EMOTION REGULATION MODERATES THE PROSPECTIVE ASSOCIATION BETWEEN ERN AND ANXIETY IN EARLY ADOLESCENCE: AN AGE-SPECIFIC MODERATION OF COGNITIVE REAPPRAISAL BUT NOT EXPRESSIVE SUPPRESSION

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The Supervisory Committee certifies that this ***disquisition*** complies with North Dakota State University's regulations and meets the accepted standards for the degree of

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SUPERVISORY COMMITTEE:

Pan Liu
Chair

Jeremy Hamm

Jeffrey Johnson

Carmen Kho

Approved:

18 March 2024
Date

Clayton Hilmert
Department Chair

ABSTRACT

The increasing prevalence of anxiety problems during adolescence underscores the importance of a better understanding of the development of anxiety. While past research has highlighted a link between error responsivity, indexed by error-related negativity (ERN), and youth anxiety, the role of emotion regulation in the ERN-anxiety relationship remains unclear. We conducted a two-wave study with 115 healthy nine-12-year-olds, and found that expressive suppression (ES), an important emotion regulatory strategy, moderates the ERN-anxiety association. A larger T1 ERN predicted heightened T2 anxiety symptoms in those with higher ES. Interestingly, the moderating effect of cognitive reappraisal (CR), another important emotion regulatory strategy, on the ERN-anxiety association was age-dependent; in older youths, the association between T1 ERN and T2 symptoms was significant only for those with lower CR. These findings offer novel insights into the differential age-related patterns in the moderating effects of emotion regulation, and inform future emotion intervention studies for youths.

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LIST OF ABBREVIATIONS

CI.....Confidence interval

CR .....Cognitive reappraisal

Cz .....Vertex electrode

e.g.....For example

ERN.....Error-related negativity

EEG.....Electroencephalogram

ERP .....Event-related potentials

ES.....Expressive suppression

FCz.....Fronto-central electrode.

Hz.....Hertz

i.e.....That is

kΩ.....Kiloohm

ms.....Milliseconds

μV.....Microvolts

n.s. .....Non-significant

PCA.....Principal component analysis

SD .....Standard deviation

SE.....Standard error

T1 .....Time 1

T2 .....Time 2

LIST OF SYMBOLS

*.....Asterisk

ß.....Beta

χ.....Chi

$.....Dollar

μ .....Mu

Ω.....Omega

% .....Percent

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1. INTRODUCTION

The number of youths suffering from anxiety has been steadily increasing over the years (Paulus et al., 2015; Parodi et al., 2022). Without effective prevention or intervention, these early problems may persist and predict a host of long-term, detrimental health consequences, including mood and anxiety disorders (Copeland et al., 2009). It is therefore critical to understand the developmental mechanisms of anxiety in youths. In recent decades, there has been considerable interest in the study of error responsivity and its role in the development of anxiety (Moser et al., 2013). Error responsivity refers to the detection and monitoring of errors aimed at optimizing behavioral performance across tasks and situations (Olvet & Hajcak, 2008). Numerous studies have leveraged event-related potentials (ERP) to examine the neurophysiological markers of error responsivity in relation to anxiety (Meyer, 2016; Meyer, Hajcak, et al., 2017). One such neural marker is the error-related negativity (ERN), an early fronto-central negative deflection between 0 - 150 ms following an erroneous response (Gehring et al., 1993). The ERN is thought to reflect cognitive control processes that monitor task performance and maintain optimal adjustments following error responses. An enlarged ERN is found to be associated with heightened anxiety symptoms in 10-18 years old youths (McDermott et al., 2009; Meyer et al., 2012) and adults (Ladouceur et al., 2007; Weinberg et al., 2010).

However, not all individuals with maladaptive error responsivity (indicated by an enlarged ERN) experience elevated anxiety. Instead, the association between error responsivity and anxiety is likely to be moderated by other individual or environmental factors. One such factor that has been commonly implicated in psychopathology is emotion regulation (Sheppes et al., 2015). Emotion regulation refers to individuals' abilities to alter the significance of, or attention towards, emotional information (Gross & Thompson, 2007). Adaptive regulation

strategies (e.g., higher tendency to reappraise or lower tendency to suppress emotional responses) is associated with positive outcomes, while suboptimal regulatory capacities (e.g., lower tendency to reappraise or higher tendency to suppress emotional responses) is associated with adverse outcomes (Brewer et al., 2016; Dryman & Heimberg, 2018; Gross & John, 2003; Hu et al., 2014; Moore et al., 2008). Likewise, in the context of error processing, adaptive emotion regulatory strategies (e.g., reappraisal) has been found to reduce error-related distress (Dignath et al., 2019; Hobson et al., 2014; Levsen & Bartholow, 2018; Wang & Yang, 2014). However, it remains unclear how error responsivity and emotion regulation jointly contribute to the development of anxiety. To address this issue, we examined the interaction between error responsivity, as indexed by the ERN, and two facets of emotion regulation (i.e., cognitive reappraisal and expressive suppression; Gross & John, 2003) in predicting prospective anxiety symptoms in early adolescence.

The ERN is considered a neurobehavioral trait that is heritable and stable over time and may therefore be less susceptible to modification (Hajcak, 2012; Weinberg et al., 2012). In contrast, emotion regulation is a modifiable attribute that may serve as the target of prevention and intervention protocols for youths at risk. Existing prevention and intervention studies have found that training youths in using more adaptive emotion regulation strategies lead to better psychological adjustment and well-being (see reviews Eadeh et al., 2021; van Agteren et al., 2021). Therefore, understanding the extent to which different emotion regulatory strategies may strengthen or weaken the associations between the ERN and anxiety symptoms will help inform the development of prevention/intervention tools for youths at risk for anxiety (e.g., those with maladaptive patterns of error responsivity).

1.1. Associations between the ERN and anxiety

Our ability to detect and respond to errors is an executive function that is critical for behavioral control (Gehring et al., 1993). It serves to mobilize increased attentional resources towards action-outcome contingencies to compensate for errors (Ullsperger & von Cramon, 2001). One neural index of error processing is the ERN, a fronto-central negative deflection between 0-150 ms following an erroneous response (Gehring et al., 1993). Source localization studies have identified the origination of ERN in the anterior cingulate cortex (Fitzgerald et al., 2005; Mathalon et al., 2003; van Veen & Carter, 2002), the prefrontal cortex (McClure et al., 2007), and supplementary motor cortex (Iannaccone et al., 2015). The ERN reflects cognitive control processes that aim to monitor performance and maintain optimal adjustments following mistakes. Further, the ERN may be developmentally sensitive and increases with age (Davies et al., 2004; Meyer et al., 2012; Tamnes et al., 2013), reflecting improved cognitive control (Troller-Renfree et al., 2016) and behavioral performance (McDermott et al., 2013) across development.

A considerable amount of research has supported the ERN-anxiety association (Meyer, 2016; Meyer, Hajcak, et al., 2017; Moser et al., 2013). In adults, an enlarged ERN has been associated with the onset of generalized anxiety disorder, obsessive compulsive disorder, and social anxiety disorder (Riesel, 2019; Weinberg et al., 2010, 2015). Similarly, in youths, an enlarged ERN was cross-sectionally or longitudinally associated with anxiety disorders (Carrasco et al., 2013; Ladouceur et al., 2006; Olvet & Hajcak, 2008; Weinberg et al., 2012) and heightened subclinical anxiety symptoms (Filippi et al., 2020; Hajcak et al., 2003; Meyer et al., 2012; Moser et al., 2012; Santesso et al., 2006; Weinberg et al., 2016). For example, six-year-old typically developing children with an enhanced ERN showed greater anxiety symptoms at age

nine (Meyer et al., 2015). Adolescent girls who demonstrated an enhanced ERN at baseline were more likely to be diagnosed with generalized anxiety disorder 18 months later (Meyer et al., 2018).

The ERN may be differentially associated with anxiety in younger children (Lawler et al., 2021; Moser, 2017), although findings remain inconsistent (Meyer et al., 2013). While an enlarged ERN is mostly found to be associated with greater anxiety symptoms in older youths of 10-18 years old (Filippi et al., 2020; McDermott et al., 2009; Meyer et al., 2018) and adults (Kujawa et al., 2016; Weinberg et al., 2010), some studies have reported a smaller ERN in association with greater temperamental fear or heightened anxiety symptoms in children of ages five to eight (Lo et al., 2016; Torpey et al., 2013). Other studies have found an interaction between the ERN and child age in predicting anxiety symptoms (Ip et al., 2019; Meyer et al., 2012). Among eight-13-year-olds, a larger ERN was associated with increased parental reports of child anxiety only in older children (Meyer et al., 2012). Researchers propose that this reversed pattern in younger children may be related to a developmental shift in the processing of anxiety-related content, such that younger children tend to show greater sensitivity towards external fear stimuli, while older children tend to be more responsive towards internal fear stimuli, such as making an error on a task (Meyer, Hajcak, et al., 2017; Weinberg et al., 2016). Based on these studies, it is more likely that during late childhood and early adolescence, a larger, rather than a smaller, ERN is associated with heightened anxiety symptoms.

1.2. Associations between emotion regulation and anxiety

The process model of emotion regulation elucidates four distinct phases of emotion generation, including (1) the detection of an internal or external stimulus, (2) the allocation of attention, (3) an appraisal of the stimulus, and (4) the response following the emotional

experience (Gross & Thompson, 2007). Individuals employ different emotion regulatory strategies during the process of emotion generation (Gross, 2014). Antecedent-focused strategies often occur before or during the first three phases of the emotion process. A widely studied antecedent-focused strategy is cognitive reappraisal (CR), the ability to redefine the emotional meaning and relevance of an event or stimulus (Gross & John, 2003; Webb et al., 2012). In contrast, response-focused strategies occur later during the response phase of the emotion process. One such strategy is expressive suppression (ES), the tendency to inhibit behavioral responses towards an emotional event or stimulus (Aldao et al., 2010; Gross & John, 2003).

Antecedent-focused strategies such as CR, compared to response-focused strategies like ES, are more effective in reducing the intensity and duration of emotional responses (Brewer et al., 2016; Dryman & Heimberg, 2018). Supporting this notion, CR has been linked to higher self-esteem (Gross & John, 2003; Nezlek & Kuppens, 2008), optimism and life satisfaction (Gross & John, 2003; Haga et al., 2009; Hu et al., 2014), and lower levels of anxiety (O'Connor et al., 2014; Werner et al., 2011) and depression (Haga et al., 2009; Moore et al., 2008) in youths and adults. For instance, greater engagement in positive reappraisal was associated with lower anxiety in nine-12-year-old children (Chan et al., 2016). ES, on the other hand, is mostly associated with negative outcomes, such as lower self-esteem (Gross & John, 2003; Nezlek & Kuppens, 2008), lower life satisfaction (Gross & John, 2003; Haga et al., 2009; Hu et al., 2014), and elevated social anxiety (Kashdan & Breen, 2008; O'Connor et al., 2014; Werner et al., 2011) and depression symptoms (Haga et al., 2009; Moore et al., 2008) in youths and adults.

Moreover, the effect of reappraisal strategies in youths may increase with age (McRae et al., 2012; Silvers et al., 2012). Compared to 10-13-year-olds, 14-17-year-old adolescents reported greater reduction in negative affect ratings following cognitive reappraisal (McRae et

al., 2012). A similar association between reappraisal success and older age was reported in healthy individuals of ages 10-23 (Silvers et al., 2012). Researchers attributed this to the development of improved effortful control capacities, a core characteristic of CR, (McRae et al., 2012), as children grow into adolescence (Morales et al., 2016). Interestingly, Silvers and colleagues (2012) found that the relationship between successful reappraisal and age tapered off in late adolescence and early adulthood, suggesting that the abilities essential for successful reappraisal develop over childhood and adolescence and mature during early adulthood. These findings evidenced early adolescence as a prominent stage for the development of emotion regulatory strategies, especially CR.

1.3. Interactions between error responsivity and emotion regulation in predicting prospective anxiety

The development of psychopathology relies on the interplay between different risk factors (Cicchetti & Rogosch, 1996). Along this line, a number of studies have examined the relationship between ERN and anxiety in the context of other individual (Canen & Brooker, 2017; McDermott et al., 2009; Meyer, Danielson, et al., 2017) and environmental factors (Banica et al., 2020; Meyer, Danielson, et al., 2017). One longitudinal study reported an interaction between childhood behavioral inhibition (e.g., a temperamental trait characterized by hypervigilance and withdrawal in response to novelty) and adolescent ERN in predicting anxiety symptoms during adolescence. In 14-16-year-old youths, an enlarged ERN was associated with higher anxiety symptoms only in those with heightened childhood behavioral inhibition (McDermott et al., 2009). The ERN also interacted with interpersonal stress in predicting prospective anxiety symptoms: for adults with an enlarged ERN only, elevated interpersonal stress predicted heightened anxiety symptoms six months later (Banica et al., 2020). Another

study found a three-way interaction between early temperament, exposure to a natural disaster, and ERN (Meyer, Danielson, et al., 2017). Children who showed high temperamental fear at age three and experienced elevated stress during Hurricane Sandy demonstrated increased internalizing symptoms (i.e., anxiety and depression) at age nine, but only when they also showed a larger ERN at age six. However, no work has examined the potential interaction between ERN and emotion regulation in predicting prospective symptoms of anxiety. In other words, it is unclear to what extent emotion regulation moderates the ERN-anxiety association as children transition into adolescence.

1.4. The present study

The present study aimed to address this gap using two-wave longitudinal data collected from 115 youths (nine-12 years old at T1, 10-13 years old at T2). Specifically, we examined (1) the prospective association between the ERN at T1 and anxiety symptoms at T2 and (2) to what extent this relationship is moderated by youths' emotion regulation abilities at T2. Based on the literature, we hypothesized that (1) a larger ERN at T1 would predict greater anxiety symptoms at T2, with T1 anxiety symptoms and the unique variance of T2 ERN accounted for; (2) more adaptive emotion regulation strategies (e.g., antecedent-focused strategies such as CR) would weaken the association between T1 ERN and T2 anxiety symptoms, whereas suboptimal emotion regulatory strategies (e.g., response-focused strategies such as ES) would strengthen the ERN-anxiety relationship. Additionally, considering extant findings on the positive association between reappraisal success and age (McRae et al., 2012; Silvers et al., 2012), the association between the ERN, emotion regulation (especially CR), and anxiety might be further conditioned on age. Therefore, we also explored the three-way interaction between the ERN, emotion regulatory strategies, and age.

2. METHOD

2.1. Participants and procedure

The data presented in this study was part of an ongoing study investigating the neural correlates of cognitive risks for anxiety and depression in late childhood and early adolescence. At the beginning of the study (T1), a community sample of 115 nine-to-12-year-old early adolescents (66 girls; Mean age/SD = 11.00/1.16 years) were recruited from a Midwestern urban area through a participant registry of local families. None of the youths reported major medical conditions or neurodevelopmental disabilities. Sample demographics were relatively representative of the local community (87.5% White, 3.6% Asian, 8.9% More than one race; 7.2% Hispanic or Latino; family annual income: $15,000 – $350,000).

At T1, youths and their caregivers were invited to campus for a laboratory visit. Following caregiver consent and youth assent, youths completed a battery of EEG tasks (including a Go/No-Go task) and an eye-tracking task that tapped into different cognitive risk processes. The order of the EEG tasks was counterbalanced across participants. Only data from the Go/No-Go task were reported in this study. Following the lab visit, youths also completed an online questionnaire package at home reporting their mental health problems. A follow-up study (T2) was conducted approximately a year later (Mean/SD of the interval = 12.20/1.72 months), using the same study protocol at T1 with the addition of a youth self-report measure of emotion regulation. Of the initial 115 participants, 92 youths (53 girls; Mean age/SD = 12.06/1.20 years) returned for the T2 follow-up study. At both T1 and T2, participants received monetary compensation for their participation.

2.2. The EEG Go/No-Go Task

The current study adopted a youth-friendly version of the Go/No-Go task in combination with EEG recordings to elicit the ERN (Grammer et al., 2014; Ip et al., 2019). The Go/No-Go task consisted of 252 Go trials and 63 No-Go trials divided into 3 blocks (84 Go and 21 No-Go trials per block, presented in a random order). As shown in Figure 1, each trial started with a fixation cross present for 200 milliseconds (ms). Then, an image of a dog or a cat was presented for 750 ms, followed by a blank screen for 500 ms. Participants were instructed to press a button as quickly as possible when they saw a cat (Go) and not to press the button when they saw a dog (No-Go). All images were matched on size and color. The task was conducted using the E-Prime software (Psychology Software Tools Inc., Pittsburgh, PA). At beginning of the task, a brief practice session was conducted to familiarize participants with the procedure. In addition to the EEG, participants' behavioral responses (e.g., button presses and reaction time [RT]) were recorded during the Go/No-Go task.

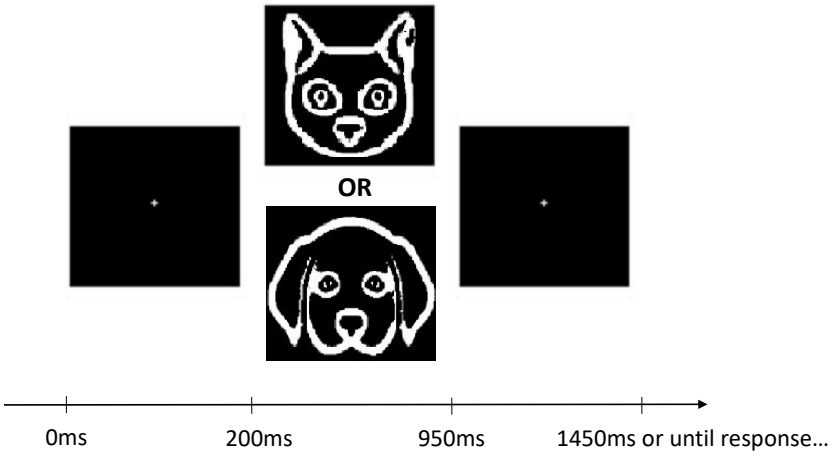

Figure 1. Trial procedure of the Go/No-Go task. Note: ms: milliseconds.

2.3. Questionnaires

Emotion Regulation Questionnaire. The Emotion Regulation Questionnaire (Gross & John, 2003) was used to measure youths' emotion regulation capacities. This scale measures two

facets of emotion regulation: cognitive reappraisal (CR, 6 items; e.g., *when I want to feel more positive emotions, I change what I'm thinking about*) and expressive suppression (ES, 4 items; e.g., *I keep my emotions to myself*). Youths rated the extent to which they agreed with each statement on a seven-point Likert scale (1 = strongly disagree, 7 = strongly agree). A total score of each subscale was calculated to indicate CR and ES, respectively. Both subscales demonstrated good internal consistency in the current sample (CR, Cronbach's α = 0.83; ES, Cronbach's α = 0.80).

Youth Self-Report Anxiety. The Youth Self-Report is a 118-item checklist that assesses behavioral and emotional symptoms in four-18-year-olds (Achenbach, 1991). The scale consists of a 16-item anxiety subscale (Kendall et al., 2007), which was used in the current study to assess youths' anxiety symptoms in the past month. Each item (e.g., *I worry a lot*) were rated on a three-point scale (0 = not true, 2 = very true). A total score was computed to indicate anxiety symptoms. The scale demonstrated good internal consistency in this study at both T1 (Cronbach's α = 0.87) and T2 (Cronbach's α = 0.88).

2.4. EEG data acquisition and processing

Youths completed the Go/No-Go task in an electrically shielded chamber, during which continuous EEG signals were recorded using a 64-channel HydroCel Geodesic Sensor Net (Electrical Geodesic Inc.) and an EGI 200 NetAmps Amplifier. The EEG signals were recorded with a sampling rate of 250 Hz, referenced to the vertex electrode (Cz). Electrode impedances were kept below 50 kΩ. The EEG data were processed using the EEGLab (Delorme & Makeig, 2004) and ERPLab (Lopez-Calderon & Luck, 2014) toolboxes operated in MATLAB version 9.10.0 (Mathworks, Inc., Natick, MA). The raw data were first filtered within the 0.1-40 Hz bandpass and re-referenced to the average of the two mastoid electrodes. We then removed the

ocular artifacts (i.e., eye blinks and eye movements) using an independent component analysis approach. Next, the data were time-locked to the response (i.e., button press) and segmented into desired epochs (200 ms pre-response to 200 ms following response). Further artifact correction was conducted to reject individual segments with (1) voltage beyond ±100 μV, (2) more than 50 μV change of voltage between time points, or (3) more than 300 μV change of voltage between the most positive and negative timepoints within a 200 ms window. Finally, for each individual, we averaged the ERP waveforms across trials for correct and error responses, respectively. Following artifact rejection, we retained ERP data of 91 (of 115) youths at T1 and 83 (of 92) youths at T2 with ≥10 trials in each condition for subsequent analyses.

2.5. Principal component analysis of the ERP data

Prior work in similar age groups has mostly quantified the ERN with arbitrarily defined time windows and selected electrodes (Grammer et al., 2014; Ip et al., 2019). This approach uses only a small portion of the collected data, yields less accurate temporo-spatial information, and cannot speak to the underlying ERP components which may not be readily perceptible by visual inspection (Luck, 2014). To avoid these limitations, we took a principal component analysis (PCA) approach to quantify the ERN component. The PCA is a factor-analytic approach that generates more accurate temporo-spatial information of ERP components by accounting for the variance across all time points and electrode sites.

A two-step temporal-spatial PCA was adopted using the ERP PCA Toolkit in MATLAB (Dien, 2010; Dien et al., 2005; Dien & Frishkoff, 2005). We first performed a temporal PCA with Promax rotation to reduce the temporal dimensions of the data (Dien & Frishkoff, 2005). All time points were treated as variables, while participants, experimental conditions, and electrodes were considered as observations. Linear combinations of variables (i.e., time points)

were generated as temporal factors for each observation. Following the temporal PCA, we ran a spatial PCA on each of the temporal factors using Infomax rotation to reduce the spatial dimensions of the data, where the electrodes were treated as variables and participants, conditions, and temporal factors as observations.

The PCA of T1 ERP data yielded 28 temporo-spatial factors (7 temporal factors × 4 spatial factors) with 83.1% of the total variance accounted for. Of the 28 factors, 21 factors accounted for a minimum of 0.50% of the variance and were retained for further inspection (Dien, 2012). As shown in Figure 2a, we identified the TF03SF1 factor (peaking channel FCz, peaking latency 136 ms, 5.36% variance) as temporally and spatially analogous to the ERN (Meyer, 2022). For our T2 data, the PCA generated 28 temporo-spatial factors (7 temporal factors × 4 spatial factors) with 83.8% of the total variance accounted for. Of the 28 factors, 19 accounted for a minimum of 0.50% of the variance. We identified a similar TF03SF1 factor (peaking channel FCz, peaking latency 136 ms, 6.34% variance) that resembled the ERN (Figure 2b). The factor scores of the two identified PCA factors were extracted for the error and correct conditions, respectively, as indicators of the amplitude of the ERN in each condition (ERN_{error} and $ERN_{correct}$) at T1 and T2. We quantified the ERN using a regression-based residual score by regressing the ERN_{error} on $ERN_{correct}$ at each time point. The regression-based method captures unique variance of one condition relative to the other and has been found to be more reliable and internally consistent compared to subtraction-based difference scores (Meyer, Lerner, et al., 2017).

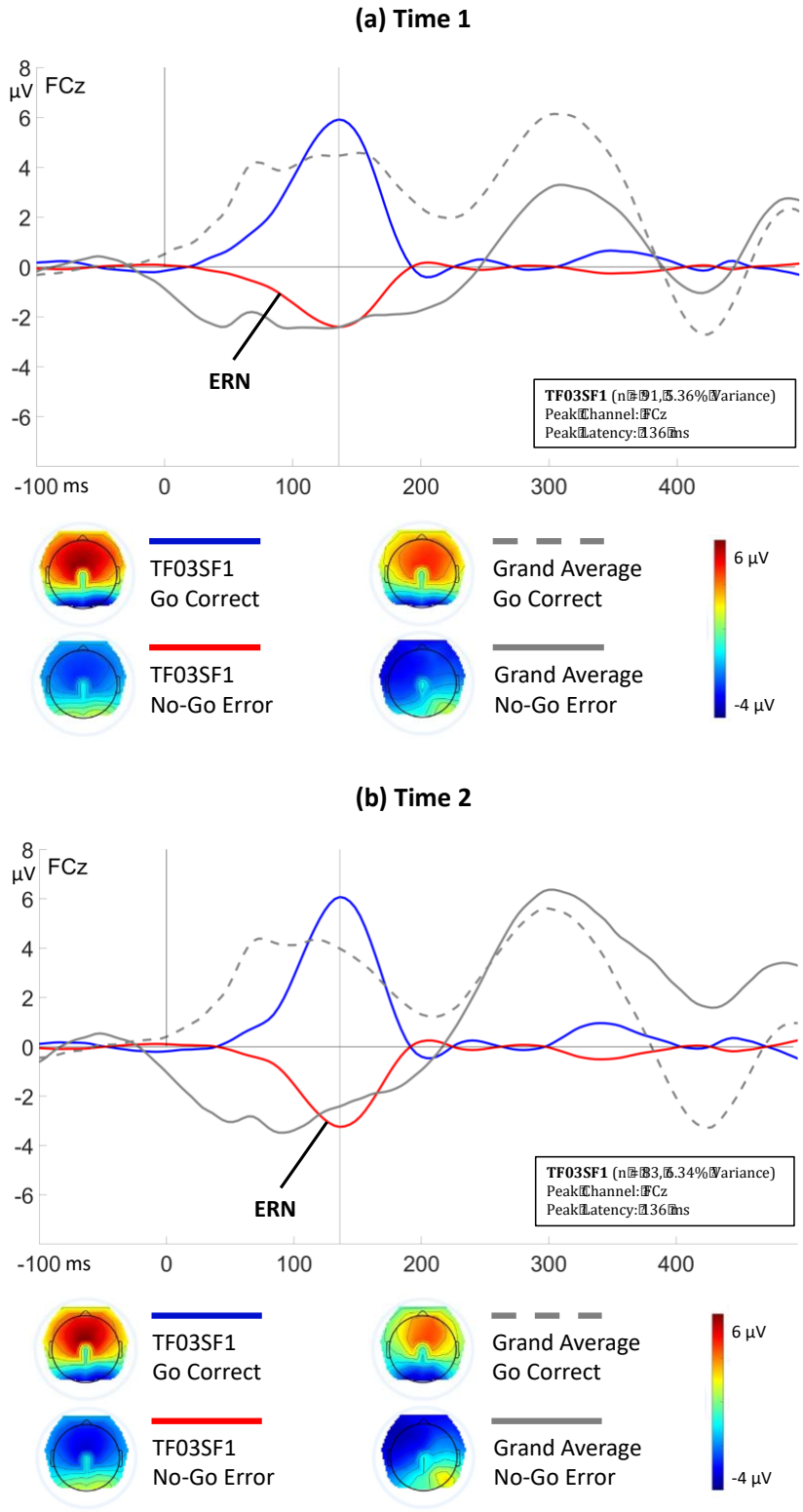

Figure 2. ERP waveforms and topographic maps (at peak latencies) of the ERN in the correct and error conditions superimposed on the grand average of the original data at (a) Time 1 and (b) Time 2. Note: ERN: error-related negativity; µV: microvolts; ms: milliseconds.

2.6. Statistical analysis

Of the 115 youths at T1, 29 had missing data for at least one study variable (e.g., six with missing anxiety scores, 24 with unusable ERP data). At T2, 17 out of the 92 youths had missing data for at least one study variable (e.g., six with missing anxiety scores, eight with missing emotion regulation scores, nine with unusable ERP data). Little's Missing Completely at Random Test (Little, 1988) via the R *naniar* package (Tierney & Cook, 2023) indicated that the data at T1 and T2 were missing completely at random ($\chi 2$ (109) = 108.42, $p > .10$). To further account for the missing data, we performed multiple imputation using R *mice* package (van Buuren & Groothuis-Oudshoorn, 2011). Fifty imputations with ten iterations per imputation were implemented for each variable; averaged data were then calculated across the 50 imputed datasets. For data missing completely at random, multiple imputation can provide relatively unbiased estimates with improved efficiency regardless of the proportion of missingness (Madley-Dowd et al., 2019). The imputed data (N = 115) were subjected to subsequent statistical analysis in R Studio (Version 2023.03).

We conducted two multiple regression models to test the interactions between T1 ERN and T2 emotion regulatory strategies (CR or ES) in predicting T2 anxiety symptoms. In each model with T2 anxiety symptoms as the outcome, we added youth's age, sex, and T1 anxiety symptoms as covariates. To better speak to the prospective association between T1 ERN and T2 symptoms and account for any cross-sectional association between T2 ERN and T2 symptoms, we also included the unique variance of T2 ERN (calculated by regressing T2 ERN on T1 ERN)

as another covariate.[1] Next, we added T1 ERN and T2 CR or ES scores as main predictors in our models. We then entered the two-way interaction terms between T1 ERN, T2 CR/ES, and age (i.e., ERN × CR/ES, ERN × Age, CR/ES × Age). Finally, considering previous findings on the association between reappraisal success and age (McRae et al., 2012; Silvers et al., 2012), as well as the interaction between the ERN and age in predicting anxiety (Ip et al., 2019; Meyer et al., 2012), we included the three-way ERN × CR/ES × Age interaction in the models.

[1] We conducted two sets of alternative models that (1) did not include the unique variance of T2 ERN as a covariate (Table A1-A3, Figure A1-A2) or (2) included the original score of T2 ERN as a covariate (Table A4-A6, Figure A3-A4). These models generated highly similar results and are reported in the supplemental materials.

3. RESULTS

3.1. Behavioral results of the Go/No-Go task

At T1, youths had an overall accuracy rate of 58.0% (Mean/SD number of errors = 26.29/11.02) on No-Go trials. They responded faster on error trials (Mean/SD = 328.69/62.81 ms) compared to correct trials (Mean/SD = 398.86/74.52 ms, $t(114) = -13.09$, $p = .000$). Additionally, youths responded slower on trials following an error response (Mean/SD = 472.79/113.70 ms) compared to trials following a correct response (Mean/SD = 376.53/71.24 ms, $t(114) = 13.07$, $p = .000$). Similar results were found at T2. Youths' overall accuracy rate was 58.7% (Mean/SD number of errors = 25.86/10.23). RTs were faster on error trials (Mean/SD = 328.69/62.81 ms) compared to correct trials (Mean/SD = 376.62/58.84 ms, $t(114) = -19.33$, $p = .000$). Post-error trial RTs (Mean/SD = 421.50/88.19 ms) were slower compared to post-correct trial RTs (Mean/SD = 365.40/58.18 ms, $t(114) = 8.71$, $p = .000$).

3.2. Descriptive statistics and bivariate correlations between study variables

Table 1 shows the means, standard deviations, and bivariate correlations of the study variables. Girls were slightly older than boys. Compared to boys, girls showed a smaller (less negative) ERN_{error} at T1 and T2, a smaller ERN at T2, and elevated anxiety symptoms at T1 and T2. Age was associated with a larger (more negative) $ERN_{correct}$ and ERN_{error}. Greater anxiety symptoms at T1 were associated with a smaller (less negative) ERN and ERN_{error} at T2. Anxiety symptoms at T1 and T2 were positively correlated with each other; both were positively correlated with T2 ES. A larger (more negative) T1 $ERN_{correct}$ was associated with greater T2 ES. Lastly, $ERN_{correct}$ and ERN_{error} were positively correlated with each other and between T1 and T2.

Table 1. Mean, standard deviation, and bivariate correlations of study variables.

	1	2	3	4	5	6	7	8	9	10	11	12
1. Sex *(1=boy, 2=girl)*												
2. Age (in years)	.19*											
3. T1 $ERN_{correct}$	.10	-.37**										
4. T1 ERN_{error}	.21*	-.21*	.40**									
5. T1 ERN	.18	-.07	.00	.92**								
6. T1 Anxiety	.35**	.11	.03	.14	.14							
7. T2 $ERN_{correct}$	.01	-.18	.34**	.09	-.05	.00						
8. T2 ERN_{error}	.20*	-.16	.34**	.56**	.46**	.20*	.57**					
9. T2 ERN	.24*	-.06	.18	.61**	.59**	.24*	.00	.82**				
10. T2 Anxiety	.31**	.09	-.03	.01	.02	.63**	-.04	.12	.17			
11. T2 CR	.03	-.10	-.04	.14	.17	-.03	.06	.13	.12	-.14		
12. T2 ES	.07	.16	-.23*	-.03	.07	.32**	.17	-.14	-.06	.52**	.10	
Mean:	-	11.00	5.86	-2.34	0.00	8.51	6.18	-3.07	0.00	9.63	18.82	10.16
SD:	-	1.16	4.40	5.59	5.13	5.83	4.03	7.81	6.42	6.06	4.48	3.24

Note: * $p < .05$, ** $p < .01$ (2-tailed); T1: Time 1; T2: Time 2; ERN: error-related negativity; SD: standard deviation; CR: cognitive reappraisal; ES: expressive suppression.

3.3. Results of multiple regression analyses

Figure 2 shows the waveforms and topographic maps (at the peak latency of 136 ms) of the TF03SF1 factor as an indicator of the ERN in the error and correct conditions at T1 and T2, superimposed on the grand average waveforms of the original ERP data. Paired-sample *t*-test conducted on T1 ERN showed a larger, more negative amplitude of ERN_{error} (Mean/SD = -2.34/5.59) compared to $ERN_{correct}$ (Mean/SD = 5.86/4.40), $t(114) = -15.75$, $p = .000$. T2 ERN_{error} also showed a more negative amplitude (Mean/SD = -3.07/7.81) relative to T2 $ERN_{correct}$ (Mean/SD = 6.18/4.03), $t(114) = -15.42$, $p = .000$.

Table 2 and Table 3 illustrate results of the multiple regression models with CR and ES as the moderator, respectively. In Model 1 with CR as the moderator (Table 2), we did not find a significant main effect of T1 ERN ($ß = -0.06$, $SE = 0.09$, $p = .428$). Consistent with previous studies (Meyer et al., 2012; Weinberg et al., 2016), we found a significant ERN × Age

interaction (ß = -0.23, *SE* = 0.08, *p* = .002), such that a larger ERN was associated with greater anxiety symptoms in older youths of the upper tercile age group (11.68-12.99 years at T1; ß = -0.35, *SE* = 0.14, *p* = .004) but not the younger youths of the middle (10.31-11.67 years at T1; ß = -0.06, *SE* = 0.09, *p* = .417) and lower (9.04-10.30 years at T1; ß = 0.21, *SE* = 0.13, *p* = .067) tercile age groups. The two-way interaction between T1 ERN and T2 CR was not significant (ß = 0.05, *SE* = 0.02, *p* = .577).

Interestingly, the ERN × Age interaction was further qualified by a significant three-way interaction of ERN × CR × Age (ß = 0.20, *SE* = 0.01, *p* = .037). We decomposed the three-way interaction by first probing the two-way interaction of ERN × CR in three tercile age groups (Figure 3). The ERN × CR interaction was significant in the older (upper tercile) age group (ß = 0.27, *SE* = 0.04, *p* = .032) but not in the younger age groups (lower tercile, ß = -0.22, *SE* = 0.02, *p* = .208; middle tercile, ß = -0.10, *SE* = 0.04, *p* = .536). Further decomposing the ERN × CR interaction in the older age group showed that a larger T1 ERN predicted greater anxiety symptoms at T2 for those with lower (ß = -0.52, *SE* = 0.17, *p* = .000) and middle tercile (ß = -0.34, *SE* = 0.16, *p* = .004) levels of CR, but not for those with higher CR (ß = -0.02, *SE* = 0.28, *p* = .935; see Figure 3).

Table 2. Regression results with cognitive reappraisal as the moderator.

		B	*ß*	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.07	0.01	0.39	[-0.70, 0.85]	0.19	.850
	Child Sex	1.19	0.10	0.97	[-0.74, 3.11]	1.22	.225
	Unique variance of T2 ERN	0.08	0.07	0.09	[-0.10, 0.25]	0.90	.371
	T1 Anxiety Symptoms	0.60	0.58	0.08	[0.44, 0.77]	7.34	.000**
					R^2 = 0.41, F(4, 110) = 19.11, p < .001		
Step 2:	T1 ERN	-0.07	-0.06	0.09	[-0.25, 0.11]	-0.80	.428
	T2 CR	-0.16	-0.12	0.10	[-0.36, 0.04]	-1.61	.109
					R^2 = 0.43, F(6, 108) = 13.56, p < .001		
Step 3:	T1 ERN × T2 CR	0.01	0.05	0.02	[-0.02, 0.04]	0.56	.577
	T1 ERN × Age	-0.24	-0.23	0.08	[-0.40, -0.09]	-3.18	.002**
	T2 CR × Age	0.03	0.03	0.08	[-0.14, 0.20]	0.37	.713
					R^2 = 0.48, F(9, 105) = 10.79, p < .001		
Step 4:	T1 ERN × T2 CR × Age	0.03	0.20	0.01	[0.00, 0.06]	2.12	.037*
					R^2 = 0.50, F(10, 104) = 10.49, p < .001		

Note: * p < .05, ** p < .01; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; CR: cognitive reappraisal.

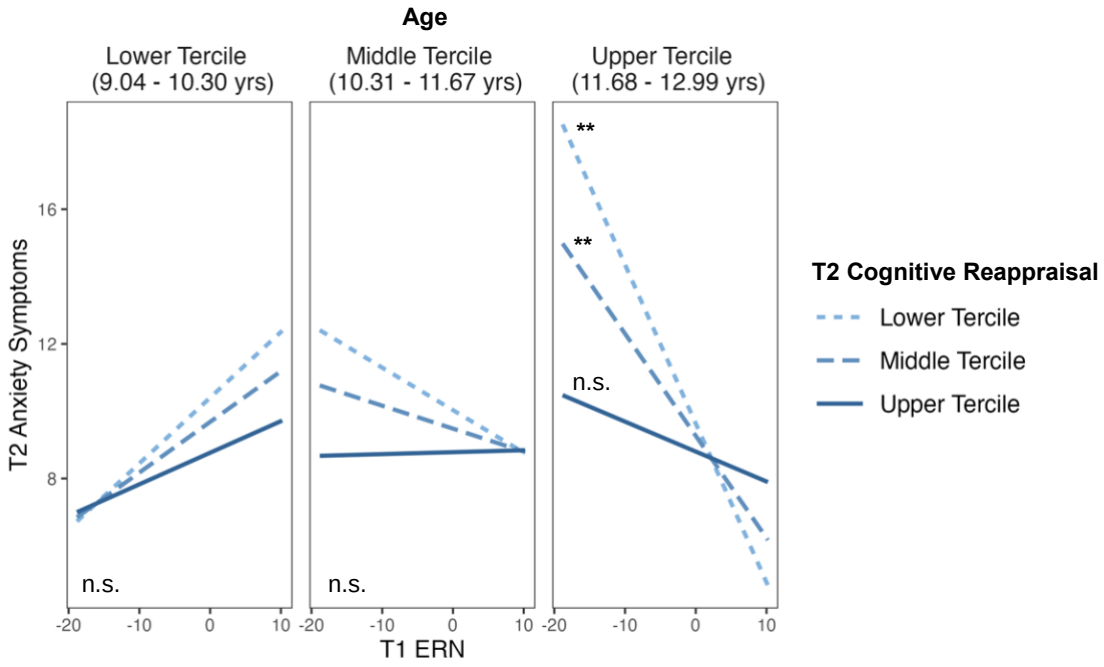

Figure 3. An illustration of the three-way interaction between T1 ERN, T2 CR, and Age in predicting T2 anxiety symptoms. Note: ** p < .01; n.s.: non-significant; T1: Time 1; T2: Time 2; CR: cognitive reappraisal; ERN: error-related negativity; a larger ERN is reflected by a more negative amplitude.

In Model 2 with ES as the moderator, the ERN × ES × Age interaction was not significant (*ß* = -0.12, *SE* = 0.02, *p* = .097). To preserve power, we removed the interaction terms of age from the model.[2] In the revised model (Table 3), the main effect of T1 ERN was not significant (*ß* = -0.10, *SE* = 0.08, *p* = .154), although the association was in the expected direction. The main effect of ES was significant, with greater T2 ES associated with greater T2 anxiety symptoms (*ß* = 0.38, *SE* = 0.13, *p* = .000). The ERN × ES interaction was also significant (*ß* = -0.16, *SE* = 0.02, *p* = .021). As illustrated in Figure 4, decomposing this two-way interaction showed that a larger T1 ERN predicted greater T2 anxiety symptoms for youths with higher (*ß* = -0.24, *SE* = 0.11, *p* = .009) but not middle (*ß* = -0.11, *SE* = 0.08, *p* = .104) or lower (*ß* = -0.02, *SE* = 0.09, *p* = .734) tercile levels of ES.

Table 3. Regression results with expressive suppression as the moderator.

		B	*ß*	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.07	0.01	0.39	[-0.70, 0.85]	0.19	.850
	Child Sex	1.19	0.10	0.97	[-0.74, 3.11]	1.22	.225
	Unique variance of T2 ERN	0.08	0.07	0.09	[-0.10, 0.25]	0.90	.371
	T1 Anxiety Symptoms	0.60	0.58	0.08	[0.44, 0.77]	7.34	.000**
					R^2 = 0.41, F(4, 110) = 19.11, p < .001		
Step 2:	T1 ERN	-0.11	-0.10	0.08	[-0.27, 0.04]	-1.44	.154
	T2 ES	0.70	0.38	0.13	[0.44, 0.96]	5.38	. 000**
					R^2 = 0.54, F(6, 108) = 21.07, p < .001		
Step 3:	T1 ERN × T2 ES	-0.05	-0.16	0.02	[-0.09, -0.01]	-2.34	.021*
					R^2 = 0.56, F(7, 107) = 19.60, p < .001		

Note: * p < .05, ** p < .01; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression.

[2] Results of the original model that included these terms (ERN × ES × Age, ERN × Age, ES × Age) are reported in the supplemental materials (Table A7-A8). In this model, the ERN × Age interaction was significant (*ß* = -0.14, *SE* = 0.08, *p* = .047). Decomposing the interaction showed the expected patterns: the ERN-symptom association was marginally significant for older youths of the upper tercile of age (*ß* = -0.22, *SE* = 0.13, *p* = .051), but not for younger youths of the lower (*ß* = 0.13, *SE* = 0.14, *p* = .271) or middle (*ß* = -0.04, *SE* = 0.09, *p* = .600) terciles of age.

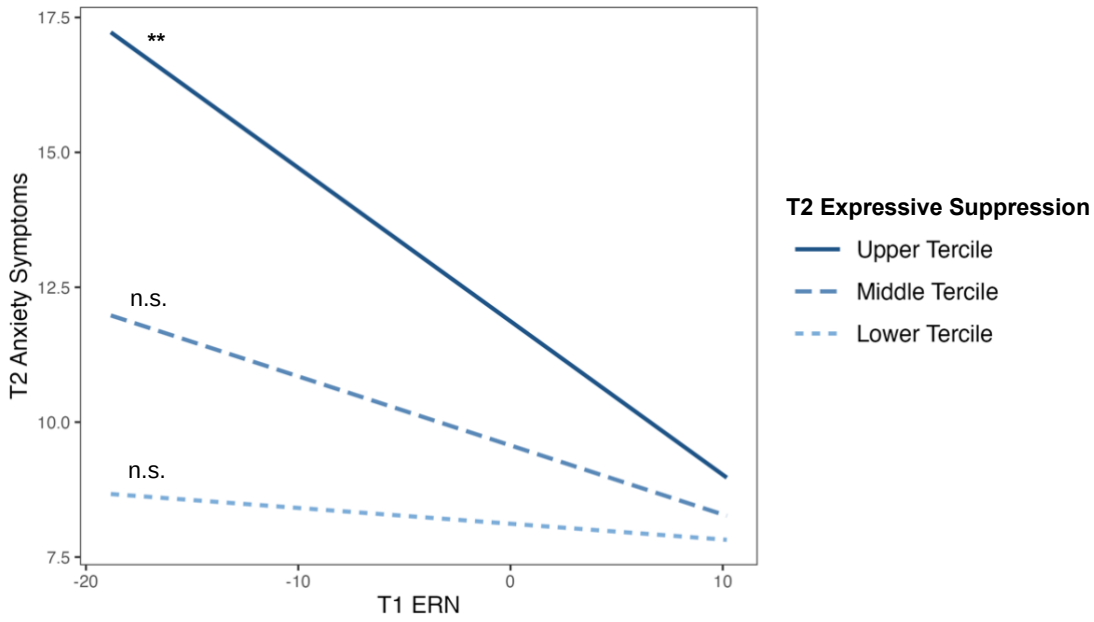

Figure 4. An illustration of the interaction between T1 ERN and T2 ES in predicting T2 anxiety symptoms. Note: ** p < .01; n.s.: non-significant; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression; a larger ERN is reflected by a more negative amplitude.

4. DISCUSSION

Using two-wave longitudinal data from a sample of community-dwelling youths, the present study was the first to examine the moderating effect of emotion regulation on the prospective association between the ERN and anxiety symptoms during early adolescence. As expected, we observed significant moderating effects of emotion regulation (CR or ES) on the association between T1 ERN and T2 anxiety symptoms. The moderating effect of ES on the ERN-symptom association was significant for our entire sample, such that T1 ERN predicted T2 anxiety symptoms for youths with higher, but not lower, levels of ES. However, the moderating effect of CR was significant only for older youths in our sample, as demonstrated by an ERN × CR × Age interaction. Specifically, among older (upper age tercile) youths, the ERN-symptom association was significant for those with lower, but not higher, levels of CR. These findings contribute novel evidence on the moderating effects of CR and ES, two important aspects of emotion regulation, on the prospective ERN-anxiety relationship in early adolescence. Our results further elucidate an age-specific pattern in the moderating role of CR, such that the moderating effect of CR was significant only for older youths.

The moderating effect of ES on the ERN-anxiety association indicated that a larger ERN at T1 predicted greater anxiety symptoms at T2 only for youths with relatively higher, but not lower, ES. In other words, a greater tendency to employ ES strategies of emotion regulation strengthened the prospective association between ERN and anxiety symptoms. This indicates that greater ES tendencies might have exacerbated the development of anxiety symptoms, especially for youths with a larger ERN. Conversely, a lower tendency of using ES strategies might have protected youths with a larger ERN against the increase of anxiety symptoms over time. These patterns were consistent with existing findings on the effects of ES on the

development of psychopathology (Chukwuemeka & Obi-Nwosu, 2021; Farmer & Kashdan, 2012; Kashdan & Breen, 2008). For example, socially-anxious young adults who relied more on ES strategies reported fewer positive social events and less positive emotions on the next day (Farmer & Kashdan, 2012) and smaller increases in positive emotions three months later (Kashdan & Breen, 2008). Similarly, thought suppression, another suppression-related regulation strategy, was linked to higher eating psychopathologies among 15-23-year-old females, but only for those with maladaptive body image comparisons (e.g., greater discrepancy between their perceived and ideal body image; Ferreira et al., 2015).

On the other hand, the moderating effect of CR on the ERN-anxiety association was significant only in youths of the upper age tercile (11.68-12.99 years at T1). Among these older youths, a larger ERN at T1 predicted greater anxiety symptoms at T2 for those with relatively lower, but not higher, CR. In this case, lower individual tendencies to engage in CR strategies of emotion regulation strengthened the ERN-symptom association, suggesting that lower CR tendencies may confer heightened risks of anxiety for older youths with a larger ERN. Alternatively, greater use of CR strategies buffered the ERN-symptom association, potentially protecting older youths with a larger ERN against the development of anxiety symptoms. Our findings were consistent with the literature supporting the protective role of higher CR against the development of psychopathology in older youths and adults (see review Daniel et al., 2020). For instance, during the transition from high school to college, using more CR strategies reduced the adverse effects of perceived stress on subsequent symptoms of depression and anxiety (Zahniser & Conley, 2018). Similarly, individual tendencies to engage in CR weakened the association between emotional reactivity and depressive symptoms in 14-15-year-old adolescents (Shapero et al., 2016).

4.1. Differential age-specific patterns in the moderating effect of CR and ES

It is interesting to note that the moderating effect of CR, but not ES, was conditioned on age. Specifically, the moderating effect of CR on the ERN-symptom association was significant only in older, and not younger, youths of our sample. This age-specific effect was consistent with previous findings of similar age-related patterns in the effect of CR. For instance, 14-17-year-old adolescents reported greater reduction in negative affect following CR compared to 10-13-year-olds (McRae et al., 2012). Among 10-23-year-old individuals, older age was associated with more effective CR in reducing negative ratings towards aversive stimuli; this relationship between CR and age was especially strong for 10-17-year-olds (Silvers et al., 2012).

We posit that the age-specific patterns in the moderating effect of CR may be related to the maturation of higher-order cognitive processes during adolescence (Morales et al., 2016; Silvers, 2022). Antecedent-focused strategies like CR involves redefining the relevance of emotional stimuli and modifying one's beliefs of their emotional experience, which relies on effortful control and other higher-order cognitive processes (e.g., metacognitive skills; McRae et al., 2012). In this case, younger youths with underdeveloped cognitive control abilities may be less likely to employ, and less successful with, CR strategies. Indeed, van Cauwenberge and colleagues (2017) found that following CR, 12-15-year-olds, but not eight-11-year-olds, showed a significantly reduced late positive potential (an ERP marker of elaborative processing of emotional stimuli) in response to negative stimuli; this suggested that 12-15-year-olds had more successfully tempered the emotional significance of the negative stimuli via CR. In another study, CR significantly reduced negative affect and amygdala activation towards aversive images in 14–17-year-olds, but not in 10-13-year-olds (Silvers et al., 2015). Consistent with these findings, in our study, older youths might have employed CR strategies more frequently and, in

some of them, more effectively, yielding greater individual variability in CR that moderated the prospective ERN-symptom relationship.

In contrast, the moderating effect of ES was not conditioned on age but significant for all youths in the current sample. ES refers to the tendency to inhibit behavioral responses towards emotional stimuli. Unlike CR, ES and other response-focused emotion regulation strategies may emerge and mature earlier in life, ahead of the maturation of higher-order cognitive control abilities (Eisenberg et al., 2010). Recent work reported increased individual differences in the frequency of daily use of ES during middle childhood (Gross & Cassidy, 2019), suggesting that ES abilities may mature during this developmental stage. Based on this literature, we speculate that the differential age-related patterns observed in the moderating effects of CR and ES may reflect distinct developmental trajectories of CR and ES, although the mechanisms underlying this differentiation warrants further exploration.

4.2. Strengths, limitations and future directions

Our study had several strengths. First, by assessing the ERN and symptoms at two time points over a 12-month period, we were able to partial out the cross-sectional associations between the ERN and anxiety symptoms, and herein better evince the prospective relationship between T1 ERN and T2 symptoms. Second, instead of group comparisons (e.g., between clinical and non-clinical samples), we took a dimensional approach, examining an unselected community-dwelling youth sample with emerging anxiety symptoms. Such approach increases the statistical power and shed light on the processes shared by both typical and atypical development (Cicchetti & Toth, 2009). Finally, leveraging a PCA approach to analyze the ERP data allowed us to generate more accurate, sample-specific temporo-spatial information of the ERN component.

One limitation of our study was that emotion regulation (CR and ES) was only measured at T2. Future research assessing emotion regulation at more than one time point can provide evidence on whether changes in emotion regulatory abilities over time moderate the prospective ERN-anxiety relationship. While our study focused on an unselected youth sample, examining high-risk or clinical youth samples with more severe anxiety symptoms is necessary to advance our understanding of the relationships between the ERN, emotion regulation, and the development of anxiety. Further work on mid-to-late adolescence and more ethnically-diverse populations will also help determine whether the observed moderating effect of emotion regulation strategies is present beyond early adolescence and White-dominant youth samples. Regardless of the limitations, our study provided initial evidence of the role of emotion regulation in moderating the ERN-anxiety relationship. We also documented age-specific patterns in the moderating roles of CR (but not ES) in the development of anxiety. Future prevention and intervention studies can leverage these findings to tailor training protocols of emotion regulation for youths of different ages; e.g., more CR-focused protocols may benefit older youths in particular.

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APPENDIX A. SUPPLEMENTAL MATERIALS

Table A1. Regression results with cognitive reappraisal as the moderator without T2 ERN as a covariate.

		B	*ß*	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.05	0.01	0.39	[-0.72, 0.82]	0.13	.896
	Child Sex	1.28	0.10	0.97	[-0.63, 3.19]	1.33	.187
	T1 Anxiety Symptoms	0.61	0.59	0.08	[0.45, 0.78]	7.57	.000**
					$R^2 = 0.41$, $F(3, 111) = 25.25$, $p < .001$		
Step 2:	T1 ERN	-0.08	-0.06	0.09	[-0.25, 0.10]	-0.85	.400
	T2 CR	-0.16	-0.12	0.10	[-0.36, 0.04]	-1.59	.114
					$R^2 = 0.43$, $F(5, 109) = 16.15$, $p < .001$		
Step 3:	T1 ERN × T2 CR	0.00	0.02	0.01	[-0.03, 0.03]	0.26	.799
	T1 ERN × Age	-0.25	-0.24	0.08	[-0.40, -0.10]	-3.25	.002**
	T2 CR × Age	0.04	0.03	0.08	[-0.13, 0.21]	0.47	.640
					$R^2 = 0.48$, $F(8, 106) = 12.11$, $p < .001$		
Step 4:	T1 ERN × T2 CR × Age	0.03	0.20	0.01	[0.00, 0.06]	2.20	.030*
					$R^2 = 0.50$, $F(9, 105) = 11.07$, $p < .001$		

Note: * $p < .05$, ** $p < .01$; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; CR: cognitive reappraisal

Table A2. Regression results with expressive suppression as the moderator without T2 ERN as a covariate.

		B	*ß*	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.05	0.01	0.39	[-0.72, 0.82]	0.13	.896
	Child Sex	1.28	0.10	0.97	[-0.63, 3.19]	1.33	.187
	T1 Anxiety Symptoms	0.61	0.59	0.08	[0.45, 0.78]	7.57	.000**
					$R^2 = 0.41$, $F(3, 111) = 25.25$, $p < .001$		
Step 2:	T1 ERN	-0.12	-0.10	0.08	[-0.28, 0.04]	-1.49	.140
	T2 ES	0.70	0.37	0.13	[0.44, 0.96]	5.35	. 000**
					$R^2 = 0.53$, $F(5, 109) = 25.00$, $p < .001$		
Step 3:	T1 ERN × T2 ES	-0.05	-0.17	0.02	[-0.09, -0.01]	-2.54	.013*
					$R^2 = 0.56$, $F(6, 108) = 22.96$, $p < .001$		

Note: * $p < .05$, ** $p < .01$; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression

Table A3. Simple slope effects for models with CR or ES as the moderator without T2 ERN as a covariate.

		Lower Tercile			Middle Tercile			Upper Tercile		
		ß	*SE*	*p*	*ß*	*SE*	*p*	*ß*	*SE*	*p*
Cognitive Reappraisal	Younger Age Tercile	0.17	0.13	.119	0.12	0.14	.293	0.06	0.17	.670
	Middle Age Tercile	-0.11	0.09	.162	-0.06	0.09	.387	-0.01	0.11	.910
	Older Age Tercile	-0.40	0.14	.001**	-0.26	0.15	.036*	-0.09	0.20	.613
Expressive Suppression		-0.03	0.09	.754	-0.13	0.08	.095	-0.25	0.10	.005**

Note: * $p < .05$, ** $p < .01$; SE: standard error; T2: Time 2; ERN: error-related negativity; CR: cognitive reappraisal; ES: expressive suppression

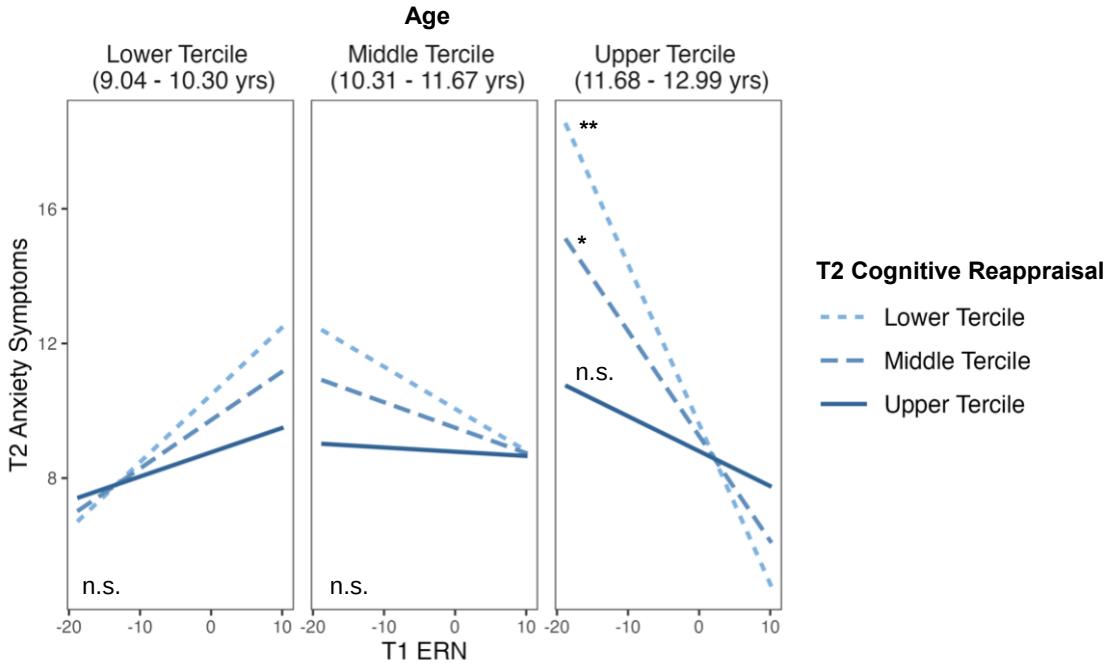

Figure A1. An illustration of the three-way interaction between T1 ERN, T2 CR, and Age in predicting T2 anxiety symptoms without T2 ERN as a covariate. Note: ** $p < .01$; * $p < .05$; n.s.: non-significant; T1: Time 1; T2: Time 2; CR: cognitive reappraisal; ERN: error-related negativity; a larger ERN is reflected by a more negative amplitude.

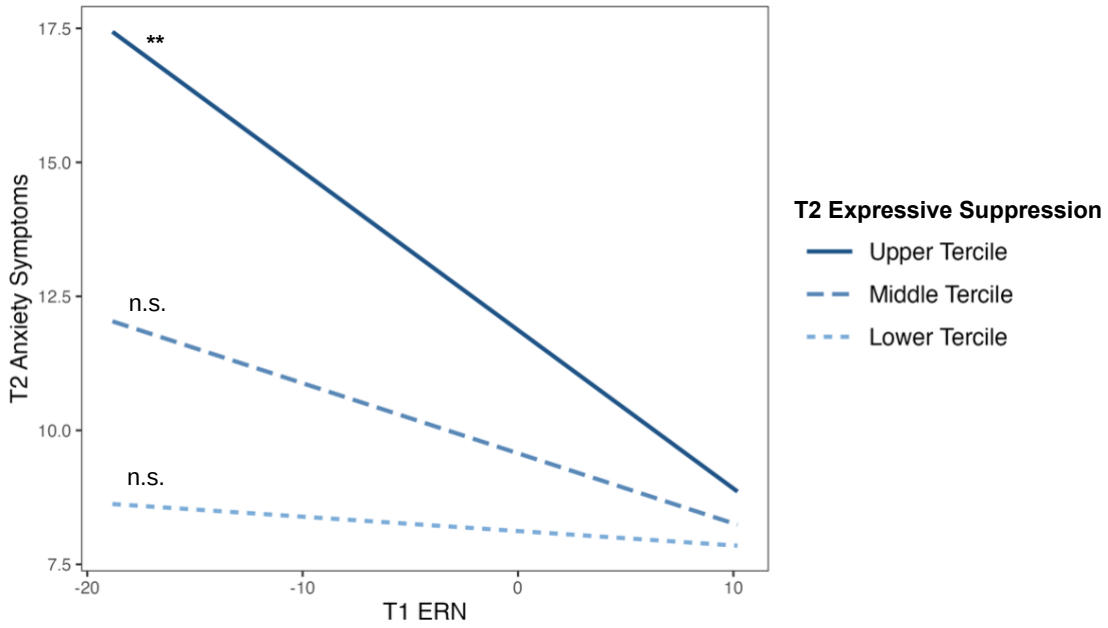

Figure A2. An illustration of the interaction between T1 ERN and T2 ES in predicting T2 anxiety symptoms without T2 ERN as a covariate. Note: ** $p < .01$; n.s.: non-significant; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression; a larger ERN is reflected by a more negative amplitude.

Table A4. Regression results with cognitive reappraisal as the moderator with the original score of T2 ERN as a covariate.

		B	*ß*	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.05	0.01	0.39	[-0.73, 0.83]	0.14	.890
	Child Sex	1.27	0.10	0.99	[-0.69, 3.22]	1.28	.202
	T2 ERN	0.01	0.01	0.07	[-0.14, 0.15]	0.07	.942
	T1 Anxiety Symptoms	0.61	0.59	0.08	[0.45, 0.78]	7.41	.000**
					$R^2 = 0.41$, $F(4, 110) = 18.77$, $p < .001$		
Step 2:	T1 ERN	-0.13	-0.11	0.11	[-0.34, 0.08]	-1.20	.233
	T2 CR	-0.16	-0.12	0.10	[-0.36, 0.04]	-1.61	.109
					$R^2 = 0.43$, $F(6, 108) = 13.56$, $p < .001$		
Step 3:	T1 ERN × T2 CR	0.01	0.05	0.02	[-0.02, 0.04]	0.56	.577
	T1 ERN × Age	-0.24	-0.23	0.08	[-0.40, -0.09]	-3.18	.002**
	T2 CR × Age	0.03	0.03	0.08	[-0.14, 0.20]	0.37	.713
					$R^2 = 0.48$, $F(9, 105) = 10.79$, $p < .001$		
Step 4:	T1 ERN × T2 CR × Age	0.03	0.20	0.01	[0.00, 0.06]	2.12	.037*
					$R^2 = 0.50$, $F(10, 104) = 10.49$, $p < .001$		

Note: * $p < .05$, ** $p < .01$; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; CR: cognitive reappraisal.

Table A5. Regression results with expressive suppression as the moderator with the original score of T2 ERN as a covariate

		B	*ß*	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.05	0.01	0.39	[-0.73, 0.83]	0.14	.890
	Child Sex	1.27	0.10	0.99	[-0.69, 3.22]	1.28	.202
	T2 ERN	0.01	0.01	0.07	[-0.14, 0.15]	0.07	.942
	T1 Anxiety Symptoms	0.61	0.59	0.08	[0.45, 0.78]	7.41	.000**
					$R^2 = 0.41$, $F(4, 110) = 18.77$, $p < .001$		
Step 2:	T1 ERN	-0.18	-0.15	0.10	[-0.37, 0.01]	-1.85	.068
	T2 ES	0.70	0.38	0.13	[0.44, 0.96]	5.38	. 000**
					$R^2 = 0.54$, $F(6, 108) = 21.07$, $p < .001$		
Step 3:	T1 ERN × T2 ES	-0.05	-0.16	0.02	[-0.09, -0.01]	-2.34	.021*
					$R^2 = 0.56$, $F(7, 107) = 19.60$, $p < .001$		

Note: * $p < .05$, ** $p < .01$; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression.

Table A6. Simple slope effects for models with CR or ES as the moderator with the original score of T2 ERN as a covariate.

		Lower Tercile			Middle Tercile			Upper Tercile		
		ß	*SE*	*p*	*ß*	*SE*	*p*	*ß*	*SE*	*p*
Cognitive Reappraisal	Younger Age Tercile	0.13	0.15	.293	0.10	0.15	.444	0.05	0.17	.741
	Middle Age Tercile	-0.14	0.11	.155	-0.09	0.10	.311	-0.03	0.12	.790
	Older Age Tercile	-0.43	0.16	.001**	-0.29	0.16	.032*	-0.11	0.21	.548
Expressive Suppression		-0.05	0.11	.561	-0.14	0.10	.092	-0.27	0.11	.005**

Note: * $p < .05$, ** $p < .01$; SE: standard error; T2: Time 2; ERN: error-related negativity; CR: cognitive reappraisal; ES: expressive suppression

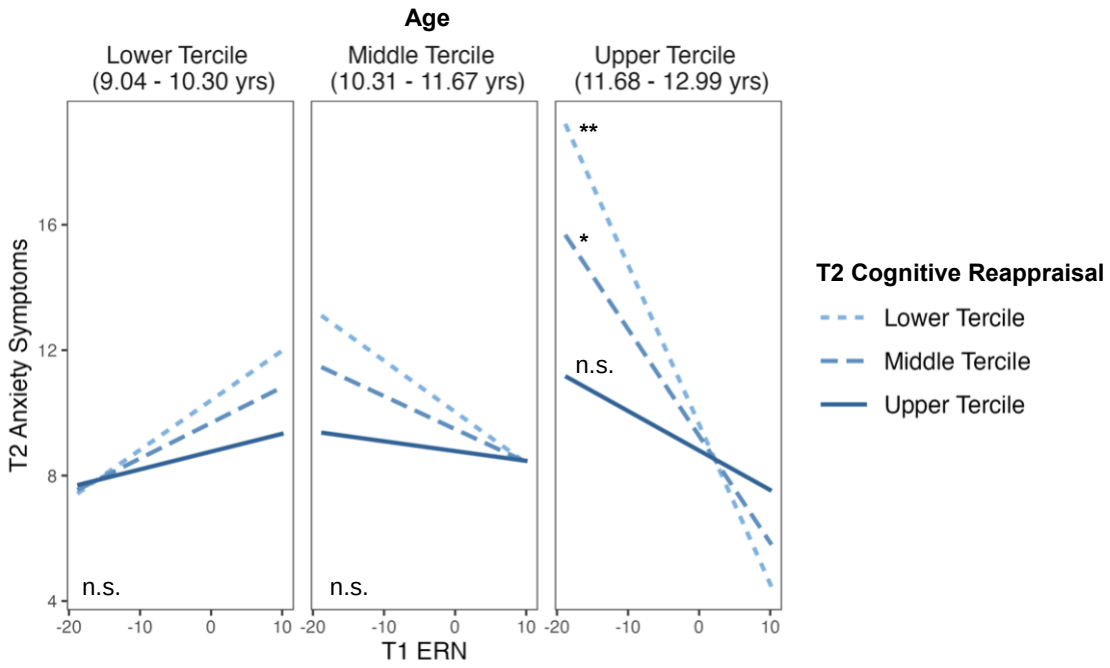

Figure A3. An illustration of the three-way interaction between T1 ERN, T2 CR, and Age in predicting T2 anxiety symptoms with the original score of T2 ERN as a covariate. Note: ** $p < .01$; * $p < .05$; n.s.: non-significant; T1: Time 1; T2: Time 2; CR: cognitive reappraisal; ERN: error-related negativity; a larger ERN is reflected by a more negative amplitude.

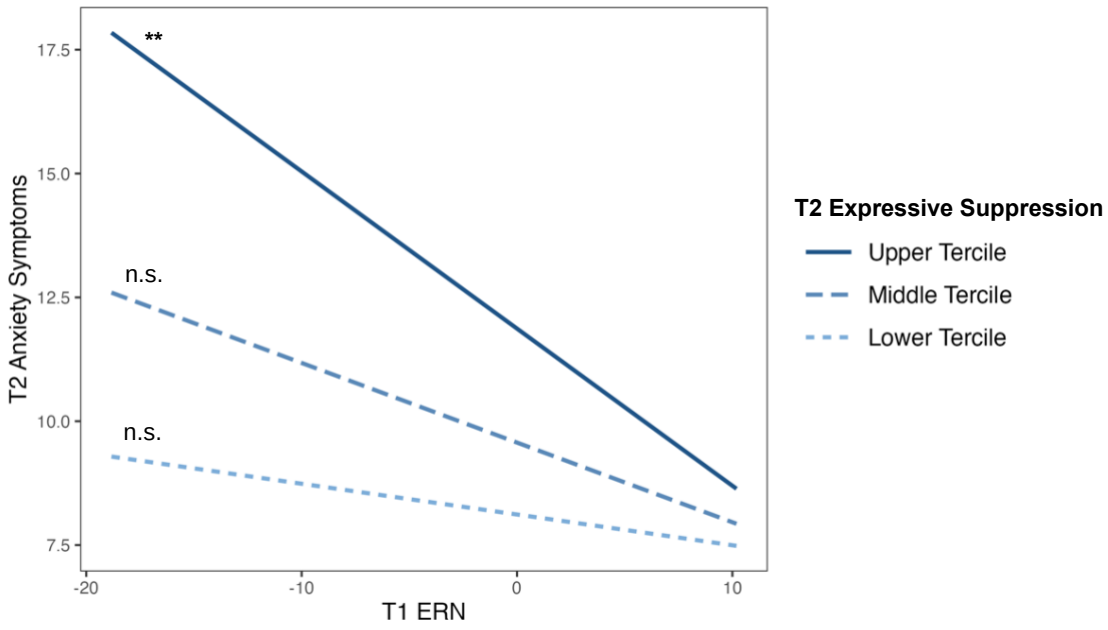

Figure A4. An illustration of the interaction between T1 ERN and T2 ES in predicting T2 anxiety symptoms with the original score of T2 ERN as a covariate. Note: ** $p < .01$; n.s.: non-significant; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression; a larger ERN is reflected by a more negative amplitude.

Table A7. Regression results with expressive suppression as the moderator including the interaction terms with age.

		B	ß	*SE*	*95% CI (B)*	*t*	*p*
Step 1:	Child Age	0.07	0.01	0.39	[-0.70, 0.85]	0.19	.850
	Child Sex	1.19	0.10	0.97	[-0.74, 3.11]	1.22	.225
	Unique variance of T2 ERN	0.08	0.07	0.09	[-0.10, 0.25]	0.90	.371
	T1 Anxiety Symptoms	0.60	0.58	0.08	[0.44, 0.77]	7.34	.000**
						$R^2 = 0.41$, $F(4, 110) = 19.11$, $p < .001$	
Step 2:	T1 ERN	-0.11	-0.10	0.08	[-0.27, 0.04]	-1.44	.154
	T2 ES	0.70	0.38	0.13	[0.44, 0.96]	5.38	. 000**
						$R^2 = 0.54$, $F(6, 108) = 21.07$, $p < .001$	
Step 3:	T1 ERN × T2 ES	-0.03	-0.09	0.02	[-0.07, 0.01]	-1.22	.224
	T1 ERN × Age	-0.12	-0.11	0.07	[-0.26, 0.03]	-1.61	.111
	T2 ES × Age	0.18	0.11	0.11	[-0.04, 0.39]	1.62	.7108
						$R^2 = 0.58$, $F(9, 105) = 16.27$, $p < .001$	
Step 4:	T1 ERN × T2 ES × Age	-0.03	-0.12	0.02	[-0.07, 0.01]	-1.68	.097
						$R^2 = 0.59$, $F(10, 104) = 15.18$, $p < .001$	

Note: * $p < .05$, ** $p < .01$; SE: standard error; CI: confidence interval; T1: Time 1; T2: Time 2; ERN: error-related negativity; ES: expressive suppression.

Table A8. Simple slope effects for the model with ES as a moderator including the interaction terms with age.

	Lower Tercile			Middle Tercile			Upper Tercile		
	ß	*SE*	*p*	ß	*SE*	*p*	ß	*SE*	*p*
Child Age	0.13	0.14	.271	-0.04	0.09	.600	-0.22	0.13	.051

Note: SE: standard error; ES: expressive suppression.