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The Associations between Sleep, Anxiety, and Error-Related Brain Activity in Youth

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THE ASSOCIATIONS BETWEEN SLEEP, ANXIETY, AND ERROR-RELATED BRAIN ACTIVITY IN YOUTH

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ABSTRACT

Due to the high prevalence of anxiety disorders and their associated impairment, elucidating neural mechanisms that underlie these disorders has been increasingly prioritized. Specifically, the error-related negativity (ERN) has been identified as a neural marker that indexes risk for anxiety across development. Approximately half of the variance in the ERN can be attributed to factors other than genetic inheritance, including factors such as parenting style and stressful life events. The present study sought to examine associations between the ERN and another factor — sleep difficulties. This study serves as a novel examination of the associations between self-reported naturalistic sleep difficulties, anxiety, and the ERN. In a sample of 221 females, ages 8 to 15 years old, we first examined the relations between chronic (i.e., over the past month) and recent (i.e., over the past week) sleep disturbances and the ERN. We then investigated whether a specific chronic or recent sleep difficulty uniquely predicts the ERN. In exploratory analyses, we assessed whether the ERN moderates and/or mediates the relationship between sleep difficulties and anxiety. Results indicated that youth who report chronically shorter sleep duration, chronically worse sleep, and recent shorter sleep duration on school days over the past week have a larger ERN. In addition, recent sleep duration on school days over the past week uniquely predicted the ERN. Exploratory analyses indicated the ERN moderates the associations between chronic sleep quality and total anxiety, as well as that between chronic sleep quality and child social anxiety. Finally, results suggested the ERN may partially mediate the link between sleep problems and anxiety. Future studies should clarify the direction of these associations via longitudinal designs.

CHAPTER 1

INTRODUCTION

Anxiety disorders are among the most prevalent disorders in the world (Kessler et al., 2005, 2009). Their first onset can occur throughout the lifespan, from pre-adolescence to late adulthood, and these disorders have been associated with several domains of decreased quality of life, such as greater physical disabilities with increased age (Brenes et al., 2008; Lenze & Wetherell, 2011). Importantly, anxiety disorders are common among children and adolescents, with a lifetime prevalence ranging from 15% - 20% during these developmental stages (Beesdo et al., 2009; Cartwright-Hatton et al., 2006). Among youth, anxiety disorders have also been related to a host of impairments, including difficulty in domains of educational achievement, family relationships, and hobbies (Creswell et al., 2014). Further, anxiety disorders in adolescents have been found to commonly co-occur with other mental health disorders, especially Major Depressive Disorder (Lewinsohn et al., 1997), as well as to predict risk for the onset of other mental health disorders and associated negative outcomes (e.g., low educational achievement) later in life (Woodward & Fergusson, 2001). In addition to anxiety disorders among youth being highly prevalent, a large majority of these disorders remain untreated — only around 18% of adolescents with anxiety disorders receive treatment (Essau, 2005). Considering the negative impact of anxiety, it is crucial to develop targeted interventions for individuals early in development.

Over the past two decades, research has increasingly focused on characterizing the underlying neural mechanisms that contribute to anxiety disorders across development (Pine, 2007). One such neural marker that has been linked to anxiety is the error-related negativity (ERN), which is a response-locked event-related potential (ERP). The ERN is localized to fronto-central electrode sites, and it occurs in the form of a sharp, negative deflection approximately 50 ms after the commission of an error in a lab-based task (Falkenstein et al., 1991; Gehring et al., 1993). The ERN is thought to be generated in the anterior cingulate cortex (ACC), which is a brain region that processes pain, threat, and punishment (Miltner et al., 2003; Shackman et al., 2011; van Veen & Carter, 2002). Errors have been conceptualized as a type of endogenous threat. Indeed, errors initiate a series of physiological responses consistent with this, e.g., skin conductance response, potentiated startle reflex, heart rate deceleration, pupil dilation,

and corrugator muscle contraction (Weinberg et al., 2012). Thus, we view individual differences in the ERN to index, in part, the degree to which an individual experiences errors as aversive and/or salient. In line with this, we have found that the ERN relates to self-reported error sensitivity — the extent to which an individual has negative reactions to mistakes (Chong & Meyer, 2019).

A considerable body of literature has identified the ERN as a neural marker of anxiety and related disorders (Cavanagh & Shackman, 2015; Hajcak, 2012; Meyer, 2016; Moser et al., 2013; Weinberg et al., 2016). In particular, several studies in both children and adults have shown that the ERN is increased in anxious individuals, including those with generalized anxiety disorder, social anxiety disorder, and obsessive-compulsive disorder (Endrass et al., 2010; Meyer, 2017; Riesel, 2019; Weinberg et al., 2010). Further, the ERN has been found to prospectively predict the onset of anxiety symptoms and disorders in children as young as 5 years old (Meyer et al., 2015; Meyer et al., 2017; Meyer et al., 2018). Therefore, an enhanced ERN has been identified as a neural marker of both anxiety and risk for anxiety.

As the ERN has been shown to prospectively predict risk for the onset of anxiety, examining the factors that contribute to shaping the ERN early in development is crucial. A combination of genetic and environmental factors have been found to relate to the ERN, such that 40-60% of the variance in the ERN is related to environmental factors (Anokhin et al., 2008). One environmental factor that has been studied in relation to the ERN is parenting style. Studies have shown that children of parents with harsh or controlling parenting styles have larger ERNs (Banica et al., 2019; Brooker & Buss, 2014; Chong et al., 2020; Meyer et al., 2015, 2019; Meyer & Wissemann, 2020). Furthermore, findings have shown that the *presence* of a controlling parent in the room, while children complete a lab-based task, relates to an increased ERN (Meyer et al., 2019). Controlling parents are more likely to punish mistakes (Robinson et al., 2001) and this punishment can lead children to persistently worry about error commission (Kawamura et al., 2002). Experimentally, an increased ERN has also been found after punishing errors in a lab setting, and this effect remained even after punishment ended (Meyer & Gawlowska, 2017; Riesel, 2019; Riesel et al., 2012). Thus, learning experiences surrounding error commissions, such as exposure to critical parenting style and punishment for errors, appear to shape the ERN early in development.

Additionally, stressful life events are another environmental factor that have been studied in relation to the ERN. An examination of a hurricane's impact on participant anxiety found that children with an increased ERN experienced elevated anxiety symptoms following the natural disaster (Meyer, Danielson, et al., 2017). Another study in youth, ages 8 to 15 years old, explored the relation between life stressors and the ERN (Mehra & Meyer, Under Review). Results suggested that more frequent stressful life events are associated with an elevated ERN. The study also revealed differential associations with the ERN based on type of life event, such that interpersonal-related stressors, but not non-interpersonal events, uniquely predicted the ERN. Finally, age appeared to moderate the relation between the ERN and interpersonal stressful life events, such that more frequent stressors predicted an enhanced ERN in younger children but not older children. These findings underscore the influential role of life stressors as environmental events that contribute to anxiety and the ERN early in development.

More broadly, sleep is also widely recognized as an important factor that may impact anxiety in youth. While most literature focuses on sleep difficulties in relation to affective disorders, these problems have also been found in relation to anxiety (Alvaro et al., 2013; Harvey, 2002; Mellman, 2006; Staner, 2003). Up to 88% of children with anxiety disorders are reported by their parents to have sleep-related problems (Alfano et al., 2006, 2007, 2010; Chase & Pincus, 2011). In addition, sleep disturbances are included in the DSM-5 diagnostic criteria for some anxiety diagnoses, including separation anxiety disorder and generalized anxiety disorder (American Psychiatric Association, 2013).

Evidence for the relation between sleep disturbances and anxiety has been observed in a variety of anxiety disorders across both children and adults (Cox & Olatunji, 2016). Specifically, in Generalized Anxiety Disorder (GAD), increased sleep impairment has been found in individuals with GAD compared to both healthy controls and individuals with other types of anxiety disorders (Alfano et al., 2006, 2015; Berger et al., 2009; Brenes et al., 2009; Wetherell et al., 2003). Additionally, sleep problems have also been found to predict the onset of GAD (Batterham et al., 2012; Shanahan et al., 2014; Steinsbekk & Wichstrøm, 2015). Further, research in children with separation anxiety disorder has shown that for those with the diagnosis, 97% of children had at least one sleep-related problem, and those with a separation anxiety diagnosis also had significantly more sleep-related problems than those without separation anxiety (Alfano et al., 2007; Chase & Pincus, 2011). Specifically, children with separation

anxiety were found to experience insomnia, nightmares, and refusal to sleep alone or away from home compared to children without separation anxiety (Alfano et al., 2007; Chase & Pincus, 2011). Moreover, there is evidence that sleep disturbances are related to social anxiety symptoms (Buckner et al., 2008; Cheng et al., 2017). Those with social anxiety have been found to have poor sleep based on self-report measure criteria (Zalta et al., 2013) and sleep problems (Ramsawh et al., 2009), and sleep problems have been shown to predict the onset of social anxiety (Steinsbekk & Wichstrøm, 2015). Likewise, research in Panic Disorder (PD) has found increased sleep difficulties compared to healthy controls (Overbeek et al., 2005; Todder & Baune, 2010), an increased likelihood of sleep disturbances in those with PD (Ramsawh et al., 2009; Roth et al., 2006), and that sleep problems predict PD onset (Batterham et al., 2012). Similarly, studies examining sleep in phobias have found increased likelihood of sleep disturbances (Ramsawh et al., 2009; Roth et al., 2006) and poor sleep quality (Kleim et al., 2014) in those with phobias. Throughout the literature, similar patterns of results were identified in studies using subjective and objective sleep measures (Cox & Olatunji, 2016).

As depression and anxiety are highly comorbid conditions, it is important to note that these reported studies vary in the way in which they chose to take depression into account (e.g., control for baseline depression, examine groups with comorbid depression). Finally, in terms of the directions of these relations, some of the literature explores how sleep disturbances precede the onset of anxiety disorders, while others primarily posit that the sleep and anxiety relation is bidirectional (Alvaro et al., 2013; Chorney et al., 2008; Gregory et al., 2005, 2006; Gregory & O'Connor, 2002; Leahy & Gradisar, 2012; Ong et al., 2006).

Since sleep problems have a clear association with anxiety disorders, studies have specifically begun examining the role of an inadequate quantity or quality of sleep (i.e., sleep deprivation) in particular facets of anxiety. A study in a group of young adults found that a 36-hour period of sleep deprivation led to heightened self-reported anxiety symptoms, as well as worse time performance on a sustained attention task (Sagaspe et al., 2006). Thus, sleep deprivation was associated with the slowing of cognitive processes and increases in anxiety. Further, Talbot et al. (2010) explored the relation between sleep deprivation (i.e., a maximum of 8.5 hours of sleep total over two nights) and affective functioning of healthy participants from the ages of 10 years old to 60 years old. Results revealed that participants in the sleep deprivation condition experienced greater anxiety in the context of a catastrophizing task, and they rated the

possibility of catastrophes as more likely. In a group of early adolescents sleep deprivation was associated with elevated appraisal of their most threatening worry (Talbot et al., 2010). As such, sleep deprivation appears to be associated with elevated threat perception.

Moreover, sleep deprivation has been found to reduce performance on tasks across a variety of studies, though the underlying mechanism is not entirely clear. A meta-analysis found that short-term sleep deprivation (i.e., <48 hours) is related to significant decline across cognitive domains, with simple attention as the most strongly impacted domain by acute sleep deprivation (Lim & Dinges, 2010). Another study used fMRI to demonstrate changes in functional connectivity during sleep deprivation conditions, and authors found that sleep deprivation was related to network alterations that are associated with worse task performance and heightened negative mood (Ben Simon et al., 2017). Further, fMRI studies examining rapid eye movement (REM) sleep – one of the two main phases of the sleep cycle – have found that specific reductions in REM sleep are associated with increased emotional reactivity (Glosemeyer et al., 2020; Rosales-Lagarde et al., 2012).

Despite findings that sleep difficulties relate to anxiety, reduced sleep decreases performance and elevates threat perception, as well as an increased interest in identifying factors that may shape the ERN, few studies have examined the potential link between sleep and error-related brain activity. Examining the relation between sleep and the ERN may enhance our understanding of the underlying etiopathogenesis of anxiety. Furthermore, by identifying factors that may impact the ERN, we may pave the way for novel prevention and intervention strategies. Existing literature relating sleep disturbances to the ERN largely focuses on the ERN's relations with mental fatigue and acute sleep deprivation. For example, Kato et al. (2009) and Xiao et al. (2015) have both found that individuals with experimentally-induced mental fatigue exhibited decreased ERN amplitudes during a lab-based task. Similarly, studies examining sleep deprivation and extended wakefulness conditions have also found associations between these conditions and smaller ERNs (Hsieh et al., 2007; Scheffers et al., 1999; Tsai et al., 2005). Examining naturalistic variations in sleep duration over a period of seven days, Fueggle et al. (2018) found similar results – a smaller ERN for those in the lower sleep duration group. These studies either focus on experimentally induced fatigue and sleep deprivation conditions, which are acute in nature, or recent naturalistic sleep variations (i.e., over the past week). However, prior work suggests that chronic sleep disturbances, which are frequently present in childhood

and found to increase with age, may contribute to risk for anxiety disorders (Caporino et al., 2017). No studies, to our knowledge, have examined the relation between chronic, naturalistic sleep disturbances and the ERN.

Current Study Aims and Hypotheses

The current study aims to bridge this gap by examining the relationship between the ERN and naturalistic sleep-related difficulties in children and adolescents, ages 8 to 15 years old. Sleep is measured using two subjective questionnaires – one that assesses the child's self-reported severity or frequency of various sleep-related problems occurring over the past month and one that examines the child-report of sleep-related problems occurring over the past week.

As a first step, we examined whether various types of sleep difficulties relate to the ERN. Based on previous findings suggesting that chronic sleep disturbances are related to anxiety and heightened threat perception, we hypothesized that subjective sleep problems over the past month will relate to the ERN, such that greater chronic sleep difficulties will be associated with a larger ERN. However, based on the findings related to acute sleep deprivation and the ERN, we tentatively hypothesized that sleep problems *over the past week* would relate to the ERN in the opposite direction, such that greater difficulties over the past week would be associated with a smaller ERN. We also examined whether specific sleep subscales (i.e., sleep duration and sleep quality over the past month; or sleep duration over the past week, last weekend, and last night) uniquely predicted the ERN. As there is no existing literature examining the differential impact of various sleep difficulties in relation to the ERN, we did not have an a priori hypothesis for these analyses. Next, we examined the relationship between sleep difficulties and parent-report of anxiety severity. Based on prior sleep and anxiety studies, we expected that sleep problems will broadly be associated with anxiety severity across all subtypes of anxiety. We also assessed the relationship between the ERN and specific anxiety domains. In line with previous work, we expected the ERN to be associated with disorders characterized by performance-based concerns, such as generalized anxiety and social anxiety, and not fear or arousal-related anxiety (e.g., panic and phobias).

Finally, as exploratory analyses, we assessed whether the ERN moderated and/or mediated the relationship between sleep difficulties and anxiety. As previous work suggests that sleep difficulties and a larger ERN are each associated with greater anxiety severity and risk for

anxiety, it is possible that these risk factors may interact, such that the relationship between sleep difficulties and anxiety severity is greater for those with a larger ERN. On the other hand, it is possible that sleep difficulties may impact anxiety via the ERN (i.e., the sleep difficulties/anxiety relationship may be mediated by the ERN). This would suggest that sleep problems may shape the ERN and thereby increase risk for anxiety. Given the lack of previous work in this area, these were exploratory analyses.

CHAPTER 2

METHODS

Participants

The sample included 251 female children and adolescents, ages 8 to 15 years old, recruited as part of a larger study funded by the NIMH (R01 MH097767). Participant EEG data were assessed for usability. 17 participants were excluded from analyses for having a Flanker task accuracy below 60%, 1 was excluded for committing fewer than 6 errors (Olvet & Hajcak, 2009), and 2 were excluded for being deemed univariate outliers (i.e., more than three standard deviations from the mean). Of the remaining 231 participants, 10 were excluded for having missing data for the self-report measures on sleep or anxiety. Thus, the final sample contained 221 participants, who were 12.48 years old (*SD* = 1.76), on average. Participants self-reported their ethno-racial identities as 82.4% White, 5.4% Black, 5.4% Hispanic, and 4.5% Other, with 2.3% of participants missing these data.

The study was approved by the Institutional Review Board at Stony Brook University. Inclusion criteria for study participation were that the parent and child must speak English and the parent must be biologically related to their child. Exclusion criteria were a history of developmental or medical disabilities for the child. Both parent consent and child assent were obtained prior to initiation of study activities. Participants were paid at a rate of $20 per hour of participation.

Measures

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) was completed by the child to assess chronic sleep difficulties across several domains occurring over the past month. The PSQI is a subjective sleep measure that is among the most common and recommended subjective measures of sleep (Buysse et al., 2006). It consists of 19 items that examine various types of sleep-related disturbances over the past month. The present study focuses on the PSQI total score and two specific subscales: sleep duration and sleep quality. The sleep duration subscale of the PSQI consists of one item asking participants, “During the past month, how many hours of actual sleep did you get at night?” Participants respond via free response reporting the

number of hours of sleep they got per night over the past month. Item responses were then recoded on a 0 to 3 scale, such that a sleep duration greater than 7 hours was coded as 0, between 6 and 7 hours as 1, between 5 and 6 hours as 2, and less than 5 hours as 3. As such, a higher value on the sleep duration subscale indicates a lower sleep duration over the past month. The sleep quality subscale of the PSQI asks participants, "During the past month, how would you rate your sleep quality overall?" Responses range from 0 ("very good") to 3 ("very bad"), such that higher values represent worse sleep quality ratings over the past month. The total PSQI score represents a composite of sleep quality and quantity domains and can range from 0 to 21. A total score on the PSQI of 5 or greater is indicative of poor sleep (Buysse et al., 1989). The PSQI has been found to be reliable and valid in populations of youth as young as 14-years-old (de la Vega et al., 2015) and has been used in studies with children as young as 5-years-old (Fauroux et al., 2012) with a variety of health problems.

The Sleep Diary (Buysse et al., 2006; Carney et al., 2012) was also completed in order to capture information on the child's more recent self-reported experiences of sleep. The present study specifically focused on analyzing sleep duration over three different time periods: school nights the past week, last weekend, and last night. Sleep duration was computed by first calculating the number of minutes of sleep between reported time the child got into bed (e.g., "On school nights in the past week, what time have you usually gotten into bed?") and the reported time they woke up (e.g., "On school days in the past week, what time have you usually woken up?"). Then, the amount of time taken to fall asleep after getting in bed (e.g., "On school nights in the past week, after you have gone to bed at night, how long has it usually taken you to fall asleep? (in minutes)") was subtracted. The remaining value represented the child's sleep duration in minutes during the specified period, such that a greater value signifies a longer sleep duration. Although the format of the sleep diary may vary, such that researchers can modify the array of questions included, it is considered the gold-standard for subjective sleep assessment due to its self-monitoring methods (Carney et al., 2012).

Additionally, parents rated their child's anxiety symptoms using the Screen for Child Anxiety Related Emotional Disorders-Parent (SCARED-P; Birmaher et al., 1997). The SCARED-P is a 38-item self-report measure that examines anxiety symptom severity across five domains: panic and somatic symptoms, generalized anxiety, separation anxiety, social anxiety, and school avoidance. The measure also calculates a total anxiety severity score, which ranges

from 0 to 76. Responses to each item range from 0 (“not true or hardly ever true”) to 2 (“true or often true”). The SCARED-P has been found to be reliable and valid in populations of youth ages 8 to 18 years (Birmaher et al., 1999; Birmaher et al., 1997; Hale et al., 2011).

Tasks and Materials

In order to measure error-related brain activity, participants completed a computer-based arrowhead version of the Flankers task (Eriksen & Eriksen, 1974) during EEG recording. Participants were instructed to respond to a stimulus composed of five arrowheads by using the right or left mouse button based on the direction of the central arrowhead. Participants were requested to respond as quickly and accurately as possible. Each participant completed 11 blocks of 30 trials of stimuli, totaling 330 trials, which were composed of 50% compatible (“<<<<<” or “>>>>>”) trial types and 50% incompatible (“<<><<” or “>><>>”) trial types. Stimuli were randomized and presented for 200 ms with randomly variable inter-trial intervals that ranged from 2300-2800 ms. Prior to the initiation of the task, participants completed 1 practice block of 30 trials. The 11 blocks of the actual task were then initiated by the participant, and participants received performance-based feedback following each block. Accuracies of 75% and below resulted in the message “Please try and be more accurate,” accuracies 90% and above were prompted with “Please try to respond faster,” and accuracies between 75% and 90% viewed “You’re doing a great job.”

Psychophysiological Recording

Participants wore elastic caps with thirty-four electrode sites that were connected to the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands) in order to collect continuous EEG recording during the Flankers task. Additionally, one electrode was placed on each mastoid, and four electrodes were added to the face to record eye movement and blinking via electrooculogram (EOG). The two facial electrodes capturing horizontal eye movements were placed about 1 cm from the outer edge of the left and right eyes, and the two facial electrodes capturing vertical eye movements were placed about 1 cm above and below each eye. The EEG signal was preamplified at each electrode and amplified with a gain of one by the ActiveTwo BioSemi system to improve the signal-to-noise ratio. Data were digitized at a 24-bit resolution with a sampling rate of 1024 Hz using a low-pass fifth order sinc filter with a half-power cutoff

of 204.8 Hz. Online, active electrodes were referenced with respect to a common mode sense (CMS) active electrode that produced a monopolar (non-differential) channel.

Offline, data were referenced to the average of the right and left mastoids and band-pass filtered between 0.1 and 30 Hz. Ocular corrections were conducted per Gratton et al. (1983). An automatic procedure was used to conduct artifact detection and rejection, such that a voltage step greater than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a voltage difference of less than .50 μV within 100 ms intervals was rejected from channels in each trial.

EEG data was segmented by error and correct trial types at -500 ms to 1000 ms surrounding trial responses. The correct-related negativity (CRN) and the error-related negativity (ERN) were obtained through this separate averaging of correct and incorrect responses, respectively. Baseline correction was performed on the interval from -500 to -300 ms. Then, the average ERN and CRN activity from the interval of 0 to 100 ms after a response was exported for each participant. Analyses focused on FCz – the electrode where error-related brain activity was maximal – and a regression-based difference score (ERNresid; the unstandardized residuals from a regression predicting the ERN from the CRN) was created (Meyer, Lerner, et al., 2017). Task percent accuracy and average reaction time (by trial type and across all trials) were calculated for use as behavioral measures in analyses.

Data Analyses

Statistical analyses were conducted using SPSS Version 26. General Linear Modeling was used, and Greenhouse-Geisser corrections were applied to *p* values when the assumption of sphericity was violated. Repeated-measures ANOVAs were conducted to examine behavioral components during error and correct trials, as well as to compare the average ERN and CRN values. Further, to examine the associations between study variables, the Pearson correlation coefficient (*r*) was used. Two-tailed correlations were conducted for analyses without a directional hypothesis and one-tailed correlations were conducted for analyses with a directional hypothesis. Three multiple regressions were performed to examine whether any sleep subscales predict unique variance in the ERN for each sleep measure, as well as for both measures in one model. Further, regressions were conducted to determine whether the ERN is independently associated with sleep difficulties above and beyond anxiety symptoms. Finally, in line with the

exploratory aims, we performed moderation models wherein the interaction between sleep disturbances and the ERN predicting anxiety severity were tested. We also conducted mediation models examining whether the ERN mediates the relationship between sleep disturbances and anxiety severity. The moderation and mediation analyses were performed using SPSS Hayes macro PROCESS (Hayes, 2012) models 1 and 4, respectively, with 5000 bootstrap samples. All study analyses where $p < .05$ were considered significant. Notably, the exploratory analyses were conducted controlling for child-report of depressive symptoms using the Children's Depression Inventory (CDI; Kovacs, 1992).

Power Analyses

In order to determine whether the study is adequately powered, post hoc power analyses were conducted using G*Power 3.1 (Faul et al., 2007). Based on a linear multiple regression with 5 predictors, an analysis with a medium effect size ($f^2 = .15$), an alpha of .05, and a sample size of 221, the analysis will be powered at a level of 1.000. Further, in order to assess the power of the exploratory mediation analysis, the power of the indirect effect was calculated by multiplying the power of the test of the α path (power = 1.000, 1 predictor) by the power of the β path (power = 1.000, 2 predictors), based on medium effect sizes ($f^2 = .15$), an alpha of .05, and a sample size of 221 (Kenny, 2018). The mediation analysis was therefore determined to be powered at a level of 1.000. Both of these calculated power values are greater than the standard power level of .80 (Cohen, 1988). As the most involved analyses of the study are at power, the remaining analyses in this study are also determined to be at power. Thus, the sample size of 221 participants renders the current study adequately powered.

CHAPTER 3

RESULTS

Age and Self-Report Measures

Means and standard deviations for the PSQI, Sleep Diary, and SCARED-P totals and subscales are presented in **Table 1**. Two-tailed correlations with age and self-report measures revealed significant positive associations between age and the PSQI total, r (219) = 0.29, p < .001, PSQI sleep duration, r (219) = 0.40, p <.001, and PSQI sleep quality, r (219) = 0.21, p = .002, such that older children reported worse overall sleep, less time slept, and worse sleep quality over the past month. Additionally, age was positively correlated with the SCARED-P total and subscales for panic and somatic symptoms, generalized anxiety, social anxiety, and school avoidance, all ps < .05, such that parents reported greater panic, generalized anxiety, social anxiety, school avoidance, and total anxiety in older children and adolescents. However, there was a negative correlation between age and the SCARED-P separation anxiety subscale, r (219) = -0.18, p = .007, such that parents reported greater separation anxiety in younger children. Finally, there was a significant negative correlation between age and the Sleep Diary's sleep duration during school days in the last week, r (219) = -0.23, p < .001, such that older children reported a shorter sleep duration on school days over the past week.

Sleep Difficulty Self-Report Measures

Two-tailed correlations were conducted to assess the associations amongst subscales of the PSQI and the Sleep Diary measures of sleep difficulty. Only one significant correlation was found across measures. PSQI sleep duration was associated with Sleep Diary sleep duration on school days over the past week, r (219) = -0.13, p = .049, such that less sleep duration over the past month on the PSQI was associated with less sleep on school days over the past week on the Sleep Diary. The PSQI sleep duration, sleep quality, and total scores were not associated with any of the other Sleep Diary sleep duration measures over the past week, last weekend, or last night, all ps > .05.

Behavioral Measures

Participants had an average reaction time of 462.07 ms (*SD* = 96.14, range = 309.75 to 785.63) and responded correctly to 84.63% (*SD* = 7.33, range = 60.06% to 97.88%) of the Flanker trials, on average. A repeated-measures ANOVA with a Greenhouse-Geisser correction revealed faster participant reaction time on error trials (*M* = 369.70, *SD* = 74.00 ms) compared to correct trials (*M* = 479.05, *SD* = 102.42 ms), F (1, 218) = 791.74, $p < .001$. Additionally, participant age was related to reaction time on both error trials, r (217) = -0.43, $p < .001$, and correct trials, r (217) = -0.57, $p < .001$), such that reaction time across trials was faster for older children.

Two-tailed correlations were also conducted between the behavioral measures and self-report sleep measures. There were significant negative associations between average reaction time and the PSQI sleep duration, r (217) = -.20, $p = .003$, PSQI sleep quality, r (217) = -.20, $p = .004$, and PSQI total score, r (217) = -.21, $p = .002$, such that less sleep, worse sleep quality, and worse overall sleep in the past month related to faster reaction time across trials. Additionally, reaction time was positively related to the Sleep Diary's sleep duration last week, r (217) = .16, $p = .020$, and sleep duration last night subscales, r (217) = 0.13, $p = .048$, such that participants who got less sleep on school days over the past week and last night had faster reaction times. The Sleep Diary's sleep duration last weekend subscale was not associated with participant reaction time, r (217) = -.06, $p = .398$. Finally, there were no significant associations between accuracy and self-reported sleep difficulties on the PSQI or the Sleep Diary. However, it is important to note that these findings do not remain significant when controlling for child age, all ps > .05.

Error-Related Brain Activity and Age

The ERN *(M* = 1.33, *SD* = 6.26 μV) was significantly more negative than the CRN *(M* = 5.14, *SD* = 5.93 μV) at electrode FCz, F (1, 220) = 111.27, $p < .001$. To examine the unique variance of activity occurring during error trials compared to correct trials, a residual-based difference score (ERN_{resid}) was created using the saved unstandardized residuals from a regression predicting the ERN from the CRN at FCz. Consistent with previous work, age was significantly associated with the ERN_{resid}, r (219) = -.23, $p < .001$, and the ERN, r (219) = -.20, $p = .003$, such that older children were characterized by more error-related brain activity. Age was not related to the CRN, r (219) = -.04, $p = .578$.

Error-Related Brain Activity and Sleep Difficulties

Table 2 reports the correlations between the ERN, CRN, and ERN_{resid} and the sleep measure subscales. There were significant negative relations between the ERN_{resid} and both subscales on the PSQI. The ERN_{resid} was negatively associated with the PSQI duration of sleep subscale, r (219) = -0.13, p = .027, such that fewer hours of sleep over the past month related to enhanced error-related brain activity. Likewise, the ERN_{resid} negatively related to the PSQI sleep quality subscale, r (219) = -0.14, p = .017, such that worse sleep quality over the past month also related to a larger ERN_{resid}. However, while the pattern of these associations remained significant when controlling for task accuracy and reaction time, neither of these associations between the PSQI subscales and the ERN remained significant when controlling for child age. Further, there was one significant association between the ERN_{resid} and the Sleep Diary subscales. The ERN_{resid} was positively associated with the Sleep Diary's sleep duration during school days in the last week, r (219) = 0.20, p < .001, such that fewer minutes of sleep in the past week related to a larger ERN. The correlation between sleep duration in the last week and the ERN_{resid} remained significant when controlling for child age, reaction time, and task accuracy simultaneously. Additionally, the pattern of associations for both measures of sleep difficulties remained the same while controlling for child anxiety symptoms.

Figure 1 depicts topographical headmaps and the waveforms for error, correct, and the difference at electrode FCz for high and low sleep duration and quality groups based on significant relationships between the ERN_{resid} and the PSQI and Sleep Diary subscales.

Moreover, the relations between the ERN and the CRN at FCz and the PSQI subscales of sleep were examined. There was a trend-level negative relationship between the ERN and the sleep quality subscale, r (219) = -0.11, p = .050, such that worse sleep quality related to a larger ERN. There was also a trend-level negative association between the ERN and the duration of sleep subscale, r (219) = -0.11, p = .058, such that fewer hours of sleep related to a larger ERN. There were no significant associations between the CRN and the PSQI subscales or total.

There were also associations between the Sleep Diary items and the ERN and CRN. A positive association between sleep duration on school days over the past week and the ERN was revealed, r (219) = 0.20, p < .001, such that fewer minutes of sleep on school days over the past week was associated with a larger ERN. Additionally, there was a positive relation between sleep duration last weekend and the ERN, r (219) = 0.12, p = 0.043, such that fewer minutes of sleep

the past weekend related to a larger ERN. Additionally, there was a trend-level association between the past weekend's sleep duration and the CRN, r (219) = 0.10, p = 0.061, such that more sleep related to a larger CRN.

Examining Unique Sleep-Related Predictors of the ERN

Three multiple regressions were then conducted to test whether any sleep subscales for the PSQI or the Sleep Diary predicted a unique amount of variance in the ERN_{resid} while controlling for the other subscales. The first regression included the PSQI sleep duration and sleep quality subscales predicting the ERN_{resid}. Neither sleep duration, β = -0.09, t (218) = -1.34, p = .182, nor sleep quality, β = -0.11, t (218) = -1.60, p = .112, uniquely predicted the ERN_{resid} while controlling for the other subscale.

The second regression included the Sleep Diary subscales of sleep duration over the past week, last weekend, and last night predicting the ERN_{resid}. The sleep duration on school days over the last week uniquely predicted the ERN_{resid} while controlling for sleep duration last weekend and last night, β = 0.20, t (217) = 2.93, p = .004. Neither sleep duration last weekend, β = 0.06, t (217) = 0.90, p = .369, nor sleep duration last night, β = -0.04, t (217) = -0.52, p = .606, uniquely predicted the ERN_{resid} while controlling for the other Sleep Diary subscales.

Finally, a third regression was conducted including all the subscales from the PSQI and the Sleep Diary predicting the ERN_{resid}. The Sleep Diary's sleep duration on school days over the last week uniquely predicted the ERN_{resid}, β = 0.19, t (215) = 2.75, p = .006, while controlling for Sleep Diary sleep duration last weekend, Sleep Diary sleep duration last night, PSQI sleep duration over the past month, and PSQI sleep quality over the past month. None of the other sleep subscales on the Sleep Diary or PSQI uniquely predicted the ERN_{resid} while controlling for the other subscales. Across all three regressions, the pattern of results remained the same when controlling for child age, reaction time, accuracy, and child anxiety symptoms simultaneously.

Relations between Sleep Difficulties, Anxiety, and the Error-Related Negativity

The associations between the SCARED-P anxiety subscales and the ERN_{resid} were examined (**Table 3**). There were significant negative correlations between the social anxiety subscale and the ERN_{resid}, r (219) = -.17, p = .007, and between the total anxiety and the ERN_{resid}, r (219) = -.14, p = .016, such that as anxiety increased, the magnitude of error-related

brain activity also increased. Additionally, there were trend-level negative relationships between the ERN_{resid} and the panic subscale, r (219) = -.11, p = .050, the generalized anxiety subscale, r (219) = -.10, p = .074, as well as with the school avoidance subscale, r (219) = -.10, p = .069, such that as anxiety increased, the ERN became more negative.

There were also several significant associations between the PSQI and SCARED-P anxiety subscales, such that increased anxiety was associated with increased sleep-related difficulties. The general pattern of associations remained the same when controlling for child age (i.e., increased anxiety is related to increased sleep-related difficulties). However, there were no significant associations between the Sleep Diary measure of recent sleep duration and any subscales of anxiety on the SCARED-P (See **Table 3** for all values).

Assessing the ERN as an Independent Predictor of Sleep Difficulties

In order to test whether the ERN_{resid} is a unique predictor of sleep difficulties above and beyond total child anxiety symptoms, regressions were conducted entering the ERN_{resid} and total child anxiety as predictors of each sleep difficulty subscale on the PSQI and Sleep Diary. The regression entering the ERN_{resid} and total anxiety predicting PSQI sleep duration was significant, F (2, 218) = 4.66, p = .010, and anxiety was an independent predictor of chronic sleep duration difficulties beyond the ERN_{resid}, β = 0.16, t (218) = 2.34, p = .020. The analysis examining PSQI sleep quality as the outcome had a similar pattern, in that the overall model was significant, F (2, 218) = 11.80, p < .001, and anxiety was an independent predictor of chronic sleep quality difficulties controlling for the ERN_{resid}, β = 0.004, t (218) = 4.33, p < .001.

Sleep duration on school days over the last week, the past weekend, and last night, as measured by the Sleep Diary, were then separately examined as the regression outcomes with the ERN_{resid} and anxiety as predictors. The regression predicting sleep duration on school days over the last week was significant, F (2, 218) = 4.73, p = .010, and the ERN_{resid} was an independent predictor of sleep duration on school days over the last week above and beyond anxiety, β = 2.92, t (218) = 3.03, p = .003. However, neither the model predicting sleep duration over the past weekend, F (2, 218) = 0.918, p = .401, nor that predicting sleep duration last night, F (2, 218) =1.08, p = .341, was significant.

Moderation Models – Interactions between Sleep Difficulties and the ERN Predicting Anxiety Severity

We also conducted exploratory analyses to examine to what extent the relationship between sleep difficulties and anxiety might differ based on the magnitude of the error-related negativity. To do so, we conducted two sets of moderation models. In the first set of models, we tested the interaction between sleep difficulties and the ERN_{resid} predicting child total anxiety. Additionally, as prior work has found a specific relationship between the ERN and social anxiety (Endrass et al., 2014; Judah et al., 2016; Kujawa et al., 2016; Lahat et al., 2014), a finding that was replicated in the current study, we utilized the social anxiety subscale of the SCARED-P as the outcome of the second set of models. We focused these analyses on the domains of sleep difficulties from both the PSQI and Sleep Diary that our initial analyses suggested were related to the ERN_{resid}.

In the first model, we examined the interaction between the child report of sleep duration from the PSQI and the ERN_{resid} predicting total anxiety. Results suggested that this interaction was not significant, $\Delta R^2 = .002$, F (1, 217) = 0.42, $p = .520$. In the next model, we examined the interaction between sleep quality from the PSQI and the ERN_{resid} predicting child total anxiety. Results suggest a significant interaction, $\Delta R^2 = 0.027$, F (1, 217) = 6.78, $p = .010$, such that the relationship between sleep quality and total anxiety was stronger for those with a relatively larger ERN_{resid}, $\beta = 7.06$, 95% CI [4.21, 9.90], $t = 4.88$, $p < .001$, compared to those with a relatively smaller ERN_{resid}, $\beta = 1.76$, 95% CI [-0.95, 4.48], $t = 1.28$, $p = .202$ (**Figure 2**). The final model tested the interaction between the Sleep Diary's sleep duration from school days during the past week and the ERN_{resid} predicting total anxiety, and results indicated the interaction was not significant, $\Delta R^2 = 0.0023$, F (1, 217) = 0.50, $p = .479$.

Further, moderation analyses were conducted to test the interaction between sleep difficulties and the ERN_{resid} predicting child *social anxiety*, specifically. The first model testing the interaction between child report of sleep duration on the PSQI and the ERN_{resid} predicting social anxiety suggested a nonsignificant interaction, $\Delta R^2 = .009$, F (1, 217) = 2.10, $p = .149$. Next, we examined the interaction between PSQI sleep quality subscale and the ERN_{resid} predicting child social anxiety. Results suggest a significant interaction, $\Delta R^2 = 0.023$, F (1, 217) = 5.51, $p = .020$, such that the relationship between sleep quality and social anxiety was stronger for those with a relatively larger ERN_{resid}, $\beta = 1.89$, 95% CI [.80, 2.99], $t = 3.40$, $p < .001$,

compared to those with a relatively smaller ERN_{resid}, β = 0.05, 95% CI [-1.00, 1.10], t = 0.10, p = .921 (**Figure 3**). The final model tested the interaction between the Sleep Diary's sleep duration on school days during the past week and the ERN_{resid} predicting social anxiety, and results indicated the interaction was not significant, ΔR^2 = 0.005, F (1, 217) = 1.04, p = .309. Notably, the pattern of results across interaction analyses remains the same when controlling for child age, task accuracy, average reaction time, and child-report of depression severity simultaneously.

Mediation Models – Sleep Difficulties and Anxiety

To examine whether the relationship between sleep difficulties and anxiety was partially accounted for by the ERN, mediation models were tested. The first set of models used total anxiety from the SCARED-P as the outcome, and the second set of models used social anxiety from the SCARED-P as the outcome, based on prior work (as noted above) that has demonstrated an association between the ERN and social anxiety. Only those sleep subscales from the PSQI and Sleep Diary with significant correlations with the ERN_{resid} were examined.

First, three models examining the ERN_{resid} mediating the relations between sleep difficulties and total anxiety severity as the outcome were assessed. The initial model testing whether the ERN_{resid} mediated the relationship between the PSQI's sleep duration and total anxiety was not significant, *indirect effect* = 0.21, 95% CI [-.010, .537]. Further, there was not a significant indirect effect of the ERN_{resid} mediating the relation between the PSQI's sleep quality and total anxiety severity, *indirect effect* = 0.23, 95% CI [-.048, .630].

Lastly, we examined a model wherein the relationship between the Sleep Diary's sleep duration on school days during the past week and total anxiety severity was mediated by the ERN_{resid}. Results supported the mediation model, such that the indirect path from sleep duration on school days over the past week to total anxiety severity via the ERN reached significance, *indirect effect* = -0.001, 95% CI [-.0026, -.0001] (**Figure 4**). To examine the specificity of this model, the mediator (i.e., the ERN_{resid}) and the outcome (i.e., total anxiety) were reversed. The original model was determined to demonstrate specificity, as the indirect path in this alternative model was not significant, *indirect effect* = <.001, 95% CI [-.0003, .0007].

Next, we examined three mediation models wherein social anxiety severity was the outcome. Results supported the first model testing whether the relation between the PSQI's sleep duration and social anxiety is mediated by the ERN_{resid}, *indirect effect* = .09, 95% CI [.004, .247]

(**Figure 5**). This model demonstrates specificity, as the indirect effect is no longer significant when switching the mediator (i.e., ERN_{resid}) and the dependent variable (i.e., social anxiety), *indirect effect* = -.16, 95% CI [-.401, .011].

Then, the model examining whether the relation between the PSQI's sleep quality and social anxiety severity is mediated by the ERN_{resid}. Results supported a significant model, *indirect effect* = .12, 95% CI [.002, .281] (**Figure 5**). However, this model does not demonstrate specificity, as the indirect effect remains significant when switching the mediator (i.e., ERN_{resid}) and the outcome variable (i.e., social anxiety severity), *indirect effect* = -.23, 95% CI [-.542, .016].

The final model examined whether the relation between the Sleep Diary's sleep duration on school days during the past week and social anxiety severity was mediated by the ERN_{resid}. Results revealed a significant model, *indirect effect* = -.001, 95% CI [-.001 0, -.0001] (**Figure 5**). This model demonstrates specificity, as the indirect effect is no longer significant when switching the mediator (i.e., ERN_{resid}) and the outcome variable (i.e., social anxiety), *indirect effect* = <.001, 95% CI [-.0002, .0008].

Of note, for all significant mediation models, the pattern of results remained the same when controlling for task accuracy, but they were no longer significant when separately controlling for average task reaction time, child age, or child-report of depression severity.

CHAPTER 4

DISCUSSION

The current study is a novel examination of the relationship between error-related brain activity (i.e., the ERN) – a neural marker for anxiety – and sleep difficulties in youth ages 8 to 15 years old. In examining both subjective chronic sleep difficulties (i.e., over the past month via the PSQI), and recent sleep difficulties (i.e., school days over the past week, last weekend, and last night via the Sleep Diary), we found significant associations such that chronically shorter sleep duration, chronically worse sleep, and recent shorter sleep duration on school days over the past week were each associated with heightened error-related brain activity. Additionally, recent sleep duration on school days over the past week was a unique predictor of the ERN, and this association between the ERN and sleep duration on school days over the past week was independent of anxiety. Results from moderation analyses revealed that the ERN moderates the relationship between chronic sleep quality and child total anxiety, as well as that between chronic sleep quality and child social anxiety. Finally, mediation analyses suggested that the link between sleep difficulties and anxiety may be partially mediated by error-related brain activity.

Results from the current study supported our hypothesis that greater *chronic* sleep problems would be associated with elevated error-related brain activity. Specifically, we found that both a shorter sleep duration over the past month and a worse sleep quality over the past month were each associated with a larger ERN. Although prior work examining associations between sleep problems and the ERN has mainly focused on manipulations of acute sleep deprivation conditions or recent sleep difficulties, our findings align with work suggesting the likely possibility that chronic sleep disturbances contribute to risk for anxiety disorders (Caporino et al., 2017), with the ERN serving as a marker of risk for anxiety. The pattern of results also remained the same when controlling for task accuracy, reaction time, and child anxiety symptoms, indicating that behavior during the task and anxiety do not account for these associations. However, since chronic sleep disturbances and the ERN have each been found to increase with age – findings replicated in the current study – it is important to note that the associations between *chronic* sleep difficulties and the ERN did not remain significant when controlling for child age. The association between chronic sleep difficulties and the ERN may therefore be better explained by developmental factors.

Additionally, our prediction that sleep difficulties would be associated with all subtypes of anxiety was mostly true, but only for chronic sleep difficulties. These results are in line with prior research describing relations between sleep disturbances (e.g., reduced sleep duration, worse sleep quality, insomnia) and anxiety disorders, including generalized anxiety, social anxiety, panic disorders, and phobias (for a review, see: Cox & Olatunji, 2016). While chronic sleep issues related to these subtypes of anxiety, *recent* sleep duration difficulties were not found to relate to any subtype of anxiety in the present study. Perhaps prolonged naturalistic sleep difficulties have a cumulative impact on anxiety, such that disruptions in sleep most negatively affect youth when they become established patterns or habits. These may include impairment in neuronal functions due to cell loss, which can limit cognitive abilities like memory consolidation, as well as weaken gene expression processes, and even structural neural alterations, such as decreased gray matter volume at the whole-brain level (Jan et al., 2010; Sung et al., 2020).

Furthermore, our results partially supported our hypothesis that the ERN would relate to performance-based domains of anxiety (i.e., generalized anxiety and social anxiety) but not fear or arousal-based anxiety symptoms. Although the association between the ERN and social anxiety symptoms, which is a well-established relationship (Endrass et al., 2014; Judah et al., 2016; Kujawa et al., 2016; Lahat et al., 2014), was replicated in the current study, we did not find a relationship between the ERN and generalized anxiety in this sample.

On the other hand, results did not support the hypothesis that *recent* sleep problems would be related to a blunted ERN – a hypothesis in the opposite direction from the ERN's relationship with chronic sleep difficulties. We formed this hypothesis based on prior findings that acute sleep impairment, via experimentally-induced mental fatigue and acute sleep deprivation, is associated with a reduced ERN in adults (Hsieh et al., 2007; Kato et al., 2009; Scheffers et al., 1999; Tsai et al., 2005; Xiao et al., 2015). Additionally, a study authored by Fueggle et al. (2018) examined the association between recent naturalistic sleep duration, measured via actigraphy over the past week, and the ERN in young adults. Their results converged with those of experimental studies, finding a reduced ERN amplitude for those with shorter sleep durations (i.e., less than seven hours nightly) compared to those with longer sleep durations (i.e., between seven and nine hours nightly). However, in assessing the relationship between the ERN and self-reported sleep duration on school days over the past week, last

weekend, and last night in the current study, we found the opposite results. Specifically, less sleep on school days over the past week was associated with an *elevated* ERN amplitude, but sleep duration last weekend and last night were not associated with the ERN.

The present study's results regarding *recent* sleep problems and the ERN may diverge from prior literature due to a variety of reasons. First, the current study examined sleep duration by using participants' self-reported recollection of their sleep and wake times each day over the past week. This method of self-report is less accurate in quantifying sleep duration than is one that occurs in a controlled lab environment or that is determined with actigraphy – an objective measurement of the sleep-wake cycle based on motion captured by an accelerometer. Additionally, participant self-report of sleep may be more related to one's *perceptions* surrounding their sleep; sleep perception may be more related to symptoms of anxiety than objective sleep measures are (Blake et al., 2017; Klumpp et al., 2017; Van Ravesteyn et al., 2014). Next, while most prior studies examining the association between the ERN and recent sleep difficulties have been manipulated in a lab condition, the current study assessed naturalistic sleep duration without manipulation. Sleep that is manipulated in a lab, such as in sleep deprivation conditions, is likely a drastic change from a person's naturalistic sleep patterns. However, assessing recent naturalistic sleep difficulties on school days over the past week may have simply served as a temporally closer representation of a chronic, established sleep pattern for individuals. Finally, extant studies have primarily examined adult populations whereas the present study explores these associations in a developmental sample of children and adolescents. Considering previous work demonstrating that the ERN and the anterior cingulate cortex undergo substantial changes throughout development, it is possible that the impact of sleep deprivation on the ERN differs between adults, children, and adolescents (Tamnes et al., 2013; Velanova et al., 2008). Future work should examine this possibility.

Moreover, in discerning whether particular types and time-courses of sleep problems differentially predicted the ERN, we found that recent sleep duration on school days over the past week was a unique predictor of the ERN amplitude above and beyond other recent and chronic sleep difficulties. This association was maintained even when controlling for child age, task reaction time, task accuracy, anxiety, and depression, demonstrating the relationship is not due to child development, task performance, or internalizing symptoms. This is the first study, to our knowledge, to examine the differential impact of various subjective sleep problems on the

ERN. One possible interpretation of these results is that recent sleep duration on school days might be the best predictor of overall sleep quality for children. It could be that children have a better memory for their experiences of sleep on school days over the past week due to the greater structure inherently required on these days (e.g., needing to wake up at a particular time to arrive to school, parental control over sleep time). It could also be that worse sleep experiences on school days are more indicative of a general pattern of dysfunctional sleep, whereas on weekends children and adolescents may sometimes choose to have poor sleep for the purpose of engaging in social scenarios (e.g., sleepovers, time with friends) and hobbies, but these choices do not necessarily represent a child's overall naturalistic sleep pattern.

In our first set of exploratory analyses, we sought to determine whether the ERN interacts with sleep difficulties to predict child total anxiety and social anxiety. Results suggested that the relation between worse sleep quality over the last month and greater total anxiety was significant only for those with an average to large ERN (i.e., one standard deviation below the mean). Examining the associations between sleep difficulties and social anxiety, specifically, the same pattern was found such that the relationship between sleep quality and social anxiety was significant for those with a relatively large ERN. Both these models remained significant when controlling for child age, task performance, and depression. These results suggest that amongst children with elevated error-related brain activity, worse sleep quality is associated with increased anxiety symptoms; however, amongst children with decreased error-related brain activity, sleep quality does not relate to anxiety. Thus, having a large ERN may put children at risk for sleep-related increases in anxiety. Another interpretation of these findings is that having a blunted ERN may be a buffer or protective factor for children – such that poor sleep quality does not relate to increased anxiety symptoms. Our results, therefore, underscore the potential roles of an elevated ERN and poor sleep quality as vulnerability factors that lead to worse outcomes (i.e., increased anxiety) for youth when they occur in combination.

Finally, as a preliminary first step toward examining whether the ERN may play a causal role in sleep difficulties and anxiety, we assessed mediation models investigating whether sleep difficulties impact anxiety via the ERN. Results examining overall anxiety as an outcome found that the ERN only mediated the relation between recent sleep duration on school days over the past week and total anxiety. When examining social anxiety as the outcome, the ERN mediated the association between social anxiety and chronic sleep duration, chronic sleep quality, and

recent sleep duration on school days. Importantly, while these mediation models remained significant when controlling for task accuracy, they did not remain significant when separately accounting for child age, task reaction time, or depression. It is likely that aspects of development, task performance, and internalizing symptoms may each individually account for these results.

One potential explanation of these initial findings is that sleep impacts anxiety via the ERN due to sleep disturbance-related alterations in neural structures. Sleep disturbances have been found to lead to a variety of alterations in the anterior cingulate cortex (ACC), the region of the brain to which the ERN is localized. Specifically, a greater volume of the rostral ACC has been found for those with heightened insomnia (Winkelman et al., 2013), and worse naturalistic sleep has been associated with less activation of the dorsal ACC, an area associated with emotion and stimulus reappraisal (Klumpp et al., 2017). In terms of network connectivity, decreased functional connectivity between the amygdala and ventral ACC — a network associated with emotional responses — has been found to be related to sleep debt (Motomura et al., 2013), and decreased connectivity from the right insula to the left ACC, which is a network associated with information processing, has been found in those with sleep deprivation (Zhang et al., 2021). Finally, research has found reduced gamma-aminobutyric acid (GABA), which is a main inhibitory neurotransmitter, levels in the ACC for people with shorter naturalistic sleep durations (Park et al., 2020).

However, since the current analyses were conducted in a cross-sectional sample, the information regarding directionality and whether there is a true underlying causal mechanism is limited. Future studies examining these effects longitudinally are needed to clarify these questions. One way to do so would be by assessing the ERN and anxiety levels of youth with sleep difficulties before, during, and after a sleep intervention. Several types of sleep interventions, including cognitive (Blake et al., 2017), school-based (Cain et al., 2011; Kira et al., 2014; Moseley & Gradisar, 2009; Rey et al., 2020; Wolfson et al., 2015), education with parental involvement (Bonnar et al., 2015), and mindfulness-based (Bei et al., 2013), have effectively improved sleep disturbances across age groups. Determining whether the sleep intervention leads to a reduction in sleep disturbances, followed by a reduction in the ERN, and then finally a reduction in anxiety would allow us to specify the direction of this potential causal model.

Notably, many of the current study's findings did not remain significant when controlling for participant age. One possible explanation may be related to a person's chronotype, which describes an individual's tendency either toward a morning sleep cycle (i.e., they tend to sleep and wake earlier) or an evening sleep cycle (i.e., they tend to sleep and wake later) (Roenneberg et al., 2003). While children initially tend to be categorized as a morning chronotype, research has shown a normative developmental shift, associated with the onset of puberty, toward an evening chronotype occurs during adolescence (Hagenauer & Lee, 2012; Kuula et al., 2018; Randler et al., 2009; Roenneberg et al., 2004). Additionally, having an evening chronotype has been associated with mental health difficulties, including an association with anxiety even when controlling for specific sleep disturbances (Cox & Olatunji, 2019; Taylor & Hasler, 2018). Perhaps assessing the degree to which participants have an evening chronotype might better account for the relations between sleep and the ERN. It could be that those with early onset or delayed chronotype changes experience difficulties adjusting to social structures (e.g., early school start times for early onset evening chronotypes) that results in functional impairment compared to their normatively developing peers.

The current study has several limitations that should be addressed in future research. Since solely female participants were involved in this study, we cannot generalize about the applicability of the findings to males. Further, the distinction between chronic (i.e., past month) and recent (i.e., past week) sleep difficulties was quite close in time; this distinction would likely benefit from more exaggerated differences in naturalistic sleep difficulties (e.g., past 3 months for a measure of chronic sleep rather than just the past month). As sleep has been found to shift across development, assessing pubertal stages and hormones may clarify some of the mechanisms underlying the association between sleep problems and the ERN. It may also be interesting to examine the contributions of parental control over sleep schedules to these relationships. Lastly, as sleep duration was a large focus of the current study, accounting for time spent napping during the day would also be important to be able to assess the potential role of cumulative sleep debt in future iterations of this study.

Overall, the present study builds upon past research examining associations between sleep difficulties and the ERN by specifically exploring self-report of both chronic and recent naturalistic sleep problems in children and adolescents, as well as how these sleep difficulties differentially relate to the ERN. Results lay a foundation for future research to clarify the

direction of these relations and the potential causal role of the ERN in sleep and anxiety associations. Understanding specific aspects of sleep as it is associated with both error-related brain activity and anxiety may help us identify specific circumstances under which early interventions may be most effective.

Table 1

Self-Report Measures: Means and Standard Deviations

Self-Report Measure	Mean	Standard Deviation
Pittsburgh Sleep Quality Index (Child-Report)		
Sleep Duration Subscale	0.45	0.69
Sleep Quality Subscale	0.75	0.58
Total Score	4.68	2.81
Screen for Child Anxiety Related Emotional Disorders (Parent-Report)		
Panic and Somatic Symptoms Subscale	1.12	2.09
Generalized Anxiety Subscale	3.42	3.36
Separation Anxiety Subscale	1.58	2.02
Social Anxiety Subscale	3.47	3.31
School Avoidance Subscale	0.77	1.11
Total Score	10.37	8.80
Sleep Diary (Child-Report)		
Past Week School Days: Minutes Sleep Duration	565.27	221.85
Last Weekend: Minutes Sleep Duration	638.84	207.90
Last Night: Minutes Sleep Duration	586.20	219.68

Note. Means and standard deviations for child report of sleep using the Pittsburgh Sleep Quality Index (PSQI) and Sleep Diary, as well as parent report of anxiety symptoms using the Screen for Child Anxiety Related Emotional Disorders (SCARED).

Table 2

Pearson's R for Correlations between the ERN, CRN, and ERN_{resid} and Sleep Measures

Child-Report Sleep Measure	ERN	CRN	ERN_{resid}
Pittsburgh Sleep Quality Index (Child-Report)			
Sleep Duration Subscale	-0.11[t]	-<0.01	-0.13*
Sleep Quality Subscale	-0.11[t]	0.01	-0.14*
Total Score	-0.04	0.06	-0.08
Sleep Diary (Child-Report)			
Past Week School Days: Minutes Sleep Duration	0.20**	0.09	.20**
Last Weekend: Minutes Sleep Duration	0.12*	0.10[t]	0.09
Last Night: Minutes Sleep Duration	-0.03	-0.04	-0.01

*p<.05, **p<.01, [t]p<.08

Note. 1-tailed Correlations between regression-based difference scores (ERN_{resid}) from the unstandardized residuals from a regression predicting the ERN from the CRN, the ERN, and the CRN at electrode FCz with the Pittsburgh Sleep Quality Index (PSQI) and Sleep Diary measures of child-report sleep difficulties.

Table 3

Pearson's R for Correlations between Anxiety Measure and the ERN_{resid} and Sleep Measures

	Screen for Child Anxiety Related Emotional Disorders (Parent-Report)					
Child-Report Sleep Measure	Panic and Somatic Symptoms Subscale	Generalized Anxiety Subscale	Separation Anxiety Subscale	Social Anxiety Subscale	School Avoidance Subscale	Total Score
ERN_{resid}	-0.11^{t}	-0.10^{t}	-0.02	-0.17**	-0.10^{t}	-0.14*
Pittsburgh Sleep Quality Index (Child-Report)						
Sleep Duration Subscale	0.20**	0.14*	-0.01	0.14*	0.17**	0.17**
Sleep Quality Subscale	0.27**	0.25**	0.15*	0.18**	0.29**	.30**
Total Score	0.33**	0.19**	0.15*	0.20**	0.32**	0.30**

Table 3 - continued

Child-Report Sleep Measure	Panic and Somatic Symptoms Subscale	Generalized Anxiety Subscale	Separation Anxiety Subscale	Social Anxiety Subscale	School Avoidance Subscale	Total Score
Sleep Diary (Child-Report)						
Past Week School Days: Minutes Sleep Duration	-0.08	0.02	0.08	-0.07	-0.08	-0.03
Last Weekend: Minutes Sleep Duration	0.03	0.04	-0.04	-0.01	-0.01	0.01
Last Night: Minutes Sleep Duration	-0.10^{t}	-0.11^{t}	0.02	-0.07	-0.10^{t}	-0.10^{t}

*p<.05, **p<.01, ^{t}p<.08

Note. 1-tailed Correlations between Screen for Child Anxiety Related Emotional Disorders (SCARED) parent-report anxiety measure and regression-based difference scores (ERN_{resid}) from the unstandardized residuals from a regression predicting the ERN from the CRN, the Pittsburgh Sleep Quality Index (PSQI), and Sleep Diary measures of child-report sleep difficulties.

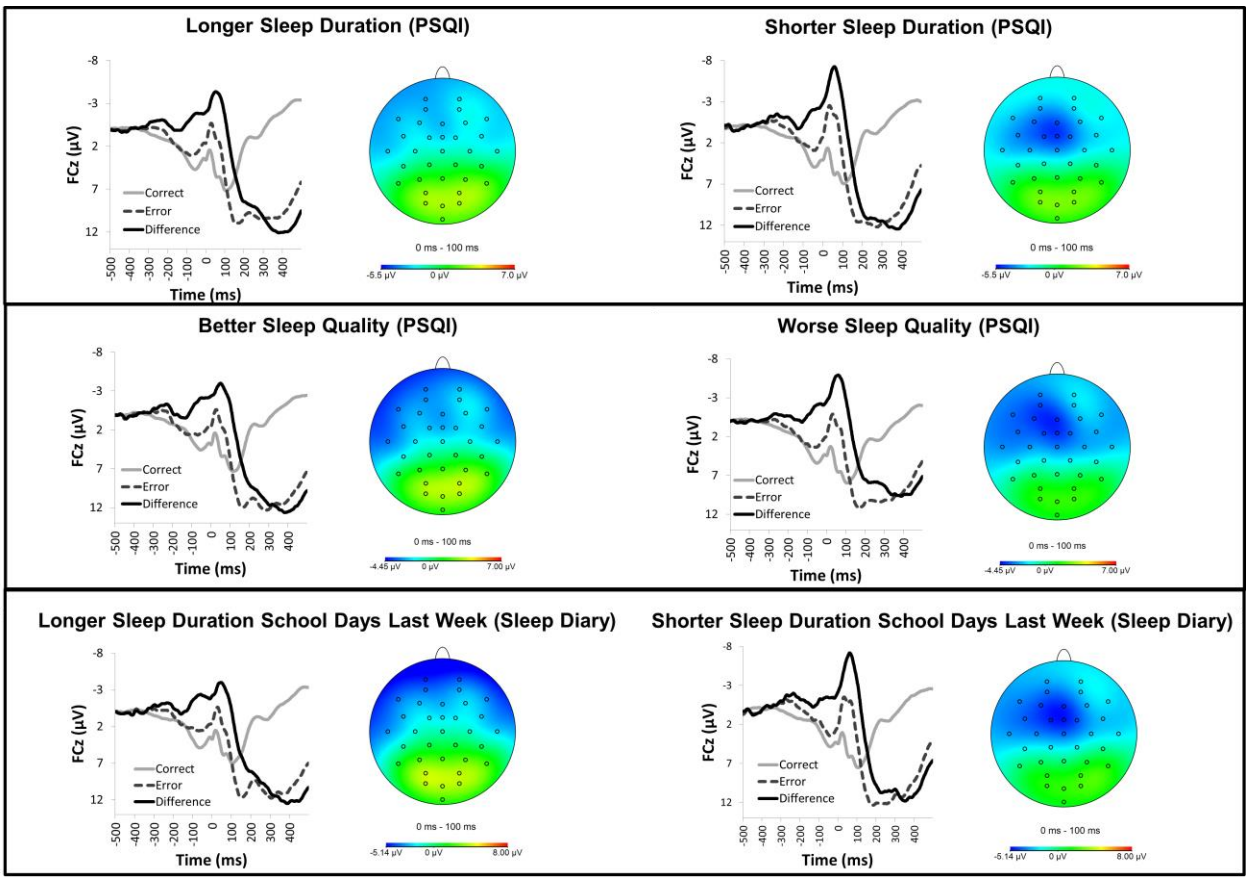

Figure 1

Topographical Headmaps for the ERN$_{resid}$ in Sleep Duration and Quality Groups

Note. Topographical headmaps for the difference wave (ΔERN; error minus correct for 0 to 100 ms) and the waveforms for error, correct, and difference at electrode FCz for longer and shorter sleep duration and better and worse sleep quality groups based on significant relationships between the ERN$_{resid}$ and the Pittsburgh Sleep Quality Index (PSQI; assessing chronic sleep difficulties occurring over the passing month) and the Sleep Diary (assessing recent sleep difficulties over the past week) subscales. The PSQI sleep duration and quality headmaps were created using participants with a PSQI subscale score greater than 0 for the subscale as being in the "shorter" sleep duration or "worse" sleep quality groups, and scores equal to 0 for the subscale comprised the "longer" sleep duration or "better" sleep quality groups. The Sleep Diary sleep duration headmaps were created using participants in the top ("longer") and bottom ("shorter") quartiles for the subscale.

Figure 2

The Interaction between Past Month Sleep Quality and the ERN Predicting Total Anxiety

Note. Depiction of the regression-based wave (ERN_{resid}; the unstandardized residuals from a regression predicting the ERN from the CRN for 0 to 100 ms) at electrode FCz by sleep quality over the past month interaction predicting child total anxiety. Sleep quality over the past month was self-reported on the Pittsburgh Sleep Quality Index (PSQI) and child total anxiety was reported by parents on the Screen for Child Anxiety Related Emotional Disorders (SCARED). A smaller ERN is defined as a less negative ERN (i.e., one standard deviation above the mean) and larger ERN as more negative (i.e., one standard deviation below the mean). The relationship between sleep quality over the past month and total anxiety was stronger for those with a relatively larger ERN. The categorical distinction in sleep quality is for visualization purposes only.

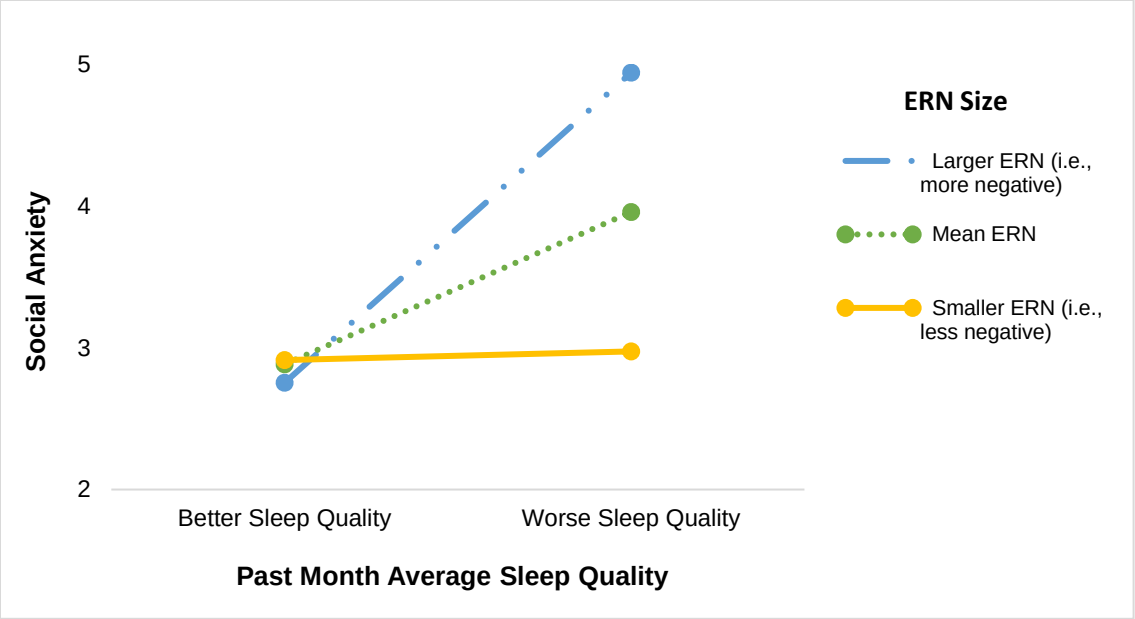

Figure 3

The Interaction between Past Month Sleep Quality and the ERN Predicting Social Anxiety

Note. Depiction of the regression-based wave (ERN_{resid}; the unstandardized residuals from a regression predicting the ERN from the CRN for 0 to 100 ms) at electrode FCz by sleep quality over the past month interaction predicting child social anxiety. Sleep quality over the past month was self-reported on the Pittsburgh Sleep Quality Index (PSQI) and child social anxiety was reported by parents on the Screen for Child Anxiety Related Emotional Disorders (SCARED). A smaller ERN is defined as a less negative ERN (i.e., one standard deviation above the mean) and larger ERN as more negative (i.e., one standard deviation below the mean). The relationship between sleep quality over the past month and social anxiety was stronger for those with a relatively larger ERN. The categorical distinction in sleep quality is for visualization purposes only.

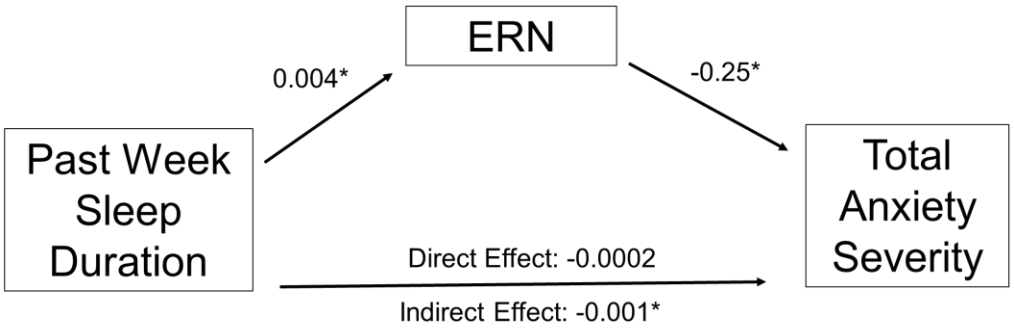

Figure 4

Mediation Model Assessing the ERN as a Mediator for the Relation between Sleep Duration on School Days Last Week and Total Anxiety Severity

Note. Regression coefficients for the relation between sleep duration on school days over the past week on the child-reported Sleep Diary and total anxiety assessed via the Screen for Child Anxiety Related Disorders (SCARED) parent-report measure, as mediated by the ERN.

*$p < .05$

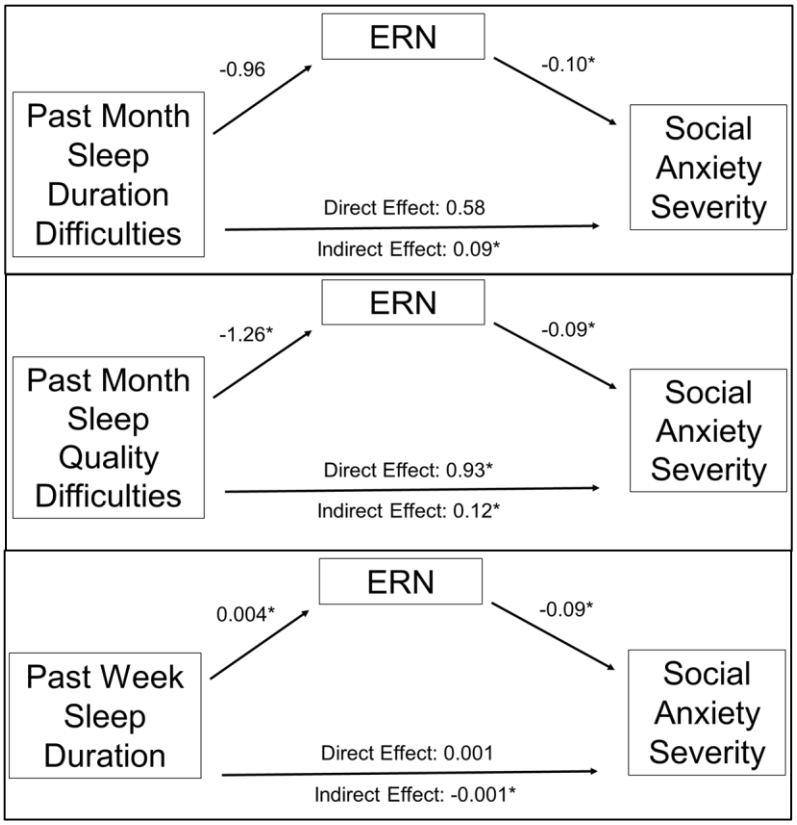

Figure 5

Mediation Models Assessing the ERN as a Mediator for the Relation between Sleep Difficulties and Social Anxiety Severity

Note. Regression coefficients for the relationship between sleep difficulty measures, from the child-report Pittsburgh Sleep Quality Index (PSQI; i.e., past month sleep duration, sleep quality) and the child-reported Sleep Diary (i.e., past week sleep duration), and social anxiety assessed via the Screen for Child Anxiety Related Disorders (SCARED) parent-report measure, as mediated by the ERN.

*$p < .05$

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