Eating Disorder Symptoms in Adolescents: The Role of Emotional Dysregulation

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Abstract

Eating disorders (ED) are highly prevalent among adolescents, yet there is a lack of evidence-based treatments for this group. Emotion dysregulation has been regarded as a transdiagnostic factor present across all EDs. However, there is a gap in the current literature looking at emotion dysregulation as an early predictor of EDs including anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) in adolescence (14 – 15) as well as controlling for factors including maternal postnatal distress (MPD), which has been previously suggested to have a genetical and environmental effect on both constructs. Addressing this gap can aid future research in developing early interventions for EDs through targeting this modifiable risk factor. The current study examined emotion dysregulation at age 10 – 11 due to the efficacy of emotion dysregulation treatment in this age period. This study investigated this relationship prospectively with nationally representative data from Longitudinal Study of Australian Children at the ages of 0-1, 10-11 and 14-15 (n=2429). Results of the present study suggest that increased emotion dysregulation at 10–11 is significantly associated with symptoms of partial syndrome BED at 14-15, even after controlling for maternal postnatal distress. However, emotion dysregulation was not significantly associated with partial syndrome AN or BN, including after adjusting for MPD. This study indicates that emotion dysregulation can be a potential target for early intervention at 10-11 to reduce the odds of developing BED in adolescence. However, further studies are needed to better understand the role of emotion dysregulation in AN and BN.

Declaration

This thesis contains no material which has been accepted for the award of any other degree of diploma in any University, and, to the best of my knowledge, this thesis contains no material previously published except where due reference is made. I give permission for the digital version of this thesis to be made available on the web, via the University of Adelaide's digital thesis repository, the Library Search and through web search engines, unless permission has been granted by the School to restrict access for a period of time.

27th September 2021

Contribution Statement

In writing this thesis, my supervisors Dr Alyssa Sawyer, and Dr Amanda Taylor and I collaborated to generate research questions of interest, which could be examined with the pre-collected data from the Longitudinal study of Australian Children (LSAC). My supervisors and I also collaborated to design appropriate analyses of the data set using Statistical Package for the Social Sciences (SPSS). I conducted the literature search, completed the LSAC approval form, and was responsible for data analysis and thesis write-up.

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Lastly, we would like to thank all the participants of the LSAC. 'This [thesis] uses unit record data from Growing Up in Australia, the Longitudinal Study of Australian Children. The study is conducted in partnership between the Department of Social Services (DSS), the Australian Institute of Family Studies (AIFS) and the Australian Bureau of Statistics (ABS). The findings and views reported in this paper are those of the author and should not be attributed to DSS, AIFS or the ABS.'

Chapter 1: Introduction

1.1 Overview

The growing prevalence of eating disorders (ED), disproportionately affecting people in their early life is concerning (O'Connor et al., 2018; Volpe et al., 2016). This concern is fuelled by the multitude of adverse psychological, physical and social implications of this disorder, which threaten an individual's quality of life (Leite et al., 2017; Paxton et al., 2012). Indeed, EDs (particularly anorexia nervosa) have one of the highest mortality rates of all psychiatric disorders; along with a high economic burden, with an annual estimated cost of $69.7 billion in Australia (Chesney et al., 2014; Fichter & Quadflieg, 2016; Paxton et al., 2012; van-Hoeken & Hoek, 2020). Yet, even with this array of consequences, there is a lack of evidence-based treatments available for the at-risk group of adolescents (Herpertz-Dahlmann, 2017; Mairs & Nicholls, 2016). Therefore, it is important to explore and better understand the modifiable risk factors of this disorder in order to inform potential early intervention strategies for EDs. One such important risk factor present across all EDs is emotion dysregulation (Mallorquí-Bagué et al., 2018). This section examines this risk factor in the context of developing EDs as well as account for the effect of early life experiences including maternal postnatal distress on both emotion dysregulation and risk of EDs.

1.2 Eating Disorders

ED has come a long way from its initial perception as a cultural-bound syndrome, which only affects western societies (Keel & Klump, 2003). Currently, these symptoms of persistent disordered eating, psychological disturbances (e.g., fear of gaining weight) and maladaptive compensatory behaviours such as purging are found to be present across different races, cultures, gender, and age groups (Keel & Klump, 2003; Leite et al., 2017; Mitchison et al., 2020; Preti et al., 2009). That said, studies also show that this disorder is

however more prevalent among females and young people, particularly in the adolescence period (Leite et al., 2017; Mitchison et al., 2020).

Currently, EDs including anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) affect 6.2% of Australian adolescents and this prevalence continues to grow (Galmiche et al., 2019; Mitchison et al., 2020). When we consider partial syndrome EDs as well, this prevalence rises to 22.2% (Mitchison et al., 2020). Despite partial syndrome cases only representing people, who meet "some, but not all symptoms" (Patton et al., 2008, p. 294) required for clinical diagnoses of the respective ED; these individuals are still at considerable risk for developing full syndrome EDs (Loth et al., 2014). Indeed, for both full and partial syndromes, the manifestation of symptoms and the accompanying consequences differ depending on the type of ED.

1.2.2 Binge Eating Disorder

BED is one of the most common types of ED (Paxton et al., 2012). This disorder is characterised by recurrent episodes of rapid binge eating of unusually large amounts of food and feelings of loss of control during this episode (American Psychiatric Association [APA], 2013). Negative emotions such as sadness, embarrassment and guilt usually precede the episode (APA, 2013). As such, studies emphasise that the behaviours involved in this disorder usually act as a coping mechanism to distress (Masheb & Grilo, 2006; Roher et al., 2014).

Unfortunately, the disordered eating involved in BED is associated with negative health sequelae including obesity, cardiovascular disease, type 2 diabetes, and high blood pressure (Chevinsky et al., 2020; McCuen-Wurst et al., 2018; Reas & Grilo, 2007).

1.2.2 Bulimia Nervosa

On the other hand, BN symptomology also involves episodes of binge eating as described in the above section (APA, 2013). However, these episodes are also accompanied

by compensatory behaviours including purging (i.e., self-induced vomiting, misuse of drugs - laxatives) and negative self-evaluation based on weight (APA, 2013).

Like in BED, individuals with BN also engage in binge-eating behaviours as a coping mechanism in response to negative affect (Jeppson et al., 2003). However, to compensate for this, purging behaviours are usually employed in an attempt to amend the negative emotions surrounding the binging episode as well as reclaiming this lost sense of control to lose weight (Jeppson et al., 2003; McFillin et al., 2012). Unfortunately, BN can be associated with oral health issues, cardiovascular issues (e.g. heart failure due to inconsistent heart rate), intestinal health problems and has also been linked to increased risk for suicide (Silverstein et al., 2019; Smith et al., 2018).

1.2.3 Anorexia Nervosa

AN has the highest mortality rates of any EDs (Paxton et al., 2012). Individuals with AN focus on restrictive caloric intake either through fasting or binging and purging cycles (APA, 2013). This disorder also involves being underweight, having a fear of weight gain and disturbances is how one's own body is perceived (e.g., perceives self as large even when underweight; APA, 2013).

Despite the exacerbated suicide rates among people with this disorder, the primary cause of early death is still due to natural causes (e.g., cardiovascular issues; Smith et al., 2018; Westmoreland et al., 2016). Additionally, the starvation and malnutrition associated with AN can also compromise essential brain functions including memory and attention (Kerem & Katzman, 2003). Despite the array of adverse outcomes attached to these EDs, there is a paucity of evidence-based interventions for adolescents with this disorder (Herpertz-Dahlmann, 2017).

1.2.4 Current Psychological Interventions for Eating Disorders

Many research, clinical and governmental efforts have been made to address these consequences of EDs. Indeed, in response to the consequences of EDs and the well-established research on the treatment-resistant nature of these disorders, the Australian government doubled the Medicare subsidies to 20 sessions for EDs (40 sessions in response to coronavirus-19; National Eating Disorders Collaboration, 2017; Services Australia, 2021a). This is double that of other highly prevalent mental disorders including anxiety and depression (Australian Institute of Health and Welfare, 2020a; Services Australia, 2021b). This suggests that there are mechanisms in place to promote treatments and address the aforementioned consequences of EDs.

When considering the current interventions available for EDs, evidence-based psychological treatments such as cognitive behavioural therapy, interpersonal therapy, enhanced - cognitive behavioural therapy, cognitive behavioural therapy-BN have shown mostly promising outcomes for adults (Atwood & Friedman, 2019; Iacovino et al., 2012; Wilson et al., 2010). However, to date, there is limited evidence to recommend a specific psychological treatment for adolescents with EDs (Herpertz-Dahlmann, 2017; Mairs & Nicholls, 2016). Family-based therapy (for AN and BN), enhanced cognitive behavioural therapy (for AN, BN and BED) and interpersonal therapy (for BED) appears to be the most established treatment for youth with EDs (Herpertz-Dahlmann, 2017; Hurst & Zimmer-Gembeck, 2019; Kass et al., 2013; Mairs & Nicholls, 2016). Yet, the effectiveness of these treatments for adolescents is still not supported by long term data (Herpertz-Dahlmann, 2017; Hurst & Zimmer-Gembeck, 2019; Kass et al., 2013; Lock, 2010; Mairs & Nicholls, 2016).

This lack of evidence-based treatments available for adolescents is of particular concern given the already substandard help-seeking rates for individuals with AN (34.5%), BN (62.8%) and BED (49%), which is further exacerbated for high-severity cases (Ali et al.,

2020; Coffino et al., 2019). This reiterates the need to identify and address the modifiable risk factors of EDs before symptoms become clinically severe (Svaldi et al., 2012).

1.2.5 Risk factors for eating disorders

Extensive research on the predictors of EDs have identified numerous risk factors. While adolescence has been established to be a critical time in exacerbating the risk of EDs, there is a lack of consensus on a specific age in the adolescent years. That said, some studies find that the peak age for the onset of AN is at around 15 years; for BN, this peak age ranges between 15 – 19 years of age and the first peak for the onset of BED is suggested to be at 14 years of age (Micali et al., 2013; Smink et al., 2014; Zerwas et al., 2015). Together, the findings from these studies consistently indicate that the ages of 14 – 15 years appear to be one of the most sensitive periods for the development of all these EDs (Micali et al., 2013; Preti et al., 2009; Smink et al., 2014; Zerwas et al., 2015).

This increased risk during adolescence can be partially attributed to the key developmental changes (i.e., hormonal, and morphological) that occur within this critical period (Rapee et al., 2019). These changes leave adolescents more susceptible to the risk factors of EDs including sociocultural pressures such as unrealistic thin ideals, emotion dysregulation and altered self-concept (i.e., perception of self; Herpertz-Dahlmann et al., 2015; Hochgraf et al., 2017; Rapee et al., 2019; Spear, 2009).

The sex of an individual is also considered a risk factor. Studies suggest that across all EDs, there is also a clear female preponderance (Bentley et al., 2015; Kinasz et al., 2016; Mitchison et al., 2020). Indeed, Mitchison et al. (2020) found that the prevalence of both partial and full syndrome EDs among adolescent females (32.9%) is 20.1% higher compared to males (12.8%). That said, recent studies also emphasise the rise in the prevalence of EDs among males (Limbers et al., 2018). This highlights the importance of exploring EDs among males and females (Kinasz et al., 2016; Limbers et al., 2018).

Additionally, early childhood experiences with primary caregivers have also been shown to increase the risk of such internalising disorders in adolescence (Cimino et al., 2018; Halligan et al., 2007; Hay et al., 2003; Korhonen et al., 2012; Murray et al., 2011). This includes the impact of maternal distress (i.e., symptoms of mental disorders), as early as the postnatal period (Dvir et al., 2014). Such distress tends to affect the child's environment at the critical and malleable period of brain development (Dvir et al., 2014). Indeed, maternal distress is associated with poor responsive parenting and insecure child attachment, which have been implicated in ED outcomes (Ajslev et al., 2010; Bergmeier et al., 2020; Milan & Acker, 2014; Tasca, 2019; Tasca & Balfour, 2014). Maternal postnatal distress (MPD) has also been associated with the development of emotional difficulties as well as employment of maladaptive coping mechanisms, which also increases the risk of EDs in adolescence (Brown et al., 2020; Hay et al., 2003; Korhonen et al., 2012; Mallorquí-Bagué et al., 2018; Tasca, 2019). Studies also suggest that such maternal distress can also have genetic (i.e., heritability) implications on offspring, which can contribute to making adolescents more vulnerable to the risk of EDs (Franić et al., 2010; Silberg & Bulik, 2005; Thornton et al., 2011). Henceforth, it can be proposed that the effect of MPD on ED outcomes is likely to have both genetic and environmental components.

There are also many modifiable risk factors within each ED, including perfectionism, which is associated with AN and purging symptoms, emotional eating (BED and BN), low self-esteem and body dissatisfaction (Forbush et al., 2007; Ghaderi, 2003; Keel & Forney, 2013). However, one of the primary modifiable risk factors associated with the development and maintenance of all EDs is emotion dysregulation. In fact, studies even suggest that targeting emotion dysregulation among people with ED could show promising results in reducing ED symptoms (Haynos & Fruzzetti, 2011; Hazzard et al., 2020; Lavender et al.,

2015). Therefore, it is worthwhile to consider emotion dysregulation as an early predictor and also a possible early intervention strategy for EDs (Mallorquí-Bagué et al., 2018).

1.3 Emotion Dysregulation

Indeed, emotion dysregulation is often regarded as a transdiagnostic factor involved in the "development and maintenance of all EDs" (Mallorquí-Bagué et al., 2018, p.54). This dysregulation refers to one's inability to appropriately experience and adaptively express emotional states that foster felicitous behaviours (Brockmeyer et al., 2014). Difficulties in this area usually involve the endorsement of maladaptive emotion regulation strategies such as suppression of emotions, impulsive reactivity and avoidance of expressing emotions over adaptive strategies including cognitive reappraisal (Brockmeyer et al., 2014; Daniel et al., 2020; Cutuli, 2014; Gross & John, 2003). Thus, when facing negative stressors, these individuals are unable to regulate emotions in an adaptive manner, and this has been linked to the adoption of problematic coping mechanisms including disordered eating and compensatory behaviours (i.e., purging; McLaughlin et al., 2011; Sim & Zeman, 2006).

This is evident in studies investigating the temporal association between negative affect and disordered eating (Engel et al., 2013; Haedt-Matt & Keel, 2011; Lavender et al., 2015; Meule et al., 2019; Monell et al., 2018). These literatures suggest that among individuals with EDs, negative affect on a particular day can increase the risk of engaging in unhealthy ED behaviours on a subsequent day (Engel et al., 2013; Haedt-Matt & Keel, 2011). Meaning, people with restrictive type AN will be more likely to employ restriction around food, whereas individuals with BN and BED will be at a greater risk of engaging in binge eating and/ or purging behaviours (Engel et al., 2013; Haedt-Matt & Keel, 2011). This evidence supports the notion that these symptoms of ED act as a maladaptive coping strategy in response to a negative situation (Engel et al., 2013; Haedt-Matt & Keel, 2011). Indeed, such impact of emotion dysregulation is of high concern, especially when paired with fact

that early life experiences beyond the individual's control are involved in the development of this dysregulation (Dvir et al., 2014).

1.3.1 Development of Emotion Regulation

Emotion regulation abilities develop throughout a person's life. Developmental literature indicates that these abilities are shaped by early life experiences (Monell et al., 2015). Dvir et al. (2014) suggest that children who have been exposed to adversities including poor responsiveness by primary caregivers in early childhood demonstrate difficulties in processing and regulating emotions compared to other children.

One such early experience implicated in this is MPD (Dvir et al., 2014). Mothers tend to be the focus of these studies due to them being overrepresented as primary caregivers (i.e., most time spent with child); and as such, they have a considerable impact on the environment the child grows up in (Australian Bureau of Statistics, 2020; Keel & Forney, 2013). The stressors connected to MPD are often associated with mothers not meeting the child's needs for emotional comfort (i.e., limited responsiveness to a child crying, laughing; Ajslev et al., 2010). This lack of appropriate environmental support for emotional growth (i.e., via parental demonstration and interaction) during this early critical period of brain development can lead to emotion dysregulation at preadolescence (11 years of age) and has also been linked to internalising disorders during adolescence (Hay et al., 2003; Korhonen et al., 2012).

These risks of MPD is concerning as Thompson and Goodman (2010) report that lifetime primary regulation strategies around help-seeking, avoidance, attentional redirection and problem-solving usually develop at the age of 7. However, more recent evidence indicates that with effective interventions, children can learn to regulate and respond to emotions more adaptively and appropriately (Moltrecht et al., 2021). Therefore, the long-term consequences including the psychopathological outcomes of emotion dysregulation can be mitigated (Hay et al., 2003; Korhonen et al., 2012).

A recent meta-analysis of 21 randomised-control trials examined the effectiveness of psychological interventions on reducing emotion dysregulation (Moltrecht et al., 2021). They found that the 10 – 13 years of age period is the most effective time for intervening (Moltrecht et al., 2021). This age group's receptiveness to treatment is consistent with developmental trends of emotion regulation (Rawana et al., 2014). These trends indicate that more sophisticated cognitive regulation strategies including cognitive reappraisal, control of emotional expressions and cognitive reframing develops around the age of 10 – 12 (Rawana et al., 2014). Thus, promoting the adaptive use of these newly developing skills during this malleable period has been shown to be effective in alleviating emotion dysregulation (Moltrecht et al., 2021; Rawana et al., 2014).

Still, there is a gap in the current literature regarding when this factor predicts the development of different EDs among adolescents. While there is support for intervening at around 10 years of age to effectively alleviate emotion dysregulation, it is still not confirmed if emotion dysregulation at this age is predictive of developing EDs in adolescence (Moltrecht et al., 2021).

1.3.2 Emotion dysregulation as an early predictor of Adolescent eating disorders

Few longitudinal studies have examined the effect of early emotion dysregulation on disordered eating during the vulnerable period of adolescence. Shriver et al. (2021) found that among healthy-weight adolescents with high body dissatisfaction, emotion dysregulation at age 7 is associated with emotional eating at 17 years of age. However, it must be noted that emotional eating is not necessarily a symptom of EDs (APA, 2013). Rather, it only shares a high association with objective binge eating (Masheb & Grilo, 2006). As such, interpretations cannot be made on EDs. Thus, it is important to measure the actual symptoms of EDs.

Emotion dysregulation as a childhood predictor of ED symptoms has also been previously investigated longitudinally. However, the findings of these studies are conflicting.

For instance, Allen et al. (2009) use the dataset from the population based Western Australian Pregnancy Cohort (Raine) Study of parents and children to suggest that emotional problems at age 10 shares no significant association with ED symptoms at age 14. However, there are a few limitations to these findings that must be considered. For instance, Allen et al. (2009) neglect to measure different EDs separately. Previous studies emphasise that the extent of the effect of emotion dysregulation may vary between different types of EDs (Weinbach et al., 2018). For instance, Weinbach et al. (2018) suggest that greater emotion dysregulation is involved in behaviours of BED and BN compared to restrictive-type AN. As such, it is important to explore these EDs separately to better understand the individual effect of this dysregulation.

Moreover, this relationship was also explored by Brown et al. (2020) with the dataset from a large community sample of children and their parents in the United Kingdom (the Avon Longitudinal Study of Parents and Children). This study found that the symptoms of borderline personality disorder (at age 11), for which emotion dysregulation is a key component predicted binge eating at the previously established peak age of 14 (Brown et al., 2021; Preti et al., 2009). Indeed, this is also corroborated by findings from previous studies, which suggest that emotion dysregulation at around the age of 10 -11 increases the risk of other internalising disorders (i.e., depression, anxiety) during adolescence (Axelson et al., 2012; Brotman et al., 2006). This suggests that perhaps it would be in our best interest to explore this 10 – 11 age group as it was also distinguished that this age group shows the most promising response to treatment (Brown et al., 2020; Moltrecht et al., 2021). Nevertheless, there are limitations to Brown et al. (2020), which restrict the conclusions that can be drawn from their findings. For instance, only binge eating and purging behaviours were explored, and restrictive eating, as well as body image disturbances were not examined (Brown et al., 2020). This means that the primary symptoms of AN, which not only

disproportionately affects adolescents, but is also responsible for one of the highest mortality rates for any psychiatric disorder is left unaccounted for (Paxton et al., 2012). Additionally, people were classified as exhibiting binge eating and/or purging symptoms, if they had reported any number of episodes within the past year (Brown et al., 2020). The lack of diagnostic specificity, in particularly failing to show consistency with criteria set out in the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5) can limit the extent to which these study findings can be linked to different ED contexts (APA, 2013, Brown et al., 2020). Furthermore, Brown et al. (2020) also neglect to control for other potential confounds such as MPD, which has previously been established to increase the risk of emotion dysregulation as well as EDs due to its biological and environmental effects (Bould et al., 2015; Brown et al., 2020; Silberg & Bulik, 2005; Tasca, 2019; Tasca & Balfour, 2014). As such, in understanding the role of early emotion dysregulation on ED outcomes, the present study will address these limitations of the previous studies.

1.4 Present Study

Indeed, there is a gap in the current literature looking at emotion dysregulation as an early predictor of ED symptoms in adolescence. Previous studies have found that emotion dysregulation at 10 - 11 years of age predicts the ED symptom of binge eating (Brown et al., 2020). Additionally, as previous studies have emphasised the differential effect of emotion dysregulation on different types of EDs, this study separately measured the outcomes of different EDs (AN, BN and BED; Weinbach et al., 2018).

The aim of the current study was to investigate whether emotion dysregulation at 10 - 11 predicts the development of AN, BN and BED at 14 – 15 years of age. To help ensure that these findings are generalisable to the broad Australian population, these associations are investigated using a large representative sample of children and mothers from the Longitudinal Study of Australian Children (LSAC). Additionally, due to the potential

association between MPD and the two constructs, MPD would be adjusted for in the analyses

(Jami et al., 2021). The hypotheses (see Figure 1) for the present study are as follows:

1) Individuals with high levels of emotion dysregulation at age 10 -11 will be at
   increased risk for experiencing partial syndrome AN at age 14 - 15, after adjusting for
   MPD at child age 0 – 1.

2) Those with high levels of emotion dysregulation at age 10 -11 will be at increased risk
   for experiencing partial syndrome BN at age 14 - 15, after adjusting for MPD at child
   age 0 – 1

3) Individuals with high levels of emotion dysregulation at age 10 -11 will be at
   increased risk for experiencing partial syndrome BED at age 14 - 15 after MPD at
   child age 0 – 1 is adjusted for.

Figure 1

Proposed hypotheses of the present study
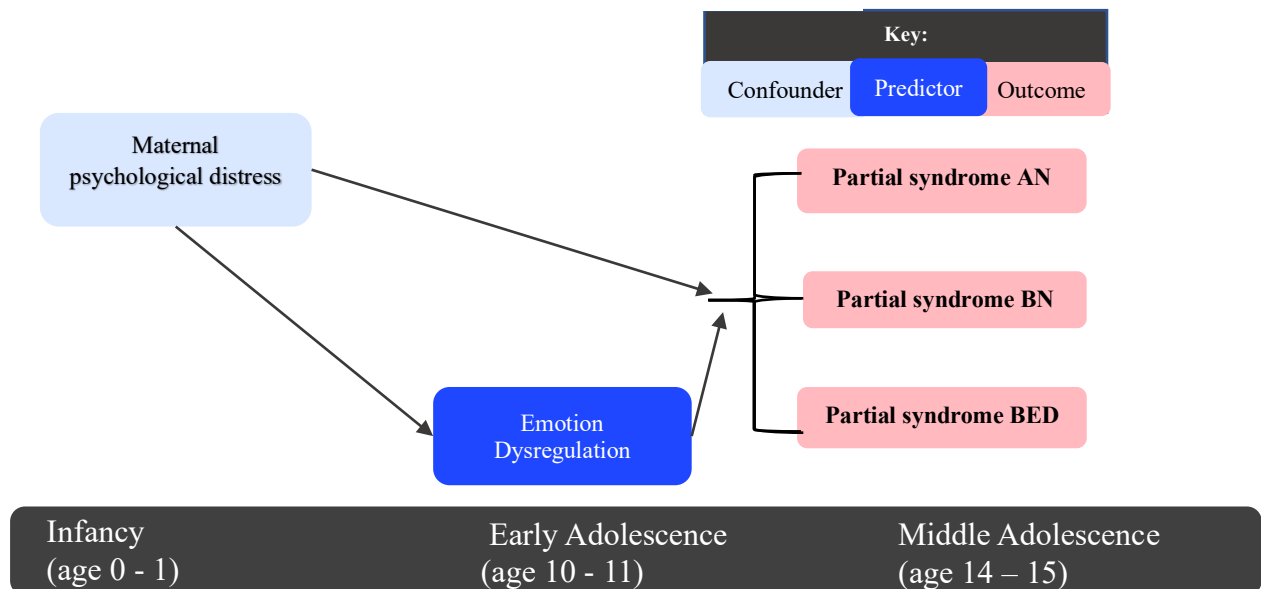
Chapter 2: Methods

2.1 Participants

The sample for the present study came from the LSAC dataset. LSAC used a "two-stage clustered sample design" (Hughes et al., 2019, p.886) based on randomly sampled Australian postcodes from the Medicare database, which was then stratified on the basis of state/ territory of residence and remoteness (Gray & Sanson, 2005). This design was used to ensure a geographically representative sample (Gray & Sanson, 2005). Since its commencement in 2004, this longitudinal cross-sequential study has biennially observed the development of youths in two different cohorts. In the present study, we included children in the birth cohort, who were enrolled in the study at age 0-1 years old. Data has been collected through face-to-face interviews with the child (commenced after 6-7 years of age) and their primary parent/ guardian, as well as self-complete questionnaires with teachers and secondary parent/guardian (Gray & Sanson, 2005).

Figure 2

The respective age of the child, the variables of interest and the associated measures
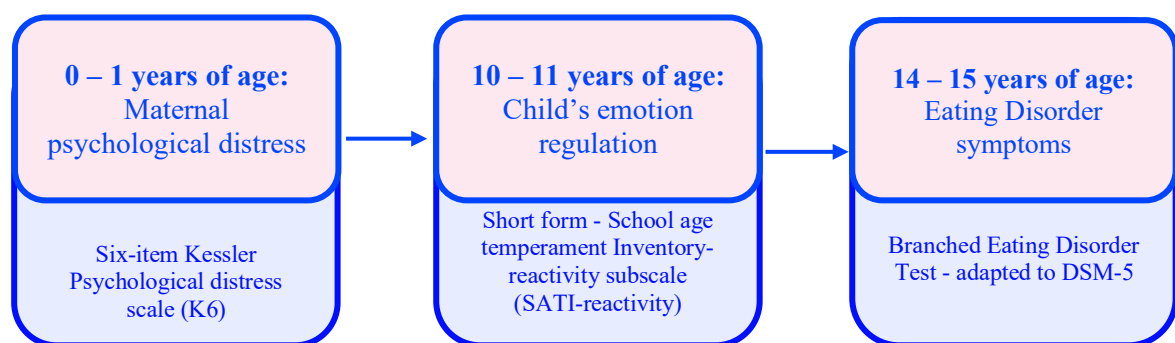
In the present study, data were examined at child age: 0 - 1, 10 - 11, and 14 - 15 (see Figure 2). To test the hypotheses, only children and their mothers were included in this study.

Participants with missing data for any relevant items and measures were excluded from our study.

Ethics approval for LSAC was provided by the Australian Institute of Family Studies Ethics Committee. For the purpose of using the data in the present study, approval was granted by the National Centre for Longitudinal data access.

2.2 Measures

2.2.1 Demographic Factors

Demographic characteristics for the present study include the study child's age, sex, Indigenous status, and family socioeconomic position. Sex was categorised into male and female. Age was measured as years (e.g., 0 – 1, 10 – 11, 14 - 15). In the present study, Indigenous status was categorised into non-Indigenous and Indigenous. People from Aboriginal and/or Torres Strait Islander backgrounds were regarded as having Indigenous status. Family socioeconomic position as measured by the LSAC dataset reflects the socioeconomic resources available to the families based on the annual family income, parent's educational achievement and employment status (Australian Institute of Family Studies [AIFS], 2017; Blakemore et al., 2009). This measure of socioeconomic position was developed by Blakemore et al. (2009). It was employed in this study as it accounts for multiple indicators of socioeconomic status (Blakemore et al., 2009). This parent-rated construct is initially presented as standardised z-scores by LSAC, where higher scores indicate higher family socioeconomic position (AIFS, 2017; Blakemore et al., 2009). To ensure comparability to Australian population data, these scores were converted into percentiles and then divided into quintiles (five groups; Australian Bureau of Statistics [ABS], 2018). These quintiles range from 1 to 5, with higher quintiles reflecting higher family socioeconomic positions. For instance, quintile 1 represents people in the least

advantaged socioeconomic position, and quintile 5 represents those in the most advantaged family socioeconomic position (ABS, 2018; AIFS, 2017).

2.2.2 Emotion Dysregulation

Emotion dysregulation was measured at child age 10 – 11, using the parent-rated reactivity subscale of the short-form School-Aged Temperament Inventory (SATI-reactivity) (Allen et al., 2020; McClowry, 1995). Responses to items (e.g., 'when angry, yells or snaps at others') are given on a five-point scale from 1 (never) to 5 (always; see Appendix A; Allen et al., 2020; McClowry, 1995). The scores of the four items were averaged, with higher scores indicating greater emotional dysregulation (Allen et al., 2015). The short-form of SATI-reactivity has demonstrated high internal reliability in both previous literature ($\alpha = 0.85 - 0.87$) and also the present study ($\alpha = 0.86$; Allen et al., 2015).

2.2.3 Partial Syndrome Eating Disorders

The symptoms of AN, BN and BED were measured using the self-reported Branched Eating Disorder Test (BEDT), when children were aged 14 – 15 years. The BEDT is a well-established measure of partial-syndrome EDs (Allen et al., 2020; Bisset et al., 2019; Mitchison et al., 2020). For the present study, individuals were categorised into partial-syndrome AN, partial-syndrome BN, partial-syndrome BED, and no partial syndrome ED.

The BEDT was originally designed to identify EDs based on diagnostic criteria set out in the DSM-3, which has since been updated (DSM-5; APA, 2013). In order to use the current criteria for ED classification, we used the more recently developed coding of BEDT in accordance with DSM-5 (Bisset et al., 2019). This amendment also allows for the additional measure of partial syndrome BED, which was not previously endorsed by BEDT (Table 1; Bisset et al., 2019).

For the three disorders of interest, the type of responses and the number of items differ depending on the disorder measured. For partial syndrome, AN and BN, twenty-five

items of the test was utilised to represent 3 symptoms (see table 1), while for BED, only five

items were required. This is because only objective binge eating is necessary for the

classification of partial syndrome BED. The specific criteria for classifying each partial

syndrome ED are presented in Table 1.

Table 1

Criteria for classifying DSM-5 partial syndrome anorexia nervosa, bulimia nervosa and binge eating disorder using branched eating disorder

Symptom	Criteria
Partial syndrome anorexia nervosa (at least two symptoms present)	
A. Body weight that is significantly lower than minimally expected.	Being assessed as having a very low body weight, sourced from their body mass index
B. Fear of gaining weight, or behaviours that interfere with weight gain, despite low body weight.	Assessed as being underweight, sourced from their body mass index. AND At least ONE of the following: Reported fearing weight gain for at least 2 days per week AND reported being "very" or "extremely" concerned about gaining weight. OR Reported at least ONE of the following behaviours, lasting at least 3 months: Self-induced vomiting to control weight at least weekly; taking medication to control weight at least weekly; fasting to control weight at least 4 days per week; excessive exercise (2+ hours) to control weight at least 6 days per week.
C. Disturbance in experience of body weight or overvaluation of weight	If underweight: Rated themselves as being "about the right weight," "somewhat overweight," or "very overweight"; If normal weight: Rated themselves as being "somewhat-overweight" or "very overweight"; If overweight: Rated themselves as being "very overweight" (note: could not be rated if obese). OR Reported weight as being "very important" to how they feel about themselves.
Partial syndrome bulimia nervosa (at least two symptoms present)	
A. Regular objective binge eating	Reported experiencing a loss of control overeating at least weekly for at least 3 months.

	AND Reported that it is "very difficult" or "impossible" to stop eating after starting to eat in this way. AND The amount of food eaten in a 2-hour period is equivalent to "8 pieces of bread and half a litre of ice cream and 5 biscuits" OR "12 pieces of bread and 1 litre of ice cream and 10 biscuits" OR "1 loaf of bread and 2 litre of ice cream and 1 packet of biscuits."
B. Overvaluation of weight	Rated their weight as "very important" to how they feel about themselves.
C. Regular engagement in compensatory behaviours	Reported at least ONE of the following behaviours, lasting at least 3 months: Self-induced vomiting to control weight at least weekly; taking medication to control weight at least weekly; fasting to control weight at least 4 days per week; excessive exercise (2+ hours) to control weight at least 6 days per week.
Partial syndrome binge eating disorder (the following symptom is present)	
A. Regular objective binge eating	Reported experiencing a loss of control overeating at least weekly for at least 3 months. AND Reported that it is "very difficult" or "impossible" to stop eating after starting to eat in this way. AND The amount of food eaten in a 2-hour period is equivalent to "8 pieces of bread and half a litre of ice cream and 5 biscuits" OR "12 pieces of bread and 1 litre of ice cream and 10 biscuits" OR "1 loaf of bread and 2 litre of ice cream and 1 packet of biscuits."

Note. Adapted from "DSM-5 eating disorder symptoms in adolescents with and without attention-deficit/hyperactivity disorder: A population-based study" by M. Bisset, N. Rinehart, and E. Sciberras, *International Journal of Eating Disorders, 52*(7), p.857. Participants could meet the criteria for multiple eating disorders because of the shared symptoms between some eating disorders.

For partial syndrome AN, the scores of body mass index (BMI) were calculated with the following formula: BMI = weight (kg)/ height (meters)^2 (Bisset et al., 2019). These scores were then converted and categorised per the current convention where a BMI of less than 18.5 is indicative of individuals being underweight; BMI of 18.5 – 24.9 reflects healthy weight; BMI of 25 – 29.9 is overweight and any BMI of over 30 is classified as obese (Australian Institute of Health and Welfare, 2020b; Flegal et al., 2014).

Additionally, the stem questions such as 'have you felt afraid of gaining weight in the last month?' were presented in a binary format (1 = yes, 2 = no). Further details of frequency (e.g. occurrence of per week), duration (e.g. length of time that the symptoms are present) and amount (e.g. hours spent exercising) for these stem questions had four-point or five-point scales (Selzer et al., 1996) Within these scales, higher values usually meant more severe symptoms, except for the item of 'amount of food eaten in a 2-hour period'. This item ranges from 1 to 5, where 1 reflects a smaller amount of food consumed, 4 represents a very large amount of food consumed and 5 reflects 'less than any of the [aforementioned] amounts'. Participants were excluded if they did not answer the stem questions as well as if they answered 'yes' to the stem questions and did not provide further details (i.e., for question around frequency), which is required for a classification to be made.

For both partial syndrome-AN and BN, individuals were classified as presenting partial symptoms for the respective ED if they met at least two of the three symptoms; and for BED, they must qualify for one symptom (i.e., engaging in binge eating; Table 1). Due to some symptoms being present in both AN and BN (i.e., compensatory behaviours and overvaluation of weight), or BN and BED (i.e., objective binging), some participants presented with multiple EDs (see Appendix B).

There is evidence to suggest that the BEDT has excellent psychometric properties and good accuracy in identifying individuals [at risk of] meeting the criteria for partial syndrome EDs (Cotrufo et al., 1998; Selzer et al., 1996). Indeed, its 'sensitivity' in detecting true cases (100%), 'specificity' in identifying false cases (99%) and 'positive predictive value' (70%) was found to be promising in its initial pilot validation study with a non-clinical population (Selzer et al., 1996). Additionally, the internal reliability for partial syndrome AN ($\alpha = 0.77$), BN ($\alpha = 0.81$) and BED ($\alpha = 0.92$) are also very high in the present study, indicating that items are measuring the same construct.

2.2.4 Maternal Postnatal Psychological Distress

Mothers reported their level of postnatal psychological distress when the respective child was 0-1 years old using the six-item Kessler scale (K6; Cairney et al., 2007; Kessler et al., 2010). They responded to items (e.g., 'during the past four weeks, how often did you feel worthless?') using a five-point scale from 0 (none of the time) to 4 (all the time) (see Appendix C; Mewton et al., 2016). These scores were then summed and categorised using the suggested cut-off points (Kessler et al., 2010). Individuals scoring between 0 to 12 were considered to have no probable serious mental illness (0), while scoring between 13 to 24 was indicative of probable serious mental illness (1) (Kessler et al., 2010).

As indicated by prior literature ($\alpha = 0.78 - 0.90$) and the current study ($\alpha = 0.81$), this scale has high internal consistency (Mewton et al., 2016). Clinical validation studies concerning K6 have suggested that it has a sensitivity of 36% and a specificity of 96% (Prochaska et al., 2012). This sensitivity in detecting true cases is poor, which is concerning. However, further examination shows that for scores of 13 and above, the total classification accuracy remains strong (92%) (Prochaska et al., 2012).

2.3 Statistical Analysis

The dataset was analysed using the social sciences statistical software package (SPSS, version 28). Descriptive statistics were calculated, and reliability analyses were conducted to establish the internal reliability - Cronbach's alpha for the measures of K6, SATI-reactivity and BEDT (each type of ED).

Logistic regression was subsequently performed to test the hypotheses of the present study and determine the strength of the association. Prior to these analyses, it was ensured that the data met the required assumptions for the logistic regression analyses. This included independent observations, binary outcomes for binary logistic regression, no

multicollinearity, linearity of independent variables, log odds (tested with box Tidwell test) and no substantial outliers for the continuous predictor variable of emotion dysregulation (Harrell, 2015). Initially, the simple binary logistic regression models were examined, including emotion dysregulation and the respective ED. Subsequently, multivariate analyses were conducted for emotional regulation and each respective ED, adjusting for MPD.

Chapter 3: Results

3.1 Demographic Characteristics

The demographic characteristics of both the response and complete case sample are displayed in Table 2. There were 2429 participants in the present study who had complete data available for all relevant variables required for analysis. As depicted in Table 2, over half of the adolescent participants were males (52.6%); predominantly identified as non-Indigenous (98.1%) and most participants were from the highest socioeconomic position (29.9%) and least (11.1%) were from the lowest socioeconomic position. This sample is mostly representative of the Australian population, with some differences (Australian Bureau of Statistics, 2020; Australian Institute of Health and Welfare, 2020c). Indeed, there is a lower rate of Indigenous people in the present study compared to the Australian population (3.3%; Australian Institute of Health and Welfare, 2020c). Additionally, while the distribution of socioeconomic position for the Australian population followed a similar pattern to that of our complete sample, our sample appears to be wealthier than the average Australian one (Australian Bureau of Statistics, 2020).

Overall, characteristics are mostly consistent between the response and complete sample, although the complete sample was of slightly higher socioeconomic position and lower weight. For the measures of interest to the present study, the distribution of scores is largely similar between the response and complete case samples. However, the complete sample appears to have a slightly lower rate of MPD and partial syndrome EDs as well as a lower mean for emotion dysregulation.

For the percentage of adolescents meeting criteria for partial-syndrome ED (including those meeting the criteria for multiple EDs) in both samples, AN showed the highest prevalence, followed by BN and BED.

Table 2

Characteristics of response sample and complete sample

	Response sample		Complete sample (n = 2429)
	n	% (95% CI) / mean (standard deviation) range	% (95% CI) / mean (standard deviation) range
Child gender (female)	5107	48.9% (47.5 – 50.3)	47.2% (45.2 – 49.2)
Indigenous status (Yes)	5107	4.6% (4.0 – 5.2)	1.9% (1.4 – 2.6)
Family socioeconomic position [a]	5092		
(Least Advantaged) Quintile 1		20.0% (18.9 – 21.1)	11.1% (9.9 – 12.4)
Quintile 2		25.2% (24.0 – 26.4)	23.3% (21.6 – 25.0)
Quintile 3		17.3 % (16.3 – 18.4)	18.2% (16.7 – 19.8)
Quintile 4		15.3% (14.3 – 16.3)	17.5% (16.0 – 19.0)
(Most Advantaged) Quintile 5		22.2% (21.1 – 23.4)	29.9% (28.1 – 31.8)
Maternal postnatal psychological distress – Mother rated (K6: high risk for serious mental illness)			
Child Age: 0 – 1	4256	2.6% (2.1 – 3.1)	2.0% (1.5 – 2.6)
Emotion Dysregulation - Child rated (SATI – reactivity subscale)			
Child Age: 10 – 11	3663	2.5 (0.8) 1.0 – 5.0	2.4 (0.8) 1.0 – 5.0
Body Mass Index (Total) – Child rated Child Age: 14 – 15	2922		
Underweight		17.6% (16.2 – 19.0)	18.0% (16.5 – 19.6)
Healthy weight		63.6% (61.8 – 65.3)	64.3% (62.4 – 66.3)
Overweight		13.8% (12.5 – 15.1)	13.2% (11.9 – 14.6)
Obese		5.1% (4.3 – 5.9)	4.4% (3.7 – 5.3)

Partial-syndrome Anorexia Nervosa present– child rated (Branched eating disorder test – DSM 5) Child Age: 14 - 15	2866	10.8% (9.7 – 12.0)	11.1% (9.9 – 12.4)
Partial-syndrome Bulimia Nervosa present– child rated (Branched eating disorder test – DSM 5) Child Age: 14 - 15	2968	2.9% (2.3 – 3.6)	2.8% (2.2 – 3.5)
Partial-syndrome Binge Eating Disorder present– child rated (Branched eating disorder test – DSM 5) Child Age: 14 - 15	2985	0.8% (0.5 – 1.16)	0.7% (0.4 – 1.1)

Note. It is possible for people to meet the criteria for multiple eating disorders.

[a] Being in a higher quintile reflects higher family socioeconomic position, quintile 1 represents those with the lowest socioeconomic position – least advantaged, quintile 5 represents those with the highest family socioeconomic position – most advantaged.

3.1.2 Demographic Characteristics Stratified by Eating Disorder Classification

Table 3 depicts the demographic characteristics of adolescents, who met the criteria for overall partial syndrome AN, BN and BED (including those meeting the criteria for multiple EDs), as well as those who did not meet the criteria for any EDs.

The results of the current study suggest that there was a larger representation of males (57.4%) with partial syndrome AN than females. Similarly, more males were also in the no partial syndrome ED group (52.8%). In contrast, there appears to be a higher rate of females with partial syndrome BN (58.8%) and BED (58.8%). The representation of Indigenous adolescents in the partial syndrome AN group (1.8%) resembles the no ED group (1.9%). While no Indigenous participants met the criteria for BN, these individuals were highly represented in the BED group (5.9%). Additionally, the distribution of family socioeconomic position between adolescents with no EDs and those with partial syndrome AN and BN groups were similar. However, most of the participants with partial syndrome BN (30.9%) were in the second lowest socioeconomic position, and most adolescents with AN and no

EDs were in the highest socioeconomic position (29.4%). This distribution differs for participants with partial syndrome BED, where the majority (29.4%) of these individuals are in the lowest socioeconomic position, with the rest being equally distributed within the other four quintiles (23.5%).

While 100% of adolescents with partial syndrome AN are underweight, the majority of all other ED and no ED groups are of healthy weight. However, a higher rate (72.4%) of participants with no partial syndrome ED are in the healthy weight than any other groups (63.2% for BN; 64.7% for BED). The pattern of BMI distribution is relatively stable between BN, BED and the no ED group. However, compared to adolescents with no partial syndrome ED, being underweight is slightly higher amongst the BN group and a bit lower in the BED group. Additionally, there is a lower rate of adolescents who are overweight in the no partial syndrome ED group (14.6%) than in the BN (22.1%) and BED (23.5%) groups.

Table 3

Characteristics of partial syndrome eating disorders no eating disorder population

	No partial syndrome eating disorder present (n = 2094)	Partial syndrome Anorexia Nervosa present (n = 269)	Partial syndrome Bulimia Nervosa present (n = 68)	Partial syndrome Binge Eating Disorder present (n = 17)
	% (95% CI) / mean (standard deviation) range	% (95% CI) / mean (standard deviation) range	% (95% CI) / mean (standard deviation) range	% (95% CI) / mean (standard deviation) range
Child gender (female)	47.3% (45.1 – 49.2)	42.8% (36.8 – 48.9)	58.8% (46.2 – 70.6)	58.8% (32.9 – 81.6)
Indigenous status (Yes)	1.9% (1.4 – 2.6)	1.8% (0.6 – 4.3)	0.0% (0.0 – 0.1)	5.9% (0.1 – 28.7)
Family socioeconomic position (Total) [a] (Least Advantaged) Quintile 1	11.5% (10.1 – 12.9)	8.6% (5.5 – 12.6)	8.8% (3.3 – 18.2)	29.4% (10.31 – 56.0)
Quintile 2	23.4% (21.7 – 25.3)	20.1% (15.5 – 25.4)	30.9% (20.2 – 43.3)	23.5% (6.8 – 50.0)
Quintile 3	18.1% (16.5 – 19.9)	19.3 % (14.8 – 24.6)	17.6% (9.5 – 28.8)	23.5% (6.8 – 50.0)
Quintile 4	17.5% (15.9 – 19.2)	17.1% (12.8 – 22.1)	14.7% (7.3 – 25.4)	23.5% (6.8 – 50.0)
Quintile 5 (Most Advantaged)	29.4% (27.5 – 31.4)	34.9% (29.3 – 41.0)	27.9% (17.7 – 40.2)	23.5% (6.8 – 50.0)

Body Mass Index (Total) – Child rated - Child Age: 14 – 15				
Underweight	8.0% (6.9 – 9.3)	100.0% (98.6 – 100.0)	10.3% (4.2 – 20.1)	5.9% (0.1 – 28.7)
Healthy weight	72.4% (70.4 – 74.3)	0.0% (0.0 – 0.1)	63.2% (50.7 – 74.6)	64.7% (38.33 – 85.8)
Overweight	14.6% (13.1 – 16.2)	0.0% (0.0 – 0.1)	22.1% (12.9 – 33.8)	23.5% (6.8 – 50.0)
Obese	5.0% (4.1 – 6.0)	0.0% (0.0 – 0.1)	4.4% (0.9 – 12.4)	5.9% (0.1 – 28.7)
Maternal postnatal psychological distress – Mother rated (K6: % at high risk for serious mental illness) Child Age: 0 – 1	1.9% (1.2 – 2.4)	3.0% (1.3 – 5.8)	5.9% (1.6 – 14.4)	5.9% (0.1 – 28.7)
Emotion Dysregulation - Child rated (SATI – reactivity subscale) Child Age: 10 – 11	2 (0.8) 1.0 – 5.0	2.5 (0.8), 1.0 – 4.75	2.5 (0.7), 1.25– 4.25	3.0 (0.9) 1.25 – 5.0

Note. n = 354 met criteria for any partial syndrome eating disorder; It is possible for people to meet the criteria for multiple eating disorders.

[a] Being in a higher quintile reflects higher family socioeconomic position, quintile 1 represents those with the lowest socioeconomic position – least advantaged,

quintile 5 represents those with the highest family socioeconomic position – most advantaged.

Additionally, there was a lower rate of MPD among adolescents that have no-ED (1.9%) compared to those with partial syndrome AN (3%), BN (5.9%) and BED (5.9%) groups. Similarly, the current study's results also suggest that the mean emotion dysregulation score (10 - 11 years of age) is higher among adolescents who have developed partial syndrome ED (2.5 – 3.0) at 14 – 15 years of age compared to those who have not (2.4).

It must be noted that due to the smaller sample size of the partial syndrome BN and BED groups, the 95% confidence intervals (CIs) are wide. This is indicative of a decreased precision of the estimation of the true population mean.

3.2 Association between Emotional Dysregulation and Eating Disorders

Table 4 shows the unadjusted and adjusted (for MPD) outcomes for the logistic regression analyses examining the relationship between emotion dysregulation at 10 – 11 and EDs at age 14-15 years. In contrast to the study hypotheses, for both the adjusted and unadjusted model, there is no significant evidence that emotion dysregulation at age 10 - 11 is associated with increased odds of meeting the criteria for partial syndrome AN and BN in adolescence. This pattern of effect also remained consistent when only primary diagnoses of one partial syndrome ED were considered (refer to Appendix D).

Consistent with our third hypothesis, for the adjusted model of BED, there is evidence that emotional dysregulation at 10 – 11 years of age is significantly associated with partial syndrome BED at 14 – 15 years of age. This suggests that a one unit increase in emotion dysregulation at 10 – 11 years of age is associated with 2.17 (95% CI: 1.25 – 3.78) times greater odds of belonging to the partial syndrome BED group in adolescence after MPD is adjusted. Similarly, the unadjusted model also suggested a significant association between the two constructs but had a slightly higher odds ratio of 2.23 (95% CI: 1.29 – 3.86).

Table 4

Binomial logistic regression (95% CI) of adjusted and unadjusted models of partial syndrome eating disorders and emotion dysregulation (n = 2429)

	Emotion Dysregulation (Unadjusted)	Emotion Dysregulation (Adjusted [a])
Partial syndrome Anorexia Nervosa		
Odds Ratio	1.13	1.12
95% Confidence interval	0.96 – 1.33	0.95 – 1.32
p-value	0.16	0.19
Partial syndrome Bulimia Nervosa		
Odds Ratio	1.15	1.12
95% Confidence interval	0.85 – 1.57	0.82 – 1.52
p-value	0.37	0.48
Partial syndrome Binge Eating Disorder		
Odds Ratio	2.23	2.17
95% Confidence interval	1.29 – 3.86	1.25 – 3.78
p-value	0.004*	0.006*

Note. n = 354 met criteria for any partial syndrome eating disorder; n = 269 met criteria for partial syndrome anorexia nervosa; n = 68 met criteria for bulimia nervosa; n = 17 met criteria for partial syndrome binge eating disorder; It is possible for people to meet the criteria for multiple eating disorders; *p < 0.05, two tailed = statistically significant association.

[a] this model adjusts for maternal postnatal distress.

Chapter 4: Discussion

4.1 Overview

This study aimed to investigate the role of emotion dysregulation at 10 – 11 in increasing the risk of partial syndrome EDs at 14 – 15 years of age. While the transdiagnostic role of emotion dysregulation across all EDs have been previously established, it is still not known if emotion dysregulation can be an early predictor of different type of EDs (Brown et al., 2020; Mallorquí-Bagué et al., 2018). The present study seeks to build on the current understanding by separately investigating the individual effect of emotion dysregulation on partial syndrome AN, BN and BED. Moreover, our study also accounts for the combined genetic and environmental effect of MPD on both EDs and emotion dysregulation by adjusting for it in the final analyses (Evans et al., 2017; Keel & Forney, 2013). However, the three main findings of the present study only partially supported our hypotheses.

4.2 Summary of Findings

Firstly, consistent with our hypothesis, this present study found that a higher level of emotion dysregulation at age 10 – 11 is associated with an increased risk of partial syndrome BED at 14 – 15 years of age, even after adjusting for MPD. This relationship between the two constructs is supported by the findings of Brown et al. (2020), which also suggest that borderline personality disorder symptoms (emotional dysregulation is a primary symptom) at the age of 11 is associated with ED symptoms including binge eating episodes at 14 years of age.

Indeed, emotion dysregulation at the young age of 10 - 11 can be especially debilitating as it is a critical developmental period nearing adolescence (Rawana et al., 2014). Entering adolescence with this dysregulation can be concerning as this period involves substantial hormonal changes, which has been associated with exacerbating emotion dysregulation (Rapee et al., 2019; Rawana et al., 2014). It should also be noted that more

advanced and adaptive emotion regulation strategies including cognitive reappraisals are only beginning to develop during this period (Rawana et al., 2014). Therefore, these children with emotion dysregulation are left with reduced adaptive strategies in this vulnerable time where their dysregulation is more likely to be exacerbated (Rapee et al., 2019; Rawana et al., 2014). As such, in order to manage these ongoing struggles to appropriately experience and express emotions, these adolescents turn to maladaptive coping mechanisms to alleviate this discomfort for a short term; one such coping mechanism is the symptoms of BED (Goldschmidt et al., 2012).

Indeed, this idea that BED symptoms are a coping strategy has been well-established in previous studies (Goldschmidt et al., 2012; Stein et al., 2007). In fact, these literatures suggest that the experience of negative affect before a meal is associated with increased loss of control and binge eating during the meal (Goldschmidt et al., 2012). Accordingly, positive affect was associated with reduced employment of BED symptoms (Goldschmidt et al., 2012; Munsch et al., 2012). Such reliance on BED symptoms as a coping mechanism is fuelled by the perception that these episodes of binge eating can directly reduce the negative affect experienced (Goldschmidt et al., 2012; Munsch et al., 2012; Stein et al., 2007). However, this is not necessarily the case. Rather than the accumulated negative affect, it is the inability to adaptively regulate emotion in response to this negative affect, which encourages the employment of BED symptoms as coping strategies (Munsch et al., 2012).

Food intake is often used as a coping mechanism as it carries pleasure promoting properties (Moore et al., 2018). In fact, food intake, especially during a binging episode is associated with the release of several happy hormones including dopamine and serotonin (Moore et al., 2018; Volkow et al., 2011). Accordingly, binge eating episodes habeen ve often shown to have a short-term effect on reducing distress (Goldschmidt et al., 2012; Moore et al., 2018; Munsch et al., 2012; Smyth et al., 2007). Meaning, albeit maladaptive, this

behaviour does act as an effective coping strategy. Indeed, such episodes having a somewhat instant effect on alleviating the struggles involved in emotion dysregulation can explain the previous findings that emotion dysregulation is an inherent part of the loss of control experienced during binge eating episodes (Racine & Horvath, 2018; Vannucci et al., 2013). These findings also contribute to the idea that among individuals with BED, food becomes the regulator of controlling difficult experiences and expression of emotion (Dingemans et al., 2017; Goldschmidt et al., 2012; Meule et al., 2021; Moore et al., 2018; Munsch et al., 2012; Smyth et al., 2007). As such, this explains the role of emotion dysregulation, especially when experienced at the vulnerable and unstable time of childhood can foster this unhealthy relationship with food as a way to cope with the ongoing struggle (Goldschmidt et al., 2012; Meule et al., 2021; Moore et al., 2018).

Conversely, our first and second hypotheses indicating that a higher level of emotion dysregulation at age 10 – 11 will increase the risk of partial syndrome AN and BN at age 14 – 15 were not supported by the results of the current study. As such, our results also contradict the findings of previous studies in the area. Indeed, these previous literatures suggest that emotion dysregulation has a similar effect on BN and BED, predominantly due to the shared symptom of objective binge eating (Weinbach et al., 2018). However, this was not supported by our finding as emotion dysregulation at the age of 10 - 11 only shared a significant association with BED in adolescence.

On the other hand, the result of no significance between emotion dysregulation and partial syndrome AN can be somewhat supported by other studies as previous findings indicate that restrictive-type AN patients are more likely to use adaptive emotion regulation strategies (i.e., reduced impulsivity) compared to those with other EDs including BED (Anderson et al., 2018; Danner et al., 2012; Sloan et al., 2017). That said, the finding of no association between AN and emotion dysregulation is still not supported by these studies

(Anderson et al., 2018; Danner et al., 2012; Meule et al., 2021; Sloan et al., 2017). Indeed, Racine and Wildes (2013) suggest that restrictive type AN is significantly associated with difficulties in accessing and employing effective regulation strategies as opposed to displaying impulsive ones. More specifically, Wang et al. (2018) and Nowakowski et al. (2013) indicate that this type of AN is facilitated by the lack of emotional awareness, avoidance of negative emotions, suppression of emotional experiences and are present with overall symptoms of alexithymia (i.e., issues in articulating and expressing emotions).

Therefore, the insignificant association can be potentially attributed to the characteristics of the present study. Upon further examination of the SATI-reactivity subscale used to measure emotion dysregulation in the present study, it is found that the items of this measure appear to better reflect the emotion regulation strategies around impulsivity (Foley et al., 2008). Prior literature indicates that emotion regulation strategies around impulsivity and negative urgency are significantly related to binge-purge type AN, BN and BED (Monell et al., 2018). Within our partial syndrome AN sample, nearly all participants (n = 268) met the criteria for restrictive-type AN (see Appendix B). Therefore, it can be suggested that our measure of emotion dysregulation is more useful for capturing the dysregulated strategies involved in developing binge-type EDs than avoidant based strategies of AN.

However, as per this guideline, we should detect a significant association between emotion dysregulation and BN as well (Monell et al., 2018). Examination of the present study's partial syndrom BN sample suggest that of 68 adolescents with partial syndrome BN, the majority (n = 56) met the criteria for BN due to having symptoms of only compensatory behaviours and overvaluation of weight (see Appendix E). Only a small number (n = 11) of people met the criteria for partial syndrome BN as a result of objective binge eating, all of whom contributed to a significant association between partial syndrome BED and emotion dysregulation. Meaning, that the general characteristics of the partial syndrome BN sample

are not consistent with that of the BED group, which can explain the difference in results. Consistently, Brown et al. (2020) found that emotion dysregulation symptoms (measured with borderline personality disorder symptoms at 11) did not predict purging at age 14 (Brown et al., 2020). This proposes that the previous findings of negative urgency and other impulsive strategies involved in emotion dysregulation among individuals with BN can potentially be due to the associated binge eating symptoms (Weinbach et al., 2018). As such, the lack of binge eating symptoms in the current BN sample may explain this poor association (Goldschmidt et al., 2012; Monell et al., 2018; Weinbach et al., 2018).

It is also worth noting that only engaging in behaviours of purging in the absence of bingeing is referred to as non-compensatory behaviour (Liebman et al., 2020). Liebman et al. (2020) indicate that these non-compensatory behaviours as well as overvaluation of weight are more common among individuals employing maladaptive regulation strategies such as behaviour inhibition (i.e., avoidance, withdrawal) rather than impulsive behaviour (Weinbach et al., 2018). As such, the absence of a significant association may be a result of the inhibitory avoidant strategies involved in the symptoms presented by the majority of this sample, which is not accounted for by our measure of emotion dysregulation.

4.3 Limitations

Although our study has promising implications, it is not without limitations. Firstly, while the short form SATI-reactivity is a well-validated measure of emotion dysregulation among children, given its brevity, it does not adequately capture the various types of emotion regulation strategies involved in different EDs such as AN and BN (Allen et al., 2015). Indeed, the other core aspect of emotion dysregulation involving the experiences of emotional distress including suppression or avoidance of emotion is not accounted for (Brockmeyer et al., 2014). This can mean that we do not have clarity regarding whether the

absence of significant association for the partial syndrome AN and BN models is reflective of the true association or a result of this measure.

Additionally, it must be noted that BEDT shows promising psychometric characteristics (Selzer et al., 1996). However, an important caveat for interpreting the results of this study is that BEDT is a screening tool for partial syndrome EDs. Therefore, it lacks the same standard of rigour as a clinical interview with a mental health professional. As such, BEDT may be more vulnerable to capturing false positive cases (Hughes et al., 2019). This is emphasised by the prevalence of partial syndrome AN being 11.1%, which is larger than the population prevalence found among other previous studies on adolescents (0.1%–2.5%) (Allen et al., 2013; Flament et al., 2015; Micali et al., 2015; Mitchison et al., 2020). Indeed, our prevalence also appeared to be greater than that of the other [kindergarten] LSAC cohort (0.2% – 2.5%; Bisset et al., 2019; Hughes et al., 2019; Mitchison et al., 2020). That said, further examinations of our data (refer to Appendix E) suggest that this difference between the two cohorts can partially be attributed to our complete sample having a considerably high rate (18%) of children who are underweight (Appendix E). Indeed, the prevalence of being underweight was the second highest in the present study, whereas, within the other LSAC cohort, the underweight group usually has the lowest prevalence of any other BMI group (i.e., 6% at age 14 - 15) (Hughes et al., 2019).

Similarly, despite BED being one of the most common types of ED, the current study as well as the previous studies using LSAC data suggest that this partial syndrome has the lowest prevalence (Bisset et al., 2019; Hughes et al., 2019; Paxton et al., 2012). This can be attributed to the fact that BEDT was initially developed to measure partial syndrome AN and BN. Therefore, the newly developed criteria for BED within this measure may lack the sensitivity in capturing true cases (Bisset et al., 2019; Hughes et al., 2019)

In addition, the association between partial syndrome BED and emotion dysregulation was made based on only 17 positive cases. Therefore, there is a possibility of inflated odds ratios. While the finding of significance is corroborated by Brown et al. (2020), it would still be beneficial to have future studies investigate this relationship to ensure that the pattern of the effect remains.

4.4 Strengths

One of the many strengths of this study is that it has a very large sample (n=2429). Due to this sample size, more reliable and valid conclusions can be made from our results. This also means that this study has considerable power to detect true effects.

Additionally, another strength of this study is the longitudinal nature of the study. This can allow for a better understanding of the sequences of events and the cause-and-effect relationships over time. For instance, in our study, it is evident that emotion dysregulation at an earlier period is associated with developing partial syndrome BED at a later time. Also, by not using retrospective recollection of MPD and emotion dysregulation at age 10 – 11, we minimise possible biases (i.e., recall bias; Hughes et al., 2019).

Our efforts to measure the different types of partial syndrome ED separately is also considered a strength as it ensured that we would be able to detect the differential effect of emotion dysregulation on the different EDs. This allowed us the understand the importance of emotion dysregulation at 10 –11 in increasing the risk of specific EDs and to what extent.

Moreover, by employing the well-established DSM-5 adaptation of the BEDT criteria classification of EDs, our study presented more relevant and applicable results, which can be interpreted in the context of a respective ED (Bisset et al., 2019; Hughes et al., 2019; Mitchison et al., 2020). Additionally, we were also able to account for partial syndrome BED.

4.5 Implications and future directions

The results of this study have promising theoretical and practical implications. As discussed, the presence of emotion dysregulation within EDs have long been confirmed (Brockmeyer et al., 2014; Mallorquí-Bagué et al., 2018). Yet, to our knowledge, this is one of the first studies to highlight the presence of emotion dysregulation prior to the development of partial syndrome BED in adolescence. This significant positive relationship between emotion dysregulation and partial syndrome BED indicates that alleviating emotion dysregulation at 10 – 11 years of age can potentially reduce the odds of developing partial syndrome BED in adolescence. Due to the previous findings suggesting that this 10 – 11 year old period is the best time to improve emotion regulation skills, our significant results can inform future prevention and intervention efforts to alleviate the development and impact of BED (Moltrecht et al., 2021).

Additionally, the findings of insignificance for partial syndrome AN and BN may be a result of the measure used for emotion dysregulation. Therefore, future studies should look into this age group using a measure that captures a wide range of emotion dysregulation strategies. However, if this age is not found to be important, studies should also investigate when this construct becomes a significant predictor for AN and BN in adolescence. Indeed, it is worth noting that some studies even suggest that emotion dysregulation in the form of disengagement (i.e., avoidance and denial of emotions) or involuntary involvement (i.e. impulsive behaviours) around the age of 12 may also be predictive of later mental health outcomes (Rawana et al., 2014). Therefore, perhaps emotion dysregulation at a later period can contribute to a more significant relationship for partial syndrome AN and BN, than emotional dysregulation measured earlier in childhood. Due to the substantial consequences associated with these EDs, it is important for further studies to explore this relationship in order to construct effective early interventions.

While it was not a core aim of our study, our findings also show that males (41.2% - 57.2%) are largely represented within ED groups (see Table 3). This was especially the case with the partial syndrome AN, where males were over-represented compared to females. These findings are inconsistent with other previous studies on the prevalence of EDs, which emphasise the significantly lower representations of male adolescents; studies using the other LSAC cohort studies demonstrate a similar distribution (Hughes et al., 2019; Mitchison et al., 2020). Upon further examination of the present study's data (refer to Appendix E), the male preponderance for AN was found to be a result of a greater number of males being underweight (primary symptoms of AN), despite more females still displaying the other two symptoms of body dissatisfaction and fear of gaining weight. That said, even among the other two EDs, male representation appears to be relatively larger than previously found. As such, this study may be highlighting the growing prevalence of EDs among males. Indeed, Limbers et al. (2018) suggest that the prevalence of ED among males are on the rise. This may be due to increased clarity among clinicians in diagnosing, treating, and understanding males with EDs as well as the reducing, albeit still not ideal, stigmatisation around seeking help (Strother et al., 2012). Indeed, Lavender et al. (2017) suggest that an increasing number of men also face psychosocial pressures relating to unrealistic muscularity, which can lead to muscularity-oriented disordered eating (Strother et al., 2012). This study not only contributes to such studies by highlighting this growing prevalence of EDs among men, but also the need for future studies to explore gender differences within the causes and manifestation of ED. This understanding can potentially inform future treatments as well as early interventions (Lavender et al., 2017; Limbers et al. 2018; Strother et al., 2012).

4.6 Conclusion

This study contributes valuable findings to the current literature, suggesting that emotion dysregulation at age 10 – 11 is significantly associated with partial syndrome BED at

age 14-15, but not AN or BN. This finding provides a potential foundation to determine the best window of time as well as modifiable risk factors such as emotion dysregulation, which can be targeted for early intervention for BED. However, further research is still needed to confirm these findings and better understand the role of emotion dysregulation in the development and maintenance of AN and BN in adolescence.

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Appendices

Appendix A

School-Aged Temperament Inventory – Reactivity subscale – Shortened form

Please tick the response that best describes how often your child's behaviour matches the behaviour described in each item:

- Reacts strongly (cries or complains loudly) to a disappointment or failure

- When angry, yells or snaps at others

- Moody when corrected for misbehaviour

- Responds intensely to disapproval (shouts, cries, etc.).

Responses:

1 - Never

2 - Rarely

3 - Half the time

4 - Frequently

5 - Always

Appendix B

As displayed in Table B1.1, only a small proportion of participants with ED met criteria for more than one disorder. In line with DSM-5 criteria, the participant (n=1) meeting criteria for all 3 partial syndrome eating disorder is likely to represent partial syndrome binge-purge type Anorexia (APA, 2013).

Table B1.1

Details on the frequency and percentage of the participants meeting multiple partial syndrome eating Disorders

	Number of people that met the criteria (%)
Meeting criteria for partial syndrome bulimia nervosa and anorexia nervosa	6 (1.7%)
Meeting criteria for partial syndrome bulimia nervosa and binge eating disorder	11 (3.1%)
Meeting criteria for partial syndrome anorexia nervosa and binge eating disorder	0 (0.0%)
Meeting criteria for all three partial syndrome eating disorders	1 (0.3%)

Note. There were a total of 18 overlapping cases; the percentage reflects the proportion that met more than one disorder in the eating disorder population (n=354); for example n = 6 or 1.7% of the eating disorder population met criteria for both partial syndrome anorexia nervosa and bulimia nervosa.

Appendix C

The 6-item Kessler psychological Distress scale

In the past 4 weeks how often …..
- Did you feel nervous?

- Did you feel hopeless?

- Did you feel restless or fidgety?

- Did you feel that everything was an effort?

- Did you feel so sad that nothing could cheer you up?

- Did you feel worthless?

LSAC's coding of the response:

1 - All of the time

2 - Most of the time

3 - Some of the time

4 - A little of the time

5 - None of the time

Recoded responses - to fit standard convention:

4 - All of the time

3 - Most of the time

2 - Some of the time

1 - A little of the time

0 - None of the time

Appendix D

Table D1.1

Binomial logistic regression (95% CI) of adjusted and unadjusted models of primary diagnosis partial syndrome eating disorders and emotion dysregulation

n = 2429	Emotion Regulation (Unadjusted)	Emotion Regulation (Adjusted [a])
Partial syndrome Anorexia Nervosa		
Odds Ratio	1.13	1.12
95% Confidence interval	0.96 – 1.33	0.95 – 1.32
p-value	0.16	0.19
Partial syndrome Bulimia Nervosa		
Odds Ratio	1.09	1.06
95% Confidence interval	0.78 – 1.51	0.76 – 1.48
p-value	0.63	0.72

Note. This analysis considers only the primary classification of partial syndrome eating disorder; people meeting multiple partial syndrome eating disorders were classified into one group based on previous study's guidelines; primary diagnosis was made based on the key that anyone with partial syndrome bulimia nervosa should not meet criteria for anorexia nervosa and participants for binge eating disorder must not meet criteria for any other partial syndrome eating disorders; no analysis was done for partial syndrome binge eating disorder because only n = 5 met the criteria.

[a] this model is adjusts for maternal postnatal distress

Consistent with table D1.1, even when primary diagnosis of one ED is identified, the pattern of effect remains the same as emotion dysregulation at 10 – 11 is not significantly associated with increasing the risk of partial syndrome AN or BN at 14 – 15 years of age

Appendix E

Table E1.1 shows the prevalence of ED symptoms in our current sample of adolescents (14 – 15 years of age). This estimated prevalence is consistent the LSAC's other [kindergarten] cohort sample of adolescents (14 – 15 years of age; Hughes et al., 2019). That said, our sample represents a higher rate of underweight adolescents, mainly males. However, in the criteria relating to the other symptoms of ED, females are overrepresented. Due to the higher rate of underweight males, they have a higher prevalence for AN. Other two EDs disproportionately affected female adolescents.

Table E1.1

The estimated prevalence of Eating Disorder symptoms in population and by weight status

	Sample n = 2429	%	Underweight (%)	Healthy (%)	Overweight (%)	obese(%)
Body Mass Index	-	-	18.0%	64.3 %	13.2%	4.4%
Male	1282	52.8%	11.9%	32.4%	5.9%	2.5%
Female	1147	47.2%	6.1%	32.0%	7.3%	1.9%
Fear of gaining weight or behaviours that interfere with weight gain	345	14.2%	0.7%	8.7%	3.5%	1.2%
Male	97	4.0%	0.3%	2.4%	0.9%	0.4%
Female	248	10.2%	0.5%	6.3%	2.6%	0.8%
Disturbance in experience of body weight or overvaluation of weight	896	36.9%	11.0%	21.3%	3.4%	1.2%
Male	357	14.7%	6.3%	7.1%	0.9%	1.2%
Female	539	22.2%	4.7%	14.2%	2.5%	0.8%
Objective binge eating	17	0.7%	0.04%	0.5%	0.2%	0.04%
Male	7	0.3%	0.04%	0.2%	0.1%	0.0%
Female	10	0.4%	0.0%	0.3%	0.1%	0.04%
Overvaluation of weight	472	19.4%	2.1%	13.0%	3.2%	1.2%
Male	157	6.5%	0.9%	4.3%	0.9%	0.4%

Female	315	13.0%	1.1%	8.7%	2.3%	0.8%
Regular engagement in compensatory behaviours	114	4.7%	0.5%	3.0%	1.0%	0.3%
Male	58	2.4%	0.3%	1.5%	0.6%	0.1%
Female	56	2.3%	0.2%	1.5%	0.4%	0.2%
Partial syndrome Anorexia Nervosa	269	11.1%	11.1%	0.0%	0.0%	0.0%
Male	154	6.3%	6.3%	0.0%	0.0%	0.0%
Female	115	4.7%	4.7%	0.0%	0.0%	0.0%
Partial syndrome Bulimia Nervosa	68	2.8%	0.3%	1.8%	0.6%	0.1%
Male	28	1.2%	0.2%	0.7%	0.2%	0.0%
Female	40	1.6%	0.1%	1.1%	0.4%	0.1%
Partial syndrome Binge Eating Disorder	17	0.7%	0.04%	0.5%	0.2%	0.04%
Male	7	0.3%	0.04%	0.2%	0.1%	0.0%
Female	10	0.4%	0.0%	0.3%	0.1%	0.04%

Note. Each percentage reflects the proportion of participants meeting the specific characteristic[s]; for example: 1.8% of the population meet the criteria for partial syndrome bulimia nervosa and are in healthy weight range.