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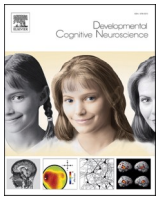

Relational victimization prospectively predicts increases in error-related brain activity and social anxiety in children and adolescents across two years

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ABSTRACT

Recent research has focused on identifying neural markers associated with risk for anxiety, including the error-related negativity (ERN). An elevated ERN amplitude has been observed in anxious individuals from middle childhood onward and has been shown to predict risk for future increases in anxiety development. The ERN is sensitive to environmental influences during development, including interpersonal stressors. Of note, one particular type of interpersonal stressor, relational victimization, has been related to increases in anxiety in adolescents. We tested whether relational victimization predicts increases in the ERN and social anxiety symptoms across two years in a sample of 152 child and adolescent females (ages 8 – 15). Results indicated that children and adolescents' baseline ERN was positively related to the ERN two years later. Furthermore, greater relational victimization at baseline predicted greater increases in the ERN two years later, controlling for baseline ERN. Moreover, relational victimization at baseline predicted increases in social anxiety, and this relationship was mediated by increases in the ERN. These results suggest that relational victimization impacts the developmental trajectory of the neural response to errors and thereby impacts increases in social anxiety among children and adolescents.

Anxiety disorders are common worldwide (Baxter et al., 2013) and are the most prevalent and persistent form of mental illness among children and adolescents (Ghandour et al., 2019; Kessler et al., 2012; Polanczyk et al., 2015). Anxiety disorders often emerge in childhood and adolescence, with a median age of onset of 11 years (Kessler et al., 2005). Furthermore, anxiety disorders are highly recurrent (Essau et al., 2018; Spinhoven et al., 2016) and are characterized by high rates of relapse (Batelaan et al., 2017; Ginsburg et al., 2014; Hendriks et al., 2013). Thus, there is a pressing need to characterize developmental pathways of risk for anxiety in order to improve prevention and intervention techniques.

Recent research has focused on identifying neural markers associated with anxiety and risk for the development of anxiety disorders, including the error-related negativity (ERN) (Hajcak, 2012; Meyer, 2017, 2022; Moser et al., 2013; Weinberg et al., 2016). The ERN is an event-related potential that is elicited within 100 ms following mistakes on lab-based reaction-time tasks (Falkenstein et al., 1991; Gehring et al., 1993). The ERN is thought to reflect a generic error-monitoring response generated by the anterior cingulate cortex, a brain region that is involved in cognitive control as well as negative affect, threat, and pain processing (Shackman et al., 2011). Recent work has also posited that the ERN reflects individual differences in distress surrounding mistakes, or error sensitivity (Chong and Meyer, 2019; Weinberg et al., 2016).

The ERN is related to individual differences in worry, perfectionism, and anxiety, and numerous studies have reported a link between an elevated ERN amplitude and anxiety symptoms from middle childhood onward (Cavanagh and Shackman, 2015; Hajcak, 2012; Meyer, 2016; Moser et al., 2013; Weinberg et al., 2016). An elevated ERN has been observed in individuals with generalized anxiety disorder (Weinberg et al., 2012; Weinberg and Hajcak, 2011a; Xiao et al., 2011), social anxiety disorder (Barker et al., 2015; Endrass et al., 2014; Judah et al., 2016; Kujawa et al., 2016; Umemoto et al., 2021), and obsessive-compulsive disorder (Endrass et al., 2008; Gehring et al., 2000; Hajcak et al., 2008; Riesel, 2019). Furthermore, an increased ERN

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amplitude is associated with risk for the development of anxiety, as an elevated ERN has been shown to predict future increases in anxiety (Filippi et al., 2020; Lahat et al., 2014; McDermott et al., 2009; Meyer et al., 2017a,2017b, 2021; Meyer and Hajcak et al., 2015; Meyer et al., 2018). It should be noted, however, that some studies have not observed this pattern of results (Seow et al., 2020), and a meta-analysis demonstrated that the relationship between anxiety and an elevated ERN was significantly greater among women than men (Moser et al., 2016). Collectively, the bulk of prior work has shown that an enhanced ERN appears to be a transdiagnostic marker associated with increased anxiety symptoms, as well as risk for future development of anxiety disorders and increases in anxiety symptoms (Meyer, 2016, 2022).

The ERN also appears to be sensitive to environmental modulations which increase the perceived salience of errors. For instance, the ERN magnitude has been shown to be enhanced in response to laboratory task manipulations that increase the threat-value of committing an error, including emphasizing accuracy over speed (Gehring et al., 1993b), emphasizing that performance is being evaluated (Hajcak et al., 2005), and incentivizing correct responses with monetary rewards (Ganushchak and Schiller, 2008; Hajcak et al., 2005). Similarly, in child samples, the ERN was increased when task performance was evaluated by an observer (Kim et al., 2005) or when the task was completed in the presence of a controlling parent (i.e., authoritarian parenting characterized by high levels of control and low levels of warmth) (Meyer et al., 2019). Additionally, the ERN has been shown to be potentiated by task-based punishment (e.g., a loud tone following errors on reaction time tasks) in adults (Meyer and Gawlowska, 2017; Riesel et al., 2012, 2019) and children (Cole et al., 2022), and the punishment potentiation of the ERN was related to increased anxiety symptoms. Collectively, it appears that environmental modulations that enhance the salience of errors tend to result in increases in the ERN magnitude. As such, we believe the link between an enhanced ERN and anxiety may reflect heightened error sensitivity, in which one's own errors are experienced as highly aversive. Indeed, increased self-reported error sensitivity is related to a larger ERN, and error sensitivity has been shown to mediate the relationship between an enhanced ERN and anxiety symptoms (Chong and Meyer, 2019). Furthermore, a recent intervention *targeting* error sensitivity (i.e., a brief, computerized intervention consisting of a one-hour tutorial covering perfectionism, fear of the social consequences of making mistakes, and over-valuation of the negative consequences of errors) has successfully led to reductions in the ERN amplitude among young adults (Meyer et al., 2020). Taken together, environmental manipulations that modulate the perceived threat-value of committing an error appear to impact the ERN, thus underscoring how the ERN may reflect an individual's heightened error sensitivity.

In addition to laboratory task modulation of the ERN, the magnitude of the ERN is also sensitive to naturalistic environmental stressors. Indeed, overly critical, or punitive parenting is related to an elevated ERN in children (Brooker and Buss, 2014; Chong and Meyer, 2019; Meyer et al., 2019; Meyer et al., 2015) and adults when assessed retrospectively (Banica et al., 2019; Meyer and Wissemann, 2020). Additionally, environmental stressors such as adverse childhood experiences and natural disasters are related to an increased ERN amplitude and anxiety (Lackner et al., 2018; Meyer et al., 2017a, 2017b; Wu et al., 2021). Of note, interpersonal stressors are also related to an enhanced ERN and increased anxiety symptoms. In particular, the ERN has been shown to moderate the relationship between interpersonal stress and anxiety symptoms six months later in adults (Banica et al., 2020), such that among individuals with a larger ERN at baseline, increased instances of interpersonal stress over one year were related to increased anxiety symptoms. Moreover, we recently demonstrated that while higher frequency of stressful life events was related to an increased ERN, more frequent interpersonal stressors, in particular, uniquely predicted an enhanced ERN amplitude among children and adolescents (Mehra et al., 2022). Collectively, these results suggest that aversive (e.g., punitive, stressful) life experiences may potentiate the ERN by increasing the threat value of errors and may contribute to increased risk for anxiety.

Importantly, adolescence is a period of increased prevalence and persistence of anxiety disorders (Beesdo et al., 2007; Kessler et al., 2012). Furthermore, social anxiety, in particular, tends to emerge in early adolescence (Beesdo et al., 2007), with a median age of onset at 13 years (Kessler et al., 2005). In contrast to social anxiety, specific phobia and separation anxiety have a median age of onset at 7 years, and other anxiety disorders have later median ages of onset (19–31 years; Kessler et al., 2005). As such, it is critical to understand risk factors implicated in the development and maintenance of social anxiety in the transition from late childhood to adolescence.

In particular, stressful life events have been found to predict increases in anxiety and the development of internalizing disorders (Casline et al., 2021; Green et al., 2010). One specific type of interpersonal stressor, peer victimization, has been shown to predict increasing anxiety symptoms in adolescents (Adrian et al., 2019; Forbes et al., 2019; Spence et al., 2022; Stapinski et al., 2015). Peer victimization refers to experiencing repeated harmful and aversive aggression from peers, and includes overt victimization (e.g., physical aggression such as hitting or pushing) and relational victimization (e.g., the extent to which individuals experience peers purposefully attempting to harm their relationships with others through social exclusion or ridiculing; Storch et al., 2005a, 2005b; Casper and Card, 2017). While a recent meta-analysis demonstrated that overt and relational victimization are highly intercorrelated (mean $r = 0.72$; Casper and Card, 2017), relational victimization, in particular, has been shown to be related to increases in anxiety in adolescents (Hamilton et al., 2016; Siegel et al., 2009). Furthermore, among older female adolescents (age 15–17), relational victimization predicted social anxiety symptoms (Ranta et al., 2013). Additionally, age appears to moderate the association between relational victimization and internalizing symptoms in children and adolescents, such that the relationship between relational victimization and internalizing symptoms becomes stronger as children age (Casper and Card, 2017). Thus, adolescence is a developmental period of vulnerability to the effects of relational victimization and to the development of anxiety disorders (Casper and Card, 2017; Kessler et al., 2012).

Given that the ERN has been shown to be elevated in individuals with social anxiety (Barker et al., 2015; Endrass et al., 2014; Judah et al., 2016; Kujawa et al., 2016; Umemoto et al., 2021) and social anxiety tends to emerge in late childhood and early adolescence (Beesdo et al., 2007), we collected self-report and EEG data across two years from a sample of children and adolescents ages 8–15 to investigate potential factors which may predict the development of social anxiety longitudinally. While interpersonal stress has been shown to relate to a larger ERN in children and adolescents (Mehra et al., 2022), and to interact with the ERN to predict anxiety symptoms six months later in adults (Banica et al., 2020), the specific impact of relational victimization on the development of the ERN and social anxiety symptoms has yet to be examined in children and adolescents. Therefore, the current study aimed to examine how relational victimization relates to the developmental trajectory of the ERN and subsequent changes in social anxiety symptoms in a sample of 152 girls over two years. We predicted that increased relational victimization would relate to increases in social anxiety and the ERN over two years, and we conducted exploratory analyses to test whether increases in the ERN would mediate the relationship between baseline relational victimization and increases in social anxiety.

1. Method

1.1. Participants

The participant sample was drawn from an NIMH-funded, longitudinal study (R01 MH097767) investigating neural measures of risk for

depression in females from a community sample. Of note, previous studies have been published using this dataset (Chong et al., 2020; Gorday and Meyer, 2018; Mehra et al., 2022; Meyer and Carlton et al., 2018; Meyer et al., 2021), however, no previous studies focused on the relationships between the ERN, relational victimization, and social anxiety symptoms.

In the current study, of the 317 recruited participants, 253 participants had EEG data at baseline and 188 participants had EEG data at time two.[1] Results of Little's MCAR test indicate that the data are missing at random at each time point, χ^2 (DF =2) = 1.44, p = .49. In total, 164 participants had EEG data at both baseline and time two. However, 12 participants were excluded for having task accuracy below 65%[2] or having fewer than 6 errors (Olvet and Hajcak, 2009). The final sample was comprised of 152 females ages 8–15 years (Baseline M = 11.77, SD = 1.76 years; time two M = 13.81, SD = 1.80 years) with usable EEG data at both time points. The sample was 82.9% White, 4.3% Black, 6.7% Hispanic, and 4.3% Other,[3] with 1.8% of participants with missing data on race and ethnicity.

Participants were recruited via a commercial mailing list. Afterwards, a phone screening was conducted to ensure the following inclusion criteria were met: the participant must live with at least one biological parent, both parent and child must speak English, and there must be no significant history of developmental disabilities or medical disabilities in the child. Relevant to the current study, as part of a larger battery, participants completed self-report measures and a computer-based reaction time task while EEG was acquired at baseline, and then again two years later. Participants were compensated $20 per hour for their study participation. The study protocol was approved by the Institutional Review Board at Stony Brook University, and parental consent and child assent were obtained prior to study participation.

1.2. Self-report measures

Social Experiences Questionnaire. The Social Experiences Questionnaire (SEQ; Crick and Grotpeter, 1996) is a 15-item self-report measure consisting of three subscales, including the relational victimization subscale, overt victimization subscale, and prosocial behaviors subscale. Each subscale includes 5 items, all of which are rated on a 5-point Likert scale, from 1 (never) to 5 (all the time). The overt victimization subscale measured the frequency of other children attempting or threatening to physically harm them (e.g., by hitting, pushing, yelling). The prosocial behaviors subscale measured the frequency that individuals received peers' helping, supporting, or caring acts. The current study focused on the relational victimization subscale, which assessed the frequency that individuals experienced peers attempting to harm their interpersonal relationships (e.g., spreading rumors, excluding peers from social interactions). Among the present sample, reliability at baseline was acceptable to very good for the total score (α = 0.73) and the relational victimization subscale (α = 0.84). Additionally, reliability two years later was acceptable for the total score (α = 0.69) and the relational victimization subscale (α = 0.72).

Multidimensional Anxiety Scale for Children. The Multidimensional Anxiety Scale for Children (MASC; (March et al., 1997) is a 39-item anxiety self-report measure consisting of four subscales, including physical symptoms (12 items), social anxiety (9 items), separation anxiety (9 items), and harm avoidance (9 items). Each item on the MASC is rated on a 4-point Likert scale, from 0 (never true about me) to 3 (often true about me). The MASC has shown high test-retest reliability (March et al., 1997; March et al., 1999) and good internal reliability (Baldwin and Dadds, 2007; Wei et al., 2014). The present study focused on the social anxiety subscale, which assesses humiliation/rejection and performance fears (e.g., "I worry about other people laughing at me;" "I feel shy"). Reliability in the current sample for the total scale was very good at baseline (α = 0.88), and two years later (α = 0.90). Reliability for the social anxiety subscale was also very good at baseline (α = 0.86), and two years later (α = 0.88).

1.3. Tasks and materials

Participants completed a standard arrow version of the computer-based Flankers task (Eriksen and Eriksen, 1974) while EEG was recorded at baseline and again two years later. During each trial, participants were presented with five white arrowheads in the center of a screen with a black background. Arrowhead presentations were equiprobably either congruent ("<<<<<" or ">>>>>") or incongruent (">><>>" or "<<><<"). Participants were instructed to respond as quickly and accurately as possible by clicking either the left or right mouse button based on the direction of the central arrowhead. Stimuli were presented in random order, each for 200 ms followed by a randomly variable intertrial interval of 2300–2800 ms wherein a gray fixation cross was shown on the center of a black background. Participants first completed a 30-trial practice block, followed by the full task which consisted of 11 blocks of 30 trials, totaling 330 trials. After each block, participants were provided performance-based feedback. If performance accuracy was 75% or below, participants were prompted with "Please try and be more accurate." If performance accuracy was above 90%, participants were prompted with "Please try to respond faster." Finally, if performance accuracy was between 75% and 90%, participants were shown feedback that read "You're doing a great job." Of note, an adaptive version of the Flankers task was utilized at time two in order to shorten the task and reduce participant burden. In particular, the task was modified at time two to terminate after participants made 20 errors, such that not all participants completed 330 trials of the task if they exceeded 20 errors.[4] All other aspects of the task were unchanged.

1.4. Psychophysiological recording

At baseline and time two, continuous EEG was recorded using the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands) and an elastic cap with 34 electrode sites. Two additional electrodes were placed on both the right and left mastoids. Four facial electrodes collected eye movement and blink data with two electrodes 1 cm away from the outer edges of the left and right eyes to measure horizontal movements and two electrodes 1 cm away from the top and bottom of the right eye to measure vertical movements. The EEG signal was pre-amplified at each electrode and amplified with a gain of one by the BioSemi ActiveTwo system. The 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 cutoff of 204.8 Hz. Each active electrode was referenced online to a common mode sense (CMS) active electrode producing a monopolar (non-differential) channel.

Offline, data were preprocessed and analyzed using BrainVision Analyzer Version 2.1.2.327. Data were referenced to the average of the mastoid electrodes and band-pass filtered between 0.1 and 40 Hz. Ocular corrections were conducted per Gratton et al. (1983). Artifact detection and rejection were conducted through an automatic procedure using the following specified parameters: voltage steps greater than 50.0 μV between sample points, voltage differences of 175.0 μV within a trial,

[1] At baseline, 24.3% of participants were missing EEG data, and two years later, 43.7% of the original sample were missing EEG data.

[2] Among the total sample at baseline, 14 participants had accuracy below 65%. No participants at time 2 had accuracy below 65%.

[3] Within the "Other" category, participants predominantly described their race as White and Black, one participant described their race as White and Guyanese, and one participant described their race as Italian.

[4] Among participants with usable EEG data, at baseline, participants completed an average of 322.05 (SD = 23.27) trials, and at time 2 participants completed an average of 305.93 (SD = 57.48).

and voltage differences of less than 0.50 μV within 100 ms intervals were rejected from all channels in each trial.

EEG data were segmented for each trial from 500 ms before the response until 800 ms after the response, and baseline correction was performed on the interval from 500 ms to 300 ms prior to the response. Correct and incorrect responses were averaged separately in the 100 ms window centered around the negative peak for each individual to obtain the correct-related negativity (CRN) and the error-related negativity (ERN). Analyses focused on FCz, where visual inspection revealed error-related brain activity to be maximal, and in line with prior studies (e.g., Chong et al., 2020; Chong and Meyer, 2019; Cole et al., 2022; Meyer et al., 2018; Meyer and Gawlowska, 2017; Olvet and Hajcak, 2009). Among participants with usable EEG data, at baseline, the average for the CRN consisted of 239.36 (SD = 56.36) segments and the average for the ERN consisted of 47.27 (SD = 29.34) segments. At time two, the average for the CRN consisted of 252.12 (SD = 58.77) segments and the average for the ERN consisted of 36.91 (SD = 19.68) segments. Internal consistency (Spearman-Brown corrected split-half reliability) of the ERN was good at baseline, $r(228) = 0.77$, and at time two, $r(174) = 0.85$; internal consistency of the CRN was very good at baseline, $r(228) = 0.96$, and at time two, $r(174) = 0.96$. A regression-based difference score was computed for the residualized ERN by regressing average correct-related negativity activity onto the average error-related negativity activity and saving the unstandardized residual scores (Meyer et al., 2017a,2017b).

1.5. Data analysis

Statistical analyses were conducted using SPSS Version 27. Paired samples *t*-tests were conducted to assess accuracy, reaction time, and the presence of the ERN at baseline and time two, and to assess changes in the residualized ERN amplitude from baseline to time 2. The Pearson correlation coefficient (*r*) was used to examine associations between study variables. A multiple regression was conducted to test the hypothesis that baseline relational victimization would predict the residualized ERN magnitude at time two, controlling for baseline residualized ERN. Additional regressions were conducted as follow-ups to determine if the pattern of results was the same when a potential outlier on baseline relational victimization was excluded and while controlling for potential confounding variables, including child age at baseline, reaction time on error trials, and number of errors, respectively. Finally, mediation models testing whether increases in the residualized ERN mediated the relationship between relational victimization at baseline and increases in social anxiety two years later were conducted using SPSS Hayes macro PROCESS (Hayes, 2012) model 4 with 5000 bootstrap samples. For all analyses, any values where $p < .05$ were considered significant.

2. Results

2.1. Descriptive statistics

Study variables are described in Table 1.

Table 1
Descriptive Statistics of Self-report Measures.

	N	M	SD	Minimum	Maximum
Baseline Relational Victimization	298	8.31	3.66	5	25
Time 2 Relational Victimization	241	6.96	2.65	5	20
Baseline Social Anxiety	294	1.12	0.74	0	3
Time 2 Social Anxiety	239	1.03	0.72	0	3

Note. Relational victimization was measured as the sum of the relational victimization subscale items of the SEQ. Social anxiety was measured as the average of the social anxiety subscale items of the MASC.

2.2. Behavioral data

Participants committed significantly more correct responses (M = 266.95, SD = 42.54) than errors (M= 54.76, SD = 29.59) at baseline, $t(236) = 46.92$, $p < .001$; similarly, participants committed significantly more correct responses (M = 259.91, SD = 60.99) than errors (M= 43.27, SD = 27.85) at time two, $t(180) = 40.62$, $p < .001$. Reaction time was faster on error trials (M= 371.14 ms, SD = 76.67) compared to correct trials (M = 482.45 ms, SD = 109.65) at baseline, $t(236) = 24.95$, $p < .001$; similarly, reaction time was faster on error trials (M= 325.91 ms, SD = 57.14) compared to correct trials (M = 413.12 ms, SD = 74.48) at time two, $t(180) = 24.58$, $p < .001$.

Social anxiety was not related to accuracy at baseline or time two, *p*s > 0.34; however, social anxiety was related to reaction time on correct responses, $r(235) = -0.17$, $p = .01$, and errors, $r(235) = -0.14$, $p = .04$, at baseline, but not at time two, such that children higher in social anxiety were characterized by faster reaction times at baseline. At baseline, older age was related to higher numbers of correct responses, $r(232) = 0.37$, $p < .001$, and lower numbers of errors, $r(232) = -0.29$, $p < .001$. Similarly, at time two, older age was related to more correct responses, $r(180) = 0.17$, $p = .02$, and lower error rates, $r(179) = -0.23$, $p = .002$. Reaction time was negatively associated with age at baseline (correct RT: $r(232) = -0.49$, $p < .001$; error RT: $r(232) = -0.41$, $p < .001$), and at time two (correct RT: $r(180) = -0.38$, $p < .001$; error RT: $r(179) = -0.30$, $p < .001$), indicating that overall, older children responded more quickly than younger children.

2.3. Error-related brain activity

At baseline, the ERP response was more negative following commission of errors (M = −3.87 μV, SD = 2.75) than correct responses (M = −1.42 μV, SD = 2.03), $t(233) = -16.19$, $p < .001$). Similarly, at time two, the ERP response was more negative following errors (M = 0.33 μV, SD = 7.93) than correct responses (M = 5.0 μV, SD = 5.65), $t(173) = -10.38$, $p < .001$. To isolate error-related negativity, we calculated the residualized ERN (i.e., the unstandardized residual score calculated by regressing the CRN onto the ERN). Numerically, participants displayed a larger, more negative residualized ERN at time two (M = −0.37 μV, SD = 5.84) than at baseline (M = −0.18 μV, SD = 2.37), however this was not statistically significant, $t(152) = 0.44$, p = .66).[5] A two-way repeated measures ANOVA revealed that when entering the raw ERN and CRN waveforms at baseline and time two, there was a significant interaction between trial type (error or correct) and time (baseline or time two), $F(1152) = 26.21$, $p < .001$, $\eta_p^2 = .15$. To probe the interaction, post hoc analyses revealed a mean difference between baseline and time two ERN of − 4.18, $p < .001$, as well as a mean difference between baseline and time two CRN of − 6.41, p < .001. Fig. 1.

We conducted correlations among the residualized ERN, child age, social anxiety symptoms, and relational victimization (Table 2). The residualized ERN at baseline was positively related to participants' residualized ERN two years later. At baseline, the residualized ERN was negatively related to child age, indicating that older age was related to a larger ERN. However, the residualized ERN two years later was not related to age. The residualized ERN was negatively related to social anxiety symptoms at baseline, but not at time two. Of note, relational victimization at baseline was negatively related to the residualized ERN at time two, indicating that higher frequency of relational victimization

[5] When using a subtraction based difference score, participants displayed a larger, more negative ΔERN at time two (M = −4.75 μV, SD =5.80) than at baseline (M = −2.59 μV, SD = 2.41), $t(151) = 4.99$, $p < .001$ (Fig. 1). The baseline subtraction-based ERN was positively related to the baseline regression-based ERN, $r(234) = 0.98$, $p < .001$, and the time two subtraction-based ERN was positively related to the time two regression-based ERN, $r(173) = 0.997$, $p < .001$.

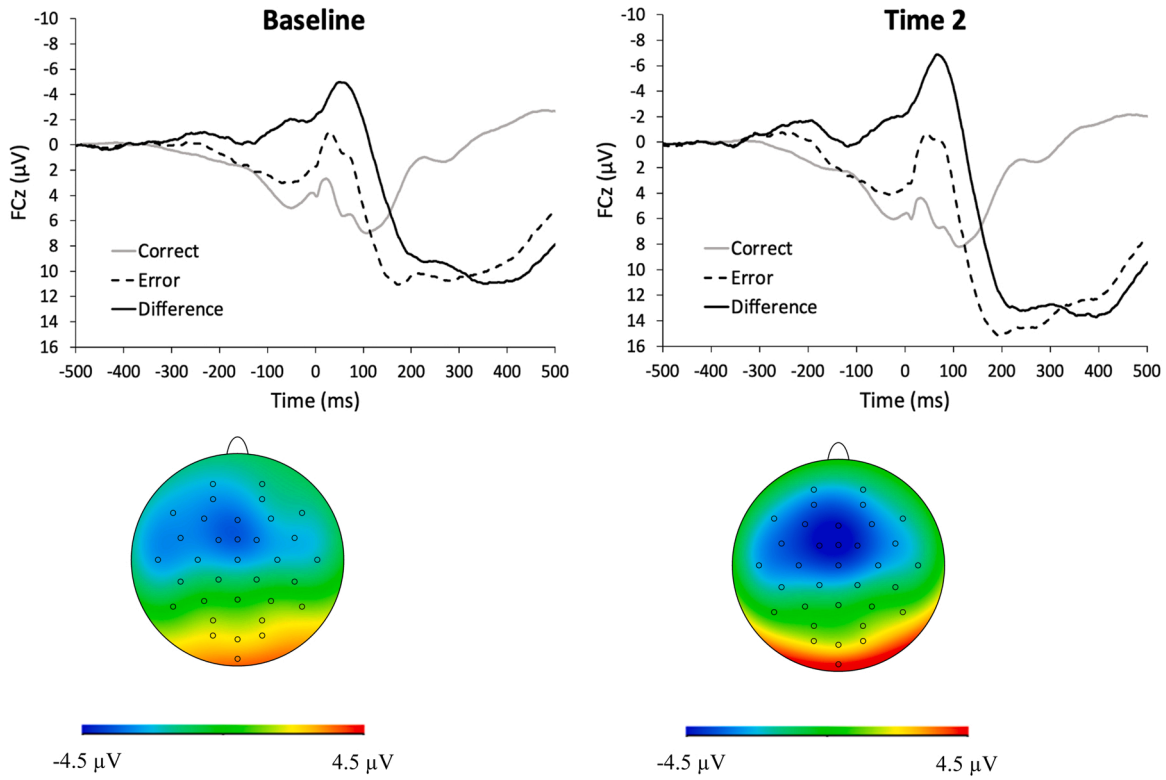

Fig. 1. Error (dotted line), correct (grey line), and difference (ERN minus CRN; black line) waveforms at baseline and two years later. *Note.* Waveforms are plotted with negative values up. Topographic maps depict the ΔERN 100 ms around the peak of the difference wave at baseline and at time two, respectively, at electrode site FCz.

Table 2
Correlations Among residualized ERN, Relational Victimization, Social Anxiety, and Age.

	1	2	3	4	5	6	7
1. Baseline ERN_{resid}	_						
2. Time 2 ERN_{resid}	0.44 * *	_					
3. Baseline Relational Victimization	0.07	-0.16 *	_				
4. Baseline Social Anxiety	-0.15 *	-0.14	0.34 **	_			
5. Time 2 Social Anxiety	-0.17 *	-0.02	0.13†	0.55 **	_		
6. Baseline Age	-0.37 **	-0.09	-0.03	0.31 **	0.30 **	_	
7. Time 2 Age	-0.40 **	-0.07	0.01	0.33 **	0.28 **	0.96 **	_

Note. ** $p < .01$. * $p < .05$, † $p < .06$

relates to a larger residualized ERN two years later.

To further examine the relationship between peer victimization and the residualized ERN two years later, we conducted a regression to examine whether relational victimization at baseline predicted a larger residualized ERN at time two, controlling for baseline residualized ERN (Fig. 2). Results indicated that the overall model was significant, $F(2, 150) = 19.01$, $p < .001$, $R^2 = .20$ (Table 3). The baseline residualized ERN significantly predicted the residualized ERN at time two. Moreover, controlling for the residualized ERN at baseline, relational victimization at baseline significantly predicted the residualized ERN two years later. Importantly, these results remained significant when controlling for reaction time on error trials, and number of errors at baseline, respectively (Supplemental tables 1–4).

We also conducted the regression model in SPSS AMOS using a full information maximum likelihood approach, and the results were consistent with those of the complete cases approach. Baseline residualized ERN significantly predicted residualized ERN at Time 2, $B = 1.06$, $\beta = 0.38$, $p < .001$, and, controlling for the residualized ERN at baseline, relational victimization at baseline significantly predicted the residualized ERN two years later, $B = -0.28$, $\beta = -0.17$, $p = .01$.

To assess the potential impact of an outlier on the relational victimization measure (i.e., with a score on the relational victimization subscale that was more than four standard deviations from the mean), we re-ran the regression excluding this participant. Removal of the outlier on relational victimization did not impact the results (Supplemental Table 5). The overall model remained significant, $F(2, 149) = 19.03$, $p < .001$, $R^2 = .20$.

Additionally, controlling for age did not impact the results (Supplemental Table 6). The overall model with baseline child age, residualized ERN, and relational victimization predicting the residualized ERN at time two was significant, $F(3, 148) = 13.13$, $p < .001$, $R^2 = .21$, and child age at baseline did not relate to residualized ERN at time two, $t = -0.87$, $B = 0.24$, $p = .39$. Controlling for baseline residualized ERN and child age, relational victimization at baseline predicted the residualized ERN two years later, $t = -2.05$, $B = -0.24$, $p = .04$. The results were unchanged when the outlier on relational victimization was excluded (Supplemental Table 7).

Finally, we conducted a mediation analysis to investigate whether increases in the residualized ERN mediated the relationship between relational victimization at baseline and increases in social anxiety two years later (Fig. 3). Increases in social anxiety was computed by subtracting baseline social anxiety from time two social anxiety symptoms,

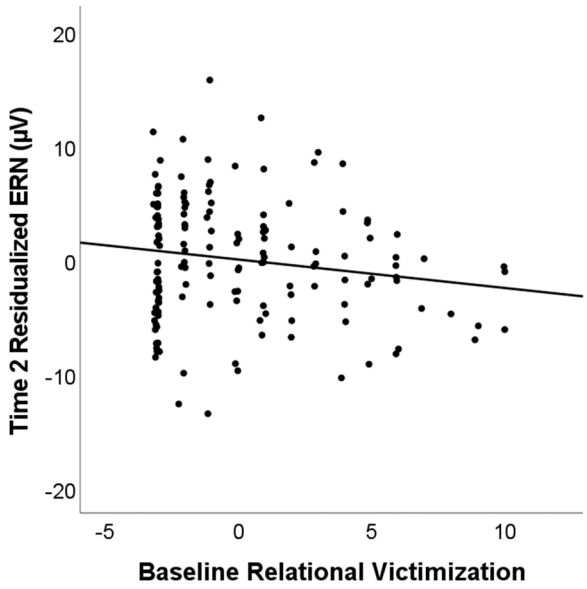

Fig. 2. Partial regression scatterplot depicting the relationship between baseline relational victimization scores (from the Social Experiences Questionnaire) and time two residualized ERN, controlling for baseline residualized ERN. *Note.* An outlier on relational victimization (> 4 SD from the mean) was excluded from the partial regression scatterplot.

Table 3
Regression Model with Baseline Residualized ERN and Relational Victimization Predicting Time 2 Residualized ERN.

Predictor	B	SE B	β	*t*
Baseline ERN_{resid}	1.05	0.18	0.43	5.84 **
Baseline Relational Victimization	-0.23	0.12	-0.15	-2.01 *

Note. * * $p < .01$. * $p < .05$.

and similarly, increases in the residualized ERN was computed by subtracting baseline residualized ERN from time two residualized ERN. The model was marginally significant in predicting increases in the residualized ERN, $R^2 = .02$, $F = 3.20$, $p = .08$, such that relational victimization was related to increases in the residualized ERN at a trend level, $t = -1.80$, $B = -0.21$, SE $= 0.12$, $p = .08$. Furthermore, the model significantly predicted increases in social anxiety, $R^2 = .10$, $F = 8.56$, $p < .001$. There was a significant direct effect of relational victimization on increases in social anxiety, $t = -3.04$, $B = -0.04$, SE $= 0.01$, $p < .01$. Importantly, there was a significant indirect effect of increases in the residualized ERN on changes in social anxiety symptoms, $B = -0.01$, SE $= 0.003$, 95% confidence interval: -0.01 to -0.0002. (Fig. 3).[6]

Importantly, when conducting the mediation model using a full information maximum likelihood approach, the results were consistent with the complete cases approach. Relational victimization predicted increases in residualized ERN, $B = -0.24$, SE $= 0.12$, $p = .04$. Increases in the residualized ERN trended towards predicting increases in social anxiety, $B = 0.02$, SE $= 0.01$, $p = .05$. There was a significant direct effect of relational victimization on increases in social anxiety, $B = -0.04$, SE $= 0.01$, $p < .001$. The indirect effect of increases in the residualized ERN on changes in social anxiety symptoms was $B = -0.004$.

3. Discussion

The current study examined how relational victimization (i.e., the frequency that individuals experience peers attempting to harm their interpersonal relationships) relates to the developmental trajectory of the ERN and subsequent changes in social anxiety symptoms in a sample of children and adolescents ages 8–15 years old across two years. In line with our hypothesis, results suggest that relational victimization predicts increases in the residualized ERN amplitude over two years in children and adolescents. Furthermore, exploratory analyses indicated that relational victimization predicted increases in social anxiety over two years, and this relationship was mediated by increases in the residualized ERN. These results are consistent with findings of Mehra et al. (2022), in which interpersonal stress was related to a larger ERN in children and adolescents. The current results extended these findings by demonstrating that relational victimization, a particular type of interpersonal stress which may be especially relevant to the development of anxiety in adolescence, appears to impact the development of the ERN and social anxiety symptoms in youth.

We examined developmental changes in the residualized ERN over two years among children and adolescents, given prior findings on the developmental trajectory of ERN in youth showing that the ERN tends to increase with age (Tamnes et al., 2013). Indeed, cross-sectional work has demonstrated age-related increases in ERN magnitude from childhood throughout late adolescence (Davies et al., 2004; Ladouceur et al., 2007; Santesso and Segalowitz, 2008; Wiersema et al., 2007). In contrast to these findings, the current results demonstrated that children and

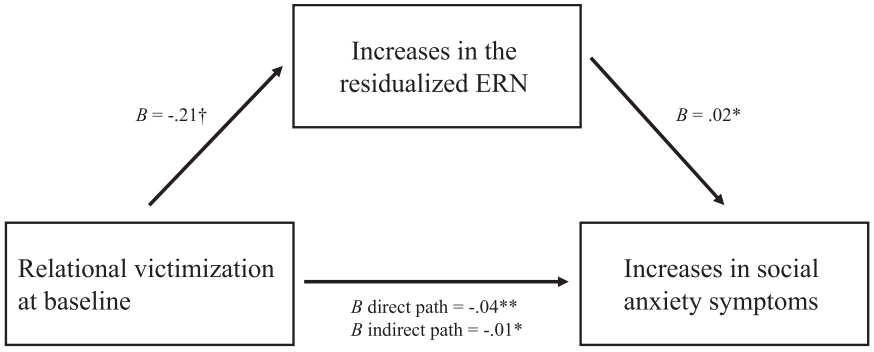

Fig. 3. Figure depicting a mediation model where the relationship between relational victimization at baseline and increases in social anxiety symptoms is mediated by increases in the residualized ERN. *Note.* * * $p < .01$. * $p < .05$, † $p < .08$.

[6] Including age as a covariate in the mediation model did not impact the results (see Supplement). Excluding the potential outlier in this model also did not impact the results.

adolescents' change in the residualized ERN magnitude across time was not significant. However, the raw scores (ERN and CRN) did appear to increase over time. It is possible that the use of residual scores may not reveal this pattern across time. Additionally, we found that children and adolescents' baseline residualized ERN was moderately positively correlated with their residualized ERN two years later, suggesting relative stability of the residualized ERN at the individual level over two years, in line with previous test-retest reliability among youth (Meyer et al., 2014) and adults (Weinberg and Hajcak, 2011b).

Furthermore, children and adolescents' baseline residualized ERN was negatively related to social anxiety symptoms at baseline and two years later, such that a larger (more negative) residualized ERN amplitude was associated with greater social anxiety symptoms. This association is consistent with prior work linking social anxiety symptoms and an elevated ERN in adults (Barker et al., 2015; Endrass et al., 2014; Judah et al., 2016; Umemoto et al., 2021) and adolescents (Filippi et al., 2020; Kujawa et al., 2016). The present findings are consistent with our prior work in the current sample at baseline demonstrating that the relationship between an elevated ERN amplitude and increased social anxiety was more robust than the relationship between the ERN and any other anxiety symptom scales, including panic, separation anxiety, generalized anxiety, and school avoidance (Meyer et al., 2018).

In order to understand environmental factors implicated in shaping the development of the ERN, we examined the relationship between baseline relational victimization and the residualized ERN magnitude two years later. Multiple regression results demonstrated that while controlling for the baseline residualized ERN, baseline relational victimization predicted an elevated residualized ERN amplitude two years later. In other words, increased frequency of experiencing relational victimization in late childhood and early adolescence appeared to impact the development of the ERN and contributed to increased residualized ERN amplitude two years later. This finding is in line with other work that has identified environmental factors implicated in shaping the ERN during development, including punitive parenting behaviors (Banica et al., 2019; Brooker and Buss, 2014; Chong and Meyer, 2019; Meyer et al., 2019; Meyer et al., 2015; Meyer and Wissemann, 2020) and life stressors (Lackner et al., 2018; Meyer et al., 2017a,2017b; Wu et al., 2021). While interpersonal stress has been shown to relate to a larger ERN in children and adolescents (Mehra et al., 2022) the current findings are consistent with and extend these results by suggesting that a specific form of interpersonal stress, relational victimization, is implicated in modulating the development of the residualized ERN.

Lastly, in order to understand how the effects of relational victimization on the residualized ERN relate to the development of social anxiety, we tested the relationship between relational victimization, prospective changes in the residualized ERN, and changes in social anxiety symptoms from baseline to two years later. Exploratory mediation analysis results indicated that baseline relational victimization predicted increases in social anxiety, and this relationship was mediated by increases in the residualized ERN. In other words, environmental stressors (i.e., relational victimization) appear to modulate the ERN resulting in increased ERN amplitude over two years, and this resulting change in the ERN predicts increases in social anxiety symptoms in children and adolescents. The current results indicate that experiencing greater relational victimization may impact the development of the ERN and thereby contribute to increased risk for social anxiety. It is possible that increased frequency of experiencing relational victimization may increase children and adolescents' sensitivity to threats, including internally generated threats (e.g., their own mistakes; Meyer, 2017; Weinberg et al., 2016). Relatedly, repeated experiences of relational victimization may make children and adolescents more self-conscious and engage in more self-monitoring of their behaviors and performance. Such elevated performance monitoring may result in greater salience and threat-value of errors, and thereby increase risk for anxiety, as elevated error sensitivity has been shown to predict risk for increases in anxiety (Meyer, 2016; Meyer et al., 2018; Meyer et al., 2018). Taken together, our results suggest that increases in threat and error sensitivity following relational victimization may underlie one pathway to social anxiety in the transition from late childhood to adolescence.

Importantly, the current results are in line with findings in which stressful life events are commonly related to the development of anxiety and other internalizing disorders during adolescence (Casline et al., 2021; Green et al., 2010). Moreover, peer victimization (including overt and relational victimization) has been shown to predict increases in anxiety symptoms in adolescents (Adrian et al., 2019; Forbes et al., 2019; Spence et al., 2022; Stapinski et al., 2015). The current results also support recent findings in which relational victimization specifically has been shown to be related to increases in anxiety in adolescents (Hamilton et al., 2016; Siegel et al., 2009). Because not everyone who experiences relational victimization in childhood or adolescence will develop anxiety, it is useful to examine the role of error sensitivity (as indexed by the ERN) in order to index individual differences in this potential pathway of risk for the development of anxiety disorders.

The current study has several strengths, including its longitudinal design and novel extension of prior work (Mehra et al., 2022) by investigating the impact of a particular form of interpersonal stress, relational victimization, on the development of the ERN and social anxiety in a large sample of children and adolescents. However, the current study also has several limitations. The participant sample included only females, thus limiting the ability to generalize results on the impact of relational victimization on the development of the ERN and anxiety among males and precluding the examination of sex and gender differences in these results. However, the inclusion of only females may be appropriate for the study of the development of anxiety, as rates of anxiety are higher in female adolescents compared to males (Merikangas et al., 2010; Ranta et al., 2007). Although a recent meta-analysis found no gender differences in the rates of experiencing relational victimization among adolescents (Casper and Card, 2017), it will be important for future work to include participants of all genders. Additionally, a limitation of this work is that the mediator (change in the residualized ERN) and the outcome measure (change in social anxiety symptoms) reflect concurrent assessments. When testing the opposite model (whether changes in social anxiety symptoms mediates the relationship between baseline relational victimization and increases in the residualized ERN), there is a significant indirect effect of changes in social anxiety on increases in the residualized ERN. Lastly, it is likely that there may be multiple potential factors (e.g., parenting style, attention biases, parents' anxiety, socioeconomic status, genetics) that might impact the development of the ERN and anxiety that were not assessed in the current study.

Overall, the present results indicate that relational victimization predicts increases in the residualized ERN amplitude two years later in children and adolescents ages 8–15 years old. Furthermore, relational victimization predicted increases in social anxiety over two years, and this relationship was mediated by increases in the residualized ERN. These results extend recent findings in which interpersonal stress has been shown to relate to a larger ERN in children and adolescents (Mehra et al., 2022), suggesting that relational victimization modulates the development of the ERN and thereby predicts increased risk for social anxiety symptoms. Furthermore, the current results demonstrate a separate process by which relational victimization relates to both an elevated residualized ERN and anxiety symptoms, as compared to recent findings in which the ERN moderated the impact of interpersonal stress on anxiety symptoms six months later in adults (Banica et al., 2020). In sum, the current results suggest that increases in error sensitivity (i.e., elevated ERN) following relational victimization may underlie one pathway to increases in social anxiety in children and adolescents. As anxiety disorders are the most prevalent and persistent form of mental illness among children and adolescents (Ghandour et al., 2019; Kessler et al., 2012; Polanczyk et al., 2015), it is important that future work continue to longitudinally assess developmental pathways of risk for anxiety, including potential bidirectional relationships among the ERN,

anxiety symptoms, and environmental stressors such as relational victimization, in order to improve early identification and intervention strategies.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

Data will be made available on request.

Acknowledgments

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2023.101252.

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