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Investigating the EEG Error-Related Negativity in College Students with ADHD, Anxiety, and Depression

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Investigating the EEG Error-Related Negativity in College Students with ADHD, Anxiety, and Depression

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Abstract

Error-related negativity (ERN) is an event-related potential elicited by the commission of errors. It appears as a negative deflection peaking between 50ms and 100ms after an erroneous response. Previous literature demonstrated that individuals who suffer from either anxiety or depression display a higher ERN amplitude compared to a control group. It has also been shown that people with ADHD display a lower ERN amplitude. Based on these findings, we investigated the relationships between these three disorders and their effects on the amplitude of the ERN. We recruited thirty-one students at East Tennessee State University and gathered data on their level of anxiety, depression, and ADHD through completion of three surveys: the Beck Anxiety Inventory, Beck Depression Inventory, and the ADHD self-report scale. Subsequently, participants were asked to perform a modified Flanker task while their EEG was collected using a 32-channel EEG cap. ERN amplitude for error responses was significantly higher than ERN amplitude for correct responses. In contrast with previous literature, no significant influence on the ERN was observed due to anxiety, depression, or ADHD. Additional research on the topic with larger sample size and different diagnostic procedures may be necessary to further investigate the phenomenon.

Keywords: *Error-related Negativity, Anxiety, Depression, ADHD*

Introduction

Our ability to learn from mistakes is dependent upon mechanisms that allow for the detection of errors. The human brain has an error detection and compensation system and its activity can be detected using electroencephalography (EEG). EEG technology is a non-invasive means of recording electrical brain activity at the scalp. This process is brought forward by using electrodes connected to the scalp that record electrical potentials associated to stimulus events; the signal is then amplified and plotted as a function of change in voltage over time (Luck, 2005). The neural responses time-locked in the EEG are referred to as event-related potentials or ERPs, and they may be elicited by different types of stimuli. They can vary in their amplitudes, latencies, and directionality depending on the source of the response. ERP research investigates how ERPs are related to cognitive functions such as learning, memory, sensory perception, and to neurological disorders (Sur & Sinha, 2009).

The study of ERPs can provide better insight into internal cognitive processing such as error detection. The brain's error detection activity is manifested in the form of an ERP known as error-related negativity (Gehring, 1993). The error-related negativity, also referred to as ERN, is seen as a negative deflection in averaged electrical brain activity and it appears following the commission of an error and it is absent after correct responses (Pailing, 2002). This negative deflection begins approximately when the error is made, and it peaks 50-100 ms later (Gehring, 1993). By examining the amplitude and latency of the ERN, we can understand the time course, as well as how aware a participant is that an erroneous response has occurred.

A wide array of evidence supports that the ERN is evoked in the anterior cingulate cortex (ACC). Dehaene, Posner and Tucker (1994) first conducted a dipole study that suggested the ERN dipole was located in the brain midline, within the ACC. Following studies that used event-

related functional magnetic resonance imaging techniques confirmed this hypothesis, finding the rostral ACC and the left lateral frontal cortex to be the main components of error detection and processing (Kiehl, Liddle, & Hopfinger, 2000). Brown and Braver (2005), further investigated the error detection mechanism from the ACC through neuroimaging and computational neural modeling. Results supported the theory that the ACC detects error likelihood, and that conflict and error detection are special instances of this function (Brown & Braver, 2005).

Researchers use many different paradigms to elicit ERNs in study participants. The paradigms are designed to make the participants commit an error, usually by asking to complete the task as fast and as accurately as possible. Some of them include: Stop Signal task, Go/No-Go task, Stroop test, and Flanker task (Shiels & Hawk, 2011). For the purposes of this study, we decided to use a Flanker task to increase the likelihood of participants making a mistake, since it is a very common paradigm utilized in a wide range of ERN studies. The task consists of presenting multiple target stimuli; the central target stimulus can either be congruent or incongruent with the other stimuli (Eriksen & Eriksen, 1974). The study participants must respond based on a characteristic of the central stimulus and ignore the surrounding stimuli (Shiels, 2010).

Different factors may influence the amplitude of the ERN. First, the emphasis that is put on the accuracy of the performance is correlated to the magnitude of the ERN (Gehring, 1993). It has been demonstrated that the value that is attributed to an error and the magnitude of the perceived consequences of making a mistake both affect the amplitude of the ERN (Hajcak, 2012). Previous literature also shows that anxiety, depression, and impulsivity, and underlaying trait of attention-deficit/hyperactivity disorder, affect the ERN, indicating possible hyperactivity or hypoactivity of the error processing system in people affected by these disorders.

Anxiety

Anxiety is an emotion commonly referred to as a 'fight or flight' response to threats (Stahl, 2013). Anxiety evolves into an anxiety disorder when the individual experiences persistent symptoms that interfere with daily routines and that can worsen over time (National Institute of Health). As reported in the DMS V, there are multiple types of anxiety disorders, and they all involve a display of excessive fear and anxiety (American Psychiatric Association, 2013). The two core symptoms of anxiety disorder are excessive fear and worry. People may also experience sleep disturbance, concentration issues, fatigue, and other arousal and psychomotor symptoms (Stahl, 2013). The difference in types of anxiety lies on the object that induces the feeling of fear, anxiety, or avoidance behavior (American Psychiatric Association, 2013).

Multiple studies have revealed that anxiety influences the amplitude of the ERN. It has been found that individuals with high levels of general anxiety and worry display a greater magnitude for the ERN compared to both phobic and non-anxious individuals (Hajcak, McDonald & Simons, 2003). Supporting evidence from fMRI studies also showed that people with high anxiety had an increased activity of the anterior cingulate cortex after committing errors (Paulus, Feinstein, Simmons, & Stein, 2004). The differences in ERN in individuals suffering from anxiety are due to aspects of the disorder such as worry, negative affect, emotionality and behavioral inhibition. Studies suggest that personality traits mainly observed in anxious individuals lead to the existing relation between ERN and anxiety (Barker, Troller-Renfree, Pine, & Fox, 2016).

Increased research has been conducted on the different ways in which anxiety may affect the ERN. It has been demonstrated that a relationship exists between the ERN and the age of the

participants affected by anxiety. A study conducted on children between the age 8 and 13, found that for older children the ERN was larger in higher anxiety individuals, and the ERN was smaller for high anxiety younger children (Meyer, Weinberg, Klein, & Hajcak (2012). Age, therefore, seems to influence the ERN in anxious individuals. A previous study conducted on college students also showed the ERN is increased in individuals who suffer from anxiety (Hajcak, McDonald & Simons, 2003). From these findings we can infer that the ERN is not substantially affected by anxiety in early childhood, but as the individual grows older the effects are evident. Due to its strong relation with anxiety, the ERN could help in the understanding of possible risks for anxiety disorder (Hajcak, 2012).

Depression

Depression is a mood disorder that affects individuals in their everyday life. Some common symptoms of depression are hopelessness, sad or anxious mood, guilt, anhedonia, fatigue, appetite change, and suicidal thoughts (National Institute of Mental Health). As described in the DMS V, there are several types of depressive disorders, such as: major depressive disorder, dysthymia, or substance/medication induced depressive disorder. Major depressive disorder consists of sharp changes in affect, cognition, and neurovegetative functions alternating with remission inter-episodes, all lasting for at least two weeks. Dysthymia, or persistent depressive disorder, involves disturbances lasting for at least two years. Some substances or medications, when abused, may also cause depression-like symptoms (American Psychiatric Association, 2013).

A strong overlap exists between the neurobiology of anxiety and depression (Stahl, 2013). This is reflected in the perception of errors in depressed individuals. People affected by depression, in fact, are more sensitive to mistakes and negative feedback (Olvet, D. M., &

Hajcak, G., 2008). Additionally, depressed individuals exhibit a greater ERN amplitude compared to control groups, although error-related positivity is the same independently of the disorder presence (Chiu & Deldin, 2007). In line with these findings, Holmes and Pizzagalli (2008) illustrated that subjects affected by Major Depressive Disorder were less accurate after incorrect responses, displayed a much larger ERN, and had a higher current density in the anterior cingulate cortex and medial prefrontal cortex.

The similarities in error perception between anxiety and depression may be attributed to negative affect, an underlying characteristic of both disorders (Olvet, & Hajcak, 2008). Affective distress and behavioral patterns associated to it are strictly related to executive functions in the frontal lobe, and people who score high for negative affect display larger ERN amplitudes (Luu et al., 2000).

Impulsivity

Impulsivity is associated with an increase in sensitivity to rewards and a decrease in sensitivity to punishments (Corr, 2002; Monterosso & Ainslie, 1999). Research shows that high levels of impulsivity are also correlated to a decrease in ERN amplitude after committing errors (Olvet & Hajcak, 2008). Highly impulsive individuals in fact display a smaller ERN in the medial frontal cortex and a lower sensitivity to punishment in the medial prefrontal cortex compared to less impulsive individuals. This reduced punishment sensitivity in impulsivity is a trait characteristic of many disorders, one of which being attention-deficit/hyperactivity disorder (ADHD) (Potts, George, Martin, & Barratt, 2006).

Attention-deficit/hyperactivity disorder.

Attention-deficit/hyperactivity disorder, most commonly referred to as ADHD, is an onset development disorder most common in children but can continue through adulthood. It

manifests in difficulty in concentrating and in controlling impulsive behaviors. The three main symptoms of ADHD are inattention, hyperactivity, and impulsivity (National Institute of Mental Health). These symptoms become problematic when they interfere with daily activities and development. Inattention symptoms include having difficulty focusing, disorganization and a lack of persistence. Hyperactivity in individuals with ADHD is displayed through excessive motor activity at inappropriate times, persistent movement such as tapping and fidgeting, and sometimes restlessness, especially in adults. Impulsivity manifests as abrupt actions that may result harmful for others, such as social intrusiveness or risky decision making. It is possible that impulsivity is the consequence of a need for immediate gratification and the inability to wait. (American Psychiatric Association, 2013). ADHD is mainly prevalent in children, with a 5% presence across most cultures; about 2.5% of adults are affected by it. Hyperactivity symptoms typically diminish during adolescence, inattention and impulsivity tend to persist through adulthood (American Psychiatric Association, 2013).

Researchers hypothesized that people affected by ADHD, of which impulsivity is a main component, would show a lower ERN, since impulsivity may be a cause of lower sensitivity to error (Potts, George, Martin, & Barratt, 2006; Ruchsow, Spitzer, Gron, Grothe, & Kiefer, 2005). The first studies on the relation between ADHD and ERN began in 2005. Most of them were conducted on children, and they found that those who had ADHD tended to display a lowered ERN compared to non-ADHD children (Shiels & Hawk, 2011).

A Flanker Task has been utilized in several procedures in these types of studies to induce an ERN after commission of errors (Albrech et al, 2008; Jonkman, Melis, Kemner, & Markus, 2007; Van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2006). In 2006, Van Meel, Heslenfeld, Oosterlaan and Sergeant (2006) investigated differences between children affected by ADHD

and a control group in term of error processing by having them perform a modified Eriksen Flanker Task. The findings showed that for children with ADHD error percentage was higher in all conditions compared to the control, and that pressure to respond quickly and incongruency increased error percentage for both groups. A significantly reduced ERN was observed in the ADHD group. These results indicated that ADHD affected online monitoring of errors but not remedial action (Van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2006). Additional studies utilizing the Flanker task for error monitoring purposes supported previous findings, illustrating that children with ADHD displayed a smaller error negativity compared to a healthy control group (Albrech et al, 2008).

From the premise that children with ADHD displayed a lower ERN, Hermann et al. (2009) investigated the effects that ADHD would have on the ERN in adult subjects. They found that individuals affected by ADHD displayed a reduced ERN (Hermann et al., 2009). Additional studies found that adults with ADHD show a normal automatic error detection but fail to evaluate errors at a conscious level (Van der Meere & Roeyers, 2009). These findings add to the evidence that ADHD disturbs error monitoring, showing that the issue persists in adulthood (Van der Meere & Roeyers, 2009).

The existence of a correlation between higher levels of anxiety and depression and a greater magnitude of ERN has been reported (Chiu & Deldin, 2007; Hajcak, McDonald & Simons, 2003); however, scholars have not investigated the relationships between all three onset disorders may concurrently affect the ERN.

In this study we investigated the effects that ADHD, anxiety, and depression combined would have on the ERN in young adults and college students. The relation between ERN magnitude and ADHD, anxiety, and depression scores was therefore investigated to determine

how predictive combinations of the disorders will affect the magnitude of the ERN. Considering that impulsivity lowers the ERN, but anxiety and depression increase it, we hypothesized that participants who scored higher on the ADHD scale survey would have a lower ERN than participants with lower ADHD scores, anxiety, and depression. On the other hand, we hypothesized that depression and anxiety would have a stronger effect on the ERN, therefore participants who scored high on all three scales will have a higher ERN than those with lower scores. To test this hypothesis, we recruited forty-three participants, surveyed them to assess their level of anxiety, depression, and ADHD, and subjected them to a Flanker task to compare their ERN responses.

Methods

In this study we investigated the difference in error-related negativity in participants with different levels of attention-deficit/hyperactivity disorder, anxiety, and depression. To elicit an ERN, the participants engaged in a Flanker task.

Participants

Forty-three undergraduate and graduate students over the age of 18 at East Tennessee State University participated in the study. They were recruited through the ETSU Psychology Research Participation website SONA and they earned three SONA credits as form of compensation. One participant did not complete the study session due to technical malfunctioning after the first block of the Flanker task, therefore their data was not used. The data of four participants were not used because of various EEG recording issues. Additionally, the data of seven more participants was not utilized as they made less than five mistakes and we did not regard it sufficient to analyze an ERN. Demographic data for one participant was missing. Out of the thirty participants of which the demographic data was recorded, 19 were

female and 11 were males, 2 were left handed and 28 were right handed. Their age ranged from 18 years to 35 years of age.

Materials

Surveys.

Participants were asked to complete three assessment surveys to determine whether they suffered from ADHD, anxiety, or depression. The surveys utilized for this study were: Beck Anxiety Inventory (BAI) (Steer & Beck, 1997), Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and Adult ADHD Self-report scale (ASRS v1.1) (Daigre et al., 2009).

The BAI is a self-report instrument utilized to assess the level of anxiety in adolescents and adults (Steer & Beck, 1997). This inventory is made of 21 questions, in which people must answer questions rating them on a scale from 0 to 3. Depending on their score, people may have low (0-21), moderate (22-35), or potentially concerning (36 and above) levels of anxiety (Steer & Beck, 1997).

The BDI is also a 21-item inventory, and it measures levels of depression in adults. In this self-report survey, people choose the option that is closest to how they feel from four possible choices. Each option is assigned a number. Based on the total of the score after adding all the points from each question, it is possible to assess if the person is depressed or not, and at what level. A score between 1 and 10 is considered normal; if the score falls between 11 and 16 a mild mood disturbance is indicated; scores of 17-20 indicate borderline clinical depression; scores of 21-30 show moderate depression; a range of 31-40 is a sign of severe depression, and scores over 40 indicate extreme depression (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

The ASRS is a self-assessment survey for ADHD that consists of 18 questions. People must answer how often certain instances occur. The options are: never, rarely, sometimes, often, very often. Based on their answer for each question it is possible to assess if the person has ADHD or not. Considering that ADHD may be stronger in some individuals than others, we assigned to each possible answer a score, starting from 0 for never up to 4 for very often. The total score for the survey was then calculated to assess the level of attention disturbance.

Electrophysiological data recording.

Data was collected using a 32-channel EEG cap. The EEG was amplified with two Guger Technologies g.USBamp amplifiers. The recordings were grounded to the left mastoid and referenced to the right mastoid at 256 Hz and filtered using a 0.05-30 Hz bandpass filter. The impedance was kept under 20 ohms.

Flanker task.

Participants were required to perform a Flanker task on a computer. The task was designed to enhance the chances of the participant's committing, thereby eliciting an error resulting in an ERN. The Flanker task used for the experiment was designed by the researchers through the program E-Prime 2.0. The task consisted of four cycles, each made of 96 trials. Participants were allowed a short break after each cycle.

Participants were seated 1m away from the screen, the stimulus size was 0.5 degrees of visual angle, and stimuli were presented for 900ms. A blank slide was presented between each stimulus for 1000 ms. Stimuli consisted of five arrows (Fig 1). The participants were presented four types of stimuli: two congruent (> > > > > & < < < < <) and two incongruent (> > < > > & < < > < <). The participants' task was to indicate the direction in which the middle arrow pointed;

if the middle arrow pointed left, they were to press the letter ‘a’ on the keyboard, if it pointed right they were to press the letter ‘l’.

A feedback slide indicating the correctness of the response appeared after each trial. If participants did not answer within a 900ms period, the program prompted them to respond faster.

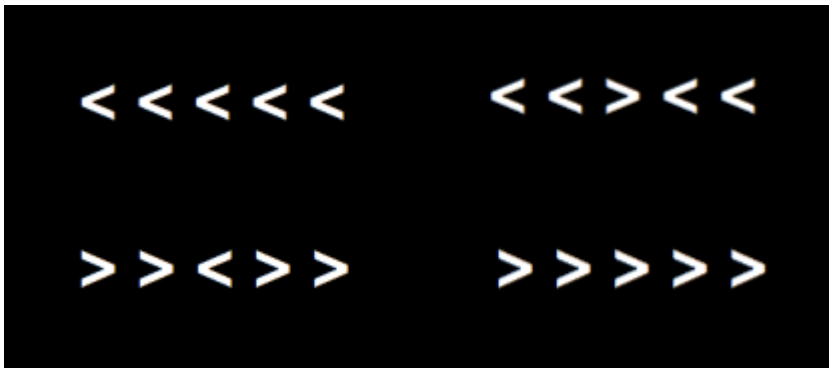

Figure 1. Congruent vs Incongruent slides in the Flanker Task.

Procedure

At the beginning of the study, participants read and signed an informed consent form approved by the ETSU Institutional Review Board and also reported demographic information. Subsequently, they completed the three surveys mentioned above to assess their level of anxiety, depression, and ADHD. Participants were not divided into groups. The scores from the assessments were used as continuous variables.

A 32-channel EEG cap was placed on the participants’ head to record brain activity while they performed the task and the electrodes were filled with gel to improve the electrical signal. All participants then performed the Flanker task. At the beginning of the task participants were prompted to respond as fast and as accurately as possible. At the completion of the experiment, the cap was removed and they were given an opportunity to wash their hair. Participants received a copy of the informed consent document before they left the lab.

Data Analysis

The EEG data was first refined using Independent Component Analysis (ICA) and ICLabel, to remove ECG artifacts such as muscle movement, eye movement, and line noise. Because the ERN is elicited in the ACC, the midline electrodes Fz, Fcz and Cz were used for the analysis. Defined as the most negative peak in the range of 0 – 150 ms, ERN amplitude was averaged for the correct and the incorrect trials.

ERN amplitude data for incorrect trials of each participant was averaged and input in a multiple linear regression model, using the BAI, BDI and ASRS v.1 surveys as covariates. The data analysis was conducted through the software RStudio (RStudio Team, 2015).

Results

Event-Related Potentials

A grand average waveform for all participants was plotted for both correct and incorrect responses for the three midline electrodes Fz, Fcz, and Fz (see Fig. 2). Figure 2 displays a large difference in ERP amplitudes between 50 and 100 ms following the response. The ERN response appears to be most prominent at electrode Cz. Descriptive statistics for the values of ERN for both incorrect and correct responses are shown in Table 1. A paired *t*-test was used to determine whether the mean difference between the ERN in incorrect trials and the ERN in correct trials was statistically significant**.** In concordance with previous literature, results indicate that the amplitude of the ERN for incorrect trials ($M = 2.86$, sd = 2.37) was significantly higher compared to the amplitude of the ERN for correct trials ($M = 0.76$, sd = 1.42), a statistically significant mean increase of 2.10 $t(30) = 5.65$, $p < .001$.

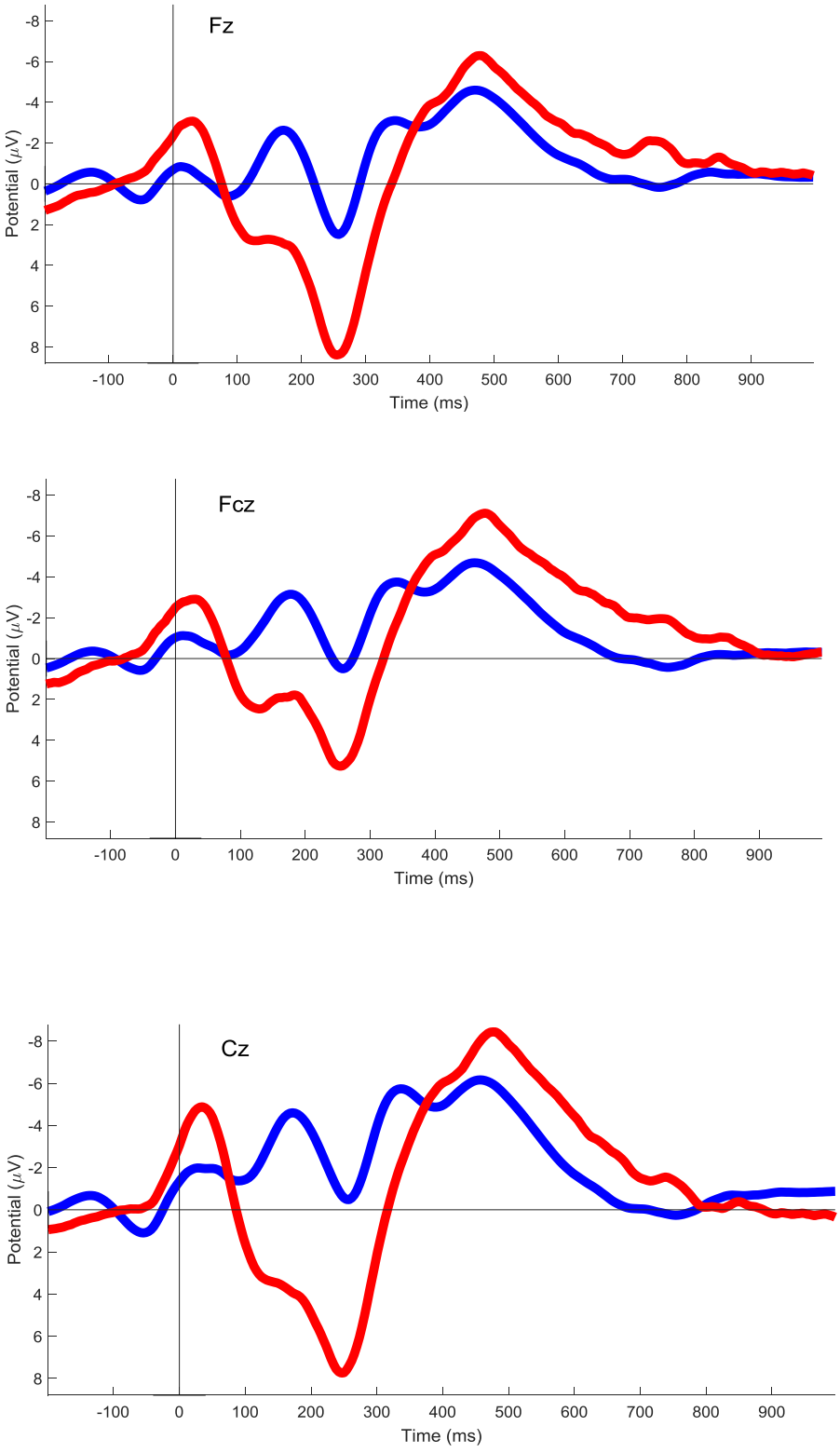

Figure 2. Grand average ERPs at electrodes Fcz, Fz, and Cz following correct and incorrect responses in the Flanker task.

Table 1 Descriptive Table

Statistic	N	Mean	St. Dev.	Min	Max
Incorrect ERN	31	2.86	2.37	-0.15	11.01
Correct ERN	31	0.76	1.46	-1.15	5.27
ADHD	30	26.97	13.98	10.00	63.00
Anxiety	31	15.91	13.91	0	45
Depression	30	12.30	11.09	0.00	40.00
Errors	31	19.97	14.18	5	67

Linear Regression Model

A multiple regression analysis was conducted to test if anxiety, depression, and ADHD significantly predicted participants' ERN amplitude (see Table 2). The regression was calculated for the instance of no interaction among variables, displayed in column 1 of Table 2, and with the existence of an interaction among variables, reported in column 2 of Table 2. The results of the regression in the case of no interaction among variables indicated a non-significant effect of the disorders on the ERN, $R^2 = 0.037$, $F(3, 25) = 0.32$, $p = 0.81$. The results of the regression in the case of interaction among variables also indicated a non-significant effect of the disorders on the ERN, producing an $R^2 = 0.277$, $F(7, 21) = 1.15$, $p = 0.37$.

Considering the multiple overlapping symptoms between anxiety, depression, and ADHD, we regard the second model considering interaction among variables to be the best fitting model for our data.

Table 2 Linear Regression

	Dependent variable:	
	Incorrect ERN	
	(1)	(2)
ADHD	0.005	0.135
	(0.044)	(0.139)
Anxiety	-0.062	0.200
	(0.064)	(0.206)
Depression	0.070	0.275
	(0.087)	(0.275)
ADHD:Anxiety		-0.008
		(0.009)
ADHD:Depression		-0.003
		(0.009)
Anxiety:Depression		-0.011
		(0.010)
ADHD:Anxiety:Depression		0.0002
		(0.0003)
Constant	2.888***	-0.955
	(1.034)	(2.663)
Observations	29	29
R^2	0.037	0.277
Adjusted R^2	-0.078	0.036
Residual Std. Error	2.535 (df = 25)	2.397 (df = 21)
F Statistic	0.322 (df = 3; 25)	1.150 (df = 7; 21)
Note:		*p<0.1; **p<0.05; ***p<0.01

Discussion

The present results support the hypothesis of an elicited ERN following incorrect responses. The ERP amplitude recorded between 50 and 100 ms following an incorrect response was significantly greater than the one for correct responses. These results were expected, since there is a large amount of evidence in the literature to support the existence of the ERN.

Surprisingly, we were unable to detect the effects that anxiety, depression, and ADHD have on the ERN, which would have been consistent with previous studies. Our results indicated a weak correlation between ERN and the disorders both when we considered no interaction among the three covariates and the existence of an interaction. These null findings were unexpected, due to the multiple published studies that found anxiety and depression to increase ERN amplitude and ADHD to decrease it (Chiu & Deldin, 2007; Hajcak, McDonald & Simons, 2003; Meyer, Weinberg, Klein, and Hajcak, 2012; Olvet, D. M., & Hajcak, G., 2008; Shiels & Hawk, 2011; Van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2006).

Limitations

Several limitations may have led to non-significant results, including small sample size, a narrow range in self-assessment survey scores and a low number of clinically diagnosed individuals. The strength of the effect may also be smaller than we expected and only manifest for sever cases or solely for specific subtypes of the disorders

Sample size.

One limitation in our study was the size of our sample. Although we initially were able to recruit 43 participants, we had to discard data from 12 of them, and our final sample size consisted of only 31 participants. We initially regarded 30 to be a valid recruitment target by comparing this number to the sample size from previous studies on the ERN, most of which

included between 20 and 50 participants. Meyer et al. (2012) recruited overall 54 participants to investigate the correlation between anxiety and ERN amplitude; Hajcak, McDonald & Simons (2003) collected data from 3 groups - phobic, worry and control – assigning 20 to 24 people to each group. Studies on depression and ERN had sample sizes that ranged between 35 and 42 participants (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; Luu et al., 2000). Finally, studies on the effects of ADHD on the ERN included between 20 and 68 participants, dividing them into ADHD groups and control groups (Van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2006; Jonkman, Melis, Kemner, & Markus, 2007; Hermann et al., 2009).

Considering that these studies only investigated an individual variable affecting the ERN, while we considered three covariates, we may need to triple the size of our sample to detect any significant effects on the ERN. A Type 2 error may therefore have led to false negative results.

To verify whether sample size may have been responsible for the null findings, we computed power using the software G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). Two power analyses were conducted, one for each linear regression. The first analysis showed the power of the study if there was no interaction among anxiety, depression, and ADHD (see Figure 3). The power of the study in this case would have been 11.8%. The second analysis was based on the results from the linear model that considered the existence of an interaction among the three disorders (see Figure 4). In this case, the power of the study was 55.7%.

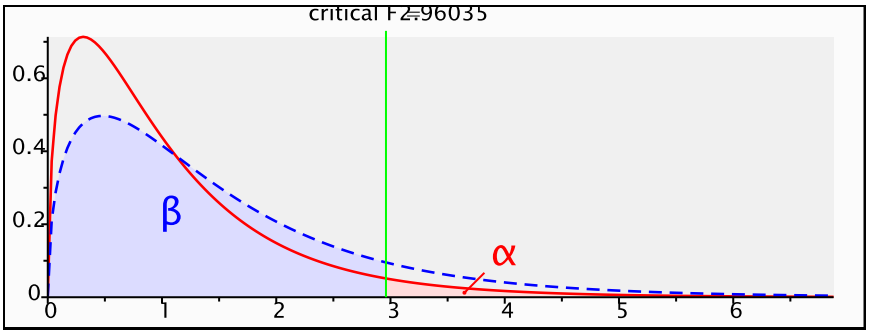

Figure 3. Power plot in the case of no interaction among variable.

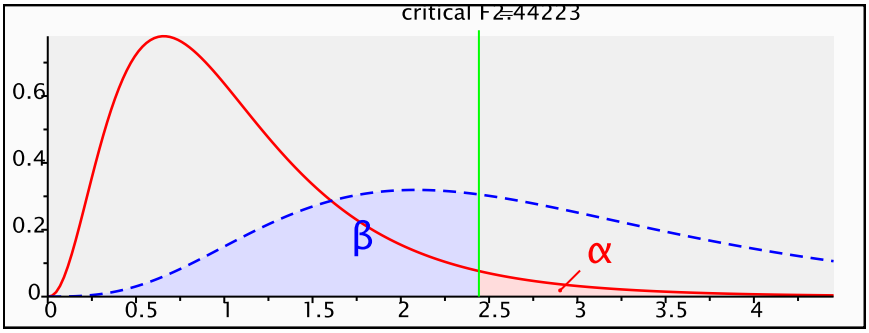

Figure 4. Power plot in the case of interaction among variable.

A third power analysis was conducted to determine the sample size necessary to achieve power of 80%. By inputting the effect size linear regression, we found that a sample size of forty-five participants would have been necessary to achieve a minimum of 80% power.

Severity of the disorders and subtypes.

In addition to the small sample size, another limitation that may have affected the power of our study and possibly led to false negative results is the effect size of the investigated phenomenon. We assumed that the intensity of the influence of the disorders on the ERN would be correlated to the level of severity of the disorders. We did not consider the possibility of the need for a minimum threshold for any effects to be detectable. It may be the case that for anxiety,

depression, and ADHD to alter ERN amplitude, people will need to suffer from medium to severe symptoms. Unfortunately, the data we collected from the self-assessment scores revealed that our sample did not display a wide range of magnitude of symptoms for the three disorders. Most of the participants scored low in all three surveys and suffered from mild to medium symptoms. We had very few cases in which participants scored high (See Table 1). Additionally, the number of participants that were clinically diagnosed was also very low. Out of the 31 participants, three had been clinically diagnosed with ADHD, seven with depression, and eight with anxiety.

Finally, we should consider that effects may occur only with specific subtypes of the disorders. The surveys utilized in this study only screen for general symptoms of anxiety, depression, and ADHD and do not provide specific diagnoses.

Future directions.

Given our findings, we suggest there is a need for further investigation of the variables that may influence ERN amplitude. Researchers should plan the design and statistical analyses of future studies accordingly to achieve a power of at least 80%. Replication studies and new research will be needed to better understand error processing. We also suggest that future research should investigate whether only severe cases of the disorders as opposed to mild cases may have a significant influence on ERN amplitude. Additionally, further studies should seek whether only certain subtypes of anxiety, depression, and ADHD alter the amplitude of the ERN and what are the specific traits in each subtype that lead to differences in error detection and error processing. Finally, in addition to replication studies, we suggest that a review of the published literature should be conducted to assess the statistical power obtained in previous work.

More rigorous studies on error processing are necessary for a better understanding of how the anterior cingulate cortex functions. By expanding the knowledge in error detection, processing, and compensation mechanisms, we will be able to understand how humans correct erroneous behavior and how they learn from their mistakes. Additional knowledge in how neurological disorders are involved in error processing is also important. Understanding the interactions between anxiety, depression, ADHD, and the ERN could aid in clinical diagnosis of the disorders, utilizing ERN amplitude as an additional diagnostic trait. Furthermore, monitoring of the ERN before, during and after treatments for these disorders may aid in checking for the effectiveness of the treatment by observing eventual improvements in error processing.

Conclusion

Our findings indicate the presence of an error-related negativity following incorrect responses but fail to support the hypothesis that a significant change in ERN amplitude occurs in relation to anxiety, depression, and ADHD. We suggest that further research is needed to investigate the effect size of this phenomenon, comparing mild to severe cases of anxiety, depression, and ADHD, and considering the different subtypes of each disorder. Previous literature may also need to be revised for validity of results and rigorousness of the study design.

Clarity on the subject will lead to a better understanding of error processing mechanisms and how different neurological disorders may lead to hyperactivity or deficiencies in error detection and compensation.

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