Maternal Emotion Socialization in Early Childhood Predicts Adolescents' Amygdala-vmPFC Functional Connectivity to Emotion Faces

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Guided by Eisenberg, Cumberland, and Spinrad's (1998) conceptual framework, we examined multiple components of maternal emotion socialization (i.e., reactions to children's negative emotion, emotion talk, emotional expressiveness) at 33 months of age as predictors of adolescents' amygdala-vmPFC connectivity and amygdala activation when labeling and passively observing angry and happy faces. For angry faces, more positive maternal emotion socialization behaviors predicted (a) less positive amygdala-vmPFC connectivity, which may reflect more mature vmPFC downregulation of the amygdala activation underlying implicit emotion regulation, and (b) more amygdala activation, which may reflect higher sensitivity to others' emotional cues. Associations between negative emotion socialization behaviors and neural responses to angry faces were nonsignificant, and findings for the models predicting neural responses to happy faces showed a less consistent pattern. By expanding Eisenberg et al.'s (1998) framework to consider neural processing of negative emotions, the current findings point toward the potential long-term implications of positive emotion socialization experiences during early childhood for optimal functioning of the amygdala-vmPFC circuitry during adolescence.

Keywords: adolescence, amygdala-PFC functional connectivity, amygdala activation, emotion socialization, toddlerhood

Supplemental materials: http://dx.doi.org/10.1037/dev0000852.supp

In their landmark paper, Eisenberg, Cumberland, & Spinrad, (1998) laid out a comprehensive framework for how parental emotion socialization behaviors—specifically, reacting to children's negative emotions, discussing emotions, and expressing emotions—contribute to children's ability to express and regulate emotions in socially competent ways. Eisenberg et al.'s framework mainly focused on child emotions at the behavioral level, yet emotion reactivity and regulation are multimodal processes that occur at both behavioral and physiological levels. Recent neurobiological research has highlighted functional connectivity between the amygdala and prefrontal cortex (PFC) as critical to emotion regulation (see Kim et al., 2011). In addition, accumulating empirical studies have linked early parenting with functional connectivity between the amygdala and medial PFC (mPFC) or ventromedial PFC (vmPFC, e.g., Gee, Gabard-Durnam, et al., 2013; Herringa et al., 2013; Kopala-Sibley et al., 2018). Seeking to extend

Eisenberg et al. (1998) framework to the neurobiological level, we examined maternal emotion socialization behaviors measured when children were 33 months of age as predictors of children's amygdala-vmPFC functional connectivity and amygdala activation to emotion faces at 13 years of age.

Maternal Socialization of Emotion

Parental reactions to children's negative emotions act as an important avenue for emotion socialization. Parents who provide comfort and help children problem-solve may facilitate children's ability to engage in constructive strategies for managing negative emotions (Eisenberg et al., 1998). For instance, mothers' use of emotion coaching practices (i.e., acceptance of children's emotions, instructions to assist children's coping) predicted better teacher-reported emotion regulation (Ramsden & Hubbard, 2002). Further, mothers' attempts to refocus the child's attention from a distressing stimulus and cognitively reframe the situation during a disappointing paradigm predicted decreases in preschool- and school-age children's anger and sadness intensity (Morris et al., 2011). In contrast, nonsupportive reactions, such as minimizing the child's negative emotional expression or punishing the child for negative emotional displays, may contribute to children's suppression or maladaptive expression of negative emotions (Eisenberg et al., 1998). Mothers' self-reported punitive responses, for example, predicted lower levels of child-reported regulation of negative emotion longitudinally (Eisenberg et al., 1999), and observer ratings of parental minimizing of children's negative emotions predicted poorer teacher-reported emotion regulation (Lunkenheimer, Shields, & Cortina, 2007).

Parental talk about emotions is another important aspect of emotion socialization. As posited by Eisenberg et al. (1998), children who are exposed to more frequent emotion talk may be better able to understand and communicate their feelings and may therefore learn to effectively manage their emotions. Existing empirical studies have revealed that maternal emotion talk, especially elaborative emotion talk, prospectively predicts children's better understanding of emotion (e.g., Dunn, Brown, & Beardsall, 1991) and more regulated behaviors during compliance tasks (Laible, 2004). It is also proposed that language may facilitate emotion regulation by helping children to make sense of their ongoing emotional experiences and communicate their needs (Cole, Armstrong, & Pemberton, 2010). In a similar vein, labeling emotions, or "putting emotion into words," has been found to increase emotion regulation and reduce emotional reactivity at both the neural (Brooks et al., 2017; Lieberman et al., 2007) and behavioral levels (Lieberman, Inagaki, Tabibnia, & Crockett, 2011). Thus, early maternal emotion talk may not only promote children's emotion talk and understanding but also their effective emotion regulation.

Lastly, the extent to which parents themselves display negative or positive emotions may have implications for children's emotional functioning (Eisenberg et al., 1998). Mother-reported positive (Nelson et al., 2012) and negative (Ramsden & Hubbard, 2002) expressiveness has been concurrently associated with children's more and less adaptive emotion regulation, respectively. Moreover, maternal reports of negative dominant emotions (e.g., anger) were associated with lower levels of school-age children's

constructive coping with daily stressors (Valiente, Fabes, Eisenberg, & Spinrad, 2004).

In sum, prior studies provide substantial support for the role of supportive emotion socialization behaviors in promoting greater emotional competence in early and middle childhood. Longer-term implications of early emotion socialization, however, have received less empirical attention. Notably, as children enter adolescence, they undergo drastic changes both physiologically and socially and thus may experience more frequent and intense emotions (see Ahmed, Bittencourt-Hewitt, & Sebastian, 2015). In addition, adolescence is a common period of onset for various forms of psychopathology characterized by deficits in emotion-related capacities (e.g., major depressive disorder, bipolar disorder, see Merikangas et al., 2010). It is, therefore, important to understand factors that predict emotional functioning among adolescents.

Parenting and the Amygdala-PFC Circuit

Although there has been rich evidence supporting the contributions of emotional socialization to children's emotional functioning at the behavioral level, investigations at the neural level are scarce. The amygdala-PFC circuit exhibits dramatic changes over development, such that amygdala-PFC functional connectivity in emotion-related tasks tends to be positive (i.e., activation of the amygdala increases in moments when activation of the PFC increases) during childhood, negative in adulthood, and close to zero in adolescence (Gee, Humphreys, et al., 2013; Silvers et al., 2017). In addition, more negative (or less positive) amygdala-PFC functional connectivity in emotion processing tasks (e.g., viewing or labeling emotion faces) has been associated with less separation anxiety among children and adolescents (Gee, Humphreys, et al., 2013), less stress rumination and fewer depressive symptoms among adolescent girls (Fowler, Miernicki, Rudolph, & Telzer, 2017), and lower levels of internalizing problems (Gard et al., 2018). Taken together, more negative or less positive amygdala-PFC connectivity in response to emotional stimuli indicates a more mature pattern of neural activity that, in turn, may underlie more optimal emotion regulation and psychological well-being.

Parenting, especially during the early years, is proposed to play a critical role in shaping the function and development of the amygdala-PFC circuit (Callaghan & Tottenham, 2016). Specifically, maternal presence has been shown to promote negative amygdala-mPFC connectivity in an emotion processing task, especially for securely attached children (Gee et al., 2014). Early trauma, maltreatment, and family adversity predicted adolescents' more negative amygdala-vmPFC connectivity at resting state (Burghy et al., 2012; Herringa et al., 2013), but more positive amygdala-mPFC connectivity in emotion processing tasks (Herringa et al., 2016; Marusak, Martin, Etkin, & Thomason, 2015), which may reflect less optimal emotion functioning at the neural level. Two studies also linked early maternal hostility (Kopala-Sibley et al., 2018) and parental deprivation (Gee, Gabard-Durnam, et al., 2013) with children's premature negative amygdala-mPFC connectivity in emotion processing tasks, which may be a self-protective mechanism in the face of long-term lack of parental support. These findings suggest that positive parenting may predict more adaptive and age-typical amygdala-mPFC/vmPFC connectivity in response to emotion stimuli. Yet, given the

predominately cross-sectional designs and focus on adversity in existing studies (see Kopala-Sibley et al., 2018, for exception), longitudinal studies that assess normative differences in early parenting as predictors of amygdala-PFC connectivity during adolescence are needed.

The Current Study

In this 10-year longitudinal study, we examined maternal emotion socialization behaviors during toddlerhood as predictors of children's neural responses to emotion faces during early adolescence. When children were 33 months of age, we measured three aspects of emotion socialization laid out in Eisenberg et al.'s (1998) framework (i.e., reactions to children's negative emotions, emotion talk, and emotional expressiveness). When children were approximately 13 years of age, they completed a functional MRI (fMRI) scan. We examined adolescents' amygdala-vmPFC functional connectivity and amygdala activation when labeling (i.e., matching emotion faces with corresponding labels) and passively observing emotion faces. We focused on amygdala-vmPFC connectivity specifically because (a) existing empirical evidence has demonstrated links between early family adversity and adolescents' amygdala-vmPFC connectivity (e.g., Burghy et al., 2012; Gee, Gabard-Durnam, et al., 2013; Herringa et al., 2013), (b) functional parcellation of the mPFC suggests that the vmPFC supports emotion regulation (Etkin, Egner, & Kalisch, 2011), and (c) emotion regulation is proposed to develop in a medial to lateral pattern in the PFC, such that amygdala-vmPFC connectivity is needed before lateral PFC can exert effects on the amygdala (Silvers et al., 2017). In addition, because facial expressions in real life often vary in intensity, to increase ecological validity, we used ambiguous emotion faces (i.e., angry and happy faces morphed, respectively, with neutral faces) in the current study.

Based on Eisenberg et al.'s (1998) conceptual framework and existing neural studies (see Callaghan & Tottenham, 2016; Herringa et al., 2016; Marusak et al., 2015), we hypothesized that positive maternal emotion socialization (i.e., supportive reactions to children's negative emotions, more frequent and elaborative emotion talk, and positive expressiveness) in toddlerhood would predict more negative (or less positive) amygdala-vmPFC connectivity to angry faces during early adolescence, and negative emotion socialization (i.e., nonsupportive reactions, dominant negative expressiveness) would predict less negative (or more positive) amygdala-vmPFC connectivity. In addition to testing these main effects, we also examined interactions between emotion socialization behaviors and task condition (labeling vs. observing) on amygdala-vmPFC connectivity. Given that emotion labeling may serve as an implicit regulatory process and has been linked with reduced distress (Lieberman et al., 2011), more negative (or less positive) amygdala-vmPFC connectivity in the Label versus Observe condition may reflect more effective implicit regulation. We hypothesized that adolescents who experienced high positive or low negative emotion socialization would show more negative (or less positive) amygdala-vmPFC connectivity in the Label versus Observe condition.

We examined amygdala activation as a secondary aim. We did not have a priori hypotheses for this outcome because (a) heightened amygdala activation to ambiguous angry faces may reflect adaptive sensitivity to subtle emotional cues or maladaptive hyper-

reactivity to threat, and (b) emotion labeling may help adolescents pay more attention to the subtle emotional cues and downregulate their emotion reactivity at the same time. In addition, because studies of neural processing of positive emotions are less frequent, and existing studies have yielded nonsignificant findings (e.g., Gee, Gabard-Durnam, et al., 2013; Herringa et al., 2016; Romund et al., 2016; see Gee et al., 2014 as notable exception), analyses examining maternal emotion socialization as predictors of adolescents' neural activation to happy faces were exploratory.

Method

Participants

Toddlers ($N = 128$, 62 boys) and their mothers participated in a longitudinal study of early socioemotional development. At the initial time point, children ranged between 31 and 35 months of age ($M = 32.7$ months, $SD = .76$). When children were approximately 13 years of age, families were contacted to participate in a follow-up study of family relationships and adolescent neural and behavioral regulation of stress. Sixty-seven families participated in the follow-up study, and 51 adolescents completed an fMRI scan (34 boys, $M = 13.2$ years, $SD = .56$, range $= 12.4–14.8$ years). Reasons for adolescents not completing the fMRI scan included claustrophobia ($n = 2$), braces ($n = 7$), and declining to participate ($n = 6$). In addition, neuroimaging data from one adolescent were not usable because of malfunction of the computer delivering the task stimuli.

We focused on the sample of 51 children with complete fMRI data. Among these families, mothers averaged 16.2 ($SD = 1.86$) years of education and were 82.4% European American, 5.9% Asian American, 2.0% African American, 2.0% Hispanic, and 7.8% other ethnicity, respectively. Fathers averaged 15.6 ($SD = 2.28$) years of education and were 88.2% European American, 3.9% African American, 2.0% other ethnicity, respectively, and 5.9% did not report their ethnicity. The research protocols for the *Children's Social Development Project* (*CSDP*; protocol # 05181) and *CSDP-Phase 3* (protocol #15435) were approved by the Institutional Review Board at the University of Illinois at Urbana–Champaign.

Maternal Emotion Socialization Measures

At the initial time point (33 months), mother–child dyads participated in a 90-min visit to a laboratory playroom and were observed interacting in a variety of sessions (see McElwain, Holland, Engle, & Wong, 2012, for further details). Toward the end of the laboratory visit, mothers and children were observed during a wordless picture book task, and maternal talk about emotions was assessed from transcripts of this task. At the end of the laboratory visit, mothers were given a questionnaire packet, which included items about parental reactions to children's negative emotions and parental emotional expressiveness, to complete at home and return by mail.

Maternal talk about emotions. Mothers and children were given a wordless picture book that contained 12 photos of infants showing various facial expressions. Photos, which were taken from the IFEEL Pictures booklet (see Emde, Osofsky, & Butterfield, 1993), included displays of positive, negative, and neutral/

ambiguous expressions. Mothers were asked to talk with their child about what the infant in each picture may be thinking, feeling, or doing. There was no time limit for the task, and mother–child discussions averaged 5.59 min ($SD = 2.49$).

Mothers' utterances during the task were transcribed. An utterance was defined as a complete clause, which could be a complete sentence ("Do you think she looks angry?") or sentence fragment (e.g., "Sad face"), and each utterance was coded for (a) focus (i.e., in what terms the mother talked about the picture, including emotions, cognitions, desires, behaviors, or physical characteristics) and (b) function (i.e., whether the mother provided a label, an explanation of the cause of the infant's state, an action that may resolve the problem, or a rationale for her own description).

We examined the number of maternal utterances in which emotion was the focus (e.g., "She looks happy.") and the number of elaborative utterances about emotion, that is, mother went beyond providing a label and talked about the cause of the emotion (e.g., "I wonder if his brother took his toy"), an action that may resolve the problem (e.g., "Don't be sad, baby. Why don't we tickle the baby's chin."), or a rationale for her own description (e.g., "She is sad because she is crying"). On average, mothers displayed 40.6 utterances ($SD = 19.55$, range = 7–177) during the task, and 13.3 (31.9%, $SD = 8.54$, range = 1–46) of those utterances focused on emotions. Further, of the utterances focused on emotions, mothers averaged 3.3 (20.9%, $SD = 3.94$, range = 0–28) elaborative utterances. Intercoder reliability was high, $\kappa = .96$ and .90 for the number of emotion focused utterances and elaborative emotion utterances, respectively.

Maternal-reported reactions to toddlers' negative emotions. Using the Coping with Toddler's Negative Emotions Scale (CTNES; Spinrad, Eisenberg, Kupfer, Gaertner, & Michalik, 2004), mothers rated how they would respond to their child's expression of negative emotions in 12 hypothetical situations (e.g., "If my child is going to spend the afternoon with a new babysitter and becomes nervous and upset because I am leaving him or her, I would: . . .") using a 7-point Likert scale, ranging from 1 (*very unlikely*) to 7 (*very likely*). For each situation, mothers rated the likelihood of the response for seven different subscales. Subscales were created by averaging ratings (with reverse scoring as appropriate) across the 12 situations. Given the focus of this report, four subscales were examined: (a) emotion-focused reactions (e.g., "distract my child by talking about all the fun he will have with the sitter"; $\alpha = .75$), (b) problem-focused reactions (e.g., "help my child think of things to do that with make it less stressful, like calling him once during the afternoon"; $\alpha = .82$), (c) punitive reactions (e.g., "tell my child that he won't get to do something else enjoyable, such as going to the playground or getting a special snack, if he doesn't stop behaving that way"; $\alpha = .78$), and (d) minimizing reactions (e.g., "tell him that it's nothing to get upset about"; $\alpha = .86$). Composites of supportive and nonsupportive reactions to children's negative emotions were created by averaging the emotion-focused and problem-focused subscales ($r[48] = .35$, $p = .016$) and punitive and minimizing subscales ($r[48] = .55$, $p < .001$), respectively. The CTNES was adapted from the Coping with Children's Negative Emotions Scale (CCNES, Fabes, Eisenberg, & Bernzweig, 1990). The composites in the current study are consistent with a validation study of the CCNES in which a principal component factor analysis showed that punitive and minimizing reactions loaded on one factor and problem-focused

and emotion-focused coping loaded on another factor (Fabes, Poulin, Eisenberg, & Madden-Derdich, 2002). The CTNES has shown good internal consistency and test—retest reliability (Spinrad et al., 2004), and scores on the CTNES subscales have been associated in expected ways with relevant constructs, such as observed maternal sensitivity and warmth, child social competence, and externalizing problems (Spinrad et al., 2007).

Maternal emotional expressiveness in the family. Mothers also completed the Short Form (24 items) of the Self-Expressiveness in the Family Questionnaire (SEFQ; Halberstadt, Cassidy, Stifter, Parke, & Fox, 1995). Items assessed the degree to which the mother expressed positive and negative emotions toward other family members and were rated on a 9-point scale, ranging from 1 (*not at all frequently*) to 9 (*very frequently*). We examined the 12-item positive expressiveness subscale (e.g., "Telling family members how happy you are"; $\alpha = .86$) and 10-item negative dominant expressiveness subscale (e.g., "Expressing anger at someone else's carelessness"; $\alpha = .83$). For each subscale, responses were averaged across items. The Short Form of the SEFQ has good reliability and construct validity, and its subscales have been related to parents' personality and general feeling states (Halberstadt et al., 1995; Nelson et al., 2012).

Neural Responses to Emotion Faces

Approximately 10 years following the first laboratory visit, adolescents participated in two laboratory visits. First, adolescents and parents participated in a 90-min behavioral visit, in which parents and adolescents were observed in several interactive tasks (see Ravindran, Hu, McElwain, & Telzer, 2019). At a second visit scheduled approximately two weeks later, adolescents participated in a 90-min brain imaging scan. During the brain scan, adolescents completed an emotion processing task adapted from previous studies (e.g., Fowler et al., 2017; Lieberman et al., 2007).

Specifically, adolescents were presented with facial stimuli consisting of angry, happy, and neutral expressions from the NimStim set (available at http://www.macbrain.org). Happy and angry faces were morphed, respectively, with neutral faces in 15% increments (i.e., 15%, 30%, 45%, 60%, and 75%, where the percentage indicates the emotional intensity of happy [or angry] valence, see Figure S1 in the online supplemental materials [e.g., stimuli]). This procedure yielded 80 unique stimuli (5 emotion intensities × 2 valences × 8 faces). There were equal numbers of male and female faces, and equal numbers of African American and European American faces. The task included two conditions: Label and Observe. During the "Label" rounds, adolescents were instructed to match the facial emotions with one of three labels ("Happy," "Neutral," and "Angry") displayed across the bottom of the screen using a button box. During the "Observe" rounds, participants were instructed to view the faces and to press their thumb (to control for potential confound of motor activity/instructions in the Label condition) for each face, regardless of emotion. Although adolescents were not explicitly instructed to regulate their emotions, amygdala-vmPFC connectivity in this emotion processing task may reflect neural activities underlying implicit emotion regulation (Gyurak, Gross, & Etkin, 2011), especially for the Label condition, which is frequently used to measure implicit emotion regulation involved in "putting feelings into words" (Brooks et al., 2017; Fowler et al., 2017; Lieberman et al., 2007).

Label and Observe conditions were presented randomly in a block manner, with two blocks for each condition. Each block began with a fixation cross lasting 1,250 ms, followed by a title slide denoting the condition for the given block (label or observe), which lasted 2,750 ms. Face stimuli appeared on the screen for 2.5 s. Each face was followed by a jitter that was determined by a gamma distribution centered at 1,000 ms. There were 40 trials per block, resulting in 160 trials (40 trials × 2 blocks per condition × 2 conditions), and each of the 80 face stimuli was presented twice (once in each condition).

fMRI data acquisition. Imaging data were collected using a 3 Tesla Siemens Trio MRI scanner. The task included T2∗weighted echoplanar images (EPI; slice thickness = 3 mm; 38 slices; TR = 2 s; TE = 25 msec; matrix = 92 × 92; FOV = 230 mm; voxel size 2.5 × 2.5 × 3 mm). In addition, structural scans consisted of a T2∗weighted, matched-bandwidth (MBW), high-resolution, anatomical scan (TR = 4 s; TE = 64 msec; FOV = 230; matrix = 192 × 192; slice thickness = 3 mm; 38 slices) and a T1∗ magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR = 1.9 s; TE = 2.3msec; FOV = 230; matrix = 256 × 256; sagittal plane; slice thickness = 1 mm; 192 slices). To maximize brain coverage, MBW and EPI scans were obtained using an oblique axial orientation.

fMRI data preprocessing. Preprocessing utilized FSL FMRIBs Software Library (FSL v6.0; https://fsl.fmrib.ox.ac.uk/fsl/). Steps taken during preprocessing included correction for slice-to-slice movement using MCFLIRT; high-pass temporal filtering with a 128-s cut-off to remove low frequency drift across the time-series; skull stripping of all images with BET; and spatial smoothing using a 6-mm Guassian kernel, full-width-at-half maximum. Functional images were resampled to a 2 × 2 × 2 mm space and coregistered in a two-step sequence to the MBW and the MPRAGE images using Advanced Normalization Tools (ANTS; Avants, Tustison, & Song, 2009; http://stnava.github.io/ANTs/) to warp them into the standard stereotactic space defined by the Montreal Neurological Institute (MNI) and the International Consortium for Brain Mapping. Preprocessing was completed utilizing individual-level independent component analysis (ICA) with MELODIC combined with an automated component classifier (Tohka et al., 2008; Neyman-Pearson threshold = 0.3), which was applied to filter signal origination from noise sources (e.g., motion, physiological rhythms). Such a method for cleaning fMRI data is a widespread practice that is incorporated in many standard preprocessing pipelines (e.g., fmriprep; https://fmriprep.readthedocs.io/en/stable/workflows.html).

The task was modeled using an event-related design within the Statistical Parametric Mapping software package (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, U.K.). Each event was modeled using the onset of the stimulus and a duration of 2.5 s. Individual fixed-effects models were created for each participant using the general linear model in SPM with regressors for conditions of interest: Label trials for angry faces, Observe trials for angry faces, Label trials for happy faces, and Observe trials for happy faces. We controlled for levels of emotion intensity in the face stimuli by including the intensity rating as a parametric modulator at the trial level, which ranged from −2 to 2 corresponding to the 15% to 75% range of emotion intensity in the morphed images (centered at 0, i.e., 45% of emotion intensity). We focused on the main effects of conditions

of interest, controlling for the parametric modulator, which examines neural activation in the Label and Observe conditions versus the baseline after holding emotion intensity in the stimuli constant. Volumes containing motion in excess of 0.9 mm framewise-displacement were modeled in a separate junk regressor. Jittered inter-trial-interval periods (i.e., fixation) were not explicitly modeled and therefore served as the implicit baseline for task conditions.

We utilized an ROI-based approach to extract parameter estimates of signal intensity from the bilateral amygdala (AAL atlas; Tzourio-Mazoyer et al., 2002; see Figure 1 for mask images). Parameter estimates were extracted from each participant's first-level contrasts for the four conditions (vs. the implicit baseline): labeling angry faces, observing angry faces, labeling happy faces, and observing happy faces, and were used in subsequent analyses.

To examine neural connectivity, we conducted psychophysiological interaction (PPI) analyses using a structurally defined region of interest in the bilateral amygdala as our seed region. PPI analyses utilized a generalized form of the context-dependent PPI from the automated generalized PPI (gPPI) toolbox (McLaren, Ries, Xu, & Johnson, 2012). Time series were extracted from the seed region and served as the physiological variable in the analysis. Each trial type was then convolved with the canonical HRF to create the psychological regressor. In the final step, the physiological and psychological variables were multiplied to create the PPI term. This interaction term was then used to identify regions that covary with the amygdala seed region in a task-dependent manner. As such, each participant's individual gPPI model included a deconvolved BOLD signal alongside the psychological and interaction term for each event type. Estimates of connectivity were extracted from a region of the vmPFC (see Figure 1 for mask images). We utilized Neurosynth (Yarkoni, Poldrack, Nichols, Van Essen, & Wager, 2011) to create the vmPFC mask using the search term "vmPFC" in the automated meta-analysis tool and thresholded the resultant statistic map at $Z = 5$. The resulting ROI is likely to reflect the region consistently labeled as vmPFC among neuroimaging researchers. Parameter estimates (i.e., connectivity levels between the amygdala and vmPFC) were extracted from the same four first-level contrasts as used to estimate amygdala activation (i.e., label angry, observe angry, label happy, observe happy) for use in further analyses.

Data Analytic Strategy

Using Mplus 8.1 (Muthén & Muthén, 1998–2018), we fitted multilevel models to account for the nested structure of data (i.e., Label and Observe conditions nested within each participant). At the within-person level, we tested effects of condition on amygdala-vmPFC connectivity and amygdala activation. At the between-person level, we tested maternal emotion socialization behaviors (ESB, listed at the top of Table 1) as predictors of amygdala-vmPFC connectivity and amygdala activation. We also examined cross-level ESB × Condition interactions. Because we tested ESB main effects and ESB × Condition interactions in the same model, we used effect coding for condition (Observe = −1, Label = 1), so that the main effect parameters represented associations across conditions. For significant interactions, we probed condition effects at high (+1 SD) and low (−1 SD) levels of the given emotion socialization behavior. Covariance between amygdala-

Figure 1. ROI images of bilateral amygdala (upper) and vmPFC (lower). Right Amygdala: $k = 254$, Center of Mass = $[24, 4, -18]$; left Amygdala: $k = 270$, Center of Mass = $[-22, 4, -18]$; vmPFC: $k = 1452$, Center of Mass = $[-2, 42, -10]$.

vmPFC connectivity and amygdala activation at both the within-person and between-person levels were estimated. Separate analyses were conducted for angry and happy faces, and for each emotion we first fitted six separate models, that is, one for each emotion socialization predictor. For the emotion socialization behaviors showing significant main or interaction effects in the separate models, we tested an inclusive model to examine their unique contributions.

Missing data. Of the 128 children who participated at the initial time point, 67 participated in the follow-up at 13 years. Participating children were more likely to be boys, $t(126) = -3.52$, $p = .001$, compared with those who did not participate in the follow-up study. The two groups did not differ on child age at the initial time point, maternal education, or the emotion socialization variables. Of the 67 who participated in the adolescent phase, 51 children had usable neuroimaging data. For adolescents who had neuroimaging data (vs. those who did not), mothers reported higher supportive reactions, $t(18.20) = 2.14$, $p = .046$, and higher positive expressiveness, $t(62) = 2.22$, $p = .030$, at the first time point, but did not differ on other study variables, child gender, child age at the adolescent phase, or maternal education. In the analyses reported below, we used data from the 51 participants who had complete fMRI data, although results were the same when full information maximum likelihood (FIML) was used to estimate model parameters among (a) the sample of 67 children who participated at both time points, and (b) the original Time 1 sample of 128 children.

Supplementary analyses. Because previous studies have found that right ventrolateral PFC (rvlPFC) was more active during emotion labeling than passive viewing (Lieberman et al., 2007) and that more positive amygdala-rvlPFC connectivity when labeling negative emotions was related to greater stress-reactive rumination and depressive symptoms (Fowler et al., 2017), we also examined emotion socialization behaviors as predictors of amygdala-rvlPFC connectivity. In these supplementary analyses, our model tests paralleled those used in the main analyses described above. Results of the additional analyses were largely nonsignificant (see Tables S3–S5 in the online supplemental materials). Considering our sample of early adolescents, these findings are consistent with the viewpoint that amygdala-vmPFC connectivity develops before amygdala-vlPFC connectivity does (Silvers et al., 2017).

For interested readers, the statistical maps showing (a) the whole-brain activation patterns of the primary contrasts, and (b) the emotion socialization behaviors as regressors (controlling for child gender) on whole-brain activation patterns and functional connectivity with bilateral amygdala in the Label > Observe contrast can be found on Neurovault (see https://neurovault.org/collections/XMHSEZWX/).

Results

Preliminary Analyses

Descriptive statistics and bivariate correlations among the study variables are presented in Table 1. One-sample t tests indicated that in both the Label and Observe conditions for both angry and happy faces (a) amygdala-vmPFC connectivity was greater than 0 (ts [50] = 7.83 to 10.16, ps < .001), indicating that, on average, amygdala activation increased in moments when activation of vmPFC increased, and (b) amygdala activation did not differ from 0. Additionally, paired sample t tests showed that, on average, amygdala-vmPFC connectivity and amygdala activation did not differ across the Label and Observe conditions. Correlations between potential covariates (i.e., child gender, child age at the adolescent phase, maternal education, and total number of maternal utterances) and amygdala-vmPFC connectivity (and amygdala activation) were all nonsignificant. We did, however, control for main effects of child gender in the main models because child gender was associated with missingness in the longitudinal sample. In all the models tested and reported below, child gender was not

Table 1

Descriptive Statistics and Intercorrelations Among the Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Supportive reactions	—	.16	−.05	−.03	.28	.01	−.07	−.24	−.18	−.10	.33*	.23	.34*	.31
2. Nonsupportive reactions		—	.04	.001	−.10	.32*	.05	−.12	.11	−.02	.13	.15	.18	.29*
3. Emotion talk frequency			—	.56***	.20	.04	−.28*	−.11	.07	−.07	.04	−.07	.01	.15
4. Elaborative emotion talk				—	.22	−.05	−.21	.05	.19	.04	.04	−.12	−.08	.06
5. Positive expressiveness					—	−.30*	−.03	−.10	.20	.20	.17	−.03	.003	.22
6. Negative expressiveness						—	−.01	.26	.02	−.35*	.22	.21	.27	.28
7. Amygdala-vmPFC AngL							—	.36*	.47**	.32*	.06	.04	−.02	−.04
8. Amygdala-vmPFC AngO								—	.49***	.31*	.10	−.03	−.10	−.17
9. Amygdala-vmPFC HapL									—	.28*	−.09	−.04	−.12	−.11
10. Amygdala-vmPFC HapO										—	−.31*	−.31*	−.34*	−.19
11. Amygdala activation AngL											—	.74***	.84***	.72***
12. Amygdala activation AngO												—	.81***	.73***
13. Amygdala activation HapL													—	.78***
14. Amygdala activation HapO														—
M	6.11	2.45	13.69	2.87	7.61	3.80	0.40	0.46	0.44	0.40	0.001	−0.04	−0.02	−0.09
SD	0.39	0.76	8.04	2.75	0.82	1.17	0.28	0.42	0.32	0.35	0.40	0.45	0.40	0.40
n	48	48	51	51	48	48	51	51	51	51	51	51	51	51

Note. AngL = angry faces in the Label condition; AngO = angry faces in the Observe condition; HapL = happy faces in the Label condition; HapO = happy faces in the Observe condition.
* $p < .05$. ** $p < .01$. *** $p < .001$.

associated with amygdala-vmPFC connectivity or amygdala activation.

Main Models

Angry faces. Six models were tested to assess the main effects of each emotion socialization behavior (ESB), as well as ESB × Condition interaction effects, on amygdala-vmPFC functional connectivity and amygdala activation to angry faces. At Level 1, and consistent with the paired sample *t* tests reported above, no main effects of condition emerged. Level 2 parameter estimates for each model are reported in Table 2. (Note that each row in Table 2 shows the estimates from a single model for that predictor.) More supportive reactions to children's negative emotions predicted less positive amygdala-vmPFC connectivity (see Table 2 and Figure 2). Maternal elaborative emotion talk interacted with condition on amygdala-vmPFC connectivity (see Table 2). Adolescents showed less positive amygdala-vmPFC connectivity in Label than in Ob-serve at high ($B = −.07$, $SE = .03$, $p = .019$) but not low ($B = .01$, $SE = .04$, $p = .720$) levels of elaborative emotion talk. See the online supplemental materials (Figure S2) for a scatter plot showing this association by condition.

In addition, maternal elaborative emotion talk interacted with condition on amygdala activation (see Table 2). Adolescents showed marginally higher amygdala activation in Label than in Observe at high ($B = .05$, $SE = .03$, $p = .067$) but not low ($B = −.02$, $SE = .03$, $p = .510$) levels of elaborative emotion talk. Maternal positive expressiveness also interacted with condition on amygdala activation. Adolescents showed higher amygdala activation in Label than in Observe at high ($B = .07$, $SE = .03$, $p = .043$) but not low ($B = −.02$, $SE = .03$, $p = .521$) levels of maternal positive expressiveness. See the online supplemental materials for scatter plots showing these relations for elaborative emotion talk (Figure S3a) and positive expressiveness (Figure S3b) by condition.

Table 2

Maternal Emotion Socialization at 33 Months as Predictors of Amygdala Activation and Amygdala-vmPFC Connectivity to Angry Faces at 13 Years

Emotion socialization behavior (ESB)	Amygdala-vmPFC connectivity				Amygdala activation			
	ESB main effect		ESB × Condition		ESB main effect		ESB × Condition	
	B (*SE*)	*p*	*B* (*SE*)	*p*	*B* (*SE*)	*p*	*B* (*SE*)	*p*
Supportive reactions	−.18 (.08)	.031	.10 (.05)	.053	.33 (.18)	.063	.04 (.06)	.505
Nonsupportive reactions	−.05 (.05)	.353	.04 (.04)	.262	.10 (.07)	.154	−.01 (.03)	.659
Emotion talk frequency	−.01 (.01)	.174	−.002 (.003)	.536	−.002 (.01)	.796	.003 (.002)	.147
Elaborative emotion talk	−.01 (.01)	.715	−.02 (.01)	.049	−.01 (.02)	.718	.01 (.01)	.045
Positive expressiveness	−.03 (.06)	.681	.02 (.05)	.682	.03 (.09)	.697	.05 (.03)	.047
Negative expressiveness	.05 (.05)	.334	−.05 (.03)	.159	.08 (.05)	.112	−.001 (.02)	.949

Note. Six models were tested to assess the main effects of each emotion socialization behavior (ESB) and the ESB × Condition interaction on amygdala-vmPFC functional connectivity and amygdala activation to angry faces. For condition, Observe and Label were coded as −1 and 1, respectively. Gender was included as a covariate.
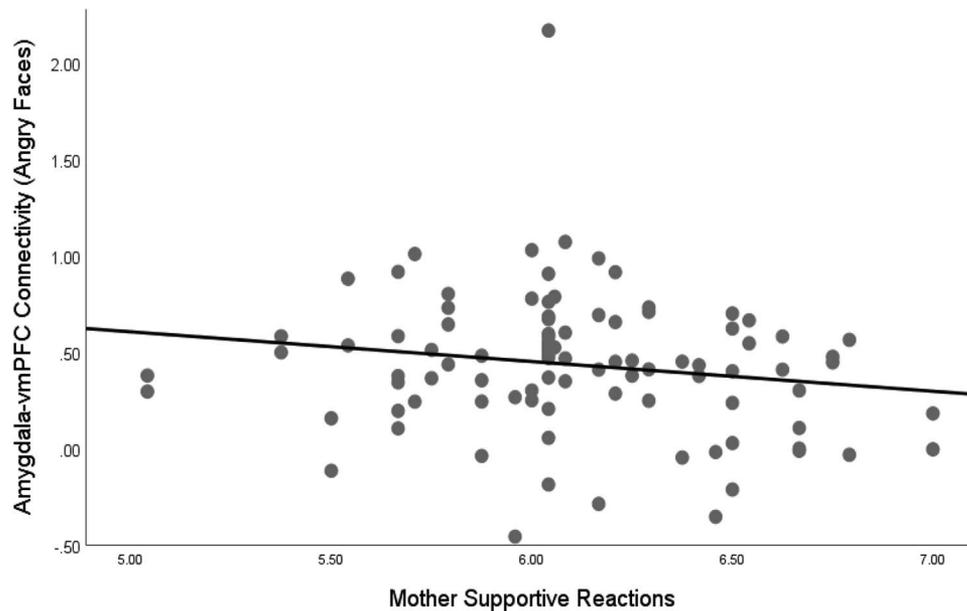
Figure 2. Higher mother supportive reactions to children's negative emotions at 33 months of age predicted less positive amygdala-vmPFC connectivity when labeling and observing angry faces at 13 years. When one outlier is taken out, the results remain the same, $B = -.17$, $SE = .08$, $p = .039$.

In the inclusive model, (a) the main effect of maternal supportive reactions on amygdala-vmPFC connectivity and (b) the interaction between maternal elaborative emotion talk and condition on amygdala-vmPFC connectivity remained significant. The interactions between (a) maternal elaborative emotion talk and condition and (b) maternal positive expressiveness and condition on amygdala activation became nonsignificant and marginal, respectively. In addition, a positive association between maternal supportive reactions and amygdala activation emerged ($B = .33$, $SE = .16$, $p = .039$). See the online supplemental materials for the scatter plot showing this relation (Figure S4) and parameter estimates for the inclusive model for angry faces (Table S1).

Happy faces. Six models were tested to assess the main effects of each emotion socialization behavior (ESB) and the ESB × Condition interaction on amygdala-vmPFC functional connectivity and amygdala activation to happy faces. At Level 1, no main effects of Condition emerged. Level 2 parameter estimates for each model are reported in Table 3. Maternal dominant negative expressiveness interacted with condition on amygdala-vmPFC connectivity. Adolescents showed marginally more positive amygdala-vmPFC connectivity in Label than in Observe when negative expressiveness was high ($B = .08$, $SE = .05$, $p = .068$), but not when negative expressiveness low ($B = -.05$, $SE = .04$, $p = .203$). See the online supplemental materials (Figure S5) for the scatter plot showing this association by condition.

In addition, maternal supportive reactions (Figure 3a), nonsupportive reactions (Figure 3b), and dominant negative expressiveness (Figure 3c) predicted more amygdala activation. Frequency of maternal emotion talk interacted with condition on amygdala activation. Adolescents showed higher amygdala activation in Label

Table 3

Maternal Emotion Socialization at 33 Months as Predictors of Amygdala Activation and Amygdala-vmPFC Connectivity to Happy Faces at 13 Years

Emotion socialization behavior (ESB)	Amygdala-vmPFC connectivity				Amygdala activation			
	ESB main effect		ESB × Condition		ESB main effect		ESB × Condition	
	B (SE)	p	B (SE)	p	B (SE)	p	B (SE)	p
Supportive reactions	−.14 (.09)	.120	−.03 (.06)	.640	.35 (.16)	.031	.02 (.05)	.763
Nonsupportive reactions	.01 (.05)	.861	.03 (.03)	.345	.14 (.06)	.026	−.03 (.03)	.335
Emotion talk frequency	.000 (.01)	.919	.003 (.003)	.321	.003 (.01)	.584	−.004 (.002)	.037
Elaborative emotion talk	.02 (.01)	.185	.01 (.01)	.210	−.003 (.02)	.911	−.01 (.01)	.109
Positive expressiveness	.09 (.05)	.059	−.004 (.05)	.931	.05 (.07)	.435	−.05 (.03)	.055
Negative expressiveness	−.05 (.03)	.122	.06 (.03)	.037	.10 (.04)	.011	−.001 (.02)	.941

Note. Six models were tested to assess the main effects of each emotion socialization behavior (ESB) and the ESB × Condition interaction on amygdala-vmPFC functional connectivity and amygdala activation to happy faces. For condition, Observe and Label were coded as −1 and 1, respectively. Gender was controlled as a covariate.
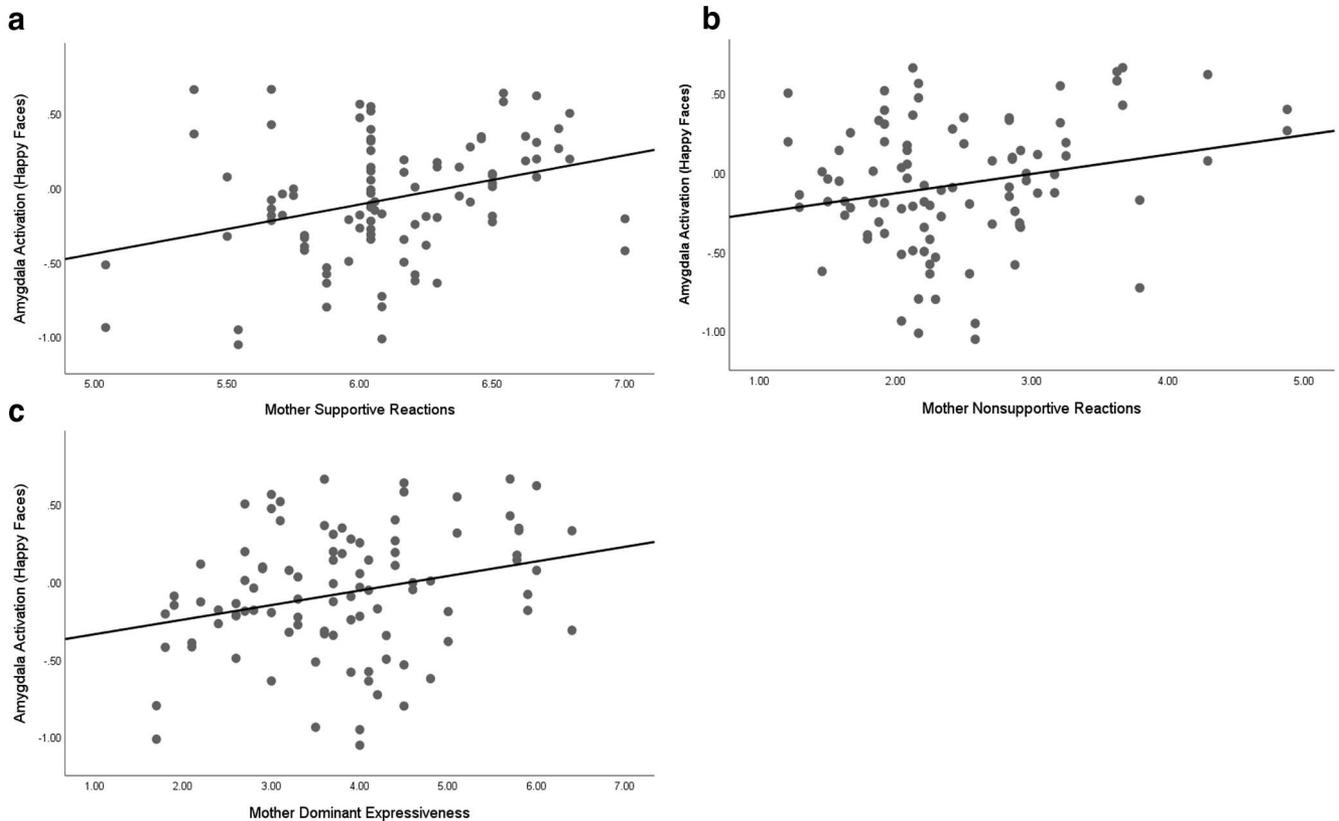
Figure 3. Mother (a) supportive reactions to children's negative emotions, (b) nonsupportive reactions to children's negative emotions, and (c) dominant negative expressiveness at 33 months of age predicted higher amygdala activation to happy faces at 13 years.

than in Observe when frequency of maternal emotion talk was low ($B = .06$, $SE = .03$, $p = .019$), but not when frequency of maternal emotion talk was high ($B = .004$, $SE = .02$, $p = .824$). See the online supplemental materials (Figure S6) showing the scatter plot for this relation by condition.

In the inclusive model, the association between nonsupportive reactions and amygdala activation became nonsignificant, whereas the significance level of all other associations remained the same (see Table S2 in the online supplemental materials for parameter estimates).

Discussion

Supporting Eisenberg et al.'s (1998) framework, substantial empirical evidence over the past several decades indicates that maternal emotion socialization is a key contributor to emotional competence during childhood, yet longer-term associations from early childhood to adolescence have rarely been examined. In addition, no study to our knowledge has investigated relations between mothers' emotion socialization behaviors specifically and children's emotion-related regulatory processes at the neural level. To address these gaps, we examined multiple aspects of maternal emotion socialization (i.e., reactions to children's negative emotions, emotion talk, emotional expressiveness) at 33 months of age as predictors of adolescents' amygdala-vmPFC functional connec-

tivity and amygdala activation when labeling and observing emotion faces at 13 years.

Amygdala-vmPFC Connectivity

We preface our discussion of the amygdala-vmPFC connectivity results by noting that, on average, connectivity was positive and greater than 0, which would be expected given that our participants composed a low-risk community sample of early adolescents (age range: 12.4 to 14.8 years). For our sample, therefore, a more mature pattern of amygdala-vmPFC connectivity would be reflected in *less positive* connectivity (i.e., connectivity values closer to zero) versus *more negative* connectivity, which may be a more applicable description during later adolescence and adulthood when connectivity tends to be negative (i.e., below 0) on average (Silvers et al., 2017). In addition, levels of amygdala-vmPFC connectivity and amygdala activation, on average, did not differ between the Label and Observe task conditions. Nevertheless, wide individual differences emerged for amygdala-vmPFC connectivity, amygdala activation, and condition effects, and our findings show that early experiences of maternal emotion socialization may contribute in expected ways to such individual differences.

For angry faces, as hypothesized, supportive reactions to children's negative emotions predicted less positive amygdala-vmPFC

functional connectivity across Label and Observe conditions. Further, mothers' elaborative emotion talk interacted with condition, such that less positive amygdala-vmPFC functional connectivity emerged in the Label versus Observe condition when elaborative emotion talk was high. These connectivity patterns may reflect more mature downregulation of vmPFC on amygdala activation underlying implicit and automatic emotion regulation. Importantly, these associations remained significant in the inclusive model, indicating that supportive reactions and elaborative emotion talk each made unique contributions.

The finding that maternal supportive reactions predicted young adolescents' less positive amygdala-vmPFC connectivity converges with evidence from neural studies showing that early family adversity predicted more positive amygdala-mPFC connectivity in an emotion processing task during adolescence (Herringa et al., 2016). This finding is also consistent with evidence from behavioral studies showing that maternal emotion coaching practices (Ramsden & Hubbard, 2002) and supportive reactions to children's displays of anger and sadness (Morris et al., 2011) predicted children's more effective behavioral regulation of emotions. According to Eisenberg et al.'s (1998) model, when children display negative emotions, parental supportive reactions (e.g., comforting, redirection, problem-solving) may help children reduce emotion arousal, which may in turn contribute to appropriate emotion expression and competent social behaviors. Our finding supports Eisenberg et al.'s arousal hypothesis at the neural level by linking maternal supportive reactions to a more mature pattern of functional connectivity reflecting vmPFC downregulation of amygdala activation.

With respect to elaborative emotion talk, adolescents showed less positive amygdala-vmPFC connectivity when labeling versus passively observing angry faces, but only when mothers' elaborative emotion talk was high. Emotion labeling is considered an implicit regulatory process and is associated with decreased stress (Lieberman et al., 2011). In this light, the interaction finding suggests that when mothers talk about emotion in an elaborated manner, children may engage in more optimal regulation when labeling versus passively observing emotions. Early experiences of elaborative emotion talk—which combines emotion labeling with rich information about contextual cues, causal reasoning, and providing justification or rationale for emotion labels—may yield a more mature pattern of amygdala-vmPFC connectivity (i.e., downregulation of the amygdala activation) when labeling angry faces because these children may have greater cognitive resources to make sense of and reflect on the emotional stimuli. This pattern of results also converges with prior findings that elaborative emotion talk predicted children's better emotion understanding (Dunn et al., 1991) and behavioral regulation (Laible, 2004) and that child language skills predicted more regulatory skills and less anger in a delay task (Roben, Cole, & Armstrong, 2013).

In contrast to angry faces, our examination of happy faces was exploratory, given limited neural investigations of positive emotional stimuli. One aspect of negative emotion socialization, that is, dominant negative expressiveness, interacted with task condition to predict amygdala-vmPFC connectivity in response to happy faces, and this interaction remained significant in the inclusive model. Specifically, when maternal expression of dominant negative emotions (e.g., anger) was high, adolescents showed marginally more positive amygdala-vmPFC connectivity when labeling versus passively observing happy faces, which indicates less effective implicit regulation in emotion labeling. This finding is consistent with a prior finding that less securely attached children and adolescents showed more positive amygdala-mPFC connectivity when viewing pictures of their mothers' happy faces versus an implicit baseline, which may indicate less competence to downregulate amygdala activation to positive stimuli (Gee et al., 2014). High levels of maternal anger and hostility may undermine the mother-child relationship, which may interfere with development of the amygdala-PFC circuit (Callaghan & Tottenham, 2016), so that adolescents may show less optimal neural regulation. The current findings, as well as Gee et al.'s finding, support such a link for positive emotional stimuli.

Amygdala Activation

Examination of amygdala activation was a secondary aim. For angry faces, maternal supportive reactions predicted higher amygdala activation. In addition, maternal elaborative emotion talk and positive expressiveness each interacted with task condition to predict amygdala activation, such that adolescents showed higher amygdala activation to angry faces in Label versus Observe, but only at high levels of maternal elaborative emotion talk and positive expressiveness, respectively. Recall that our emotion processing task involved images of angry and happy faces that were morphed with neutral faces, yielding more ambiguous yet ecologically valid stimuli compared with more prototypical facial expressions. With this design characteristic in mind, we suspect that adolescents' amygdala activation to angry faces may capture sensitivity to subtle emotional cues and that being asked to label (vs. passively view) such faces may further prompt adolescents to focus on the subtle cues. Early positive emotion socialization experiences, such as maternal talk about emotions and positive expressiveness, may promote ability to detect subtle negative emotions when asked to do so.

Our results for amygdala activation to happy faces were mixed. On the one hand, supportive reactions predicted higher levels of amygdala activation to happy faces. This finding parallels the positive association between mothers' supportive reactions and adolescents' amygdala activation to angry faces, and we speculate that supportive reactions may predict higher neural sensitivity to emotion stimuli regardless of valence. On the other hand, nonsupportive reactions and dominant negative expressiveness also predicted higher levels of amygdala activation to happy faces. In addition, frequency of emotion talk interacted with condition on amygdala activation, such that adolescents showed more amygdala activation when labeling versus passively observing happy faces, but only when frequency of emotion talk was low. These latter findings are somewhat counterintuitive. There may be multiple processes underlying heightened amygdala activation to ambiguous happy faces during adolescence, which may either reflect overall sensitivity to emotion stimuli or adolescents' hyper reactivity to appetitive stimuli. Yet, given that existing neural studies examining parenting and adolescent responses to positively valenced stimuli are limited, caution is needed in strong interpretation of the findings for happy faces.

Limitations and Contributions

This study is not without limitations. First, the sample was predominantly well educated, European American, and middle class. The findings, therefore, may not be generalized to other populations. Second, in line with prior literature on emotion socialization, we focused our investigation on mothers. However, paternal emotion socialization may interact with maternal emotion socialization to predict children's socioemotional functioning (e.g., McElwain, Halberstadt, & Volling, 2007) and may play a unique role in shaping neural circuits underlying adolescents' processing of emotionally salient information. Third, the neuroimaging data were collected about 10 years after the initial phase of the study, and there was sizable attrition, which was largely due to difficulty in reestablishing contact with the families. Notably, however, the families who participated in the adolescent study phase (vs. those who did not) did not differ on any of the emotion socialization variables at 33 months nor on the demographic characteristics, with the exception of child gender. We controlled for child gender in the main models, and as reported in our analyses of missingness, we also found no difference in results when we tested our models using FIML with larger sample sizes that included some missing data. Fourth, although amygdala-vmPFC connectivity may reflect implicit regulatory processes which play an essential role in emotion regulation in daily life (Gyurak et al., 2011), adolescents in our study were not explicitly instructed to regulate their emotions. A promising future direction will be to compare the role of emotion socialization in implicit versus explicit regulation in neural responses to emotions.

Lastly, parental emotion socialization behaviors were not measured during adolescence, and we were thus unable to tease apart the unique contributions of early versus concurrent emotion socialization on adolescent neural functioning. Given that early caregiving experience plays an important role in shaping children's brain development (see Bick & Nelson, 2016) and, in particular, the developing amygdala-PFC circuitry central to emotion regulation (see Callaghan & Tottenham, 2016), maternal emotion socialization in toddlerhood may influence the young child's neural processing and regulation of emotions and those effects may persist into adolescence. Because the adolescent period is also characterized by plasticity of brain circuits involved in emotion regulation (see Ahmed et al., 2015), it is also possible that the quality of maternal emotion socialization is stable across childhood such that concurrent (vs. early) emotion socialization experiences contribute to neural function in early adolescence. In either scenario, however, intervening during early childhood to promote positive maternal emotion socialization behaviors may be an especially effective avenue for supporting development of adaptive neural responses to emotions across childhood and adolescence. Indeed, evidence for an early intervention approach emerged in a recent randomized clinical trial: For families at risk of maltreatment, an intervention to enhance parental sensitivity during toddlerhood facilitated children's more mature patterns of neural function during middle childhood (Bick, Palmwood, Zajac, Simons, & Dozier, 2019). Future intervention studies targeting parental emotion socialization behaviors specifically would shed further light on how caregiving processes contribute to children's brain development and emotion-related neural circuitry.

In summary, findings from our 10-year longitudinal study suggest that multiple and distinct aspects of maternal emotion socialization outlined in Eisenberg et al.'s (1998) framework may set the foundation for neural processing and regulation of emotions in early adolescence. Positive maternal emotion socialization behaviors in toddlerhood predicted less positive amygdala-vmPFC functional connectivity to angry faces, which may reflect more mature PFC downregulation of amygdala activation. This neural pattern is likely to be important for behavioral adjustment and psychological well-being during adolescence when emotional reactivity and the risk of emotion-related psychopathology become heightened. By pinpointing caregiving experiences that contribute to adaptive neural regulation of emotion—that is, responding supportively to children's negative emotions and talking about emotions in an elaborative manner—this line of research can ultimately inform prevention and intervention efforts to foster children's emotional competence and mental health.

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