F_{2,105} = 2.71$	(.07)	(CM > C)	
Peer problems	2.31 (1.58)	3.32 (1.85)	1.63 (1.41)	$F_{2.105} = 9.87$	<.001	PV > CM, C	
Prosocial	7.36 (2.06)	7.62 (2.01)	8.51 (1.48)	$F_{2,105} = 3.58$	.03	CM, PV < C	
Total difficulties score	12.1 (5.02)	13.0 (6.09)	8.11 (4.36)	$F_{2.105} = 8.91$	<.001	CM, PV > C	
CTQ				,		CTQ Severity Classification	
Physical abuse	14.5 (3.63)	6.64 (1.49)	5.22 (0.54)	$F_{2,105} = 176$	<.001	CM > PV,  C	
Emotional abuse	16.9 (3.93)	8.15 (2.23)	5.78 (1.09)	$F_{2,105} = 176$	<.001	CM > PV, C	
Physical neglect	9.53 (2.86)	6.44 (2.02)	5.72 (1.14)	$F_{2,105} = 33.5$	<.001	CM > PV, C	
Emotional neglect	16.4 (4.33)	9.69 (3.41)	6.81 (2.21)	$F_{2,105} = 76.3$	<.001	CM > PV, C	
CTQ total score	57.3 (10.3)	30.9 (6.91)	23.5 (3.66)				
CECA							
Mother antipathy	24.6 (7.81)	17.9 (6.15)	12.2 (3.81)	$F_{2,105} = 35.9$	<.001	CM > PV > C	
Father antipathy	24.0 (7.74)	17.2 (5.66)	11.8 (3.35)	$F_{2,105} = 38.4$	<.001	CM > PV > C	
rPEQ							
Relational victimization	1.69 (2.12)	8.42 (2.61)	0.53 (0.84)	$F_{2,105} = 166$	<.001	PV > CM, C	
Overt victimization	1.03 (1.77)	9.61 (4.97)	0.06 (0.23)	$F_{2,105} = 106$	<.001	PV > CM, C	
Reputational victimization	1.09 (2.13)	8.95 (2.71)	0.14 (0.48)	$F_{2,105} = 211$	<.001	PV > CM, C	
ECIPQ	1.26 (2.73)	9.89 (6.83)	0.75 (1.07)	$F_{2,105} = 51.4$	<.001	PV > CM, C	
Age at Onset of CM or PV, Years	6.34 (2.67)	10.6 (2.41)	_	$F_{2,105} = 53.2$	<.001	CM < PV	
Duration of CM or PV, Years	8.82 (3.56)	4.07 (1.66)	_	$F_{2,105} = 56.5$	<.001	CM > PV	
Sex, Males	16 (42%)	17 (44%)	16 (44%)	$\chi^2 = 0.04$	n.s.	-	
Ethnicity <sup>e</sup>				$\chi^2 = 9.41$	n.s.	-	
Chinese	32 (84%)	31 (79%)	34 (94%)	_	_	-	
Malay	4 (11%)	2 (5%)	1 (3%)	_	_	_	
Indian	2 (5%)	6 (16%)	1 (3%)	_	_	-	

Values are presented as mean (SD) or n (%).

BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; C, control (group); CECA, Childhood Experience of Care and Abuse; CM, childhood maltreatment (group); CTQ, Childhood Trauma Questionnaire; ECIPQ, European Cyberbullying Intervention Project Questionnaire; NAPAS, Negative and Positive Affect Scale; n.s., nonsignificant; PV, peer victimization (group); rPEQ, Revised Peer Experiences Questionnaire; SDQ, Strengths and Difficulties Questionnaire; SES, socioeconomic status.

**Letter GNG Task.** The XNoGo-LetterGo contrast yielded no significant group differences. However, for the LetterGo-XNoGo contrast, both the CM and PV groups exhibited greater activation than the control group in regions of the default mode network (DMN) including the left precuneus,

SPL, and posterior cingulate cortex (PCC) (cluster 1) and the right SPL and precuneus (cluster 2), suggesting that increased DMN activation may be related to exposure to early-life interpersonal stress from caregivers or peers (Table 3 and Figure 2D).

<sup>&</sup>lt;sup>a</sup>Tests adjusted for multiple comparisons.

 $<sup>{}^</sup>b\mathsf{The}$  values in parentheses are marginally statistically significant.

<sup>&</sup>lt;sup>c</sup>The age range was 17 to 21 years.

<sup>&</sup>lt;sup>d</sup>The SES total score ranges from 6 to 26, with higher values indicating higher status.

eThe Fisher's exact test was used.

Table 2. Regions of Differential Activation in the Emotional Go/NoGo Task

	Cluster Level						
	Peak MNI		No. of		Group Comparisons <sup>t</sup>		
Contrasts and Brain Regions	Coordinates, x, y, z	t-max	Voxels	p Value <sup>a</sup>			
FearNoGo-HappyGo							
Bilateral Calcarine/Cuneus/Lingual Gyri (BA 18)	10, -88, 14	4.50	523	.038	CM > PV		
Right Putamen/Globus Pallidus/Amygdala	24, -6, -6	3.59	160	.038 <sup>c</sup>	CM > PV		
Left Middle Temporal (BA 37/39)	-48, -54, 8	4.25	184	.028 <sup>c</sup>	CM > PV		
Right Cerebellum/Fusiform	20, -44, -18	3.61	212	.019 <sup>c</sup>	CM > PV		
Left Cerebellum	-26, -42, -28	3.34	191	.025 <sup>c</sup>	CM > PV		
Right Precuneus (BA 18)	12, -62, 24	3.31	174	.032 <sup>c</sup>	CM > PV		
Left Precuneus (BA 18)	-14, -72, 30	3.23	166	.035 <sup>c</sup>	CM > PV		
HappyNoGo-FearGo							
Left Middle Temporal Gyrus	-40, -52, 6	3.79	198	.021 <sup>c</sup>	CM, C > PV		
HappyGo-FearGo							
Right Orbitofrontal, Inferior and Middle Frontal Gyri (BA 10, 45, 46)/Anterior Insula	28, 32, 6; 26, 30, 16; 32, 38, 0	4.18	473	.032	CM < PV		
Right Inferior Parietal Lobule (BA 40)/Putamen/Globus Pallidus/Sensorimotor Cortex	56, -26, 28; 24, -8, -2; 44, -20, 38	4.05	1260	<.001	CM < PV		
Left Inferior Parietal Lobule (BA 40)/Superior Parietal Lobule and Precuneus (BA 7)	-26, -42, 34; -30, -40, 54; -36, -42, 62	4.27	489	.027	CM < PV		
Bilateral Precuneus and Cuneus (BA 18/31)	-22, -68, 22	3.69	517	.020	CM < PV		
Left Inferior Parietal Lobule, Supramarginal Gyrus (BA 40)/Posterior Insula	-38, -28, 22	4.05	422	(.055)	CM < PV		

BA, Brodmann area; C, control (group); CM, childhood maltreatment (group); MNI, Montreal Neurological Institute; PV, peer victimization (group).

## **DISCUSSION**

To our knowledge, this is the first fMRI study on response inhibition that directly compares CM and PV using a sizable community youth sample free from medications and drug abuse while controlling for the timing and duration of early-stress exposure and the number of recent stressors. These control participants are crucial to elucidate the effects of early-life interpersonal stress independently from confounding effects associated with psychiatric diagnosis, medications, drug abuse, and recent stress (50).

There were no significant group differences in behavioral performance on the emotional and nonemotional GNG tasks. However, groups differed in brain activation during emotional but not nonemotional response inhibition, indicating that atypical inhibitory neural process in CM and PV are dependent on emotional content. During inhibition to fearful faces, the CM group showed significantly greater activation than the PV group in the extrastriate cortex comprising the bilateral calcarine, cuneus, and lingual gyri. They also exhibited increased activation in right limbic-striatal, left MTG, and bilateral cerebellar regions, although at a more liberal threshold. During inhibition to happy faces, there was a trend of PV-specific underactivation in the left MTG. These results demonstrate that while performance on the task remained similar for the 3 groups, the CM and PV groups recruited different brain regions to successfully execute inhibition to fearful and happy stimuli, respectively. When responding to happy faces, the CM group

had lower activation than the PV group in widespread areas including the right fronto-limbic-striatal and bilateral parietal and occipital regions. They also demonstrated (at a trend level) decreased activation in right fronto-limbic areas compared with control participants, suggesting that frontolimbic hypoactivation may be maltreatment-specific. Furthermore, both the CM and PV groups activated the DMN (precuneus, SPL, pre-/postcentral and PCC) more than the control group during the cognitively low-load LetterGo blocks.

The CM and PV groups had comparable depressive, anxiety, negative affect, and SDQ scores within normative ranges, but they exhibited distinct atypical neural activation patterns during emotional response inhibition. The CM group exhibited greater activation than the PV group in visual-emotional information processing regions when inhibiting responses to fearful faces. Notably, the posterior cluster activation in the CM group was positively correlated with parental antipathy, suggesting that increased activation in these perceptual processing regions may be related to early aversive caregiving. The observed hyperactivation of the limbic-visual-cerebellar regions during fearful inhibition in the CM group may reflect the brain's attempt to facilitate emotional inhibitory control and functionally adapt to growing up in threatening environments in ways that promote survival.

Despite the neural processing differences that we observed, the CM group did not show performance deficits. Prior work indicates that reduced ability to inhibit responses to

<sup>&</sup>lt;sup>a</sup>Familywise error rate-corrected p values at cluster level.

<sup>&</sup>lt;sup>b</sup>Group differences in brain activation were conducted with recent stressful life events, duration, and age at onset of CM or PV as covariates.

<sup>&</sup>lt;sup>c</sup>p Value uncorrected at the cluster level.

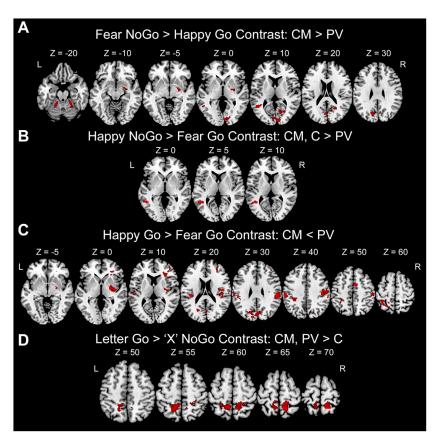


Figure 2. Between-group differences in brain activation during the Go/NoGo (GNG) tasks. Axial slices are marked with the z coordinate as distance in millimeters from the anterior-posterior commissure. The right side of the image corresponds to the right side of the brain. Depicted activations are clustercorrected to p < .05, using an individual voxel height threshold of p < .005. (A) Areas of increased activation in childhood maltreatment (CM) group compared with peer victimization (PV) group during FearNoGo relative to HappyGo trials, (B) areas of reduced activation in the PV group compared with CM and control (C) groups during HappyNoGo relative to FearGo trials, (C) areas of reduced activation in the CM group compared with the PV group during HappyGo relative to FearGo trials, and (D) areas of increased activation in CM and PV groups compared with the C group during LetterGo relative to XNoGo trials. L, left; R, right.

threatening stimuli is associated with internalizing symptoms in maltreated girls (51) and that inhibitory control is more impeded in trauma-exposed children than in older youths who may use compensatory strategies with increasing age (52). We note that our CM youths represent high-functioning community youths with IQ and SES comparable to control participants. Therefore, the lack of performance deficits is consistent with the view that our sample reflects resilient maltreated youths, although with some (subclinical) symptoms of anxiety and depression.

Two comparisons in the current study yielded group-related differences in activation within the striatum and dorsal

amygdala. Compared with the PV group, the CM group exhibited higher activation in these regions for the FearNoGo-HappyGo contrast but lower activation for the HappyGo-FearGo contrast. It is noteworthy that for both of these directional contrasts, happy faces are the primary behavioral target, i.e., participants should respond when seeing happy faces. Happy faces are positive social signals that are rewarding and motivate others to approach (48), and youths have an especially increased proclivity to respond to happy faces in emotional GNG tasks (53). The striatum, particularly the ventral striatum, is strongly engaged during reward learning and risk/reward choice behaviors in youths (54,55).

Table 3. Regions of Differential Activation in the Nonemotional Go/NoGo Task

	Cluster Level					
Contrast and Brain Regions	Peak MNI Coordinates, x, y, z	t-max	No. of Voxels	p Value <sup>a</sup>	Group Comparisons <sup>b</sup>	
LetterGo-XNoGo						
Left Precuneus and Superior Parietal Lobule (BA 7), Posterior Cingulate Cortex (BA 31)	-14, -50, 56	4.69	610	.015	CM, PV > C	
Right Superior Parietal Lobule and Precuneus (BA 7)	14, -54, 68	4.76	492	.044	CM, PV > C	

BA, Brodmann area; C, control (group); CM, childhood maltreatment (group); MNI, Montreal Neurological Institute; PV, peer victimization (group).

<sup>&</sup>lt;sup>a</sup>Familywise error rate-corrected *p* values at the cluster level.

<sup>&</sup>lt;sup>b</sup>Group differences in brain activation were conducted with recent stressful life events, duration, and age at onset of CM or PV as covariates. Planned comparison showed that the PV group also had greater activation than the control group only in both clusters (cluster 1:  $F_{2,103} = 9.54$ , p = .001; cluster 2:  $F_{2,103} = 9.21$ , p = .001).

Importantly, this region shows heightened recruitment when taking risks in the presence of peers compared with risk taking in the presence of parents (56). Therefore, striatal regions are deeply involved in motivation and decision making in social contexts (57).

Consistent with this viewpoint, we propose that for our CM group, happy faces may be less rewarding at either the cognitive or neural level, similar to prior findings in autism and major depressive disorder (48). Maltreated individuals may have limited experience with positive affect in their early-life relationships and avoid positive emotional cues because they are inconsistent with their internal cognitive schemas for social interactions (58). Indeed, prior studies have revealed a trend toward attentional avoidance of positive facial emotions in CM (58,59). Alternatively, maltreated youths may be more wary of smiling faces, perceiving them as potential threats in a mechanism similar to a hostile attributional bias (60). In contrast, the enhanced activation in striatal reward regions for happy faces in youths exposed to PV may motivate them to make indiscriminate advances toward peers (53,61), making them more vulnerable to bullies

For the letter GNG task, there were no significant group differences in response inhibition at the neural and behavioral levels, consistent with our prior finding that maltreated youths did not exhibit functional abnormalities during successful response inhibition using the stop task (2). Perhaps the early-stress groups exhibit atypical activation only when there is an interpersonal element involved, such as facial emotions, which could be linked to the interpersonal stressors that they experienced.

Intriguingly, we observed increased activation in DMN regions for both the CM and PV groups compared with control participants in the LetterGo condition, consistent with a recent meta-analysis (13). The DMN has long been associated with mind wandering and self-referential thought (62), especially during well-rehearsed or cognitively less-demanding tasks (63). Recent findings suggest that mind wandering is not a negative phenomenon per se but instead has adaptive consequences leading to mood improvements (64) and aiding in creativity, problem-solving, and future planning (65). Given that our letter GNG task always followed the emotional GNG task, we speculate that the increased mind wandering during the low-load LetterGo condition might have been used strategically by participants in the early-stress groups to shift their thoughts toward pleasant topics, thereby facilitating response inhibition during the letter GNG task.

## **Strengths and Limitations**

Strengths of this study are that all participants were free from psychopathology, medications, and drug abuse and the fact that we controlled for the timing and duration of CM/PV exposure and current stressors. The groups were matched on important demographic variables, and there were no significant differences between the 2 early-stress groups on psychopathologic symptoms, which were within nonclinical ranges. These variables were not significantly correlated with brain activation in areas that differed between groups and are unlikely to confound our findings. We used both emotional and nonemotional GNG tasks within the same MRI session to

examine response inhibition, enabling direct comparison of affective and nonaffective response inhibition. Moreover, our task was designed with blocks of trials in which participants knew in advance that no executive decision was required, providing a recommended proactive rather than a reactive control mechanism in GNG tasks (66).

We acknowledge that block design limits our ability to examine neural correlates of specific cognitive components of response inhibition, i.e., stimulus perception, making a decision, and executing that decision. However, the primary advantage to this design is enabling the investigation of response inhibition in multiple contexts within a tightly controlled and efficient task. Using an event-related GNG task with temporally spaced NoGo stimuli would make our task exceedingly long and would not incorporate the continuous and prepotent responses desired here.

We further acknowledge that this study is cross-sectional, and the findings are correlational, as is true with most neuro-imaging studies. The use of retrospective self-report data may be subject to recall biases, and the SES measure does not include information on parents' incomes; however, youths often have difficulties reporting this information (46). In addition, our results may not be generalizable to the childhood sexual abuse population. It is also unclear how the length of time between CM/PV exposure and MRI might have influenced the findings. Lastly, while we carefully matched our groups on sex, we did not directly examine the influence of sex on brain activation.

## **Conclusions**

In sum, our findings indicate that early-stress-exposed individuals show neural alterations compared with their non-stressed counterparts, particularly during decision making in socioemotional contexts. These findings are most applicable to the more resilient portion of trauma-exposed community youths, for whom atypical patterns of activation may reflect compensatory brain mechanisms to facilitate emotional inhibitory control in the face of prior aversive experiences. From an intervention perspective, our findings can inform meditation and intentional mind-wandering training to direct attention away from stressful emotional stimuli, as well as training individuals to accurately decode socioemotional cues during peer interactions.

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The data that support the findings of this study are available from the corresponding author upon reasonable request.

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