



The role of cognitive control in the co-occurrence of disordered eating and internalizing symptoms in preadolescence.

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Summary of Thesis

This thesis aimed to investigate the associations between cognitive control, disordered eating (DE), and internalizing symptoms in a typically developing sample of preadolescents. Chapter 1 presents a review of the core constructs and related literature surrounding what is currently known about the overlap between DE, internalizing disorders, and cognitive control. In Chapter 2, I investigated associations between self-reported DE and internalizing symptoms in a demographically diverse sample of preadolescents, finding strong evidence for these associations in both boys and girls.

In Chapter 3, I examined the role of set shifting, finding no behavioral evidence for an association between poorer set shifting and higher levels of DE and internalizing symptoms in preadolescents. Cognitive control processes were examined further in Chapter 4, in the form of response inhibition and response monitoring. During a Go/NoGo task, electroencephalography was used to measure event-related potentials. I found no behavioral evidence to suggest DE was associated with impairments in response inhibition or response monitoring performance in preadolescence. However, higher levels of depression were associated with greater response inhibition performance but impaired response monitoring performance. When examining neural markers, significant associations were found between some neural markers of response inhibition and increased levels of anxiety and depression.

In Chapter 5, I built upon the work described in Chapter 4 by exploring these cognitive control processes in the context of emotion. As in Chapters 3 and 4, I found no associations between behavioral performance on the emotional Go/NoGo task and DE and internalizing symptoms. However, higher levels of DE were associated with poorer recognition of happy facial expressions and enhanced neural markers of response inhibition during the presentation of happy faces in the emotional Go/NoGo task. Lastly, blunted neural markers of response monitoring were associated with increased DE in the context of emotion only.

In summary, findings from this thesis demonstrate neural indicators of cognitive control impairments are associated with the early emergence of DE and internalizing symptoms in preadolescence. Implications for early intervention and future research are discussed in Chapter 6.

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List of abbreviations

ACC = anterior cingulate cortex	ERN = error-related negativity
ALN = additional learning needs	ERP = event-related potential
AN = anorexia nervosa	fMRI = functional magnetic resonance imaging
AN-R = anorexia nervosa-restricting subtype	FSM = Free school meal
AN-BP = anorexia nervosa-binge/purge subtype	HCs = healthy controls
ARFID = avoidant/restrictive food intake disorder	MDD = major depressive disorder
BED = binge eating disorder	OCD = obsessive compulsive disorder
BN = bulimia nervosa	OFC = orbitofrontal cortex
BRIEF-P = Behavior Rating Inventory of Executive Function – Preschool version	OSFED = other specified feeding and eating disorder
ChEAT = Children’s Eating Attitude Test	Pe = error positivity
CRN = correct-related negativity	PFC = prefrontal cortex
DE = disordered eating	P3d = P3 _{NoGo} – P3 _{Go} difference wave
DLPFC = dorsolateral prefrontal cortex	RCADS-25 = Revised Child Anxiety Depression Scale – 25 item version
DSM-5 = Diagnostic and Statistical Manual of Mental Disorders, fifth edition	RDoC = Research Domain Criteria
EAL = English as an additional language	RT = reaction time
EAT-26 = Eating Attitudes Test – 26 items	SEN = special education needs
ED = eating disorder	SES = Socio-economic status
EDNOS = eating disorder not otherwise specified	STT-CV = Shape Trail Test – Child Version
EEG = electroencephalography	TMT = trail making Test
	TMT-CV = Children’s Trail Making Test
	WCST = Wisconsin Card Sorting Test
	WIMD = Welsh Index of Multiple Deprivation

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Impact of Thesis

Findings described in this thesis have been presented at national and international conferences. Chapters in this thesis have also been based on work published in peer-reviewed manuscripts.

Chapter 1

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Potential risk factors for disordered eating behaviours in children. *Poster presentation at International Conference on Children's Eating Behaviour, Birmingham, March 2019.*

Chapter 2

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Chapter 4

Neural correlates of response monitoring and response inhibition in disordered eating during preadolescence. *Poster presentation at Society for Research in Child Development 2021 Virtual Biennial meeting, April 2021.*

Associations between disordered eating, obsessive-compulsive behaviours, and neural indicators of response inhibition during preadolescence. *Data blitz presentation and poster presentation at British Association for Cognitive Neuroscience Annual Meeting, Birmingham, May 2022.*

Chapter 5

Neural correlates of emotion regulation and associations with disordered eating during preadolescence. *Oral presentation at The British Feeding and Drinking Group 46th Virtual Annual Meeting, April 2022*

Chapter 1. General Introduction

Eating disorders (EDs), such as anorexia nervosa (AN) and bulimia nervosa (BN), are a heterogeneous set of conditions characterized by a complex combination of psychological and physical symptoms (e.g., binge eating and purging, dieting, low body weight, and preoccupying cognitions around eating, shape, and weight). EDs have the highest mortality rate of any psychiatric illness (Smink et al., 2012) and treatment outcomes for EDs are modest. In adolescents with AN and BN treated with the leading evidence-based interventions, long-term remission rates are only 35-40% (Le Grange et al., 2014; Le Grange et al., 2015). Age-standardized annual incidence rates for all diagnosed EDs in the UK are 37.2 per 100,000 in 2009. However, the highest incidence was found in 15- to 19-year-old girls and 10- to 14-year-old boys (Micali et al., 2013). More recent evidence suggests these diagnoses are increasingly occurring at earlier developmental periods (Nicholls et al., 2011; Petkova et al., 2019; Reas & Rø, 2018). Applying the Diagnostic and Statistical Manual of Mental Disorders fifth edition (DSM-5) diagnostic criteria, Petkova et al. (2019) reported an increase in the overall incidence of AN in children aged between 8-12 years, from 2.1 per 100,000 in 2006, to 3.2 per 100,000 in 2015. However, this figure may be an underestimate of the true prevalence of EDs, as statistics based on individuals with a diagnosis or assessment through primary mental health services often only represent the most serious cases (Hoek & Van Hoeken, 2003).

Overall, research examining treatment outcomes overwhelmingly advocates for early detection and intervention of EDs to improve prognosis. Clinical studies suggest that response to treatment is greater during the early stages of the ED, but may diminish across the duration of the illness (Ambwani et al., 2020) as the ED becomes more fixed and entrenched over time (Steinglass & Walsh, 2016). In line with this, early detection and intervention of EDs has been a key recommendation in several Government commissioned reviews of ED services across the UK (NHS England, 2015; Scottish Government, 2021; RQIA, 2015; Tan, 2019), as well as international ED strategies (InsideOut Institute, 2021; Streatfeild et al., 2021). Specifically, taking a whole school approach to the prevention of EDs and helping teachers and parents identify early signs of EDs has been highlighted as an important step in improving Welsh ED services (Tan, 2019). Therefore, research focused on prodromal and early stages of the ED to facilitate better treatment response and prevent chronic and severe full syndrome EDs is needed.

In addition to the lack of effective treatment options, ED presentations are complex and often co-occur with many other mental health conditions. Internalizing disorders, such as anxiety and depression, represent the most commonly reported co-occurring conditions (Hudson et al., 2007). Co-occurring internalizing symptoms are also found to negatively influence an individual's ED severity and prognosis (Castellini et al., 2011; Dellava et al., 2010; Kaye et al., 2004; McDermott et al., 2006). Previous research in adults and adolescents has examined this important co-occurrence and demonstrated shared behavioral, cognitive, and neurobiological correlates (Bakalar et al., 2015; Kaye et al., 2004; Silberg & Bulik, 2005; Ulfvebrand et al., 2015). A greater understanding of these shared underlying cognitive processes and neurobiological correlates at earlier developmental periods could also help identify potential risk factors and target early interventions before the onset of the disorder. This is because examination of cognitive processes and neurobiology in EDs can be constrained by illness-related sequelae, such as malnutrition. The cognitive processes underlying the overlap between EDs and internalizing disorders are not clearly understood as it can be challenging to disentangle trait factors present before the onset of the ED from state or scar effects, due to the impact of a current or previous ED.

To address these confounds, research has started to investigate behavior and cognition in community samples where ED-related symptoms can be investigated dimensionally, rather than employing categorical designs, such as case-control comparisons. Research examining eating pathology dimensionally in younger age groups prior to the typical onset of an ED is an important avenue for investigation given disordered eating (DE) has been shown to emerge in late childhood (Graber et al., 1994; Sands et al., 1997) and is a predictor of an ED diagnosis in adolescence (Evans et al., 2017). Internalizing symptoms are also found to emerge during childhood (Solmi et al., 2021) and are predictive of anxiety disorders in adolescence (Beesdo et al., 2009), further supporting the rationale for studying the co-occurrence of these symptoms in preadolescence.

By studying pre-clinical symptoms using a transdiagnostic approach, research is better able to capture and identify the trajectories of DE and internalizing symptoms, as well as their relations to cognitive functions and neurophysiology (Wildes & Marcus, 2013). This thesis will focus on examining cognitive control, a set of cognitive functions implicated in both EDs and internalizing disorders (discussed in section 1.5 onwards). Compared to broader executive functions, less empirical attention has been given to how cognitive control impairments relate to the *early emergence* of co-occurring DE and internalizing symptoms. Therefore, using a transdiagnostic and dimensional approach, I will recruit typically

developing preadolescents to investigate the role of cognitive control in the co-occurrence of DE and internalizing symptoms. This thesis will specifically examine whether associations between cognitive control difficulties and DE occur independently of internalizing symptoms, or whether internalizing symptoms mediate the relation between these processes and DE.

This chapter provides an overview of eating disorders, internalizing disorders, and their co-occurrence, including common psychological factors and shared neurocircuitry and neurobiology. It then focuses on the anterior cingulate cortex (ACC) as a key brain region at the center of eating psychopathology, internalizing disorders, and cognitive control. Finally, the chapter provides an overview of the literature examining cognitive control in both disorders, as well as cognitive control in the context of emotion.

1.1 Eating Disorders (EDs)

1.1.1 Anorexia Nervosa (AN)

AN is an ED that presents with extreme dietary restriction and weight loss, characterized by a distorted perception of body image that is linked to an intense fear of gaining weight (American Psychiatric Association, 2013). These symptoms are often seen as the primary constituents of AN; however, the development of eating psychopathology is still poorly understood. Individuals with AN exhibit extreme restraint towards eating, alongside ritualized and repetitive behaviors surrounding diet and weight (Kaye, 2008). Prevalence rates vary considerably; however, rates between 1-3% have been reported in the general population (Dahlgren et al., 2017). Individuals with AN are consistently shown to have elevated mortality rates compared to other mental health difficulties, including other EDs (Arcelus et al., 2011; Harris & Barraclough, 1998; Lomholt et al., 2019).

Mortality rates highlight the severity of AN and longitudinal studies report negative outcomes following treatment and remission. The long-term consequences of AN include chronic AN presentations, diagnostic crossover to other EDs, and physical health issues such as cardiac complications, osteoporosis, and renal disease (Fichter et al., 2017; Herzog et al., 1997; Meczekalski et al., 2013). But these outcomes can be minimized or avoided by earlier access to treatment (Andrés-Pepiñá et al., 2020), again highlighting the importance of early identification and intervention of ED symptoms.

1.1.2 *Bulimia Nervosa (BN)*

BN is characterized by a distorted perception of body weight and shape, as well as a fear of gaining weight. The main symptoms of the disorder include recurrent episodes of binge eating followed by compensatory behaviors to prevent weight gain (Levinson et al., 2017). These compensatory behaviors are commonly seen in the form of self-induced vomiting; however, alternative methods include excessive exercise and misuse of diuretics and/or laxatives (American Psychiatric Association, 2013). The binge-purge cycle is a conceptualization of the disorder that attempts to understand the pattern of behavior and how it is maintained (Pearson et al., 2015). In this cycle the binge eating can often be identified as an avoidance strategy used by the individual to provide a distraction from a distressing life event or an increase in negative affect. Once the distress has decreased the binge eating is negatively reinforced; however, the physical discomfort from the binge and fear of gaining weight can then lead to compensatory behaviors. The discomfort starts to decrease following these compensatory behaviors and the elevated affective state following the binge starts to become more salient, starting the cycle over again (Pearson et al., 2015). These DE behaviors, therefore, become maladaptive coping strategies for dealing with distress or negative affect in BN.

Prevalence rates for BN vary considerably between studies; however, most prevalence rates are reported to be between 0.5 – 1.5% (Smink et al., 2012). The median age at BN diagnosis is reported to be later than at AN diagnosis, at around 18 years compared to 17 years (Solmi et al., 2021), with males diagnosed later than females (Demmler et al., 2020). Although the mortality rate of BN is lower than AN, individuals can present with long-term cardiac complications (Tith et al., 2020), severe electrolyte and acid base imbalances, dental issues, as well as difficulties eating and chronic abdomen pain associated with persistent acid reflux (Westmoreland et al., 2016).

1.1.3 *Binge Eating Disorder (BED)*

BED was first listed as a distinct ED diagnosis in the DSM-5, whilst previous versions of the DSM required binge eating to be diagnosed as Eating Disorder Not Otherwise Specified (EDNOS; American Psychiatric Association, 2013). The key diagnostic features of BED are recurrent episodes of binge eating, the presentation of marked distress regarding persistent binge eating, and an absence of consistent compensatory behaviors (American Psychiatric Association, 2013). It should also be noted that, although less common than

overeating, BED is much more severe and often co-occurs with other mental health conditions, such as anxiety and depression (Leehr et al., 2015).

Few studies have estimated prevalence rates of BED, however estimates are between 0.5 – 3% in the general population, and between 1.3 – 30.1% in obese adults, a particular risk group for developing this ED (Dingemans et al., 2002). Median age of BED onset is 20 years, typically later than age of onset of AN and BN (Solmi et al., 2021). Although research is still in the early stages, there does appear to be some significant predictors of outcome in individuals with BED. Specifically, higher levels of body dissatisfaction and impulsivity are predictive of poorer prognosis in individuals with BED, as well as experience of sexual abuse (Fichter et al., 2008). It is also important to consider the elevated co-occurrence between BED and obesity, and the implications that this has on physical and mental health.

1.1.4 Other Specified Feeding and Eating Disorders (OSFED)

OSFED, previously known as EDNOS in earlier versions of the DSM, has a lifetime prevalence rate around 1.5% (Mustelin et al., 2016). The mean age of onset of OSFED is 18 years in females, with a median duration of illness of 2 years (Mustelin et al., 2016). OSFED is a diagnosis that can be applied more broadly to individuals who present with clinically significant impairment and distress but do not meet the full criteria for an ED (American Psychiatric Association, 2013). Examples of OSFED include atypical AN, where all criteria are met including significant weight loss; however, the individual's weight is still within or above the healthy range (American Psychiatric Association, 2013). Research has reported a decrease in the prevalence of partial and sub-threshold EDs as a result of this new diagnosis (Hammerle et al., 2016), leading to better identification of individuals who do not meet the full criteria for an ED but who require professional intervention.

1.1.5 Avoidant/Restrictive Food Intake Disorder (ARFID)

ARFID was first introduced as a diagnostic category in the DSM-5 (American Psychiatric Association, 2013) and replaces the Feeding Disorder of Infancy or Early Childhood, which was restricted to children up to age six. ARFID extends this previous category by providing a more specific and detailed description of the eating difficulties, as well as a broader criteria that is appropriate across the age range (Bryant-Waugh, 2013). DSM-5 diagnostic criteria of ARFID describes an eating or feeding disturbance that manifests as a persistent inability to meet appropriate nutritional and/or energy needs

resulting in significant weight loss, nutritional deficiency, a reliance on nutritional supplements, or significant psychosocial difficulties (American Psychiatric Association, 2013). Examples of avoidance and restriction of eating are a lack of interest in eating or food, avoidance based on the sensory aspects of food, as well as fear of aversive consequences of food (American Psychiatric Association, 2013). Importantly, ARFID is not typically accompanied by concerns with weight or shape, which is an important distinction between ARFID and AN or BN (Bryant-Waugh, 2013).

Large scale epidemiological studies for ARFID are scarce; however, in a school-based survey of 1444 children aged 8-13 years in Switzerland, 3.2% reported symptoms consistent with ARFID (Kurz et al., 2015). Prevalence rates of ARFID in clinical ED samples of children and adolescents are higher and range between 5% to 14% (Nicely et al., 2014; Ornstein et al., 2017). Compared to individuals with AN and BN, those with a diagnosis of ARFID are generally younger, are more likely to have an anxiety disorder, report a longer duration of illness prior to diagnosis, include a greater number of males, and are more likely to have a medical condition (Fisher et al., 2014). This suggests ARFID is both demographically and clinically distinct from AN and BN and captures individuals who may not have been previously recognized as having an ED. ARFID in childhood has been suggested as a risk factor for AN in adolescence (Norris et al., 2014); however, the shared etiology between AN and ARFID is not clear and more longitudinal research is needed.

Table 1.1 presents a summary of the key features of each of the ED diagnoses discussed in this section.

Table 1.1.*Summary of key ED features.*

ED diagnosis	Median age of onset	Typical presentation	Prevalence
AN	17 years (Solmi et al., 2021)	Extreme dietary restriction, weight loss, distorted body image perception, fear of gaining weight, obsessive preoccupation with food, and/or excessive exercise.	1 - 3% (Dahlgren et al., 2017)
BN	18 years (Solmi et al., 2021)	Recurrent episodes of binge eating and compensatory behaviors e.g., self-induced vomiting, excessive exercise, and misuse of diuretics and laxatives.	0.5 - 1.5% (Smink et al., 2012)
BED	20 years (Solmi et al., 2021)	Recurrent episodes of binge eating and the absence of compensatory behaviors, as well as marked distress regarding persistent binge eating.	0.5 - 3% (Dingemans et al., 2002)
OSFED	18 years (Mustelin et al., 2016)	A broader diagnosis that captures clinically significant impairment and distress surrounding food and eating, but the individual does not meet full diagnostic criteria for an ED.	1.5% (Mustelin et al., 2016)
ARFID	10-11 years (Nicely et al., 2014)	Avoidance and restriction of eating leading to an inability to meet appropriate nutritional/energy needs. E.g., lack of interest in eating or food, avoidance based on the sensory aspects of food, as well as fear of aversive consequences of food. Absence of weight and shape concerns.	<1% - 15.5% (Bourne et al., 2020)

Note. ED: Eating Disorder, AN: Anorexia Nervosa, BN: Bulimia Nervosa, BED: Binge Eating Disorder, OSFED: Other Specified Feeding and Eating Disorders, ARFID: Avoidant/Restrictive Food Intake Disorder.

1.1.5. Disordered Eating (DE)

DE, such as binge eating, dieting, restriction of eating, and purging, are behaviors present in individuals with diagnosed EDs, such as AN and ARFID. These patterns of behavior are also found to commonly occur in the general population, although in a less severe and frequent form than clinical populations (Naor-Ziv & Glicksohn, 2016). Research

has reported estimates of the prevalence of diagnosed EDs in women to be 8.4%, whereas subclinical prevalence estimates are considerably higher at 19.4%. For men, estimates of the prevalence of both diagnosed EDs (2.2%) and subclinical presentations (13.8%) are lower than those estimated for women (Galmiche et al., 2019).

DE has been found to emerge in late childhood and remain stable into adolescence and young adulthood (Hilbert et al., 2013; Neumark-Sztainer et al., 2011). DE in childhood increases the risk of developing a diagnosable ED in adolescence (Evans et al., 2017; Herle et al., 2020; Kotler et al., 2001; Tanofsky-Kraff et al., 2011). A recent adolescent cohort study found the most prominent rise in DE to occur between ages 12 to 15 years, with high-risk individuals already displaying elevated DE behaviors at 12 years of age (Breton et al., 2022). As most ED prevention programs start in later adolescence (Ciao et al., 2014), this study underlines the importance of starting these programs in preadolescence, before DE behaviors start to increase and antecedents can be identified.

1.1.6 Models of EDs

The Transdiagnostic Model (Fairburn et al., 2003) and the Cognitive-Interpersonal Maintenance Model (Schmidt & Treasure, 2006) are well-validated maintenance models of EDs. The Transdiagnostic Model was designed to apply to all forms of EDs, stating that, despite some variation in presentations of symptomatology, all share the same distinctive psychopathology. Central to this shared psychopathology is an over-evaluation of eating, shape and weight and their control (Fairburn et al., 2003). In addition, this model proposes four key constructs that are possibly relevant to all ED presentations, namely clinical perfectionism, interpersonal difficulties, mood intolerance, and core low self-esteem (Fairburn et al., 2003).

The Cognitive-Interpersonal Maintenance Model was developed specifically for AN, combining intra- and interpersonal factors (Schmidt & Treasure, 2006). This model proposed a combination of predisposing factors, such as obsessive-compulsive traits, perfectionism, and anxious avoidance, that contribute to the maintenance of the disorder through strengthening of pro-anorectic beliefs and behaviors. These traits result in interpersonal difficulties which can be further compounded by reactions from others, due to the profound impact ED symptoms and behaviors can have on them (Schmidt & Treasure, 2006).

These models suggest areas of cognition and emotion are central to the maintenance of EDs; however, further research is required to understand whether these factors are also important in the early emergence of eating pathology.

1.2 The Co-occurrence of Internalizing Disorders and EDs

The previous section noted a substantial overlap between some EDs and internalizing disorders. The label ‘internalizing disorders’ has been widely used in developmental psychopathology and encompasses anxiety and depression symptoms and disorders (Kovacs & Devlin, 1998). This is due to the central feature of internalizing disorders being emotion and mood-related difficulties (Kovacs & Devlin, 1998), as well as difficulties of overcontrol (Rubin & Mills, 1991), such as social withdrawal and hypervigilance. The focus of this review will be on anxiety and depression diagnoses and symptomatology.

1.2.1 Anxiety and EDs

Anxiety is defined as the anticipation of future threat and danger, often associated with hypervigilance, avoidance, and cautious behaviors (American Psychiatric Association, 2013). The experience of anxiety itself can be protective and adaptive, but anxiety that is excessive, persistent, and often stress-induced may meet the criteria for an anxiety disorder. In children, anxiety can present on a continuum, from milder, transient symptoms to diagnosable anxiety disorders. Therefore, some children may experience distressing anxiety symptoms but not meet the criteria for an anxiety disorder, highlighting the importance of investigations utilizing dimensional behaviors and symptoms in the community, rather than in clinical samples only.

Anxiety disorders often co-occur with each other but can differ according to the types of environments or situations that induce anxiety and the associated cognitions. There appears to be some anxiety disorders that typically develop later on in adolescence, such as generalized anxiety disorder, panic disorder, and agoraphobia (American Psychiatric Association, 2013). However, many are found to develop during childhood (Solmi et al., 2021), with separation anxiety, selective mutism and specific phobias emerging in early childhood, and social anxiety disorder typically occurring in late childhood into adolescence (American Psychiatric Association, 2013).

Co-occurrence of eating pathology and anxiety has been well established and includes community samples of adults (Godart et al., 2007) and adolescents (Rojo-Moreno et al.,

2015). Potential mechanisms for this co-occurrence include shared etiological factors, such as harm avoidance cognitions and safety behaviors (Pallister & Waller, 2008), as well as DE behaviors functioning as maladaptive coping strategies for dealing with anxiety and emotional distress (Fairburn et al., 2003). An anxiety diagnosis may also serve as a risk factor for developing an ED. Anxiety disorders very often precede the co-occurring ED (Swinbourne et al., 2012) and individuals with EDs frequently report a childhood-onset anxiety disorder, with an average onset at 8-10 years (Adambegan et al., 2012; Godart et al., 2000; Kaye et al., 2004; Raney et al., 2008).

Several studies have reported co-occurrence of anxiety symptoms and DE in older female adolescents (e.g. Touchette et al., 2011; Zaidler et al., 2000); however, inconsistent findings have been reported in preadolescence. Holm-Denoma et al. (2014) reported a weak and largely non-significant association between DE and anxiety symptoms across boys and girls, and two age cohorts: 9-11 years and 12-15 years. However, their measure was not adapted for their younger participants. In contrast, significant associations between DE and both general and social anxiety have been found in preadolescents (Houldcroft et al., 2014). Significant gender differences in reports of general and social anxiety were also found, with a higher number of symptoms reported by girls than boys. Houldcroft et al. (2014) measured eating behaviors through an examination of dietary restraint, emotional eating, and external eating; constructs that are more associated with behaviors present in BN and binge eating (e.g. Johnson et al., 2012; Stein et al., 2007), rather than broader eating psychopathology.

1.2.2. Depression and EDs

Depression can present as a clinical depressive disorder, such as major depressive disorder (MDD), as well as less severe presentations that do not meet diagnostic criteria. Its key features include low or irritable mood, loss of interest or pleasure in previously enjoyable activities, changes in appetite, fatigue, and sleep problems (American Psychiatric Association, 2013). Increased risk of suicide is also seen in individuals with depression, especially in older adolescents and adults (Bernaras et al., 2019).

Co-occurrence of EDs and depression has been commonly reported (see Godart et al., 2007 for a review of the literature), with increased levels of suicidal behavior and self-harming also seen in ED samples (Koutek et al., 2016). MDD is reported to be the most common co-occurring depressive disorder in individuals with EDs (Ulfvebrand et al., 2015), particularly in females with BED (Grilo et al., 2009; Ulfvebrand et al., 2015).

Research exploring gender differences in adolescents has reported the co-occurrence of DE behaviors and depression symptoms to be present in both males and females. For example, body weight dissatisfaction is associated with higher levels of depression, and body image factors are predictive of depression, in both male and female high school students (Santos et al., 2007; Tomori & Rus-Makovec, 2000). Several studies have shown age-dependent gender differences in depression, DE, and the co-occurrence of these from preadolescence to mid-adolescence. For example, gender differences in concurrent DE and depression emerge between ages 12-15 years; however, levels of DE and depression are similar in boys and girls before this point (Evans et al., 2017; Ferreira et al., 2012; Holm-Denoma et al., 2014), suggesting puberty may play a role in these distinct trajectories.

A series of mechanisms have been proposed to help understand the co-occurrence of EDs and depression. Evidence from twin studies suggests there is a genetic association, as well as shared environmental factors that specifically influence risk for both early ED and depressive symptoms (Silberg & Bulik, 2005; Wade et al., 2000). Another suggestion is that DE serves as a maladaptive way of coping with and managing depressive symptoms (Beato-Fernández et al., 2007; Puccio et al., 2016; Tomori & Rus-Makovec, 2000). Alternatively, negative self-evaluations, a key feature of depression, could lead to negative evaluations of body weight and in turn DE behaviors (Kenny et al., 2021). The direction of this effect is still unclear. These mechanisms have largely been studied in females and different developmental pathways may be present for males.

1.2.3 The Co-occurrence of Anxiety and Depression in DE/EDs

Anxiety and depression commonly occur in DE and EDs, and the co-occurrence of these internalizing disorders has been shown to lead to more severe ED symptoms compared to individuals with no co-occurring conditions (Elran-Barak & Goldschmidt, 2021). The relations between ED symptoms, anxiety, and depression have also been found to be reciprocal (Drieberg et al., 2019), suggesting DE, anxiety, and depression could serve as maintenance factors as well as risk factors.

Within this co-occurrence there appears to be differential effects of anxiety and depression on eating pathology, specifically concerning symptom severity. Elran-Barak and Goldschmidt (2021) stated individuals with co-occurring anxiety reported more severe ED symptoms relative to individuals with co-occurring depression. However, contrasting evidence has suggested more complex and severe EDs for children and adolescents with co-

occurring depression compared to those with no co-occurring disorder or co-occurring anxiety alone (Hughes et al., 2013).

Differential associations between anxiety and depression with ED symptoms could reflect the different functions of these behaviors. In overweight youth with anxiety, emotional eating was proposed to function as a way of coping with hyper-arousal, whereas for children with depression, emotional eating was suggested to be a way of generating more positive emotions (Goossens et al., 2009). In both male and female adults with diagnosed EDs, there are gender differences in the most common co-occurring diagnosis; for males, generalized anxiety disorder is the most common and MDD is most common in females (Ulfvebrand et al., 2015). Suggesting internalizing disorders may function differently in males and females within EDs.

As highlighted in section 1.2.1, not all measures used in studies with children are validated for use with children, which could account for some of the inconsistencies in the literature. Although research in preadolescents has focused on the relation between DE and anxiety, there has been less work on the co-occurrence of anxiety, depression, and DE. Examination of this relation with age-appropriate and validated measures could shed more light on the presentation of these symptoms in preadolescents, as well as potential gender differences in this age group.

Overall, the research has shown a high co-occurrence of anxiety, depression, and EDs. Internalizing symptoms may confer risk for the development of DE, as research demonstrates anxiety disorders often predate the onset of an ED and typically present at earlier developmental periods. As a result, DE behaviors may be employed as a coping strategy for anxiety and depression, potentially due to ineffective cognitive processes (discussed in 1.5). In addition to the overlapping psychological and clinical factors between eating pathology and internalizing disorders discussed in this section, there are also shared neurological features present.

1.3 Shared Neurocircuitry in EDs and Internalizing Disorders

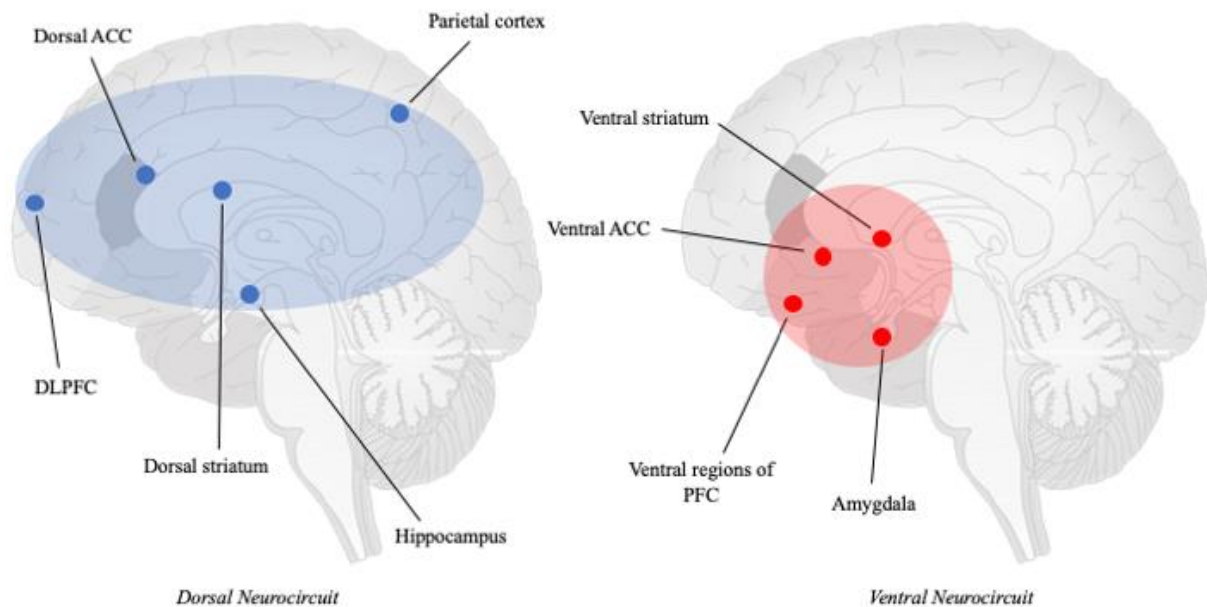
The role of neurobiology has been consistently demonstrated in internalizing disorders (e.g. Donofry et al., 2016; Etkin, 2010; Shin & Liberzon, 2010) and EDs (Stern & Bulik, 2020). A review by Kaye et al. (2010) highlighted evidence demonstrating individuals who had recovered from AN exhibited altered activity in limbic and cognitive neurocircuitry, including frontal, temporal, and parietal areas of the brain compared to healthy controls

(HCs; e.g., Råstam et al., 2007), suggesting this dysfunction is independent of current disorder or nutrition status. Disturbances in these neural networks have also been implicated in anxiety disorders (Etkin, 2010; Shin & Liberzon, 2010; Wright et al., 2003), depression (Donofry et al., 2016; Drevets, 2001), and other psychiatric disorders (McTeague et al., 2020).

These circuits can be specified further into ventral and dorsal neurocircuits (Phillips et al., 2003), presented in Figure 1.1. The ventral neurocircuit, which includes areas such as the amygdala, insula, and ventral regions of the striatum, ACC, and prefrontal cortex (PFC), is involved in the processing of emotionally salient information and the generation of affective responses, as well as automatic regulation of autonomic responses to emotional stimuli (Kaye et al., 2010). In contrast, the dorsal neurocircuit, including areas such as the hippocampus, dorsal regions of the ACC, dorsolateral prefrontal cortex (DLPFC), and parietal cortex, is thought to be involved in executive and cognitive processes such as planning, selective attention, and regulation of affective states (Phillips et al., 2003).

Figure 1.1.

The dorsal and ventral neurocircuits.



1.3.1 Prefrontal Cortex (PFC)

Several studies have implicated regions of the PFC in EDs. Greater activation in the medial PFC has been reported in women with an ED (AN and BN) in response to threatening and disgusting food stimuli (Uher et al., 2004) and negative body image words (Miyake, Okamoto, Onoda, Shirao, et al., 2010), likely as an attempt to regulate the emotional response to the emotive stimuli. These studies suggest the increased activation observed in the PFC could be driven by hyper-responsive emotional and fear networks in individuals with EDs (Friederich et al., 2013). The interplay between areas of the PFC, such as the orbitofrontal cortex (OFC) and ACC, during the processing of emotion information is important to consider, as these regions have also been implicated in internalizing disorders (Drevets, 2001; Saxena et al., 1998).

Another area of the PFC strongly implicated in EDs is the DLPFC (Von Hausswolff-Juhlin et al., 2015). Hyperactivity in the dorsal neurocircuit, including the DLPFC, has been identified in individuals with AN and is thought to compensate for impaired ventral neurocircuitry when regulating automatic affective responses to emotion stimuli, such as during emotion regulation (Kaye et al., 2010). In contrast, individuals with BN are reported to display hypoactivation of the lateral PFC (Uher et al., 2004), which could be driven by deficits in suppression of behavior, such as loss of control in eating. These regulatory processes are thought to be particularly important around the start of puberty as development of the DLPFC accelerates during this time (Huttenlocher & Dabholkar, 1997) and increased activity in the DLPFC and orbital regions could lead to excessive cognitive control and the excessive worry seen in individuals with AN (Kaye et al., 2010). Overall, these findings suggest impaired functioning in areas of the PFC may be important for the development of EDs, particularly in the context of cognitive control and emotion regulation.

Although this thesis will focus on the role of cortical regions, it is important to highlight that subcortical regions, such as the amygdala, have also been implicated in EDs (Frank, 2013; Miyake, Okamoto, Onoda, Kurosaki, et al., 2010; Miyake, Okamoto, Onoda, Shirao, et al., 2010) and internalizing disorders (Davis, 1992; Manji et al., 2001; Rauch et al., 2003; Sheline et al., 1998).

1.3.2 Anterior Cingulate Cortex (ACC)

The literature presented has demonstrated the key role of frontal cortical regions in the overlap between EDs and internalizing disorders; however, the anterior region of the

cingulate cortex is an area of particular interest in this thesis due to its connections with both the dorsal and ventral neurocircuits. The ACC is thought to play a role in emotional regulation, motivation, and cognitive control processes, such as set shifting and response monitoring (Løvstad et al., 2012; Stevens et al., 2011).

The ACC has also been implicated in the etiology of EDs. For example, elevated ACC activity has been reported in both individuals with a current diagnosis of AN and those who have recovered from AN, during visual presentations of food stimuli (Frank et al., 2004; Uher et al., 2003). This suggests increased activation of the ACC in response to food stimuli is a potential trait marker for AN. Importantly, in the study by Uher et al. (2003), explicit subjective evaluations of the food stimuli did not differ between HCs and recovered participants, suggesting the trait markers described are neurologically based. However, the cross-sectional nature of the study means it is not possible to distinguish a scar effect (resulting from the past disorder in recovered individuals) from true trait factors. Therefore, research conducted with individuals prior to the onset of an ED, who are at-risk of developing an ED, are needed to support these conclusions.

In addition to the potential functional deficits of the ACC reported, there have also been structural impairments identified in individuals with EDs. Reduced grey matter volume in the ACC has been reported in individuals with AN-R (Joos et al., 2010; Mühlau et al., 2007) and persists following weight restoration (Mühlau et al., 2007; Uher et al., 2003). However, more recent studies suggest a reversal of cortical thinning in individuals with AN after weight restoration (Bernardoni et al., 2016; King et al., 2015; Pfuhl et al., 2016), although, there may be additional scarring effects that cannot be excluded. These structural changes appear to be unique to restrictive EDs, as individuals with BN do not differ from HCs in grey matter volume of the ACC (Joos et al., 2010). Despite this, findings reported by Zhang et al. (2020) in adolescents suggest a reduction in grey matter volume of the ACC and OFC may be an early indicator of the development of binge eating and purging behavior in adolescents. Currently, there are no studies that have examined early neurological markers for the development of DE in preadolescents.

The ACC is also reported to be a key brain region implicated in depression and anxiety. A review by Lichenstein et al. (2016) highlighted aberrant ACC connectivity to be an important neural mechanism of risk for depression. Findings from studies examining both functional and structural connectivity of the ACC support an extensive disruption of this connectivity in adolescent depression, for both adolescents with a current illness and those at high risk for depression prior to the illness onset (Lichenstein et al., 2016). In anxiety,

reduced functional activity in the dorsal ACC has been reported in individuals with anxiety disorders compared to HCs, during emotion regulation and attentional control tasks (Blair et al., 2012). Structurally, a reduction in grey matter volume of the ACC in individuals with an anxiety disorder has also been reported (Shang et al., 2014).

1.4 Interim Conclusion

There is a clear gap in the current literature examining the development of DE, with very few studies directly investigating neurophysiological factors associated with DE before the onset of a diagnosed ED. The significant co-occurrence present between EDs and internalizing disorders warrants exploration in preadolescents to confirm whether there is a strong association between DE and internalizing symptoms in this younger age group. Research also suggests that internalizing disorders may share underlying neurobiology with EDs, and the ACC, which plays an important role in cognitive control, appears to be a key brain structure involved in EDs, anxiety, and depression.

1.5 Cognitive Control

Cognitive control refers to the ability to direct cognitive and attentional resources to achieve a predefined goal and adapt responses to maximize performance (Steward et al., 2018). The ACC has been proposed to play a role in cognitive control due to its connections with limbic and motor systems, as well as the PFC (Banfield et al., 2004; Gehring & Knight, 2000). It involves multiple processes, such as set shifting, inhibitory control, and monitoring, to enable adaptive decision making and emotion regulation (Steward et al., 2018). Notably, the National Institute of Mental Health's Research Domain Criteria (RDoC; Insel et al., 2010) initiative identified cognitive control as a construct within its cognitive systems domain, highlighting the importance of transdiagnostic research examining neural and behavioral measures of cognitive control within the context of mental health.

In typically-developing children, cognitive control processes are found to emerge early in childhood and continue to improve throughout adolescence, extending into adulthood (Zelazo & Müller, 2002). Developmental studies have demonstrated this behavioral improvement to be in line with greater activation in the PFC and ACC across childhood and adolescence (Davies et al., 2004; Moriguchi & Hiraki, 2011), highlighting the importance of these brain regions in cognitive control development. Emotional development, including skills like recognition and regulation of emotions, are important foundations for children's

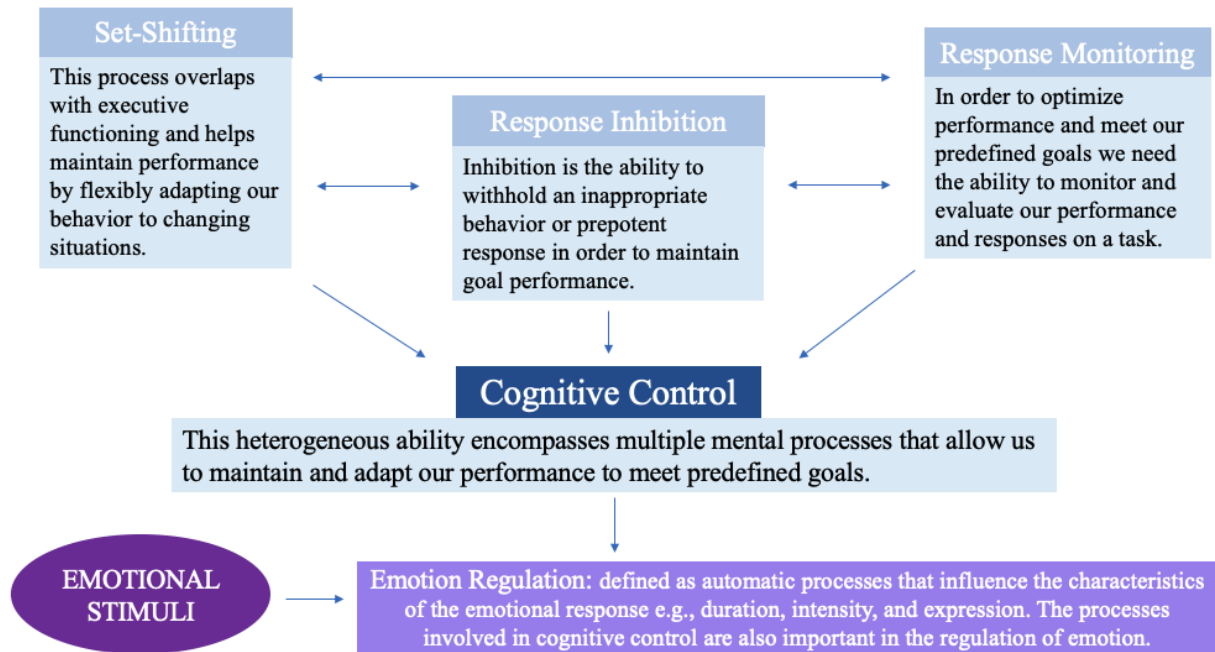
wellbeing and ability to form interpersonal relationships. These skills are also found to emerge early in development and improve across childhood (Batty & Taylor, 2006; Thümmeler et al., 2022), suggesting preadolescence is an important stage in the typical development of cognitive and emotional skills.

This thesis will focus on three of these cognitive control processes: response monitoring, response inhibition, and set shifting (Figure 1.2). I chose to focus on these three processes as there is a well-established link between set shifting and response inhibition in both EDs and internalizing disorders (described in 1.5.1 and 1.5.3), as well as research examining these processes in an emotional context. Response monitoring is also commonly studied within the context of anxiety and depression (described in section 1.5.2); however, considerably less research has focused on response monitoring in relation to eating pathology. In addition, response monitoring is commonly examined alongside response inhibition, reducing participant burden, a key consideration for developmental samples.

These cognitive control processes will also be examined in the context of emotion regulation. Event-related potentials (ERPs) generated in the electroencephalogram (EEG) will be used to capture neural correlates of these cognitive processes. EEG has high temporal resolution, enabling us to study brain activity that corresponds to specific cognitive processes as they unfold during a trial. In addition, EEG is also non-invasive, making it ideal for studying brain activity in children.

Figure 1.2.

Diagram defining the main processes involved in cognitive control that will be the focus of this thesis.



The rationale for the focus of this thesis is based on research demonstrating associations between impairments in cognitive control processes and higher levels of anxiety and depression (e.g. Hardin et al., 2007; McTeague et al., 2016; Snyder & Hankin, 2016), as well as research consistently reporting that internalizing symptoms predate the onset of EDs (e.g. Adambegan et al., 2012; Godart et al., 2000; Kaye et al., 2004; Keel et al., 2005; Raney et al., 2008). Therefore, shared cognitive control impairments across both DE and internalizing symptoms may account for the high co-occurrence of these symptoms, although this has yet to be thoroughly examined.

Impaired cognitive control could mean that emotion coping strategies are ineffective due to difficulties disinhibiting and shifting attention away from negative thoughts and emotions (Goschke, 2014). Furthermore, when impairments in cognitive control are accompanied by internalizing symptoms, they may reduce the individual's ability to cope with negative affect and lead to DE behaviors as a strategy for handling this distress (Dingemans et al., 2015). This idea that DE behaviors are employed as a maladaptive coping strategy is in line with research describing DE behaviors as a way of gaining a sense of control in stressful situations (Williams & Reid, 2012) and coping with emotions (Henderson

et al., 2019; Reid et al., 2020). In the affect regulation model of EDs, Stice (2001) proposed DE behaviors are used to manage and decrease negative affect, further supporting the use of DE as a maladaptive coping strategy.

1.5.1 Response Inhibition

Response inhibition, part of a collection of inhibitory control processes, is 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). This ability is required within an effective cognitive control system to enable adaptive goal-directed behaviors, alongside monitoring of performance and flexible shifting of behaviors to meet task demands (Ridderinkhof et al., 2004). Response inhibition can be differentiated into action restraint, measured using Go/NoGo tasks (Hare et al., 2005), and action cancellation, measured using stop-signal tasks (Ridderinkhof et al., 2004). On Go/NoGo tasks, the participant is required to perform speeded responses on Go trials, such as a button press when presented with a target stimulus, or withhold a response on NoGo trials, where a non-target is presented. Stop trials, on stop-signal tasks, also require a response to be withheld, but the stop signal is presented after the target stimulus. The main behavioral indices of response inhibition are the percentage of commission errors, which are responses during NoGo trials or stop trials, where the response should be withheld. In stop-signal tasks the stop-signal reaction time is also calculated as an estimation of the stopping process (Ridderinkhof et al., 2004).

The neural correlates of response inhibition include two stimulus-locked ERPs generated in the EEG, the N2 and P3, located over frontocentral sites. The N2 component is a negative deflection in amplitude around 200ms post-stimulus that is greater on NoGo trials compared to Go trials that reflects monitoring conflict between competing responses (e.g. Albert et al., 2013; Donkers & Van Boxtel, 2004; Hong et al., 2017). The P3 is a positive deflection following the N2 that is greater on NoGo trials compared to Go trials, associated with inhibitory processing and evaluation of the conflict stimulus (Bruin et al., 2001; Hong et al., 2017; Wessel, 2018). Source modelling has consistently identified the ACC as a key neural generator of both the N2 (Bekker et al., 2005) and P3 (Zhang et al., 2012) components and both reflect the neural correlates of response inhibition (e.g. Eimer, 1993; Falkenstein et al., 1999; Jodo & Kayama, 1992). The following sections will review response inhibition in both EDs and internalizing disorders.

1.5.1.1 Response Inhibition and DE/EDs

In EDs, behavioral measures of response inhibition have been shown to differ in adult and adolescent populations with AN compared to HCs (Steinglass et al., 2019; Svaldi et al., 2014; Wu et al., 2013). However, there has also been evidence of enhanced response inhibition (Weinbach et al., 2020), as well as comparable performance to HCs (Bartholdy et al., 2019), which may depend on the subtype of AN or the age of the population tested.

Overall, studies examining the association between DE and N2/P3 components are limited; however, several ERP studies conducted in children with obesity demonstrate attenuated P3 amplitudes (Reyes et al., 2015; Tascilar et al., 2011; Walk et al., 2020). For example, attenuated P3 amplitudes have been reported in children with obesity compared to HCs using the oddball paradigm (Tascilar et al., 2011) and when presented with Go trials during a Go/NoGo task (Reyes et al., 2015). Increased adiposity has also been related to attenuated P3 amplitudes during incongruent trials on a Flanker task (Walk et al., 2020). However, no significant effects were reported for N2 amplitudes (Reyes et al., 2015; Walk et al., 2020). In addition, adults with AN are reported to display attenuated P3 amplitudes during Stop trials in a stop-signal task compared to HCs, although the N2 component was not significantly different (Yue et al., 2020). Taken together, these findings suggest individuals with DE behaviors may show comparable conflict detection abilities to HCs based on the N2 findings. However, attenuated P3 amplitudes may reflect deficits in evaluation of and adjusting behavior to that conflict. However, this is based on only a few studies.

1.5.1.2 Response Inhibition and Internalizing Disorders

In adults, response inhibition impairments are a key feature of anxiety and depression (Årdal & Hammar, 2011; Grillon et al., 2017; Hammar et al., 2010; Lee et al., 2012; Li et al., 2021; Snyder, 2013). In anxiety, excessive response inhibition is thought to be promoted by overactivation of the behavioral inhibition system (Kagan et al., 1987). Individuals with clinical levels of anxiety and HCs with elevated anxiety symptoms induced by a threat condition (Grillon et al., 2017) are found to display excessive response inhibition. Excessive inhibition during increased threat may be an adaptive cognitive function; however, elevated response inhibition during both threatening and non-threatening situations is maladaptive and a hallmark of anxiety.

In contrast to adults, children and adolescents with internalizing symptomatology typically show no significant differences in behavioral markers of response inhibition when

compared to HCs (Brunnekreef et al., 2007; Diler et al., 2014; Hum et al., 2013a; Pan et al., 2011). Associations between anxiety and response inhibition performance in children from both clinical (Oosterlaan et al., 1998) and community (Iijima et al., 2019) samples are also reported to be weaker than associations reported in adults. Importantly, most research examining the relation between response inhibition and depressive symptoms in children and adolescents focuses on emotional Go/NoGo tasks (Han et al., 2012; Ho et al., 2018; Zhang et al., 2016), rather than non-emotional Go/NoGo tasks. This is an area of research that this thesis will address.

The literature examining N2 and P3 amplitudes in individuals with anxiety is generally mixed. P3 amplitudes have been reported to be reduced (Bechor et al., 2019; Éismont et al., 2009; Wauthia et al., 2022; Xu et al., 2014) as well as enhanced (Sehlmeyer et al., 2010; Xia et al., 2020), compared to HCs. Attenuated P3 amplitudes have been found across a range of tasks, such as during the presentation of emotional faces in a dot-probe task (Bechor et al., 2019), on invalid trials in a spatial cueing task (Wauthia et al., 2022), during an acoustic Go/NoGo task (Éismont et al., 2009), as well as a passive viewing oddball paradigm task (Xu et al., 2014). However, enhanced P3 amplitudes have been specifically found on NoGo trials during Go/NoGo tasks (Sehlmeyer et al., 2010; Xia et al., 2020). Individuals with anxiety have shown elevated N2 amplitudes during proactive control trials in a continuous performance task (Valadez et al., 2021), as well as incompatible trials on an emotional flanker task (Yu et al., 2018), which are thought to represent increased conflict detection and elevated engagement in cognitive control processes (Dennis & Chen, 2009). Greater N2 amplitudes in anxiety appear to be more consistent when responding to emotionally salient stimuli, while some studies using non-emotional stimuli do not report this enhancement (Baving et al., 2004; Larson et al., 2013; Voegler et al., 2018).

Neuroimaging studies examining response inhibition in both adults (Katz et al., 2010; Korgaonkar et al., 2013; Langenecker et al., 2007; Wagner et al., 2006) and adolescents (Harvey et al., 2005; Pan et al., 2011) with depression have reported hyperactivation of the ACC compared to HCs. N2 amplitudes are typically comparable in individuals with depression compared to HCs (Palmwood et al., 2017; Ruchow et al., 2008). While P3 amplitude reduction has been reported in individuals with depression (Nan et al., 2018) as well as healthy individuals with a family history of depression (Houston et al., 2003) during presentations of targets in complex oddball tasks.

In summary, neural markers of response inhibition indicate impairments across internalizing disorders in both children and adults. However, behavioral markers of impaired

response inhibition appear to be more prominent in adults with internalizing disorders, compared to children. This suggests variations in task performance may occur in later stages of cognitive development, with neural differences occurring before cognitive differences emerge. The literature highlights the examination of non-emotional stimuli as a key area for future examination in anxiety and depression.

1.5.2 Response Monitoring

Response monitoring refers to the ability to evaluate and adjust one's own behavior in order to meet a predefined goal and optimize performance (Thakkar et al., 2008). This can involve behavior adjustments based on task demands and detection of errors (Hajcak et al., 2005). Behavioral measures of response monitoring are typically in the form of post-error slowing (Endrass & Ullsperger, 2014), as well as overall performance outcomes, such as accuracy and reaction time (RT). Response monitoring is typically assessed using conflict tasks in conjunction with response inhibition. For example, Go/NoGo and Stop-signal tasks (see 1.5.1) have been previously used to examine response monitoring in AN (Suttkus et al., 2021; Wierenga et al., 2014). The Flanker task (Eriksen & Eriksen, 1974) is another conflict task that requires participants to respond to the target stimuli, typically presented as the middle arrow or letter, on congruent (>>>>>) or incongruent (>><<>>) conditions. Lastly, response monitoring has also been assessed using the probabilistic reversal learning task (Hampton et al., 2006). This is not a conflict task and instead requires participants to learn and respond to stimulus-reward contingencies, monitoring and adjusting their behavior while receiving probabilistic feedback.

Most research focuses on neural measures of response monitoring in the form of response-locked ERPs generated in the EEG (Falkenstein et al., 2000), a summary of which is provided in Table 1.2.

Table 1.2.

Summary of response-locked ERPs related to response monitoring.

ERP	Characterization of potential	Functional significance
ERN	<ul style="list-style-type: none">• A negative deflection in amplitude which peaks in voltage around 50 to 150ms following an error response and is located around midline fronto-central recording sites (Hajcak & Simons, 2002).• fMRI, EEG source localization, and brain lesion research suggests the ERN is generated by the ACC (O'Connell et al., 2007; Stemmer et al., 2004; van Veen & Carter, 2002).	<ul style="list-style-type: none">• Initially thought to reflect an error detection system (Falkenstein et al., 2000; Gehring et al., 1993).• Alternatively, associated with response conflict and monitoring e.g., overriding a prepotent response on a task in order to answer correctly (Botvinick et al., 2004), or identifying situations with the potential to produce errors, such as those high in response conflict (van Veen & Carter, 2002).• Proposed to function as a reward prediction error signal used by the ACC to enable the tracking of ongoing events and indicate when the events are worse than anticipated, such as when a response commission error is performed (Holroyd & Coles, 2002; Ridderinkhof et al., 2004).
CRN	<ul style="list-style-type: none">• A smaller peak of negative amplitude compared to the ERN, produced as a result of correct responses (Ford, 1999).	<ul style="list-style-type: none">• Hypothesized to reflect the same process as the ERN, but modulated by response accuracy (Hoffmann & Falkenstein, 2010).

- ACC is a common generator of the CRN and ERN (Roger et al., 2010).
- Pe
- A positive peak in amplitude occurring around 200-500ms after an incorrect response (Falkenstein et al., 1999).
 - Generated by the ACC, with some evidence suggesting the rostral ACC (Herrmann et al., 2004).
 - Mixed evidence, but overall thought to be involved in error awareness and detection to modify behavior and optimize performance on a motivationally significant task (Davies et al., 2001; Endrass et al., 2007; Falkenstein et al., 2000; Ridderinkhof et al., 2009).

Note. ACC: anterior cingulate cortex, CRN: correct-related negativity, EEG: electroencephalography, ERN: error-related negativity, ERP: event-related potential, fMRI: functional magnetic resonance imaging, Pe: error positivity.

1.5.2.1 Response Monitoring and DE/EDs

To-date, there are currently no studies examining response monitoring in relation to DE and EDs in children, with the majority of research capturing older adolescent (Geisler et al., 2017; Wierenga et al., 2014) or young adult females (Pieters et al., 2007; Ritschel et al., 2017; Suttikus et al., 2021) with AN. Behaviorally, comparisons in response monitoring between individuals with EDs and HCs are mixed, with some reporting no differences (Geisler et al., 2017; Suttikus et al., 2021; Wierenga et al., 2014), one study reporting poorer (Ritschel et al., 2017) and one reported better (Pieters et al., 2007) performance in individuals with EDs compared to HCs.

Findings from neuroimaging studies examining response monitoring in EDs are also mixed. For example, Pieters et al. (2007) found adults with AN to display attenuated ERN amplitudes compared to HCs during a Flanker task. An explanation for these attenuated ERN amplitudes may relate to hypofunction of the ACC, observed in individuals with AN (Mühlau et al., 2007). fMRI studies have produced conflicting findings, with either elevated ACC activity found in individuals with AN compared to HCs (Geisler et al., 2017), or no differences between HCs and individuals with a current or previous AN diagnosis (Ritschel et al., 2017; Suttikus et al., 2021; Wierenga et al., 2014). It is important to highlight the use of alternative tasks in these studies, such as a probabilistic reversal learning task (Geisler et al., 2017; Ritschel et al., 2017), which may require different types of error processing compared to response conflict tasks. Based on the limited evidence to-date, attenuation of the ERN appears to be present in AN, but this may be specific to response-conflict tasks.

The ED literature overall is inconsistent and highlights the current lack of studies examining response monitoring in eating psychopathology. As ACC function may be yoked to malnutrition in AN (Mühlau et al., 2007), further studies are needed to disentangle disorder-related sequelae and scar effects from trait factors that may confer risk for developing an ED.

1.5.2.2 Response Monitoring and Internalizing Disorders

Behavioral markers of response monitoring, such as post-error slowing and performance accuracy, are typically comparable in children and adolescents with internalizing disorders compared to HCs (Carrasco et al., 2013; Hum et al., 2013a; Ladouceur et al., 2006; Ladouceur et al., 2012). However, relative to HCs, adults with depression are found to exhibit difficulties adjusting their behavior after errors, displaying significant

decreases in performance after committing an error (Douglas et al., 2009; Pizzagalli et al., 2006). In contrast, anxiety disorders in adults are largely unrelated to post-error slowing (see Rueppel et al., 2022 for a review of the literature).

In contrast to behavioral findings, developmental studies examining neural correlates have reported the ERN to be a biomarker for anxiety in children and adolescents (Meyer, 2017). The relation between the ERN amplitude and anxiety has also been found to change as a function of age (Meyer et al., 2012). Specifically, in older children anxiety is associated with enhanced ERN amplitudes, whereas higher levels of anxiety in younger children are associated with attenuated ERN amplitudes (Meyer et al., 2012). Enhanced ERN amplitudes have also been found in adults with health anxiety (Riesel et al., 2017), generalized anxiety disorder (Weinberg et al., 2012; Weinberg et al., 2010), and elevated anxiety symptoms (Hajcak et al., 2003). This highlights an impairment in response monitoring that is a common feature across many anxiety disorders and developmental stages, at both clinical and subclinical levels.

Enhanced ERN amplitudes in anxiety may reflect increased threat sensitivity. For example, errors can be aversive, signifying a threat to the individual. Therefore, worry could potentiate the ERN through increasing the threat value associated with errors (Proudfit et al., 2013; Seow et al., 2020). In addition, forms of dispositional anxiety (Fox et al., 2005; Fox et al., 2008; Lahat et al., 2014) may play a role through increased sensitivity to external cues relating to punishment and threat, as well as excessive responses to potential or uncertain threats (Proudfit et al., 2013). Taken together, this suggests that the enhanced ERN amplitude in anxiety reflects an increased threat sensitivity.

Children and adolescents with depression show significantly attenuated ERN amplitudes compared to HCs (Ladouceur et al., 2012; Weinberg, Meyer, et al., 2016). This finding has even been extended to children at-risk for depression (Meyer et al., 2018), suggesting attenuated ERN amplitudes may be a trait factor of depression. However, attenuated ERN amplitudes in adults have been linked to specific subtypes of depression, such as melancholic depression (Schrijvers et al., 2008; Weinberg, Kotov, et al., 2015; Weinberg, Liu, et al., 2016). There may be a more nuanced association between the ERN and depression in adults, rather than a broader response monitoring deficit.

In summary, impaired response monitoring is a feature of internalizing disorders in both children and adults. Specifically, this is in the form of enhanced ERN amplitudes in anxiety and attenuated ERN amplitudes in depression. Although there is some behavioral evidence for impaired response monitoring in adults with depression, the majority of research

indicates intact behavioral markers in anxiety and depression, especially in children. Therefore, a complete account of response monitoring needs to consider both the behavioral and neural profile, which may not align.

1.5.3 Set Shifting

Set shifting is an executive function that involves the ability to flexibly shift attention between multiple tasks, behaviors or mental states (Miyake et al., 2000). Set shifting often requires integration of multiple cognitive control processes during tasks, such as response inhibition and monitoring (Gruner & Pittenger, 2017; Lindner et al., 2014). Set shifting forms a core part of a broader construct, known as cognitive flexibility, which also includes processes such as task switching and reversal learning (Gruner & Pittenger, 2017).

Behavioral tasks are often used to measure set shifting ability, and one of the most commonly employed measures is the Trail Making Task (TMT; Delis et al., 2001; Reitan, 1955). The TMT requires the individual to shift between alternating numeric and alphabetical sequences in order (e.g., 1-A-2-B-3-C...). Another common measure of set shifting ability is the Wisconsin Card Sorting Test (WCST; Berg, 1948). This task requires the participant to match stimulus cards according to one of four category cards. The rule (color, shape, or number) for sorting these cards into categories changes throughout the task and the participant must adjust their performance accordingly. Performance on these tasks includes outcome measures such as response errors, time to complete task on the TMT (after controlling for baseline motor speed), and categories completed on the WCST (Roberts et al., 2010).

1.5.3.1 Set Shifting and DE/EDs

Impairments in set shifting have been observed in individuals both in the acute phase of AN (Tchanturia et al., 2004; Tchanturia et al., 2002) and in recovery (Fuglset, 2019; King et al., 2019; Roberts et al., 2010), in unaffected family members (Galimberti et al., 2013; Holliday et al., 2005; Tenconi et al., 2010), as well as individuals with BN (Brand et al., 2007; Roberts et al., 2010; Tchanturia et al., 2004). These findings suggest set shifting difficulties could be a trait factor and potential endophenotype of EDs. However, set shifting may not be a risk factor for EDs, as studies in children and adolescents with AN report no significant differences compared to HCs (Bühren et al., 2012; Herbrich et al., 2018; Lang et al., 2014; Rößner et al., 2017). Although some studies have noted a pattern in their data

consistent with poorer set shifting in children with AN compared to HCs (e.g. Lang et al., 2014), these small differences could be interpreted as more subtle cognitive disturbances that are exacerbated by starvation and function to maintain the longer duration of illness more commonly observed in adults with AN (Lang et al., 2014).

Impairments in set shifting have been proposed to arise due to a tendency to maintain high levels of cognitive control and are thought to be related to a more rigid, rule-bound and less adaptive cognitive style, typically observed in AN (King et al., 2019; Tchanturia et al., 2004). This cognitive style could also contribute to the maintenance of AN through more rigid and compulsive behaviors resulting in chronic and severe forms of the illness (Harrison et al., 2011).

ERPs are not commonly studied in relation to set shifting performance in EDs, with most research focusing on functional brain activity (e.g., Castro-Fornieles et al., 2019; Sato et al., 2013; Zastrow et al., 2009). As this thesis is focused on time-locked neural activity only (i.e., ERPs), this neuroimaging evidence will not be further commented on in this section.

1.5.3.2 Set Shifting and Internalizing Disorders

Impaired set shifting is reported in generalized anxiety disorder (Tempesta et al., 2013) and can increase risk for its development (Zainal & Newman, 2018; Zhang et al., 2015). The attentional control theory of anxiety proposes that elevated levels of anxiety compromise performance of attentional control functions, such as set shifting, and this results in increased attention to threatening environmental cues (Eysenck et al., 2007). This is supported by an association between rumination, a key feature of generalized anxiety disorder, and set shifting difficulties. Therefore, difficulties in shifting between cognitions may lead to fixation on recurrent negative thoughts (Yano et al., 2016).

Set shifting impairments in adults with depression are well established (Austin et al., 2001; Michopoulos et al., 2006; Stange et al., 2017), with impairments reported in individuals with current and remitted depression symptoms (Rock et al., 2014), as well as unaffected family members (Liu et al., 2021). Although less work has examined set shifting performance in children and adolescents with depression, investigations in offspring at-risk for depression have produced interesting findings. Both Klimes-Dougan et al. (2006) and Micco et al. (2009) found the presence of a parental diagnosis of MDD was not associated with set shifting impairments. However, the presence of current MDD symptoms in at-risk children was associated with set shifting impairments (Micco et al., 2009). These findings argue

against set shifting impairments being trait dependent, instead suggesting this impairment is state dependent in children and adolescence.

As highlighted in anxiety, set shifting is thought to play a role in depressive symptomatology through elevations in rumination. Specifically, impairments in set shifting lead to difficulties disengaging from negative cognitions, fueling depressive symptomatology (Vergara-Lopez et al., 2016). This is captured in the ‘content meets process’ model, which posits that individuals at increased risk for depression have limited cognitive control, leading to difficulties switching attentional focus from dominant cognitions (negative) to non-dominant cognitions (Maccoon & Newman, 2006). This pattern is consistent with food-related cognitions in individuals with EDs suggesting a shared underlying mechanism for both DE and internalizing disorders that I explore in this thesis. Many studies have also focused on these difficulties in relation to emotional rather than neutral stimuli and will be covered in section 1.6.1.

1.6 Emotion Regulation

Emotion regulation and cognitive control are thought to overlap conceptually, with similar regulatory processes and goal directed behavior employed in each (Pruessner et al., 2020). Emotion regulation is often defined as a combination of processes that govern the expression, timing, and intensity of emotional experiences in order to serve a larger goal (Gross & Thompson, 2007). Therefore, both cognitive control and emotion regulation require the use of top-down control when responding to issues that are identified as potential disruptions to our goals (Compton et al., 2011). Neuroimaging studies provide support for this overlap, with involvement of the frontoparietal network (Braunstein et al., 2017; Niendam et al., 2012), ACC, and DLPFC in both cognitive control (e.g. Holroyd & Coles, 2002; Ridderinkhof et al., 2004) and emotion regulation (Ochsner et al., 2004; Phan et al., 2005). Therefore, given the literature already presented describing impairments in cognitive control processes in individuals with internalizing disorders and EDs (for a summary, see Table 1.3), it may also be the case that these processes are deficient within the context of emotion.

Table 1.3.

Summary of research on cognitive control performance in internalizing disorders and eating disorders (EDs)/Disordered eating (DE).

		Anxiety	Depression	EDs/DE
Response Monitoring (RM)	Behavioral findings	RM typically comparable in individuals with anxiety disorders and HCs (Carrasco et al., 2013; Hum et al., 2013a; Ladouceur et al., 2006) and not significantly related to internalizing symptoms (Weinberg, Meyer, et al., 2016).	Adults display a greater number of errors directly following an error, relative to HCs (Douglas et al., 2009). Children and adolescents do not differ from HCs (Ladouceur et al., 2012).	No significant differences between adults with EDs and HCs (Geisler et al., 2017; Suttkus et al., 2021; Wierenga et al., 2014).
	Neural findings	Elevated ERN amplitude reported in adults (Riesel et al., 2017; Weinberg et al., 2012; Weinberg et al., 2010) and children/adolescents (Meyer, 2017; Meyer et al., 2012).	Attenuated ERN amplitudes reported in children/adolescents (Ladouceur et al., 2012; Meyer et al., 2018) and some studies with adults (Weinberg, Kotov, et al., 2015; Weinberg, Liu, et al., 2016).	Adults with AN display attenuated ERN amplitudes compared to HCs (Pieters et al., 2007).
Response Inhibition (RI)	Behavioral findings	Adults display elevated RI on behavioral tasks (Grillon et al., 2017).	Impairments reported in adults in both longitudinal studies (Årdal & Hammar, 2011; Hammar et al.,	Impairments in individuals with binge eating and/or purging symptoms (Steinglass et al.,

	Children/adolescents typically show no significant differences compared to HCs (Brunnekreef et al., 2007; Hum et al., 2013a).	2010) and meta-analyses (Lee et al., 2012; Snyder, 2013). Adolescents with depressive symptoms do not significantly differ when compared to HCs (Brunnekreef et al., 2007; Diler et al., 2014; Pan et al., 2011).	2019; Svaldi et al., 2014; Wu et al., 2013). However, no significant differences in RI between adolescents with binge/purge behaviors and those without (Bartholdy et al., 2019). Enhanced RI in adolescents with AN-R compared to HCs (Weinbach et al., 2020).
Neural findings	P3 amplitudes reduced (Bechor et al., 2019; Éismont et al., 2009; Wauthia et al., 2022; Xu et al., 2014) as well as enhanced (Sehlmeyer et al., 2010; Xia et al., 2020), compared to HCs. Elevated N2 amplitudes compared to HCs (Hoyniak & Petersen, 2019; Valadez et al., 2021; Yu et al., 2018) Studies using non-emotional stimuli do not report this N2 enhancement (Baving et al., 2004; Larson et al., 2013; Voegler et al., 2018)	P3 amplitude reduction has been reported in individuals with depression (Nan et al., 2018) as well as healthy individuals with a family history of depression (Houston et al., 2003).	Adults with AN display smaller P3 amplitudes compared to HCs, but no significant differences found for N2 amplitudes (Yue et al., 2020). Children with obesity present with attenuated P3 amplitudes (Reyes et al., 2015; Tascilar et al., 2011; Walk et al., 2020).

Set Shifting (SS)	<p>Adults display impaired SS compared to HCs (Tempesta et al., 2013) and SS impairments increase risk for developing generalized anxiety disorder (Zainal & Newman, 2018; Zhang et al., 2015). SS difficulties reported in children with heightened behavioral inhibition (Buzzell et al., 2021; Henderson et al., 2015; Henderson & Wilson, 2017).</p>	<p>Impairments reported in adults with current depression (Austin et al., 2001; Michopoulos et al., 2006; Stange et al., 2017) as well as individuals with remitted symptoms (Rock et al., 2014) and unaffected family members (Liu et al., 2021). Offspring of parents with depression do not display SS impairments (Klimes-Dougan et al., 2006; Micco et al., 2009). But, at-risk children with depression symptoms do display SS impairments (Micco et al., 2009).</p>	<p>Impairments reported across the spectrum of EDs in adults (Aloi et al., 2015; Duchesne et al., 2010; Wu et al., 2014), including in AN (Tchanturia et al., 2004; Tchanturia et al., 2002) and BN (Brand et al., 2007; Roberts et al., 2010; Tchanturia et al., 2004). Findings are less consistent in children/adolescents, with many reporting no differences from HCs (Bühren et al., 2012; Herbrich et al., 2018; Lang et al., 2014; Rößner et al., 2017).</p>
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Note. ACC: anterior cingulate cortex, AN: Anorexia nervosa, AN-BP: Anorexia Nervosa-binge/purge subtype, AN-R: Anorexia Nervosa-restricting subtype, BN: Bulimia Nervosa, CRN: Correct-related negativity, ERN: Error-related negativity, HCs: healthy controls, N2: N200, Pe: error positivity, P3: P300.

Response monitoring may be particularly relevant for emotion regulation as individual differences in detection and behavioral adaptation to errors are associated with individual differences in emotion regulation, such as response to daily stressors (Compton et al., 2008). Neural correlates of response monitoring provide further support, as more proficient differentiation of correct and incorrect responses in the ERN and Pe amplitudes is found to predict better emotion regulation, such as lower levels of daily stress reactivity (Compton et al., 2011). These findings suggest efficient monitoring of performance represents a global cognitive-emotional control capacity that extends to regulating everyday life stressors.

Response inhibition is thought to play a role in emotion regulation through enabling individuals to override their default emotional expressions in order to maintain goal-directed behavior (Pruessner et al., 2020), such as inhibiting a negative emotional response in a socially inappropriate context. Furthermore, neuroimaging studies have reported emotional context to have a modulatory effect on response inhibition (Albert et al., 2010; Shafritz et al., 2006). This moderation appears to change over development, starting with enhanced and more rapid N2 and P3 responses for angry faces compared to happy faces in early childhood (Lewis et al., 2007). In adults, increased allocation of neural resources is instead required for inhibition during positively valenced contexts compared to negative contexts (Albert et al., 2010). Frontal midline ERPs have been reported to steadily decrease in amplitude across time in these emotion-stimuli tasks and become more localized to frontal cortical areas (Lewis et al., 2006), mapping behavioral improvements in response inhibition onto the timescale of PFC maturation (Casey et al., 2000).

Set shifting is thought to be relevant for emotion regulation due to the need to adjust emotional responses to changing emotional stimuli and contexts (Compton et al., 2011). This includes shifting attention away from negative stimuli in order to reduce the experience of negative emotions (Mather & Carstensen, 2005). This is seen as early as infancy, where emotional reactivity is decreased through the infant shifting their gaze away from an emotion-inducing stimulus (Rothbart & Sheese, 2007). Some researchers suggest this shifting away from negative stimuli could be maladaptive when used consistently as a way to regulate anxiety, but adaptive when used to regulate emotional response during performance towards a desired goal (Johnson, 2009).

The literature presented here demonstrates an important overlap between processes involved in cognitive control, such as response monitoring, response inhibition and set shifting, and the cognitive processes engaged during emotion regulation. The following

sections will review the research describing cognitive control of emotion within both EDs and internalizing disorders.

1.6.1 Emotion Regulation and DE/EDs

Emotion regulation is a key factor involved in the development and maintenance of EDs (Harrison, Sullivan, et al., 2010; Henderson et al., 2021; Lavender et al., 2015). Studies exploring emotion regulation deficits in individuals with a diagnosed ED have reported significantly higher levels of experienced emotion intensity, less acceptance and awareness of emotions, limited expression of emotions, increased rumination about these emotions, as well as more self-reported emotion regulation problems, when compared to HCs (Boscoe et al., 2021; Fox et al., 2013; Svaldi et al., 2012). These difficulties are also present in children and early adolescents with DE (McLaughlin et al., 2011; Sim & Zeman, 2006). However, research examining the cognitive processes underlying emotion regulation are limited.

Emotion regulation is thought to be interconnected with other processes, such as emotion recognition and attention, to enable successful social emotional functioning (Ochsner, 2008). For example, emotion recognition difficulties have been associated with impairments in emotion regulation in women with AN (Harrison et al., 2009). These emotion recognition difficulties are well-established in EDs (Harrison et al., 2009; Harrison, Sullivan, et al., 2010; Pollatos et al., 2008; Zonnevijlle-Bendek et al., 2002), including individuals who have recovered from AN (Harrison, Tchanturia, et al., 2010) and those with increased DE (Ridout et al., 2010; Ridout et al., 2012; Wallis et al., 2018). Attentional biases to social affective stimuli, specifically negative faces, are also seen in both individuals with a current ED and those who have recovered, when compared to HCs (Harrison, Sullivan, et al., 2010; Harrison, Tchanturia, et al., 2010). Meta-analyses and systematic reviews have identified attentional biases across a range of stimuli in EDs, such as food, body, and weight stimuli (Kerr-Gaffney et al., 2019; Stott et al., 2021). These findings suggest individuals with EDs may experience difficulties across a range of emotion processing domains, including emotion recognition and attentional biases, and these may contribute to difficulties in emotion regulation.

Research examining emotion regulation in ED samples has primarily focused on self-report measures, meaning there is a relative shortage of neurobiological or experimental evidence to support these findings. The few studies that have explored the cognitive processes underlying emotion regulation in ED have reported attenuated early ERP

components to all emotional faces (Hatch et al., 2010; Sfarlea et al., 2016), indicating dysfunctions in automatic and perceptual processing. Similarly, increased N2 amplitudes, indicative of enhanced conflict monitoring, in response to all emotion categories have been reported in individuals with AN compared to HCs (Pollatos et al., 2008). There is evidence that individuals with EDs may also present with dysfunctions in later and more elaborate processing. The P3 is found to be smaller during emotional processing in individuals with AN (Hatch et al., 2010; Pollatos et al., 2008) in the absence of behavioral differences (Hatch et al., 2010; Sfarlea et al., 2016). Taken together, the evidence suggests that the adults in these samples have developed compensatory mechanisms to overcome deficits in emotion regulation and maintain task performance. To-date, there has not been an investigation of neural correlates of emotion regulation in children presenting with DE, who are less likely to have fully developed compensatory networks.

1.6.2 Emotion Regulation and Internalizing Disorders

Emotion regulation is as an important factor in the development of anxiety disorders. For example, individuals with anxiety selectively shift their attention toward threatening or negative stimuli (Bar-Haim et al., 2007; Mathews & MacLeod, 2005; Williams et al., 2014) and employ increased cognitive effort to reorient attention and regulate emotions after processing negative stimuli (Fox et al., 2001; Hallion et al., 2019; MacNamara et al., 2017; Telzer et al., 2008).

Response inhibition impairments, specific to negative emotions, have also been highlighted in individuals with anxiety. For example, children and adults diagnosed with an anxiety disorder (Ladouceur et al., 2006; Waters & Valvoi, 2009), as well as adults with elevated anxiety symptoms (Pacheco-Unguetti et al., 2012), display response inhibition difficulties when presented with negative emotional faces. However, enhanced neural correlates of response inhibition are found during presentations of *both* positive and negative stimuli compared to HCs (Hum et al., 2013a; Lewis et al., 2008). Interestingly, these atypical neural markers are not accompanied by behavioral task impairments in children with anxiety (Hum et al., 2013a, 2013b; Lewis et al., 2008; Waters & Valvoi, 2009).

Individuals with depression do not appear to display consistent differences from HCs in response inhibition performance (Grunewald et al., 2015; Han et al., 2012; Trinkl et al., 2015). However, both individuals with depression symptoms and unaffected first-degree relatives of individuals with MDD present with increased activity in neural regions associated

with cognitive control when presented with negative stimuli during an inhibition task (Kaiser et al., 2014; Lisiecka et al., 2012), suggesting this neural activity may be a trait marker of depression. Findings for N2 and P3 amplitudes however, are mixed, with reduced NoGo N2 and NoGo P3 amplitudes for positive emotions in individuals with depression (Camfield et al., 2018). Zhang et al. (2016) reported larger P3 amplitudes to positive faces were associated with increased anhedonia symptoms and larger P3 amplitudes to negative faces were associated with lower depressive symptoms. These findings suggest that within the relation between depression and emotion regulation, there may be specific symptom profiles that lead to more nuanced emotion regulation impairments.

In summary, these findings suggest individuals with internalizing symptoms present with impairments in cognitive control during regulation of emotions, as well as an attentional bias towards negatively valenced stimuli. These cognitive control impairments could underlie the presence of rumination in internalizing disorders, which may function as a maladaptive emotion regulation strategy, maintaining negative affect.

1.7 Summary

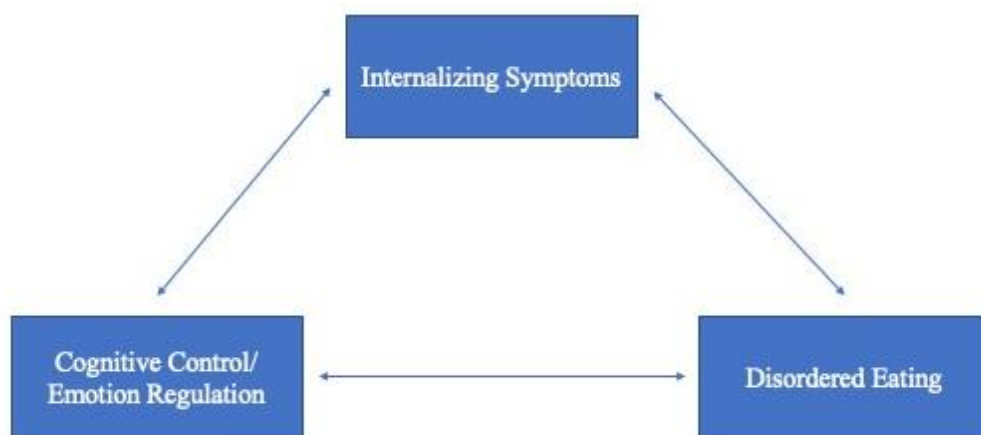
This chapter presents evidence of high co-occurrence between EDs and internalizing disorders, which could relate to overlapping impairments in cognitive control processes, such as response monitoring, response inhibition, and set shifting. Less is known about cognitive control in individuals who present with DE, especially earlier in development, and investigations in these samples can help identify antecedents and capture risk trajectories of eating pathology. Research examining the co-occurrence of DE and internalizing symptoms in preadolescence, prior to the typical onset of an ED, is an important avenue for investigation given DE and internalizing symptoms are found to emerge in childhood (Graber et al., 1994; Sands et al., 1997; Solmi et al., 2021). Identifying correlates of these symptoms at this early stage could help us discover viable prevention targets and increase our understanding of the etiology of EDs, anxiety, and depression. In addition, by studying these processes using a transdiagnostic approach, we can explore the overlap between DE and internalizing symptoms and examine more specific and nuanced relations between symptoms and cognitive control difficulties.

I argue the co-occurrence of internalizing symptoms and DE is driven in part by shared impairments in cognitive control. This may be due to DE functioning as a maladaptive coping strategy for dealing with feelings of distress and high levels of anxiety, as a result of

ineffective cognitive control processes. Although previous models, such as the affect regulation model (Stice, 2001), have proposed DE behaviors to function as a maladaptive strategy for managing negative affect, the underlying cognitive control processes have not yet been examined in the context of co-occurring DE and internalizing disorders. Specifically, research has not thoroughly examined whether cognitive control impairments are associated with DE independently of internalizing symptoms, or whether internalizing symptoms may mediate the relation between cognitive control/emotion regulation and DE (Figure 1.3). Internalizing symptoms are proposed as the mediator in this model due to research suggesting cognitive control impairments may function as a risk factor for internalizing symptoms (Houston et al., 2003; Liu et al., 2021; Meyer, 2017; Meyer et al., 2018; Zainal & Newman, 2018; Zhang et al., 2015) and these internalizing symptoms are often found to predate the onset of an ED (e.g. Adambegan et al., 2012; Godart et al., 2000; Kaye et al., 2004; Keel et al., 2005; Raney et al., 2008). Importantly, the relations between these constructs may be bidirectional, serving to maintain and facilitate these difficulties.

Figure 1.3.

Diagram presenting the model that will be examined in this thesis.



The model examined in this thesis is highly novel in its exploration of cognitive control processes in emotional and non-emotional contexts, DE, and internalizing symptoms. As previously described in this chapter, relations have been established between DE and internalizing symptoms in adults and adolescents, although less is known about this relation in preadolescence. In addition, cognitive control processes (in emotional and non-emotional

contexts) have been linked to EDs and internalizing disorders, but the examination of relations between cognitive control, DE, and internalizing symptoms in preadolescence has not yet been studied, especially in both boys and girls. Lastly, the use of both behavioral measures and neuroimaging further highlights the novelty of this research, as to the best of my knowledge, this is the first study to investigate the relations between behavioral and neural markers of cognitive control, DE, and internalizing symptoms in preadolescence.

1.8 Aims of the Thesis

The aims of this thesis are to investigate the associations between DE, internalizing symptoms, and cognitive control in a sample of typically developing preadolescents, using a combination of neural, behavioral, and questionnaire measures. Specifically, I will examine whether cognitive control impairments are associated with DE and internalizing symptoms in preadolescence, and whether internalizing symptoms mediate associations between cognitive control impairments and DE. Response inhibition and response monitoring will also be explored in an emotional context to investigate emotion regulation processes.

The main research questions that the thesis aims to answer are:

- 1) Are DE and internalizing symptoms associated in preadolescence?
- 2) Are components of cognitive control, such as set shifting, response inhibition and response monitoring, associated with both DE and internalizing symptoms in preadolescence?
- 3) Do internalizing symptoms mediate the relation between DE and cognitive control?
- 4) Do internalizing symptoms mediate the relation between DE and emotion regulation?

The specific aims of each of the studies in this thesis are presented below.

Study One: Associations between disordered eating and internalizing symptoms in preadolescence.

The aim of this study is to address question one, by examining concurrent associations between DE and internalizing symptoms in preadolescents, aged 9-11 years, using a range of validated measures. This is because previous studies in preadolescence are inconsistent and have often relied on measures more suitable for older adolescents and adults. In addition, gender differences in these associations can be explored.

Study Two: The role of set shifting in the association between internalizing symptoms and disordered eating in preadolescence.

This study aims to address questions two and three, by expanding on the link established between DE and internalizing symptoms in Study one. I will examine associations between behavioral markers of set shifting, the first component of cognitive control to be explored, and both DE and internalizing symptoms in a sample of preadolescence. Set shifting will be measured using the Shape Trail Test - Child Version (STT-CV; Chan & Morgan, 2018), a culturally unbiased measure of set shifting. This task differs from more traditional set shifting measures as it relies less on language understanding and integration of numerical and alphabetical series, an important consideration when recruiting children from diverse communities. This study will also investigate whether DE is associated with set shifting independently of internalizing symptoms in preadolescence, which to-date has not yet been examined.

Study Three: Neural correlates of response monitoring and response inhibition in a preadolescent sample displaying disordered eating and internalizing symptoms.

Study three addresses questions two and three and expands on Study two to investigate both behavioral and neural markers of cognitive control processes. I will again investigate the associations between DE, internalizing symptoms, and cognitive control processes, using a Go/NoGo task to assess both response monitoring and response inhibition. By using one Go/NoGo task to assess both components, the child completes fewer tasks, reducing the chance of fatigue and inattention, an important consideration in developmental research. The Go/NoGo task allows simultaneous brain-behavior assessments in children. Therefore, behavioral measures of response monitoring and response inhibition, namely accuracy rates and reaction times, will be examined alongside neural measures in the form of the following ERPs: N2, P3, ERN, Pe, and CRN. I will investigate whether behavioral and neural markers of response inhibition and response monitoring are associated with DE independently of internalizing symptoms in preadolescence.

Study Four: Neural correlates of emotion regulation in a preadolescent sample displaying disordered eating and internalizing symptoms.

To expand on Study three and address questions two and four, this study will investigate response monitoring and response inhibition in the context of emotional stimuli, using both neural and behavioral measures. This study will follow the same procedure used in

Study three, with the addition of emotional stimuli to the Go/NoGo task. This simplifies the task instructions the child needs to understand, another important consideration for developmental research. The mediating role of internalizing symptoms will again be examined in the relation between emotion regulation and DE.

Chapter 2. Associations between disordered eating and internalizing symptoms in preadolescence.

The study described in this chapter has been published as an original article:

Thomas, K. S., Williams, M. O., & Vanderwert, R. E. (2021). Disordered eating and internalizing symptoms in preadolescence. *Brain and Behavior, 11*(1). e01904.
doi: 10.1002/brb3.1904

2.1 Introduction

As I previously discussed in Chapter 1 of this thesis, research has reported considerable overlap between DE, anxiety, and depression in both adolescent and adult samples (Godart et al., 2007; Rojo-Moreno et al., 2015; Schaumberg et al., 2019; Touchette et al., 2011; Zaider et al., 2000). However, much less is known about these associations in preadolescence. The literature is further limited when considering the measures used to examine DE, which are often only validated in older adolescents and adults, rather than preadolescents.

This study aimed to build upon the current literature by examining concurrent associations between DE, anxiety, and depression, in a demographically diverse sample of boys and girls using validated measures for preadolescents. This aimed to address inconsistencies in the existing literature concerning the association between DE and internalizing symptoms in this age group. For example, Holm-Denoma et al. (2014) reported a weak and largely non-significant association between DE and anxiety symptoms across boys and girls, and two age cohorts: 9-11 years and 12-15 years. Whereas, Houldcroft et al. (2014) found significant associations between DE and both general and social anxiety in preadolescents. However, Houldcroft et al. (2014) measured eating behaviors through an examination of dietary restraint, emotional eating, and external eating; constructs that are more associated with behaviors present in BN and binge eating (e.g. Johnson et al., 2012; Stein et al., 2007), rather than broader eating psychopathology. Importantly, although the measures employed by Houldcroft et al. (2014) were adapted for their preadolescent sample, the measure of DE used by Holm-Denoma et al. (2014) was not adapted for the youngest ages in their sample.

I hypothesized that children who reported higher levels of DE will also report higher levels of anxiety and depression symptoms, and that there will be higher levels of anxiety and

depression symptoms among children who are categorized as above the clinical threshold for DE compared to children who are categorized as below the clinical threshold. I also hypothesized that anxiety and depression will both be significant independent variables in regression models examining relations between anxiety, depression, and DE.

A further aim of the study was to examine the role gender may play in moderating the relations between DE, anxiety, and depression, and how this symptomatology presents in preadolescence. In line with previous research in community samples of preadolescent children described in Chapter 1, it was hypothesized that there will be no gender differences in DE or depression symptoms (e.g. Ferreiro et al., 2012; Holm-Denoma et al., 2014). In contrast, it was hypothesized that gender will moderate the association between anxiety symptoms and DE, with stronger associations between DE and anxiety present in girls compared to boys. This is based on literature demonstrating a strong relation between anxiety disorders and DE in adolescent females (e.g. Touchette et al., 2011; Zaider et al., 2000) and reports of significant gender differences in anxiety levels in preadolescents (e.g. Houldcroft et al., 2014).

2.2 Methods

2.2.1 Ethical Guidelines

This project received approval from the Cardiff University School of Psychology Ethics Committee. Headteachers provided gatekeeper consent before parents/guardians were contacted. Opt-in parental consent was obtained as well as assent from the child prior to participating in the study. Both parent and child were provided with a description of the study and were made aware of their right to withdraw at any stage of the study. All data were stored anonymously.

2.2.2 Recruitment and Sample

The study recruited a sample of 213 children aged 9-11 years ($M = 10.3$ years; 51.2% male) from twelve state-run primary schools in south Wales. Children were recruited from both years 5 and 6. An additional 18 children with parental consent to participate in the study were absent for the day of testing.

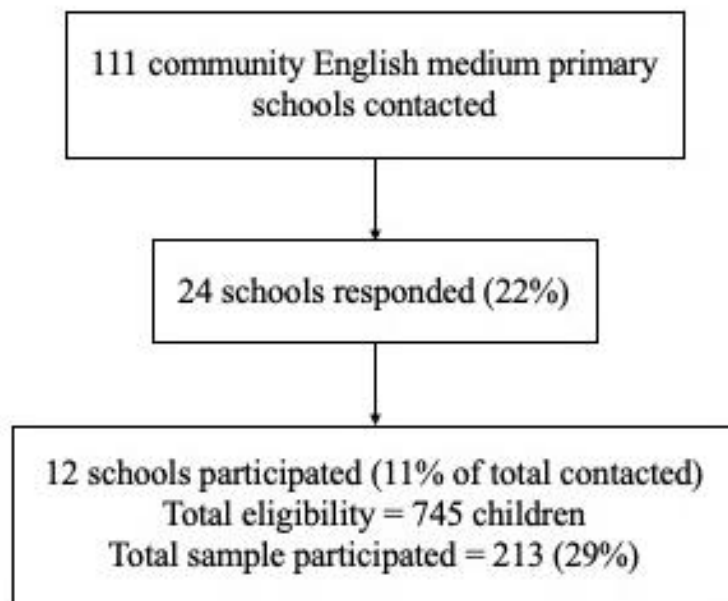
Headteachers from all 111 community English-medium primary schools in south Wales were contacted to enquire about interest in the study. Twenty-four schools responded to the invitation (22%), but only twelve were able to participate in the research (11% of total

schools contacted). Barriers to participation for some of these schools included no current year 5/6 classes and lack of time due to commitments to other projects.

From the consenting schools, 745 children were eligible to participate in data collection based on their current school year. Therefore, approximately 29% of the eligible sample participated in the study. See Figure 2.1 for a summary of the recruitment process.

Figure 2.1.

Recruitment process for schools.



G*Power 3.1 (Faul et al., 2007) was used to conduct a power calculation to determine the required sample size for correlation and multiple regression analyses. Based on previous literature (Houldcroft et al., 2014), a medium effect size of .18 measured by Cohen's f^2 was used as well as an alpha value of .05, power of .95, and a maximum of three independent variables. The estimated total sample size was 100 participants.

Free school meal (FSM) uptake data was collected for each school to assess socioeconomic status at the school level. This figure is a three-year average of the pupils eligible for FSM and the Welsh average is 18% (Welsh Government, 2019). High FSM averages indicate high material deprivation; however, this is only at the school level and not at the individual family level. Table 2.1 provides a summary of the participation rates for each school, as well as FSM data, proportion of children with reported additional learning needs (ALN) or special education needs (SEN), and the proportion of children who are

learning English as an additional language (EAL). The data were collated from a Welsh Government school information database (Welsh Government, n.d.).

Table 2.1.

Individual school characteristics: participation rates and proportion of children in each school who meet criteria for FSM, ALN/SEN and EAL provision.

	N (proportion of eligible children)	FSM (%)	ALN/SEN (%)	EAL (%)
School 1	46 (38%)	24.5	22.3	8.4
School 2	10 (20%)	50.4	33.7	16.3
School 3	28 (27%)	13.0	14.9	N/A
School 4	35 (37%)	16.8	27.6	16.1
School 5	17 (34%)	40.3	34.8	3.8
School 6	10 (14%)	40.5	35.8	25.5
School 7	2 (9%)	45.1	30.1	15.7
School 8	21 (35%)	2.0	5.9	1.3
School 9	5 (17%)	13.0	18.4	N/A
School 10	8 (27%)	27.0	22.4	8.8
School 11	11 (22%)	30.4	17.9	2.9
School 12	20 (33%)	7.2	9.7	6.3

Note. FSM: Free School Meals; ALN: Additional Learning Needs; SEN: Special Educational Needs; EAL: English as an Additional Language.

2.2.3 Measures

2.2.3.1 Children's Eating Attitude Test (Maloney et al., 1989)

DE behaviors and attitudes were measured using the Children's Eating Attitude Test (ChEAT), a self-report modified version of the abbreviated adult Eating Attitudes Test (EAT-26; Garner & Garfinkel, 1979). The ChEAT is a 26-item questionnaire designed to measure dimensional DE behaviors and attitudes in children aged between 8-and 13-years-old, such as food preoccupation, concerns about being overweight, bingeing and purging, and dieting. Each item is rated by the child using a 6-point response format (always, very often, often, sometimes, rarely, never) and represents the frequency with which the child demonstrates the

attitude or behavior described in each item (e.g., I feel very guilty after eating.). Scores range from 0-78, with the three most symptomatic responses ('often', 'very often' and 'always') scored from 1-3 respectively, and the remaining three responses scored as zero.

The scale has good test-retest reliability ($\alpha = 0.81$) and good internal consistency ($\alpha = 0.90$) in boys and girls aged between 7 and 12 years (Maloney et al., 1988; Smolak & Levine, 1994). Higher total scores on the ChEAT indicate higher levels of symptomatology with a cutoff score of 20 indicative of more severe eating pathology that could warrant further clinical assessment (Maloney et al., 1988).

Adjustments to the wording of item 4 were made where 'I have gone on eating binges where I feel that I might not be able to stop' was changed to 'I have started to eat and then felt like I cannot stop'. This was based on previous studies (e.g. Coombs et al., 2011) highlighting difficulties with the comprehension of 'binges' with children of a similar age. In addition, items 9 and 26, which refer to 'vomit,' were also accompanied by 'am/be sick', a more familiar term for children. Finally, item 21 was changed from 'I give too much time and thought to food' to 'I spend too much time thinking about food', to simplify the wording and improve comprehension. Alpha values for the adjusted items were acceptable ($\alpha = 0.706$).

2.2.3.2 Revised Child Anxiety and Depression Scale - 25 item version (Muris et al., 2002)

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 DSM. The RCADS-25 was used as it provides separate anxiety and depression subscales, comprised of 15 and 10 items, respectively. This is important as both anxiety and depression are relevant to EDs and may display different relations to cognitive control (described in Chapter 1). The RCADS-25 captures broad anxiety symptoms, ranging from social anxiety symptoms (e.g., I am afraid of being in crowded places) and generalized anxiety symptoms (e.g., I worry that something awful will happen to someone in my family), to obsessive compulsive symptoms (e.g., I have to think of special thoughts (like numbers or words) to stop bad things from happening). The depression subscale captures symptoms relevant to MDD diagnostic criteria (e.g., Nothing is much fun anymore; I have no energy for things). Cronbach's alpha values for the current sample were acceptable for both scales (anxiety: $\alpha = .864$; depression: $\alpha = .839$), as well as the total score ($\alpha = .916$). All 25-items are rated on a 4-point scale (never, sometimes, often, always) and represent the frequency to which these behaviors, thoughts or feelings occur (e.g., I have trouble sleeping). Overall scores range from 0-75 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).

2.2.4 Procedure

Headteachers provided consent for the study to take place at the school and opt-in consent was required from parents/guardians. All questionnaires were administered within schools during class time. The children whose parents/guardians provided consent were assessed in small groups of 5-6 at a time, where they were seated in a separate classroom with the researcher who was either alone or accompanied by an additional teacher. Children were asked to self-identify their gender and provided with an age-appropriate introduction to the study and the researcher, as well as an opportunity to ask any questions. They were seated around a table but asked to not share their answers with anyone else or read each other's answers.

Children were asked to provide assent prior to their participation in the study. The contents of the assent form were read aloud to the children and questions about the contents of the form were asked to check for understanding of the information. This included their understanding of the confidentiality of the information they provided from their parents/guardians and teachers, their right to withdraw at any time, their right to ask questions at any time, and their ability to leave any questions blank that they did not want to answer.

Questionnaire measures took approximately 30 minutes to complete. Children were read each question aloud before making their response, however if any of the children did not understand or were unsure of a specific question, then they were asked to raise their hand. The researcher then helped clarify the wording to the child by using a list of standard definitions or paraphrases. Due to the variability in reading age, some children required more input than others, however the class teacher was able to advise the researcher on who these children were beforehand. When an additional teacher was present, they were able to help by working through the questions with children who exhibited difficulties with reading more generally, however this was dependent on time and staffing constraints. Children who wished to work at a faster rate were permitted to, but they were asked to stop and listen to the instructions given for each new questionnaire.

Once the questionnaires had been completed the children were provided with a verbal debrief and given a chance to ask the researcher any remaining questions or discuss any concerns they had. They were also given a written debrief form to take home to their parents/guardians which included a list of contact details for the researcher and their supervisors, as well as some support organizations for advice and helpful resources.

Children were observed during the testing session for signs of emotional distress and in the event of this occurring, the child was encouraged to discontinue their participation ($n = 0$). The questionnaire measures used in this study were not diagnostic tools, so diagnoses could not be determined based on the children's scores. In the event of an extremely high score on these measures, a protocol was put in place to inform the parent and advise a visit to their medical doctor for further support ($n = 0$).

2.2.5 Statistical Analyses

All statistical analyses were performed using SPSS (version 27.0; IBM, 2020). All analyses were two-tailed and a p -value of .05 was used to determine statistical significance. Data were screened for floor and ceiling effects, as well as missing values. There were 33 data points missing from the ChEAT data (0.6% of overall data points) and 34 data points missing from the RCADS data (0.6% of overall data points). These missing values were distributed across participants and the items in these scales, so total scores were calculated irrespective of the missing values.

Visual inspection of plots revealed that the data were positively skewed and not normally distributed; this was supported by Shapiro-Wilk normality tests which were significant for both ChEAT score and RCADS measures. Therefore, a log transformation was performed on both the ChEAT and RCADS measures. All measures, excluding the RCADS total score, did not meet the assumption of a log transformation due to a minimum value of zero. Therefore, a constant of 1 was added when the transformations were performed. All measures were found to be normally distributed after the log transformation and could therefore be analyzed with parametric tests. In light of some limitations raised regarding the use of log transformations (Field & Wilcox, 2017), bootstrapping using the original data was also performed alongside each statistical test to ensure the results were robust. Results using parametric tests and bootstrapping were identical unless reported otherwise. Unless specified, untransformed data are presented in tables.

Correlational analyses were used to explore associations between DE, anxiety, and depression. The significant associations were followed up with linear regressions, whereby ChEAT scores were entered into the model as the dependent variable and anxiety and depression symptoms were independent variables. To examine whether gender may play a moderating role in the relations between anxiety, depression, and DE, *t*-tests were conducted to test for differences between boys and girls on both ChEAT and RCADS measures. Separate correlation and regression analyses were performed to examine associations between the questionnaire measures for girls and boys. In each of the linear regression models, ChEAT was entered as a dependent variable and anxiety and depression symptoms were independent variables. An additional linear regression was performed to examine the interaction between anxiety and gender when ChEAT was the dependent variable.

2.3 Results

2.3.1 Descriptive Statistics

Table 2.2 presents the descriptive statistics for the whole sample as well as split by gender. ChEAT scores and RCADS total scores presented some variation in the sample, with the majority falling within normative ranges.

Table 2.2.*Descriptive statistics for the whole sample with gender differences examined.*

	Whole Sample (n = 213)		Male (n = 109)		Female (n = 104)		<i>t</i> (211)	<i>p</i>	<i>d</i>
	M (SD)	Range	M (SD)	Range	M (SD)	Range			
ChEAT	9.18 (6.85)	0-43	9.43 (6.71)	0-31	8.91 (7.01)	0-43	-.558	.578	.077
RCADS Total	20.73 (12.50)	1-68	20.03 (11.18)	2-55	21.46 (13.77)	1-68	-.518	.605	.071
RCADS Anxiety	12.74 (7.89)	0-38	12.11 (7.15)	1-29	13.39 (8.58)	0-38	-.957	.340	.033
RCADS Depression	7.99 (5.47)	0-30	7.92 (5.13)	0-26	8.07 (5.82)	0-30	-.101	.919	.014

Note. ChEAT: Children's Eating Attitude Test; RCADS: Revised Child Anxiety and Depression Scale.

2.3.2 Associations between DE, Anxiety, and Depression Symptoms

To test my first hypothesis, correlations were performed to investigate the associations between DE, anxiety, and depression in the sample (Table 2.3). There was a significant positive correlation between ChEAT scores and total RCADS scores, as well as ChEAT scores and both anxiety and depression subscales.

Table 2.3.*Pearson's correlations between questionnaire measures.*

	ChEAT	RCADS Total	RCADS Anxiety
RCADS Total	.421**		
RCADS Anxiety	.417**	.932**	
RCADS Depression	.356**	.880**	.679**

Note. ** $p < .001$. ChEAT: Children's Eating Attitude Test; RCADS: Revised Child Anxiety and Depression Scale.

Transformed data were used in the analyses.

A sensitivity analysis was conducted to account for any overlap between the ChEAT and RCADS scores concerning an item directly examining eating behavior in the RCADS depression subscale ('I have problems with my appetite'). This item was removed from the RCADS depression subscale and correlational analyses with the ChEAT total score were repeated. A Pearson correlation revealed a significant positive correlation between ChEAT total score and RCADS depression score ($r = .361, p < .001$), suggesting this item was not independently contributing to the positive association between the two measures.

To further investigate the significant associations between DE, anxiety, and depression, a linear regression was performed with RCADS total scores and ChEAT scores. The model was significant ($F(1, 211) = 45.45, p < .001, R^2 = .177$). Another linear regression was performed to examine the independent associations of the RCADS anxiety and depression subscales on ChEAT scores. The model was significant ($F(2, 210) = 23.58, p < .001, R^2 = .183$). While anxiety contributed significantly to the model ($B = .357, SE B = .093, p < .001$), depression was not significant ($B = .144, SE B = .091, p = .113$). As anxiety and depression often co-occur and were correlated, collinearity diagnostics were examined to ensure assumptions of multicollinearity were not violated. VIF values were acceptable (VIF = 1.853).

One of the aims of the study was to investigate levels of anxiety and depression symptoms among children who were categorized as above/below the clinical threshold for DE. Based on the limited sample of children who were categorized as above the clinical threshold ($n = 18$), I was not adequately powered to test this.

2.3.3 Examination of Gender Differences

To test my hypothesis that there will be no gender differences present in DE or depression, a *t*-test was conducted to examine whether there were significant gender differences in average scores on the ChEAT and RCADS (Table 2.2). No significant differences were found for any of the mean scores between boys and girls.

Additional correlation analyses were performed to examine associations between the questionnaire measures for girls and boys, respectively (see Tables 2.4 and 2.5). Overall, the correlations were very similar for both genders. The correlations between ChEAT scores and total RCADS scores appear slightly stronger in girls compared to boys. This was also the case for correlations between ChEAT scores and anxiety and depression subscales.

Table 2.4.

Pearson's Correlations Between Questionnaire Measures for Girls.

	ChEAT	RCADS Total	RCADS Anxiety
RCADS Total	.442**		
RCADS Anxiety	.480**	.953**	
RCADS Depression	.378**	.883**	.730**

Note. ** $p < .001$. ChEAT: Children's Eating Attitude Test; RCADS: Revised Child Anxiety and Depression Scale. Transformed data were used in the analyses.

Table 2.5.

Pearson's Correlations Between Questionnaire Measures for Boys.

	ChEAT	RCADS Total	RCADS Anxiety
RCADS Total	.404*		
RCADS Anxiety	.363*	.911*	
RCADS Depression	.336*	.878*	.633*

Note. * $p < .01$. ChEAT: Children's Eating Attitude Test; RCADS: Revised Child Anxiety and Depression Scale. Transformed data were used in the analyses.

To test whether the RCADS anxiety coefficient was significantly different between boys and girls, a linear regression was performed with the RCADS anxiety term, a recoded dummy variable for gender, and an interaction term combining these two variables. The overall model was significant ($F(3, 209) = 15.37, p < .001, R^2 = .181$) however the interaction term was not a significant coefficient ($B = .118, SE B = .138, p = .394$). The linear regression was repeated to test whether the RCADS depression coefficient differed significantly between boys and girls. Again, the overall model was significant ($F(3, 209) = 10.30, p < .001, R^2 = .129$) but the interaction term was not a significant coefficient ($B = .055, SE B = .138, p = .689$). These findings indicate that both the RCADS anxiety and RCADS depression coefficients did not differ significantly between boys and girls. However, when collinearity diagnostics were examined for both multiple regressions, there were substantial violations of the assumption of multicollinearity. Specifically, the interaction terms (anxiety: $VIF = 17.85$; depression: $VIF = 11.66$) and dummy variable for gender (anxiety: $VIF = 16.34$; depression: $VIF = 10.73$) violated this assumption. Mean centering the variables is commonly used to reduce the issue of multicollinearity, but this would not affect the test statistic and the non-significance of these results (Shieh, 2011), so no further action was taken.

2.4 Discussion

Research conducted with adolescents and adults has reported strong associations between internalizing symptoms and DE behaviors, as well as diagnosed EDs. However, studies examining these associations in preadolescence have provided mixed results. The results of the current analyses provide strong support for a pattern of associations between DE and internalizing symptoms in preadolescence that is consistent with what has been reported in adolescents and adults. Specifically, my results showed that preadolescent children who reported higher levels of DE also reported higher levels of anxiety and depression symptoms.

In line with my hypotheses, the relation between anxiety and DE was statistically significant, but depression was not significant in the model. Associations between depression and DE have previously been reported in preadolescents (e.g. Evans et al., 2017; Gardner et al., 2000); however, not all of these studies included anxiety as an independent variable. The overlap between anxiety and depression could mean that once the variance in DE is accounted for by anxiety, the unique variance explained by depression is no longer

statistically significant. To my knowledge this is one of the first studies to highlight this dissociation between anxiety and depression using validated measures for preadolescents. Previous studies have either measured anxiety and depression in isolation or when measured concurrently, Holm-Denoma et al. (2014) reported significant relations between DE and depression, but not anxiety. Their findings are inconsistent with the wider literature in EDs where anxiety symptoms are commonly reported to arise prior to the onset of the ED (e.g. Raney et al., 2008; Swinbourne et al., 2012). My results are therefore in line with the broader hypothesis that elevated anxiety in childhood increases risk for later development of DE (e.g. Adambegan et al., 2012; Kaye et al., 2004).

Consistent with my hypotheses, there were no gender differences in either DE or depression symptoms in preadolescents. However, inconsistent with my hypotheses and previous evidence from older adolescents (O'Dea & Abraham, 1999), gender was not a moderator in the association between anxiety and DE in my study. I also found that regression models examining the interaction between gender and both anxiety and depression were not statistically significant. These non-significant results are interesting in the context of pubertal, environmental, and social changes that occur in adolescence. These changes may be key in explaining why gender is a moderator in this older sample, and it could be the case that these factors have not yet emerged for the sample in this study. For example, Deardorff et al. (2007) reported an increase in social anxiety symptoms in females compared to males during puberty. This gender difference may be important in the context of DE due to the overlap between EDs and social anxiety (Pallister & Waller, 2008).

The overlap between social anxiety and EDs is important when considering the mechanisms underlying the trajectories of anxiety and DE. Pallister and Waller (2008) argue the relative chronology of anxiety and eating disorders is dependent on the type of anxiety. Social anxiety, for example, has been reported to frequently precede the ED, whilst generalized anxiety disorder more commonly occurs later or simultaneously. This may be because social anxiety typically starts during later childhood, whereas generalized anxiety disorder emerges during later adolescence into adulthood (Solmi et al., 2021). Schwalberg et al. (1992) suggest DE behaviors and attitudes such as concerns over shape, eating, and weight may emerge because of anxiety about social evaluation; providing further support for a specific association between social anxiety and DE. These findings highlight the complexity of examining the trajectories of DE and anxiety, and the role different types of anxiety may play in the development and maintenance of DE and EDs. Although the RCADS-25 captures a broad range of anxiety symptoms, including social phobia and generalized anxiety, these

symptom types were not disaggregated in my study. Therefore, I am unable to draw specific conclusions about the relations between DE and anxiety subtypes in preadolescence.

This study has implications for broader understanding of the presentation of DE and internalizing symptoms in preadolescence. Firstly, the presence of these potentially very serious behaviors and cognitions in a moderately large and representative sample of children is concerning and highlights the importance of early screening measures for prevention and intervention. In addition, the dissociation between anxiety and depression and their relations to DE highlights the importance of measuring all three concurrently to delineate the individual risk trajectories of internalizing symptoms in the development of DE. For example, anxiety may be a stronger risk factor for DE in preadolescence, as childhood anxiety tends to precede DE (e.g. Raney et al., 2008; Swinbourne et al., 2012). Whereas depression may play a more pertinent role in DE development during adolescence, due to its strong association with bulimic symptoms (Presnell et al., 2009), which more commonly occur in later adolescence and adulthood (Solmi et al., 2021). Furthermore, the presence of DE in both girls and boys supports early screening for DE for all children. However, more gender-targeted interventions could be better suited to adolescents due to the increased prevalence of EDs in females compared to males, indicating different risk trajectories (e.g. Touchette et al., 2011; Zaider et al., 2000).

The strong associations between DE and anxiety in preadolescence during the end of primary school not only supports previous research in adolescence (Touchette et al., 2011; Zaider et al., 2000), but also contributes to the limited literature in preadolescence. Both DE and anxiety during this stage of development have the potential to increase risk of developing EDs and anxiety disorders in later adolescence, and as highlighted already, early detection of DE and anxiety may be crucial for intervention and prevention. This is especially important considering the increased stress that can occur during this time as a result of puberty and the transition from primary to secondary school (Rice et al., 2011). A transdiagnostic prevention program during the last year of primary school would be one potential way of addressing this.

One limitation of the methodology used in this study is the reliance on self-report measures and the biases that can occur when self-report measures are used have been well-documented (Paulhus, 1991). Importantly, the measures used in this study were adapted and validated questionnaires for the age group recruited in this sample and previous research has demonstrated self-report symptom scales are predictive of subsequent diagnoses (Shankman et al., 2009). In addition, children who required extra provision with comprehension and reading were provided with support by the researcher and/or schoolteacher during the testing

sessions. Parent and teacher reports or diagnostic interviews with the children would have provided richer information; however, this option was constrained by time and resources. The findings from this study were based on a cross-sectional design so I am not able to examine the trajectories of DE, depression, and anxiety across this period from preadolescence to adolescence or draw conclusions about causality. Therefore, longitudinal studies which start during preadolescence and follow-up during adolescence would be valuable in examining the co-occurrence of these symptoms. Additionally, the use of opt-in parental consent was important when recruiting for this study due to the sensitive nature of the questionnaires employed; however, there is potential for this to introduce sampling bias as some parents may be wary of research or be less able to engage with school communications. Similarly, parents may want to protect their child from engaging with questions on topics of concern related to their child's eating behaviors. Finally, IQ was not measured in my study and should be considered a limitation. This decision was made based on the time constraints of completing testing sessions within schools, as well as considerations around participant burden.

Future research should consider the measures used to examine DE in preadolescence, as there are still inconsistencies present in the literature. Adjustments to the scoring method and factor structure of the ChEAT in non-clinical populations have been proposed to increase the variance of the item scores and hence the total score, as well as reducing the skewness in the data (Anton et al., 2006). Research examining the underlying mechanisms involved in this overlap is crucial to further our understanding of the development of these symptoms and any shared associations with cognitive functions and neurobiology. Chapters 3 and 4 will build upon this study by specifically examining the role of cognitive control components in this association between DE and internalizing symptoms.

In summary, this study provides support for the associations between DE and both anxiety and depression in preadolescence. While the relation between anxiety and DE behaviors was significant, and that this is the case for both boys and girls, depression was not a significant independent variable when included in the model alongside anxiety. These results highlight the importance of early detection for DE behaviors and attitudes, as well as anxiety and depression in both male and female children during preadolescence. However, longitudinal research is vital to examine the trajectories of these problems, as well as additional factors, across time.

Chapter 3. The role of set shifting in the association between internalizing symptoms and disordered eating in preadolescence.

3.1 Introduction

In Chapter 2, I presented findings demonstrating significant associations between DE and internalizing symptoms in preadolescents. In line with the model presented in Chapter 1, this co-occurrence may reflect shared cognitive correlates, in the form of cognitive control processes. I will build upon the findings described in Chapter 2 by examining the role of set shifting, the first component of cognitive control to be explored in the thesis, in the association between DE and internalizing symptoms. Specifically, I will investigate whether set shifting impairments are associated with DE independently of internalizing symptoms, or whether internalizing symptoms mediate the relation between set shifting and DE.

There is convincing evidence for impairments in set shifting in adults with EDs (Aloi et al., 2015; Brand et al., 2007; Duchesne et al., 2010; Roberts et al., 2010; Tchanturia et al., 2004; Tchanturia et al., 2002; Wu et al., 2014), but findings are more varied in children and adolescents (Allen et al., 2013; Bühren et al., 2012; Darcy et al., 2012; Herbrich et al., 2018; Kjaersdam Telléus et al., 2015; Lang et al., 2014; Rößner et al., 2017; Shott et al., 2012; Wang et al., 2021). As these studies focus on children and adolescents who have already been diagnosed with an ED, it is still not known whether set shifting difficulties are present before the onset of the ED (trait factor) or whether these difficulties co-occur with the onset of the ED (state factor). Set shifting difficulties are proposed to be a heritable trait (Galimberti et al., 2013; Holliday et al., 2005; Tenconi et al., 2010); however, only one study to-date has examined set shifting difficulties prior to the onset of an ED in preadolescence (Steegers et al., 2021). This prospective study examined the associations between set shifting ability and DE behaviors and cognitions across time. Using a maternal-report measure of set shifting ability, they found set shifting difficulties at 4 years of age were associated with more restrictive eating behaviors (maternal-report), but not body image (child-reported), in boys and girls at age 9. These findings suggest that set shifting difficulties may be related to the early development of specific DE behaviors. However, this research can be strengthened by measuring set shifting using neurocognitive tasks, rather than parental reports, to investigate difficulties in preadolescents at-risk for developing an ED. In addition, research targeting a broader range of DE behaviors than captured by Steegers et al. (2021) is still needed in preadolescents.

Set shifting difficulties are a well-documented factor involved in the development and maintenance of anxiety and depression (Liu et al., 2021; Rock et al., 2014; Tempesta et al., 2013; Zainal & Newman, 2018; Zhang et al., 2015). Less work has focused on child and adolescent samples; however, impaired set shifting in children has been associated with depressive symptoms (Micco et al., 2009) and higher levels of behavioral inhibition (Buzzell et al., 2021; Henderson et al., 2015; Henderson & Wilson, 2017). Therefore, it may be the case that DE behaviors develop as a maladaptive way of coping with anxiety and depression symptoms that accompany persistent negative cognitions (in line with the thesis model presented in Chapter 1). This in turn, may be exacerbated by the more rigid and inflexible cognitive and behavioral style resulting from set shifting difficulties.

This study aimed to examine the association between set shifting performance and both DE behaviors and internalizing symptoms in a community sample of preadolescents. The Shape Trail Test - Child Version (STT-CV; Chan & Morgan, 2018), a modified version of the Children's Trail Making Test (TMT-CV; Reitan & Herring, 1985), was used to measure set shifting ability, extending previous research using parental-report measures (Stegers et al., 2021). In addition, the current study captured self-report measures of a broad range of DE, as well as measures of internalizing symptoms. I had two primary hypotheses:

1. Impaired set shifting performance will be associated with increased levels of self-reported DE, anxiety, and depression symptoms.
2. Internalizing symptoms will mediate the relation between set shifting and DE, in line with the overarching model of the thesis.

3.2 Methods

3.2.1 Participants

A total of sixty-three participants (M age = 11.0 years; 46.0% female) were recruited for this study across two stages. During the first stage, twenty-six children (M age = 10.9 years; 53.8% female) were recruited from a previous stage of the project (detailed in Chapter 2). These children completed the self-report questionnaires in their school and 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 completing the questionnaires in their school and participating in the current study. Stage two of recruitment invited thirty-seven 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 socio-economic status ($U = 395.5, z = -1.254, p = .210$), or ethnicity ($X^2(3, N = 60) = 6.474, p = .091$). Therefore, I collapsed across the two groups. Study variables were not compared between recruitment groups as I was only interested in their variability across the whole sample, rather than examining neural and behavioral correlates of eating behaviors and internalizing symptoms at each recruitment stage.

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 terms of task exclusion criteria, data from five participants were identified as statistical outliers and excluded (defined as > 3 SDs away from the mean duration to complete Part B), as well as an additional participant who was excluded due to excessive errors (defined as > 3 SDs away from the mean number of errors) on the task. Table 3.1 presents the demographics of the final sample. Socio-economic status (SES) was determined via postcode matching to the Welsh Index of Multiple Deprivation (WIMD).

Table 3.1.*Demographics of the whole sample.*

Child Demographics (N = 57)	<i>M</i> (Range)
Age (years)	10.98 (10.00 – 11.92)
Gender (male %)	50.9
Ethnicity (%)	
White	80.7
Mixed or Multiple Ethnic Groups	1.8
Asian or Asian British	3.5
Other Ethnic Group	8.8
Black, African, Caribbean, or Black British	0
Missing data	5.3
Parent Demographics	<i>M</i> (Range)
Age (years)	42.97 (29.90 – 61.20)
SES (WIMD) Quartile (%)	
1 st (most deprived)	22.8
2 nd	15.8
3 rd	17.5
4 th (least deprived)	43.9

Note. SES: Socio-economic status, WIMD: Welsh Index of Multiple Deprivation.

A post-hoc sensitivity analysis was conducted to determine the effect size that could be detected based on the collected sample size. Based on a two-tailed bivariate correlation with an alpha value of .05, power of .95, and sample size of 57, the critical $r = .261$.

3.2.2 Materials

3.2.2.1 Self-report Questionnaire Measures

The ChEAT (Maloney et al., 1989) and RCADS-25 (Muris et al., 2002) were used to measure levels of DE, anxiety, and depression, respectively. Chapter 2 provides full details of these measures. As highlighted at the end of Chapter 2, the shortened response set used in the ChEAT raises the potential for limited variability obtained on each item, as well as the total score (Anton et al., 2006; Smolak & Levine, 1994).

Therefore, the current study (and all subsequent studies described in the thesis) 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). I also found greater variance and a reduction in skewness in my sample (descriptive statistics for both original and alternative ChEAT scoring procedures are presented in Appendix A).

In the alternative scoring procedure, a Likert scale from 1 (never) to 6 (very often) is used, with higher scores representing more difficulties. Adjustments to the wording of certain items in the ChEAT are identical to those described in Chapter 2. Cronbach's alpha value for the adjusted items with the alternative scoring strategy was acceptable ($\alpha = .713$). To confirm the amendments to the measure produced similar factor structures to previous studies using the ChEAT in this age range, I conducted a confirmatory factor analysis (full details are presented in Appendix B).

3.2.2.2 Shape Trail Test – Child Version (Chan & Morgan, 2018)

The Shape Trail Test - Child Version (STT-CV; Chan & Morgan, 2018) was used to measure set shifting ability, mirroring the number and position of items in the child version of the trail making task (TMT-CV). This version of the test was used due to less reliance on language understanding and integration of numerical and alphabetical series compared to the TMT-CV. In addition, the STT-CV is a culturally unbiased version of the task, which is important to consider when recruiting children from diverse communities. Task performance on the STT-CV correlates with children's performance on measures of executive function, including task switching (Chan & Morgan, 2018).

The task was completed on pen and paper, consisting of two parts (A and B). In Part A, numbers 1 to 15 enclosed by small circles are arranged across an A4 page, and participants are asked to connect the numbers in ascending order by drawing a line between them. This was timed and identical to Part A of the TMT-CV (Reitan & Herring, 1985). Part B consists of numbers 1 through 8 enclosed by small circles, arranged alongside numbers 1 to 7, enclosed by small squares. The participant is then told that the rules had changed, and they now had to connect the numbers in sequential numerical order, whilst alternating between shapes (e.g., 1 in the circle, 1 in the square, 2 in the circle, 2 in the square, etc.) The participant is also timed for this section of the task, and for both parts of the task the participant is instructed to work as quickly and accurately as they can.

The main outcome measure used in the study was the difference in completion time of Part A and Part B ($B_{\text{time}} - A_{\text{time}}$). The B - A difference score measures set shifting ability whilst controlling for baseline motor speed. In addition, frequency of errors were scored on Part B based on the number of incorrect connections: (i) sequential errors occurred when the child omitted the next element in a series of shapes or numbers; (ii) perseveration errors occurred when the child failed to correctly alternate between the category of shape and number. Perseveration errors are thought to be indicative of a repetitive response to a previously learned rule that persists even when a different response is required (Tchanturia et al., 2012). Higher error rates indicate more set shifting difficulties.

Videos of the behavioral tasks were coded using EUDICO Linguistic Annotator (ELAN) software (Sloetjes & Wittenburg, 2008). This was to verify timings documented during the session, and to score correct responses and errors. Interrater reliability between two coders was calculated (Cronbach's $\alpha = 1$).

3.2.3 Procedure

All participants in the study were accompanied to the laboratory session by a parent or guardian. First, the child completed an emotion recognition task (detailed in Chapter 5), as well as the self-report questionnaires. Children who were recruited in stage 1 completed the ChEAT and RCADS-25 in their schools within small groups (detailed in Chapter 2). Next, children completed two cognitive control tasks where EEG data was collected (described in Chapter 4 and 5) before completing the STT-CV. The child was video recorded whilst performing the task and time taken to complete each part was coded offline and discontinued if five minutes elapsed. Sample pages for both Part A and Part B were completed before starting the STT-CV, with any errors corrected by the experimenter. As compensation for their time, each child received a gift voucher and a small gift.

3.2.4 Statistical Analyses

All statistical analyses were conducted using SPSS (version 27.0; IBM, 2020). Approach to normality tests and correction of RCADS-25 data was as described in Chapter 2. Error rates on the STT-CV violated the Shapiro-Wilk test of normality and were not able to be corrected. Therefore, Pearson's correlations were used for RT data and Spearman's rho correlations were used for error rates.

Preliminary analyses found age was not correlated with questionnaire or behavioral data, and there were no significant differences in data collected between boys and girls. Therefore, gender and age were not included as covariates in subsequent analyses. To test my primary hypothesis and investigate the associations between DE, internalizing symptoms, and set shifting performance, correlational analyses were conducted.

3.3 Results

3.3.1 Questionnaire Measures

The majority of participants fell within normative ranges on the questionnaires. Descriptive statistics of questionnaire measures are presented in Appendix C. Correlations examined the associations between DE, anxiety, and depression. The current subsample of children was consistent with the larger sample presented in Chapter 2, with ChEAT scores positively correlated with both RCADS Anxiety ($r = .644, p < .001$) and RCADS Depression scores ($r = .439, p < .001$).

3.3.2 Behavioral Measures

Table 3.2 presents a summary of the SST-CV behavioral data for the sample. Overall, children committed a small number of errors on the task, which were mostly categorized as perseveration errors. Behavioral data split by gender is presented in Appendix D.

Table 3.2.

SST-CV behavioral data for the whole sample.

	M (SD)	Minimum	Maximum
Part B – Part A duration (s)	9.24 (4.67)	0.31	24.42
Perseveration errors	.40 (.78)	0	2
Sequential errors	.07 (.26)	0	1

Note. SST-CV: Shape Trail Test – Child Version.

To examine my first hypothesis, correlations examined associations between questionnaire and behavioral measures (Table 3.3). There were no significant correlations between the STT-CV outcome measures and self-report questionnaire measures.

Table 3.3.

Correlations between questionnaire measures and Shape Trail Test - Child Version (SST-CV) outcome measures.

	Part B – Part A duration ^a	Perseveration errors ^b	Sequential errors ^b
ChEAT Total	-.043	.009	.182
RCADS Anxiety	-.007	.006	.228
RCADS Depression	.002	-.066	-.032

Note. ChEAT: Children’s Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

^a Pearson’s correlation coefficients, ^b Spearman’s rho correlation coefficients.

Transformed RCADS-25 data were used in the analyses.

3.4 Discussion

I have explored whether the co-occurrence of internalizing symptoms and DE in preadolescents is driven in part by shared impairments in set shifting performance. Overall, my findings suggest that neither DE behaviors nor internalizing symptoms are associated with set shifting performance in preadolescence.

My findings are inconsistent with the only other study to examine the associations between set shifting ability and DE in a preadolescent sample (Steegers et al., 2021). Steegers et al. (2021) found set shifting difficulties at 4 years of age were associated with more restrictive eating behaviors, but not body image, in boys and girls at age 9. This suggests set shifting difficulties may contribute to the early emergence of DE behaviors, specifically restrictive eating behaviors, across both boys and girls in preadolescence. It is important to highlight the methodological differences when comparing my null findings with Steegers et al. (2021). Firstly, Steegers et al. (2021) measured set shifting at 4 years of age only,

compared to age 10-11 years in my study. My findings are consistent with a range of studies that report no set shifting differences in older children and adolescents with EDs compared to HCs (Andrés-Perpiña et al., 2011; Bühren et al., 2012; Castro-Fornieles et al., 2019; Darcy et al., 2012; Fitzpatrick et al., 2012; Herbrich et al., 2018; Kjaersdam Tellés et al., 2015; Lang et al., 2014; Miles et al., 2020; Rößner et al., 2017; Shott et al., 2012). Set shifting impairments measured in older children and adolescents may be masked by compensatory strategies over time, such as involvement of other cognitive abilities like response inhibition (Weinbach et al., 2019).

Secondly, compared to the child-reported DE behaviors and neurocognitive set shifting task in my study, Steegers et al. (2021) used maternal-report measures of set shifting ability and restrictive eating behaviors, while body image was a child-reported measure. Maternal-reported set shifting performance was measured using the Behavior Rating Inventory of Executive Function – Preschool version (BRIEF-P; Gioia et al., 1996). Previous research has raised questions about the relation of the BRIEF to neurocognitive measures of executive function, suggesting the BRIEF captures behavioral difficulties and risk for the development of social and school-related difficulties, rather than executive functioning abilities (McAuley et al., 2010). An additional explanation for these differences could be the environment in which the set shifting abilities are assessed. For example, neurocognitive tasks may assess underlying set shifting skills that are specific to that task, whereas the BRIEF captures the application of set shifting within the home and school settings. This may also include emotional features, as some of the items within the BRIEF-P set shifting subscale assess elements of emotional control. This may lead to parents reporting increased set shifting difficulties compared to the level of impairment assessed using neurocognitive measures.

Further supporting evidence for a dissociation between questionnaire and neurocognitive measures of set shifting has been found when using self-report measures of set shifting performance. For example, adolescents (Wang et al., 2021) and adults with an ED (Miles, Phillipou, et al., 2022) self-report poorer set shifting ability compared to HCs on questionnaire measures. However, no performance differences were found on neurocognitive tasks in children and adolescents with EDs (Andrés-Perpiña et al., 2011; Bühren et al., 2012; Castro-Fornieles et al., 2019; Darcy et al., 2012; Fitzpatrick et al., 2012; Herbrich et al., 2018; Kjaersdam Tellés et al., 2015; Lang et al., 2014; Miles et al., 2020; Rößner et al., 2017; Shott et al., 2012), as well as adults with acute AN (Miles, Phillipou, et al., 2022) compared to HCs. In addition, questionnaire and neurocognitive assessments of set shifting

performance were not found to correlate in a sample of individuals with or without a history of an ED diagnosis (Miles, Nedeljkovic, et al., 2022), suggesting these measures may capture different aspects of set shifting performance. For example, questionnaire measures of set shifting (self-reported or parental-reported) may capture inflexibility at an observable and perceived behavioral level, whereas neurocognitive tasks may focus on more general cognitive impairments in set shifting performance. One explanation for this distinction in performance could be that set shifting is more easily compensated for by other executive functioning abilities in neurocognitive tasks compared to questionnaire measures. As set shifting was the only domain of executive functioning examined in my study, future studies should measure and control for other domains such as response inhibition and working memory, alongside self-report and neurocognitive assessments of set shifting.

Another key aim of the study was to examine whether internalizing symptoms were also associated with set shifting performance, potentially mediating the association between DE and set shifting. My findings suggest internalizing symptoms are not associated with set shifting performance. This contrasts with previous research in anxiety, demonstrating set shifting difficulties are involved in the development of anxiety (Zainal & Newman, 2018; Zhang et al., 2015). Although there is less research in the age range of the current sample to directly compare to, research has reported a link between set shifting difficulties and higher levels of behavioral inhibition in children (Buzzell et al., 2021; Henderson et al., 2015; Henderson & Wilson, 2017). As children with higher levels of behavioral inhibition are at increased risk of developing social anxiety in later childhood and adolescence (Chronis-Tuscano et al., 2009), the relation between set shifting difficulties and anxiety may be more pronounced for social anxiety symptoms specifically, rather than broader constructs of anxiety, as measured in my study. However, similar to the study by Steegers et al. (2021), parental-report was used to assess set shifting performance in Buzzell et al. (2021), making direct comparisons between findings based on questionnaire vs. neurocognitive assessments challenging.

In terms of depression symptoms, set shifting impairments have been proposed to represent an endophenotype for depression (Liu et al., 2021; Rock et al., 2014). However, studies examining set shifting performance in children with a family history of MDD (Klimes-Dougan et al., 2006; Micco et al., 2009) found impairments were only present when children themselves presented with MDD symptoms. This suggests set shifting impairments may represent a state factor of depression and are only associated with more severe

depression symptoms (characteristic of MDD), rather than the levels of symptomatology captured in a community sample.

The findings of this study should be considered in light of some of the limitations. Firstly, the null findings may be explained by a lack of power. Based on a sensitivity analysis (detailed in 3.2.1), there was sufficient power to detect moderate-to-large effect sizes. However, a larger sample size would be required to detect smaller effect sizes. Secondly, some children in my study did not commit any errors on the SST-CV. Chan and Morgan (2018) validated the SST-CV in children aged 6-9 years, suggesting this task may not have been challenging enough to accurately capture set shifting performance in my older age range. In addition, due to the cross-sectional and correlational research design, I could not examine the trajectories or temporal relationships between these symptoms and set shifting performance. Finally, I only examined set shifting performance using one form of assessment. Based on the recently published findings demonstrating relations between questionnaire measures of set shifting and DE (Steegers et al., 2021), it would have been beneficial to also examine parental-reported set shifting performance in this study. As this study only focused on set shifting ability, I was not able to account for the possible compensatory role played by other executive functions.

Despite these limitations, this study is the first to examine the concurrent relations between DE behaviors and set shifting performance using neurocognitive tasks in a community sample of preadolescents. Important for considering ED research, I recruited both boys and girls across a diverse socioeconomic background to provide a more representative sample of the community. In contrast to my hypothesis, I found no associations between set shifting performance and both DE and internalizing symptoms in preadolescents. In Chapter 4, I will explore the associations between DE, internalizing symptoms, response inhibition, and response monitoring in preadolescents. Although response inhibition and response monitoring are components of cognitive control, they are distinct from set shifting. Due to the lack of associations between DE, internalizing symptoms, and behavioral data in this chapter, I hope to provide a more thorough and detailed examination of these cognitive control abilities using a combination of behavioral and neural measures.

Chapter 4. Neural correlates of response monitoring and response inhibition in a preadolescent sample displaying disordered eating and internalizing symptoms.

4.1 Introduction

As highlighted in Chapter 1 and 2 of the thesis, the co-occurrence of DE and internalizing symptoms has been reported in preadolescence (Thomas et al., 2021), as well as in EDs and internalizing disorders in adolescence (Ulfvebrand et al., 2015). This co-occurrence could relate to shared cognitive correlates, such as cognitive control processes. Cognitive control has been linked to the ACC (Banfield et al., 2004; Gehring & Knight, 2000), a key area of the brain implicated in both EDs and internalizing disorders (Lichenstein et al., 2016; Mühlau et al., 2007; Shang et al., 2014; Uher et al., 2003). In Chapter 3, I found no behavioral evidence for an association between poorer set shifting and higher levels of DE and internalizing symptoms, suggesting difficulties in this area of cognitive control may not be involved in the co-occurrence of these symptoms. The study presented in this chapter aims to build upon the work described in Chapter 3 by examining the role of cognitive control processes further, in the form of response inhibition and response monitoring. Neuroimaging will be used alongside behavioral measures to provide a more comprehensive examination of these cognitive control processes and their underlying mechanisms.

Response inhibition is 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). In Chapter 1, I presented evidence for response inhibition difficulties in adults with anxiety and depression (Grillon et al., 2017; Li et al., 2021), as well as individuals with binge eating and/or purging symptoms (Steinglass et al., 2019; Svaldi et al., 2014; Wu et al., 2013). Unlike adults, adolescents with internalizing symptomatology (Brunnekreef et al., 2007; Diler et al., 2014; Hum et al., 2013a; Pan et al., 2011; Singh et al., 2010) and DE behaviors (Bartholdy et al., 2019), typically show no differences in behavioral markers of response inhibition performance when compared to HCs. This suggests neural differences may be observable before cognitive differences have emerged, highlighting the importance of examining both neural and cognitive components at earlier stages of development.

The specific neural correlates of response inhibition are the N2 and P3, both stimulus-locked ERPs generated by the ACC (Bekker et al., 2005; Zhang et al., 2012). The N2 is proposed to reflect identification of conflict or inconsistency between competing responses

(Albert et al., 2013), while the P3 indexes later evaluation processes (Smith et al., 2008). 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 AN (Yue et al., 2020) demonstrate consistent attenuation of the P3 while no N2 effects are reported.

The literature examining N2 and P3 amplitudes in individuals with internalizing symptoms is generally mixed (see Chapter 1). Greater N2 amplitudes are observed in anxiety (Hoyniak & Petersen, 2019; Valadez et al., 2021; Yu et al., 2018), while individuals with depression typically demonstrate comparable N2 amplitudes to HCs (Palmwood et al., 2017; Ruchow et al., 2008). Although attenuated P3 amplitudes have been reported in depression (Houston et al., 2003; Nan et al., 2018), both enhanced and attenuated P3 amplitudes are found in anxiety (Bechor et al., 2019; Éismont et al., 2009; Sehlmeier et al., 2010; Wauthia et al., 2022; Xia et al., 2020; Xu et al., 2014). Furthermore, depressive symptoms are found to drive associations between attenuated P3 amplitudes and anxiety in female adolescents (Santopetro et al., 2022). This may provide one explanation for the inconsistent findings reported in the P3 and anxiety literature, while also highlighting the importance of simultaneously examining both anxiety and depression symptoms.

Response monitoring is described as the ability to evaluate and adjust one's own behavior in order to meet a predefined goal and optimize performance (Thakkar et al., 2008). As described in Chapter 1, behavioral markers of response monitoring, in the form of post-error slowing, are typically comparable to HCs in individuals with EDs (Geisler et al., 2017; Suttikus et al., 2021; Wierenga et al., 2014) and internalizing disorders (Carrasco et al., 2013; Hum et al., 2013a; Ladouceur et al., 2006; Ladouceur et al., 2012).

Most research in this area focuses on neural measures of response monitoring, indexed by ERN/CRN and Pe amplitudes. Although response monitoring has not been investigated in relation to DE and EDs in children, adults with AN displayed attenuated ERN amplitudes compared to HCs during a Flanker task (Pieters et al., 2007). Children, adolescents (Ladouceur et al., 2012; Weinberg, Liu, et al., 2016) and adults (Schrijvers et al., 2008; Weinberg, Kotov, et al., 2015) with depressive symptoms are also reported to display attenuated ERN amplitudes compared to HCs. However, enhanced ERN amplitudes are reported to be a biomarker for anxiety in older children and adolescents (Meyer, 2017; Meyer et al., 2012). The Pe and CRN are less widely studied response monitoring ERPs in the internalizing disorder and ED literature, especially within non-clinical populations. Therefore, my analysis of these components will be exploratory.

This novel investigation aimed to address the lack of research currently examining the associations between behavioral and neural correlates of cognitive control and co-occurring DE and internalizing symptoms in preadolescence. Specifically, I will examine whether cognitive control impairments are associated with DE independently of internalizing symptoms, or whether internalizing symptoms mediate the relation between these processes and DE. To do this, a Go/NoGo task will be used alongside EEG recording, to study both behavioral and neural indicators of response inhibition and response monitoring. This is important given the perseverance of task performance previously found alongside neural differences in adolescents with internalizing symptoms and DE. My hypotheses were:

- 1) There will be no significant associations between NoGo accuracy and anxiety, depression, and DE behaviors.
- 2) There will be no significant associations between post-error slowing and anxiety, depression, and DE behaviors.
- 3) Increased levels of DE and internalizing symptoms will be positively correlated with neural markers of impaired response inhibition (in the form of less positive P3 amplitudes and more negative N2 amplitudes on NoGo trials).
- 4) Increased levels of DE and internalizing symptoms will be positively correlated with impaired response monitoring.

Specifically, increased anxiety will be positively correlated with ERN amplitudes (i.e., greater anxiety symptoms reflected in more negative ERN amplitudes), while depression and DE will be negatively correlated with ERN amplitudes (i.e., greater depression symptoms reflected in less negative ERN amplitudes).

4.2 Methods

4.2.1 Participants

Details of sample, recruitment, and overall exclusion criteria are described in Chapter 3. In relation to task exclusionary criteria, fifteen children were excluded based on low task accuracy (Go Accuracy < 50%). Table 4.1 presents the demographics of the final sample once exclusion criteria were employed.

Table 4.1.*Demographics of the final sample.*

Child Demographics (N = 48)	<i>M</i> (Range)
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)	<i>M</i> (Range)
Age (years)	42.52 (29.90 – 61.20)
SES (WIMD) Quartile (%)	
1 st (most deprived)	18.8
2 nd	20.8
3 rd	18.8
4 th (least deprived)	41.7

Note. SES: Socio-economic status, WIMD: Welsh Index of Multiple Deprivation.

A post-hoc sensitivity analysis was conducted to determine the effect size that could be detected based on the collected sample size. Based on a two-tailed bivariate correlation with an alpha value of .05, power of .95, and sample size of 48, the critical $r = .285$.

4.2.2 Materials

4.2.2.1 Self-report Questionnaire Measures

The ChEAT (Maloney et al., 1989) and RCADS-25 (Muris et al., 2002) were used to measure levels of DE, anxiety, and depression, respectively. See Chapter 2 for full details on these measures.

4.2.2.2 *Go/NoGo Task*

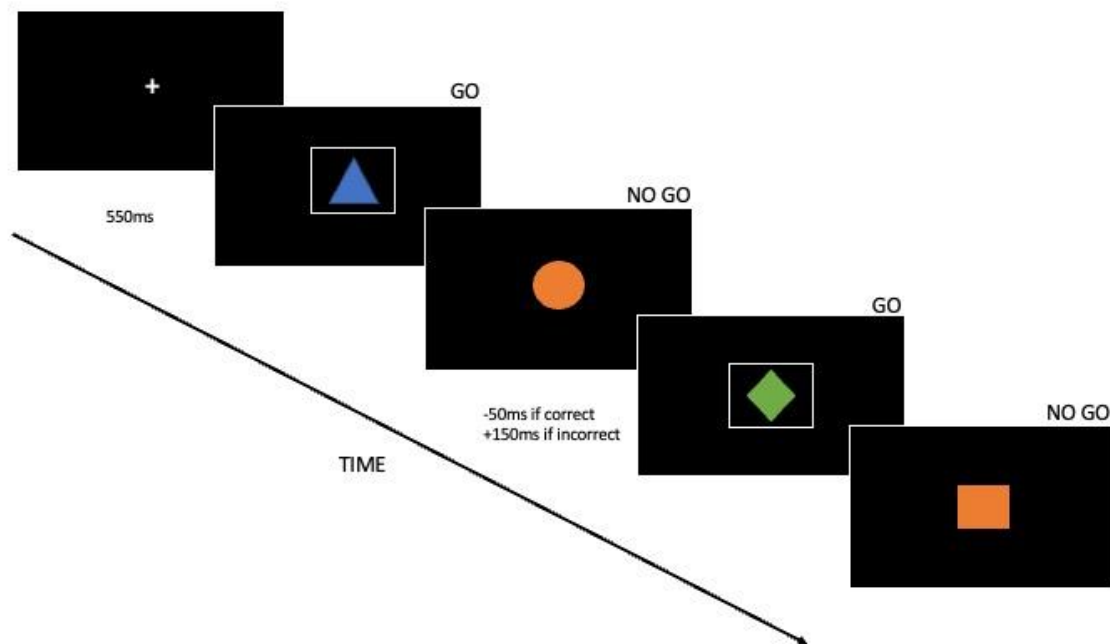
A modified version of the Go/NoGo task (Figure 4.1), a behavioral task used to elicit cognitive control abilities (Hare et al., 2005), was programmed and presented using E-Prime 2.0 software. In this task, children were presented with stimuli on a screen and asked to respond as fast and accurately as possible to a cue presented using a button box.

Children were presented with stimuli consisting 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. Participants were given verbal instructions before the task started, with visual instructions also included on the presentation screen. Checks of understanding were made by the experimenter before the task began.

Stimuli were edited to have a consistent black background and a size of 506 x 650 pixels. 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 550ms but this decreased by 50ms or increased by 150ms depending on the participant's performance on NoGo trials to ensure sufficient commission errors were available for data analysis (Hum et al., 2013a). The response window for NoGo trials was set to be 200ms longer than Go trials to ensure the non-response 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.

Figure 4.1.

A visual representation of the Go/NoGo task used in the study.



4.2.3 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 ActiCamp amplifier (Brain Products) was used and EEG activity was sampled at a rate of 500Hz. The reference channel was Cz and ground was placed over Fpz. Impedances were kept below 30 k Ω 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 (MathWorks, 2021). Data were initially band pass filtered at 0.3-100Hz and then re-referenced to the average activity of all the electrodes. Artifacts in the data were automatically identified using a threshold value of $\pm 200\mu\text{V}$, and then excluded from the data. Eye blinks were automatically identified as signals that met predefined thresholds of $>100\text{ms}$ rise time, $>150\text{ms}$ fall time, and $>125\mu\text{V}$ amplitude at electrodes Fp1 and Fp2, and visual inspection follow-up to ensure appropriate exclusion of blink trials. Practice trials and those with anticipatory responses (reaction times $<200\text{ms}$) were also removed from the data. The data was then low-pass filtered at 30Hz for ERP construction. Cleaned data were then segmented. Stimulus-locked ERPs (N2/P3) for Go and NoGo trials were segmented into 100ms pre-

stimulus baseline to 1000ms post-stimulus epochs. Response-locked ERPs for correct (Go) and incorrect (NoGo) responses were segmented into 100ms pre-response to 400ms post response epochs, and baseline-corrected using the 100ms pre-response time window.

The mean amplitude was calculated for each ERP in the channels Fz, Cz, and Pz. The N2 was scored from 240 to 340ms and P3 scored from 350 to 450ms in Fz. The Pe was scored from 200 to 350ms in Cz for correct and incorrect trials. The ERN/CRN was scored from 10 to 90ms, with the ERN scored on incorrect trials and the CRN used for correct trials. These time windows and electrode positions were established through comparison with previous literature using the Go/NoGo task in the same age range (e.g. Jonkman, 2006) as well as visual inspection of individual participant and grand mean plots. Mean amplitude was used in statistical analyses as this is reported to be a more robust measure of ERP waveforms than the peak amplitude (Clayson et al., 2013).

4.2.4 Behavioral Data Processing

Three behavioral outcomes were obtained from the data: Go accuracy (%), NoGo accuracy (%), and post-error slowing. The primary behavioral outcome measure of response inhibition performance was NoGo accuracy, which represents the proportion of responses successfully withheld. Lower levels of NoGo accuracy reflect poorer response inhibition performance. Go accuracy, defined as the proportion of correct responses to Go stimuli, reflects attention or vigilance on the task. Higher levels of Go accuracy represent increased attention. Lastly, post-error slowing was the main behavioral outcome measure of response monitoring performance. Once RTs were computed for correct Go and incorrect NoGo responses separately, post-error slowing was calculated as the difference between the average RT of correct Go responses following an incorrect NoGo response and the average RT of correct Go responses following a correct Go response (Mean RT post-error – Mean RT post-correct). Increased post-error slowing is thought to reflect an adaptive behavioral adjustment, where the individual is able to monitor their performance and take more caution after an error, and less caution after a correct response.

For overall behavioral data analysis, only responses made within 200 and 1200ms of each trial were included to exclude nondeliberate responses (based on the procedure used by Hum et al., 2013a). For calculation of post-error slowing, only correct Go responses that were followed by a correct Go response were included in the calculation of mean RT post-error, as

research suggests participants can display pre-error speeding, leading to an overestimation of post-error slowing (Pfister & Foerster, 2022).

4.2.5 Procedure

All participants in the study were accompanied to the laboratory session by a parent or guardian. First, the researcher took head circumference measurements and began capping the child while they completed an emotion recognition task (detailed in Chapter 5), as well as self-report questionnaires. Children who were recruited in stage 1 completed the ChEAT and RCADS-25 in their schools within small groups (detailed in Chapter 2). 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 30s sessions in which participants were instructed to alternate between opening and closing their eyes. Overall, the tasks lasted approximately 10 minutes. In addition to this Go/NoGo task, the child completed an emotional version of the Go/NoGo task (detailed in Chapter 5). The order of these two tasks was counterbalanced across participants. Once the Go/NoGo tasks were finished, the child completed a set shifting task (detailed in Chapter 3). As compensation for their time, each child received a gift voucher and a small gift.

4.2.6 Statistical Analyses

All statistical analyses were conducted using SPSS (version 27.0; IBM, 2020). All participants were included in analyses of questionnaire and behavioral data. One participant was excluded from all ERP analyses due to excessive EEG artifacts and five participants were excluded from analyses involving response ERPs based on a low number of commission errors (< 6). Research has suggested at least 6-8 error trials are needed to reliably quantify the ERN (Olvet & Hajcak, 2009). Data were screened for outliers prior to data analysis and final sample sizes are specified for each statistical test. 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.

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 counterbalance groups as well as the order of the two Go/NoGo tasks. Unless specified, untransformed data are presented in tables. Homogeneity of variance was assessed by Levene's test for equality of variances. Where this was violated, comparative non-parametric tests were used e.g., Mann-Whitney U. Bonferroni correction was used to adjust for multiple comparisons.

To test my primary hypotheses and investigate the associations between DE, internalizing symptoms, and measures of cognitive control, Pearson's r correlations were conducted. Where significant correlations were present between DE and behavioral/ERP measures of cognitive control, follow-up hierarchical regression analyses were conducted to examine the association between DE and cognitive control, while controlling for anxiety and depression. If significant correlations were present between cognitive control measures and both DE and internalizing symptoms, hierarchical regressions were followed by mediation analysis to examine whether internalizing symptoms mediated the relation between DE and cognitive control. Multicollinearity was tested using the variance inflation factor and was at an acceptable level (Neter et al., 1985), unless reported otherwise.

4.3 Results

4.3.1 Questionnaire Measures

The majority of participants fell within normative ranges on the questionnaires. Although there were no significant gender differences, girls reported slightly higher scores on RCADS anxiety and depression subscales compared to boys. Boys reported higher ChEAT scores than girls; however, girls displayed more variability in their scores (Table 4.2).

Table 4.2.*Descriptive statistics for ChEAT, RCADS anxiety, and RCADS depression, split by gender.*

	Whole Sample (n = 48)		Male (n = 27)		Female (n = 21)	
	M (SD)	Min - Max	M (SD)	Min - Max	M (SD)	Min - Max
ChEAT	60.65 (12.57)	35 - 100	61.48 (9.92)	45 - 84	59.57 (15.53)	35 - 100
RCADS Anxiety	11.44 (6.93)	2 - 32	11.04 (5.37)	2 - 23	11.95 (8.65)	2 - 32
RCADS Depression	7.44 (4.83)	0 - 20	7.26 (4.75)	0 - 20	7.67 (5.04)	2 - 20

Note. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

4.3.2 Behavioral Measures

Table 4.3 presents a summary of the Go/NoGo behavioral data for the sample (see Appendix E for detailed overview of behavioral performance, split by gender). A repeated measures ANOVA was conducted to examine the main effects of trial type on RT and accuracy. In line with previous studies, it revealed faster mean RTs on NoGo trials (incorrect responses only) compared to Go trials for the whole sample ($F(1, 47) = 28.56, p < .001, \eta_p^2 = .378$); however, there was not a significant difference in accuracy between Go and NoGo trials ($F(1, 47) = 1.81, p = .185, \eta_p^2 = .037$).

Table 4.3.*Go/NoGo behavioral data for the whole sample (N = 48).*

	M (SD)	Minimum - Maximum
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
Post error slowing (ms)	-5.99 (67.80)	-104.97 – 177.62

Note. RT = reaction time.

To examine my first and second hypotheses that there would be no significant associations between NoGo accuracy or post-error slowing and anxiety, depression, and DE behaviors, correlations were conducted between questionnaire and behavioral measures (Table 4.4, full results are provided in Appendix F). There were few significant correlations; however, RCADS depression scores positively correlated with NoGo accuracy ($p = .002$) and negatively correlated with post-error slowing ($p = .011$). ChEAT and RCADS anxiety scores were not significantly associated with NoGo accuracy nor post-error slowing.

Table 4.4.

Pearson correlations between questionnaire measures and Go/NoGo behavioral performance.

	ChEAT Total	RCADS Anxiety	RCADS Depression
NoGo Accuracy (%)	.055	.263	.442**
Post-error slowing	-.174	-.098	-.362*

Note. * $p < .05$ level, ** $p < .01$. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

4.3.3.1 Stimulus-locked ERPs (N = 48)

Figure 4.2 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. Although ERP amplitudes were the focus of this thesis, I report N2 and P3 latencies in Appendix G for transparency. Table 4.5 presents trial counts for conditions of interest specific to stimulus-locked ERPs.

Figure 4.2.

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

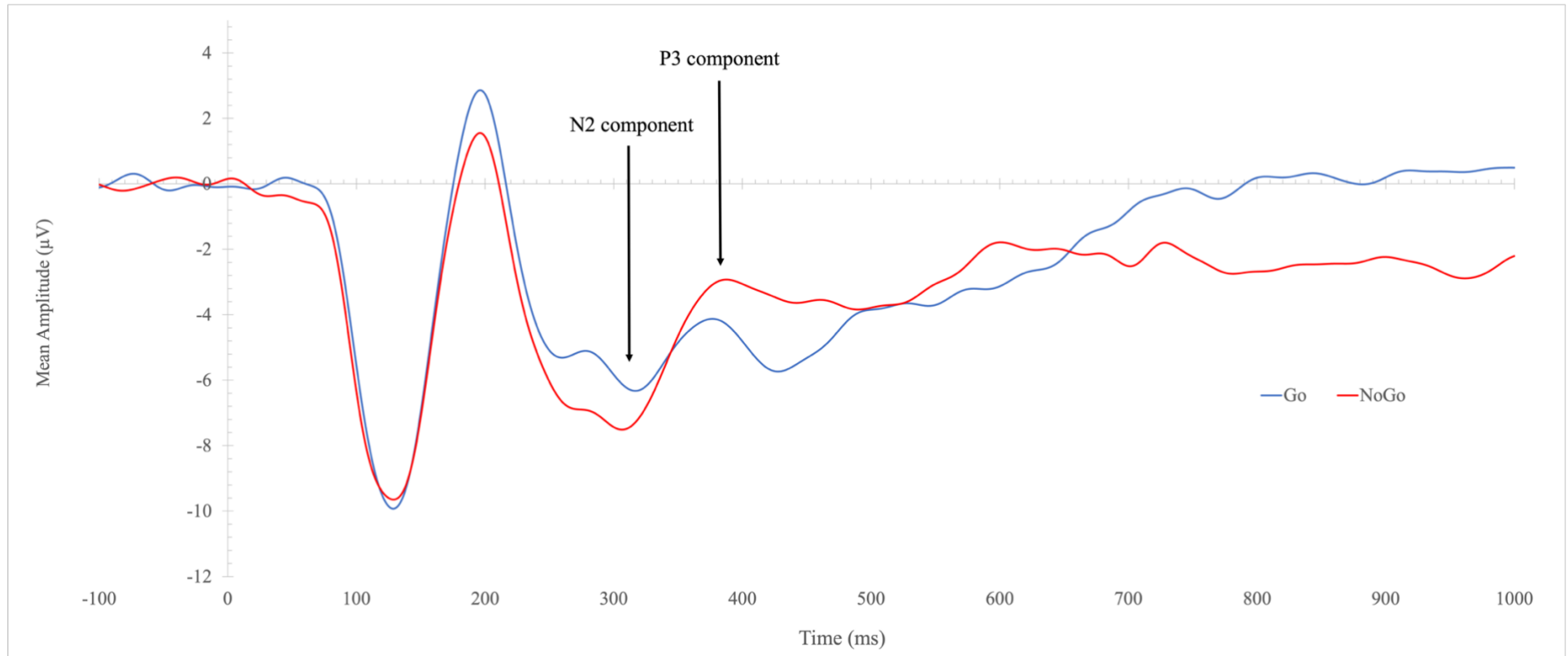


Table 4.5.

Average trial counts used in ERP analyses for Go and NoGo conditions (N = 48).

	M (SD)	Minimum - Maximum
Go	55.56 (18.70)	16.00 – 94.00
NoGo	28.04 (9.13)	7.00 – 40.00

Correlational analyses were conducted to test my third hypothesis that DE and internalizing symptoms would be associated with attenuated P3 amplitudes and enhanced N2 amplitudes on NoGo trials. Exploratory analyses were conducted to examine correlations between questionnaire measures and P3 and N2 amplitudes on Go trials. As shown in Table 4.6, 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, while small to moderate effect sizes were found for associations with ChEAT scores ($P3_{Go}$: $r = -.273$, $p = .060$; $P3_{NoGo}$: $r = -.204$, $p = .164$). Bonferroni corrections were used for these exploratory analyses, with associations between $P3_{Go}$ amplitudes and internalizing symptoms remaining significant for depression symptoms only (corrected alpha level = .017).

Table 4.6.

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

	ChEAT	RCADS Anxiety	RCADS Depression
$N2_{Go}$	-.153	-.215	-.122
$N2_{NoGo}$	-.083	-.182	-.156
$P3_{Go}$	-.273	-.288*	-.353*
$P3_{NoGo}$	-.204	-.317*	-.392**

Note. * $p < .05$ level, ** $p < .01$. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

4.3.3.2 Response-locked ERPs ($N = 44$)

Figure 4.3 presents grand mean response-locked waveforms for correct and incorrect responses. As expected, both the ERN and Pe components were larger for incorrect responses compared to correct responses. The CRN, present for correct responses, displayed a smaller peak in negative amplitude compared to the ERN. Although ERP amplitudes were the focus of this thesis, I report ERN/CRN and Pe latencies in Appendix H for transparency. Table 4.7 presents trial counts for conditions of interest specific to response-locked ERPs.

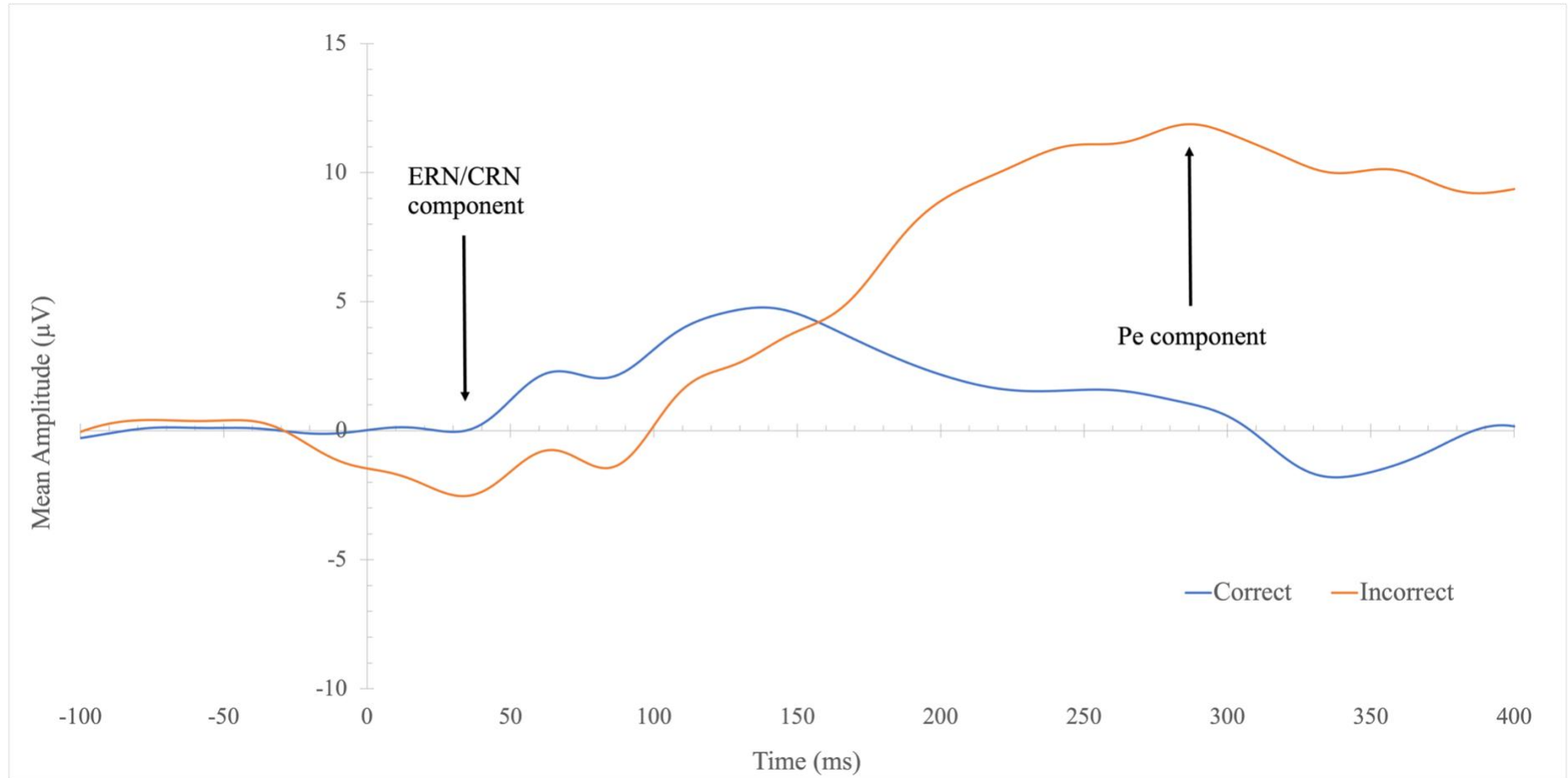
Table 4.7.

Average trial counts used in ERP analyses for correct and incorrect responses ($N = 44$).

	M (SD)	Minimum - Maximum
Correct	60.84 (17.37)	27.00 – 95.00
Incorrect	8.66 (3.67)	6.00 – 21.00

Figure 4.3.

Grand mean response-locked waveforms for correct and incorrect responses at Cz.



Correlational analyses were conducted to test my fourth hypothesis that higher levels of DE and depression would be associated with attenuated ERN amplitude whilst higher levels of anxiety would be associated with elevated ERN amplitudes. Exploratory analyses were conducted to examine the correlations between questionnaire measures and both CRN and Pe amplitudes. There were no significant correlations present between questionnaire measures and mean amplitudes for response-locked ERPs (Table 4.8).

Table 4.8.

Correlations between questionnaire measures, ERN/CRN, and Pe mean amplitudes.

	ChEAT	RCADS Anxiety	RCADS Depression
CRN	.061	.050	-.058
ERN	.108	.093	.114
Pe _{Correct}	.126	-.063	-.071
Pe _{Incorrect}	-.015	-.127	.018

Note. ChEAT: Children’s Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale, CRN: correct-related negativity, ERN: error-related negativity, Pe: error positivity.

Transformed data were used in the analyses.

4.4 Discussion

I have explored whether the co-occurrence of internalizing symptoms and DE in preadolescents is driven in part by shared impairments in cognitive control, such as response inhibition and response monitoring. Overall, my findings suggest there is some divergence between behavioral and neural evidence, particularly for response inhibition. Behaviorally, there were significant associations between depression and enhanced response inhibition, through increased NoGo accuracy on the Go/NoGo task. In contrast, at a neural level, depression was associated with impaired response inhibition i.e., attenuated P3_{NoGo} amplitudes.

While there were no significant associations between both DE and anxiety and behavioral markers of response inhibition (i.e., NoGo accuracy) or response monitoring (i.e., post-error slowing), increased depression was associated with greater inhibition through

increased accuracy on NoGo trials. This is inconsistent with my hypothesis that there would be no associations between internalizing symptoms and behavioral markers of response inhibition. Using non-emotional tasks, some investigations of preadolescents have found significant associations between symptoms of depression and anxiety and poorer accuracy on a Go/NoGo task (Shanok et al., 2021), while others report preadolescents with internalizing symptoms do not differ in response inhibition performance compared to HCs (Brunnekreef et al., 2007). However, it is not clear from the literature whether depression symptoms have been previously linked to enhanced response inhibition. In my sample, increased depression was associated with less post-error slowing, suggesting there was a reduction in response monitoring behaviorally that has previously been shown in neural activity, such as studies reporting blunted ERN amplitudes in individuals with depression (Ladouceur et al., 2012; Meyer et al., 2018; Schrijvers et al., 2008; Weinberg, Dieterich, et al., 2015; Weinberg, Liu, et al., 2016).

In my study, P3 and N2 amplitudes were used as neural markers of response inhibition during Go/NoGo performance. In line with my hypothesis, I found associations between increased anxiety and depression, and attenuated P3 amplitudes, indicative of impaired evaluation of environmental inconsistency or conflict (e.g., a NoGo trial; Bruin et al., 2001). My findings are consistent with P3 amplitude reduction reported in depression (Houston et al., 2003; Nan et al., 2018) and anxiety (Bechor et al., 2019; Éismont et al., 2009; Wauthia et al., 2022; Xu et al., 2014), potentially reflecting an association between P3 attenuation and a more global measure of internalizing problems (e.g. Bernat et al., 2020), rather than specific traits.

A similar association was found between increased DE and attenuated P3, but these effects were not significant. Comparisons with previous research examining associations between DE and N2/P3 components are limited; however, adults with AN are reported to display smaller P3 amplitudes in a stop-signal task compared to HCs (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). Although these findings suggest neural markers of response inhibition difficulties may be present across a spectrum of eating difficulties, they may be spurious given the researchers did not control for co-occurring anxiety, which could be driving these significant associations.

The non-significant association between N2 amplitudes and DE are consistent with Yue et al. (2020), who found no group differences in N2 amplitude between individuals with AN and HCs. Methodological factors may also help explain the absence of associations

between N2 amplitudes and both DE and internalizing symptoms. Individuals with anxiety have been shown to display elevated N2 amplitudes (Hoyniak & Petersen, 2019; Valadez et al., 2021; Yu et al., 2018), which are thought to represent increased conflict detection and elevated engagement in cognitive control processes (Dennis & Chen, 2009). However, most studies reporting enhanced N2 amplitudes in anxiety use emotionally salient stimuli, while some studies using non-emotional stimuli, like my study, do not report this enhancement (Baving et al., 2004; Larson et al., 2013; Voegler et al., 2018). These findings suggest enhancement of N2 amplitudes may be specific to emotionally salient stimuli and may help explain the absence of significant associations in my sample.

Overall, my findings regarding response monitoring ERPs did not support my hypothesis that higher levels of DE and depression would be associated with attenuated ERN amplitude whilst higher levels of anxiety would be associated with elevated ERN amplitudes. These results are surprising given that several studies have found greater ERN amplitudes in children with anxiety (Moser et al., 2013), as well as individuals who present with traits common in EDs, such as obsessive-compulsive behaviors (Hajcak et al., 2008; Hajcak & Simons, 2002; Santesso et al., 2006a). However, Moser et al. (2013) note that not all studies describing enhanced ERN amplitudes in children with clinical anxiety disorders find significant relations between dimensional anxiety symptoms and ERN amplitude. More recently, a study investigating relations between dimensional self-report psychiatric symptoms and ERN amplitudes in adults were also unable to replicate prior ERN findings (Seow et al., 2020). This raises the importance of considering dimensional vs. categorical approaches to capturing mental health difficulties and their relations to cognitive biomarkers, such as the ERN.

There also appears to be a distinction between internalizing symptoms, as enhanced ERN amplitudes are not consistently reported in clinical depression or depressive symptoms (Weinberg, Dieterich, et al., 2015). Instead, attenuated ERN amplitudes are frequently reported in depression (Ladouceur et al., 2012; Weinberg, Liu, et al., 2016). Therefore, this suggests there are opposing associations between internalizing traits and ERN amplitudes; with enhanced ERN amplitudes associated with anxiety and attenuated ERN amplitudes associated with depression. In addition, my findings did not provide support for research reporting attenuated ERN amplitudes in individuals with AN (Pieters et al., 2007). This suggests that ERN amplitude attenuation may be linked to factors inherent to the clinical ED, such as the impact of starvation on the brain, rather than DE behaviors.

As mentioned in Chapter 2 of the thesis, there are some limitations with using child self-report measures (Paulhus, 1991). However, measures used in this study were adapted and validated questionnaires for the age group recruited in this sample, and previous research has demonstrated self-report symptom scales are predictive of subsequent diagnoses (Shankman et al., 2009). In addition, extra support was provided for children who required help with comprehension and reading. Future research would benefit from multi-informant approaches to corroborate my findings and consider both self-report and parent/guardian-report assessments of mental health. This is important because associations between enhanced ERN/Pe amplitudes and anxiety in children without a clinical diagnosis appear to be specific to parent reports of anxiety, and not child reported anxiety (Meyer et al., 2012).

Additional executive functioning abilities, such as working memory, have been proposed to impact on cognitive control abilities (Weinberg, Dieterich, et al., 2015). These were not captured in the measures employed and IQ was not formally assessed in the sample. In addition, traits such as perfectionism and obsessive-compulsive behaviors were not explored in this study. However, they demonstrate a consistent overlap with DE (Drieberg et al., 2019; Serpell et al., 2006) and are linked to elevated cognitive control (Barke et al., 2017; Santesso et al., 2006a; Thomas et al., 2022). Therefore, future studies should consider integrating a broader range of neurocognitive measures to control for other aspects of executive functioning in cognitive control performance, while also considering the interplay of traits such as perfectionism and obsessive-compulsive behaviors.

The findings from Chapter 2 demonstrated strong associations between internalizing symptoms and DE in preadolescents; however, Chapter 3 described an absence of significant associations between behavioral correlates of set shifting and both DE and internalizing symptoms. I built upon this study in the current chapter by examining both behavioral and neural correlates of response inhibition and response monitoring in preadolescents. The results support this approach, through the divergence between behavioral and neural findings. At a behavioral level, depression, and to some degree DE, demonstrated associations with greater task performance. However, at a neural level, higher levels of anxiety and depression were associated with impaired response inhibition. As this association between attenuated P3 amplitudes and DE was not significant, it suggests that internalizing symptoms are important to consider when assessing response inhibition in DE. In Chapter 5, I will continue to explore the role of cognitive control in the co-occurrence of internalizing symptoms and DE by examining these processes in an emotional context. As emotional processing difficulties are a

key factor involved in DE, anxiety, and depression, cognitive control impairments may be more pronounced within this context.

Chapter 5. Neural correlates of emotion regulation in a preadolescent sample displaying disordered eating and internalizing symptoms.

5.1 Introduction

In Chapter 4, behavioral and neural correlates of response inhibition and response monitoring were examined in the context of their associations with DE and internalizing symptoms in my preadolescent sample. The study presented in this chapter aims to build upon the work described in Chapter 4 by exploring these cognitive control processes in the context of emotion. As described in Chapter 1, studies have demonstrated an overlap in regulatory processes used within cognitive control and emotion regulation (Braunstein et al., 2017; Holroyd & Coles, 2002; Niendam et al., 2012; Ochsner et al., 2004; Phan et al., 2005; Pruessner et al., 2020; Ridderinkhof et al., 2004). Cognitive control processes, such as response inhibition and response monitoring, enable us to monitor, evaluate, and adapt our emotional reactions to achieve a certain goal (Thompson, 1994). For example, response monitoring processes allow for detection and identification of conflict in the environment and are thought to reflect an emotional response to errors (Luu et al., 2000). Response inhibition then enables individuals to override their default emotional expressions in order to maintain goal-directed behavior (Pruessner et al., 2020), such as inhibiting a negative emotional response in a socially inappropriate context. Although emotion regulation difficulties are a well-documented factor involved in the development and maintenance of EDs (Bodell et al., 2019; Fox et al., 2013; Goossens et al., 2009; Harrison, Sullivan, et al., 2010; Henderson et al., 2021; Hilbert & Tuschen-Caffier, 2007; Svaldi et al., 2012), our understanding of the underlying cognitive processes and their neural correlates is limited.

Emotional Go/NoGo tasks have been used alongside EEG with children and adolescents to capture cognitive and neural markers of cognitive control in emotion regulation (Hum et al., 2013a, 2013b; Lewis et al., 2008; Lewis et al., 2006; Lewis et al., 2007; Zhang et al., 2016). These tasks use arbitrary Go/NoGo cues, such as gender, to avoid additional processing of the emotional expression and capture automatic emotion regulation processes. For example, Hum et al. (2013a) examined neural correlates of response inhibition (N2 amplitudes) and response monitoring (ERN/CRN amplitudes) during the presentation of angry, calm, and happy facial expressions. As described in Chapter 1, emotional Go/NoGo tasks in anxiety have found enhanced neural correlates of response inhibition during the presentation of both positive and negative stimuli (Hum et al., 2013a; Lewis et al., 2008), as

well as enhanced neural correlates of response monitoring (Hum et al., 2013a). For depression, some studies report reduced N2 and P3 amplitudes for positive emotions (Camfield et al., 2018), but others report enhanced P3 amplitudes (Zhang et al., 2016). Interestingly, these atypical neural markers are not accompanied by behavioral task impairments in children with anxiety (Hum et al., 2013a, 2013b; Lewis et al., 2008; Waters & Valvoi, 2009) or depression (Grunewald et al., 2015; Trinkl et al., 2015).

Although emotional Go/NoGo tasks have not been previously used in ED studies, attenuated early ERP components to emotional faces have been reported in adolescents with AN compared to HCs (Hatch et al., 2010; Sfarlea et al., 2016), indicating dysfunctions in automatic and perceptual processing. There is evidence that individuals with EDs may also present with dysfunctions in later and more elaborate processing. The P3, for example, is found to be smaller during emotional processing in individuals with AN (Hatch et al., 2010; Pollatos et al., 2008). Similar to individuals with anxiety and depression, these atypical neural markers are not accompanied by behavioral impairments in individuals with EDs (Hatch et al., 2010; Sfarlea et al., 2016).

Emotion regulation is also thought to be interconnected with other processes, such as emotion recognition, to enable successful social emotional functioning (Ochsner, 2008; Tottenham et al., 2011). Emotion recognition difficulties have been associated with impairments in emotion regulation in women with AN (Harrison et al., 2009), and these emotion recognition difficulties are well-established in EDs and DE in adults (Harrison et al., 2009; Harrison, Sullivan, et al., 2010; Harrison, Tchanturia, et al., 2010; Pollatos et al., 2008; Ridout et al., 2010; Ridout et al., 2012; Wallis et al., 2018; Zonnevijlle-Bendek et al., 2002). These findings suggest individuals with EDs may experience difficulties across a range of emotion processing domains, including emotion recognition, and these may contribute to difficulties in emotion regulation.

In sum, cognitive control processes, such as response inhibition and response monitoring, are thought to be involved in emotion regulation. Behavioral and neural correlates of response inhibition and response monitoring have been previously studied in children with anxiety and depression. However, to my knowledge, no study yet has examined behavioral and neural correlates of emotion regulation in children with DE. The current study aimed to address this gap by investigating associations between DE behaviors, internalizing symptoms, and both neural and behavioral correlates of emotion regulation in a community sample of preadolescents. Due to the overlap between automatic emotion regulation processes and emotion recognition (Ochsner, 2008), as well as the consistent emotion

recognition difficulties reported in EDs (Harrison et al., 2009), emotion recognition performance will also be assessed to further understand the cognitive mechanisms underlying regulation. In line with the overarching argument of the thesis, I will examine whether emotion regulation impairments are associated with DE independently of internalizing symptoms, or whether internalizing symptoms mediate the relation between these processes and DE. My hypotheses were:

1. There will be no significant associations between overall NoGo accuracy and anxiety, depression, and DE.
2. There will be no significant associations between overall post-error slowing and anxiety, depression, and DE.
3. Increased levels of DE will be negatively associated with emotion recognition performance.
4. Increased levels of DE and internalizing symptoms will be positively correlated with neural markers of impaired response inhibition (in the form of less positive P3 amplitudes and more negative N2 amplitudes on NoGo trials).
5. Increased levels of DE and internalizing symptoms will be positively correlated with impaired response monitoring. Specifically, increased anxiety will be positively correlated with more negative ERN amplitudes, while depression and DE will be positively correlated with less negative ERN amplitudes. The Pe and CRN are less widely studied response monitoring ERPs in the internalizing disorder and ED literature, especially within non-clinical populations. Therefore, my analysis of these components will be exploratory.

5.2 Methods

5.2.1 Participants

Details of sample, recruitment, and overall exclusion criteria are described in Chapter 3. In relation to task exclusionary criteria, ten children were excluded based on low task accuracy (Accuracy < 50%), resulting in a final sample of fifty-three participants (M age = 10.98; 56.6% male). Table 5.1 presents the demographics of the final sample once exclusion criteria were employed.

Table 5.1.*Demographics of the final sample.*

Child Demographics (N = 53)		<i>M</i> (Range)
Age (years)		10.98 (10.00 – 11.83)
Gender (male %)		56.6
Ethnicity (%)		
	White	83.0
	Mixed or Multiple Ethnic Groups	3.8
	Asian or Asian British	1.9
	Other Ethnic Group	7.8
	Black, African, Caribbean, or Black British	0.0
	Missing data	3.8
Parent Demographics (N = 53)		<i>M</i> (Range)
Age (years)		42.65 (29.90 – 61.20)
SES (WIMD) Quartile (%)		
	1 st (most deprived)	24.5
	2 nd	18.9
	3 rd	18.9
	4 th (least deprived)	37.7

Note. SES: Socio-economic status, WIMD: Welsh Index of Multiple Deprivation.

A post-hoc sensitivity analysis was conducted to determine the effect size that could be detected based on the collected sample size. Based on a two-tailed bivariate correlation with an alpha value of .05, power of .95, and sample size of 53, the critical $r = .271$.

5.2.2 Materials

5.2.2.1 Self-report Questionnaire Measures

The ChEAT (Maloney et al., 1989) and RCADS-25 (Muris et al., 2002) were used to measure levels of DE, anxiety, and depression, respectively. See Chapter 2 for full details on these measures.

5.2.2.2 *Go/NoGo Task*

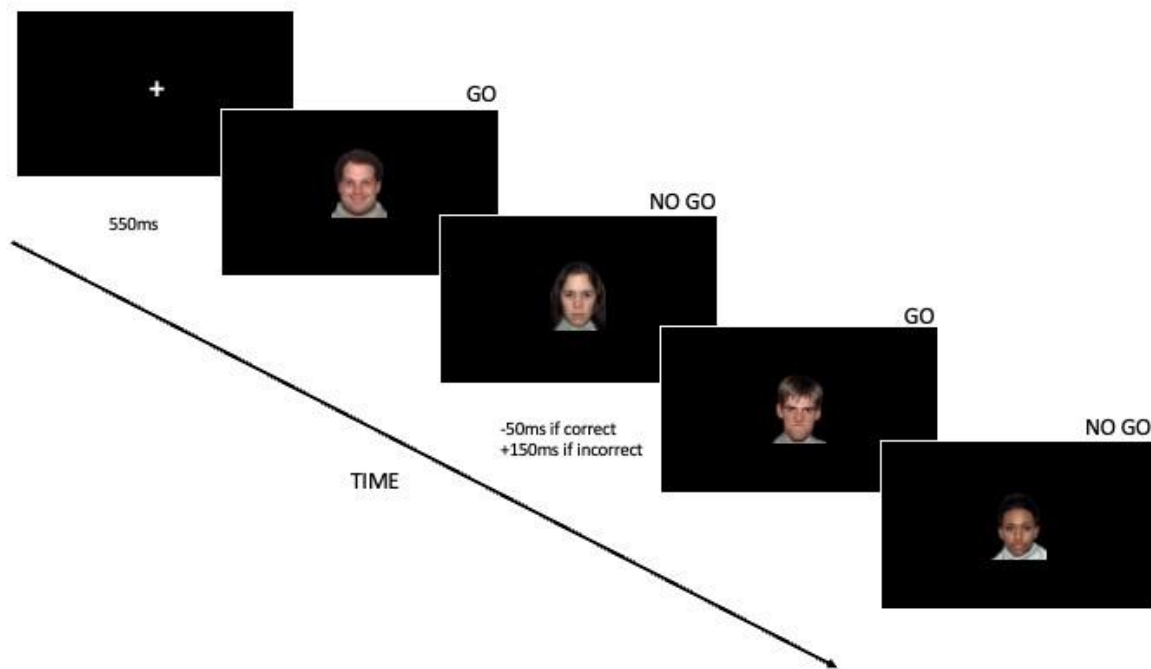
An emotional version of the Go/NoGo task described in Chapter 4 was programmed and presented using E-Prime 2.0 software (Figure 5.1). The design of this task was based on the emotional Go/NoGo task used by Hum et al. (2013a). Children were presented with NimStim face stimuli on a screen (Tottenham et al., 2009), which included four female models and four male models depicting angry, calm and happy emotions (closed mouth only). They were asked to respond as fast and accurately as possible to a cue presented using a button box.

Gender was the Go/NoGo cue, and this was counterbalanced across participants. Children were asked to respond when a male face appeared (Go trials) and not respond to a female face (NoGo trials), or vice versa according to the counterbalance condition. Participants were given verbal instructions before the task started, with visual instructions also included on the presentation screen. Checks of understanding were made by the experimenter before the task began.

Facial stimuli were edited to have a consistent black background and a size of 506 x 650 pixels. 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 550ms but this decreased by 50ms or increased by 150ms depending on the participant's performance on NoGo trials to ensure sufficient commission errors were available for data analysis (Hum et al., 2013a). The response window for NoGo trials was set to be 200ms longer than Go trials to ensure the non-response 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.

Figure 5.1.

A visual representation of the emotional Go/NoGo task used in the study.



Behavioral outcome measures were calculated as described in Chapter 4. However, accuracy rates were calculated for total trials and each emotion separately. Although RTs were also calculated using this procedure, I collapsed RTs across emotions when computing post-error slowing due to the low number of commission errors across the different emotions.

5.2.2.3 Emotion Recognition Task

The stimuli were photographs of two female models selected from the NimStim Face Stimulus Set (models 09 and 10; Tottenham et al., 2009) displaying expressions of happiness, sadness, and anger, as well as a neutral face. For each model, the three emotion expressions were morphed with the neutral face to create 10 levels of intensity, ranging from 10 to 100% (see Gao & Maurer, 2010) (Figure 5.2). This resulted in 33 images for each model (3 expressions x 10 intensities + 3 neutral faces). Each image was printed in color (size: 9.5 x 12cm), mounted onto card and laminated.

Figure 5.2.

Stimuli used in the emotion recognition task.



Note. Children sorted photographs of sad (A), happy (B), and anger (C) expressions at increasing 10% intensity levels from neutral to sad, happy, or anger. Adapted from “Altering Facial Movements Abolishes Neural Mirroring of Facial Expressions,” by K. Birch-Hurst, 2021, *Cognitive, Affective, & Behavioral Neuroscience*. Copyright 2021 by K. Birch-Hurst et al. Reprinted with permission.

This task was based on the procedure used by Gao and Maurer (2010). Children were asked to place the cards, one-by-one, into one of four boxes corresponding to each emotion (happy, angry, sad, or calm). Each box was marked with a schematic face on the front and children were provided with prompts if they were struggling to choose a box (e.g., ‘try to go with your first thought’ and ‘try not to think about it too much’).

I used emotion recognition scores described by Birch-Hurst et al. (2021). *Mean accuracy* scores were calculated for each emotion, as well as an overall mean accuracy score. This was computed by averaging across each participants’ accuracy for happy, angry, and sad expressions. *Sensitivity* was scored as the minimal intensity levels at which each participant was able to correctly label the expressive faces, e.g., if they correctly identified both models displaying an intensity of 20% and above for happy expressions, their sensitivity score for happy would be 20.

5.2.3 EEG Data Acquisition and Processing

Full details of EEG acquisition and processing used for both Go/NoGo tasks are provided in Chapter 4. However, for the emotion Go/NoGo task, stimulus-locked ERPs

(N2/P3) on Go and NoGo trials were split for each of the three different emotions in the task (happy, angry, and calm). Response-locked ERPs were computed for correct (Go) and incorrect (NoGo) responses only. I collapsed across emotions for response-locked ERPs, due to the likelihood of insufficient trial counts to examine response ERP components for each emotion (Hum et al., 2013a).

The mean amplitude was calculated for each ERP in the channels Fz, Cz, and Pz. The N2 was scored from 300 to 400ms and P3 scored from 480 to 600ms in Fz. In addition to separate stimulus-locked Go- and NoGo-ERPs, difference waveforms were computed for P3 and N2 ('P3d', 'N2d'), with NoGo amplitudes minus Go amplitudes (e.g., $P3d = P3_{NoGo} - P3_{Go}$ amplitudes). This procedure is used to isolate the unique effects of NoGo-ERPs by controlling for effects common across both Go and NoGo trials (Bekker et al., 2005; Gajewski & Falkenstein, 2013).

The response-locked ERPs were scored as the mean amplitude averaged across Fz and Cz. For the ERN/CRN this was from 10 to 90ms, while the Pe was scored from 200 to 350ms. These time windows were established through comparison with previous literature within the same age range (Davies et al., 2004; Hum et al., 2013a; Santesso et al., 2006b; Taylor et al., 2001) and visual inspection of individual participant and grand mean plots.

5.2.4 Procedure

Full details of the testing session are provided in Chapters 3 and 4.

5.2.5 Statistical Analyses

All statistical analyses were conducted using SPSS (version 27.0; IBM, 2020). One participant was excluded from all ERP analyses due to excessive EEG artifacts, and one participant was excluded from analyses involving response ERPs based on a low number of commission errors (< 6). Approach to data cleaning, normality testing, statistical corrections, and preliminary analyses was as described in Chapter 4.

Gender was added as a covariate in both Go/NoGo and emotion recognition behavioral analyses as there were significant differences found between boys and girls for both NoGo accuracy and recognition accuracy for sad emotional expressions. Age was added as a covariate in analyses involving stimulus-locked ERPs across all emotion conditions, as age was significantly associated with N2 and P3 amplitudes on Calm Go trials.

To test my primary hypotheses and investigate the associations between DE, internalizing symptoms, and measures of emotion regulation, Pearson's r correlations were conducted. Where significant correlations were present between DE and behavioral/ERP measures of emotion regulation, follow-up hierarchical regression analyses were conducted to examine the association between DE and emotion regulation, while controlling for anxiety and depression. If significant correlations were present between emotion regulation measures and both DE and internalizing symptoms, hierarchical regressions were followed by mediation analysis to examine whether internalizing symptoms mediated the relation between DE and emotion regulation. For regression analyses, multicollinearity was tested using the variance inflation factor and at an acceptable level (Neter et al., 1985), unless reported otherwise.

To further examine the relation between DE and stimulus-locked ERPs (P3, N2) across different emotions, repeated-measures ANCOVAs were performed with ChEAT included as a covariate. The main effects of the emotion and trial-type presented were examined as well as interactions with ChEAT scores. Significant three-way interactions between emotion, trial type, ChEAT, and additional covariates on N2 and P3 components were further examined using the difference waveforms (N2d, P3d) in place of individual ERPs. This is in line with previous emotional Go/NoGo tasks used with adolescents to capture the N2_{NoGo} and P3_{NoGo} effects more clearly across different emotions (Sun et al., 2020).

5.3 Results

5.3.1 Questionnaire Measures

The majority of participants fell within normative ranges on the questionnaires. Although there were no significant gender differences, boys reported slightly higher scores across all measures. However, girls displayed more variability in their ChEAT and RCADS anxiety scores, exceeding the maximum scores reported by boys (Table 5.2).

Table 5.2.*Descriptive statistics for ChEAT, RCADS anxiety, and RCADS depression, split by gender.*

	Whole Sample (n = 53)		Male (n = 30)		Female (n = 23)	
	M (SD)	Min - Max	M (SD)	Min - Max	M (SD)	Min - Max
ChEAT	58.40 (12.96)	35 - 100	59.43 (10.69)	41 - 84	57.04 (15.58)	35 - 100
RCADS Anxiety	11.19 (7.25)	1 - 32	11.37 (6.61)	1 - 23	10.96 (8.17)	2 - 32
RCADS Depression	7.36 (4.99)	0 - 20	7.43 (5.35)	0 - 20	7.26 (4.60)	2 - 20

Note. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

5.3.2 Behavioral Measures

5.3.2.1 Go/NoGo Task

Table 5.3 presents a summary of the Go/NoGo behavioral data for the sample (see Appendix I for detailed overview of behavioral performance, split by gender). A 3 Emotion (happy, angry, calm) x 2 Trial Type (Go, NoGo) repeated-measures ANCOVA was used to examine the effect of emotion and trial type on accuracy and RT, with gender as a covariate. For RTs, there was a significant main effect of trial type ($F(1, 52) = 5.145, p = .027, \eta_p^2 = .090$) but not emotion ($F(1.05, 54.56) = 1.322, p = .257, \eta_p^2 = .025$). In line with previous studies, significantly faster RTs were found on NoGo trials (incorrect responses only) compared to Go trials. For accuracy, there were no significant main effects of emotion ($F(2, 104) = .740, p = .480, \eta_p^2 = .014$) or trial type ($F(1, 52) = .298, p = .588, \eta_p^2 = .006$).

Table 5.3.*Go/NoGo behavioral data for the whole sample (N = 53).*

	M (SD)			
	Overall	Angry	Calm	Happy
Go Accuracy (%)	72.58 (19.18)	71.29 (19.39)	72.88 (18.90)	71.82 (20.30)
NoGo Accuracy (%)	70.89 (8.28)	72.10 (12.92)	69.29 (14.55)	68.75 (14.88)
Go Mean RT (ms)	440.70 (59.91)	443.30 (62.74)	438.07 (62.09)	440.68 (64.38)
NoGo Mean RT (ms)	372.95 (67.46)	377.16 (76.65)	366.80 (88.48)	375.00 (74.75)
Post error slowing (ms)	-33.40 (58.95)			

5.3.2.2 Emotion Recognition Task

Participants were generally consistent in their performance across each emotional expression, however both accuracy and sensitivity were higher for anger expressions compared to sadness and happiness (Table 5.4). A repeated measures ANCOVA was conducted to statistically test this by examining the main effect of emotion (happiness, anger, or sadness) on accuracy and sensitivity, with gender included as a covariate. For both accuracy ($F(2, 102) = 7.96, p < .001, \eta_p^2 = .135$) and sensitivity ($F(1.68, 85.70) = 5.243, p = .010, \eta_p^2 = .093$), there was a significant main effect of emotion, with significantly more sensitivity for anger expressions compared to sadness ($p = .012$) and happiness ($p < .001$).

Table 5.4.

Mean accuracy and sensitivity of each labelled emotional expression. Overall values are averaged across all three emotions.

	Happiness	Anger	Sadness	Overall
Mean Accuracy				
(% of correctly labelled photographs out of 20)	67.26 (8.00)	75.75 (7.68)	68.30 (11.64)	70.44 (5.54)
Sensitivity				
(Minimum intensity levels necessary to correctly identify emotional expression of both models)	46.23 (10.60)	37.36 (8.36)	43.96 (13.64)	42.52 (6.33)

Note. Standard deviations are in parentheses.

5.3.2 Associations between Questionnaire and Behavioral Measures

To test my first and second hypotheses that there would be no significant associations between total NoGo accuracy, post-error slowing and questionnaire measures, correlations were performed (Table 5.5; see Appendix J for correlations with detailed task performance). Exploratory analyses were conducted to examine correlations between questionnaire measures and NoGo accuracy split by emotion. There were no significant correlations between Go/NoGo performance and ChEAT scores, RCADS anxiety, and RCADS depression.

Table 5.5.*Correlations between questionnaire and Go/NoGo behavioral measures.*

	ChEAT	RCADS Anxiety	RCADS Depression
NoGo Accuracy (%)			
Total	-.026	.027	.103
Anger	-.026	.088	.162
Calm	-.099	-.037	-.002
Happy	.069	.098	.247
Post-error slowing (ms)	-.250	-.177	-.234

Note. ChEAT: Children’s Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

To test my third hypothesis that increased levels of DE would be negatively associated with emotion recognition performance, correlations were performed to examine associations between questionnaire measures and mean emotion recognition accuracy across happy, angry, and sad emotional expressions (Table 5.6). The only significant association was between higher ChEAT scores and lower mean recognition accuracy on happy trials ($r = -.337, p = .014$). As my hypothesis did not specify an emotion-specific effect, Bonferroni correction was used to adjust for multiple comparisons. The correlation between higher ChEAT scores and lower mean recognition accuracy on happy trials remained significant (corrected alpha level = .017).

Table 5.6.

Correlations between questionnaire and emotion recognition accuracy (%) for each emotion.

	ChEAT	RCADS Anxiety	RCADS Depression
Happy	-.337*	-.216	-.092
Anger	-.046	-.057	.075
Sad	-.040	.042	-.054

Note. * $p < .05$. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

The significant correlation between ChEAT scores and happy emotion recognition accuracy was further explored using a hierarchical multiple regression that controlled for anxiety and depression (Table 5.7). The model was significant at Step 1 and Step 2, with anxiety the only significant coefficient in the model. The addition of happy emotion recognition to the model did not contribute to a significant increase in the ΔF value, from Step 1 to Step 2. This means happy emotion recognition was not able to account for significant variability in ChEAT scores over and above internalizing symptoms alone.

Table 5.7.

Hierarchical multiple regression of happy face recognition (happy recog), anxiety, and depression on children's eating attitude test (ChEAT) scores.

Variable	<i>B</i>	95% CI for <i>B</i>		<i>SE B</i>	β	<i>t</i>	<i>p</i>
		<i>LL</i>	<i>UL</i>				
Step 1							
Constant	29.149	18.514	39.783	5.294		5.506	<.001
Anxiety	28.839	15.327	42.350	6.727	.631	4.287	<.001
Depression	.363	-12.855	13.581	6.581	.008	.055	.956
<i>R</i> ²	.405						
<i>F</i>	16.996						<.001
Step 2							
Constant	54.106	26.202	82.011	13.886		3.897	<.001
Anxiety	26.130	12.672	39.589	6.697	.571	3.902	<.001
Depression	1.272	-11.639	14.182	6.425	.028	.198	.844
Happy recog	-.342	-.697	.013	.177	-.211	-1.936	.059
<i>R</i> ²	.447						
<i>F</i>	13.202						<.001
ΔR^2	.042						
ΔF	3.747						.059

Notes. Steps were defined in the same hierarchical regression analysis; *B* = unstandardized regression coefficient; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; *SE B* = standard error of the coefficient; β = standardized coefficient; *R*² = coefficient of determination; ΔR^2 = *R* square change; ΔF = *F* value change.

Transformed data were used in the analyses.

5.3.3 ERP Measures

5.3.3.1 Stimulus-locked ERPs ($N = 52$)

Figure 5.3 presents grand mean stimulus-locked waveforms for each emotion (anger, calm, and happy) on Go and NoGo trials. As expected, both the N2 and P3 components were larger for NoGo trials, compared to Go trials, across most emotions. The exception was N2 amplitudes on calm trials, which were similar across Go and NoGo trials. Although ERP amplitudes were the focus of this thesis, I report N2 and P3 latencies in Appendix K for transparency. Table 5.8 presents trial counts for conditions of interest specific to stimulus-locked ERPs.

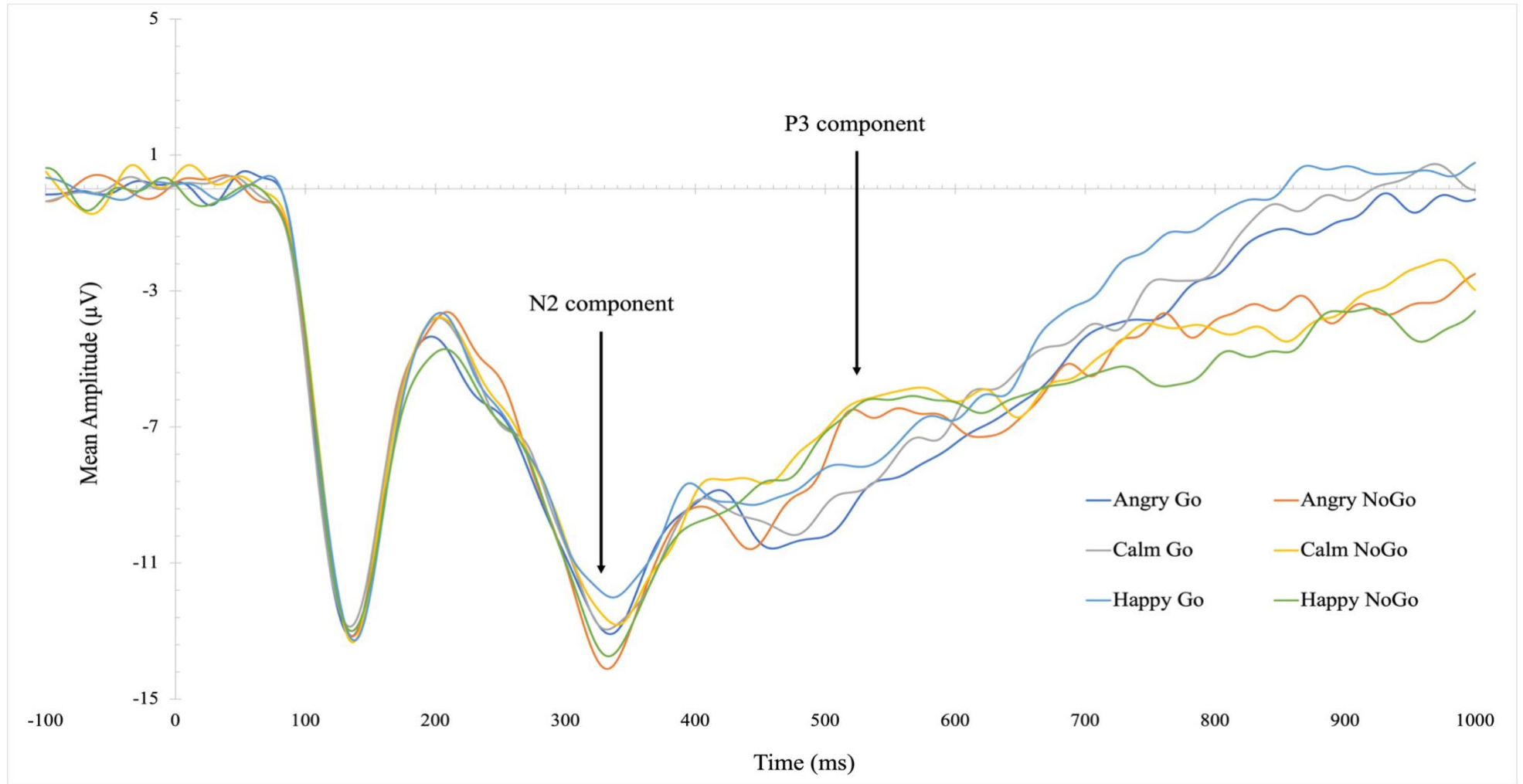
Table 5.8.

Average trial counts used in ERP analyses for Go and NoGo conditions across angry, calm, and happy emotions ($N = 52$).

	M (SD)	Minimum - Maximum
Angry Go	18.27 (6.26)	4.00 – 30.00
Angry NoGo	9.92 (2.44)	4.00 – 14.00
Calm Go	18.52 (6.57)	3.00 – 31.00
Calm NoGo	10.10 (2.78)	3.00 – 15.00
Happy Go	18.37 (6.95)	3.00 – 31.00
Happy NoGo	10.13 (3.22)	3.00 – 16.00

Figure 5.3.

Grand mean stimulus-locked waveforms for each emotion (anger, calm, and happy) on Go and NoGo trials at Fz.



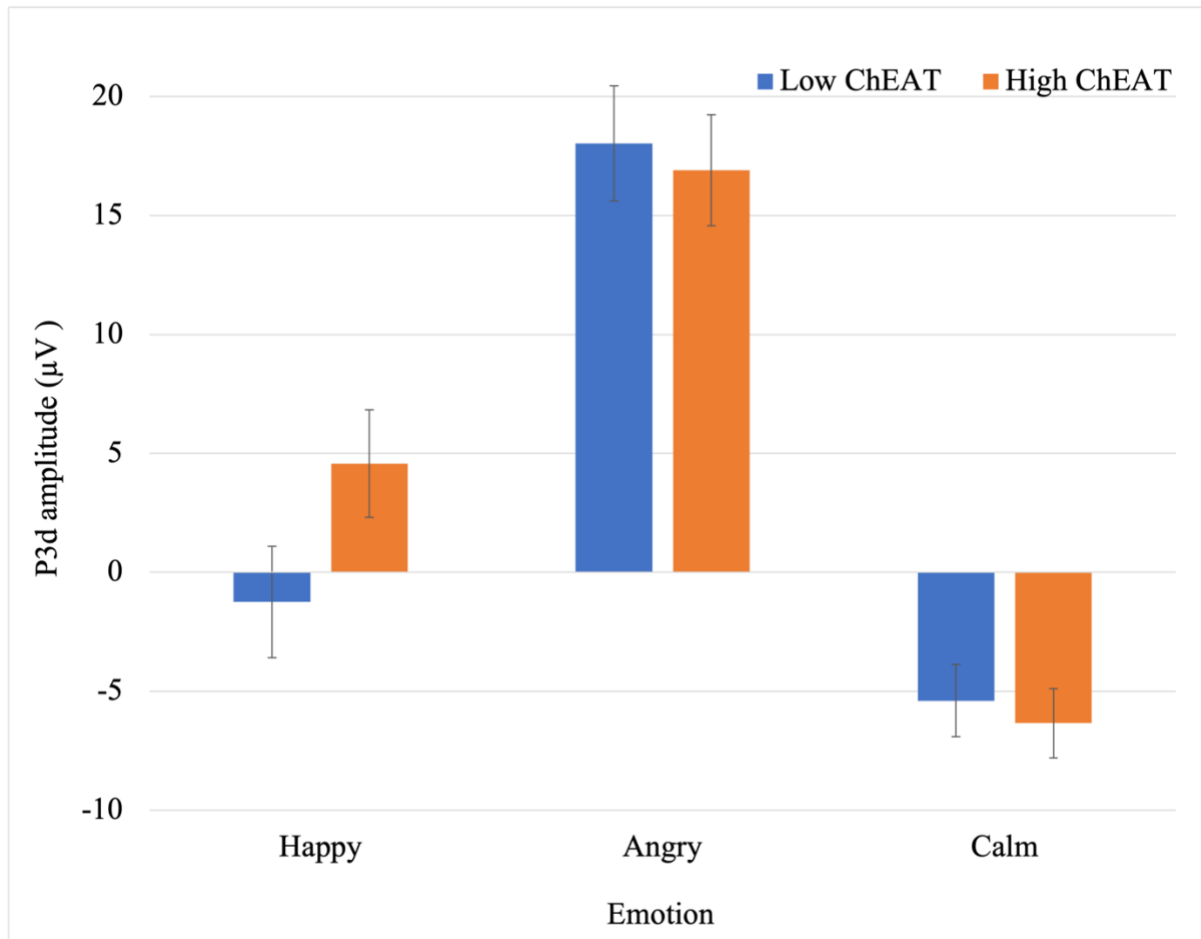
To examine my fourth hypothesis, that DE and internalizing symptoms would be associated with attenuated P3 amplitudes and enhanced N2 amplitudes on NoGo trials, I first conducted a 3 Emotion (happy, angry, calm) x 2 Trial Type (Go, NoGo) repeated-measures ANCOVA for each stimulus-locked ERP, with ChEAT score and age as covariates.

There were no significant interactions between N2 amplitudes, ChEAT score and: Emotion ($F(2, 98) = .052, p = .949, \eta_p^2 = .001$); Trial Type ($F(1, 49) = 1.156, p = .288, \eta_p^2 = .023$), or Emotion x Trial Type ($F(1.75, 85.49) = .176, p = .810, \eta_p^2 = .004$). Therefore, N2 amplitudes were not further explored in their relation to DE and internalizing symptoms. When these analyses were repeated for P3 amplitudes, there was a significant ChEAT score x Emotion x Trial Type ($F(2, 98) = 3.194, p = .045, \eta_p^2 = .061$) interaction. Larger P3 amplitudes were found on NoGo compared to Go trials when happy and angry faces were shown, but not calm.

As my hypothesis did not specify an emotion-specific effect, exploratory analyses were conducted to examine the relation between DE, internalizing symptoms, and P3 amplitudes by emotion. To further examine the three-way interaction between ChEAT score x Emotion x Trial Type and isolate the effect of NoGo trials from Go trials across each emotion, a P3 difference score was calculated ($P3d = P3_{NoGo} - P3_{Go}$ amplitudes). A repeated measures ANOVA was performed for P3d with ChEAT score and age as covariates. As expected, there was a significant interaction between ChEAT score x emotion ($F(2, 98) = 4.155, p = .019, \eta_p^2 = .078$). To visualize the interaction (Figure 5.4), the P3d wave for each emotion was split by high and low ChEAT groups (calculated using a median split). Both groups display similar amplitudes for angry and calm trials; however, on happy trials, there is an enhancement of the P3d amplitude in the high ChEAT group compared to the low ChEAT group.

Figure 5.4.

P3 difference wave ($P3_{NoGo} - P3_{Go}$) mean amplitudes on angry, happy, and calm trials, split by high and low Children's Eating Attitude test (ChEAT) group. Error bars = ± 1 SE.



In addition, there was a significant positive correlation between P3d amplitudes for happy trials and ChEAT scores ($r = .317, p = .022$). This demonstrates an enhancement in the $P3_{NoGo}$ effect with happy faces as DE increases. This significant correlation was followed by a hierarchical multiple regression (Table 5.9). Anxiety and depression were added into the model at Step 1 and P3d amplitudes were added at Step 2. The full model of P3d amplitudes, anxiety, and depression in relation to ChEAT was statistically significant; however, anxiety was the only significant coefficient at Step 1 and 2. In addition, the F -value did not significantly change between steps, suggesting P3d amplitudes were not able to account for significant variability in ChEAT scores over and above internalizing symptoms alone.

Table 5.9.

Hierarchical multiple regression of P3d amplitudes on Happy trials and anxiety, and depression on children's eating attitude test (ChEAT) scores.

Variable	<i>B</i>	95% CI for <i>B</i>		<i>SE B</i>	β	<i>t</i>	<i>p</i>
		<i>LL</i>	<i>UL</i>				
Step 1							
Constant	29.200	18.293	40.106	5.427		5.380	<.001
Anxiety	28.785	14.991	42.579	6.864	.626	4.193	<.001
Depression	.380	-12.993	13.752	6.654	.009	.057	.955
<i>R</i> ²	.399						
<i>F</i>	16.235						<.001
Step 2							
Constant	30.730	19.776	41.684	5.448		5.641	<.001
Anxiety	26.239	12.214	40.263	6.975	.570	3.762	<.001
Depression	1.214	-12.033	14.461	6.588	.027	.184	.855
P3d amplitudes	.190	-.060	.439	.124	.173	1.527	.133
<i>R</i> ²	.426						
<i>F</i>	11.894						<.001
ΔR^2	.028						
ΔF	2.331						.133

Notes. P3d: P3_{NoGo} – P3_{Go} difference wave for happy trials. Steps were defined in the same hierarchical regression analysis; *B* = unstandardized regression coefficient; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; *SE B* = standard error of the coefficient; β = standardized coefficient; *R*² = coefficient of determination; ΔR^2 = *R* square change; ΔF = *F* value change. Transformed data were used in the analyses.

There was also a significant negative correlation between P3d amplitudes on calm trials and anxiety ($r = -.405$, $p = .003$), depression ($r = -.318$, $p = .021$), and a moderate effect size for DE ($r = -.256$, $p = .067$). This suggests a smaller P3_{NoGo} effect with calm faces as anxiety and depression increase. Bonferroni correction was used to adjust for multiple comparisons in this exploratory analysis. The only remaining significant correlation after correction was between P3d amplitudes on calm trials and anxiety (corrected alpha level = .017).

5.3.3.2 Response-locked ERPs ($N = 51$)

Figure 5.5 presents grand mean response-locked waveforms for correct and incorrect responses. As expected, both the ERN and Pe components were larger for incorrect responses compared to correct responses. The CRN, present for correct responses, displayed a smaller peak in negative amplitude compared to the ERN. Although ERP amplitudes were the focus of this thesis, I report ERN/CRN and Pe latencies in Appendix L for transparency. Table 5.10 presents trial counts for conditions of interest specific to response-locked ERPs.

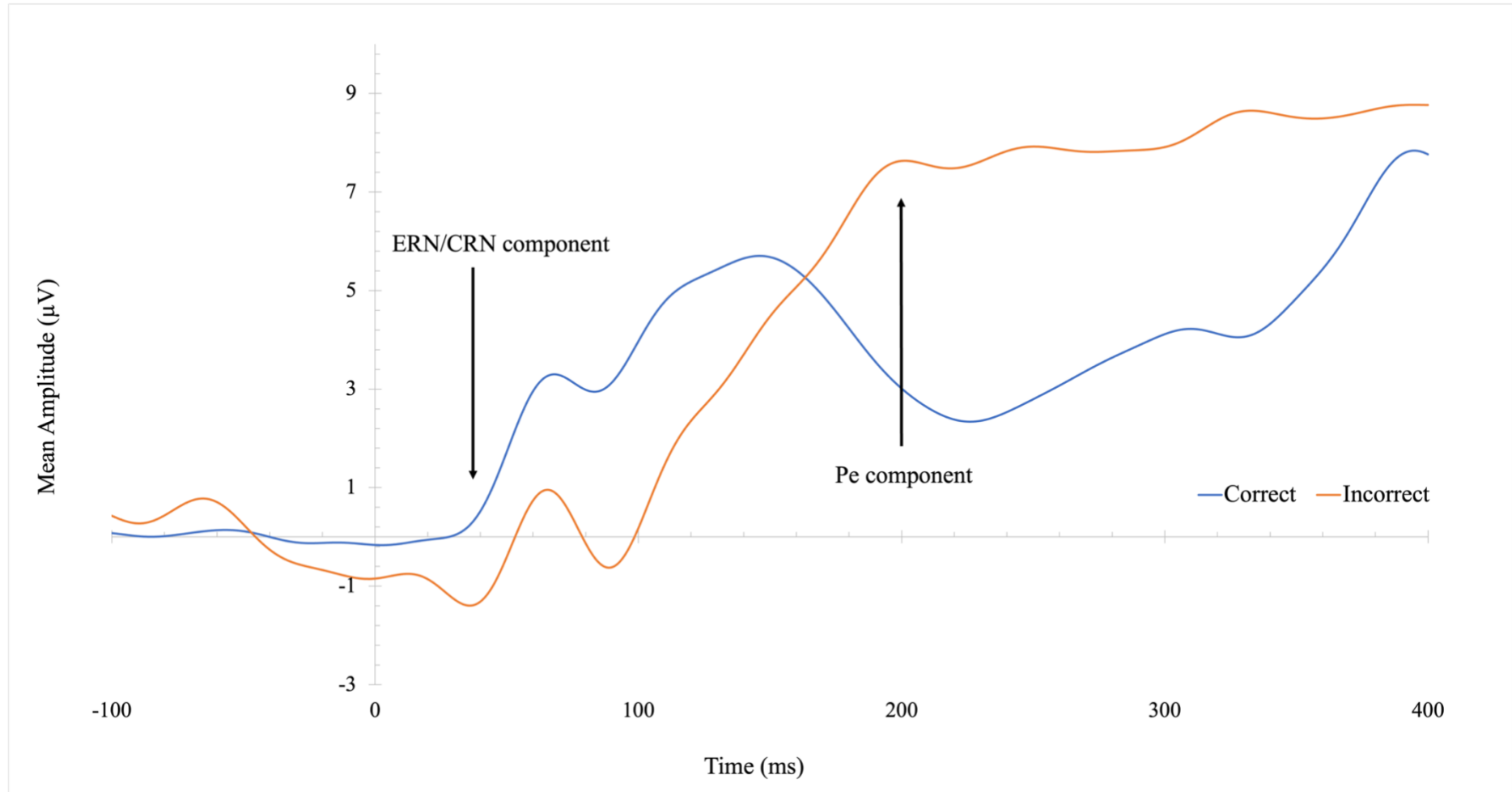
Table 5.10.

Average trial counts used in ERP analyses for correct and incorrect responses ($N = 51$).

	M (SD)	Minimum - Maximum
Correct	60.67 (17.35)	17.00 – 94.00
Incorrect	10.37 (2.90)	6.00 – 18.00

Figure 5.5.

Grand mean response-locked waveforms for correct and incorrect responses averaged across Fz and Cz.



Correlational analyses were conducted to test my final hypothesis that increased DE and depression symptoms would be associated with attenuated ERN amplitudes, whilst increased anxiety symptoms would be associated with increased ERN amplitudes. Exploratory analyses were conducted to examine the correlations between questionnaire measures and both CRN and Pe amplitudes. Table 5.11 presents correlations between questionnaire measures and mean amplitudes for response-locked ERPs. The only significant correlation was between attenuated ERN amplitudes and increased ChEAT scores ($p = .022$).

Table 5.11.

Correlations between questionnaire measures and ERN/CRN mean amplitudes averaged across Fz and Cz.

	ChEAT	RCADS Anxiety	RCADS Depression
CRN	-.018	-.099	-.081
ERN	.320*	.069	.016
Pe _{Correct}	-.135	-.127	-.232
Pe _{Incorrect}	.164	-.036	-.124

Note. $N = 51$. * $p < .05$ level. ChEAT: Children’s Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale, CRN: correct-related negativity, ERN: error-related negativity, Pe: error positivity.

Transformed data were used in the analyses.

The significant correlation between ERN amplitudes and ChEAT scores was followed by a hierarchical multiple regression (Table 5.12), with anxiety and depression added into the model at Step 1 and ERN amplitudes entered at Step 2. Anxiety was the significant predictor of ChEAT scores at Step 1. At Step 2, the full model was statistically significant and both anxiety and ERN amplitudes were significant predictors of ChEAT scores. As RCADS anxiety and depression subscales were not significantly correlated with ERN amplitudes, further mediation analyses were not conducted.

Table 5.12.

Hierarchical multiple regression of ERN amplitudes, anxiety, and depression on children's eating attitude test (ChEAT) scores.

Variable	<i>B</i>	95% CI for <i>B</i>		<i>SE B</i>	β	<i>t</i>	<i>p</i>
		<i>LL</i>	<i>UL</i>				
Step 1							
Constant	27.752	16.925	38.579	5.385		5.154	<.001
Anxiety	29.379	15.834	42.925	6.737	.638	4.361	<.001
Depression	.979	-12.154	14.112	6.532	.022	.150	.881
<i>R</i> ²	.427						
<i>F</i>	17.854						<.001
Step 2							
Constant	24.019	13.450	34.588	5.254		4.572	<.001
Anxiety	28.031	15.239	40.823	6.358	.609	4.408	<.001
Depression	1.652	-10.721	14.025	6.151	.037	.269	.789
ERN amplitude	1.325	.333	2.316	.493	.277	2.688	.010
<i>R</i> ²	.503						
<i>F</i>	15.853						<.001
ΔR^2	.076						
ΔF	7.223						.010

Notes. ERN: error-related negativity. Steps were defined in the same hierarchical regression analysis; *B* = unstandardized regression coefficient; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; *SE B* = standard error of the coefficient; β = standardized coefficient; *R*² = coefficient of determination; ΔR^2 = *R* square change; ΔF = *F* value change.

Transformed data were used in the analyses.

5.4 Discussion

In this chapter I explored the link between DE behaviors, internalizing symptoms, and both neural and behavioral correlates of emotion regulation in a community sample of preadolescents. This builds on Chapter 4 by examining cognitive control processes in the context of emotion. The first key finding from this study is the relation between increased DE and impaired happy face processing at a behavioral and neural level in preadolescence. The second key finding is the relation between increased DE and attenuated response monitoring, which was independent of internalizing symptoms. These findings suggest that although there is a strong association between DE and internalizing symptoms in preadolescence, the relations between DE and cognitive control difficulties do not appear to be fully accounted for by co-occurring internalizing symptoms.

In line with my first and second hypotheses, there were no significant associations between DE, internalizing symptoms, and behavioral markers of response inhibition (i.e., NoGo accuracy) or response monitoring (i.e., post-error slowing). My third hypothesis, that increased DE would be associated with poorer emotion recognition performance, was supported. However, the impairment was present for happy facial expressions only. Although previous research has identified emotion recognition deficits in individuals with EDs (Harrison et al., 2009; Harrison, Tchanturia, et al., 2010) and those with high levels of DE (Ridout et al., 2010), this is reported to be a global emotion deficit, rather than specific to happy faces. However, my findings are more consistent with evidence in individuals with AN showing a reduced capacity to process positive emotion expressions compared to HCs and individuals with obesity (Cserjési et al., 2011). This is the first study to examine emotion recognition performance in preadolescents with DE behaviors. The emotion recognition measure used in my study varied the intensity of expression by morphing from neutral to full expression; while previous studies have used static images of full expressions, using more complex stimuli may have given me the ability to identify a more nuanced relation between DE and emotion recognition.

Evidence of happy emotional processing impairments in preadolescents with increased DE was also present when examining neural measures, specifically the P3 amplitude. I calculated difference waves to isolate the unique effects of P3_{NoGo}, the neural marker of response inhibition, by controlling for effects common across both Go and NoGo trials (Bekker et al., 2005; Gajewski & Falkenstein, 2013). Individuals with higher levels of DE displayed enhanced P3d amplitudes for happy faces, but similar amplitudes to those with lower levels of DE for angry and calm faces. Moreover, this effect was independent of

internalizing symptoms. My findings are consistent with fMRI research, which reports individuals with AN display greater neural activity to increasing intensity of happy expressions compared to HCs, suggesting individuals with AN find these positive expressions more salient (Fonville et al., 2014). Research with the general population has proposed increased P3_{NoGo} amplitudes for happy faces reflect a natural tendency to approach positive and rewarding stimuli, making inhibition of responses more difficult and requiring more effort (Albert et al., 2012). However, the experience of happy emotional expressions as rewarding and positive may not be the case for people with EDs or patterns of DE. People with EDs avoid experiencing both negative *and* positive affect (Lampard et al., 2011), and show avoidance and difficulties in maintaining social relationships (Flament et al., 2001). This is more broadly reflected in an established link between EDs and social anhedonia (Dolan et al., 2022; Harrison et al., 2014; Tchanturia et al., 2012). Taken together, these findings suggest there may be additional factors involved in the relation between impaired processing of happy faces and DE, such as less interest and motivation in social interactions and potentially, finding these ‘approach’ behaviors from others threatening.

In line with my final hypothesis, I found increased DE was associated with attenuated ERN amplitudes, which remained when controlling for anxiety and depression. To the best of my knowledge there has only been one electrophysiological study that has examined ERN amplitudes in AN. Using a flanker task, Pieters et al. (2007) found attenuated ERN amplitudes in individuals with AN compared to HCs. It was proposed that this attenuation may reflect state-like factors linked to the severity of the disorder, such as reduced grey matter in the ACC (Pieters et al., 2007). However, as has been highlighted in studies with individuals with depression (Ruchow et al., 2006; Schoenberg, 2014), attenuated ERN amplitudes may reflect hypoactivity in central reward pathways (Ruchow et al., 2004). This is particularly interesting in the context of emotion stimuli, as anhedonia has also been linked to reduced ERN amplitudes in depression (Schrijvers et al., 2008). This early impairment in response monitoring of affective material may be linked to a disruption in reward processing in these areas, therefore leading to subsequent difficulties in later emotion processing, reflected in my P3 findings.

Participants committed a low number of errors for each emotion, which meant I was unable to compare response-locked ERPs by emotion. This limits the conclusions that can be drawn from these findings and whether the response-locked ERPs follow the emotion-specific effects found for stimulus-locked ERPs. In addition, the emotion recognition task

included female models only, however comparisons between counterbalancing orders showed no differences when males or females were the Go or the NoGo cue.

Future research should explore a wider range of emotions in the context of DE. For example, disgust has been frequently implicated in the development and maintenance of EDs (Fox & Froom, 2009; Fox & Harrison, 2008; Fox & Power, 2009; Harvey et al., 2002; Troop et al., 2002). Disgust can function as a threat-related emotion, potentially contributing to ED-related avoidance behaviors, such as food avoidance and calorie restriction (Anderson et al., 2021).

Positive emotions that go beyond happiness should also be explored, along with integration of information from the wider context, such as body cues. For example, emotional expressions that map onto positive and rewarding events, such as winning a game or completing a task, may provide us with more information about the processing of positive emotional stimuli in EDs. Additional co-occurring factors should also be considered in research examining at-risk samples, such as the effects of autistic traits and alexithymia, as both have previously been shown to be associated with emotion recognition difficulties in EDs (Brewer et al., 2015; Kerr-Gaffney et al., 2020).

In conclusion, the findings from this study provide a novel contribution to our understanding of emotion regulation and DE in preadolescents. Results suggest a relation between increased DE and impaired happy face processing at a behavioral and neural level in preadolescence. In addition, attenuated ERN amplitudes were found to be related to increased DE, and this relation was independent of internalizing symptoms. An early disruption in emotion regulation may be key to the development of DE behaviors and the potential for developing diagnosable EDs.

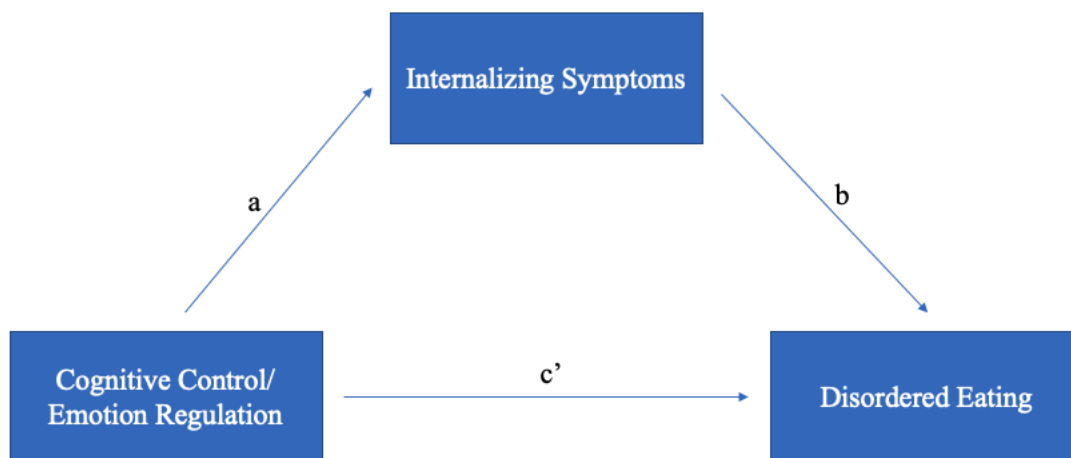
Chapter 6. General Discussion

6.1 Aims of Thesis

DE and internalizing symptoms commonly co-occur across development (Elran-Barak & Goldschmidt, 2021; Evans et al., 2017; Holm-Denoma et al., 2014; Thomas et al., 2021). In line with the model presented in Chapter 1 (Figure 6.1), this co-occurrence may reflect shared cognitive correlates, in the form of cognitive control processes. Impairments in cognitive control processes have been previously reported in children and adolescents with anxiety (Meyer, 2017; Meyer et al., 2012) and depression (Ladouceur et al., 2012; Meyer et al., 2018), as well as adults with DE (Steinglass et al., 2019; Svaldi et al., 2014; Wu et al., 2013) separately. However, little research has examined the relations between all three symptoms and cognitive processes in younger age groups, such as preadolescence.

Figure 6.1.

Diagram presenting the model examined in this thesis.



The aim of this thesis was to address this gap by exploring the associations between DE, internalizing symptoms, and cognitive control in a sample of typically developing preadolescents aged 10-11 years. In addition, I investigated whether associations between cognitive control and DE were independent of internalizing symptoms, or whether internalizing symptoms mediated the relation between cognitive control and DE. The main research questions that the thesis aimed to answer were:

- 1) Are DE and internalizing symptoms associated in preadolescence?

- 2) Are components of cognitive control, such as set shifting, response inhibition and response monitoring, associated with both DE and internalizing symptoms in preadolescence?
- 3) Do internalizing symptoms mediate the relation between DE and cognitive control?
- 4) Do internalizing symptoms mediate the relation between DE and emotion regulation?

Chapter 2 addressed question one by examining concurrent associations between DE and internalizing symptoms using self-report questionnaires in preadolescents. Chapters 3 and 4 extended these findings by considering the role of cognitive control in the co-occurrence of DE and internalizing symptoms, addressing questions two and three. Chapter 3 focused on behavioral measures of set shifting, while Chapter 4 combined behavioral and neuroimaging measures to provide a more comprehensive examination of response inhibition and response monitoring and their underlying mechanisms. Chapter 5 built upon the findings from Chapter 4 by exploring the relations between DE, internalizing symptoms, and cognitive control in the context of emotional stimuli, addressing questions two and four. To further understand these emotion regulation processes, Chapter 5 also examined associations between emotion recognition, DE, and internalizing symptoms. This chapter will provide a summary of the main findings of this thesis, as well as outline important implications, strengths, and limitations of the research. Considerations and future directions for research in the field will also be highlighted.

6.2 Summary of Findings

6.2.1 The relation between DE and internalizing symptoms in preadolescence

My first key finding was evidence of DE and internalizing symptoms in preadolescence and the presence of concurrent relations between these symptoms by age 10. This research extended previous work (e.g. Holm-Denoma et al., 2014; Houldcroft et al., 2014) examining these co-occurring symptoms in preadolescents, by utilizing a measure of DE that was validated for this younger age-range and captured a broader range of eating difficulties.

My second key finding was that when both anxiety and depression symptoms were entered into a regression model predicting DE, only anxiety was a significant predictor of DE in my sample of preadolescents. Associations between depression and DE have previously been reported in preadolescents (e.g. Evans et al., 2017; Gardner et al., 2000); however, not

all of these studies included anxiety as an independent variable in the model. My findings suggest anxiety may play a key role in the association between depression and DE in preadolescence.

My third key finding was the absence of gender differences in DE and internalizing symptoms in preadolescents. This confirmed my hypothesis that there would be no gender differences in DE or depression symptoms in preadolescents. However, it contrasted with my hypothesis that there would be a significant interaction between gender and anxiety when using regression models to predict DE. These findings are inconsistent with previous evidence from older adolescents that has shown gender to be a moderator in the association between anxiety and DE (O'Dea & Abraham, 1999). Pubertal, environmental, and social changes that occur in adolescence may be key in explaining why gender is a moderator in older samples, and it could be the case that these factors have not yet emerged for the sample in my study.

6.2.2 Behavioral markers of cognitive control

To address research questions two to four, I examined the role of cognitive control processes in the association between DE and internalizing symptoms, both in emotional and non-emotional contexts. Associations between cognitive control processes, DE, and internalizing symptoms were first conducted. Significant associations between DE and cognitive control were further analyzed to investigate whether cognitive control processes were associated with DE independently of internalizing symptoms. A summary of the key behavioral findings is presented in Table 6.1.

Contrary to my hypothesis, I found no behavioral evidence to suggest DE and internalizing symptoms were associated with set shifting performance in preadolescence (Chapter 3). However, when examining response inhibition and response monitoring performance across non-emotional and emotional contexts (Chapters 4 and 5), I hypothesized that there would be no significant associations between behavioral markers of cognitive control, DE, and internalizing symptoms. Findings from Chapter 5 supported my hypothesis, as there were no significant associations between behavioral markers of response inhibition (NoGo accuracy), response monitoring (post-error slowing), DE, and internalizing symptoms.

Contrary to my hypothesis, on the non-emotional Go/NoGo task (Chapter 4), increased depression was associated with greater NoGo accuracy, indicative of enhanced inhibition, as well as less post-error slowing, suggesting there was a behavioral reduction in

response monitoring. There were no significant associations between cognitive control and both DE and anxiety in the non-emotional Go/NoGo task, in line with my hypothesis. Therefore, my key finding was the absence of significant associations between behavioral markers of cognitive control and DE in preadolescence. These findings suggest cognitive control processes used in emotional and non-emotional contexts may be preserved at earlier stages of ED symptom development in preadolescence. Instead, these difficulties may be more apparent in chronic ED presentations, more commonly reported in older adolescents and adults (Grange & Loeb, 2007; Lang et al., 2014; Wonderlich et al., 2012).

Emotion regulation is also thought to be interconnected with other processes, such as emotion recognition, to enable successful social emotional functioning (Ochsner, 2008; Tottenham et al., 2011). To further our understanding of emotion regulation processes I examined emotion recognition performance. My key finding was that poorer recognition of happy facial expressions was associated with increased DE. This suggests emotion recognition impairments may present earlier in development, potentially contributing to later difficulties in emotion processing and regulation.

Table 6.1.*Summary of my key behavioral findings.*

	Disordered eating (DE)	Anxiety	Depression
Set shifting	No significant associations.	No significant associations.	No significant associations.
Response inhibition			
<i>Non-emotional task</i>	No significant associations	No significant associations	Increased depression was associated with greater NoGo accuracy.
<i>Emotional task</i>	No significant associations.	No significant associations.	No significant associations.
Response monitoring			
<i>Non-emotional task</i>	No significant associations.	No significant associations.	Increased depression was associated with less post-error slowing.
<i>Emotional task</i>	No significant associations.	No significant associations.	No significant associations.
Emotion recognition	Increased DE associated with poorer happy emotion recognition accuracy.	No significant associations.	No significant associations.

6.2.3 Neural markers of cognitive control

In Chapters 4 and 5, I examined associations between DE, internalizing symptoms, and neural markers of response inhibition and response monitoring in both non-emotional and emotional contexts, respectively. A summary of the key neural findings is presented in Table 6.2. For response inhibition, I hypothesized that increased levels of DE and internalizing symptoms would be positively correlated with neural markers of impaired response inhibition (in the form of attenuated P3 amplitudes and greater N2 amplitudes on NoGo trials) across both non-emotional and emotional Go/NoGo tasks. The N2 is proposed to reflect identification of conflict or inconsistency between competing responses (Albert et al., 2013), while the P3 indexes later evaluation processes (Smith et al., 2008).

Consistent with my hypotheses, I found attenuated P3_{NoGo} amplitudes were significantly correlated with increased levels of anxiety and depression on the non-emotional Go/NoGo task. Although a similar association was found between increased DE and attenuated P3_{NoGo} amplitudes, these effects were not significant. Using emotional stimuli (Chapter 5), I found the opposite pattern of results, with enhanced P3_{NoGo} amplitudes associated with increased DE. This was specific to happy emotional expressions and not associated with internalizing symptoms. However, I found attenuated P3_{NoGo} amplitudes on calm trials were significantly associated with anxiety following Bonferroni correction, while there was a moderate, but not significant, association with DE and depression. This association between attenuated P3_{NoGo} amplitudes and anxiety was consistent with my hypothesis and suggests a smaller P3_{NoGo} effect with calm faces as anxiety increases. Lastly, in both emotional and non-emotional contexts, there were no significant associations between N2 amplitudes and anxiety, depression, or DE.

For response monitoring, I hypothesized that increased levels of DE and internalizing symptoms would be positively correlated with impaired response monitoring. Specifically, increased anxiety would be associated with greater ERN amplitudes, while depression and DE would be associated with attenuated ERN amplitudes. The ERN is thought to reflect response monitoring (Botvinick et al., 2004), such as identifying situations high in response conflict with the potential to produce errors (van Veen & Carter, 2002). The CRN is hypothesized to reflect the same process as the ERN, but modulated by response accuracy (Hoffmann & Falkenstein, 2010), while the Pe is thought to be involved in error awareness and detection (Davies et al., 2001; Endrass et al., 2007; Falkenstein et al., 2000; Ridderinkhof et al., 2009). The Pe and CRN are less widely studied response monitoring ERPs in the

internalizing disorder and ED literature, especially within non-clinical populations.

Therefore, my analysis of these components was exploratory.

Associations between neural correlates of response monitoring and DE were specific to emotional tasks. Attenuated ERN amplitudes were associated with increased DE in the emotional Go/NoGo task (Chapter 5), but no significant associations were found in the non-emotional Go/NoGo task (Chapter 4). Across both Go/NoGo tasks, I found no significant associations between Pe amplitudes, CRN amplitudes, and DE. In addition, internalizing symptoms were not associated with any neural correlates of response monitoring (ERN, CRN, Pe).

Table 6.2.

Summary of my key neural findings.

	Disordered eating (DE)	Anxiety	Depression
Response inhibition			
<i>Non-emotional task</i>	A moderate, but not significant, association between attenuated P3 amplitudes and increased levels of DE.	Attenuated P3 amplitudes were significantly correlated with increased levels of anxiety.	Attenuated P3 amplitudes were significantly correlated with increased levels of depression.
	No significant associations with N2 amplitudes.	No significant associations with N2 amplitudes.	No significant associations with N2 amplitudes.
<i>Emotional task</i>	Enhanced P3 amplitudes on happy trials were associated with increased levels of DE.	Attenuated P3 amplitudes on calm trials were associated with increased levels of anxiety.	A moderate, but not significant, association between attenuated P3 amplitudes on calm trials and increased levels of depression.
	A moderate, but not significant, association between attenuated P3 amplitudes on calm trials and increased levels of DE.	No significant associations with N2 amplitudes.	No significant associations with N2 amplitudes.

No significant associations with N2 amplitudes.

Response monitoring

<i>Non-emotional task</i>	No significant associations.	No significant associations.	No significant associations.
<i>Emotional task</i>	Attenuated ERN amplitudes were associated with increased levels of DE.	No significant associations.	No significant associations.

Note. ERN: error-related negativity.

6.3 Cognitive Control in Emotional and Non-emotional Contexts

Cognitive control processes are consistently reported to be impaired in adults with EDs (Aloi et al., 2015; Duchesne et al., 2010; Pieters et al., 2007; Roberts et al., 2010; Steinglass et al., 2019; Svaldi et al., 2014; Tchanturia et al., 2004; Wu et al., 2013; Yue et al., 2020); however, little research has investigated these processes in preadolescents. This thesis aimed to examine associations between cognitive control in both emotional and non-emotional contexts, DE, and internalizing symptoms in preadolescents.

Overall, I found similar behavioral findings across both emotional and non-emotional contexts. However, differences became apparent when examining neural correlates of response inhibition and response monitoring. These findings highlight the increased sensitivity of neuroimaging to uncover differences, but also contribute to neurobiological etiological models of EDs. My findings suggest that associations between DE and neural indicators of cognitive control difficulties are present before behavioral effects are observable in preadolescence. Therefore, these early neural-level changes may play an important role in the development of DE earlier in development, adding to previous models implicating the ACC in ED development during adolescence (Hill et al., 2016).

For response inhibition, attenuated P3 amplitudes were associated with internalizing symptoms (and a moderate, but not significant, association with DE) in the non-emotional Go/NoGo task (Chapter 4), while enhanced P3 amplitudes associated with higher levels of DE in the emotional Go/NoGo task (Chapter 5). These findings suggest P3 amplitudes, reflective of evaluation of environmental inconsistency or conflict, are a feature of DE, anxiety, and depression in preadolescence, but these associations are context dependent. In EDs, P3 amplitudes are reported to present differently according to stimuli type (Berchio et al., 2022), with significantly higher P3 amplitudes during presentation of food stimuli compared to neutral stimuli in adolescents with higher levels of loss of control eating (Biehl et al., 2019) and emotional eating (Wu et al., 2018). In contrast, during neutral stimuli tasks individuals with AN are reported to display attenuated P3 amplitudes (Bradley et al., 1997; Yue et al., 2020). Combined with my findings, these results suggest that higher levels of DE are associated with enhanced P3 amplitudes (i.e., greater inhibitory effort) when processing emotive stimuli such as emotional face expressions and food stimuli. However, when presented with neutral stimuli, increased DE is associated with attenuated P3 amplitudes (i.e., reduced inhibitory effort). This association in non-emotional contexts may be driven by higher levels of internalizing symptoms, as anxiety and depression were significantly

associated with attenuated P3 amplitudes in my study (Chapter 4). This is consistent with P3 amplitude reduction reported in depression (Houston et al., 2003; Nan et al., 2018) and anxiety (Bechor et al., 2019; Éismont et al., 2009; Wauthia et al., 2022; Xu et al., 2014), potentially reflecting an association between P3 attenuation and internalizing problems more generally (e.g. Bernat et al., 2020).

The association between enhanced P3 amplitudes and higher levels of DE may reflect a disruption in reward processing, implicated in the processing of both face stimuli (see Chapter 5) and food stimuli in EDs (O'Hara et al., 2015). Furthermore, enhanced attentional mechanisms to emotionally significant stimuli appear to be present across a range of DE behaviors and may represent a risk factor for the development of diagnosable EDs.

As the ACC is the neural generator of the P3 (Zhang et al., 2012), my findings also have implications for its function. For example, elevated ACC activity in response to food stimuli has been proposed to be a trait marker for AN (Frank et al., 2004; Uher et al., 2003). Combined with the P3 findings discussed above, this may suggest hyperactivity of the ACC in response to emotive stimuli is an early indicator of increased ED risk. In addition, reduced functional activity and connectivity in the ACC has been reported in internalizing disorders (Blair et al., 2012; Lichenstein et al., 2016). Therefore, hypoactivation of the ACC may be an early risk factor for internalizing symptom development, reflected in P3 attenuation in my study.

For response monitoring, increased DE was associated with attenuated ERN amplitudes in the emotional Go/NoGo task only (Chapter 5). Although attenuated ERN amplitudes have been previously reported in AN (Pieters et al., 2007), this was using a neutral stimuli flanker task more in line with the task described in Chapter 4. However, I did not find a significant association between DE and ERN amplitudes using this task. This difference could reflect an early monitoring deficit to social stimuli that becomes more generalized (i.e., non-emotional) as the disorder progresses. Another possible explanation could be the modulation of the ERN by emotional interference in the early emergence of DE. Previous research in the general population has demonstrated modulation of the ERN amplitude across positive and negative emotional stimuli (Boksem et al., 2011; Larson et al., 2006; van Wouwe et al., 2011). Although I was not able to differentiate the ERN amplitude by emotion in my study, these findings may suggest an attenuation of the ERN amplitude that is more pronounced during more salient targets, such as emotional compared to neutral stimuli. This may also be linked to a disruption in reward processing, possibly leading to subsequent difficulties in later emotion processing, reflected in the P3 findings.

6.4 Implications for Interventions and Treatment Options

The strong associations between DE and internalizing symptoms found in my preadolescent sample highlight the importance of early detection of both DE and internalizing symptoms at this early stage. Children with high levels of DE are likely to also display high levels of anxiety and depression. Therefore, prevention and intervention efforts need to consider this co-occurrence and ensure transdiagnostic support is provided, rather than focusing solely on supporting eating behaviors.

DE is known to increase between the ages of 12 and 15 (Breton et al., 2022), aligning with the onset of puberty (Graber, 2013; Pfeifer & Allen, 2021) and new environmental stressors (McVey et al., 2002; Riglin et al., 2013). However, most prevention programs start in later adolescence (Ciao et al., 2014). This thesis presents evidence of DE in children at age 10 years of age, underlining the importance of starting these programs in preadolescence, when DE is already present.

Anxiety symptoms appear to play a particularly important role in the associations between DE and internalizing symptoms. Previous research has identified anxiety as a potential risk factor for EDs with childhood-onset anxiety disorders often found to precede a co-occurring ED (Adambegan et al., 2012; Godart et al., 2000; Kaye et al., 2004; Raney et al., 2008; Swinbourne et al., 2012). Although my study was not able to determine directionality, only correlates, my findings combined with previous research (see section 1.11) suggest that anxiety may be a key modifiable factor in the development of DE and subsequent EDs. It is likely that the association between DE and anxiety is complex and bidirectional; however, providing support and reducing anxiety symptoms at an earlier point in development, such as preadolescence, may be effective in lowering the risk of developing DE at this early stage. Specifically, interventions focused on ways to manage and cope with feelings of distress and anxiety may be key to preventing DE behaviors being used as a maladaptive coping strategy.

Findings from Chapter 5 suggest higher levels of DE are associated with impaired happy face processing at a behavioral and neural level in preadolescence. Research from individuals with EDs demonstrates avoidance of negative *and* positive affect (Lampard et al., 2011), as well as avoidance and difficulties maintaining social relationships (Flament et al., 2001). Taken together, these findings suggest individuals with EDs or patterns of DE do not experience happy emotional expressions as positive and rewarding, instead, potentially finding these ‘approach’ behaviors from others threatening. Therefore, interventions focused on improving interpersonal skills and supporting positive social experiences may reduce the

threat associated with social interactions, thus reducing anxiety and risk for DE. In addition, therapeutic approaches that emphasize emotion recognition in others, particularly positive emotions, may help improve processing of happy emotional expressions. In turn, this may improve social interactions and promote the rewarding nature of these experiences.

The emotion-specific associations between cognitive control and DE are also important when considering ED models, such as the Transdiagnostic Model (Fairburn et al., 2003) and the Cognitive-Interpersonal Maintenance Model (Schmidt & Treasure, 2006). These models highlight the interplay between interpersonal difficulties, cognitive processes, and socio-emotional elements in the maintenance of ED symptoms. My findings update these models by suggesting difficulties in these areas, particularly concerning emotion processing, may also be challenging for individuals with patterns of DE early in development, before the onset of an ED. These difficulties may maintain DE behaviors and increase risk for greater difficulties processing and regulating emotions later in development, thus increasing the risk of developing a diagnosable ED.

6.5 Strengths of the Thesis

One of the key strengths of this thesis is the combination of behavioral and neural markers of cognitive control. To the best of my knowledge, the studies described in Chapters 4 and 5 are the first to investigate the associations between behavioral and neural markers of cognitive control, DE, and internalizing symptoms in preadolescence. This combination provides a more comprehensive investigation of the cognitive processes and their underlying mechanisms compared to using behavioral measures alone, and is especially important given the comparable task performance observed in younger samples with internalizing disorders and EDs compared to HCs (Bartholdy et al., 2019; Brunnekreef et al., 2007; Diler et al., 2014; Hum et al., 2013a; Pan et al., 2011; Singh et al., 2010). Related to this strength is the examination of cognitive control processes in both emotional and non-emotional contexts, with many previous studies focusing on either one or the other. By studying cognitive control processes in both contexts, I am better able to provide a broader overview of these abilities. This is particularly important given the overlap in regulatory processes used within cognitive control and emotion regulation (Braunstein et al., 2017; Holroyd & Coles, 2002; Niendam et al., 2012; Ochsner et al., 2004; Phan et al., 2005; Pruessner et al., 2020; Ridderinkhof et al., 2004), and the involvement of both cognitive and emotional factors in contemporary models

of EDs, such as the cognitive interpersonal maintenance model proposed by Schmidt and Treasure (2006).

Throughout the studies reported in this thesis, I have focused on using a dimensional approach to capturing mental health difficulties, in line with the National Institute of Mental Health's RDoC (Insel et al., 2010). This approach extends previous work using categorical approaches, such as diagnostic thresholds, by measuring mental health difficulties across a wider spectrum, enabling us to include individuals at earlier stages of symptom development. Related to the dimensional approach, this thesis is also strengthened by the inclusion of co-occurring internalizing symptoms. Transdiagnostic approaches to conceptualizing mental health difficulties help increase our understanding of the etiology of mental health problems and develop novel approaches to treatment and research (Dalglish et al., 2020).

A final strength of the thesis is the recruitment of both male and female preadolescents. Preadolescence is an important but understudied developmental stage for EDs, with DE found to emerge in late childhood (Breton et al., 2022; Evans et al., 2017; Herle et al., 2020) and recent evidence suggesting ED diagnoses are occurring at earlier developmental periods (Nicholls et al., 2011; Petkova et al., 2019; Reas & Rø, 2018). In addition, the majority of ED research in adolescence studies females only, due to the higher occurrence of diagnoses in females compared to males (Galmiche et al., 2019). Previous research in younger samples, however, highlights the lack of gender differences in reported DE (e.g., Chapter 2; Holm-Denoma et al., 2014). Therefore, the inclusive recruitment in this thesis expands our knowledge of the cognitive control processes associated with DE in children, which is not limited to those who identify as female.

6.6 Limitations of the Research

I have described specific limitations within each study chapter; however, I will now highlight some of the overarching limitations of my research. Firstly, DE and internalizing symptoms were only assessed using child self-report measures. Associations between response monitoring and anxiety measured by child self-report vs parent-report have been found to differ (Meyer et al., 2012). This was also the case for set shifting in preadolescents (Chapter 3), as parental reports of restrictive eating behaviors, but not child-reported body image, were associated with set shifting difficulties (Steegers et al., 2021). The discrepancy between child and parent reports may be explained by the observability of some of these behaviors/symptoms, with more observable behaviors attracting more attention from parents

(Grills & Ollendick, 2003; Maurizi et al., 2012). For example, parents report more problems with sleeping patterns, activity, and appetite on measures of internalizing symptoms, compared to their child (Bagheri et al., 2019). Whereas, when measuring feelings of distress and more secretive DE behaviors, children are found to report higher levels of these symptoms than their parents (Bagheri et al., 2019; Bartholdy et al., 2017). Although agreement between child and parent report tend to be low, making interpretation of these reports challenging (Achenbach et al., 1987), both reports may provide different but valuable insights. Therefore, the addition of parental reports of DE and internalizing symptoms would be beneficial to include in future studies to complement findings from child self-report measures.

In addition, there are some limitations relevant to both Go/NoGo tasks. Firstly, the low trial numbers used to form the response-locked ERPs could have contributed to the null findings observed for most relations between neural indices and both DE and internalizing symptoms. These ERPs may be more unstable and higher trial numbers may produce a more reliable waveform. Secondly, behavioral performance across both tasks was lower than anticipated, based on the procedure used by Hum et al. (2013a). This performance, coupled with the lack of post-error slowing, may be reflective of the absence of task feedback. Without trial-by-trial feedback, participants may not be aware when they have committed an error and therefore, do not adjust their behavior. The motivational properties of feedback may also play a role in task performance and ERP components. For example, some participants may interpret negative feedback as a form of punishment, heightening self-doubt and ultimately demotivating the individual. When the participant is performing well, feedback may decrease anxiety through providing an external evaluation of their performance, reducing the burden on their own response monitoring (Doñamayor et al., 2014; Nieuwenhuis et al., 2005). Alternatively, negative feedback may motivate the individual to rectify this error on subsequent trials, improving performance.

Another limitation is the issue of task impurity, a commonly reported measurement problem in the executive functioning literature, particularly in developmental studies (Best & Miller, 2010; Hughes & Graham, 2002). To avoid ceiling effects, standard cognitive control tasks, such as the WCST, are often complex, requiring engagement of multiple executive function processes (Miyake et al., 2000). This is often further complicated by the variation of tasks used across age ranges, making comparisons across development challenging. To try and mitigate for these issues in my research, the demands of the Go/NoGo tasks used in Chapters 4 and 5 were dynamically adjusted based on the child's performance. This meant

children were challenged when they performed well on the task, but the task was made easier when their performance was poor. Therefore, although the age range recruited in this study was narrow, the task could be adapted for a broader range of ability. In addition, the set shifting task (SST-CV) described in Chapter 3 was specifically chosen due to less reliance on language understanding and integration of numerical and alphabetical series compared to commonly used tasks, such as the TMT-CV. As the SST-CV is culturally unbiased, this task is more appropriate for assessing the cognitive control performance of children from diverse communities.

It is important to acknowledge the unforeseeable impact of the COVID-19 pandemic on the research conducted in this thesis. Some of the data collection and recruitment took place pre-pandemic, between April 2019 – March 2020; however, the remaining research was conducted from March 2021 – September 2021, during the pandemic. Research has found that some adults' and children's mental health was negatively impacted by the COVID-19 pandemic (Pierce et al., 2020; Raw et al., 2021); therefore, this should be considered when interpreting the findings from this thesis. However, the aim of this thesis was to examine associations between DE, internalizing symptoms, and cognitive control, so it was beyond the scope of my research to investigate the impact of COVID-19 on these associations. Future research could examine whether there are differences in the strengths of these associations pre-, during, and post-pandemic. For example, stronger associations between DE and internalizing symptoms during the pandemic compared to pre-pandemic may suggest a greater use of DE as a maladaptive coping strategy at a time of particular stress and anxiety.

Lastly, it is important to acknowledge the cross-sectional nature of the research. Currently, I am unable to comment on any causal relationships, so replication and extension of this research is required. Longitudinal designs enable us to study the trajectories of DE and internalizing symptoms across development, providing more insight into the etiology of DE. This investigation would be particularly important given the onset of potential life stressors during this developmental stage (e.g., transition to secondary school and puberty), which can increase risk for mental health difficulties (Low et al., 2012; Riglin et al., 2013). Therefore, collecting data across two time points, from preadolescence (age 10-11 years) to early adolescence (12-13 years), would allow us to probe these trajectories of DE across a life stressor, such as the transition from primary to secondary school.

6.7 Future Directions

Future studies should extend this research by examining individual symptom profiles, such as restrictive-type eating behaviors or social anxiety, rather than only using global measures of these symptoms. Thomas et al. (2022) highlight specific links between executive functioning in AN-R and AN-BP, such as more pertinent response inhibition difficulties in individuals with AN-BP (Galimberti et al., 2012) compared to AN-R (Weinbach et al., 2020). In EDs that typically present with more binge eating behaviors, such as BED, researchers have hypothesized this cognitive inflexibility results in an inability to control eating, due to difficulties in changing strategies when regulating emotions during periods of distress (Dingemans et al., 2015). Whilst enhanced set shifting in AN-R (Weinbach et al., 2020) may enable the individual to more effectively shift attention away from food and facilitate restrictive eating behaviors (Thomas et al., 2022). There also appears to be an increased co-occurrence of certain anxiety subtypes and EDs, such as social anxiety (Kerr-Gaffney et al., 2018; Swinbourne et al., 2012) and generalized anxiety disorder (Touchette et al., 2011; Ulfvebrand et al., 2015). Individuals with co-occurring social anxiety are found to present with more severe ED psychopathology, irrespective of body mass index (Kerr-Gaffney et al., 2018). While generalized anxiety symptoms during middle childhood are predictive of ED diagnoses, specifically AN and BN, as well as a range of ED symptoms in adolescence (Schaumberg et al., 2019). Therefore, it is important to investigate whether these links are also present in patterns of DE (e.g., restrictive vs binge/purge eating behaviors) and anxiety subtypes (e.g., social anxiety vs generalized anxiety symptoms) in preadolescence.

Additional correlates of DE, such as perfectionism and obsessive-compulsive behaviors are also important to consider for future studies in this area. There is a consistent overlap between these traits and DE (Drieberg et al., 2019; Serpell et al., 2006), as well as their link to elevated cognitive control (Barke et al., 2017; Santesso et al., 2006a; Thomas et al., 2022). Higher levels of perfectionism have also been associated with enhanced set shifting performance in individuals with AN (Vall & Wade, 2015) and individuals who have recovered from AN (Lindner et al., 2014). The association between enhanced set shifting performance and perfectionism appears to be specific to AN-R (Herbrich et al., 2018), further underlining the importance of examining individual symptom profiles in future research.

Lastly, future studies would benefit from recruitment of a more diverse sample, considering neurodiversity and gender diversity. There is a well-established link between AN and autism (Brede et al., 2020; Kinnaird & Tchanturia, 2020; Tchanturia et al., 2017), and autistic individuals are significantly more likely to express gender diversity (e.g., identifying

as a gender different to their birth sex, or identifying as non-binary) than non-autistic individuals (Dewinter et al., 2017; Strang et al., 2014; van der Miesen et al., 2018). In addition, individuals who identify as gender diverse are at higher risk of experiencing mental health symptoms, including DE (Feder et al., 2017; Hartman-Munick et al., 2021). Importantly, early screening and interventions for DE that target binary gender identity (e.g., male and female), may not be appropriate for transgender and non-binary children and adolescents, requiring adaptations. Future work is needed to examine the development of DE in younger samples who are autistic and gender diverse, to ensure the methods used to detect and support DE are inclusive.

6.8 Conclusion

The aim of this thesis was to investigate the relation between DE, internalizing symptoms, and cognitive control in preadolescence. My research found strong evidence for an association between DE and internalizing symptoms in preadolescent boys and girls, with anxiety symptoms a stronger predictor of DE than depressive symptoms (Chapter 2). This research extended previous findings from preadolescent samples by capturing a broader range of DE using validated measures for this younger age range. Across three studies (Chapters 3 – 5), I examined the role of cognitive control in the co-occurrence of DE and internalizing symptoms in preadolescence. Although cognitive control impairments have been previously reported in adults with DE and internalizing symptoms, associations between DE, internalizing symptoms, and behavioral markers of cognitive control do not appear to be present in preadolescents. However, at a neural level, higher levels of anxiety and depression were associated with impaired response inhibition (Chapter 4). Importantly, DE was only associated with cognitive control processes when these were examined in an emotional context (Chapter 5). For response inhibition, this was specific to presentations of happy emotional expressions. These associations were independent of internalizing symptoms, suggesting the co-occurrence of DE and internalizing symptoms in preadolescence was not due to shared cognitive control difficulties in an emotional context. It could be the case that associations between psychopathology and neural indicators of cognitive control difficulties are present before behavioral effects are observable in preadolescence. This highlights the importance of examining both behavioral and neural markers of cognitive control at this early stage. The emotion-specific associations between cognitive control and DE underline the need for research to consider these processes across multiple contexts. It suggests the addition

of emotional interference may be particularly challenging for individuals with patterns of DE, potentially leading to greater difficulties processing and regulating emotions later in development. Finally, the importance of longitudinal designs and increased diversity of samples needs to be considered in future research to further our understanding of the etiology of DE. This will provide more inclusive and comprehensive intervention and prevention programs.

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Appendix A

Descriptive statistics for Original and Alternative ChEAT scoring procedures

Item	Original scoring procedure				Alternative scoring procedure			
	M	SD	Skew	s ²	M	SD	Skew	s ²
Item 1	0.31	0.81	2.57	0.65	2.28	1.44	1.14	2.06
Item 2	0.08	0.34	4.53	0.12	1.94	0.97	0.95	0.95
Item 3	0.43	0.85	1.89	0.72	2.81	1.39	0.67	1.93
Item 4	0.19	0.60	3.34	0.36	2.04	1.24	1.28	1.54
Item 5	0.40	0.89	2.09	0.80	2.70	1.41	0.83	1.98
Item 6	0.90	1.15	0.85	1.32	3.41	1.66	0.15	2.76
Item 7	0.10	0.47	5.33	0.22	1.72	1.07	1.87	1.14
Item 8	0.17	0.58	3.87	0.34	1.81	1.19	1.75	1.42
Item 9	0.02	0.22	13.60	0.05	1.60	0.74	1.62	0.55
Item 10	0.13	0.55	4.53	0.30	1.64	1.13	2.19	1.27
Item 11	0.50	1.04	1.81	1.08	2.45	1.70	1.07	2.88
Item 12	0.70	1.10	1.22	1.21	3.19	1.59	0.43	2.54
Item 13	0.21	0.65	3.34	0.43	1.78	1.30	1.80	1.69
Item 14	0.37	0.88	2.29	0.78	2.31	1.52	1.20	2.30
Item 15	0.59	1.01	1.43	1.01	3.05	1.53	0.44	2.34
Item 16	0.17	0.52	3.63	0.27	2.63	1.01	0.34	1.02
Item 17	0.18	0.55	3.23	0.30	2.30	1.18	0.66	1.40
Item 18	0.18	0.65	3.64	0.42	1.73	1.26	2.03	1.58
Item 19	1.70	1.26	-0.31	1.59	4.51	1.62	-0.78	2.63
Item 20	0.14	0.56	4.36	0.31	1.59	1.08	2.35	1.16
Item 21	0.22	0.73	3.33	0.53	1.83	1.36	1.97	1.84
Item 22	0.18	0.61	3.75	0.38	2.10	1.16	1.37	1.35
Item 23	0.18	0.58	3.42	0.33	1.82	1.23	1.57	1.52
Item 24	0.10	0.46	5.26	0.21	1.81	1.07	1.53	1.15
Item 25	0.78	1.00	1.07	1.01	3.62	1.52	-0.37	2.32
Item 26	0.08	0.45	6.01	0.21	1.62	1.05	2.57	1.11
Total	9.01	6.83	1.65	46.63	60.42	12.15	0.79	147.62

Note. ChEAT: Children's Eating Attitudes Test. Item 25 in the table has been reverse-scored. The total ChEAT score also includes the reverse scoring of item 25.

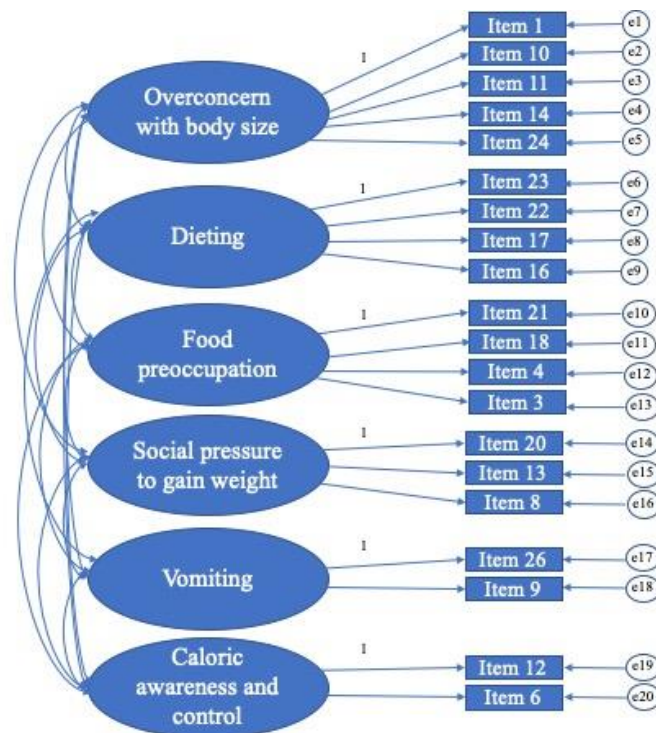
Appendix B

Confirmatory Factor Analysis of the ChEAT

The full 6-point scale was used as an alternative scoring strategy to increase variance and sensitivity in the individual scores, in line with Anton et al. (2006). A confirmatory factor analysis was performed in SPSS AMOS v26.0 to evaluate the 6-factor model proposed by Anton et al. (2006). Each path coefficient between the indicator and latent variable was fixed to 1, to provide a measurement scale for the latent factor. As missing data was present in the dataset, I employed the full-information maximum likelihood estimation. The model is displayed in Figure B1.

Figure B1.

6-factor ChEAT structure proposed by Anton et al. (2006).



The model fit statistics indicated a poor-fitting model (CFI = .822, TLI = .759; values of $\geq .90$ indicate an acceptable fitting model; Whittaker, 2016). The absolute fit index of the model, indicated by the RMSEA value, was acceptable (RMSEA = .072, 90% CI [.06, .08]). RMSEA values of $\leq .05$ indicate a close-fitting model, whilst values of up to .08 are considered acceptable (Brown & Cudeck, 1993; as cited by Whittaker, 2016). Standardized

path coefficients are displayed in Table B1, demonstrating some weak correlations between some of the items and their factors.

Table B1.

Standardized path coefficients between each item and the loaded factor.

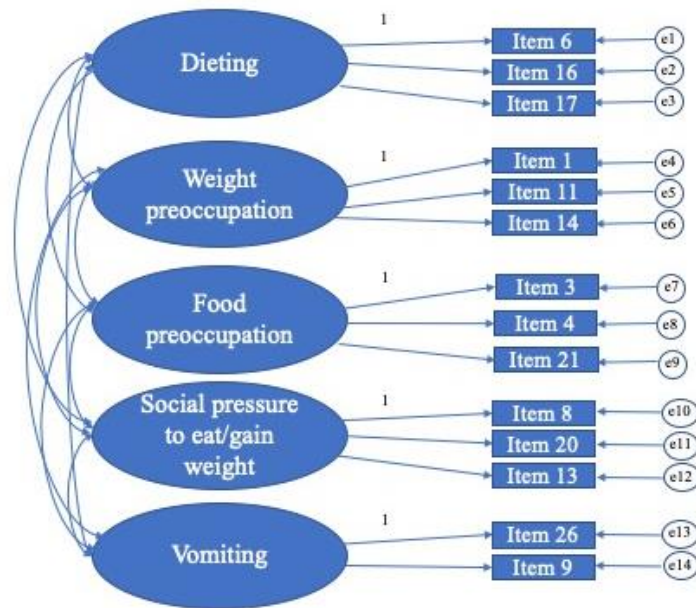
Item	Factor	Coefficient
10	Overconcern with body size	.527
1	Overconcern with body size	.727
11	Overconcern with body size	.835
14	Overconcern with body size	.708
24	Overconcern with body size	.216
23	Dieting	.956
22	Dieting	.245
17	Dieting	.591
16	Dieting	.219
21	Food preoccupation	.797
18	Food preoccupation	.492
4	Food preoccupation	.519
3	Food preoccupation	.788
20	Social pressure to gain weight	.740
13	Social pressure to gain weight	.296
8	Social pressure to gain weight	.645
26	Vomiting	.580
9	Vomiting	.591
12	Caloric awareness and control	.873
6	Caloric awareness and control	.302

More recently, Murphy et al. (2019) evaluated a 5-factor structure based on previous factor analyses of the ChEAT. They reported a 5-factor model based on fourteen items of the ChEAT parsimoniously captures the different types of eating attitudes and behaviors reported by children. Murphy et al. (2019) employed a 3-point Likert scale and acknowledged the potential for extra measurement error and less variation captured in their questionnaire as

opposed to using the 6-point scale. Therefore, a confirmatory factor analysis was conducted to examine the fit of their 5-factor model to my data. The model is displayed in Figure B2.

Figure B2.

5-factor structure proposed by Murphy et al. (2019).



Comparative fit indices for the 5-factor model indicated an acceptable fitting model (CFI = .936, TLI = .899). The absolute fit index of the model was suggestive of a good fitting model (RMSEA = .050, 90% CI [.029, .069]). Standardized path coefficients are displayed in Table B2, demonstrating that most factor loadings were strongly positive (except for items 16 and 13).

Table B2.*Standardized path coefficients between each item and the loaded factor.*

Item	Factor	Coefficient
6	Dieting	.533
16	Dieting	.281
17	Dieting	.412
1	Weight preoccupation	.739
11	Weight preoccupation	.837
14	Weight preoccupation	.704
3	Food preoccupation	.728
4	Food preoccupation	.502
21	Food preoccupation	.862
26	Vomiting	.638
9	Vomiting	.537
8	Social pressure to eat/gain weight	.665
20	Social pressure to eat/gain weight	.723
13	Social pressure to eat/gain weight	.295

Appendix C

Descriptive statistics of questionnaire measures for the whole sample and split by gender.

	Whole Sample (n = 57)		Male (n = 29)		Female (n = 28)		<i>F</i> (1, 55)	<i>p</i>
	M (SD)	Min - Max	M (SD)	Min - Max	M (SD)	Min - Max		
ChEAT	59.42 (12.80)	35 - 100	60.45 (10.92)	41 - 84	58.36 (14.62)	35 - 100	.376	.542
RCADS Anxiety	11.54 (7.10)	1 - 32	11.03 (6.38)	1 - 23	12.07 (7.85)	2 - 32	.339	.563
RCADS Depression	7.54 (4.82)	0 - 20	7.45 (5.08)	0 - 20	7.64 (4.64)	2 - 20	.376	.542

Note. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the inferential statistical analyses.

Appendix D

Descriptive statistics of the Shape Trail Test – Child Version for the whole sample and split by gender.

	Whole Sample (n = 57)	Male (n = 29)	Female (n = 28)
	M (SD)	M (SD)	M (SD)
Part B – Part A duration (s)	9.24 (4.67)	9.16 (4.00)	9.32 (5.36)
Perseveration errors	.40 (.78)	.59 (.91)	.22 (.58)
Sequential errors	.07 (.26)	.07 (.26)	.07 (.27)

Note. SST-CV: Shape Trail Test – Child Version.

Appendix E

Descriptive statistics of behavioral measures on the Go/NoGo task for the whole sample and split by gender.

	Whole Sample (n = 48)	Male (n = 27)	Female (n = 21)	<i>F</i> (1, 46)	<i>p</i>
	M (SD)	M (SD)	M (SD)		
Go Accuracy (%)	74.02 (15.62)	76.48 (14.84)	70.86 (16.37)	1.550	.219
NoGo Accuracy (%)	78.15 (9.80)	76.04 (11.06)	80.86 (7.25)	2.981	.091
All trials Accuracy (%)	75.38 (9.76)	76.30 (8.49)	74.19 (11.29)	.544	.464
Go Mean RT (ms)	363.04 (48.96)	368.02 (58.14)	356.62 (34.09)	.636	.429
NoGo Mean RT (ms)	319.56 (56.73)	315.78 (50.33)	324.41 (65.01)	.269	.607
All trials Mean RT (ms)	341.29 (44.87)	341.90 (48.45)	340.51 (40.98)	.011	.917
Post error slowing (ms)	-5.99 (67.80)	-17.10 (54.79)	8.29 (80.74)	325.000†	.388

Note. RT = reaction time.

†Mann-Whitney U

Appendix F

Pearson correlations between questionnaire and Go/NoGo behavioral measures.

	ChEAT	RCADS Anxiety	RCADS Depression
Go Accuracy (%)	.322*	.012	-.119
NoGo Accuracy (%)	.055	.263	.442*
All trials Accuracy (%)	.370**	.110	.032
Go Mean RT (ms)	.201	.191	.268
NoGo Mean RT (ms)	.290*	.210	.262
All trials Mean RT (ms)	.293*	.237	.312*
Post error slowing (ms)	-.174	-.098	-.362*

Note. * $p < .05$ level, ** $p < .01$. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

Appendix G

Analyses of stimulus-locked ERP latencies for the Go/NoGo task.

Repeated-measures ANOVAs were conducted to examine the main effect of trial type on N2 and P3 latencies. There was a significant main effect of trial type for both the N2 ($F(1, 47) = 174.800, p < .001, \eta_p^2 = .788$) and P3 ($F(1, 47) = 417.753, p < .001, \eta_p^2 = .899$), with longer latencies on NoGo trials compared to Go trials. There were no significant correlations between questionnaire measures and N2 and P3 latencies (Table G1).

Table G1.

Correlations between questionnaire measures and N2 and P3 latencies on Go and NoGo trials at Fz.

	ChEAT	RCADS Anxiety	RCADS Depression
N2 _{Go}	-.047	.087	.011
N2 _{NoGo}	.020	.046	.131
P3 _{Go}	.022	.075	.168
P3 _{NoGo}	.014	.119	.233

Note. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

Appendix H

Analyses of response-locked ERP latencies for the Go/NoGo task.

Repeated-measures ANOVAs were conducted to examine the main effect of response accuracy on ERN and Pe latencies. There was a significant main effect of response accuracy for both the ERN ($F(1, 43) = 828.461, p < .001, \eta_p^2 = .951$) and Pe ($F(1, 43) = 778.459, p < .001, \eta_p^2 = .948$), with longer latencies on incorrect responses compared to correct responses.

Longer Pe latencies on correct responses were positively correlated with increased ChEAT scores and there was a moderate effect size for the association with RCADS anxiety ($r = .293, p = .053$) (Table H1).

Table H1.

Correlations between questionnaire measures and ERN/CRN and Pe latencies at Cz.

	ChEAT	RCADS Anxiety	RCADS Depression
CRN	.094	.038	.024
ERN	-.041	-.051	.046
Pe _{Correct}	.339*	.293	.182
Pe _{Incorrect}	-.128	-.118	-.144

Note. * $p < .05$ level. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale, CRN: correct-related negativity, ERN: error-related negativity. Transformed data were used in the analyses.

Appendix I

Descriptive statistics of behavioral measures collapsed across emotions on the emotional Go/NoGo task for the whole sample and split by gender.

	Whole Sample (n = 53)	Male (n = 30)	Female (n = 23)	<i>F</i> (1, 52)	<i>p</i>
	M (SD)	M (SD)	M (SD)		
Go Accuracy (%)	72.58 (19.18)	75.07 (19.70)	69.35 (18.41)	1.161	.286
NoGo Accuracy (%)	70.89 (8.28)	68.90 (7.55)	73.47 (8.65)	4.205	.045
All trials Accuracy (%)	71.99 (11.68)	73.00 (11.45)	70.67 (12.11)	.515	.476
Go Mean RT (ms)	440.70 (59.91)	452.08 (64.24)	425.85 (51.34)	2.571	.115
NoGo Mean RT (ms)	372.95 (67.46)	377.80 (64.41)	366.63 (72.21)	.353	.555
All trials Mean RT (ms)	406.82 (58.22)	340.70 (47.22)	396.24 (57.75)	1.351	.251
Post-error slowing	-33.40 (58.95)	-47.31 (60.49)	-15.24 (52.74)	4.081	.049

Note. RT = reaction time

Appendix J

Correlations between questionnaire and emotional Go/NoGo behavioral measures.

	ChEAT	RCADS Anxiety	RCADS Depression
<i>Go Accuracy (%)</i>			
Total	.110	.046	-.163
Anger	.127	.047	-.118
Calm	.112	.019	-.195
Happy	.057	.025	-.177
<i>NoGo Accuracy (%)</i>			
Total	-.026	.027	.103
Anger	-.026	.088	.162
Calm	-.099	-.037	-.002
Happy	.069	.098	.247
<i>All trials Accuracy (%)</i>			
Total	.115	.055	-.152
Anger	.127	.067	-.099
Calm	.075	-.010	-.228
Happy	.036	.017	-.155
<i>Go Mean RT (ms)</i>			
Total	-.073	.066	.049

Anger	-.037	.116	.100
Calm	-.068	.002	-.009
Happy	-.102	.068	.047
NoGo Mean RT (ms)			
Total	-.088	.021	.092
Anger	-.038	.021	.066
Calm	-.139	.034	.105
Happy	-.063	.004	.060
All trials Mean RT (ms)			
Total	-.089	.046	.079

Note. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale, RT: reaction time.

Transformed data were used in the analyses.

Appendix K

Analyses of stimulus-locked ERP latencies for the emotional Go/NoGo task.

A repeated-measures ANOVA was also conducted to examine N2 latencies at Fz. There were significant main effects of emotion ($F(2, 102) = 349.430, p < .001, \eta_p^2 = .873$) and trial type ($F(1, 51) = 32.574, p < .001, \eta_p^2 = .390$), as well as a significant emotion x trial type interaction ($F(2, 102) = 1528.102, p < .001, \eta_p^2 = .968$). Pairwise comparisons revealed significantly longer latencies on Go compared to NoGo trials for Happy stimuli, but latencies were longer for NoGo trials compared to Go trials for Angry and Calm stimuli.

P3 latencies followed the same pattern displayed by N2 latencies, with significant main effects of emotion ($F(2, 102) = 973.782, p < .001, \eta_p^2 = .950$) and trial type ($F(1, 51) = 522.371, p < .001, \eta_p^2 = .911$), as well as a significant emotion x trial type interaction ($F(2, 102) = 763.233, p < .001, \eta_p^2 = .937$). Pairwise comparisons revealed longer P3 latencies on Go trials compared to NoGo trials for Happy stimuli, but longer latencies on NoGo trials compared to Go trials for Calm and Angry stimuli.

Correlations were conducted to examine associations between questionnaire measures and N2 and P3 latencies. No significant correlations were found between questionnaire measures and all N2 and P3 latencies for Happy and Calm stimuli (Table K1). However, shorter P3_{Go} latencies for Angry stimuli were significantly correlated with increased depression scores.

Table K1.

Correlations between questionnaire measures and N2 and P3 latencies on Go and NoGo trials for each emotion.

	ChEAT	RCADS Anxiety	RCADS Depression
Angry			
N2 _{Go}	-.017	-.048	-.127
N2 _{NoGo}	-.079	-.074	-.183
P3 _{Go}	.086	.032	-.314*
P3 _{NoGo}	.117	.061	.095
Calm			

	N2 _{Go}	.030	.098	-.004
	N2 _{NoGo}	-.003	.049	.075
	P3 _{Go}	.046	-.149	-.252
	P3 _{NoGo}	.015	.052	.015
Happy				
	N2 _{Go}	-.105	-.150	-.237
	N2 _{NoGo}	.082	.058	.022
	P3 _{Go}	-.158	-.180	-.179
	P3 _{NoGo}	.007	.075	.040

Note. * $p < .05$. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale.

Transformed data were used in the analyses.

Appendix L

Analyses of response-locked ERP latencies for the emotional Go/NoGo task.

Repeated-measures ANOVAs were conducted to examine the main effect of response accuracy on ERN and Pe latencies. There was a significant main effect of response accuracy for both the ERN ($F(1, 50) = 3765.861, p < .001, \eta_p^2 = .987$) and Pe ($F(1, 50) = 1105.097, p < .001, \eta_p^2 = .957$), with longer latencies on incorrect responses compared to correct responses. There were no significant correlations between questionnaire measures and both ERN/CRN and Pe latencies (Table L1).

Table L1.

Correlations between questionnaire measures and ERN/CRN and Pe latencies.

	ChEAT	RCADS Anxiety	RCADS Depression
CRN	.087	.227	-.036
ERN	-.042	.065	.023
Pe _{Correct}	.051	.077	.085
Pe _{Incorrect}	-.056	-.117	.040

Note. ChEAT: Children's Eating Attitude Test, RCADS: Revised Child Anxiety Depression Scale, CRN: correct-related negativity, ERN: error-related negativity.

Transformed data were used in the analyses.