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Author/s:

Barendse, MEA;Allen, NB;Sheeber, L;Pfeifer, JH

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The impact of depression on mothers' neural processing of their adolescents' affective behavior

Marjolein E.A. Barendse,¹ Nicholas B. Allen,¹ Lisa Sheeber,² and Jennifer H. Pfeifer¹

¹Department of Psychology, University of Oregon, Eugene, OR 97403, USA

²Oregon Research Institute, Eugene, OR, USA

Correspondence should be addressed to Marjolein E.A. Barendse, Department of Psychology, University of Oregon, 1444 Franklin Blvd, Eugene, OR 97403, USA.

E-mail: barendse@uoregon.edu.

Abstract

Depression affects neural processing of emotional stimuli and could, therefore, impact parent–child interactions. However, the neural processes with which mothers with depression process their adolescents' affective interpersonal signals and how this relates to mothers' parenting behavior are poorly understood. Mothers with and without depression ($N = 64$ and $N = 51$, respectively; $M_{\text{age}} = 40$ years) from low-income families completed an interaction task with their adolescents ($M_{\text{age}} = 12.8$ years), which was coded for both individuals' aggressive, dysphoric, positive and neutral affective behavior. While undergoing fMRI, mothers viewed video clips from this task of affective behavior from their own and an unfamiliar adolescent. Relative to non-depressed mothers, those with depression showed more aggressive and less positive affective behavior during the interaction task and more activation in the bilateral insula, superior temporal gyrus and striatum but less in the lateral prefrontal cortex while viewing aggressive and neutral affect. Findings were comparable for own and unfamiliar adolescents' affect. Heightened limbic, striatal and sensory responses were associated with more aggressive and dysphoric parenting behavior during the interactions, while reduced lateral prefrontal activation was associated with less positive parenting behavior. These results highlight the importance of depressed mothers' affective information processing for understanding mothers' behavior during interactions with their adolescents.

Key words: depression; parenting; adolescence; fMRI; affective processing

Introduction

Nurturing, positive parenting behavior is associated with child and adolescent well-being. For example, some studies have shown that adolescents' relationships with their parents are more predictive of their depressive symptoms than are relationships with their peers (Stice *et al.*, 2004). Depression, one of the most common mental disorders, affects parenting behavior and parent–child interactions. Observational studies with mothers and children have consistently demonstrated that depression predicts more negative parental behavior and, to a lesser extent, reduced positive parenting behaviors (Lovejoy *et al.*, 2000). Further, mothers' depressive symptoms have been shown to be associated with less positive behavior during problem-solving interactions (PSIs) and more dysphoric behavior during event-planning interactions (EPIs) with their adolescents (Schwartz *et al.*, 2014).

As parenting is a social and interpersonal process, parents' perception, interpretation, and neurobiological processing of their children's affective and interpersonal signals is a likely pathway through which depression impacts parenting. Deficits in processing of affective and interpersonal stimuli among depressed

persons have been shown consistently (Groenewold *et al.*, 2013). These can be divided into a strengthened neural activation or emotional response to negative stimuli and a reduced response to rewarding or positive stimuli. A meta-analysis of neural processing of emotional stimuli in people with depression demonstrated hyperactivation for negative stimuli and hypoactivation for positive stimuli in limbic regions (amygdala, parahippocampal, insula), reward-related regions [striatum, anterior cingulate cortex (ACC)] and the cerebellum and fusiform gyrus (Groenewold *et al.*, 2013). Depressed persons also had reduced activation in the left dorsolateral prefrontal cortex (PFC) for negative stimuli.

Studies covered in this meta-analysis examined processing of general, non-personally relevant stimuli; however, the affective expressions of one's own child are likely to be more salient than general emotional stimuli. Mothers activate similar brain regions when viewing their own child's affect compared to an unfamiliar child's affect but with stronger activation in limbic regions, the striatum and ventrolateral PFC (Morgan *et al.*, 2015; Rigo *et al.*, 2019). Similarly, the cingulate, precuneus and ventrolateral PFC activate more strongly when adolescents see their own mother's face compared to an unfamiliar mother (Whittle *et al.*,

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2012). How mothers with depression process their own children's affective and interpersonal signals has mainly been examined in mothers with postnatal depression and their infants. The findings showed differential activation in limbic regions, but in inconsistent directions, and this research was conducted in small samples (Barrett et al., 2012; Laurent and Ablow, 2013; Wonch et al., 2016). One study examined this in 19 mothers of adolescents with internalizing disorders and demonstrated the importance of regions in the 'self' network: mothers who had experienced a higher number of depressive episodes activated the ventromedial PFC less strongly to negative emotions of their child and activated the precuneus less to positive and more to negative emotions of their child (Morgan et al., 2015).

It is unknown how mothers with depression process their own adolescents' affective and interpersonal signals compared to non-depressed mothers. This is important to examine because parent-child relationships remain essential for child well-being in adolescence. Therefore, we aimed to examine the extent to which neural activation differs between mothers with and without depression when viewing their own and another adolescent's affective behavior.

Further, previous studies have suggested that mothers' processing of their adolescents' affective and interpersonal signals is a likely pathway through which depression impacts parenting. To test this, we examine how neural activation when viewing their own (vs other) adolescents' facial expressions relates to the mother's parenting behavior as observed during parent-child interactions. Similar to the literature on the link between depression and processing of own children's emotions, there is a paucity of studies on parenting behavior and processing of (own) children's facial expressions, and the existing studies are mostly with mothers of infants. The existing studies point to the relevance of similar brain regions as discussed above, where positive parenting behaviors or relationships were associated with the activation of lateral prefrontal regions and self-referential processing regions and limited (relatively low) limbic activation to children's negative emotional expressions (Musser et al., 2012; Laurent and Ablow, 2013; Morgan et al., 2015; Turpyn et al., 2019).

Aims and hypotheses

The preregistered (<https://osf.io/2qz56>) research questions and hypotheses are as follows:

- To what extent does neural activation when viewing their own and another adolescents' affective behavior differ between mothers with and without depression?

We expected that depressed mothers would have greater activation than non-depressed mothers in the amygdala, hippocampus, anterior insula and ACC when viewing negative interpersonal behavior (especially aggressive behavior) and show lower activity in the dorsolateral and ventromedial PFCs to negative interpersonal behavior. We also hypothesized that depressed mothers would show reduced reactivity of the nucleus accumbens in response to positive interpersonal stimuli, compared to non-depressed mothers. Finally, we not only expected these group differences to be most prominent when viewing one's own adolescent but also expected greater activation in amygdala and ventrolateral PFC when viewing one's own adolescent (compared to unfamiliar) in both groups.

- How does neural activation when viewing their own (and other) adolescents' facial expressions relate to the mother's

parenting behavior as observed during parent-child interactions?

We hypothesized that stronger activation to adolescents' negative affective behavior, as well as reduced activation to adolescents' positive affective behavior, as described above in hypothesis 1, is associated with higher rates of observed maternal aggressive and dysphoric parenting behavior and with lower levels of observed maternal positive parenting behavior.

Methods

Participants

Low-income mothers ($N = 180$) and their early adolescents (ages 11–14) participated in the study. The majority of participants ($n = 132$) were recruited through the organization that administered the Oregon Health Plan (Medicaid) in the county where data were collected. The remainder of the sample ($n = 48$) were recruited through online advertisements; those recruited online were screened to ensure that their incomes would have rendered them eligible for Medicaid. Two groups of women/mothers were recruited: (i) a depressed ($n = 90$) group in which women reported currently elevated depressive symptoms and had a history of treatment for depressive disorder and (ii) a non-depressed ($n = 90$) group in which women had no more than mild symptoms of depression currently, and no lifetime history of treatment for depression, and no current (last month) mental health treatment. Current depressive symptoms were measured with the Patient Health Questionnaire [PHQ-8 (Kroenke et al., 2001); range 0–24] and those in the depressed group were required to have a score >10 , whereas mothers recruited for the non-depressed group had to score <8 . Inclusion criteria for the depression group were based on the symptom level and a history of treatment for depression rather than diagnostic status because of evidence that elevated depressive symptoms are associated with parenting difficulties and risk for adverse child outcomes regardless of diagnostic status (Lovejoy et al., 2000; Goodman et al., 2020). Moreover, the history of treatment-seeking selected for those women who had experienced a significant level of impairment or longer duration (Dew et al., 1988; Mojtabei and Olsson, 2006).

Exclusion criteria included current psychosis or having another illness or cognitive impairment that would interfere with participation (for either mother or adolescent). Mothers who participated in the fMRI task could not possess MRI contraindications. Mothers from the two groups did not differ in age, income or their adolescents' sex, age or race. For further details on recruitment and procedures, see Nelson et al. (2020). Mothers and adolescents provided informed consent and assent, respectively, and all procedures were approved by the Institutional Review Board of Oregon Research Institute.

All participants completed the parent-child interaction task. One hundred fifteen participants had complete and good-quality data for the personalized affect fMRI task. Out of the remainder of the sample, 17 participants declined or did not go through with MRI, 12 were ineligible for MRI, 33 completed part of the MRI but not the personalized affect task (most did not complete this task because not all affects were represented in interactional tasks, see stimuli description below) and 3 participants were excluded for motion artifacts (see the "Processing of fMRI data" section below). Out of the participants without fMRI data, 40% were from the depression group and 60% from the control group. There were no significant differences between participants with and without fMRI data in age, adolescents' age, adolescents' sex, PHQ score

or any of the parenting variables (independent samples *t*-tests or chi-square test, all *P* values > 0.05).

Parent-child interaction task

Mothers and adolescents completed two 15-min interaction tasks that were video recorded for coding. One task was an EPI in which they were asked to plan a vacation they would like to take together. The second task was a PSI, in which families were asked to discuss and try to resolve two areas of conflict chosen from the Issues Checklist (Prinz et al., 1979). The ordering of tasks was counterbalanced and separated by a puzzle task to reduce affect carry-over from one task to the next. Trained observers coded mother and adolescent behavior with the Living in Family Environments coding system [LIFE (Hops et al., 1995)]. The LIFE system was developed to assess behaviors characteristic of depressed individuals as well to facilitate the examination of functional relations between the behavior of depressed persons and that of their family members. It is an event-based, microanalytic coding system in which a new code is entered each time there is a change in a participant's verbal content or non-verbal and paraverbal affective behavior. These micro-level data are combined into mutually exclusive constructs, composed of both affective and verbal content codes (Hops et al., 1995). The proportions of duration (PRDs) of three constructs, reflecting aggressive, dysphoric and positive interpersonal behavior, are the primary indices of maternal parenting behavior. PRD is calculated as the amount of time the behavior is displayed during the course of the interaction over the duration of the interaction. Aggressive behavior includes aggressive (e.g. raised voice; clenched teeth) or contemptuous (e.g. eye rolling; sneering) affective behavior and critical, provoking or irritating statements. Dysphoric behavior is defined by sad non-verbal behavior (e.g. tearfulness, sighing) or complaining statements. Positive behavior is defined by happy or caring non-verbal behavior and humorous, validating, caring or approving statements. These constructs have been used extensively in our work and have been shown to relate to both maternal and adolescent depressive symptoms (Hops et al., 1995; Sheeber et al., 2007; Schwartz et al., 2014).

Data were coded by extensively trained observers blind to diagnostic status. Approximately 20% of the videos were coded by an additional observer for reliability. The interclass correlation (ICC) for maternal EPI aggressive behavior was 0.72, EPI dysphoric behavior was 0.95, EPI positive behavior was 0.92, PSI aggressive behavior was 0.93, PSI dysphoric behavior was 0.97 and PSI positive behavior was 0.93. The ICC for adolescent EPI aggressive behavior was 0.87, EPI dysphoric behavior was 0.93, EPI positive behavior was 0.89, PSI aggressive behavior was 0.82, PSI dysphoric behavior was 0.88 and PSI positive behavior was 0.90. These ICCs reflect good agreement.

fMRI Task and scan paradigm

Stimuli for the personalized affect fMRI task were approximately 3 s video clips of the mother's adolescent or an unfamiliar adolescent displaying positive, aggressive, dysphoric or neutral behavior. Stimuli of the 'own adolescent' condition were derived from the video recording of the parent-child interaction task described above. The video clips were selected by a trained staff member and then validated by a second rater; if there were discrepancies, they would discuss it to reach a consensus. The unfamiliar adolescents were actors, matched on sex to the target adolescent, discussing topics typical to the mother-adolescent interaction task. All clips had video and audio and session runners ensured that sound was working in the scanner. There were 15 trials of each

condition, which led to 120 trials in total (15 × 4 emotions × own and unfamiliar adolescent). Trials were approximately 3 s long and interspersed with 1 s rest (fixation cross).

All scans were acquired using a Siemens Skyra 3.0 Tesla scanner at the Lewis Center for Neuroimaging at the University of Oregon. Scan specifications were as follows: 2 mm isometric voxels, multiband acceleration factor = 3, in plane acceleration factor = 2, TR = 2000 ms, TE = 25 ms, 72 slices of 208 × 208 mm.

Processing of fMRI data

FMRIprep 20.2.1 was used to preprocess the fMRI data (Esteban et al., 2019). The processing of the T1-weighted reference scan included intensity non-uniformity correction, skull stripping, tissue segmentation, surface reconstruction and normalization to standard space (MNI152Nlin2009cAsym). Processing of the functional volumes included susceptibility distortion correction (SDC) based on two fieldmaps, co-registration to the T1-weighted scan, head motion estimation and correction, slice time correction and resampling to standard space (MNI152Nlin2009cAsym). All resamplings were performed with a single interpolation step by composing all the pertinent transformations. Eight participants did not have a complete fieldmap scan, so the fieldmapless SDC correction was used for these participants. After running FMRIprep, images were smoothed at 6 mm Full Width at Half Maximum (FWHM).

We used an automated motion classifier (auto-motion-fmriprep, <https://github.com/dcosme/auto-motion-fmriprep>) to identify motion artifacts in each volume. Using the motion-related output from fmriprep, this classifier marks for each volume the presence or absence of substantive motion artifacts. Participants were excluded if their scans contained substantive motion artifacts in >25% of volumes.

Subject-level models were set up and estimated in SPM12. We created event-related models following the general linear model, using a canonical hemodynamic response function, high-pass filtering of 100 s and the FAST algorithm for autocorrelation modeling. Motion regressors (Euclidean distance, Euclidean rotation, the first derivatives of both and the binary substantive motion artifact regressor from the automated motion classifier described above) were included in these subject level models as regressors of no interest.

Analyses

Group differences

At the second-level (group-level), we set up a mixed-effects model with 3dLME in AFNI 20.3.00, which included the within-subjects factor 'relevance' (i.e. own vs unfamiliar adolescent), the within-subjects factor 'affective behavior category' (positive, aggressive, dysphoric, neutral) and the between-subjects factor 'group' (depressed and non-depressed). We tested the main effects of personal relevance, affect and group, as well the interaction between these factors in a whole-brain analysis. If any main effect of or interaction with affect emerged, specific contrasts explored the difference between each affective behavior category and neutral behavior.

Association with parenting behavior

To test the association between neural activation to adolescents' affective behavior and parenting behavior, we added parent affective behavior from the parent-child interaction as covariates in the 3dLME model in AFNI 20.3.00. Note that Group was not included as a factor in these analyses, because group differences in parenting behavior would create collinearity. Six covariates

were considered: PRD of aggressive, dysphoric and positive behaviors during the EPI and PRD of aggressive, dysphoric and positive behaviors during the PSI. PRDs during the PSI were all correlated less than 0.80 with PRDs of the same affective behavior construct during the EPI (e.g. aggressive behavior during PSI and aggressive behavior during EPI); therefore, they were analyzed separately. PRDs were mean-centered.

Mediation analyses

We extracted activation from six clusters where group differences were found and tested the following mediation model: depression status → ROI activation → parent affective behavior. Since group differences were found in interaction with affect, affect was included as a moderator of the 'a' and 'b' paths. These six clusters are highlighted in Table 1; they included the four strongest clusters (i.e. those with the highest peak F values from the group-by-affect analysis) as well as two additional clusters that were selected because they aligned with our hypotheses. We extracted mean activation across all voxels in the cluster for each of the affect conditions (i.e. aggressive, dysphoric, positive and neutral) using AFNI's 3dmaskave. For mediation, we used lavaan in R v3.6.3 with full information likelihood and robust standard errors. We compared a model that constrained the indirect path to be the same for all four affect conditions to a model without these constraints, with the chi-square difference test, to determine if there was moderated mediation. We then reported indirect effects of the best model. Note that we deviated from the preregistered analysis plan here, which described testing mediation for all clusters where group differences were found. We chose instead to select clusters to reduce the multiple comparison problem.

Thresholding and reporting

For all whole-brain analyses, we used AFNI 3dFWHMx and 3dClustSim in accordance with their guidelines (Cox *et al.*, 2017) to determine the statistical significance and cluster-forming threshold for cluster FWE correction, setting a voxel-wise threshold of $P < 0.001$. This led to a cluster-size threshold of 47 (at $NN = 2$) to achieve an FWE-corrected alpha of 0.05. In the analyses of associations with parenting behavior, we additionally corrected for the number of examined parenting variables by applying a Bonferroni correction adjusted for the average correlation between the parenting variables (<https://www.quantitativeskills.com/sisa/calculations/bonfer.htm>). This correlation was 0.30, increasing the cluster-size threshold to 62 to achieve an FWE-corrected alpha of 0.0143. In the mediation analyses, we corrected for the number of parenting variables and the number of brain activation clusters, again adjusting for the correlation between parenting variables as described above. This led to a corrected alpha of 0.004. The results showed four extreme outliers on the neural activation data, so sensitivity analyses were run with and without these participants. Almost all clusters were robust to this sensitivity analysis; thus, the final neuroimaging results reported are without outliers ($N = 111$) to be conservative. Location labels were based on the Eickhoff-Zilles macro labels from N27 (Eickhoff *et al.*, 2007). Unthresholded maps are available on request.

Results

Descriptives

Table 1 summarizes demographic characteristics and mothers' affective behaviors during the observational tasks. As expected

Table 1. Demographic data and parenting variables by group

N = 115	Depression group mean (SD) n = 64	Control group mean (SD) n = 51	Difference
Mother age	40.34 (6.45)	40.22 (6.81)	$t = 0.09$, $P = 0.93$
Adolescent age	12.94 (1.30)	12.77 (1.26)	$t = 0.70$, $P = 0.49$
Sex adolescent	34 F, 30 M	18 F, 33 M	$\chi^2 = 2.96$, $P = 0.09$
PHQ-8 score	14.23 (3.22)	2.80 (2.17)	$t = 22.65$, $P < 0.001$
GAD7 score	10.03 (4.52)	2.12 (2.91)	$t = 10.01$, $P < 0.001$
Mother aggressive affect EPI	0.06 (0.08)	0.02 (0.02)	$t = 3.74$, $P < 0.001$
Mother dysphoric affect EPI	0.20 (0.16)	0.17 (0.15)	$t = 1.01$, $P = 0.31$
Mother positive affect EPI	0.18 (0.09)	0.22 (0.10)	$t = -2.00$, $P = 0.048$
Mother aggressive affect PSI	0.20 (0.20)	0.09 (0.09)	$t = 4.19$, $P < 0.001$
Mother dysphoric affect PSI	0.18 (0.13)	0.16 (0.17)	$t = 0.62$, $P = 0.54$
Mother positive affect PSI	0.14 (0.09)	0.20 (0.10)	$t = -3.57$, $P < 0.001$

Note: group differences were tested with a two-sample t-test, except for adolescent sex, which was tested with a chi-square test. Significant group differences are in bold.

based on high comorbidity rates in the population, mothers with depression had higher general anxiety symptoms than mothers in the control group. Within the depression group, 45% were using antidepressants (most commonly SSRIs or SSNRIs). Four participants in the control group (8%) were taking antidepressants as treatment for headaches/migraine ($n = 2$) or anxiety ($n = 2$; not reported during eligibility screening, but reported during session).

Affective behavior

Mothers with depression showed more aggressive affect and less positive affect than the control group during both types of parent-child interaction tasks (Table 1). Table 2 presents correlations between mothers' affective behavior variables.

Task effects and group differences

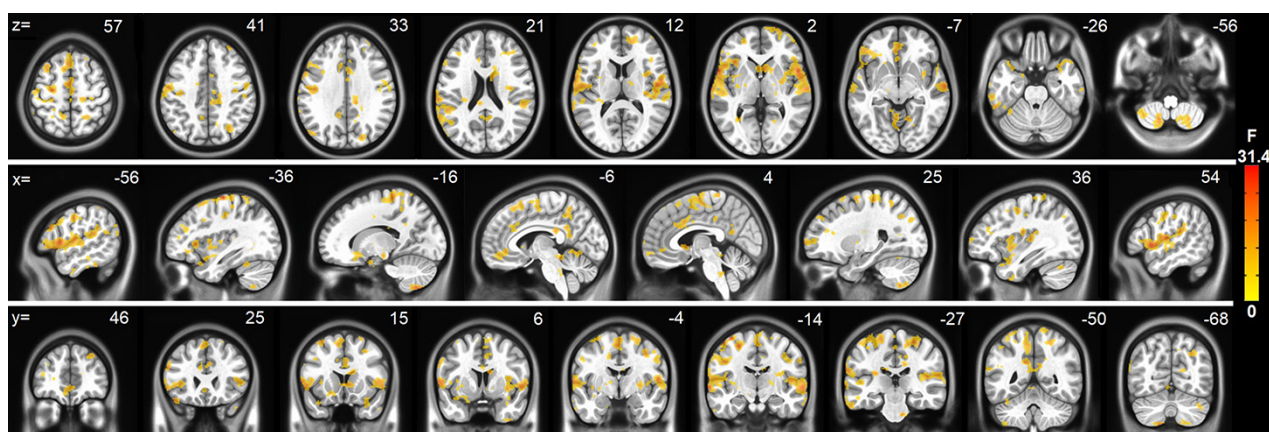
There was no three-way interaction of group-by-affect-by-personal-relevance. Two-way interactions appeared for affect-by-personal-relevance and group-by-affect but not for group-by-personal-relevance. Considering our aim to examine differences between mothers with and without depression, we focus on the group-by-affect findings. The results for the interaction between affect and personal relevance can be found in Supplementary Table S1 and Supplementary Figure S1.

Group differences were most prevalent for the aggressive and neutral affective stimuli. Figure 1 and Table 3 present all significant clusters for the group-by-affect interaction. Compared to non-depressed mothers, mothers with depression showed more bilateral insula, inferior frontal gyrus (IFG), superior temporal gyrus (STG) and striatum activation in response to adolescents' aggressive and neutral affect (see Figure 2). The non-depressed mothers activated the lateral prefrontal cortex (LPFC) more in response to aggressive and neutral affect. Also, only the control

Table 2. Spearman correlations between parenting variables

	EPI dysphoric	EPI positive	EPI other	PSI aggressive	PSI dysphoric	PSI positive	PSI other	Age mother	PHQ
EPI aggressive	0.29	-0.37	-0.29	0.48	0.00	-0.33	-0.34	0.04	0.27
EPI dysphoric		-0.39	-0.73	0.08	0.62	-0.20	-0.39	-0.03	0.12
EPI positive			-0.09	-0.26	-0.20	0.55	0.04	-0.02	-0.17
EPI other				-0.09	-0.44	-0.01	0.58	0.13	-0.07
PSI aggressive					-0.14	-0.47	-0.42	0.01	0.30
PSI dysphoric						-0.10	-0.49	0.02	0.19
PSI positive							-0.01	0.05	-0.29
PSI other								0.13	-0.23
Age mother									0.08

Note: Significant correlations (at $P < 0.05$) are in bold and correlations between tasks within affective category in blue.

**Fig. 1.** Significant clusters for the interaction between EPI group and affect, thresholded at FWE-corrected $P < 0.05$.

group activated self-related regions (medial PFC and precuneus) more in response to positive or dysphoric affect than to neutral or aggressive affect (see Figure 2).

Associations with parenting behavior

There were no three-way interactions of affect-by-personal-relevance-by-parent-behavior for any of the parent behavior variables. Parent behavior also did not interact with personal relevance. However, there were significant interactions between affect and each of the parent behavior variables. Several clusters overlapped with those found in the analyses of group differences. For example, more aggressive and dysphoric parent behavior during the event planning task was associated with higher insula, IFG and STG activation in response to adolescents' aggressive and neutral affect (see Figures 3 and 4). More dysphoric parent behavior was also associated with higher striatum and medial PFC/ACC activation to aggressive and neutral affective behavior (see Figure 4). More positive parent behavior, on the other hand, was associated with more LPFC activation to aggressive or neutral affective behavior and less activation in the hippocampus and brain stem to aggressive affective behavior (see Figure 5).

Mediation effects

For six clusters from the whole-brain analysis of group-by affect, we extracted activation and tested the following mediation model: depression status \rightarrow ROI activation \rightarrow parent affective behavior. Table 4 reports the indirect effects for each ROI and parent affective behavior. None of the indirect effects survived correction for multiple comparisons. Direct effects between group and parent affective behavior were significant for parent

aggressive and positive behavior (not reported in Table 4, but consistent with the t-tests reported in Table 1).

Discussion

In the current study, we aimed to examine the extent to which neural activation when viewing their own and another adolescent's affective behavior differs between mothers with depression and non-depressed mothers. Largely in line with our hypotheses, mothers with depression had higher activation in the anterior insula, ventral striatum and IFG and lower activation in the LPFC while viewing aggressive and neutral affective behavior. These group differences were similar across viewing their own and an unfamiliar adolescent. Our second aim was to investigate how neural activation when viewing their own and other adolescent's facial expressions relates to the mother's parenting behavior as observed during parent-child interactions. Mothers who showed more aggressive affective behavior activated the insula, IFG and STG more in response to adolescents' aggressive and neutral affect, compared to mothers who showed less aggressive affective behavior. More dysphoric parent behavior was associated with increased activation in the same regions, as well as higher striatum and medial PFC/ACC activation to aggressive and neutral affective behavior. More positive parent behavior, on the other hand, was associated with more LPFC activation to aggressive or neutral affective behavior.

Salience

The higher anterior insula activation in response to aggressive and neutral affective behavior is in line with findings that people with depression react more strongly to negative affective stimuli

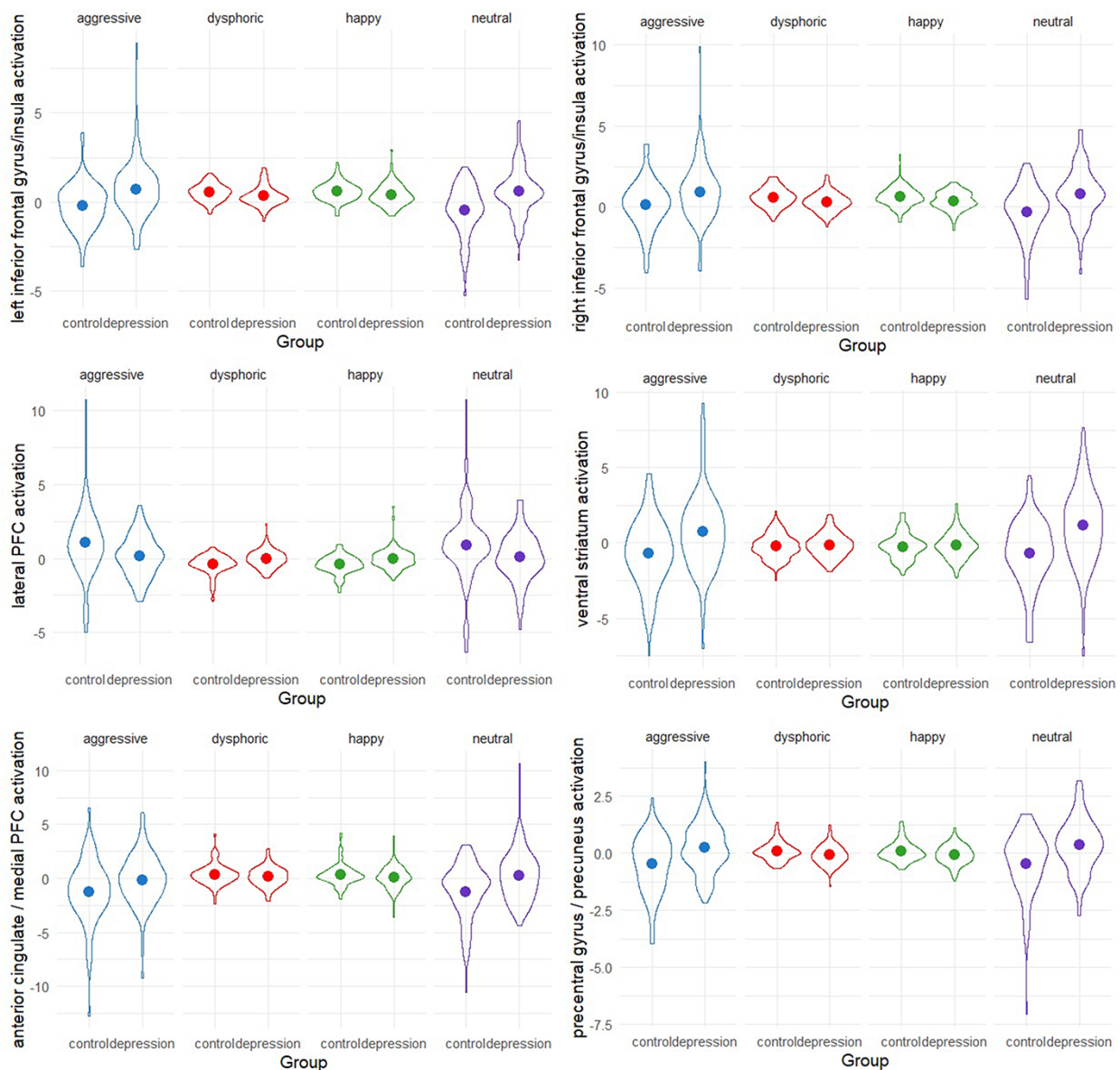


Fig. 2. Illustration of the interaction pattern for six selected clusters where group interacted with affect.

and process neutral stimuli as more negative (Roiser *et al.*, 2012; Groenewold *et al.*, 2013; Young *et al.*, 2017). This relatively high insula activation was also associated with more aggressive and dysphoric behavior by the mother, especially during the event-planning task, which is designed to elicit more positive interactions (making aggressive and dysphoric behavior more out of context). Similarly, the increased ventral striatum activation is in line with previous research (Groenewold *et al.*, 2013) and could represent an increased salience of the aggressive and neutral stimuli (Jensen *et al.*, 2007).

Perceptual processes

We also found higher STG activation to aggressive and neutral faces in the depression group as well as associations of aggressive parent behavior with activation in the STG, MTG and superior temporal sulcus in between. These regions are involved in speech processing, theory of mind and the integration of (auditory and visual) social signals (Hein and Knight, 2008), and their activation

might be explained by our use of video stimuli, providing dynamic auditory and visual information. A meta-analysis on neural processing of emotion in depression reported that the studies on this topic predominantly used static visual stimuli (most commonly affective faces) (Groenewold *et al.*, 2013). It also demonstrated that people with depression activated the fusiform gyrus, a face processing region, more in response to negative emotions, which they argued might reflect a perceptual bias toward these negative emotional stimuli. Our findings might be interpreted in the same way, but showing up in different regions because of the inclusion of auditory emotional information.

Regulatory processes

Lower lateral PFC activation in response to aggressive and neutral affective behavior aligns with findings that people with depression have difficulty with emotion regulation and are less likely to activate regulatory areas in the dorsolateral PFC (Groenewold *et al.*, 2013). Although participants were not specifically instructed

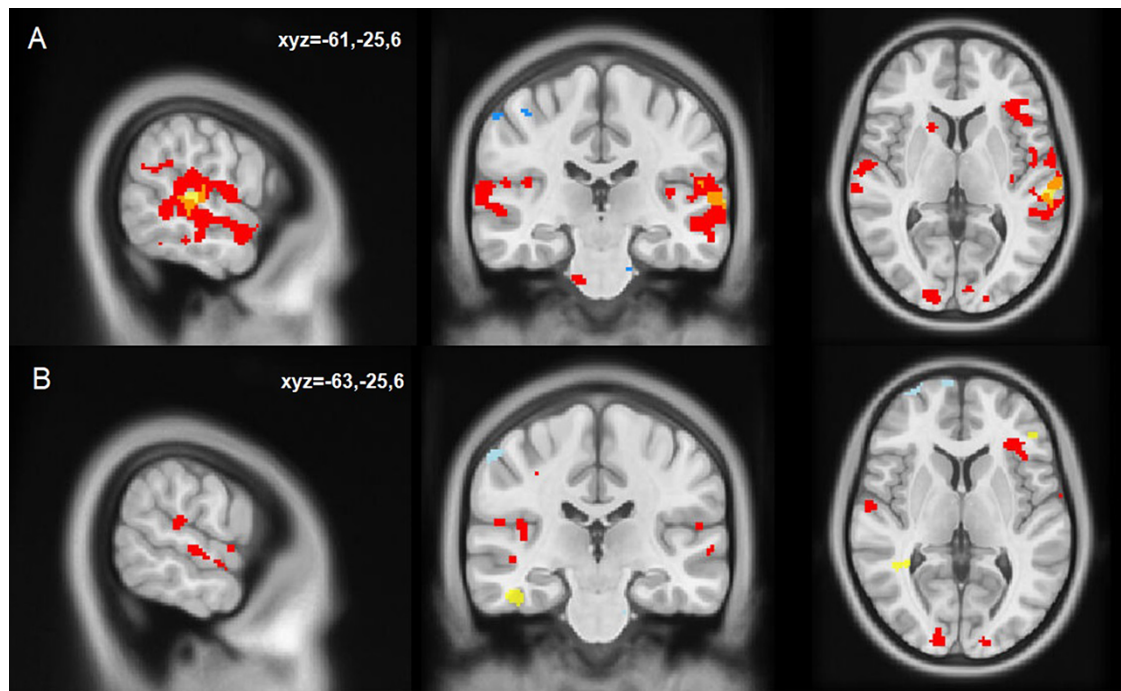


Fig. 3. Clusters where mothers' aggressive behavior was associated with their neural response to (A) aggressive or (B) neutral adolescent affective behavior. Positive associations are shown in red-orange-yellow (red for mothers' EPI aggressive behavior, yellow for mothers' PSI aggressive behavior and orange for both) and negative associations are presented in blue (dark blue for mothers' EPI aggressive behavior, light blue for mothers' PSI aggressive behavior and mid-blue for both). There were no significant clusters where mothers' behavior was associated with adolescent positive or dysphoric affect. Note that we did not test whether one parenting variable had a significantly different association with neural activation than another parenting variable.

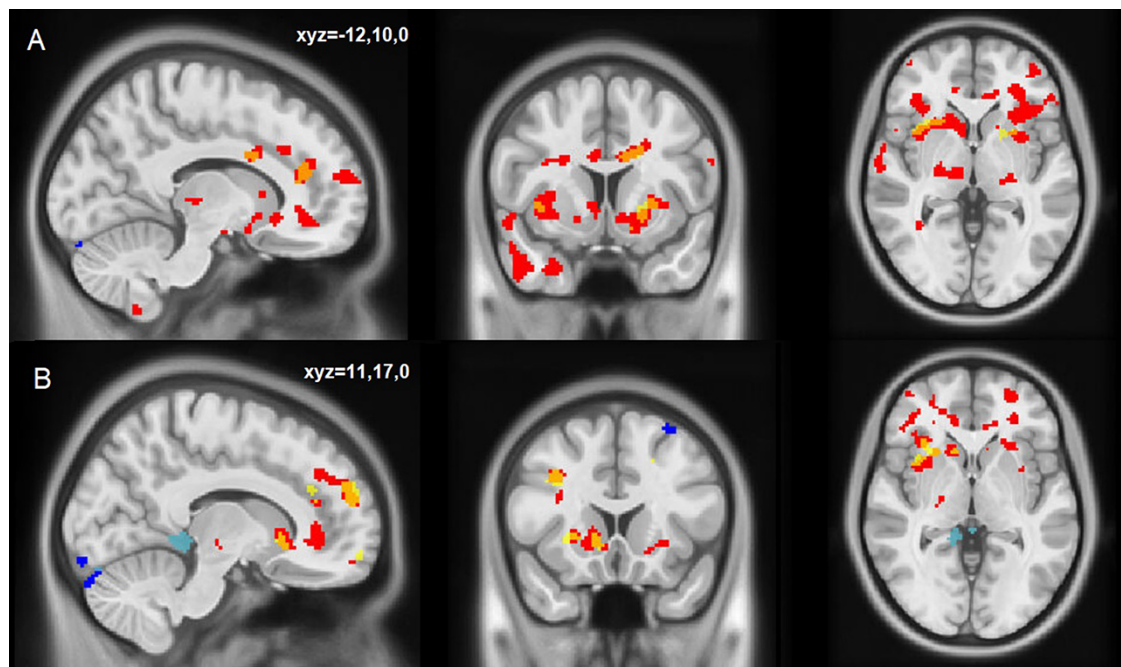


Fig. 4. Clusters where mothers' dysphoric behavior was associated with their neural response to (A) aggressive or (B) neutral adolescent affective behavior. Positive associations are shown in red-orange-yellow (red for mothers' EPI dysphoric behavior, yellow for mothers' PSI dysphoric behavior and orange for both) and negative associations are presented in blue (dark blue for mothers' EPI dysphoric behavior, light blue for mothers' PSI dysphoric behavior and mid-blue for both). There were no significant clusters where mothers' behavior was associated with adolescent positive or dysphoric affect. Note that we did not test whether one parenting variable had a significantly different association with neural activation than another parenting variable.

Table 3. Details of significant clusters for the interaction between group and affect

Peak F	No. of voxels	Location of peak (x, y, z)			Location(s), starting with the peak
31.44	2554	51.5	11.5	-0.5	^a Right IFG, STG, (anterior) insula
29.50	1949	-36.5	-16.5	67.5	^a Left precentral gyrus, posterior cingulate gyrus, precuneus, postcentral gyrus
26.55	4428	-60.5	9.5	5.5	^a Left IFG, STG, (anterior) insula
25.17	127	-22.5	49.5	3.5	^a Left superior frontal gyrus, middle frontal gyrus
24.46	139	-16.5	-64.5	-58.5	Left cerebellum (VIII)
22.87	332	-2.5	21.5	59.5	Left supplementary motor area, superior frontal gyrus
21.84	1230	-2.5	-6.5	59.5	Left and right supplementary motor area, medial frontal gyrus
21.41	234	13.5	-30.5	31.5	Right middle cingulate cortex
20.14	103	-42.5	25.5	-20.5	Left temporal pole
20.06	140	9.5	-26.5	-38.5	Brain stem
19.98	288	-4.5	37.5	-4.5	^a ACC, medial frontal gyrus
19.64	244	15.5	-62.5	-52.5	Right cerebellum (VIII)
19.39	95	21.5	-80.5	35.5	Right superior occipital gyrus
19.30	73	-28.5	-28.5	19.5	Left (posterior) insula
19.23	61	-6.5	-32.5	23.5	Left posterior cingulate gyrus
18.63	123	1.5	-32.5	71.5	Right and left paracentral lobule
18.3	205	5.5	13.5	1.5	^a Right and left caudate (ventral striatum)
18.29	86	37.5	-4.5	45.5	Right precentral gyrus
18.16	460	43.5	55.5	7.5	Right middle frontal gyrus, superior frontal gyrus
17.66	93	39.5	-64.5	-32.5	Right cerebellum (Crus 1)
17.60	116	-34.5	19.5	59.5	Left middle frontal gyrus
17.49	117	-2.5	-54.5	-4.5	Left cerebellum (IV-V)
17.21	96	33.5	-12.5	17.5	Right (posterior) insula
17.16	178	9.5	5.5	19.5	Right caudate (dorsal striatum)
16.77	85	-44.5	-44.5	-30.5	Left cerebellum (Crus 1)
16.31	211	47.5	21.5	-30.5	Right temporal pole
16.02	78	29.5	47.5	39.5	Right superior frontal gyrus
15.98	61	23.5	-52.5	57.5	Right superior parietal gyrus
15.48	55	-16.5	-18.5	-12.5	Left parahippocampal gyrus, hippocampus
15.46	106	29.5	-66.5	39.5	Right superior occipital gyrus
15.38	53	-32.5	-56.5	3.5	Left middle occipital gyrus
15.32	54	31.5	13.5	47.5	Right middle frontal gyrus
15.07	60	-54.5	-40.5	-26.5	Left inferior temporal gyrus
15.04	67	-60.5	-8.5	-32.5	Left inferior temporal gyrus
14.83	86	13.5	-56.5	-6.5	Right lingual gyrus, right cerebellum
14.58	169	-26.5	21.5	-14.5	Left IFG, superior orbital gyrus
14.40	340	3.5	13.5	35.5	Right and left middle cingulate cortex
14.00	107	-6.5	-40.5	-14.5	Left cerebellum (III)
13.32	112	-46.5	-46.5	49.5	Left inferior parietal lobule
12.69	53	19.5	-56.5	-18.5	Right cerebellum (VI)
12.31	86	-38.5	-60.5	-56.5	Left cerebellum (VIII)
12.17	63	35.5	7.5	-48.5	Right inferior temporal gyrus
11.54	148	-20.5	3.5	-22.5	Left parahippocampal gyrus
11.53	120	23.5	29.5	15.5	Right middle frontal gyrus and adjacent white matter
11.37	331	37.5	-14.5	43.5	Right precentral gyrus, postcentral gyrus
11.17	58	21.5	29.5	59.5	Right superior frontal gyrus
10.73	71	-38.5	37.5	23.5	Left middle frontal gyrus
10.63	48	69.5	-16.5	-18.5	Right middle temporal gyrus
10.43	55	19.5	-72.5	17.5	Right calcarine gyrus
8.44	76	11.5	-56.5	37.5	Right precuneus
8.33	62	21.5	-20.5	3.5	Right thalamus

Note: ^aindicates a cluster presented in Figure 2 and used for mediation analyses.

to regulate their emotions in our fMRI task, negative affective stimuli often elicit spontaneous emotion regulation, which involves the activation of ventrolateral and dorsolateral PFC (Etkin et al., 2015). Further, higher lateral PFC activation to aggressive or neutral affective behavior was associated with more positive affective behavior by the mother, indicating the adaptiveness of this neural response.

Self-relevance

The group differences in self-perception-related brain regions, such as the medial PFC and the precuneus/posterior cingulate, are interesting considering the stimuli in our fMRI task were much more self-relevant than those typically used. Mothers in the control group activated the mPFC/ACC and precuneus less in response to aggressive adolescent affect, compared to the

Table 4. Results of mediation analyses

Mediator	Outcome	Test of moderated mediation		Indirect effect(s)	SE	P
Left STG/insula/IFG	EPI aggressive	χ^2 diff(3) = 12.13, P = 0.007	agg	0.01	0.008	0.15
			dys	-0.004	0.004	0.31
			pos	0.005	0.003	0.17
			neu	-0.004	0.007	0.53
	PSI aggressive	χ^2 diff(3) = 0.67, P = 0.88		0.001	0.003	0.60
				0.005	0.003	0.13
	PSI dysphoric	χ^2 diff(3) = 0.29, P = 0.96		0.002	0.003	0.39
	EPI positive	χ^2 diff(3) = 33.21, P < 0.001	agg	-0.03	0.01	0.01
			dys	0.006	0.007	0.34
			pos	-0.009	0.007	0.18
neu			0.03	0.01	0.02	
PSI positive	χ^2 diff(3) = 1.71, P = 0.63		-0.001	0.002	0.40	
			0.001	0.001	0.48	
Right STG/insula/IFG	EPI aggressive	χ^2 diff(3) = 1.66, P = 0.65		-0.002	0.003	0.54
	PSI aggressive	χ^2 diff(3) = 0.32, P = 0.95		0.004	0.003	0.29
	EPI dysphoric	χ^2 diff(3) = 6.61, P = 0.09		0.002	0.004	0.64
	PSI dysphoric	χ^2 diff(3) = 0.39, P = 0.94		-0.02	0.01	0.12
	EPI positive	χ^2 diff(3) = 23.72, P < 0.001	agg	0.01	0.01	0.29
			pos	-0.01	0.009	0.18
		neu	0.03	0.01	0.04	
PSI positive	χ^2 diff(3) = 0.99, P = 0.80		-0.001	0.002	0.72	
			0.001	0.001	0.31	
Lateral PFC	EPI aggressive	χ^2 diff(3) = 0.28, P = 0.96		0.003	0.003	0.17
	PSI aggressive	χ^2 diff(3) = 0.85, P = 0.84		0.02	0.02	0.30
	EPI dysphoric	χ^2 diff(3) = 13.07, P = 0.004	agg	0.01	0.02	0.47
		dys	-0.001	0.02	0.97	
		pos	0.02	0.02	0.37	
		neu	0.002	0.002	0.36	
PSI dysphoric	χ^2 diff(3) = 0.10, P = 0.99		<0.001	0.002	0.88	
			-0.001	0.001	0.55	
			<0.001	<0.001	0.93	
			<0.001	0.002	0.85	
EPI positive	χ^2 diff(3) = 5.67, P = 0.13		0.005	0.005	0.32	
			0.003	0.004	0.46	
PSI positive	χ^2 diff(3) = 1.54, P = 0.67		0.003	0.004	0.46	
			0.003	0.004	0.46	
Ventral striatum	EPI aggressive	χ^2 diff(3) = 0.07, P = 0.99	agg	-0.02	0.01	0.18
			dys	0.001	0.002	0.75
	PSI aggressive	χ^2 diff(3) = 0.84, P = 0.84	pos	0.001	0.002	0.68
			neu	0.03	0.02	0.046
	EPI dysphoric	χ^2 diff(3) = 3.69, P = 0.30		<0.001	0.001	0.78
				0.001	0.001	0.24
PSI dysphoric	χ^2 diff(3) = 1.18, P = 0.76		<0.001	0.001	0.72	
			0.02	0.02	0.40	
EPI positive	χ^2 diff(3) = 29.45, P < 0.001	agg	-0.001	0.02	0.55	
		dys	-0.004	0.006	0.55	
		pos	0.009	0.01	0.40	
		neu	0.02	0.02	0.40	
PSI positive	χ^2 diff(3) = 1.02, P = 0.80	agg	-0.01	0.01	0.25	
		dys	-0.002	0.004	0.67	
Medial PFC/anterior cingulate	EPI aggressive	χ^2 diff(3) = 0.27, P = 0.97	pos	0.009	0.009	0.33
			neu	0.04	0.03	0.08
	PSI aggressive	χ^2 diff(3) = 0.92, P = 0.82		-0.001	0.002	0.41
				0.003	0.002	0.18
	EPI dysphoric	χ^2 diff(3) = 18.94, P < 0.001		<0.001	0.001	0.54
				-0.001	0.002	0.68
PSI dysphoric	χ^2 diff(3) = 17.65, P < 0.001		-0.002	0.003	0.55	
			-0.002	0.003	0.49	
EPI positive	χ^2 diff(3) = 2.50, P = 0.47	agg	-0.03	0.01	0.02	
		dys	-0.004	0.006	0.50	
PSI positive	χ^2 diff(3) = 5.45, P = 0.14	pos	0.002	0.006	0.77	
		neu	0.03	0.01	0.01	
Precuneus/precentral gyrus	EPI aggressive	χ^2 diff(3) = 1.26, P = 0.74		-0.002	0.003	0.49
				-0.002	0.003	0.49
	PSI aggressive	χ^2 diff(3) = 0.98, P = 0.81		-0.002	0.003	0.49
				-0.002	0.003	0.49
	EPI dysphoric	χ^2 diff(3) = 2.40, P = 0.49		-0.002	0.003	0.49
				-0.002	0.003	0.49
	PSI dysphoric	χ^2 diff(3) = 0.83, P = 0.84		-0.002	0.003	0.49
				-0.002	0.003	0.49
EPI positive	χ^2 diff(3) = 24.77, P < 0.001	agg	-0.03	0.01	0.02	
		dys	-0.004	0.006	0.50	
PSI positive	χ^2 diff(3) = 1.93, P = 0.59	pos	0.002	0.006	0.77	
		neu	0.03	0.01	0.01	

Note: The independent variable was always 'group'. Alpha was set at 0.004 to correct for multiple comparisons, see the "Methods" section for details. agg = aggressive, dys = dysphoric, pos = positive and neu = neutral affective behavior.

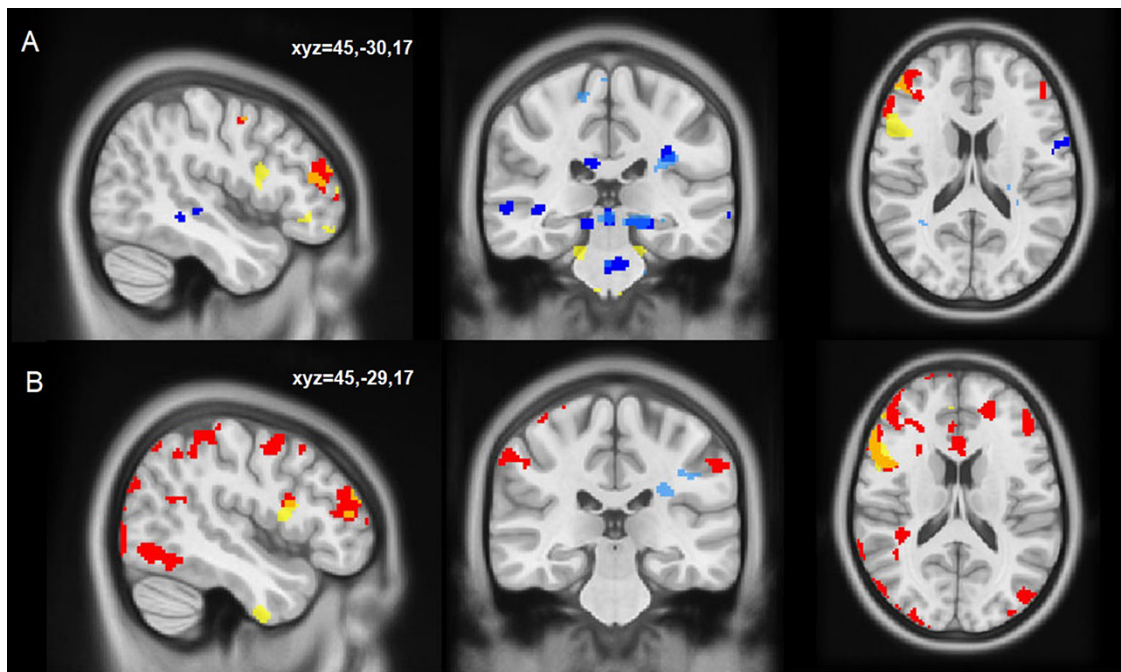


Fig. 5. Clusters where mothers' positive behavior was associated with their neural response to (A) aggressive or (B) neutral adolescent affective behavior. Positive associations are shown in red–orange–yellow (red for mothers' EPI positive behavior, yellow for mothers' PSI positive behavior and orange for both) and negative associations are presented in blue (dark blue for mothers' EPI positive behavior, light blue for mothers' PSI positive behavior and mid-blue for both). There were no significant clusters where mothers' behavior was associated with adolescent positive or dysphoric affect. Note that we did not test whether one parenting variable had a significantly different association with neural activation than another parenting variable.

depression group and compared to happy and dysphoric affect. Lower mPFC/ACC response to aggressive adolescent affect was also related to less dysphoric behavior. The mPFC is thought to decode self-relevance, and thinking about yourself, as well as about what others think of you, elicits activation in the mPFC and PCC/precuneus (Pfeifer *et al.*, 2009; Lieberman *et al.*, 2019; Van der Cruijssen *et al.*, 2019). Although speculative, activation in these regions could indicate to what extent mothers are relating the adolescent's behavior to themselves and/or to their own actions.

Familiarity

Contrary to our hypotheses, group differences were not stronger for viewing one's own adolescent compared to the unfamiliar adolescent. Some previous studies have found associations between depression and activation in limbic or midline regions that were specific to viewing one's own child (Laurent and Ablow, 2013; Morgan *et al.*, 2015), although others did not (Barrett *et al.*, 2012; Wonch *et al.*, 2016). However, all of these studies had small samples (total *N* ranging from 19 to 45), some used lenient fMRI thresholds and three out of the four focused on infant children. The current findings suggest that alterations in the neural processing of affective signals in depression might be general and not specific to the interaction partner. We should note that our findings do not imply that there are no unique patterns of neural activation when viewing and hearing one's own child compared to another child. That question was not an aim of this study and can better be answered with a multivariate pattern analysis.

Mediation effects

Finally, our exploratory analyses of mediational pathways from depression status to parent affective behavior through altered

neural processing in six clusters did not show significant mediation. While these analyses did not support the hypothesis that neural processes mediate the effect of depression on parenting behavior, we also note that this might be the result of low power since the number of tested models required us to apply a low alpha and some of the indirect paths were significant before multiple comparisons correction. This was, for example, the case for depression predicting lower parent positive behavior through stronger STG/insula/IFG and precuneus/PCC response to aggressive adolescent affect. We therefore recommend future, better powered studies to examine these mediational pathways.

Strengths and limitations

A clear strength of the present study is the use of personally relevant affective stimuli and the use of video clips with audio rather than static photos. This makes the stimuli more ecologically valid and elicits activation in a wider network of regions involved in processing affective stimuli, such as the parts of the auditory cortex and (posterior) superior temporal sulcus, which processes biological motion. This is in contrast to the most common emotion processing fMRI tasks, which use affective faces and thus heavily lean on visual information processing. In addition, we measured parenting behavior through observation, leading to more objective indices of (affective) parenting behavior than self-report.

Our findings also have to be considered in light of several limitations. First, the trade-off of having naturalistic fMRI stimuli from the interaction tasks is reduced experimental control over the stimuli. However, the findings were similar across the stimuli of the familiar and unfamiliar adolescent (the latter of which was the same for both groups), so it is unlikely that our main

findings of group differences or associations with parenting variables are driven by variation in the stimuli. In addition, although we used a well-validated observational system (Hops et al., 1995; Sheeber et al., 2007; Schwartz et al., 2014) and the observational approach to measuring parenting behavior has many strengths, the observed behavior can still be influenced by transient factors such as mood, recent stressors or reactivity to the lab context. As such, technologies that enable the examination of parenting behavior in real-world settings would provide important information about whether these findings generalize to the everyday interactions of families (Nelson and Allen, 2018). Third, we had to exclude a substantial number of participants because of a lack of appropriate affective stimuli that could be included in the fMRI task. This might have biased the final sample to mothers of adolescents who showed more emotion in the interaction tasks. However, participants with and without fMRI data did not differ on demographic variables or any of the parenting behaviors. Fourth, the interaction tasks to measure parent behavior took place before the fMRI scan, even though we use parent behavior as an outcome in the mediation models. This was done for practical reasons, since creating a naturalistic discussion between parent and adolescent during scanning is not practically feasible. The setup of the mediation models thus requires the assumption that the neural processing when the mothers observed adolescents' behavior in the scanner is similar to their neural processing during the interaction tasks. Further, 45% of the depression group used a form of antidepressants. Little is known about how SSRIs or other antidepressants impact the hemodynamic response and if this would vary by task condition (Harris and Reynell, 2017), but ROI activation does not appear to vary much by antidepressant use within the depression group (see Supplementary Figure S2). We purposefully did not exclude participants on medications because this would make the sample less representative of the population of mothers with depression. Finally, the current study is cross-sectional in nature. Longitudinal data would have provided a stronger foundation for the mediational analyses; thus, we recommend our mediational analyses to be repeated in future well-powered, longitudinal studies.

Conclusion

In the current study, we demonstrated that depression in mothers is associated with altered neural processing of both their own adolescent's affective behavior and that of unfamiliar adolescents. The patterns of strengthened limbic, striatal and sensory processing align with the theory of a depression-related negativity bias and the reduced lateral prefrontal activation with that of impaired regulatory function. These same patterns of neural activation were associated with more negative and less positive behavior during parent-child interactions, although the null findings using mediation analysis should temper the conclusion that neural processes mediate the relationship between depression and parenting behavior. These findings highlight the importance of processing of affective information to mothers' behavior during interactions with their adolescent, which are thought to be a central method through which parents influence their child's well-being in adolescence. If findings hold after replication, they could suggest approaches that recruit the lateral PFC (e.g. reappraisal) or target salience and sensory processing (e.g. attention modification) to improve parent-adolescent interactions. This study was one of the first to use personally relevant video stimuli, and this illustrated the relevance of a wider network of regions in affective processing; thus we recommend future studies to also

use dynamic video stimuli, which are more ecologically valid than static pictures. Future research should also examine the extent to which changes in the neural processing of affective signals, over time or by intervention, are related to changes in parent behavior during interactions with their adolescent. This would provide stronger indications of causal associations and could eventually lead to interventions that minimize the impact of depression on parenting behavior among parents of adolescents, which in turn could play a role in preventing the intergenerational transmission of psychopathology.

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Conflict of interest

All authors reported no biomedical financial interests or potential conflicts of interest. N.B.A. is a cofounder of, and shareholder in, Ksana Health Inc. Ksana Health products and intellectual property were neither used in, nor directly relevant to, the current study.

Supplementary data

Supplementary data is available at SCAN online.

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