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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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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.

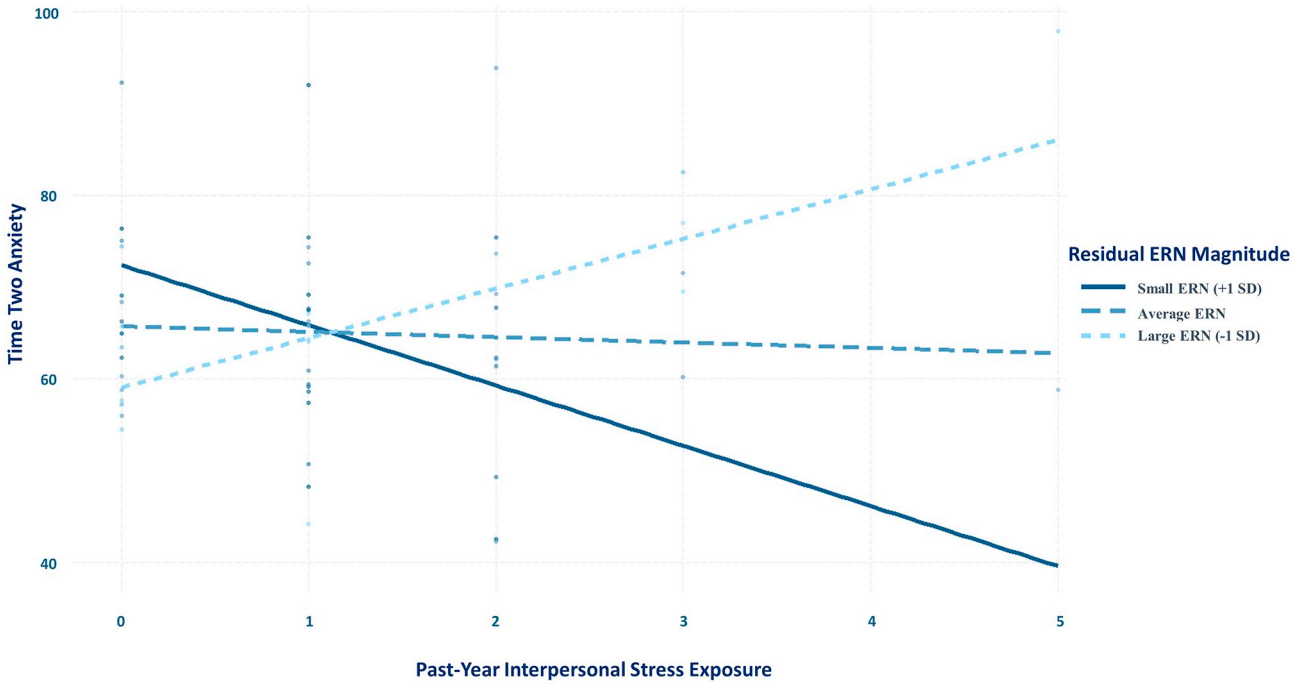

Fig. 2. Simple slopes depicting conditional effect of past-year interpersonal stress on Time 2 anxiety at large (−5.69), medium (0), and small (5.69) ERN magnitude values, controlling for Time 1 anxiety, gender, days between assessments, past-year non-interpersonal stress, and the interaction between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