A dimensional examination of eating disorder symptoms in relation to cognitive processing: An event-related potentials study

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Identifying neurocognitive mechanisms involved in individuals experiencing eating disorder (ED) symptoms may be important for preventing EDs and improving rates of recovery. The present pilot study assessed how cognitive functioning may be associated with ED symptoms in college students (N = 41). Cognitive functioning was examined using electroencephalography during an auditory response inhibition task to measure the P3 component of event-related potentials. Multiple regression analysis revealed that longer P3 latencies in the frontal region of the cortex were significantly and linearly associated with greater ED symptoms $F(3, 37) = 13.62, p < .001, R^2 = 0.525$, Adj. $R^2 = 0.486$. These pilot findings build upon prior work in clinical samples in that they indicate that functional brain differences are observable across a wide span of ED symptoms, not just in those with diagnosed ED. The present findings provide support for further exploration of changes in P3 latencies among individuals with ED symptoms to enhance our understanding of neural mechanisms that may pertain to the dimensional aspects of disordered eating attitudes and behaviors.

1 | INTRODUCTION

Identifying neurocognitive mechanisms involved in individuals experiencing eating disorder (ED) symptoms may be important for preventing EDs and improving rates of recovery (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013). A recent systematic review of the literature that examined information processing and abnormal eating
highlighted a need for research of neurocognitive mechanisms pertaining to ED symptomatology (Wolz, Fagundo, Treasure, & Fernández-Aranda, 2015). The goal of this study is to address this gap by exploring the relationship between brain function and ED symptoms utilizing electroencephalography (EEG) and event-related potentials (ERPs).

The present work takes a dimensional approach to the study of ED symptoms with the goal of identifying mechanisms that may contribute to the risk of ED for some, as well as those that may promote resilience to ED in others. In recent years, there has been a shift in the conceptualization of mental health problems from a categorical to a dimensional approach, where symptoms are examined on a spectrum rather than a dichotomous basis (Brown & Barlow, 2005; Cuijpers, 2014; Cuthbert, 2014; Williamson, Gleaves, & Stewart, 2004; Zipf, Giel, Bulik, Hay, & Schmidt, 2015). An advantage of this approach is that it allows researchers to explore the full range of symptomatology that may impact changes in the brain and behavior leading to ED, rather than just those differences at extreme ends of the spectrum.

To date, the studies that sought to examine functional brain changes among eating disordered individuals did so by comparing differences in brain function between individuals diagnosed with an ED and matched healthy control participants (Bradley et al., 1997; Dodin & Nandrino, 2003; Nikendei et al., 2012; Otagaki, Tohoda, Osada, Horiguchi, & Yamawaki, 1998; Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010). Therefore, a gap remains in identifying neurocognitive mechanisms across the full spectrum of ED symptoms. This includes studying those with symptoms below the threshold for a diagnosable disorder, as a way to guide early identification and prevention of these disorders.

2 | EATING DISORDERS

Although there are distinct diagnostic criteria for anorexia nervosa, bulimia nervosa, and binge ED, it is common for individuals to move between these diagnostic categories over their lifetime (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Eddy et al., 2002; Fairburn, Cooper, & Shafran, 2003; Fairburn & Harrison, 2003). These commonalities suggest shared mechanisms pertaining to the range of ED symptoms experienced across these disorders (Fairburn & Harrison, 2003; Fairburn et al., 2003; Gorwood, 2004). Accordingly, some researchers have turned their attention to neurobiological factors that may be present in individuals with ED as another way of identifying commonalities and differences across disorders (Bradley et al., 1997; Dodin & Nandrino, 2003; Nikendei et al., 2012; Otagaki et al., 1998; Svaldi et al., 2010). The current work builds on past research of cognitive processing in severe eating disordered individuals by examining the relation of ED symptoms to cognitive processing among a sample displaying a range of ED symptoms (i.e., no ED symptoms, sub-clinical ED symptoms, and clinical ED symptoms).

3 | ELECTROENCEPHALOGRAPHY

Electroencephalography is the recording of cortical activity of the brain, produced by large populations of neurons firing together, which can be detected utilizing metal electrodes that are strategically placed on the scalp (Cohen, 2014; Jauregui-Lobera, 2011; Light et al., 2010). EEG has high temporal resolution, which makes it a useful proxy for the neuronal function of the cortex (Luck, 2014). EEG can be used to measure the brain’s response to a specific sensory, cognitive, or motor event through the measurement of ERPs. ERPs are time-locked to a stimulus occurrence and accordingly represent the electrophysiological response to that stimulus (Bokura, Yamaguchi, & Kobayashi, 2001; Duncan et al., 2009; Picton et al., 2000). In this study, we use ERPs to examine brain differences associated with the cognitive action of inhibition, which has been used in prior studies examining brain function among EDs (Dodin & Nandrino, 2003; Nikendei et al., 2012; Otagaki et al., 1998). Because mental processes occur in milliseconds, the excellent temporal resolution of ERPs allows researchers to study brain processes effectively by examining cognitive processes in the same timeframe in which they are occurring (Cohen, 2014; Dodin & Nandrino, 2003; Duncan et al., 2009; Jauregui-Lobera, 2011; Light et al., 2010).
An ERP can be characterized by analyzing its polarity (positive-P or negative-N), latency (in milliseconds), or amplitude (in microvolts) (Duncan et al., 2009). The latency of the ERP allows researchers to examine the length of the time required for the cognitive processes associated with that peak (Bradley et al., 1997; Luck, 2014). The amplitude of the ERP reveals the amount of neural resources and cortical activation dedicated to a specific cognitive process (Bradley et al., 1997; Duncan et al., 2009). Because ERPs are non-invasive, relatively inexpensive, and provide an array of neurocognitive information, they are widely used to study various brain processes (Jauregui-Lobera, 2011; Luck, 2014; Picton et al., 2000).

The largest component of the ERP waveform is the P3, which is also referred to as the P300 (Bradley et al., 1997). This large, positive wave often peaks between 250 and 500 ms after stimuli presentations (Luck, 2014; Polich, 2007). However, this positive peak can vary between individuals and can peak as late as 1,000 ms (Luck, 2014; Polich, 2007). P3 amplitude has been widely studied, revealing some limitations for the purposes of examining cognitive processing in individuals with ED symptoms, in that P3 amplitude is sensitive to differences in blood sugar. Accordingly, cognitive differences seen in assessments using P3 amplitude among individuals with ED symptoms may not accurately reflect variation in participant cognitive abilities, but instead may be a reflection of their blood sugar differences (Bokura et al., 2001; Polich, 1987). This is an important factor to consider given the expected variability in blood sugar among individuals with ED symptoms (i.e., high blood sugar due to a recent binge, low blood sugar due to starvation). Given this potential limitation, peak amplitude is often used in conjunction with other ERP components, specifically P3 latency. P3 latency is thought to reflect the timing involved in the individual’s decision to respond or withhold their response to a stimulus in cognitive tasks (Duncan et al., 2009; Otagaki et al., 1998; Shucard, McCabe, & Szymanski, 2008). It is therefore useful in detecting functional brain differences associated with cognitive processing (Duncan et al., 2009). Furthermore, latency has been found to be a more stable measure of the P3 component of the ERP than amplitude in that P3 latency is not altered by differences in blood sugar.

3.1 Examining eating disorders using electroencephalography

Research of brain function within EDs has generally reported consistent findings of longer P3 latencies in eating disordered individuals (Bradley et al., 1997; Dodin & Nandrino, 2003; Otagaki et al., 1998; Svaldi et al., 2010). Longer P3 latencies have been seen across ED diagnoses (i.e., anorexia, bulimia, and binge ED) when compared to healthy controls, during visual and auditory tasks. Further, longer P3 latencies have been seen in both normal weight and overweight eating disordered individuals (i.e., bulimia and binge ED), indicating that these differences are not solely a reflection of weight or nutritional status (Otagaki et al., 1998; Svaldi et al., 2010).

While consistent findings have been seen regarding P3 latency, findings for P3 amplitude have varied between studies. Some studies found no significant differences in P3 amplitude between eating disordered individuals (i.e., anorexia and bulimia) and healthy controls (Bradley et al., 1997; Otagaki et al., 1998). Others have reported increased amplitudes in ED participants as compared to controls (Dodin & Nandrino, 2003). Still, other research in this area has indicated reduced amplitudes among individuals with anorexia when compared to healthy controls (Nikendei et al., 2012). Taken together, these results point to the variable nature of P3 amplitude due to its sensitivity to external and internal factors (i.e., blood sugar, stimulus probability, task difficulty, stimulus content) that alter amplitude in a manner that is unintended by the experimental paradigm.

4 The present study

The objective of the current work is to utilize a dimensional approach to examine neurocognitive differences along a continuum of ED symptoms that have previously only been observed in individuals with ED diagnoses (Bradley et al., 1997; Dodin & Nandrino, 2003; Otagaki et al., 1998; Svaldi et al., 2010). It is hypothesized that participant P3 latencies will be linearly related to ED symptoms in the manner and direction reported in categorical studies of individuals.
with ED. Specifically, it is expected that observed P3 latencies measured during the response inhibition task will be longer as the number of ED symptoms increase. Finally, given the inconsistencies in studies of P3 amplitude, it is hypothesized that there will not be an association between P3 amplitude and ED symptoms.

5 MATERIALS AND METHODS

5.1 Participants

This study was reviewed and approved by the governing Institutional Review Board for a Southeastern university. The participants in this study included 41 college student volunteers who were recruited through the university subject pool over the course of two semesters. The gender, ethnicity, and age characteristics of this sample are presented in Table 1 and are in keeping with the demographics of the undergraduate students participating in the university subject pool at the university where this study took place.

5.2 Procedure

Participants completed the Institutional Review Board approved informed consent process. This pilot study consisted of two surveys, a demographics survey and the Eating Disorder Examination Questionnaire (EDE-Q), followed by an EEG. Participants were excluded from the EEG if they had a history of seizures or neurological problems. No participants withdrew from the study and there were no adverse events. Principal investigator K.N., licensed psychologist, was present to monitor safety. To ensure confidentiality, identifiers were removed, including participant names, date of birth, and date of assessment.

5.3 Eating Disorder Examination Questionnaire

To assess ED symptoms, participants completed the EDE-Q, a validated and widely-used 28-item self-report measure of eating attitudes and behaviors (Fairburn & Beglin, 1994). This measure uses a 7-point Likert response scale ranging from 0 (No Days) to 6 (Every Day) to ask participants about their thoughts and behaviors over the past 28 days. The EDE-Q is divided into four subscales including restraint, eating concern, shape concern, and weight concern. A global score on the EDE-Q is derived by calculating the average of the four subscales. Scores on this measure can range from 0 to 6; 0 if participants indicate no problematic eating attitudes and behaviors, up to 6 if they indicate daily problems for each of the questions on the measure. However, a score of 6 is relatively uncommon, even among clinical samples (Binford, Le Grange, & Jellar, 2004; Mountford, Haase, & Waller, 2006). In a study of the EDE-Q among a clinical sample of adolescents \( N = 70, M_{age} = 15.78 \) years, \( SD = 2.28 \) who met DSM-IV diagnostic criteria for bulimia nervosa, partial bulimia nervosa, or anorexia nervosa, the EDE-Q mean global score of the sample was 3.38 (Binford et al., 2004). In a similar study of individuals \( N = 84, M_{age} = 28.3 \) years, \( SD = 8.69 \) meeting DSM-IV diagnostic criteria for bulimia nervosa, anorexia nervosa, or ED not otherwise specified, the EDE-Q mean global score of the sample was 4.28 (Mountford et al., 2006).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Percentage</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (male/female)</td>
<td>14.6/85.4</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Ethnicity (White/non-White)</td>
<td>78/22</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Age (years)</td>
<td>18.54</td>
<td>2.36</td>
<td>1.39</td>
</tr>
</tbody>
</table>

EDE-Q, Eating Disorder Examination Questionnaire global score.
While the EDE-Q was examined continuously in this study, individuals who met or exceeded clinical cutoffs were still identified to characterize the sample. A score of greater than or equal to four has been established as the cutoff score to be considered as having clinically significant ED symptoms (Lavender, De Young, & Anderson, 2009; Luce, Crowther, & Pole, 2008; Mond, Hay, Rodgers, & Owen, 2006). A study of EDE-Q norms for young adult women \( (N = 5,255) \) found that the EDE-Q mean global score of their sample was 1.52, well below the clinical range (Mond et al., 2006). Studies of the EDE-Q, including a meta-analytic study, have displayed high internal consistency, with Cronbach’s \( \alpha \) ranging from .93 to .95, high discriminative validity, high convergent validity, and high test-retest reliability (Aardoom, Dingemans, Op’t Landt, & Van Furth, 2012; Berg, Peterson, Frazier, & Crow, 2011; Lavender et al., 2009).

5.4 | EEG and go/NoGo task

Participants completed a 6-min, standardized, auditory response inhibition task known as a Go/NoGo task while the EEG was being recorded (Falkenstein, Koshlykova, Kiroj, Hoormann, & Hohnsbein, 1995). A 64-channel Biosemi EEG system with ActiveTwo Pin-type electrodes was utilized to record EEG activity (BioSemi Inc., Amsterdam, The Netherlands). The auditory Go/NoGo task was presented binaurally using headphones. In this Go/NoGo task, participants heard two tones that were either the same frequency or different frequencies for a total of 50 pairs of stimuli presentations. The goal of this Go/NoGo task was for participants to inhibit their response when they heard two tones of the same frequency (low-low), and respond by pressing the spacebar when they heard tones of different frequencies (low-high). The timing for each stimulus presentation, in which participants heard the paired tones and responded, was 2,000 ms. After this time had elapsed, the computer gave the participants feedback indicating whether their response was correct. For example, if the paired tones were “low-high,” a spacebar press would constitute a correct response and no response would constitute an incorrect response. The inter-stimulus interval between paired tone presentations was 500 ms. All participants practiced the task and received corrective feedback before the task began.

Electroencephalography data were processed using the EEGLab and ERPLab plugins designed for MATLAB following the methods outlined by Lopez-Calderon and Luck on the ERPLab Toolbox website (Lopez-Calderon & Luck, 2014). The EEG data were segmented into epochs surrounding each auditory presentation, starting 200.00 ms before each set of paired tones and lasting until 800.00 ms after each auditory presentation. A series of uniform steps were completed on the continuous EEG datasets to remove bad channels, electrical noise, and non-brain wave artifacts (e.g., muscle activity, eye movement, and blinks) that contaminate the brain signal. The steps that were utilized to analyze the continuous EEG data were based on the ERPLab Toolbox tutorial, which states best practices for analyzing EEG data and creating ERPs (Lopez-Calderon & Luck, 2014; Luck, Lopez-Calderon, Huang, & Foo, 2012). Artifact-free ERPs were created using ERPLab to examine the P3 component of the ERP waveform for three electrode sites Fz (frontal), Cz (central), and Pz (parietal), that are commonly studied in the literature (Dodin & Nandrino, 2003; Duncan et al., 2009; Falkenstein, Hoormann, & Hohnsbein, 1999; Otagaki et al., 1998). The peak latency and amplitude variables within the P3 component of the ERP were each examined at the three midline electrode sites (i.e., Fz, Cz, and Pz).

5.5 | Analytic strategy

Two linear multiple regression analyses were utilized with SPSS version 22 to first evaluate the relationship between ED symptoms and P3 latency and second to examine ED symptoms and P3 amplitude. The first step in this analytic strategy was to determine the correlations between the EDE-Q global score and the measures of P3 latency and P3 amplitude for the Fz, Cz, and Pz electrode sites in the Go and NoGo conditions. The significant correlations were included in the regression models as independent factors. In the first multiple regression analysis, the dependent variable was the EDE-Q global score and the independent variables were the significantly correlated measures of
RESULTS

Analyses included the entire sample (N = 41). The EDE-Q global score (M = 2.36, SD = 1.39) was below the clinical cutoff, falling within the range of what has been reported in the literature for a college sample (Lavender et al., 2009; Luce et al., 2008). The span of scores among the sample indicated good variability (e.g., scores ranging from 0 to 4.88), capturing individuals with no ED symptoms to individuals with scores at or above the clinical cutoff (see Table 2). Of note, there were no scores on the extreme end of the measure (i.e., >5), which is to be expected given that the sample consisted of college students and scores in this extreme range would typically require more stringent intervention (e.g., inpatient care). Therefore, our sample does not include all levels of ED symptoms.

Visual inspection of the scatter plots for the relationship between ED symptoms and P3 latency showed that relations were linear, indicating that linear multiple regression was appropriate. For the correlations between ED symptoms and P3 latency, three of the six correlations were significantly positively correlated: Go Fz, Go Cz, and NoGo Fz (see Table 3). An examination of multicollinearity indicated that the data demonstrated an absence of multicollinearity with variance inflation factors values below two for all independent factors, indicating that the predictors were not highly correlated in a manner that would affect data interpretability. Additionally, the plots of standardized residuals against the predicted values indicated that the data displayed homoscedasticity. Finally, Q-Q plots of these P3 latency factors indicated that the residuals displayed a normal distribution with data following a straight line.

The linear combination of P3 latency factors was significantly related to EDE-Q global scores, F(3, 37) = 13.62; p < .001; R² = 0.525; Adj. R² = 0.486. Two of the three independent factors in the model were significant, Fz Go (p < .001), and Fz NoGo (p = .027); Cz Go did not significantly contribute to the model (p = .601). Visual inspection of the scatterplot between EDE-Q global scores and P3 latency at the Fz site in the Go condition (Figure 1), which was the strongest factor in the model, demonstrates that P3 latencies increase throughout the range of scores. The model remained significant after Bonferroni alpha correction of .017 with a p-value < .001 (Bland & Altman, 1995). The adjusted R-square indicates that approximately half of the total variability in EDE-Q global scores can be explained by the P3 latency data (see Table 4). Given the sample size of 41, the 3 independent factors, the Bonferroni alpha correction of .017, and the effect size of 1.11 derived using Cohen’s f² method of effect size calculation (Cohen, 1988), a post hoc power analysis conducted with G*Power (Erdfelder, Faul, & Buchner, 1996) determined that this study was sufficiently powered to detect an effect with a power of .999 (Cohen, 1992). Based on the power analysis, this study was sufficiently powered to detect differences in the frontal region of the cortex as a function of the EDE-Q scores (Range: 0–4.88, SD = 1.39).

<table>
<thead>
<tr>
<th>EDE-Q score range</th>
<th>Participants (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.0</td>
<td>7</td>
</tr>
<tr>
<td>1.0–1.99</td>
<td>12</td>
</tr>
<tr>
<td>2.0–2.99</td>
<td>9</td>
</tr>
<tr>
<td>3.0–3.99</td>
<td>6</td>
</tr>
<tr>
<td>4.0–4.99</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 2: Range of scores on the EDE-Q, N = 41

EDE-Q, Eating Disorder Examination Questionnaire global score.
Visual inspection of the scatter plots for the relationship between ED symptoms and P3 amplitude indicated that relations were linear. For the correlations between P3 amplitude in the Go and NoGo conditions for the Fz, Cz, and Pz sites, all correlations were negative and two of the six correlations were significant: Go Pz and Go Cz. These significant negative correlations indicate that greater ED symptoms were associated with reduced P3 amplitudes in the Go Pz and Go Cz conditions (see Table 3). The linear combination of P3 amplitude factors was significantly related to EDE-Q global scores, $F(2, 38) = 3.373; p = .033; R^2 = 0.164; Adj. R^2 = 0.120$. However, both of the independent factors in the model were not significant (see Table 4). Further, the model did not remain significant after employing FIGURE 1 Scatter plot of EDE-Q global scores and P3 Peak Latency at the Fz site for the Go condition.

<table>
<thead>
<tr>
<th></th>
<th>Go Fz</th>
<th>Go Cz</th>
<th>Go Pz</th>
<th>NoGo Fz</th>
<th>NoGo Cz</th>
<th>NoGo Pz</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q P3 latency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fz</td>
<td>$r = .717$</td>
<td>.717</td>
<td>.392</td>
<td>.326</td>
<td>.151</td>
<td>.278</td>
</tr>
<tr>
<td>Cz</td>
<td>.011*</td>
<td></td>
<td></td>
<td>.037*</td>
<td>.345</td>
<td>.078</td>
</tr>
<tr>
<td>Pz</td>
<td>.455</td>
<td>.455</td>
<td>.395</td>
<td>.305</td>
<td>.305</td>
<td>.305</td>
</tr>
<tr>
<td>EDE-Q P3 amplitude</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fz</td>
<td>−.298</td>
<td>−.298</td>
<td>−.395</td>
<td>−.288</td>
<td>−.288</td>
<td>−.271</td>
</tr>
<tr>
<td>Cz</td>
<td>−.313</td>
<td>−.313</td>
<td>−.313</td>
<td>−.305</td>
<td>−.305</td>
<td>−.305</td>
</tr>
<tr>
<td>Pz</td>
<td>.058</td>
<td>.058</td>
<td>.011*</td>
<td>.068</td>
<td>.053</td>
<td>.087</td>
</tr>
</tbody>
</table>

This table includes the correlations between the P3 latency and amplitude components and EDE-Q global scores. Go represents the condition where participants heard two different tones and were told to respond by pressing the spacebar. NoGo represents the condition where participants heard two of the same tones and were told not to respond. Fz represents the data collected from the EEG for the frontal location, Cz represents the central location, and Pz represents the parietal location.

*p < .05, **p < .01.
7 | DISCUSSION

The present pilot study examined the dimensional relationship between ED symptoms and brain function in a college sample. This study found that P3 latencies measured during a response inhibition task were longer in association with higher ED symptoms, which is supported by related research in clinical samples (Bradley et al., 1997; Dodin & Nandrino, 2003; Otagaki et al., 1998; Svaldi et al., 2010). In confirming the first study hypothesis, this study extends past work by revealing that these prolonged P3 latencies are observable in a linear manner across a range of ED symptoms from low symptoms to those exceeding the clinical cutoff. In keeping with the last study hypothesis, the present pilot findings did not find a significant association between P3 amplitude and ED symptoms (Bradley et al., 1997; Otagaki et al., 1998).

7.1 | P3 latency

The results of this study provide evidence that changes in P3 latencies are consistent and observable across the full spectrum of ED symptoms. P3 latencies were longer in individuals reporting more ED symptoms, indicating that as ED symptoms increased, cognitive processing of the tones and the decision to respond or withhold a response on the Go/NoGo auditory task also increased. These observable differences in neurocognitive processing across the range of eating disordered symptoms may indicate that functional brain changes are gradual and could play a role in the development of EDs (Nikendei et al., 2012).

P3 latency reflects stimulus evaluation time (Luck, 2014; Otagaki et al., 1998) and accordingly longer P3 latencies likely reflect a slowing of task-related information processing. Researchers have not determined the cause(s) of these longer P3 latencies in eating disordered individuals. However, there is converging evidence suggesting that longer P3 latencies are not due to low blood sugar or malnutrition in individuals with EDs (Geisler & Polich, 1992). In addition, research among non-eating disordered individuals has indicated that within the same group of participants, P3 latencies did not differ whether the participant had been fasting for 14 hr, or had recently consumed food (Geisler & Polich, 1992).

Prior studies have revealed that longer P3 latencies are present in low-weight, normal weight, and overweight eating disordered individuals, including individuals with anorexia, bulimia, and binge ED (Bradley et al., 1997; Dodin

| TABLE 4 Summary of the multiple linear analysis for the EDE-Q and correlated electrode sites |
|---------------------------------------------|-----------------|-------|-----|-------|-------|-------|-------|-------|
|                                      | B          | SE   | β   | t     | p     | R²   | Adj. R² | SE   | F     | p     |
| P3 latency                           | 0.525      | 0.486 | 0.998 | 13.617 | .000** |
| (Constant)                           | −2.749     | 0.962 | −2.859 | .007** |
| Fz NoGo latency                      | 0.004      | 0.002 | 0.262 | 2.297  | .027* |
| Fz Go latency                        | 0.010      | 0.002 | 0.699 | 4.656  | .000** |
| Cz Go latency                        | −0.001     | 0.002 | −0.079 | −0.527 | .601  |
| P3 amplitude                         | 0.164      | 0.120 | 1.306 | 3.736  | .033* |
| (Constant)                           | 3.482      | 0.474 | 7.351 | .000** |
| Pz Go amplitude                      | −0.042     | 0.069 | −0.115 | −0.612 | .544  |
| Cz Go amplitude                      | −0.106     | 0.061 | −0.325 | −1.736 | .091  |

This table contains the multiple regression models as well as the unstandardized and standardized regression coefficients for the independent predictors for both P3 latency and P3 amplitude.

*p < .05, **p < .01.

a Bonferroni corrected alpha of .025 (Bland & Altman, 1995). Given that neither of the P3 amplitude factors were significant, a power analysis was not employed.
indicating that changes in P3 latencies are not due to weight, but instead are related to other behavioral factors, such as ED symptoms. These findings are further supported by research on weight restored individuals with EDs, who continue to show non-verbal information processing deficits, as measured with ERPs (Bradley et al., 1997). Taken together, these findings suggest that the longer P3 latencies displayed by individuals with more ED symptoms in this study are not solely due to weight, nutritional, or blood sugar deficits. Therefore, these observed differences may represent a brain-based mechanism that is present across a spectrum of eating attitudes and behaviors.

Researching examining cognitive processing in P3 latency has indicated longer processing times in individuals with ED symptoms, utilizing a variety of different stimuli, including both disorder-relevant and disorder-irrelevant stimuli. These longer P3 latencies have been seen among eating disordered individuals during verbal tasks (i.e., disorder-irrelevant words, pronounceable nonwords, and three-digit numbers), through both disorder-relevant and disorder-irrelevant visual tasks (i.e., geometric shapes, simple and complex images of female bodies, and images of high caloric food), and auditory tasks (i.e., two-tone Oddball task and in this study a two-tone auditory Go/NoGo task) (Bradley et al., 1997; Dodin & Nandrino, 2003; Otagaki et al., 1998; Svaldi et al., 2010). These complementary findings of longer P3 latencies regardless of the stimuli being disorder-relevant (i.e., food or body images) or disorder-irrelevant (i.e., sounds, shapes, and words), suggest a deficit in cognitive processing among individuals with ED symptoms that is not stimuli specific.

7.2 | P3 amplitude

The present finding of no significant association between P3 amplitude and ED symptoms is in agreement with several studies in this area that have indicated no significant differences in P3 amplitude between eating disordered individuals when compared to healthy controls (Bradley et al., 1997; Otagaki et al., 1998). This may be due to the sensitivity of P3 amplitude to stimulus probability and task difficulty (Bokura et al., 2001; Polich, 1987). This means that differences in the structure of a cognitive task can result in differences in P3 amplitude findings which are a function of the task and not necessarily an indication of true cognitive differences. This was the case in the Dodin and Nandrino (2003) study in which the researchers used a 30- (target) to 120- (non-target) stimulus presentation ratio, and in turn reported corresponding greater P3 amplitudes. However, this study had an equal ratio of Go (25 pairs of tones) and NoGo (25 pairs of tones) conditions, which may have contributed to the null result. Taken together, future studies of EEG and ED symptoms that evaluate P3 amplitude, should systematically vary the ratio of Go and NoGo trials to determine the impact of this ratio on P3 amplitude results.

Furthermore, unlike P3 latency which is generally stable, P3 amplitude is influenced by blood sugar and weight (Geisler & Polich, 1992; Jones et al., 1990). P3 amplitude is reduced even in mild hypoglycemia (Bradley et al., 1997; Geisler & Polich, 1992; Jones et al., 1990) and has been reported to increase after food intake (Geisler & Polich, 1992). Accordingly, it has been suggested that individuals eat within a few hours prior to their EEG session (Geisler & Polich, 1992). This same research has shown that P3 latency findings are more robust and less influenced by weight and blood sugar (Geisler & Polich, 1992). Finally, P3 amplitude is also related to stimulus content (e.g., disorder-relevant vs. disorder-irrelevant stimuli). Multiple studies have indicated increased P3 amplitude in eating disordered individuals when presented with food-related stimuli (Nijs, Franken, & Muris, 2008, 2009; Svaldi et al., 2010). Given that this study utilized nondisorder-relevant auditory tones, these stimuli may not have impacted P3 amplitude in the same way that disorder-relevant food stimuli may have. It will be important for future research to further examine differences in P3 amplitude while controlling for blood sugar and utilizing disorder-relevant stimuli presentations.

7.3 | Eating disorders and frontal deficits

The present pilot findings of prolonged P3 latencies associated with greater ED symptoms were significant for the frontal region of the cortex. These results are not the first to identify a connection between ED symptoms and the
frontal region of the brain. However, they are among the first to demonstrate this in a dimensional manner (Uher et al., 2003, 2004). Changes in the right prefrontal cortex and medial prefrontal cortex have been reported to differentiate individuals with active EDs from recovered and control individuals (Uher et al., 2003, 2004). Additionally, further research has indicated that right hemispheric prefrontal and frontal lesions are consistently associated with EDs (Castaño & Capdevila, 2010; Trummer, Eustacchio, Unger, Tillich, & Flaschka, 2002; Uher et al., 2003; Uher, 2005; Ward, Tiller, Treasure, & Russell, 2000). Multiple case studies have also reported the development of EDs after the incidence of brain lesions in the right prefrontal cortex and frontal lobe (Castaño & Capdevila, 2010; Trummer et al., 2002; Ward et al., 2000). The brain changes in the prefrontal cortex, observed in this study and found in these related studies, could constitute a “trait vulnerability” or a shared mechanism in the development of ED that may be a useful target for future investigation (Castaño & Capdevila, 2010; Otagaki et al., 1998; Trummer et al., 2002; Uher et al., 2003, 2004; Ward et al., 2000). These pilot findings that slower cognitive processing in the frontal region is associated with higher ED symptoms warrant further exploration. Specifically, individuals with ED symptoms and behaviors may be less able to allocate adequate resources to attention-related cognitive processes. The present findings provide preliminary evidence that cognitive processing may be impaired in individuals with ED symptoms, particularly related to sustained attention, which could be a mechanism to study in future clinical research.

8 | LIMITATIONS

This pilot study of brain function and ED symptoms in college students has several limitations. This was a cross-sectional study of brain function and ED symptoms. Therefore, one cannot determine the cause of the neurocognitive differences seen in participants or whether these cognitive differences predispose individuals to ED symptoms. Additionally, due to the correlational nature of this study, it is not possible to rule out unstudied variables that may be contributing to this relationship (e.g., age, gender, psychopathology, weight). While the present sample size was sufficiently powered, equal in size to similar studies, and representative of the gender and racial background of students in this region; it was a relatively small, homogeneous college sample, which limits generalizability. However, given that the symptoms assessed are commonly seen in college students, it is possible that the results would relate to other college students or emerging adults, specifically White females. This sample consisted of more females than males, which is common in ED research because EDs are more prevalent in females (Bradley et al., 1997; Dodin & Nandrino, 2003; Nikendei et al., 2012; Otagaki et al., 1998; Svaldi et al., 2010). Finally, the procedures for this study did not include a diagnostic interview to confirm ED diagnoses. Although many individuals scored in the range characteristic of an ED, diagnosis for an ED cannot be confirmed in this study, which is why the term ED symptoms was used. Accordingly, future longitudinal research with a larger, more diverse, and random sample with the inclusion of diagnostic interviews is warranted.

9 | CONCLUSIONS

The present pilot study included a range of individuals from those displaying no ED symptoms to those with ED symptoms in the clinical range. Rather than having an ED group and an asymptomatic control group, as past studies have done (Bradley et al., 1997; Dodin & Nandrino, 2003; Nikendei et al., 2012; Otagaki et al., 1998; Svaldi et al., 2010), this study included individuals at many levels of ED symptomatology with the goal of strengthening the ecological validity of the present findings. These pilot findings of measurable increases in the P3 latency component of ERPs in association with increasing ED symptoms suggest a shared neural mechanism that may exist across a range of symptoms. These findings could inform future research as well as our understanding of cognitive differences in individuals with ED symptoms.
Eating disorders are more effectively treated and have reduced mortality rates if they are identified early (Zipfel, Löwe, Reas, Deter, & Herzog, 2000). Gaining a more detailed understanding of functional brain changes among individuals displaying varying levels of ED symptoms has the potential to inform efforts to curtail the negative implications of EDs. Since neurocognitive differences were present when individuals displayed ED symptoms that were still considered below the clinical range, it may point to early differences that could be targeted in prevention efforts. Accordingly, these results support future research aimed at examining when changes in P3 latency correspond to key symptom changes which may represent a risk factor for the development of an ED. Alternatively, these findings could also be used to identify those whose neurocognitive profile may indicate resilience to ED symptoms. By identifying these resilient individuals, researchers may be able to find new ways to promote healthy attitudes and behaviors about body, weight, and shape.

10 | FUTURE DIRECTIONS

The present pilot study indicates that a dimensional approach to the study of neurocognitive function holds potential for detecting cognitive changes associated with ED symptoms. Future research could aim to identify the specific processes that drive neurocognitive changes across the spectrum of ED symptomatology, as well as examining if more nuanced changes could also be detected with other neuroimaging tools, such as functional magnetic resonance imaging. Further, future research could also examine how this dimensional approach may differ between different subtypes of ED diagnoses. One study examining the issue of dimensionality with EDs found that the symptoms of anorexia fit within a continuous view, while the symptoms of binge ED did not occur on a continuum (Williamson et al., 2004). Another future direction could be an examination of the apparent deficits in right hemispheric functioning associated with EDs. Studies have indicated that individuals with EDs have deficits in their right hemisphere due to over-arousal of their left hemisphere (Bradley et al., 1997; Nikendei et al., 2012). Research has also indicated that right hemispheric prefrontal and frontal lesions are consistently associated with EDs (Castañón & Capdevila, 2010; Trummer et al., 2002; Uher et al., 2003, 2004; Ward et al., 2000). Additionally, research has indicated that individuals with EDs display reduced nonverbal skills and enhanced verbal skills (Bradley et al., 1997; Maxwell, Tucker, & Townes, 1984). Nonverbal skills rely more on right hemispheric functioning, whereas verbal skills rely more on left hemispheric functioning (Bradley et al., 1997; Uher, 2005). Subsequent work should investigate how hemisphere deficits may be related to ED symptoms. A majority of EEG research focuses on the evaluation of the three central midline electrodes, Fz, Cz, and Pz, as that has become the normative practice due to their consistent findings. Accordingly, a final future direction could be to extend these findings would be to evaluate hemispheric differences by utilizing both verbal and nonverbal tasks in a sample of individuals displaying the full range of ED symptoms (e.g., no symptoms to inpatient clinical populations).

These preliminary findings of significantly longer P3 latencies associated with elevated ED symptoms provide support for future studies looking at shared neural mechanisms over time, in a variety of cognitive tasks, and with a range of neuroimaging methodologies, among each of the subtypes of EDs. This study provides support for forthcoming research in each of these domains to examine in more detail what implications functional brain changes may have for the enhanced understanding, prevention, early diagnosis, and treatment of EDs. With a greater understanding of these cognitive differences, we have the potential to understand whether they reflect a vulnerability to developing EDs or a characteristic change that occurs from the ED. With this knowledge, we may be able to target the specific cognitive mechanisms that contribute to EDs through early intervention. Many more studies will be needed to inform our currently ambiguous understanding of the cognitive mechanisms and changes involved in ED before fruitful early intervention avenues can be developed.

CONFLICT OF INTEREST

The authors Lauren M. Schaefer and Kate B. Nooner declared no potential conflicts of interest with respect to the research authorship, and/or publication of this article.
INFORMED CONSENT STATEMENT

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all patients for being included in the study.

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