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Early Temperament and Parental Psychopathology as Predictors of Neural Reactivity to Reward in Middle Childhood

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Abstract of the Dissertation

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Loss of interest or pleasure in previously rewarding experiences is a key feature of depression, and there is growing evidence that reduced reactivity to reward may be a vulnerability marker preceding the onset of depression. Relatively little is known, however, about factors that influence the development of reward sensitivity across childhood, and better understanding developmental trajectories could have important implications for preventing depression. Low positive emotionality (PE), parental history of depression and parenting style have been associated with risk for depression, but associations with reward sensitivity have yet to be examined.

The current study was part of a multi-method prospective study of a large community sample of children. Participants started the study when they were 3 years old and completed laboratory observation measures of temperament and parenting behavior, as well as parent-report measures of parenting style. Approximately 6 years later, when the children were 9 years old,

they returned to the lab to complete a monetary reward task while event-related potentials (ERP) were recorded, and biological mothers and fathers completed a semi-structured interview to assess for history of psychopathology. First, I evaluated whether early child temperament predicted reward sensitivity in middle childhood, as measured by the feedback negativity (FN), an ERP component sensitive to receipt of rewards versus losses. Lower PE at age 3 predicted a reduced FN to rewards and losses at age 9. No effects of negative emotionality or behavioral inhibition on the relative reactivity to rewards versus losses were observed. Next, I evaluated whether parental history of depression and anxiety predicted children's FN. Results indicated that maternal history of depression was associated with a blunted FN in offspring, but only when there was no maternal history of anxiety. Greater severity of maternal depression was also associated with greater blunting of the FN in children. No effects of paternal psychopathology were observed on relative reactivity to rewards versus losses. Lastly, I evaluated whether early parenting behavior and style moderated the effects of parental depression on the FN. Results indicated that maternal positive parenting interacted with maternal and paternal depression, such that blunted reactivity to reward and loss feedback was observable among offspring of parents with histories of depression and low positive parenting by mothers. These findings indicate that both early temperament and parental depression (but not anxiety) may contribute to reduced sensitivity to reward, and maternal parenting moderates the effect of parental depression on reward reactivity. The FN may be an important factor in trajectories from early risk to the onset of depression later in life.

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Chapter 1

Neural Reactivity to Monetary Rewards and Losses in Childhood: Longitudinal and Concurrent Associations with Observed and Self-Reported Positive Emotionality

A number of theorists conceptualize personality and psychopathology in terms of independent biobehavioral motivational systems (Davidson, 1992; Gray, 1994; Lang, Bradley, & Cuthbert, 1990). Each of these models includes a system that is associated with positive affect and reward sensitivity, referred to as the behavioral approach system (Gray, 1994), behavioral activation system (Fowles, 1980), approach system (Davidson, 1992), or behavioral facilitation system (Depue & Iacono, 1989). The approach system is hypothesized to regulate reward-processing, including reward seeking, responsiveness, and learning (Dillon et al., 2013) and generate positive emotions, such as exuberance and joy, which are presumed to facilitate and reinforce appetitive behavior and engagement with rewarding stimuli (Depue & Collins, 1999; Polak-Toste & Gunnar, 2006; Smillie, 2013; Watson, Wiese, Vaidya, & Tellegen, 1999; Wilt & Revelle, 2009). Consistent with this, there is evidence that reward-sensitivity and positive emotionality load on the same latent factor in children and adults (Lucas, Diener, Grob, Suh, & Shao, 2000; Olino, Klein, Durbin, Hayden, & Buckley, 2005). Most theorists assume that the approach system is fundamental to human functioning and survival, and therefore emerges early in development. It is also presumed that there are individual differences in the strength of this system that are responsible for stable variations in reward sensitivity and dispositional positive affect (also referred to as positive emotionality, extraversion, and surgency; Putnam, 2012).

The approach system is typically contrasted with the withdrawal (Davidson, 1992) or behavioral inhibition (Gray, 1994) system. This system is characterized by sensitivity to signals of threat and punishment, and dispositional negative emotionality (NE) and behavioral inhibition (BI; Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Kennis, Rademaker, & Geuze, 2013; Watson et al., 1999). While NE and BI are correlated temperament traits, NE refers to the

tendency to experience a broad range of negative emotions (Clark & Watson, 1999), whereas BI refers specifically to the tendency to be fearful and hesitant in novel situations (Kagan, 1984).

Despite the rich theoretical literature on the approach system, empirical studies examining links between the neural processes associated with reward sensitivity and temperamental positive emotionality (PE) are surprisingly limited (Kennis et al., 2013; Wilt & Revelle, 2009). Much of the existing work has examined resting electroencephalogram (EEG) activity, with adult studies linking greater self-reported approach system sensitivity to greater left prefrontal activation (Sutton & Davidson, 1997; Tomarken, Davidson, Wheeler, & Doss, 1992). More recently, functional magnetic resonance imaging (fMRI) studies of adults and adolescents have suggested that self-reported approach system sensitivity and extraversion are associated with enhanced activation of the ventral striatum and medial orbitofrontal cortex in response to rewards (Cohen, Young, Baek, Kessler, & Ranganath, 2005; Forbes et al., 2010; Kennis et al., 2013; Simon et al., 2010). Given that individual differences in the approach system are presumed to emerge early in life, it is important to evaluate associations across development. However, the literature on reward sensitivity and PE has typically used older adolescents and adults, focused on cross-sectional designs, and not systematically addressed the specificity of associations with inhibition/withdrawal system related-constructs.

The much richer literature on BI provides an instructive contrast, as a number of studies have examined relationships between BI in early childhood and subsequent individual differences in neural reactivity to both threat and reward-related stimuli. For example, children high in BI show enhanced electrocortical measures of error monitoring (McDermott et al., 2009) and increased amygdala reactivity when attending to subjective fear states and novel faces in adolescence and adulthood (Pérez-Edgar et al., 2007; Schwartz, Wright, Shin, Kagan, & Rauch,

2003). Greater right frontal EEG asymmetry, hypothesized to be a marker of withdrawal system sensitivity, has also been linked to BI across development (Fox et al., 2005). Interestingly, however, studies have suggested that early BI is related to striatal activation in response to cues indicating potential for reward and loss (Bar-Haim et al., 2009; Guyer et al., 2006) and in response to negative feedback (Helfinstein et al., 2011). These studies raise the question of whether the link between reward sensitivity and temperamental emotionality is specific to approach system-related affective styles (e.g., PE).

Event-related potentials (ERPs) are relatively inexpensive and non-invasive neural measures that can be used across development (Nelson & McCleery, 2008), and may be employed to assess reward sensitivity. In particular, the feedback negativity (FN) is characterized by a *relative* negativity in response to monetary losses compared to rewards that peaks approximately 250-300 ms over frontocentral recording sites and has been linked to activation in the ventral striatum and medial prefrontal cortex (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Gehring & Willoughby, 2002; Foti, Weinberg, Dien, & Hajcak, 2011; Hajcak, Moser, Holroyd, & Simons, 2006). Though the FN has previously been conceptualized as an enhanced negativity following losses, recent research indicates that it may be better characterized as a relative positivity in response to rewards that is reduced or absent following losses (Foti, Weinberg, et al., 2011; Holroyd, Pakzad-Vaezi, & Krigolson, 2008). The FN is often analyzed as the loss minus gain difference score in order to isolate variation in the waveform attributed to processing of outcome valence (Dunning & Hajcak, 2007; Foti, Kotov, Klein, & Hajcak, 2011; Luck, 2005), and the FN difference score has previously been related to neural reward circuitry using fMRI and to behavioral and self-report measures of reward sensitivity (Bress & Hajcak, 2013; Carlson et al., 2011).

The FN may be an economical and valid measure of reward system sensitivity that can be applied across development. Cross-sectional research in adults has linked an enhanced FN to greater extraversion (Smillie, Cooper, & Pickering, 2011); however, it remains unclear whether approach-related temperament systems such as PE, measured in early childhood, relate to the FN later in development. Also, additional research is needed to evaluate the extent to which associations between the FN in monetary reward tasks are specific to PE, rather than temperament styles linked to the withdrawal system. Recently, several adult studies have examined associations between the FN and self-reported withdrawal/behavioral inhibition system temperament constructs in adults with mixed findings. Santesso and colleagues (2012) found that greater NE predicted an enhanced FN to negative performance feedback in a monetary incentive task. On the other hand, Lange, Leue, and Beauducel (2012) found that adults high in behavioral approach system sensitivity exhibited an enhanced FN when reward feedback changed to non-reward feedback, while people higher in behavioral inhibition system sensitivity showed a reduced FN.

The primary goal of the current study was to evaluate prospective and concurrent associations between the FN and individual differences in PE from early to late childhood. A laboratory observation was used to assess temperament in a large sample of three-year-old children. Approximately six years later, children filled out a self-report measure of temperamental emotionality and completed a monetary reward task while ERPs were recorded. I hypothesized that, consistent with the notion that reward sensitivity and positive affect are aspects of the same stable, early-emerging biobehavioral system, variability in early observed and later self-reported PE would both predict the magnitude of the FN, such that greater PE would be associated with an enhanced FN. Given the low correlations between laboratory and

questionnaire measures of child temperament (Hayden, Klein, & Durbin, 2005; Mangelsdorf, Schoppe, & Buur, 2000), obtaining converging results across both measurement methods would provide particularly powerful support for a link between PE and the FN. Similarly, demonstrating this effect across a six-year period spanning early to late childhood, a period of immense neural and socioemotional change (Giedd & Rapoport, 2010; Monk, 2008), also provides a particularly stringent test. Finally, a secondary aim was to explore the specificity of the PE-FN association by examining whether observed NE and BI at age 3 and self-reported NE at age 9 predict the FN.

Method

Participants

Participants were part of a larger sample (N=559) recruited through a commercial mailing list. Three-year-old children with no significant medical conditions or developmental disabilities and living with at least one English-speaking biological parent were eligible to participate. A subset of 427 children participated in the second assessment approximately six years later. Data from 42 participants were excluded due to poor EEG quality and from 3 participants due to temperament data points that were significant outliers according to Grubbs Test (Grubbs, 1969, p<.05). The final sample included 382 children (45.0% female). With regard to ethnicity, 7.6% of the final sample was of Hispanic or Latino background. The sample was 94.8% Caucasian, 2.9% African American, and 2.4% Asian. The mean age was 3.55 years (SD=0.26) at the first assessment and 9.20 years (SD=0.40) at the second assessment.

Procedure

Parents provided written informed consent prior to the start of each assessment. When the children were approximately three years old, families visited the laboratory for the temperament assessment described below. Families were invited back to the lab as close as possible to the

child's ninth birthday for a battery that included questionnaires and the reward task. During the second visit, EEG sensors were attached and children were told that they could win up to $5 in a guessing task. Children were instructed to press the left or right mouse button to select a door and completed five practice trials prior to beginning the reward task described below. Children also completed a questionnaire assessing trait positive and negative affect (described below).

Measures

Observational Temperament Assessment. At age three, each child and a parent visited the laboratory for an observational assessment of temperament. The assessment included a standardized set of 12 episodes: 11 episodes were from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith, Reilly, Lemery, Longley, & Prescott, 1995) and one was adapted from a Lab-TAB episode. A previous study found moderate stability of laboratory ratings of temperament from ages 3 to 7 and moderate concurrent and longitudinal associations between Lab-TAB ratings and home observations (Durbin, Hayden, Klein, & Olino, 2007). Tasks were videotaped and later coded. No episodes presumed to evoke similar affective responses occurred consecutively, and each episode was followed by a brief play break to allow the child to return to baseline. The parent remained in the room with the child for all episodes except "Stranger Approach" and "Box Empty" but was instructed not to interact with the child (except in "Pop-Up Snakes") and was seated facing at a right angle from the experimenter and child and given questionnaires to complete.

The episodes, in order of presentation, were as follows:

1. "Risk Room." Child explored a set of novel and ambiguous stimuli, including a Halloween mask, balance beam, and black box.

2. "Tower of Patience." Child and experimenter alternated turns in building a tower. The experimenter took increasing amounts of time before placing her block on the tower.

3. "Arc of Toys." Child played independently with toys for 5 min before the experimenter asked the child to clean up.

4. "Stranger Approach." Child was left alone briefly in the room before a male accomplice entered, speaking to the child while slowly walking closer.

5. "Make That Car Go." Child and experimenter raced remote-controlled cars.

6. "Transparent Box." Experimenter locked an attractive toy in a transparent box, leaving the child alone with a set of nonworking keys. After a few minutes, the experimenter returned and told the child that she had left the wrong set of keys. The child used the new keys to open the box and play with the toy.

7. "Exploring New Objects." Child was given the opportunity to explore a set of novel and ambiguous stimuli, including a mechanical spider, a mechanical bird, and sticky soft gel balls.

8. "Pop-Up Snakes." Child and experimenter surprised the parent with a can of potato chips that actually contained coiled snakes.

9. "Impossibly Perfect Green Circles." Experimenter repeatedly asked the child to draw a circle on a large piece of paper, mildly criticizing each attempt.

10. "Popping Bubbles." Child and experimenter played with a bubble-shooting toy.

11. "Snack Delay." Child was instructed to wait for the experimenter to ring a bell before eating a snack. The experimenter systematically increased the delay before ringing the bell.

12. "Box Empty." Child was given an elaborately wrapped box to open under the impression that a toy was inside. After the child discovered the box was empty, the experimenter returned with several toys for the child to keep.

Coding procedures. Each display of facial, bodily, and vocal positive affect, fear, sadness, and anger in each episode was rated on a 3-point scale (*low*, *moderate*, *high*). The positive affect scale was defined as the frequency and intensity of positive verbalizations, smiling and joyful body movements. Ratings were summed separately within each channel (facial, bodily, vocal) across the 12 episodes, standardized, and summed across the three channels to derive total scores for positive affect, fear, sadness, and anger. Interest was rated on a single 4-point scale (*none*, *low*, *moderate*, and *high*) for each episode based on the child's comments about the activity and how engaged the child was in play. Interest ratings were then summed across the 12 episodes. PE consisted of the sum of the standardized positive affect and interest variables. NE was the sum of the standardized sadness, fear, and anger variables.

The coding of BI was designed to capture specific types of behaviors identified by Kagan (1984) and followed Goldsmith's scoring system (Goldsmith et al., 1995; Pfeifer, Goldsmith, Davidson, & Rickman, 2002). BI was coded by dividing the three episodes specifically designed to assess BI ("Risk Room," "Stranger Approach," and "Exploring New Objects") into 20- or 30-s epochs, and rating a series of affective and behavioral codes for each epoch (Goldsmith et al., 1995). Within each epoch, a maximum intensity rating of facial, bodily, and vocal fear was coded on a scale of 0 (*absent*) to 3 (*highly present and salient*). Based on previous studies using the Lab-TAB (Durbin, Klein, Hayden, Buckley, & Moerk, 2005; Pfeifer et al., 2002), BI was computed as the average standardized ratings of latency to fear (reversed); and facial, vocal, and bodily fear ("Risk Room," "Stranger Approach," and "Exploring New Objects"); latency to touch objects; total number of objects touched (reversed); tentative play; referencing the parent; proximity to parent; referencing the experimenter; and time spent playing (reversed; "Risk Room" and "Exploring New Objects"); startle ("Exploring New Objects"); sad facial affect

("Exploring New Objects" and "Stranger Approach"); and latency to vocalize; approach toward the stranger (reversed); avoidance of the stranger; gaze aversion; and verbal/nonverbal interaction with the stranger (reversed; "Stranger Approach").

Most episodes were coded by different raters. PE and NE had adequate internal consistency (α = .82 and .74, respectively) and interrater reliability (intraclass correlations [*ICC*] = .89 and .82, respectively; *N* = 35). BI exhibited good internal consistency (α = .80) and interrater reliability (ICC = .88, *N* = 28). To reduce skewness and kurtosis, a log transformation was applied to NE and its components (sadness, anger, fear) and BI scores. PE was not significantly correlated with NE but was negatively associated with BI. Scoring of BI and NE both include measures of fear-related behavior; thus, BI and NE were moderately correlated (Table 1).

Self-Report Measure of Positive and Negative Emotionality. At the age 9 assessment, children completed the positive and negative affect scales of the Affect and Arousal Scale (AFARS; Chorpita, Daleiden, Moffitt, Yim, & Umemoto, 2000). The AFARS is a child self-report measure designed to assess trait dimensions of the tripartite model of emotion and has demonstrated acceptable test-retest reliability, convergent and discriminant validity (Daleiden, Chorpita, & Lu, 2000). Children are asked to rate how true each item is with respect to their usual feelings from 0 (never true) to 3 (always true). The positive affect scale consists of 10 items and possible scores can range from 0 to 30. The negative affect scale consists of 8 items and scores can range from 0 to 24.

Reward Task. The reward task was administered using Presentation software (Neurobehavioral Systems) and was similar to a version used in previous studies (Dunning & Hajcak, 2007; Foti & Hajcak, 2009). The task consisted of 60 trials, presented in three blocks of

20 trials. At the beginning of each trial, participants were presented with an image of two doors and were instructed to choose one door by clicking the left or right mouse button. The doors remained on the screen until the participant responded. Next, a fixation mark (+) appeared for 1000 ms, and feedback was presented on the screen for 2000 ms. Participants were told that they could either win $0.50 or lose $0.25 on each trial. Wins were worth more than losses because the response to losses is thought to be stronger than the response to wins (Tversky & Kahneman, 1981, 1992), and because this allowed participants to accrue money across the task. A win was indicated by a green "↑," and a loss was indicated by a red "↓." Next, a fixation mark appeared for 1500 ms and was followed by the message "Click for the next round", which remained on the screen until the participant responded and the next trial began. Across the task, 30 gain and 30 loss trials were presented in a random order.

EEG Data Acquisition & Processing. The continuous EEG was recorded using a 34-channel Biosemi system based on the 10/20 system (32 channel cap with Iz and FCz added). Two electrodes were placed on the left and right mastoids, and the electrooculogram (EOG) generated from eye blinks and movements was recorded from facial electrodes: two approximately one cm above and below the left eye, one approximately one cm to the left of the left eye and one approximately one cm to the right of the right eye. The ground electrode during acquisition was formed by the Common Mode Sense and the Driven Right Leg electrodes. The data were digitized at 24-bit resolution with a LSB value of 31.25nV and a sampling rate of 1024 Hz, using a low-pass fifth order sinc filter with -3dB cutoff points at 208 Hz. Off-line analysis was performed using Brain Vision Analyzer software (Brain Products). Data were converted to a linked mastoid reference, band-pass filtered with cutoffs of 0.1 and 30 Hz, segmented for each trial 200 ms before feedback onset and continuing for 1000 ms after onset. The EEG was

corrected for eye blinks (Gratton, Coles and Donchin, 1983). Artifact rejection was completed using semi-automated procedures and the following criteria: a voltage step of more than 50 μV between sample points, a voltage difference of 300 μV within a trial, and a voltage difference of less than .50 μV within 100 ms intervals. Visual inspection was used to remove additional artifacts. Data were baseline corrected using the 200 ms interval prior to feedback. ERPs were averaged across gain and loss trials, and the FN was scored 275-375 ms following feedback at a pooling of Fz, FCz and Cz. Analyses focused on the loss minus gain difference score in order to isolate variation in the waveform attributed to processing of outcome valence (Luck, 2005); more negative values for the difference score indicate greater differentiation in the ERP between gains and losses and greater sensitivity to valence.

Results

Observational Measure of Temperamental Emotionality at Age 3

Bivariate correlations between variables are presented in Table 1. First, a multiple regression analysis was computed with child sex and continuous temperament variables (i.e., PE, NE, and BI) from the observational assessment (i.e., Lab-TAB) at age 3 as predictors and the loss minus gain FN difference score as the criterion variable. Unstandardized and standardized regression coefficients, as well as total model R^2, are presented in Table 2. Sex significantly predicted the loss minus gain difference, with males showing a larger (i.e., more negative) FN, $t(381)=-3.45$, $p<.01$. In addition, observed PE at age 3 significantly predicted the loss minus gain difference in the FN at age 9, $t(381)=-2.24$, $p<.05$, such that higher PE was associated with

greater differentiation between losses and gains (Figure 1)[1]. Observed NE and BI at age 3 did not significantly predict the FN difference score (*p*s>.31).[2]

To evaluate whether the effects of sex and PE were driven by gain or loss trials specifically, I also computed the model with mean amplitudes for loss and gain trials individually as the criterion variables. Males showed an enhanced positivity (i.e., greater reactivity) on gain trials compared to females, β=.14, $t(381)=2.63$, $p<.01$, but the effect of sex on the loss FN was not significant, β=-.01, $t(381)=-.10$, $p>.05$. The effect of PE did not reach significance on either trial type alone (*p*s>.25), but a significant effect of NE on loss trials emerged, β=.14, $t(381)=2.42$, $p<.05$, with greater NE related to a less negative FN on loss trials only.

Self-Reported Temperamental Emotionality at Age 9

Next, multiple regression analyses were computed with child sex and self-reported continuous affectivity variables from the AFARS at age 9 as the predictors and the loss minus gain FN difference score as the criterion variable (Table 2). Self-reported PE at age 9 predicted the difference in the FN, $t(381)=-2.52$, $p<.05$, such that higher PE was associated with greater differentiation between losses and gains. Self-reported NE at age 9 did not significantly predict the FN difference score ($p>.99$).

I also computed the model with the mean amplitudes on loss and gain trials individually as criterion variables and sex and self-reported affectivity variables as predictors. Neither AFARS PE (*p*s>.11) nor AFARS NE (*p*s>.87) significantly predicted the FN on loss or gain

[1]In order to evaluate whether effects of gender moderate the association between PE and the FN, we repeated the model with the addition of the gender X PE interaction in Step 2. The gender X PE interaction was not significant (*p*>.52).

[2] To evaluate whether multicollinearity may contribute to the lack of effects for BI and NE on the FN difference score, we also computed two separate models with BI and PE in one and NE and PE in the other. The effect of PE remained significant in both models, but BI and NE did not significantly predict the FN difference score in either model (*p*s>.18).

trials individually, again suggesting that the associations with PE are specific to relative reactivity to wins vs. losses.

Unique Effects of Observed and Self-Reported PE

Lastly, to evaluate whether observed PE at age 3 and self-reported PE at age 9 have unique effects on the FN, a multiple regression analysis was computed with sex, Lab-TAB PE, and AFARS PE as predictor variables and the loss minus gain FN difference score as the criterion variable (Table 2). Both Lab-TAB PE, $t(381)=-2.52$, $p<.05$, and AFARS PE, $t(381)=-2.58$, $p<.05$, independently predicted the loss minus gain FN difference score.

Discussion

The current study evaluated whether early temperamental emotionality predicts children's electrocortical responses to rewards and losses in later childhood. Results indicated that observed PE in preschool prospectively predicted the FN in late childhood. In addition, child self-reports of PE were concurrently related to the FN. Across time and measurement approaches, children higher in PE showed enhanced electrocortical sensitivity to the difference between wins and losses. These findings are consistent with the hypothesis that PE and reward sensitivity reflect different facets of the same early-emerging biobehavioral approach-related motivational system.

Approach system-related temperament traits have previously been linked to resting EEG asymmetries (e.g., Sutton & Davidson, 1997) and neural processing of rewards as measured by fMRI (Cohen, et al., 2005; Forbes et al., 2010; Simon, et al., 2010); however, to our knowledge, this is the first study to use a longitudinal design to evaluate neural correlates of PE in children. The current results demonstrate that individual differences in approach system-related temperament traits in early childhood predict neural sensitivity to reward in late childhood,

despite enormous developmental changes in both biological and socioemotional systems across childhood (Giedd & Rapoport, 2010; Monk, 2008).

These findings also support the validity of the FN as an index of at least some of the neural processes associated with PE and the approach system. The FN may be useful in exploring a number of domains of developmental research. For example, depression has previously been linked to reductions in the FN (Bress, Smith, Foti, Klein & Hajcak, 2012; Foti & Hajcak, 2009), and attention-deficit hyperactivity disorder (ADHD) has been associated with a disrupted FN response (Holroyd, Baker, Kerns, & Müller, 2008; van Meel, Oosterlaan, Heslenfeld, & Sergeant, 2005). Hence, the FN might be a useful in tracing and understanding developmental trajectories of approach system-related psychopathologies.

Interestingly, observational PE at age 3 and self-reported PA at age 9 were not related to each other but both had unique and comparable effects on the FN. This is consistent with a large literature documenting low associations between laboratory and questionnaire measures of child temperament (Mangelsdorf et al., 2000), and suggests that each measure taps different aspects of approach-related temperament styles that predict reward sensitivity.

I also evaluated the effect of NE and BI on the FN in late childhood. Unlike PE, observed NE in early childhood did not predict the FN difference score in late childhood, though greater observed NE predicted a blunted FN (i.e., less negative) on loss trials only. The association between NE and the FN on loss trials must be interpreted cautiously, as the effect was not apparent for the difference score measure, which has previously been linked to reward sensitivity (Bress & Hajcak, 2013), and the relationship between NE and the FN was not replicated cross-sectionally with child self-report. Nonetheless, this finding is consistent with recent evidence for a reduced FN among adults high in self-reported behavioral inhibition sensitivity (Lange et al.,

2012). As the FN is also modulated by violations in expectation (e.g., Hajcak, Holroyd, & Simons, 2007), it is possible that children who display higher NE expect more negative outcomes, which leads to a blunted FN to monetary losses.

In addition, I did not find an effect of BI on the FN when controlling for PE. Previous fMRI research has found that high BI is associated with increased striatal activation to cues indicating the *possibility* of a negative or positive outcome (Bar-Haim et al., 2009; Guyer et al., 2006). Thus, BI may be uniquely associated with the potential to win or lose, which is consistent with the conceptualization of the behavioral inhibition system as regulating ongoing behavior associated with competing goals (Gray & McNaughton, 1982). To our knowledge, only one previous study has examined BI and neural reactivity to outcomes and found that high BI was associated with increased striatal activation to *negative* feedback (Helfinstein et al., 2011). The current study is among the first to examine multiple temperament constructs in a single study and it is possible that variability in PE may influence the results of previous studies exploring links between NE, BI, and reward-related neural activity.

In the current study, males showed greater electrocortical sensitivity to monetary outcomes than females. This is consistent with questionnaire studies of adults indicating that males report greater reward sensitivity than females (Li, Huang, Lin, & Sun, 2007; Torrubia, Avila, Moltó, & Caseras, 2001), and both laboratory and parent report measures of child temperament indicating that boys exhibit greater surgency than girls (Else-Quest, Hyde, Goldsmith & Van Hulle, 2006; Olino, Durbin, Klein, Hayden, & Dyson, 2013). Our findings suggest that this early-emerging gender difference is observable on the neural, as well as behavioral, level.

The magnitude of the association between PE and the FN was relatively small. As discussed by Patrick et al. (2013), associations between neurophysiological and behavioral measures are typically small in magnitude owing to the substantial difference in methods. In addition, the small associations in the current study are not surprising given the high variability characteristic of young children, the amount of time between assessments (two-thirds of the children's lives), evidence that temperament is less stable in childhood compared to adolescence and adulthood (Roberts & DelVecchio, 2000), and the fact that both temperament and FN were measured in single assessments, making it impossible to tease apart trait from state influences.

The results of the current study indicate that reward-related neural activity in late childhood is prospectively predicted by laboratory observations of temperamental PE in early childhood and concurrently associated with child self-reported PE. These findings contribute to understanding the neural processes associated with PE and suggest that the FN is a valid measure of approach-related system functioning that may be applied across childhood.

Chapter 2

Neural Reactivity to Rewards and Losses in Offspring of Mothers and Fathers with Histories of Depressive and Anxiety Disorders

Abnormalities in neural reward circuitry may be a key feature underlying depressive disorders, particularly the core symptom of anhedonia (Naranjo, Tremblay, & Busto, 2001; Russo & Nestler, 2013). Functional magnetic resonance imaging (fMRI) studies have shown that compared to healthy controls, adults with depression exhibit reduced activation to rewards in striatal regions of the brain and are less likely to adjust their behavior in response to rewards (Eshel & Roiser, 2010; Henriques & Davidson, 2000; Pizzagalli et al., 2009; Pizzagalli, Jahn, & O'Shea, 2005; Smoski et al., 2009; Steele, Kumar, & Ebmeier, 2007). Older children and adolescents with depression also show abnormal reward decision-making in behavioral tasks and reduced striatal responses to reward (Forbes et al., 2009; Forbes et al., 2006).

In order to determine if an abnormality may be a vulnerability marker, rather than simply a concomitant of the disorder, it should be evident prior to onset and in healthy individuals at elevated risk (Ingram & Luxton, 2005). Two behavioral studies have reported that abnormal reward-related decision-making prospectively predicts depression in late childhood and adolescence (Forbes, Shaw, & Dahl, 2007; Rawal, Collishaw, Thapar, & Rice, 2012). In addition, several studies have reported that offspring of depressed parents, a group at elevated risk for depression (Hammen, 2009), exhibit abnormal neural response to reward. Gotlib and colleagues (2010) found that compared to girls with no parental history of depression, never-depressed girls (mean age of 12 years) with maternal histories of recurrent depression showed less activation in putamen and left insula and greater activation in right insula when anticipating rewards. Moreover, McCabe, Woffindale, Harmer, and Cowen (2012) reported that adolescent and young adult offspring of depressed parents exhibited reduced orbitofrontal cortex and anterior cingulate cortex responses to reward.

These studies suggest that abnormalities in response to reward may be a vulnerability marker and precede the onset of depression. However a number of issues remain to be elucidated. First, although abnormal reward processing has been observed in offspring of depressed parents, the influence of parental anxiety has not been examined. Anxiety and depressive disorders often co-occur, with 50% or more depressed adults and youth exhibiting comorbid anxiety disorders (Fava et al., 2000; Kessler, Avenevoli, & Merikangas, 2001; Sanderson, Beck, & Beck, 1990). Moreover, there is substantial overlap in the genetic influences on depressive and anxiety disorders (Hettema, 2008). Nonetheless, reward-related abnormalities may be relatively specific to depression. Low approach-related motivation and positive affect are much more characteristic of depression than anxiety (Kasch, Rottenberg, Arnow, & Gotlib, 2002; Watson & Naragon-Gainey, 2010). In addition, a recent electroencephalogram (EEG) study found evidence of reduced left frontal EEG asymmetry, hypothesized to reflect approach motivation, while anticipating reward in adults with major depressive disorder (MDD) and their relatives, but not in adults with panic disorder and their relatives (Nelson et al., 2013; Shankman et al., 2013). In fact, there is some evidence that anxiety may be associated with *heightened* neural processing of reward under certain conditions. Adolescents with social phobia showed increased striatal activation during anticipation of high magnitude rewards (Guyer et al., 2012). Moreover, increased striatal activation during anticipation of rewards moderated the association between early childhood inhibited temperament and anxiety symptoms in adolescence (Pérez-Edgar et al., 2013). It is unclear, however, whether parental anxiety predicts reward processing in offspring, and if so, whether it influences the association between risk for depression and reward reactivity. Given the distinction between reward processing and depression compared to anxiety,

it is possible that the effect of parental anxiety may attenuate the association between parental depression and reduced reward reactivity in offspring.

Second, little is known about how specific features of parental depression relate to reward processing in offspring. More than half of adults with depression report relatively normal levels of pleasure (Fawcett, Clark, Scheftner, & Gibbons, 1983; Oei, Verhoeven, Westenberg, Zwart, & Van Ree, 1990; Pelizza & Ferrari, 2009), suggesting depression may be heterogeneous with regard to reward reactivity. Melancholia is a subtype of depression that appears to aggregate in families (Schreier et al., 2006) and is characterized by anhedonia and lack of mood reactivity to positive events. Anhedonia is also associated with greater severity of depression (Pelizza & Ferrari, 2009), and severity aggregates in families (Schreier et al., 2006). Thus, it is possible that offspring of parents with melancholic or more severe depression will exhibit particularly diminished reactivity to reward.

Third, most research on reward processing among youth at risk for depression has used samples with a broad range of ages, often spanning middle childhood through adolescence or young adulthood. Yet, there is evidence of developmental changes in reward processing across this range. For example, in one study, ventral striatal reactivity to receipt of rewards increased between ages 10-12 and ages 14-15 (Van Leijenhorst et al., 2010). Although developmental changes in neural systems of reward processing in adolescence have been proposed as a key feature underlying the increased prevalence of depression in adolescence (Davey, Yücel, & Allen, 2008), it is unclear whether abnormal reward processing is associated with risk for depression *before* adolescence. Determining whether this abnormality emerges earlier in development could have important implications for understanding developmental processes in depression and for early identification and intervention.

Previous research has differentiated between two phases of reward processing: the anticipation phase and the feedback, or consummatory, phase (Pizzagalli et al., 2009; Richards, Plate, & Ernst, 2013). The feedback negativity (FN) is an event-related potential (ERP) component that reflects the feedback phase of reward processing. The FN presents as a *relative* negativity following negative outcomes compared to positive outcomes (e.g., rewards) and peaks approximately 250-300 ms after feedback over frontocentral recording sites (Foti, Weinberg, Dien, & Hajcak, 2011; Gehring & Willoughby, 2002; Hajcak, Moser, Holroyd, & Simons, 2006; Santesso, Dzyundzyak, & Segalowitz, 2011). The FN is often examined as the difference between the mean amplitude on loss relative to gain trials to isolate neural sensitivity to outcome valence (Dunning & Hajcak, 2007; Foti et al., 2011). More negative values for the difference score indicate greater differentiation between gains and losses (i.e., increased reactivity), and this difference score has been related to behavioral and self-report measures of reward sensitivity (Bress & Hajcak, 2013), as well as reward-related neural activity using fMRI (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011). Functionally, the FN appears to reflect a reward-related signal used to reinforce behaviors that lead to desirable outcomes (Holroyd & Coles, 2002; Weinberg, Luhmann, Bress, & Hajcak, 2012)

Depressive symptoms have been associated with a reduced FN (i.e., hyposensitivity) to monetary gains vs. losses in older youth and adults (Bress, Smith, Foti, Klein, & Hajcak, 2012; Foti & Hajcak, 2009), and this association appears to be specific to symptoms of depression rather than anxiety (Bress, Meyer, & Hajcak, 2013). In addition, the FN may reflect vulnerability for depression, as a reduced FN to monetary rewards and losses in a small sample of adolescent girls prospectively predicted the development of depressive episodes and increases in depressive symptoms over the next two years (Bress, Foti, Kotov, Klein, & Hajcak, 2013). It remains

unclear, however, if parental depression is associated with a blunted FN in children, and if so, whether it is moderated by parental anxiety.

In order to evaluate the FN as a vulnerability marker for depression, I investigated whether maternal or paternal histories of depressive or anxiety disorders predicted relative reactivity to rewards versus losses in a large community sample of 9-year-old children who had never experienced a depressive episode, while controlling for child symptoms of depression and anxiety. The focus on middle childhood provides insight into whether abnormalities in neural reward systems are already apparent prior to the developmental changes in reward systems and increased prevalence of depression in adolescence (Davey et al., 2008; Van Leijenhorst et al., 2010). I hypothesized that parental depression would be associated with a blunted FN in offspring (i.e., reduced differentiation between rewards and losses). Given the weaker associations of low approach motivation and positive affectivity with anxiety than depression (Shankman et al., 2013; Watson & Narragon-Gainey, 2010), and previous work linking some forms of anxiety to *increased* striatal activation during reward anticipation (Guyer et al., 2012; Pérez-Edgar et al., 2013), I hypothesized that parental anxiety would be associated with a normal or enhanced FN in offspring. In addition, I tentatively posited that the effect of parental anxiety would attenuate the association between parental depression and the FN in offspring, such that a blunted FN would be apparent only among children of parents with pure depression (i.e., without comorbid anxiety). Finally, I conducted exploratory analyses to evaluate whether parental melancholia or depression severity were associated with an even greater reduction of the FN in offspring of depressed parents.

Method

Participants

Participants in the current study are part of a larger prospective longitudinal study (see Olino et al., 2010). The sample was initially recruited using commercial mailing lists when the children were 3 years old (*N*=559). Children with no significant medical conditions or developmental disabilities and living with at least 1 biological, English-speaking parent were eligible to participate. Only 1 child per family was included. Parents and children were re-assessed when the children were approximately six years old, at which time an additional sample of 50 children were recruited through advertisements in the community in order to increase ethnic and racial diversity. The sample was evaluated again at age 9, at which time the reward task was administered. A total of 468 children completed the reward task. Data for 42 children were excluded for poor EEG data quality, and psychopathology data were missing for 8 parents and 1 child. In addition, data were excluded for 6 children due to a parental history of bipolar disorder and 4 children due to a lifetime diagnosis of a depressive disorder. Thus, the final sample in this report consisted of 407 children. The sample was 45% female and mean age at the time of the EEG assessment was 9.18 years (*SD*=.40). With regard to ethnicity, the sample was 11.1% Hispanic. With respect to race, the sample was 89.7% Caucasian, 7.6% African American, and 2.7% Asian.

Procedure

When the children were 3 years old, both biological parents were asked to complete a semi-structured diagnostic interview to assess their own lifetime history of psychopathology. The children were invited back to the laboratory as close as possible to their 9th birthday and completed the EEG assessment. EEG sensors were attached and children were instructed that they could win up to $5 in the reward task. Children then completed 5 practice trials prior to the start of the actual task. Children and parents also completed a semi-structured diagnostic

interview to assess lifetime child psychopathology, and parents completed a semi-structured diagnostic interview assessing their own psychopathology since the initial evaluation.

Measures

Parental psychopathology. When children entered the study at age 3 (or age 6 for the supplementary sample), biological mothers and fathers were interviewed using the Structured Clinical Interview for DSM-IV non-patient version (SCID; First, Spitzer, Gibbon, & Williams, 1996). An advanced doctoral student in clinical psychology and a masters-level clinician conducted the interviews by telephone, which generally yields comparable results to face-to-face interviews (Rohde, Lewinsohn, & Seeley, 1997; Sobin, Weissman, Goldstein, & Adams, 1993). The other rater derived independent diagnoses from recorded interviews; interrater reliability (kappa) was 0.93 for lifetime depressive disorder and 0.91 for lifetime anxiety disorder (n=30). Interrater reliability could not be calculated for melancholia because there were too few cases in the reliability sample.

For the age 9 assessment, advanced doctoral students in clinical psychology and a masters-level clinician administered the SCID to biological mothers and fathers to assess psychopathology in the years since the initial assessment. One parent (generally the mother) was interviewed face-to-face; the other parent was interviewed by telephone. An expert rater with over 15 years of experience administering the SCID derived independent diagnoses from videotaped interviews. Interrater reliability (kappa) was 0.91 for depressive disorders, 0.73 for anxiety disorders, and .067 for melancholia (n=45). According to Landis and Koch (1977), kappas between 0.41 and 0.60 indicate moderate; 0.61–0.80 substantial, and 0.81 and greater almost perfect, agreement. At both assessments, SCID interviewers were supervised individually by a licensed clinical psychologist.

To measure severity of MDD, parents were asked about number of MDD symptoms (range=0-9), impairment, and treatment for their worst episode. Impairment and treatment were each rated on a 3-point scale (0=no significant impairment in major role at home or school, 1=impairment but still able to function, 2=incapacitation in major role; 0=no treatment, 1=outpatient pharmacotherapy or psychotherapy, 2=hospitalization). For each assessment, a composite severity score was calculated as the sum of z-scores for number of MDD symptoms, impairment, and treatment. When parents met criteria for MDD during the intervals covered by both SCID assessments, the higher composite severity score was used for analysis.

In cases where completion of the SCID with one parent was not possible, family history information was obtained from the other parent using a semi-structured interview (Andreasen, Endicott, Spitzer, & Winokur, 1977). Diagnoses for 24 fathers were derived solely using the family history method. Diagnoses from the initial and age 9 diagnostic assessments were combined to yield lifetime diagnoses.

Child psychopathology. At the age 9 assessment, 1 parent (generally the mother) and the child were interviewed using the DSM-IV version of the Schedule of Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime (K-SADS-PL; Axelson, Birmaher, Zelazny, Kaufman, & Gill, 2009). Advanced doctoral students in clinical psychology and a masters-level clinician administered the K-SADS first to the parent and then to the child. Further information was then obtained to reconcile discrepancies. Summary ratings for each symptom were derived based on the combination of parent and child reports. Lifetime symptoms of depressive disorders and anxiety disorders were rated on a 3-point scale (0=Not present; 1=Subthreshold; 2=Threshold) and depression and anxiety items from the screener were summed to create dimensional scores that were used as covariates in the current analyses (depression

dimensional scores could range from 0-16 and anxiety dimensional scores could range from 0-42). The screener was used to compute dimensional scores because other symptoms were assessed only for children with at least 1 symptom in the screener. Administration of the K-SADS was supervised in a group format by an experienced child psychiatrist and licensed clinical psychologist. To assess interrater reliability, a second rater independently derived ratings from videotapes for 74 participants. Intraclass correlations (ICCs) for dimensional scores of depressive and anxiety symptoms were 0.81 and 0.82, respectively. As our goal was to determine whether reward responding abnormalities are present prior to the onset of a depressive disorder, children meeting criteria for lifetime MDD or dysthymic disorder were excluded from analyses (n=4).

Reward task. The reward task was similar to a version used in previous studies (Bress et al., 2012; Dunning & Hajcak, 2007; Foti & Hajcak, 2009). The task consisted of 60 trials, presented in three blocks of 20 trials. At the beginning of each trial, participants were presented with an image of two doors and instructed to choose one door by clicking the left or right mouse button. The doors remained on the screen until the participant responded. Next, a fixation mark (+) appeared for 1000 ms, and feedback was presented on the screen for 2000 ms. Participants were told that they could either win $0.50 or lose $0.25 on each trial. A win was indicated by a green "↑," and a loss was indicated by a red "↓." Next, a fixation mark appeared for 1500 ms and was followed by the message "Click for the next round", which remained on the screen until the participant responded and the next trial began. Across the task, 30 win and 30 loss trials were presented in a random order.

EEG Data Acquisition & Processing. The continuous EEG was recorded using a 34-channel Biosemi system based on the 10/20 system (32 channel cap with Iz and FCz added).

Two electrodes were placed on the left and right mastoids, and the electrooculogram (EOG) from eye blinks and movements was recorded from facial electrodes above and below the left eye, to the left of the left eye and to the right of the right eye. The Common Mode Sense and the Driven Right Leg electrodes served as the ground electrode during data acquisition. The data were digitized at 24-bit resolution with a LSB value of 31.25nV and a sampling rate of 1024 Hz, using a low-pass fifth order sinc filter with -3dB cutoff points at 208 Hz. Off-line analysis was performed using Brain Vision Analyzer software (Brain Products). Data were re-referenced to an average mastoid reference, band-pass filtered with cutoffs of 0.1 and 30 Hz, segmented for each trial 200 ms before feedback onset and continuing for 600 ms after onset. The EEG was corrected for eye blinks (Gratton, Coles, & Donchin, 1983). Artifact rejection was completed using semi-automated procedures and the following criteria: a voltage step of more than 50 μV between data points, a voltage difference of 300 μV within a trial, and a voltage difference of less than .50 μV within 100 ms intervals. Visual inspection was used to remove additional artifacts. Data were baseline corrected using the 200 ms interval prior to feedback. ERPs were averaged across win and loss trials, and analyses focused on the FN difference score, which was calculated as the mean amplitude on loss trials minus the mean amplitude on gain trials. The FN was scored as the mean amplitude 275-375 ms following feedback, where the loss minus gain difference wave was maximal. In order to reduce noise associated with a recording at a single electrode, the FN was scored as the average activity across frontocentral sites (i.e., Fz, FCz, and Cz), which is consistent with previous research (e.g., Bress et al., 2013; Bress et al., 2012) and the scalp distribution of the difference wave.

Results

Participant Characteristics

Table 3 presents demographic variables and associations with maternal and paternal psychopathology. Overall, 38.1% (*n*=155) of children had mothers with lifetime histories of depressive disorders (24.1% MDD, 7.4% dysthymic disorder, 6.6% both) and 17.2% (*n*=70) had fathers with lifetime histories of depressive disorders (10.3% MDD, 2.7% dysthymic disorder, 4.2% both). Rates of current depression in parents were low, with 0.7% of mothers (*n*=3) and 0.5% of fathers (*n*=2) meeting criteria for depression in the past month. With regard to anxiety disorders, 38.8% (*n*=158) of children had mothers with lifetime anxiety disorders and 20.9% (*n*=85) had fathers with lifetime anxiety disorders. Anxiety disorders among mothers included specific phobia (16.7%), social phobia (14.7%), panic disorder (9.8%), generalized anxiety disorder (GAD; 6.1%), post-traumatic stress disorder (PTSD; 5.2%), obsessive compulsive disorder (OCD; 3.2%), and agoraphobia without panic disorder (1.5%). Anxiety disorders among fathers included social phobia (10.1%), specific phobia (5.9%), GAD (4.2%), panic disorder (3.2%), OCD (2.5%), PTSD (1.5%), and agoraphobia without panic (1.2%). In total, 20.8% (*n*=85) of children had mothers with histories of both depression and anxiety and 8.3% (*n*=34) had fathers with histories of both depression and anxiety. In addition, 22.1% (*n*=90) of children had mothers with histories of substance abuse or dependence (12.8% alcohol only) and 44.0% (*n*=179) had fathers with histories of substance abuse or dependence (26.0% alcohol only).

With regard to child psychopathology, 20.6% (*n*=84) of children met criteria for a lifetime diagnosis of an anxiety disorder. In addition, 9.1% (*n*=37) met criteria for lifetime attention deficit hyperactivity disorder (ADHD) and 3.7% (*n*=15) for oppositional defiant disorder (ODD).

Associations between variables are presented in Table 3. Male children showed slightly higher rates of child depressive symptoms than females, though children's level of depressive

symptoms were low overall. Mothers with a history of depressive or anxiety disorders had children with higher rates of anxiety symptoms, but no significant associations between parental psychopathology and offspring depressive symptoms were observed. Higher rates of maternal completion of college were linked to lower child symptoms of anxiety, but higher rates of paternal anxiety disorders. Paternal completion of college was negatively related to maternal depressive and anxiety disorders. No significant effects of parental education on the FN were observed. As would be expected, distributions of maternal and paternal psychopathology differed as a function of other parental diagnoses.

Effects of Parental Depression and Anxiety on the FN

A hierarchical multiple regression analysis was calculated to examine effects on children's FN. All categorical variables were dummy coded as 0 and 1. Child characteristics (i.e., age in months, sex, lifetime depressive symptoms, and lifetime anxiety symptoms) were entered in Step 1, followed by parental psychopathology (i.e., maternal depressive, maternal anxiety, paternal depressive, and paternal anxiety disorder) in Step 2 and interactions between diagnoses in each parent to examine comorbidity in Step 3. Results are presented in Table 4. I initially tested the model including the four cross-parent interactions (i.e., maternal depression X paternal depression, maternal anxiety X paternal anxiety, maternal depression X paternal anxiety, and maternal anxiety X paternal depression). None of the interactions were significant (*p*s>.40); therefore I removed these interactions from the final model to simplify the presentation of results.

The effect of sex was significant, $t(405)=3.43$, $p=.001$, with boys showing larger FNs overall compared to girls. The effects of child age, child depressive symptoms, child anxiety symptoms, maternal anxiety, paternal anxiety and paternal depression were not significant. The

effect of maternal depression was significant in the final model, $t(405)=2.32$, $p=.02$, but was qualified by a significant interaction with maternal anxiety, $t(405)=-2.14$, $p=.03$ (Figures 3 and 4). The interaction between paternal depression and paternal anxiety was not significant.

To interpret the maternal depression by maternal anxiety interaction, dummy coded variables were computed for each of the maternal psychopathology groups (i.e., no depression or anxiety, pure depression, pure anxiety, and comorbid depression and anxiety) and their associations with the FN were compared. Children of mothers with a history of pure depression showed a reduced FN relative to children of mothers with no history of depression or anxiety, $t(405)=2.35$, $p=.02$, and mothers with comorbid depression and anxiety, $t(405)=2.13$, $p=.03$. The comparison between children of mothers with pure depression and mothers with pure anxiety did not reach significance, $t(405)=1.34$, $p=.18$. Children of mothers with no history of depression or anxiety, mothers with histories of pure anxiety, and mothers with comorbid depression and anxiety did not significantly differ from one another (ps>.42).

The main regression analysis was repeated separately on children's mean activity following losses and mean activity following rewards. The effects of parental psychopathology, including the maternal depression X maternal anxiety interaction, did not reach significance in either analysis, suggesting that the effect is apparent in offspring only for the relative response to rewards and losses.

Because the main effect of sex was significant, the hierarchical multiple regression model was repeated with the addition of the two- and three-way interactions between sex, maternal depression and maternal anxiety in the final step. None of the two- or three-way interactions with sex were significant (ps>.58).

The model was also calculated examining current child symptoms of depression and anxiety on the K-SADS (instead of lifetime), as well as child-reported symptoms from the Children's Depression Inventory (CDI; Kovacs et al., 1992) and Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997). No substantive changes in the results were observed with either model. Lastly, to evaluate whether specific parental anxiety disorders predict the FN, I tested an additional model with maternal and paternal depression, specific phobia, social phobia, OCD, PTSD, and GAD in Step 2. None of the effects of specific parental anxiety disorders reached significance (*p*s>.13).

Melancholia and Depression Severity

Additional analyses were computed to evaluate whether maternal melancholic features and MDD severity influence children's FN. These analyses were limited to the 125 children of mothers with a history of MDD. Because child sex was significantly related to their FN in the previous analyses and maternal melancholia predicted greater child symptoms of depression, $r(123)=.18$, $p=.04$, child sex and children's symptoms of depression were included as covariates.

Mothers with a history of MDD were divided into two groups: those with a history of melancholic features ($n=58$) and those with no history of melancholia ($n=67$). A hierarchical multiple regression analysis was calculated with child sex and depressive symptoms in Step 1, and maternal melancholia and maternal anxiety in Step 2. Neither maternal melancholia, $\beta=-.06$; $t(123)=-.63$, $p>.05$, nor maternal anxiety, $\beta=-.15$; $t(123)=-1.76$, $p=.08$, were significantly related to children's FN.

Next, the model was repeated with composite scores for maternal MDD severity replacing melancholia. Greater severity of maternal depression was associated with blunting of

the FN in offspring, β=.19; $t(123)=2.11$, $p=.04$ (Figure 4), and maternal comorbid anxiety was associated with a relatively enhanced FN, β=-.18; $t(123)=-2.06$, $p=.04$.

Lastly, I evaluated whether timing of maternal depression (prior to the child's birth versus during the child's life) influenced the FN. The effect of timing was not significant ($p>.94$).

Discussion

The current findings indicate that the FN to monetary rewards and losses is reduced in children of mothers with a lifetime history of depression, but *only* if the mothers have no history of anxiety disorders. Thus, offspring of mothers with a history of pure depression exhibited significantly smaller FNs than offspring of mothers with no history of depressive or anxiety disorders and offspring of mothers with a history of both depressive and anxiety disorders. Although children of mothers with pure depression also exhibited a smaller FN than children of mothers with pure anxiety, this comparison was not statistically significant. Finally, among offspring of mothers with a history of MDD, greater severity of maternal depression during the worst lifetime episode was associated with greater blunting of children's FN. These results are consistent with previous fMRI studies that found abnormal reward reactivity among offspring of depressed parents (Gotlib et al., 2010; McCabe et al., 2012), but extend this work by using electrocortical measures, focusing on younger children, and examining the effect of parental comorbid anxiety and depression severity on processing of reward feedback.

The FN is thought to reflect reinforcement learning processes (Holroyd & Coles, 2002), and the FN difference score has previously been linked both to self-reported tendency to engage in reward-related behavior and to behavioral measures of bias to make responses that are frequently rewarded (Bress & Hajcak, 2013). Thus, children with a reduced FN may exhibit less

sensitivity to reward or impaired ability to learn contingencies associated with rewarding versus non-rewarding outcomes, and therefore decreased likelihood to engage in rewarding behaviors, which may increase risk for developing depression (Dimidjian, Barrera, Martell, Muñoz, & Lewinsohn, 2011). Consistent with this, it has previously been reported that a reduced FN predicts the onset of MDD in a small sample of adolescent girls (Bress et al., 2013). However, additional prospective studies are needed to determine if a blunted FN in childhood predicts the development of depression and whether abnormalities in the processing of reward feedback mediate the intergenerational transmission of depression.

Our finding that only the offspring of depressed mothers with no history of anxiety exhibited a blunted FN raises the possibility that reduced responding to reward feedback increases risk specifically for pure (without comorbid anxiety) depression. Children with maternal histories of comorbid depression and anxiety, who are at heightened risk for both disorders (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984), exhibited a typical pattern of reward processing. These results are consistent with evidence that depression with and without prominent anxiety have distinct neurobiological profiles (for a review see Ionescu, Niciu, Mathews, Richards, & Zarate, 2013).

In addition, among offspring of mothers with a history of MDD, more severe maternal depression was associated with a particularly blunted FN to rewards versus losses. As greater severity of parental depression is associated with higher rates of depression in offspring (Hammen & Brennan, 2003; Keller et al., 1986), this finding suggests that children with a reduced FN may be a subgroup at particularly high risk. The lack of an association between maternal melancholic depression and the FN in offspring was surprising because anhedonia and loss of mood reactivity to positive experiences are key features of melancholia. However, the

validity of self-reports of melancholic features has been questioned (Parker et al., 1990), and these concerns are compounded when respondents are reporting on past episodes. Thus, our assessment of melancholia may not have been sensitive or specific enough to detect effects of parental melancholic depression on offspring responses to reward.

Importantly, this study is among the first to examine neural measures of reward processing as vulnerability markers for depression prior to adolescence. Developmental changes in brain reward systems may be a key feature underlying the rapid increase in the prevalence of depression beginning in adolescence (Davey, Yücel, & Allen, 2008). Our findings suggest that children at high risk for depression may begin to show abnormalities in the processing of reward feedback even before adolescence.

It should be noted that the current results were specific to the loss minus gain FN difference score, which has been used in previous FN studies to isolate neural activation specific to the valence of feedback (Bress & Hajcak, 2013; Carlson et al., 2011; Dunning & Hajcak, 2007; Foti et al., 2011). It is possible that reactivity to only loss or gain trials could drive effects; however, I did not find any significant effects of parental psychopathology on loss or gain trials when examined separately.

In contrast to effects of maternal depression, I did not find significant effects of paternal depression on sensitivity to rewards versus losses in offspring. This is consistent with our previous findings with this sample at earlier ages indicating that maternal, but not paternal, depression was associated with reduced neural reactivity to emotional faces and poorer emotion recognition skills in offspring (Kujawa et al., 2014; Kujawa, Hajcak, Torpey, Kim, & Klein, 2012). It is also consistent with the weaker effects of paternal depression on child psychopathology (Brennan, Hammen, Katz, & Le Brocque, 2002; Connell & Goodman, 2002). It

is important to note, however, that the rate of depression in adult males is lower than in females (Nolen-Hoeksema, 2002); consequently, our sample included a larger number of mothers than fathers with depression. In addition, diagnoses for a small proportion (5.9%) of fathers were derived using family history interviews, which have only moderate sensitivity (Andreasen et al., 1977). Thus, it is possible that the lack of effects for paternal depression is due to methodological factors.

Other limitations of the current findings should be noted. The effect of maternal depression on the FN in offspring was relatively small; the entire model including covariates accounted for only 5% of the variance in the FN. However, small effects would be expected for several reasons. First, as discussed by Patrick et al. (2013), associations between neurophysiological and behavioral measures are typically small in magnitude owing to the substantial difference in methods. Second, as many offspring of depressed mothers will not develop depression and a number of offspring of mothers with no history of depression will become depressed (Hammen, 2009), the effect sizes of maternal depression on offspring psychopathology are generally small (see meta-analysis by Goodman et al., 2011). Finally, depressive disorders are characterized by etiological heterogeneity and equifinality; hence, as our findings suggest, abnormal reward processing is likely to be evident in only a subgroup of individuals with, or at risk for, depression.

In addition, the comparison between offspring of mothers with pure depression and pure anxiety did not reach significance, though the direction of means was consistent with a reduced FN among the pure depression group. It is important to note that I did not find evidence of an enhanced FN among offspring of mothers with pure anxiety disorders. Though previous research has linked anxiety to increased striatal activation during anticipation of large rewards (Guyer et

al., 2012; Pérez-Edgar et al., 2013), it is possible that this effect is specific to *anticipation* of reward and is not captured by the FN, which reflects the feedback phase of reward processing. Also, the effects of anxiety may only be apparent when there is greater variability in the magnitude of reward and loss outcomes.

The current study did not find elevated rates of depressive symptoms in children of depressed parents; however, consistent with previous studies, maternal depression was associated with an increased level of anxiety symptoms in offspring (Warner, Mufson, & Weissman, 1995; Weissman et al., 2006). The lack of association between parental depression and child depressive symptoms is likely due to the low level of depressive symptoms in our sample owing to the young age of the children and the fact that I excluded those who had already developed depressive disorders.

Lastly, in the current study, males showed greater reactivity to rewards versus losses than females. In self-report studies of adults, males tend to report greater reward sensitivity (Li, Huang, Lin, & Sun, 2007; Torrubia, Avila, Moltó, & Caseras, 2001), and our study extends that finding to neural measures in children. It has been suggested that there may be sex differences in vulnerability factors prior to adolescence that contribute to the sharper rise in rates of depression in females than males following puberty (Hyde, Mezulis, & Abramson, 2008; Nolen-Hoeksema & Girgus, 1994). Reduced neural sensitivity to rewards and losses may represent an example of a sex difference in a vulnerability factor that is evident prior to adolescence. However, additional research is needed to evaluate this possibility.

In conclusion, this is the first study to directly compare the effects of parental depression and anxiety on reward processing in offspring. The offspring of mothers with a history of depression but no history of anxiety exhibited blunted neural reactivity to reward and loss

feedback. Moreover, among children of depressed mothers, greater severity of maternal depression was related to greater blunting of the FN. As these children are several years away from entering the period of risk for depression and have never experienced a depressive episode, these findings suggest that the FN to reward feedback may be a vulnerability marker that precedes the onset of some forms of depression. The processes responsible for these effects may involve genetic influences or interactions with mothers who also have abnormal reward processing. Regardless, these results highlight the importance of considering comorbidity between anxiety and depression and depression severity in examining neural correlates and vulnerability markers.

Chapter 3

Early Parenting Moderates the Association between Parental Depression and Neural Reactivity to Rewards and Losses in Offspring

The ability to respond to feedback is essential in navigating the world and adjusting behavior to achieve desired outcomes. There is evidence that the neural systems that process and adjust behavior in response to feedback continue to develop across childhood and into adolescence (Peters, Braams, Raijmakers, Koolschijn, & Crone, 2014). In addition, deficits in the ability to respond effectively to feedback, particularly feedback indicating reward, appear to be key psychobiological processes underlying depressive disorders in adults and youth (Eshel & Roiser, 2010; Forbes et al., 2009; Forbes et al., 2006; Henriques & Davidson, 2000; Pizzagalli et al., 2009; Pizzagalli, Jahn, & O'Shea, 2005; Smoski et al., 2009; Steele, Kumar, & Ebmeier, 2007), and may play a role in the pathogenesis of depression (e.g., Bress, Foti, Kotov, Klein, & Hajcak, 2013; Gotlib et al., 2010; Kujawa, Proudfit, & Klein, in press; McCabe, Woffindale, Harmer, & Cowen, 2012).

Parental depression is associated with heightened risk of depression in offspring (Hammen, 2009), thus, vulnerability markers and intermediate phenotypes for depression should be evident among children of depressed parents. Consistent with this, offspring of depressed parents exhibit abnormalities in the neural processing of reward (Gotlib et al., 2010; Kujawa et al., in press; McCabe et al., 2012). In particular, we recently demonstrated that children of mothers with histories of depression, but not anxiety, exhibit a blunted feedback negativity (FN) event-related potential (ERP) component in response to monetary rewards and losses (Kujawa et al., in press).

While parental depression is associated with greater vulnerability for depressive disorders in offspring, pathways to depression are marked by both equifinality and multifinality, as not all people who develop depression have a parental history of depression and some offspring of

depressed parents never experience a depressive episode themselves. Thus, research evaluating moderating variables is essential for understanding the transmission of depression across generations (Goodman, 2014; Goodman & Gotlib, 1999). Similarly, it may be important to consider moderating influences on potential intermediate phenotypes for depression, such as reward processing. Consistent with this, we previously found that the effect of maternal depression on neural reactivity to reward in offspring was moderated by maternal history of anxiety disorders (Kujawa et al., in press).

Parenting is another factor that may moderate the association of parental depression with depression and intermediate phenotypes in offspring. There is evidence that low parental warmth, as well as high parental intrusiveness and rejection, are related to depressive symptoms in youth both cross-sectionally and longitudinally (for a review, Klein, Kujawa, Black, & Pennock, 2013). On the other hand, authoritative parenting, characterized by warmth, non-punitive discipline and consistency, has been associated with positive outcomes in adolescents, including lower depression (Milevsky, Schlechter, Netter, & Keehn, 2007). In addition, problematic parenting, including low warmth and high psychological control and over-involvement, has been shown to moderate the effect of maternal depression on psychopathology and social functioning in offspring (Brennan, Le Brocque, & Hammen, 2003).

It is plausible that early parenting contributes to depression in offspring by shaping vulnerabilities, such as reward processing. Indeed, there is some evidence that early parenting and maltreatment predict neural activation during reward anticipation across development. For example, functional magnetic resonance imaging (fMRI) studies indicate that child maltreatment and severe early deprivation are associated with reduced basal ganglia activation during reward anticipation later in life (Dillon et al., 2009; Mehta et al., 2010; Pechtel & Pizzagalli, 2011).

Work examining less extreme forms of problematic parenting is more limited; however, a recent study found an effect in the opposite direction, with low maternal warmth associated with *enhanced* striatal and medial prefrontal cortex activation during reward anticipation among a sample of adolescent girls (Casement et al., 2014).

To our knowledge, only one previous study has evaluated the effects of both parenting and maternal depression on neural reactivity to reward in offspring. Using fMRI, Morgan, Shaw, and Forbes (2014) examined interactions between maternal depression and early maternal warmth in prospectively predicting neural reactivity to reward among young adult males recruited for a study of the development of antisocial behaviors. Among offspring of mothers with a history of depression, low maternal warmth in early childhood was associated with an *enhanced* striatal response during anticipation and receipt of reward, while low maternal warmth in early adolescence was associated with *reduced* striatal activation during reward anticipation.

Existing studies examining effects of parenting on reactivity to reward have focused either on extreme forms of problematic parenting or on maternal warmth; it remains unclear if other dimensions of parenting (e.g., intrusiveness and control, hostility, permissiveness) also have effects. In addition, studies of the influence of parenting on reward processing in offspring have focused exclusively on maternal parenting (Casement et al., 2014; Morgan et al., 2014), and have not examined the impact of parenting by fathers. Compared to paternal psychopathology, maternal psychopathology is more strongly linked to internalizing problems in young children, an effect which has been attributed to greater maternal involvement in child rearing, particularly early in the child's life (Connell & Goodman, 2002). We previously failed to find effects of paternal depression on the FN to rewards and losses in offspring (Kujawa et al., in press);

however, it is possible that effects of paternal depression may be more apparent when examined in conjunction with parenting behavior.

In the current study, we extend our previous work (Kujawa et al., in press) by examining the effects of parenting both alone and as moderators of parental depression effects on the feedback negativity (FN). Observational and self-report measures of parenting were obtained in a large group of preschool-aged children from the community. Observational measures focused on maternal parenting behavior and included both positive (i.e., support, confidence, and instruction) and negative (i.e., hostility and intrusiveness) behaviors. Self-report measures were completed by both mothers and fathers and assessed authoritative, authoritarian and permissive parenting styles (Baumrind, 1971). Approximately six years later, when the children were nine years old, we completed a follow-up assessment in which children participated in an electroencephalogram (EEG) assessment of reactivity to reward and loss feedback to measure the FN. The FN is an ERP component that is observable approximately 250-350 ms after feedback over frontocentral recording sites as a *relative* negativity to monetary losses compared to rewards (Foti, Weinberg, Dien, & Hajcak, 2011; Gehring & Willoughby, 2002) and has been linked to reward-related neural activity, including ventral striatum and medial prefrontal cortex, using fMRI (Becker, Nitsch, Miltner, & Straube, 2014; Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011). At both assessments, mothers and fathers completed diagnostic interviews to assess lifetime histories of psychopathology. Because there is some evidence that blunted reward reactivity is relatively specific to depression rather than anxiety (Bress et al., 2013; Kujawa et al., in press; Shankman et al., 2012), and anxiety may be associated with *heightened* reward reactivity in some conditions (Guyer et al., 2012; Pérez-Edgar et al., 2013), I controlled for both parental and child anxiety in all analyses.

In light of evidence that maternal depression has a greater impact on offspring risk than paternal depression (Connell & Goodman, 2002), the first aim was to evaluate whether early maternal parenting behavior and style predict the FN in mid-late childhood and whether maternal parenting moderates the effect of maternal depression on the FN. A second, more exploratory, aim was to examine the effects of paternal parenting on the FN, alone and in conjunction with paternal history of depression. As mothers may have greater involvement than fathers in early parenting (Connell & Goodman, 2002), a third, also exploratory, aim was to examine whether *maternal* parenting moderates the association between *paternal* depression and the FN.

Method

Participants

Participants were part of a larger sample (*N*=559) recruited through a commercial mailing list. At the initial assessment, three-year-old children with no significant medical conditions or developmental disabilities and living with at least one English-speaking biological parent were eligible to participate (Olino, Klein, Dyson, Rose, & Durbin, 2010). A subset of 426 children who completed at least one of the parenting assessments at age 3 also participated in the electroencephalogram (EEG) assessment approximately six years later. Data from 42 participants were excluded due to poor EEG quality. Because the aim of the study was to identify early markers of risk for unipolar depression, 5 children with a parental history of bipolar disorder, 3 children missing data on parental mood disorders and 5 children who had already experienced a lifetime depressive episode were excluded from analyses. Lastly, data from 26 children were excluded because of parental reports of a significant learning or developmental disability at the follow up assessment. The final sample consisted of 345 children (55.9% male). At the initial assessment of parenting, the mean child age was 3.63 (*SD*=.29), and at the follow up assessment, the mean age was 9.18 (*SD*=.40). With regard to ethnicity, 7.5% of the sample was Hispanic, and

with regard to race, 94.2% was Caucasian, 3.2% was African American, and 2.6% was Asian. With regard to parental education, 59.7% of mothers and 49.0% of fathers had completed college.

Procedure

This study was approved by the Stony Brook University Institutional Review Board. All parents provided written informed consent and children provided verbal assent. The initial assessment took place when the children were approximately three years old. At this time, the child and one parent completed an observational measure of parenting behavior (described below). In addition, parents completed self-report measures of parenting style and semi-structured interviews to assess their own lifetime histories of psychopathology. Participants were invited back to the laboratory as close as possible to the child's ninth birthday, at which time the monetary reward ERP task was administered, parents completed another diagnostic interview assessing their own psychopathology, and children and the primary parent participated in a semi-structured interview assessing lifetime child psychopathology.

Measures

Observational measure of maternal parenting behavior. At the age 3 assessment, the child and one biological parent participated in a parent-child interaction assessment of parenting behavior based on the Teaching Tasks (TT) battery (Egeland et al., 1995). The battery includes six standardized tasks (e.g., book reading, block building) designed to elicit parent and child behaviors. Trained coders rated videotapes of each episode for parental hostility (parent's expression of anger, frustration, annoyance, discounting or rejection of the child), intrusiveness (parent's failure to respect the child as an individual, or interference with the child's needs, interests or behaviors), confidence (degree to which the parent seems to believe that she can

work successfully with the child and that the child will behave appropriately), supportive presence (parent's expression of positive regard and emotional support to the child) and quality of instruction (parent's ability to structure the situation so that the child understands the objective and is able to attempt the task). All ratings were on a 5-point scale, with the exception of confidence, which was rated on a 3-point scale. To reduce skewness and kurtosis, hostility and confidence were dichotomized into a 2-point scale for each episode. All ratings were averaged across tasks. Interrater reliability was acceptable for all variables (ICCs=.59-.85, n = 55; Cicchetti, 1994). A negative parenting composite score was computed by combining standardized scores on hostility and intrusiveness ((α = .69; Kujawa et al., 2014), and a positive parenting composite score was computed by combining standardized scores on confidence, support and instruction (α = .85). Five children were missing TT data (but parents completed self-report measures of parenting) and one participant was a significant outlier on positive parenting (p<.01) according to Grubb's test (1969) and was excluded from analyses. Because I was interested in examining maternal and paternal parenting behavior separately and almost all TT batteries (91.9%) were completed by mothers, data from 23 participants in which the father completed the TT were also excluded from analyses of observed parenting behavior.

Self-report measures of maternal and paternal parenting style. At the age 3 assessment, the Parenting Styles and Dimensions Questionnaire (PSDQ; Robinson, Mandleco, Olsen, & Hart, 2001) was administered to biological mothers (PSDQ-M) and biological fathers (PSDQ-F). The PSDQ is a 37-item self-report measure designed to assess three major parenting styles: authoritative (high control, high warmth), authoritarian (high control, low warmth), and permissive (low control, high warmth). Items are rated from 1 (*never*) to 5 (*always*). Data were missing for 25 mothers and 76 fathers.

Parental psychopathology. We have previously described the procedures for assessing parental psychopathology and provided a detailed analysis of the effects of parental depression on the FN in offspring (Kujawa et al., in press). In summary, biological mothers and fathers completed the Structured Clinical Interview for DSM-IV non-patient version (SCID; First, Spitzer, Gibbon, & Williams, 1996) at both the age 3 and age 9 assessments. The SCID was administered by advanced doctoral students in clinical psychology and masters-level clinicians. Diagnoses from the age 3 and age 9 assessments were combined to yield lifetime diagnoses. When one biological parent was unavailable to complete the SCID, family history information was obtained from the other parent using a semi-structured family history interview (Andreasen, Endicott, Spitzer, & Winokur, 1977). It was not possible to directly interview 13 fathers using the SCID, thus diagnoses were derived using the family history method. Data on anxiety disorder were missing for 1 of these fathers.

Monetary reward task. A detailed description of the monetary reward EEG task and EEG data acquisition/processing is provided in Kujawa et al. (in press). Participants were instructed to guess which door on the computer screen had a prize behind it and were told that they would earn $0.50 or lose $0.25. The task consisted of 60 trials (30 gain, 30 loss) presented in a random order. At the start of each trial, participants were presented with two doors and pressed a mouse button to select one of the doors. Feedback was presented for 2000 ms, with a win indicated by a green "↑," and a loss indicated by a red "↓."

EEG data acquisition and processing. EEG was recorded using a 34-channel Biosemi system based on the 10/20 system (32 channel cap with Iz and FCz). Electrodes were placed on the left and right mastoids, and the electrooculogram was recorded from four facial electrodes. Data were referenced to an average mastoid reference, band-pass filtered with cutoffs of 0.1 and

30 Hz, segmented for each trial 200 ms before feedback onset and continuing for 600 ms after onset. The EEG was corrected for eye blinks (Gratton, Coles, & Donchin, 1983), and artifact rejection was completed using semi-automated procedures with the following criteria: a voltage step of more than 50 μV between data points, a voltage difference of 300 μV within a trial, and a voltage difference of less than .50 μV within 100 ms intervals. Visual inspection was used to remove additional artifacts. ERPs were averaged across win and loss trials and baseline corrected. The FN was scored as the mean amplitude 275-375 ms following feedback at a pooling of Fz, FCz, and Cz. Consistent with previous work (Bress & Hajcak, 2013; Carlson et al., 2011; Kujawa et al., in press), results focused on ΔFN, which was calculated as the difference between mean amplitude on loss trials and mean amplitude on gain trials (i.e., more negative difference scores indicate greater differentiation between rewards and losses).

Child psychopathology. At the age 9 assessment, a parent and the child completed the DSM-IV version of the Schedule of Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime (K-SADS; Axelson, Birmaher, Zelazny, Kaufman, & Gill, 2009). Advanced doctoral students in clinical psychology and a masters-level clinician administered the K-SADS first to the parent and then to the child, with further information obtained to reconcile discrepancies as needed. Lifetime symptoms were rated on a 3-point scale (0=Not present; 1=Subthreshold; 2=Threshold). Summary ratings for depression and anxiety items from the screener were summed to create dimensional scores that were used as covariates in the current analyses (depression dimensional scores could range from 0-16 and anxiety dimensional scores could range from 0-42). To assess interrater reliability, a second rater independently derived ratings from videotapes of 74 participants. Intraclass correlations (ICCs) for dimensional scores of depressive and anxiety symptoms were 0.81 and 0.82, respectively.

Data Analysis

Hierarchical multiple regression analyses were computed to examine the main and interactive effects of parenting behavior and parental depression on ΔFN to monetary gains and losses. First, I evaluated main and interactive effects of maternal parenting behavior and maternal depression. Next, I evaluated main and interactive effects of paternal parenting behavior and paternal depression, and lastly, I evaluated interactive effects of maternal parenting behavior and paternal depression. Continuous variables were centered to examine interactions. Child characteristics (sex, anxiety symptoms, depressive symptoms)[1] were entered into step 1, followed by parent variables (maternal or paternal lifetime diagnoses of depression and anxiety, observed or self-reported parenting)[2] in step 2 and interactions between parental depression and parenting in step 3.

Results

Participant Characteristics and Associations between Variables

Demographic characteristics and correlations between all variables are presented in Table 5. We previously reported a detailed description of rates of parental psychopathology (Kujawa et al., in press). In the current study, 36.8% of mothers and 16.8% of fathers made criteria for a lifetime depressive disorder (MDD or dysthymia). In addition, 35.7% of mothers and 21.5% of fathers met criteria for a lifetime anxiety disorder. With regard to child diagnoses, 5.5% met criteria for lifetime separation anxiety disorder, 3.8% for social phobia, 10.1% for specific phobia, 0.3% for panic disorder, 0.6% for agoraphobia, and 3.5% for generalized anxiety

[1] We also evaluated the models controlling for child symptoms of disruptive behavior disorders. No substantive changes in the results were observed.
[2] We also evaluated the models controlling for parental completion of college and history of substance use disorders. No substantive changes in the results were observed.

disorder. In addition, 6.4% of children met criteria for lifetime attention-deficit hyperactivity disorder (ADHD) and 2.9% for oppositional defiant disorder (ODD).

With regard to associations between variables, higher child depression symptom scores were associated with greater TT negative parenting, lower TT positive parenting, lower PSDQ-M authoritative parenting, and higher PSDQ permissive parenting by both mothers and fathers. Both higher child anxiety symptoms and maternal anxiety diagnoses were related to higher PSDQ-M permissive parenting. Maternal depression was related to greater PSDQ authoritarian parenting by mothers and fathers, as well as greater PSDQ-F permissive parenting. Paternal depression was related to greater PSDQ-F authoritarian parenting, and paternal anxiety was related to greater PSDQ-M authoritative parenting. TT positive parenting was modestly but significantly related to lower PSDQ-M authoritarian and permissive parenting, and TT negative parenting was related to greater PSDQ-M permissive style. No child symptoms, parental diagnoses or parenting variables correlated with the FN, with the exception of PSDQ-F authoritarian, which was associated with an enhanced FN in offspring.

Maternal Depression and Maternal Parenting Behavior

First, a hierarchical multiple regression analysis was computed to examine effects of maternal depression and observed maternal parenting behavior from the TT on children's ΔFN. As previously demonstrated (Kujawa et al., in press), males showed an enhanced FN compared to females, $t(314)=2.95$, $p<.01$, and the effects of child anxiety and depressive symptoms on the FN did not reach significance ($ps > .17$). None of the main or interactive effects of TT positive parenting, TT negative parenting, maternal anxiety or maternal depression reached significance ($ps > .41$) in the observed parenting behavior model.

Next, the analysis was repeated using self-report measures of maternal parenting style from the PSDQ. Final unstandardized and standardized regression coefficients, as well as R^2 for each step and the total model, are presented in Table 6. In the self-reported maternal parenting style model, the main effect of PSDQ-M authoritarian parenting reached significance at entry, β=.12, $t(318)=-1.99$, $p<.05$, but was no longer significant after the interactions were entered, $p=.35$. The interaction between maternal depression and PSDQ-M authoritative parenting style was significant, $t(318)=-2.46$, $p<.05$. To interpret the interaction, the model was computed at each level of maternal depression. For offspring of mothers with a history of depression, lower PSDQ-M authoritative parenting was associated with a reduced FN, β=-.20, $t(114)=-2.08$, $p<.05$ (Figures 5 and 6). For offspring of mothers with no history of depression, the effect of PSDQ-M authoritative parenting was not significant, β=.09, $t(202)=1.32$, $p>.05$. Main and interactive effects of PSDQ-M authoritarian and permissive parenting styles were not significant (*p*s>.25).

Paternal Depression and Paternal Parenting Behavior

Next, a hierarchical multiple regression analysis was computed to examine effects of paternal depression and self-report measures of paternal parenting style from the PSDQ on children's ΔFN. PSDQ-F authoritarian was significant in step 2, β=-.13, $t(266)=-1.97$, $p<.05$, but none of the main or interactive effects of paternal depression, paternal anxiety, PSDQ-F authoritarian, PSDQ-F authoritative, or PSDQ-F permissive parenting style reached significance in the final model (*p*s>.07).

Paternal Depression and Maternal Parenting Behavior

Lastly, two models were computed to evaluate whether maternal parenting behavior (TT and PSDQ) interacted with paternal depression to predict ΔFN. In the observed parenting model, the interaction between TT positive parenting and paternal history of depression was significant,

$t(313)=-2.19$, $p<.05$ (Table 7). To interpret the interaction, the model was computed separately for children with and without paternal histories of depression. For offspring of fathers with a history of depression, less positive maternal parenting behavior was associated with a blunted FN, $\beta=-.50$, $t(49)=-2.20$, $p<.05$. For offspring of fathers with no history of depression, the effect of maternal positive parenting behavior on the FN was not significant, $\beta=.06$, $t(262)=0.76$, $p>.05$ (Figure 5).

In the maternal self-report model, maternal authoritarian parenting behavior was associated with a blunted FN, $\beta=.13$, $t(317)=2.06$, $p<.05$, prior to controlling for the interactions. None of the main or interactive effects reached significance in the final model ($ps>.13$).

Discussion

In the current study, I evaluated whether observational and self-reported measures of early parenting prospectively moderated the effects of maternal and paternal depression on reactivity to reward and loss feedback in offspring. Results indicated that among offspring of mothers with histories of depression, low self-reported maternal authoritative parenting style in early childhood predicted a blunted FN (i.e., reduced reactivity to reward versus loss feedback) in middle childhood. In addition, among offspring of fathers with histories of depression, low observed maternal positive parenting behavior (i.e., support, confidence and instruction) in early childhood predicted a blunted FN in middle childhood. Thus, across multiple measures, results indicate that children of parents with histories of depression are particularly sensitive to the effects of low maternal positive parenting behavior and styles in shaping abnormal processing of reward and loss feedback. These results are consistent with previous work finding blunted neural activation during anticipation of reward among children with histories of maltreatment and early deprivation (Dillon et al., 2009; Mehta et al., 2010; Pechtel & Pizzagalli, 2011), and extend a

previous study evaluating maternal depression and maternal warmth in predicting young men's neural activation to reward (Morgan et al., 2014).

We have previously demonstrated that maternal depression is associated with a blunted FN in offspring but only among offspring of mothers without comorbid anxiety disorders (Kujawa et al., in press). The current study builds on this finding by suggesting that the combination of parental histories of depression and low maternal positive parenting are also associated with reduced reward reactivity. Importantly, the moderating effect of comorbid anxiety does not account for the current findings, as maternal anxiety was not related to observed positive parenting or self-reported authoritative parenting, which were both found to interact with parental depression. Children of parents with histories of depression may be at increased risk of exhibiting deficits in reward responding due to genetic predisposition and modeling of parental reactions to environmental stimuli. The current results indicate that this effect is apparent only among children whose mothers exhibit low support, warmth and structure. In cases of low positive and authoritative maternal parenting, at-risk children may have greater difficulty learning how to respond to environmental contingencies and to shape behavior accordingly. This, in turn, may increase the likelihood of developing depression later in life, as abnormal reward responding has previously been shown to predict depression (Bress et al., 2013; Forbes, Shaw, & Dahl, 2007; Rawal, Collishaw, Thapar, & Rice, 2012). Importantly, these results were specific to positive aspects of parenting, rather than negative parenting (e.g., hostility, intrusiveness, and punitive discipline), and findings apply to both maternal parenting behavior, referring to specific observable behaviors in the moment, and parenting style, which reflects both attitudes and reports of behavior across contexts and time.

While no significant interactions were observed between paternal depression and paternal behavior, *maternal* observed positive parenting significantly moderated the effect of *paternal* depression on the FN. We have previously failed to find effects of paternal depression on the FN and other early markers of emotional processing biases in children (Kujawa et al., 2014; Kujawa, Hajcak, Torpey, Kim, & Klein, 2012; Kujawa et al., in press), which is consistent with smaller effects of paternal compared to maternal psychopathology on internalizing symptoms in young offspring (Connell & Goodman, 2002). However, the current results suggest that effects of fathers' depression on their children's FN are dependent on the quality of mothers' parenting behavior, and indicate that maternal parenting may buffer the negative effects of paternal depression on reward reactivity.

Bivariate correlations indicated that greater self-reported paternal authoritarian parenting style was associated with an *enhanced* FN response to reward and losses; however, this effect no longer reached significance when controlling for interactions with depression. As authoritarian parenting is characterized by low warmth, this finding could be consistent with recent research indicating the low maternal warmth predicted *heightened* striatal reactivity during reward anticipation (Casement et al., 2014). However, as the effect of authoritarian parenting did not interact with risk for depression, this finding could suggest a pathway from greater authoritarian parenting by fathers to externalizing behavior, which may also be characterized by a disrupted FN (Holroyd, Baker, Kerns, & Müller, 2008; van Meel, Oosterlaan, Heslenfeld, & Sergeant, 2005). Importantly, however, the results of the current study remained the same when controlling for symptoms of disruptive behavior disorders in children. I also observed a main effect of maternal authoritarian parenting predicting a *blunted* FN, suggesting that overcontrolling and rejecting maternal parenting may also contribute to reduced reactivity to reward in offspring.

However, this effect was not observed in bivariate correlations or in the final regression models after controlling for the interactions with maternal depression and must be interpreted cautiously.

Several strengths of the current study should be noted. First, both maternal and paternal histories of depression and reports of parenting style were assessed, whereas much of the existing literature focuses only on maternal psychopathology and parenting. I also looked at whether paternal depression is moderated by the effect of maternal parenting style. In addition, the interaction effects between positive parenting behavior and parental depression converged across constructs (parenting behavior vs. style), methods (observation vs. questionnaire), and parents (both maternal and paternal depression). Lastly, the study included a relatively large sample and prospective design, indicating that the effects of early maternal parenting relate to reward processing in offspring across a large portion of childhood, which could provide insight into trajectories from early risk to the onset of depression in later life.

Nonetheless, there are also several limitations to the current study. First, I did not find a significant effect of maternal depression and observed maternal parenting on the FN. This was surprising, as the interaction was apparent with maternal-report measures of parenting. In addition, the observational measure of parenting behavior was limited to mothers, and self-report measures of parenting style were missing for a large proportion of fathers (22%). Thus, it is possible that methodological factors contributed to the lack of significant effects of paternal parenting.

This study is the first to evaluate interactions between maternal and paternal depression and parenting behavior in predicting neural reactivity to reward versus loss across development. Blunted reward reactivity was observed among offspring of parents with histories of depression and lower maternal authoritative and positive parenting. Future research is needed to evaluate

whether abnormal reward processing mediates associations of parental depression and early parenting with the development of depression in adolescence and young adulthood.

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Table 1. *Bivariate associations between child temperament variables and the FN*

	Gender	Lab-TAB PE	Lab-TAB NE	Lab-TAB BI	AFARS PA	AFARS NA	Loss-Gain FN
Lab-TAB PE	-.08	-	-	-	-	-	-
Lab-TAB NE	-.09	-.01	-	-	-	-	-
Lab-TAB BI	-.16**	-.23***	.44***	-	-	-	-
AFARS PA	-.20***	.00	-.07	-.02	-	-	-
AFARS NA	-.02	-.01	.07	.01	-.07	-	-
Loss-Gain FN	-.18**	-.11*	.08	.10*	-.09	.01	-
M(SD)	-	.09(1.76)	.55(.25)	.63(.20)	24.71 (3.95)	6.23 (3.43)	-4.56(7.48)
Range	-	-5.09-6.09	0.00-1.39	0.00-1.37	10.00-30.00	0.00-19.00	-23.39-15.76
% Male	55.0%						

***$p < .001$;**$p < .01$;*$p < .05$

Table 2. *Multiple regression analyses predicting age 9 FN from observational and self-report measures of temperament across development.*

Predictor	Loss-Gain Difference Score FN	
	b(SE)	β
Model 1 – Observational Measures of Temperament (Age 3)		
Gender	-2.65(.77)	-.18***
Lab-TAB PE	-.50(.22)	-.12*
Lab-TAB NE	1.67(1.66)	.06
Lab-TAB BI	.94(2.18)	.03
	Total R^2=.05, F(4,377)=5.10, p<.01	
Model 2 – Self-Report Measures of Temperament (Age 9)		
Gender	-3.03(.77)	-.20***
AFARS PA	-.25(.10)	-.13*
AFARS NA	.00(.11)	.00
	Total R^2=.05, F(3,378)=6.23, p<.001	
Model 3 – Observational & Self-Report Measures Combined		
Gender	-3.17(.76)	-.21***
Lab-TAB PE	-.54(.21)	-.13*
AFARS PA	-.25(.10)	-.13*
	Total R^2=.06, F(3,378)=8.45, p<.001	

***p< .001; **p <.01; *p < .05

Table 3. *Bivariate correlations between study variables for parental psychopathology and the FN.*

Variable	Mean(*SD*)/ %	1	2	3	4	5	6	7	8	9	10	11
1. Age	9.18(0.40)	-										
2. Sex (% female)	44.5%	-.01	-									
3. Race (% Caucasian)	89.7%	.07	.09	-								
4. Child depression	0.41(1.00)	.00	-.11*	.00	-							
5. Child anxiety	3.39(3.58)	.02	-.02	-.03	.14**	-						
6. Mother education	55.8%[b]	-.04	.00	.14**	-.08	-.12*	-					
7. Father education	43.5%[b]	-.01	-.08	.13*	-.06	-.09	.39***	-				
8. Maternal depression	38.1%	.01	.02	.03	.06	.22***	-.03	-.12*	-			
9. Maternal anxiety	38.8%	.00	.01	.06	.05	.20***	-.10†	-.11*	.26***	-		
10. Paternal depression	17.2%	-.02	.00	-.08	.05	.06	.06	-.07	.11*	.09	-	
11. Paternal anxiety	20.9%	.01	-.02	.04	-.08	-.02	.16**	.05	.17**	.05	.31***	-
12. Loss-Gain FN	-4.44(7.61)	.03	.17**	.02	-.04	.04	.03	-.02	.05	-.02	-.05	-.05

***p<.001; **p<.01; *p<.05; †p=.05

[a] Pearson's correlations were calculated to examine associations involving at least 1 continuous variable; phi coefficients were calculated to evaluate associations between 2 categorical variables

[b] Percentage of parents completing 4-year college or higher

Table 4. *Hierarchical regression with child characteristics, parental psychopathology and interactions between parental depression and anxiety predicting the FN.*

Predictor	b(SE)	β
1. Child Characteristics	R^2=.03, F(4, 402)=3.33, p<.05	
Age (months)	.05(.08)	.03
Sex	-2.54(.76)	.17**
Child depression symptoms	-.17(.38)	-.02
Child anxiety symptoms	.07(.11)	.03
2. Parental Psychopathology Variables	Change R^2=.01, F(4, 398)=0.82, p>.05	
Maternal Depression	2.52(1.09)	.16*
Maternal Anxiety	.89(1.06)	.06
Paternal Depression	-1.32(1.34)	-.07
Paternal Anxiety	-1.13(1.16)	-.06
3. Interactions	Change R^2=.01, F(2, 396)=2.45, p=.09†	
Maternal Depression X Maternal Anxiety	-3.43(1.61)	-.18*
Paternal Depression X Paternal Anxiety	1.15(2.15)	.04
	Total model R^2=.05, F(10, 396)=2.16, p<.05	

**p<.01; *p<.05

†When the non-significant paternal depression X paternal anxiety interaction is removed from Step 3, R^2 change is significant (p<.05)

Table 5. *Bivariate correlations between study variables for parenting behavior and style, parental depression and anxiety and the FN.* (continued on next page)

Variable	Mean(*SD*)/ %	1	2	3	4	5	6	7	8	9	10
1. Sex (% male)	55.9%	-									
2. Child depression	.26(.69)	.11	-								
3. Child anxiety	3.25(3.41)	.03	.25***	-							
4. Maternal depression	36.8%	-.01	.05	.20***	-						
5. Maternal anxiety	35.7%	-.02	.06	.19**	.25***	-					
6. Paternal depression	16.8%	.01	.04	-.01	.12*	.09	-				
7. Paternal anxiety	21.4%	.05	-.07	-.04	.19**	.08	.29***	-			
8. TT Positive	0.00(2.52)	.03	-.11*	.00	-.06	.02	-.04	.01	-		
9. TT Negative	0.00(1.65)	.02	.15**	-.02	.00	.05	.04	-.09	-.61***	-	
10. PSDQ-M Authoritative	60.97(6.41)	-.10	-.12*	.03	.03	-.05	.05	.11*	.03	-.03	-
11. PSDQ-M Authoritarian	20.06(4.25)	.04	.06	-.01	.13*	.07	.06	-.03	-.16**	.09	-.15*
12. PSDQ-M Permissive	10.74(3.21)	.01	.15**	.12*	.09	.23***	.04	-.01	-.19**	.20**	-.11*
13. PSDQ-F Authoritative	56.39(8.18)	.04	-.05	.00	-.02	-.07	.09	.06	-.02	.07	.26***
14. PSDQ-F Authoritarian	20.56(4.68)	.02	.08	-.07	.16**	.10	.14*	.02	-.01	.07	-.14*
15. PSDQ-F Permissive	11.29(3.39)	-.03	,14*	,12	.14*	.02	.07	.03	-.17**	.13*	-.02
16. ΔFN	-4.71(7.28)	-.17**	-.07	.02	.01	-.01	-.05	-.06	-.03	.03	.01

***p<.001; **p<.01; *p<.05

Table 5 (Continued).

Variable	11	12	13	14	15
1. Sex (% male)					
2. Child depression					
3. Child anxiety					
4. Maternal depression					
5. Maternal anxiety					
6. Paternal depression					
7. Paternal anxiety					
8. TT Positive					
9. TT Negative					
10. PSDQ-M Authoritative					
11. PSDQ-M Authoritarian	-				
12. PSDQ-M Permissive	.44***	-			
13. PSDQ-F Authoritative	.03	.04	-		
14. PSDQ-F Authoritarian	.29***	.18**	-.26***	-	
15. PSDQ-F Permissive	.23***	.36***	-.01	.29***	-
16. ΔFN	.07	-.06	.04	-.13*	.00

***p<.001; **p<.01; *p<.05

Table 6. *Hierarchical regression with maternal psychopathology and maternal parenting predicting the FN*

Predictor	b(SE)	Final β
1. Child characteristics	R^2=.04, F(3, 316)=3.90, p<.01	
Sex	-2.61(.83)	-.18**
Child depression symptoms	-.39(.42)	-.05
Child anxiety symptoms	.06(.12)	.03
2. Mother variables	Change R^2=.02, F(5, 311)=1.00, p>.05	
Maternal depression	.08(.89)	.01
Maternal anxiety	.20(.90)	.01
PSDQ-M authoritative	.10(.08)	.09
PSDQ-M authoritarian	.13(.14)	.08
PSDQ-M permissive	-.18(.18)	-.08
3. Interactions	Change R^2=.02, F(3, 308)=2.49, p=.06	
Maternal Depression X Authoritative	-.33(.13)	-.17*
Maternal Depression X Authoritarian	.20(.22)	.08
Maternal Depression X Permissive	-.25(.30)	-.07
	Total model R^2=.07, F(11, 308)=2.22, p<.05	

**p<.01; *p<.05;

Table 7. *Hierarchical regression with paternal psychopathology and maternal parenting predicting the FN.*

Predictor	b(SE)	Final β
1. Child characteristics	R^2=.04, F(3, 311)=3.79, p<.05	
Sex	-2.40(.81)	-.17**
Child depression symptoms	-.44(.44)	-.06
Child anxiety symptoms	.07(.12)	.03
2. Parent variables	Change R^2=.00, F(4, 307)=0.31, p>.05	
Paternal depression	.04(1.14)	.00
Paternal anxiety	-.83(1.06)	-.05
TT positive parenting	.18(.22)	.06
TT negative parenting	.27(.33)	.06
3. Interactions	Change R^2=.02, F(2, 305)=2.48, p=.09	
Paternal Depression X Positive Parenting	-1.29(.59)	-.21*
Paternal Depression X Negative Parenting	-1.29(.96)	-.13
	Total model R^2=.06, F(9, 305)=1.95, p<.05	

**p<.01; *p<.05

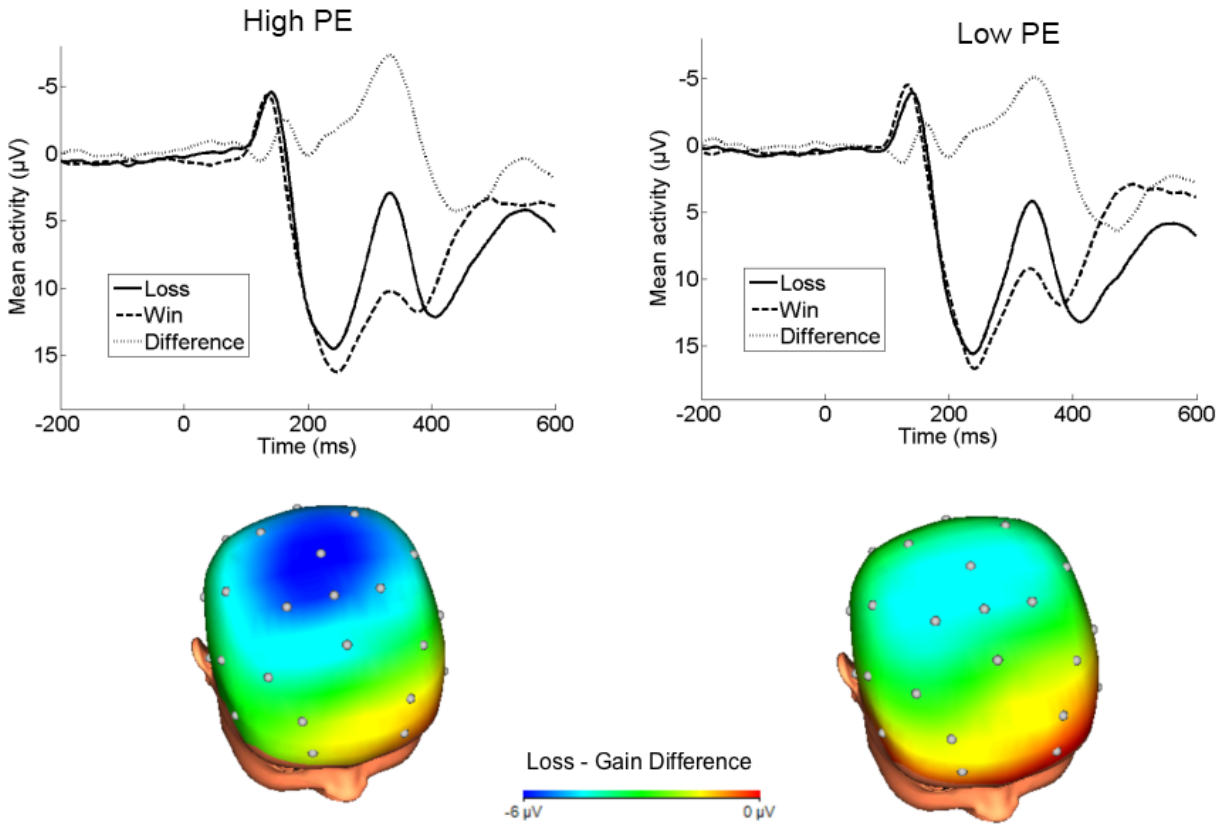

Figure 1. ERPs (negative up) at Fz/FCz/Cz following feedback and scalp distributions depicting the loss minus gain difference 275-375 ms after feedback for children high and low in PE on the Lab-TAB at age 3. Note: A median split of PE was used for illustrative purposes. Analyses used continuous measures of temperament.

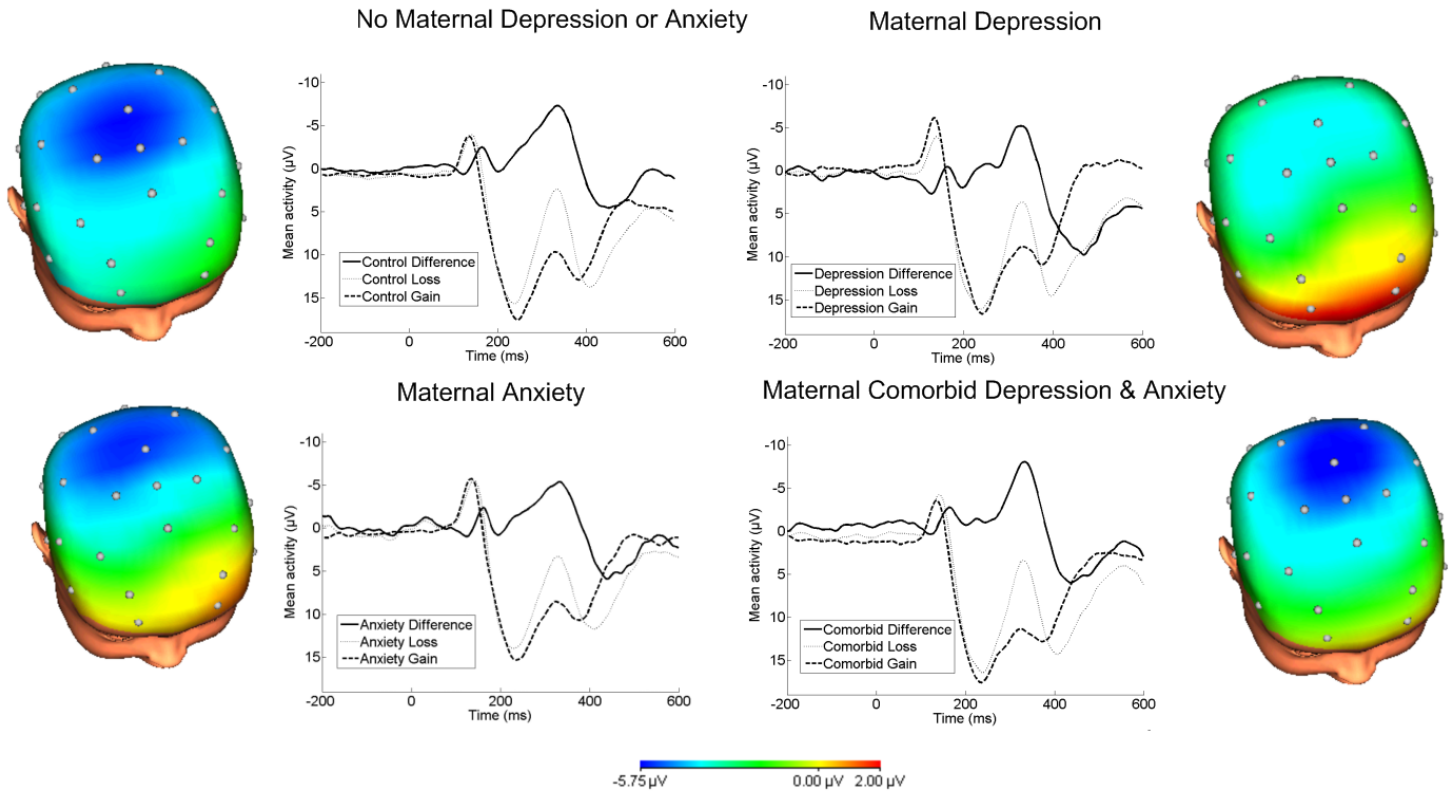

Figure 2. ERPs (negative up) at FCz for losses, gains and the difference, and scalp distributions depicting the loss-gain difference for children with no maternal history of anxiety or depression (top left), maternal depression only (top right), maternal anxiety only (bottom left), and maternal comorbid depression and anxiety (bottom right).

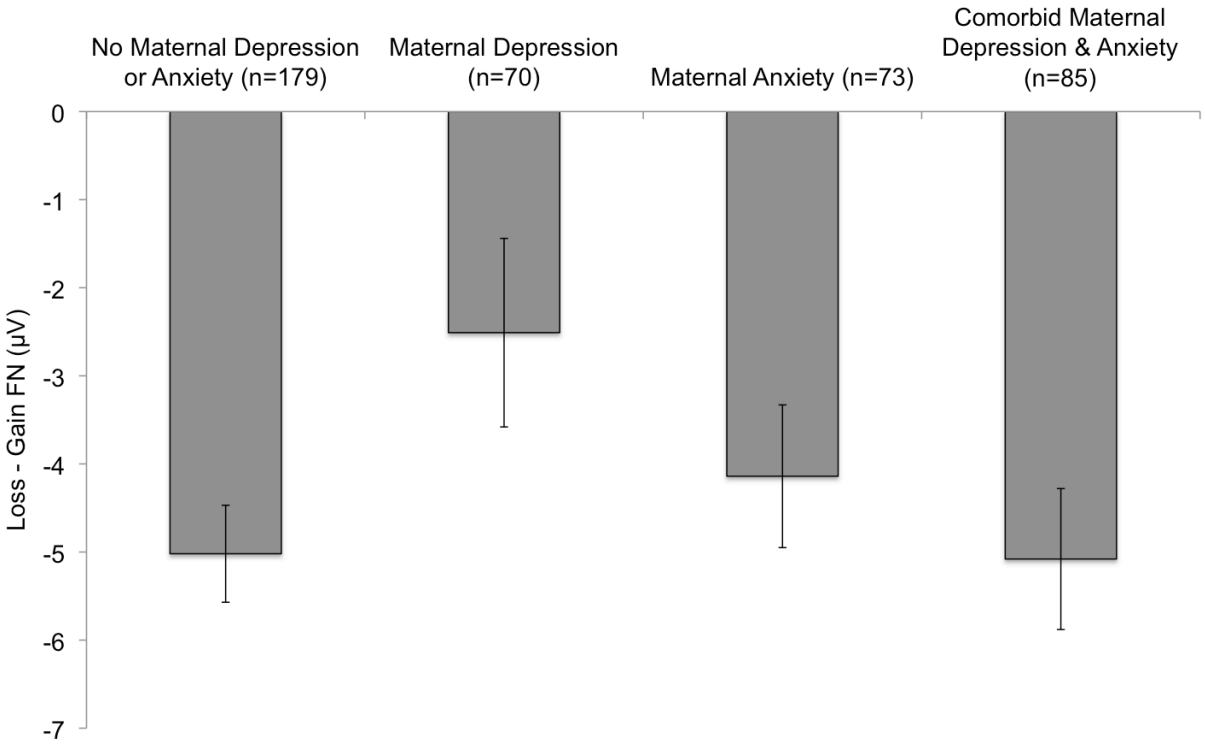

Figure 3. Bar graph depicting means and standard errors of the loss minus gain FN difference score at the Fz, FCz, and Cz pooling for each of the maternal depression/anxiety groups.

Figure 4. Scatter plot depicting the association between maternal MDD severity and the loss minus gain FN difference score.

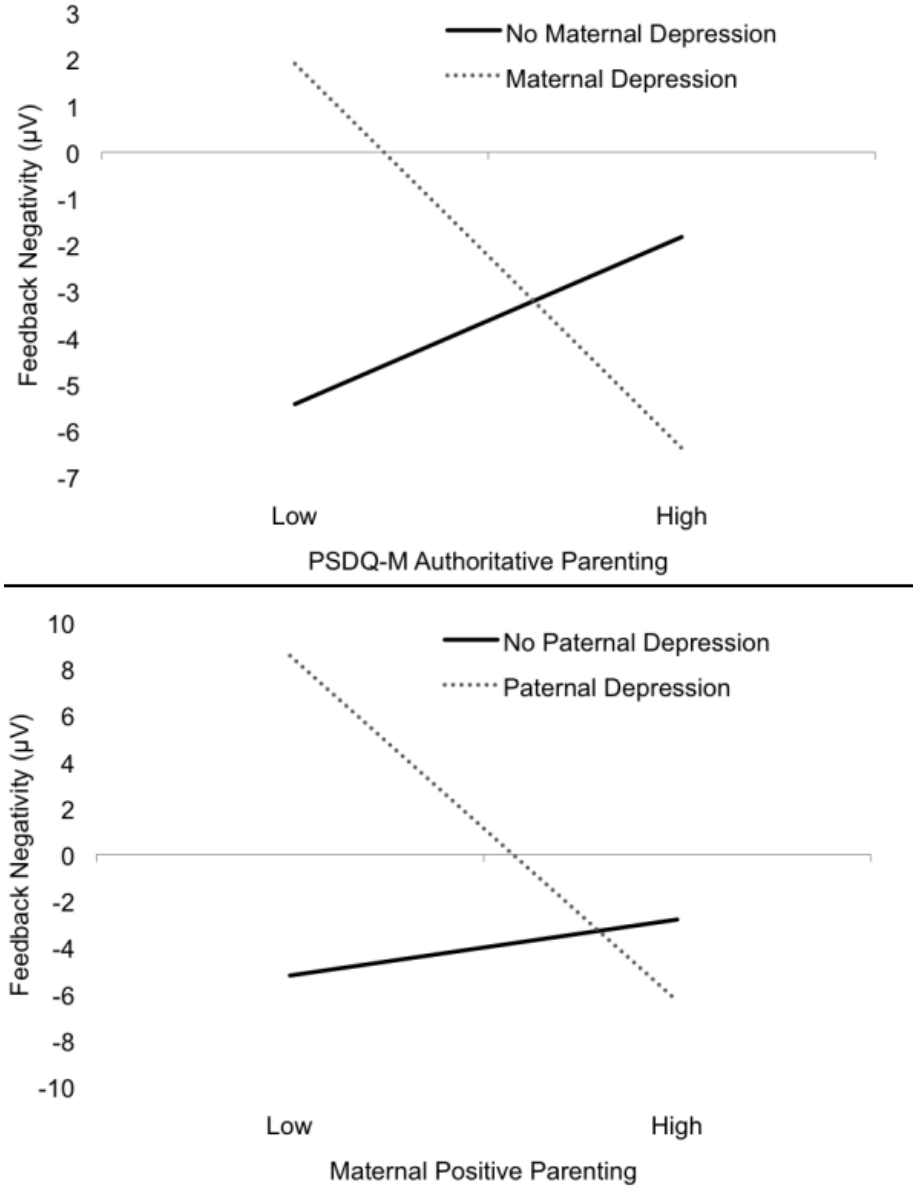

Figure 5. The interaction between maternal depression and maternal-reported authoritative parenting style (PSDQ-M) in predicting ΔFN in offspring (top), and the interaction between paternal depression and observed maternal positive parenting (TT) in predicting ΔFN in offspring (bottom). Note: Parental depression is plotted as the moderating variable because it is dichotomous; however, our hypotheses focus on parenting moderating the effects of parental depression.

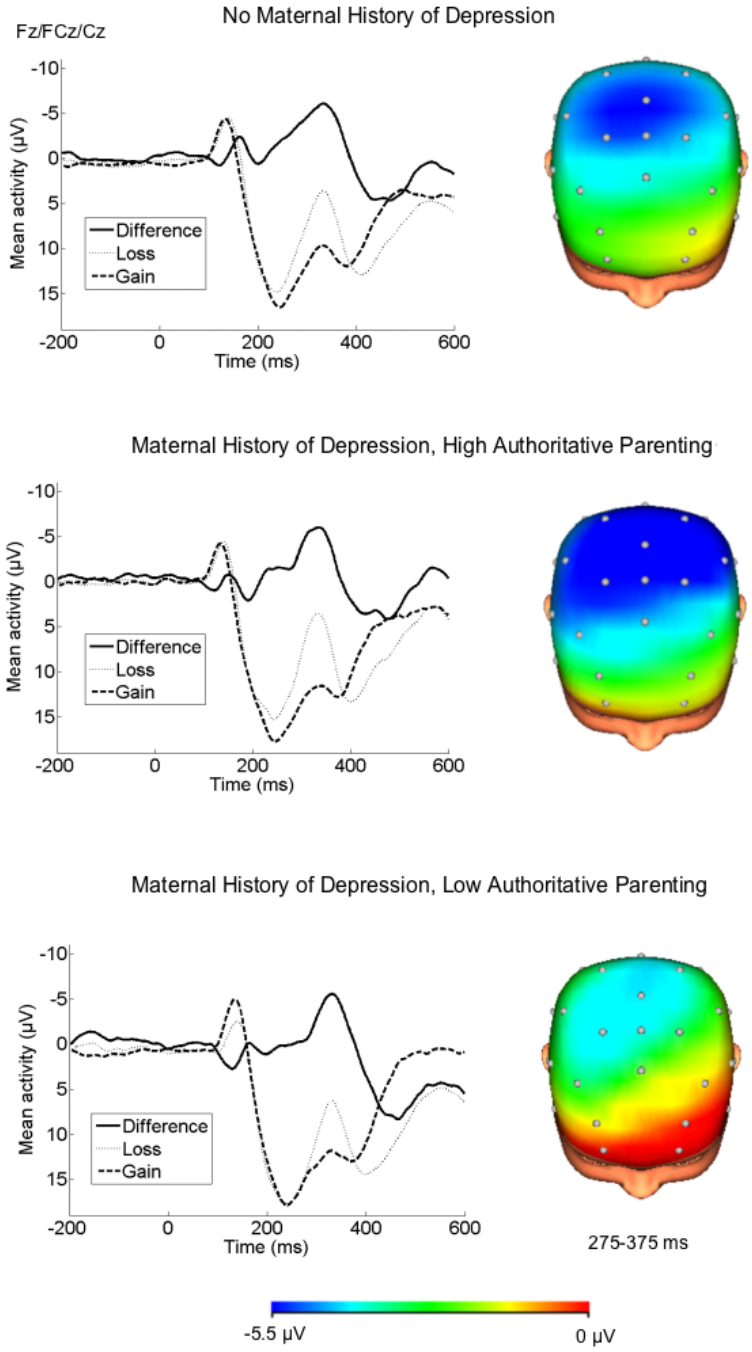

Figure 6. ERPs (negative up) and scalp distributions depicting the loss-gain difference score for children with no maternal history of depression, children with a maternal history of depression and high PSDQ-M authoritative parenting, and children with a maternal history of depression and low PSDQ-M authoritative parenting. Note: A median split of PSDQ-M authoritative parenting was used for presentation purposes.