Journal of Abnormal Child Psychology
https://doi.org/10.1007/s10802-020-00648-1

Parental Presence Impacts a Neural Correlate of Anxiety (the Late Positive Potential) in 5–7 Year Old Children: Interactions with Parental Sensitivity to Child Anxiety

Taylor N. Day[1] · Lyndsey J. Chong[1] · Alexandria Meyer[1]

Abstract
Anxiety disorders tend to onset early in development and often result in chronic impairment across the lifespan. Thus, there is substantial interest in identifying early neural markers of anxiety and leveraging these markers to better understand processes leading to anxiety. The late positive potential (i.e., LPP) indexes sustained attention to motivationally relevant stimuli; and the LPP to negative images is increased in individuals with anxiety. In the current study, we examined how *parental presence* impacts the LPP to threatening images in children (52.6% male) between 5 and 7 years-old ($N = 78$). Moreover, we explored interactions with parental sensitivity to child anxiety symptoms. Results suggest that when children are in the presence of their parent (compared to the presence of an experimenter), they displayed a larger LPP to threatening images. LPP activity was modulated by parental response to their child's anxiety symptoms, such that children with parents who were overly reactive to their children's anxiety symptoms had the greatest LPP response when viewing threatening stimuli in their parent's presence. Additionally, exploratory analyses indicated that children with clinical and subclinical anxiety were characterized by an increased LPP to negative images, but only when the LPP was measured with parents in the room. Findings are novel and extend previous work by suggesting that parents who react strongly when observing their children's anxiety symptoms in turn increase their child's engagement with threatening stimuli, thereby placing them at greater risk for anxiety.

Keywords Anxiety · Parenting · ERP · Late positive potential

Anxiety disorders are the most common form of psychopathology (Kessler et al. 2005). These disorders are often characterized by an early onset and chronic course (Bittner et al. 2007; Yonkers et al. 2003), along with functional impairment and high economic costs (Kessler and Greenberg 2002). Additionally, anxiety often has a profound impact on the lives of individuals suffering from these disorders. In light of this, identifying early environmental *moderators* of neurodevelopmental factors that increase the likelihood for an anxious trajectory (i.e., elevated risk that may potentially lead to clinically significant anxiety in later years) may clarify our understanding of the etiopathogenesis of clinical anxiety and elucidate novel preventative and intervention strategies.

Work in children and adults has shown that cognitive biases in the processing and detection of threat stimuli are one factor linked to the development of anxiety (Bar-Haim et al. 2007; Muris and Field 2008). There is also substantial evidence suggesting that individuals with anxiety may have a differential response to threat-related emotional stimuli (Bishop et al. 2004) and such interpretations play a crucial role in the development and maintenance of anxiety (Beck and Rush 1985; Mogg and Bradley 1998). A growing interest in biomarkers has spurred empirical investigations on the development of core neural systems underlying clinical anxiety, including studying differences in brain functioning and examining neural causes of information processing biases (Blackford and Pine 2012; Guyer et al. 2008; Pine 2007; Strawn et al. 2014); however, neuroscience studies on emotional information processing has been largely dominated by neuroimaging studies. Although this body of work has contributed crucial knowledge on neural structures related to emotional information processing, scalp-recorded electrical activity in the brain (i.e., electroencephalogram [EEG]) may offer a number of advantages over the use of neuroimaging techniques. For instance, EEG directly reflects neural activity and has excellent temporal resolution due to its ability to

✉ Alexandria Meyer
meyer@psy.fsu.edu

[1] Department of Psychology, Florida State University, 1107 W Call St., Tallahassee, FL 32304, USA

Published online: 22 April 2020

detect the dynamic changes in neural processes on the order of milliseconds. Thus, given its excellent temporal resolution, this method is optimal for examining online modulation of neural activity in different contexts. Additionally, EEG is relatively inexpensive and may be more tolerable in younger populations.

The Late Positive Potential (LPP): a Neural Risk Marker of Anxiety

The late positive potential (LPP) is a slow-wave event-related potential (ERP) that reflects sustained attention towards motivationally salient stimuli (Auerbach et al. 2016, 2015; Hajcak et al. 2012), such that a larger LPP is typically observed following aversive and pleasant stimuli as compared to neutral stimuli (Cuthbert et al. 2000; DeCicco et al. 2012; Foti and Hajcak 2008; Foti et al. 2009; Hajcak and Dennis 2009; Schupp et al. 2000). It begins approximately 250 to 300 ms post-stimulus (Cuthbert et al. 2000) and extends throughout picture processing and after picture offset, lasting for several hundred milliseconds to seconds (Foti and Hajcak 2008; Hajcak and Olvet 2008). It shows good to excellent test-retest reliability in children and adults (Huffmeijer et al. 2014; Kujawa et al. 2013a).

In the presence of unpleasant stimuli, anxiety is associated with a larger LPP in both children and adults (Hajcak and Dennis 2009; Kujawa et al. 2015; Leppanen et al. 2007; MacNamara and Hajcak 2010). Previous studies have also demonstrated that larger LPP amplitudes are elicited by unpleasant stimuli related to greater state anxiety in adults (MacNamara and Hajcak 2009, 2010) and greater trait anxiety in children (DeCicco et al. 2012). Additionally, larger LPPs to unpleasant images have been shown to predict increases in anxiety symptoms in children after a traumatic event (Kujawa et al. 2016). In light of these findings, the LPP to unpleasant images has been proposed as a neural marker of emotional reactivity or threat sensitivity. Despite this, little work has been done to investigate environmental moderators (such as parenting) that may shape the LPP response in children.

Role of Parenting in Child Anxiety

Abundant work has shown that parenting may have significant effects on the development of child physiology and the brain. Researchers have found that harsh parenting prospectively predicts the development of neural markers that lead to anxiety (Belsky and de Haan 2011; Meyer et al. 2015); on the other hand, positive parenting has been shown to predict healthy brain development in regions associated with reward processing, emotional reactivity, and emotional regulation (Belsky and de Haan 2011). Certainly, the *presence* of the primary caregiver plays an important role in the development of threat-sensitivity in the early childhood of many mammalian species, such as rodents (Levine 2001), non-human primates (Bayart et al. 1990), and children (Conner et al. 2012; Gee et al. 2014; Gunnar and Donzella 2002; Tottenham 2012). Hence, parental presence may have a significant impact on the LPP, a known neural marker for child anxiety.

Moreover, much research on anxiety disorders has been dedicated to exploring the influence of parenting on the development, maintenance, and remission of childhood anxiety (McLeod et al. 2007; Rapee 1997; Wood et al. 2003). Indeed, studies have found that parenting affects outcomes for child anxiety disorders through mechanisms such as parental control (Whaley et al. 1999) and increased family accommodation (Lebowitz et al. 2013). However, there are numerous theoretical models hypothesizing the mechanism underlying the relationship of parenting styles and anxiety (McLeod et al. 2007) – some studies have shown strong associations between types of parenting and anxiety (Chorpita and Barlow 1998; Gerlsma et al. 1990; Rapee 1997; Wood 2006; Wood et al. 2003), while others have suggested that non-parenting factors such as non-shared environmental and genetic effects may account for more substantial variance in child anxiety (Eley et al. 2003; Harris 2002; Kagan 2003; Maccoby 2002; Rice et al. 2002; Rutter 2002; Van Beijsterveldt et al. 2004). Moreover, a meta-analysis by McLeod et al. (2007) showed that parenting accounted for only 4% of the variance in child anxiety. This reflects a need to identify other factors (i.e., potential moderators) that may clarify our understanding of the relationship between parenting and child anxiety. We aimed to examine a construct (described below) that may influence but is not unique to parenting approaches.

Parent Sensitivity to Child Anxiety

Anxiety sensitivity (AS) is defined as the belief or perception that anxiety symptoms and experiences have negative consequences (Reiss 1991). Individuals who are high in AS may have increased sensitivity to interoceptive cues such as heart rate, blushing, or difficulty concentrating. Moreover, individuals high in AS often associate anxious cues with negative outcomes (e.g., fearing that one will have a heart attack when their heart beats fast or fearing negative social evaluation when one is sweating). AS is linked with anxiety and has been identified as a risk factor for a variety of anxiety disorders in adults (Boswell et al. 2013; Schmidt et al. 1997, 2007, 2006). These findings have been extended to children and adolescents as well (Allan et al. 2016; Knapp et al. 2016), suggesting that it is also an applicable construct in the childhood literature.

With regard to child AS, researchers have found that parental reinforcement and modeling of bodily symptoms were relevant factors (Watt and Stewart 2000; Watt et al. 1998). Parents who are high in AS tend to be more reactive to their own symptoms and potentially communicate and model catastrophic outcomes associated with their anxiety symptoms to their children (Drake and Kearney 2008; Graham and Weems 2015; Watt et al. 1998). Although a novel concept, there is some research to support that parents who are high in AS may also be reactive to their children's anxiety symptoms and may exacerbate the child's anxiety. Wissemann et al. (2018) created and validated the Parent Sensitivity to Child Anxiety Index (PSCAI) to measure the degree to which parents are sensitive to their *child's anxious behaviors*. This measure was found to have 3 subscales: Physical Concerns, Social Concerns, and Fear Concerns. The first, Physical Concerns, reflects the degree to which parents have negative thoughts when their child has a physical concern (e.g., When my child complains about aches and pains, I worry there is something terribly wrong with her/him). The second subscale, Social Concerns, reflects the extent to which a parent worries about negative social evaluation when their child displays anxious symptoms (e.g., I worry that other people will notice my child's anxiety). The third subscale, Fear Concerns, reflects the degree to which a parent experiences anxiety when their child displays anxiety (e.g., It scares me when my child appears to be afraid).

Measurement of this construct revealed that parents with high AS to their children's anxiety symptoms demonstrated greater accommodation of their child's anxiety (Wissemann et al. 2018). This pattern suggests that even when children's symptoms are at non-clinical levels, parents who demonstrate elevated responsivity to their children's anxiety often modify their own behavior and in turn may reinforce and increase anxiety in their children.

Considering that parents high in AS to their children may be modeling hyper-reactivity to their children's anxiety symptoms, we would expect these effects to be stronger when children are in the *presence of these types of caregivers*. However, research has suggested that caregiver presence may decrease anxiety in children through social regulation, such that social proximity to caregivers provides additional resources to children and allows them to be less vigilant to potential threats (Conner et al. 2012). And yet, research has shown that parental presence does not aid in reducing anxiety in children prior to an operation, especially when the parent is anxious (Bevan et al. 1990; Cameron et al. 1996; Vagnoli et al. 2010). Therefore, parental sensitivity to their child's anxiety may moderate child reactivity. However, given the novelty of this topic, we approached our scientific inquiry by examining the relationship between anxiety sensitivity for parents to their children and child neurobiological reactivity. This investigation may provide the foundation to exploring causal relationships, but that is beyond the scope of the present study.

Study Aims

In the current study, we first examined the impact of parental presence on a neural marker of threat sensitivity (i.e., the LPP; aim 1). To do so, we measured the LPP in young children (between the ages of 5 and 7 years old) while their parent was in the room and then while an experimenter was in the room (i.e., control condition). The current study used a modified version of the emotional-interrupt paradigm (Mitchell et al. 2006; Weinberg and Hajcak 2011) to elicit neural responses to negative and neutral (International Affective Picture System; Lang et al. 2008) images. We also aimed to investigate if the impact of parental presence on the LPP differs by parental sensitivity to child anxiety (i.e., environmental moderator; aim 2). The PSCAI was administered to measure parental sensitivity to child anxiety. We focused on young children due to previous work suggesting parental influence is greater early in development (Doom et al. 2015; Gee et al. 2014).

Methods

Participants

Participants included 78 parent and child dyads recruited from the community through recruitment events and flyers distributed to local businesses, libraries, and schools. Data from this study (a different ERP task) has been published in a previous study (Meyer et al. 2019). As shown in Table 1, children (52.6% male, 66.7% Caucasian, 89.7% Non-Hispanic/Non-Latino) were between 5 and 7 years of age ($M = 5.79$, $SD = 0.79$). Participating parents (also characterized in Table 1) were predominantly mothers (89.7%) as well as biological parents of the child (96.2%). Annual family income was reported as $40,000 or less for 21.8% of the sample. Correlations between demographic variables and variables of interest (i.e., LPP, PSCAI) are presented in Table 2.

Children were excluded from the present study if they were unable to complete both conditions of the task ($n = 12$) or had movement artifacts leading to poor quality of the EEG data ($n = 13$). Therefore, the final sample ($N = 78$) had valid LPP data for both the parent and experimenter conditions. Children excluded did not differ on any demographic or anxiety variables (diagnoses or PSCAI) from children included in the present study, all $ps > 0.05$.

Table 1 Descriptive characteristics of child-parent dyads. Means and standard deviations reported for continuous data, with *t* values reflecting results from *t*-tests. Frequency values reported for categorical data, with χ^2 values reflecting results from chi-square tests

	Full sample	Subthreshold or threshold anxiety disorder ($n = 32$)	No Diagnosis ($n = 46$)	*t* or χ^2
Child age	5.79 (0.79)	5.75 (0.79)	5.83 (0.79)	**0.42**
Child gender (male)	52.6%	53.1%	52.2%	0.007
Child race	–	–	–	3.56
Asian	7.7%	9.4%	6.5%	–
Black	19.2%	12.5%	23.9%	–
White	66.7%	71.9%	63.0%	–
American Indian/Alaskan Native	1.3%	3.1%	0.0%	–
Other	5.1%	3.1%	6.5%	–
Child ethnicity (non-Hispanic)	89.7%	87.5%	91.3%	0.30
Parent age	37.00 (6.36)	37.22 (6.31)	36.85 (6.45)	−0.25
Parent gender (female)^	89.7%	87.5%	91.3%	1.48
Parent education	–	–	–	0.25
High school or equivalent	3.8%	3.1%	4.3%	–
Some college or 2-year degree	28.2%	28.1%	28.3%	–
College degree	26.9%	25.0%	28.3%	–
Graduate degree	41.0%	43.8%	39.1%	–
Caretaking responsibilities	–	–	–	2.38
Parent is primary caretaker	69.2%	78.1%	63.0%	–
Parent is not primary caretaker	1.3%	0.0%	2.2%	–
Parent shares with partner	29.5%	21.9%	34.8%	–

*$p < 0.05$, **$p < 0.01$

^one parent reported their gender as neither male nor female

Materials

Emotional Interrupt Task Twenty-six developmentally appropriate pictures comprised of negative and neutral content were selected from the International Affective Picture System (IAPS; Lang et al. 2008). The 15 negative images primarily depicted humans and animals (e.g., crying child, snake, mean dog) and the 11 neutral

Table 2 Correlation matrix examining the relationship between demographic variables and key variables of interest – late positive potential (LPP) to negative and neutral images when children were in the presence of their parent and an experimenter and Parent Sensitivity to Child Anxiety Index (PSCAI) subscales

	1	2	3	4	5	6	7
1. LPP: parent negative							
2. LPP: parent neutral	0.68**						
3. LPP: experimenter negative	0.74**	0.64**					
4. LPP: experimenter neutral	0.56**	0.66**	0.71**				
5. PSCAI: Physical Concerns	0.18	0.12	0.22	0.15			
6. PSCAI: Social Concerns	−0.04	−0.01	−0.13	−0.07	0.38**		
7. PSCAI: Fear Concerns	0.12	−0.03	−0.10	0.03	0.38**	0.49**	
8. Child age	−0.24*	−0.05	−0.16	−0.06	−0.09	−0.03	−0.20
9. Child gender	0.09	0.01	−0.07	−0.07	−0.11	−0.07	−0.13
10. Child race	0.10	0.15	−0.03	−0.05	−0.17	0.02	0.02
11. Child ethnicity	−0.20	−0.26*	−0.09	−0.14	−0.05	−0.12	−0.08
12. Parent age	−0.10	−0.14	−0.01	−0.21	−0.05	0.002	−0.14
13. Parent gender	0.07	0.004	0.01	0.01	−0.03	−0.14	0.02
14. Parent education	0.10	−0.04	0.03	−0.05	−0.04	0.10	−0.04
15. Caretaking responsibility	−0.01	0.06	0.08	0.08	−0.09	−0.07	−0.14

*$p < 0.05$, **$p < 0.01$

images were inanimate or plant-based objects (e.g., cup, leaf).[1] Images were presented using Presentation software (Neurobehavioral Systems, Inc., Albany, CA) to control the presentation and timing of all stimuli. Throughout the task, children viewed a total of 20 neutral images and 20 negative images. To ensure children were actively viewing the pictures, IAPS pictures were presented via an emotional interrupt task (Mitchell et al. 2006; Weinberg and Hajcak 2011). Similar to previous studies in children and adults (Kujawa et al. 2013a, b), each trial consisted of the following stimuli in successive order: a fixation cross (+) presented for 800 ms, an IAPS picture for 1000 ms, a large arrow (11 cm length × 5.3 cm maximum height) for 150 ms, and the IAPS picture presented again for 400 ms. The interval between trials varied randomly between 1500 and 2000 ms. Children were instructed to press the left or right button on the mouse corresponding to the arrow stimulus pointing towards the left or right. Forty trials comprised the task.

Parent Sensitivity to Child Anxiety Index The Parent Sensitivity to Child Anxiety (PSCAI; Wissemann et al. 2018) is an 11-item scale designed to tap a parent's sensitivity or reactivity to his/her child's anxiety symptoms and experiences across three subscales: Physical Concerns, Social Concerns, and Fear Concerns. Items were rated from "very little" (0) to "very much" (4).

The mean total score of the PSCAI was 17.77 and the standard deviation was 6.72. Scores ranged from 0 to 37, with higher scores indicating greater parental sensitivity to their child's anxiety. PSCAI subscales scores were as follows: Physical Concerns, $M = 7.09$, $SD = 3.83$, skewness = 1.70; Social Concerns, $M = 3.58$, $SD = 1.88$, skewness = 2.33; and Fear Concerns, $M = 7.10$, $SD = 2.83$, skewness = 0.16. The Cronbach's alpha for the total PSCAI score was good, alpha = 0.85. Additionally, the PSCAI subscales had good internal consistency: Physical Concerns, alpha = 0.85; Social Concerns, alpha = 0.81; and Fear Concerns, alpha = 0.72.

Clinical Interview The Schedule for Affective Disorders and Schizophrenia for School-Age Children: Present and Lifetime Version (K-SADS-PL; Kaufman et al. 1997) was administered to parents regarding their children's current and past psychopathology. The interviews were administered by a Ph.D. level clinician and clinical research interviewers who were trained and supervised by the Ph.D. level clinician. All interviews were recorded. The K-SADS is designed to assess a range or psychopathology in children; lifetime, as well as current, diagnoses were derived from the parent report on the child. All diagnoses were reviewed in case conferences led by an experienced clinical psychologist.

In the current study, we focused on current subthreshold and threshold anxiety disorders. Consistent with prior research (e.g., Meyer et al. 2019), subthreshold disorders were defined as children who displayed at least 1 threshold symptom of a disorder in combination with at least 1 other subthreshold symptom and significant impairment related to the disorder. We opted to group the subthreshold diagnoses with the threshold diagnoses given that there were no significant differences on any of the key variables (i.e., LPP, PSCAI), all $ps > 0.05$. Overall, 27 children had at least one (or more) current subthreshold or threshold anxiety disorder: 1 subthreshold panic disorder, 11 subthreshold separation anxiety, 10 subthreshold simple phobias, 5 threshold phobias, 2 subthreshold social anxiety disorder, 2 threshold social anxiety disorder, 11 subthreshold generalized anxiety disorder, 3 threshold generalized anxiety disorder, 6 subthreshold obsessive compulsive disorder, 3 subthreshold anxiety disorder not otherwise specified (NOS), and 4 threshold anxiety disorder NOS. Based on 30 audio-recorded interviews that were scored by a second rater, interrater reliability for subthreshold and threshold anxiety disorders was good (kappa = 0.81).

Procedure

Following informed consent (parents signed consent documents and children provided verbal/written assent) and a brief description of the experiment, including viewing the IAPS pictures to ensure that both parents and children were willing to complete the emotional interrupt task, EEG electrodes were attached. Children completed the IAPS task, along with a range of other EEG tasks and self-report measures (reported elsewhere) lasting approximately 2–3 h. Children were randomized to have their parent or a research assistant (i.e., experimenter) in the room during the first condition of the emotional interrupt task, and then the other individual was present for the second condition; the order was counterbalanced across participants to eliminate order effects.[2] In the parent condition, parents were instructed to sit in a chair next to their child (approximately 2–3 ft away, i.e., in the child's periphery) and watch their child complete the task. During the experimenter condition, the experimenter sat in the same chair next to the child.

Within each condition, the task consisted of 40 trials and each IAPS picture was presented either once or twice per condition (i.e., children viewed 20 neutral images and 20 negative images). Prior to the start of each condition, children were informed that the pictures they viewed earlier would

[1] The following IAPS pictures presented in color: 1050, 1120, 1201, 1300, 1321, 1930, 2120, 2130, 2780, 2810, 2900, 3280, 5740, 5750, 5800, 5970, 7002, 7004, 7006, 7009, 7010, 7025, 7035, 7090, 7380, 9582

[2] Randomization was efficacious, i.e., no order effects for condition, X^2 (2, $N = 78$) = 0.82, $p = 0.37$.

appear on the screen and they were instructed to respond to the arrow stimulus by pressing the right or left button on the mouse. Given the primary objective of this task was to view negatively- and neutrally-valenced pictures and the response task was present only to maintain attention (i.e., accuracy was not assessed), no practice trials were completed. Instead, children began the task immediately after the examiner initiated it and the child hit the space bar in response to the "Ready" stimuli.

Psychophysiological Reading and Data Reduction

Continuous EEG recordings were collected using a 34-channel BioSemi system (BioSemi, Amsterdam, Netherlands) based on the 10/20 system. Electrodes were also placed on the left and right mastoids. Electrooculogram (EOG) was recorded to quantify eye blinks and movements; facial electrodes were placed approximately one cm above and below the right eye (one above, two below), and one cm to the right and left of right and left eyes, respectively (two total). During acquisition, the ground electrode was formed by the Common Mode Sense (CMS) active electrode and the Driven Right Leg (DRL) passive electrode, although all data were re-referenced to the left and right mastoid for data analysis. The data were digitized using Brain Vision Analyzer software (Brain Products, Gilching, Germany) at a sampling rate of 1024 Hz. A low and high band-pass filter was applied, with cutoffs of 0.1 and 30 Hz, respectively. EEG data were segmented for each trial, beginning at 500 ms before each picture onset and ending 1000 ms after the initial presentation of the image. Data were corrected for eye blinks (Gratton et al. 1983) and horizontal and vertical eye movement. Semi-automatic artifact rejection was then used to remove artifacts with a voltage step of more than 50 μV between sample points, a voltage difference of 300 μV within a trial, or a maximum voltage difference of less than 0.5 μV within 100 ms intervals. Visual inspection of the waveforms was used to reject additional trials that were unnecessarily noisy.

ERPs were constructed by averaging negative and neutral pictures separately and then baseline corrected to 200 ms prior to image onset. Parietal and occipital electrodes were pooled (Pz, P3, P4, Oz, O1, O2) and the mean activity between 400 and 1000 ms was exported for data analyses. Split-half reliability (Spearman Brown Adjusted) of the LPP during this task was acceptable: parent condition negative images, $r = 0.82$; parent condition neutral images, $r = 0.70$;experimenter condition negative images, $r = 0.82$; and experimenter neutral images, $r = 0.79$. Moreover, after removing artifacts, we retained approximately 19 trials per condition: number of trials for experimenter negative condition, M = 19.35, SD = 1.18, range = 10–20; number of trials for experimenter neutral condition, M = 19.53, SD = 0.75, range = 17–20; number of trials for parent negative condition, M = 19.50, SD = 1.07, range = 15–20; and number of trials for parent neutral condition, M = 19.30, SD = 1.20, range = 14–20. The number of trials did not differ between conditions, all ps > 0.10.

Data Analytic Plan

For statistical analyses, we used SPSS (Version 24.0 for Mac) General Linear Model software. All main study variables were visually inspected for outliers using frequency distributions and none were identified. To address aim 1, we conducted a 2 × 2 repeated measures analysis of variance (RM-ANOVA) to examine the within-subjects LPP potentiation for picture valence (negative vs. neutral) and condition (parent vs. experimenter); initially the RM-ANOVA had no covariates. To address aim 2, we conducted the same 2 × 2 RM-ANOVA with the PSCAI subscales as covariates. Paired samples t-tests and correlations were utilized to probe significant interactions.

Results

Late Positive Potential: The Impact of Parental Presence

To examine the effect of valence and condition on the LPP, we conducted a 2 × 2 RM- ANOVA (negative vs. neutral; parent vs. experimenter). Overall, the LPP was larger (i.e., more positive) during negative picture viewing ($M = 125.66$, $SD = 87.74$) compared to neutral picture viewing ($M = 95.50$, $SD = 79.05$), $F(1, 77) = 24.96$, $p < 0.001$, $\eta p^2 = 0.25$, consistent with previous work (Fig. 1). While there was not a main effect of condition, $F(1, 77) = 0.18$, $p = 0.90$, there was a significant two-way interaction between valence and condition, $F(1,77) = 6.42$, $p = 0.01$, $\eta p^2 = 0.08$.

Post-hoc paired samples *t*-tests revealed that the LPP was more positive during negative picture viewing compared to neutral picture viewing for both the parent, $t(77) = 5.62$, $p < 0.001$, $d = 0.64$, and experimenter conditions, $t(77) = 2.56$, $p = 0.01$, $d = 0.29$. To deconstruct the two-way interaction between valence and condition, we created subtraction-based difference scores by subtracting the LPP during neutral picture viewing from the LPP during negative picture viewing for the parent condition and for the experimenter condition. Results of the paired samples *t*-test comparing the parent and experimenter subtraction-based difference scores indicated that there was a larger difference between negative and neutral picture viewing for the parent condition, $M = 41.08$, $SD = 64.58$, than the experimenter condition, $M = 19.26$, $SD = 66.41$, $t(77) = 2.53$, $p = 0.01$, $d = 0.29$ (see Fig. 1). That is, parental presence potentiated the LPP for negative picture viewing relative to neutral picture viewing to a greater degree than experimenter presence.

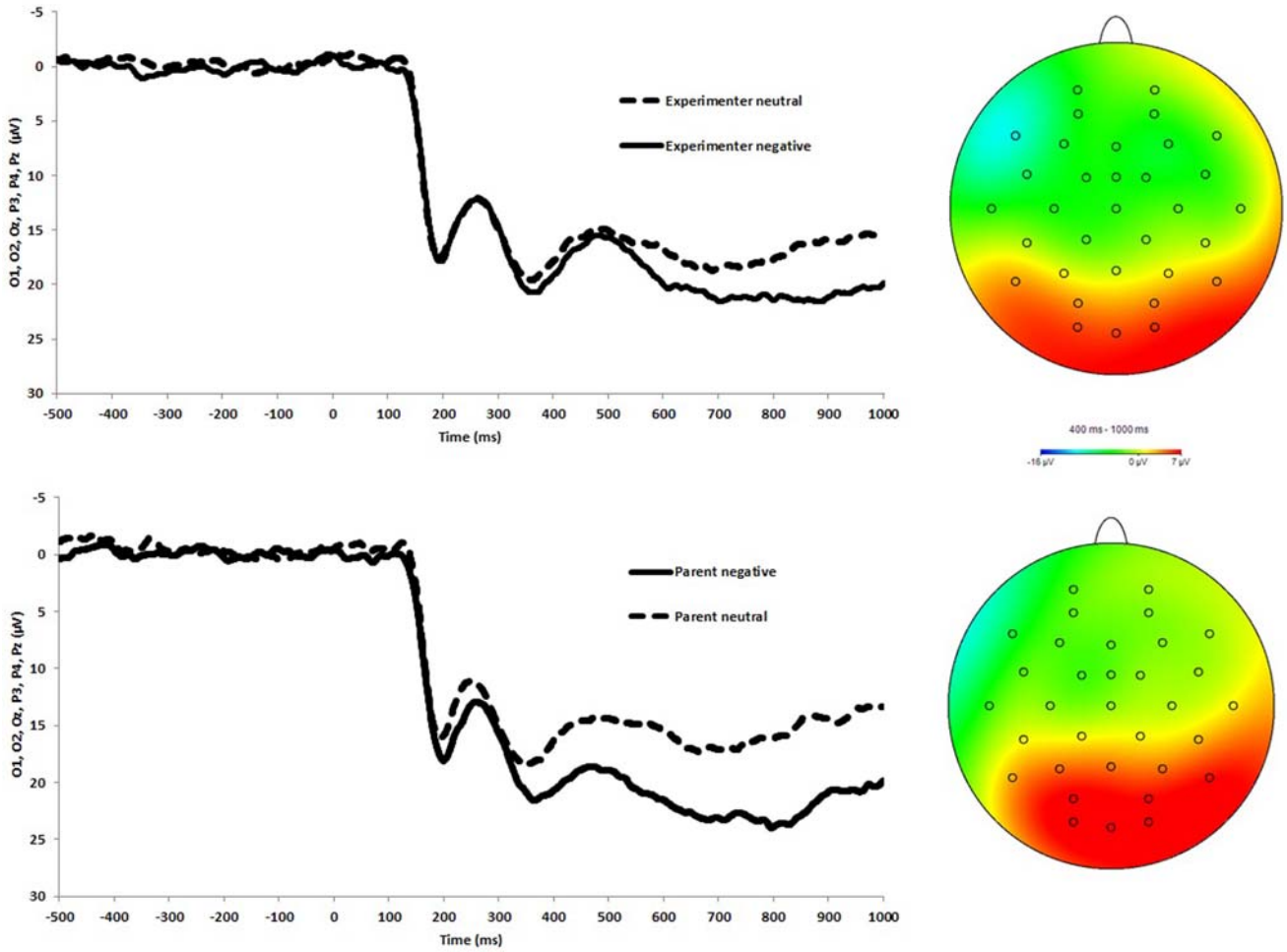

Fig. 1 On the *left*, the graphs depict response-locked ERP waveforms for negative and neutral picture viewing trials for the experimenter (top) and parent conditions (bottom). On the *right*, Topographic maps of activity (negative minus neutral) in the LPP window of 400 to 1000 ms are also presented for the experiment (top) and parent conditions (bottom)

Parent Sensitivity to Child Anxiety and the LPP

Next, we wished to examine to what extent individual differences in parent sensitivity to child anxiety may relate to the parent-potentiation of the LPP. We conducted an additional 2 (negative vs. neutral) × 2 (parent vs. experimenter) RM-ANOVA, with the PSCAI subscales (Physical Concerns, Social Concerns, Fear Concerns) entered simultaneously as covariates. There was a significant three-way interaction between valence, condition, and the PSCAI Fear Concerns subscale, $F(1, 74) = 10.25$, $p = 0.002$, $\eta p^2 = 0.12$.[3] Significant three-way interactions did not occur for the other PSCAI subscales, $ps > 0.20$. The two-way interaction of valence by condition, $F(1, 74) = 0.80$, $p = 0.37$, as well as the main effect of valence, $F(1, 74) = 2.29$, $p = 0.13$, were non-significant when parental anxiety sensitivity to their child's anxiety was included in the model. The main effect of condition remained non-significant, $F(1, 74) = 0.68$, $p = 0.41$.

To probe the three-way interaction, we conducted two additional RM-ANOVAs for each condition. In the first, we entered valence (negative vs. neutral) in the parent condition and the Fear Concerns subscale of the PSCAI was entered as a covariate. Results suggested that the 2-way interaction between valence and Fear Concerns did not reach significance, $F(1,76) = 2.64$, $p = 0.11$. Additionally, when we ran a similar RM-ANOVA for the experimenter condition, results also failed to support a significant 2-way interaction between valence and Fear Concerns, $F(1, 76) = 2.37$, $p = 0.10$.

Given the non-significant condition-specific 2-way interactions, we created additional subtraction-based difference scores to clarify the significant three-way interaction. Notably, these difference scores were calculated utilizing a different approach than described above; that is, the

[3] The pattern of results is consistent when controlling for child age and ethnicity (i.e., the three-way interaction with PSCAI Fear remains significant), $F(1, 72) = 9.43$, $p = 0.003$, $\eta p^2 = 0.12$.

experimenter condition was subtracted from the parent condition for each valence (i.e., negative parent minus negative experimenter, and neutral parent minus neutral experimenter). Results indicated that the LPP during negative picture viewing in the parent condition (relative to the experimenter condition) was related to PSCAI Fear Concerns, $r(76) = 0.29$, $p = 0.01$. However, the difference between the neutral conditions (parent minus experimenter) was not significantly related to the PSCAI Fear Concerns subscale, $r(76) = -0.07$, $p = 0.52$. Thus, parents with higher sensitivity to their child's fear appeared to potentiate the LPP during negative picture viewing (but not neutral picture viewing) to a greater degree than parents with lower sensitivity to their children's fear.

We also created a third type of subtraction-based difference score. To isolate the emotional modulation of the LPP specific to the parent condition, we subtracted the previously-derived experimenter difference score (i.e., negative experimenter minus neutral experimenter) from the parent difference score (i.e., negative parent minus neutral parent). This value indicates the extent to which parental presence (relative to experimenter presence) potentiates the LPP to *negative* compared to neutral images. The unique variance related to parent potentiation was significantly related to the PSCAI Fear Concerns subscale, $r(76) = 0.31$, $p < 0.01$ (Fig. 2).

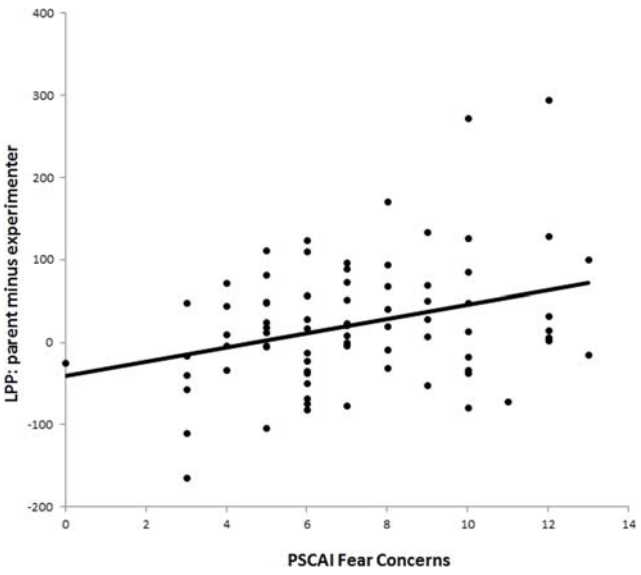

Fig. 2 Scatterplot depicting the relationship between the parent potentiation of the LPP to negative images (i.e., extent to which parental presence, relative to experimenter presence, increased the LPP to negative compared to neutral images) and the Parent Sensitivity to Child Anxiety Index (PSCAI) Fear Concerns subscale. Parents with higher sensitivity to their child's fear appeared to potentiate the LPP during negative picture viewing (but not neutral picture viewing) to a greater degree than parents with lower sensitivity to their children's fear

Supplemental Exploratory Analyses: Child Anxiety Disorders, the LPP, and Parent Sensitivity to Child Anxiety

Given the demonstrated relationship between anxiety and the LPP, as well as the theoretical link between parental anxiety sensitivity and child anxiety, we aimed to examine the clinical relevance of our findings as they relate to anxiety diagnoses measured by the K-SADS. We probed the extent to which the LPP during both the parent and experimenter condition, as well as parent sensitivity to child anxiety, differed between anxious and non-anxious children. As indicated in Table 3, results suggested that the LPP to negative images was larger in anxious children ($M = 156.97$, $SD = 101.65$) compared to non-anxious children ($M = 112.47$, $SD = 62.67$), but only when the parent was present, $t(76) = -2.39$, $p = 0.02$, $d = 0.53$. Similarly, LPP activity in response to negative images during the parent condition predicted child anxiety diagnostic status (above and beyond child age), $\chi^2(1, N = 78) = 4.56$, $p = 0.03$. The LPP did not differ between anxious and non-anxious children during any other condition, all ps > 0.05.

Likewise, total scores on the PSCAI were increased in parents of children with threshold and subthreshold anxiety ($M = 20.59$, $SD = 6.36$) compared to parents of non-anxious children ($M = 15.80$, $SD = 6.32$), $t(76) = -3.29$, $p = 0.002$, $d = 0.76$. Scores on the PSCAI Fear Concerns were significantly increased amongst parents of children with anxiety ($M = 8.56$, $SD = 2.60$) compared to parents of non-anxious children ($M = 6.09$, $SD = 2.55$), $t(76) = -4.19$, $p < 0.001$, $d = 0.96$. Additionally, scores on the PSCAI Social Concerns subscale differed between parents of children with anxiety ($M = 4.19$, $SD = 2.35$), and without anxiety ($M = 3.15$, $SD = 1.35$), $t(76) = -2.47$, $p = 0.02$, $d = 0.54$. However, scores on the PSCAI Physical Concerns subscale were comparable between groups, $p > 0.05$. Collectively, these additional analyses provide some evidence of validity and clinical relevance with regard to our novel findings demonstrating parental potentiation of the LPP to negative images, specifically among parents with high anxiety sensitivity to their children's fear.

Discussion

Results from the current study suggest that when children are in the presence of their parent (compared to an experimenter), they display a larger LPP to negative stimuli. Interestingly, as supported by the significant three-way interaction, the present study further revealed that LPP activity was moderated by parental response to their child's anxiety symptoms, such that children with parents who were overly reactive to their children's anxiety symptoms (i.e., elevated score on the fear subscale of the PSCAI) had the greatest LPP response when viewing threatening stimuli in their parent's presence.

Table 3 Means and standard deviations for the late positive potential (LPP) to negative and neutral images when children were in the presence of their parent and an experimenter, as well as the Parent Sensitivity to Child Anxiety Index (PSCAI) total and subscales in children with a subthreshold/threshold anxiety disorder and children with no anxiety disorder. In the right column, *t* values reflect results from *t*-tests examining differences between the groups

	Subthrehsold or threshold anxiety disorder ($n = 32$)	No diagnosis ($n = 46$)	*t*
LPP parent present: negative images	156.97 (101.65)	112.47 (62.67)	−2.39*
LPP parent present: neutral images	100.87 (98.67)	81.84 (59.54)	−1.06
LPP experimenter present: negative images	140.77 (116.35)	106.57 (68.69)	−1.63
LPP experimenter present: neutral images	118.35 (96.65)	89.52 (64.83)	−1.58
PSCAI total	20.59 (6.36)	15.80 (6.32)	−3.29**
PSCAI: Physical Concerns	7.84 (3.77)	6.57 (3.82)	−1.46
PSCAI: Social Concerns	4.19 (2.35)	3.15 (1.35)	−2.47*
PSCAI: Fear Concerns	8.56 (2.60)	6.09 (2.55)	−4.19**

*$p < 0.05$, **$p < 0.01$

Moreover, exploratory analyses indicated that children with anxiety were characterized by increased LPPs when viewing negative images compared to non-anxious children, but only when the LPP was measured with parents in the room. Overall, findings from the current study are novel and extend previous work by examining the relationship between parenting, the LPP, parental presence, and anxiety in children.

This is the first study, to our knowledge, to examine the impact of any observer on the magnitude of the LPP. Although previous work has examined the extent to which an observer impacts other neural markers of threat-sensitivity (e.g., the error-related negativity; Barker et al. 2015; Barker et al. 2018; Buzzell et al. 2017; Kim et al. 2005; Voegler et al. 2018), no previous study has examined this in relation to the LPP. Indeed, previous work suggests that the error-related negativity is increased when an observer is present – particularly in anxious individuals (Barker et al. 2015). Moreover, the current findings are broadly consistent with previous work suggesting that peer presence influences the neural response to reward in youth insofar as social context appears to modulate neural markers of both threat and reward (Van Hoorn et al. 2018).

In the current study, we examine the extent to which the presence of a child's parent compared to a stranger (i.e., the experimenter) impacts neural reactivity to negative and neutral images. Results suggested that children displayed increased neural reactivity to negative compared to neutral images in both conditions; however, the LPP to negative images was *increased more when parents were in the room*. This is a surprising finding to the extent that one might expect the presence of a parent to be comforting to a child and thus reduce reactivity to threat (Gee et al. 2014). However, in light of the significant three-way interaction incorporating parental sensitivity to children's anxiety symptoms (i.e., the fear subscale of the PSCAI), these findings are less surprising and seem to be related to a specific aspect of parenting approach. As revealed by post-hoc analyses, parents who reported being elevated on items such as "It scares me when my child appears to be afraid" appear to potentiate their children's LPP to negative images. In contrast, parents who reported being low on the PSCAI Fear Concerns did not increase the LPP during the parental presence condition to the same extent as parents elevated in this construct, thereby suggesting that parental sensitivity to child anxiety is an environmental moderator (as further discussed below). Thus, being overly reactive to children's anxiety may increase this neural correlate of anxiety (i.e., the LPP).

This is also the first study to examine parental sensitivity to children's anxiety (i.e., the PSCAI) in relation to a physiological or neural marker in children. Results suggest that the presence of parents who report being elevated on the fear subscale of the PSCAI may up-regulate children's neural reactivity to negative stimuli (i.e., the LPP). It is possible that parents who experience distress in response to their children's anxiety display negative and exaggerated reactions. Parents who are elevated on this scale endorse items such as: "It is distressing for me when my child appears nervous." Perhaps this distress was noticeable to children, either during the experimental manipulation should they shift their eyes from the computer or as a learned behavior such that children expect their parents to respond during the experiment in a similar manner as they do during real life situations; for example, parents may begin sweating, display fearful facial expressions, or make verbalizations related to their fear in response to their children's anxiety. This may further up-regulate children's fear and/or indicate to them that the feared stimuli are actually dangerous, and they should *increase their vigilance towards it*. Thus, the presence of a parent who is high in sensitivity to their child's anxiety may actually increase their child's engagement with fearful stimuli and increase their child's reactivity to threat, thereby resulting in an increased LPP to negative stimuli. Future work using behavioral coding or child-report is needed to clarify this mechanism.

We also examined how the two variables of interest – the LPP and the PSCAI – differed among children with and without clinical levels of anxiety. We opted to include children

with both threshold and subthreshold anxiety diagnoses in our analyses, consistent with previous work (e.g., Meyer et al. 2019). Children with subthreshold diagnoses displayed at least one threshold anxiety symptom as well as significant impairment related to their anxiety. Importantly, there is some evidence that subthreshold levels of anxiety are clinically relevant and predict future psychopathology (Carter et al. 2001; Karsten et al. 2011). Moreover, our findings demonstrated that children with threshold and subthreshold diagnoses did not differ on key variables of interest (i.e., the LPP and PSCAI).

Interestingly, children in the current study who met criteria for a threshold or subthreshold anxiety disorder were characterized by an increased LPP to negative images, but *only during the parent condition*. The LPP to negative images when an experimenter was in the room was not increased in anxious children. In terms of utilizing the LPP as a neural correlate or marker of risk, results from the current study suggest that the LPP should be measured in young children with a parent in the room. It is possible that parental presence increases the psychological relevance of this measure (for the reasons discussed above), and thus this approach may be useful in utilizing the LPP to threatening images as a biomarker of risk for anxiety. Additionally, children with anxiety were characterized by increased PSCAI scores, which were predominantly driven by increases in the PSCAI Fear Concerns subscale (i.e., large effect characterizing group differences). That is, parents of children with clinically significant anxiety reported being more anxious in response to their children demonstrating fear or apprehension. As indicated above, we posit that parents may be reinforcing, maybe unknowingly, normative anxious thoughts and behaviors in their young children (e.g., parent appearing anxious and permitting avoidance when child expresses an age-appropriate fear of the dark), which in turn may exacerbate anxiety. However, longitudinal studies are needed to clarify whether parental sensitivity to child anxiety is a precursor to the onset of anxiety in children (i.e., mediation models).

It is important to recognize that the current study is limited insofar as the LPP was not measured while the child was alone in the room. Thus, current results do not indicate the extent to which the LPP measured while someone is alone relates to the LPP measured while in the presence of another individual. Future work should examine this issue by comparing the way the LPP is typically measured (i.e., the LPP measured while someone is alone) to the LPP measured with an observer present. Indeed, in previous work, the LPP to negative images (measured while children are alone in a room) has been linked to anxiety (DeCicco et al. 2012). Therefore, it was somewhat unexpected that there was no demonstrated link between examiner presence and anxiety when children were viewing negatively-valenced images. It is possible that having an experimenter in the room is distracting and reduces the LPP/anxiety link. Future research is needed to clarify this issue and also elucidate the role that the parent plays in the experimental manipulation, including observational coding to clarify how parents may react differently than the experimenter or eye tracking to examine the extent to which the child shifts their attention away from the screen when the parent versus experimenter is in the room. Moreover, in the current investigation, we did not include images depicting positive emotions. Future work should examine the extent to which parenting or parent sensitivity to child anxiety may relate to children's neural reactivity to positive images. To add on, in the current study, we did not measure accuracy or reaction time during the task. Further research should examine the impact of parental presence on these measures. And, we did not video record children complete the task and are therefore unable to determine if children were looking at their parent or the experimenter during task completion, thus resulting in noisy EEG data and trials that were potentially removed. Future work should investigate this possibility. Finally, given the relatively small sample size in the current study, replication in larger samples is warranted. The base rate of clinically significant anxiety diagnoses limited the size of our threshold group. Additionally, replication in larger samples would allow us to further examine the influence of key demographic variables (e.g., age, race, ethnicity) on the relationship between LPP activity, parental sensitivity, and child anxiety.

The results from the current investigation were novel as no previous study had examined the impact of parental presence on the LPP in children, nor the interaction with parent anxiety sensitivity. However, due to the lack of previous work in this area and the exploratory nature of the current investigation, results should be interpreted with caution and future studies are needed to replicate the current findings. If supported, these findings set the stage for future studies examining the LPP as a longitudinal biomarker of risk for anxiety, the role of parenting in shaping that relationship, as well as the development of novel intervention strategies aimed at reducing parental sensitivity to children's anxiety. Considering data suggesting that anxiety sensitivity can be reduced via brief, computerized intervention strategies (Norr et al. 2017; Schmidt et al. 2017; Short et al. 2017), and in light of the fact that parent sensitivity to child anxiety may be related to increases in children's LPP to threatening images, it may be fruitful to examine whether intervening on parent sensitivity to child anxiety may reduce risk for anxiety in children across time.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Informed consent Informed consent was obtained from all individual participants included in the study.

Ethical Approval All procedures performed involving human participants were in accordance with the ethical standards of the Florida State University Institutional Review Board (IRB) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

References

Allan, N. P., Felton, J. W., Lejuez, C. W., MacPherson, L., & Schmidt, N. B. (2016, May). Longitudinal investigation of anxiety sensitivity growth trajectories and relations with anxiety and depression symptoms in adolescence. *Development and Psychopathology, 28*(2), 459–469. https://doi.org/10.1017/S0954579415000590.

Auerbach, R. P., Stanton, C. H., Proudfit, G. H., & Pizzagalli, D. A. (2015, May). Self-referential processing in depressed adolescents: A high-density event-related potential study. *Journal of Abnormal Psychology, 124*(2), 233–245. https://doi.org/10.1037/abn0000023.

Auerbach, R. P., Bondy, E., Stanton, C. H., Webb, C. A., Shankman, S. A., & Pizzagalli, D. A. (2016). Self-referential processing in adolescents: Stability of behavioral and ERP markers. *Psychophysiology, 53*(9), 1398–1406.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1–24.

Barker, T. V., Troller-Renfree, S., Pine, D. S., & Fox, N. A. (2015). Individual differences in social anxiety affect the salience of errors in social contexts. *Cognitive, Affective, & Behavioral Neuroscience, 15*(4), 723–735.

Barker, T. V., Troller-Renfree, S. V., Bowman, L. C., Pine, D. S., & Fox, N. A. (2018). *Social influences of error monitoring in adolescent girls. Psychophysiology*, e13089.

Bayart, F., Hayashi, K. T., Faull, K. F., Barchas, J. D., & Levine, S. (1990). Influence of maternal proximity on behavioral and physiological responses to separation in infant rhesus monkeys (Macaca mulatta). *Behavioral Neuroscience, 104*(1), 98–107.

Beck, A. T., & Rush, A. J. (1985). A cognitive model of anxiety formation and anxiety resolution. *Issues in Mental Health Nursing, 7*(1–4), 349–365.

Belsky, J., & de Haan, M. (2011). Annual research review: Parenting and children's brain development: The end of the beginning. *Journal of Child Psychology and Psychiatry, 52*(4), 409–428.

Bevan, J. C., Johnston, C., Haig, M. J., Tousignant, G., Lucy, S., Kirnon, V., Assimes, I. K., & Carranza, R. (1990). Preoperative parental anxiety predicts behavioural and emotional responses to induction of anaesthesia in children. *Canadian Journal of Anaesthesia, 37*(2), 177–182.

Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004, Feb). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience, 7*(2), 184–188. https://doi.org/10.1038/nn1173.

Bittner, A., Egger, H. L., Erkanli, A., Jane Costello, E., Foley, D. L., & Angold, A. (2007). What do childhood anxiety disorders predict? *Journal Of Child Psychology And Psychiatry. And Allied Disciplines, 48*(12), 1174–1183.

Blackford, J. U., & Pine, D. S. (2012). Neural substrates of childhood anxiety disorders: A review of neuroimaging findings. *Child and Adolescent Psychiatric Clinics, 21*(3), 501–525.

Boswell, J. F., Farchione, T. J., Sauer-Zavala, S., Murray, H. W., Fortune, M. R., & Barlow, D. H. (2013, Sep). Anxiety sensitivity and interoceptive exposure: A transdiagnostic construct and change strategy. *Behavior Therapy, 44*(3), 417–431. https://doi.org/10.1016/j.beth.2013.03.006.

Buzzell, G. A., Troller-Renfree, S. V., Barker, T. V., Bowman, L. C., Chronis-Tuscano, A., Henderson, H. A., Kagan, J., Pine, D. S., & Fox, N. A. (2017). A neurobehavioral mechanism linking behaviorally inhibited temperament and later adolescent social anxiety. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(12), 1097–1105.

Cameron, J. A., Bond, M. J., & Pointer, S. C. (1996). Reducing the anxiety of children undergoing surgery: Parental presence during anaesthetic induction. *Journal of Paediatrics and Child Health, 32*(1), 51–56.

Carter, R. M., Wittchen, H. U., Pfister, H., & Kessler, R. C. (2001). One-year prevalence of subthreshold and threshold DSM-IV generalized anxiety disorder in a nationally representative sample. *Depression and Anxiety, 13*(2), 78–88.

Chorpita, B. F., & Barlow, D. H. (1998). The development of anxiety: The role of control in the early environment. *Psychological Bulletin, 124*(1), 3–21.

Conner, O. L., Siegle, G. J., McFarland, A. M., Silk, J. S., Ladouceur, C. D., Dahl, R. E., Coan, J. A., & Ryan, N. D. (2012). Mom—It helps when you're right here! Attenuation of neural stress markers in anxious youths whose caregivers are present during fMRI. *PLoS One, 7*(12), e50680.

Cuthbert, B. N., Schupp, H. T., Bradley, M. M., Birbaumer, N., & Lang, P. J. (2000). Brain potentials in affective picture processing: Covariation with autonomic arousal and affective report. *Biological Psychology, 52*(2), 95–111.

DeCicco, J. M., Solomon, B., & Dennis, T. A. (2012, Jan). Neural correlates of cognitive reappraisal in children: An ERP study. *Developmental Cognitive Neuroscience, 2*(1), 70–80. https://doi.org/10.1016/j.dcn.2011.05.009.

Doom, J. R., Hostinar, C. E., VanZomeren-Dohm, A. A., & Gunnar, M. R. (2015, Sep). The roles of puberty and age in explaining the diminished effectiveness of parental buffering of HPA reactivity and recovery in adolescence. *Psychoneuroendocrinology, 59*, 102–111. https://doi.org/10.1016/j.psyneuen.2015.04.024.

Drake, K. L., & Kearney, C. A. (2008). Child anxiety sensitivity and family environment as mediators of the relationship between parent psychopathology, parent anxiety sensitivity, and child anxiety. *Journal of Psychopathology and Behavioral Assessment, 30*(2), 79–86.

Eley, T. C., Bolton, D., O'Connor, T. G., Perrin, S., Smith, P., & Plomin, R. (2003). A twin study of anxiety-related behaviours in pre-school children. *Journal of Child Psychology and Psychiatry, 44*(7), 945–960.

Foti, D., & Hajcak, G. (2008, Jun). Deconstructing reappraisal: Descriptions preceding arousing pictures modulate the subsequent neural response. *Journal of Cognitive Neuroscience, 20*(6), 977–988. https://doi.org/10.1162/jocn.2008.20066.

Foti, D., Hajcak, G., & Dien, J. (2009, May). Differentiating neural responses to emotional pictures: Evidence from temporal-spatial PCA. *Psychophysiology, 46*(3), 521–530.

Gee, D. G., Gabard-Durnam, L., Telzer, E. H., Humphreys, K. L., Goff, B., Shapiro, M., Flannery, J., Lumian, D. S., Fareri, D. S., & Caldera, C. (2014). Maternal buffering of human amygdala-prefrontal circuitry during childhood but not during adolescence. *Psychological Science, 0956797614550878*.

Gerlsma, C., Emmelkamp, P. M., & Arrindell, W. A. (1990). Anxiety, depression, and perception of early parenting: A meta-analysis. *Clinical Psychology Review, 10*(3), 251–277.

Graham, R. A., & Weems, C. F. (2015, Jul). Identifying moderators of the link between parent and child anxiety sensitivity: The roles of gender, positive parenting, and corporal punishment. *Journal of Abnormal Child Psychology, 43*(5), 885–893. https://doi.org/10.1007/s10802-014-9945-y.

Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology, 55*(4), 468–484. https://doi.org/10.1016/0013-4694(83)90135-9.

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*(1), 199–220.

Guyer, A. E., Lau, J. Y. F., McClure-Tone, E. B., Parrish, J., Shiffrin, N. D., Reynolds, R. C., Chen, G., Blair, R. J. R., Leibenluft, E., & Fox, N. A. (2008). Amygdala and ventrolateral prefrontal cortex function during anticipated peer evaluation in pediatric social anxiety. *Archives of General Psychiatry, 65*(11), 1303–1312.

Hajcak, G., & Dennis, T. A. (2009, Mar). Brain potentials during affective picture processing in children. *Biological Psychology, 80*(3), 333–338. https://doi.org/10.1016/j.biopsycho.2008.11.006.

Hajcak, G., & Olvet, D. M. (2008, Apr). The persistence of attention to emotion: Brain potentials during and after picture presentation. *Emotion, 8*(2), 250–255. https://doi.org/10.1037/1528-3542.8.2.250.

Hajcak, G., Weinberg, A., MacNamara, A., & Foti, D. (2012). ERPs and the study of emotion. *The Oxford Handbook of Event-Related Potential Components*, 441-474.

Harris, J. R. (2002). Beyond the nurture assumption: Testing hypotheses about the child's environment. *Parenting and the child's world: Influences on academic, intellectual, and social-emotional development*, 3–20.

Huffmeijer, R., Bakermans-Kranenburg, M. J., Alink, L. R., & van Ijzendoorn, M. H. (2014, May 10). Reliability of event-related potentials: The influence of number of trials and electrodes. *Physiology & Behavior, 130*, 13–22. https://doi.org/10.1016/j.physbeh.2014.03.008.

Kagan, J. (2003). Biology, context, and developmental inquiry. *Annual Review of Psychology, 54*(1), 1–23.

Karsten, J., Hartman, C. A., Smit, J. H., Zitman, F. G., Beekman, A. T., Cuijpers, P., van der Does, A. W., Ormel, J., Nolen, W. A., & Penninx, B. W. (2011). Psychiatric history and subthreshold symptoms as predictors of the occurrence of depressive or anxiety disorder within 2 years. *The British Journal of Psychiatry, 198*(3), 206–212.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., Williamson, D., & Ryan, N. (1997). Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(7), 980–988.

Kessler, R. C., & Greenberg, P. E. (2002). The economic burden of anxiety and stress disorders. *Neuropsychopharmacology: The fifth generation of progress, 67*, 982–992.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). *Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication* (Vol. 62 SRC - GoogleScholar).

Kim, E. Y., Iwaki, N., Uno, H., & Fujita, T. (2005). Error-related negativity in children: Effect of an observer. *Developmental Neuropsychology, 28*(3), 871–883.

Knapp, A. A., Blumenthal, H., Mischel, E. R., Badour, C. L., & Leen-Feldner, E. W. (2016, Feb). Anxiety sensitivity and its factors in relation to generalized anxiety disorder among adolescents. *Journal of Abnormal Child Psychology, 44*(2), 233–244. https://doi.org/10.1007/s10802-015-9991-0.

Kujawa, A., Klein, D. N., & Proudfit, G. H. (2013a, Oct). Two-year stability of the late positive potential across middle childhood and adolescence. *Biological Psychology, 94*(2), 290–296. https://doi.org/10.1016/j.biopsycho.2013.07.002.

Kujawa, A., Weinberg, A., Hajcak, G., & Klein, D. N. (2013b, Jul). Differentiating event-related potential components sensitive to emotion in middle childhood: Evidence from temporal-spatial PCA. *Developmental Psychobiology, 55*(5), 539–550. https://doi.org/10.1002/dev.21058.

Kujawa, A., MacNamara, A., Fitzgerald, K. D., Monk, C. S., & Phan, K. L. (2015, Nov). Enhanced neural reactivity to threatening faces in anxious youth: Evidence from event-related potentials. *Journal of Abnormal Child Psychology, 43*(8), 1493–1501. https://doi.org/10.1007/s10802-015-0029-4.

Kujawa, A., Hajcak, G., Danzig, A. P., Black, S. R., Bromet, E. J., Carlson, G. A., Kotov, R., & Klein, D. N. (2016, Sep 1). Neural reactivity to emotional stimuli prospectively predicts the impact of a natural disaster on psychiatric symptoms in children. *Biological Psychiatry, 80*(5), 381–389. https://doi.org/10.1016/j.biopsych.2015.09.008.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). International affective picture system (IAPS): Affective ratings of pictures and instruction manual. *Technical Report A-8*.

Lebowitz, E. R., Woolston, J., Bar-Haim, Y., Calvocoressi, L., Dauser, C., Warnick, E., Scahill, L., Chakir, A. R., Shechner, T., Hermes, H., Vitulano, L. A., King, R. A., & Leckman, J. F. (2013, Jan). Family accommodation in pediatric anxiety disorders. *Depression and Anxiety, 30*(1), 47–54. https://doi.org/10.1002/da.21998.

Leppanen, J. M., Moulson, M. C., Vogel-Farley, V. K., & Nelson, C. A. (2007). An ERP study of emotional face processing in the adult and infant brain [research support, N.I.H., extramural research support, non-U.S. Gov't]. *Child Development, 78*(1), 232–245.

Levine, S. (2001). Primary social relationships influence the development of the hypothalamic–pituitary–adrenal axis in the rat. *Physiology & Behavior, 73*(3), 255–260.

Maccoby, E. E. (2002). Parenting effects: Issues and controversies.

MacNamara, A., & Hajcak, G. (2009, Nov). Anxiety and spatial attention moderate the electrocortical response to aversive pictures. *Neuropsychologia, 47*(13), 2975–2980. https://doi.org/10.1016/j.neuropsychologia.2009.06.026.

MacNamara, A., & Hajcak, G. (2010, Mar). Distinct electrocortical and behavioral evidence for increased attention to threat in generalized anxiety disorder. *Depression and Anxiety, 27*(3), 234–243. https://doi.org/10.1002/da.20679.

McLeod, B. D., Wood, J. J., & Weisz, J. R. (2007). Examining the association between parenting and childhood anxiety: A meta-analysis. *Clinical Psychology Review, 27*(2), 155–172.

Meyer, A., Proudfit, G. H., Bufferd, S. J., Kujawa, A. J., Laptook, R. S., Torpey, D. C., & Klein, D. N. (2015). Self-reported and observed punitive parenting prospectively predicts increased error-related brain activity in six-year-old children. *Journal of Abnormal Child Psychology, 43*(5), 821–829.

Meyer, A., Carlton, C., Chong, L. J., & Wissemann, K. (2019). The presence of a controlling parent is related to an increase in the error-related negativity in 5 - 7 year-old children. *Journal of Abnormal Child Psychology, 47*(6), 935–945.

Mitchell, D. G., Richell, R. A., Leonard, A., & Blair, R. J. (2006, Aug). Emotion at the expense of cognition: Psychopathic individuals outperform controls on an operant response task. *Journal of Abnormal Psychology, 115*(3), 559–566. https://doi.org/10.1037/0021-843X.115.3.559.

Mogg, K., & Bradley, B. P. (1998, Sep). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy, 36*(9), 809–848.

Muris, P., & Field, A. P. (2008). Distorted cognition and pathological anxiety in children and adolescents. *Cognition and Emotion, 22*(3), 395–421.

Norr, A. M., Gibby, B. A., & Schmidt, N. B. (2017). Is computerized psychoeducation sufficient to reduce anxiety sensitivity in an at-risk sample?: *A randomized trial. Journal of Affective Disorders, 212*, 48–55.

Pine, D. S. (2007). Research review: A neuroscience framework for pediatric anxiety disorders. *Journal of Child Psychology & Psychiatry, 48*(7), 631–648.

Rapee, R. M. (1997). Potential role of childrearing practices in the development of anxiety and depression. *Clinical Psychology Review, 17*(1), 47–67.

Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review, 11*(2), 141–153.

Rice, F., Harold, G. T., & Thapar, A. (2002). Assessing the effects of age, sex and shared environment on the genetic aetiology of depression in childhood and adolescence. *Journal of Child Psychology and Psychiatry, 43*(8), 1039–1051.

Rutter, M. (2002). The interplay of nature, nurture, and developmental influences: The challenge ahead for mental health. *Archives of General Psychiatry, 59*(11), 996–1000.

Schmidt, N. B., Lerew, D. R., & Jackson, R. J. (1997, Aug). The role of anxiety sensitivity in the pathogenesis of panic: Prospective evaluation of spontaneous panic attacks during acute stress. *Journal of Abnormal Psychology, 106*(3), 355–364.

Schmidt, N. B., Zvolensky, M. J., & Maner, J. K. (2006, Dec). Anxiety sensitivity: Prospective prediction of panic attacks and Axis I pathology. *Journal of Psychiatric Research, 40*(8), 691–699. https://doi.org/10.1016/j.jpsychires.2006.07.009.

Schmidt, N. B., Maner, J. K., & Zvolensky, M. J. (2007, May 30). Reactivity to challenge with carbon dioxide as a prospective predictor of panic attacks. *Psychiatry Research, 151*(1–2), 173–176. https://doi.org/10.1016/j.psychres.2006.11.006.

Schmidt, N. B., Norr, A. M., Allan, N. P., Raines, A. M., & Capron, D. W. (2017). A randomized clinical trial targeting anxiety sensitivity for patients with suicidal ideation. *Journal of Consulting and Clinical Psychology, 85*(6), 596.

Schupp, H. T., Cuthbert, B. N., Bradley, M. M., Cacioppo, J. T., Ito, T., & Lang, P. J. (2000). Affective picture processing: The late positive potential is modulated by motivational relevance. *Psychophysiology, 37*(2), 257–261.

Short, N. A., Fuller, K., Norr, A. M., & Schmidt, N. B. (2017). Acceptability of a brief computerized intervention targeting anxiety sensitivity. *Cognitive Behaviour Therapy, 46*(3), 250–264.

Strawn, J. R., Dominick, K. C., Patino, L. R., Doyle, C. D., Picard, L. S., & Phan, K. L. (2014). Neurobiology of pediatric anxiety disorders. *Current Behavioral Neuroscience Reports, 1*(3), 154–160.

Tottenham, N. (2012). Human amygdala development in the absence of species-expected caregiving. *Developmental Psychobiology, 54*(6), 598–611.

Vagnoli, L., Caprilli, S., & Messeri, A. (2010). Parental presence, clowns or sedative premedication to treat preoperative anxiety in children: What could be the most promising option? *Pediatric Anesthesia, 20*(10), 937–943.

Van Beijsterveldt, C. E. M., Verhulst, F. C., Molenaar, P. C. M., & Boomsma, D. I. (2004). The genetic basis of problem behavior in 5-year-old Dutch twin pairs. *Behavior Genetics, 34*(3), 229–242.

Van Hoorn, J., McCormick, E. M., Rogers, C. R., Ivory, S. L., & Telzer, E. H. (2018). Differential effects of parent and peer presence on neural correlates of risk taking in adolescence. *Social Cognitive and Affective Neuroscience, 13*(9), 945–955.

Voegler, R., Peterburs, J., Lemke, H., Ocklenburg, S., Liepelt, R., & Straube, T. (2018). Electrophysiological correlates of performance monitoring under social observation in patients with social anxiety disorder and healthy controls. *Biological Psychology, 132*, 71–80.

Watt, M. C., & Stewart, S. H. (2000, Aug). Anxiety sensitivity mediates the relationships between childhood learning experiences and elevated hypochondriacal concerns in young adulthood. *Journal of Psychosomatic Research, 49*(2), 107–118.

Watt, M. C., Stewart, S. H., & Cox, B. J. (1998, May). A retrospective study of the learning history origins of anxiety sensitivity. *Behaviour Research and Therapy, 36*(5), 505–525.

Weinberg, A., & Hajcak, G. (2011, Oct). The late positive potential predicts subsequent interference with target processing. *Journal of Cognitive Neuroscience, 23*(10), 2994–3007. https://doi.org/10.1162/jocn.2011.21630.

Whaley, S. E., Pinto, A., & Sigman, M. (1999). Characterizing interactions between anxious mothers and their children. *Journal of Consulting and Clinical Psychology, 67*(6), 826–836.

Wissemann, K., Gorday, J. Y., & Meyer, A. (2018). The parent sensitivity to child anxiety index. *Child Psychiatry & Human Development*, 1–8.

Wood, J. J. (2006). Parental intrusiveness and children's separation anxiety in a clinical sample. *Child Psychiatry and Human Development, 37*(1), 73–87.

Wood, J. J., McLeod, B. D., Sigman, M., Hwang, W. C., & Chu, B. C. (2003). Parenting and childhood anxiety: Theory, empirical findings, and future directions. *Journal of Child Psychology and Psychiatry, 44*(1), 134–151.

Yonkers, K. A., Bruce, S. E., Dyck, I. R., & Keller, M. B. (2003). Chronicity, relapse, and illness–course of panic disorder, social phobia, and generalized anxiety disorder: Findings in men and women from 8 years of follow-up. *Depression and Anxiety, 17*(3), 173–179. https://doi.org/10.1002/da.10106.