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Associations between maternal negative affect and adolescent’s neural response to peer evaluation

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A B S T R A C T

Parenting is often implicated as a potential source of individual differences in youths' emotional information processing. The present study examined whether parental affect is related to an important aspect of adolescent emotional development, response to peer evaluation. Specifically, we examined relations between maternal negative affect, observed during parent–adolescent discussion of an adolescent-nominated concern with which s/he wants parental support, and adolescent neural responses to peer evaluation in 40 emotion-ally healthy and depressed adolescents. We focused on a network of ventral brain regions involved in affective processing of social information: the amygdala, anterior insula, nucleus accumbens, and subgenual anterior cingulate, as well as the ventrolateral prefrontal cortex. Maternal negative affect was not associated with adolescent neural response to peer rejection. However, longer durations of maternal negative affect were associated with decreased responsivity to peer acceptance in the amygdala, left anterior insula, subgenual anterior cingulate, and left nucleus accumbens. These findings provide some of the first evidence that maternal negative affect is associated with adolescents’ neural processing of social rewards. Findings also suggest that maternal negative affect could contribute to alterations in affective processing, specifically, dampening the saliency and/or reward of peer interactions during adolescence.

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1. Introduction

There is emerging evidence that individual differences in neural responsivity to peer evaluation are associated with affective health and disorder (Pfeifer and Blakemore, 2012). Despite behavioral evidence linking parenting with youths’ affective responses to peers (Brown et al., 1993), little is known about the extent to which parenting is associated with individual differences in adolescents’ neural response to a particularly salient type of emotional information, social evaluation by peers (Nelson et al., 2005; Somerville, 2013). The present study examines relations between one aspect of parenting, maternal negative affect, and neural response to peer rejection and acceptance in emotionally-healthy and depressed adolescents.

1.1. Parental influences on adolescent socioemotional development

Although peers play an increasingly important role during adolescence, parents continue to have a strong

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influence on adolescents’ socioemotional development (Stocker et al., 2007). Research has consistently revealed associations between high levels of maternal affect and adolescent depressive symptoms (e.g., Schwartz et al., 2012), particularly when mothers express negative affect during discussions that typically elicit parental positive affect, support, or empathy (Dietz et al., 2008; McMakin et al., 2011; Schwartz et al., 2011). As such, examinations of maternal negative affect in typically positive or supportive contexts may be particularly informative in elucidating the effects of parental affect on adolescent social and emotional development.

The mechanisms mediating links between parental affect and adolescents’ socioemotional adjustment are still being established; however, theories suggest that high levels of maternal negative affect could alter adolescents’ affective responses to peers by increasing their sensitivity to negative affect, diminishing their capacity to experience or maintain positive affect, transmitting maladaptive regulatory behaviors, and altering their experience and/or expectations of social interactions (Eisenberg et al., 1998; Morris et al., 2007; Sheeber et al., 2001). Alterations in emotional reactivity and regulation could thereby sensitize adolescents to social evaluation, impair their ability to regulate distress stemming from negative feedback, or dampen the rewarding aspects of social interactions.

Indeed, behavioral studies have shown that parenting practices are associated with youths’ ability to modulate their affective reactions to peer rejection (Yeung and Leadbeater, 2010), confidence in their ability to form and maintain friendships, and perceptions of intimacy and social support within relationships (Ladd and Pettit, 2002; Parker et al., 2006). Furthermore, parental affect during parent–child interactions provides youth with important information about the consequences of their own affective behaviors (Eisenberg et al., 1998). Thus, adolescents who frequently experience parental negative affect may experience, or come to expect, less reward from social interactions. It therefore stands to reason that maternal negative affect during parent–adolescent interaction may influence adolescents’ affective responses to peer evaluation by influencing activity in neural regions that underlie affective and social information processing.

Despite immense interest (Belsky and de Haan, 2011), relatively few studies have attempted to link normative variations in parental affect with functional differences in youth’s neural response to affective information. A number of studies have, however, examined associations between extreme parental negative affect (e.g., child maltreatment) and children’s responses to affective information. These studies provide compelling evidence for the influence of parental negativity on development of affective neural systems (Glaser, 2000) and for alterations in the processing of negative affect (Kauffman and Charney, 2001; Pollak and Tolley-Schell, 2003) and reward-related information (Dillon et al., 2009). However, given the importance of parents on adolescents’ emotional adjustment (Morris et al., 2007), it is essential to explore the impact of normative variations in parental negative affect on youth’s neural response to affectively charged social information (Belsky and de Haan, 2011).

A series of studies by Whittle and colleagues provide preliminary evidence that normative variations in parental affect can influence the development of neural structures that are central for affective and social information processing, including the neural correlates of peer evaluation. Specifically, high parental negativity (e.g., aggressive behavior) during parent–child interactions was associated with smaller amygdala (Yap et al., 2008) and hippocampus (Whittle et al., 2011) volume whereas high parental positive affect was associated with larger orbitofrontal cortex volume (Whittle et al., 2009). Although suggestive of relations between parental negative affect and alterations in adolescents’ affective processing, research has yet to identify associations between normative variations in parental negativity and adolescents’ neural response to peer evaluation.

1.2. Neural correlates of peer evaluation processing

Given that developmental psychopathology models suggest that increased sensitivity to peer evaluation plays an important role in the rise of anxiety and depression during adolescence (Davey et al., 2008; Nelson et al., 2005; Somerville, 2013), researchers have focused on identifying the neural correlates of peer evaluation. Using virtual interaction paradigms such as the Cyberball (Eisenberger et al., 2003) and Chatroom Task (Guyer et al., 2008), studies consistently find that social exclusion/rejection activates a ventral affective network that includes the amygdala, and anterior insula, as well as the ventro-lateral prefrontal cortex (VLPFC) which is posited to subserve regulation of distress associated with social exclusion/rejection (Guyer et al., 2008; Masten et al., 2009; Sebastian et al., 2011). Whereas social exclusion in adults appears to activate a dorsal portion of the ACC (Eisenberger et al., 2003), extant studies with adolescents often implicate the sgACC in the processing of exclusion (Masten et al., 2009; Sebastian et al., 2010). Fewer studies have examined responsivity to social acceptance but research has begun to show that acceptance activates reward-related brain regions, particularly the NAcc (Davey et al., 2010; Gunther Moor et al., 2010; Guyer et al., 2012) and amygdala (Davey et al., 2011).

There is also emerging evidence for group differences in depressed vs. non-depressed adolescents’ in neural responses to peer evaluation. For example, in an earlier analyses the Chatroom Interact task which included a sample that overlaps with the present study, we found that depressed youth showed increased activation to rejection (and not acceptance) relative to controls in the bilateral amygdala, sgACC, left anterior insula, and left NAcc (Silk et al., in press). The present study presents only new analyses focused individual differences in neural responses to peer evaluation. Details about ROIs that were activated by peer acceptance and evaluation can also be found in that study.

1.3. The present study

To examine relations between parental affect and adolescents’ neural responses to peer evaluation, we used behavioral observations of maternal negative affect from
a parent–adolescent interaction task that was designed to elicit parental support and functional imaging data on adolescent’ responses to peer evaluation during a new virtual peer interaction paradigm (Chatroom Interact, Silk et al., 2012). Unlike an earlier Chatroom Task (Guyer et al., 2008), in which adolescents received rigged feedback from virtual peers about whether they were chosen to participate in an online chat, in the Chatroom Interact Task, participants engage in a “live” online interaction with virtual peers during which they are repeatedly selected (accepted) and not selected (rejected) to discuss topics that are of interest to teens. The task was designed to increase ecological validity and participant engagement with virtual peers and appears to engage a similar set of ventral affective processing neural regions. We hypothesized that youth who experienced more negative maternal affect would exhibit heightened neural responsivity to peer rejection in brain regions implicated in the processing of affectively-salient information (i.e., amygdala, sgACC, and anterior insula) as well as decreased activation of VLPFC. With respect to peer acceptance, we hypothesized that youth who experienced more maternal negativity would exhibit blunted neural response in brain regions implicated in the processing of reward (i.e., NAcc) and affective valuation (i.e., amygdala, sgACC, and anterior insula).

2. Method

2.1. Participants and recruitment

Participants were 40 adolescents (25 female, ages 11–17, M [SD]=14.75 [1.63]) who completed parent–adolescent interaction and neuroimaging tasks from a larger study examining the socioemotional development of emotionally healthy and depressed youth. 80% of adolescents and mothers (N=32) self-identified as Caucasian, the other 20% self-identified as African-American. Of these 40 adolescents, 26 were healthy adolescents with no psychiatric history and 14 were diagnosed with a current primary DSM-IV diagnosis of Major Depressive Disorder (MDD). Because the groups were matched on gender, and MDD is more common among females than males (Kessler et al., 2003), both groups had a higher proportion of females than males. Seven of the depressed adolescents were also diagnosed with secondary DSM-IV anxiety (2 with GAD, 1 with GAD, Specific Phobia, and Panic, 1 with agoraphobia and Specific Phobia, 1 with agoraphobia and Panic, and 1 with Specific Phobia) or behavioral (Oppositional Defiant) disorders (N=1). Among mothers with emotionally-healthy adolescents, 46% of mothers received partial college training, 33% had a college degree and 21% had a graduate degree. Among mothers with depressed adolescents, 14% completed high school, 36% received partial college training, 36% had a college degree and 14% had a graduate degree. The emotionally healthy and depressed samples did not differ in child age, gender, and race or maternal race and education level (all ps > .50). However, consistent with previous research (Hammen and Brennan, 2003), mothers of MDD adolescents reported higher levels of depression symptoms (M [SD]= 7.57 [6.02]) on the Quick Inventory of Depressive Symptomatology (QIDS, Rush et al., 2003) than mothers of CON adolescents (M [SD]=2.13 [1.79]), F(1,40) = 16.64, p < .001.

All participants were recruited from pediatrician’s offices and community advertisements; MDD adolescents were also referred from University and community mental health clinics. DSM-IV diagnoses were assessed using the Schedule for Affective Disorders and Schizophrenia in School-Age Children—Present and Lifetime version (Kaufman et al., 1997). Participants were excluded if they had a current diagnosis of obsessive-compulsive, post-traumatic stress, conduct, substance use, or attention-deficit hyperactivity disorders or a lifetime diagnosis of schizophrenia, bipolar, psychotic depression, schizoaffective disorder, or pervasive developmental disorders. Participants were also excluded if they were taking psychoactive medications other than SSRI’s (N=1) or had metal braces or other metal objects in their body.

2.2. Procedures

On the first of two laboratory visits, mother–adolescent dyads completed behavioral interaction tasks (i.e., parent–adolescent discussions). Clinical assessments were also completed at this visit. Approximately two weeks later, adolescents completed the Chatroom Interact task while in the scanner during the second visit.

2.2.1. Behavioral observation of maternal negativity

Maternal negative affect was assessed during an 8-min supportive discussion task (see Gilliom et al., 2002), where dyads were asked to discuss a topic that the adolescent nominated as a problem that they would like help solving. Specifically, the dyad was told: “We are really interested in how children talk to their parents when they have problems and when they need support. . . . we would like you two to talk about a problem(s) [child] is having, and would like some support with.” Although this task was designed primarily to elicit supportive parenting behaviors, it can also elicit a range of negative affect in families (e.g., parental expressions of frustration, irritation, and disappointment in adolescents for their own role in generating the problem, or parental dissatisfaction with the steps adolescents have taken to resolve the problem). Maternal affect during this discussion was coded from videotaped observations on a second-by-second basis using a version of the Specific Affect coding system (SPAFF) that was adapted for parent–adolescent interactions (Gottman et al., 1996).

This adapted system has been showed to capture meaningful differences in parental affect that predict children’s emotional health and disorder (e.g., Granic et al., 2007; Snyder et al., 2003). Each SPAFF affect code is based on a combination of facial expressions, gestures, posture, voice tone, and speech rate that capture the overall affective tone of each second of maternal behavior. Because laboratory-based parent–child interaction tasks typically elicit low rates of parental negative affect (e.g., Capaldi et al., 1994; Hollenstein et al., 2004; Snyder et al., 2003), particularly among parents who were recruited to assess normative variations in maternal negative affect, we also expected relatively low rates of maternal negative affect. We therefore
used a standard approach for measuring parental negative affect that has been commonly utilized in developmental research (e.g., McMakin et al., 2011; Snyder et al., 2003). Specifically, we created a composite variable to quantify the amount of maternal negative affect observed by summing the duration of contempt, anger, fear/anger, sad/withdrawn, and whine/complain codes. SPAFF coders were extensively trained and blind to the clinical characteristics of participants. Approximately 25% of the interactions were coded by a master coder to provide an estimate of observer agreement and weekly calibration meetings were held to avoid coder drift. Reliability was calculated continuously over the coding period. Kappa was .92 for duration-based codes of affective behavior, indicating a high inter-rater reliability.

2.2.2. Chatroom interactive task

The Chatroom Interactive task was designed to investigate reactions to “live” social acceptance and rejection from virtual peers in an online setting (Silk et al., 2012). Following completion of parent–adolescent interaction tasks during the first laboratory visit, participants were asked to view photos and biographical profiles of other age-matched adolescents. Adolescents were told they would have the opportunity to interact online with several of these youth as part of an internet communication study. Participants provided their own photograph and profile and were asked to choose the top 5 males and top 5 females who they would be interested in interacting with online at their next visit. Approximately two weeks following this first visit, participants returned to the laboratory where they were told that they had been matched to play a “chat game” with four selected peers (2 males, 2 females) via remote connection during neuroimaging. As shown in Fig. 1, pictures of the peers and participant were then projected on the screen as the participant and fictitious peers took turns selecting who they would rather talk to about common teen interests (e.g., music, TV, friends).

The task proceeded in 5 blocks, each comprised of 15 acceptance or rejection trials (total run time 16.7 min). As described in Silk et al. (2012), all participants experienced two accept blocks in which they were chosen 2/3 of the time and two reject blocks in which they were rejected 2/3 of the time. In block 1, the participant made choices among the two virtual peers. Analyses focus on blocks 2–5, in which the participant was chosen (i.e., accepted)/not chosen (i.e., rejected) by fictitious peers. The order of accept and reject blocks and trials was randomized.

Each block began with an instruction about who would be making choices for that block (agent) and every trial began with the question “Who would you rather talk to with about [topic]?” (‘choice phase’) for 3.34 s. Feedback was then provided about which person was chosen (the participant or a fictitious peer) for 10.02 s (‘feedback phase’). The photograph of the not chosen/‘rejected’ person was superimposed with an “X” and the photograph of the chosen/‘accepted’ person was highlighted around the border. To maintain task engagement, in all trials in which the participant was not choosing an interaction partner (i.e., blocks 2–5), s/he was asked to press a button to indicate whether the person on the left or the right was chosen.

Stimuli were presented using E-prime 1.0 (Psychology Software Tools, Pittsburgh, PA).

2.3. BOLD fMRI acquisition, preprocessing, and analysis

2.3.1. Imaging acquisition

Images were acquired on a 3T Trio scanner (Siemens, Erlangen, Germany). Thirty-two 3.2-mm slices were acquired parallel to the AC–PC line using a reverse-weighted echo planar (EPI) pulse sequence (T2*-weighted imaged depicting BOLD signal; TR = 1670 ms, TE = 29 ms, FOV = 205 mm, flip angle = 75°). Each image was acquired in 1.67 s, allowing 8 scans per trial. High-resolution T1-weighted MPRAGE images (1 mm, axial) were also collected for use in cross-registration.

2.3.2. fMRI data preprocessing

fMRI analyses were conducted using routines from multiple packages including NeuroImaging Software (NIS) (Fissell et al., 2003), Analysis of Functional Neuroimaging (AFNI) software (Cox, 1996), and custom Matlab routines. Functional imaging data were corrected for motion using the first image as a reference (AFNI 3dVolReg). Four participants (3 CON, 1 MDD) were excluded due to excessive head movement (over 30% of scans with greater than ±5 mm, or ±5° movement from a reference image and ±1 mm and ±1° incremental (scan-to-scan) movement, >30% of scans with greater than >1 mm incremental movement), resulting in the present sample of 41 adolescents with usable fMRI data (1 participant was missing parent–child interaction data). Linear and quadratic trends within runs were then regressed out of fMRI time series to eliminate effects of scanner drift, unrelated to brain activity (NIS Correct; Fissell et al., 2003). This procedure also reduces the impact of outliers by Winsorizing or clipping outliers over 1.5 interquartile range (IQR) from the 25th or 75th percentiles to the nearest value within this range. Data were temporally smoothed using a 7-point Gaussian filter and converted to % change from the median of the run for each voxel. Data were co-registered to the Colin-27 Montreal Neurological Institute (MNI) template using the Automated Image Registration package’s (Air; Woods et al., 1993) 32 parameter non-linear automated warping algorithm and spatially smoothed using a 6 mm full width at half maximum (FWHM) kernel.

2.4. Plan of analyses

We conducted region of interest (ROI) analyses on a priori regions specified using AFNI’s Talairach atlas including the sgACC (centroid: x, y, z = 0, 11, −7), bilateral anterior insula (x, y, z = ±38, 10, 5), NAcc (x, y, z = ±12, 9, −8), and vIPFC (x, y, z = ±37, 25, −8). Because AFNI’s Talairach atlas ROI incorporates a smaller volume than the anatomical boundaries of the amygdala, this ROI was anatomically defined by hand tracing on the MNI Colin 27 brain (x, y, z = ±23, -4, -17) (see Siegle et al., 2007). Regions were identified based on prior research on the neural substrates of adolescents’ affective responses to peer rejection and acceptance during Chatroom and Chatroom Interact tasks (see Guyer et al., 2012; Silk et al., in press).
BOLD activity in ROIs during peer acceptance and rejection trials was extracted for hypothesis testing. The long duration of each trial enabled slow event related model-free analysis (i.e., examining the empirical shape of the hemodynamic response using scan-within-trial as a repeated measure), eliminating the need for event deconvolution. As is standard for slow-event-related analyses, BOLD “reactivity” to each stimulus was computed as the average activity over voxels within an ROI and across TRs (8 TRs within a single 13.36 second trial) after subtracting the BOLD signal during a pre-stimulus baseline (1 TR). This technique separates stimulus-related activity from activity lingering from previous trials (e.g., Birn et al., 1999; Gallivan et al., 2009; Harris et al., 2007; MacDonald et al., 2000; Siegle et al., 2007). BOLD reactivity within each ROI was then averaged separately for acceptance and rejection trials. Specifically, BOLD reactivity for each trial was computed by averaging BOLD activity minus the pre-stimulus baseline within an ROI for a trial (8 scans within a single 13.36 s trial).

Preliminary analysis suggested that the duration of maternal negative affect was skewed, thus this variable was dichotomized to represent the presence vs. absence of maternal negative affect. Study hypotheses regarding associations between maternal negative affect and neural response to peer evaluation were tested using a series of planned linear regressions in IBM SPSS.20 (SPSS, Chicago, IL). After controlling for two covariates, adolescent depression and maternal depressive symptoms, maternal negative affect was entered as an explanatory variable for adolescent neural activity to peer rejection and acceptance in each of the ROIs.

Because family-wise error corrections are often overly conservative (see Troendle, 2000; Storey, 2003), we used a widely-validated false discovery rate procedure (B–H; Benjamini and Hochberg, 1995; Benjamini and Yekutieli, 2001), to adjust the p-value for multiple comparisons. This method for reducing the possibility of Type I error utilizes a sequential approach for controlling false discovery rates. Finally, as shown in Supplementary Materials, regression analyses using AFNI’s 3dRegana were used to identify the voxels within our ROIs that showed a main effect of maternal negative affect on adolescents’ response to acceptance. To control for multiple comparisons, tests were subjected to small volume correction (SVC) (corrected p < .05), using AFNI’s Alphasim program.

3. Results

3.1. Preliminary analyses

Preliminary analyses suggested that adolescent age and gender were not significantly correlated with duration of maternal negative affect and were therefore not included in analyses. Results did not change when regressions included a continuous measure of maternal negative affect (duration) or adolescent depression (severity of depressive symptoms on the Mood and Feelings Questionnaire). Indeed, there were no group differences in either the (a) dichotomized maternal affect variable indexing presence of any negative affect (50% of mothers of emotionally-healthy adolescents and 73% of mothers of MDD adolescents exhibited no negative affect, $\chi^2(1) = 2.13$, $p > .05$) or (b) continuous maternal affect variable indexing the duration of time that mothers exhibited negative affect ($M \ [SD]_{mothers \ of \ MDD \ adolescents} = 4.04 \ s \ [9.37 \ s]$, range = 0–45.06 s; $M \ [SD]_{mothers \ of \ non-MDD \ adolescents} = 1.65 \ s \ [3.53 \ s]$, range = 0–12.30 s, $t(39) = -.95 \ p > .05$). In addition, there were no significant group differences in severity of depressive symptoms between the mothers who...
did and did not \((M [SD]_{NegAffectPresent} = 3.12 [3.52]; M [SD]_{NegAffectAbsent} = 5.10 [5.45])\) exhibit any negative affect during the supportive discussion task, \(F(1.39) = 1.66, p > .05, d = 1.22\).

To validate observations of maternal negative affect during the supportive behavior task, we examined associations between duration of expressed negative affect and adolescent report of maternal acceptance/rejection on the acceptance/rejection scale of a well-validated questionnaire measure of parenting behavior, the Child Report of Parental Behavior Inventory (CRBPI; Margolies and Weintraub, 1977). Bivariate associations revealed that longer durations of maternal negative affect were marginally correlated with lower levels of maternal acceptance/higher levels of maternal rejection \((r = -.24, p = .06)\). Furthermore, among mothers who expressed negative affect during the interaction task, longer durations of negative affect were significantly correlated with lower levels of acceptance/higher levels of rejection \((r = -.44, p = .038)\).

### 3.2. Maternal negativity and behavioral response to peer evaluation

Repeated measures analyses of variance (MANOVA) indicated that adolescents were significantly slower to respond for rejection \((M [SD] = 954.59 s [228.58 s])\) trials than for acceptance \((M [SD] = 896.70 [215.53 s], t = -3.77, p < .05, d = 1.21)\). Maternal negative affect was related to how quickly adolescents pressed a button to indicate whether the person on the right or left was chosen. Specifically, including maternal depressive symptoms and adolescent depressive status as covariates, higher levels of maternal negative affect were associated with slower reaction times following acceptance \((F = 5.53, R^2 = .28, p < .001, \beta = .38, t = 2.55, p < .05)\) and rejection \((F = 6.89, R^2 = .34, p < .001, \beta = .47, t = 3.28, p < .01)\). Furthermore, post hoc t-tests suggested that adolescents who experienced any maternal negative affect during the supportive discussion task were slower to respond than adolescents who experienced no maternal negative affect following both acceptance \((M [SD]_{MatNegAffectPresent} = 862.04 ms [171.40 ms], M [SD]_{MatNegAffectAbsent} = 966.96 ms [227.06 ms], t = -2.30, p < .05)\) and rejection during the Chatroom Interaction task \((M [SD]_{MatNegAffectPresent} = 862.04 ms [171.04 ms], M [SD]_{MatNegAffectAbsent} = 1052.86 ms [217.81 ms], t = -3.16, p < .05, d = 1.01)\).

### 3.3. Maternal negativity and neural response to peer evaluation

Contrary to hypotheses, maternal negative affect was not related to adolescent neural response to peer rejection in any ROI, specifically: the amygdala (right: \(F = 1.24, \beta = -.25, t = -1.50, p > .05; F = 2.00, \beta = -.32, t = -1.91, p > .05\), anterior insula (right: \(F = 1.40, \beta = -.28, t = -1.62, p > .05\); left: \(F = 2.64, \beta = -.36, t = -2.20, p > .05\), NAcc (right: \(F = 3.1, \beta = -.14, t = -1.78, p > .05\); left: \(F = 1.71, \beta = -.14, t = -1.84, p > .05\), sgACC (right: \(F = 2.04, \beta = -.29, t = -1.72, p > .05\), and VLPFC (right: \(F = 3.6, \beta = -.15, t = -1.85, p > .05\); left: \(F = 6.2, \beta = -.16, t = -1.95, p > .05\). However, as hypothesized, maternal negative affect predicted variance in the neural response to acceptance in all four of the ROIs for peer acceptance (see Table 1). In addition to maternal negative affect, maternal depressive symptoms accounted for significant variance in adolescent neural response to acceptance only in the left amygdala \((\beta = -.47, t = -2.58, p > .05)\). Relations between maternal negative affect and adolescent neural responsivity to acceptance were significant even after controlling for maternal depressive symptoms. As shown in Figs. 2a–c, longer duration of maternal negative affect is associated with less activity in (a) bilateral amygdala, (b) left anterior insula, (c) left NAcc and (d) sgACC during acceptance trials. Maternal negative affect remained a significant predictor of variance in adolescents’ neural responses to acceptance in these regions following a false discovery rate procedure (Benjamini and Hochberg, 1995; Thissen et al., 2002, see Table 1). Finally, as reported in Supplementary Materials, results from small-volume corrected (SVC) regression analyses (accomplished using AFNI’s 3dRegana and Alphasim programs) were largely consistent with results from anatomical ROI analyses. The only difference is that maternal negative affect did not significantly predict activity in the sgACC in SVC analyses.

### 4. Discussion

A number of factors likely contribute to relations between alterations in parental and adolescent affect (e.g., common environmental stressors, inherited genetic risk, and direct modeling of maladaptive coping); however, the present study focuses on alterations in adolescents’ neural reactivity. By examining the association between maternal negative affect and the neural response to peer evaluation, this study provides some of the first evidence that maternal affect influences the neural processes that support adolescents’ affective responses to a social reward that is particularly salient during this developmental period (Somerville, 2013) – peer acceptance. Specifically, adolescents who experienced any maternal negative affective in a social context that should elicit supportive parenting showed less responsivity in a ventral network of brains regions involved in the generation and processing of affective (e.g., reward-related) and social stimuli, including the amygdala, anterior insula, NAcc, and sgACC. These relations held after controlling for maternal depressive symptoms.

Adolescence is a developmental period when youth typically find peer relationships to be especially rewarding and become highly motivated to build and maintain characterized by such bonds (Steinberg and Morris, 2001). It is also a developmental period that is characterized by significant neural plasticity, which is thought to be particularly relevant for some types of social-affective learning (Crone and Dahl, 2012). As such, even relatively low levels of maternal negative affect may alter typical social trajectories during adolescence, leading youth to experience or expect less reward from interactions with peers, to withdraw from social activities, or to experience anhedonia – in short, exhibiting the symptoms of depression. Thus, just as adolescents become increasingly attuned to social rewards from peers, negative affect from mothers could dampen the rewarding aspects of social interactions and thereby increase adolescents’ risk for depression.
Fig. 2. Associations between maternal negative affect and adolescent neural response to peer acceptance. Regression models suggest that maternal negative affect during the Supportive Discussion task is predictive of variability in adolescents’ responses to acceptance. Specifically, longer durations of maternal negative affect are associated with decreased responsivity in (a) bilateral amygdala, (b) bilateral anterior insula, and (c) left nucleus accumbens.
Table 1
Summary of regressions predicting neural responsivity to peer acceptance from maternal negative affect.

|                        | F     | Adj. R² | ΔF   | ΔR²  | Std. β | t     | Uncorrected p-value | Corrected p-value |
|------------------------|-------|---------|------|------|--------|-------|---------------------|--------------------|
| **Right amygdala**     |       |         |      |      |        |       |                     |                    |
| Model 1                | 6.55  | 0.12    |      | 0.38 | −2.66  | .012  | .028                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Model 2                | 2.33  | 0.09    | .34  | 0.02 | −1.75  | .050  | MNA × MDD           | .07                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| **Left amygdala**      |       |         |      |      |        |       |                     |                    |
| Step 1                 | 7.07  | 0.14    |      | −0.4 | −2.66  | .020  | .035                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Step 2                 | 3.55  | 0.16    | .07  | 1.67 | −1.66  | .13   |                     | .28                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| **Right anterior insula** |     |         |      |      |        |       |                     |                    |
| Step 1                 | 4.63  | 0.09    |      | −.33 | −2.15  | .157  | .183                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Step 2                 | 1.92  | 0.62    | 0.03 |       | −1.24  |       |                     | .20                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| **Left anterior insula** |      |         |      |      |        |       |                     |                    |
| Step 1                 | 16.04 | 0.28    |      | −.55 | −4.01  | .007  | .024                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Step 2                 | 5.54  | 0.50    | 0.02 |       | −2.99  |       |                     | .15                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| MNA × MDD              | −.15  | −0.90   |      |      |        |       |                     |                    |
| **Right nucleus accumbens** |      |         |      |      |        |       |                     |                    |
| Step 1                 | 0.75  | 0.00    |      | −.14 | −0.87  | .531  | .531                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Step 2                 | 0.57  | 0.66    | 0.04 |       | −0.34  |       |                     | .08                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| MNA × MDD              | −.08  | −0.41   |      |      |        |       |                     |                    |
| **Left nucleus accumbens** |      |         |      |      |        |       |                     |                    |
| Step 1                 | 10.19 | 0.19    |      | −.46 | −3.19  | .005  | .024                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Step 2                 | 4.80  | 1.87    | 0.07 |       | −1.94  |       |                     | .31                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| MNA × MDD              | −.31  | −1.86   |      |      |        |       |                     |                    |
| **Subgenual cingulate cortex** |      |         |      |      |        |       |                     |                    |
| Step 1                 | 7.81  | 0.15    |      | −.41 | −2.79  | .026  | .036                |                    |
| MNA                    |       |         |      |      |        |       |                     |                    |
| Step 2                 | 4.54  | 2.58    | .10  |       | −1.39  |       |                     | .38                |
| MDD                    |       |         |      |      |        |       |                     |                    |
| MNA × MDD              | −.38  | −2.26   |      |      |        |       |                     |                    |

Notes: Abbreviations: MDD, adolescent major depressive disorder status; MNA, duration of maternal negative affect; L, left; R, right. All predictors were centered. Maternal negative affect was skewed, thus we log transformed this variable before centering it. Results from step 1 of the models were interpreted unless analyses suggested that step 2 of the model significantly accounted for additional variance in neural responses (see results of model for sgACC activity). Post hoc, a false discovery rate procedure (Benjamini & Hochberg, 1995) was used to adjust the p-value for multiple comparisons in order to reduce the probability of Type I error.

¹ p < .10.
² p < .05.
³ p < .01.
⁴ p < .001.

Moreover, given evidence that parental behavior moderates the association between poor peer relations and emotional adjustment (Brown and Bakken, 2011), adolescents who do not receive adequate levels of parental support may struggle to cope with the affectively-charged and unstable quality of peer relationships characterize this developmental period (Cairns et al., 1995). Although further research is needed to specify the mechanism(s) by
which parental negative affect may disrupt adolescents’ experiences of social rewards, findings from the present study suggest that the presence of maternal negative affect during interactions when adolescents are seeking support may blunt the salience or reward of positive feedback from peers.

Our working hypothesis that maternal negative affect alters the neural processing of affectively-charged social information is consistent with behavioral and neuroanatomical research showing that children who have experienced extreme levels of parental negative affect (i.e., abuse) are particularly sensitive to mothers’ negative emotional expressions (Pollak and Tolley-Schell, 2003) and show diminished sensitivity to rewards (Dillon et al., 2009). Consistent with prior work linking decreased striatal responsivity with altered responses to monetary reward (Forbes et al., 2009) we found that adolescents who experienced maternal negative affect when seeking parental support showed decreased responsivity to a social reward in the NAcc as well as other regions that have been implicated in affective processing.

Although it is surprising that maternal negative affect was not associated with increased reactivity to peer rejection, it is important to note that we focused on normative variations in parental behavior. It is possible that the levels of maternal negative affect that were observed during the parent–adolescent interactions were not intense, frequent, or long enough to sensitize adolescents’ responses to negative emotional information as may happen in more severe, or pathological, experiences of parental negative affect (e.g., emotional neglect, child abuse). It is also possible that the types of negative affect and cognitions that adolescents experience during discussions with their mother are less generalizable to their experiences of peer rejection. Thus, in addition to examining social vs. non-social affective stimuli, it will be important to investigate potential variations in how adolescents process different types of social feedback (e.g., maternal vs. peer evaluation). Furthermore, given the possibilities of alternative hypotheses it will be important for future studies to continue to specify how these relations could vary as a function of maternal parenting behavior. For instance, one could expect that adolescents who experience relatively high levels of parental negative affect would be more surprised, and therefore show greater responsivity, positive feedback. As research in this area grows, it will be important to increase the specificity with which forms of maternal negative affect (e.g., presence of maternal negative affect in conflictual parent–child discussions, different types of “negative” parental behaviors such as criticism or intrusiveness) is associated with individual differences in adolescents’ responses to peer evaluation.

It is, however, notable for parents to express negative affect during a context in which they are explicitly asked to provide support for their child. It is possible that expressions of negative affect reflect sensitive behaviors, such as sympathy. However, we found that duration of maternal negative was inversely associated with adolescents’ perception of maternal acceptance. Researchers have suggested that relatively high levels of context-inappropriate maternal negativity may represent a parenting behavior that is reflective of a relatively adverse family environment, for example, low parental acceptance/high parental rejection (McMakin et al., 2011; Schwartz et al., 2011). As such, the expression of maternal negative affect during this type of supportive parent–adolescent interaction may be indicative of low emotional support, which may, in turn, adversely influence adolescents’ ability to form rewarding friendships (Ladd and Pettit, 2002). This hypothesis is consistent with results showing an inverse relationship between maternal negative affect and adolescents’ ratings of maternal acceptance. In sum, even at low levels, these kinds of experiences of parental negative affect may socialize an adolescent to view, or even anticipate, that social interactions will have a negative affective quality (e.g., less accepting) and, perhaps, be experienced as less rewarding.

We also found that decreased responsivity in the bilateral amygdala and anterior insula is associated with maternal negative affect. Although these regions have been implicated in the processing of negative affect, research has also suggested that they are activated by salient (i.e., affectively-charged) information that include rewarding experiences, especially social rewards (e.g., Davey et al., 2010). In addition, prior work has also suggested that the anterior insula has an important role in representation of one’s internal states, including subjective feelings of emotion (Critchley et al., 2004; Lee and Siegle, 2012; Terasawa et al., 2013). Hence, together with the finding that higher levels of maternal negative affect are related to diminished amygdala responsivity to peer acceptance, results from the present study provide further support for the idea that positive peer evaluation is not incorporated into attention and emotional saliency networks to the same degree in adolescents who experience higher levels of maternal affect (Lindquist and Barrett, 2012), relative to their peers who experience low levels of maternal negativity. Moreover, evidence of blunted responsivity in these regions could also indicate that these adolescents view positive social information as less personally-relevant and therefore less motivating. This hypothesis is consistent with our finding that the presence of maternal negative affect is associated slower reaction times.

It is also interesting that we found relations between maternal negative affect and blunted neural response to peer acceptance in the sgACC. Increased sgACC responsivity to negative emotional information has been consistently implicated in adults (Drevets et al., 2008) and, more recently, adolescents with major depressive disorder (e.g., Silk et al., in press; Yang et al., 2009). Combined with findings from animal-based studies, these studies suggest that the sgACC is involved in affective responses to negative emotional events. However, there have also been several studies in humans and animals that indicate the sgACC may also play an important role in generating positive emotional responses in the context of appetitive stimuli (Drevets et al., 2008). The sgACC is functionally interconnected with the ventral striatum and the ventral tegmental area and activation in these regions is associated with rewarding experiences. Hypoactivation in this region in
the context of a rewarding experience like peer acceptance may be associated with symptoms of depression, for example anhedonia.

The present study has several limitations. First, our cross-sectional design precludes conclusions about the mediating or causal effect of maternal negative affect on adolescents’ neural response to peer evaluation. Without prospective longitudinal studies, it is difficult to determine whether maternal negative affect alters adolescents’ neural development and responsivity of peer evaluation and if these alterations precede the onset of depressive symptoms. Second, as is typical for many observational measures of parental affect that are obtained from laboratory-based parent–child interaction tasks, although negative affect was commonly expressed, the durations of negative affective bouts were typically very brief. In order to better understand the links between parenting and individual differences in how adolescents process affectively-charged social information, it will be important for future studies to replicate/extend these findings and to assess parenting behaviors with ecologically-valid methods that may allow for greater variation in negative affect. Third, although we focused on parental influences, we recognize that adolescents’ affective behavior also likely contributed to their mothers’ expressions of negative affect during the supportive discussion task. It will be important for future research to examine how transactional processes may be associated with adolescents’ neural response to peer evaluation. Furthermore, it is important to acknowledge that a number of factors likely interact to mediate observed associations between maternal negative affect and adolescents’ affective response to peer evaluation. Although beyond the scope of the present study, it will be important for future research to examine the contributions of shared environmental and genetic risk factors on relations between maternal affect and adolescents’ neural responsivity to peer acceptance. Moreover, the present study focuses on the influence of maternal affective behavior, excluding exploration of associations between fathers’ affect and neural responsivity to peer evaluation. Finally, given our relatively small sample size (particularly for boys), we were not able to investigate the impact of age (i.e., pubertal status) and gender on associations between parenting and responsivity to peer evaluation.

Despite these limitations, the study also has several strengths. Maternal affect was observed during naturalistic parent–adolescent interactions focused on discussions of a topic that was nominated by the adolescent. Moreover, we used a micro-analytic (second-by-second) behavioral coding system to capture normative, subtle variations in mothers’ negative affective behavior. We also utilized a novel virtual peer interaction paradigm that included live simulated interaction with age-matched virtual peers. This allowed us to tap into affective responses to ecologically valid social evaluative stimuli likely to be emotionally salient for adolescents. Overall, findings highlight maternal negative affect as a potential risk mechanism that is associated with altered responsivity to social rewards. As a modifiable environmental risk factor, parental negative affect could be targeted in treatment and prevention approaches for targeting alterations in adolescents’ affective processing, specifically disruptions in their responses to peer evaluation that may hinder their capacity to build and enjoy the friendships that are central to this developmental period.

**Conflict of interest**

None declared.

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**Appendix A. Supplementary data**

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.dcn.2014.01.006.

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