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Depression Risk and Electrocortical Reactivity during Self-Referential Emotional Processing in 8 to 14 Year-Old Girls

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Abstract

Cognitive vulnerabilities, such as a negative self-referential processing bias, have been theorized to play a causal role in the development of depression. Indeed, depression is associated with the endorsement and recall of more negative and fewer positive emotional words (i.e. recall biases) in the self-referential encoding task (SRET). In addition, currently depressed adults and adolescents, compared to healthy controls, show an enhanced late positive potential (LPP), an event-related potential (ERP) component that reflects sustained attentional engagement, during the processing of negative relative to positive words in the SRET. However, it is unclear whether these behavioral and neural measures in the SRET are indicators of *risk* for depression, or are concomitants of the disorder. The present study included 121 8 to 14 year-old girls with no lifetime history of depression, and examined the association between maternal history of depression (i.e. risk) and both behavioral and ERP measures while viewing positive and negative adjectives during the SRET. Lifetime history of major depressive disorder and/or dysthymia in the biological mother was assessed via a semi-structured diagnostic interview. Results indicated that participants with maternal history of depression, compared to those with no maternal history of depression, demonstrated an enhanced LPP to negative words. There were no group differences in the LPP to positive words. Maternal history of depression was also related to faster response time when rejecting negative words. Participant's current depression symptoms were associated with increased negative recall bias and decreased positive recall bias. The present study provides novel evidence that abnormal electrocortical reactivity to negative self-referential words indexes vulnerability for depression in 8 to 14 year-old girls.

Keywords

adolescents; children; depression; late positive potential; risk; self-referential bias

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[5]Bivariate Pearson correlations were conducted to determine if the LPP to positive and negative words was associated with positive or negative recall biases, or response time when endorsing positive or rejecting negative words. Results revealed no significant correlations between the LPP and recall bias (*p*s > .54), or response time. (*p*s > .33).

Major depressive disorder (MDD) and dysthymia are among the most prevalent mental disorders, occurring in approximately 18-20% of the population and afflicting women twice as frequently as men (Kessler, Chiu, Delmer, & Walters, 2005; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Depressive disorders are relatively infrequent during childhood; however, lifetime prevalence rates increase substantially and reach approximately 10% during adolescence (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015). Notably, the increased prevalence of depressive disorders across adolescence is primarily due to new onset cases amongst girls and not boys (Avenevoli et al., 2015; Hankin, Abramson, Moffitt, Silva, McGee, & Angell, 1998). Despite the increased rates of depression during adolescence, the underlying cognitive-affective processes that may confer *risk* for depression immediately prior to and during the early part of this sensitive period remain poorly understood.

Several theoretical models and empirical evidence have indicated that cognitive biases play a causal role in vulnerability for depression (Abramson, Metalsky, & Alloy, 1989; Alloy, Abramson, Walshaw, Neeren, 2006; Beck, 1967; Gotlib & Joorman, 2010; Mathews & Macleod, 2005; Nolen-Hoeksema, 1991; Seligman, 1975; Wells & Matthews, 1994). Biases are particularly salient when processing self-relevant material (Wisco, 2009). The self-referential encoding task (SRET) has often been used to investigate self-referential biases in depression (Rogers, Kuiper, & Kirker, 1977). In the encoding phase, participants are presented with positive and negative adjectives (e.g., happy, loser, etc.) and asked whether the words are self-descriptive or not. Following the encoding phase, participants are presented with a free recall of the positive and negative words.

Depression has been associated with abnormalities in both behavior and brain activation during the SRET (Lemogne et al., 2010). Specifically, depressed adults, compared to healthy and non-depressed psychiatric controls, display a self-referential bias characterized by endorsing and recalling more negative and fewer positive adjectives (Derry & Kuiper, 1981; Dobson & Shaw, 1987; Kuiper & Derry, 1982; Matt, Vazquez, & Campbell, 1992; Moulds, Kandris, & Williams, 2007). Several cross-sectional studies have also investigated SRET performance in depressed children and adolescents, and these studies have largely replicated findings in adults: depression in childhood and adolescence is associated with greater endorsement and recall of negative adjectives and lower endorsement and recall of positive adjectives (Auerbach, Stanton, Proudfit, and Pizzagali, 2015; Connolly, Abramson, & Alloy, in press; Timbremont & Braet, 2004; Zupan, Hammen, & Jaenicke, 1987). Negative self-referential bias also has been observed in remitted depressed children and adolescents, as they rate negative adjectives as being more self-relevant compared to non-depressed controls (Timbremont & Braet, 2004). Further, some studies have found that children at risk for depression display depressotypic self-referential biases following a negative mood induction, demonstrating that cognitive biases may be evident before the onset of the disorder and evident when primed by negative mood (Hayden et al., 2013; Jaenicke, Hammen, Zupan, Hiroto, Gordon, Adrian, & Burge, 1987; Taylor & Ingram, 1999).

In addition to cognitive-affective biases reflected by the endorsement and recall of positive and negative words, response time (RT) latency has been used as a measure of attentional bias in depression. Previous research has found that depressed adults display faster

processing of negative self-relevant information versus negative other-relevant information (Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997; Greenberg & Alloy, 1989; Kuiper & MacDonald, 1982; MacDonald & Kuiper, 1985). Further, Connolly and colleagues (2015) found that increased depressive symptoms in adolescents was associated with slower RTs when rejecting a negative adjective as self-descriptive, and this relationship in turn predicted increased depressive symptoms at a nine month follow-up.

Collectively, these studies suggest that performance in the SRET can reveal depressotypic processing biases. Two recent studies have gone beyond behavioral measures to examine the neural response to negative and positive adjectives presented during the SRET (Shestyuk & Deldin, 2010; Auerbach et al., 2015). These studies leveraged scalp-recorded event-related brain potentials (ERPs) to examine early and late stages of emotional processing, indexed by the P200 component and the late positive potential (LPP) elicited by the positive and negative adjectives during the encoding phase of the SRET. ERPs have several advantages over the SRET behavioral measures, including superior temporal resolution and not relying on task performance, which can be problematic in children as they may endorse few negative words and recall few words overall (Goldstein et al., 2014).

Automatic processes such as increased attention towards salient information can be indexed by a range of early ERPs, including the P100 and P200. For instance, the P200 is a positive deflection in the ERP signal that is maximal at midline anterior sites between 150 and 250 ms following stimulus onset and has been thought to reflect the automatic processing of semantic information (Crowley & Colrain, 2004; Huang & Luo, 2006). The P200 is modulated by word valence, such that it is enhanced to emotional compared to neutral words (Crowley & Colrain, 2004; Huang & Luo, 2006). The temporally later LPP has been consistently shown to index increased processing of, and engagement with, emotional stimuli (Foti & Hajcak, 2008; MacNamara, Foti, & Hajcak, 2009; Weinberg, Hilgard, Bartholow, & Hajcak, 2012). The LPP is observed as a sustained positivity that emerges at centroparietal electrodes as early as 200 ms after stimulus onset and is sustained for the duration of stimulus presentation (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Hajcak, Dunning, Foti, & Weinberg, 2014; Hajcak & Olvet, 2008). Notably, the LPP is an ideal tool for investigating individual differences in neurophysiological reactivity to emotional stimuli across development, as it has been identified in children as young as 5 years old (Hajcak & Dennis, 2009; Kujawa, Klein, & Hajcak, 2012; Nelson & McCleery, 2008).

Using the SRET, Shestyuk and Deldin (2010) first reported that both the P200 and the LPP were potentiated to negative relative to positive words in depressed adults whereas healthy controls displayed a potentiated P200 and LPP to positive relative to negative words. Auerbach and colleagues (2015) recently extended these findings and demonstrated that depressed 13 to 18 year-old adolescent females similarly exhibited an enhanced P100 and LPP, but not P200, to negative relative to positive adjectives in the SRET, while healthy controls showed an enhanced LPP in response to positive compared to negative adjectives. These studies extend the traditional SRET behavioral findings and indicate that depressed adolescents and adults are characterized by an abnormal neural response to negative

compared to positive stimuli in the SRET—and suggest that ERPs may be viable biomarkers of negative processing biases in depression.

To date, studies utilizing neural measures have focused on currently depressed individuals. Thus, it is unclear whether depressotypic processing biases on the SRET reflect the impact of current depressive symptoms or may index vulnerability for the development of depressive disorders. Indeed, there is emerging evidence for a prospective relationship between *behavioral* measures of self-referential processing biases and the development of depressive symptoms. Specifically, Goldstein and colleagues (2014) found that both positive words endorsed and recalled at age 6 on the SRET predicted increased depressive symptoms at age 9. In addition, Hayden and colleagues (2013) found that depressotypic biases characterized by decreased positive schematic processing and increased negative schematic processing at age 7 predicted depressive symptoms at a 1-2 year follow-up. Lastly, in a community sample of adolescents, decreased recall of positive self-descriptive adjectives predicted increased depressive symptoms at a 9-month follow-up (Connolly et al., 2015). Thus, behavioral measures of processing biases on the SRET may index vulnerability for later increases in depression.

A reliable strategy for investigating risk for depression is to examine children and adolescents of depressed parents, particularly of depressed mothers (for a review see Gotlib, Joormann, & Folan-Ross, 2014). Notably, the effects of maternal depression on offspring have been shown to be stronger than those for paternal depression (Connell & Goodman, 2002). Parental history of depression is associated with a two- to three-fold increase in risk for offspring developing depression by late adolescence (Hammen & Brennan, 2003; Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002; Williamson, Birmaher, Axelson, Ryan, & Dahl, 2004). Moreover, maternal depression has been associated with earlier depression onset and worse prognosis in offspring (Lieb, et al., 2002; Weissman et al., 2006). Previous research has indicated that risk for depression begins to increase with the onset of puberty, and that incidence peaks for girls in the mid-teen years (Angold, Costello, & Worthman, 1998; Avenevoli et al., 2015; Hankin et al., 1998; Hyde, Mezulis, & Abramson, 2008; Lewinsohn, Clarke, Seeley, & Rohde, 1994). In order to assess mechanisms which may contribute to vulnerability for depression during this crucial developmental period, the current study included girls in late childhood and early adolescence. This age range targets the period immediately prior to the time of highest risk for depression onset.

The present study examined whether maternal history of depression (i.e. risk) was associated with abnormal behavioral and electrocortical measures during the SRET in a sample of 121 8 to 14 year-old girls with no lifetime history of depression. For the ERPs, we hypothesized that maternal history of a depressive disorder would be associated with an increased LPP to negative words. We also examined earlier ERPs (i.e., P200 and P300), although given previous mixed findings, we considered this exploratory and did not have specific hypotheses. For behavioral measures, we hypothesized that maternal history of a depressive disorder would be associated with increased negative recall bias, decreased positive recall bias, and slower response time for negative word rejection. Finally, depression and anxiety disorders are highly comorbid (Avenevoli et al., 2015) and anxiety also increases in prevalence during adolescence (Merikangas et al., 2010). Indeed, a lifetime history of an

anxiety disorder has been shown to predict first onset depression in adolescents and young adults (Bittner, Goodwin, Wittchen, Beesdo, Höfler, & Lieb, 2004; Weissman et al., 2006). Therefore, we conducted additional analyses controlling for the participants' lifetime history of anxiety disorders and current depressive and anxiety symptoms. We hypothesized that maternal history of depressive disorders would be associated with the SRET ERP and behavioral measures over and above these other risk factors.

Finally, we also examined whether the association between maternal history of depression and behavioral and electrocortical measures during the SRET varied across development. Moreover, we sought to evaluate whether the relationship between risk for depression and measures on the SRET were stronger in older versus younger participants. Previous research has revealed evidence for both stability and change in cognitive vulnerability during childhood and adolescence (Cole et al., 2009; Hankin, 2008; Hankin, Oppenheimer, Jenness, Barrocas, Shapero, & Goldband, 2009; Hayden et al., 2013). Some investigators have reasoned that cognitive vulnerability factors do not impact depression until the transition from middle childhood to early adolescence, when cognitive processing abilities and attributional styles are more established (Cole & Turner, 1993; Nolen-Hoeksema, Girgus, & Seligman, 1992; Turner & Cole, 1994); others have argued that cognitive abilities relevant to vulnerability develop earlier, during the preschool years (Alessandri & Lewis, 1996). Given the rapid development during adolescence of cognitive function and neural systems that may underlie cognitive vulnerability for depression, we tentatively hypothesized that depressogenic biases would be stronger among more developed and older adolescents, and that the relationship between risk and depressogenic biases would be stronger among more developed and older adolescents.

Method

Participants

The sample included 314 8 to 14 year-old (M = 12.46, SD = 1.79) females and a biological parent who participated as part of a larger longitudinal study of pubertal development and neural activity related to emotion and cognition. The ethnic distribution was 84.7% Caucasian, 6.5% African American, 7.5% Hispanic, and 6.4% 'Other'.

The current study utilized data from the initial laboratory visit, and focused on data obtained from the SRET, which was completed by a subsample of participants (n = 146). A community sample was recruited using local referral sources (e.g., school districts), online classified advertisements, postings in the community, and a commercial mailing list targeting homes with an 8 to 14 year-old female. Families received financial compensation for their participation. Criteria for participation were English fluency, ability to read and comprehend questionnaires, absence of an intellectual disability, and a biological parent consenting to participate in the study. In addition, because the present study focused on maternal depression risk, we excluded participants from the SRET subsample (n = 11) who had a lifetime history of MDD and/or persistent depressive disorder (i.e., dysthymia). In addition, we exclude participants (n = 14) who attended the lab visit with their biological father, resulting in a final sample of 121 participants (age: M = 12.67, SD = 1.70).[1]

Measures

Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present and Lifetime Version (K-SADS-PL, Kaufman et al., 1997)— The K-SADS-PL was administered to determine the presence of lifetime depressive and anxiety disorders in the 8 to 14 year-old females. The parent and participant completed separate, in-person interviews that were conducted by trained interviewers. The same interviewer completed the assessment with the participant and biological parent, and discrepancies in reporting were discussed and final diagnoses were determined using team consensus best-estimate meetings (Klein, Ouimette, Kelly, Ferro, & Riso, 1994). The interviewers were clinical psychology Ph.D. students in the department of psychology at Stony Brook University. All interviewers were trained extensively by a clinical psychologist (G.H.). Specifically, all interviewers watched the SCID-101 training videos (Biometrics Research Department, New York, NY), observed 2-3 joint SCID and K-SADS-PL interviews being conducted, and completed 1-2 joint SCID and K-SADS-PL interviews under the observation of a trained interviewer prior to conducting independent interviews. All interviews were audio-recorded in order to assess inter-rater reliability. A subsample of the interviews (n = 26) were randomly selected for re-assessment, and the Cohen's kappa coefficient for depressive disorders was excellent (κ = .84).

Structured Clinical Interview for the DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1996)—Parental lifetime history of MDD and persistent depressive disorder (i.e., dysthymia) were assessed using the SCID, which was administered to the biological parent who accompanied the participant to the lab session. Diagnoses were determined based on DSM-IV criteria. The same clinical interviewers whom completed the K-SADS-PL also administered the SCID. All interviews were audio-recorded in order to assess inter-rater reliability. A subsample of the interviews (n = 20) were randomly selected for re-assessment, and the Cohen's kappa for depressive disorders was excellent (κ = .91). In the present study, MDD (n = 29) and dysthymia (n = 1) were collapsed into a single category; the one case of dysthymia also met lifetime criteria for MDD. In the current sample, 73.3% of cases occurred within the adolescent's lifetime (n = 4 with onset before the child's birth and recurrence during the child's lifetime) and 26.7% occurred before the adolescent was born.[2]

Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988)—The PDS is a self-report measure of changes in growth across several physical domains associated with pubertal stage, including growth spurt in height, pubic hair, skin change, breast development, and menarche. Participants and a biological parent separately rated the participant's development across the five items using a 4-point Likert scale ranging from 1 (*development has not yet started*) to 4 (*development seems complete*). Scores on each

[1]When participants who completed the study with a biological father were included in all of the analyses the pattern of results were unchanged, all significant main effects and interactions remained significant.

[2]To determine if the occurrence of maternal depression during the participant's lifetime had an effect on the LPP to emotional words, we conducted a Valence (Negative vs. Positive) X Maternal Lifetime Depression (During Adolescent's Lifetime vs. Not During Adolescent's Lifetime) mixed-measure analysis of variance (ANOVA) with valence as a within-subjects factor, maternal lifetime depressive disorder during the participant's lifetime as a between-subjects factor, and age included as a mean-centered continuous covariate. There was no main effect of maternal lifetime depression onset, or significant interactions with the LPP to emotional words. In sum, the relationship between maternal lifetime depression (i.e. risk) and the LPP to emotional words did not vary significantly across cases where the mother's depression occurred before versus during the child or adolescent's lifetime.

item are combined, with higher values indicating increased pubertal development. PDS self and informant reports were highly correlated (r = .89), and were therefore averaged forming a single composite variable. The PDS has demonstrated good validity and reliability (Petersen et al., 1998; Robertson, Skinner, Love, Elder, Conger, Dubas, & Petersen, 1992). In the current sample, Cronbach's alpha for the PDS was .78.

Children's Depression Inventory: Self-Report (CDI: SR; Kovacs, 2010)—The CDI: SR is a 27-item self-report questionnaire assessing the presence and severity of depressive symptoms in children aged 7 to 17 years. For each item participants are presented with three statements and asked to select the one that best describes them. All items were rated on a scale ranging from 0 to 2 and summed to yield a total score of depressive symptom severity. The CDI: SR has demonstrated excellent test-retest reliability and construct, convergent, and discriminant validity (Carey, Faulstich, Gresham, Ruggiero, & Enyart, 1987; Saylor, Finch, Spirito, & Bennett 1984; Smucker, Craighead, Craighead, Green, 1986). In the current sample, Cronbach's alpha for the CDI was .89.

Screen for Child Anxiety Related Disorders Child Version (SCARED; Birmaher, Brent, Chiappetta, Bridge, Monga, & Baugher, 1999)—The SCARED is a 41-item self-report measure assessing childhood anxiety symptoms in the past 3 months. Items are rated on a 3-point scale ranging from 0 (*Not True or Hardly Ever True)* to 2 (*Very True or Often True*). The SCARED has demonstrated good internal consistency and discriminant validity (Birmaher et al., 1999). In the current sample, Cronbach's alpha was .93.

Procedure

The present study utilized a computerized version of the SRET (Auerbach et al., 2015). The task included 60 trials; 30 positive and 30 negative words selected from the Affective Norms for English Words based on valence, arousal, and length (ANEW; Bradley, & Lang, 2010).[3] Previous research has indicated that emotional words from the ANEW and other datasets are rated similarly across children aged 9 to 11 (Vasa, Carlino, London, & Min, 2006). In addition, few age-related effects were found when comparing affective word ratings in 5 to 9 year-old children (Syssau & Monnier, 2009), suggesting that emotional words are evaluated similarly in younger and older children. Positive and negative words differed in valence (t = −34.27, p < .001), but were matched on arousal (t = −0.29, p = .77), and length (t = 0.27, p = .52). Words were presented in a pseudo-random order, with a maximum of two words of the same valence presented consecutively. On each trial, the word was presented for 1000 ms, followed by a fixation cross for 500 ms, followed by a question prompt, “Does this word describe you?” Participants responded using the left and right mouse buttons to select ‘Yes’ or ‘No’. The next trial did not begin until the participant provided a response, and each trial was preceded by a 500 ms fixation cross. Three practice trials were completed using

[3]The following 30 positive and 30 negative words were included in the self-referential encoding task (alphabetical order): adorable, afraid, alive, angry, awful, beautiful, boring, brave, bright, clumsy, confident, cute, depressed, difficult, dummy, failure, friendly, frustrated, fun, gentle, grateful, guilty, happy, helpless, honest, hopeful, idiot, insecure, jolly, lazy, lonely, loser, loved, lucky, mad, moody, nervous, nice, positive, proud, relaxed, respectful, sad, satisfied, scared, selfish, silly, smart, stupid, surprised, sweet, terrible, terrific, ugly, unhappy, upset, useful, useless, winner, worry.

affectively neutral words before the task was initiated; continuous electroencephalography (EEG) was recorded after the practice trials and once the participant indicated they understood the instructions and were ready to start.

Upon completion of the 60 trials, participants completed a brief distractor task which involved counting backwards out loud from 60 to 1. The inclusion of a brief delay or distractor task preceding the free recall phase to control for recency effects on memory has been used in previous studies with children as young as age 6 (Auerbach et al., 2015; Cole & Jordan, 1995; Goldstein et al., 2014; Prieto, Cole, & Tageson, 1992). Following the distractor task, participants were asked to recall as many words as they could that were presented during the task, within 5-minutes. Positive and negative recall biases were calculated using the most common approach, which involved taking the number of positive or negative words endorsed and recalled as the numerator, and the total number of both positive and negative words endorsed as the denominator (Goldstein et al., 2014; Prieto et al., 1992). This approach is the standard in the adult SRET research and is preferable to alternative methods because it controls for overall endorsement levels, which may vary by groups resulting in spurious shifts in recall bias scores (Goldstein et al., 2014; Prieto et al., 1992). In addition, response time (RT) for word endorsement was calculated as the time between word presentation and the button-press for word endorsement (i.e., yes/no). Complete RT data was unavailable for 25 participants because they either failed to endorse a single negative word ($n = 10$), or failed to reject a single positive word ($n = 15$). As a result, these conditions were excluded from the RT analysis, which focused on RT for endorsed positive trials and rejected negative trials, as these data were available for all participants.[4] Faster RTs for endorsing positive or negative stimuli is thought to reflect preferential processing of pleasant and unpleasant content, respectively (Greenberg & Alloy, 1989; MacDonald & Kuiper, 1985).

Physiological Recording and Data Processing

Continuous EEG was recorded while participants completed the encoding phase of the SRET on a 19 in. computer monitor at a distance of approximately 39 in. ERP activity was recorded from 34 electrodes positioned according to the 10/20 system, including FCz and Iz, using the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). Electrodes were placed above and below the left eye to monitor vertical electrooculographic (VEOG) activity, adjacent to the outer canthi of the left and right eyes to monitor horizontal electrooculographic (HEOG) activity, and from the left and right mastoids. The EEG signal was pre-amplified at the electrode to improve signal-to-noise ratio. Data were digitized at a 24-bit resolution with a sampling rate of 1024 Hz using a low pass fifth order sinc filter with a half-power cut-off of 204 Hz. Active electrodes were measured online with reference to a common mode sense active electrode constructing a monopolar channel. The raw EEG data were re-referenced offline to the average of the left and right mastoids and band-pass filtered

[4]Previous studies that have included response time (RT) as a behavioral measure of depressotypic bias during the SRET have typically included 4 conditions of RT: (1) endorsed positive words, (2) endorsed negative words, (3) rejected positive words, and (4) rejected negative words (e.g. Alloy et al., 1997; Connolly et al., 2015). However, the current study was only able to investigate RT for endorsed positive words and rejected negative words, due to very low rates of negative word endorsement ($M = 4.84$, $SD = 4.97$) and positive word rejection ($M = .34$, $SD = 4.91$), resulting in an inadequate number of available trials in these conditions to compute stable RTs for a large portion of the sample.

from 0.1 to 30 Hz. Eye blink and ocular-movement corrections were performed using established standards described by Gratton, Coles and Donchin (1983). The EEG was segmented for each trial beginning 200 ms before the stimuli was presented and continuing for 1200 ms (i.e., the entire stimulus duration).

A semi-automated procedure was employed to identify and reject artifacts. Data for individual channels were marked for rejection if any of the following criteria were met: a voltage step of more than 50.0 μV between sample points was present; a deflection greater than 300.0 μV occurred within a trial; a voltage difference of less than 0.50 μV was detected within 100 consecutive ms. In addition, visual inspection of the remaining trials was conducted to detect and reject any other artifacts.

Typically, ERP studies of emotional processing examine effects by computing the average activity in microvolts within a given time-window, at pre-specified electrode sites for each participant in each condition (Hajcak et al., 2010). However, this approach to quantifying ERPs can be limited insofar as underlying ERP components are not isolated (Luck, 2014). In addition, when averaging over a pre-specified time-window and electrode location a very small proportion of the data is utilized. Furthermore, the scoring and labeling of ERP components is inconsistent across studies, particularly in samples with children. To overcome the limitations of traditional ERP quantification, and to better distinguish between early and late components of emotional processing that can overlap, the current study utilized temporospatial principal components analysis to empirically isolate and score ERP components (PCA; Dien, 2010; Dien, Beal, & Berg, 2005; Dien & Frishkoff, 2005; Foti, Hajcak, & Dien, 2009; Weinberg & Hajcak, 2011). PCA is a factor-analytic statistical approach that examines variance across electrode sites and across time points, thereby using all of the data, and separates latent components that are not easily discernable when computing traditional ERP averages. Consistent with previous research and simulation studies assessing the optimal parameters for computing evoked-potentials (Dien, 2010; Dien & Frishkoff, 2005; Foti et al., 2009), Promax rotation was used to achieve simple structure in the temporal domain, followed by Infomax rotation in the spatial domain to achieve orthogonality. The PCA was computed using Matlab ERP PCA Toolbox (version 2), and used all time points as variables, and considered all participants, word types (positive vs. negative), and recording sites as observations, resulting in linear combinations of time points (referred to as temporal factors) and reducing the 1229 temporal dimensions of the original data set (1024 samples per second multiplied by a total trial-plus-baseline length of 1200 msec). The number of factors to retain for rotation was determined based on the resulting Scree plot (Cattell, 1966) and parallel analysis using the Scree plot of a fully random dataset (Dien, 1998; Zwick & Velicer, 1986). The slope of eigenvalues of the random data were used as a baseline to compare the factors of the dataset of interest; 28 temporal factors were larger than that obtained from the purely random dataset and were extracted for rotation. Covariance matrix and Kaiser normalization were used for this PCA (Dien et al., 2005). Each temporal factor may be reflected as a virtual epoch, and can be described by both its factor loading and factor scores. Spatial information is conserved in the temporal PCA – scalp topography can be reconstructed for any time point, participant, and condition by multiplying the corresponding electrode scores by the factor loading and standard deviation (Dien, 1998).

Following the temporal PCA, a spatial PCA was conducted in order to reduce the number of spatial dimensions in the data set. In the spatial PCA, recording sites were used as variables, and all participants, conditions, and temporal factors scores were used as observations. The covariance matrix was used, and four spatial factors were extracted from each temporal factor for Infomax rotation, based on the resulting Scree plot and parallel analysis (Cattell, 1966; Dien, 1998; Zwick & Velicer, 1986). Each spatial factor may be considered to be a virtual electrode, representing a linear combination of recording sites. The factor loadings characterize the scalp topography of each factor, and the factor scores characterize the activity of each spatial factor across time, participants and conditions. These PCA results can be interpreted by reconstructing (i.e., in microvolts) each temporospatial factor combination by multiplying factor scores by their corresponding loadings and standard deviations; thus, both the time course and the scalp topography of the electrocortical activity captured by that temporospatial factor combination can be directly assessed.

Overall, the temporospatial PCA resulted in a total of 112 factor combinations (4 spatial factors extracted for each of 28 temporal factors). Sixteen factors accounted for more than 1% of the variance each (accounting for 65.5% of the total variance), and were therefore retained for further inspection (Kaiser, 1960). Three factors were temporally and spatially similar to those observed in previous work on emotional processing in children and adults (e.g., Foti et al., 2009; Kujawa et al., 2013; Weinberg & Hajcak, 2011), were theoretically analogous to the ERPs of interest in the current study (Auerbach et al., 2015; Shestyuk & Deldin, 2010), and were consequently included in subsequent analyses. The factors included an early midline central positivity peaking at 230 ms (P200), an early parietal positivity peaking at 462 ms (P300), and a late central positivity peaking at 924 ms (LPP).

Data Analysis

To test the potential impact of maternal lifetime depression on the PCA-derived factor scores and recall biases, we conducted a Valence (Negative vs. Positive) X Maternal Lifetime Depression (Present vs. Absent) mixed-measure analysis of variance (ANOVA) with valence as a within-subjects factor, and maternal lifetime depressive disorder as a between-subjects factor. To examine the impact of depression risk on RT, we conducted separate one-way ANOVAs for endorsed positive words and rejected negative words.

To ensure that maternal lifetime depression findings were not better accounted for by concurrent depression or anxiety symptoms or lifetime history of an anxiety disorder diagnosis, we conducted follow-up Valence (Negative vs. Positive) X Maternal Lifetime Depression (Present vs. Absent) ANCOVAs with valence as a within-subjects factor, parental lifetime depressive disorder as a between-subjects factor, and lifetime anxiety disorder, mean-centered CDI, and mean-centered SCARED total as simultaneous covariates. Similar RT ANCOVA analyses were conducted for endorsed positive trials and rejected negative trials separately.

To examine whether the relationship between maternal lifetime depression and LPP/ behavioral measures of depressotypic bias differed developmentally we conducted follow-up analyses with age and pubertal status included. In order to maximize the statistical power to detect significant moderation, age and pubertal status were included as continuous variables

(MacCallum, Zhang, Preacher, & Rucker, 2002). To this end, hierarchical regression analyses were performed separately for the LPP to negative words, the LPP to positive words, positive recall bias, negative recall bias, RT endorsed positive words, and RT rejected negative words as the dependent variable, respectively, regressed on mean-centered age and maternal lifetime depression (present vs. absent) in the first level, and the Age X Maternal Lifetime Depression interaction in the second level. To examine the relationship between pubertal status, maternal lifetime depression and the interaction of these variables on measures of depressotypic bias, the above analyses were performed again replacing age with mean-centered pubertal status (PDS) as an independent variable. All analyses were conducted using IBM SPSS Statistics, Version 22.0 (Armonk, NY, USA).

Results

Demographics and Clinical Characteristics

As shown in Table 1, participants with maternal depression did not differ from those with no maternal depression on any demographic variable or current depression or anxiety symptoms, but they did have higher rates of lifetime anxiety disorder diagnosis.

ERPs

The two PCA-derived components that were temporally and spatially similar to the P200 and P300 explained 2.7% and 5.4% of the variance, respectively. Results indicated no main effects of word valence or maternal lifetime depression, or significant interactions involving these factors (*p*s > .11).

The third PCA-component resembled the LPP in terms of its temporal and spatial characteristics and accounted for 14.6% of the variance. As shown in Figure 1, this factor was a positive-going slow-wave, peaking at 924 ms, and was maximal at fronto-central electrodes. Overall, the LPP to positive and negative words was comparable, $F(1, 119) = 2.50$, *ns* (see Figure 2 for raw waveforms at Pz). Results indicated a Valence X Maternal Lifetime Depression interaction, $F(1, 119) = 5.52$, $p < .05$, $\eta_p^2 = .04$. Follow-up analyses indicated that participants with maternal lifetime history of depression had a larger LPP to negative words, $F(1, 119) = 6.76$, $p < .01$, $\eta_p^2 = .05$; however, the groups demonstrated a comparable LPP to positive words, $F(1, 119) = 0.15$, *ns* (see Table 1 and Figure 1). Thus, participants with maternal depression were characterized by a potentiated LPP to negative words specifically.

ANCOVA analyses controlling for concurrent depression and anxiety symptoms and lifetime history of an anxiety disorder again indicated a significant Valence X Maternal Lifetime Depression interaction, $F(1, 115) = 4.54$, $p < .05$, $\eta_p^2 = .04$; maternal lifetime history of depression group had a significantly larger LPP to negative words, $F(1, 115) = 7.47$, $p < .01$, $\eta_p^2 = .06$, but were similar in their LPP to positive words, $F(1, 115) = 0.54$, *ns*.

Behavioral Measures

In the analysis of RT, girls with a maternal lifetime history of depression demonstrated significantly faster RTs when rejecting negative words, $F(1, 119) = 6.26$, $p < .05$, $\eta_p^2 = .05$,

but RT for the endorsement of positive words was comparable across groups, $F(1, 119) = 1.29$, *ns*. ANCOVA analyses indicated that girls with a maternal lifetime history of depression continued to demonstrated faster RTs when rejecting negative words when controlling for concurrent depression and anxiety symptoms, and lifetime history of anxiety disorders, $F(1, 115) = 7.17$, $p < .01$, $\eta_p^2 = .06$. RTs when endorsing positive words remained comparable across groups following the inclusion of covariates, $F(1, 115) = 2.01$, *ns*.

To determine if the LPP findings were independent of RT we conducted a Valence (Negative vs. Positive) X Maternal Lifetime Depression (Present vs. Absent) mixed-measures ANCOVA with average RT to rejected negative words included as a mean-centered covariate. Results again revealed a significant Valence X Maternal Depression interaction, $F(1, 118) = 4.68$, $p < .05$, $\eta_p^2 = .04$. To examine if the RT findings were independent of the LPP we conducted an ANCOVA with Maternal Lifetime Depression (Present vs. Absent) as the between-subjects factor and the LPP to negative words as a mean-centered covariate. Results again indicated that girls with a maternal lifetime history of depression had significantly faster RTs when rejecting negative words, $F(1, 118) = 5.51$, $p < .05$, $\eta_p^2 = .05$. These findings suggest that both the LPP and RT are independently associated with maternal depression history.

In the analysis of recall biases, there was a main effect of valence, $F(1, 119) = 195.48$, $p < .001$, $\eta_p^2 = .62$, such that participants demonstrated increased recall bias for positive words ($M = 0.21$, $SD = 0.09$) relative to negative words ($M = 0.03$, $SD = 0.05$). There was also a trend-level Valence X Maternal Lifetime Depression interaction, $F(1, 119) = 2.69$, $p < .10$, $\eta_p^2 = .02$; follow-up analyses revealed that high-risk girls demonstrated increased recall of negative words, $F(1, 119) = 3.00$, $p < .10$, $\eta_p^2 = .03$, whereas the recall of positive words was comparable across groups, $F(1, 119) = 1.20$, *ns.* ANCOVA analyses indicated that the Valence X Maternal Lifetime Depression interaction did not reach significance, $F(1, 115) = 2.03$, $p > .16$. However, there was a significant Valence X CDI interaction, $F(1, 115) = 5.11$, $p < .05$, $\eta_p^2 = .04$. Correlations between CDI and behavioral performance indicated that greater current depression symptoms were associated with increased recall bias for negative words, $pr(121) = .56$, $p < .001$, and decreased recall bias for positive words, $pr(121) = -.22$, $p < .01$.

Developmental Analyses

In the LPP analyses, age was negatively associated with the LPP to positive words, $t(120) = -2.31$, $p < .05$, but the Age X Maternal Lifetime Depression interaction was not significant, $t(120) = 0.68$, *ns*. Furthermore, neither Age nor the Age X Maternal Lifetime Depression interaction was associated with the LPP to negative words (*p*s > .17). Similarly, the PDS was positively associated with the LPP to positive words, $t(120) = -2.86$, $p < .01$, but the PDS X Maternal Lifetime Depression interaction was not significant, $t(120) = 1.28$, *ns*. In addition, the PDS was negatively associated with the LPP to negative words, $t(120) = -2.46$, $p < .05$, but the PDS X Maternal Lifetime Depression interaction was not significant, $t(120) = -1.11$, *ns*.

In the RT analyses neither age, puberty, or their interaction with maternal lifetime history of depression was associated with RT to endorsed positive words or rejected negative words (*p*s > .35).

In the recall bias analyses, age was positively associated with positive recall bias, $t(120) = 2.24$, $p < .05$, but the Age X Maternal Lifetime Depression interaction was not significant, $t(120) = 0.66$, *ns*. Neither the PDS, $t(120) = 0.64$, *ns*, nor the PDS X Maternal Lifetime Depression interaction, $t(120) = 0.24$, *ns*, were associated with positive recall bias. Age was also positively associated with negative recall bias, $t(120) = 3.17$, $p < .01$, but the Age X Maternal Lifetime Depression interaction was not significant, $t(120) = 0.56$, *ns*. In addition, pubertal status was positively associated with negative recall bias, $t(120) = 2.83$, $p < .01$, but the PDS X Maternal Lifetime Depression interaction was not significant, $t(120) = .17$, *ns*.

Discussion

The current study examined the relationship between maternal lifetime history (i.e., risk) of depression and both behavioral and neural measures of depressotypic processing biases on the SRET in a large sample of never-depressed 8 to 14 year-old females. Results indicated that participants with a maternal lifetime history of depression, relative to those with no maternal lifetime history, demonstrated an enhanced LPP to negative words, but there were no group differences in the LPP to positive words. Critically, these findings remained significant after controlling for current depressive and anxiety symptoms and lifetime history of an anxiety disorder. Notably, maternal lifetime history of depression was specifically related to the LPP and not earlier ERP components, suggesting risk-related abnormalities in more elaborative versus automatic levels of emotional processing.

Maternal history of depression was also associated with response time during the encoding phase, such that high-risk girls were faster to reject negative adjectives as self-descriptive. LPP and RT associations with maternal history of depression were independent of one another and were unrelated to current depressive symptoms. However, current depression symptoms *were* associated with increased recall bias for negative words and decreased recall bias for positive words. These findings suggest that recall of endorsed words as a measure of depressotypic processing biases in the SRET may emerge concurrently with depressive symptoms, whereas the LPP and RT recorded during the SRET may be neural and behavioral markers that index vulnerability for depression. Broadly, these findings are consistent with several theoretical models implicating a negative self-referential cognitive vulnerability in the etiology of depressive disorders, including schema theory, the hopelessness model, and response styles theory (Abramson, et al., 1989; Alloy, et al., 2006; Beck, 1967; Nolen-Hoeksema, 1991).

The depression risk findings complement and extend previous research on depression and the LPP to emotional words during the SRET. Depressed adults have been found to have a potentiated LPP to negative relative to positive self-referential words, while non-depressed adults display the opposite pattern (Shestyuk & Deldin, 2010). In addition, a recent study of depressed adolescent females largely replicated this finding, such that depressed adolescents had a potentiated LPP to negative relative to positive words, while healthy controls had a

potentiated LPP to positive relative to negative words (Auerbach et al., 2015). Although these studies revealed important neural abnormalities in self-referential processing that co-occur with depression, they were unable to determine whether self-referential information processing biases reflect *vulnerability* for depression, or if they are a concomitant of depression. The present study indicates that even without the inclusion of a negative mood induction, adolescent females at increased risk for depression are characterized by an increased LPP in response to negative adjectives during the SRET. That is, allocating increased attention to negative information during the SRET, as reflected in a larger LPP, is associated with increased depression risk.

These results provide insights regarding potential etiological mechanisms of depression. Specifically, maternal depression history was associated with more elaborative processing of negative information, and this effect was not better accounted for by current internalizing symptoms. The augmented processing of negative information, in the context of evaluating whether it is self-referential, may be important for the development of negative schemas; moreover, this cognitive vulnerability may in turn be modeled and reinforced by a depressed parent. Notably, these effects were not moderated by age or pubertal development, suggesting that the LPP functioned similarly in relation to risk across developmental changes in early puberty.

Previous studies have likewise found a relationship between depression and earlier ERP components elicited by emotional words in the SRET. Specifically, depressed adults, compared to healthy controls, displayed a potentiated P200 to negative words, relative to positive (Shestyuk & Deldin, 2010). Depressed adolescent females exhibited a potentiated P100 to negative words, relative to positive, but not an enhanced P200 (Auerbach et al., 2015). However, the current study did not find differences between two earlier components of emotional processing (i.e., the P200 and P300) and depression *risk*. Previous studies utilized traditional averaging techniques to compute early and late components, potentially confounding multiple ERP components (Luck, 2014). A strength of the current study is that we utilized temporospatial PCA to empirically-define ERP components. An alternative explanation for these discrepancies may be that current mood dysfunction is related to a range of ERP abnormalities in the SRET, whereas risk is only associated with later, more elaborative, processing of negative emotional information.

The current study found that maternal lifetime history of depression was associated with faster RTs when rejecting negative words, but was not related to RTs when endorsing positive words. These results may suggest that for high risk girls the negative words were experienced as more salient and aversive and were therefore rejected more quickly. Similar to the LPP findings, RT was unrelated to current depressive symptoms. Importantly, follow-up analyses specified that both the LPP and RT effects were independent, and potentially unique markers of depression risk. Future prospective studies are needed to verify the possibility that LPP and RT during SRET might predict subsequent risk for depressive episodes and disorders.

Previous studies with depressed adults have found that depression is associated with faster RTs when endorsing negative self-referential words (Alloy et al., 1997; Greenberg & Alloy,

1989; Kuiper & MacDonald, 1982; MacDonald & Kuiper, 1985). Further, a recent study with adolescents found that depression was associated with faster RTs when rejecting positive words (Connolly et al., 2015). However, the current study was unable to investigate these conditions because a large number of participants failed to reject a single positive word or endorse a single negative word. Therefore we excluded these conditions from RT analyses, making the current findings more difficult to directly compare to previous studies. Connolly and colleagues (2015) had similar issues given that 26.8 % of their sample failed to reject a single positive word and 43.0% failed to endorse a single negative word; findings based on averages with relatively few trials should be interpreted with caution.

There was no association between maternal lifetime history of depression and SRET recall biases. It is possible that recall bias may only be observable among at-risk individuals in the presence of a negative mood state (Gotlib, Joormann, & Foland-Ross, 2014), and the present study did not employ a negative mood induction. For instance, a previous investigation only found recall differences in children with parental risk for depression following a mood induction; without a mood induction, high- and low-risk children had similar recall bias during the SRET (Taylor and Ingram, 1999; see also: Hayden et al., 2013; Jaenicke et al., 1987). In contrast to maternal lifetime depression history, current depressive symptoms *were* associated with increased negative recall bias and decreased positive recall bias. These results are consistent with a large body research indicating that, across adolescence and adulthood, depressive symptoms and diagnosis are associated with increased endorsement and recall of negative words and decreased endorsement and recall of positive words (Auerbach et al., 2015; Connolly et al., 2015; Derry & Kuiper, 1981; Dobson & Shaw, 1987; Goldstein et al., 2014; Kuiper & Derry, 1982; Lemogne et al., 2010; Matt et al., 1992; Moulds et al., 2007; Timbremont & Braet, 2004; Zupan et al., 1987).

It is crucial to understand developmental changes in cognitive vulnerability to depression. Contrary to our hypothesis, the association between maternal lifetime history of depression and neural and behavioral indices of cognitive biases was not moderated by age or pubertal status in the current study. Some researchers have reasoned that cognitive vulnerability factors do not impact depression until the transition from middle childhood to early adolescence, when cognitive processing abilities and attributional styles are more established (Cole & Turner, 1993, Nolen-Hoeksema et al., 1992; Turner & Cole, 1994). In support of this hypothesis, Nolen-Hoeksema and colleagues (1992) found that the interaction between depressotypic attributional style and negative life events predicted increased depressive symptoms in early adolescence (ages 11 – 14), but not in middle childhood (ages 8 – 11). However, these studies are limited in that they assess depressotypic bias using a self-report measure with relatively low internal reliability, and did not include behavioral or neural measures of vulnerability which may be more appropriate for developmental samples who have limited meta-cognitive abilities (Abela & Hankin, 2008). However, other studies utilizing behavioral measures of depressotypic bias have found support for the presence of cognitive vulnerability to depression and interactions with life stress in children as young as 5 (Conley et al., 2001; Panak & Garber, 1992). Nonetheless, larger longitudinal studies with a greater representation of age and range of pubertal development are needed to better understand the impact of development on cognitive vulnerability for depression during this

sensitive period. In particular, future studies might focus on differences that emerge later in puberty by examining older adolescents.

While the present study has important implications for understanding the role of cognitive vulnerabilities in the development of depression, there are several limitations that should be noted. The current study focused on females in late childhood and early adolescence because they are at increased risk for developing depression; however, the role of cognitive vulnerabilities in the development of depression may differ across sexes. Indeed, Hankin and Abramson (2001) have proposed an integrative model of depression which suggests that adolescent girls have increased rates of depression in part due to an interaction between experiencing more negative events and more rumination and negative inferential style compared to adolescent boys. Future studies might examine the interaction between negative events, early-emerging processing biases reflected in the LPP, and the emergence of depression. The current study utilized a community sample, resulting in low levels of current depressive symptoms in the girls, and an unequal distribution of the risk groups (i.e. 29 cases of lifetime maternal depression or 24% of the total sample). This may have limited our ability to find associations between current depressive symptoms and the LPP and RT. Future studies could select a sample to maximize variability in depressive symptoms, or the number of high risk cases for comparison. In addition, the current study focused on maternal depression, and did not evaluate depression status of the father, limiting our ability to draw conclusions on parental depression more broadly. Although effect-sizes obtained in analysis with depression risk were comparable to previous studies investigating psychological and biological vulnerability factors (Asarnow, Thompson, Joorman, & Gotlib, 2014; Nelson, Perlman, Hajcak, Klein, & Kotov, 2015), future studies should consider including an assessment of both maternal and paternal depression history.

Although several past studies have found evidence for depressotypic bias after a delay or distractor task (Auerbach et al., 2015; Cole & Jordan, 1995; Goldstein et al., 2014; Prieto et al., 1992), it is possible that the distractor task in the current study may have rendered recall more difficult for younger participants, thereby weakening our ability to examine depressotypic bias.Further, the current study did not explicitly test each participant's knowledge of the selected words from the SRET, therefore we were unable to control for differences in comprehension. However, inclusion criteria for the study were ability to read and comprehend questionnaires, and previous research has shown that words selected from ANEW are rated similarly across late childhood (Vasa et al., 2006).

The current study integrated psychological and biological assessments, and focused on multiple domains of risk for depression (i.e. cognitive biases and abnormal neural activity), allowing for a more comprehensive investigation of potential mechanisms and measures related to the intergenerational transmission of risk for depression. To conclude, the current study found that maternal depression was associated with an increased LPP in response to negative words, and faster decision making times when rejecting negative words in late child and adolescent girls. These findings were independent of the girls' age, pubertal status, current depressive or anxiety symptoms and lifetime history of an anxiety disorder, suggesting that negative words may be more schema-consistent in high-risk girls, regardless of current clinical status. In addition, the LPP and RT findings were independent of each

other, indicating that they may be unique markers of risk. Although depression risk (but not depressive symptoms) was related to the LPP and RT to negative words, depressive symptoms (but not risk) was related to positive and negative behavioral recall biases. Treatment outcome studies have shown that anti-depressant medication, mindfulness meditation, and cognitive-behavioral therapy are all associated with changes in self-referential processing, assessed via behavioral changes in word endorsement, and changes in activation of brain systems implicated in self-referential processing, such as the medial prefrontal cortex and the ventral anterior cingulate cortex (Goldin, Ramel, & Gross, 2009; Di Simplicio, Norbury, & Harmer, 2012; Yoshimura et al., 2014). Since the LPP shows abnormalities in individuals at risk for depression, future research might assess whether prevention or early intervention efforts can alter the LPP to emotional words presented during the SRET in these individuals, and whether that in-turn alters subsequent risk. Along similar lines, longitudinal work is needed to assess if the LPP to emotional words during the SRET predicts the onset and maintenance of depressive disorders.

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General Scientific Summary

Late childhood and early adolescent girls at increased risk for developing depression exhibit an enhanced neural response when evaluating the self-relevance of negative words. High risk girls also display faster behavioral response times when rejecting negative words as self-descriptive. These neural and behavioral abnormalities may indicate a vulnerability for the development of depression.

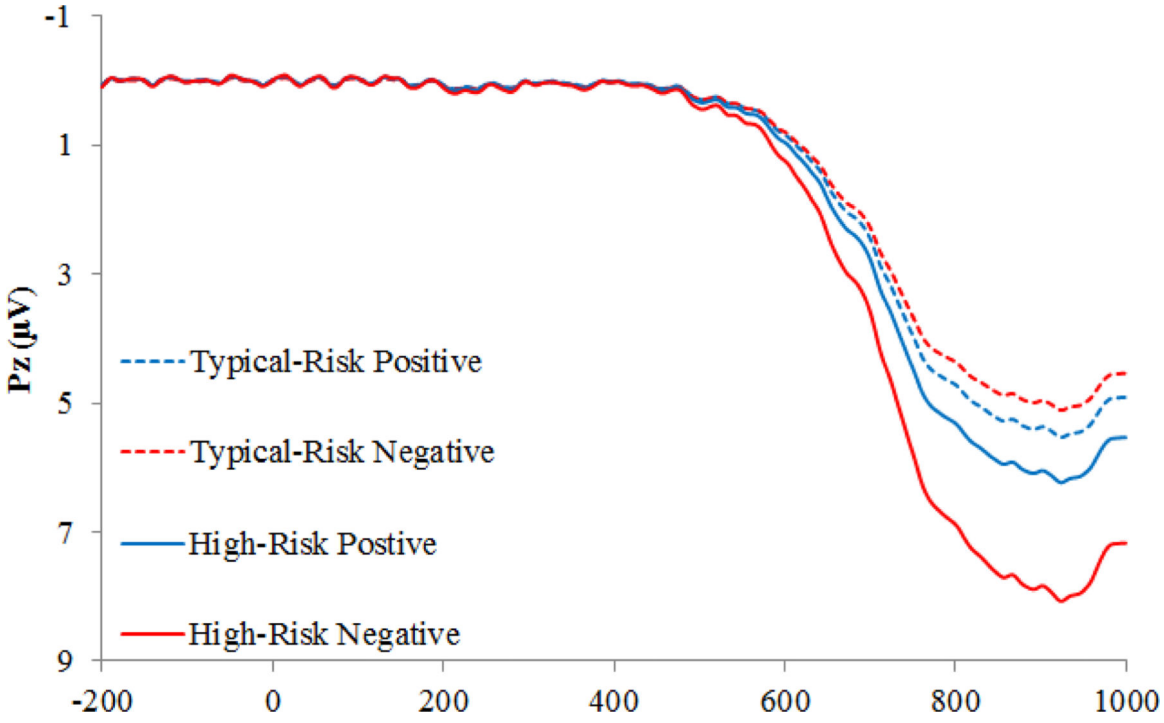

Figure 1.

PCA-derived LPP waveforms to negative and positive words for participants with no maternal depression (dashed lines) and maternal depression (solid lines). PCA = principal components analysis; LPP = late positive potential.

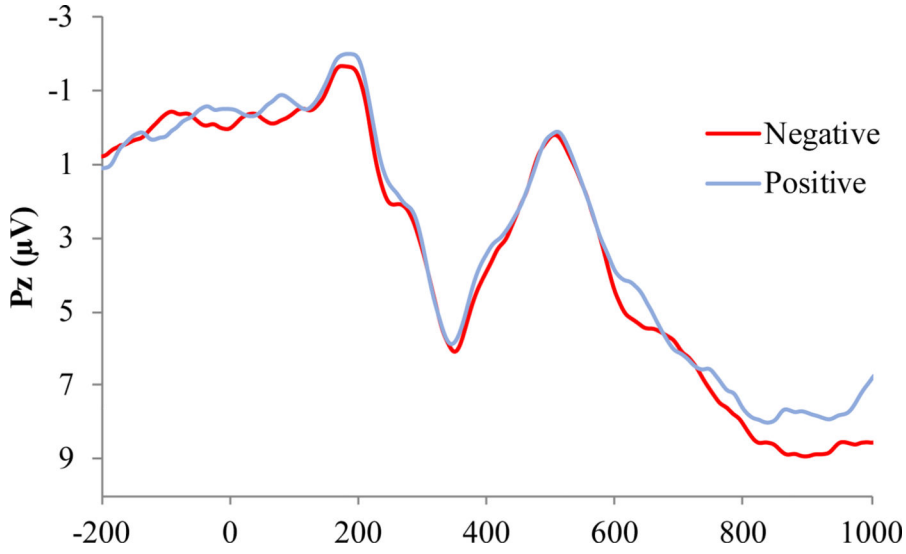

Figure 2.

Raw LPP waveforms at the Pz electrode site to negative and positive word presentation during the SRET. LPP = late positive potential; SRET = self-referential encoding task.

Table 1

Demographics, Clinical Characteristics, and SRET Behavior for Participants with and without Maternal Lifetime Depression

	No Maternal Depression (*n* = 92)	Maternal Depression (*n* = 29)	*F* or χ^2	*p*
Demographics				
Age (*SD*)	12.67 (1.76)	12.67 (1.51)	*F* < 0.01	.98
% Caucasian	88.0%	79.3%	χ^2 = 1.12	.29
Clinical Characteristics				
Lifetime Anxiety Disorder	23.9%	51.7%	χ^2 = 8.37	<.01
CDI (*SD*)	5.99 (6.29)	6.49 (6.12)	*F* = 0.14	.71
SCARED (*SD*)	21.12 (13.30)	25.30 (14.68)	*F* = 2.07	.15
LPP (μV)				
Positive Words (*SD*)	6.58 (6.10)	7.07 (5.16)	*F* = 0.15	.70
Negative Words (*SD*)	6.13 (5.67)	9.37 (6.45)	*F* = 6.76	.01
SRET Behavior				
Positive Endorsement (*SD*)	25.66 (4.72)	25.66 (5.55)	*F* < 0.01	.99
Negative Endorsement (*SD*)	4.54 (4.95)	5.66 (5.01)	*F* = 1.11	.30
Positive Processing (*SD*)	0.22 (0.09)	0.20 (0.08)	*F* = 1.20	.28
Negative Processing (*SD*)	0.03 (0.05)	0.05 (0.06)	*F* = 3.00	.09
Positive Endorsed RT (*SD*)	2616.11 (259.65)	2554.44 (239.22)	*F* = 1.29	.26
Positive Rejected RT (*SD*)	3136.27 (846.20)	2603.42 (379.56)	*F* = 9.28	<.01
Negative Endorsed RT (*SD*)	2816.14 (483.30)	2564.90 (200.94)	*F* = 6.64	.01
Negative Rejected RT (*SD*)	2674.58 (266.80)	2538.66 (212.52)	*F* = 6.26	.01

Note. CDI = Children's Depression Inventory; SCARED = Screen for Child Anxiety Related Disorders; *SD* = standard deviation; LPP = Late Positive Potential; SRET = Self-Referential Encoding Task; RT = Response Time.