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RESEARCH ARTICLE

Associations between disordered eating, internalizing symptoms, and behavioral and neural correlates of response inhibition in preadolescence

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Abstract

Response inhibition difficulties are reported in individuals with eating disorders (EDs), anxiety, and depression. Although ED symptoms and internalizing symptoms co-occur in preadolescence, there is limited research examining associations between these symptoms and response inhibition in this age group. This study is the first to investigate the associations between behavioral and neural markers of response inhibition, disordered eating (DE), and internalizing symptoms in a community sample of preadolescents. Forty-eight children (*M* age = 10.95 years, 56.3% male) completed a Go/NoGo task, whereas electroencephalography was recorded. Self-report measures of DE and internalizing symptoms were collected. Higher levels of anxiety and depression were associated with neural markers of suboptimal response inhibition (attenuated $P3_{NoGo}$ amplitudes) in preadolescence. In contrast, higher levels of depression were associated with greater response inhibition at a behavioral level. These findings suggest internalizing symptoms in preadolescence are associated with P3-indexed difficulties in evaluation and monitoring, but these are not sufficient to disrupt behavioral performance on a response inhibition task. This pattern may reflect engagement of compensatory processes to support task performance. DE was not significantly associated with response inhibition, suggesting that difficulties in response inhibition may only be reliably observed in more chronic and severe DE and ED presentations.

KEYWORDS
disordered eating, eating disorders, EEG, event-related potentials, internalizing symptoms, preadolescence, response inhibition

1 | INTRODUCTION

Eating disorders (EDs) are mental health conditions characterized by a complex combination of psychological and physical symptoms (e.g., binge eating, purging, dieting, low body weight, and preoccupying cognitions surrounding eating, shape, and weight; American Psychiatric Association [APA], 2013). EDs have the highest mortality rate of any psychiatric illness (Smink et al., 2012) and current treatment outcomes for EDs are modest. In adolescents with anorexia nervosa and bulimia nervosa, long-term remission rates are approximately

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35%–40% based on the use of leading evidence-based treatments (Le Grange et al., 2014, 2015). Importantly, early detection and intervention of EDs is necessary to improve prognosis, as responsiveness to treatment decreases over the duration of the illness (Ambwani et al., 2020).

Preadolescence (typically defined by researchers as age 8–11 years; Barry et al., 2022; Murray et al., 2022) may be a critical stage for studying the development of EDs. Although EDs are most likely to develop during adolescence (Micali et al., 2013; Stice et al., 2009), there is evidence to suggest these diagnoses are increasing in preadolescence (Nicholls et al., 2011; Petkova et al., 2019; Reas & Rø, 2018). Prodromal ED symptoms also start to emerge during preadolescence, indicating the first phase of the disorder onset (Herle et al., 2020; Kotler et al., 2001; Schaumberg et al., 2019). These symptoms, referred to as disordered eating (DE), can include behaviors and cognitions, such as binge eating, dietary restriction, body dissatisfaction, and excessive exercise. DE is found to be present in the general population, although in a less severe and infrequent form than DE observed in clinical EDs (Naor-Ziv & Glicksohn, 2016). However, very little is known about early factors that may be associated with DE and confer risk for later development of EDs. Early identification of cognitive and neural factors that underpin the development of EDs is important for developing pre-onset interventions (Culbert et al., 2015; Wildes & Marcus, 2013). Beyond this, studying the neural and cognitive associations with DE in preadolescence is important as research in adolescence and adulthood can be constrained by illness-related sequelae, such as malnutrition. Therefore, research focused on prodromal stages of the ED to identify correlates to prevent severe and chronic diagnosable EDs is needed.

Internalizing symptoms, such as anxiety and depression, have been implicated in the development of DE due to their high co-occurrence with EDs and DE across the lifespan (Evans et al., 2017; Hudson et al., 2007; Thomas et al., 2021; Touchette et al., 2011; Ulfvebrand et al., 2015). Internalizing symptoms are also found to play a significant mediating role between executive functioning difficulties and ED behaviors (Giel et al., 2012; Weider et al., 2015). Related to this, research suggests there may be common underlying neurocognitive mechanisms between DE and internalizing symptoms (Donofry et al., 2016). Response inhibition, defined as the ability to withhold a prepotent incorrect response in order to perform a correct response and maintain goal performance (Davidson et al., 2006), is an executive function that has been widely studied in relation to mental health conditions across development (Hwang et al., 2016; Mar et al., 2022; Thomas et al., 2022; Wright et al., 2014). In EDs, behavioral measures show decreased inhibitory control in adults with binge eating and/or purging symptoms (Galimberti et al., 2012; Hege et al., 2015; Steinglass et al., 2019; Svaldi et al., 2014; Wu et al., 2013). In contrast, adolescents with binge eating and/or purging DE (Bartholdy et al., 2019) and restrictive-type anorexia nervosa (Weinbach et al., 2020) do not display behavioral difficulties in response inhibition when compared to healthy controls. A similar pattern of findings has been reported in individuals with internalizing symptoms, such as anxiety and depression: Adults display response inhibition difficulties (Grillon et al., 2017; Li et al., 2021), whereas adolescents typically show no differences in behavioral markers of response inhibition performance when compared to healthy controls (Brunnekreef et al., 2007; Diler et al., 2014; Hum et al., 2013a; Pan et al., 2011; Singh et al., 2010). These data, therefore, suggest that response inhibition difficulties in adolescents with DE and internalizing symptoms are either not reliably detectable at the behavioral level or not yet present.

The anterior cingulate cortex (ACC) is known to mediate functions involved in response inhibition (Banfield et al., 2004; Gehring & Knight, 2000) and is a key area of the brain implicated in the etiology of EDs (Frank et al., 2004; Joos et al., 2010; Mühlau et al., 2007; Uher et al., 2003) as well as internalizing disorders (Blair et al., 2012; Lichenstein et al., 2016; Shang et al., 2014). The N2, a marker of conflict or inconsistency between competing responses (Albert et al., 2013), and the P3, an index of later evaluation processes (Smith et al., 2008), are stimulus-locked event-related potentials (ERPs) generated by the ACC (Bekker et al., 2005; Zhang et al., 2012). In contrast to behavioral markers, neural markers indicate suboptimal response inhibition performance across internalizing disorders in both children and adults. For example, greater N2 amplitudes are observed in anxiety (Hoyniak & Petersen, 2019; Valadez et al., 2021; Yu et al., 2018), whereas attenuated P3 amplitudes have been reported in depression (Houston et al., 2003; Nan et al., 2018; Santopetro et al., 2022, 2020). In addition, both enhanced and attenuated P3 amplitudes are found in anxiety (Bechor et al., 2019; Éismont et al., 2009; Sehlmeyer et al., 2010; Wauthia et al., 2022; Xia et al., 2020; Xu et al., 2014). Although ERP studies examining the association between DE and N2/P3 components are limited, findings from children with obesity (Reyes et al., 2015; Tascilar et al., 2011; Walk et al., 2020) and adults with anorexia nervosa (Yue et al., 2020) demonstrate consistent attenuation of the P3; however, investigations of the N2 report no effects. Taken together, the contrasting behavioral and neural findings suggest it is possible that neural differences are observable before behavioral differences have emerged (Bartholdy et al., 2019; Hum et al., 2013a), highlighting the importance of examining both neural and behavioral component of response inhibition.

The current study aimed to examine the associations between both behavioral and neural correlates of response inhibition with DE and internalizing symptoms in preadolescence. Research to-date has focused on examining the associations between response inhibition and DE or response inhibition and internalizing symptoms separately, in clinical adult and adolescent samples. By studying preclinical symptoms using a transdiagnostic approach (Astle et al., 2022), we are better able to identify risk factors that emerge before the development of a diagnosable ED and possible confounding illness-related sequelae. Furthermore, we aimed to investigate the specificity of these associations by examining whether DE and internalizing symptoms make independent contributions to variations in behavioral and neural markers of response inhibition. To do this, a common measure of response inhibition, the Go/NoGo task, will be used alongside electroencephalography (EEG) recording, to study both behavioral and neural indicators of response inhibition. We hypothesized a dissociation between these behavioral and neural measures of response

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inhibition. That is, increased levels of DE and internalizing symptoms will be positively correlated with neural markers of suboptimal response inhibition performance but not with behavioral markers of response inhibition difficulties. Specifically, increased levels of DE and internalizing symptoms will be associated with less positive P3 amplitudes on NoGo trials, whereas higher levels of anxiety symptoms will be associated with more negative N2 amplitudes on NoGo trials. In our secondary analysis, we will explore the independence of these associations by controlling for behavioral task performance in the association between neural markers and DE/internalizing symptoms.

2 | METHODS

2.1 | Participants

Sixty-three participants (*M* age = 11.0 years; 46.0% female) were recruited for this study across two stages. During the first stage, 26 children (*M* age = 10.9 years; 53.8% female) were recruited after participating in a school-based study (details provided in Thomas et al., 2021). These children were invited to participate in the current study at the University between August 2019 and March 2020. Typically, there was a delay of 2–3 months between participants participating in the two studies. Stage two of recruitment invited 37 children (*M* age = 11.0 years; 40.5% female) to participate in the study through social media advertisements and invitations through a recruitment database. This took place between March 2021 and September 2021, due to disruptions to testing during the COVID-19 pandemic. Invitation emails and social media advertisements to families across both recruitment stages described the research as an investigation of children's brain activity and how it related to their eating behaviors, thoughts, and feelings. A *t*-test revealed the two recruitment groups did not differ significantly in child age (t (61) = 1.035, p = .305) or parent age (t (60) = −1.753, p = .085). There were no significant differences in the number of boys and girls recruited at each recruitment stage (X^2 (1, N = 63) = 1.088, p = .297), reported socioeconomic status (U = 395.5, z = −1.254, p = .210), or ethnicity (X^2 (3, N = 60) = 6.474, p = .091). Therefore, we merged the two groups.

Parents confirmed their child did not meet any of the following exclusion criteria: premature birth, significant developmental delays, uncorrected visual difficulties, or significant head trauma leading to neurological abnormalities. In relation to task exclusionary criteria, 15 children were excluded based on low task accuracy (go accuracy <50%). And one participant was excluded from all ERP analyses due to excessive EEG artifacts. Table 1 presents the demographics of this final sample of 48 children.

The project received approval from the Cardiff University School of Psychology Ethics Committee (EC.19.02.12.5566GR5A6). Written informed consent from the parent/guardian and child assent was obtained before the experiment began. As compensation for their time, each child received a gift voucher and a small gift. Socioeconomic status was determined via postcode matching to the Welsh Index of Multiple Deprivation.

TABLE 1 Demographics of the final sample.

Child demographics (N = 48)	*M* (minimum–maximum)
Age (years)	10.95 (10.00–11.92)
Gender (male %)	56.3
Ethnicity (%)	
White	85.4
Mixed or multiple ethnic groups	4.2
Asian or Asian British	4.2
Other ethnic group	4.2
Black, African, Caribbean, or Black British	0
Missing data	2.0
Parent demographics (N = 48)	***M* (minimum–maximum)**
Age (years)	42.52 (29.90–61.20)
SES (WIMD) quartile[1] (%)	
1st (most deprived)	18.8
2nd	20.8
3rd	18.8
4th (least deprived)	41.7

Note: [1]Postcodes are allocated to one of 1909 lower layer super output areas (LSOAs) in Wales. Each LSOA has a population of around 1600 people. The LSOAs are ranked from 1 to 1909, with 1 being the most deprived and 1909 being the least deprived. LSOAs ranked 1–478 fall within quartile 1, 479–955 in quartile 2, 956–1432 in quartile 3, and 1433–1909 in quartile 4.

Abbreviations: SES, socioeconomic status; WIMD, welsh index of multiple deprivation.

2.2 | MATERIALS

2.2.1 | Self-report questionnaire measures

Children's eating attitude test

DE behaviors and attitudes were measured using the children's eating attitude test (ChEAT; Maloney et al., 1989), a self-report modified version of the abbreviated adult eating attitudes test (26; Garner & Garfinkel, 1979). Using the traditional scoring strategy, the three most symptomatic responses ("often," "very often," and "always") are scored from one to three, and the remaining three responses scored as zero. This limits the variability of the data (Anton et al., 2006; Smolak & Levine, 1994). Therefore, the current study employed an alternative scoring strategy that has been previously used in a large community sample of children aged 7–12 years and resulted in greater variability in item scores and a reduction in skewness for the total ChEAT score (Anton et al., 2006). We also found greater variance and a reduction in skewness in our sample (ChEAT total original scoring: skew = 1.65, s^2 = 46.63; ChEAT total alternative scoring: skew = 0.79, s^2 = 147.62). In the alternative scoring procedure, a Likert scale from 1 (never) to 6 (very often) was used with higher scores representing more difficulties.

Minor adjustments to the wording of items were made to enhance comprehension. Item 4 was changed from "I have gone on eating binges

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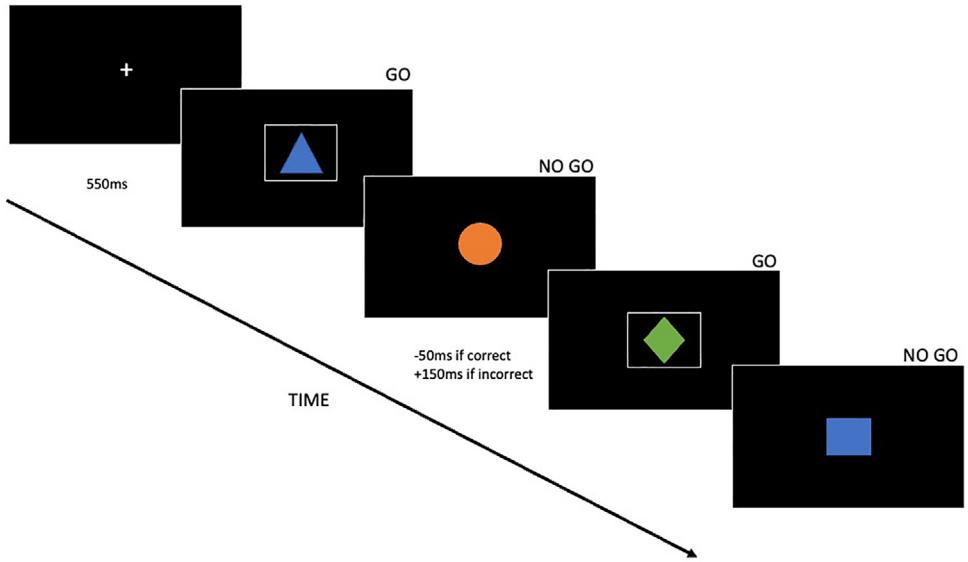

FIGURE 1 A visual representation of the Go/No-Go task used in the study.

where I feel that I might not be able to stop" to "I have started to eat and then felt like I cannot stop" (see Coombs et al., 2011). Items 9 and 26, which refer to "vomit," were also accompanied by "am/be sick." Finally, item 21 was changed from "I give too much time and thought to food" to "I spend too much time thinking about food." Cronbach's alpha value for the adjusted items with the alternative scoring strategy was acceptable ($\alpha = .71$).

Revised child anxiety and depression scale—25 item version

The revised child anxiety depression scale—25 item version (RCADS-25; Muris et al., 2002) is a brief assessment of anxiety and depression symptoms as defined by the Diagnostic and Statistical Manual of Mental Disorders (APA, 2013). The anxiety and depression subscales are comprised of 15 and 10 items, respectively. All 25-items are rated on a 4-point scale (never, sometimes, often, and always) and represent the frequency to which these behaviors, thoughts, or feelings occur (e.g., I have trouble sleeping). Overall scores range from 0 to 75 and individual responses are scored from 0 (never) to 3 (always). Higher scores indicate more severe anxiety and depression symptomatology. The RCADS-25 is comparable to the full-length version regarding test–retest reliability ($r_s = .78–.86$, $p < .001$) and internal consistency ($\alpha = .87–.95$; Brown et al., 2014). Cronbach's alpha values for the current sample were acceptable for both scales (anxiety: $\alpha = .86$; depression: $\alpha = .84$), as well as the total score ($\alpha = .92$).

2.2.2 | Go/NoGo task

A Go/NoGo task (Figure 1), a behavioral task used to elicit cognitive control abilities (Hare et al., 2005), was presented using E-Prime Professional 2.0 software. Children were presented with stimuli on a screen and asked to respond as fast and accurately as possible to a cue presented using a button box. Stimuli consisted of four different shapes (circle, diamond, square, and triangle), each presented in three different colors (blue, green, and orange). The presence or absence of a white box surrounding the shape was used to cue Go or NoGo trials. Children were asked to respond when a box surrounded the shape (Go trials) and not respond when there was no box (NoGo trials), or vice versa according to the counterbalanced condition.

Stimuli were presented on a black background and were approximately 506 × 650 pixels in size. Following a white fixation cross appearing on the screen, stimuli were presented pseudo-randomly in the center of the screen. The "Go" stimuli were initially displayed for 550 ms but this was dynamically adjusted to decrease by 50 ms or increase by 150 ms depending on the participant's performance on the previous three NoGo trials (Hum et al., 2013a). The response window for NoGo trials was set to be 200 ms longer than Go trials to ensure the nonresponse was deliberate. The task comprised of 20 practice trials followed by 2 blocks of 72 trials with a self-controlled break. Each block consisted of 48 Go trials and 24 NoGo trials that were pseudo-randomly presented.

2.2.3 | ERP components

N2 and P3 amplitudes were the stimulus-locked ERPs measured during Go and NoGo trials on the Go/NoGo task. A higher P3 amplitude reflects greater engagement of evaluation processes (Smith et al., 2008), whereas elevated N2 amplitudes represent increased conflict detection and engagement in cognitive control processes (Albert et al., 2013).

2.3 | Behavioral data processing

Four behavioral outcomes were obtained from the data: Go accuracy (%), NoGo accuracy (%), Go reaction time (RT), and NoGo RT. The primary behavioral outcome measure of response inhibition performance

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was NoGo accuracy, which represents the proportion of responses successfully withheld. Lower levels of NoGo accuracy reflect poorer ability to inhibit responses. Only responses made within 200 and 1200 ms of each trial were included to exclude nondeliberate responses. Go accuracy, defined as the proportion of correct responses to Go stimuli, and RT on Go and NoGo trials were reported to provide a more comprehensive account of behavioral performance.

2.4 | EEG data acquisition and processing

The EEG was recorded from 32 channels using an electrode cap (ActiCap, Brain Products) with Ag/AgCl electrodes placed according to the International 10–20 system. An ActiCHamp amplifier (Brain Products) was used, and EEG activity was sampled at a rate of 500 Hz. The reference channel was Cz and ground was placed over Fpz. Impedances were kept below 30 kΩ and channels were monitored during acquisition with noisy channels noted. Electrodes FT9, TP9, TP10, and FT10 were not included in the analysis due to poor signals across multiple participants.

The data were processed offline using MATLAB version R2021b. Data were initially band-pass filtered at 0.3–100 Hz and then re-referenced to the average activity of all the electrodes. Artifacts in the data were automatically identified using a threshold value of ±200 μV and then excluded from the data. Eye blinks were automatically identified as signals that met predefined thresholds of >100 ms rise time, >150 ms fall time, and >125 μV amplitude at electrodes Fp1 and Fp2, and visual inspection follow-up to ensure appropriate exclusion of blinks. Practice trials and those with anticipatory responses (RTs <200 ms) were also removed from the data. The data were then low-pass filtered at 30 Hz for ERP construction. Cleaned data were then segmented. Stimulus-locked ERPs (N2/P3) for Go and NoGo trials were segmented into 100 ms pre-stimulus baseline to 1000 ms poststimulus epochs.

The mean amplitude was calculated for each ERP in the channels Fz, Cz, and Pz. The N2 was scored from 240 to 340 ms and P3 scored from 350 to 450 ms in Fz. These time windows and electrode positions were established through comparison of grand mean waveforms and previous literature using the Go/NoGo task in the same age range (e.g., Jonkman, 2006). Mean amplitude was used in statistical analyses as this is reported to be a more robust measure of ERP waveforms than peak amplitude (Clayson et al., 2013). The average trial count used in ERP analyses for the Go condition was $M = 55.56$ (SD = 18.70, minimum–maximum = 16–94). For the NoGo condition, this was $M = 28.04$ (SD = 9.13, minimum–maximum = 7–40).

2.5 | Procedure

First, the researcher began capping the child while they completed self-report questionnaires. Children who were recruited in stage 1 completed the ChEAT and RCADS-25 in their schools (as described in Thomas et al., 2021) and did not have to repeat the questionnaires. Although self-report questionnaires were completed by the child independently, extra support was provided for children who required help with comprehension and reading in the school and laboratory sessions. Once the EEG cap had been fitted and electrode gel applied, the child was sat in a separate testing room to complete the tasks.

The EEG session began with a resting session, where baseline EEG data were collected with six 30 s blocks in which participants were instructed to alternate between keeping their eyes opened or closed for the 30 s duration. Before the Go/NoGo task started, participants were given verbal instructions. Visual instructions were also included on the display screen used to present the tasks. Checks of understanding were made by the experimenter before the task began. In total, the baseline and Go/NoGo task lasted approximately 10 min. In addition to the Go/NoGo task, the child completed an emotional version of the Go/NoGo task as part of a larger project. The order of these two tasks was counterbalanced across participants.

2.6 | Statistical analyses

All statistical analyses were conducted using SPSS (version 27.0). RCADS-25 data violated the assumption of normality based on visual inspection of histograms and the Shapiro–Wilk test of normality. This was corrected using a Log10(+1) transformation. Unless specified, untransformed data are presented in tables. Preliminary analyses found age was not correlated with questionnaire, behavioral, or ERP data, and boys and girls did not differ significantly in their data; therefore, gender and age were not included as covariates in subsequent analyses. *T*-tests revealed no significant differences in behavioral data collected between the two counterbalanced groups (i.e., whether the presence or absence of a white box surrounding the shape was used to cue Go or NoGo trials) as well as the order of the two Go/NoGo tasks. Homogeneity of variance was assessed by Levene's test for equality of variances. Where this was violated, comparative nonparametric tests were used for example, Mann–Whitney *U*. Bonferroni correction was used to adjust for multiple comparisons.

To test our primary hypotheses and investigate the associations among DE, internalizing symptoms, and measures of response inhibition, Pearson's *r* correlations were conducted. Where significant correlations were present between behavioral/ERP measures of response inhibition and DE/anxiety/depression, follow-up linear regression analyses were conducted to examine the independent contribution of DE, anxiety, and depression. In our secondary analysis, additional regression analyses were conducted to control for behavioral task performance in the association between neural markers and DE/anxiety/depression. Multicollinearity was tested using the variance inflation factor and was at an acceptable level (Neter et al., 1985), unless reported otherwise.

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TABLE 2 Descriptive statistics for self-report questionnaires and Go/NoGo behavioral data.

	M (SD)	Minimum–maximum
ChEAT	60.65 (12.57)	35–100
RCADS anxiety	11.44 (6.93)	2–32
RCADS depression	7.44 (4.83)	0–20
Go accuracy (%)	74.02 (15.62)	50.00–100.00
NoGo accuracy (%)	78.15 (9.80)	46.00–96.00
Go mean RT (ms)	363.04 (48.96)	275.88–539.40
NoGo mean RT (ms)	319.56 (56.73)	245.29–558.33

Note: $N = 48$.
Abbreviations: ChEAT, children's eating attitude test; RCADS, revised child anxiety depression scale; RT, reaction time.

TABLE 3 Correlations between questionnaire measures and N2 and P3 mean amplitudes on Go and NoGo trials.

	ChEAT	RCADS anxiety	RCADS depression
$N2_{Go}$	−0.153	−0.215	−0.122
$N2_{NoGo}$	−0.083	−0.182	−0.156
$P3_{Go}$	−0.273	−0.288*	−0.353*
$P3_{NoGo}$	−0.204	−0.317*	−0.392**

Note: Transformed data were used in the analyses.
Abbreviations: ChEAT, children's eating attitude test; RCADS, revised child anxiety depression scale.
*$p < .05$ level.
**$p < .01$.

3 | RESULTS

3.1 | Questionnaire and behavioral data

Descriptive statistics for self-report questionnaire and behavioral data are presented in Table 2. ChEAT scores were positively correlated with both RCADS anxiety ($r = .649$, $p < .001$) and RCADS depression scores ($r = .437$, $p = .002$). A strong positive correlation was also found between RCADS anxiety and RCADS depression scores ($r = .634$, $p < .001$). In addition, a repeated measures ANOVA was conducted to examine the main effects of trial type on accuracy and RT. There was not a significant difference in accuracy between Go and NoGo trials (F [1, 47] = 1.81, $p = .185$, and $\eta_p^2 = .037$), but faster mean RTs were found on NoGo trials compared to Go trials (F [1, 47] = 28.56, $p < .001$, and $\eta_p^2 = .378$).

To test the first hypothesis that there would be no significant correlations between behavioral markers of response inhibition and both DE and internalizing symptoms, correlational analyses between questionnaire measures and NoGo accuracy were performed. Secondary analyses revealed greater RCADS depression scores were significantly correlated with increased accuracy on NoGo trials ($r = .442$, $p = .002$). ChEAT and RCADS anxiety scores were not significantly correlated with NoGo accuracy ($r = .055$, $p = .711$; $r = .263$, $p = .071$, respectively). A follow-up linear regression confirmed RCADS depression scores ($B = 16.406$, SE $B = 4.907$, and $p = .002$) significantly predicted NoGo accuracy (F [1, 46] = 11.180, $p = .002$, and $R^2 = .196$).

3.2 | ERP measures

3.2.1 | Stimulus-locked ERPs ($N = 48$)

Figure 2 presents grand mean stimulus-locked waveforms for Go and NoGo trials. As expected, both the N2 and P3 components were larger for NoGo trials, compared to Go trials. However, the effect was only significant for P3 amplitudes (F [1, 47] = 42.565, $p < .001$, and $\eta_p^2 = .475$), but not for N2 amplitudes (F [1, 47] = 1.815, $p = .184$, and $\eta_p^2 = .037$).

Correlational analyses were conducted to test our second hypothesis that DE and internalizing symptoms would be associated with attenuated P3 amplitudes and enhanced N2 amplitudes. As shown in Table 3, there were no significant correlations present between questionnaire measures and N2 amplitudes. However, attenuated P3 amplitudes on both Go and NoGo trials were significantly correlated with higher scores on measures of anxiety and depression, whereas nonsignificant small to moderate effect sizes were found for associations with ChEAT scores.

Secondary analyses, in the form of linear regressions, were performed to further investigate the independent contribution of anxiety and depression symptoms on P3 amplitudes. For $P3_{Go}$ amplitudes, the model was significant (F [2, 45] = 3.404, $p = .042$, and $R^2 = .131$), but both RCADS anxiety ($B = -2.450$, SE $B = 4.088$, and $p = .552$) and depression ($B = -6.135$, SE $B = 3.973$, and $p = .120$) were not significant independent predictors. The same pattern of findings was also found for $P3_{NoGo}$ amplitudes, as although the model was significant (F [2, 45] = 4.343, $p = .019$, and $R^2 = .162$), both coefficients were not significant independent predictors of $P3_{NoGo}$ amplitudes (RCADS anxiety: $B = -2.505$, SE $B = 3.848$, and $p = .518$; RCADS depression: $B = -6.599$, SE $B = 3.645$, and $p = .077$).

These linear regressions were then repeated with the behavioral measure of NoGo accuracy included as an independent variable. The model predicting $P3_{Go}$ amplitudes was no longer significant (F [3, 44] = 2.229, $p = .098$, and $R^2 = .132$), with all three coefficients nonsignificant independent predictors (RCADS anxiety: $B = -2.433$, SE $B = 4.135$, and $p = .559$; RCADS depression: $B = -6.388$, SE $B = 4.214$, and $p = .137$; NoGo accuracy: $B = .015$, SE $B = .091$, and $p = .872$). Lastly, the model predicting $P3_{NoGo}$ amplitudes remained significant (F [3, 44] = 2.878, $p = .047$, and $R^2 = .164$), but all three coefficients were not significant independent predictors of $P3_{NoGo}$ amplitudes (RCADS anxiety: $B = -2.539$, SE $B = 3.887$, and $p = .517$; RCADS depression: $B = -6.097$, SE $B = 3.961$, and $p = .131$; NoGo accuracy: $B = -.029$, SE $B = .086$, and $p = .733$).

Follow-up analyses were conducted to examine the association between behavioral (NoGo accuracy) and neural markers of response inhibition (N2/P3 amplitudes). NoGo accuracy was not significantly

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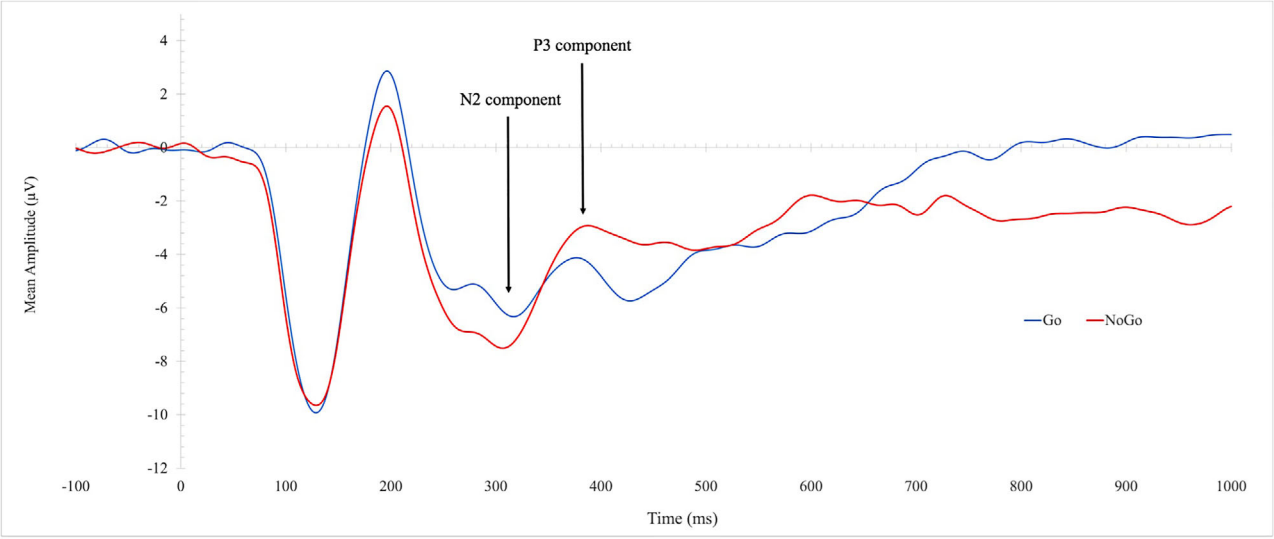

FIGURE 2 Grand mean stimulus-locked waveforms for Go and NoGo trials at Fz.

correlated with P3 amplitudes ($P3_{Go}$: $r = -.134$, $p = .365$; $P3_{NoGo}$: $r = -.214$, $p = .145$) or $N2_{Go}$ amplitudes ($r = -.219$, $p = .135$). However, better response inhibition performance was associated with greater $N2_{NoGo}$ amplitudes ($r = -.320$, $p = .026$).

4 | DISCUSSION

The current study examined the associations and independent contributions of DE and internalizing symptoms to variations in behavioral and neural markers of response inhibition in preadolescent boys and girls. Overall, our findings suggest there is some divergence between behavioral and neural evidence. As predicted, increased depression and anxiety were both significantly associated with suboptimal response inhibition in the form of attenuated $P3_{NoGo}$ amplitudes. The association of $P3_{NoGo}$ with DE also followed this pattern, but with a smaller effect size and it was not significant. In contrast, and contrary to expectation, self-reported depressive symptoms were positively correlated with enhanced response inhibition, as indexed by increased NoGo accuracy on the Go/NoGo task. Our findings suggest that associations among DE, internalizing symptoms, and neural indicators of response inhibition difficulties may be present before behavioral difficulties are observable in preadolescence. Therefore, these early neural changes, alongside increased internalizing symptoms, may play an important role in the early stages of DE development.

In our study, P3 and N2 amplitudes were used as neural markers of response inhibition during Go/NoGo performance. In line with our hypothesis, we found associations between increased anxiety and depression and attenuated P3 amplitudes, indicative of suboptimal evaluation of environmental inconsistency or conflict (e.g., a NoGo trial; Bruin et al., 2001). Our findings are consistent with P3 amplitude reduction reported in people with diagnosed depression (Houston et al., 2003; Klawohn et al., 2020; Nan et al., 2018; Santopetro et al., 2022, 2020) and anxiety (Bechor et al., 2019; Éismont et al., 2009; Wauthia et al., 2022; Xu et al., 2014) compared to unaffected comparators. Atypical ACC activation is characteristic of people with internalizing disorders (Blair et al., 2012; Lichenstein et al., 2016; Shang et al., 2014). Therefore, our finding that attenuated P3 activity, which is an index of ACC activity, is correlated with the RCADS suggests that hypoactivation of the ACC may be an early risk factor for internalizing symptom development. However, following regression analyses, we found neither anxiety nor depression were significant independent predictors of $P3_{NoGo}$ amplitudes, although the model was significant. Although collinearity statistics were within acceptable ranges, this finding suggests that anxiety and depressive symptoms overlapped in their shared variance with $P3_{NoGo}$ amplitudes rather than independently contributing to the multivariate model.

The same pattern was found between increased DE and attenuated P3, but the effects were smaller and did not reach significance. Comparisons with previous research examining associations between DE and P3 components are limited; however, adults with anorexia nervosa have shown smaller P3 amplitudes in a stop-signal task compared to healthy controls (Yue et al., 2020) and several ERP studies have reported attenuated P3 amplitudes in children with obesity (Reyes et al., 2015; Tascilar et al., 2011; Walk et al., 2020). However, internalizing symptoms were not assessed in these studies, and although these findings suggest neural markers of response inhibition difficulties may be present across a spectrum of eating difficulties, they may be driven by co-occurring internalizing symptoms.

The nonsignificant associations between N2 amplitudes and both DE and depression are consistent with previous findings (Palmwood et al., 2017; Santopetro et al., 2022; Yue et al., 2020). However, our findings are inconsistent with previous research in anxiety, where elevated N2 amplitudes have been reported (Hoyniak & Petersen, 2019; Valadez et al., 2021; Yu et al., 2018) and are thought to represent increased conflict detection and elevated engagement in cognitive control processes (Dennis & Chen, 2009). Methodological factors may explain the absence of an association in the current study. Studies that

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find enhanced N2 amplitudes in people with anxiety typically use emotional stimuli (Hum et al., 2013, 2013ab; Yu et al., 2018), whilst those using nonemotional stimuli tend not to find effects (Baving et al., 2004; Larson et al., 2013; Voegler et al., 2018).

Although there were no significant associations between both DE and anxiety and our behavioral marker of response inhibition, NoGo accuracy; increased depression was associated with stronger inhibitory processes through increased accuracy on NoGo trials. This contrasts with previous investigations of preadolescents and adolescents with internalizing symptoms, who do not differ in response inhibition performance compared to healthy controls (Brunnekreef et al., 2007; Diler et al., 2014; Hum et al., 2013a; Pan et al., 2011; Singh et al., 2010), and was an unexpected pattern given that adults with internalizing symptoms show reduced behavioral inhibition on Go/NoGo tasks (Kaiser et al., 2003; Pacheco-Unguetti et al., 2012; Xia et al., 2020). However, there is some support for enhanced response inhibition in other general population samples of children with internalizing symptoms (Kooijmans et al., 2000; Oosterlaan et al., 1998). This may suggest that less severe levels of internalizing symptoms in preadolescence facilitate increased inhibition, whereas more severe and clinically relevant internalizing symptoms result in reduced and maladaptive inhibition (e.g., Gray, 1982).

It is important to consider why there was a dissociation between behavioral and neural markers of response inhibition in our study. Although neural activity related to evaluation processes was reduced in children with higher levels of depression, other compensatory processes may enable performance to be enhanced behaviorally. For example, emerging depressive symptoms are thought to facilitate inhibitory control by promoting an analytical processing style (Ambady & Gray, 2002; Andrews & Thomson, 2009). As information is processed more slowly and thoroughly, this may lead to a speed-accuracy trade off that biases accuracy. Indeed, this pattern of findings has been reported in preadolescents with internalizing symptoms compared to children with no internalizing problems (Brunnekreef et al., 2007). The authors proposed that children with internalizing symptoms have the capacity to perform accurately on response inhibition tasks, given they have sufficient time to respond. Inhibition tasks that require more processing time, such as more complex inhibition tasks or those including emotional stimuli, may lead children with internalizing symptoms to instead resort to trading accuracy for speed. To disentangle the complex processes involved in inhibitory control and their associations with internalizing symptoms and DE, drift diffusion models may be a useful tool in future research. Drift diffusion models provide greater sensitivity than typical outcome measures (RT and accuracy) and have previously reported a speed-accuracy trade off in individuals with depression during flanker tasks (Dillon et al., 2015; Pe et al., 2013).

The contradictory pattern of behavioral and neural findings may also be explained by our finding that NoGo accuracy was not significantly associated with P3 amplitudes. Instead, NoGo accuracy was significantly negatively correlated with $N2_{NoGo}$ amplitudes, indexing increased engagement of cognitive control processes and conflict detection. Our findings are consistent with Xia et al. (2020), who suggest NoGo accuracy and P3 amplitudes may index different response inhibition processes. This is in line with the idea that N2 and P3 amplitudes represent distinct neural correlates of response inhibition (Albert et al., 2013; Smith et al., 2008). For example, although P3 amplitudes index a specific response inhibition process during the Go/NoGo task, the behavioral measure may capture a wider range of cognitive and perceptual processes relevant to task performance. This has implications for the use of behavioral and neural measures of response inhibition in isolation and suggests multiple measures are needed to provide a thorough examination of these abilities. Although replication is necessary, our findings suggest that internalizing symptoms in preadolescence may be specifically related to evaluation and monitoring processes rather than earlier inhibition processes, such as conflict detection.

The current study has important implications for our understanding of the development of DE and internalizing symptoms in preadolescence. Although DE and internalizing symptoms are highly correlated in this age group, behavioral and neural markers of response inhibition appear to significantly relate to depressive symptoms only. This association may have clinical utility in identifying and predicting depressive symptoms in preadolescence. For example, response inhibition difficulties may be used as an objective tool for assessing depressive symptoms alongside self-report measures, with the potential to predict the onset of depressive symptoms (Shimony et al., 2021). Furthermore, support aimed at training effective response inhibition ability in preadolescence, such as using cognitive training programs (Koster et al., 2017; Woolf et al., 2022), may help prevent or lower depressive symptoms. In turn, lowering levels of depressive symptoms may offer a route to preventing the development of early ED symptoms, reducing the likelihood of a clinical ED developing. However, the cross-sectional nature of the data means that we cannot draw causal conclusions from these findings. Future studies should use longitudinal data to study the trajectories of the relations among DE, internalizing symptoms, and cognitive control across development.

One limitation of our study is the issue of task impurity, a commonly reported measurement problem in the executive functioning literature (Best & Miller, 2010; Hughes & Graham, 2002). For example, performance on inhibitory control tasks requires attention and working memory, as well as adequate processing speed (McAuley & White, 2011). Therefore, it is difficult to isolate response inhibition ability from these other abilities. Future studies could address this issue by using multiple response inhibition measures to create a latent variable that is more representative of the underlying construct (e.g., Aichert et al., 2012) and explore it is associations with DE and internalizing symptoms.

The measure of DE used in the current study may also present some limitations. The ChEAT is only able to capture a global measure of DE but not subtypes of DE behaviors, such as restrictive type eating behaviors, binge/purge behaviors, and weight and shape concerns. Specific links have been found between executive functioning and anorexia nervosa subtypes, such as increased response inhibition difficulties in individuals with binge/purge type anorexia nervosa (Galimberti et al., 2012) compared to individuals with restrictive type anorexia nervosa

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(Weinbach et al., 2020). Therefore, global measures of DE may not be able to capture more nuanced associations between response inhibition and specific DE symptoms, such as binging/purging and restriction. Although validated measures of DE are limited in preadolescents, future studies may benefit from using a combination of measures to quantify DE symptom profiles. For example, the Eating Disturbances in Childhood-Questionnaire (Dyck et al., 2013) assesses avoidant or restrictive food intake in relation to avoidant/restrictive food intake disorder (ARFID), whilst the child version of the ED Examination Questionnaire (Decaluwé, 1999) captures ED symptoms related to anorexia nervosa, bulimia nervosa, and binge-ED, but not ARFID.

In summary, we found that higher levels of internalizing symptoms were associated with neural markers of suboptimal evaluation and monitoring performance, as indexed by attenuated $P3_{NoGo}$ amplitudes, in preadolescence. In contrast, higher levels of depression were associated with greater response inhibition at a behavioral level. This suggests that the specific difficulties in evaluation and monitoring found in those with higher levels of depression were not sufficient to disrupt behavioral performance, which relied on a broader range of response inhibition-relevant processes. In addition, although we found modest effect sizes, DE was not significantly associated with response inhibition. This indicates that difficulties in response inhibition may only be evident in more chronic and severe DE and ED presentations.

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CONFLICT OF INTEREST STATEMENT

The authors report there are no conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

Data are available at https://osf.io/76ze2/.

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