Pathways to developing anxiety: Understanding the roles of life stress and neural response to errors

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July 2023

A thesis submitted to McGill University in partial fulfillment of the requirements of the degree of Doctor of Philosophy

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Abstract

Anxiety disorders are some of the most common and debilitating kinds of mental disorders worldwide. Although many treatments for anxiety exist, they are not effective for everyone, leaving it unclear how to effectively treat and prevent these disorders. Increased clarity around which individuals are most vulnerable to developing heightened anxiety, why, and under what circumstances, would allow us to develop more targeted prevention and intervention programs for anxiety disorders.

One proposed vulnerability marker for anxiety is an event-related potential (ERP) component called the error-related negativity (ERN), a neural index of performance monitoring elicited following error commission. An enhanced ERN is observed in adults and children with anxiety and related disorders ranging from obsessive-compulsive disorder to social anxiety to generalized anxiety. An enhanced ERN in childhood can prospectively predict subsequent increases in anxiety, and is observed in unaffected first-degree relatives of individuals with anxiety and in individuals in remission from anxiety. However, it is unclear how individuals develop an enhanced ERN. Some work has shown that stress (e.g., shocks, punishment) can enhance ERN magnitude, suggesting that environmental factors may alter neural indices of performance monitoring and place individuals at heightened risk for anxiety.

The present thesis aimed to better understand how stress affects and interacts with the ERN to confer for heightened anxiety. In Study One, we examined associations between ERN magnitude in adulthood and severity of different types of stressors experienced during different developmental periods. Results showed that more severe total and social-evaluative stress experienced in early adolescence was associated with enhanced error monitoring, which suggests

that early adolescence is a developmental period during which individuals may be particularly vulnerable to the effects of stress on neural markers of performance monitoring.

In Study Two, we investigated associations between ERN magnitude in adulthood and parental behaviour experienced in childhood. We found that participants who reported experiencing authoritarian/overprotective maternal behaviours displayed an enhanced ERN, which suggests that long-term exposure to parenting styles characterized by punishment and over-control may be one pathway to the development of enhanced performance monitoring. Finally, Study Three sought to examine whether the magnitude of one's ERN might interact with interpersonal stress experienced over the past year to predict anxiety symptoms. Results showed that individuals with enhanced performance monitoring at Time 1 reported heightened anxiety symptoms at Time 2 (six months later) when they experienced more severe interpersonal stressors over the past year, compared to individuals with a smaller index of performance monitoring.

This work improves our understanding of anxiety vulnerability by demonstrating that neural response to errors may play an important role in risk for heightened anxiety, and that this response can be shaped by environmental factors. Ultimately, these results may aid in early identification of individuals at risk for anxiety disorders, and the development of prevention and intervention programs.

Résumé

Les troubles d’anxiété sont parmi les troubles de santé mentale les plus communs et incapacitants dans le monde. Bien qu'il existe de nombreux traitements pour l'anxiété, ils ne sont pas adéquats pour tous et nous ignorons comment traiter et prévenir ces troubles de façon efficace. Une meilleure compréhension des personnes susceptibles à développer une anxiété élevée ainsi que les raisons pour et les circonstances entourant cette vulnérabilité permettrait de développer des meilleurs programmes de prévention et d'intervention.

L'un des marqueurs de vulnérabilité proposés pour l'anxiété s’agit de la négativité liée à l'erreur (« error-related negativity »; ERN), une composante du potentiel évoqué. L’ERN est un indice neuronal de la surveillance de la performance qui est provoqué lorsqu'une erreur est commise. Un ERN augmenté est observée chez les personnes souffrant d’anxiété ou de troubles liés à l’anxiété comme le trouble obsessionnel compulsif, l'anxiété sociale, et l'anxiété généralisée. Un ERN élevé en enfance peut prédire de manière prospective une augmentation ultérieure d'anxiété. Un ERN élevé est également observé chez les membres de la famille au premier degré non-affectée des personnes souffrant de trouble d’anxiété ainsi que chez les personnes en rémission d’un trouble d'anxiété. Cependant, il n’est pas clair comment les individus développent un ERN élevé. Certains œuvres de recherche démontrent que le stress (par exemple, les chocs électriques, la punition) peut augmenter l’ampleur de l'ERN. Ceci suggère que les facteurs environnementaux peuvent modifier les indices neuronaux de surveillance de la performance et accroître le risque d’anxiété.

Cette thèse cherche à mieux comprendre comment le stress affecte et interagit avec l'ERN pour conférer un risque accru d'anxiété. Dans la première étude, nous avons examiné les associations entre l'ampleur de l'ERN à l’âge adulte et l’intensité des différents facteurs de stress

vécus au cours de différentes périodes de développement. Les résultats ont démontré qu'un niveau plus élevé de stress global et de stress socio-évaluatif en début d'adolescence était associé à une surveillance d'erreurs de performance augmentée. Ces résultats suggèrent que le début de l'adolescence est une période développementale d'une vulnérabilité particulière pour l'impact du stress sur les indices neuronaux de la surveillance de performance.

Dans la deuxième étude, nous avons examiné les associations entre l'ampleur de l'ERN à l'âge adulte et le comportement des parents en enfance. Nous avons constaté que les participants qui ont rapporté avoir vécu des comportements maternels autoritaires et surprotecteurs présentaient un ERN accrue, ce qui suggère que l'exposition à long terme aux pratiques parentales répressives et excessivement contrôlantes pourrait être un parcours menant au développement d'une surveillance accrue de la performance. Enfin, la troisième étude visait à déterminer si l'ampleur du ERN pouvait interagir avec le stress interpersonnel vécu pendant la dernière année pour prédire les symptômes d'anxiété. Les résultats démontrent que les personnes ayant une surveillance accrue de la performance au temps 1 rapportaient des symptômes d'anxiété plus élevés au temps 2 (six mois plus tard) lorsqu'elles avaient subi des facteurs de stress interpersonnels plus intenses lors de la dernière année en comparaison avec ceux qui avaient un indice de surveillance de la performance plus faible.

L'ensemble de ces travaux améliore notre compréhension de la prédisposition pour l'anxiété en démontrant que la réponse neuronale aux erreurs pourrait jouer un rôle important dans l'augmentation du risque d'une anxiété accrue et que cette réponse pourrait être influencé par des facteurs environnementaux. Ces résultats pourraient aider avec l'identification précoce des personnes à risque accru de développer un trouble d'anxiété et l'élaboration de meilleurs programmes de prévention et d'intervention.

Acknowledgements

I am deeply grateful for the many wonderful people who helped me on my PhD journey. First, I want to thank my supervisor Dr. Anna Weinberg. Anna, your passion for research and enthusiasm for making positive change inspired me every day. Thank you for everything you have taught me, and for your guidance, encouragement, humour, and compassion. I have grown as a scientist, clinician, and human because of your mentorship, and I am so grateful to have had the opportunity to learn from you.

Next, I would like to thank my advisory committee members, Dr. Sarah Racine and Dr. David Zuroff, for their helpful feedback and suggestions on my research. Thanks also to Dr. Melanie Dirks for teaching me how to assess stress, and to the McGill Psychology faculty members who taught me throughout my graduate studies. Further, I am grateful for the support and help I received from Chantale Bousquet, Nina Pinzarrone, Sarah Lessard, and Giovanna LoCascio—thank you all. Thank you also to the clinical supervisors who guided and supported me as I learned to be a better clinician.

I want to thank Aislinn, Clara, Paige, Juhyun, Héléna, Lidia, Grace, Corinne, and Simon for making the TRAC lab feel like a second home. I'm so grateful that I got to work with all of you. To the Honours students, research assistants, and lab coordinators who dedicated their time to collecting the data in these studies: Thank you so much! I'm so appreciative of your hard work. Thank you also to everyone who participated in my lab's studies – your time and effort made this research possible.

I wish to thank the grad students in the McGill Psychology Department for creating a wonderful community. To Catherine, Ally, Erika, Amanda, and Serena: You have been the most supportive cohort that anyone could ask for. Thank you for everything. A special thank you to

Aislinn and Catherine, who supported me and made me laugh every step of the way. I could not have done this without you, and I am so grateful for your friendships. Thank you also to my family for their encouragement and support. Lastly, thank you, Jyota, for taking care of me and always being on my team.

Contribution to Original Knowledge

The work presented here contributes in several ways to the literature on neural indices of susceptibility to stress and anxiety. First, Study One assessed associations between neural response to errors and distinct categories of life stress, as well as cumulative stress, experienced during different developmental periods, to identify factors that may lead to enhanced performance monitoring in young adulthood. Study Two further investigated this topic by assessing associations between punitive parenting experienced in childhood and adolescence and neural response to errors in young adulthood, to seek further support for the idea that social stress and punishment are associated with enhanced performance monitoring. Finally, Study Three investigated whether an enhanced neural index of performance monitoring may be a diathesis that interacts with interpersonal stress experiences to heighten risk for anxiety disorders, to elucidate one potential pathway to the development of anxiety.

Study One demonstrated that associations between neural response to errors and life stress differ as a function of the developmental timing and type of stress experienced. Specifically, we found that more severe total and social-evaluative stress experienced in early adolescence was associated with enhanced error monitoring, while life-threatening stress severity was not. This suggests that early adolescence is a developmental period during which individuals may be particularly vulnerable to the effects of stress on neural markers of performance monitoring, compared to childhood and later adolescence. Further, we hypothesize that social-evaluative stress may have more of an impact on neural functioning than other kinds of stress (e.g., physically dangerous stressors), and that these effects can last beyond adolescence and into young adulthood. Together, these findings suggest that research would benefit from considering the type and timing of stressors when investigating their effects on performance monitoring.

Study Two replicated previously-reported effects of punitive parenting on neural response to errors, but further demonstrated than these effects last beyond childhood and into young adulthood. Specifically, we found that young adults who reported experiencing authoritarian and overprotective maternal behaviours up until the age of 16 displayed an enhanced neural response to errors. Our findings suggests that long-term exposure to parenting styles characterized by punishment and over-control may be one pathway to the development of enhanced performance monitoring, which contributes to our understanding of environmental factors that may lead to heightened risk for anxiety disorders.

Study Three investigated whether the magnitude of one's neural response to errors may interact with interpersonal stress experienced over the past year to predict anxiety symptoms. We used a longitudinal design that allowed us to measure participants' anxiety symptoms and neural response to errors at the start of their first academic term in university (Time 1), as well as past-year interpersonal stress and anxiety symptoms six months later (Time 2). Results showed that individuals with enhanced performance monitoring at Time 1 reported heightened anxiety symptoms at Time 2 when they experienced more severe interpersonal stressors over the past year—even when controlling for anxiety reported at Time 1—compared to individuals with a smaller index of performance monitoring. Importantly, we showed that this effect is specific to interpersonal stressors—cumulative stress did *not* interact with performance monitoring to predict anxiety. This advances our understanding of risk for anxiety disorders by indicating that heightened neural response to errors may be a diathesis which renders individuals more susceptible to developing heightened anxiety following interpersonal stress.

Overall, this work suggests that neural indices of performance monitoring—specifically, the neural response to errors—may play an important role in risk for anxiety disorders, and that

this response can be shaped by environmental factors, most notably social-evaluative stress. These findings contribute to the body of work that seeks to use neural markers of performance monitoring to identify those at risk for anxiety before they develop the illness, and create new interventions aimed at decreasing anxiety symptoms. For instance, these findings may be used to identify children at risk for anxiety disorders to guide prevention efforts (e.g., interventions aimed at changing highly punitive parenting styles), or provide interventions for modulating the neural response to errors to treat anxiety. Ultimately, such approaches may improve our attempts at precision interventions targeted to people with specific vulnerabilities, thereby improving anxiety interventions.

Contribution of Authors

This thesis comprises three original manuscripts detailing a portion of my (IB) doctoral research, conducted under the supervision of Dr. Anna Weinberg (AW). Below are citations for the manuscripts, as well as a description of the contribution of the authors involved.

Study One

Banica, I., Sandre, A., Shields, G.S., Slavich, G.M., & Weinberg, A. (2021). Associations between lifetime stress exposure and the error-related negativity (ERN) differ based on stressor characteristics and exposure timing in young adults. *Cognitive, Affective, & Behavioral Neuroscience*, 1–18. https://doi.org/10.3758/s13415-021-00883-z

AW conceptualized the design for the longitudinal study from which the data for this manuscript were drawn. IB and AW developed the study design included in the manuscript. IB and AS completed data collection, and IB conducted data analysis, under the supervision of AW. IB, AS, and AW contributed to result interpretation. IB conducted the literature review and drafted the manuscript. AS and AW edited the manuscript. All authors provided revisions and approved the final manuscript for submission.

Study Two

Banica, I., Sandre, A. & Weinberg, A. (2019). Overprotective/authoritarian maternal parenting is associated with an enhanced error-related negativity (ERN) in young adult females. *International Journal of Psychophysiology, 137*, 12–20. https://doi.org/10.1016/j.ijpsycho.2018.12.013

AW conceptualized the overall study concept, and AW and IB developed the study design detailed in the manuscript. AS completed data collection under the supervision of AW. IB completed data analysis, under the supervision of AW, and conducted the literature review. IB

drafted the manuscript. AW and AS edited the manuscript, supplied revisions, and approved the final manuscript for submission.

Study Three

Banica, I., Sandre, A., Shields, G.S., Slavich, G.M., & Weinberg, A. (2020). The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later. *International Journal of Psychophysiology, 153,* 27–36. https://doi.org/10.1016/j.ijpsycho.2020.03.006

AW conceptualized the design for the longitudinal study from which the data for this manuscript were drawn. IB and AS developed the study design detailed in the manuscript, completed data collection, and conducted data analyses, under the supervision of AW. IB conducted the literature review and drafted the manuscript. AS and AW edited the manuscript. All authors provided revisions and gave approval for the final manuscript for submission.

Introduction

Anxiety disorders are among the most common, persistent, and debilitating mental illnesses in the world (Louise et al., 2017; Polancyzk et al., 2015). In 2015, it was estimated that around 264 million people globally were experiencing an anxiety disorder (World Health Organization, 2017). Anxiety disorders strain health care systems (Dobson et al., 2020; Kessler & Greenberg, 2002), and are associated with distress and impaired functioning (Chistolm et al., 2016), making it critical to better understand, treat, and prevent them. Although much progress has been made toward elucidating factors that may contribute to the development and maintenance of anxiety disorders (e.g., Batelaan et al., 2010; Blanco et al., 2014; Jacobson & Newman, 2017; Moreno-Peral et al., 2014; Pahl et al., 2012), we are still unable to accurately predict who is most at risk of developing anxiety. For instance, although stress is a well-established risk factor for anxiety (Faravelli, 1985; Faravelli, & Pallanti, 1989; Finley-Jones & Brown, 1981; Green et al., 2010; Hankin et al., 2004; Young & Dietrich, 2015), not everyone who experiences stress will go on to develop an anxiety disorder (Ingram & Luxton, 2005; Harkness, Hayden, & Lopez-Duran, 2015; Harkness & Monroe, 2016), suggesting a critical need to better identify who is most vulnerable to stress exposure, and why.

Being better able to predict risk would allow for early intervention and more targeted treatments for anxiety. One proposed risk marker for anxiety is an event-related potential (ERP) component called the error-related negativity (ERN; Falkenstein et al., 1991; Gehring et al., 1993). The ERN is a neural marker of performance monitoring that is enhanced in individuals with a variety of anxiety and related disorders (e.g., obsessive-compulsive disorder, generalized anxiety disorder; Endrass et al., 2010; Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002; Weinberg et al., 2010, 2012) and high trait anxiety (Hajcak, McDonald, & Simons, 2003a;

Olvet & Hajcak, 2008). Because the ERN is partially heritable (Anokhin, Golosheykin, & Heath, 2008) and can prospectively predict the development of anxiety (Lahat et al., 2014; McDermott et al., 2009; Meyer et al., 2015b, 2017a), it has been proposed as a neural marker of risk for anxiety disorders (Olvet & Hajcak, 2008; Riesel et al., 2011, 2015).

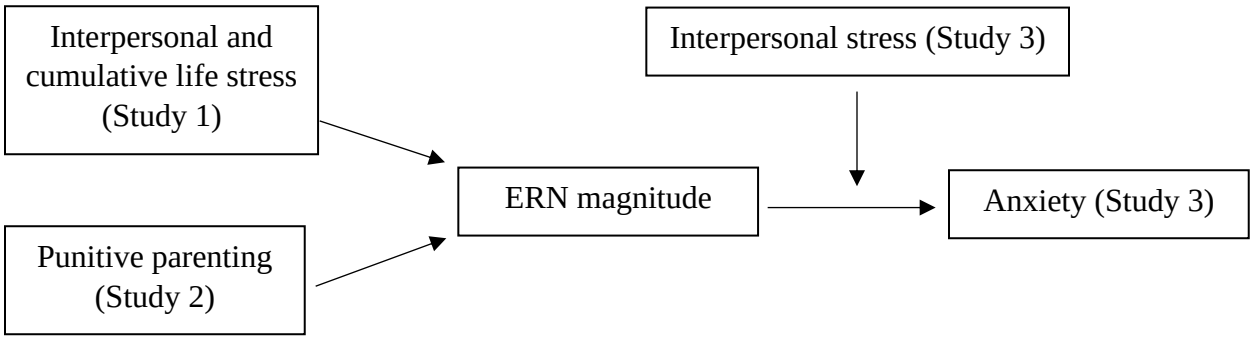
There appear to be multiple sources of variation in ERN magnitude. While genes account for approximately 50% of variation in the ERN (Anokhin, Golosheykin, & Heath, 2008), environmental factors, such as life stress, are also associated with altered performance monitoring (e.g., Brooker & Buss, 2014; Khan et al., 2018; Riesel et al., 2012). For instance, the ERN is enhanced when errors are punished by shocks and noxious sounds (Meyer & Gawlowska, 2017; Riesel et al., 2012, 2019a). Social-evaluative stress is also associated with enhanced performance monitoring; for instance, a larger ERN is observed when individuals are told that their task performance is being observed by peers, experimentors, and parents (e.g., Buzzell et al., 2017b; Kim et al., 2005; Meyer et al., 2019; Hajcak et al., 2005), and when they report having experienced punitive parenting (e.g., Brooker & Buss, 2014b; Meyer et al., 2015). Life stress—and punishment experiences in particularly—may thus lead to enhanced performance monitoring, although more work is needed to better understand these links.

There is also limited evidence to suggest that stress can *interact* with ERN magnitude to predict later psychopathological symptoms (Meyer et al., 2017). It is thus possible that the ERN is a diathesis that is altered by, and interacts with, life stress, to heighten risk for anxiety. However, this possibility has not been thoroughly investigated. In what follows, I present three studies investigating these ideas, with the aim of elucidating psychophysiological pathways to anxiety disorder risk. As depicted in Figure 1, the primary objectives of my doctoral research were to examine:

1) Associations between life stress and ERN magnitude, focusing on differential effects of *types* of stressors during different *developmental periods* on ERN magnitude, as well as long-term effects of stress on the ERN;
2) Whether stress can interact with ERN magnitude to prospectively predict anxiety symptoms.

Figure 1.

Overall model examined by the studies presented in this dissertation.



In the first study presented in this dissertation (Banica et al., 2021), my co-authors and I examined associations between adult ERN magnitude and distinct (life-threatening and social-evaluative) and cumulative (total) stressors experienced during different developmental periods. In Study Two (Banica et al., 2019), we expanded on these findings by investigating associations between the experience of punitive parental behaviour during childhood and ERN magnitude in young adulthood. Finally, in Study Three (Banica et al., 2020), we investigated whether ERN magnitude moderates the association between past-year interpersonal stress and anxiety symptoms six months later. I will begin with a literature review on the ERN and its associations with anxiety disorders and life stress.

Literature Review

Anxiety Disorders

Anxiety disorders are a leading cause of disability worldwide (Vos et al., 2017; World Health Organization, 2017). Up to 33% of the global population is estimated to experience an anxiety disorder during their lifetime (Bandelow & Michaelis, 2015), and in Canada, around 4.6% of the population develops an anxiety disorder each year (Dobson et al., 2020). The prevalence of anxiety has increased over the last 20 years (Dobson et al., 2020) and since the start of the COVID-19 pandemic (Dozois, 2020), making it an increasingly pressing public health concern. Anxiety disorders are associated with high health care costs and indirect financial losses due to lost productivity (Chistolm et al., 2016; Dobson et al., 2020; Hohls et al., 2018; Kessler & Greenberg, 2002; Konnopka & Konig, 2019), cause immense human suffering and decreased quality of life (Barrera & Norton, 2010; Hohls et al., 2021; Olatunji, Cisler, & Tolin, 2007), and are associated with heightened risk for physical illnesses (e.g., cardiovascular disease; Garfield et al., 2014; Nabi et al., 2010; Scott et al., 2013; Tully et al., 2016). Further, anxiety can cause functional impairment in multiple life domains (McKnight et al., 2016), including familial and social relationships (Hauschildt et al., 2010; Lochner et al., 2003), physical functioning (Olatunji et al., 2007; Romera et al., 2011), and occupational performance (McKnight et al., 2016). Individuals with anxiety are more likely to be unemployed, less likely to be able to work, and report lower incomes than the general population (Patel et al., 2002).

The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines multiple anxiety disorders (American Psychiatric Association, 2015). While these disorders share the core features of excessive anxiety and fear and associated maladaptive behaviours, they also differ from one another in several ways. For instance, social anxiety disorder (SAD) is characterized

mostly by fear and avoidance of social interactions involving the possibility of judgment, while the key features of specific phobia are fear and avoidance of specific situations or objects (e.g., heights, spiders), and generalized anxiety disorder (GAD) is characterized by excessive worry about multiple domains that is experienced as uncontrollable and is accompanied by physical symptoms (American Psychiatric Association, 2013). A diagnosis closely related to, and often comorbid with, anxiety disorders is obsessive-compulsive disorder (OCD), an illness characterized by intrusive obsessional thinking and repetitive behavioural or cognitive compulsions (American Psychiatric Association, 2013). Previously considered an anxiety disorder in the DSM-IV, OCD was moved to a new section of the DSM-5 called "Obsessive-compulsive and related disorders" (OCRD) in an effort to group together mental disorders characterized by obsessions and repetitive behaviours (Abramowitz & Jacoby, 2014; American Psychiatric Association, 2013). However, some research suggests that OCD is more phenomenologically similar to anxiety disorders than other OCRDs (Abramowitz & Deacon, 2005; Abramowitz & Jacoby, 2014; Storch et al., 2008), and—importantly for the present thesis—that there are shared neural markers of risk for both OCD and anxiety disorders (Meyer, 2016; Olvet & Hajcak, 2008; Riesel et al., 2011; Riesel et al., 2015). This thesis will thus review relevant literature on both anxiety disorders and obsessive-compulsive disorder.

The onset of many anxiety disorders, such as specific phobias and social anxiety, is typically in childhood and adolescence. However, agoraphobia, panic disorder, and OCD tend to develop around early adulthood, and generalized anxiety disorder often starts around the age of 30 (American Psychiatric Association, 2013; Bandelow & Michaelis, 2015). Anxiety tends to follow a chronic course, with prevalence rates generally peaking between 18 and 35 years of age and decreasing between 50 and 65 years of age (Bandelow & Michaelis, 2015). Further, anxiety

disorders tend to be highly co-morbid with other anxiety diagnoses and forms of psychopathology (Bandelow & Michaelis, 2015). Given the immense human suffering and societal burden caused by anxiety disorders, and their chronicity and frequent comorbidity with other impairing conditions, it is critical to better understand and treat them.

Many treatments for anxiety are available, including medications (e.g., benzodiazepines, selective serotonin reuptake inhibitors) and psychotherapy (e.g., cognitive behavioural therapy). While such interventions are effective for many people, they fail for many others (Ali et al., 2017; Bandelow et al., 2015; Batelaan et al., 2017; Leichsenring et al., 2014; Levy, O'Bryan, & Tolin, 2021; Perna et al., 2016; Van Dis et al., 2020), which has prompted researchers and clinicians to focus on anxiety prevention programs. Some work suggests that such programs produce medium to large reductions in anxiety symptoms (Barrett et al., 2005, 2006; Lowry-Webster et al., 2003; Martinsen et al., 2019; Mychailyszyn et al., 2012; Topper et al., 2017), while others demonstrate small or no effects (Ahlen, Lenhard, & Ghaderi, 2015; Johnstone et al., 2018; Lau & Rapee, 2011; Miller, 2008; Stallard et al., 2007), or only short-term reduction in symptoms (Feiss et al., 2019), making it unclear whether they successfully promote significant long-term alleviation of anxiety.

A major obstacle to our ability to effectively prevent and treat anxiety disorders is a limited understanding of factors that place individuals at risk of developing these disorders, and how these factors confer risk. While anxiety is partially heritable (Franić et al., 2016; Guffanti et al., 2016; Purves et al., 2020), environmental factors also play a strong role in anxiety disorder etiology and maintenance. Environmental risk factors include having a parental history of mental illness, especially mood and anxiety disorders; childhood behavioural inhibition; smoking and substance use; and sociodemographic variables such as age, female sex and gender, and low

socioeconomic status (Batelaan et al., 2010; Blanco et al., 2014; Jacobson & Newman, 2017; Moreno-Peral et al., 2014; Pahl et al., 2012; Zimmerman et al., 2020). Further, anxiety is often preceded by episodes of life stress (Faravelli, 1985; Faravelli, & Pallanti, 1989; Finley-Jones & Brown, 1981; Green et al., 2010; Hankin et al., 2004; Young & Dietrich, 2015). Interpersonal stressors such as entrapment, humiliation, and peer victimization are particularly salient in predicting symptoms (Farmer & Kashdan, 2015; Hamilton et al., 2016; Kendler et al., 2003; Schneider et al., 2021; Siegel et al., 2009; Uliaszek et al., 2010). However, consistent with diathesis-stress models of psychopathology, not everyone with these risk factors or who experiences life stress goes on to develop anxiety (Ingram & Luxton, 2005; Harkness, Hayden, & Lopez-Duran, 2015; Harkness & Monroe, 2016), and individuals with different combinations of these risk factors can develop anxiety disorders (i.e., equifinality).

This problem of equifinality and lack of predictive ability further complicates our efforts to identify individuals at risk of developing anxiety, and is exacerbated by the fact that anxiety disorders are often co-morbid with other mental illnesses, most notably depression. It is estimated that between 47% and 88% of individuals experiencing anxiety have also had a depressive disorder (Adams et al., 2016; Belzer & Schneier, 2004; Craske et al., 2011; Cummings et al., 2015; de Graaf et al., 2003; Hek et al., 2011; Jacobson & Newman, 2017; Kessler et al., 1999; Melton et al., 2016; Mineka et al., 1998). Additionally, there is substantial overlap between characteristics of anxiety and depression, such as negative affect (Brown et al., 1998; Clark & Watson, 1991), distress (Craske et al., 2011), fatigue, and difficulties concentrating (American Psychiatric Association, 2013). The frequent co-morbidity and symptom overlap between these disorders makes it difficult to elucidate specific pathways of risk for anxiety. This, in turn, prevents a better understanding of anxiety and related disorders and

hinders our ability to identify vulnerable individuals early on and develop more effective and targeted prevention and treatment efforts (Insel et al., 2010). Frequent symptom overlap has prompted a shift towards conceptualizing mental illness in terms of continuous dimensions of symptoms and impairment rather than diagnostic categories (Craske et al., 2011; Cuthbert & Insel, 2013; Insel et al., 2010; Sanislow et al., 2010).

To this end, the National Institute of Mental Health's Research Domain Criteria (RDoC) framework encourages researchers to improve our understanding of psychopathology by investigating basic functioning in biological, psychological, and behavioural systems, such as neural circuits involved in performance monitoring (National Institute of Mental Health, 2022). This approach aims to identify how abnormalities in specific neurobiological systems may underlie transdiagnostic psychopathological symptoms in an effort to develop and provide more precise mental health interventions to alleviate illness (Cuthbert & Insel, 2013). One promising method to investigate neural circuits underlying maladaptive behaviour and cognitive and emotional processing is electroencephalography (EEG), which allows us to measure event-related potentials (ERPs). The ERP waveform shows changes over time in voltage recorded at the level of the scalp, which reflects neural processes that occur in response to specific stimuli (Luck & Kappeman, 2012). ERP components reflect electrical brain activity that occurs due to a specific sensory, motor, or cognitive event (e.g., making an error), and reflect the summation of postsynaptic potentials generated mostly by pyramidal cells in the cortex (Luck, 2014; Luck & Kappeman, 2012).

A Neural Index of Performance Monitoring: The Error-related Negativity (ERN)

Enhanced neural indices of performance monitoring have been associated with anxiety disorders and greater risk for anxiety (e.g., Gehring, Himle, & Nisenson, 2000; Hajcak,

McDonald, & Simons, 2003a; Hajcak & Simons, 2002; Meyer et al., 2012; Weinberg, Klein, & Hajcak, 2012; Xiao et al., 2011). Specifically, an ERP component called the error-related negativity (ERN) has been proposed as a biomarker of risk for anxiety disorders and OCD (Olvet & Hajcak, 2008; Riesel et al., 2011, 2015). The ERN is a negative deflection in the ERP waveform that occurs following error commission across multiple laboratory tasks. It is maximal at frontocentral electrode sites on the scalp, and 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 demonstrates good test-retest reliability and internal consistency in both adults (Larson et al., 2010; Olvet & Hajcak, 2009; Riesel et al., 2013; Sandre et al., 2020; Weinberg & Hajcak, 2011) and children and teenagers (Meyer et al., 2014; Segalowitz et al., 2010) for up to multiple years, indicating that it is a relatively trait-like, reliable, and stable neural response (Weinberg et al., 2015).

The ERN 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 & van Veen, 2007; Dehaene, 2018; Holroyd & Coles, 2002; Lo, 2018). This is a crucial function, as monitoring our actions and learning from mistakes is vital for adapting to our demanding environments (Botvinick et al., 2001; Falkenstein et al., 2000; Holroyd & Coles, 2002; van Veen & Carter, 2002). The ERN arises so quickly after an erroneous response is made, and in the absence of external performance feedback, that is appears to reflect an endogenous process that spontaneously detects errors without needing sensory input (Dehaene, 2018; Dehaene et al., 1994; Gehring et al., 1993). The ERN is observed when participants are both aware and unaware that they have committed an error (Endrass et al., 2005, 2007; Hughes & Yeung, 2011; Nieuwenhuis et al., 2001; O'Connell

et al., 2007; Shalgi et al., 2009; Wang et al., 2020), suggesting that it can be elicited in the absence of conscious knowledge, although some studies report a smaller ERN following unperceived errors (Maier et al., 2008; Navarro-Cebrian et al., 2013; Wessel et al., 2011). Some work suggests that the ERN is associated with oscillatory activity in the mid-frontal theta band (4-8 Hz; Cavanagh et al., 2009; Debener et al., 2005; Luu & Tucker, 2001; Luu et al., 2004; Navarro-Cebrian et al., 2013; Trujillo & Allen, 2007), which is related to action monitoring, cognitive control, memory, and learning (Cavanagh et al., 2010, 2013; Cavanagh & Frank, 2014; Cavanagh & Shackman, 2015; Cooper et al., 2019).

Neural Generators of the ERN

A wealth of evidence suggests that the anterior cingulate cortex (ACC) generates the ERN (Gehring et al., 2012). The ACC is a hub in the medial frontal cortex that is made up of several subdivisions involved in crucial functions such as motor control and cognitive, emotional, and social processing (Bush et al., 2002; Heilbronner & Hayden, 2016; Holroyd & Umemoto, 2016; Kolling et al., 2016; Lavin et al., 2013; Paus, 2001; Shenhav et al., 2013, 2016). Many source localization studies suggest that the ERN is generated by the ACC (Bediou et al., 2012; Dehaene et al., 1994; Donamayor et al., 2011; Holroyd et al., 1998; Ibanez et al., 2012; Ladouceur et al., 2006, 2007; van Veen & Carter, 2002), as do studies using intracerebral EEG recording (Brazdil et al., 2005; Jung et al., 2010), and data collected in patients with ACC lesions (Stemmer et al., 2004). Some studies have localized the ERN signal to one ACC subdivision, the dorsal ACC (dACC; Brazdil et al., 2005; Hirsh & Inzlicht, 2010), which is involved in several key cognitive and motor control functions including performance monitoring, reward processing, and motor response selection (Badgaiyan & Posner, 1998; Braver et al., 2001; Bush et al., 2002; Garavan et al., 2002, 2003; Gehring & Knight, 2000; Turken & Switck, 1999). The dACC is

theorized to act with a network of other neural regions to integrate information—such as how much cognitive control and effort is needed to obtain the pay-off of an action—to allocate cognitive control resources and guide our behaviour in accordance with our goals (Shenhav, Cohen, & Botvinick, 2016). Additional studies suggest that other regions of the ACC, such as the rostral, posterior, and caudal ACC, are also involved in error processing (Ullsperger & von Cramon, 2001, 2004) and ERN signal generation (Brazdil et al., 2005; Buzzell et al., 2017a). The pre-supplementary motor area (pre-SMA), a region in the frontal cortex involved in movement control (Nachev et al., 2008; Spieser et al., 2015), has also been identified as a possible generator of the ERN (Brazdil et al., 2002, 2005; Hermann et al., 2004; Gehring et al., 2012; Grützmann et al., 2022; Rollnik et al., 2004).

The ACC has dense interconnections to both limbic and prefrontal areas (Bush et al., 2000; Heilbronner & Hayden, 2016), such as the prefrontal cortex (PFC; Barbas & Pandya, 1989; Kondo, Osaka, & Osaka, 2004; Paus, 2001; Silton et al., 2010), amygdala (Barbas & de Olmos, 1990; Kober et al, 2008; Marusak et al., 2016), and insula (Morecraft & Van Hoesen, 1998), which are also activated during error commission (Brazdil et al., 2002; Garavan et al., 2002; Kiehl et al., 2000; Menon et al., 2001; Polli et al., 2009; Pourtois et al., 2010). In line with this, input from frontal cortical areas appears to be crucial in modulating the ERN signal (Gehring et al., 2012). For instance, lesion studies suggest that damage to the prefrontal cortex is associated with an attenuated ERN magnitude (Gehring & Knight, 2000; Hogan et al., 2006; Ullsperger & von Cramon, 2006; Ullsperger et al., 2002).

Functional Significance of the ERN

There are several theories of the functional significance of the ERN signal. The first hypothesis to emerge is the error-detection (or mismatch) theory, which posits that the ERN is

the output of a system that contrasts behavioural output with an estimate of what the correct response should be (Falkenstein et al., 1991; Gehring et al., 1993). According to this view, a neural process compares a representation of the ongoing behavioural response with a representation of the correct response in a given task and computes the difference between the two (Gehring et al., 2012); if there is a mismatch between these two responses, an error signal (i.e., the ERN) is generated. However, several researchers have raised the concern that, if the error-detection theory is accurate, the brain would need to have access to what the "correct" response is in every situation, leaving it unclear why the brain is executing an incorrect response instead of the correct one (Carter et al., 1998). Further, correct responses elicit a small negativity in the ERP waveform called the "correct response negativity" (CRN; Vidal et al., 2003), and a negativity similar to the ERN and CRN is elicited on task trials without a correct or erroneous response (Sandre & Weinberg, 2019), which is not congruent with the error-detection theory.

These concerns contributed to the development of the conflict monitoring theory, which posits that the ERN signal is elicited when multiple behavioural response options are in competition with one another in a given task. When behavioural conflict is high, the ERN is theorized to alert cognitive regions involved in executive control that increased attention to task-relevant stimuli, decreased attention to distracting information, and heightened control over behaviour is required to improve task performance (Botvinick et al., 2001; Carter et al., 1998; Lo, 2018; Yeung et al., 2004). The conflict monitoring theory can be modelled computationally, and successfully models performance on various tasks (Botvinick et al., 2001; Yeung et al., 2004). However, some have argued that many of the effects observed in studies providing support for this theory can be explained by contingency learning or memory biases that do not

necessarily involve conflict (e.g., Bugg, 2015; Bugg et al., 2011; Schmidt, 2013, 2019), leaving it unclear whether this model appropriately explains error monitoring.

The reinforcement learning theory (RL-ERN) is another computational model of the ERN. This theory suggests that a mesencephalic dopamine system generates the ERN through withdrawal of dopamine activity when an anticipated outcome is worse than initially expected (Holroyd & Coles, 2002, 2008; Holroyd et al., 2005; Shahnazian & Holroyd, 2018). The mesencephalic dopamine system consists of nuclei projecting from the basal ganglia and cortex, including the ACC, and is believed to be involved in reinforcement learning (Schultz, 1997, 2010; Schultz, Dayan, & Montague, 1997), the process by which actions followed by positive feelings and consequences are more likely to be repeated, while actions followed by negative feelings and consequences are less likely to be repeated (Sutton & Barto, 1998; Thorndike, 1970). The RL-ERN theory posits that the basal ganglia use information gained from past experience to compute the value of ongoing events, and release a signal when events are worse than anticipated in the form of reduced mesencephalic dopaminergic input to the ACC; this reduction disinhibits neuronal activity in the ACC, which generates the ERN. The ACC then chooses which of the relevant neural regions involved in motor activity should be given control to optimize task performance (Holroyd & Coles, 2002, 2008). In support of this theory, de Bruijn et al. (2004) showed that administering a dopamine agonist led to enhanced ERN magnitudes.

The ERN appears to be involved in the *evaluation* of errors, not necessarily in *adjustment of behaviour* following errors, given the mixed findings on associations between the ERN and strategic behavioural adjustments following error commission (Cavanaugh & Shackman, 2015; Gehring et al., 2012; Weinberg et al., 2012, 2016). For instance, some studies have found that increased post-error slowing—the amount of time one slows down on the trial following an

error, presumably to improve accuracy (Botvinick et al., 2001; Rabbit, 1966)—is associated with a larger ERN (Beatty et al., 2020; Carrasco et al., 2013a; Debner et al., 2005; Gehring et al., 1993; Ladouceur et al., 2007), while others have not (Banica et al., 2019; Carrasco et al., 2013b; Coleman et al., 2015; Dudschig & Jentzsch, 2009; Endrass et al., 2007; Gehring & Fencsik, 2001; Hajcak et al., 2003b; Weinberg et al., 2012). Similarly, an enhanced ERN has been linked to better task performance in some studies (Hajcak et al., 2003b; Holroyd & Coles, 2002; Ladouceur et al., 2007; Pieters et al., 2007; Renn & Cote, 2013; Santesso et al., 2005; Suchan et al., 2019; Themanson et al., 2012), but not others (Banica et al., 2019; Carrasco et al., 2013a, 2013b; Masaki et al., 2007). These inconsistent associations between behavioural adjustment and ERN magnitude suggest that the ERN does not only reflect individual differences in control over task performance.

Variation in ERN magnitude

ERN magnitude appears to be influenced by several factors, such as individual differences in personality traits, cognition, and affective states. For instance, larger ERN amplitude is related to greater working memory (Miller et al., 2012), attention, and executive functioning capacity (Larson & Clayson, 2010), as well as better impulse control (Amodio et al., 2008a) and academic performance (Hirsh & Inzlicht, 2010). In terms of personality characteristics, an enhanced ERN is associated with greater childhood fearfulness (Brooker & Buss, 2014a; Lahat et al., 2014; McDermott et al., 2009; Meyer et al., 2015a, 2018), increased behavioural inhibition (Amodio et al., 2008b; Boksem et al., 2006), higher perfectionism (Meyer & Wissemann, 2020; Perrone-McGovern et al., 2017; Schrijvers et al., 2010; Stahl et al., 2015), lower propensity for risk and impulsivity (Santesso & Segalowitz, 2009; Taylor et al., 2018), higher levels of negative emotionality (Hajcak et al., 2004; Luu et al., 2000), and increased

empathy (Santesso & Segalowitz, 2009). Further, sleep deprivation (Hsieh et al., 2007; Tsai et al., 2005) and alcohol consumption (Bartholow et al., 2012; Easdon et al., 2005; Ridderinkhof et al., 2002) are both associated with smaller ERN amplitude, while negative affect and sad mood inductions appear to increase ERN amplitude (Olvet & Hajcak, 2011; Wiswede et al., 2009).

Similarly, factors that increase the salience of mistakes—such as when errors are punished with monetary loss and correct responses are rewarded (Chiu & Deldin, 2007; Ganushchak & Schiller, 2008; Groom et al., 2013; Hajcak et al., 2005), when participants are told to prioritize response accuracy over speed (Gehring et al., 1993), when errors are punished (Meyer and Gawlowska, 2017; Riesel et al., 2012, 2019a), and when task performance is evaluated (Hajcak et al., 2005; Kim et al., 2005)—appear to increase ERN magnitude. Taken together, this work suggests that while the ERN signal is present across participants, its magnitude is modulated by several factors that affect one's evaluation of errors, including situational, emotional, and personality characteristics. Thus, it is theorized that individual variation in ERN magnitude may reflect the degree to which one finds errors threatening or aversive (Meyer & Hajcak, 2019; Weinberg et al., 2012, 2015, 2016). Consistent with this idea, the physiological reaction to error commission is similar to reactions following other kinds of threats, involving increased startle (Hajcak & Foti, 2008), a skin conductance response (Hajcak et al., 2003), heart rate deceleration (Hajcak et al., 2003), and frowning (Lindström et al., 2013).

The ERN, Anxiety Disorders, and OCD

Heightened concern with errors is often observed in individuals with anxiety disorders and OCD (Campbell & Brown, 2002; Proudfit et al., 2013), who display increased performance monitoring compared to individuals without anxiety (Michael et al., 2021). The association between OCD and enhanced ERN magnitude is observed so frequently in adults (Agam et al.,

2014; Endrass et al., 2010; Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002; Klawohn et al., 2014, 2016; Mathews et al., 2016; Nawani et al., 2018; Nieuwenhuis et al., 2005; Riesel et al., 2011, 2014; Roh et al., 2016, 2017; Santamaria-Garcia et al., 2018; Weinberg et al., 2015) and children (Carrasco et al., 2013a, 2013b; Hajcak et al., 2008; Hanna et al., 2012, 2016, 2018; Liu et al., 2014) that it has been proposed as an endophenotype for OCD (Riesel, 2019). An enhanced ERN is also observed in individuals with subclinical OCD symptoms (Gründler et al., 2009; Hajcak & Simons, 2002; Kaczkurkin, 2013; O'Toole et al., 2012; Riesel et al., 2015; Santesso et al., 2006a; Zambrano-Vazquez & Allen, 2014) and unaffected first-degree relatives of individuals with OCD (Carrasco et al., 2013; Riesel et al., 2011, 2019).

Further, an enhanced ERN is observed in adults (Weinberg et al., 2010, 2012, 2015; Xiao et al., 2011) and children (Hanna et al., 2020) with GAD, as well as adults (Endrass et al., 2014) and children (Kujawa et al., 2016) with SAD. One study also reported an enhanced ERN in individuals with panic disorder (Valt et al., 2018). Further, ERN magnitude is larger in children (Chong & Meyer, 2019; Ladouceur et al., 2006; Meyer et al., 2013, 2016) and adults (Lee et al., 2020; Weinberg et al., 2010) experiencing a mix of anxiety disorders and symptoms, suggesting that it may be a transdiagnostic marker for anxiety disorders and OCD. More generally, adults and children who report high trait anxiety also display a larger ERN amplitude compared to individuals low in trait anxiety (Hajcak, McDonald, & Simons, 2003a; Meyer et al., 2012, 2016; Moser et al., 2013; Olvet & Hajcak, 2008). Importantly, an enhanced ERN in childhood and adolescence can prospectively predict increases in anxiety years later (Filippi et al., 2021; Lahat et al., 2014; McDermott et al., 2009; Meyer, 2017; Meyer et al., 2015b, 2017a; 2018, 2021), and appears to interact with cognitive control to predict anxiety symptoms during major life stressors such as the COVID-19 pandemic (Morales et al., 2022). Further, an enhanced ERN magnitude is

observed in unaffected first-degree relatives of individuals with anxiety disorders (Riesel et al., 2019b) and in individuals in remission from anxiety (Hajcak et al., 2008; Kujawa et al., 2016; Riesel et al., 2015). Taken together, this suggests that enhanced performance monitoring predates anxiety symptom expression and persists beyond the illness. A larger ERN thus may be a latent vulnerability, or risk marker, for anxiety and anxiety-related disorders (Meyer, 2016; Olvet & Hajcak, 2008; Riesel et al., 2011, 2015).

However, an enhanced ERN is not observed in *all* anxiety disorders. For instance, individuals reporting specific phobias display a smaller ERN than those reporting high general anxiety and worry (Hajcak et al., 2003a), and inducing fear related to specific phobias does not enhance ERN magnitude (Moser et al., 2005). Similarly, individuals with a diagnosis of PTSD and elevated PTSD symptoms do not seem to display an enhanced ERN compared to those without the disorder and fewer symptoms (Crane et al., 2018; Gorka et al., 2016; Rabinak et al., 2013; Swick et al., 2015), although one study found that hyperarousal symptoms specifically are associated with a *smaller* ERN (Lieberman et al., 2017). The ERN thus seems to be more closely related to dispositional anxiety and anxious apprehension (i.e., worry) than to anxious arousal (i.e., acute fear; Moser et al., 2012; Weinberg et al., 2010, 2016; Vaidyanathan et al., 2011), although some work does not support this idea (Gorka et al., 2017).

The ERN and Other Disorders

ERN magnitude may be useful for differentiating between anxiety and comorbid conditions such as depression. Although exaggerated concern about one's errors is also common in depression (Hammen & Watkins, 2018), findings on the relationship between performance monitoring and depression are mixed. While some studies report an enhanced ERN magnitude in depressed samples (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008, 2010; Tucker et al., 2003),

others have found a smaller ERN magnitude (Clayson et al., 2020; Ladouceur et al., 2012; Schoenberg, 2014; Weinberg et al., 2016), and still others report an ERN magnitude comparable to control populations (Compton et al., 2008; Olvet, Klein, & Hajcak, 2010; Ruchsow et al., 2004, 2006; Schrijvers et al., 2008, 2009; Weinberg, Klein, et al., 2012; Weinberg et al., 2015). It is possible that ERN magnitude is sensitive to depression *severity*, whereby more severe depressive symptomology is characterized by a smaller ERN, while less severe depression is characterized by an enhanced ERN or ERN magnitude comparable to controls (Olvet et al., 2010; Schrijvers et al. 2008, 2009). These mixed findings may also be due to the fact that several studies involved participants with comorbid major depressive disorder (MDD) and anxiety disorders. In support of this point, Weinberg et al. (2012, 2015) showed that an enhanced ERN was observed in participants with GAD, but not comorbid GAD and MDD, suggesting that depressive symptoms may attenuate the larger ERN magnitude associated with heightened anxiety (Peters et al., 2018; Weinberg et al., 2016). Thus, the ERN may be one way to distinguish anxiety from depression (Bress et al., 2013; Weinberg et al., 2012, 2015, 2016).

Further, while an enhanced ERN magnitude is seen in anxiety disorders, a *blunted* ERN seems to be correlated with other psychological disorders. Broadly, a smaller ERN has been found in individuals reporting greater externalizing symptoms (e.g., impulsivity, hyperactivity, aggression; Achenbach & Edelbrock, 1981; American Psychiatric Association, 2013) compared to those with less externalizing (Hall, Bernat, & Patrick, 2007; Meyer & Klein, 2018; Pasion & Barbosa, 2019; Ruchsow et al., 2005; Shiels & Hawk, 2010; Suor et al., 2020). In line with this, individuals with attention-deficit/hyperactivity disorder (ADHD) appear to display an attenuated ERN compared to controls (Chang et al., 2009; Geburek et al., 2013; Groen et al., 2008; Herrmann et al., 2010; Liotti et al., 2005; McLoughlin et al., 2009; Meyer & Hajcak, 2019;

Smith et al., 2016; van Meel et al., 2007), though this effect is not consistently observed (Groom et al., 2010; O'Connell et al., 2009; Wiersema et al., 2005, 2009; Wild-Hall et al., 2009; Zhang et al., 2009).

A blunted ERN is also associated with alcohol and substance use disorders (Franken et al., 2007; Luijten et al., 2014; Marhe et al., 2013; Sokhadze et al., 2008), although some studies have found no ERN amplitude differences between those endorsing substance use and controls (Smith et al., 2017), or report an enhanced ERN in individuals with substance use (Crane et al., 2018; Gorka et al., 2016; Padilla et al., 2010). However, some of these findings may be due to comorbidity with anxiety (Gorka & Phan, 2017; Schellekens et al., 2010). Lastly, schizophrenia and psychotic symptoms are associated with ERN blunting (Bates et al., 2002, 2004; Foti et al., 2012, 2016; Houthoofd et al., 2013; Kansal et al., 2014; Kopp & Rist, 1999; Mathalon et al., 2002; Minzenberg et al., 2014; Morri et al., 2006; Perez et al., 2012; Simmonite et al., 2012). The ERN may thus be a *transdiagnostic* endophenotype for dysfunction underlying several psychopathologies, including clinical anxiety and OCD (Riesel et al., 2019b; Weinberg et al., 2016).

The ERN and Stress

Stress measurement and conceptualization

Because ERN magnitude appears to be a promising biomarker of psychopathology risk, there has been increased interest in factors that lead to individual differences in performance monitoring, such as stress exposure. The term "stress" is often used to denote multiple concepts, including the event that induced stress (i.e., a "stressor") and one's reactions to the stressful event (i.e., the "stress response"; Epel et al., 2018). A stressor has been defined as a situation that requires energy expenditure to cope with, or adapt to, it (Monroe, 2008), or an event that is

threatening and/or harmful (Brown & Harris, 1989; Cohen et al., 2016). Examples of stressors include physical abuse, a divorce, combat exposure, and the loss of a job. On the other hand, the stress response includes biological, psychological, and cognitive processes, as well as behaviours, evoked by stressors (Epel et al., 2018).

Stress can also be conceptualized as a challenge to, or disruption of, homeostasis, the body's state of steady internal conditions (Cannon, 1929; Davies, 2016; Goldstein & McEwen, 2002). Stressful life events lead to a state called "allostasis", which refers to a set of adaptations that occur within the body to cope with challenges (McEwen, 1998; Goldstein & McEwen, 2002). Over the long term, chronic allostatic states can cause wear and tear on the body, referred to as "allostatic load" (McEwen, 1998, 2000; Goldstein & McEwen, 2002; Schulkin & Sterling, 2019). Heightened allostatic load is associated with long-term consequences to health and brain functioning (McEwen, 2000), such as neural alterations following prolongued stress exposure (e.g., McEwen, 2017; Lupien et al., 2009).

However, it is important to note that life stress is not a monolithic construct (Epel et al., 2018; Hammen, 2005; Monroe & Roberts, 1990; Slavich, 2019); it can occur in multiple domains (e.g., interpersonal problems, physical threat) and can differ in its characteristics. For instance, stressors can be considered "acute" or "chronic". Acute stressors are characterized by high intensity and short duration, such as a car accident (Epel et al., 2018; Slavich, 2016; Shields & Slavich, 2017), while chronic stressors are persistent difficulties that are present over a long period of time, such as ongoing abuse, punitive parenting, or prolongued financial problems (Epel et al., 2018; Slavich, 2016; Shields & Slavich, 2017). Further, stressors can be classified as "deprivation" or "threat" experiences. Deprivation refers to a lack of expected social and/or cognitive input, and threat refers to the presence of experiences that endanger physical integrity

(McLaughlin et al., 2014; Sheridan & McLaughlin, 2014). Importantly, these various stressor characteristics have been shown to exert different effects on neural functioning and development (Andersen et al., 2008; Gollier-Briant et al., 2016; Humphreys et al., 2019; McLaughlin et al., 2019; Teicher et al., 2018), and are associated with different emotional consequences (Kendler et al., 2003; Schafer et al., 2022; Slavich, 2016).

Stress assessment presents a complex measurement issue. For instance, self-report measures may be influenced by retrospective recall biases, as they are often completed several years after the stressors have occurred (Hardt & Rutter, 2004; Newbury et al., 2018; Reuben et al., 2016), and by one's *perception* of the stressor. Some stress assessment instruments—such as the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978) and UCLA Life Stress Interview (Adrian & Hammen, 1993)—can elicit detailed information about stressful events which are rated by independent judges on domains such as stress severity and duration, thus providing more "objective" stress ratings. However, these tools are limited by the fact that they usually assess only recent stressors and thus do not provide information about cumulative stress exposure and allostatic load. The Stress and Adversity Inventory (STRAIN; Slavich & Shields, 2018), on the other hand, is a computerized self-report measure that inquires about stressors experienced in multiple life domains across the entire lifespan, allowing for investigation of cumulative effects of stress exposure and stressors experienced in specific developmental periods. The STRAIN also provides subjective (e.g., self-reported severity) and objective (e.g., duration, frequency) stress data and differentiates acute from chronic stressors.

Such assessment systems can help us better understand the link between stress and mental disorders. One predominant model of the role of stress in psychopathology is the diathesis-stress hypothesis, which proposes that mental disorders develop as a result of both stressful experiences

and an individual's diathesis (i.e., a predispositional factor that presents an inherent vulnerability for illness; Bleuler, 1963; Broerman, 2020; Ingram & Luxton, 2005; Meehl, 1962; Rosenthal, 1963; Zuckerman, 1999). While diatheses were originally conceptualized as biological vulnerabilities (e.g., Meehl, 1962), more recent work suggests that cognitive, emotional, and social factors can also serve as diatheses (Alloy et al., 1988; Brown & Harris, 1978; Ingram & Luxton, 2005; McKeever & Huff, 2003; Monroe & Simons, 1991). Diatheses are considered trait-like: They are relatively stable but can change over time and be influenced by life experiences (Ingram & Luxton, 2005). The diathesis-stress model posits that the point at which an individual develops a disorder depends on the interaction between their degree of diathesis and the amount of stress they experience (Ingram & Luxton, 2005; Monroe & Simons, 1991).

The relationship between stress and diathesis may be dynamic. For instance, early life stress can lead to maladaptive cognitive and emotional patterns which can become diatheses for psychopathology that are triggered by later life stress (Ingram & Luxton, 2005; McKeever & Huff, 2003). This concept is central to the "two hit" hypothesis, which posits that vulnerable individuals experience disruptions in neural development (e.g., due to prenatal infection or early-life adversity) that produce alterations in brain functioning (hit one). This initial hit is hypothesized to render individuals more susceptible to negative effects of stress later in life (hit two), thereby leading to psychopathology symptoms (Bayer et al., 1999; Cannon et al., 2014; Georgopoulos et al., 2018; Giovanoli et al., 2013; Maynard et al., 2001; Monte et al., 2017). It is also possible that several "hits" are involved in this pathway (Bayer et al., 1999). Multi-hit models posit that several kinds of stressors experienced during different developmental periods, as well as complex interactions between diatheses and stressors, can increase vulnerability to psychopathology and make individuals more susceptible to subsequent stressors (Chen et al.,

2010; Davis et al., 2016; Pantelis et al., 2003). In line with this idea, the present thesis explored whether dynamic relationships may exist between the ERN—a proposed diathesis for anxiety and related disorders (Riesel et al., 2019b; Weinberg et al., 2016)—and life stress.

Effects of stress on ERN magnitude

Environmental factors have been associated with altered ERN magnitude. For instance, the ERN is enhanced under threat of shock (Meyer & Gawlowska, 2017), and when errors are punished by noxious sounds (Riesel et al., 2012), an effect which persists up to 24 hours after the cessation of punishment (Riesel et al., 2019a). Additionally, adolescents who have experienced high levels of trauma (Lackner et al., 2018), and veterans with greater exposure to combat (Khan et al., 2018), also exhibit an enhanced ERN. These findings suggest that danger-related stressful experiences that increase the negative consequences of errors can have a lasting impact on the way in which the brain responds to mistakes.

ERN magnitude is also enhanced under conditions of social-evaluative stress. Interpersonal stress involves threatening events centered around interactions with others, such as spousal conflict or the loss of a friend (Brown & Harris, 1978; Cohen et al., 2019; Kendler et al., 2003). Stressful interpersonal experiences, especially those involving threats to one's social status (i.e., social-evaluative stress), strongly predict distress and heightened physiological stress responses, even when compared with other types of severe stressors (Dickerson & Kemeny, 2004; Kendler et al., 2003; Slavich, 2016). Consistent with this idea, participants display a larger ERN when they are told that their performance is being observed by peers (Buzzell et al., 2017b; Kim et al., 2005; Van Meel & Van Heijningen, 2010), parents (Meyer et al., 2019), and experimenters (Hajcak et al., 2005; Schillinger et al., 2016; Voegler et al., 2018), especially when the participants report high anxiety (Barker et al., 2015; Masaki et al., 2017). In general,

making mistakes in social contexts is especially distressing (Hewitt et al., 2003; Kim et al., 2005). It is thus theorized that an enhanced ERN magnitude during social-evaluative situations reflects increased performance monitoring in an attempt to avoid mistakes with potential repercussions to social status (Masaki et al., 2017; Van Meel & Van Heijningen, 2010).

Further, there is evidence that harsh and punitive parenting styles (e.g., authoritarian parenting) are associated with an enhanced ERN in children (Banica et al., 2019; Brooker & Buss, 2014b; Chong et al., 2020; Kessel et al., 2019; Meyer et al., 2015). Authoritarian parents typically attempt to control and shape child behaviour to match their standards, often with harsh punishment, and expect obedience (Baumrind, 1971; Robinson et al., 1995). Similarly, parents high in control are characterized by attempts to regulate their child's activities, thoughts, and emotions, and encouraging dependence (Barber, 1996; Rubin & Mills, 1991). These parental behaviours have been associated with punitive reactions to children's errors (Kawamura et al., 2002; Robinson et al., 1995). Consistent with this, children whose performance is observed by highly controlling parents display larger ERN magnitudes compared to observation by a research experimenter or parents high in other dimensions of parenting (i.e., firmness and acceptance; Meyer et al., 2019). Additionally, low maternal sensitivity and low socio-economic status appear to interact to predict atypical ERN development in early childhood (Brooker, 2018). Thus, stressful parenting practices involving harsh punishment following error commission may enhance performance monitoring and sensitize children to mistakes (Banica et al., 2019; Brooker & Buss, 2014b; Enns et al., 2000; Frost et al., 1991; Meyer et al., 2015; Rice et al., 1996).

The ERN may mediate and moderate associations between stress and anxiety

Because of this work indicating that stress can alter ERN magnitude, the ERN has been proposed to be a mediator of the association between stress and anxiety. For instance, punitive

parenting is a risk factor for anxiety (Gershoff et al., 2010; Edwards et al., 2010; Lansford et al., 2014; McLeod et al., 2007; Pinquart, 2017; van der Bruggen et al., 2008; van der Sluis et al., 2015; Yap et al., 2014), and ERN magnitude has been found to mediate associations between parenting style and offspring anxiety disorder status (Chong et al., 2020; Meyer et al., 2015a, 2019), suggesting that stressful parenting may confer risk for anxiety disorders via ERN magnitude potentiation. However, long-term effects of stressful parenting have not been thoroughly examined, leaving it unclear if fear of error-related parental punishment may lead to heightened self-monitoring over the long term.

Similarly, limited work suggests that ERN magnitude may *interact* with stressful experiences to increase risk of heightened anxiety. Meyer et al. (2017a) reported that fearful children who experienced heightened Hurricane Sandy-related stress showed increases in internalizing symptoms only when they also had an enhanced ERN. This finding suggests that life stress may be a catalyst through which the vulnerability of an enhanced ERN confers risk for anxiety. However, very little work has been done to examine whether interactions between ERN magnitude and life stress predict anxiety, leaving a critical gap in our knowledge and ability to identify who is most at risk for the disorder.

The ERN Across Development

Further, there are important developmental concerns when considering neural markers of risk for anxiety. The ERN can be measured as early as 4 or 5 years of age (Brooker et al., 2011; Torpey et al., 2012), and generally increases in magnitude with age (Boen et al., 2022; Davies et al., 2004a, 2004b; DuPuis et al., 2014; Hajcak et al., 2008; Hanna et al., 2012; Kim et al., 2007; Ladouceur et al., 2004, 2007, 2012; Meyer, 2017; Meyer et al., 2012; Moser, 2017; Santesso & Segalowitz, 2008; Santesso et al., 2006b; Tamnes et al., 2013; van Meel et al., 2012; Wiersma et

al., 2007) and pubertal maturation (Gorday & Meyer, 2018; Peters et al., 2018), although a few studies have not found age effects (Eppinger et al., 2009; Richardson et al., 2011). This increase in ERN magnitude is consistent with evidence for continued maturation of the ACC and PFC from childhood through young adulthood (Caballero et al., 2016; Lichenstein et al., 2016; Sturman, & Moghaddam, 2011; Petanjek et al., 2011; Velanova, Wheeler, & Luna, 2008), and is theorized to reflect improvements in mechanisms involved in cognitive control (Luna et al., 2010; Rubia, 2012; Tamnes et al., 2013) and increased capacity for performance monitoring (Crone et al., 2014; Weinberg et al., 2016).

The association between ERN magnitude and anxiety disorder status and symptoms appears to change with age. Consistent with findings from adult studies, an enhanced ERN is associated with heightened anxiety among older children and adolescents, while in younger children, heightened anxiety and fear are associated with a *blunted* ERN (Lo et al., 2015; Meyer, 2017; Meyer et al., 2012; Moser et al., 2015; Torpey et al., 2013) or are not associated with ERN magnitude (Weinberg et al., 2016). This "flipping" of the ERN-anxiety association from young childhood to adulthood is theorized to exist due to developmental changes in sources of anxiety (Gullone, 2000; Meyer, 2017), such that young children typically fear concrete external threat and older children start to fear more abstract, internal threats such as behavioural incompetence. However, a few studies have found enhanced ERN magnitudes in young children with clinical anxiety disorders (Hajcak et al., 2008; Ladouceur et al., 2006; Meyer et al., 2013), suggesting the possibility that *clinical* levels of childhood anxiety are associated with enhanced error monitoring, while subclinical anxiety symptoms are characterized by a blunted ERN (Weinberg et al., 2016).

The relationship between ERN magnitude and stress may also vary with age. Developmental research suggests that different stressor characteristics can exert varying effects on neural development, especially during certain sensitive periods (Andersen & Teicher, 2008, Andersen et al., 2008; Cohen et al., 2006; De Bellis et al., 2000; Lim et al., 2015; Lupien, et al., 2009; McCrory et al., 2017; Mueller et al., 2010; Teicher et al., 2018). For instance, adolescence (approximately ages eight to 18) is a time during which neural systems involved in performance monitoring and cognitive control undergo substantial change (Gogtay et al., 2004; Giedd, 2004; Kelly et al., 2008; Segalowitz & Davies, 2004; Sowell et al., 1999, 2003; Steinberg, 2005). Plasticity in these regions appears particularly pronounced during the pubertal transition to adolescence, between approximately eight and 12 years of age (Dahl & Gunnar, 2009; Shaw et al., 2008; Sowell et al., 2004; Tottenham & Galvan, 2016).

Consistent with this notion, the magnitude of the ERN appears to be particularly susceptible to social threat in early adolescence: Young adolescents (approximately 9 to 12 years old), but not older adolescents (approximately 15 to 18 years old; Barker et al., 2018) or children (around 7 to 8 years old; Kim et al., 2005) display an enhanced ERN in social situations compared to non-social contexts. Further, a study by Moor et al. (2012) suggests that early adolescents (aged 10 to 12) show increased activity in response to social rejection in the ACC, compared to mid-adolescent teenagers (14 to 16 years of age) and emerging adults (19 to 21-year-olds). These data are consistent with evidence that early adolescence is a period of social reorientation toward peers (Parker et al., 2015), and subsequently heightened sensitivity to social stress (e.g., peer evaluation and exclusion; Bolling et al., 2011; Silk et al., 2013).

Developmental considerations are also important when thinking about associations between performance monitoring and stress related to deprivation. In contrast with work

showing *enhanced* error monitoring following threat, toddlers of low socioeconomic status (SES) display *smaller* ERN magnitudes compared to those of high SES (Brooker, 2018; Conejero et al., 2016; Meyer et al., 2020). Further, early childhood psychosocial deprivation has been associated with a blunted ERN in 11- and 12-year-old children (Loman et al., 2013; Troller-Renfree et al., 2016). However, in 8-year-old children, one study reported smaller ERN magnitudes in children who experienced a prolonged period of time in institutional care (McDermott et al., 2012), while another found no ERN magnitude differences in children raised in institutions or foster care, or never-institutionalized children (McDermott et al., 2013). These mixed findings highlight the importance of identifying the type and timing of stress to understand its association with neural response to errors. However, specific effects on ERN magnitude of different types of stressors experienced during different developmental periods have not been investigated, leaving it unclear whether certain experiences more readily alter ERN trajectory than others.

Taken together, prior work suggests that the ERN is enhanced under conditions of stress. However, effects of multiple stressors on ERN magnitude have not been investigated, leaving it unclear whether certain environmental experiences might have a more pronounced impact on neural performance monitoring. Further, it is not clear whether the developmental timing of stressors may impact the ERN in different ways, or whether there are dynamic relationships between stressors and error monitoring that unfold over time. In line with the hypothesis that complex interactions between diatheses and stressor type and timing may increase vulnerability to anxiety (Chen et al., 2010; Davis et al., 2016; Pantelis et al., 2003), the three studies presented next aimed to identify effects of stressors on ERN magnitude and interactions between stress and the ERN. Study One (Banica et al., 2021) investigated associations between the ERN in young adulthood and severity of specific types of stressors (i.e., social and physically threatening

stress), as well as cumulative lifetime stressors, in a sample of undergraduate students. We also collected data on the developmental periods in which the stressors were experienced, ranging from childhood to early and mid-adolescence to the past year, which allowed us to investigate developmental timing effects of specific and cumulative life stress on ERN magnitude.

Study One

Associations between lifetime stress exposure and the error-related negativity (ERN) differ based on stressor characteristics and exposure timing in young adults

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Banica, I., Sandre, A., Shields, G.S., Slavich, G.M., & Weinberg, A. (2021). Associations between lifetime stress exposure and the error-related negativity (ERN) differ based on stressor characteristics and exposure timing in young adults. *Cognitive, Affective, & Behavioral Neuroscience*, 1 – 18. https://doi.org/10.3758/s13415-021-00883-z

Abstract

Life stress increases risk for multiple forms of psychopathology, in part by altering neural processes involved in performance monitoring. However, the ways in which these stress-cognition effects are influenced by the specific timing and types of life stressors experienced remains poorly understood. To address this gap, we examined how different social-psychological characteristics and developmental timing of stressors are related to the error-related negativity (ERN), a negative-going deflection in the event-related potential (ERP) waveform that is observed from 0 to 100 ms following error commission. A sample of 203 emerging adults performed an ERN-eliciting arrow flanker task and completed an interview-based measure of lifetime stress exposure. Adjusting for stress severity during other developmental periods, there was a small-to-medium effect of stress on performance monitoring, such that more severe total stress exposure, as well as more severe social-evaluative stress in particular, experienced during early adolescence significantly predicted an enhanced ERN. These results suggest that early adolescence may be a sensitive developmental period during which stress exposure may result in lasting adaptations to neural networks implicated in performance monitoring.

Introduction

Major life stressors are a key risk factor for a wide range of health problems, including multiple forms of psychopathology (Brown & Harris, 1978; Ingram & Luxton, 2005; Kendler et al., 2002; Kendler et al., 2003; Mazure, 1998). One proposed manner in which stress can affect mental health outcomes is by promoting adaptations in brain structure and function that are beneficial during times of stress but that also may represent latent vulnerabilities for future dysfunction if persistent (Lupien et al., 2009; Masten & Cicchetti, 2010; McCrory et al., 2010; McCrory et al., 2017; McEwen, 1998). In fact, a wealth of evidence shows that individuals who have experienced heightened life stress exhibit volumetric and functional alterations in multiple brain regions, including the anterior cingulate cortex (ACC; Cohen et al., 2006; Treadway et al., 2009) and prefrontal cortex (PFC; De Bellis et al., 2002; Hanson et al., 2010; McLaughlin et al., 2014; Tomoda et al., 2009), which are involved in adaptive performance monitoring and cognitive control (Carter & van Veen, 2007; Miller & Cohen, 2001; Shenhav et al., 2013; Stemmer et al., 2004). Such individuals also demonstrate increased risk for multiple forms of psychopathology (Bae et al., 2006; Botteron et al., 2002; Dell'Osso et al., 2015; Kujawa et al., 2016b; Murrough et al., 2016; Pizzagalli et al., 2018).

Life stress, however, is not a monolithic construct (Epel et al., 2018; Hammen, 2005; Monroe & Roberts, 1990; Slavich, 2019). For example, stressors can occur in multiple domains (e.g., interpersonal conflict, physical danger, financial difficulties) and can be episodic (e.g., serious car accident) or chronic (e.g., ongoing physical abuse; McEwen, 1998; Slavich, 2016; Shields & Slavich, 2017). Consistent with this specificity, emerging research suggests that these different stressor characteristics can exert varying effects on neural development (Andersen et al., 2008; Gollier-Briant et al., 2016; Humphreys et al., 2019; Teicher et al., 2018). Moreover,

although many studies have primarily been concerned with differentiating the effects of early life stress from adulthood or more proximal experiences of stress, there are periods across childhood and adolescence when susceptibility to the remodeling effects of stress might be especially high (Heim & Binder, 2012; Lupien et al., 2009; Steinberg, 2005), in part because of functional changes in physiological systems involved in the body's stress response across development (Gunnar & Donzella, 2002; Gunnar, Talge, & Herrera, 2009a; Gunnar et al., 2009b; Lupien et al., 2009).

Additionally, given that different regions of the brain mature at different rates, it is likely that stress-susceptible regions of the brain have differing sensitive periods (Andersen et al., 2008; Andersen & Teicher, 2008; Cohen et al., 2006; De Bellis et al., 2000; Lim et al., 2015; Luby et al., 2019; Lupien et al., 2009; McCrory et al., 2017; Mueller et al., 2010; Tottenham & Galván, 2016). For instance, brain regions involved in perception and sensory processing show significant development in childhood, before approximately age 7 years (Gogtay et al., 2004; Shaw et al., 2008). Plasticity in neural regions involved in cognitive control is also seen in infancy and early childhood (Guyer et al., 2018; Inguaggiato et al., 2017). Adolescence (approximately ages 8 to 18) is another important time during which neural systems involved in performance monitoring and cognitive control undergo substantial change (Giedd, 2004; Gogtay et al., 2004; Kelly et al., 2008; Segalowitz & Davies, 2004; Sowell et al., 1999; Sowell et al., 2003; Steinberg, 2005), and this change is accompanied by increased cognitive control over behaviors by higher-order prefrontal regions (Griffin, 2017; Luna & Sweeney, 2004).

Plasticity in regions involved in cognitive control appears particularly pronounced during the pubertal transition to adolescence, between approximately ages 8 and 12 years (Dahl & Gunnar, 2009; Shaw et al., 2008; Sowell et al., 2004; Tottenham & Galván, 2016). Importantly,

this developmental period is also characterized by reorganization of the hypothalamic–pituitary–adrenal (HPA) axis, a key part of the body's stress response system (Doom & Gunnar, 2013; Gunnar & Vazquez, 2006). Heightened reactivity to stressors and increased neural sensitivity to the effects of cortisol is also seen during early adolescence, making this a period of heightened vulnerability (Gunnar & Donzella, 2002; Gunnar & Quevedo, 2007; Gunnar, Talge, & Herrera, 2009a; Gunnar et al., 2009b; Lupien et al., 2009).

Combined, these data suggest that it will be beneficial to clarify associations between characteristics and timing of stressors and the activity of neural systems involved in performance monitoring and cognitive control. Effective goal-directed behaviour requires the ability to monitor the outcomes of our actions, which allows us to adapt our behaviour to changing and demanding environments (Ridderinkhof et al., 2004; Ullsperger et al., 2014). The ability to detect errors in one's performance is a crucial step in recognizing that behaviour should change (Falkenstein et al., 2000; Holroyd & Coles, 2002). The error-related negativity (ERN), an event-related potential (ERP) component involved in error detection, measures one aspect of performance monitoring. The ERN is a fronto-centrally maximal negative deflection in the ERP waveform that differentiates erroneous from correct responses within 100 ms of response onset (Falkenstein et al., 1991; Gehring et al., 1995). It functions as an early alarm signal in an action monitoring network that indicates the need to adjust behaviour and increase executive control to remediate mistakes (Botvinick et al., 2001; Gehring et al., 1993; Holroyd & Coles, 2002).

Although the bulk of existing data implicates the ACC as the primary neural generator of the ERN, the ACC has dense interconnections to both limbic and prefrontal areas (Bush et al., 2000), which also modulate ERN magnitude (de Bruijn et al., 2004; Gehring & Knight, 2000; Manoach & Agam, 2013). The amplitude of the ERN appears to increase across adolescence

(Davies et al., 2004; Meyer et al., 2012; Tamnes et al., 2013; Weinberg et al., 2016), an effect that is hypothesized to be due in part to increased activity in ventral-frontal cortical regions involved in error monitoring (Buzzell et al., 2017a). This is consistent with evidence for continued maturation of the ACC and PFC from childhood through young adulthood (Caballero et al., 2016; Lichenstein et al., 2016; Petanjek et al., 2011; Sturman & Moghaddam, 2011; Velanova et al., 2008).

The ERN has also been shown to be sensitive to the effects of a diverse array of naturalistic and experimental stressors, as well as cumulative effects of different stressors (Brooker, 2018; Kessel et al., 2019). For instance, neural response to errors is enhanced under threatening or dangerous conditions: an enhanced ERN is seen under threat of shock (Meyer & Gawlowska, 2017), and when errors are punished by noxious sounds (Riesel et al., 2012), an effect that persists up to 24 hours after the cessation of punishment (Riesel et al., 2019). Naturalistic studies also bear out the results of these laboratory findings. For instance, adolescents who experienced high levels of trauma (Lackner et al., 2018) and veterans with greater exposure to combat (Khan et al., 2018) exhibit an enhanced ERN. It may be adaptive to monitor performance more carefully under conditions of danger and threat – and, indeed, errors potentiate defensive reflexes that are also activated in response to stressful or dangerous situations, such as exposure to threatening stimuli (Bradley et al., 2006; Grillon et al., 1993; Riesel et al., 2012). Neural responses to errors may result in the downstream activation of such defensive systems to avoid physical harm (Riesel et al., 2012). However, other studies have found no associations between posttraumatic stress disorder (PTSD) diagnosis and ERN magnitude in veterans (Gorka et al., 2016; Rabinak et al., 2013; Swick et al., 2015), suggesting that threats to physical integrity may not always lead to increased error monitoring. Moreover,

there is to date limited research examining whether the timing of threats to physical integrity is important in understanding associations with the ERN.

Nor does this effect appear to be unique to life-threatening stressors. ERN magnitude, for example, is also enhanced under conditions of social-evaluative stress, such as when participants are told that their performance is being observed (Barker et al., 2015; Buzzell et al., 2017b; Hajcak et al., 2005; Kim et al., 2005; Schillinger et al., 2016; Van Meel & Van Heijningen, 2010). Furthermore, experiencing harsh and controlling parenting has been associated with a larger ERN in children (Brooker & Buss, 2014; Meyer et al., 2015b; Meyer et al., 2019), an association that persists into early adulthood (Banica et al., 2019). The magnitude of the ERN also appears to be particularly susceptible to social stress in early adolescence: young (approximately aged 9 to 12 years), but not older (approximately aged 15 to 18 years), adolescents display an enhanced ERN in social situations compared to nonsocial contexts (Barker et al., 2018), suggesting that the influence of social evaluation on the ERN changes across adolescence. Further, a study by Moor et al. (2012) suggests that early adolescents (aged 10 to 12 years) show increased activity in response to social rejection in the ACC compared to mid-adolescent teenagers (aged 14 to 16 years) and emerging adults (aged 19 to 21 years). These data are consistent with evidence that early adolescence is a period of social reorientation toward peers (Parker et al., 2015) and subsequently heightened sensitivity to social stress (e.g., peer evaluation and exclusion; Bolling et al., 2011; Silk et al., 2013).

Taken together, these findings suggest that the ERN is enhanced under stressful circumstances—particularly those involving threats to physical integrity or social standing—in which errors may be more consequential. However, most studies that have examined these associations have only looked at a single type of stress exposure. Consequently, it is not clear

whether all types of stress show similar associations with the ERN, nor whether there might be cumulative effects of multiple stressors (Dunn et al., 2019; Evans et al., 2013). Critical to this investigation, it is also not clear whether the developmental timing of stressors differentially affects associations with the ERN. For instance, in contrast to the work cited above, some studies have found that early childhood psychosocial deprivation has been associated with a blunted ERN (in 11- and 12-year-old children; Loman et al., 2013; Troller-Renfree et al., 2016), whereas others have found no such association (in 8-year-old children; McDermott et al., 2013). Additionally, there is some evidence that early adolescents who experienced institutional stress for longer during childhood appear to display later blunted error processing compared to early adolescents with less exposure during infancy (McDermott et al., 2012). This highlights the potential importance of identifying the type and timing of stress to understand its association with neural response to errors.

The present study sought to address this gap by investigating associations between the ERN and both specific and cumulative life stress during distinct developmental periods as retrospectively reported by an emerging adult sample. We tested the following two hypotheses: (1) experiencing socially evaluative and life-threatening stressors would be associated with a heightened ERN; and (2) the developmental timing of exposure to these forms of adversity would influence their associations with the ERN, with stronger associations being observed for stressors occurring earlier in development. In particular, we predicted that early adolescent stress would be most strongly associated with an enhanced ERN magnitude because of prior research indicating that young adolescents exhibit an enhanced ERN under stressful social-evaluative conditions (Barker et al., 2018) and that plasticity in neural regions involved in higher-order cognitive functioning is particularly heightened during the transition into puberty (Tottenham &

Galván, 2016), as opposed to mid- and late-adolescence. Finally, we conducted exploratory analyses with cumulative lifetime stress exposure across different developmental periods in order to understand the specificity of our hypothesized effects.

Method

Participants

Two hundred forty-five participants were recruited from McGill University's psychology human participant pool, flyers posted around the McGill campus, online advertisements, and in-class advertisements. To participate in the study, which is a part of an ongoing longitudinal project (Banica et al., 2020), participants were required to be at least 18 years old and in the first semester of their first year of their undergraduate degree. Participants received course credit or monetary compensation for their time. All participants provided informed, written consent after reviewing the protocol, and all procedures were approved by the McGill University Research Ethics Board.

Two participants were excluded due to excessive noise in their EEG data, one participant was excluded for not completing the stress assessment, 11 participants were excluded because they were currently taking psychotropic medication (e.g., antidepressant or antianxiety medication; De Bruijn et al., 2004; Zirnheld et al., 2004), and one participant was excluded because their correct-related brain activity was more than three standard deviations from the sample mean. Because six or more errors trials are required to elicit a reliable ERN (Meyer et al., 2013; Olvet & Hajcak, 2009), 27 participants were excluded for committing fewer than six errors.[1] The final sample for the ERN and stress analyses thus included 203 participants. Six

[1] Participants excluded for having fewer than six errors did not differ on life-threatening stressor count, $t(274) = -0.83$, $p = 0.41$, social-evaluative stressor count, $t(274) = 0.43$, $p = 0.67$, or total stressor count, $t(274) = 0.91$, $p = 0.37$, compared with participants who had six or more errors. They also did not differ on the stressor severity timing variables, all $ps > 0.14$.

participants were removed from the regression analysis involving life-threatening situations variables because their stress severity scores were more than three standard deviations from the sample mean, leaving a sample of 197 for these analyses. Seven participants were removed from the regression analysis involving the social-evaluative stress variables because their stress severity scores were more than three standard deviations from the sample mean, leaving a sample of 196 for these analyses. Finally, four participants were removed from the regression analysis involving the total stress variables because their stress severity scores were more than three standard deviations from the sample mean, leaving 199 participants in these analyses.[2,3]

The average age of the 203-person sample was 18.14 (SD = 0.39) years. Of these participants, 75.4% were female and 24.6% were male; 51.7% of participants were Caucasian, 22.7% were Chinese, 4.4% were South Asian, 2.5% were Arab/West Asian, 2.5% were Hispanic, 1.0% were South East Asian, 1.0% were Caribbean, 2.0% were Korean, 9.4% indicated they were of another ethnicity, and 2.5% did not indicate their ethnicity. The median family income was between $90,000 and $99,999 CAD (range between less than $10,000 and more than $250,000).

Measures

All participants completed the Stress and Adversity Inventory for Adults (Adult STRAIN; Slavich & Shields, 2018), an online interview that assesses individuals' exposure to major stressors over the entire lifespan. The STRAIN has demonstrated excellent test-retest reliability; good concurrent, predictive, incremental, and discriminant validity; and does not

[2] Participants excluded based on stress severity scores (N = 14) did not significantly differ on conditional ERN magnitude, $t(201) = -1.75$, $p = 0.08$, or conditional CRN values, $t(201) = -1.73$, $p = 0.09$, compared with participants included in the analyses.
[3] For results of the regressions with these outliers included, see Table S1 in supplemental analyses.

appear to be influenced by participants' social desirability or personality characteristics (Slavich & Shields, 2018; Sturmbauer et al., 2019). Participants respond to questions probing 55 types of acute life events and chronic difficulties, and for each stressor that is endorsed, follow-up questions are asked about its severity, frequency, timing, and duration. Up to 115 summary scores indicating overall count and severity of total, acute, and chronic stress experienced in multiple domains, such as life-threatening situations, humiliation, interpersonal loss, marital/partner, and housing, can be computed for each participant from the raw life stress data (Slavich & Shields, 2018). For persons experiencing multiple instances of the same stressor, the stress severity scores reflect severity of the worst (i.e., most severe) instance of each stressor. Participants rated severity using the following response options: Very slightly or not at all, a little, moderately, quite a bit, or extremely stressful or threatening. The severity ratings for the stressors occurring in the same life stress domain are then summed together to yield a total life domain severity score for the time window specified (e.g., early life, adulthood).

Stressor timing and duration data also allow for the investigation of stressor severity during different life periods, ranging from infancy to the past year. To that end, we created severity variables for different types of life stress exposure occurring during different developmental periods. Consistent with prior research (Dahl, 2004), childhood was defined as 7 years or younger; early adolescence was 8 to 12 years; and mid-adolescence was 13 to 15 years. Given naturalistic data showing that recent life stress impacts psychological functioning (Kendler et al., 1998; Kendler et al., 2003) and experimental data demonstrating an effect of proximal stressors on ERN magnitude (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel et al., 2019), we also calculated participants' total stress severity during the past year, defined as the 365-day period prior to completing the STRAIN. Because our participants were nearly

uniformly 18 years old, this 365-day period included age 17 for most (N = 242) but not all (N = 44) participants. For these reasons, we could not reliably calculate stress severity scores for all participants between the ages of 16 to 17 years that were distinct from the past-year variables we created.

Severity of life-threatening stressors during different developmental periods was calculated by summing severity scores of stressors in the following life domains that occurred during each period: ongoing sexual abuse; accident experienced by a close other; having one's life threatened; being robbed; being jailed; physical sexual attack; and/or physical abuse. This yielded separate sum scores of the severity of life-threatening stressors in childhood, early adolescence, mid-adolescence, and the past year. Similarly, severity of social stressors at different time points was calculated by summing severity scores of stressors in the domains of experiencing harsh discipline, losing a job, dropping out of school, having an unfaithful romantic partner, experiencing emotional abuse, and experiencing bullying, during each separate time period. Lastly, severity of total stress was calculated by adding together severity scores across all stress domains during each of the developmental periods.

Task and materials

Using an Intel Core i7 computer, an arrow version of the flanker task (Eriksen & Eriksen, 1974) was administered to participants using Presentation software (Neurobehavioral Systems, Inc., Albany, CA) to control the timing and presentation of task stimuli. All stimuli were displayed on a 19-inch (48.3 cm) computer monitor. Five horizontally aligned arrowheads were presented on every trial, and the center arrow was always the target. Participants used the computer mouse to indicate the target arrow's direction (i.e., pressing the left mouse button if the center arrow points to the left). Approximately 50% of the trials were congruent (">>>>>" or

“<<<<<”) and approximately 50% were incongruent (“<<><<” or “>><>>”). The order of incongruent and congruent trials was random. Arrow stimuli were presented for 200 ms, followed by a period of a maximum of 1,800 ms, which ended once a response was provided. An intertrial interval ranging randomly from 1,000 to 2,000 ms was then presented. Participants were presented with a black screen with a white cross in the center during response and intertrial periods.

Procedure

Participants were first given a brief description of the study. Next, they completed the STRAIN. Electroencephalograph (EEG) sensors were then attached, and participants were provided with more detailed flanker task instructions. Participants were instructed to indicate the central arrowhead’s direction using the right or left mouse button. Participants first did a 6-trial practice block and were told to be both as fast and accurate as possible. The actual task consisted of five blocks of 30 trials (150 trials total), and each block was initiated by the participant. To encourage both accurate and fast responding, participants were provided with feedback at the end of each block, based on their performance. If participants got 75% or less of trials correct, the message “Please try to be more accurate” was displayed; if participant performance was above 80% correct, they received the message “Please try to respond faster”; otherwise, if participant performance was between 76% and 79% accurate, the message “You're doing a great job” was displayed. Participants performed several additional tasks during the experiment: a monetary reward task; a social feedback task (both described in Ethridge & Weinberg, 2018); and an emotional picture viewing task (described in Sandre et al., 2019). The task order was counterbalanced across participants.

Electroencephalographic recording and data processing

Continuous EEG was recorded with a 32-electrode cap and a BrainVision actiCHamp system (Brain Products, Munich, Germany) with a ground electrode at Fpz and based on the standard 10/20 layout. The electrooculogram (EOG) generated from blinks and eye movements was recorded using facial electrodes placed around 1 cm below and above one eye (VEO) and 1 cm to the left and right of both eyes (HEO). A sampling rate of 1,000 Hz was used to record data.

BrainVision Analyzer software (Brain Products, Munich, Germany) was used to conduct offline analysis. Unsegmented data were band-pass filtered with low and high cutoffs of 0.01 and 30 Hz, respectively, with a Butterworth zero phase filter with a 24 db/octave roll-off. For each trial, the EEG was segmented into 1,500 ms windows starting 500 ms before each response onset and continuing for 1,000 ms post-response. Data were then referenced to the average of the left (TP9) and right (TP10) mastoids. Ocular and eye-blink corrections were conducted using HEO and VEO using a modification of the formula defined in Miller et al. (1988).

Detecting and rejecting non-encephalic artifacts (e.g., slow-wave activity, muscle movements) was conducted using a semi-automatic procedure. The criteria applied were a maximum voltage difference of 175 μV within 400-ms intervals, a minimum voltage difference of less than 0.50 μV within 100-ms intervals, and a voltage step of more than 50.0 μV between sample points. These intervals were rejected in each trial from individual channels. To detect and reject remaining artifacts, visual inspection of the data was then conducted. Following artifact rejection, the minimum number of correct trials at Cz for our sample was 79, the maximum was 144, and the average was 132.56. The minimum number of error trials at Cz was 6, the maximum was 60, and the average was 14.94.

Error and correct trials were then averaged separately.[4] 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 (Sandre et al., 2020). Consistent with prior research (Banica et al., 2019; Riesel et al., 2013; Weinberg et al., 2010; Weinberg et al., 2012; Weinberg & Hajcak, 2011), and based on visual inspection of the data, the ERN was quantified on error trials as the average activity from 0 to 100 ms at electrode site Cz, where brain activity following errors was maximal across all participants. In addition, the correct response negativity (CRN)—a negative deflection in the ERP typically present following both correct and error trials (Burle et al., 2008), which appears to index generic response monitoring (Simons, 2010)—was evaluated in the same time window and sites on correct trials. Throughout the manuscript, "conditional ERN" refers to average electrical activity (in microvolts) between 0 and 100 ms following erroneous responses; "conditional CRN" refers to average electrical activity between 0 and 100 ms following correct responses; and "residual ERN"/"ERN_{resid}" refer to average electrical activity between 0 and 100 ms following erroneous responses after controlling for the conditional CRN.

The residual method of calculating the ERN creates scores unrelated to activity following correct responses, identifying brain activity that indexes error processing specifically and providing a more reliable measure than subtraction-based methods (Meyer et al., 2017b). A regression-based procedure (Meyer et al., 2017b; Weinberg et al., 2015) was thus used to compute a standardized ERN residual score to quantify error-specific neural activity. To calculate the residual ERN (ERN_{resid}), participants' conditional CRN was entered as the

[4] We collapsed across congruent and incongruent error and correct trials. See Table S2 in the supplemental material for results of the main regression analyses using residual ERN activity following incongruent trials only.

predictor, and the conditional ERN was entered as the dependent variable; the standardized residual scores from this regression were saved and used as the ERN_{resid}.

The conditional ERN and CRN displayed good internal consistency (Clayson & Miller, 2017), determined using split-half reliability analyses examining associations between even and odd trials. The Spearman-Brown coefficient for the conditional ERN was r = 0.81. For the conditional CRN, the coefficient was r = 0.98. For the residual ERN, the Spearman-Brown coefficient was r = 0.67.

Data analysis

All statistical analyses were conducted using IBM SPSS Statistics software (Version 24). Paired samples t-tests were used to compare the magnitude of correct- and error-related brain activity. Stress severity scores were z-scored before analysis to facilitate comparison of severity ratings for different types of stress and developmental periods. Pearson's correlations were used to report the magnitude of bivariate associations between the ERN_{resid} and stress severity variables. To investigate associations between the ERN_{resid} and stressor severity during different developmental periods, we conducted simultaneous multiple regressions with severity of stressors during childhood, early adolescence, mid-adolescence, and past year as independent variables, and the residual ERN as the dependent variable. One regression was conducted for life-threatening stressors and one for social-evaluative stressors in order to investigate unique effects of different stressor characteristics. To further investigate the cumulative effects of stress, we also conducted a simultaneous multiple regression that included total stressor severity during each developmental period as the predictors. Prior research indicates that there are gender differences in both ERN magnitude and its relationship with individual difference variables

(Larson et al., 2011; Moran et al., 2012; Moser et al., 2016); therefore, gender was included as a covariate in each regression.

Results

Error-related brain activity

Figure 1a displays waveforms of all participants' average ERN, CRN, and difference between them (conditional ERN values minus conditional CRN values), and a topographical map displaying activity post-error minus post-correct. A paired-samples *t*-test indicated that participants' average brain activity between 0 and 100 ms following erroneous responses was significantly more negative (i.e., larger) than activity following correct responses, $t(202) = -17.77$, $p < 0.001$. A repeated-measures ANOVA comparing ΔERN values (error-related neural activity minus correct-related neural activity) at electrode sites Fz, Cz, and Pz confirmed that brain activity following errors was maximal at Cz, $F(1.54, 308.49) = 81.79$, $p < 0.001$. Given that Mauchly's test indicated a violation of sphericity, $\chi^2(2) = 70.02$, $p < 0.001$, we report results with a Greenhouse-Geisser correction. Descriptive statistics, as well as bivariate associations between time-limited stress severity variables and the residual ERN, are presented in Table 1.

Life stress exposure

Means, standards deviations, and ranges of number and severity of cumulative, life-threatening, and social-evaluative stressors are displayed in Table 2. The total sample experienced an average of 11.49 stressors over their lives (*SD* = 7.05; range = 0-34), with an average total severity of 26.47 (*SD* = 17.46; range = 0-87), which corresponds to a severity rating of "moderately" stressful.

Flanker task behavioural data

Descriptive statistics for flanker task behavioural variables (number of errors committed, reaction time on error trials, reaction time on correct trials, and post-error slowing), as well as bivariate associations between behavioural variables, stress variables, and ERN_{resid}, are reported in the supplemental material (Table S3). A paired samples *t*-test indicated that participants were significantly more accurate on congruent trials than incongruent trials, $t(198) = -27.61$, $p < 0.001$, and significantly faster on congruent than incongruent trials, $t(198) = -37.16$, $p < 0.001$.

Stress and the ERN

Results of the three multiple regressions predicting ERN_{resid} magnitude from severity of life-threatening, social-evaluative, and cumulative life stress exposure experienced during childhood, early adolescence, mid-adolescence, and the year prior to completing the stress questionnaire are presented in Table 3.

We first investigated the effects of timing of life-threatening stressors. Although the overall model predicted a significant amount of variance in the ERN_{resid}, after adjusting for the effects of gender and severity of life-threatening situations during all other time periods, only severity of life-threatening situations experienced in mid-adolescence was uniquely significantly associated with the ERN_{resid}, $\beta = 0.17$, $p = 0.02$, with more severity predicting a less negative (i.e., smaller) ERN (Fig. 1b). We did not observe significant associations with either more proximal (i.e., past-year) or more distal (i.e., childhood or early adolescence) stressors.

Next, we examined the effects of timing of social-evaluative stressors. As above, the overall model accounted for a significant portion of variance in the ERN_{resid}. However, after adjusting for effects of other predictors, only the severity of social-evaluative stressors occurring during early adolescence was significantly associated with magnitude of the ERN_{resid}, $\beta =$

−0.22, $p = 0.007$. Specifically, more severe social-evaluative stress occurring during this time period was associated with a more negative (i.e., larger) ERN (Fig. 1c).

The final regression was conducted to estimate the effects of cumulative stress exposure occurring during each developmental period. The results indicated that the ERN_{resid} was also significantly negatively associated with severity of *total* stress experienced during early adolescence, $\beta = -0.23$, $p = 0.006$, with more severe life stress exposure occurring across all life domains predicting a larger ERN (Fig. 1d). Because this effect was similar in magnitude to that observed for social-evaluative stress, we calculated a new total severity score of stress in early adolescence excluding severity ratings for social-evaluative stress. This new total severity score was also significantly correlated with the ERN_{resid}, $r = -0.17$, $p = 0.02$.

Discussion

Although prior studies have examined associations between life stress and the ERN, this body of research has not thoroughly explored the important question of how such effects might differ as a function of the specific timing or type of stressors experienced. To address this gap, we investigated how type of life stress experienced (social-evaluative, life-threatening, and cumulative) and timing of stress exposure (from childhood to the past year) was associated with participants' neural response to errors. We found that more severe total stress exposure occurring during early adolescence (8 to 12 years old) was associated with an enhanced ERN. These findings are consistent with studies indicating that stress experienced during early adolescence is associated with alterations in the function and structure of the ACC and PFC (De Bellis et al., 2000; Lupien et al., 2009; Tottenham & Galván, 2016), which are involved in generating the ERN (Brázdil et al., 2005; de Bruijn et al., 2004; Dehaene et al., 1994; Gehring & Knight, 2000). In particular, prior studies have found that extreme threat or deprivation occurring during

childhood and adolescence are associated with increased activation of the ACC (Lim et al., 2015; McCrory et al., 2017; Mueller et al., 2010). The present study extends these findings by demonstrating that even common forms of stress, such as bullying, may be associated with increased engagement of a performance monitoring network when they occur during certain developmental periods.

In particular, the data suggest that more severe social-evaluative stress experienced during early adolescence is associated with a larger ERN. This finding is consistent with prior research indicating that social stressors, such as punitive parenting and peer evaluation, are related to enhanced error monitoring (Barker et al., 2015; Banica et al., 2019; Brooker & Buss, 2014; Buzzell et al., 2017b; Hajcak et al., 2005; Kim et al., 2005; Meyer et al., 2015b; Meyer et al., 2019; Schillinger et al., 2016; Van Meel & Van Heijningen, 2010). Importantly, consistent with Barker et al. (2018), who found that young (but not older) teenagers show enhanced error monitoring in social contexts, our results indicate that social stress experienced during early adolescence is particularly strongly associated with a larger ERN in emerging adulthood. Sensitivity to social stress is heightened in adolescence (Bolling et al., 2011; Silk et al., 2013), which is a developmental period characterized by increased emphasis on peer relationships and social feedback (Parker et al., 2015). This sensitivity appears particularly pronounced in early adolescence (Moor et al., 2012). Therefore, the present results suggest that increased social stress, which is especially salient during this time, may result in lasting adaptations in neural networks involved in performance monitoring.

These adaptations may be functional in the context of stress. In social settings, for example, errors may pose a threat to safety or social standing, and better performance monitoring in times of social stress may help individuals to avoid mistakes that could result in negative

evaluation (Banica et al., 2019; Brooker & Buss, 2014; Hajcak, 2012; Kujawa et al., 2016a; Lim et al., 2015; Meyer et al., 2015b; Meyer et al., 2019). Yet such adaptations may become maladaptive in other circumstances. For instance, an enhanced ERN has been observed in individuals with social anxiety (Endrass et al., 2014; Kujawa et al., 2016a), who, due in part to past social stress, find social situations threatening and closely monitor their performance to prevent mistakes (Clark & Wells, 1995). This heightened self-monitoring can then interfere with social performance (Clark & Wells, 1995). An enhanced ERN may thus be one pathway through which social interactions become even more stressful. More specifically, social stress in early adolescence may sensitize neural performance monitoring networks to errors, leading to enhanced performance monitoring and increased neural reactivity to mistakes in social settings, as well as poorer social skills. Longitudinal studies examining associations between social stress, the ERN, social functioning, and social anxiety will be necessary to explore this possibility.

We also examined the possibility that variability in the magnitude of the ERN might be better predicted by cumulative stress (Brooker, 2018; Kessel et al., 2019), both in terms of cumulative effects over time for each distinct type of stress across all developmental periods (regressions one and two) and in terms of cumulative effects of multiple stressors within distinct developmental periods (regression three). Our results are consistent with evidence for a cumulative effect of multiple stressors within distinct developmental periods in that the data indicated that more severe *total* stress exposure occurring during early adolescence was associated with an enhanced ERN magnitude, even when adjusting for social stress severity. These findings are in line with broader neuroscience literature demonstrating that alterations in ACC and PFC volume and function, as well as the magnitude of the ERN, are associated with many different types of stress (Banica et al., 2019; Barker et al., 2015; Buzzell et al., 2017b;

Cohen et al., 2006; De Bellis et al., 2002; Hanson et al., 2010; Kim et al., 2005; McLaughlin et al., 2014; Meyer et al., 2015b; Meyer & Gawlowska, 2017; Riesel et al., 2019; Tomoda et al., 2009; Treadway et al., 2009). Moreover, they suggest that cumulative effects of stress—particularly during early adolescence, when neural plasticity is increased—are also related to alterations in performance monitoring networks. Future studies with larger samples that are better powered to examine interactions between stressor types and timing will be useful for exploring such cumulative effects more thoroughly.

Extending prior research, our results suggest some specificity in terms of the effects of the *timing* of stressors and associations with the ERN. For instance, although we did not observe a significant association with life-threatening stressors experienced in early adolescence, our results did indicate that increased severity of life-threatening stressors experienced during mid-adolescence was associated with a *smaller* ERN magnitude. This is contrary to our hypotheses and to literature indicating that shock and noxious sounds (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel et al., 2019) and trauma (Khan et al., 2018; Lackner et al., 2018) are related to an *enhanced* ERN, as well as research suggesting that neural systems that generate the ERN may activate defensive systems designed to protect oneself from harm (Riesel et al., 2012). Furthermore, it should be noted that the effect size is small and changed in magnitude when including outlier participants in the regression analyses (see supplemental material). Thus, we are hesitant to speculate about why mid-adolescent exposure to stress related to threats to physical integrity may be associated with decreased performance monitoring. Future studies should further investigate this research question.

However, there is also research showing that war veterans with PTSD display a similar ERN magnitude as control participants without PTSD (Gorka et al., 2016; Rabinak et al., 2013;

Swick et al., 2015), indicating that individuals exposed to life-threatening stressors do not always display enhanced performance monitoring. In our study, life-threatening stress was measured by asking participants about the severity of situations, such as physical and sexual abuse, being robbed, and living in a war zone, which may exert different effects on performance monitoring than threats in a controlled laboratory setting. However, another possibility is that the relatively restricted range of reported severity of life-threatening situations limited our ability to detect the expected associations with the ERN. This range is also small compared with the range of the social-evaluative stress and total stress severity variables—stressors that were more common in our sample of college students. Future studies should investigate this question in samples with a greater number and more severe dangerous stressors.

Contrary to research demonstrating that childhood is a vulnerable period during which environmental influences may have a particularly strong effect on neural development (Andersen, 2003; Crews et al., 2007; De Bellis, 2005; Hart & Rubia, 2012), and that stress during childhood is associated with alterations in neural regions involved in performance monitoring (Cohen et al., 2006; Tomoda et al., 2009; Treadway et al., 2009), the present analyses did not reveal significant associations between childhood stress exposure and ERN magnitude. In our sample, however, relatively few participants reported severe stressors during childhood. Further, we were not able to separate stress severity experienced in later childhood from that experienced in infancy, which has previously shown different associations with ERN magnitude (Loman et al., 2013; McDermott et al., 2013; Troller-Renfree et al., 2016). Future studies are required to clarify associations between ERN magnitude in adulthood and more severe stress occurring early in development. Similarly, our results did not identify significant associations between ERN magnitude and stress experienced during the past year, despite prior research

suggesting that there are proximal effects of stress on performance monitoring (e.g., Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel et al., 2019). However, in this study, proximal stress included stressors experienced over the preceding 12 months, whereas other studies investigated effects of same-day or previous-day stress, which may explain the observed differences. Future experimental research examining the duration of the effects of laboratory stressors will be helpful to clarify the nature of these associations.

Limitations of this study point to directions for future research. First, our sample consisted of relatively few male participants, which did not allow us to investigate whether there are different stress-susceptible developmental periods for males versus females. Given prior research indicating possible sex differences in rates of neural maturation (Andersen, 2003; Lenroot et al., 2007; Lenroot & Giedd, 2010), future research should look closely at sex-specific sensitive periods. In particular, adolescence is thought to begin with puberty onset (Blakemore et al., 2010), which tends to be between the ages of 8 and 12 for females (Hayward, 2003). However, hormonal events involved in puberty typically occur 1 or 2 years later for males (Blakemore et al., 2010). Our stress severity time intervals classified early adolescence as 8 to 12 years old, which may be accurate for denoting the period of transition to puberty for females (Hayward, 2003) but may not effectively capture male transition to puberty (Blakemore et al., 2010). Additionally, a sample that consisted of 75% female participants may not have provided adequate statistical power to detect gender effects, or potential moderating effects of gender.

Second, our sample consisted of undergraduate students at the beginning of their college studies, who endorsed fewer, and less severe, stressors compared with other studies that have employed the STRAIN (Cazassa et al., 2020; Slavich & Shields, 2018; Sturmbauer et al., 2019).

Our results may reflect experiences unique to those with higher-than-average socioeconomic status and education, limiting their generalizability. Indeed, prior research has found different patterns of error monitoring alterations among individuals reporting more extreme forms of stress—for instance, a blunted, as opposed to enhanced, ERN in early adolescents who experienced prolonged early institutionalization (Loman et al., 2013; Troller-Renfree et al., 2016). At a broader level, relative to most of the global population, our sample was Western, Educated, Industrialized, Rich, and Democratic (i.e., WEIRD; Henrich et al., 2010a), and findings in WEIRD samples do not always replicate in non-WEIRD samples (Henrich et al., 2010b). Future research should thus aim to replicate our findings in a more racially and socioeconomically diverse sample reporting more, and more severe, stressors. However, research has shown that experiencing even one major social stressor may substantially impact health (Slavich et al., 2009; Slavich et al., 2014), and consistent with such findings, we observed significant associations between social-evaluative stress exposure and the ERN even with a relatively limited range of stress severity. Furthermore, investigating effects of stressors in the mild-moderate range is important for elucidating how different levels of stress severity—not just the presence and absence of severe stress—may impact development (McLaughlin et al., 2021).

Third, although prior research indicates that social desirability and personality characteristics do not influence responding on the STRAIN (e.g., Slavich & Shields, 2018), we cannot rule out the possible influence of such biases in this study. Furthermore, as is the case with all retrospective self-report measures, recall of stressful events and severity may not have been completely accurate. For example, participant reports of childhood stress may not be as accurate as their reports on stressors that occurred more recently (Maughan & Rutter, 1997). However, we note that the STRAIN focuses on stressors that are moderate to severe in nature,

which prior research has shown can be reliably recalled (Brown & Harris, 1978; Reuben et al., 2016). Moreover, validation studies using the STRAIN have shown very high test-retest reliability over time, suggesting that individuals are recalling the same stressors at different time points (Cazassa et al., 2020; Slavich & Shields, 2018). Additionally, because of the correlational nature of the study, it is also possible that individuals with a larger ERN have more biased recall. Yet a negative recall bias should result in global biases reflected across *all* stressor types and timing, and we did not find associations between ERN magnitude and stress experienced in every domain or developmental period, again suggesting minimal impact of recall biases. Nevertheless, future prospective studies documenting stress across time will be necessary to validate the results reported here, and, in particular, the present study should be replicated using contemporaneous assessments of life stress exposure.

Fourth, unlike traditional interview-based measure of life stress, such as the UCLA Life Stress Interview (Hammen, 1991) and Life Events and Difficulties Schedule (Brown & Harris, 1978), the STRAIN does not generate interviewer-rated life stress exposure or severity scores. A wealth of research has shown that perceptions of stress exposure and severity are strongly tied to biological and clinical health (Epel et al., 2018; Slavich & Cole, 2013), suggesting that it is important to investigate individuals' stressor appraisals and not just objective stress severity. However, future research should certainly employ other interview-based measures of life stress to examine the robustness of the effects described here.

Fifth, given the present study design, we cannot be certain whether the effects observed are due to adolescent-limited stress versus chronic stress that began in childhood and perhaps peaked in adolescence (Tottenham & Galván, 2016). Research suggests that interpersonal stress is highly continuous (Chapell et al., 2006), and the social stress reported by participants in the

present sample during adolescence may have started earlier and continued past early adolescence. However, the results of our multiple regression analysis—in which stress across other periods was controlled for—are suggestive in this regard, in that social stress during early adolescence showed unique associations with the ERN. Nonetheless, prospective studies would be helpful for further disentangling the effects of stress experienced during different developmental periods on the ERN.

Finally, the effect sizes observed in the present sample are small to medium in magnitude (Cohen, 1988), and should be interpreted with caution. However, self-report data and psychophysiological variables share no method variance and thus are expected to moderately correlate with one another (Campbell & Fiske, 1959; Patrick et al., 2013). Consistent with this, similar effect sizes are common in the literature investigating associations between the ERN and important individual difference variables (Cavanagh & Shackman, 2015; Meyer et al., 2015a; Moser et al., 2013; Weinberg et al., 2016) and can have meaningful implications for real-world outcomes (Hajcak et al., 2019; Meyer et al., 2017a). For instance, the ERN has demonstrated incremental predictive ability for the later development of anxiety disorders over and above other common risk factors (Meyer et al., 2015a; Meyer et al., 2018) and appears to predict adolescents' tobacco use initiation (Anokhin & Golosheykin, 2015), effects that were in the small to medium range. These studies suggest that even relatively modest effects can improve prediction of important health-related outcomes.

Conclusions

The present findings indicate that greater total and social-evaluative stress severity during early adolescence is associated with increased error monitoring in emerging adulthood. These results provide support for the notion that early adolescence is a sensitive period during which

stress may more significantly influence neural networks involved in performance monitoring. In a notable extension of prior work, the present results also indicate that it is important to consider both the developmental timing and type of stressors experienced over the lifespan. Specially, we found that social-evaluative stress occurring during early adolescence was associated with a larger ERN, whereas life-threatening stress severity during mid-adolescence was related to a *smaller* ERN. However, total stress severity in early adolescence was also associated with enhanced error monitoring, which suggests that there are both cumulative *and* specific effects of stressor type on performance monitoring across development. This research therefore lays the groundwork for future prospective studies seeking to understand the long-term effects of stress-induced neural adaptations to the environment.

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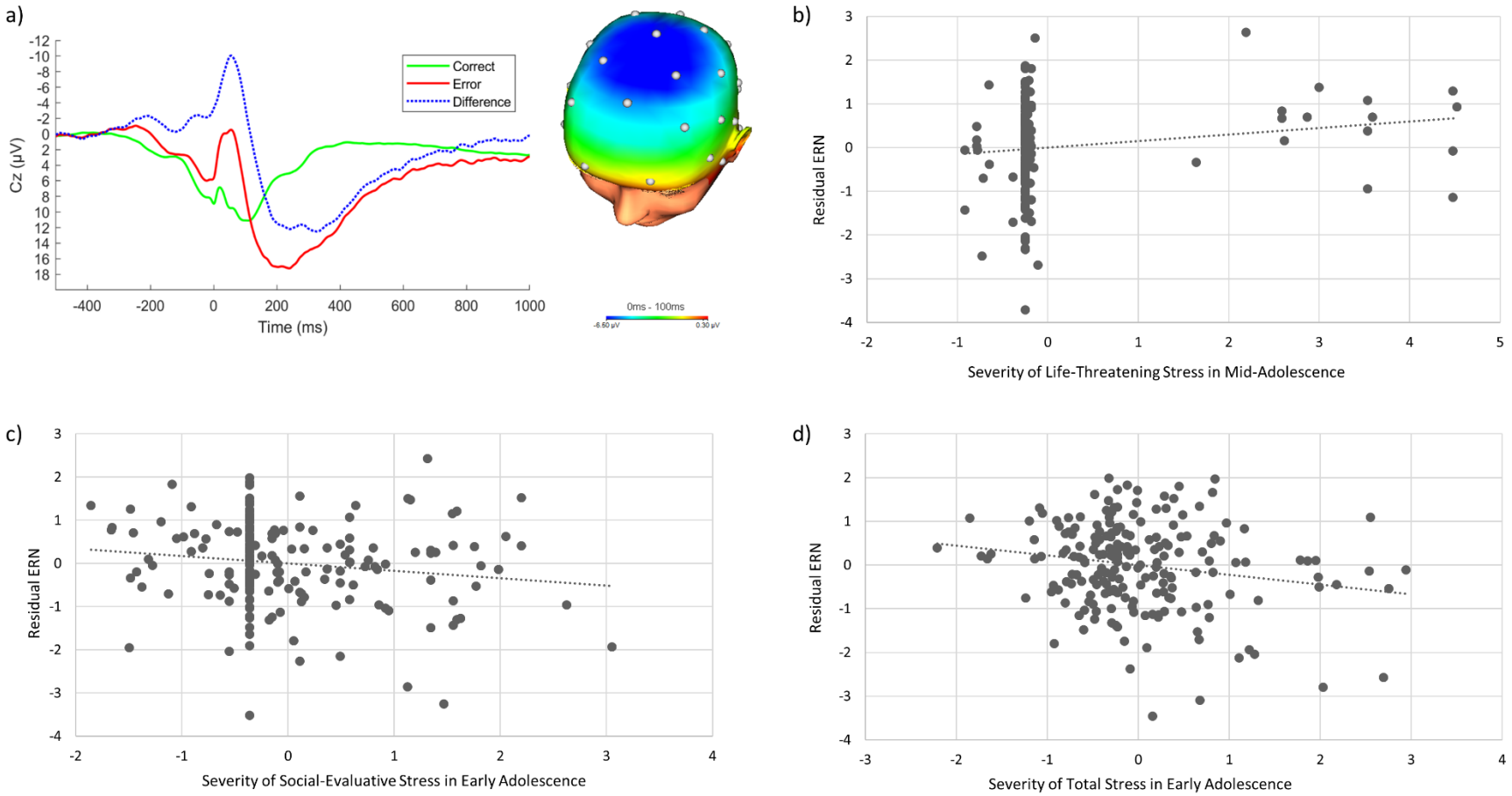

Fig. 1. (**a**) Response-locked ERP average waveforms after erroneous and correct responses, and the difference (conditional ERN values minus conditional CRN values), at electrode site Cz, and a topographical map displaying the error minus correct neural response difference (in μV) for all participants (N = 203). (**b**) A partial regression plot depicting the association between life-threatening stress severity during mid-adolescence and the residual ERN, adjusting for gender and stress severity during other time periods. (**c**) Partial regression plot depicting the association between social-evaluative stress severity during early adolescence and the residual ERN, adjusting for gender and stress severity during other time periods. (**d**) Partial regression plot depicting the association between total stress severity during early adolescence and the residual ERN, adjusting for gender and stress severity during other time periods

Table 1

Descriptive statistics and correlations between participants' residual error-related negativity, z-scored stress severity variables, and gender

	Mean (*SD*) Range	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**	**7.**	**8.**	**9.**	**10.**	**11.**	**12.**	**13.**
1. Life-threatening: Childhood	.08 (.44) 0 to 4													
2. Life-threatening: Early Adolescence	.28 (1.03) 0 to 5	.12												
3. Life-threatening: Mid-Adolescence	.29 (1.06) 0 to 5	.01	.13											
4. Life-threatening: Past Year	.69 (1.82) 0 to 10	.06	.13	-.01										
5. Social: Childhood	1.11 (1.93) 0 to 9	.42**	.22**	.12	-.07									
6. Social: Early Adolescence	1.64 (2.34) 0 to 9	.15**	.37**	.20**	.15*	.40**								
7. Social: Mid-Adolescence	0.87 (1.78) 0 to 8	-.02	.28**	.20**	.17*	.03	.32**							
8. Social: Past Year	0.95 (2.09) 0 to 13	-0.01	.00	.20**	.19**	.30**	.08	.13						
9. Total: Childhood	4.69 (7.30) 0 to 33	.29**	.08	.12	-.07	.64**	.26**	.09	.28**					
10. Total: Early Adolescence	7.30 (8.92) 0 to 43	.06	.30**	.04	.14	.24**	.54**	.29**	.07	.40**				
11. Total: Mid-Adolescence	10.36 (11.48) 0 to 52	-.06	.30**	.29**	.19**	.31**	.37**	.36**	.26**	.38**	.50**			
12. Total: Past Year	23.36 (19.09) 0 to 89	-.06	.26**	.05	.31**	.21**	.20**	.22**	.49**	.24**	.27**	.37**		
13. ERN_{resid}	0 (1) -3.55 to 2.59	-.01	-.06	.14*	-.13	-.01	-.16*	.01	-.03	.02	-.21**	-.13	-.10	
14. Gender	75% female; 0 = male, 1 = female	-.06	.05	-.13	-.11	-.02	-.01	-.06	.07	.05	.05	.01	.04	.13

*$p < 0.05$; **$p < 0.01$.
"ERN" = error-related negativity; "Life-threatening" = severity of life-threatening stressors; "Social" = severity of social-evaluative stressors; "Total" = severity of cumulative stressors across all stress domains.
For correlations between life-threatening stress variables, gender, and the ERN residual, $N = 197$. For correlations between social stress variables, gender, and the ERN residual, $N = 196$. For correlations between total stress variables, gender, and the ERN residual, $N = 199$. For correlations between life-threatening and social stress variables, $N = 192$. For correlations between life-threatening and total stress variables, and social and total stress variables, $N = 193$. For correlations between residual ERN and gender, $N = 203$. The means, standard deviations, and ranges presented for severity scores during different developmental periods are those before z-scoring. The range of the residual ERN is that of the 203-participant sample.

Table 2

Descriptive statistics for number and severity of life-threatening, social-evaluative, and total stressors experienced across the lifespan

	Mean	***SD***	**Range**
Life-threatening stress: Total count	1.22	1.79	0 - 9
Life-threatening stress: Total severity	2.92	4.13	0 - 20
Social-evaluative stress: Total count	1.55	1.84	0 - 8
Social-evaluative stress: Total severity	3.82	4.00	0 - 18
Total stress: Total count	11.49	7.05	0 - 34
Total stress: Total severity	25.47	17.46	0 - 87

Note. For all statistics, $N = 203$.

Table 3

Results of three separate simultaneous linear regressions predicting the residual error-related negativity from severity of stressors in the life-threatening, social-evaluative, and total life stress domains during different developmental periods

	Residual Error-Related Negativity (ERN)					
Predictor	***b* (SE)**	***β***	***p***	***95% CI***	**Tolerance**	**VIF**
REGRESSION 1: LIFE-THREATENING SITUATIONS						
Gender	.34 (.17)	**.15***	**.04**	**0.01, 0.67**	.96	1.04
Life-threatening stress: Childhood Severity	.02 (.07)	.02	.81	-0.12, 0.16	.98	1.02
Life-threatening stress: Early Adolescence Severity	-.08 (.07)	-.08	.28	-0.22, 0.06	.95	1.06
Life-threatening stress: Mid-Adolescence Severity	.17 (.07)	**.17***	**.02**	**0.03, 0.31**	.96	1.04
Life-threatening stress: Past Year Severity	-.11 (.07)	-.11	.14	-0.25, 0.03	.97	1.03
	Total $R^2 = 0.06$; $F(5, 191) = 2.54$, $p = 0.03$					
REGRESSION 2: SOCIAL-EVALUATIVE STRESS						
Gender	.33 (.16)	**.15***	**.04**	**0.02, 0.65**	.99	1.01
Social stress: Childhood Severity	.10 (.08)	.10	.23	-0.06, 0.26	.75	1.33
Social stress: Early Adolescence Severity	-.22 (.08)	**-.22***	**.01**	**-0.38, -0.06**	.74	1.35
Social stress: Mid-Adolescence Severity	.10 (.08)	.10	.20	-0.05, 0.25	.86	1.16
Social stress: Past Year Severity	-.06 (.08)	-.06	.40	-0.21, 0.09	.88	1.13
	Total $R^2 = 0.06$; $F(5, 190) = 2.32$, $p = 0.045$					
REGRESSION 3: TOTAL STRESS						
Gender	.28 (.16)	.12	.08	-0.03, 0.59	1.00	1.01
Total stress: Childhood Severity	.13 (.08)	.13	.09	-0.02, 0.29	.79	1.26
Total stress: Early Adolescence Severity	-.23 (.08)	**-.23***	**.01**	**-0.39, -0.07**	.69	1.44
Total stress: Mid-Adolescence Severity	-.05 (.09)	-.05	.57	-0.22, 0.12	.67	1.50
Total stress: Past Year Severity	-.06 (.08)	-.06	.45	-0.21, 0.09	.85	1.18
	Total $R^2 = 0.08$; $F(5, 193) = 3.22$, $p = 0.008$					

* $p < .05$. Univariate outliers were eliminated for some analyses. Therefore, for regression one, $N = 197$. For regression two, $N = 196$. For regression three, $N = 199$. Gender is coded such that 0 = males and 1= females

Supplemental Analyses

Table S1

Results of three separate simultaneous linear regressions predicting the residual error-related negativity from severity of stressors in the life-threatening, social-evaluative, and total life stress domains during different developmental periods, including outlier participants' data.

	Residual Error-Related Negativity (ERN)	
Predictor	***b* (SE)**	***β***
REGRESSION 1: DANGEROUS SITUATIONS		
Gender	.31 (.16)	.14†
Life-threatening stress: Childhood Severity	-.01 (.07)	-.01
Life-threatening stress: Early Adolescence Severity	-.19 (.07)	**-.19***
Life-threatening stress: Mid Adolescence Severity	.12 (.07)	.12
Life-threatening stress: Past Year Severity	-.13 (.07)	-.13
	Total $R^2 = 0.08$; $F(5, 197) = 3.57$, $p = 0.004$	
REGRESSION 2: SOCIAL-EVALUATIVE STRESS		
Gender	.31 (.16)	.13†
Social stress: Childhood Severity	.07 (.08)	.07
Social stress: Early Adolescence Severity	-.18 (.08)	**-.18***
Social stress: Mid Adolescence Severity	-.004 (.07)	-.004
Social stress: Past Year Severity	-.05 (.07)	-.05
	Total $R^2 = 0.05$; $F(5, 197) = 1.85$, $p = 0.10$	
REGRESSION 3: TOTAL STRESS		
Gender	.30 (.16)	.13†
Total stress: Childhood Severity	.13 (.08)	.13
Total stress: Early Adolescence Severity	-.15 (.08)	-.15†
Total stress: Mid Adolescence Severity	-.05 (.08)	-.05
Total stress: Past Year Severity	-.04 (.08)	-.04
	Total $R^2 = 0.05$; $F(5, 197) = 1.93$, $p = 0.09$	

Note. * indicates $p < .05$; † indicates $p < .07$ (trending). Gender is coded such that 0 = males and 1= females.

Table S2

Results of the main regression analyses using residual ERN activity following incongruent Flanker task trials only.

	Residual Error-Related Negativity (ERN)	
Predictor	***b* (SE)**	***β***
REGRESSION 1: DANGEROUS SITUATIONS		
Gender	.20 (.18)	.09
Life-threatening stress: Childhood Severity	-.07 (.07)	-.08
Life-threatening stress: Early Adolescence Severity	-.06 (.08)	-.06
Life-threatening stress: Mid Adolescence Severity	.10 (.07)	.10
Life-threatening stress: Past Year Severity	-.07 (.08)	-.07
	Total R^2 = .03; F(5,172) = 1.14, p = .34	
REGRESSION 2: SOCIAL-EVALUATIVE STRESS		
Gender	.24 (.17)	.10
Social stress: Childhood Severity	.04 (.08)	.04
Social stress: Early Adolescence Severity	-.27 (.09)	**-.27***
Social stress: Mid Adolescence Severity	.23 (.08)	**.23***
Social stress: Past Year Severity	-.02 (.08)	-.02
	Total R^2 = .08; F(5,172) = 3.16, p = .01	
REGRESSION 3: TOTAL STRESS		
Gender	.16 (.17)	.07
Total stress: Childhood Severity	.05 (.08)	.05
Total stress: Early Adolescence Severity	-.15 (.09)	-.15
Total stress: Mid Adolescence Severity	-.02 (.09)	-.02
Total stress: Past Year Severity	-.09 (.08)	-.09
	Total R^2 = .04; F(5,175) = 1.42, p = .22	

Note. * indicates $p < .05$. Gender is coded such that 0 = males and 1= females.

Table S3

Descriptive statistics and correlations between participants' residual error-related negativity, z-scored stress severity variables, and flanker task behavioural data.

	Mean (SD) Range	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**	**7.**	**8.**	**9.**	**10.**	**11.**	**12.**	**13.**	**14.**	**15.**	**16.**
1. Life-threatening: Childhood	.08 (.44) 0 to 4																
2. Life-threatening: Early Adolescence	.28 (1.03) 0 to 5	.12															
3. Life-threatening: Mid Adolescence	.29 (1.06) 0 to 5	.01	.13														
4. Life-threatening: Past Year	.69 (1.82) 0 to 10	.06	.13	-.01													
5. Social: Childhood	1.11 (1.93) 0 to 9	.42**	.22**	.12	-.07												
6. Social: Early Adolescence	1.64 (2.34) 0 to 9	.15**	.37**	.20**	.15*	.40**											
7. Social: Mid Adolescence	0.87 (1.78) 0 to 8	-.02	.28**	.20**	.17*	.03	.32**										
8. Social: Past Year	0.95 (2.09) 0 to 13	-0.01	.00	.20**	.19**	.30**	.08	.13									
9. Total: Childhood	4.69 (7.30) 0 to 33	.29**	.08	.12	-.07	.64**	.26**	.09	.28**								
10. Total: Early Adolescence	7.30 (8.92) 0 to 43	.06	.30**	.04	.14	.24**	.54**	.29**	.07	.40**							
11. Total: Mid Adolescence	10.36 (11.48) 0 to 52	-.06	.30**	.29**	.19**	.31**	.37**	.36**	.26**	.38**	.50**						
12. Total: Past Year	23.36 (19.09) 0 to 89	-.06	.26**	.05	.31**	.21**	.20**	.22**	.49**	.24**	.27**	.37**					
13. Total errors	15.22 (6.15) 6 to 34	-.11	-.15*	-.01	-.04	.08	-.00	-.04	-.05	-.08	-.15*	-.06	-.07				
14. RT on error trials	298.74 (28.09) 239.98 to 405.69	.09	.01	.00	.01	-.03	.01	.01	.12	.00	.03	-.01	.04	-.15*			
15. RT on correct trials	376.98 (37.15) 303.31 to 489.08	.04	.11	.04	-.03	.05	.12	.10	.18*	.00	.06	.16*	.09	-.32**	.58**		
16. Post-error slowing	39 (36.10) -64.43 to 168.81	.12	.02	-.02	.07	.02	.07	-.06	.05	.04	.13	.09	.04	-.27**	.09	.22**	
17. ERN_{resid}	0 (1) -3.55 to 2.59	-.01	-.06	.14*	-.13	-.01	-.16*	.01	-.03	.02	-.21**	-.13	-.10	.15*	.08	.05	-.03

Note. * indicates $p < .05$, ** indicates $p < .01$. "ERN" means "error-related negativity", "Life-threatening" means severity of life-threatening stressors, "Social" means severity of social-evaluative stressors, and "Total" refers to severity of cumulative stressors across all stress domains. For correlations between life-threatening stress variables and the ERN residual, N = 197. For correlations between social-evaluative stress variables and the ERN residual, N = 196. For correlations between total stress variables and the ERN residual, N = 199. For correlations between life-threatening and social-evaluative stress variables N = 192. For correlations between life-threatening and total stress variables, and social-evaluative and total stress variables, N = 193. For correlations between residual ERN and behavioural variables, N = 199. For correlations between behaviour and life-threatening stress variables, N = 193. For correlations between behaviour and social-evaluative stress variables, N = 192. For correlations between behaviour and total stress variables, N = 195. The means and standard deviations presented for severity scores during different developmental periods are those prior to z-scoring. The range of the residual ERN is that of the 203-participant sample.

Preface to Study Two

In Study One, we investigated associations between the ERN and specific and cumulative life stress severity during distinct developmental periods, as retrospectively reported by an emerging adult sample. We tested the following two hypotheses: (1) experiencing more severe social-evaluative and life-threatening stressors would be associated with a heightened ERN and (2) the developmental timing of exposure to these forms of adversity would influence their associations with the ERN, with stronger associations being observed for stressor severity occurring during early adolescence. Our findings suggested that developmental timing and stressor type may play important roles in ERN magnitude. Specifically, more severe social-evaluative stress occurring during early adolescence was associated with a larger ERN in young adulthood, as was early adolescence total stress severity. These results provide support for the notion that early adolescence is a sensitive period during which cumulative and social stress may more significantly influence neural networks involved in performance monitoring.

Although this study suggests that broad categories of stressors can influence ERN magnitude into young adulthood, it is unclear whether these effects can also be seen with more specific types of stressors, such as parenting, and whether such stress can have lasting impacts on the ERN that persist beyond childhood (Brooker & Buss, 2014b; Chong et al., 2020; Meyer et al., 2015, 2019) and into adulthood. Lastly, mechanisms through which stress can affect the ERN were not investigated in Study One. It is hypothesized that chronic harsh punishment of mistakes may impact neural performance monitoring networks by sensitizing individuals to their performance (Brooker & Buss, 2014b; Meyer et al., 2015, 2019), although this has yet to be confirmed. To address these gaps, Study Two investigated whether punitive parenting—a specific social stressor—may be associated with an enhanced ERN in adult offspring. Harsh

parenting has been associated with an enhanced offspring ERN (Brooker and Buss, 2014; Meyer et al., 2015), possibly through heightened fear of punishment following mistakes. In Banica et al. (2019), we investigated whether individuals who experienced punitive parenting as children displayed long-term alterations in performance monitoring. We analysed associations between the ERN in emerging adults and two critical dimensions of parenting—care and authoritarianism/overprotection (Parker, 1979, 1990; Parker et al., 1979)—for mothers and fathers separately. Authoritarian and overprotective parenting is often associated with harshly disciplining children in an attempt to regulate their behavior (Baumrind, 1971; Borelli et al., 2015; Frías-Armenta et al., 2004); we thus investigated whether experiencing this kind of parenting would promote long-term neural adaptations in performance monitoring networks.

Study Two

Overprotective/Authoritarian Maternal Parenting is Associated with an Enhanced Error-Related Negativity (ERN) in Emerging Adult Females

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Banica, I., Sandre, A. & Weinberg, A. (2019). Overprotective/authoritarian maternal parenting is associated with an enhanced error-related negativity (ERN) in young adult females. *International Journal of Psychophysiology*, *137*, 12 – 20. https://doi.org/10.1016/j.ijpsycho.2018.12.013

Abstract

Error monitoring is crucial for survival and adaptation, and can be indexed by the error-related negativity (ERN), a fronto-centrally located negative deflection in the event-related potential (ERP) waveform that differentiates erroneous from correct responses within 100 ms of a response. The ERN is seen as an early neural signal indicating the need to adjust performance and increase executive control. Previous findings indicate that punishing errors increases ERN magnitude, and that punitive parenting predicts an enhanced ERN in children. If punitive parenting can in fact sensitize children to error commission over the long term, an enhanced ERN should be seen in adults who experienced harsh parenting as children. The present study thus sought to establish whether punitive parenting is associated with an enhanced ERN in emerging adulthood. A sample of 70 emerging adult females reported on their mothers' and fathers' parenting styles separately and performed a flanker task to elicit the ERN. Higher reported overprotective/authoritarian maternal behavior was associated with an enhanced ERN. These results provide further support for the hypothesis that punitive parenting may lead to long-term sensitization of neural networks involved in performance monitoring.

1. Introduction

Monitoring one's actions and learning from mistakes is crucial for adapting to our demanding environments (Botvinick et al., 2001; Falkenstein et al., 2000; Holroyd and Coles, 2002; van Veen and Carter, 2002). Before an adaptive response to errors can be mounted, however, one must identify the discrepancy between intended and actual behaviors (Falkenstein et al., 2000; Holroyd and Coles, 2002); that is, the error must be registered. The error-related negativity, or ERN, is a fronto-centrally maximal negative deflection in the event-related potential (ERP) that differentiates erroneous from correct responses within 100 ms of response onset (Falkenstein et al., 1991; Gehring et al., 1995). A wealth of evidence suggests that the ERN is generated by the Anterior Cingulate Cortex (ACC), an information-integration region in the medial frontal cortex that guides behavior (Brázdil et al., 2005; Dehaene et al., 1994; Shenhav et al., 2013; Stemmer et al., 2003). There are multiple theories of the functional significance of the ERN, but it is generally seen as an early signal indicating the need for behavioral adjustment and increased executive control (Carter and Van Veen, 2007; Dehaene, 2018; Holroyd and Coles, 2002; Lo, 2018).

Previous studies have shown that the magnitude of the ERN can be increased by experimental factors that change the significance of errors, as when correct responses are rewarded (Ganushchak and Schiller, 2008; Hajcak et al., 2005), when accuracy is emphasized over response speed (Gehring et al., 1993), or when performance is evaluated (Hajcak et al., 2005). Additionally, punishment (e.g., with electric shock, or a noxious sound) appears to increase the magnitude of the ERN, an effect that persists even after errors are no longer punished (Meyer and Gawlowska, 2017; Riesel et al., 2012; Riesel et al., in prep). This suggests that learning experiences that increase the negative consequences of errors can have a lasting

impact on the way in which the brain responds to mistakes (Enns et al., 2000; Frost et al., 1991; Rice et al., 1996).

Consistent with these experimental data, there is evidence that harsh parenting styles—characterized, in part, by critical, punitive parental reactions to children's mistakes (Kawamura et al., 2002; Robinson et al., 1995; Thompson et al., 2003)—are associated with an enhanced ERN in children (Brooker and Buss, 2014; Meyer et al., 2015). This is consistent with developmental theories suggesting that harsh parenting breeds fear of punishment in offspring (Hoffman, 1983), which may increase self-monitoring and sensitize children to error commission to avoid parental punishment (Frost et al., 1991; Enns et al., 2000; Kawamura et al., 2002; Rice et al., 1996), thereby potentiating the ERN (Brooker and Buss, 2014).

If increased fear of punishment does lead to heightened self-monitoring over the long term, an enhanced ERN in individuals who experienced punitive parenting should persist into adulthood; however, this has not yet been demonstrated. Moreover, “harsh parenting” may consist of multiple dimensions, including high demandingness, low warmth, low responsiveness to the child's needs and/or emotions, high levels of criticism and control, and harsh discipline including shouting or corporal punishment (Baumrind, 1971; Brooker and Buss, 2014; Frías-Armenta et al., 2004; Giles-Sims et al., 1995; Levinson et al., 2017; Mackenbach et al., 2014; Thompson et al., 2003). Yet distinct aspects of parenting styles may influence child development in different ways (Enns et al., 2002; Rapee, 1997; Silove et al., 1991).

To this end, in the present study, we investigated associations between the ERN in emerging adults and two critical dimensions of parenting: authoritarianism/overprotection, and care (Parker, 1979; Parker, 1990; Parker et al., 1979). The “authoritarianism/over-control” dimension of parenting refers to the degree to which parents try to exert control over their

children, shield them from experiences, and discourage independence (high overprotection/authoritarianism; Kendler, 1996; Parker et al., 1979). Overprotective and authoritarian parenting is often associated with harshly disciplining children in an attempt to regulate their behavior (Baumrind, 1971; Borelli et al., 2015; Frías-Armenta et al., 2004), as well as with the development of anxiety disorders (Parker, 1979; Silove et al., 1991), which have consistently been linked to an enhanced ERN (Carrasco et al., 2013; Endrass et al., 2010; Gehring et al., 2000; Hajcak et al., 2008; Hajcak and Simons, 2002; Meyer et al., 2012; Moser et al., 2013; Olvet and Hajcak, 2008). We thus hypothesized that individuals who reported having experienced authoritarian/overprotective parenting in childhood would display a larger ERN in young adulthood.

On the other hand, the "care" dimension of parenting refers to the degree to which parents are affectionate, empathetic, and emotionally warm versus cold, indifferent, and rejecting toward their offspring. While low care is characterized by negative parenting behaviors such as indifference and neglect, it is independent of punitive behaviors and overly protective tendencies (Kendler, 1996; Parker et al., 1979). Because previous work suggests that *punishment* is related to an enhanced ERN magnitude, we did not have reason to suspect that the care dimension of parenting would be related to performance monitoring, and hypothesized that parental care scores would not be associated with the ERN in our sample.

And finally, previous studies investigating associations between parenting and the ERN have looked at the impact of one parent's behavior on children's neural responses to errors (Brooker and Buss, 2014; Meyer et al., 2015). However, some research suggests that maternal and paternal behavior can impact children's psychological functioning in different ways (Enns et al., 2002). To get a clearer picture of the unique contributions of both maternal and paternal

behavior on the ERN, the present study measured parenting behavior separately for participants' mothers and fathers. Because previous research has shown that recalled maternal behavior is more strongly associated with psychopathology in adult offspring than paternal behavior (Enns et al., 2002; Kerver et al., 1992), we hypothesized that maternal parenting styles would be more strongly associated with the magnitude of the ERN than paternal behavior.

2. Materials and methods

2.1. Participants

Eighty-three participants were recruited from the McGill University psychology human participant pool. Participants received course credit for their time. All participants provided written informed consent after review of the protocol and all procedures were approved by the Research Ethics Board at McGill University.

Of the eighty-three participants, seventy-nine were female and four were male. Previous research has demonstrated sex differences in ERN magnitude as well as associations between the ERN and individual difference variables (Larson et al., 2011; Moran et al., 2012; Moser et al., 2016); we thus excluded the four male participants from our sample, as their small number left us underpowered to adequately investigate sex/gender effects. Following this, data from three female participants were excluded due to excessive noise in the EEG data, one participant was excluded for not completing the task, three participants were excluded because they were taking antidepressant medication, and one participant was excluded because she did not follow questionnaire instructions. To elicit a reliable ERN, six or more errors trials are required (Meyer et al., 2013; Olvet and Hajcak, 2009; Sandre et al., in prep); therefore, one participant was excluded for committing too few errors.

The final sample included 70 female participants. For analyses of behavioral data, three participants were excluded because their performance was more than three standard deviations above the sample mean. Sixty-seven participants were therefore included in the behavioral analyses. The mean age of the 70-person sample was 20.3 (*SD* = 1.60) years. 57% of participants were Caucasian, 16% were Chinese, 7% were South Asian, 6% were Arab/West Asian, 3% were Hispanic, 1% were South East Asian, 1% were Caribbean, 1% were Native/First Nations/Aboriginal, 6% indicated they were another ethnicity, and 1% did not indicate their ethnicity.

2.2. Measures

All participants completed the Parental Bonding Instrument (PBI; Parker et al., 1979). The PBI is a 25-item self-report scale designed to measure parental behavior during the first 16 years of life as perceived by adult offspring. Per previous research (Gotlib et al., 1988; Mackinnon et al., 1989; Parker et al., 1979), we used the PBI to measure two dimensions of parenting—care and overprotection/authoritarianism—assessed separately for each parent. Average scores for care and overprotection/authoritarianism were obtained for each parent, and each item was rated on a 4-point Likert scale ranging from 1 (“very unlike”) to 4 (“very like”). Greater care scale scores indicate higher levels of parental care and warmth, while greater scores on the overprotection/authoritarianism scale indicate more overprotective and authoritarian behaviors. Two participants did not report on their fathers' parenting behaviors, and one participant answered questions related to paternal care but not paternal overprotection/authoritarianism; for the former two participants, we only analyzed associations with maternal parenting. In our final sample, 70 participants reported on maternal behavior, 68

participants reported on paternal care, and 67 participants reported on paternal overprotection/authoritarianism.

The PBI has demonstrated high internal consistency, as well as short- and long-term test-retest reliability (Parker, 1979; Wilhelm and Parker, 1990). Furthermore, scores on the PBI have been shown to correlate with parent and sibling reports in clinical and nonclinical populations (Parker, 1981, Parker, 1989). Several studies have also indicated that the PBI is not influenced by current mood state (Gerlsma et al., 1993; Gotlib et al., 1988; Parker, 1981).

2.3. Task and materials

An arrow version of the flanker task (Eriksen and Eriksen, 1974) was administered on an Intel Core i7 computer using Presentation software (Neurobehavioral Systems, Inc.; Albany, CA) to control the presentation and timing of all stimuli. Each stimulus was displayed on a 19 in (48.3 cm) monitor. On each trial, five horizontally aligned arrowheads were presented in the center of the screen, and targets were always the center arrow. Participants used a computer mouse to indicate the direction of the target arrow (i.e. press 'left mouse button' if arrow points to the left). Half of all trials were congruent ("<<<<<" or ">>>>>") and half were incongruent (">><>>" or "<<><<"); the order of congruent and incongruent trials was random. Congruent and incongruent trials were distributed equiprobably within blocks. All stimuli were presented for 200 ms followed by an interval that varied randomly from 2300 to 2800 ms. Participant response type (correct or incorrect) and reaction time (in ms) on every trial was recorded for later analysis.

2.4. Procedure

Following a brief description of the experiment, electroencephalograph (EEG) sensors were attached, and the participant was given detailed task instructions. Participants were required

to indicate the direction of the central arrowhead using the left or right mouse button. Participants then performed a practice block containing 10 trials and were instructed to be both as accurate and fast as possible. The actual task consisted of 11 blocks of 30 trials (330 trials total) with each block initiated by the participant. To encourage both fast and accurate responding, participants received feedback based on their performance at the end of each block. If performance was 75% correct or lower, the message "Please try to be more accurate" was displayed; performance above 80% correct was followed by "Please try to respond faster"; otherwise, the message "You're doing a great job" was displayed.

All participants performed multiple additional tasks during the experiment: Island Getaway (Kujawa et al., 2014; Ethridge et al., 2017), in which participants play a "Survivor"-type game and receive social feedback; Emotional Interrupt (Mitchell et al., 2006; Mitchell et al., 2008), in which participants view emotionally salient photos; and Doors (Hajcak Proudfit, 2015), a monetary reward task. The order of the tasks was counterbalanced across subjects. Data from additional tasks are reported elsewhere (e.g., Ethridge and Weinberg, 2018).

2.5. Electroencephalographic recording and data processing

Continuous EEG was recorded using a 32-electrode cap and a BrainVision actiCHamp system (Brain Products, Munich, Germany) based on the standard 10/20 layout, with active Ag/AgCl sensors and a ground electrode at Fpz. The electrooculogram (EOG) generated from eye movements and blinks was recorded using facial electrodes placed approximately 1 cm above and below one eye (VEO) and 1 cm to the right and left of both eyes (HEO). All electrode impedances were below 10 kΩ, and data were recorded with a sampling rate of 1000 Hz. Offline analysis was conducted using BrainVision Analyzer software (Brain Products, Munich, Germany). Data were referenced to the average of TP9 and TP10, and continuous

(nonsegmented) data were band-pass filtered with low and high cutoffs of 0.1 and 30 Hz, respectively, using a Butterworth zero phase filter with a 12 db/octave roll-off. Eye-blink and ocular corrections were conducted using HEO and VEO per a modification of the formula described in Gratton et al. (1983).

A semi-automatic procedure was employed to detect and reject artifacts. The criteria applied were a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a minimum voltage difference of less than 0.50 μV within 100 ms intervals. These intervals were rejected from individual channels in each trial. Visual inspection of the data was then conducted to detect and reject any remaining artifacts.

The EEG was segmented into 1500 ms windows for each trial, beginning 500 ms before each response onset and continuing for 1000 ms. Error and correct trials were then separately averaged. The mean activity in a 200 ms window from −500 to −300 ms prior to response onset served as the baseline and was subtracted from each data point. Based on visual inspection of the grand averaged data, and in line with previous studies (Weinberg and Hajcak, 2011), 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 maximal in previous studies on the ERN (Riesel et al., 2013; Weinberg et al., 2012a; Weinberg et al., 2010). In addition, the correct response negativity (CRN) was evaluated in the same time window and sites on correct trials. The CRN appears to indicate generic response monitoring (Simons, 2010), and a negative deflection in the ERP is typically present following both correct and error trials (Burle et al., 2008).

Finally, the error positivity (Pe), a centroparietally-located positive deflection appearing after error commission, was quantified on error trials as the average activity from 200 to 400 ms at electrode site Pz (Falkenstein et al., 1991; Riesel et al., 2013). The correct positivity (Pc) was

quantified on correct trials as the average activity from 200 to 400 ms at electrode site Pz. The Pe appears to index error awareness, sustained processing, and elaboration (Endrass et al., 2007; Hughes and Yeung, 2011; Nieuwenhuis et al., 2001; Riesel et al., 2013). The Pe was included in these analyses to determine whether the hypothesized effects were specific to the ERN or might also be reflected in later stages of error processing and performance monitoring. The minimum number of correct trials at both Cz and Pz for our sample was 217, and the maximum was 324. The average number of correct trials was 285.67 at Cz and 285.59 at Pz. The minimum number of error trials at Cz and Pz was 6, while the maximum was 75. The average number of erroneous trials was 36.07 at Cz and 36.04 at Pz.

The residual method of calculating the ERN and Pe appears to identify brain activity that specifically indexes error processing, as it creates scores unrelated to correct response activity, and therefore provides a more reliable ERP measure (Meyer et al., 2017). To quantify error-specific neural activity, a regression-based procedure (Meyer et al., 2017; Weinberg et al., 2015) was used to compute standardized residuals of the ERN and Pe. To calculate ERN_{resid}, participants' CRN was entered as the predictor, and the ERN was the dependent variable; the ERN_{resid} scores are the saved standardized residuals from this regression. To calculate Pe_{resid}, participants' Pc (correct positivity) was entered as the predictor, and the Pe was the dependent variable; the saved standardized residuals were used as the Pe_{resid}.

The ERP components of interest in the present sample demonstrated good internal consistency. The split-half reliability Spearman-Brown coefficient, calculated by examining associations between odd and even trials, was $r = 0.89$ for the ERN, $r = 0.98$ for the CRN and Pc, $r = 0.95$ for the Pe, $r = 0.76$ for the ERN_{resid}, and $r = 0.93$ for the Pe_{resid}.

Behavioral measures included the number of error trials for each subject, as well as accuracy expressed as a percentage of correct trials out of the total number of trials. Accuracy following correct and erroneous responses was also calculated (post-correct and post-error accuracy, respectively). Average reaction times (RTs) on error and correct trials were calculated separately. Reaction time on correct trials following errors was also evaluated. 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 analysis if RTs were faster than 200 ms or slower than 1000 ms. The maximum number of correct responses for the sample was 324, the minimum was 223, and the average was 286.26. The maximum number of erroneous responses was 76, the minimum was 6, and the average was 36.29.

2.6. Data analysis

All statistical analyses were conducted using IBM SPSS Statistics software (Version 24). Repeated-measures analyses of variance (ANOVAs) were used to compare within-participant response speed and accuracy following error and correct trials. Pearson's correlations were used to measure bivariate associations between ERP, behavioral, and questionnaire data. To examine unique associations between parenting and the ERN and Pe, we conducted two simultaneous multiple regressions (one for each component) with either the ERN_{resid} or Pe_{resid} as the dependent variable and maternal overprotection/authoritarianism, maternal care, paternal overprotection/authoritarianism, and paternal care as predictors.

3. Results

3.1. Behavioral data

Averages and standard deviations for number of error trials, total accuracy (correct trials out of total trials), accuracy following error trials, accuracy following correct trials, reaction times on error and correct trials, reaction times on trials following error and correct trials, and post-error slowing are presented in Table 1, as are correlations between study variables. Repeated-measures analyses of variance (ANOVAs) indicated that participants were more accurate on trials following errors than trials following correct responses, F (1, 69) = 18.69, $p < .001$, faster on error compared to correct trials, F (1, 66) = 474.71, $p < .001$, and faster on trials following correct responses than trials following errors, F (1, 66) = 42.22, $p < .001$.

3.2. Parenting behavior

Means and standard deviations of maternal and paternal PBI scales are presented in Table 1, as are correlations between study variables. As indicated, the two dimensions of maternal parenting behaviors were moderately negatively associated with one another, as were the two dimensions of paternal parenting behaviors. Greater maternal care was moderately associated with greater paternal care, and greater maternal overprotection/authoritarianism was related to greater paternal overprotection/authoritarianism. However, none of these associations were greater than |0.40|, suggesting these are related but non-redundant measures. Paired samples *t*-tests indicated that mothers were rated as significantly more caring, $t(67) = 2.40$, $p < .05$, and more overprotective/authoritarian, $t(66) = 2.77$, $p < .01$, than fathers.

Fig. 1(a) displays waveforms of the ERN, CRN, and ERN minus CRN difference, and a topographical map depicting error minus correct activity, for all participants. In examining the associations between parenting and error-related brain activity, we observed a significant negative correlation between maternal overprotection/authoritarianism and

ERN_{resid}, $r = -0.24$, $p < .05$, such that higher levels of reported maternal overprotection/authoritarianism were related to a more negative ERN_{resid} (i.e., a larger ERN_{resid}), as depicted in the scatterplot in Fig. 1(b). Maternal care was not significantly associated with the magnitude of the ERN_{resid}, nor was either dimension of paternal parenting behaviors. Higher levels of paternal care were, however, significantly and positively associated with the magnitude of the Pe_{resid}, $r = 0.30$, $p < .05$, such that greater paternal care was associated with a more positive Pe_{resid}, as depicted in the scatterplot in Fig. 1(c). Paternal overprotection/authoritarianism was also significantly negatively correlated with post-error slowing, $r = -0.26$, $p < .05$, such that greater paternal overprotection/authoritarianism was associated with less response slowing following erroneous responses, as depicted in the scatterplot in Fig. 1(d).

To determine whether maternal overprotection/authoritarianism predicts ERN_{resid} amplitude over and above other adverse parenting practices, a multiple linear regression was calculated predicting ERN_{resid} magnitude from maternal and paternal care and overprotection/authoritarian scores. Results of this analysis are presented in Table 2. Only maternal overprotection/authoritarianism significantly predicted ERN_{resid} magnitude, $\beta = -0.27$, $p < .05$. Maternal care and paternal variables did not significantly and uniquely predict the ERN_{resid}; however, after controlling for associations with other predictors, a positive association with paternal care and the ERN_{resid} emerged that was of a similar magnitude to the association with maternal overprotection/authoritarianism, $\beta = 0.24$, $p = .054$. Similarly, a multiple linear regression was conducted predicting Pe_{resid} magnitude from maternal and paternal care and overprotection/authoritarian scores. Results of this analysis are also presented in Table 2. Only

paternal care significantly predicted Pe_{resid} magnitude, $\beta = 0.30$, $p < .05$. Paternal overprotection/authoritarianism and maternal variables did not significantly predict the Pe_{resid}.

4. Discussion

The present study investigated the relationship between neural response to errors in emerging adulthood and two dimensions of parenting – care and overprotection/authoritarianism – for mothers and fathers separately. As hypothesized, overprotective/authoritarian parental behavior – a dimension of parenting characterized, in part, by harsh discipline (Baumrind, 1971; Borelli et al., 2015; Frías-Armenta et al., 2004) – was associated with an enhanced ERN_{resid}. Specifically, higher levels of maternal overprotection/authoritarianism, and not maternal care, were related to increased neural response to errors in female offspring, and only this dimension of parenting was significantly associated with ERN_{resid} magnitude. This finding is consistent with previous work on punishment, punitive parenting, and enhanced neural response to errors (Brooker and Buss, 2014; Meyer et al., 2015; Meyer and Gawlowska, 2017; Riesel et al., 2012), but extends these findings by suggesting that overprotective/authoritarian parenting in childhood is associated with the altered function of neural performance monitoring networks in adulthood.

These findings, combined with previous work indicating that punitive parenting is associated with an enhanced ERN years later in children (Meyer et al., 2015), suggest one potential developmental pathway to exaggerated activity in neural performance monitoring networks. Parents who endorse authoritarian behavior are more likely to believe that their children should strive to be flawless (Robin et al., 1990); consistent with this, authoritarian parenting can involve harsh punishment and focusing on children's mistakes (Baumrind, 1971; Borelli et al., 2015; Frías-Armenta et al., 2004). Parental punishment may increase both

children's fear of making errors and anxiety about the consequences of errors. This may, in turn, lead children to engage in increased self-monitoring to avoid mistakes, and promote a catastrophizing attitude toward errors over the long term (Brooker and Buss, 2014; Fox, 2010; Stahl et al., 2015; Schrijvers et al., 2010). Growing up in an environment where errors are punished and catastrophized may thus lead children to overvalue perfect performance (Brooker and Buss, 2014; Frost et al., 1991; Meyer et al., 2015; Rice et al., 1996).

This exaggerated activity in neural performance monitoring networks might also be reinforced through overprotective maternal behavior. Overly involved mothers may attempt to control their children's behavior and solve their problems for them to promote success and prevent distressing things from happening (Segrin et al., 2012; Segrin et al., 2013; Rapee, 2009). This can prevent children from pursuing situations where they can make mistakes and use problem-solving skills to correct them (Cooklin et al., 2013; Feng et al., 2008; LeMoyne and Buchanan, 2011). Alternatively, overprotective maternal behavior may increase offspring error salience through modeling. Overly involved and controlling mothers tend to be risk-aversive and preoccupied with their own performance (Little, 2015; Rapee et al., 2009; Segrin et al., 2012; Soenens et al., 2005), and children can adopt their mothers' fearful and perfectionistic attitudes (de Rosnay et al., 2006; Frost et al., 1991; Gerull and Rapee, 2002). Thus, overprotection may not only lead to ineffective coping skills in offspring (Segrin et al., 2012; Segrin et al., 2013), but also reinforce the notion that errors are catastrophic (Brooker and Buss, 2014; Fox, 2010; Stahl et al., 2015; Schrijvers et al., 2010; Weinberg et al., 2016).

In the present study, we did not observe an association between paternal overprotection/authoritarianism and the ERN_{resid}. On the other hand, participants who reported more overprotective/authoritarian paternal behavior slowed down significantly less following

error trials, possibly reflecting reduced behavioral regulation and adjustment to avoid future errors (Gadeyne et al., 2004; Rinaldi and Howe, 2012). The association with maternal overprotection was in the same direction, though not significant. Thus, overprotective/authoritarian parenting behavior may be related to exaggerated neural responses to errors without any attendant improvement in task performance for offspring. While some research indicates that ERN magnitude is associated with increased post-error slowing and fewer errors committed, other studies have found no links between magnitude of the ERN and behavioral data (see Cavanagh and Shackman, 2015 and Weinberg et al., 2012b for reviews). Thus, these results are consistent with the idea that the ERN is an early evaluative signal that a mistake has been made and behavior *should* be adjusted (Burle et al., 2008), but that variation in the magnitude of the component does not strongly predict performance (Allain et al., 2004; Simons, 2010; Weinberg et al., 2016).

While we found no significant associations between maternal warmth and neural indices of performance monitoring, we did observe a trend-level effect of paternal care predicting ERN_{resid} magnitude, whereby less caring paternal behavior was associated with a larger ERN_{resid} amplitude. We also observed *significant* associations with paternal behavior that we had not hypothesized. For instance, in these data, less paternal care significantly predicted a smaller Pe_{resid} following error commission. As these associations were not hypothesized a priori, we present the following speculations with caution. The Pe is thought to index elaborative error processing and awareness, intended to aid with problem-solving and behavioral adjustment to avoid erring in the future (Endrass et al., 2007; Falkenstein et al., 2000; Hughes and Yeung, 2011; Nieuwenhuis et al., 2001; Riesel et al., 2013). There is evidence that warm, sensitive parenting is associated with better problem-solving and executive functioning in offspring

(Bernier et al., 2010; Raikes and Thompson, 2008; Ruchkin et al., 1999). The fact that authoritarian/overprotective parenting was unrelated to Pe_{resid} magnitude in our sample is in line with research indicating that Pe amplitude is not altered under conditions of punishment (Riesel et al., 2012). As participants who experienced less caring paternal behavior displayed a marginally larger ERN_{resid} and a significantly smaller Pe_{resid}, future studies should investigate the possibility that paternal warmth promotes the development of more adaptive activity in neural networks involved in self-regulation and problem-solving. However, we would note here that we did not observe these same patterns of association with maternal warmth.

Overall, our findings suggest that overprotective and authoritarian parenting may lead to both enhanced neural error monitoring and less adaptive behavioral recovery from errors, while less caring parental behavior may interfere with sustained neural processing of errors in service of improving performance. The fact that maternal and paternal behaviors seem to significantly predict different neural indices of performance monitoring may reflect common differences in parental roles. For instance, mothers are more likely to be the primary caregivers, typically spending more time with their children than fathers do (Coyl-Shepherd and Newland, 2013; Enns et al., 2002; Arendell, 2000; Umemura et al., 2013), and mothers and fathers tend to display different kinds of behaviors when interacting with their children (Lamb, 2010). However, effects of maternal and paternal behaviors may not be as specific as we had hypothesized: The non-statistically significant effect of paternal care on ERN_{resid} magnitude was similar in size to the effect of maternal overprotection/authoritarianism on ERN_{resid} magnitude, which did reach conventional significance levels. Future studies should investigate the specificity of other kinds of parental behavior (for instance, style of play with offspring) on neural indices of early and later performance monitoring in other adult samples. As fathers increasingly assume greater

caregiving responsibility (Cabrera et al., 2000; Cabrera et al., 2018), it may be possible to decouple the effects of gender and primary caregiver. Nonetheless, even in this sample we found significant effects of paternal styles that we had not hypothesized, suggesting fathers likely have more of an impact on offspring neural development than is often thought (Cabrera et al., 2018).

Limitations of the study suggest future directions. For instance, our final sample was all female; the findings reported here may therefore reflect the effects of parenting on female offspring only. Previous research suggests that females interact with their parents differently than males, and receive different treatment in turn; for instance, daughters experience less permissive and more authoritative parenting than do sons (McKinney et al., 2018). Male and female psychological outcomes seem to differ based on whether they receive authoritarian parenting from their same- or opposite-sex parent (Wintre and Gates, 2006). Additionally, previous literature (e.g. Larson et al., 2011; Imburgio et al., under review) highlights potential sex differences in the ERN, with men and women displaying different average ERN magnitudes. And, importantly, associations between the ERN and individual differences are not identical between males and females (Moran et al., 2012; Moser et al., 2016). Future studies should aim to further understand whether the observed associations truly reflect the effects of maternal or paternal behavior specifically, or whether our results reflect the outcomes of interactions between mothers and fathers and their daughters specifically.

Further, the Parental Bonding Instrument is a retrospective self-report measure and may not provide an objective measure of parenting behavior, as participants may be biased in recalling this information. However, there are data showing that PBI scores correlate with sibling and parent reports (Parker, 1981, Parker, 1989), and are independent of current mood state (Gerlsma et al., 1993; Gotlib et al., 1988; Parker, 1981). Moreover, the results of the present

study suggest specific patterns of associations between the ERN_{resid} and subscales of the PBI; in other words, it was not the case that people who globally rated their parents more negatively had an enhanced ERN_{resid}. Lastly, the PBI assesses parenting behaviors during participants' first 16 years of life, which encompasses a broad developmental period, making it difficult to conclude whether parenting during specific time periods explains the observed associations. Despite this, we would note that information about participants' *perceptions* of their parents' behaviors may be more informative for offspring psychological outcomes (e.g. McClure et al., 2001). Nonetheless, studies on this topic should closely examine the timing of parental behaviors on neural markers of error monitoring, and include parent- and self-report questionnaires assessing perceptions of parenting, as well as objective observational measures.

In sum, the present study provides further empirical support for evidence that adverse parenting practices relate to neural responses to errors by showing that maternal overprotective/authoritarian behavior is associated with increased error monitoring beyond childhood and into adulthood. However, non-significant but comparably-sized associations suggest that effects of maternal and paternal behaviors may not be as specific as we had expected. These findings could be particularly useful in identifying areas to be targeted by interventions aimed at preventing the development of anxiety disorders (Carrasco et al., 2013; Endrass et al., 2010; Gehring et al., 2000; Hajcak et al., 2008; Hajcak and Simons, 2002; Meyer et al., 2012; Moser et al., 2013; Olvet and Hajcak, 2008). Long-term exposure to parenting styles characterized by punishment may be a potentially important pathway to the development of an enhanced ERN magnitude in emerging adulthood.

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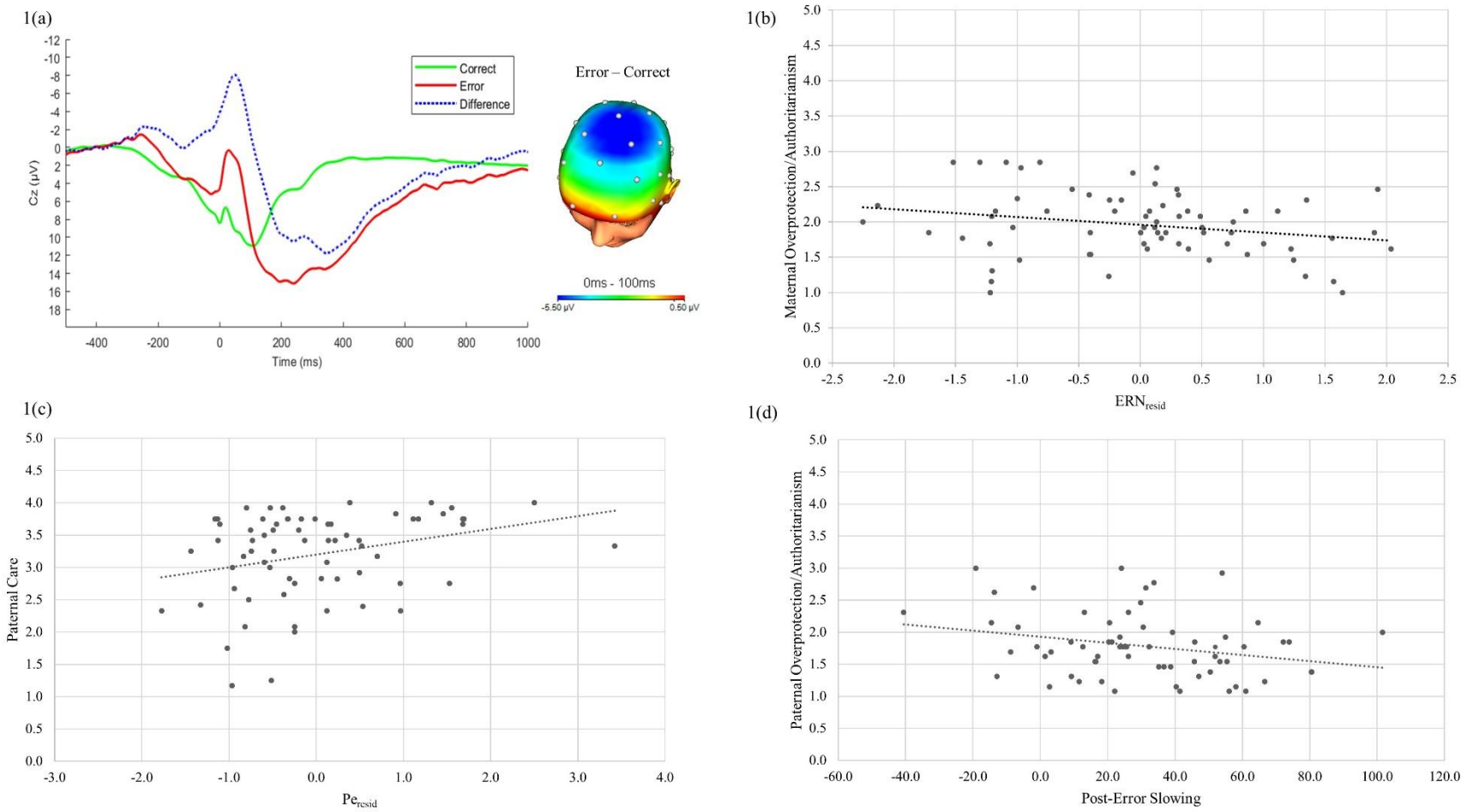

Fig. 1. (a) Response-locked ERP average waveforms following correct and incorrect responses, as well as the difference (error – correct) wave, at electrode site Cz, and a topographical map depicting average difference (μV) between correct and erroneous neural responses (error – correct) for all participants. (b) The association between maternal overprotection/authoritarianism scores measured in the Parental Bonding Instrument (PBI) and the residual error-related negativity (ERN_{resid}). (c) The association between paternal care scores in the PBI and the residual error positivity (Pe_{resid}). (d) The association between paternal overprotection/authoritarianism scores in the PBI and post-error slowing (in msec).

Table 1

Correlational table depicting associations between the residual error-related negativity, residual error positivity, parental behavior, and flanker task behavioral data.

	Mean (SD)	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**	**7.**	**8.**	**9.**	**10.**	**11.**	**12.**	**13.**	**14.**
1. Maternal Care	3.41 (0.53)														
2. Maternal Overprotection/ Authoritarianism	1.96 (0.46)	-0.25*													
3. Paternal Care	3.20 (0.67)	0.25*	-0.08												
4. Paternal Overprotection/ Authoritarianism	1.77 (0.50)	-0.21	0.39**	-0.23											
5. Total errors	38.36 (13.04)	0.07	-0.09	0.00	0.01										
6. Accuracy	0.88 (0.05)	-0.09	0.09	-0.04	0.06	-0.87**									
7. Post-correct accuracy	0.88 (0.04)	-0.07	0.10	-0.02	0.00	-0.99**	0.86**								
8. Post-error accuracy	0.91 (0.06)	-0.07	-0.03	0.10	-0.02	-0.55**	0.68**	0.45**							
9. Correct reaction time	372.03 (32.90)	-0.12	0.02	-0.16	-0.12	-0.20	0.19	0.22	0.04						
10. Error reaction time	299.75 (25.02)	0.04	-0.04	0.14	-0.11	-0.25*	0.23	0.25*	0.16	0.59**					
11. Post-error reaction time	378.84 (40.09)	-0.01	-0.01	0.02	-0.18	-0.16	0.15	0.17	0.03	0.82**	0.56**				
12. Post-correct reaction time	361.89 (31.37)	-0.10	0.002	-0.13	-0.15	-0.32**	0.30*	0.33**	0.12	0.96**	0.65**	0.85**			
13. Post-error slowing	30.19 (27.48)	0.11	-0.20	-0.01	**-0.26***	0.07	-0.20	-0.06	-0.18	0.26*	-0.04	0.39**	0.17		
14. ERN_{resid}	0.00 (1.00)	0.07	**-0.24***	0.17	0.01	0.22	-0.23	-0.21	-0.16	0.21	0.21	0.07	0.18	-0.07	
15. Pe_{resid}	0.00 (1.00)	0.15	-0.19	**0.30***	0.03	-0.04	0.16	0.03	0.19	-0.17	-0.09	-0.11	-0.12	-0.26*	0.13

Note. For correlations between maternal behavior, ERN, and Pe, N = 70. For correlations between paternal care, ERN, and Pe, N = 68. For correlations between paternal overprotection/authoritarianism, ERN, and Pe, N = 67. For correlations between parental behavior, ERN, Pe, and behavioral data, N = 67.
Bolded numbers indicate statistically significant correlations between parental variables and ERP and behavioral variables.
$*$ Indicates $p < .05$.
$**$ Indicates $p < .01$.

Table 2

Results of two simultaneous linear regressions predicting the residual error-related negativity and error positivity, respectively, from maternal and paternal care and overprotection/authoritarianism.

	Error-Related Negativity (ERN_{resid})			**Error Positivity (Pe_{resid})**		
Predictor	***b* (SE)**	***B***	**VIF**	***b* (SE)**	***β***	**VIF**
Maternal Care	0.07 (0.24)	0.03	1.11	0.10 (0.24)	0.05	1.11
Maternal Overprotection/ Authoritarianism	-0.60 (0.29)	**-0.27***	1.22	-0.48 (0.29)	-0.22	1.22
Paternal Care	0.39 (0.20)	0.24†	1.08	0.48 (0.19)	**0.30***	1.08
Paternal Overprotection/ Authoritarianism	0.36 (0.27)	0.18	1.24	0.39 (0.26)	0.20	1.24
	Total R^2 =0.13; $F(4, 62) = 2.26$, $p = 0.07$			Total R^2 =0.14; $F(4, 62) = 2.56$, $p = .047$		

Bolded numbers indicate statistically significant correlations between parental variables and ERP and behavioral variables.

∗ Indicates p < .05.

† Indicates p = .05.

Preface to Study Three

In Study Two, we investigated associations between ERN magnitude and maternal and paternal behaviours in a young adult sample, hypothesizing that maternal behaviour characterized by harsh punishment would be associated with an enhanced ERN. Our findings suggest that maternal overprotective/authoritarian behavior was associated with increased error monitoring beyond childhood and into adulthood. First, the data provide further support for the idea that social-evaluative stress is associated with enhanced ERN magnitude, and that specific types of stress (i.e., parental behaviour) can affect error monitoring. Second, the data point to an important developmental risk factor for an enhanced ERN in adulthood: Punitive parenting. The findings suggest that parental punishment may exert long-lasting influence on performance monitoring by altering ERN trajectory. This is theorized to occur due to harsh punishment of children's mistakes, which leads to hypervigilance to one's own performance years later.

Taken together, Studies One and Two highlight the potential role of social-evaluative stress in enhancing neural performance monitoring networks. Thus, interpersonal stress appears to be associated with both enhanced ERN magnitude (e.g., Brooker & Buss, 2014b; Buzzell et al., 2017; Hajcak et al., 2005; Meyer et al., 2015, 2019; Schillinger et al., 2016) and increased anxiety disorder symptoms (Farmer & Kashdan, 2015; Hamilton et al., 2016; Kendler et al., 2003; Siegel et al., 2009; Uliaszek et al., 2010). Second, the ERN may be an important risk marker for anxiety (Olvet & Hajcak, 2008; Riesel et al., 2011, 2015), but not everyone with an enhanced ERN has an anxiety disorder (Carrasco et al., 2013; Hajcak et al., 2008; Kujawa et al., 2016; Riesel et al., 2011, 2015), suggesting that it is a diathesis that requires a catalyst (e.g., exposure to a subsequent stressor) to confer risk for anxiety. Social stress might be such a catalyst. Limited work has investigated interactions between the ERN and stress (Meyer et al.,

2017a), and it is unclear whether interpersonal stress may interact with ERN magnitude to heighten risk for anxiety.

To address this gap, Study Three investigated whether the ERN measured during participants' first term in university moderates the relationship between past-year social stress and anxiety symptoms at the end of the year. The transition to university is accompanied by heightened interpersonal stress, making this an ideal time to investigate this research question. We predicted that individuals experiencing heightened social stress would report greater later anxiety symptoms when they displayed an enhanced ERN, whereas individuals with a smaller ERN would not report heightened anxiety following increased social stress exposure. We controlled for baseline anxiety symptoms at the start of the academic year, and conducted analyses to examine whether our results were specific to social, and not non-social, stress.

Study Three

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

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Banica, I., Sandre, A., Shields, G.S., Slavich, G.M., & Weinberg, A. (2020). The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later. *International Journal of Psychophysiology*, *153,* 27 – 36. https://doi.org/10.1016/j.ijpsycho.2020.03.006

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, Riesel et al., 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 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, Meyer et al., 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, Endrass et al., 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, Weinberg et al., 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, Riesel et al., 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, Meyer et al., 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, Riesel et al., 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, Riesel et al., 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

[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).

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 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, Weinberg et al., 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 (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]

4. Discussion

[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 ($p = .10$).

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 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, Weinberg et al., 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, Riesel et al., 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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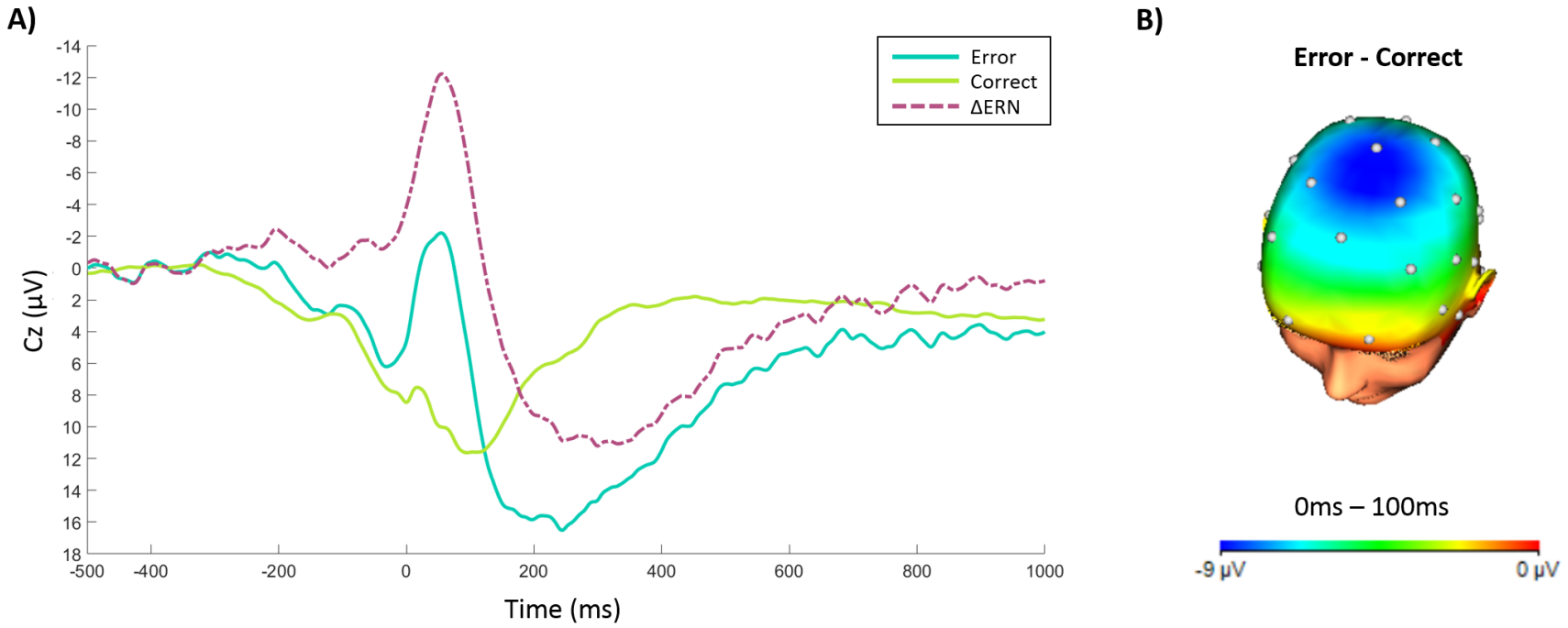

Fig. 1. A) Response-locked ERP average waveforms following error and correct responses, as well as the error minus correct difference wave (i.e., ΔERN), at electrode Cz. B) Topographic map depicting the average difference (μV) between error and correct responses from 0 ms to 100 ms post-response onset.

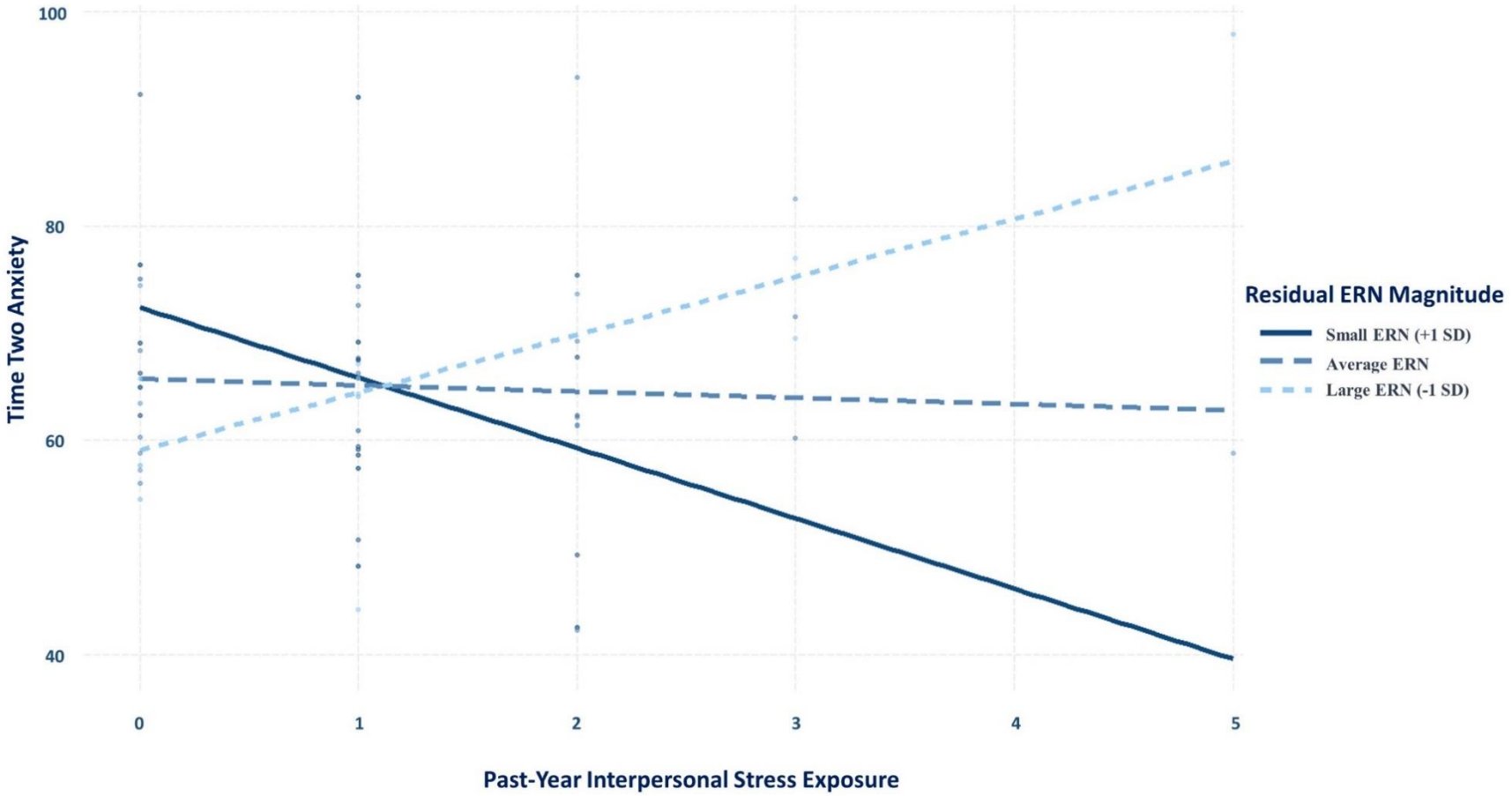

Fig. 2. Simple slopes depicting conditional effect of past-year interpersonal stress on Time 2 anxiety at large (-5.69), medium (0), and small (5.69) ERN magnitude values, controlling for Time 1 anxiety, gender, days between assessments, past-year non-interpersonal stress, and the interaction between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

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)	.60**							9.58	6.58	-7.25 – 29.86
3. ERN_{resid} (T1)	.80**	.00						0.00	5.69	-16.15 – 16.22
4. Anxiety symptoms (T1)	-.16	-.18	-.06					74.84	22.20	47.00 – 139.00
5. Anxiety symptoms (T2)	-.08	-.10	-.02	.71**				65.86	16.72	44.00 – 119.00
6. Total past-year interpersonal stressors (T2)	-.15	-.10	-.12	.24	.32*			1.23	1.18	0.00 – 5.00
7. Total past-year non-interpersonal stressors (T2)	-.03	-.12	.05	.18	.29*	.24		3.02	2.77	0.00 – 12.00
8. Time between symptom assessment (days)	-.06	.00	-.07	-.04	-.01	.07	.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.

** $p < .01$.

* $p < .05$.

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.

	B	***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.

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	.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.

General Discussion

Summary of Findings

The present thesis aimed to elucidate risk factors for anxiety by investigating environmental sources of variation in a neural marker of anxiety disorder risk, the ERN, and how stress may interact with the ERN to predict future anxiety symptoms. Studies One and Two demonstrated that social-evaluative stress is associated with enhanced performance monitoring in young adulthood, suggesting that this kind of stress may alter the brain's response to errors over the long term. Study Three demonstrated that neural indices of performance monitoring may confer risk for anxiety following the experience of social stress, suggesting that an enhanced ERN may be a diathesis that interacts with stress to increase anxiety.

More specifically, Study One demonstrated that experiencing more severe total and social-evaluative stress in early adolescence (ages 8 to 12) was associated with heightened ERN magnitude in young adulthood (around 18 years old). First, this suggests that early adolescence may be a vulnerable period during which general and social stress can alter neural pathways involved in performance monitoring more readily compared to other developmental periods. In line with this idea, sensitivity to social stress is heightened in adolescence. Second, the findings suggest that social-evaluative stress may be particularly salient, more readily leading to changes in neural pathways than other types of stress. Enhanced monitoring of one's performance in social situations may prevent mistakes that would result in negative evaluation and loss of status. Social stress in early adolescence may sensitize neural performance monitoring networks to errors over the long term, leading to enhanced performance monitoring and increased neural reactivity to mistakes in social settings. Importantly, our work points to the possibility that it is not just extreme stressors that can alter neural functioning—relatively common social stressors

such as bullying can also have a meaningful impact. Another notable contribution is the consideration of different types and developmental timing of stressors, which prior studies did not thoroughly investigate. Overall, Study One provides valuable insight into environmental sources of variation in ERN magnitude that may promote heightened risk for anxiety disorders.

Study Two confirmed that social stress may affect performance monitoring by demonstrating an association between experiencing overprotecting/authoritarian maternal behaviour throughout childhood and enhanced ERN magnitude in young adulthood. While the mechanisms of these effects are unclear, we suggest two possibilities that should be investigated in future work. First, it is possible that experiencing harsh maternal punishment following errors may lead to anxiety about making mistakes, and consequent heightened monitoring of one's performance to avoid making errors. Alternatively, overprotective maternal behaviours may prevent children from pursuing situations in which they can make mistakes and learn from them, and model that errors should be avoided, instilling in children the idea that errors are catastrophic and performance should be closely monitored, thereby potentiating the ERN. Importantly, our work shows that parental punishment may have long-lasting impacts on performance monitoring that are visible in adulthood. Long-term exposure to parenting styles characterized by punishment may be an important pathway to the development of an enhanced ERN magnitude.

Finally, Study Three investigated whether enhanced performance monitoring might be a diathesis that increases risk for heightened anxiety following interpersonal stress. This study demonstrated that interpersonal stress experienced over the past few months interacted with ERN magnitude measured at the start of the academic year to predict anxiety symptoms toward the end of the year. Specifically, we found that, for individuals with an enhanced ERN at Time 1, greater interpersonal stress exposure was significantly associated with increased anxiety

symptoms at Time 2 (six months later), even controlling for anxiety symptoms at Time 1. These findings are consistent with research indicating that interpersonal stress is a strong predictor of heightened anxiety, but that experiencing interpersonal stress does not always precipitate increases in anxiety, and that an enhanced ERN is a vulnerability marker—but not a diagnostic marker—of anxiety. 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. One possible explanation for this is that enhanced error monitoring renders individuals more susceptible to the negative effects of interpersonal stress, thereby enhancing risk for heightened anxiety. Study Three also demonstrated that non-interpersonal stress did *not* interact with ERN magnitude to predict anxiety, suggesting that the characteristics of interpersonal stressors may be particularly salient.

Together, the three studies presented suggest the possibility that dynamic interactions between stress and performance monitoring may heighten risk for anxiety over time. It is important to note that our findings did not investigate associations between stress, the ERN, and risk for specific anxiety disorders. Study Three used a composite measure of anxiety, which included symptoms associated with disorders that have been both consistently (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), and less consistently (e.g., PTSD 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) associated with an enhanced ERN. Prior work suggests that the ERN may be more closely related to anxious apprehension (i.e., worry) and dispositional anxiety than to anxious arousal (i.e., acute fear; Moser et al., 2012; Weinberg et al., 2010, 2016;

Vaidyanathan et al., 2011). It is thus possible that certain categories of anxiety symptoms or profiles (i.e., those more associated with worry) are better predicted by an interaction between ERN magnitude and interpersonal stress than others (i.e., those more associated with fear). This presents an exciting avenue for further research.

Clinical Implications

The over-arching goal of the work presented in this thesis was to aid our understanding of how individuals develop anxiety so that we may improve our ability to predict who is at risk and develop effective clinical interventions to treat anxiety disorders. We investigated the neural response to errors because of the clear association between an enhanced response and heightened risk for several anxiety disorders and symptoms. Heightened activity in performance monitoring neural networks may thus be an important target of intervention to treat anxiety disorders. Several studies have reported that ERN magnitude is not reduced following cognitive-behavioural and pharmacological interventions, despite participants reporting significant reductions in anxiety symptoms (Gorka et al., 2018; Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). However, other interventions do appear to alter performance monitoring.

For instance, some studies have investigated ERN magnitude following attention bias modification (ABM) interventions, which train individuals to shift attention away from threatening stimuli (Amir et al., 2009; MacLeod et al., 1986, 2002). These studies report that ABM interventions successfully decrease the ERN in both unselected (Nelson et al., 2015, 2017) and clinically anxious (Klawohn et al., 2020) populations. Further, a computerized intervention involving psychoeducation about error sensitivity and maladaptive beliefs about errors, and the promotion of more helpful beliefs about mistakes, was associated with a reduction in ERN

magnitude (Meyer et al., 2019). More general interventions, such as mindfulness training (Fissler et al., 2017) and expressive writing (Schroder et al., 2017), have also successfully altered ERN magnitude. Overall, this body of work suggests that an enhanced ERN magnitude may be reduced through several interventions which may be helpful adjunct treatments to existing therapies for anxiety (Hajcak et al., 2019).

This thesis' findings may also have critical implications for identifying individuals at risk for developing anxiety and attempting to prevent the transition from risk to illness. For instance, the studies suggest that individuals with critical parents, and those who have experienced more total and social-evaluative stressors, may be at higher risk of heightened anxiety due to enhanced performance monitoring. Many interventions aimed at reducing maladaptive parenting behaviour (e.g., severe punishment and overprotection) exist, such as the Positive Parenting Program (Triple P International, 2022) and Positive Discipline in Everday Parenting (Durrant, 2016). It is possible that these programs have positive downstream effects on performance monitoring in offspring, lowering their risk of developing an enhanced ERN and thereby decreasing risk of developing anxiety. Further, programs attempting to decrease social stressors (e.g., bullying) in early adolescence may decrease hypervigilance to errors and protect against anxiety disorders. Although much research suggests that life stress and punitive parenting can lead to maladaptive outcomes, the work presented in this thesis suggests one possible *mechanism* through which these factors can increase risk for anxiety (i.e., via enhanced error processing), providing us with an additional area of focus for interventions aimed at preventing mental disorders. However, future work needs to directly investigate the utility of such interventions for decreasing ERN magnitude and preventing anxiety symptoms via a smaller ERN.

Lastly, neural indices of performance monitoring may be used in the future to inform clinical practice. EEG is a relatively well-tolerated, affordable, and quick way to measure neural activity in infants, children, adolescents, and adults (Kappenman & Luck, 2016; Luck, 2014), making it a strong candidate for usage in clinical settings. Further, a recent study by Imburgio et al. (2020) published population norms for ERN magnitude, allowing for classification of neural responses to errors as "large" (i.e., enhanced), "medium", or "small" (i.e., blunted), which could prove useful for identifying at-risk individuals prior to anxiety disorder onset. For instance, measuring a child's ERN magnitude in a medical setting and comparing it to relevant population norms could allow clinicians to determine who is at heightened risk of anxiety early on, allowing for referrals into anxiety prevention programs and/or interventions to alter ERN magnitude. Of course, substantial work will need to be done before translating such ERP approaches to clinical settings (Imburgio et al., 2020), but this presents a promising avenue for future research and prevention efforts.

Limitations and Future Directions

Taken together, this work suggests that life stress may lead to enhanced performance monitoring, which increases risk for anxiety following life stress exposure. These findings increase our understanding of one potential pathway to the development of anxiety disorders. However, there are several important limitations that should be noted, which present crucial avenues for further investigation. First, although the findings of Studies One and Two suggest associations between life stress and alterations in performance monitoring neural networks, these studies were cross-sectional and thus cannot speak to whether social stress, total life stress, or punitive parenting *altered* the ERN. It is possible that the association between the ERN and stress is opposite to what was hypothesized, whereby children and adolescents with an enhanced

ERN elicit more punitive and/or overprotective parenting from their caregivers and generate more social stress. In line with this possibility, there is evidence that individuals with anxiety disorders, heightened anxiety symptoms, and cognitive styles associated with anxiety (e.g., anxiety sensitivity), are prone to generating elevated levels of life stress compared to those reporting low anxiety (Connolly et al., 2010; Farmer & Kashdan, 2012, 2015; Phillips et al., 2015; Riskind et al., 2010, 2013; Siegel et al., 2018; Uliaszek et al., 2012). Given that an enhanced ERN is associated with heightened anxiety, it is possible that individuals with heightened performance monitoring are prone to generating more social-evaluative and cumulative stress in their lives. However, several studies have shown directional effects of stress on the ERN (e.g., Buzzell et al., 2017b; Meyer & Gawlowska, 2017; Riesel et al., 2017, 2019; van Meel & van Heijningen, 2010), which does suggest that stress precedes an enhanced ERN.

Similarly, child temperament has been shown to influence parenting (Bell & Chapman, 1986; Sameroff, 1975). For instance, anxious behaviours exhibited by children may elicit over-protective (Brunk & Henggeler, 1984; Hudson et al., 2008, 2009; Hudson & Rapee, 2004; Rubin et al., 1999) and punitive (Xing & Wang, 2013) parental behaviours. As clinically anxious children have been shown to display an enhanced ERN (Hajcak et al., 2008; Ladouceur et al., 2006; Meyer et al., 2013; Weinberg et al., 2016), it is possible that children with exaggerated performance monitoring may elicit punitive and overprotective behaviours from their parents, rather than the other way around. However, some studies have shown that ERN magnitude mediates the association between hostile parenting and later offspring anxiety disorder status (Chong et al., 2020; Meyer et al., 2015a, 2019), providing support for the idea that punitive parenting precedes an enhanced ERN. However, the designs of Studies One and Two did not allow us to determine the direction of the ERN-stress association. This highlights an important

avenue for future research: More longitudinal studies are required to determine whether stress causes enhancements in ERN magnitude.

Additionally, we cannot determine from Study Two whether the observed parenting effects are due to environmental (i.e., punishment) or heritable (e.g., genetic) factors. For instance, heritable transmission of ERN magnitude might play a role in the association between authoritarian/overprotective maternal behaviour and offspring ERN magnitude. Some work has found that mothers with a history of anxiety tend to display heightened criticism toward their children (Hirschfeld et al., 1997) and be more overprotective (Ginsberg et al., 2005; Lindhout et al., 2006; Root et al., 2016) compared to mothers without anxiety disorders. These anxious mothers may have an enhanced ERN which they transmit to their offspring, since ERN magnitude is around 50% heritable (Anokhin et al., 2008) and children of mothers with an enhanced ERN tend to display an enhanced ERN (Moser et al., 2018; Suor et al., 2021). Thus, it is possible that it is not punishment that drives the association between parenting and performance monitoring, but rather heritable mother-offspring transmission of ERN magnitude. Study Two cannot speak to such alternative possibilities, as we did not assess maternal ERN magnitude or anxiety disorder status. It is likely that there are influences of both genetic and environmental parental factors that contribute to ERN magnitude. Our understanding would greatly benefit from future research that is better prepared to parse genetic and environmental influences, such as family and twin studies.

Third, Studies One and Two do not reveal the mechanisms through which stress may alter ERN magnitude. Although we theorize that punishment plays a role in this relationship, we did not directly assess punishment in either study. In Study Two, we analysed scores on two parenting dimension subscales of the Parental Bonding Instrument—one of which combined

authoritarian with overprotective parenting behaviours—while in Study One, we analysed composite measures of interpersonal and cumulative stressors, which did not reveal the degree of punishment associated with these stressors. However, the ERN is enhanced when errors are punished in the lab (Meyer & Gawlowska, 2017; Riesel et al., 2012, 2019a), and punitive parenting has been associated with an enhanced ERN in children in several prior studies (Brooker & Buss, 2014b; Chong et al., 2020; Kessel et al., 2019; Meyer et al., 2015). It is thus possible, as we hypothesize, that harsh punishment of errors and negative consequences associated with stress (e.g., threats to interpersonal standing) lead to fear of committing errors and heightened monitoring of one's performance over time.

Perhaps this association happens via the hypothalamic–pituitary–adrenal (HPA) axis. Stress is thought to alter neural structure and functioning in part by activating the HPA axis, which induces the release of glucocorticoids, steroid hormones (e.g., cortisol) that regulate gene expression and can thus influence protein synthesis, neurotransmitters, and receptors (Korte, 2001; Lupien et al., 2009; Seckl, 2007; Schoneveld et al., 2004; Timmermans et al., 2019). Glucocorticoids bind to receptors found throughout the brain and perform key functions for neural development and maturation, including neuron and synapse formation (Lupien et al., 2009; Seckl & Meaney, 2004). Animal models show that heightened glucocorticoid exposure due to stress can slow down neuronal maturation and synapse formation (Antonow-Schlorke et al., 2003; Huang et al., 2001), altering the structure of the brain (Matthews, 2000; Seckl, 2007). Although more research is needed to understand this process, it is possible that glucocorticoid-induced alterations in neural regions involved in performance monitoring are one way in which life stress can change ERN magnitude.

In support of this idea, most studies investigating HPA axis reactivity and performance monitoring have found that increases in the stress hormone cortisol are associated with enhanced ERN magnitude (Cavanagh & Allen, 2008; Tops & Boksem, 2011; Tops et al., 2006), although one study reported that increased cortisol is associated with a *smaller* ERN (Compton et al., 2012). Importantly, a larger ERN appears to predict greater cortisol reactivity following a social stressor (Cavanagh & Allen, 2008), making it possible that social-evaluative stressors alter ERN magnitude via increased cortisol in the HPA axis. Additionally, there is evidence of substantial HPA axis reorganization and increased neural sensitivity to cortisol during early adolescence (Doom & Gunnar, 2013; Gunnar & Donzella, 2002; Gunnar & Quevedo, 2007; Gunnar, Talge, & Herrera, 2009a; Gunnar & Vazquez, 2006; Gunnar et al., 2009b; Lupien et al., 2009), a period of development during which interpersonal stress was more strongly associated with an enhanced ERN in Study One. The ERN may thus be more susceptible to change during developmental periods characterized by HPA growth and reorganization, and increased reactivity to cortisol. Our understanding of the ways in which stress may impact neural networks involved in performance monitoring would greatly benefit from longitudinal mediational studies investigating associations between stress, punishment, and the ERN, as well as the role of HPA axis hormones in these relationships.

Study Three presents a similar limitation as Studies One and Two in that it reveals an interaction effect whereby enhanced neural response to errors interacts with social stress to predict anxiety, but does not explain *why* stress may place individuals with a larger ERN at risk for heightened anxiety. It is possible that individuals who display heightened performance monitoring are more susceptible to the adverse effects of life stress, for instance, by responding to social-evaluative stress with greater negative affect (NA). Prior work suggests that individuals

reporting high NA display a larger ERN amplitude compared to those who report low NA (Hajcak et al., 2004; Luu et al., 2000), and increasing NA in the lab leads to an enhanced ERN (Wiswede et al., 2009). Heightened NA is a characteristic of anxiety disorders (Clark & Watson, 1991, 2008; Eysenck & Fajkowska, 2018) and appears to predict subsequent increases in anxiety (Barrocas & Hankin, 2011; Chin et al., 2017; Naragon-Gainey, 2019). Negative affectivity is also correlated with experiencing increased stressful and unpleasant events (Bolger et al., 1989; Farmer & Kashdan, 2015; Watson, 1988; Watson & Pennebaker, 1989), and stress appears to lead to increases in NA (Scott et al., 2017). Thus, it is possible that individuals with an enhanced ERN display more NA following life stress, which in turn increases anxiety. However, it should be noted that two studies have reported that a larger neural response to errors is associated with *less* reactivity to real-world stressors (Compton et al., 2008, 2011). These mixed findings and the paucity of work investigating whether NA is a mechanism through which stress and an enhanced ERN might promote greater anxiety symptoms point to a critical gap in our knowledge that should be investigated with longitudinal studies and daily diary or ecological momentary assessment (EMA) methodologies.

In addition, associations between NA, stress, anxiety, and ERN magnitude may differ by age range, whereby individuals in certain developmental periods respond with increased NA to certain stressors. For instance, early adolescents appear to pay increased attention to peer interactions and display heightened sensitivity to social stress compared to those in other developmental periods (Bolling et al., 2011; Parker et al., 2015; Silk et al., 2013). It is possible that heightened NA following social stress in early adolescence may more strongly mediate associations between social stress and an enhanced ERN compared to other developmental periods. However, more research is needed to investigate this possibility.

Despite these limitations, identifying neural markers of risk for anxiety and improving our understanding of how environmental factors can influence them is still valuable. Given that anxiety is not completely heritable (Franić et al., 2016; Guffanti et al., 2016; Purves et al., 2020) and many people who experience stressful events do not go on to develop anxiety (Ingram & Luxton, 2005; Harkness, Hayden, & Lopez-Duran, 2015; Harkness & Monroe, 2016), even an incomplete understanding of neural mechanisms of risk can help us better understand anxiety disorders, identify those at risk, and guide prevention and treatment efforts.

An additional limitation of this work is that the samples used were mostly (Studies One and Three) or completely (Study Two) composed of participants who identified as female. The lack of sex and gender diversity in our samples may limit the generalizability of our findings. Prior work has shown that females are more likely than males to experience life stress (Bale & Epperson, 2015), and report increased interpersonal stress exposure in particular (Slavich & Shields, 2018; Stroud et al., 2002; Troisi, 2001). There also appear to be sex differences in the functioning of neurobiological stress response systems (Bangasser & Valentino, 2014; Bangasser et al., 2018). Further, adolescent females report more interpersonal stress than males, as well as heightened emotional responsivity to these stressors (Colten & Gore, 1999; Rudolph, 2002). Additionally, females are more likely than males to experience heightened anxiety (Kessler et al., 2005; McLean et al., 2011), and may display smaller ERN magnitudes (Fischer et al., 2016, 2017; Imburgio et al., 2020; Larson et al., 2011). Lastly, sex and gender effects have been reported for parenting—for instance, daughters tend to experience more authoritative parenting than sons (McKinney et al., 2018). The findings presented here may thus mostly reflect relationships between error monitoring, stress, and anxiety for females. It will be important for future work to replicate these findings in more sex- and gender-balanced samples.

Further, these studies used self-reports of stressful experiences, which did not allow us to parse effects of objective and perceived stress severity and parenting styles. For instance, Study Two asked participants to report on the parental behaviours they experienced between birth and 16 years of age, a methodology which is open to recall biases (Taber, 2010). Prior work suggests that anxious individuals often interpret information in a negative light and recall more negative events compared to controls (Amin et al., 1998; Hirsch et al., 2016; MacLeod et al., 1997; Stopa & Clark, 2000; Voncken et al., 2003). It is possible that participants with an enhanced ERN were more anxious and thus more prone to recalling increased maladaptive parenting compared to individuals with a smaller ERN, although we did not assess this possibility. In addition, data obtained using more objective measures of parenting, such as observational studies (McKee et al., 2013), and parent-reported child-rearing behaviours have been shown to differ from child-reported parenting (Parent et al., 2014; Taber, 2010). We may thus have observed different associations between ERN magnitude and parenting had we employed different measurement strategies. However, some work suggests that child reports of parental behaviour are often closely associated with observed parenting (Parent et al., 2014; Scott et al., 2010; Sessa et al., 2001), and perceived parenting appears to be informative for understanding offspring psychological outcomes (McClure et al., 2001). Results from Study Two thus still provide important insight into effects of parenting on a neural marker of risk for anxiety. Future work should employ both subjective and objective measures of parenting and other stressors to better understand associations between parenting, stress, and ERN magnitude.

Similarly, data collected using interview-based measures of social, life-threatening, and total stress across the lifetime may have revealed different associations with ERN magnitude than the self-reported stressor count and severity data collected in Study One. For instance, the

UCLA Life Stress Interview (Hammen, 1991) and Life Events and Difficulties Schedule (Brown & Harris, 1978) allow interviewers to rate stressful events reported by participants in a more objective manner than self-reports. Our understanding of associations between neural indices of performance monitoring and different types of stress experienced during different developmental periods would be greatly enhanced by future work using interview-based measures of life stress. However, past work has shown that one's perceptions of stressful experiences are strongly tied to health outcomes (Epel et al., 2018; Slavich & Cole, 2013), suggesting that it is also valuable to investigate self-reported appraisals of stressor count and severity.

Another important limitation of the studies presented is that they employed samples of undergraduate students who reported being mostly white and of high SES, and student samples may differ in meaningful ways from more general community populations (Hanel & Vione, 2016) and clinically anxious samples. Indeed, in Study One, our sample reported fewer and less severe stressors compared to community samples (Slavich & Shields, 2018; Sturmbauer et al., 2019) with broader SES ranges (Cazassa et al., 2020), which may affect our ability to generalize to other populations. Further, results from Study Three may not generalize directly to community samples because first-year undergraduate students are at heightened risk for psychopathology compared to those in later university years (Adlaf et al., 2001), and the first few months at university are characterized by heightened interpersonal stress (Bouteyre et al., 2007; Fisher & Hood, 1988; Wilcox et al., 2005). Although these factors made our sample an ideal one for investigating our research question, it is possible that non-student samples would have produced different results. However, past work has shown that even low levels of stress, and stressors considered mild and moderate in severity, can impact health (McLaughlin et al., 2021; Slavich et al., 2009; Slavich et al., 2014), suggesting that our findings are still important and applicable to a

portion of the population that experiences lower than average stress levels. Further, the finding in Study Three that stress and the ERN interact to predict anxiety symptoms in an unselected sample suggests that our understanding of pathways to anxiety can be improved even using non-clinical populations. Nonetheless, future work will need to employ clinically anxious and more diverse samples to confirm whether the findings generalize to a wide range of populations.

Lastly, the clinical utility of these findings might be challenged because of the small-to-medium effect sizes observed (Cohen, 1988). For instance, the association between early adolescent social stress severity and ERN magnitude in young adulthood in Study One was r = -0.22, and the interaction effect between ERN magnitude and past-year social stress to predict anxiety in Study Three was β = -0.54. However, self-report data and psychophysiological variables are expected to correlate moderately with one another because they share no method variance (Campbell & Fiske, 1959; Patrick et al., 2013). Additionally, other studies investigating associations between the ERN and important individual difference variables show similar effect sizes (Cavanagh & Shackman, 2015; Meyer et al., 2015a; Moser et al., 2013; Weinberg et al., 2016), and their findings may have important implications for real-world outcomes (Hajcak et al., 2019; Meyer et al., 2017a). For instance, the ERN has demonstrated incremental predictive ability for the later development of anxiety disorders over and above other common risk factors (Meyer et al., 2015a; Meyer et al., 2018), an effect that was small to medium in size. Thus, even modest effects like those reported here may improve our ability to predict important anxiety-related outcomes.

Final Conclusions and Summary

The work presented in this thesis examined how various types of stress experienced during different developmental periods is associated with a neural index of performance monitoring, and how error monitoring interacts with stress to predict later anxiety symptoms. The reported results aid our understanding of risk for the development of anxiety disorders. Specifically, these studies suggest that total and social-evaluative stress experienced in early adolescence, and punitive parenting experienced during the first 16 years of life, are associated with an enhanced ERN in young adulthood, possibly through punishment of errors which leads to sensitization of neural systems involved in error monitoring. Further, this work suggests that individuals with enhanced error monitoring display heightened anxiety symptoms following interpersonal stress compared to those with a smaller ERN, possibly due to increased susceptibility to the negative effects of social stress. Taken together, these findings point to several important environmental risk factors (i.e., social-evaluative stress, early adolescent stress, and punitive parenting) for the development of enhanced performance monitoring and anxiety disorders. This work will be important for future efforts to identify individuals at heightened risk for developing anxiety in the face of stress, which may ultimately be used for clinical prevention and treatment efforts for illnesses with substantial human and economic costs.

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