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The Dichotomy of Threat and Deprivation as Subtypes of Childhood Maltreatment: Differential Functional Connectivity Patterns of Threat and Reward Circuits in an Adult Trauma Sample

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ABSTRACT

BACKGROUND: Childhood maltreatment is associated with reduced activation of the nucleus accumbens, a central region in the reward network, and overactivity in the amygdala, a key region in threat processing. However, the long-lasting impact of these associations in the context of later-life stress is not well understood. The current study explored the association between childhood threat and deprivation and functional connectivity of threat and reward regions in an adult trauma sample.

METHODS: Trauma survivors (N = 169; mean age [SD] = 32.2 [10.3] years; female = 55.6%) were recruited from a level I trauma center. Two weeks after injury, participants completed the Childhood Trauma Questionnaire (measuring experiences of threat and deprivation) and underwent resting-state functional magnetic resonance imaging. Seed-to-voxel analyses evaluated the effect of childhood threat and deprivation on amygdala and nucleus accumbens resting-state connectivity.

RESULTS: Higher levels of threat were associated with increased connectivity between the right nucleus accumbens with temporal fusiform gyrus/parahippocampal gyrus and the left amygdala and the precuneus (false discovery rate–corrected $p < .05$). After controlling for posttraumatic symptoms 2 weeks posttrauma and lifetime trauma exposure, only the nucleus accumbens findings survived. There were no significant relationships between experiences of childhood deprivation and amygdala or nucleus accumbens connectivity.

CONCLUSIONS: Experiences of threat are associated with increased nucleus accumbens and amygdala connectivity, which may reflect a preparedness to detect salient and visual stimuli. This may also reflect a propensity toward dysregulated reward processing. Overall, these results suggest that childhood threat may be contributing to aberrant neural baseline reward and threat sensitivity later in life in an adult trauma sample.

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Experiences of childhood maltreatment are related to several negative mental health outcomes across the lifespan, including depression, posttraumatic stress disorder (PTSD), and suicidal ideation and behavior (1–4). The term childhood maltreatment generally refers to experiences of abuse and/or neglect. An estimated 40% of youth experience maltreatment by age 18 years (5,6), with impacts often extending beyond childhood and adolescence into adulthood (2,5).

While studies have explored the link between childhood maltreatment and adult outcomes, less research has considered this in the context of later trauma in adulthood, which is quite common. In fact, an estimated 70% of adults in the United States will experience a traumatic event during their lifetime (7,8), which is related to the development of psychopathology (e.g., PTSD) (9,10). Despite this, the specific impact of childhood maltreatment on outcomes in the context of adult trauma needs further exploration. Moreover, the neurobiological underpinnings of these associations are unclear.

Neural Impacts of Childhood Maltreatment

One impact of childhood maltreatment is on the neurobiological systems that instantiate reward and threat processing. The nucleus accumbens has long been considered a central convergence point in the neural reward pathway that has a role in integrating information about reward, increasing motivation, and leading to action (11). As such, the structure and function of this region may be atypical in individuals who have experienced childhood maltreatment. Experiences of childhood maltreatment are related to reduced volume and variations in the white matter microstructure that connects the nucleus

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accumbens and the orbitofrontal cortex, a network that is highly implicated in reward processing (12,13). Functional neuroimaging studies have also found that adults who were maltreated during childhood show reduced activation in the nucleus accumbens during reward-based tasks, when the region is typically activated, suggesting disrupted motivation for, and/or anticipation of, reward (14,15). The effect of these neurobiological changes can be seen behaviorally as well. Children exposed to maltreatment show slower response times during high-reward tasks and a reduced sensitivity in differentiating between high and low reward value (16). Overall, previous literature highlights a relationship between childhood maltreatment and the nucleus accumbens, such that childhood maltreatment is related to reduced volume and aberrant activation patterns. Taken together, this previous literature highlights a relationship between childhood maltreatment and the nucleus accumbens that is reflected in multiple aspects of brain structure and function.

Another brain region that shows aberrant structure and function as a result of maltreatment is the amygdala, a key region for threat processing and fear generation in the medial temporal lobe (17). Neuroimaging analyses have shown that childhood maltreatment is related to decreased amygdala inhibition and hyperactivity of the region during emotional and face processing tasks, suggesting an overactive fear and threat response (18–22). In sum, experiences of childhood maltreatment are related to atypical function of a key threat-detection brain region that may reflect an overactive threat system.

The amygdala has also been implicated in studies of child maltreatment that use resting-state functional magnetic resonance imaging (rs-fMRI) (23,24). rs-fMRI can be used to measure intrinsic functional connectivity and activation, thus providing a window into tonic brain connectivity when an individual is not cognitively or affectively engaged in a specific externally directed task. Research has consistently found significant associations between childhood maltreatment and amygdala connectivity with various regions such as decreased connectivity with the precuneus (24). Research has also found decreased connectivity between the right amygdala with the insula and subcortical structures, including the hippocampus and the putamen, among adults who experienced childhood emotional maltreatment (24). Thus, research has found that disrupted functional connectivity of the amygdala may affect emotional processing in adulthood.

The effect of childhood maltreatment on amygdala connectivity has been inconsistent in terms of directionality. For example, another study found positive associations between childhood maltreatment and connectivity of the amygdala and parahippocampal gyrus, right inferior temporal gyrus, right orbitofrontal cortex, hippocampus, cerebellum, and brainstem using an adult sample (19). In combat veterans, increased scores on the Childhood Trauma Questionnaire (CTQ) were associated with decreased connectivity between the amygdala and the ventromedial prefrontal cortex (25). Thus, although research has found significant associations between childhood maltreatment and amygdala connectivity, no consistent signature of amygdala connectivity has been identified. Moreover, even identified circuits (amygdala-hippocampus) often vary in whether they are positively (19) or negatively (24) associated with childhood maltreatment. These discrepant findings may reflect whether studies explored a specific type of maltreatment (e.g., emotional maltreatment) (24) or generally assessed for experiences of maltreatment (19).

The Dichotomy of Childhood Maltreatment: Threat Versus Deprivation

In examining the effects of childhood maltreatment, research has been both general and specific; some studies have prioritized the cumulative experience of abuse/neglect, while others have focused on individual types. McLaughlin *et al.* (3) offered an important distinction between threat and deprivation as subtypes of childhood maltreatment. Experiences of threat refer to the presence of threat (e.g., sexual or physical abuse), whereas deprivation highlights the absence of appropriate needs, stimuli, and responsiveness (e.g., physical, caregiver, and emotional neglect) (3). The model proposes this distinction because of the differential neural, behavioral, and psychopathological impact of threat and deprivation, while recognizing that such experiences may co-occur (3,26).

Evidence from animal and human models suggests that deprivation is linked to poorer cognitive functioning resulting from reduced sensory input and possible overpruning of sensory regions (3). Interestingly, children who are deprived are more likely to show blunted reward processing, whereas those who experience threat have shown increased activation in the ventral striatum (27). Behavioral tasks suggest that deprivation predicts poor cognitive control but not challenges to automatic emotional regulation; by contrast, exposure to threat is related to deficits in automatic emotional regulation but not cognitive control (28). Neuroimaging research tells a similar story, wherein experiences of threat—but not neglect—affect stress-related and fear regions of the brain, such as the amygdala, whereas experiences of neglect are related to synaptic pruning of sensory areas and association cortices (3,29). Overall, evidence highlights that threat and deprivation have unique neurobiological and behavioral impacts, but this has not been specifically explored with the threat and reward system.

The Current Study

Thus far, research has established that childhood maltreatment is related to deficits in reward processing and aberrant fear responses (12,30). The underlying neurobiological mechanisms of this have also been explored, with two clear regions emerging: the nucleus accumbens as a central node for reward processing and the amygdala as a fear and threat response region (11,17). However, some important gaps remain in the literature. First, many studies have focused on the structure or activation patterns of the nucleus accumbens and amygdala (12,14,30), but the intrinsic connectivity patterns are less clear for the nucleus accumbens. Moreover, while the association between childhood maltreatment and connectivity of the amygdala has been explored, findings remain mixed regarding the directionality. Furthermore, the cumulative and long-term impact of early-life maltreatment on engagement of neural reward and threat circuits following a traumatic experience later in life is not fully understood.

Thus, this study aimed to understand connectivity patterns related to childhood maltreatment in a way that considers the

unique role of childhood maltreatment on those circuits while also considering this in the context of later trauma exposure during adulthood. Based on extant literature, the current study used lateralized (vs. bilateral) whole-brain analyses for each of our regions (i.e., amygdala and nucleus accumbens). Broadly, a systematic review of amygdala lateralization and its role in emotional processing suggested that the left amygdala is more often active than the right amygdala (31). Specific to experiences of abuse and childhood maltreatment, prior work has found lateralized effects to the right and left amygdala and the left nucleus accumbens (32,33). Prior literature has also demonstrated that fear processing in individuals with PTSD had lateralized amygdala effects when compared with control participants (34). Based on this literature, we wanted to investigate whether these lateralized effects were consistent in an adult trauma sample with a history of childhood maltreatment. The first aim of the current study was to explore the relationship between childhood maltreatment and connectivity patterns with the nucleus accumbens and amygdala as seeds of interest in an adult trauma sample to understand the cumulative effect of early adversity on later (i.e., adulthood) outcomes. Threat and deprivation were examined separately to assess for distinct connectivity patterns between subtypes of maltreatment. To increase the specificity of this distinction, the other subtype was covaried in the respective analyses. Given that the existing body of literature in this area is limited and has yielded inconsistent findings (i.e., increased and/or decreased functional connectivity), we did not have a priori directional hypotheses for the amygdala or nucleus accumbens.

The second aim of the study was to isolate the role of childhood maltreatment, given the trauma sample. Thus, the same analyses were conducted, examining the relationship between threat and deprivation and respective connectivity patterns. However, posttraumatic symptoms reported soon after the index trauma were used as a covariate to reduce the impact of the acute trauma on observed connectivity patterns. Moreover, to reduce the effect of other trauma exposure, lifetime trauma was also used as a covariate. Similarly, no a priori directional hypotheses were proposed, but we expected connectivity patterns to remain significant even after controlling for lifetime trauma and posttraumatic symptoms reported soon after the trauma (e.g., at the baseline time point), indicating that childhood maltreatment is related to aberrant connectivity patterns beyond the impacts of the current trauma.

METHODS AND MATERIALS

All protocols were approved by the Medical College of Wisconsin Institutional Review Board. Informed consent was obtained from participants prior to data collection.

Participants

Participants (N = 215) were recruited from an emergency department at a level I trauma center as part of a longitudinal study (35–37) investigating neurobiological predictors of PTSD in adults. Inclusion criteria included English-speaking individuals between the ages of 18 and 65 years who had experienced a traumatic injury. Exclusion criteria included moderate to severe traumatic brain injury, spinal cord injury, history of psychotic or manic symptoms, traumatic injuries resulting from suicide attempts or self-harm, or a contraindication for MRI scanning (e.g., pregnancy). Within 2 to 4 weeks of recruitment from the emergency department (mean [SD] = 14 [6] days), enrolled participants underwent a series of functional and structural MRI scans and a battery of self-report measures, including measures of childhood maltreatment. Of the recruited participants, 169 (94 women; mean age [SD] = 32.2 [10.3] years) had usable rs-fMRI scans postinjury and self-reported data on childhood maltreatment (15 participants were missing usable fMRI data). Most participants (72.2%) had experienced a motor vehicle crash. Other mechanisms of injury included assault/altercation (12.2%) and being struck as a pedestrian (4.1%).

Measures

Childhood Maltreatment. Childhood maltreatment was measured using the CTQ (38), a retrospective self-report measure that asks about experiences of trauma in childhood. The CTQ-Short Form (39) was used for this study. The CTQ-Short Form comprises 28 items, which can be divided into 5 subscales: 1) physical abuse, 2) emotional abuse, 3) sexual abuse, 4) emotional neglect, and 5) physical neglect. Each item requires respondents to use a Likert scale ranging from 1 to 5 (1 = "never true" and 5 = "very often true") to rate the frequency of an event that occurred during childhood. The CTQ has been found to have strong psychometric properties (Cronbach's alpha = 0.95, intraclass correlation coefficient = 0.88) (38).

For the current analyses, we created 2 subscales within the CTQ: 1) threat (physical abuse, emotional abuse, and sexual abuse) and 2) deprivation (emotional neglect and physical neglect). These subtypes were made by summing subscales, which resulted in scores ranging from 15 to 75 for the threat subscale and 10 to 50 for the deprivation subscale. This methodology has been used in prior research (3,27).

Posttraumatic Symptoms Reported Soon After the Adult Trauma. The PTSD Checklist for DSM-5 (40), a validated self-report measure, was used to assess the severity of acute traumatic distress 2 weeks after the traumatic event. Participants rated how much they were bothered by each of the 20 items using a 5-point Likert scale with response options ranging from 1 (not at all) to 5 (extremely). Responses were summed to provide an overall symptom severity score. In the current sample, the PTSD Checklist for DSM-5 showed high reliability (Cronbach's alpha = 0.95).

Other Traumatic Exposure. The Life Events Checklist (LEC) (41) was used to capture lifetime trauma exposure in the sample. This is a validated self-report measure that contains 17 items describing stressful, traumatic events (e.g., natural disasters, combat, sexual assault). Four items (items 6 through 9) were excluded from the total calculation because they refer to experiences of physical or sexual assault, which may be confounded with childhood maltreatment experiences. The LEC was used as a covariate in the analyses to isolate the role of childhood maltreatment in the model and avoid misattributing results to previous trauma exposure.

Neuroimaging Procedures

Images were collected using a General Electric Discovery MR750 3T scanner equipped with a 32-channel head coil. T1-weighted high-resolution anatomical scans were acquired (field of view = 240 mm^2; matrix = 256 × 224 mm^2; slice thickness = 1 mm; 150 slices, repetition time/echo time = 8.2/3.2 ms; flip angle = 12°, voxel size = 0.9375 × 1.071 × 1 mm^3) for coregistration with functional images and structural analysis. Resting-state images were acquired during an 8-minute eyes-open scan (240 volumes; field of view = 22.4 cm^2; matrix = 64 × 64 mm^2; slice thickness = 3.5 mm; 41 sagittal slices; repetition time/echo time = 2000/25 ms; flip angle = −77°; voxel size = 3.5 × 3.5 × 3.5 mm^3).

Analytic Plan

fMRI Data Preprocessing. Standard preprocessing procedures were completed using the MATLAB-based (version 2019b; The MathWorks, Inc.) SPM (version 12) CONN Toolbox (version 20). Preprocessing steps include discarding the first 3 repetition times, motion correction using a 6-parameter linear transformation, normalization to Montreal Neurological Institute 152 template, and spatial blurring with a 4 mm full width at half maximum smoothing kernel. To address confounding effects of motion, volumes with framewise displacement over 0.3 mm were excluded from the analysis. Nuisance covariates (head motion parameters, white matter signal, and cerebrospinal fluid signal) were regressed out during the first level of analyses. Finally, participants were removed from analyses if more than 20% of the resting-state volumes were scrubbed. This resulted in the exclusion of 7 participants, which resulted in a final *n* of 169.

To examine the relationship between childhood threat and deprivation, we specified 8 separate whole-brain connectivity models. Each a priori seed region of interest (left/right amygdala, left/right nucleus accumbens using default regions of interest [using the Harvard-Oxford subcortical atlas] provided in the CONN Toolbox) was run separately for childhood threat and deprivation. In each model, we examined the effects of 1 between-participants factor (childhood threat or deprivation) on functional connectivity of each seed region with the rest of the brain, adjusting for age, gender, and the other maltreatment subtype. These analyses were repeated adjusting for acute traumatic distress (PTSD Checklist for DSM-5) and lifetime trauma exposure (LEC). In addition, given that previous research has posited that early childhood maltreatment is a risk factor for severe, chronic, and treatment-resistant depression and substance abuse later in life (42,43) and that major depressive disorder (MDD) and substance use disorder (SUD) are related to aberrant functional connectivity of reward and threat systems (44), we ran post hoc sensitivity analyses controlling for MDD and SUD. All analyses were corrected with a height threshold of $p < .001$ and a cluster-size threshold of a false discovery rate (FDR)–adjusted $p < .05$.

RESULTS

Table 1 summarizes participant demographic characteristics. Table 2 describes the correlations between threat, deprivation, posttraumatic symptoms reported soon after the adult trauma (at the baseline time point), and age.

Table 1. Demographic and Clinical Characteristics of Study Participants

Characteristic	Value
Sex, Female, *n* (%)	94 (55.6%)
Age, Years	
Mean (SD)	32.2 (10.3)
Range	18.1–58.6
Threat, CTQ	
Mean (SD)	24.55 (11.53)
Range	15–70
Deprivation, CTQ	
Mean (SD)	18.87 (7.86)
Range	10–41
PTSD Symptoms, PCL-5	
Mean (SD)	27.98 (18.40)
Range	0–73
Lifetime Depression Diagnosis, %	
Yes	15.0%
No	59.2%
Recurrent	25.9%
Substance Use Disorder Diagnosis, %	
Yes	21.9%
No	78.1%
Mechanism of Injury, %	
Motor vehicle crash	72.2%
Gun shot	0.6%
Stab	1.2%
Fall	1.2%
Pedestrian struck	4.1%
Domestic violence	1.2%
Assault/altercation	12.4%
Other	7.1%
Race, %	
Asian	1.8%
Black or African American	58.0%
More than one race	5.9%
White	27.8%
Did not disclose	6.5%
Hispanic Ethnicity, %	
Yes	7.7%
No	90.5%
Did not disclose	<1.8%

CTQ, Childhood Trauma Questionnaire; PCL-5, PTSD Checklist for DSM-5; PTSD, posttraumatic stress disorder.

Threat

Nucleus Accumbens. More experiences of threat in childhood, adjusting for age, gender, and experiences of deprivation, were associated with increased right nucleus accumbens connectivity with the temporal fusiform gyrus/parahippocampal gyrus (xyz = 34, −28, −20; k = 152; p_{FDR} = .0008) (Figure 1A). This cluster survived after controlling for baseline posttraumatic symptoms and lifetime trauma exposure (xyz = 34, −28, −20; k = 95; p_{FDR} = .0175). No significant relationship was observed with left nucleus accumbens connectivity. In addition, in post hoc analyses testing for the

Table 2. Bivariate Correlations Among Threat, Deprivation, PTSD Symptoms, and Age

Variable	Threat	Deprivation	PTSD Symptoms	Age
Threat (CTQ)	–	0.485[a]	0.318[a]	0.155[b]
Deprivation (CTQ)	–	–	0.164[b]	−0.014
PTSD Symptoms (PCL-5)	–	–	–	−0.179[b]
Age	–	–	–	–

CTQ, Childhood Trauma Questionnaire; PCL-5, PTSD Checklist for DSM-5; PTSD, posttraumatic stress disorder.

[a]$p < .001$.

[b]$p < .05$.

confounding effect of MDD and SUD, the main effect survived.

Amygdala. No significant relationship was observed in the analysis examining experiences of childhood threat, adjusting for age, gender, and experiences of deprivation in childhood, on right amygdala connectivity. More experiences of threat in childhood, adjusting for age, gender, and experiences of deprivation in childhood, were associated with increased left amygdala connectivity with the precuneus (xyz = 8, −62, 52; k = 102; p_{FDR} = .02) (Figure 1B). In addition, in post hoc analyses controlling for MDD and SUD, the main effect of threat survived. However, this cluster did not survive after controlling for baseline posttraumatic symptoms or lifetime trauma exposure.

Deprivation

No significant relationships were observed in analyses examining experiences of childhood deprivation, adjusting for age, gender, and experiences of threat in childhood, on left or right amygdala connectivity or left or right nucleus accumbens connectivity. These results remained consistent after controlling for baseline posttraumatic symptoms and lifetime trauma exposure.

DISCUSSION

The current study investigated the relationships between childhood maltreatment (threat and deprivation) and rs-fMRI of the amygdala and nucleus accumbens in traumatically injured adults. Notably, greater exposure to threat experiences during childhood was associated with increased right nucleus accumbens–temporal fusiform gyrus/parahippocampal gyrus and left amygdala–precuneus connectivity. However, deprivation during childhood was not significantly associated with different connectivity patterns of either the amygdala or the nucleus accumbens. When controlling for baseline PTSD symptoms and lifetime trauma exposure, only the right nucleus accumbens–temporal fusiform gyrus/parahippocampal gyrus cluster remained significant. The current study demonstrates the negative downstream effects via dysregulated connectivity of threat and reward circuits in adults.

Childhood threat may play a role in later-in-life reward processing. Increased connectivity between the right nucleus accumbens and temporal fusiform gyrus/parahippocampal

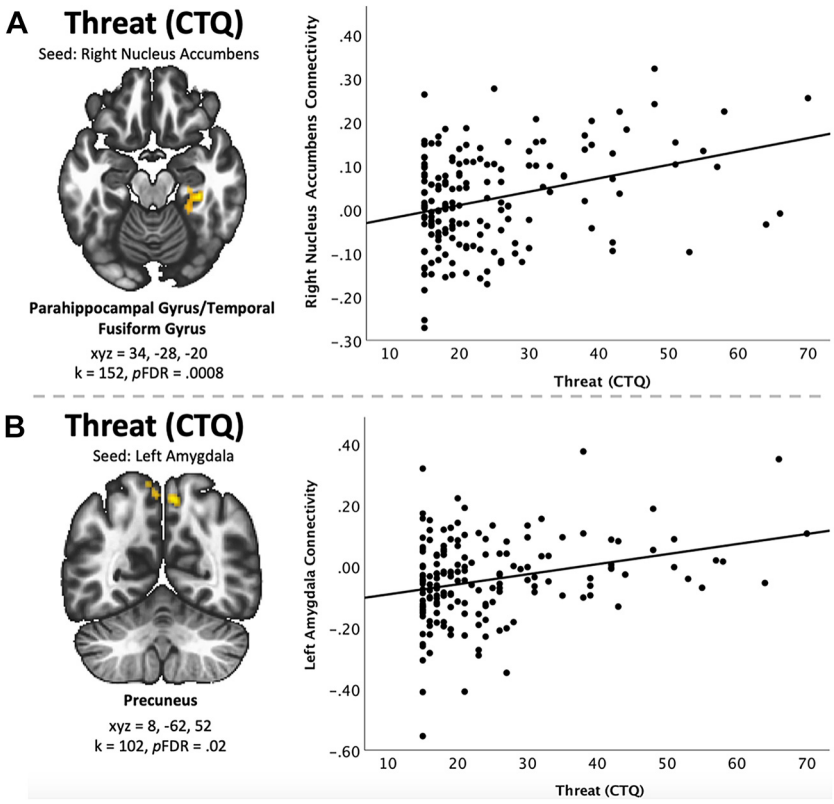

Figure 1. Whole-brain connectivity analyses. **(A)** Right nucleus accumbens connectivity with the parahippocampal gyrus/temporal fusiform gyrus. **(B)** Left amygdala connectivity with the precuneus. Graph depicts the association between childhood maltreatment (threat) and connectivity for right nucleus accumbens connectivity with the parahippocampal gyrus/temporal fusiform gyrus. Results were adjusted for age, gender, and childhood deprivation. Significant clusters were identified using a voxelwise threshold of $p < .001$ and cluster threshold of false discovery rate (FDR)–corrected $p < .05$. Graph is displayed for illustrative purposes to depict the direction of the association. CTQ, Childhood Trauma Questionnaire.

gyrus may reflect changes in vigilance or preparedness to detect salient visual stimuli, such as faces. These regions are implicated in facial and object recognition (45,46) and memory recall (47). In addition, preclinical research has implicated the nucleus accumbens in discriminating between danger and uncertainty (48), fear scaling (49), and discriminating between uncertain threat and safety (49). Our findings are consistent with prior research demonstrating that adults with a history of childhood maltreatment show increased fusiform gyrus activation to novel faces, suggesting increased detection sensitivity, dysregulated hypervigilance, and heightened salience detection (50). However, future research should continue to investigate the role of the nucleus accumbens in the context of threat and vigilance.

The current findings also highlight that greater childhood threat is related to increased connectivity between the left amygdala and the precuneus after covarying for experiences of deprivation. Such findings highlight that increased connectivity between the amygdala and the precuneus may reflect amygdala activity to emotional stimuli, especially when it involves attentional deployment or redirecting attention away from emotional stimuli (49). This interpretation is consistent with previous work demonstrating that the severity of childhood physical abuse is associated with an attentional bias away from threatening faces (50). Another study found that a bias toward happy faces partially mediated the relationship between experiences of childhood maltreatment and later PTSD symptoms (e.g., avoidance) (51).

The current findings are also consistent with previous resting-state studies implicating childhood emotional maltreatment and amygdala-precuneus connectivity, but with the right amygdala (24). The increased connectivity between the left amygdala and the precuneus that was observed in this study may reflect that adults who were maltreated in childhood are more conditioned to avoid, or turn attention away from, highly emotional arousal to cope and protect themselves. Research has reported mixed findings regarding the lateralization of amygdala connectivity as it relates to childhood maltreatment (30); for example, some research has posited that the right amygdala may be associated with positive picture encoding and anger, while the left amygdala is more involved in fear or threat processing (52). This is consistent with the current findings of increased connectivity between the left amygdala and the precuneus, which has historically been implicated in visual attention, inhibitory control, and episodic memory (53,54). In the context of the current findings, increased connectivity between the left amygdala and the precuneus may reflect enhanced attention to threat-relevant information. Moreover, because participants in the current study had recent trauma exposure, it may be that subsequent trauma interacts with how early-life experiences affect important threat circuits (55).

To understand the unique effect of childhood maltreatment (vs. cumulative traumatic experiences), we ran follow-up analyses controlling for baseline PTSD symptoms and lifetime traumatic exposure. Interestingly, only the right nucleus accumbens results remained significant. These results suggest that there may be something unique about neural reward circuitry as it relates to childhood threat compared with other traumatic experiences (26). In particular, it is possible that experiences of childhood threat have more long-lasting effects on reward circuitry versus threat circuitry (i.e., childhood threat may not affect the threat circuitry above and beyond the current or lifetime trauma). It is possible that due to experiencing recent trauma, threat circuitry is at a ceiling and wholly engaged by the recent threatening event as a salient one compared with the effect of childhood maltreatment. These differential findings (i.e., reward vs. threat circuitry outcomes) may help to inform our longer-term understanding of the unique role of childhood threat and approaches to screening experiences of childhood maltreatment, even during adulthood.

While threat was significantly related to connectivity patterns of reward and threat-related circuits, deprivation was not. These results provide evidence that the presence of harmful stimuli (e.g., threat) has differential impacts on neural circuits than the absence of positive stimuli (e.g., deprivation). Previous literature has found that experiences of threat and deprivation have opposing effects on different aspects of the brain (29). Current findings suggest that differential impacts are observed functionally as well. Research has posited that experiences of threat operate on the stress systems of the brain and impact release of glucocorticoids, whose receptors are found throughout reward- and threat-processing circuitry (56,57). Thus, because experiences of deprivation may not operate on the stress system the same way, it may explain the lack of significant findings. Similarly, childhood threat experiences have been related to changes in fear learning circuits, such as the amygdala, while deprivation may shape neural development via synaptic pruning (3,29). Thus, the lack of a significant relationship between experiences of deprivation and amygdala connectivity in this study can be explained by the fact that 1) mechanisms of impact of deprivation (e.g., pruning) cannot be accurately assessed via measures of functional connectivity and 2) the effects of deprivation are often observed on cognition, which is not necessarily dependent on or captured by reward and threat circuitry.

Limitations

The current study has several limitations. First, we used a retrospective self-report measure of childhood maltreatment, which is subject to participants' memory and willingness to disclose. Second, the current study was not designed to assess temporal features of childhood maltreatment, such as age of onset or duration of threat or deprivation, which are predictive of outcomes (30). Third, we aimed to control for previous trauma exposure to isolate the role of childhood maltreatment. We used the LEC (41) but excluded 4 items (items 6–9) that ask about experiences of physical or sexual assault because we do not know whether such experiences did in fact occur during childhood (e.g., the LEC asks whether such events have ever occurred to individuals and may be confounded with childhood threat). Fourth, given that the sample used in this study comprises adults who were recruited after experiencing a traumatic injury, the current study does not have a control group (i.e., nontrauma group). Finally, prior work has shown that childhood poverty levels may be associated with amygdala activity in adulthood (58). The current

study did not include a measure of childhood poverty and, as such, future work could build on the current study by investigating the relationship between childhood poverty and subtypes of childhood maltreatment.

Conclusions

The current study provided evidence for differential amygdala and nucleus accumbens connectivity patterns associated with childhood threat and deprivation. These connectivity patterns may be related to psychological processes such as salience, vigilance, or preparedness to detect visual stimuli. This extends the extant literature by providing additional evidence that distinguishes between child maltreatment subtypes rather than solely exploring the cumulative experience of early adversity. Moreover, findings suggest that early-life stress is related to disrupted connectivity in adults soon after they experience an additional traumatic event; clinically, these persistent and unique effects of childhood maltreatment emphasize the importance of early screening to help prevent chronic adverse outcomes. Finally, future research should further explore early-life stress in the context of trauma in adulthood in comparison to a nontrauma control group.

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