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The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later

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ABSTRACT

Anxiety disorders are often preceded by interpersonal stress; however, most individuals who experience stress do not develop anxiety, making it difficult to predict who is most susceptible to stress. One proposed trans-diagnostic neural risk marker for anxiety is the error-related negativity (ERN), a negative deflection in the event-related potential waveform occurring within 100 ms of error commission. The present study sought to investigate whether interpersonal stress experienced over the course of a year interacts with ERN magnitude to prospectively predict anxiety symptoms. A sample of 57 emerging adults performed an arrow flanker task to elicit the ERN at the start of the academic school year (time one). Toward the end of the academic year (time two), participants reported on past-year interpersonal stress and anxiety symptoms. Stress interacted with ERN magnitude to predict anxiety symptoms, whereby, for individuals with an enhanced ERN at time one, greater interpersonal stress over the course of a year was significantly associated with increased anxiety symptoms at time two, even controlling for anxiety symptoms at time one. These findings suggest that enhanced performance monitoring may render individuals more susceptible to the adverse effects of interpersonal stress, thereby increasing risk for heightened anxiety.

1. Introduction

Anxiety disorders are among the most common and persistent forms of mental illness worldwide (Baxter et al., 2013; Kessler et al., 2005; Polanczyk et al., 2015). In addition to being associated with emotional distress and severe impairments in interpersonal functioning and job performance (Antony and Stein, 2008), anxiety disorders place significant economic strain on the health care system (Kessler and Greenberg, 2002). Anxiety is often preceded by episodes of life stress (Faravelli, 1985; Faravelli and Pallanti, 1989; Finlay-Jones and Brown, 1981; Green et al., 2010; Hankin et al., 2004; Young and Dietrich, 2015), and interpersonal stressors such as entrapment, humiliation, and peer victimization are particularly salient in predicting symptoms (Farmer and Kashdan, 2015; Hamilton et al., 2016; Kendler et al., 2003; Siegel et al., 2009; Uliaszek et al., 2010). Prior research demonstrates relationships between interpersonal stress and panic disorder (Klauke et al., 2010), social anxiety disorder (Brook and Schmidt, 2008; Siegel et al., 2009), agoraphobia (Kleiner and Marshall, 1987; Last et al., 1984), and obsessive-compulsive disorder (Cromer et al., 2007; Real et al., 2011), suggesting that the link is not symptom- or disorder-specific.

However, not everyone who experiences life stress goes on to develop psychopathology (Ingram and Luxton, 2005; Harkness et al., 2015; Harkness and Monroe, 2016) — in fact, most will not — making it difficult to predict who is at risk of increased anxiety following stress exposure. Diathesis-stress models of psychopathology suggest that certain vulnerability factors, such as a genetic liability, and significant stress exposure (e.g., a divorce), interact to place individuals at risk of developing psychopathology (Ingram and Luxton, 2005; Monroe and Simons, 1991). Assessing both proposed diatheses and stressors may thus be important for understanding the etiology of anxiety.

Recently, there has been increased interest in elucidating neural systems involved in the development and maintenance of anxiety (Pine, 2007). One proposed neural marker of risk for anxiety is the error-related negativity (ERN; Olvet and Hajcak, 2008; Riesel et al., 2011, 2015), an event-related potential (ERP) component that is larger for erroneous than correct responses between 0 and 100 ms following the response (Falkenstein et al., 1991; Gehring et al., 1993). The ERN is a negative deflection in the ERP waveform that is maximal at fronto-central electrode sites and is thought to reflect activity of the anterior

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cingulate cortex (ACC; Brázdil et al., 2005; Dehaene et al., 1994; Stemmer et al., 2004). It is hypothesized to represent an alarm signal generated by a neural network engaged in performance monitoring, signifying that an error has been made and increased cognitive control is needed to adjust behaviour (Carter and van Veen, 2007; Dehaene, 2018; Holroyd and Coles, 2002; Lo, 2018).

An enhanced ERN has been observed in trait anxious adults and children (Hajcak et al., 2003; Meyer et al., 2012, 2016; Moser et al., 2013; Olvet and Hajcak, 2008), as well as individuals with obsessive-compulsive disorder (OCD; Carrasco et al., 2013; Endrass et al., 2010, 2014; Gehring et al., 2000; Hajcak and Simons, 2002; Hajcak et al., 2008; Riesel, 2019; Riesel et al., 2011), generalized anxiety disorder (GAD; Weinberg et al., 2012, 2015b; Xiao et al., 2011), and social anxiety disorder (SAD; Endrass et al., 2014; Kujawa et al., 2016). A larger ERN is also associated with heightened negative affect (Hajcak et al., 2004; Luu et al., 2000; Wiswede et al., 2009), a transdiagnostic characteristic of anxiety disorders (Clark and Watson, 1991).

This enhanced ERN is not only evident in already-affected anxious individuals, but may also be a viable *risk* marker for anxiety (Olvet and Hajcak, 2008; Riesel et al., 2011, 2015). For instance, the ERN is heritable, with genes accounting for approximately 50% of the variation in its magnitude (Anokhin et al., 2008), and an enhanced ERN in childhood and adolescence can prospectively predict subsequent increases in anxiety (Lahat et al., 2014; McDermott et al., 2009; Meyer, 2017; Meyer et al., 2015, 2018). However, an enhanced ERN is also observed in unaffected first-degree relatives of individuals with OCD (Carrasco et al., 2013; Riesel et al., 2011), as well as in individuals in remission from clinically-significant anxiety (Hajcak et al., 2008; Kujawa et al., 2016; Riesel et al., 2015), suggesting that not everyone with an enhanced ERN is anxious. Therefore, a larger ERN appears not to be a symptom, state marker, or "scar" of psychopathology, but rather a latent vulnerability for anxiety and anxiety-related disorders (Olvet and Hajcak, 2008; Riesel et al., 2011, 2015) that can interact with other factors, including stressful events, to set the stage for heightened symptoms (Meyer et al., 2017a). However, it is currently unclear what social-environmental circumstances trigger heightened anxiety in emerging adults with this vulnerability marker.

Taken together, previous research indicates that interpersonal stress often precedes anxiety, and that the ERN may be a viable neural risk marker for anxiety; however, the extent to which interpersonal stress and ERN magnitude might interact to predict anxiety is not yet clear. Further, very little is known about whether ERN magnitude can track symptom changes in adult populations. To address these issues, we examined the extent to which an enhanced ERN in combination with greater interpersonal stress exposure predicts subsequent symptoms of anxiety in a sample of first-year undergraduate students. Neural systems implicated in performance monitoring mature substantially in late adolescence and young adulthood (e.g., Hogan et al., 2005; Kelly et al., 2009; Ladouceur et al., 2007; Segalowitz and Dywan, 2009; Steinberg, 2005), which is a time of increased stress sensitivity (Walker et al., 2004), and heightened risk for psychopathology (Birmaher et al., 1996; Braet et al., 2013; Kessler et al., 2001; Wagner and Compas, 1990). Importantly, the entry to university is a time of heightened interpersonal stress (Bouteyre et al., 2007; Fisher and Hood, 1988; Schlossberg, 1989; Wilcox et al., 2005), and first-year students endorse more symptoms of psychopathology than students in later years (Adlaf et al., 2001). All of these factors make our sample an important population in which to investigate how interpersonal stress interacts with error monitoring to predict anxiety.

To that end, we measured ERN magnitude at the beginning of the academic year (i.e., in the first month and a half at university), interpersonal and non-interpersonal stressors experienced across the first year at university, and anxiety symptoms toward the end of the academic year. Because of prior research indicating that the ERN is a transdiagnostic marker of anxiety (Meyer, 2017; Riesel et al., 2017, 2019; Weinberg et al., 2016), we investigated a variety of anxiety symptoms as a composite score. We hypothesized that, for undergraduate students with a large ERN at baseline, greater interpersonal stress exposure over the year would predict more subsequent symptoms of anxiety at the end of the year while controlling for baseline anxiety levels. In order to determine if these effects were specific to social stressors, we also conducted exploratory analyses to investigate whether this effect is evident for non-interpersonal stressors.

2. Method

Two hundred and fifty-six first-year undergraduate students from McGill University were recruited at the start of the academic year (Time 1) over three consecutive years. The first (N = 92), second (N = 73), and third (N = 91) wave of participants were recruited in 2016, 2017, and 2018, respectively. Participants were recruited from the University's psychology human participant pool, verbal advertisements in classrooms, and flyers posted around the campus. Participants either received course credit or monetary compensation for their time. For those requesting monetary compensation, $23 was given to wave one and two participants, and $28 was given to wave three participants. Permission to recontact was obtained from 211 participants at Time 1. Toward the end of the first academic year, approximately six months after the initial lab visit (Time 2), those 211 participants were re-contacted several times via email with an invitation to complete online questionnaires. Participants were compensated $10 for participating in the Time 2 questionnaires, and entered into a draw to win a $100 gift card. Informed consent was obtained prior to participation and the research protocol was approved by the Research Ethics Board at McGill University.

At Time 1, two participants were excluded due to excessive noise in the electroencephalogram (EEG) data, 28 participants were excluded due to committing too few errors (i.e., fewer than 6; Olvet and Hajcak, 2009), 11 were excluded because they were taking psychotropic medication (i.e., anti-depressant/anxiety medication; De Bruijn et al., 2004; Zirnheld et al., 2004), and one participant was excluded because their Time 1 anxiety was more than three standard deviations above the sample mean. Of the remaining 214 participants, 59 participants (28% of the original sample) completed the online questionnaires at the end of the year. Two of these 59 participants were excluded because their scores on either the Time 2 anxiety or stress exposure measures were more than three standard deviations above the sample mean. Therefore, the final number of participants with usable data at Time 2 was 57.[1] Because of the size of this final sample, we conducted a sensitivity analysis using G*Power (Faul et al., 2007), to establish the smallest effect size we had at least 80% power to detect. With a total sample size of 57, eight predictors in a multiple regression, and α error probability set to 0.05, the smallest effect size we could detect was $f^2 = 0.11$. Meta-analyses investigating the association between ERN magnitude and anxiety report effect sizes ranging from $f^2 = 0.18$ (Moser et al., 2016) to $f^2 = 0.26$ (Moser et al., 2013), suggesting our sample would support this investigation.

The mean age of this final sample was 18.12 years old ($SD = 0.47$) at the start of the year, and 84% of participants were female. Forty-four percent of participants were Caucasian, 28% were Chinese, 7% were

[1] The participants who did not complete the questionnaires at Time 2 (N = 155) did not differ in age ($t_{208} = 0.14$, $p = .89$), ethnicity ($\chi^2(10) = 18.59$, $p = .05$), annual family income ($\chi^2(13) = 13.20$, $p = .43$), or baseline symptoms of anxiety ($t_{209} = 1.45$, $p = .15$) compared to those who completed the questionnaires at Time 2 (N = 57; exclusive of the two participants excluded at Time 2). Gender was a significant predictor of attrition ($\chi^2(1) = 4.28$, $p = .04$), with 108 females and 46 males lost to follow-up versus 48 females and 9 males who completed the follow-up. The magnitude of the ERN and CRN did not differ between participants who did and did not complete the follow-up questionnaires at Time 2 (ERN, $t_{210} = 1.59$, $p = .11$; CRN, $t_{210} = 0.23$, $p = .82$).

South East Asian, 5% were South Asian, 2% were Caribbean, 2% were Arab/West Asian, 2% were Hispanic, 2% were Korean, and 8% indicated they were another ethnicity. The median annual family income of the sample was between $100,000 and $149,999 (range: $50,000 to $250,000 or greater). For comparison, the median family income in Canada in 2017 was $92,990 for families consisting of a couple (and children, if applicable) living at the same address, and $46,140 for single-parent families (Statistics Canada, n.d.). However, we did not adjust participants' reported income by number of people in their immediate family, and did not collect information about number of wage-earners in their family.

2.1. Measures

2.1.1. Questionnaires

At Time 1 and 2, participants completed the Inventory of Depression and Anxiety Symptoms (IDAS-II; Watson et al., 2012). The IDAS-II is a 99-item self-report measure of 18 empirically derived internalizing dimensions of depression and anxiety. Items assess symptoms over the past two weeks and participants make their responses using a 5-point Likert-type scale ranging from 1 (*not at all*) to 5 (*extremely*). The IDAS-II has demonstrated good internal consistency, test-retest reliability, and convergent and discriminant validity with diagnoses and self-report measures in similar populations (Watson et al., 2012). The present study focused on a composite measure of anxiety symptoms by summing across the eight anxiety subscales of the IDAS-II. This composite score represents the total sum of panic (8 items; range: 8–40), social anxiety (6 items; range: 5–30), claustrophobia (5 items; range: 5–25), traumatic intrusions (4 items; range: 4–20), traumatic avoidance (4 items; range: 4–20), checking (3 items; range: 3–15), ordering (5 items; range: 5–25), and cleaning (7 items; range: 7–35) subscales; therefore, 42 items were included in our composite anxiety score (range: 42–210; Time 1 $\alpha = 0.94$; Time 2 $\alpha = 0.90$). We used this composite anxiety score because interpersonal stress is associated with multiple forms of anxiety and symptom profiles (e.g., panic disorder [Klauke et al., 2010], social anxiety disorder [Brook and Schmidt, 2008; Siegel et al., 2009], agoraphobia [Kleiner and Marshall, 1987; Last et al., 1984], and obsessive-compulsive disorder [Cromer et al., 2007; Real et al., 2011]). Additionally, an enhanced ERN has been found in individuals with a broad range of anxiety symptoms and disorders (Carrasco et al., 2013; Endrass et al., 2010; Hajcak and Simons, 2002; Weinberg et al., 2012, 2015b; Endrass et al., 2014; Kujawa et al., 2016). This anxiety composite score allowed us to examine potential moderating effects of the ERN on a broader measure of anxiety symptoms following exposure to past-year stress.

At Time 2, participants also completed the past-year version of the Stress and Adversity Inventory for Adults (Adult STRAIN; Slavich and Shields, 2018). The STRAIN is an online interview that assesses the severity and frequency of individuals' exposure to different stressors over the entire lifetime, and the past-year version of the STRAIN used here includes the same stressor questions but focuses specifically on the past 12 months. Participants respond to questions probing 55 different types of acute life events and chronic difficulties; for each stressor that is endorsed, follow-up questions are asked about its timing, severity, duration, and frequency. Summary scores can be computed that reflect the count and severity of total, acute, and chronic stress experienced across 12 major life domains (i.e., housing, education, work, treatment/health, marital/partner, reproduction, financial, legal/crime, other relationships, death, life-threatening situations, possessions) and 5 social-psychological characteristics (i.e., interpersonal loss, physical danger, humiliation, entrapment, role change/disruption). The STRAIN has demonstrated excellent test-retest reliability and concurrent and discriminant validity in community and clinical samples (Slavich and Shields, 2018; Slavich et al., 2019), as well as excellent predictive validity in relation to a variety of cognitive, biological, and clinical outcomes including anxiety levels (e.g., Mayer et al., 2019; Stewart et al., 2019; Sturmbauer et al., 2019).

The present study focused on the total count of stressors experienced over the past year within interpersonal and non-interpersonal life domains, separately. To address our specific research question, and in line with prior work involving the STRAIN (Pegg et al., 2019), we created distinct subscales for interpersonal and non-interpersonal stress. Interpersonal stressors included all acute life events and chronic difficulties occurring in the marital/partner life domain (e.g., divorce or serious break-up, ongoing arguments with a spouse or partner) and other relationships domain (e.g., major interpersonal fights with roommate(s) or suitemates). In turn, non-interpersonal stressors included all acute life events and chronic difficulties occurring in the life domains of housing, education, work, treatment/health, reproduction, financial, legal/crime, life-threatening situations, death, and possessions. Higher scores on these two variables indicate greater past-year life stress exposure.

2.2. Task and materials

Participants completed an arrow version of the flanker task (Eriksen and Eriksen, 1974) on an Intel Core i7 computer using Presentation software (Neurobehavioural Systems, Inc.; Albany, CA). All stimuli were displayed on a 19-in. (48.3 cm) computer monitor. On each trial, five horizontally aligned arrowheads were presented in the center of the screen, and targets were always the center arrow. Half of these trials were congruent (“ < < < < < ” or “ > > > > > ”) and half were incongruent (“ > > < > > ” or “ < < > < < ”); the order of congruent and incongruent trials was random. Participants were instructed to use the computer mouse to quickly indicate the direction of the target arrow using the right or left mouse button (i.e., they pressed the right mouse button if the arrow pointed to the right). All stimuli were presented for 200 ms, followed by a black screen that either terminated following response selection or after 1800 ms had elapsed. An intertrial interval ranging at random between 1000 and 2000 ms was then presented. Participants were presented with a black screen with a white fixation cross in the center during response and intertrial periods. Participant response type (correct or incorrect) and reaction time (in ms) on every trial was recorded for later analysis.

2.3. Procedure

Participants visited the lab to complete the EEG assessment within the first month and a half of the academic year. Participants completed multiple computer tasks during the experiment, with the order of the tasks counterbalanced across participants. Other tasks included a social feedback task (as described in Ethridge and Weinberg, 2018), a monetary reward task (also described in Ethridge and Weinberg, 2018), and an emotional picture viewing task (as described in Sandre et al., 2019). Participants completed a 6-trial practice block and were told to be both as fast and as accurate as possible. The actual task consisted of five blocks of 30 trials (150 trials total), and each block was initiated by the participant. At the end of every block, participants received feedback based on their performance on the screen; if accuracy was 75% or lower, the message “Please try to be more accurate” was displayed to increase attention to the task; when more than 80% of responses were correct, the message “Please try to respond faster” was shown to increase the likelihood of the participant committing more errors; otherwise, the message “You are doing a great job” was presented.

Approximately six months after the first lab visit ($M_{days} = 176.05$, $SD = 13.65$, range = 149–208), during the final weeks of the academic year, all participants were re-contacted and invited to complete an online version of the IDAS-II and STRAIN.

2.4. Electroencephalogram recording and data processing

Continuous EEG was recorded with a 32-electrode cap and a

BrainVision actiCHamp system. The cap used the standard 10/20 layout and the ground electrode was placed at Fpz. The electrooculogram (EOG) generated from blinks and eye movements was recorded using facial electrodes placed around 1 cm to the left and right of both eyes (HEO) and 1 cm below and above one eye (VEO). Data were recorded with a sampling rate of 1000 Hz. Across all participants, the average electrode impedance was below 10 kΩ.

EEG data were analyzed offline using BrainVision Analyzer software (Brain Products, Munich, Germany). Continuous (unsegmented) data were band-pass filtered with fourth order low and high cut-offs of 0.01 and 30 Hz, respectively, using a Butterworth zero phase filter with a 24 dB/octave roll-off. Following this, for each trial, the EEG was segmented into 1500 ms windows starting 500 ms before each response onset and continuing for 1000 ms post-response. Then data were referenced offline to the average of left (TP9) and right (TP10) mastoids. Ocular and eye-blink corrections were conducted using HEO and VEO using the method developed by Miller et al. (1988). A semi-automatic artifact rejection procedure was conducted in which data from individual channels were automatically rejected if there was a voltage step greater than 50 μV/ms, a difference greater than 175 μV within 400 ms, or activity of less than 0.5 μV in 100 ms intervals. Visual inspection of the data by trained research assistants was then conducted to detect and reject any remaining artifacts.

Error and correct trials were then averaged separately. The mean voltage in the 200 ms window from −500 to −300 ms before response onset served as a baseline and was subtracted from each data point (Gorka et al., 2017; Meyer et al., 2014; Weinberg and Hajcak, 2011). Based on visual inspection of the grand averaged data, the ERN was quantified on error trials as the average activity from 0 to 100 ms at electrode site Cz, where error-related brain activity has been shown to be maximal and have high internal consistency reliability (Riesel et al., 2013; Sandre et al., revise & resubmit). In addition, the correct-response negativity (CRN) was evaluated in the same time window and electrode site on correct trials. The CRN is a negative deflection in the ERP that typically follows both error and correct responses (Burle et al., 2008) and appears to reflect generic response monitoring (Simons, 2010). Therefore, to isolate error-specific neural activity, we used a regression-based procedure to compute unstandardized residuals of the ERN (Meyer et al., 2017b). To calculate the ERN_{resid}, participants' CRN was entered as the predictor, and the ERN was the dependent variable; the ERN_{resid} scores are the saved unstandardized residuals from this regression.

Internal consistency (split-half reliability) of the ERP components of interest were calculated by examining correlations between averages based on odd- and even-numbered trials for each response type (i.e., error and correct), corrected using the Spearman-Brown prophecy formula (Nunnally et al., 1967). The ERN (r = 0.84), CRN (r = 0.98), and ERN_{resid} (r = 0.75) demonstrated good internal consistency in the present sample.

Behavioural measures on the flanker task included the number of error trials for each participant, as well as accuracy expressed as a percentage of correct trials out of the total number of trials. Accuracy following error and correct responses was also calculated (post-error accuracy and post-correct accuracy). Average reaction times (RTs) on error and correct trials were calculated separately. Post-error slowing was calculated as the average of [RT (E + 1) – RT (E − 1)] for all errors, where (E + 1) is the trial after the error and (E–1) is the trial before the error (Dutilh et al., 2012). Trials were removed from analyses if RTs were faster than 200 ms or slower than 1000 ms.

2.5. Data analysis

All statistical analyses were conducted using SPSS General Linear Model Software (Version 23). Paired-sample t-tests were used to compare within-subject conditional ERN and CRN magnitude, reaction times (RTs) on error and correct trials, as well as RTs and accuracy following each response type. Pearson coefficients were used to examine zero-order correlations between ERPs (at Time 1), anxiety symptoms (at Time 1 and Time 2), and total past-year interpersonal and non-interpersonal stress (at Time 2).

To examine whether the magnitude of the ERN_{resid} at Time 1 moderated the association between past-year stress exposure and anxiety symptoms at Time 2, we conducted a simultaneous multiple regression with Time 2 anxiety symptoms as the dependent variable. ERN_{resid} magnitude, past-year interpersonal stress, the interaction between ERN_{resid} magnitude and past-year interpersonal stress, past-year non-interpersonal stress, and the interaction between ERN_{resid} magnitude and past-year non-interpersonal stress were entered as predictors. Anxiety symptoms at baseline (Time 1), as well as time between baseline and follow-up assessments (in days) were included as covariates. We also entered gender (0 = male; 1 = female) as a covariate given evidence of gender differences in the ERN and its association with individual differences (Fischer et al., 2016; Larson et al., 2011; Moser et al., 2016; Sandre et al., revise & resubmit).

3. Results

3.1. Life stress exposure

Over the past year, participants experienced an average of 4.25 total stressors (SD = 3.26; range = 0–14), with an average total stressor severity score of 11.65 (SD = 10.39; range = 0–42). On average, participants experienced 1.23 interpersonal stressors (SD = 1.18; range = 0–5) and 3.02 non-interpersonal stressors (SD = 2.77; range = 0–12) over the past year.

3.2. Flanker task performance

Participants made an average of 14.47 errors (SD = 6.15; range = 6–34) and 134.98 correct responses (SD = 6.65, range = 109–144). Mean post-error slowing was 45.30 ms (SD = 44.06). Participants were faster on error (M = 302.07, SD = 30.15) as compared to correct trials (M = 376.89, SD = 37.07; t(56) = 16.34, p < .001), and were slower to respond following error trials (M = 389.79, SD = 48.43) compared to trials following correct trials (M = 366.67, SD = 36.71; t(56) = 5.13, p < .001). Additionally, participants were more accurate following error trials (M = 0.93, SD = 0.08) than following correct trials (M = 0.90, SD = 0.04; t(56) = 3.27, p = .002).

Fig. 1A depicts response-locked ERP activity at Cz and Fig. 1B shows the scalp distribution of the error minus correct difference from 0 to 100 ms for the full sample. As depicted, the ERN was observed as a larger negativity in the waveform compared to the CRN (t(56) = 11.24, p < .001). Table 1 reports the means, standard deviations, and ranges for all Time 1 and Time 2 measures, as well as bivariate associations among these variables.

3.3. Moderation analyses

Moderated multiple regression analysis was used to examine whether the magnitude of the ERN_{resid} at the start of the year moderated the effects of total past-year interpersonal and non-interpersonal stress exposure in predicting anxiety symptoms at follow-up, adjusting for baseline anxiety symptoms, gender, and time between symptom assessments (in days). As indicated in Table 2, the ERN_{resid} X total past-year interpersonal stress interaction term significantly predicted anxiety symptoms at follow-up, controlling for the interaction between ERN_{resid} and total past-year non-interpersonal stress exposure. In contrast, the ERN_{resid} X total past-year non-interpersonal stress interaction did not significantly predict anxiety symptoms at Time 2.

Simple slopes were calculated at small (1 SD above the mean, as the ERN is a negative-going component; M + 1 SD = 5.69), intermediate

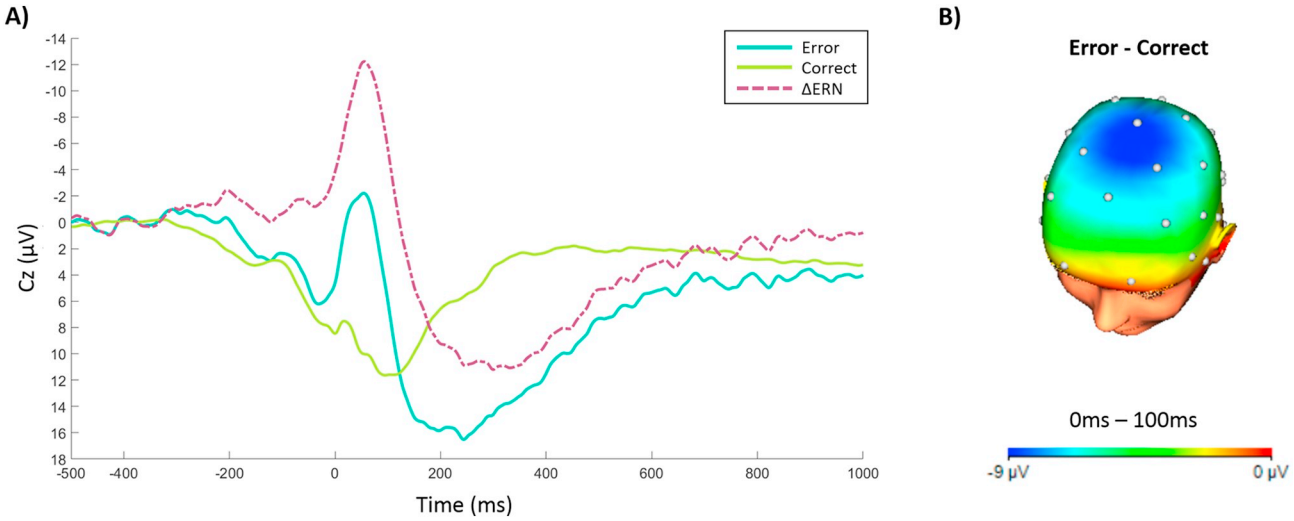

Fig. 1. A) Response-locked ERP average waveforms following error and correct responses, as well as the error minus correct difference wave (i.e., ΔERN), at electrode Cz. B) Topographic map depicting the average difference (μV) between error and correct responses from 0 ms to 100 ms post-response onset.

Table 1
Descriptive statistics and bivariate correlations for neural activity at Time 1, anxiety symptoms at Times 1 and 2, total count of past-year interpersonal and non-interpersonal stressors, and time between assessments.

	1	2	3	4	5	6	7	Mean	*SD*	Range
1. ERN (T1)								0.42	7.10	−17.47–17.61
2. CRN (T1)	0.60**							9.58	6.58	−7.25–29.86
3. ERN_{resid} (T1)	0.80**	0.00						0.00	5.69	−16.15–16.22
4. Anxiety symptoms (T1)	−0.16	−0.18	−0.06					74.84	22.20	47.00–139.00
5. Anxiety symptoms (T2)	−0.08	−0.10	−0.02	0.71**				65.86	16.72	44.00–119.00
6. Total past-year interpersonal stressors (T2)	−0.15	−0.10	−0.12	0.24	0.32*			1.23	1.18	0.00–5.00
7. Total past-year non-interpersonal stressors (T2)	−0.03	−0.12	0.05	0.18	0.29*	0.24		3.02	2.77	0.00–12.00
8. Time between symptom assessment (days)	−0.06	0.00	−0.07	−0.04	−0.01	0.07	0.15	176.05	13.65	149.00–208.00

Note. T1 = Time one; T2 = Time two; ERN = error-related negativity; ERN_{resid} = error-related negativity residual; CRN = correct-response negativity; *SD* = standard deviation.
** $p < .01$.
* $p < .05$.

(mean; $M = 0$), and large (1 *SD* below the mean; $M - 1\ SD = -5.69$) residual ERN values; results are reported in Table 3. As hypothesized, the conditional effect of past-year interpersonal stress exposure on Time 2 anxiety scores was significant at large (i.e., more negative) residual ERN magnitude, $b = 5.40$ ($SE = 1.81$), $p = .004$, whereby greater interpersonal stress exposure was associated with more anxiety. In contrast, at smaller (i.e., less negative) residual ERN magnitude, greater stress exposure was significantly associated with fewer symptoms of anxiety, $b = -6.57$ ($SE = 3.16$), $p = .04$. Fig. 2 displays simple slopes, adjusting for effects of gender, time between assessments, baseline anxiety, past-year non-interpersonal stress, and the interaction between past-year non-interpersonal stress and residual ERN magnitude.[2]

Table 2
Results of a simultaneous multiple regression investigating whether the residual error-related negativity at Time 1 interacts with total past-year interpersonal and non-interpersonal stress exposure to predict anxiety symptoms at Time 2.

	β	*t*	*p*	95% CI
Gender	0.01	0.11	0.92	−8.16, 9.07
Time between symptom assessment (days)	−0.02	−0.26	0.80	−0.26, 0.20
Anxiety symptoms (T1)	0.65	6.89	0.00	0.34, 0.63
ERN_{resid} (T1)	0.35	2.01	0.05	−0.001, 2.07
Total past-year interpersonal stress (T2)	−0.04	−0.36	0.72	−3.81, 2.66
ERN_{resid} (T1) × Total past-year interpersonal stress (T2)	−0.54	−2.97	0.01	−1.76, −0.34
Total past-year non-interpersonal stress (T2)	0.20	1.98	0.05	−0.02, 2.38
ERN_{resid} (T1) × Total past-year non-interpersonal stress (T2)	0.10	0.59	0.56	−0.11, 0.21
	$R = 0.79$		$R^2 = 0.62$	

Note. *β* is a standardized regression coefficient. T1 = Time one; T2 = Time two; ERN_{resid} = error-related negativity residual; CI = confidence interval. The dependent variable is anxiety symptoms at Time 2.

4. Discussion

In a group of first-year university students, we examined whether ERN magnitude at the start of the academic year interacted with interpersonal stress experienced over the year to predict symptoms of anxiety toward the end of the academic year. As hypothesized, we found evidence for an interaction, whereby, for those individuals with a larger ERN (i.e., more negative values), greater interpersonal stress exposure was significantly associated with more symptoms of anxiety toward the end of the year, even when controlling for the interaction

[2] The results of the regression were similar, and the effect size for the interaction term was in the same direction and of a similar magnitude, when including the participant excluded for reporting Time 2 anxiety scores more than three SD above the sample mean, though the interaction term was no longer a statistically significant predictor ($p = .10$).

Table 3
Results of simple slopes analyses showing slope of past-year interpersonal stress at three values of residual ERN magnitude, controlling for effects of gender, time between assessments, non-interpersonal stress experienced over the past year, and the interaction between residual ERN magnitude and past-year non-interpersonal stressors.

ERN magnitude (μV)	Effect	Standard Error	*t*	*p*	95% CI
−5.69 (large)	5.40	1.81	2.99	0.004	1.77, 9.04
0	−0.58	1.61	−0.36	0.72	−3.82, 2.66
5.69 (small)	−6.57	3.16	−2.08	0.04	−12.93, −0.21

Note. ERN magnitudes −5.69 and 5.69 represent values 1 standard deviation below and above the sample mean, respectively.

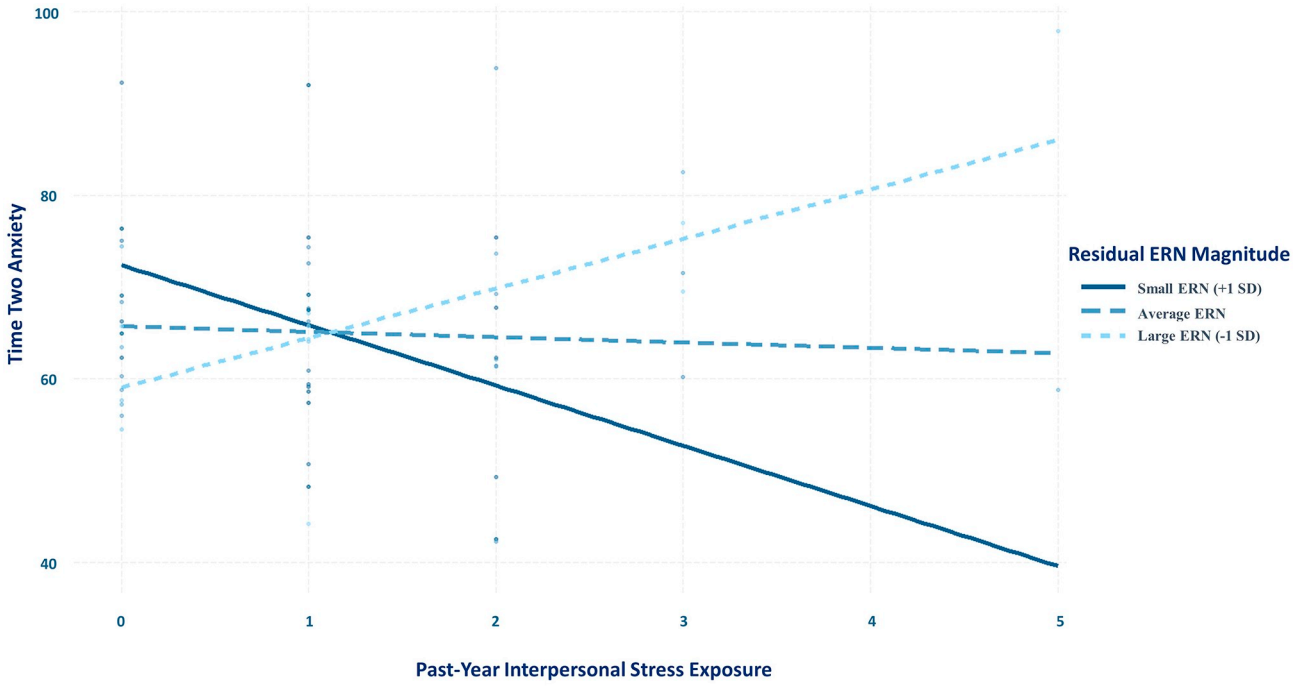

Fig. 2. Simple slopes depicting conditional effect of past-year interpersonal stress on Time 2 anxiety at large (−5.69), medium (0), and small (5.69) ERN magnitude values, controlling for Time 1 anxiety, gender, days between assessments, past-year non-interpersonal stress, and the interaction between residual ERN magnitude and past-year non-interpersonal stress.

between ERN magnitude and non-interpersonal stress, baseline anxiety symptoms, and relevant demographic factors.

These findings are consistent with research indicating that interpersonal stress is a strong predictor of heightened anxiety (Farmer and Kashdan, 2015; Hamilton et al., 2016; Kendler et al., 2003; Siegel et al., 2009; Uliaszek et al., 2010), but that experiencing interpersonal stress does not always precipitate increases in anxiety (Broeren et al., 2014; Brozina and Abela, 2006). Our results are also consistent with data suggesting that an enhanced ERN is a vulnerability marker for anxiety (Olvet and Hajcak, 2008; Riesel et al., 2011; Riesel et al., 2015), but is not itself a diagnostic marker of anxiety, as it is also seen in first-degree relatives of those with OCD who do not have the disorder (Carrasco et al., 2013; Riesel et al., 2011), is unrelated to OCD symptom severity (Riesel et al., 2014), and is observed among remitted individuals (Hajcak et al., 2008; Kujawa et al., 2016; Riesel et al., 2015). Instead, our results suggest that the *interaction* between ERN magnitude and interpersonal stress exposure might be particularly potent in predicting later anxiety symptoms – that is, the ERN may represent a latent vulnerability for anxiety that is triggered by stressful experiences (Meyer et al., 2017a).

Although interpersonal and non-interpersonal stress were both significantly associated with increased Time 2 anxiety levels, exploratory analyses revealed that non-interpersonal stress did not significantly interact with ERN magnitude to predict anxiety, suggesting that the characteristics of interpersonal stressors specifically may be particularly important to consider. Humans are motivated to perform well in social settings (Barker et al., 2018; Blascovich et al., 1999; Blascovich and Tomaka, 1996), as errors in interpersonal contexts may threaten safety or social standing (Hajcak, 2012; Lim et al., 2015). Consistent with this finding, research suggests that errors are more significant in social situations than non-interpersonal contexts: The ERN is enhanced when participants are told that their behaviour in error-eliciting tasks is being observed or evaluated (Barker et al., 2015; Buzzell et al., 2017; Hajcak et al., 2005; Kim et al., 2005; Meyer et al., 2019; Schillinger et al., 2016; Van Meel and Van Heijningen, 2010). Performance monitoring may thus be particularly important in stressful social situations relative to situations that are non-interpersonal in nature. And in fact, in our sample, participants experienced fewer interpersonal than non-interpersonal stressors, suggesting that it is the interpersonal qualities of the stressors, as opposed to the number of stressors experienced, that interacts with an enhanced ERN to predict heightened anxiety.

It is possible that individuals who exhibit an enhanced ERN are more emotionally reactive to interpersonal stressors that have a social-evaluative component, which may help to explain why an enhanced ERN interacts with interpersonal (but not non-interpersonal) stress exposure to predict anxiety levels. Indeed, some evidence suggests that individuals with social anxiety – which is associated with an enhanced ERN (Endrass et al., 2014; Kujawa et al., 2016) – are more sensitive and emotionally reactive to daily social stressors than their non-anxious counterparts (Farmer and Kashdan, 2015). Combined with our findings, these data suggest that increased performance monitoring may enhance negative affective responses to social stressors, leading to increased

anxiety over time. However, future research is needed to directly test this possibility.

Limitations of the present study suggest avenues for future research. First, although our attrition rate was comparable to those from other similar prospective studies (e.g., LeMoult et al., 2015; McLaughlin et al., 2014; Meyer et al., 2017a; Sandre et al., 2019), we lost a substantial portion of our participants between the in-lab assessment at Time 1 and the follow-up assessment at Time 2. We can only speculate on reasons for this attrition, but possible explanations include university drop-out, a lack of time to complete the Time 2 assessments, or insufficient compensation. Although participants who were lost to follow-up did not differ significantly from those who completed the follow-up session on demographic variables, baseline anxiety symptoms, or ERP values, it is nevertheless possible that our results were impacted by our low retention. It will be important to replicate the present results in a larger sample, and to prevent attrition through methods like increased participant compensation, to address these issues.

Second, participants in our sample were mostly female, and women have been found to experience more interpersonal stressors on the STRAIN (Slavich and Shields, 2018), to respond differently to social stress (Rudolph, 2002; Stroud et al., 2002; Troisi, 2001), and to experience more anxiety than men (Kessler et al., 2005; McLean et al., 2011). Our results may thus reflect the effects of the interaction between performance monitoring and social stress on anxiety mostly for women. Third, our sample was 44% Caucasian, with a median family income that is above the national median (Statistics Canada, n.d.), which may limit the generalizability of our findings. Future studies will need to examine the extent to which these effects extend to more diverse samples.

Fourth, although there is evidence that responses on the STRAIN are largely independent of participants' mood state and personality characteristics (Slavich and Shields, 2018), these factors could have nonetheless played a role here. Future studies could seek to replicate these effects using interview-based measures of interpersonal life stress (Hammen, 1991; Hammen et al., 1989). Relatedly, the interpersonal life stress variable we used included a range of stressors that possess different social-psychological characteristics (e.g., social evaluation, isolation, rejection). As a result, it is not clear if the present results are more strongly driven by some interpersonal stressors, or stressor qualities, than others (Slavich, 2019). In addition, since participants were not interviewed about the characteristics of each stressor that they experienced, it is possible that our non-interpersonal stress exposure variable could have included some interpersonal elements (e.g., a major financial problem that, at some point, triggered an interpersonal argument).

Lastly, because we used a composite measure of anxiety symptoms, our results cannot speak to the ability of the ERN and interpersonal stress to interact to predict specific symptoms of anxiety. This composite included symptoms associated with disorders that have been consistently linked to an enhanced ERN (e.g., OCD and SAD symptoms; Carrasco et al., 2013; Endrass et al., 2010; Hajcak and Simons, 2002; Weinberg et al., 2012, 2015b; Endrass et al., 2014; Kujawa et al., 2016), but also symptoms less consistently associated with a heightened ERN (e.g., trauma-related symptoms; Gorka et al., 2016; Khan et al., 2018; Lackner et al., 2018; Meyer et al., 2013; Rabinak et al., 2013; Swick et al., 2015). It is possible that certain categories of anxiety symptoms are better predicted by an interaction between ERN magnitude and interpersonal stress. Future studies looking across anxiety diagnoses in a clinical sample will be important for more fully understanding the specificity of the ERN as a predictor of later anxious dysfunction. However, prior research suggests that the ERN is a *transdiagnostic* risk marker for anxiety (Meyer, 2016; Riesel et al., 2017; Weinberg et al., 2015a), rather than a marker of specific forms of dysfunction, suggesting that a composite anxiety symptom score is appropriate to investigate our research questions.

In sum, the present results indicate that ERN magnitude at the start of the academic year interacts with past-year interpersonal (but not non-interpersonal) stress exposure to predict anxiety symptoms six months later, controlling for baseline anxiety symptoms. Specifically, experiencing more interpersonal stress was significantly related to subsequently heightened symptoms of anxiety, but only for individuals with an enhanced ERN. These findings are consistent with diathesis-stress models, whereby enhanced error monitoring renders individuals more susceptible to the negative effects of interpersonal stress, enhancing risk for heightened anxiety (Olvet and Hajcak, 2008; Riesel et al., 2011, 2015). This framework can be used by future studies to examine mechanisms through which stress may interact with the ERN to predict anxiety, with the aim of identifying individuals at risk of developing anxiety disorders.

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Declaration of competing interest

None.

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