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Maternal criticism and children's neural responses to reward and loss

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ABSTRACT

Parental criticism is linked to a number of detrimental child outcomes. One mechanism by which parental criticism may increase risk for negative outcomes in children is through children's neural responses to valenced information in the environment. The goal of the current study, therefore, was to examine the relation between maternal criticism and children's neural responses to monetary gains and losses. To represent daily environmental experiences of reward and punishment, we focused on reactivity to monetary gains versus losses in a guessing task. Participants were 202 children and their mothers recruited from the community. The average age of the children was 9.71 years (*SD* = 1.38, range = 7–11), with 52.0% of them male and 72.8% Caucasian. Mothers completed the Five Minute Speech Sample to assess expressed emotion–criticism, and of these dyads 51 mothers were rated as highly critical. In addition, children completed a simple guessing game during which electroencephalography was recorded. Children of critical mothers displayed less neural reactivity to both monetary gain and loss than children without critical mothers. Our results were at least partially independent of children's and mothers' current levels of internalizing psychopathology. These findings suggest that children exposed to maternal criticism may exhibit disruptions in adaptive responses to environmental experiences regardless of

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valence. Targeted interventions aimed at reducing expressed emotion–criticism may lead to changes in a child's reward responsiveness and risk for psychopathology.

Introduction

Early parenting styles have wide-reaching implications for child development in areas such as behavior regulation, social competence, cognitive functioning, symptoms of internalizing and externalizing disorders, and self-injurious behaviors (e.g., Carson & Parke, 1996; Crowell et al., 2008; Hadwin, Garner, & Perez-Olivas, 2006; Hollenstein, Granic, Stoolmiller, & Snyder, 2004; Otowa, Gardner, Kendler, & Hettema, 2013; von Suchodoletz, Trommsdorff, & Heikamp, 2011; Weaver, Shaw, Crossan, Dishion, & Wilson, 2014). Specific components of parenting behavior, such as parental criticism, are linked to a number of these detrimental child outcomes (Burkhouse, Uhrlass, Stone, Knopik, & Gibb, 2012; Harris & Howard, 1984; Hooley & Gotlib, 2000; James & Gibb, 2019; Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990; Sheeber, Hops, & Davis, 2001; Silk et al., 2009; Wedig & Nock, 2007). One index of parental criticism is expressed emotion, a measure of the affective climate within a family. Indeed, expressed emotion–criticism (EE–criticism) specifically reflects the extent to which a parent expresses criticism of, or hostility toward, his or her child (Hooley, 1985, 2007). The construct of EE–criticism is commonly measured using the Five Minute Speech Sample (FMSS; Magaña et al., 1986), during which parents are asked to speak about their child and their relationship with their child for an uninterrupted 5 min. Numerous studies demonstrate a link between ratings of high EE–criticism on the FMSS and levels of criticism, hostility, and anger observed by independent raters during actual parent–child interactions, supporting the construct validity of this approach (Cruise, Sheeber, & Tompson, 2011; Hermanns, Florin, Dietrich, Rieger, & Hahlweg, 1989; Kim Park, Garber, Ciesla, & Ellis, 2008; McCarty, Lau, Valeri, & Weisz, 2004; Narayan, Herbers, Plowman, Gewirtz, & Masten, 2012; Rea & Shaffer, 2016). In addition to the potentially detrimental outcomes described above, several studies demonstrate a link between parental EE–criticism and negative outcomes in youths, including psychopathology and self-injurious thoughts and behaviors (Butzlaff & Hooley, 1998; Hack & Martin, 2018; James & Gibb, 2019; Michelson & Bhugra, 2012; Santos, Saraiva, & de Sousa, 2009; Silk et al., 2009; Wedig & Nock, 2007).

Despite evidence supporting a link between EE–criticism and these detrimental child outcomes, little is known about specific mechanisms through which risk may be conferred. One potential mechanism of risk is a child's neural response to valenced information in the environment. Indeed, across development people use information from, and experiences with, their environment (e.g., previous rewarding and punishing experiences) to inform their future decision making and behaviors (for a review, see Nussenbaum & Hartley, 2019). These reinforcement learning processes are associated with normative and adaptive development, including the development of social relationships (e.g., Jones et al., 2014). Building from this line of inquiry, there is preliminary support for differential neural responses to environmental cues such as maternal criticism, maternal praise, and neutral statements (Aupperle et al., 2016; Hooley et al., 2009; Hooley, Gruber, Scott, Hiller, & Yurgelun-Todd, 2005; Lee, Siegle, Dahl, Hooley, & Silk, 2014; Silk et al., 2017). Specifically, one study comprising 28 girls aged 9–17 years found that, in response to maternal criticism, girls demonstrate increased activation in subcortical–limbic regions associated with processing negative feedback (i.e., lentiform nucleus/putamen and posterior insula; Lee et al., 2014). Another study examining neural responses to different valences of maternal feedback among adolescents with depression (n = 20) and without depression (n = 28) found that youths with depression demonstrated blunted response to praise, relative to neutral statements, in regions associated with reward and self-referential processing compared with youths without depression (i.e., ventromedial prefrontal cortex, precuneus, and thalamus/caudate; Silk et al., 2017). Both studies took steps to correct for multiple comparisons, increasing confidence in their

results. These findings offer support for a link between disruptions in normative neural reactivity to positive versus negative information (i.e., positive and negative verbal statements) in regions associated with reward, self-referential processing, and processing negative feedback and psychopathology in youths. Nonetheless, little is known about the impact of maternal criticism on children's neural responses to positively and negatively valenced information in their environments.

To address this question, we examined children's neural reactivity to monetary gains and losses with the Doors task, which is a commonly used laboratory-based task designed to assess neural responses to reward (Proudfit, 2015). The Doors task has been used in both children (e.g., Kessel et al., 2016; Kujawa, Proudfit, & Klein, 2014; Tsypes, Owens, Hajcak, & Gibb, 2017, 2018) and adults (e.g., Weinberg, Riesel, & Proudfit, 2014) and is a simple guessing paradigm during which participants are presented with an image of two identical doors and are asked to choose which of the doors has a monetary prize behind it. Following their choices, participants are provided with feedback regarding the accuracy of their decisions, resulting in either a gain or loss of prize money. This task elicits the reward positivity (RewP) event-related potential (ERP) component at frontocentral sites on the scalp approximately 300 ms following feedback of monetary gain (RewP–gain) or loss (RewP–loss), which reflects the relative positivity of the waveform in response to one outcome versus the other (Foti, Weinberg, Dien, & Hajcak, 2011; Holroyd, Pakzad-Vaezi, & Krigolson, 2008; Kujawa et al., 2018; Proudfit, 2015).[1] Thus, the RewP serves as an index of reactivity to receiving either a reward or loss. Reactivity is typically stronger to rewards than to losses (Proudfit, 2015), and smaller differences in reactivity to rewards versus losses (or smaller reactivity to gains and greater reactivity to losses) have been implicated in risk for psychopathology, particularly depression, in youths (Bress, Meyer, & Hajcak, 2015; Bress, Smith, Foti, Klein, & Hajcak, 2012; Nelson, Perlman, Klein, Kotov, & Hajcak, 2016). Moreover, the RewP is associated with both behavioral and self-report measures of reward sensitivity (Bress & Hajcak, 2013) as well as with heightened activation in brain areas linked to reward, including the ventral striatum and medial prefrontal cortex (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Luking, Nelson, Infantolino, Sauder, & Hajcak, 2017). As noted above, similar regions have been implicated in responses to maternal praise and criticism (Hooley et al., 2005, 2009; Lee et al., 2014; Silk et al., 2017). Nonetheless, although previous research highlights a relation between a blunted RewP and negative outcomes, including psychopathology (e.g., depression) (Bress, Foti, Kotov, Klein, & Hajcak, 2013; Kujawa et al., 2014; Nelson et al., 2016) and suicidal thinking (Tsypes, Owens, & Gibb, 2019), no study of which we are aware has examined the relation between EE–criticism and neural reactivity to monetary gains and losses (e.g., environmental cues). Research in this area using electroencephalography (EEG) methodologies may offer a cost-effective option for building on extant neuroimaging results that could more feasibly be integrated into prospective studies. Moreover, ERPs provide high temporal resolution, which allows for the examination of different stages of reward response (e.g., initial responses to gain or loss, reward anticipation).

Accordingly, the current study aimed to assess the relation between maternal EE–criticism and initial neural reactivity to monetary gains and losses in children. Consistent with results of recent imaging studies on neural responses to positive and negative maternal feedback (i.e., praise and criticism) (Lee et al., 2014; Silk et al., 2017), we hypothesized that children of critical mothers would demonstrate blunted neural reactivity (i.e., smaller RewP amplitudes) to gains and increased reactivity (i.e., larger RewP amplitudes) to losses than children without critical mothers. Given known links between the RewP and both parent and child internalizing disorders (Bress et al., 2013; Kujawa et al., 2014; Nelson et al., 2016), we also examined whether our findings would be maintained after statistically controlling for the potential impact of effects of mothers' and children's current symptoms and diagnoses of depression and anxiety. We hypothesized that our results would be at least partially independent of children's and mothers' current psychopathology.

[1] In previous research, the RewP has been referred to as the feedback error-related negativity, feedback-related negativity, feedback negativity, and medial frontal negativity.

Method

Participants

Participants in this study were 249 children aged 7–11 years and their mothers recruited from the community. Because our goal was to maximize the generalizability of our findings, inclusion/exclusion criteria were kept to a minimum. The only inclusion criterion was that children be between 7 and 11 years of age (to precede the surge in depression that occurs during adolescence; Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015) and live with their biological mother at least 50% of the time. Children were excluded only if they had a learning or developmental disability (e.g., autism) per parent report that would prevent them from completing the study. Of these 249 dyads, 37 children had insufficient or unusable EEG data during the Doors task (i.e., <14 usable trials per condition due to poor data quality, bad electrodes, or failure to complete the task). In addition, 10 children were excluded from the current analyses because a sibling had previously participated in the study. Therefore, 202 dyads with children (age range = 7–11 years, M = 9.71, SD = 1.38) were included in analyses. Included and excluded children did not differ on any demographic or clinical variable or in levels of EE–criticism. For children in our sample, 52% were male; in terms of race, 72.8% were Caucasian, 13.9% were African American, 0.5% were Asian or Pacific Islander, and 11.9% were biracial. The average age of mothers in our sample was 37.6 years (SD = 6.17, range = 24–54). In terms of mothers' race, 82.7% were Caucasian, 14.4% were African American, 1.5% were Asian or Pacific Islander, and 1.0% were biracial. The median annual family income was $40,000 to $45,000.

Measures

Doors task

The Doors task is a simple guessing task commonly used in studies of reward processing (e.g., Bress & Hajcak, 2013; Bress et al., 2015; Foti et al., 2011; Kujawa et al., 2014; Nelson et al., 2016; Tsypes et al., 2017, 2018; Weinberg, Liu, Hajcak, & Shankman, 2015). The task consisted of 50 trials presented in two blocks of 25 trials. Participants were shown an image of two doors at the beginning of each trial and were instructed to guess which door had a monetary prize behind it by pressing either the left or right button on a game controller. They were informed that, on each trial, they could either win $0.50, as indicated by a green up arrow, or lose $0.25, as indicated by a red down arrow. Feedback about having chosen correctly or incorrectly was presented for 2000 ms, followed by the message "Click for the next round." This message remained on the screen until participants responded and the next trial began. Across the task, 25 gain trials and 25 loss trials were presented in a random order.

EEG data acquisition and processing

During the task, continuous EEG was recorded using a custom cap and the BioSemi ActiveTwo system (BioSemi, Amsterdam, Netherlands). The EEG was digitized at 24-bit resolution with a sampling rate of 512 Hz. Recordings were taken from 34 scalp electrodes based on the 10/20 system. The electrooculogram was recorded from four facial electrodes. Offline analysis was performed using the MATLAB extension EEGLAB (Delorme & Makeig, 2004) and the EEGLAB plug-in ERPLAB (Lopez-Calderon & Luck, 2014). All data were re-referenced to the average of the left and right mastoid electrodes and bandpass filtered with cutoffs of 0.1 and 30 Hz. EEG data were processed using both artifact rejection and correction. Large and stereotypical ocular components were identified and removed using independent component analysis (ICA) scalp maps (Jung et al., 2001). Epochs with large artifacts (>100 μV) were excluded from analysis. Consistent with extant studies (e.g., Tsypes & Gibb, 2020; Tsypes, Owens, Hajcak, & Gibb, 2018), EEG was segmented for each trial, beginning 200 ms before onset of the feedback stimulus and ending 1000 ms after onset of the feedback stimulus. We focused only on children who had at least 14 trials per condition. This decision was made based on the findings of a recent comprehensive study on internal consistency of functional magnetic resonance imaging and EEG measures of reward during late childhood and early adolescence demonstrating that internally consistent measures of response to gain and loss can be obtained using as few as 14 gain and

14 loss trials in the Doors task (Luking et al., 2017). In our sample, the average number of gain trials remaining following artifact rejection was 23.33 (*SD* = 1.93, range = 16–25) and the average number of loss trials was 23.06 (*SD* = 2.41, range = 14–25).

ERPs were separately averaged across gain and loss trials, and the activity 200 ms before feedback onset served as the baseline. These averages were then exported for temporospatial principal component analysis (PCA). The key strength of PCA is that it allows the isolation of the RewP from other temporally or spatially overlapping components, allowing a specific assessment of neural reactivity to gains and losses. The PCA was conducted using the ERP PCA Toolkit Version 2.69 (Dien, 2010b). Consistent with the published guidelines for the use of PCA with ERP data (Dien, 2010a), a temporal promax rotation was performed first to rotate to simple structure in the temporal domain. The temporal PCA used all time points as variables, including all participants, two conditions (i.e., gains and losses), and 34 recording sites as observations. Based on a parallel test (Horn, 1965), 10 temporal factors were extracted for rotation, which accounted for 95.0% of the variance in the ERP signal. Following the temporal PCA, a spatial infomax rotation was performed on each temporal factor to reduce the spatial dimensions of the datasets. The spatial PCA used recording sites as variables and used all participants, conditions, and temporal factor scores as observations. Based on a parallel test (Horn, 1965), three spatial factors were extracted from each temporal factor, which resulted in a total of 30 temporospatial factor combinations. To facilitate interpretation of the PCA solutions, after analysis the ERP PCA Toolkit automatically reproduces the original data, re-creating the waveform in microvolts of each factor loading by multiplying the correlation factor loadings with the standard deviations of the variables (Dien, 2006). The toolkit then reports the peak channel and peak time point for each factor (Dien, 2010a, 2010b). The PCA-derived factor TF5SF1 at the PCA-selected peak channel (i.e., FC2) and peak time point (i.e., one sample of voltage at 320–322 ms) accounted for 2.4% of total variance, resembled the RewP in its temporal and spatial distribution, and was used in the statistical analyses reported below.

Expressed emotion–criticism

The FMSS (Magaña et al., 1986) was used to assess mothers' levels of EE–criticism. To administer the FMSS, the mother is asked to speak for an uninterrupted 5 min about the child and how the mother and child get along together. The response is audiotaped and coded by independent raters for levels of EE–criticism. Mothers are rated as high on EE–criticism if any of three criteria are met, namely their initial statement about the child is negative (e.g., "He is lazy"), they report a negative relationship (e.g., "We just can't be around each other without fighting"), or they report one or more criticisms (e.g., "She drives me crazy") as defined by the FMSS coding system. Mothers are rated as borderline critical if they express dissatisfaction with the child not severe enough to be rated as a criticism. Responses to the FMSS were assigned values of 2, 1, and 0 to reflect high, borderline high, and low EE–criticism, respectively. Consistent with recommendations that the FMSS, if anything, tends to under-identify individuals with high EE–criticism (Hooley & Parker, 2006) and with previous research using the FMSS (Gar & Hudson, 2008; James, Woody, Feurer, Kudinova, & Gibb, 2017; Kershner, Cohen, & Coyne, 1996), responses were dichotomized such that mothers exhibiting borderline or high EE–criticism were classified as critical (n = 51) and mothers exhibiting low EE–criticism were classified as not critical (n = 151). A number of studies have supported the reliability and validity of the FMSS EE–criticism subscale, including concurrent validity with observer ratings of criticism during actual parent–child interactions (e.g., Asarnow, Tompson, Woo, & Cantwell, 2001; Burkhouse et al., 2012; Magaña et al., 1986; McCarty et al., 2004; Rogosch, Cicchetti, & Toth, 2004; Silk et al., 2009). In this study, the FMSS was coded by post-baccalaureate-, graduate-, and post-doctoral-level individuals who were trained to reliability standards and were blind to the other study variables. All samples were independently coded by two raters; when discrepancies arose, a third rater was consulted and a consensus rating was reached. Inter-rater reliability was assessed with a subset of 40 speech samples, and the reliability of EE–criticism ratings was good (κ = .90).

Symptoms

Children's depressive and anxiety symptoms were assessed using the Children's Depression Inventory (CDI; Kovacs, 1981) and the Multidimensional Anxiety Scale for Children (MASC; March, Parker,

Sullivan, Stallings, & Conners, 1997). In the current study, the CDI demonstrated acceptable internal consistency (α = .78), whereas the MASC demonstrated good internal consistency (α = .89). To ensure comprehension, questionnaires were read to the children by trained research staff. Mother's depressive and anxiety symptoms were assessed using the Beck Depression Inventory–Second Edition (BDI-II; Beck, Steer, & Brown, 1996) and the Beck Anxiety Inventory (BAI; Steer & Beck, 1993). In this sample, both measures exhibited excellent internal consistency (αs = .92 and .93, respectively).

Psychiatric diagnoses

To assess children's psychopathology, mothers and their children were interviewed separately using the Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997). In this sample, 1 child (0.5%) met criteria for current major depressive disorder (MDD) and 19 children (9.5%) met criteria for a current anxiety disorder (generalized anxiety disorder: n = 8; social anxiety disorder: n = 7; separation anxiety disorder: n = 5; obsessive–compulsive disorder: n = 2; posttraumatic stress disorder: n = 2; panic disorder: n = 1). The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1995) was used to assess mothers for psychiatric diagnoses. In this sample, 9 mothers (4.5%) met criteria for current MDD and 39 mothers (19.3%) met criteria for a current anxiety disorder (social anxiety disorder: n = 17; panic disorder: n = 11; posttraumatic stress disorder: n = 8; generalized anxiety disorder: n = 6; obsessive–compulsive disorder: n = 3). Interviewers for this project were trained to reliability standards, and interviews were videotaped so that inter-rater reliability could be calculated. To assess inter-rater reliability, a subset of 20K-SADS-PL and 20 SCID-I interviews from this project was coded by a separate rater, and kappa coefficients for depression and anxiety diagnoses in children (κs = 1.0 and 1.0, respectively) and mothers (κs = .89 and .86, respectively) were good.

Procedure

Participants were recruited with Facebook, television, newspaper, billboard, and university listserv advertisements as well as with flyers posted in local establishments. Upon arrival at the laboratory, mothers were asked to provide informed consent and children were asked to provide assent to be in the study. Immediately following consent, mothers completed the FMSS. Next, mothers and their children completed the diagnostic interview and questionnaire assessments, and children completed a series of computer tasks, including the Doors task. As part of the larger study, mothers were compensated $80 and children received a $10 gift card for a local store. All children also received a $5 bonus for completing the reward task. All study procedures were approved by the university's institutional review board.

Results

A preliminary inspection of the data revealed several variables that were significantly skewed ($z > 3.29$) (cf. Tabachnick & Fidell, 2007). These variables were transformed before any further analyses to satisfy the assumptions of normality (square root: CDI, MASC, BDI-II; log10: BAI); following these transformations, none of the variables exhibited significant skew (lowest p = .43). Furthermore, due to the presence of some missing questionnaire data (CDI: 4.5%, MASC: 6.9%; BDI-II: 1.5%, BAI: 2.5%), we examined whether those data were missing at random, thereby justifying the use of data imputation methods for estimating missing values (cf. Schafer & Graham, 2002). Little's missing completely at random test, for which the null hypothesis is that the data are missing completely at random (Little & Rubin, 1987), was nonsignificant, $\chi^2(180) = 186.35$, p = .36, providing support for the imputation of missing values. Therefore, expectation maximization was used to estimate missing values, which were used in all subsequent analyses (see Schafer & Graham, 2002). Means and standard deviations for all study variables are presented in Table 1. To facilitate comparison with other studies, values presented in the table are based on untransformed data. Children in the two groups were similar on demographic variables, current MDD and anxiety diagnoses, and current symptoms of depression and anxiety. Mothers in the two groups were similar on demographic variables and current diagnoses

Table 1
Descriptive statistics for mothers and children.

	Low EE–criticism (n = 151)	High EE–criticism (n = 51)	$r_{\text{effect size}}$
Child age	9.69 (1.39)	9.76 (1.37)	.02
Child sex (% boys)	51.0%	54.9%	−.03
Child race (% Caucasian)	76.2%	64.7%	−.11
Mother age	38.12 (6.12)	35.86 (5.99)	−.16*
Mother race (% Caucasian)	85.4%	76.6%	−.10
Annual family income	$40,001–$45,000	$35,001–$40,000	−.05
CDI	5.03 (4.23)	5.36 (4.72)	.03
MASC	45.24 (17.00)	44.54 (16.36)	−.02
BDI-II	8.25 (8.97)	11.31 (12.44)	.13
BAI	4.52 (6.23)	8.70 (10.59)	.24**
Child current MDD	0.7%	0.0%	−.04
Child lifetime MDD	7.3%	4.0%	−.06
Mother current MDD	3.3%	7.8%	.10
Mother lifetime MDD	43.7%	66.7%	.20**
Child current anxiety Dx	10.0%	7.8%	−.03
Child lifetime anxiety Dx	11.9%	17.6%	.07
Mother current anxiety Dx	17.9%	23.5%	.06
Mother lifetime anxiety Dx	27.8%	31.4%	.03

Note. EE–criticism, expressed emotion–criticism; CDI, Children's Depression Inventory; MASC, Multidimensional Anxiety Scale for Children; BDI-II, Beck Depression Inventory–Second Edition; BAI, Beck Anxiety Inventory; MDD, major depressive disorder; Dx, diagnosis.
* $p < .01$.
** $p < .001$.

of anxiety, although they differed on current diagnoses of MDD and symptoms of depression and anxiety.

Next, we conducted a 2 (EE–Criticism: low or high) × 2 (Condition: gain or loss) repeated-measures analysis of variance (ANOVA) with children's RewP amplitude serving as the dependent variable. Although the EE–Criticism × Condition interaction was not significant, $F(1, 200) = 0.13$, $p = .72$, $\eta_p^2 = .001$, the main effect of condition was significant, $F(1, 200) = 26.05$, $p < .001$, $\eta_p^2 = .12$, with all children displaying larger RewP amplitudes to gain ($M = 6.73$, $SE = 0.57$) than to loss ($M = 3.53$, $SE = 0.62$) regardless of maternal EE–criticism. Importantly, there was also a significant main effect of EE–criticism, $F(1, 200) = 4.10$, $p = .04$, $\eta_p^2 = .02$, such that children exposed to mothers with high EE–criticism displayed smaller RewP amplitudes across both conditions ($M = 4.11$, $SE = 0.88$) than children of mothers with low EE–criticism ($M = 6.16$, $SE = 0.51$) (Fig. 1).[2]

We then conducted a series of follow-up analyses to determine whether the EE–criticism differences in children's RewP amplitudes would be maintained after statistically controlling for the potential influence of mothers' and children's current symptoms and diagnoses of anxiety and depression. Our findings were maintained in all these analyses (all ps < .05). In addition, because the two EE–criticism groups differed significantly on mothers' current symptoms of depression and anxiety, as well as on current and lifetime MDD, which may make it inappropriate to include these variables as covariates in our tests of robustness (cf. Miller & Chapman, 2001), we conducted an additional set of follow-up analyses. Specifically, we examined whether any of the indices of maternal psychopathology was

[2] Although the results presented in this study are based on a PCA-derived factor (TF5SF1), we also ran analyses to determine whether we would see the same patterns of neural responses when examining raw activity at a single frontocentral electrode site (FCz). Specifically, we conducted a 2 (EE–Criticism: low or high) × 2 (Condition: gain or loss) repeated-measures ANOVA with children's raw FCz amplitude serving as the dependent variable. Although the EE–Criticism × Condition interaction was not significant, $F(1, 200) = 0.99$, $p = .32$, $\eta_p^2 = .005$, the main effect of condition was significant, $F(1, 200) = 51.58$, $p < .001$, $\eta_p^2 = .21$, with all children displaying larger raw FCz amplitudes to gain ($M = 12.32$, $SE = 0.80$) than to loss ($M = 9.11$, $SE = 1.38$) regardless of maternal EE–criticism. Importantly, there was also a significant main effect of EE–criticism, $F(1, 200) = 4.06$, $p = .065$, $\eta_p^2 = .02$, such that children exposed to mothers with high EE–criticism displayed smaller raw FCz amplitudes across both conditions ($M = 8.33$, $SE = 0.89$) than children of mothers with low EE–criticism ($M = 13.09$, $SE = 0.84$) (Fig. 2).

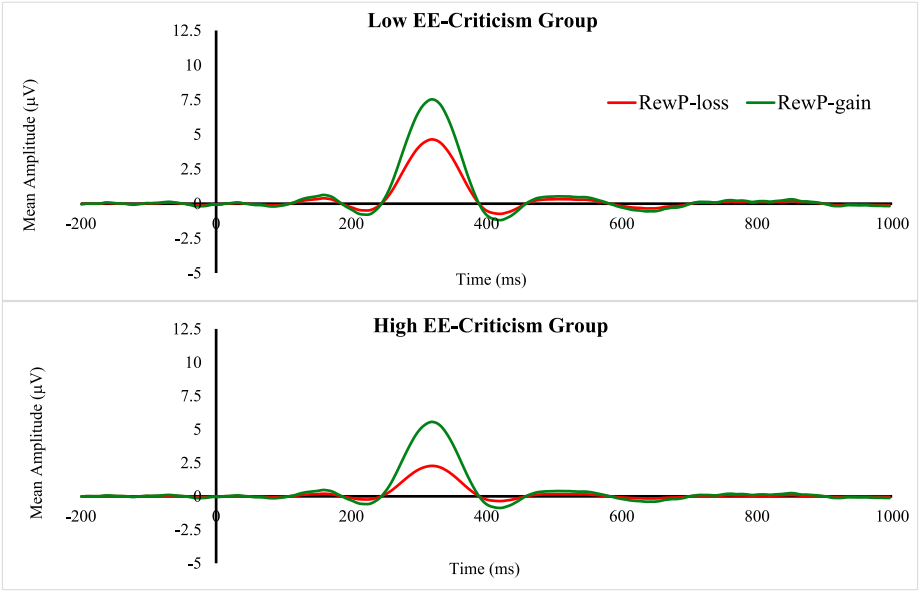

Fig. 1. The principal component analysis (PCA)-derived factor TF5SF1, which resembled the reward positivity (RewP) in response to monetary loss (red) and gain (green) for children of mothers with low expressed emotion–criticism (EE-Criticism) (top panel) and children of mothers with high EE-Criticism (bottom panel) at the PCA-selected peak channel (i.e., FC2) and peak time point (i.e., one sample of voltage at 320–322 ms). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

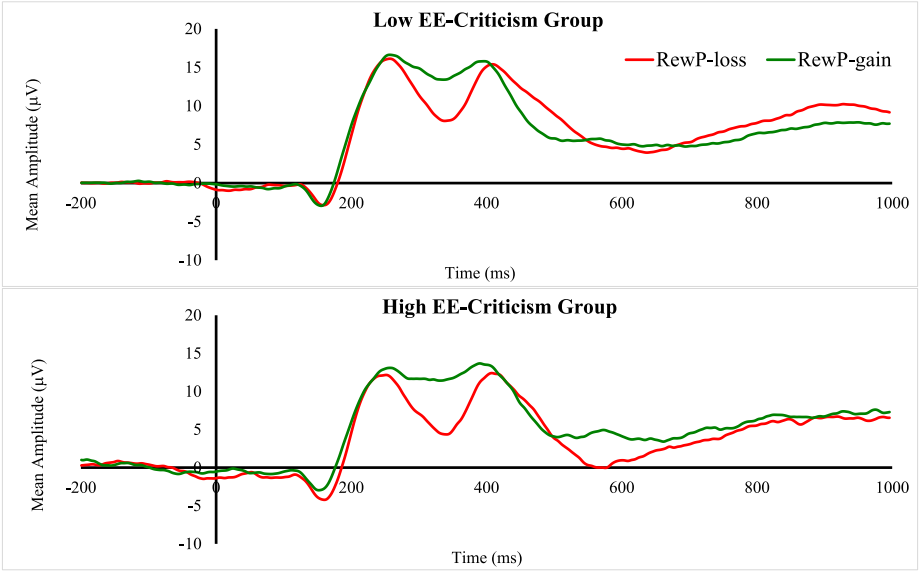

Fig. 2. Stimulus-locked mean event-related potentials to monetary loss (red) and gain (green) for children of mothers with low expressed emotion–criticism (EE-Criticism) (top panel) and children of mothers with high EE-Criticism (bottom panel). The measurement window for the reward positivity (RewP) was 275–375 ms. Waveforms were averaged at FCz. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

related to children's RewP in the full sample or in either EE–criticism subgroup considered individually. None of these analyses was significant (lowest p = .35). These results suggest that the link between EE–criticism and children's neural responses to gains and losses is at least partially independent of children's or mothers' internalizing psychopathology. Finally, exploratory analyses were conducted to determine whether any of the findings were moderated by children's age or sex. None of these analyses was significant (lowest p = .52).[3]

Discussion

The primary goal of this study was to examine the relation between maternal EE–criticism and children's neural reactivity to monetary gains and losses. Partially supporting our hypothesis, we found that, compared with children without critical mothers, children of highly critical mothers demonstrated blunted neural reactivity to monetary gains; however, contrary to our hypothesis, we found that these children also demonstrated blunted reactivity to losses. These results were maintained even after statistically controlling for the effect of children and mothers' symptoms of depression and anxiety, suggesting that our findings are at least partially independent of participants' psychopathology. Thus, our findings suggest that neural reactivity to monetary gains and losses (i.e., non-interpersonal environmental cues) may differ for children with and without critical mothers, such that children of mothers exhibiting high EE–criticism display blunted reactivity to both monetary gains and losses.

Notably, this pattern of results aligns with research examining related parenting constructs. For instance, there is preliminary evidence that among youths with depressed parents, lower levels of positive forms of parenting (i.e., authoritative parenting) are associated with blunted neural responses to reward in children (Kujawa, Proudfit, Laptook, & Klein, 2015). Moreover, similar patterns of blunted responding to positively and negatively valenced environmental information have also been observed with other measures of neural reactivity. For example, there is evidence that children of mothers with high EE–criticism exhibit a blunted late positive potential (LPP), an index of sustained attention to affectively salient visual stimuli (Hajcak, Weinberg, MacNamara, & Foti, 2012), to both positive and negative facial stimuli compared with children of mothers exhibiting low EE–criticism (James, Owens, Woody, Hall, & Gibb, 2018). Typically, larger LPPs are observed for emotionally salient stimuli than for neutral stimuli (for a review, see Lang & Bradley, 2010). To the extent that the blunted pattern of neural responses to monetary gains and losses observed in the current study generalizes to other forms of rewarding and punishing environmental experiences, these results may have important implications for the impact of maternal criticism on children's normative and adaptive development. Indeed, when viewed within the context of reinforcement learning processes (Nussenbaum & Hartley, 2019), the current results suggest that children of critical mothers are not responding to environmental experiences in a way that promotes learning from those experiences. If replicated and extended in longitudinal research, this disruption in adaptive responses to environmental experiences during childhood may be one mechanism for the development of negative outcomes that surge during adolescence such as depression (Barrocas, Hankin, Young, & Abela, 2012; Hankin et al., 1998; Hawton, Saunders, & O'Connor, 2012; Rudolph & Flynn, 2014), particularly because the neural circuitry that processes information from the environment to inform future behavior typically continues into adolescence (Peters, Braams, Raijmakers, Koolschijn, & Crone, 2014). Importantly, both blunted reward reactivity (for a review, see Proudfit, 2015) and maternal EE–criticism (Burkhouse et al., 2012) have been linked to the development of depression in youths. Future research with prospective designs is needed to examine whether children's neural reactivity to monetary gains and losses mediates the impact of maternal criticism on future risk for depression in youths.

The current study has several strengths, including a large sample size, neural assessment of reward responsivity, and the use of interviewer-coded levels of maternal EE–criticism. Despite these

[3] Although not the focus of this study, the PCA also suggested the presence of components reflecting the feedback-P3 and late-positive potential (LPP) (cf. Glazer, Kelley, Pornpattananangkul, Mittal, & Nusslock, 2018). Exploratory analyses of these two factors did not reveal any significant relations with EE–criticism. Details of these analyses are available from the corresponding author upon request.

strengths, the current study also exhibits limitations that offer important directions for future research. First, our study employed a cross-sectional design. For this reason, we are unable to assess the temporal relation between maternal EE–criticism and blunted reactivity to monetary gains and losses. As noted above, future research with a longitudinal design is necessary to determine the specific nature of the relation between maternal EE–criticism and reactivity to monetary gains and losses. Second, the current study focused solely on the relation between maternal EE–criticism and children's responses to monetary gains and losses and did not include the potential impact of paternal EE–criticism on children's reactivity. Thus, future research involving fathers is necessary to determine the generalizability of the current findings.

Conclusions

Our results underscore the relation between maternal EE–criticism and neural reactivity to monetary gains and losses. Specifically, they suggest that children of critical mothers exhibited blunted reactivity to both monetary gain and loss. If replicated and extended in longitudinal studies, these results could provide insight into a potential mechanism by which maternal EE–criticism increases risk for future psychopathology. This line of research may also highlight a potential target of intervention. Specifically, interventions that target reductions in maternal EE–criticism (e.g., family therapy) may, in turn, lead to changes in a child's reward responsiveness and risk for the occurrence (and recurrence) of psychopathology.

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