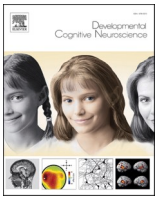
Developmental Cognitive Neuroscience 47 (2021) 100903



Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn





Social experience calibrates neural sensitivity to social feedback during adolescence: A functional connectivity approach

Karen D. Rudolph [a,*], Megan M. Davis [a], Haley V. Skymba [a], Haina H. Modi [a], Eva H. Telzer [b]

[a] University of Illinois at Urbana-Champaign, Department of Psychology, 603 E. Daniel St, Champaign, IL, 61820, USA
[b] University of North Carolina at Chapel Hill, Department of Psychology and Neuroscience, 235 E Cameron Ave, Chapel Hill, NC, 27599, USA

ARTICLE INFO

Keywords:
Family adversity
Peer adversity
Functional connectivity
Ventral striatum
Amygdala
Adolescence

ABSTRACT

The adaptive calibration model suggests exposure to highly stressful or highly supportive early environments sensitizes the brain to later environmental input. We examined whether family and peer experiences predict neural sensitivity to social cues in 85 adolescent girls who completed a social feedback task during a functional brain scan and an interview assessing adversity. Whole-brain functional connectivity (FC) analyses revealed curvilinear associations between social experiences and FC between the ventral striatum and regions involved in emotion valuation, social cognition, and salience detection (e.g., insula, MPFC, dACC, dlPFC) during social reward processing, such that stronger FC was found at both very high and very low levels of adversity. Moreover, exposure to adversity predicted stronger FC between the amygdala and regions involved in salience detection, social cognition, and emotional memory (e.g., sgACC, precuneus, lingual gyrus, parahippocampal gyrus) during social threat processing. Analyses also revealed some evidence for blunted FC (VS-PCC for reward; amygdala-parahippocampal gyrus for threat) at very high and low levels of adversity. Overall, results suggest social experiences may play a critical role in shaping neural sensitivity to social feedback during adolescence. Future work will need to elucidate the implications of these patterns of neural function for the development of psychopathology.

1. Introduction

Exposure to childhood adversity represents one of the most salient and pervasive risk factors for future mental and physical health difficulties, ranging from mood disorders and anxiety to substance use and disruptive behavior disorders to chronic diseases (Herzberg and Gunnar, 2020; McLaughlin, 2016; McLaughlin et al., 2019). Understanding the pathways through which adversity undermines development is therefore essential for creating effective prevention programs aimed at reducing health disparities among youth. Several related theories propose that early adversity sensitizes biological systems to environmental input, setting the stage for subsequent maladjustment (e.g., Juster et al., 2010; Shonkoff and Bales, 2011). Consistent with these theories, a growing body of research supports the idea that exposure to childhood adversity predicts individual differences in brain structure and function (for reviews, see Herzberg and Gunnar, 2020; McLaughlin et al., 2019), with implications for future health.

Building on these univalent perspectives, which focus on adversity, the adaptive calibration model (Ellis et al., 2017) posits that exposure to either highly stressful or highly supportive early environments can upregulate biological sensitivity, allowing youth to react to both environmental threats (within punitive environments) and supports (within rewarding environments). However, few investigations consider the possibility that neural sensitivity can emerge in the context of both high threat and high support (i.e., curvilinear associations between social experience and neural function) or examine differential effects of adversity in different domains (e.g., family and peer). Moreover, much of this research focuses on adversity-related neural processing of general emotions rather than specific social cues, which represent a particularly salient and impactful form of environmental input during adolescence (Somerville, 2013). The present study used functional magnetic resonance imaging (fMRI) to address these notable gaps through several innovations: (1) examining the impact of cumulative lifetime adversity on neural processing of social cues during adolescence; (2) considering neural processing of both social reward (belonging) and threat (non-belonging); (3) investigating both linear and curvilinear associations between adversity and neural function; and (4) comparing the effects of family and peer adversity. Consistent with a recent call to focus on

* Corresponding author.
E-mail address: krudolph@illinois.edu (K.D. Rudolph).

https://doi.org/10.1016/j.dcn.2020.100903
Received 10 April 2020; Received in revised form 27 July 2020; Accepted 7 December 2020
Available online 17 December 2020
1878-9293/© 2020 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

patterns of functional connectivity (FC) associated with early life stress (Herzberg and Gunnar, 2020), we conducted whole-brain FC analyses, using primary subcortical regions implicated in social reward processing (ventral striatum; VS; Galván, 2010; Telzer, 2016) and social threat processing (amygdala; Somerville et al., 2010) as seed regions.

1.1. Conceptualization of adversity

Contemporary conceptualizations (McLaughlin, 2016; McLaughlin and Sheridan, 2016; McLaughlin et al., 2019; Sheridan and McLaughlin, 2014) view childhood adversity as exposure to chronic or severe social experiences involving either significant threat of harm (e.g., abuse, victimization) or notable absence of expected positive environmental input (e.g., sensitive parenting, supportive friendships). In the present study, we incorporated both threat and deprivation aspects of adversity as well as the Research Domain Criteria construct of loss (Cuthbert and Insel, 2013) into indexes of cumulative lifetime adversity. Specifically, adversity was conceptualized as exposure to chronic or severe threats within the family (e.g., violence, conflict) and peer group (e.g., bullying, social rejection[1]) as well as absence of expected supports within the family (e.g., parental separation/absence) and peer group (e.g., isolation, friendlessness) and experience of significant losses (e.g., end of relationship; death of family member or friend). Conversely, favorable childhood experiences were viewed as involving the absence of notable threat or loss and the presence of support (e.g., close relationships with family or peers).

1.2. Social sensitivity during adolescence

During adolescence, a process of "social reorientation" (Nelson et al., 2016) occurs as adolescents gradually shift their focus from the family to the peer group (Brown, 2013). This reorientation is characterized by intensified emotional salience of peers and sensitivity to peer evaluation (Steinberg and Morris, 2001) along with a desire to conform to peer group norms (Knoll et al., 2015; Spear, 2009). Compared to other age groups, adolescents report more mood variability following social acceptance or rejection (Guyer et al., 2014), are more willing to take risks to gain social rewards (Gardner and Steinberg, 2005), and are more cognitively distracted by images indicating social reward or threat (Perino et al., 2016; Rogers et al., 2019; Somerville et al., 2010; Somerville, 2013). Paralleling this behavioral sensitivity to social cues, adolescents show increasing neural sensitivity to social feedback. In the context of reward processing, adolescents show greater activation than younger children and adults in the VS (Ernst et al., 2005; Galván et al., 2006; Van Leijenhorst et al., 2010). In the context of threat processing, adolescents show greater activation than younger children and adults in the amygdala (Hare et al., 2008). Both behavioral (Guyer et al., 2014) and neural (Guyer et al., 2009; Kumar et al., 2019) attunement to social cues may be particularly strong in adolescent girls relative to boys. Thus, the present study focused on whether childhood social experiences predict neural processing of social cues in adolescent girls.

[1] Whereas the traditional conceptualization of threat in the context of adversity focuses on threats to one's physical integrity (McLaughlin and Sheridan, 2016), the present study also incorporated threats to the integrity of one's social relationships. This conceptualization is in line with theory and research suggesting that threats to one's need to belong may have significant negative implications for physical and psychological well-being (Baumeister and Leary, 1995). Thus, in the context of lifetime adversity, social threat included physical and non-physical (e.g., rumor spreading, exclusion, relational manipulation) forms of victimization as well as conflict and explicit social rejection. In the context of the Social Feedback Task, social threat reflected negative feedback suggesting non-belonging in one's peer group.

1.3. Impact of social experience on neural sensitivity during adolescence

Although adolescence is characterized by a general shift toward increasing social sensitivity, developmental programming models (O'Connor, 2003) propose that early social experiences foster individual differences in emerging biological systems in ways that optimize adaptation to current environmental conditions. According to allostatic load (Juster et al., 2010) and toxic stress (Shonkoff and Bales, 2011) models, exposure to chronic or severe adverse conditions during childhood may sensitize youth to potential threat, resulting in long-term and potentially permanent "wear and tear" on biological systems, as reflected in either heightened or blunted stress responsivity. Expanding these univalent theories, the adaptive calibration model (Ellis et al., 2017) posits that both especially stressful and especially supportive childhood environments calibrate biological sensitivity, allowing youth to respond flexibly to both environmental dangers and resources. Thus, extreme environments (adverse or favorable) would result in heightened sensitivity whereas moderate environments would result in less sensitivity (except in the case of severely stressful environments, which may result in long-term blunted responsivity).

Consistent with these developmental programming models, emerging theory and research consider neurodevelopmental pathways through which early adversity influences adjustment (for reviews, see Herzberg and Gunnar, 2020; McLaughlin et al., 2019). According to this perspective, early experiences can shape emerging brain structure and calibrate brain function in ways that alter encoding of environmental cues, either amplifying or dampening the salience of contextual information. Most empirical research examining the effects of early adversity on brain function examines patterns of brain activation during emotion processing (e.g., passive viewing or regulation of emotions), particularly processing of threat cues (e.g., negative emotions or images), with a smaller body of research examining the effects of adversity on reward processing (e.g., positive emotions, monetary or social rewards).

1.3.1. Reward processing

Given the central role of the VS in processing reward cues (Galván, 2010; Telzer, 2016), research on reward processing often focuses on VS activation or connectivity between the VS and prefrontal, salience detection, or social cognition regions (Herzberg and Gunnar, 2020; McLaughlin et al., 2019). Research examining striatal activation following family adversity reveals heightened activation in youth exposed to threat (Dennison et al., 2016) but blunted activation in youth exposed to deprivation (Goff et al., 2013; Mehta et al., 2010). However, one study linked low parental warmth (a form of emotional deprivation) with heightened striatal response to anticipated reward (Casement et al., 2014). Some studies examining resting-state connectivity and FC during reward processing reveal more positive connectivity between the VS and prefrontal regions (e.g., MPFC, Fareri et al., 2017; Hanson et al., 2018; lateral PFC, Marshall et al., 2018), as well as higher connectivity within the salience network (Marusak et al., 2015) in family adversity-exposed relative to non-exposed individuals. However, another study linked maternal hostility to more negative VS-prefrontal connectivity (Kopala-Sibley et al., 2020). With regard to peer adversity, one study linked past victimization with lower MPFC response to non-social reward anticipation in adolescence (Casement et al., 2014), and two studies using event-related potentials linked peer victimization with blunted neural response to monetary (Ethridge et al., 2018) and social (but not monetary; Rappaport et al., 2019) rewards in young adults. In contrast, studies examining neural activation during risk taking (often associated with reward sensitivity) revealed that peer adversity was associated with more activation in the striatum, insula, amygdala, and orbitofrontal cortex in adolescents (Telzer et al., 2015, 2018).

1.3.2. Threat processing

Given the central role of the amygdala in processing threat cues (Somerville et al., 2010), research on threat processing often focuses on

amygdala activation or connectivity between the amygdala and prefrontal, salience detection, or social cognition regions (for reviews, see Herzberg and Gunnar, 2020; McLaughlin et al., 2019). Despite some exceptions, this research typically reveals elevated amygdala activation to threat in youth exposed to family adversity (e.g., Herringa et al., 2016; McCrory et al., 2011, 2013; McLaughlin et al., 2015), especially overt threat (McLaughlin et al., 2019). Some studies examining threat processing link family adversity with stronger positive FC between the amygdala and other regions (e.g., MPFC, Herringa et al., 2016; hippocampus, parahippocampal gyrus, Jedd et al., 2015), whereas other studies link family adversity with alternate patterns of FC (e.g., MPFC, Javanbakht et al., 2015; insula, operculum, MPFC, Kopala-Sibley et al., 2020). Studies of resting state connectivity also reveal differing patterns of stronger positive (e.g., insula, subgenual anterior cingulate cortex [sgACC]/ventromedial PFC, Thomason et al., 2015; rostral ACC, Kaiser et al., 2018; dorsolateral PFC [dlPFC], Herringa et al., 2013), weaker negative (ACC, Pagliaccio et al., 2015), and stronger negative (e.g., dlPFC, Kaiser et al., 2018; sgACC, postcentral gyrus, Herringa et al., 2013) FC with the amygdala. With regard to peer adversity, studies consistently link past threat and deprivation with elevated activation in regions involved in threat and salience processing (e.g., amygdala, dACC, sgACC, IFG; Jarcho et al., 2019; Lee et al., 2014; Masten et al., 2010; McIver et al., 2019; Rudolph et al., 2016; Schriber et al., 2018; Will et al., 2016), as well as memory (e.g., parahippocampal gyrus; McIver et al., 2018) and cognitive control (e.g., PFC; Lee et al., 2014; Will et al., 2016) in response to social threat.

1.3.3. Summary

Overall, research suggests that exposure to family and peer adversity calibrates neural systems involved in processing of both reward (e.g., VS and its connections) and threat (e.g., amygdala and its connections). Whereas some research suggests amplified adversity-linked sensitivity, other research suggests blunted adversity-linked sensitivity. Discrepant findings across studies may be due, in part, to a focus on examining only linear associations or comparing two extreme groups, without taking into account the full spectrum of social experiences or investigating whether exposure to both stressful and favorable environments shapes neural sensitivity to the environment, consistent with the adaptive calibration model.

1.4. Study overview

This study investigated the association between cumulative lifetime family and peer adversity and neural processing of social reward and threat. Given evidence for increasing neural sensitivity to reward and threat during adolescence (Galván et al., 2006; Hare et al., 2008), particularly in girls (Guyer et al., 2009; Kumar et al., 2019), we focused on adolescent girls. Specifically, we examined whether exposure to particularly high stress, unfavorable social environments and, potentially, particularly low stress, favorable social environments heightens adolescent girls' sensitivity to positive and negative social cues, leading to stronger co-activation between brain regions involved in processing reward and threat cues and those involved in salience detection and social cognition. To test these ideas, we examined linear and curvilinear associations between adversity and patterns of neural connectivity. Consistent with prior research (e.g., Kopala-Sibley et al., 2020), we conducted whole-brain FC analyses, focusing on two seed regions implicated in processing of reward (VS) and threat (amygdala). We assessed social experiences using an interview that yielded ratings of cumulative lifetime adversity, allowing us to assess favorable and unfavorable family and peer environments along a continuum.

Neural processing of social feedback was assessed using a novel task tapping activation to social reward (belonging) and threat (non-belonging). This task was inspired by previous tasks assessing neural activation to social acceptance and rejection feedback (e.g., Guyer et al., 2009; Gunther Moor et al., 2010; Jarcho et al., 2019; Silk et al., 2014) but differs from these tasks in several ways. First, relative to some tasks in which peer judgments are based on superficial characteristics (e.g., a picture of the participant), in this task peer judgments are based on more detailed information about preferences in peer-relevant domains. Second, relative to some tasks that require more complicated set-up and deception, this task requires only a single session with a modest deception. Third, our task includes a conservative neutral condition, allowing us to separately compare positive and negative feedback to neutral feedback.

2. Methods

2.1. Participants and procedures

Participants in the present study included 85 adolescent girls (M_{age} = 16.30, SD = .84, range = 14.85–17.73) who completed sessions during the summer following 9th, 10th, or 11th grade. Participants were from diverse ethnic (67.1 % White, 21.2 % African American, 3.5 % Latina/Hispanic, 1.2 % Asian, 7.1 % multiracial) and socioeconomic (family income: $0−14,999 [10.6 %], $15−29,999 [15.3 %], $30−44,999 [20 %], $45−59,999 [5.9 %], $60−74,999 [8.2 %], $75−89,999 [7.1 %], more than $90,000 [31.8 %]) backgrounds. Of the 207 girls contacted for the study, 43 were excluded (due to having braces, being claustrophobic, or having a learning disorder that would compromise their ability to complete tasks), 44 declined to participate, and 30 were contacted but not scheduled before the target sample size of 90 was reached. Four of the 90 girls were excluded from present analyses due to excessive movement during the scan or issues with fMRI data collection, and one was excluded because she did not complete the lifetime adversity interview.

Girls completed a novel social feedback task while undergoing fMRI. Within three weeks, they completed the lifetime adversity section of the Youth Life Stress Interview (Rudolph and Flynn, 2007). Trained graduate and senior undergraduate students and post-baccalaureate research staff completed and coded the lifetime adversity interviews. Participants were compensated $50 for completing the fMRI session and $25 for completing the interview. Parents provided written consent and youth provided written assent prior to participating. All study procedures were conducted in accordance with ethical guidelines of the American Psychological Association and approved by the University of Illinois Institutional Review Board.

2.2. Measures

2.2.1. Social feedback task

Prior to the scan, girls were told that they would be selecting their preferences in different domains from two options and would then receive feedback about whether other teens agreed or disagreed with them based on hundreds of teens who had previously completed the task. During the scan, girls completed the task, which included a single run of 60 trials with three phases. First, girls were presented with two choices (e.g., rap vs. rock) within a variety of domains relevant to adolescents (e.g., music), and indicated their preference via a button press (decision); this phase lasted five seconds or until participants made a selection. Second, a fixation cross appeared on the screen during a 1365−2730 millisecond (ms) jitter, followed by a screen indicating the task was retrieving peer feedback (anticipation), which remained on the screen for 2 s. Third, after a 1365−2730 ms jitter, girls received feedback indicating whether other teens who had ostensibly completed the task mostly agreed (thumbs up; positive feedback, indicative of belonging) mostly disagreed (thumbs down; negative feedback, indicative of non-belonging), or roughly half agreed/half disagreed (thumb to the side; neutral feedback) with their selection (feedback). In reality, the feedback was randomly generated such that girls received 20 trials of each type (see Fig. 1). Feedback remained on the screen for 2 s, followed by a 1365–3640 ms inter-trial interval. The present analyses focused on

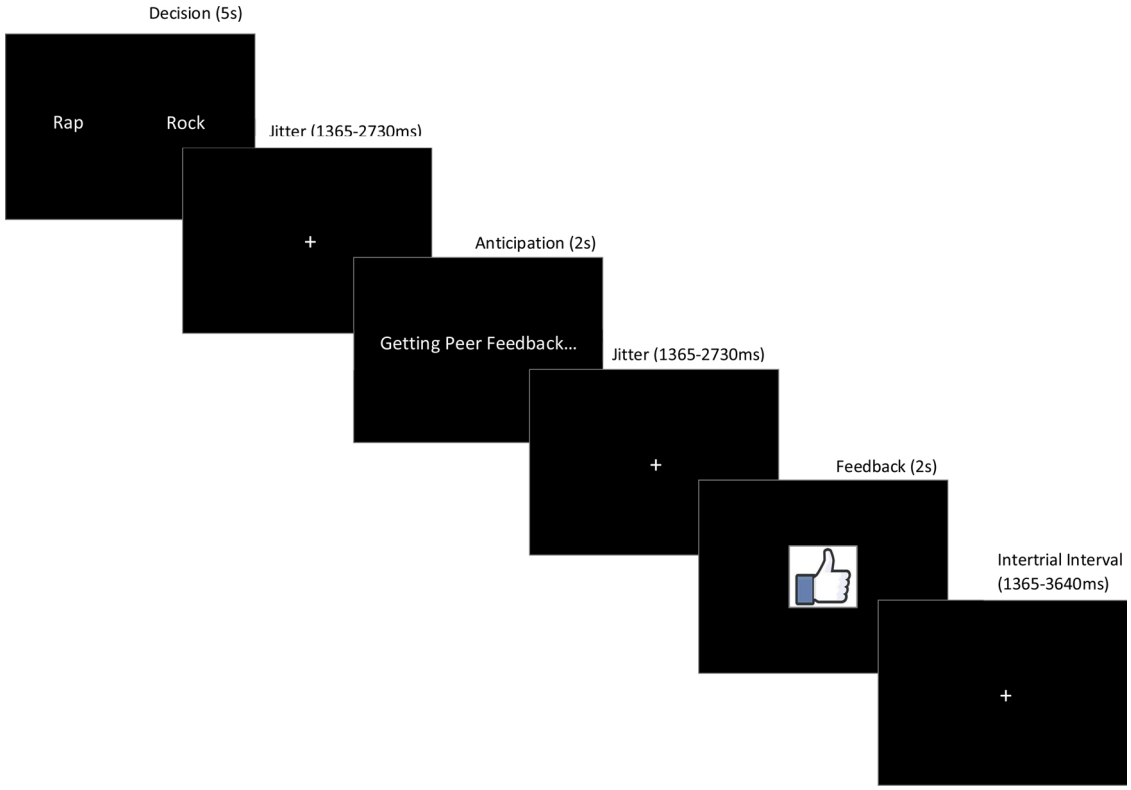


Fig. 1. In the Social Feedback Task, participants selected their preference from two options in several domains and waited for feedback from peers. One third of trials provided positive feedback (thumbs up), one third provided negative feedback (thumbs down), and one third provided neutral feedback (thumb to the side).

the feedback stage.

2.2.2. Cumulative lifetime adversity

Lifetime adversity was assessed using a section of the Youth Life Stress Interview (YLSI; Rudolph and Flynn, 2007) that assesses exposure to severe adverse events and circumstances across the lifetime using the contextual threat method (Brown and Harris, 1978). Interviewers first used a general probe to identify exposure to particularly impactful stresssors and then assessed exposure to specific adversities in the family (e.g., extended separation from a parent; parental separation or divorce; death of a close family member; family conflict; severe mental illness; financial insecurity) and peer group (e.g., social rejection, exclusion, or victimization; friendlessness; death of a friend; severe peer or romantic partner conflict). Follow-up questions ascertained the context, duration, and consequences of adverse events and circumstances. An independent coding team provided a consensual rating on a 10-point scale to reflect the overall level of adversity experienced, considering the likely impact of the events and circumstances for a typical child in the same circumstances. Separate ratings were given for cumulative lifetime family adversity ($M = 3.85$, $SD = 2.05$, range = 1–9) and peer adversity ($M = 2.95$, $SD = 1.99$, range = 1–9). To assess reliability, two coding teams independently rated 25 % of interviews. Strong reliability (was found for ratings of family (intraclass correlation coefficient; ICC = .98) and peer (ICC = .99) adversity.

2.3. Data acquisition and analysis

2.3.1. fMRI data acquisition

fMRI data were collected using a 3 T Siemens Trio MRI scanner. Structural scans consisted of a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR = 1.9 s; TE = 2.3 ms; matrix = 256 × 256; FOV = 230; sagittal plane; slice thickness = 1 mm; 192 slices) and a T2*weighted, matched-bandwidth (MBW), high-resolution, anatomical scan (TR = 4 s; TE = 64msec; matrix = 192 × 192; FOV = 230; slice thickness = 3 mm; 38 slices). The task included T2*-weighted echoplanar images (EPI) [slice thickness = 3 mm; 38 slices; TR = 2 s; TE = 25msec; matrix = 92 × 92; FOV = 230 mm; voxel size 2.5 × 2.5 × 3mm^3].

2.3.2. Data analysis

Neural data were preprocessed using statistical parametric mapping (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK). Images were spatially realigned to correct for head movement. Functional data were coregistered to the structural MPRAGE, which was then segmented into cerebrospinal fluid, gray matter, and white matter. Structural and functional images were transformed into standardized stereotactic space as defined by the Montreal Neurological Institute. Normalized functional data were smoothed using an 8 mm Guassian kernel, full-width-at-half-maximum, to increase signal-to-noise ratio. High-pass temporal filtering with a cutoff of 128 s was applied to remove low-frequency drift in the data. For each participant's data, a general linear model (GLM) was created using regressors that corresponded to the onset and duration of each trial for each of the conditions (decision, anticipation, and positive/negative/neutral feedback) as well as an unweighted regressor for trials with excessive motion (over 2.5 mm overall movement). Trials with no response and inter-trial intervals were not explicitly modeled and were therefore included in the implicit baseline. Separate regressors were modeled for each feedback type (i.e., positive, negative, and neutral) to allow us to compare feedback based on valence. We considered two primary contrasts of interest: positive > neutral feedback and negative > neutral feedback.

Because we were interested in the extent to which social experience predicts variability in co-activation across regions involved in sensitivity to reward and threat, we conducted psychophysiological interactions (PPI) using the VS and amygdala, respectively, as seed regions. The VS and amygdala ROIs were created by combining the left and right anatomical regions. The gPPI toolbox in SPM 8 (McLaren et al., 2012)

was used to (1) extract the time series from each ROI; (2) convolve each trial type with the HRF; and (3) multiply the physiological and psychological variables to create the PPI term. Random effects group-level analyses were performed on all individual subject contrasts generated from gPPI. We conducted separate whole-brain regressions using family and peer adversity, adjusting for the alternate domain, as predictors of (1) VS FC during positive > neutral feedback, and (2) amygdala FC during negative > neutral feedback. In the first set of models, we entered the linear term for family and peer adversity. In the second set of models, to explore curvilinear associations, we computed the square of family and peer adversity and entered these quadratic terms, adjusting for the linear effects. Monte Carlo simulations using the 3dClustSim tool in AFNI (Ward, 2000) were used to determine the minimum cluster size necessary in each contrast for a voxel-wise threshold of $p < .005$ and a family-wise error rate of $p = .05$. For descriptive purposes, we extracted neural connectivity from each surviving cluster using MarsBaR (Brett et al., 2002) to examine the pattern of association with adversity.

3. Results

3.1. Positive > neutral feedback: ventral striatum seed

For positive feedback (Table 1), we focused on FC with the VS as the seed region given its role in reward detection, including social reward (Galván, 2010; Telzer, 2016).[2]

3.1.1. Family adversity

There was no significant linear association between family adversity and FC when receiving positive > neutral feedback. However, results indicated significant quadratic associations between family adversity and FC between VS-left insula, VS-right dlPFC, and VS-MPFC when receiving positive > neutral feedback. For descriptive purposes, we extracted parameter estimates of FC and plotted them against family adversity. As shown in Fig. 2, the pattern of associations reflected a U-shaped curve, such that girls experiencing relatively low or relatively high levels of family adversity (compared to those experiencing moderate levels) showed strengthened FC between the VS and these brain regions when receiving positive > neutral feedback.

Table 1
Regions Showing Significant Functional Connectivity with the Ventral Striatum in the Positive Feedback > Neutral Feedback Condition.

Region label	k	t	x	y	z
Family Adversity-Quadratic					
Left insula	83	3.57	−39	−4	−11
Right dlPFC	59	3.48	45	−1	28
Right MPFC	84	3.31	6	56	1
Peer Adversity-Linear					
Left intraparietal lobule	73	3.97	−27	−67	34
Right PCC	62	3.57	3	−37	40
Peer Adversity-Quadratic					
Right dlPFC	57	4.64	42	−1	31
Left dACC/thalamus	124	4.49	−18	−1	28
Left inferior occipital gyrus	65	3.41	−33	−88	1
Left PCC	255	−4.22	−18	−46	43

Note. k refers to the number of voxels in each cluster; t refers to the peak activation in each cluster; x, y, and z refer to MNI coordinates. dlPFC = dorsolateral prefrontal cortex. MPFC = medial prefrontal cortex. PCC = posterior cingulate cortex. dACC = dorsal anterior cingulate cortex.

3.1.2. Peer adversity

Results indicated significant positive linear associations between peer adversity and FC between the VS-left intraparietal lobule and VS-right posterior cingulate cortex (PCC) when receiving positive > neutral feedback. Results also indicated significant quadratic associations between peer adversity and FC between VS-right dlPFC, VS-left dACC/thalamus, VS-left inferior occipital gyrus, and VS-bilateral PCC when receiving positive > neutral feedback. For descriptive purposes, we extracted parameter estimates of FC and plotted them against peer adversity. As shown in Fig. 3, the pattern of associations for FC between VS-right dlPFC, VS-left dACC/thalamus, and VS-left inferior occipital gyrus reflected a U-shaped curve, such that girls experiencing relatively low or relatively high levels of peer adversity (compared to those experiencing moderate levels) showed strengthened FC between VS and these brain regions when receiving positive > neutral feedback. However, the pattern of association for FC between VS-bilateral PCC reflected an inverted U-shaped curve (Fig. 3), such that girls experiencing relatively low or relatively high levels of peer adversity (compared to those experiencing moderate levels) showed blunted FC between VS and PCC when receiving positive > neutral feedback.

3.2. Negative > neutral feedback: amygdala seed

For negative feedback (Table 2), we focused on FC with the amygdala given its role in threat detection, including social threat (Somerville et al., 2010).

3.2.1. Family adversity

Results indicated significant positive linear associations between family adversity and FC between amygdala-right sgACC, amygdala-left lingual gyrus, amygdala-bilateral cerebellum, and amygdala-left precuneus when receiving negative > neutral feedback (Fig. 4). Results also indicated a significant quadratic association between family adversity and FC between amygdala-left parahippocampal gyrus when receiving negative > neutral feedback. For descriptive purposes, we extracted parameter estimates of FC and plotted them against family adversity. As shown in Fig. 4, the pattern of association for FC between amygdala-left parahippocampal gyrus reflected an inverted U-shaped curve, such that girls experiencing relatively low or relatively high levels of family adversity (compared to those experiencing moderate levels) showed blunted FC between the amygdala and the left parahippocampal gyrus when receiving negative > neutral feedback.

3.2.2. Peer adversity

Results indicated positive linear associations between peer adversity and FC amygdala-right parahippocampal gyrus and amygdala-right postcentral gyrus when receiving negative > neutral feedback (Fig. 5). There were no significant quadratic effects of peer adversity on FC for negative > neutral feedback.

4. Discussion

Given the pervasive effects of childhood adversity on short- and long-term adaptation, substantial research efforts focus on elucidating the mechanisms through which adversity shapes development. Recently, this research has begun to map neurodevelopmental pathways underlying adversity-related outcomes (for reviews, see Herzberg and Gunnar, 2020; McLaughlin et al., 2019). Building on this research, this study drew from developmental programming models to investigate whether exposure to highly stressful as well as highly favorable environments predicts neural attunement to social cues. Overall, results support the

[2] Because research suggests that social reward processing may differ across age and level of pubertal development (e.g., Forbes et al., 2010), we also conducted these analyses adjusting for age and pubertal status. When adjusting for age, all of the reported effects remained significant. There was also a significant quadratic association between peer adversity and FC between VS-left caudate when receiving positive > neutral feedback. When adjusting for pubertal status, all of the reported effects remained significant with the exception of the linear effect for the left intraparietal lobule and the quadratic effect for the left inferior occipital gyrus.

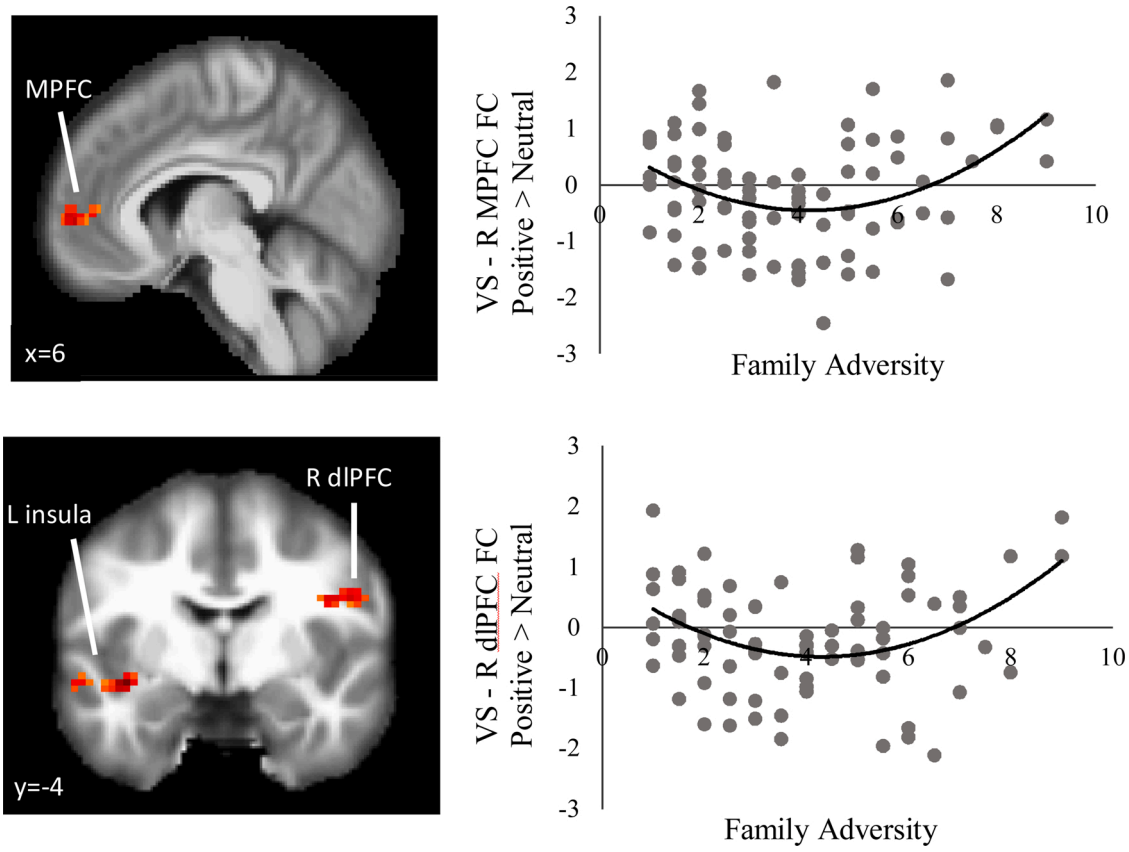


Fig. 2. Family adversity showed curvilinear associations with functional connectivity between the striatum and the medial prefrontal cortex (MPFC), insula, and right dorsolateral prefrontal cortex (dlPFC) in the Positive Feedback > Neutral Feedback condition. For descriptive purposes, parameter estimates of signal intensity were extracted and plotted against family adversity. The MPFC and right dlPFC are plotted as examples.

idea that social experiences calibrate neural processing of both social reward and social threat in adolescent girls. Whereas some results revealed a linear association between adversity and neural sensitivity to social cues, the emergence of some curvilinear associations supports the idea that environments characterized by both highly favorable and highly unfavorable conditions can shape neural function. Elucidating the origins of individual differences in neural organization may have significant implications for understanding its potential developmental consequences.

4.1. Social experiences and intensified neural processing of social cues

We examined the association between social experiences throughout childhood and neural sensitivity to social reward and threat as reflected in whole-brain FC between the VS and amygdala, respectively, and other brain regions while adolescent girls received feedback suggesting that their social preferences were consistent or inconsistent with peer group norms. During adolescence, "fitting in" with peers becomes of the utmost importance (Do et al., 2020; Steinberg and Monahan, 2007), satisfying the need to belong to a salient social group (Baumeister and Leary, 1995). According to the Belonging Regulation Model (Gardner et al., 2005), this need to belong operates through an innate social monitoring system (SMS) that regulates belonging needs. When these needs are challenged, the SMS triggers intensified monitoring of social cues signaling both social inclusion and exclusion. However, highly supportive environments also may increase sensitivity to environmental input (Ellis et al., 2017), perhaps resulting in heightened salience of social cues in both particularly unfavorable and particularly favorable environments.

4.1.1. Social reward processing

Prior studies suggest that social reward activates not only the VS (Achterberg et al., 2016; Casey et al., 2008; Galván, 2010; Guyer et al., 2011; Liu et al., 2011), but also regions involved in salience detection, social cognition, and self-referential processing (e.g., insula, dACC, MPFC; Achterberg et al., 2016, 2018; Guyer et al., 2011). Moreover, research links adversity to activation and co-activation in these regions (Casement et al., 2014; Dennison et al., 2016; Fareri et al., 2017; Hanson et al., 2018; Marshall et al., 2018; Marusak et al., 2015). Our analyses revealed that exposure to very high, but also very low, levels of adversity predicted stronger FC between the VS and the insula (family), MPFC (family), dACC (peer), and dlPFC (family and peer). Thus, our findings suggest that exposure to both stressful and favorable environments may tune the brain towards heightened processing of positive social feedback via stronger FC between regions involved in reward processing, emotion valuation, social cognition, and salience detection.

It is possible, however, that the underlying meaning of this elevated sensitivity to social reward, and thus its developmental implications, differ depending on youths' childhood social experiences. Adolescent girls from supportive families and peer groups may learn to tune into positive cues in their environment in order to benefit from the resources provided by strong relationships. In these girls, belongingness cues likely signal anticipated support and nurturance from others. According to the adaptive calibration model (Ellis et al., 2017), this "sensitive" profile develops in safe, predictable, and supportive environments, allowing for flexible attention and responses to social feedback. In contrast, heightened attunement to social reward in adolescent girls exposed to high levels of adversity may stem from "social hunger" (Gardner et al., 2005), driving them to attend to cues that satisfy their need to belong to a social group. That is, adolescent girls whose relationships have been compromised through threat, loss, or deprivation

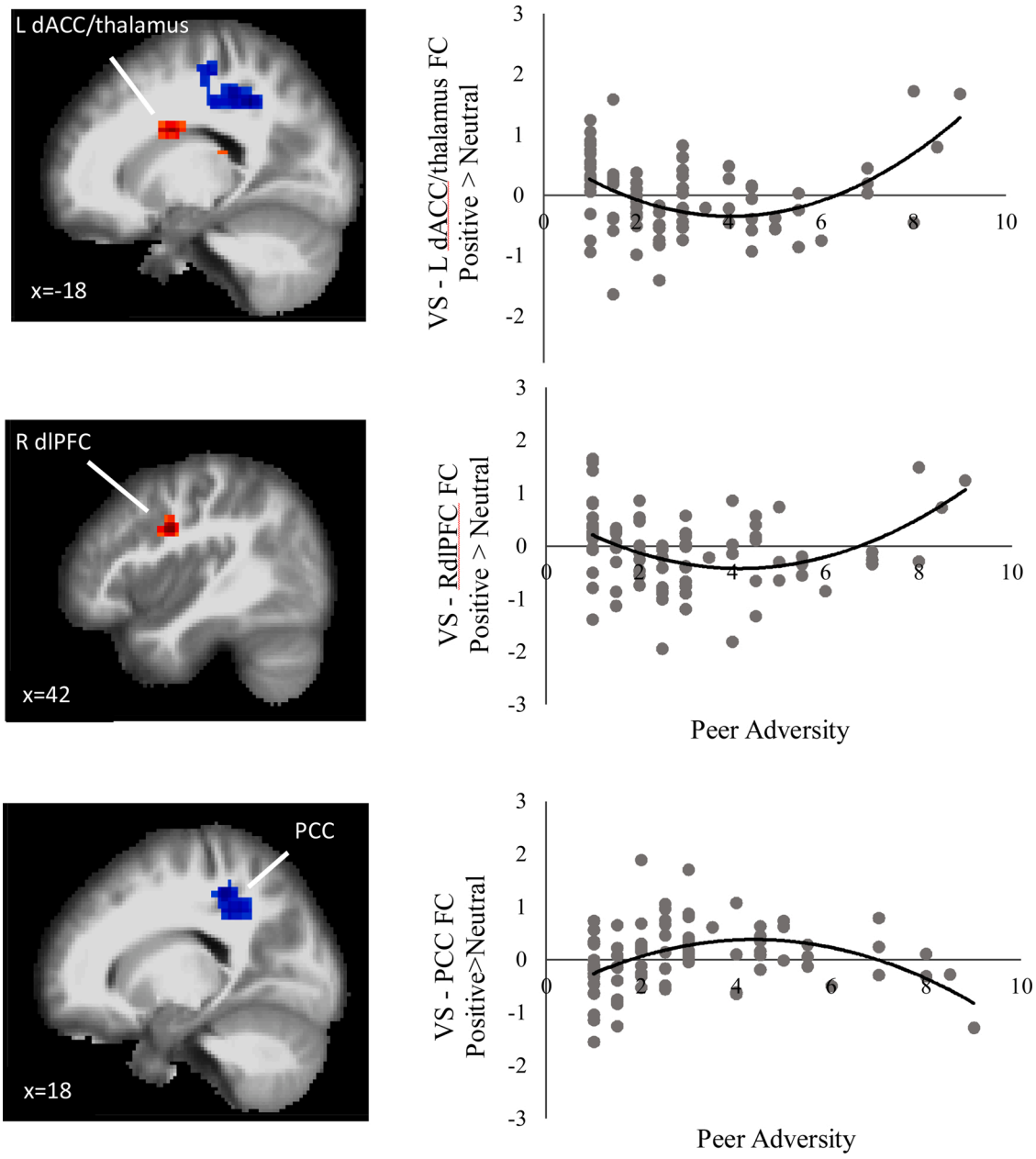


Fig. 3. Peer adversity showed curvilinear associations with functional connectivity between the striatum and the dorsolateral anterior cingulate cortex (dACC), right dorsolateral prefrontal cortex (dlPFC), and posterior cingulate cortex (PCC) in the Positive Feedback > Neutral Feedback condition. For descriptive purposes, parameter estimates of signal intensity were extracted and plotted against peer adversity.

may show elevated social monitoring (Gardner et al., 2005), as reflected in intensified neural processing of social cues indicative of peer affiliation and inclusion (Telzer et al., 2018, 2019). Heightened sensitivity to social belonging cues may cause these girls to show excessive encoding and monitoring of social situations and, potentially, set the stage for maladaptive developmental outcomes associated with overreliance on peer evaluation and approval. Given these potential differences in the developmental origins and function of heightened attunement to social reward in adolescents exposed to favorable versus unfavorable childhood environments, future research will need to determine whether the consequences of this pattern of neural sensitivity differ depending on girls' social history.

4.1.2. Social threat processing

Prior studies suggest that social threat activates not only the amygdala (e.g., Achterberg et al., 2017) but also regions involved in salience detection (e.g., sgACC; Achterberg et al., 2017; Bolling et al., 2011; Masten et al., 2009), mentalizing and self-referential processing (e.g., precuneus; Vijayakumar et al., 2017), and memory (e.g., parahippocampal gyrus; Bolling et al., 2011). Moreover, research links adversity to activation and co-activation in these regions (Herringa et al., 2016; Jarcho et al., 2019; Jedd et al., 2015; Lee et al., 2014; McIver et al., 2018; McLaughlin et al., 2015; Rudolph et al., 2016; Schriber et al., 2018; Thomason et al., 2015). Consistent with some prior research, analyses revealed that exposure to higher family adversity predicted stronger FC between the amygdala and the sgACC, precuneus, lingual gyrus, and cerebellum, whereas exposure to higher peer adversity predicted stronger FC between the amygdala and the parahippocampal gyrus and postcentral gyrus. Thus, our findings suggest that exposure to stressful environments may tune the brain towards heightened processing of negative social feedback via stronger FC between regions involved in threat and salience detection as well as social

Table 2
Regions Showing Significant Functional Connectivity with the Amygdala in the Negative Feedback > Neutral Feedback Condition.

Region Label	k	t	x	y	z
Family Adversity-Linear					
Right subgenual ACC	179	4.34	9	17	−11
Left lingual gyrus	122	4.26	−21	−73	1
Left cerebellum	204	3.86	−18	−64	−32
Right cerebellum	91	3.85	15	−61	−38
Left precuneus	194	3.74	−21	−85	40
Family Adversity-Quadratic					
Left parahippocampal gyrus	103	−3.61	−27	−34	−26
Peer Adversity-Linear					
Right parahippocampal gyrus	90	4.04	30	−43	−2
Right postcentral gyrus	122	3.50	48	−10	28

Note. k refers to the number of voxels in each cluster; t refers to the peak activation in each cluster; x, y, and z refer to MNI coordinates. ACC = anterior cingulate cortex.

cognition (e.g., understanding peers' perspectives) and emotional memory.

The amygdala, precuneus, and lingual gyrus have been implicated as part of a neural social valuation network, which integrates peer feedback in real-time to inform decision making (Kumar et al., 2019), Thus, stronger amygdala-precuneus and amygdala-lingual gyrus FC may suggest that social signals of non-belonging in the peer group carry more weight for adolescent girls exposed to family adversity and, potentially, may influence future choices such as whether to act in ways that would facilitate re-entry into the peer group. Likewise, stronger amygdala-sgACC FC in girls exposed to family adversity may reflect heightened emotion (perhaps especially sadness; Arias et al., 2020) processing in response to negative feedback. Research suggests that emotional memory consolidation may be contingent on coactivation of the amygdala and parahippocampal gyrus (Dolcos et al., 2004). Stronger amygdala-parahippocampal gyrus FC in adolescent girls exposed to high levels of peer adversity may therefore suggest that feedback indicating a lack of belonging in the peer group triggers prior emotionally salient

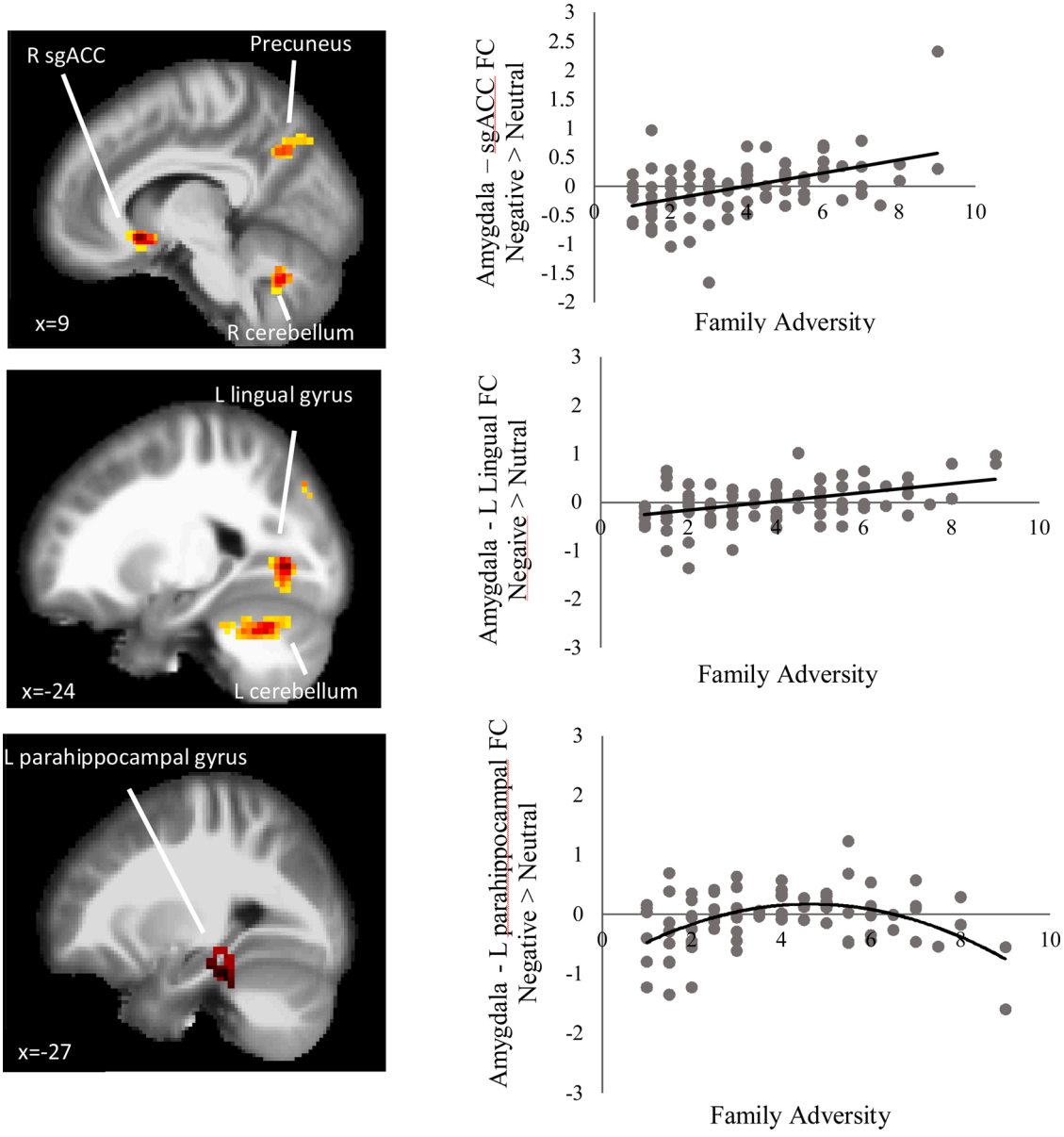


Fig. 4. Family adversity showed linear associations with functional connectivity between the amygdala and the subgenual anterior cingulate cortex (sgACC), precuneus, cerebellum, lingual gyrus, and parahippocampal gyrus in the Negative Feedback > Neutral Feedback condition. For descriptive purposes, parameter estimates of signal intensity were extracted and plotted against family adversity. The sgACC, lingual gyrus, and parahippocampal gyrus are plotted as examples.

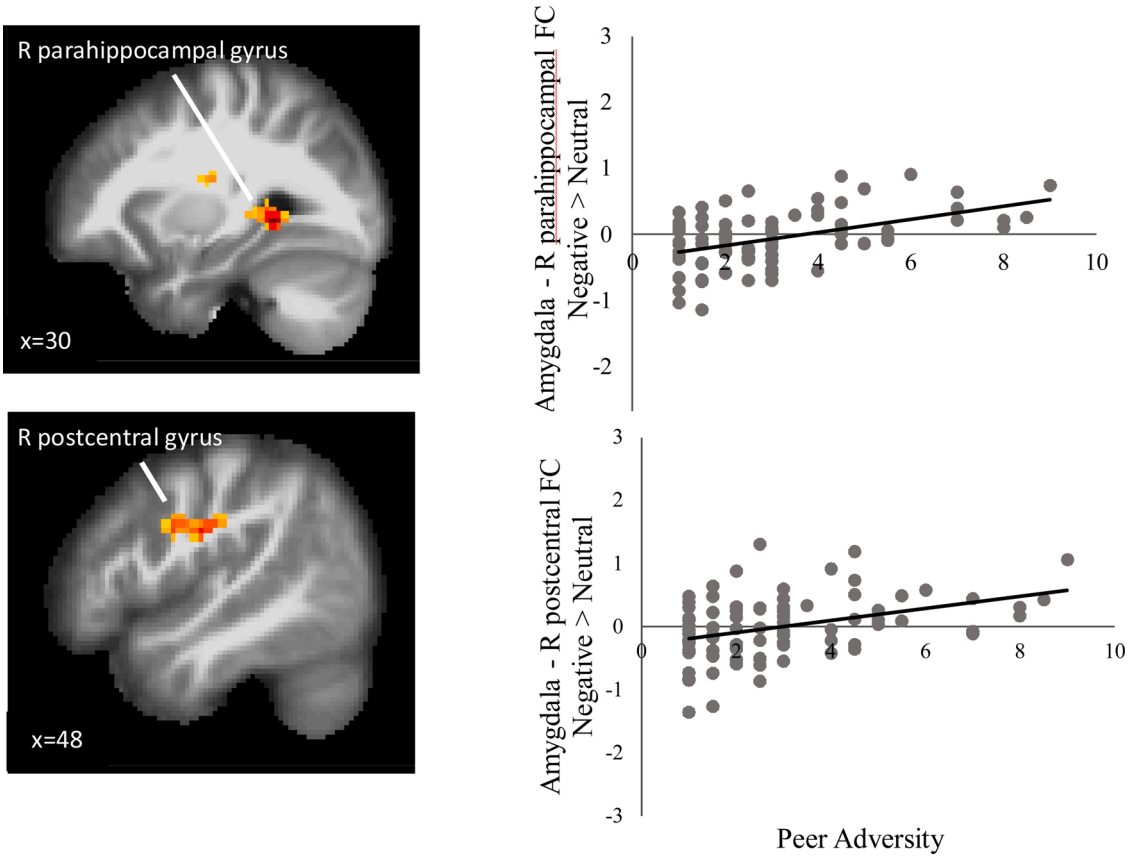


Fig. 5. Peer adversity showed linear associations with functional connectivity between the amygdala and the parahippocampal gyrus and postcentral gyrus in the Negative Feedback > Neutral Feedback condition. For descriptive purposes, parameter estimates of signal intensity were extracted and plotted against peer adversity.

memories about similar experiences. Of note, one previous study documented heightened activation in the cerebellum in response to social threat (Bolling et al., 2011), and there is some evidence for differences in the structure and function of this region in children exposed to adversity (McLaughlin et al., 2019). Thus, future studies examining the role of the cerebellum in processing of social threat following adversity may be fruitful.

Overall, our findings support the presence of a "vigilant" profile, which develops in stressful environments and promotes attention to threatening social cues (Ellis et al., 2017), presumably as an adaptation to growing up in dangerous or unpredictable contexts. Negative social feedback may assume more motivational significance and prompt deeper processing or even spark memories of prior punitive experiences in adolescent girls exposed to high levels of childhood adversity. Contrary to patterns of FC in the context of social reward, we did not find evidence for heightened threat sensitivity in adolescent girls exposed to favorable family and peer environments, suggesting that these girls may be more likely to disregard such feedback or that this feedback carries less relevance to their sense of self or emotional well-being.

4.2. Social experience and blunted neural processing of social cues

Although most of our findings supported intensified neural sensitivity following exposure to both supportive (for social reward processing) and stressful (for social reward and threat processing) environments, two findings revealed blunted sensitivity in more extreme environments. Specifically, very low and very high levels of peer adversity were associated with blunted VS-PCC FC in the context of social reward, and very low and very high levels of family adversity were associated with blunted amygdala-parahippocampal gyrus FC in the context of social threat. According to developmental programming theories, persistent exposure to adversity can overtax biological systems, eventually leading to blunted sensitivity to the environment (Juster et al., 2010; Shonkoff and Bales, 2011). Likewise, the adaptive calibration model (Ellis et al., 2017) proposes an "unemotional" profile, characterized by unresponsivity to the environment that inhibits social learning and sensitivity to social feedback; this profile is presumed to emerge in the context of traumatic stress.

The PCC has been implicated as part of a reward valuation network (Pan et al., 2017) and plays a role in attentional focus and initiating a signal for strategic changes in behavior based on environmental cues (Leech and Sharp, 2014). Moreover, prior research links early adversity with lower activation in the PCC during reward anticipation (Birn et al., 2017). Blunted VS-PCC connectivity may therefore reflect weaker cognitive modulation of behavior following reward in adolescent girls exposed to high levels of peer adversity, but additional research is needed to better elucidate the implications of this pattern. Lower amygdala-parahippocampal gyrus FC may reflect weaker stimulation of emotional memory for threat in adolescent girls exposed to high levels of family adversity. Of note, however, lower VS-PCC and amygdala-parahippocampal gyrus co-activation also was observed in girls exposed to very low levels of peer and family adversity, respectively. Consistent with the adaptive calibration model, both the developmental origins and behavioral outcomes of this dampened neural sensitivity may diverge (Ellis et al., 2017). Thus, future research will need to clarify when particularly favorable and unfavorable childhood environments promote heightened versus blunted sensitivity as well as to distinguish how the consequences of these patterns vary depending on childhood experiences.

4.3. Health implications of neural processing of social cues following adversity

Given that exposure to childhood adversity is linked to widespread differences in neural processing of social cues, the next critical step will be to examine the implications of these differences for future health. Sensitivity in several regions implicated in this study may serve as biomarkers of psychopathology in adolescence. For example, adolescents who show high activation in regions involved in threat and salience processing (e.g., amygdala and amygdala-hippocampal complex, insula, dACC, sgACC) as well as self-referential processing (e.g., MPFC) in response to social cues are at risk for anxiety and depression (Lau et al., 2012; Masten et al., 2011; McLaughlin et al., 2019; Rudolph et al., 2016; Silk et al., 2014). Disrupted FC within the reward valuation network (including connectivity between the VS and the MPFC, insula, ACC, thalamus, and PCC) also predicts future depression in adolescence (Pan et al., 2017) as well as social anhedonia in emerging adults (Wang et al., 2016). Moreover, stronger VS-MPFC FC in adversity-exposed individuals is associated with social problems in adolescence (Fareri et al., 2017) and internalizing symptoms in emerging adults (Hanson et al., 2018). In adolescent girls, higher activation in the MPFC during potentially rewarding risk taking (Telzer et al., 2018), as well as higher activation in the VS and amygdala during a group belonging task (Telzer et al., in press), are associated with concurrent and subsequent antisocial behavior and internalizing symptoms. Thus, it will be important to determine the developmental consequences associated with the patterns of neural processing identified in this study.

4.4. Contributions and limitations of the present research

This study makes several innovative contributions to theory and research aimed at elucidating how childhood adversity influences brain development. First, we extended prior research focusing mainly on adversity-linked neural processing of emotions and monetary rewards to incorporate a focus on neural processing of social reward and threat. Given the salience and emotional impact of social cues during adolescence (Guyer et al., 2014; Somerville, 2013) and their potent contribution to developmental outcomes, understanding the neural legacy of childhood adversity when adolescents are processing social cues is critical to mapping pathways through which adversity undermines long-term adjustment.

Second, we examined not only linear but also curvilinear associations between exposure to adversity and patterns of neural processing, allowing us to test critical predictions of the adaptive calibration model and to better understand the role of childhood social experiences in brain development. This approach yielded new insights into how childhood environments calibrate neural function, revealing that exposure to stressful environments intensifies processing of both social reward and threat, but exposure to supportive environments only intensifies processing of social reward. Identifying these curvilinear associations was facilitated through cumulative lifetime adversity indexes reflecting a continuum of experiences, ranging from favorable (i.e., stable, supportive family and peer relationships; normative losses) to stressful (i.e., unstable, conflictual family and peer relationships; significant losses). However, because these indexes reflect cumulative severity of adversity across a range of stressful events and circumstances, we were unable to disentangle the neural signatures of threat, deprivation, and loss. In light of a growing body of research suggesting distinct neural and behavioral sequelae of different dimensions of adversity (for a review, see McLaughlin and Sheridan, 2016; McLaughlin et al., 2019, 2014; Sheridan et al., 2017), future research will need to examine curvilinear associations using continuous indexes within each dimension. Moreover, the YLSI provided a more comprehensive assessment of adversity than favorable conditions; thus, a low rating more heavily reflected low levels of adversity, although some information was available regarding positive aspects of family and peer relationships. It will be important for future research to use assessments that cover the full spectrum of lifetime social experiences.

Third, we examined the neural correlates of exposure to both family and peer adversity within the same study, while adjusting for the alternate domain. Because prior research typically investigates adversity within one or the other of these domains, it is hard to determine their unique effects. Our findings reveal that exposure to childhood adversity in both domains uniquely predicts neural processing of social feedback during adolescence. Some similarities were found across types of adversity. When processing social reward, family and peer adversity both showed curvilinear associations with FC between the VS and regulatory (dlPFC for both) as well as emotion valuation/salience processing (insula for family; dACC for peer) regions. However, other patterns of co-activation with the amygdala and VS differed in relation to family and peer adversity. When processing social threat, family adversity was associated with heightened FC between the amygdala and regions implicated in a neural social valuation network (e.g., precuneus, lingual gyrus), whereas peer adversity was associated with heightened FC between the amygdala and a region implicated in emotional memory (parahippocampal gyrus). Because our task focused on neural activation in response to peer feedback, it is possible that negative feedback triggered specific emotional memories in girls exposed to prior peer but not family adversity. Overall, our results suggest that early experiences in the family as well as the peer group contribute to subsequent neural activation to social cues in the peer context, but an interesting question remains regarding whether similar or different patterns of activation would emerge when processing social cues in the family context during adolescence.

Finally, although our index of cumulative lifetime adversity was comprehensive, it relied on retrospective reporting. Moreover, neural processing of social cues was only examined in adolescence, making it difficult to draw strong conclusions regarding the impact of adversity on the emerging brain throughout childhood. Developmental programming models presume that neural sensitivity develops as an adaptation aimed at optimizing adjustment to current environmental demands. Thus, prospective longitudinal research needs to examine changes in neural sensitivity and its consequences across development as a function of changing environmental contexts.

4.5. Conclusion

Overall, this research suggests that cumulative lifetime exposure to family and peer adversity calibrates neural processing of both social reward and threat during adolescence. However, exposure to favorable environments also predicts neural sensitivity to reward. Because attunement to social cues may confer both costs and benefits, it will be important for future research to determine whether the adaptive value of neural sensitivity differs contingent on girls' early social history and subsequent social environments.

Declaration of Competing Interest

The authors report no declarations of interest.

Acknowledgements

We would like to thank the families and schools who participated in this study. We are grateful for the assistance of the Biomedical Imaging Center at the University of Illinois. We thank Yuji Kim and Michelle Miernicki for their assistance with data collection. This work was supported by a University of Illinois Research Board Award and a National Institute of Mental Health Grant (MH68444) awarded to K.D.R., a National Institute of Mental Health Grant (MH105655) awarded to K.D.R. and E.H.T., and a NARSAD Young Investigator Award to EHT

References

Achterberg, M., van Duijvenvoorde, A.C., Bakermans-Kranenburg, M.J., Crone, E.A., 2016. Control your anger! The neural basis of aggression regulation in response to negative social feedback. Soc. Cogn. Affect. Neurosci. 11 (5), 712–720. https://doi.org/10.1093/scan/nsv154.

Achterberg, M., van Duijvenvoorde, A.C., van der Meulen, M., Euser, S., Bakermans-Kranenburg, M.J., Crone, E.A., 2017. The neural and behavioral correlates of social evaluation in childhood. Dev. Cogn. Neurosci. 24, 107–117. https://doi.org/10.1016/j.dcn.2017.02.007.

Achterberg, M., van Duijvenvoorde, A.C., van der Meulen, M., Bakermans-Kranenburg, M.J., Crone, E.A., 2018. Heritability of aggression following social evaluation in middle childhood: an fMRI study. Hum. Brain Mapp. 39 (7), 2828–2841. https://doi.org/10.1002/hbm.24043.

Arias, J.A., Williams, C., Raghvani, R., Aghajani, M., Baez, S., Belzung, C., Booij, L., Busatto, G., Chiarella, J., Fu, C.H., Ibanez, A., Lindell, B.J., Lowe, L., Penninx, B.W.J. H., Rosa, P., Kemp, A.H., 2020. The neuroscience of sadness: a multidisciplinary synthesis and collaborative review. Neurosci. Biobehav. Rev. 111, 199–228.

Baumeister, R.F., Leary, M.R., 1995. The need to belong: desire for interpersonal attachments as a fundamental human motivation. Psychol. Bull. 117 (3), 497–529. https://doi.org/10.1037/0033-2909.117.3.497.

Birn, R.M., Roeber, B.J., Pollak, S.D., 2017. Early childhood stress exposure, reward pathways, and adult decision making. Proc. Natl. Acad. Sci. 114 (51), 13549–13554. https://doi.org/10.1073/pnas.1708791114.

Bolling, D.Z., Pitskel, N.B., Deen, B., Crowley, M.J., Mayes, L.C., Pelphrey, K.A., 2011. Development of neural systems for processing social exclusion from childhood to adolescence. Dev. Sci. 14 (6), 1431–1444. https://doi.org/10.1111/j.1467-7687.2011.01087.x.

Brett, M., Anton, J.L., Valabregue, R., Poline, J.B., 2002. Region of interest analysis using the MarsBar toolbox for SPM 99. Neuroimage 16 (2), S497.

Brown, B.B., 2013. Adolescents 'relationships with peers. In: Lerner, R., Steinberg, L. (Eds.), Handbook of Adolescent Psychology, 2nd ed. Wiley, pp. 363–394.

Brown, G.W., Harris, T., 1978. Social Origins of Depression: a Study of Psychiatric Disorder in Women. Routledge.

Casement, M.D., Guyer, A.E., Hipwell, A.E., McAloon, R.L., Hoffmann, A.M., Keenan, K. E., Forbes, E.E., 2014. Girls' challenging social experiences in early adolescence predict neural response to rewards and depressive symptoms. Dev. Cogn. Neurosci. 8, 18–27. https://doi.org/10.1016/j.dcn.2013.12.003.

Casey, B.J., Getz, S., Galvan, A., 2008. The adolescent brain. Dev. Rev. 28 (1), 62–77. https://doi.org/10.1016/j.dr.2007.08.003.

Cuthbert, B.N., Insel, T.R., 2013. Toward the future of psychiatric diagnosis: the seven pillars of RDoC. BMC Med. 11, 126 https://doi.org/10.1186/1741-7015-11-126.

Dennison, M.J., Sheridan, M.A., Busso, D.S., Jenness, J.L., Peverill, M., Rosen, M.L., McLaughlin, K.A., 2016. Neurobehavioral markers of resilience to depression amongst adolescents exposed to child abuse. J. Abnorm. Psychol. 125 (8), 1201–1212. https://doi.org/10.1037/abn0000215.

Do, K.T., Prinstein, M.J., Telzer, E.H., 2020. Neurobiological susceptibility to peer influence in adolescence. In: Kadosh, K.C. (Ed.), The Handbook of Developmental Cognitive Neuroscience. Oxford University Press.

Dolcos, F., LaBar, K.S., Cabeza, R., 2004. Interaction between the amygdala and the medial temporal lobe memory system predicts better memory for emotional events. Neuron 42 (5), 855–863. https://doi.org/10.1016/S0896-6273(04)00289-2.

Ellis, B.J., Del Giudice, M., Shirtcliff, E.A., 2017. The adaptive calibration model of stress responsivity: concepts, findings, and implications for developmental psychopathology. Dev. Psychopathol. 3, 237–276. https://doi.org/10.1017/S0954579416000985.

Ernst, M., Nelson, E.E., Jazbec, S., McClure, E.B., Monk, C.S., Leibenluft, E., Blair, J., Pine, D.S., 2005. Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. Neuroimage 25 (4), 1279–1291. https://doi.org/10.1016/j.neuroimage.2004.12.038.

Ethridge, P., Sandre, A., Dirks, M.A., Weinberg, A., 2018. Past-year relational victimization is associated with a blunted neural response to rewards in emerging adults. Soc. Cogn. Affect. Neurosci. 13 (12), 1259–1267. https://doi.org/10.1093/scan/nsy091.

Fareri, D.S., Gabard-Durnam, L., Goff, B., Flannery, J., Gee, D.G., Lumian, D.S., Caldera, C., Tottenham, N., 2017. Altered ventral striatal–medial prefrontal cortex resting-state connectivity mediates adolescent social problems after early institutional care. Dev. Psychopathol. 29 (5), 1865–1876. https://doi.org/10.1017/S0954579417001456.

Forbes, E.E., Ryan, N.D., Phillips, M.L., Manuck, S.B., Worthman, C.M., Moyles, D.L., Tarr, J.A., Sciarrillo, S.R., Dahl, R.E., 2010. Healthy adolescents' neural response to reward: associations with puberty, positive affect, and depressive symptoms. J. Am. Acad. Child Adolesc. Psychiatry 49, 62–172.

Galván, A., 2010. Adolescent development of the reward system. Front. Hum. Neurosci. 4, 6 https://doi.org/10.3389/neuro.09.006.2010.

Galván, A., Hare, T.A., Parra, C.E., Penn, J., Voss, H., Glover, G., Casey, B.J., 2006. Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. J. Neurosci. 26 (25), 6885–6892. https://doi.org/10.1523/JNEUROSCI.1062-06.2006.

Gardner, M., Steinberg, L., 2005. Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: an experimental study. Dev. Psychol. 41 (4), 625–635. https://doi.org/10.1037/0012-1649.41.4.625.

Gardner, W.L., Pickett, C.L., Jefferis, V., Knowles, M., 2005. On the outside looking in: loneliness and social monitoring. Pers. Soc. Psychol. Bull. 31 (11), 1549–1560. https://doi.org/10.1177/0146167205277208.

Goff, B., Gee, D.G., Telzer, E.H., Humphreys, K.L., Gabard-Durnam, L., Flannery, J., Tottenham, N., 2013. Reduced nucleus accumbens reactivity and adolescent depression following early-life stress. Neuroscience 249, 129–138. https://doi.org/10.1016/j.neuroscience.2012.12.010.

Gunther Moor, B., van Leijenhorst, L., Rombouts, S.A.R.B., Crone, E.A., Van der Molen, M.W., 2010. Do you like me? Neural correlates of social evaluation and developmental trajectories. Soc. Neurosci. 5, 461–482. https://doi.org/10.1080/17470910903526155.

Guyer, A.E., McClure-Tone, E.B., Shiffrin, N.D., Pine, D.S., Nelson, E.E., 2009. Probing the neural correlates of anticipated peer evaluation in adolescence. Child Dev. 80 (4), 1000–1015. https://doi.org/10.1111/j.1467-8624.2009.01313.x.

Guyer, A.E., Choate, V.R., Grimm, K.J., Pine, D.S., Keenan, K., 2011. Emerging depression is associated with face memory deficits in adolescent girls. J. Am. Acad. Child Adolesc. Psychiatry 50 (2), 180–190. https://doi.org/10.1016/j.jaac.2010.11.008.

Guyer, A.E., Caouette, J.D., Lee, C.C., Ruiz, S.K., 2014. Will they like me? Adolescents' emotional responses to peer evaluation. Int. J. Behav. Dev. 38 (2), 155–163. https://doi.org/10.1177/0165025413515627.

Hanson, J.L., Knodt, A.R., Brigidi, B.D., Hariri, A.R., 2018. Heightened connectivity between the ventral striatum and medial prefrontal cortex as a biomarker for stress-related psychopathology: understanding interactive effects of early and more recent stress. Psychol. Med. 48 (11), 1835–1843. https://doi.org/10.1017/S0033291717003348.

Hare, T.A., Tottenham, N., Galvan, A., Voss, H.U., Glover, G.H., Casey, B.J., 2008. Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. Biol. Psychiatry 63 (10), 927–934. https://doi.org/10.1016/j.biopsych.2008.03.015.

Herringa, R.J., Birn, R.M., Ruttle, P.L., Burghy, C.A., Stodola, D.E., Davidson, R.J., Essex, M.J., 2013. Childhood maltreatment is associated with altered fear circuitry and increased internalizing symptoms by late adolescence. Proc. Natl. Acad. Sci. 110 (47), 19119–19124. https://doi.org/10.1073/pnas.1310766110.

Herringa, R.J., Burghy, C.A., Stodola, D.E., Fox, M.E., Davidson, R.J., Essex, M.J., 2016. Enhanced prefrontal-amygdala connectivity following childhood adversity as a protective mechanism against internalizing in adolescence. Biol. Psychiatry Cogn. Neurosci. Neuroimaging 1 (4), 326–334. https://doi.org/10.1016/j.bpsc.2016.03.003.

Herzberg, M.P., Gunnar, M.R., 2020. Early life stress and brain function: activity and connectivity associated with processing emotion and reward. NeuroImage 209, 116493. https://doi.org/10.1016/j.neuroimage.2019.116493.

Jarcho, J.M., Grossman, H.Y., Guyer, A.E., Quarmley, M., Smith, A.R., Fox, N.A., Leibenluft, E., Pine, D.S., Nelson, E.E., 2019. Connecting childhood wariness to adolescent social anxiety through the brain and peer experiences. J. Abnorm. Child Psychol. 47 (7), 1153–1164. https://doi.org/10.1007/s10802-019-00543-4.

Javanbakht, A., King, A.P., Evans, G.W., Swain, J.E., Angstadt, M., Phan, K.L., Liberzon, I., 2015. Childhood poverty predicts adult amygdala and frontal activity and connectivity in response to emotional faces. Front. Behav. Neurosci. 9, 154 https://doi.org/10.3389/fnbeh.2015.00154.

Jedd, K., Hunt, R.H., Cicchetti, D., Hunt, E., Cowell, R.A., Rogosch, F.A., Toth, S.L., Thomas, K.M., 2015. Long-term consequences of childhood maltreatment: altered amygdala functional connectivity. Dev. Psychopathol. 27 (4), 1577–1589. https://doi.org/10.1017/S0954579415000954.

Juster, R.P., McEwen, B.S., Lupien, S.J., 2010. Allostatic load biomarkers of chronic stress and impact on health and cognition. Neurosci. Biobehav. Rev. 35 (1), 2–16. https://doi.org/10.1016/j.neubiorev.2009.10.002.

Kaiser, R.H., Clegg, R., Goer, F., Pechtel, P., Beltzer, M., Vitaliano, G., Olson, D.P., Teicher, M.H., Pizzagalli, D.A., 2018. Childhood stress, grown-up brain networks: corticolimbic correlates of threat-related early life stress and adult stress response. Psychol. Med. 48 (7), 1157–1166. https://doi.org/10.1017/S0033291717002628.

Knoll, L.J., Magis-Weinberg, L., Speekenbrink, M., Blakemore, S.J., 2015. Social influence on risk perception during adolescence. Psychol. Sci. 26 (5), 583–592. https://doi.org/10.1177/0956797615569578.

Kopala-Sibley, D.C., Cyr, M., Finsaas, M.C., Orawe, J., Huang, A., Tottenham, N., Klein, D.N., 2020. Early childhood parenting predicts late childhood brain functional connectivity during emotion perception and reward processing. Child Dev. 91 (1), 110–128. https://doi.org/10.1111/cdev.13126.

Kumar, P., Pisoni, A., Bondy, E., Kremens, R., Singleton, P., Pizzagalli, D.A., Auerbach, R. P., 2019. Delineating the social valuation network in adolescents. Soc. Cogn. Affect. Neurosci. 14 (11), 1159–1166. https://doi.org/10.1093/scan/nsz086.

Lau, J.Y., Guyer, A.E., Tone, E.B., Jenness, J., Parrish, J.M., Pine, D.S., Nelson, E.E., 2012. Neural responses to peer rejection in anxious adolescents: contributions from the amygdala-hippocampal complex. Int. J. Behav. Dev. 36 (1), 36–44. https://doi.org/10.1177/0165025411406854.

Lee, H.S., Lee, J.E., Lee, K.U., Kim, Y.H., 2014. Neural changes associated with emotion processing in children experiencing peer rejection: a functional MRI study. J. Korean Med. Sci. 29, 1293–1300. https://doi.org/10.3346/jkms2014.29.9.1.

Leech, R., Sharp, D.J., 2014. The role of the posterior cingulate cortex in cognition and disease. Brain 137 (1), 12–32. https://doi.org/10.1093/brain/awt162.

Liu, X., Hairston, J., Schrier, M., Fan, J., 2011. Common and distinct networks underlying reward valence and processing stages: a meta-analysis of functional neuroimaging studies. Neurosci. Biobehav. Rev. 35 (5), 1219–1236. https://doi.org/10.1016/j.neubiorev.2010.12.012.

Marshall, N.A., Marusak, H.A., Sala-Hamrick, K.J., Crespo, L.M., Rabinak, C.A., Thomason, M.E., 2018. Socioeconomic disadvantage and altered corticostriatal circuitry in urban youth. Hum. Brain Mapp. 39 (5), 1982–1994. https://doi.org/10.1002/hbm.23978.

Marusak, H.A., Martin, K.R., Etkin, A., Thomason, M.E., 2015. Childhood trauma exposure disrupts the automatic regulation of emotional processing. Neuropsychopharmacology 40 (5), 1250–1258. https://doi.org/10.1038/npp.2014.311.

Masten, C.L., Eisenberger, N.I., Borofsky, L.A., Pfeifer, J.H., McNealy, K., Mazziotta, J.C., Dapretto, M., 2009. Neural correlates of social exclusion during adolescence: understanding the distress of peer rejection. Soc. Cogn. Affect. Neurosci. 4 (2), 143–157. https://doi.org/10.1093/scan/nsp007.

Masten, C.L., Eisenberger, N.I., Pfeifer, J.H., Dapretto, M., 2010. Witnessing peer rejection during early adolescence: neural correlates of empathy for experiences of social exclusion. Soc. Neurosci. 5 (5–6), 496–507. https://doi.org/10.1080/17470919.2010.490673.

Masten, C.L., Eisenberger, N.I., Borofsky, L.A., McNealy, K., Pfeifer, J.H., Dapretto, M., 2011. Subgenual anterior cingulate responses to peer rejection: a marker of adolescents' risk for depression. Dev. Psychopathol. 23 (1), 283–292. https://doi.org/10.1017/S0954579410000799.

McCrory, E.J., De Brito, S.A., Sebastian, C.L., Mechelli, A., Bird, G., Kelly, P.A., Viding, E., 2011. Heightened neural reactivity to threat in child victims of family violence. Curr. Biol. 21 (23), R947–R948. https://doi.org/10.1016/j.cub.2011.10.015.

McCrory, E.J., De Brito, S.A., Kelly, P.A., Bird, G., Sebastian, C.L., Mechelli, A., Samuel, S., Viding, E., 2013. Amygdala activation in maltreated children during pre-attentive emotional processing. Br. J. Psychiatry 202 (4), 269–276. https://doi.org/10.1192/bjp.bp.112.116624.

McIver, T.A., Bosma, R.L., Sandre, A., Goegan, S., Klassen, J.A., Chiarella, J., Booij, L., Craig, W., 2018. Peer victimization is associated with neural response to social exclusion. Merrill-Palmer Q. 64 (1), 135–161. https://doi.org/10.13110/merrpalmquar1982.64.1.0135.

McIver, T.A., Bosma, R.L., Goegan, S., Sandre, A., Klassen, J., Chiarella, J., Booij, L., Craig, W., 2019. Functional connectivity across social inclusion and exclusion is related to peer victimization and depressive symptoms in young adults. J. Affect. Disord. 253, 366–375. https://doi.org/10.1016/j.jad.2019.04.085.

McLaren, D.G., Ries, M.L., Xu, G., Johnson, S.C., 2012. A generalized form of context-dependent psychophysiological interactions (gPPI): a comparison to standard approaches. Neuroimage 61 (4), 1277–1286. https://doi.org/10.1016/j.neuroimage.2012.03.068.

McLaughlin, K.A., 2016. Future directions in childhood adversity and youth psychopathology. J. Clin. Child Adolesc. Psychol. 45 (3), 361–382. https://doi.org/10.1080/15374416.2015.1110823.

McLaughlin, K.A., Sheridan, M.A., 2016. Beyond cumulative risk: a dimensional approach to childhood adversity. Curr. Dir. Psychol. Sci. 25 (4), 239–245. https://doi.org/10.1177/0963721416655883.

McLaughlin, K.A., Sheridan, M.A., Lambert, H.K., 2014. Childhood adversity and neural development: deprivation and threat as distinct dimensions of early experience. Neurosci. Biobehav. Rev. 47, 578–591. https://doi.org/10.1016/j.neubiorev.2014.10.012.

McLaughlin, K.A., Peverill, M., Gold, A.L., Alves, S., Sheridan, M.A., 2015. Child maltreatment and neural systems underlying emotion regulation. J. Am. Acad. Child Adolesc. Psychiatry 54 (9), 753–762. https://doi.org/10.1016/j.jaac.2015.06.010.

McLaughlin, K.A., Weissman, D., Bitrán, D., 2019. Childhood adversity and neural development: a systematic review. Ann. Rev. Develop. Psychol. 1, 277–312. https://doi.org/10.1146/annurev-devpsych-121318-084950.

Mehta, M.A., Gore-Langton, E., Golembo, N., Colvert, E., Williams, S.C., Sonuga-Barke, E., 2010. Hyporesponsive reward anticipation in the basal ganglia following severe institutional deprivation early in life. J. Cogn. Neurosci. 22 (10), 2316–2325. https://doi.org/10.1016/j.brainres.2012.02.027.

Nelson, E.E., Jarcho, J.M., Guyer, A.E., 2016. Social re-orientation and brain development: an expanded and updated view. Dev. Cogn. Neurosci. 17, 118–127. https://doi.org/10.1016/j.dcn.2015.12.008.

O'Connor, T.G., 2003. Early experiences and psychological development: conceptualquestions, empirical illustrations, and implications for intervention. Dev. Psychopathol. 15 (3), 671–690. https://doi.org/10.1017/S0954579403000336.

Pagliaccio, D., Luby, J.L., Bogdan, R., Agrawal, A., Gaffrey, M.S., Belden, A.C., Botteron, K.N., Harms, M.P., Barch, D.M., 2015. Amygdala functional connectivity, HPA axis genetic variation, and life stress in children and relations to anxiety and emotion regulation. J. Abnorm. Psychol. 124 (4), 817–833. https://doi.org/10.1037/abn0000094.

Pan, P.M., Sato, J.R., Salum, G.A., Rohde, L.A., Gadelha, A., Zugman, A., Mari, J., Jackowski, A., Picon, F., Miguel, E.C., Pine, D.S., Leibenluft, E., Bressan, R.A., Stringaris, A., 2017. Ventral striatum functional connectivity as a predictor of adolescent depressive disorder in a longitudinal community-based sample. Am. J. Psychiatry 174 (11), 1112–1119. https://doi.org/10.1176/appi.ajp.2017.17040430.

Perino, M.T., Miernicki, M.E., Telzer, E.H., 2016. Letting the good times roll: Adolescence as a period of reduced inhibition to appetitive social cues. Soc. Cogn. Affect. Neurosci. 11, 1762–1771.

Rappaport, B.I., Hennefield, L., Kujawa, A., Arfer, K.B., Kelly, D., Kappenman, E.S., Luby, J.L., Barch, D.M., 2019. Peer victimization and dysfunctional reward processing: ERP and behavioral responses to social and monetary rewards. Front. Behav. Neurosci. 13, 120 https://doi.org/10.3389/fnbeh.2019.00120.

Rogers, C.R., Perino, M.T., Telzer, E.H., 2019. Maternal buffering of adolescent dysregulation in socially appetitive contexts: From behavior to the brain. J. Res. Adolesc. 30 (1), 41–52.

Rudolph, K.D., Flynn, M., 2007. Childhood adversity and youth depression: the role of gender and pubertal status. Dev. Psychopathol. 19 (2), 497–521. https://doi.org/10.1017/S0954579407070241.

Rudolph, K.D., Miernicki, M.E., Troop-Gordon, W., Davis, M.M., Telzer, E.H., 2016. Adding insult to injury: neural sensitivity to social exclusion is associated with internalizing symptoms in chronically peer-victimized girls. Soc. Cogn. Affect. Neurosci. 11 (5), 829–842. https://doi.org/10.1093/scan/nsw021.

Schriber, R.A., Rogers, C.R., Ferrer, E., Conger, R.D., Robins, R.W., Hastings, P.D., Guyer, A.E., 2018. Do hostile school environments promote social deviance by shaping neural responses to social exclusion? J. Res. Adolesc. 28 (1), 103–120. https://doi.org/10.1111/jora.12340.

Sheridan, M.A., McLaughlin, K.A., 2014. Dimensions of early experience and neural development: deprivation and threat. Trends Cogn. Sci. 18 (11), 580–585. https://doi.org/10.1016/j.tics.2014.09.001.

Sheridan, M.A., Peverill, M., Finn, A.S., McLaughlin, K.A., 2017. Dimensions of childhood adversity have distinct associations with neural systems underlying executive functioning. Dev. Psychopathol. 29 (5), 1777–1794. https://doi.org/10.1017/S0954579417001390.

Shonkoff, J.P., Bales, S.N., 2011. Science does not speak for itself: translating child development research for the public and its policymakers. Child Dev. 82 (1), 17–32. https://doi.org/10.1111/j.1467-8624.2010.01538.x.

Silk, J.S., Siegle, G.J., Lee, K.H., Nelson, E.E., Stroud, L.R., Dahl, R.E., 2014. Increased neural response to peer rejection associated with adolescent depression and pubertal development. Soc. Cogn. Affect. Neurosci. 9 (11), 1798–1807. https://doi.org/10.1093/scan/nst175.

Somerville, L.H., 2013. The teenage brain: sensitivity to social evaluation. Curr. Dir. Psychol. Sci. 22 (2), 121–127. https://doi.org/10.1177/0963721413476512.

Somerville, L.H., Jones, R.M., Casey, B.J., 2010. A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. Brain Cogn. 72 (1), 124–133. https://doi.org/10.1016/j.bandc.2009.07.003.

Spear, L.P., 2009. Heightened stress responsivity and emotional reactivity during pubertal maturation: implications for psychopathology. Dev. Psychopathol. 21 (1), 87–97. https://doi.org/10.1017/S0954579409000066.

Steinberg, L., Monahan, K.C., 2007. Age differences in resistance to peer influence. Dev. Psychol. 43 (6), 1531–1543. https://doi.org/10.1037/0012-1649.43.6.1531.

Steinberg, L., Morris, A.S., 2001. Adolescent development. Annu. Rev. Psychol. 52 (1), 83–110 https://doi.org/10.1146.annurev.psych.52.1.83.

Telzer, E.H., 2016. Dopaminergic reward sensitivity can promote adolescent health: a new perspective on the mechanism of ventral striatum activation. Dev. Cogn. Neurosci. 17, 57–67. https://doi.org/10.1016/j.dcn.2015.10.010.

Telzer, E.H., Fuligni, A.J., Lieberman, M.D., Miernicki, M.E., Galván, A., 2015. The quality of adolescents' peer relationships modulates neural sensitivity to risk taking. Soc. Cogn. Affect. Neurosci. 10 (3), 389–398. https://doi.org/10.1093/scan/nsu064.

Telzer, E.H., Miernicki, M.E., Rudolph, K.D., 2018. Chronic peer victimization heightens neural sensitivity to risk taking. Dev. Psychopathol. 30 (1), 13–26. https://doi.org/10.1017/S0954579417000438.

Telzer, E.H., Fowler, C.H., Davis, M.M., Rudolph, K.D., 2019. Hungry for inclusion: exposure to peer victimization and heightened social monitoring in adolescent girls. Dev. Psychopathol. 1–14. https://doi.org/10.1017/s0954579419001433.

Thomason, M.E., Marusak, H.A., Tocco, M.A., Vila, A.M., McGarragle, O., Rosenberg, D. R., 2015. Altered amygdala connectivity in urban youth exposed to trauma. Soc. Cogn. Affect. Neurosci. 10 (11), 1460–1468. https://doi.org/10.1093/scan/nsv030.

Van Leijenhorst, L., Moor, B.G., Op de Macks, Z.A., Rombouts, S.A., Westenberg, P.M., Crone, E.A., 2010. Adolescent risky decision-making: neurocognitive development of reward and control regions. Neuroimage 51 (1), 345–355. https://doi.org/10.1016/j.neuroimage.2010.02.038.

Vijayakumar, N., Cheng, T.W., Pfeiffer, J.H., 2017. Neural correlates of social exclusion across ages: a coordinate-based meta-analysis of functional MRI studies. Neuroimage 153, 359–368. https://doi.org/10.1016/j.neuroimage.2017.02.050.

Wang, Y., Liu, W.H., Li, Z., Wei, X.H., Jiang, X.Q., Geng, F.L., Zou, L.Q., Lui, S.S.Y., Cheung, E.F.C., Pantelis, C., Chan, R.C.K., 2016. Altered corticostriatal functional connectivity in individuals with high social anhedonia. Psychol. Med. 46 (1), 125–135. https://doi.org/10.1017/S0033291715001592.

Ward, B.D., 2000. Simultaneous Inference for fMRI Data. Retrieved February 23, 2018, from. http://afni.nimh.nih.gov/pub/dist/doc/manuals/AlphaSim.pdf.

Will, G.J., van Lier, P.A., Crone, E.A., Güroğlu, B., 2016. Chronic childhood peer rejection is associated with heightened neural responses to social exclusion during adolescence. J. Abnorm. Child Psychol. 44 (1), 43–55. https://doi.org/10.1007/s10802-015-9983-0.