

PREVALENCE AND PREDICTORS OF EARLY ONSET EATING DISORDERS: A  
LONGITUDINAL INVESTIGATION OF THE ADOLESCENT BRAIN AND COGNITIVE  
DEVELOPMENT STUDY

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### **Abstract**

The purpose of this dissertation is to address gaps in the eating disorder literature across three related studies. The planned studies will utilize data from the Adolescent Brain and Cognitive Development study, a large, multi-site, ten-year longitudinal study of 9–10-year-old children. This study is the largest long-term study of brain development and child health in the United States, and it includes data from clinical interviews, behavioral and neurocognitive tasks, and biospecimens of roughly 12,000 children. The first study plans to examine prevalence of early onset eating disorders over three timepoints, and demographic characteristics of the sample with eating disorders. The second and third studies aim to assess temporal risk factors of eating disorders; particularly, comorbid mental disorders and high risk behaviors. Past literature indicates high rates of psychiatric comorbidity among individuals with eating disorders, particularly mood, anxiety, and obsessive-compulsive disorders. While some literature indicates anxiety disorders and obsessive-compulsive disorder onset typically precedes the onset of eating disorders, associations between comorbid disorders and specific eating disorder subtypes remain unclear, and they have yet to be studied longitudinally prior to adolescence. Paper three is a longitudinal examination of the experiential avoidance model. Specifically, self-harm and suicidality are associated with a variety of disordered eating behaviors and may serve some of the same underlying functions. Previous longitudinal studies are relatively limited, and this is the first to examine these behaviors temporally among children. Taken together, these three studies aim to address the present lack of clarity in the literature on distinctive risk factors of various eating disorders among a large and diverse sample of children. Further, the present studies seek to clarify the nature of eating disorder psychopathology in youth, to improve the identification of individuals at risk for eating disorders, and to inform the development of improved prevention and treatment efforts.

### **Abbreviations**

Anorexia nervosa (AN), anxiety disorder (AD), binge eating disorder (BED), body mass index (BMI), borderline personality disorder (BPD), bulimia nervosa (BN), cognitive behavioral therapy (CBT), Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5), dialectical behavior therapy (DBT), eating disorder (ED), generalized anxiety disorder (GAD), Kiddie Schedule for Affective Disorders and Schizophrenia Present and Lifetime Version (KSADS-PL), non-suicidal self-injury (NSSI), obsessive compulsive disorder (OCD), other specified feeding and eating disorder (OSFED).

## Introduction

Eating disorders (EDs) are psychological disorders that can involve a range of maladaptive eating patterns. Disordered eating encompasses a broad variety of behaviors, for example, restricting one's caloric intake by reducing the overall amount of food one eats, limiting certain types of food, or fasting for extended periods. Disordered eating is not always restrictive in nature, and can also include binge eating episodes, and/or engagement in purging behaviors to compensate for an eating binge (APA, 2013). Typically, individuals with EDs experience high levels of distress surrounding eating such that it may negatively impact many facets of life, such as physical health, social interactions, performance in work or school, and overall well-being (Bohn et al., 2008). Engagement in disordered eating is often initially due to a desire to change one's physical appearance, and thus often coincides with body image disturbances and/or a strong fear of weight gain (APA, 2013).

Though all EDs are similar in that they involve distress and impairment stemming from an assortment of eating behaviors, there are notable differences between specific subtypes of EDs. Anorexia nervosa (AN) is a restrictive ED that is associated with intense fear of weight gain, and maintenance of a body weight that is below a healthy level based on age, sex, developmental trajectory, and physical health ("DSM-5 Changes", 2016). Bulimia nervosa (BN) involves the regular occurrence of binge episodes, defined as eating a significant amount of food in one sitting while experiencing a reported loss of control, and subsequent purge episodes to compensate for binge eating. Purge episodes can include purposeful vomiting, use of laxatives, diet pills, and/or diuretics to influence one's weight (APA, 2013). Binge eating disorder (BED) is a relatively new ED, introduced in the most recent Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013). BED, like BN, involves engagement in binge eating episodes

that involve eating an objectively large amount of food, perceived loss of control during eating, and strong negative emotions following the binge, but are not followed by purge episodes (APA, 2013). Diagnostic criteria for these three main subtypes of EDs vary significantly, as do the populations most likely to develop them.

In general, EDs typically develop in adolescence or early adulthood (Hudson et al., 2007; Merikangas et al., 2010). Age of onset is generally consistent between AN and BN (Volpe, 2016) and does not frequently occur outside of this range from early adolescence to early adulthood. Though BED also typically develops in adolescence, there is more variability in the age of onset (Striegel-Moore & Bulik, 2007). Age is not the most robust demographic risk factor related to EDs; there is a significant difference in the risk of developing any ED across biological sex. Females are more likely to be diagnosed with an ED at any point post-puberty (Hoek, 2006; Merikangas et al., 2010; Mitchison et al., 2020), and females are more likely to engage in disordered eating throughout the lifespan (Anderson & Bulik, 2004; Lewisohn et al., 2002; Peat et al., 2008). The exception is BED, where prevalence rates are much closer to equal across men and women relative to other eating disorders (Barry et al., 2002). Until recently, males were often ignored in ED-focused research, but they have higher rates of EDs than once believed (Murray et al., 2017). Though rates of disordered eating among men are still comparably lower than women, men do engage in higher rates of anabolic-androgenic steroid use for appearance-related purposes (Pope et al., 2014). In sum, young adult and teenage women appear to be at the greatest risk of developing EDs, and additional research has been instrumental in detailing the prevalence of various EDs in diverse samples.

### *Prevalence of Eating Disorders*

ED prevalence differs across various groups, and often depends on how EDs are defined in a given study. Among adults, one systematic review of 33 prevalence studies found that lifetime prevalence of any eating disorder was 1.69%, with women having significantly higher rates than men (Qian et al, 2021). BED is the most prevalent subtype of ED, followed by BN, and least commonly, AN. The same systematic review found the AN to BN to BED ratio is 1:4:10, and all are significantly more common among women (Qian et al., 2021). Among individuals who have any ED, BED and other specified feeding and eating disorders (OSFED) are among the most common diagnoses (Smink et al., 2012), and generally require less intensive treatment than ‘traditional’ EDs such as AN and BN. Among individuals with EDs, rates of treatment-seeking are relatively low, ranging from roughly 34% to 63% of patients ever reporting seeking treatment (Coffino et al., 2019). Cases that require intensive care such as hospitalization are most commonly AN and BN (Lock et al., 2016; Patel et al., 2019). Considering the high rates of hospitalization for some EDs, and indication that the age of onset of AN and BN may be decreasing (Favaro et al., 2009), an accurate understanding of the prevalence rates of EDs, particularly among children, is highly important.

Early onset EDs, typically considered those that develop pre-adolescence, may be on the rise among girls and boys (Favaro et al., 2009; Morris et al., 2022). Several recent studies have documented the prevalence of early onset EDs; this knowledge is crucial to better understanding the profiles of children at risk and targeting these individuals in prevention work, and in early detection efforts. One study found a prevalence rate of 0.69% for any lifetime ED in Iran among children up to 14 years old (Mohammadi et al., 2020), and other studies on incidence of early onset EDs report between 1.4-3.0 per 100,000 children have a current ED (Madden et al., 2009;

Nicholls et al., 2011). Similar to prevalence studies among adults, BED appears to be the most common subtype of ED in children (Rozzell et al., 2018; Mohammadi et al., 2020), however, prevalence rates among any subtype of ED do not differ dramatically by gender at this stage of pubertal development (Madden et al., 2009; Rozzell et al., 2018). Further investigation of the prevalence of early onset EDs at various points throughout childhood and adolescence is necessary to recognize potential changes in risk by gender, and other demographic factors such as race and ethnicity.

### ***Medical, Psychological, and Treatment Outcomes of EDs***

There is a myriad of negative medical and psychological outcomes associated with EDs. EDs have the second highest mortality rate of any mental disorder, followed by opioid use disorder (Chesney et al., 2014). Specific subtypes of EDs are associated with certain medical complications, for example, self-imposed starvation in AN can lead to arrhythmia, amenorrhea, low blood pressure, and severe nutritional abnormalities that may result in hospitalization (Chidiac, 2019). Purging behaviors consistent with BN, particularly engagement in self-induced vomiting and laxative use, can result in complications that lead to hospitalization (Patel et al., 2018). Notably, medical complications do not arise only when individuals with EDs suffer from “low weight”, typically defined as <18.5 Body Mass Index (BMI), but occur across the weight spectrum (Madden et al., 2009; Peebles et al., 2017). Further, EDs are associated with increased mortality not only due to medical complications, but also from suicide. One study reports a 50-fold increase of death by suicide among people with AN compared to controls (Keel et al., 2003). Despite such harmful sequelae associated with EDs, they are also some of the most treatment-resistant psychiatric disorders (Vitousek et al., 1998; Halmi, 2013). Even following evidence-based treatment, relapse rates and poor treatment outcomes are relatively common,

especially among individuals with AN (Wonderlich et al., 2020). Given the many poor outcomes across various EDs and the need for continued improvement of treatment for restrictive EDs, additional research is needed to better understand their etiology and maintenance factors that contribute to their development and resistance to treatment.

### ***Theoretical Models of ED Development and Maintenance***

An empirically supported framework for the development and maintenance of eating pathology is the dual pathway model (Stice, 2001). The dual pathway model begins with acknowledging societal pressure to obtain the ‘ideal’ body. These pressures can lead to internalization of the body ideal, and to body dissatisfaction. The dual pathway model posits that being dissatisfied with one’s body can lead to dieting behaviors in efforts to change one’s physical appearance. Dieting may present as fasting, counting calories, and/or limiting specific foods such as those high in fats/carbohydrates. These associations are supported by longitudinal studies among adolescent boys and girls (Neumark-Sztainer et al., 2006; Dakanalis et al., 2014) and college-age women (Stice, 2001). Body dissatisfaction not only leads to dieting, but also can separately lead to negative affect (e.g., depression, anxiety, stress). The dual pathway model next proposes that negative affect and dieting can, in turn, predict bingeing behaviors. Bingeing may serve as a distraction from negative mood states, and give brief comfort in the moment by acting upon an urge to eat uncontrollably (Bohon et al., 2009). It is possible that purging behaviors (vomiting, laxative use, and excessive exercise) are affect regulation mechanisms, and can be done to compensate for a binge episode (Stice, Nemeroff, & Shaw, 1996). Bingeing behaviors may also occur due to highly restrictive eating outside of binge episodes; extreme restriction for extended periods of time can lead to a loss of control while eating and consuming large quantities of food in one sitting (Zunker et al., 2011). The model can be applied differently based

on the ED in question, and can be useful in considering various types of disordered eating behaviors including restriction, binge and purge behaviors, as well as body image concerns.

While the dual pathway model is a well-supported theory that explains risk and maintenance factors involved in the restriction, binge, and purge cycle, the experiential avoidance model is a broad theory that was developed to conceptualize the underlying function of a range of harmful behaviors (self-harm, suicidal behaviors, purging, etc.). The experiential avoidance model posits that individuals who experience strong emotional dysregulation use an array of behaviors to distract from their intense negative emotions, which provides temporary relief (Chapman et al., 2006). There are several factors that may influence one's likelihood to act on self-harm or suicidal urges, such as high emotional intensity, poor distress tolerance, and difficulty regulating when emotionally aroused. Typically, individuals who engage in NSSI and other maladaptive, impulsive behaviors tend to exhibit escape and avoidant behaviors in other domains of life, which leads to deficits in interpersonal effectiveness, decreased opportunity to improve distress tolerance, and lack of emotional awareness (Chapman et al., 2006; Chapman et al., 2011). While the experiential avoidance model was developed to better understand NSSI and suicidal gestures/behaviors, particularly in the context of borderline personality disorder (BPD), a recent systematic review revealed strong significant relationships between experiential avoidance and eating disorder symptoms (Gulacan et al., 2025). Dialectical behavior therapy (DBT) is highly influenced by the experiential avoidance model, and focuses on teaching patients to utilize a variety of skills in times of distress as an alternative to harmful and potentially life-threatening behaviors. DBT is widely supported in empirical literature, particularly among those who exhibit high emotional dysregulation (Harvey et al., 2019) as well as those with EDs (Brown et al., 2020). While clinical studies show that a diverse range of

clinical samples can drastically reduce their harmful behaviors by use of DBT skills (Asarnow et al., 2021; Witt et al., 2021), less is known about the developmental or neurobiological risk factors that make emotion dysregulation and associated maladaptive coping strategies more likely to develop in the first place. Longitudinal studies that assess the onset of various harmful behaviors over time will be useful in elucidating these temporal patterns.

Recently, there has been an increase in research interest in positive affect interventions for psychopathology. This began in community samples and individuals with depression and anxiety, and has been expanding to treatment of other disorders, including EDs. A positive emotion amplification model (PE-AMP) has recently been developed to help better conceptualize the onset and maintenance of eating pathology (Selby & Coniglio, 2020). PE-AMP proposes that individuals with EDs display high levels of motivation to lose weight far beyond the capabilities of most people, and that individuals with restrictive EDs are biologically prone to amplified reward responding. This amplified responding is activated through the process of behaviors to lose weight, and positive feedback from weight loss itself. The continued engagement in these behaviors can be explained by aspects of self-determination theory, which posit engagement in behaviors are reinforced by purpose, relatedness, autonomy, and competence (Ryan & Deci, 2000). These behaviors intermittently elicit positive emotion, even as weight loss continues to become more difficult, which lead to disordered eating behaviors becoming inherently fulfilling and rewarding. This model has yet to be tested in its full form in a research setting, though Selby and Coniglio (2020) suggest future applications of the model that include prevention efforts intended to break the positive feedback cycle proposed by the PE-AMP model. Given high rates of comorbidity across EDs, the PE-AMP model may also be useful in targeting symptoms of other co-occurring disorders.

### *Psychiatric Comorbidity in EDs*

EDs are highly comorbid with other mental disorders, with many studies reporting that the vast majority of individuals with EDs have at least one other disorder (Keski-Rahkonen & Mustelin, 2016; Udo & Grilo, 2019). One study of comorbid DSM-5 disorders among adults with EDs found that individuals with any ED subtype had an increased risk for mood disorders, anxiety disorders, post-traumatic stress disorder, substance use disorders, and personality disorders (Udo & Grilo, 2019). While it is not uncommon for psychopathology to be associated with other kinds of psychopathology, there are certain mental disorders that do commonly coincide with EDs. Across the extensive literature on comorbidity in EDs, EDs are most commonly and consistently associated with anxiety disorders, and this relationship has been shown in studies on children, adolescents, and adults (Swinbourne & Touyz, 2007; Rojo-Moreno et al., 2015; Convertino & Blashill, 2022). While anxiety disorders are associated with many ED variants (AN, BN, BED) it is additionally important to consider comorbidity among specific subtypes of EDs. BED is often comorbid with depression (Araujo et al., 2010), though associations between depression and AN and BN have also been established (Mischoulon et al., 2011). Obsessive-compulsive disorders are also commonly comorbid with EDs, in particular, AN (Micali et al., 2011). Studies considering symptom overlap and the potential predictive nature of comorbid disorders are necessary to best inform future ED research and interventions.

Depression and EDs have been linked in past cross-sectional and longitudinal studies. Depressive symptoms are associated with increased appearance-related and weight concerns (Sonnevile et al., 2015) and are also associated with disordered eating, particularly endorsement of loss of control eating, a main feature of binge episodes (APA, 2013). There is some indication of directionality in this relationship; for example, in one study of adolescents, depressive

symptoms predicted later engagement in loss of control eating, but loss of control eating did not predict later depression (Hilbert et al., 2013). However, other findings indicate a bidirectional relationship (Skinner et al., 2012), or loss of control eating leading to depressive symptoms later in life (Tanofsky-Kraff et al., 2011). Given the inconsistent findings related to the directionality in the relationship between depression and binge eating, further research should assess these variables over several timepoints in a large sample.

Suicidality and self-harm commonly coincide with depressive episodes, and typically present during major depression with higher severity and acuity. Suicidality is common among ED samples; individuals with BED are more likely to have a history of suicidal behavior and suicidal ideation (Conti et al., 2017) and suicide is the most common form of mortality among individuals with BN (Huas et al., 2013). Self-harm is also significantly more prevalent among ED samples as compared to the general population and other clinical samples; one meta-analysis found that 27.3% of individuals with EDs had a lifetime history of self-harm (Cucchi et al., 2016). Self-harm in particular may serve as a coping mechanism to manage emotional dysregulation (Chapman et al., 2006), similar to a sense of relief and distraction that is a commonly endorsed function of purging behavior among individuals with BN (Weding & Nock, 2010). Previous literature indicates that self-harm may be more prevalent among ED subtypes that endorse binge and purge symptoms as opposed to restrictive EDs (Cuchi et al., 2017; Amiri & Khan, 2023), however, there is scant literature on these associations among children. More research is needed to better understand these relationships between various ED profiles.

Anxiety disorders and EDs are highly comorbid across many individual studies and systematic reviews (Swinbourne & Touyz, 2007; Rojo-Moreno et al., 2015). There is significant symptom overlap between anxiety disorders and EDs; for example, anxiety disorders involve

intense and distressing worries that are typically out of proportion to the situation, while many EDs involve intense fear of food and/or weight gain. Behaviorally, both anxiety disorders and EDs include avoidance tactics from aversive stimuli that have been shown to work as maintenance mechanisms (Rawal et al., 2010). Studies that incorporate genetic risk have additionally shown shared transmission across anxiety and EDs (Keel et al., 2005). Although longitudinal associations between anxiety disorders and later EDs have been supported (Buckner et al., 2010), it is still unclear if this relationship is due to their respective natural courses or if anxiety disorders are a causal risk factor for later EDs. As such, further examination of comorbidity in EDs could test the prospective relationship between anxiety disorders and EDs bidirectionally and over several time points to better understand their relationship.

Obsessive-compulsive disorders (OCDs) and EDs are often comorbid (Hudson et al., 2007; Udo & Grilo, 2019), though this relationship is not clearly supported in some previous studies (Buckner et al., 2010). One reason for the lack of association between OCD and EDs broadly defined is that there may be a relationship between particular ED subtypes, namely AN. Past findings that assess the relationship between OCD and AN samples largely show a significant relationship both cross-sectionally and longitudinally (Micali et al., 2011; Buckner et al., 2010). One reason for comorbidity between OCD and AN may be due to shared underlying personality features: individuals with OCD or AN typically have high levels of perfectionism, a sense of need for control, and rigid thinking (Dell’Osso et al., 2016). Similar to the relationship between anxiety disorders and EDs, past literature points to a shared genetic etiology between OCD and AN (Yilmaz et al., 2020). The age of onset of OCD varies, and there is some evidence that points to unique phenomenological features of OCD that begins in childhood compared to adult-onset OCD (Garcia et al., 2009). It is certainly possible that shared features of OCD and

AN may be more common of early (or typical) onset OCD, though this has not yet been studied. Studies that assess OCD and EDs throughout various developmental stages may help to better understand this relationship.

In sum, EDs are serious psychiatric disorders associated with detrimental outcomes such as increased mortality, medical complications, high rates of psychiatric comorbidity, and psychological distress. Considering these harmful outcomes in tandem with recent findings that suggest ED development is beginning earlier in life, additional research is necessary to best understand those who are at risk. There are several research areas in need of further examination. Notably, there are no previous studies that detail the prevalence of early onset EDs over several timepoints in a large and diverse sample. Paper #1 of this proposal aims to be the first to do so. To add, though there is extensive literature on comorbidity in EDs, relatively few studies have parsed apart EDs into their specific subtypes and assessed them as both risk factors of later pathology, and as outcome variables. One known study assessed these associations among adult women, and was underpowered to detect some associations (Buckner et al., 2010). Paper #2 of the present proposal aims to expand on these past findings by assessing comorbid disorders and EDs over two timepoints in a sample of roughly 12,000 children by utilizing a longitudinal logistic regression approach. Lastly, it has been made clear that self-harm behaviors and suicidality are strongly related to a host of mental disorders. This is consistent in the ED literature, as self-harm and suicidal behaviors appear to be related with a range of disordered eating behaviors, particularly binge and purge symptomatology. However, research in this area is sparse, and more evidence is needed to clarify these temporal associations, particularly among children. Paper #3 intends to build upon previous findings by examining the longitudinal associations between self-harm, suicidality, and binge-related ED diagnoses. Taken together,

these three research papers aim to contribute to the ongoing development of literature that highlights causal risk factors of ED development and to inform the detection, prevention, and possibly treatment efforts focused on EDs.

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## Paper 1

### **Prevalence and Demographic Risk Factors of Binge-related Eating Disorders Among Children Over Time: Results from a Representative Sample**

Eating disorders (EDs) are related to poor psychological and physical outcomes, such as high rates of mortality and comorbidity with other disorders. Several common EDs such as bulimia nervosa (BN), binge eating disorder (BED), and subclinical variants of these disorders (OSFED) include binge eating, which can be defined as eating an objectively large amount of food and loss of control while eating (Wolfe et al., 2009). Binge eating is also associated with strong negative emotions such as shame, guilt, and embarrassment (O’Loghlen et al., 2022). Recent estimates suggest that BN is associated with a two to four times increased likelihood of mortality compared to controls (van Hoeken & Hoek, 2020), and both BN and BED are highly comorbid with other mental disorders, particularly mood, substance, and anxiety disorders (Mandelli et al., 2020; Swinbourne & Touyz, 2007; Keski-Rahkonen, 2021). These binge-related EDs are also associated with detrimental health consequences. Binge and purge behaviors characteristic of BN can lead to electrolyte imbalances and renal failure (Castillo & Weiselberg, 2017), while BED is associated with higher rates of obesity (Burrows et al., 2017). Given the vast harmful outcomes associated with binge-related EDs, it is necessary to have accurate and current information on the prevalence of EDs starting from childhood, in addition to knowledge of the demographic makeup common among individuals with EDs.

There are several studies that have assessed the prevalence of EDs among children and adolescents in the United States. One nationally representative study found that 0.1% of adolescents between 8-11 years old met DSM-IV criteria for an ED in their lifetime (Merikangas et al., 2011). Two large recent studies have utilized data from the Adolescent Brain and Cognitive Development (ABCD) study, a longitudinal, multi-site study (Volkow et al., 2018), to

estimate prevalence of DSM-5 EDs among children. These studies found a 1.4% prevalence rate of EDs among the baseline sample of 9-10 year-old children (Rozzell et al., 2018) and a 7.2% prevalence rate at one-year follow-up among 10-11 year-old children (Murray et al., 2022). This large difference in prevalence among the same sample is likely due to differences in inclusion of additional subclinical derivatives of particular EDs in the latter study not included in the earlier study. As such, additional studies utilizing accurate and consistent diagnostic criteria are necessary in best understanding potential changes in prevalence over time, and characterizing those at risk.

Findings indicate the age of onset of EDs is often in late adolescence/early adulthood, however, early onset EDs do occur, and are associated with high rates of medical instability (Hudson et al., 2012) and typically require hospitalization (Madden et al., 2009). Trends in age of onset have proven difficult to assess given the changes in diagnostic criteria of EDs, particularly changes in DSM-5. Diagnostic revisions have led to an increase in individuals who meet criteria for a full ED, and a corresponding decrease in those who met criteria for an unspecified eating disorder (Keel et al., 2011; Mancuso et al., 2015). Longitudinal studies utilizing DSM-5 diagnostic criteria may help to better understand the rate of change, or lack thereof, in prevalence of EDs over time.

EDs, particularly AN and BN, appear to be heavily skewed toward females from an early age, with one report estimating that among 5 to 12-year-old children in Canada, the ratio of girls to boys with EDs was 6:1 (Pinhas et al., 2011). A large gender gap beginning in childhood is consistent across findings in various countries such as Iran (Mohammadi et al., 2020), Ireland, and England (Hudson et al., 2012). Literature on sex differences in BED generally point to higher rates among adolescent girls (Mitchison et al., 2020) and adult women (Keski-Rahkonen,

2021). Though there is some consensus that the gender gap in EDs begins early, more recent findings show no significant difference in ED prevalence by sex among children and adolescents (Rozzell et al., 2018; Kjeldberg et al., 2021; Murray et al., 2022). The lack of sex differences in younger samples may be indicative of dynamic risk factors throughout the life span, such as hormonal influences during and post puberty and increasing appearance-related pressures on women throughout development.

While studies on sex differences in eating pathology tend to be relatively consistent in their findings, studies on racial and ethnic differences are not. Several previous studies report that risk for EDs did not differ between white and racial/ethnic minority young adult women (Cheng et al., 2019) or adolescents (Swanson et al., 2011). However, some studies note differences in rates by ED subtype. For example, one study found lower rates of AN among Black and Hispanic respondents as compared to White respondents, whereas there were no differences in rates of BN (Udo & Grilo, 2019). BED additionally has been shown to be similarly prevalent in adults across Hispanic and non-Hispanic groups (Perez et al., 2016). In a recent study utilizing the ABCD sample, Black, Hispanic, and mixed race children had higher self and parent endorsement of weight control behaviors and binge eating (Makowski et al., 2023). This contrasts with one previous study of parent-reported binge eating, which found that among treatment seeking children with obesity, Caucasian children were more likely to binge eat than Black children (Elliot et al., 2013). Further studies should help to establish a clear pattern in possible racial and ethnic differences in EDs, especially longitudinal studies which may help to better understand changes in risk of ED development over time. This may help to delineate possible cultural and developmental relevant risk factors across the lifespan.

Previous large-scale longitudinal studies of disordered eating have typically focused on adolescents and young adults. These studies have highlighted important psychosocial and behavioral risk factors of disordered eating: dieting (Neumark-Sztainer et al., 2011) and desire to be thinner (Halvarsson et al., 2002). While previous findings are instrumental in better understanding risk and protective factors of eating pathology, as are studies that detail the prevalence of EDs cross-sectionally (Merikangas et al., 2011; Rozzell et al., 2018; Murray et al., 2022), there is notably scant literature that highlights the prevalence of young children with current EDs over several timepoints, nor literature that particularly assesses the risk of ED diagnoses over time between different racial and ethnic groups.

The present study aimed to assess the prevalence of several of the most common eating disorders (BN, BED, subclinical BN (OSFED BN), subclinical BED (OSFED BED), and any binge-related ED) among a large and diverse sample of 9-10 year old children in the United States over three timepoints, each one year apart. To our knowledge, this study is the first to assess prevalence of early onset EDs at more than one point in time. The present study additionally aims to describe the racial and ethnic makeup of those in the sample who meet criteria for a binge-related ED, and assess the likelihood of ED diagnoses by gender and race/ethnicity over each of the three timepoints. No directional hypotheses will be made about potential differences in rates of BED and BN across race/ethnicity groups due to mixed results in prior literature. According to past findings on sex differences in EDs and prior studies that utilized the ABCD sample, directional hypotheses are as follows:

- 1) No sex differences in ED prevalence will be detected at Time 1 and the ratio of girls to boys with EDs will increase at year 1 and year 2 follow-up timepoints.

## **Methods**

### **Participants and sampling**

The present study utilized data from three timepoints in the Adolescent Brain and Cognitive Development study. The ABCD study is a ten-year longitudinal cohort study of 9-10 year old children. This large sample is collected at 21 sites across the United States yearly and is intended to match the sociodemographic makeup of the country, recruited for key variables such as age, gender, race and ethnicity, socioeconomic status, and urbanicity (Garavan et al., 2018). There are some exceptions to the intent to match the U.S. population at large in the ABCD study. For example, the ABCD sample targets incorporate an oversampling of African-American and ‘Other’ race (smaller racial groups and multiple-race groups) children and a corresponding slight undersampling of White, Hispanic, and Asian children (Heeringa & Berglund, 2020). These deviations are due to specific study objectives on particular subgroups, such as Native American children and Black children across different states (Garavan et al., 2018). Participants at baseline, collected in 2016-2018, were  $N = 11,747$  children 9-10 years old and one caregiver. At one year follow-up, children were 10-11 years old ( $N = 11,103$ ) and at two-year follow-up, children were 11-12 years old ( $N=10,756$ ). The ABCD data is collected in two separate waves each year. As part of the ABCD study, parents and guardians provided written informed consent (Volkow et al., 2018).

### **Inclusion and Exclusion criteria**

Inclusion criteria for the ABCD study were age (9–10 years of age at baseline) and the ability to provide informed consent (parents) and assent (children). Exclusion criteria were lack of English language proficiency among children in the study and severe intellectual, sensory, or

medical issues that would impact the ability to collect valid data, the child's ability to comply with testing, or contraindications to MRI scanning. It was required that parents be fluent in either English or Spanish language (Garavan et al., 2018). All children enrolled in the ABCD study that had completed the eating disorders subsection of the KSADS-PL (Kaufman et al., 2013) and the demographics information were included in the present study. The final sample was N= 11,747 at baseline, N = 10,995 at year 1 follow-up, and N = 10,652 at year 2 follow up.

### ***KSADS-PL for DSM-5***

Eating disorder diagnoses were determined using parent or guardian responses to the computerized Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) based on DSM-5 criteria (Kaufman et al., 2013). This widely used, semi-structured clinical interview tool is comprised of supplements for most DSM-5 disorders, including an “eating disorders and substance-related disorders” supplement, utilized for the present study (Kaufman et al., 2013). For examples of clinically relevant questions asked in a KSADS administration, see Supplemental Table 1. Parental reports on the KSADS eating disorder supplement were used to make ED diagnoses in the present study, as parents and/or guardians are typically consistent observers of their child's eating behaviors during childhood (Barch et al., 2018). The KSADS-PL categorizes ED diagnoses into 6 clinical subtypes: AN, BN, BED, and subclinical levels of these (OSFED AN; OSFED BN; OSFED BED) based on DSM-5 diagnostic criteria. A global ‘any ED’ variable was also created.

### ***Classification of Eating Disorders***

While the KSADS instrument includes diagnostic criteria for six subtypes of EDs, four are utilized for the present study: BN, BED, OSFED BN, and OSFED BED. OSFED AN, while

available within the present dataset, was not used in the study given the diagnostic threshold used to determine these diagnoses, similar to some past literature utilizing the dataset (Convertino & Blashill, 2022; Rozzell et al., 2018). Only one criterion must be present to confer an OSFED AN diagnosis in the present KSADS, which is ‘low body weight’. This is discrepant with DSM-5 criteria, which states all criteria *except* low body weight confer a diagnosis of OSFED AN (American Psychiatric Association; 2013). Moreover, there are other restrictive EDs (such as Avoidant and Restrictive Food Intake Disorder) not assessed within the KSADS that may better capture individuals who meet criteria for OSFED AN in the present study based on low weight alone, but do not have other hallmark features of AN such as fear of weight gain and or body image disturbances (Fisher et al., 2014). Full threshold AN was not included in the present analyses due to reported assessment related issues of this KSADS module; all related data was subsequently removed from the ABCD databases at all three timepoints. The four remaining subtypes of EDs were classified based on common hierarchy of DSM-5 criteria, with BN > BED > OSFED BN > OSFED BED (Udo & Grilo, 2019).

### **Statistical Analysis**

Statistical analysis was performed using RStudio version 7.2 (RStudio Team, 2020). Sex at birth and race/ethnicity were used as predictors of ED diagnoses in logistic regression models at each timepoint. ED diagnosis outcomes were ‘any binge-related ED’, ‘BN’, and ‘BED’. The BED variable was created by combining variables that measured any diagnosis of BED, OSFED BED, and BED in partial remission. An individual was coded a ‘1’ if they met criteria for full threshold BED, OSFED BED, or BED in partial remission. An individual was coded a ‘0’ if they did not meet criteria for any one of these three diagnoses. The BN variable was created by combining any diagnosis of BN, OSFED BN, and BN in partial remission. An individual was coded a ‘1’ if they

met criteria for full threshold BN, OSFED BN, or BN in partial remission. An individual was coded a '0' if they did not meet criteria for any one of these three diagnoses. The 'any binge-related ED' variable was created by combining all six of the aforementioned diagnoses.

## **Results**

### **Demographic Characteristics**

At baseline, participants were all 9-10 years old. Sex at birth among participants was 52.2% male and 47.8% female. The racial and ethnic breakdown of the sample at baseline is as follows: 52.0% White, 15.0% Black, 20.3% Hispanic, 2.1% Asian, and 10.5% Other. The ABCD sample mirrors the population estimates produced by the American Community Survey (ACS; 2020), particularly major demographic groups (White, Black, Hispanic). Children of Asian ancestry are underrepresented in the present ABCD sample, while children identified as 'Other' are slightly overrepresented compared to 2020 ACS estimates (Heeringa & Berglund, 2020).

Within the ABCD sample, there are several race and ethnicity categorizations. As part of the parent-report questionnaires, Parents were asked the following question: "What race do you consider the child to be? Please check all that apply. European American; Black/African American; American Indian, Native American; Alaska Native; Native Hawaiian; Guamanian; Samoan; Other Pacific Islander; Asian Indian; Chinese; Filipino; Japanese; Korean; Vietnamese; Other Asian; Other Race; Refuse to Answer; Don't Know." Parents were separately asked: "Do you consider the child Hispanic/Latino/Latina?" For the present study, children were grouped into one of the five major race/ethnicity categories: Non-Hispanic White, Non-Hispanic Black, Non-Hispanic Asian, Hispanic, and 'Other/Mixed race'. These five categories were used to target specific subsamples in the larger ABCD study (Garavan et al., 2018).

## ED Prevalence

Across all binge-related ED diagnoses, the overall prevalence rate was 1.4% (95% CI: 1.2%-1.6%) at baseline, 1.8% at 1-year follow up (95% CI: 1.5%-2.0%), and 1.7% at 2-year follow up (95% CI: 1.4%-2.0%). Prevalence rates at baseline were significantly lower than prevalence rates at 1-year follow up ( $\chi^2 = 5.81$ ;  $df = 1$ ;  $p = .02$ ), but did not significantly differ from prevalence rates at 2-year follow up ( $\chi^2 = 2.53$ ;  $df = 1$ ;  $p = .11$ ). Prevalence rates at 1-year follow up and 2-year follow up did not significantly differ ( $\chi^2 = 0.24$ ;  $df = 1$ ;  $p = .63$ ).

## Demographic Predictors of ED Diagnoses

Logistic regression analyses were conducted at each timepoint to assess demographic risk factors of binge-related EDs. See Table 1 for the complete logistic regression results at baseline. At baseline, there were no differences in likelihood of any BN diagnosis, any BED diagnosis, or any binge-related ED based on sex at birth. Race/ethnicity was a significant predictor of all three ED outcomes. In particular, Black and mixed race children had significantly increased odds of having any BED diagnosis and any ED diagnosis at baseline. Hispanic children had increased odds of having any BN diagnosis and any ED diagnosis at baseline. Hispanic children additionally had marginally significant ( $p < .10$ ) increased odds of any BED diagnosis.

At year 1 follow up, similar to the baseline timepoint, there were no differences in likelihood of any BN diagnosis, any BED diagnosis, or any ED based on sex at birth. Race/ethnicity was a significant predictor of all three binge-related ED outcomes. In particular, Black children had significantly increased odds of having any BN diagnosis and any binge-related ED diagnosis. Hispanic children had significantly increased odds of any BED diagnosis,

any BN diagnosis, and any binge-related ED diagnosis. See Table 2 for the complete logistic regression results at 1 year follow up.

At 2 year follow up, there were no significant differences in likelihood of any BN diagnosis, any BED diagnosis, or any binge-related ED based on sex at birth. Race/ethnicity was a significant predictor of any BED diagnosis and any binge-related ED, but not any BN diagnosis. Hispanic ethnicity significantly increased likelihood of any BED diagnosis and any binge-related ED. No other race and ethnicity groups were at significantly increased or lower risk of any ED diagnoses at this timepoint. See Table 3 for the complete logistic regression results at 2 year follow-up.

### **Discussion**

The current study set out to assess prevalence of binge-related EDs across three time points in the Adolescent Brain and Cognitive Development study sample. Overall, there was little change over time in prevalence of parent-reported EDs, and no significant sex differences. Similar to Makowski and colleagues (2023) we found that Black, Hispanic, and mixed race children were at greater risk for binge-related EDs compared to white children in the sample, and no difference in rates of binge-related EDs among Asian and White children. The lack of sex differences in ED prevalence in the present study is consistent with past findings utilizing the ABCD sample (Makowski et al., 2023), but contrasts with other studies that highlight higher rates of binge related EDs among girls (Mitchison et al., 2020; Mohammadi et al., 2020). Given that the ABCD study sets out to conduct clinician administered interviews for DSM-5 disorders over ten years, this data should continue to be assessed for changes in ED prevalence by sex over the course of adolescence and young adulthood and compared with other samples.

A particularly notable finding in the current study was the significantly increased risk of binge-related EDs among several racial/ethnic minority groups across all three timepoints. Hispanic children were at significantly greater risk for BN, BED, or both EDs compared to their white counterparts at baseline, year 1, and year 2 follow-up. This consistently higher risk across three years highlights the need for further research that aids in a better understanding of culturally relevant risk factors that may play a role in binge eating behaviors, beginning at a young age. The present findings are in line with a systematic review and meta-analysis that found higher prevalence rates of BN and BED in Latin countries as compared to Western countries (Kolar et al., 2016). Some culturally-focused contributors to eating pathology have been probed among Latino samples; Simmons and Limbers found a relationship between acculturation related stress and endorsement of emotional eating in Latino youth (2019). Additionally, BED is more prevalent among US-born Mexican Americans compared to individuals who did not emigrate to the US (Swanson et al., 2012), indicating there are possible cultural stressors related to immigration status that may be involved in binge eating.

Black and mixed race children in the present study were also at increased risk for binge-related EDs at baseline and year 1 follow up. While acculturation related stress could potentially be a factor that needs more exploration among racial/ethnic minorities other than Hispanic/Latino samples, the heightened risk among Black and mixed race children in the present sample could be considered within the context of minority stress theory (Meyer, 2003). Although not assessed in the present study, the minority stress theory has been tested in models of disordered eating among sexual minority populations (Calzo et al., 2017) and racial/ethnic minority adults (Rodgers et al., 2018) which have found associations between discrimination, perceived prejudice, and disordered eating. Additional studies on minority stress theory,

acculturation, and disordered eating are warranted, particularly among young samples. Given that the ABCD study utilizes diverse subsamples from sites across the country, an individual child may not be a racial/ethnic minority in their place of residence despite this status within the larger sample. Additional studies should seek to assess possible differences in rates of mental disorders across study sites to better understand potential risk factors such as minority or socioeconomic status that may vary across the country.

Previous findings within the ABCD sample have shown intersecting identities of race/ethnicity, sex, and socioeconomic status may be related to psychopathology and treatment utilization (Mennies et al., 2021). There are several indirect ways that race/ethnicity may be associated with higher rates of EDs or other mental disorders. Racial and ethnic minorities, particularly Black and Hispanic individuals, are more likely to live below the poverty level, less likely to be home owners, and less likely to have health insurance than their white counterparts (Gibson-Davis et al., 2021; Bazargan et al., 2021), increasing their risk for adverse health and mental health consequences. Racial and ethnic minorities additionally endorse less trust in medical providers (Bazargan et al., 2021), which may lead to lower rates of seeking treatment. Specific to binge-related eating pathology, minority families are more likely to live in food deserts, limiting access to healthy and affordable foods (Reynolds et al., 2024). This is associated with an and increased likelihood of childhood obesity (Key et al., 2023), and could plausibly be further implicated in binge eating.

Several strengths and limitations of the present study should be noted. Strengths of the study include use of a large, demographically representative sample of children over several timepoints. To the author's knowledge, this is the first study to assess demographic risk factors associated with early onset EDs over three consecutive timepoints. The present study is not

without limitations. Notably, the ED diagnoses utilized at all three timepoints in the present study were made via parent-reported symptoms to maintain consistency across time. However, disordered behaviors such as binge eating and purging are often done in secrecy, and may not be observed by parental figures. Studies utilizing the ABCD study should consider several important factors such as age, developmental stage, and symptomatology when choosing to utilize parent-reported or youth-reported versions of the K-SADS. Of note, Asian children were underrepresented in the ABCD study as compared to the US population. Within the sample, only one Asian participant met criteria for a binge-related ED. This particularly small sample reduces the ability to extrapolate findings, and likely contributes to the large confidence intervals and odds ratios in the current study. Further, there was a low prevalence of binge-related EDs in the sample, particularly BN. The current results should be interpreted with caution given that these rare occurrences used as outcomes could skew the findings. Lastly, given the diagnostic criteria utilized within the ABCD for an OSFED AN diagnosis was only based on low weight, we chose not to analyze any results with this diagnosis. Further studies should aim to better understand early onset AN, given its many detrimental health outcomes and treatment resistant nature.

There are many possibilities within the ABCD study to further study risk factors for eating pathology longitudinally. Particularly, based on the present findings, studies may wish to examine sex differences over time among various ED subtypes. Past literature indicates risk is heightened for girls around the time of pubertal development (Klump, 2013) which should be confirmed among a large and diverse sample of children throughout their childhood and adolescence. This will be especially important as the prevalence of EDs in the ABCD sample will likely increase over the course of the study. Future studies should also consider investigating disordered eating behaviors (i.e., self-induced vomiting) as opposed to binary diagnostic

categories to increase understanding of a broad range of disordered eating presentations among children. Given a heightened risk for binge-related EDs among Hispanic, Black, and mixed race children found in our study, as well as other similar investigations (Makowski et al., 2023), additional research is urgently needed to identify risk factors and barriers to treatment that may be specific to these commonly underrepresented groups. The current findings characterized children who have binge-related EDs over several timepoints, and identified demographic risk factors for these EDs. This study may inform future detection, prevention, and intervention work in the ED field and in further ABCD study endeavors.

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**Table 1***Demographic Risk Factors for EDs at Baseline*

Independent Variable	BED			BN			Any ED		
	OR	95% CI	<i>p</i> value	OR	95% CI	<i>p</i> value	OR	CI	<i>p</i> value
Sex at Birth [referent group = Male]									
Female	0.84	0.53, 1.31	.43	0.94	0.028, 2.79	.89	0.85	0.56, 1.28	.43
Race/Ethnicity [referent group = White]									
Black	2.06	1.11, 3.71	.02*	3.49	0.42, 29.10	.21	2.15	1.19, 3.79	.009**
Hispanic	1.71	0.94, 3.03	.07	10.40	2.60, 68.93	.003**	2.27	1.34, 3.82	.002**
Asian	<.001	<.001, 1.08	.97	<.001	NA	.99	<.001	<.001, 4.52	.97
Mixed race	2.24	1.12, 4.23	.02*	2.45	0.12, 25.59	.46	2.25	1.16, 4.17	.01*

*Note.* BED= binge eating disorder; BN= bulimia nervosa; ED= eating disorder; OR= odds ratio; CI= confidence interval.  $p < .05^*$ ,  $p < .001^{**}$

**Table 2***Demographic Risk Factors for EDs at 1 year follow up*

Independent Variable	BED			BN			Any ED		
	OR	95% CI	<i>P</i> value	OR	95% CI	<i>P</i> value	OR	CI	<i>p</i> value
Sex at Birth [referent group = Male]									
Female	0.79	0.54, 1.15	.22	1.07	0.33, 3.41	.91	0.81	0.57, 1.16	.26
Race/Ethnicity [referent group = White]									
Black	1.53	0.89, 2.54	.11	7.58	1.48, 54.72	.02*	1.77	1.07, 2.85	.02*
Hispanic	1.72	1.09, 2.68	.02*	5.51	1.07, 39.76	.05*	1.87	1.20, 2.87	.004**
Asian	<.001	<.001, 1.50	.97	<.001	NA	.99	<.001	<.001, 9.17	.97
Mixed race	1.13	0.56, 2.09	.72	5.10	0.61, 42.77	.10	1.28	0.67, 2.29	.43

*Note.* BED= binge eating disorder; BN= bulimia nervosa; ED= eating disorder; OR= odds ratio; CI= confidence interval.  $p < .05^*$ ,  $p < .001^{**}$

**Table 3***Demographic Risk Factors for EDs at 2 year follow up*

Independent Variable	BED			BN			Any ED		
	OR	95% CI	<i>p</i> value	OR	95% CI	<i>p</i> value	OR	CI	<i>p</i> value
Sex at Birth [referent group = Male]									
Female	1.15	0.80, 1.65	.46	0.82	0.27, 2.38	.72	1.11	0.78, 1.57	.56
Race/Ethnicity [referent group = White]									
Black	1.12	0.62, 1.71	.68	0.77	0.04, 4.76	.81	1.10	0.62, 1.84	.74
Hispanic	1.69	1.09, 2.29	.02*	2.21	0.55, 8.36	.24	1.74	1.14, 2.62	.009**
Asian	<.001	<.001, 1.45	.97	3.12	0.27, 3.20	.14	0.43	.02, 1.94	.40
Mixed race	1.23	0.64, 2.81	.50	3.09	0.63, 12.60	.12	1.39	.77, 2.37	.24

*Note.* BED= binge eating disorder; BN= bulimia nervosa; ED= eating disorder; OR= odds ratio; CI= confidence interval.  $p < .05^*$ ,  $p < .001^{**}$

## Paper 2

### **Specific Eating Disorders and Related Psychopathology: A Longitudinal Investigation of the ABCD Study**

Eating Disorders (EDs) are serious psychiatric disorders characterized by distress and impairment surrounding eating-related thoughts and behaviors (APA, 2013). EDs are often associated with medical complications such as exhaustion, severe malnutrition, and electrolyte imbalances (Castillo & Weiselberg, 2017). EDs are additionally associated with poor mental outcomes, such as high rates of comorbidity with other psychiatric disorders and suicidality (van Hoeken & Hoek, 2020). EDs are typically more common among females, and age of onset is usually post-puberty, in adolescence or early adulthood (Volpe et al., 2016). While EDs are less common in middle childhood, early onset eating disorders can be more severe than EDs diagnosed later in life, and often require more intensive treatment, such as inpatient hospitalization (Madden et al., 2009).

There is vast literature documenting psychiatric comorbidity among individuals with EDs, most commonly anxiety disorders (Swinbourne & Touyz, 2007), followed by mood disorders (Godart et al., 2007). These findings are consistent across younger samples of adolescents and children, with comorbid anxiety disorders present in 40-74% of children with EDs, and depression/mood disorders present among 24-30% of children with EDs (Madden et al., 2009; Convertino & Blashill, 2022). While co-occurring anxiety and mood disorders are commonly associated with EDs broadly, some studies have found associations with specific mental disorders and ED subtypes. For instance, anorexia nervosa (AN) is commonly comorbid with obsessive-compulsive disorder (Jordan et al., 2008; Williams et al., 2022; Yilmaz et al., 2020), while bulimia nervosa (BN) appears to coincide with borderline personality characteristics and depression (Braun et al., 1994; Stice et al., 2004; Jordan et al., 2008; Miller et

al., 2021). Comorbid psychiatric disorders predict worse outcomes in treatment of EDs, particularly BN (Keel & Brown, 2010). This may be especially concerning for younger children diagnosed with an ED, as some recent literature suggests earlier age of onset is associated with longer illness duration prior to treatment (Kwok et al., 2020). Additionally, length of ED is associated with lower likelihood of remission (DeYoung et al., 2020). While many cross-sectional studies have been useful in better understanding comorbidity among diverse samples of individuals with EDs, these studies do not consider comorbid pathology as possible causal risk factors for EDs.

Longitudinal studies on comorbidity in EDs have been informative to help better understand the potential causal role of comorbid psychiatric disorders in the development of EDs, though often report inconsistent findings. Cross-sectional studies on comorbid psychiatric disorders among individuals with EDs consistently show high rates of depression, anxiety disorders, and OCD (Convertino & Blashill, 2022; Mohammadi et al., 2020; Williams et al., 2022), but cannot assume temporal associations. Longitudinal studies have helped to provide such information. One systematic review found that in addition to specific dieting and body image related pathology, other psychiatric disorder-related symptoms (depression and anxiety) were prospectively associated with later EDs among adolescents (McClelland et al., 2020). Similar findings have been replicated in research on specific EDs, for example, depression is consistently identified as a risk factor for subsequent binge eating among children and adolescents (Spoor et al., 2006; Pearson et al., 2015) and OCD has been identified as a risk factor for later AN, but is less consistently associated with BN (Buckner et al., 2010; Micali et al., 2011). This literature is mixed, as another recent finding indicated no association between childhood OCD symptoms and later eating pathology (Schaumburg et al., 2020). Longitudinal

studies on psychological disorders as risk factors for EDs that utilize standardized, clinician-based interviews should be replicated in large, racially and ethnically diverse samples over several timepoints to better understand these temporal associations and clarify previous mixed findings.

Previous studies have helped to make associations between EDs and other disorders that coincide with, and/or precede them. Similarly, some studies have assessed the impact of EDs as risk factors of psychopathology at a later timepoint. Literature on EDs as predictors of later disorders is unclear, for example, Buckner and colleagues assessed these associations bidirectionally, and found that AN was not associated with any lifetime mental disorder 14 years later, however, BN was associated with a subsequent lifetime diagnosis of panic disorder and social anxiety disorder, but no other anxiety disorders or OCD (2010). This differs from another longitudinal study that found any full threshold ED diagnosis predicted anxiety disorders and depression two years later (Micali et al., 2015). Some retrospective studies have found that anxiety disorders are more likely to precede EDs, while depression is equally likely to develop before or after an ED diagnosis (Silberg & Bulik, 2005), while other studies find both anxiety disorders and depression are more likely to develop before EDs compared to after (Feldman & Meyer, 2010). Thus, while longitudinal studies have identified bidirectional associations between various psychological disorders and EDs, additional studies are necessary to build upon previous mixed findings to clarify risk factors and possible outcomes of EDs, particularly among the specific ED subtypes throughout various stages of development.

In sum, relatively limited research has examined temporal associations between psychological disorders and specific EDs as opposed to a broad ‘any ED’ outcome, and the current findings are mixed. Literature indicates that anxiety disorders, depression, and OCD are

all associated with EDs later in life (McClelland et al., 2020; Silberg & Bulik, 2005; Micali et al., 2011), however, results are less clear when assessing risk factors across the various ED subtypes, and further, such studies conducted prior to 2013 do not include BED in assessment of ED subtype (Buckner et al., 2010). Additionally, parsing out the unique temporal relationships between specific disorders and ED subtype has been difficult in past studies, in part due to lack of power to detect these differences (Buckner et al., 2010). Given some contradictory findings in previous longitudinal studies, and relatively limited literature on risk factors in early onset EDs, the present study aims to examine psychological disorders (anxiety disorders and OCD) among 9–10-year-olds as longitudinal predictors of specific EDs (BN, & BED) two years later in a large and nationally representative sample of children. The present study additionally aims to analyze adjusted models; covarying for potential differences in these risk factors across biological sex, age, and race/ethnicity groups. Given the consistent findings that indicate an association between AN and OCD, but limited findings on the co-occurrence of OCD and binge-related EDs, no directional hypotheses were made on the directional relationships between OCD and BN/BED. Based on previous literature on anxiety disorders and eating pathology, the following directional hypotheses were made:

- 1) Any anxiety disorder would predict onset of BN two years later
- 2) Any anxiety disorder would predict onset on BED two years later
- 3) BN would predict the onset of any anxiety disorder two years later
- 4) BED would not predict the onset of any anxiety disorder two years later

## **Method**

### **Participants and Sample**

The present study utilizes data from the Adolescent Brain and Cognitive Development (ABCD) study, a large, longitudinal study that aims to sample a cohort of diverse 9-10 year old children across the United States over the course of ten years. Data from the sample of children at baseline were collected from 21 sites across the country, and aimed to match the population based on sex, race/ethnicity, and socioeconomic status (Garavan et al., 2018). Participants at baseline were N = 11,878 children, and one caregiver each. Participants at baseline were 52.2% male, 48.0% non-White (e.g., Asian, Black, Hispanic, or other/mixed race). Please see the ‘participants and sample’ section from Study 1 for additional details on the ABCD sample and race/ethnicity categorizations for the present study. As part of the ABCD study, parents provided informed consent (Volkow et al., 2018). IRB approval was waived by the University of Hawai‘i at Manoa for de-identified secondary data analysis.

### **Inclusion and Exclusion criteria**

Inclusion criteria for children in the ABCD study was age 9-10 years old at baseline and ability to provide assent. For parents and guardians, inclusion criteria included the ability to provide informed consent and fluency in English or Spanish language. Exclusion criteria for children included lack of English language proficiency, severe intellectual, sensory, and/or medical concerns that would impact data collection, the child’s ability to comply with testing, or inability to complete MRI scanning. All children whose parents completed the Kiddie Schedule for Affective Disorders and Schizophrenia Parent Version (KSADS-PL) at any of the four timepoints were included in the present study. The K-SADS-PL generally shows adequate to strong interrater and test-retest reliability, and construct validity (Ambrosini, 2000; Lauth et al., 2010; de la Pena et al., 2018).

## Measures

*K-SADS-PL, Anxiety Disorders.* Given similar etiology across many anxiety disorders, a binary ‘any anxiety disorder’ variable was created. Disorders included in this variable will be general anxiety disorder (GAD), social anxiety disorder (SAD), agoraphobia, panic disorder, specific phobias, and separation anxiety disorder. Diagnoses for all six disorders will be determined using parent or guardian responses to the computerized KSADS-PL based on DSM-5 criteria (Kaufman et al., 2013). Scores were assigned a "1" if the child met for a current diagnosis of any anxiety disorder based on the KSADS-PL parent interview; scores were assigned a "0" if the child did not meet current criteria. See Supplemental Table 1 for a list of example clinical interview questions from the KSADS for additional detail.

*K-SADS-PL, Obsessive Compulsive Disorder.* OCD diagnoses were determined using parent or guardian responses to the computerized KSADS-PL based on DSM-5 criteria (Kaufman et al., 2013). Parental reports on the KSADS OCD supplement were utilized to make OCD diagnoses in the present study. OCD scores were assigned a "1" if the child met for a current diagnosis of OCD based on the KSADS-PL parent interview; scores were assigned a "0" if the child did not meet current criteria. See Supplemental Table 1 for a list of example clinical interview questions from the KSADS for additional detail.

*K-SADS-PL, Eating Disorders.* ED diagnoses were determined using parent or guardian responses to the computerized KSADS-PL based on DSM-5 criteria (Kaufman et al., 2013). Parental reports on the KSADS ED supplement were utilized to make ED diagnoses in the present study. ED scores were assigned a "1" if the child met for a current diagnosis of any full or subthreshold eating disorder (BN, BED, OSFED variants) based on the KSADS-PL parent

interview; scores were assigned a "0" if the child did not meet current criteria. See Supplemental Table 1 for a list of example clinical interview questions from the KSADS for additional detail.

### **Statistical Analysis**

Statistical analyses will be performed using RStudio version 7.2 (RStudio Team, 2020; Rosseel, 2012). To examine the relationship between variables at baseline (BL), and at two-year follow-up, longitudinal Firth's logistic regression analyses will examine EDs, OCD, and anxiety disorders as both baseline predictors and outcomes at two-year follow up. Two models will be analyzed to assess BN and BED separately, with and without covariates (sex, age, race/ethnicity). A penalized likelihood approach will be used to address potential non-convergence due to rare events of BN and BED diagnoses in the present sample.

### **Results**

The sample of children at baseline was  $N=11,876$ . All Children were between the ages of 9-10 at baseline, with an average age of  $M = 9.9$  ( $SD = 0.62$ ). The sample was racially and ethnically diverse, (48.0% non-white). The sample was 52.1% males and 47.9% females. At baseline, between 0.04% (panic disorder) and 8.8% (specific phobia) of the sample met criteria for any anxiety disorder. All prevalence rates for anxiety disorders at baseline and year 2 follow up are presented in Table 1. 10.4% of the sample met criteria for at least one anxiety disorder. At baseline, 0.94% of children met criteria for other specified obsessive-compulsive disorder and 7.7% of the sample met criteria for obsessive-compulsive disorder. At baseline, 1.3% of children met criteria for any binge-related ED, 1.1% percent of which were BED/OSFED BED and 0.2% were BN/OSFED BN.

Chi-square tests of independence were conducted to examine the associations between anxiety disorders, OCD, and eating disorders cross-sectionally at each time point. A chi-square test of independence was conducted to examine the association between any ED status and OCD status at baseline. The association between the two variables was statistically significant,  $\chi^2(1, N = 11,876) = 81.94, p < .001, \phi = .08$ . The association between any anxiety disorder and OCD at baseline was statistically significant,  $\chi^2(1, N = 11,876) = 434.77, p < .001, \phi = .19$ , as was the association between any ED and any anxiety disorder  $\chi^2(1, N = 11,876) = 98.35, p < .001, \phi = .09$ . At year two follow up, A chi-square test of independence was conducted to examine the association between any ED status and OCD status. The association between the two variables was statistically significant,  $\chi^2(1, N = 10,414) = 90.27, p < .001, \phi = .09$ . The association between any anxiety disorder and OCD at baseline was statistically significant,  $\chi^2(1, N = 10,414) = 342.45, p < .001, \phi = .17$ , as was the association between any ED and any anxiety disorder  $\chi^2(1, N = 10,414) = 60.89, p < .001, \phi = .07$ .

### ***Bulimia Nervosa***

Due to the low base rate of outcomes in the present study cases, Firth's penalized likelihood logistic regression models were used to conduct analyses. Results for all logistic regression models are reported in Table 2. Baseline anxiety disorders, obsessive compulsive disorders, and BN were used in an initial model to predict BN diagnosis at year two follow-up. Baseline OCD significantly predicted BN two years later,  $B = 1.46, SE = 0.53, p = .015, OR = 4.31, 95\% CI [1.36, 11.90]$ . A baseline anxiety disorder was not significantly associated with a BN diagnosis two years later,  $B = 0.08, SE = 0.62, p = .89$ . Baseline BN was a significant covariate,  $B = 3.88, SE = 0.78, p < .001, OR = 48.52, 95\% CI [8.18, 206.92]$ . An additional

model including covariates race/ethnicity, age, and sex was conducted. OCD at baseline remained a significant predictor of BN two years later,  $B = 1.46$ ,  $SE = 0.49$ ,  $p = .014$ ,  $OR = 4.32$ , 95% CI [1.39, 11.91]. Baseline anxiety disorders remained non-significant,  $B = 0.12$ ,  $SE = 0.58$ ,  $p = .85$ , while baseline BN remained a significant covariate,  $B = 3.59$ ,  $SE = 0.79$ ,  $p < .001$ ,  $OR = 36.23$ , 95% CI [6.02, 155.76]. Race/ethnicity, age, and biological sex were all non-significant covariates.

### ***Binge Eating Disorder***

Baseline anxiety disorders, obsessive compulsive disorders, and BED were used in an initial model to predict BED diagnosis at year two follow-up. Baseline OCD significantly predicted BED two years later,  $B = .53$ ,  $SE = 0.22$ ,  $p = .025$ ,  $OR = 1.69$ , 95% CI [1.07, 2.59]. Baseline anxiety disorder significantly predicted a BED diagnosis two years later,  $B = 0.83$ ,  $SE = 0.20$ ,  $p < .001$ ,  $OR = 2.30$ , 95% CI [1.53, 3.37]. Baseline BED was a significant covariate,  $B = 3.88$ ,  $SE = 0.78$ ,  $p < .001$ ,  $OR = 48.52$ , 95% CI [8.18, 206.92]. An additional model including covariates race/ethnicity, age, and sex was conducted. OCD at baseline remained a significant predictor of BED two years later,  $B = 0.53$ ,  $SE = 0.22$ ,  $p = .024$ ,  $OR = 1.70$ , 95% CI [1.07, 2.61]. Baseline anxiety disorders remained a significant predictor,  $B = 0.84$ ,  $SE = 0.20$ ,  $p < .001$ ,  $OR = 2.31$ , 95% CI [1.54, 3.39], while baseline BED remained a significant covariate,  $B = 3.07$ ,  $SE = 0.24$ ,  $p < .001$ ,  $OR = 21.59$ , 95% CI [1.33, 34.38]. Race/ethnicity, age, and biological sex were all non-significant covariates.

### ***Anxiety Disorders***

Baseline BED, BN, and anxiety disorders were used in an initial model to predict an anxiety disorder at year two follow-up. Baseline BN significantly predicted an anxiety disorder two years later,  $B = 1.69$ ,  $SE = 0.55$ ,  $p = .005$ ,  $OR = 5.44$ , 95% CI [1.73, 15.77]. Baseline BED was not significantly associated with later onset of an anxiety disorder,  $B = 0.15$ ,  $SE = 0.28$ ,  $p = .58$ . Baseline anxiety disorder was a significant covariate,  $B = 1.89$ ,  $SE = 0.08$ ,  $p < .001$ ,  $OR = 6.64$ , 95% CI [5.66, 7.79]. An additional model including covariates race/ethnicity, age, and sex was conducted. Baseline BN remained a significant predictor of an anxiety disorder two years later,  $B = 1.71$ ,  $SE = 0.54$ ,  $p = .004$ ,  $OR = 5.51$ , 95% CI [1.78, 15.78]. Baseline BED remained a non-significant predictor,  $B = 0.14$ ,  $SE = 0.28$ ,  $p = .61$ . Baseline anxiety disorder remained a significant covariate,  $B = 1.89$ ,  $SE = 0.08$ ,  $p < .001$ ,  $OR = 6.61$ , 95% CI [5.62, 7.76]. Age was a significant covariate,  $B = -0.018$ ,  $SE = .005$ ,  $p < .001$ ,  $OR = 0.98$ , 95% CI [0.97, 0.99]. With each one month increase in age, an anxiety disorder was at significantly lower odds to occur. Biological sex was additionally a significant covariate,  $B = -0.28$ ,  $SE = 0.08$ ,  $p < .001$ ,  $OR = 0.76$ , 95% CI [0.65, 0.88]. Compared to females, males have significantly lower odds of a developing an anxiety disorder at two year follow up. Race/ethnicity was a non-significant covariate.

### ***Obsessive Compulsive Disorders***

Baseline BED, BN, and obsessive compulsive disorders were used in an initial model to predict an obsessive compulsive disorder at year two follow-up. Baseline BN significantly predicted an obsessive compulsive disorder two years later,  $B = 1.21$ ,  $SE = 0.55$ ,  $p = .035$ ,  $OR = 3.38$ , 95% CI [1.09, 10.02]. Baseline BED also significantly predicted an obsessive compulsive disorder two years later,  $B = 0.80$ ,  $SE = 0.26$ ,  $p = .005$ ,  $OR = 2.22$ , 95% CI [1.29, 3.66]. Baseline

OCD was a significant covariate,  $B = 1.97$ ,  $SE = 0.089$ ,  $p < .001$ ,  $OR = 7.16$ , 95% CI [6.00, 8.52]. An additional model including covariates race/ethnicity, age, and sex was conducted. Baseline BN remained a significant predictor of later OCD,  $B = 1.28$ ,  $SE = 0.55$ ,  $p = .028$ ,  $OR = 3.59$ , 95% CI [1.16, 10.62], as did BED,  $B = 0.79$ ,  $SE = 0.27$ ,  $p = .005$ ,  $OR = 2.20$ , 95% CI [1.28, 3.65]. Baseline OCD was a significant covariate,  $B = 1.97$ ,  $SE = 0.089$ ,  $p < .001$ ,  $OR = 7.19$ , 95% CI [6.02, 8.56]. Race/ethnicity, age, and biological sex were all non-significant covariates.

### *Exploratory Follow-Up Analyses*

Based on patterns revealed in several longitudinal logistic regression models, follow up descriptive analyses were conducted to categorize children with ED diagnoses, similar to one previous study of BED in the ABCD study (Smith et al., 2023). Three categories were established, those who were diagnosed with BN/BED at year 2 follow-up but not baseline ('developers'), those who were diagnosed with BN/BED at baseline but not year 2 follow-up ('remitters'), and lastly, those who were diagnosed with BN/BED at both times ('maintainers').

### *Bulimia Nervosa*

Among the 18 children who met criteria for BN/OSFED BN at year two follow-up, 16 did not meet criteria for BN/OSFED BN at baseline. As such, 88.9% are considered 'developers'. Among 17 children who met criteria for BN at baseline, 15 did not meet criteria for BN at year 2 follow-up. 88.2% are considered 'remitters'. Conversely, among the 17 children who met criteria for BN at baseline, 2 also met criteria at year two follow-up; 11.8% of children are considered 'maintainers'.

### *Binge eating disorder*

Among the 159 children who met criteria for BED at year two follow-up, 126 did not meet criteria for BED at baseline. As such, 79.2% are considered ‘developers’. Among 111 children who met criteria for BED at baseline, 76 did not meet criteria for BED at year 2 follow-up. 68.5% are considered ‘remitters’. Conversely, among the 111 children who met criteria for BED at baseline, 35 also met criteria at year two follow-up; 31.5% of children are considered ‘maintainers’.

### **Discussion**

The present study examined longitudinal associations between eating disorders and other commonly comorbid diagnoses among a large sample of children. While any anxiety disorder did not predict the onset of BN two years later, the opposite association was found, that BN was predictive of a later anxiety disorder. Though an anxiety disorder did predict the onset of BED, BED was not a significant predictor of a later anxiety disorder. Further, OCD was predictive of both BN and BED, while both EDs also predicted the onset of later OCD. All four diagnoses strongly predicted the same diagnoses two years later, highlighting consistency in symptoms over two years. Based on the present findings, there appear to be some specific associations between binge-related ED subtypes, anxiety disorders, and obsessive compulsive disorders over time. However, given the small proportion of binge-related EDs in the present sample, particularly BN, the current findings should be interpreted with caution.

In the present study, BED and BN had opposing longitudinal relationships with anxiety disorders. A baseline anxiety disorder was not associated with BN two years later, though BN did predict a later onset of an anxiety disorder. This is in line with one previous longitudinal study that found BN predicted panic disorder and social anxiety 14 years later, though no anxiety

or mood disorders were predictive of later BN (Buckner et al., 2010). This study replicates the Buckner findings among a younger and larger sample. However, the majority of literature on comorbid EDs and anxiety disorders indicates that anxiety disorders are more likely to precede EDs as opposed to the other way around (Silberg & Bulik, 2005; Feldman & Meyer, 2010). This is more in line with the present BED findings, such that an anxiety disorder was predictive on BED onset two years later, and not vice versa. Some other temporal associations have also linked anxiety and stress to the development of loss of control and emotional eating among adolescents (Lim et al., 2021). It is possible that there could be a distinct pattern of symptom development which differs between BN and BED that is detectable beginning in childhood that was revealed in the present analysis. However, the current longitudinal associations should be interpreted with caution due to the small number of children with binge-related EDs in the sample.

Obsessive-compulsive disorders at baseline were predictive of both BN and BED two years later among the present sample of children. OCD is consistently associated with anorexia nervosa (AN) in past research (Micali et al., 2011; Yilmaz et al., 2020; Sharma et al., 2021). However, one recent systematic analysis found that OCD may be more prevalent among children with binge/purge type EDs compared to restrictive EDs, though this difference plateaus in adulthood (Di Luzio et al., 2024). There is less literature on the association between BED and OCD, however, symptoms such as hoarding and binge eating may have a shared etiology (Vanzhula et al., 2021). While there may be distinct underlying relationships between various psychiatric symptomatology, the broad association between OCD and disordered eating is also important to consider. Maladaptive eating behaviors exist on a continuum, as do anxiety and obsessive compulsive disorders (Comer et al., 2004). As such, research should use “lumping”,

“splitting”, and transdiagnostic approaches (Forbush et al., 2024) to examine populations with an array of disordered eating behaviors, in addition to specific ED subtypes.

Exploratory analyses that assessed the overlap in ED diagnoses between baseline and two year follow-up timepoints showed that among children who met criteria for BN at baseline, 11.8% still met criteria two years later. Among children with BED at baseline, 31.5% met criteria two years later. These high rates of overlap potentially highlight several notable issues: The treatment resistant nature of EDs, susceptibility of relapse, and a lack of appropriate detection measures, particularly in BED. While there are several evidence-based treatment for BED, Cognitive Behavioral Therapy (CBT) for BED has the most research support (McElroy et al., 2015). CBT for BED is supported among treatment seeking adults (da Luz et al., 2021), individuals with obesity and co-occurring BED (Grilo et al., 2011), and in adolescents (Hilbert et al., 2020). While CBT may be helpful in reducing symptoms among adolescents, this treatment assumes the ability for a patient to self-regulate their drive to engage in loss of control eating, and often keep track of their eating behaviors and associated cognitions. Without significant parental involvement and support, in addition to access to appropriate food on a consistent basis, this program of treatment has many potential barriers to efficacy and likely plays a role in high relapse rates (Linardon et al., 2017). Additional treatment research is needed to better address current weaknesses of CBT for binge eating among youth. For example, one recent line of study focused on addressing underlying executive dysfunction among people with BED and found improvements in executive function and reductions in binge eating (Eichen et al., 2023).

The present study has several limitations. Parent-report measures were used for assessment of each diagnostic category: OCD, anxiety disorders, and EDs. Parental guardians are

often considered reliable reporters of eating disorder symptoms among children (Barch et al., 2018). However, binge eating and purging behavior are often concealed due to associated negative emotions, (i.e., shame and embarrassment) (Gupta et al., 2008). Additionally, related ED cognitions such as body image disturbance, and fear surrounding eating may be more difficult to parents and guardians to detect among their children. Similarly, mental compulsions and other cognitive symptoms of OCD and anxiety may not be readily noticed by caretakers. This could lead to an underrepresentation of diagnoses in the sample. Additionally, the present study used a binary ‘any anxiety disorder’ variable to capture six different anxiety disorders assessed in the ABCD study. Similar to the results found in Bucker et al., (2010), there could be associations between ED subtypes and specific anxiety disorders that was not captured in the present analyses. More research should assess particular ED subtypes as well as specific anxiety disorders to better understand these relationships, in addition to utilize scales that measure symptoms on a spectrum as opposed to strict diagnostic criteria. The current research study also had several strengths. These results expand on a large body of literature that has examined comorbidity in eating disorders, and it novel in that it is the first to assess co-occurring disorders longitudinally among a diverse sample of children, and to assess BED and BN separately.

In sum, the current study revealed unique longitudinal associations among a large sample of children. While OCD predicted the later onset of both BED and BN, anxiety had differential associations with these EDs. Additionally, these findings may indicate that anxiety disorders and OCD have distinctive relationships with BED and BN onset. Future research should replicate these findings among diverse groups, given the small proportion of children with BN in the sample may not be representative of early onset BN. Notably, exploratory analyses show high rates of BED maintenance/relapse over the course of two years. Considering that close to one

third of children with BED at baseline also met criteria two years later in the current sample, the need for improved screening, access to treatment, and addressing barriers to treatment is highly necessary. Research should focus on the development and safe dissemination of novel therapies, for example, glucagon-like peptide-1 (GLP-1) agonists as treatment for BED (Richards et al., 2023; Auon et al., 2024) or adapting temperament based ED treatment (Knatz Peck et al., 2021) for binge-related concerns.

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**Table 1***Prevalence of anxiety, eating, and obsessive compulsive disorders*

DSM-5 Diagnosis	Baseline Prevalence	Year 2 Follow Up Prevalence
<b>Anxiety Disorders</b>		
Social Anxiety Disorder	0.94%	0.99%
Generalized Anxiety Disorder	1.1%	1.1%
Other Specified Anxiety Disorder (Any)	0.63%	0.69%
Separation Anxiety Disorder	0.41%	0.25%
Specific Phobia	8.8%	6.1%
Panic Disorder	0.04%	0.08%
<b>Obsessive Compulsive Disorders</b>		
Obsessive-Compulsive Disorder	7.7%	6.2%
Other Specified Obsessive-Compulsive and Related Disorder	0.94%	0.88%
<b>Eating Disorders</b>		
Binge Eating Disorder	0.63%	1.0%
Bulimia Nervosa	0.05%	0.09%
OSFED BED	0.51%	0.54%
OSFED BN	0.13%	0.09%

*Note.* BED= binge eating disorder; BN= bulimia nervosa; DSM= Diagnostic and Statistical Manual of Mental Disorders; OSFED= Other specified feeding or eating disorder.

**Table 2***Longitudinal Logistic Regression Results*

Outcome at Year Two Follow Up	Predictor	$\beta$	<i>SE</i>	<i>p</i>	Odds Ratio	95% CI
BN						
	BL AD	0.08	0.62	.89	1.09	0.26, 3.45
	BL OCD	1.46	0.53	.015*	4.31	1.36, 11.90
	BL BN	3.88	0.78	<.001**	48.52	8.18, 206.92
BED						
	BL AD	0.83	0.20	<.001**	2.30	1.53, 3.37
	BL OCD	0.53	0.22	.025*	1.69	1.07, 2.59
	BL BED	3.13	0.23	<.001**	22.86	14.14, 36.17
Anxiety disorders						
	BL BN	1.69	0.55	.005*	5.44	1.73, 15.77
	BL BED	0.15	0.28	.58	1.17	0.66, 1.98
	BL AD	1.89	0.08	<.001**	6.64	5.66, 7.79
OCD						
	BL BN	1.21	0.55	.035*	3.38	1.09, 10.02
	BL BED	0.80	0.26	.005*	2.22	1.29, 3.66
	BL OCD	1.97	0.09	<.001**	7.16	6.00, 8.52

*Note.* BL=baseline; BN = bulimia nervosa; BED=binge eating disorder; AD = Anxiety Disorder; OCD = Obsessive Compulsive Disorder; SE= standard error; CI= confidence interval. \*  $p<.05$ , \*\*  $p<.001$ .

### **Paper 3**

#### **Temporal Associations Between Non-Suicidal Self-Injury and Binge/Purge Eating Pathology in Youth**

Non-suicidal self-injury (NSSI) can be defined as continued engagement in harm to one's own physical body, without the intent to end one's life. This may commonly present as cutting, burning, scratching oneself, or purposely hitting one's body against objects, which can cause direct injury to the skin or bones (Claes & Muehlenkamp, 2013). NSSI is often conceptualized as an unhealthy and potentially dangerous coping mechanism when faced with emotional dysregulation (Cawood & Hoprich, 2011). NSSI is prevalent among adolescents. Globally, 22% of adolescents report engagement in at least one prior instance of NSSI (Xiao et al., 2022). Adolescence is a vulnerable period of development and a common time for the onset of NSSI to arise, typically close to the onset of puberty (Gillies et al., 2018). [Report prevalence among adults if available.] The prevalence of NSSI has increased among both men and women, and particularly among younger samples, in the past decade (Min et al., 2023; Wester et al., 2018). NSSI causes physical harm to the body, sometimes to the point of necessary medical attention (stitches, infections to wound sites, blood loss) (Wilkinson, 2013).

NSSI has a long-established relationship with Borderline Personality Disorder (BPD), with high rates of overlap in community and clinical samples (Cerutti et al., 2011; Goodman et al., 2017). One study found that among adolescents with BPD traits in a hospital setting, 95% endorsed NSSI (Goodman et al., 2017). NSSI is associated with interpersonal difficulties and high emotional volatility (Kandsperger et al., 2022). These emotional and interpersonal difficulties may stem from the presence of underlying BPD traits, while NSSI engagement serves as a function to regulate strong negative emotions and cope with interpersonal struggles (Mckenzie & Gross, 2014).

### ***Suicidality***

Although the purpose of NSSI is distinctly different from suicidal actions and intentions, there is a significant relationship between NSSI and suicidal ideation, suicide attempts, and death by suicide. In parallel with increasing rates of NSSI, emergency department visits for suicidality have also recently increased among adolescents, particularly females (Kim et al., 2023). Among fifteen-year-olds, death by suicide almost doubled between the years 2015 and 2020 (Marcote & Hansen, 2024). Adolescents who engage in NSSI are at markedly higher risk for suicide, with studies indicating that 10% to 37% report at least one prior suicide attempt (Hilt et al., 2008; Jacobson et al., 2008).

### ***Co-occurring Eating Pathology***

NSSI and suicidality commonly co-occur with other psychiatric disorders, including eating disorders (EDs). Among a sample of adolescent inpatients with EDs, 22% reported lifetime NSSI, which was associated with higher rates of suicidal ideation (Arnold et al., 2022). In the same study, there were significant differences in rates of NSSI by ED subtype. Patients who had a diagnosis of bulimia nervosa (BN) or anorexia nervosa (AN) binge/purge subtype more likely to endorse NSSI than patients with anorexia nervosa restricting subtype. Other studies and systematic reviews have established similar findings, with higher rates of NSSI among individuals who have a binge/purge presentation than among those with AN restricting subtype (Dzomback et al., 2020; Meier et al., 2024). A recent meta-analysis of 79 studies concluded that NSSI rates among individuals with any ED was 35%, with significant differences across some ED subtypes (Kirkpatrick et al., 2024). NSSI rates among AN-BP (42%) and BN (37%) were significantly higher than AN-R (23%).

The co-occurrence of binge eating disorder (BED) and NSSI/suicidality is less studied; those with BED had lower rates of NSSI than those with AN-BP and BN at 21% in the above-mentioned meta-analysis (Kirkpatrick et al., 2024). Among one survey of US households, adults with BED were twice as likely to endorse a history of suicide than those who did not have BED (Brown et al., 2018). The strong association between NSSI and suicidality call for further examination of both within the context of EDs. Previous literature posits that NSSI and suicidality may lie along the same continuum, given their consistently large overlap in the vast majority of previous findings (Nock et al., 2006). Increased suicidality risk among individuals who engage in purging behaviors is consistent across longitudinal findings, with one study showing that purging significantly predicted the presence and intensity of later suicidal ideation, whereas restrictive eating and binge eating did not have a significant relationship with these outcomes (Joiner et al., 2022). Further longitudinal studies should aim to replicate these previous studies to gain a better understanding of temporality of NSSI and binge/purge symptomatology.

### *Experiential Avoidance Model*

An underlying common factor between binge eating, purging, and NSSI is impulsivity. Impulsivity is a multidimensional construct encompassing a range of behaviors and affect, such as an inability to inhibit responses, and preference for immediate over delayed rewards (Whiteside et al., 2005). Impulsivity is significantly related to suicidal attempts, and although impulsivity may seem less conceptually related to suicidal ideation, it is associated with specific facets of impulsivity such as positive and negative urgency (Bruno et al., 2023). Impulsivity is consistently associated with NSSI among adolescents (Lockwood et al., 2017) and adults (Hamza et al., 2015) in both treatment-seeking and community samples. Further, impulsivity is a cardinal symptom of BN (Wu et al., 2013) and BED (Boswell & Grilo, 2020).

A theoretical model relevant to the co-occurrence of NSSI, binge eating, and purging is the experiential avoidance model, which posits that these harmful behaviors are maintained as a function of negative reinforcement via escape from undesirable emotional experiences (Chapman et al., 2006). These harmful coping strategies are typically acted upon during times of increased distress, when critical thinking skills are compromised and impulsive behaviors are more likely to occur. In sum, impulsivity is a consistent marker associated with a range of maladaptive behaviors, particularly among individuals with severe eating pathology and suicidality. Further, prior research has supported the role of behavioral and emotional avoidance as a risk and maintenance factor of EDs (Christian & Levinson, 2022; Melles & Jansen, 2023) and other commonly comorbid disorders, such as anxiety (Hofmann & Hay, 2018). In the context of the experiential avoidance model, impulsivity and emotional avoidance may be notable underlying factors that help to explain why NSSI and binge/purge behaviors co-occur.

### ***Previous Longitudinal Findings***

Despite consistent findings on the co-occurrence of NSSI, suicidality, and binge/purge behaviors, temporality of the manifestation of these behaviors is less studied. NSSI commonly develops in adolescence, with younger adolescents typically reporting increased NSSI engagement over time (De Luca et al., 2023). Similarly, adolescence is a common period for eating disorder onset (Volpe et al., 2016). Only two studies to date have assessed NSSI and binge/purge behaviors longitudinally. One study among college aged women found that NSSI predicted purging behavior 9 months later. The converse was also found, whereby purging behavior predicted later NSSI (Riley et al., 2015). Binge eating episodes were not predictive of either purging or NSSI, highlighting the possible unique underlying function that both NSSI and purge behavior may serve. This study is similar to the previously mentioned longitudinal study

by Joiner and colleagues, which found that purging behavior predicted subsequent suicidal ideation at [x timeframe] later (2022). One cohort study of 14 year olds found that recurrent NSSI predicted the onset of an ED by age 17, and that increases in ED symptoms were followed by an increase in NSSI 3 months later (Kiekens et al., 2019).

The common thread among these past longitudinal studies may be best understood within the context of the experiential avoidance model; such that individuals with emotion regulation difficulties may use a range of harmful behaviors (purging, self-harm) to avoid strong negative emotions. Past literature indicates that not all expressions of ED psychopathology may be appropriately conceptualized in the context of experiential avoidance, for instance, previous studies often do not find a significant association between binge eating and NSSI/suicidality (Riley et al., 2015; Joiner et al., 2022). This may underscore the possibility of unique and separate functions of binge eating behavior and purging behavior. For example, one prior study found significantly higher stress reactivity and negative emotionality in BN compared to both BED and healthy controls (Peterson et al., 2010). These results call attention to a psychological profile specific to individuals who engage in purging, and may also highlight this group's increased risk for engagement in NSSI.

Temporality of binge/purge eating pathology and NSSI/suicidality is important to better understand symptom trajectories and underlying psychopathology. Gaining knowledge of these mechanisms can help to further develop theories and models of psychopathology and the development of ED prevention and treatment efforts. If there is indeed a unique longitudinal association between purging and NSSI and not between binge eating and NSSI in a variety of samples, this could inform the experiential avoidance model as it pertains to eating disorders, particularly those with BN. Although the only two previous longitudinal studies of NSSI and

binge/purge behavior are relatively consistent in their findings that ED symptoms and NSSI have a bidirectional relationship (Riley et al., 2015; Kiekens et al., 2019), further research is necessary to confirm these results among other at-risk samples, such as children and adolescents.

### *Present Study*

Based on current gaps in the literature, the current research aims to focus on NSSI and suicidality as a risk factor of later binge-related ED diagnoses, and conversely, binge-related ED diagnoses as a risk factor for later NSSI and suicidality among a large, racially and ethnically diverse sample of children. The focal research questions are as follows: Does NSSI at 9-10 years old increase the likelihood of a BN or BED diagnosis at 11-12 years old? And, alternatively, does BN or BED at 9-10 years old increase the likelihood of NSSI endorsement at 11-12 years old? Further, suicidality will be probed as an outcome and indicator of binge eating pathology: Do suicidal ideations or actions among 9-10 year olds increase the likelihood of a BN or BED diagnosis at 11-12 years old? Does BN or BED at 9-10 years old increase the likelihood of suicidal ideation and behaviors at 11-12 year old? The present study will utilize data from the Adolescent Brain and Cognitive Development study across two timepoints, from baseline at 9-10 years old to two-year follow-up at 11-12 years old. According to past findings, directional hypotheses are as follows:

- 2) NSSI endorsement at Time 1 will be significantly associated with increased odds of a BN diagnosis at follow-up
- 3) Suicidality at Time 1 will be significantly associated with increased odds of a BN diagnosis at follow-up

- 4) A diagnosis of BN at Time 1 will be significantly associated with the likelihood of NSSI engagement at follow-up
- 5) A diagnosis of BN at Time 1 will be significantly associated with the likelihood of suicidality at follow-up
- 6) A diagnosis of BED at Time 1 will not be significantly associated with the likelihood of NSSI engagement at follow-up
- 7) NSSI endorsement at Time 1 will not be significantly associated with a BED diagnosis at follow-up
- 8) No directional hypotheses will be made regarding the association between BED and suicidality due to a dearth of prior literature on this relationship among children and adolescents.

### **Method**

This study will employ data from the Adolescent Brain and Cognitive Development (ABCD) study (Volkow et al., 2018), initially collected in 2016-2017. The ABCD study is a large longitudinal study in the United States, aiming to investigate the interaction between various childhood experiences (e.g., physical exercise, substance use) and their influence on health outcomes (Luciana et al., 2018). The data that will be used in the current study is from the initial baseline assessment and follow-up time points over the following two years (2017-2020), consisting of a U.S. recruited sample of 11,875 children aged 9-10 years (at baseline), and their caregivers. Participants were recruited through selected schools located near the 21 data collection sites across the United States. Children in the study are racially and ethnically diverse and generally match the U.S. population at large; 51.5% of the baseline sample are boys. Parents of children included in the study have a median family income of \$75,000 to \$99,999, and the

median level of education is completion of a bachelor's degree. Participants in the ABCD cohort will be followed for 10 years, with annual on-site assessments.

## Measures

*Eating Disorder Diagnoses.* Parental responses to the computerized Kiddie Schedule for Affective Disorders and Schizophrenia based on DSM-5 criteria (KSADS-5) were used to confirm current ED diagnoses at each timepoint. The KSADS is a widely used measure of psychopathology with a history of strong psychometric properties (Kaufman et al., 1997). This assessment was adapted from its original structure as a clinician-based interview for computerized administration for the ABCD study. In the current study, parent reports will be used to evaluate ED symptoms, as parents are significant reporters of ED behaviors among children (Barch et al., 2018). Based upon a range of semi-structured interview questions regarding eating behaviors, body image, and associated cognitions and emotions, trained assessors make binary “Yes/No” choices regarding several diagnostic options: BED, BN, OSFED variants (OSFED BN and OSFED BED), BN in partial remission, BED in partial remission. Please see Supplemental Table 1 for examples of KSADS interviewer questions for the Eating Disorders and Substance Related Disorders Supplement.

*NSSI, Suicidal Ideation, and Suicidal Behaviors.* Self-injurious behaviors and suicidal thoughts and actions were assessed using the parent version of the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS-5). This assessment was adapted from its original structure as a clinician-based interview for computerized administration for the ABCD study. Nine items from the suicide module of the KSADS-5 were used for these analyses: one item that assessed non-suicidal self-injurious behavior, and eight items that assessed suicidal ideation and

behavior of varying severity (i.e., passive suicidal ideation; active suicidal ideation with intent; interrupted/aborted attempt). A positive indication on any of the eight suicidality focused questions was coded “Yes” for current suicidality. All items were assessed for current endorsement via parent report and dichotomously coded “Yes/No.” Please see Supplemental Table 1 for examples of KSADS interviewer questions from the Depressive and Bipolar Related Disorders Supplement.

### **Statistical Analyses**

Separate sets of multinomial logistic regression analyses will be performed with each measure of NSSI and suicidality at baseline as predictors of outcome measures at T2: (BN; BED). In addition, each ED diagnosis (BN; BED) at baseline will be used to predict outcome measures at T2: (NSSI, suicidality). In further sensitivity analyses, dependent variables at T2 will be included as covariates at T1 to determine prospective associations. In addition, sensitivity analyses will examine interactions of biological sex, and race/ethnicity. Further, given the evidence to support the notable negative impact of the COVID-19 pandemic on child and adolescent mental health (Panchal et al., 2023), each variable will be assessed for significant differences by timepoint. Given that follow up data was collected after the start of the COVID-19 pandemic, this may be a contributing factor in differences in rates of eating pathology and/or NSSI and suicidality at follow-up compared to baseline. Children with missing data on the relevant KSADS parent-report items will be excluded from further analyses.

### **Results**

The sample of children at baseline was  $N=11,876$ . All children were between the ages of 9-10 at baseline, with an average age of 9.9 ( $SD=0.62$ ). The sample was racially and ethnically

diverse, and was intended to mirror the US population (53.8% White). The sample was 52.1% males and 47.9% females. At baseline, 1.58% of children currently engaged in self-harm behaviors, per parent report. Suicidality was measured using nine items that assess a range of severity of suicidal risk, with endorsement of any of the nine items indicating current suicidality. Among the baseline sample, 1.44% of children had current suicidality, ranging from 0.54% having current passive suicidal thoughts to 0.06% with a parent endorsed recent suicidal attempt. At baseline, 1.3% of children met criteria for any binge-related ED, 1.1% percent of which were BED/OSFED BED and 0.2% were BN/OSFED BN. Sample characteristics are reported in Table 1.

Fisher's exact tests were conducted to examine the associations between self-harm behavior, suicidality, and eating disorders cross-sectionally at each time point. At baseline, the association between self-harm and any ED status was statistically significant,  $p < .001$ . Children who engaged in self-harm had 4.09 times greater odds of an eating disorder diagnosis (OR = 4.09, 95% CI [2.05, 8.16]). The association between suicidality and any ED status was statistically significant,  $p = .024$ . Children who had any suicidality had 2.84 times greater odds of an eating disorder diagnosis (OR = 2.84, 95% CI [1.24, 6.52]). Lastly, the association between self-harm and suicidality was statistically significant,  $p < .001$ . Children who engaged in self-harm had a 15.45 times greater likelihood of also endorsing suicidality (OR = 15.45, 95% CI [10.05, 23.77]). At follow-up two years after baseline, any ED, self-harm, and suicidality were all significantly associated with one another at the  $p < .001$  level. Children who engaged in self-harm had 4.73 greater odds of an ED diagnosis (OR = 4.73, 95% CI [2.73, 8.18]). Children who had suicidality had 6.36 greater odds of an ED diagnosis (OR = 6.36, 95% CI [3.66, 11.06]).

Children who engaged in self-harm had 24.49 greater odds of suicidality (OR = 24.49, 95% CI [16.82, 35.66]).

### ***Bulimia Nervosa***

Due to the low base rate of outcomes in the present study cases, Firth's penalized likelihood logistic regression models were used to conduct analyses. Results for all logistic regression models are reported in Table 2. Baseline self-harm significantly predicted BN two years later,  $B = 1.89$ ,  $SE = 0.74$ ,  $p = .039$ , OR = 6.62, 95% CI [1.11, 25.51]. Baseline suicidality was not significantly associated with a BN diagnosis two years later,  $B = 1.36$ ,  $SE = 0.91$ ,  $p = .21$ . Baseline BN was a significant covariate,  $B = 4.50$ ,  $SE = 0.74$ ,  $p < .001$ , OR = 84.35, 95% CI [15.05, 320.69]. An additional model including covariates race/ethnicity, age, and sex was conducted. Self-harm at baseline remained a marginally significant predictor of BN two years later,  $B = 1.80$ ,  $SE = 0.71$ ,  $p = .052$ , OR = 6.06, 95% CI [0.98, 24.23]. Baseline suicidality remained non-significant,  $B = 1.30$ ,  $SE = 0.86$ ,  $p = .24$ , while baseline BN significantly predicted BN two years later,  $B = 4.16$ ,  $SE = 0.75$ ,  $p < .001$ , OR = 64.05, CI [10.74, 259.76]. Race/ethnicity, age, and biological sex were all non-significant covariates.

### ***Binge Eating Disorder***

Baseline suicidality significantly predicted BED two years later,  $B = 1.64$ ,  $SE = 0.34$ ,  $p < .001$ , OR = 5.17, 95% CI [2.54, 9.72]. Baseline self-harm was not significantly associated with a BED diagnosis two years later,  $B = 0.76$ ,  $SE = 0.41$ ,  $p = .08$ . Baseline BED was a significant covariate  $B = 3.39$ ,  $SE = 0.23$ ,  $p < .001$ , OR = 29.80, 95% CI [18.61, 46.79]. An additional model including covariates race/ethnicity, age, and sex was conducted. Suicidality at baseline

remained a significant predictor of BED two years later,  $B = 1.68$ ,  $SE = 0.33$ ,  $p < .001$ ,  $OR = 5.36$ , 95% CI [2.63, 10.07]. Baseline self-harm remained non-significant,  $B = 0.77$ ,  $SE = 0.41$ ,  $p = .08$ , while baseline BED significantly predicted BED two years later,  $B = 3.32$ ,  $SE = 0.24$ ,  $p < .001$ ,  $OR = 27.87$ , CI [17.31, 44.02]. Age and biological sex were all non-significant covariates, while Hispanic ethnicity was significantly associated with BED at follow-up,  $B = 1.99$ ,  $SE = 1.41$ ,  $p = 0.46$ ,  $OR = 7.29$ , 95% CI [1.02, 925.13].

### ***Self-harm***

Baseline BED significantly predicted self-harm two years later,  $B = 1.48$ ,  $SE = 0.35$ ,  $p < .001$ ,  $OR = 4.39$ , 95% CI [2.06, 8.36], as did baseline BN,  $B = 1.97$ ,  $SE = 0.71$ ,  $p = .02$ ,  $OR = 7.18$ , 95% CI [1.36, 24.33]. Baseline self-harm endorsement was a significant covariate,  $B = 1.78$ ,  $SE = 0.27$ ,  $p < .001$ ,  $OR = 5.98$ , CI [3.43, 9.83]. An additional model including covariates race/ethnicity, age, and sex was conducted. Baseline BED remained a significant predictor of self-harm,  $B = 1.48$ ,  $SE = 0.35$ ,  $p < .001$ ,  $OR = 4.41$ , 95% CI [2.06, 8.42], as did baseline BN,  $B = 1.99$ ,  $SE = 0.70$ ,  $p = .02$ ,  $OR = 7.31$ , 95% CI [1.40, 24.84]. Race/ethnicity and age were non-significant covariates. Baseline self-harm remained a significant covariate,  $B = 1.80$ ,  $SE = 0.27$ ,  $p < .001$ ,  $OR = 6.05$ , 95% CI [3.47, 9.94] and sex emerged as a significant predictor of self-harm,  $B = -0.34$ ,  $SE = 0.14$ ,  $p = .017$ ,  $OR = 0.71$ , 95% CI [0.54, 0.94], with females significantly more at risk than males.

### ***Suicidality***

Baseline BED ( $B = 0.82$ ,  $SE = 0.50$ ,  $p = 0.14$ ) and baseline BN ( $B = 0.69$ ,  $SE = 1.44$ ,  $p = 0.66$ ) diagnoses did not significantly predict suicidality two years later. Baseline suicidality was

a significant covariate,  $B = 1.99$ ,  $SE = 0.30$ ,  $p < .001$ ,  $OR = 7.29$ ,  $CI [3.97, 12.47]$ . An additional model including covariates race/ethnicity, age, and sex was conducted. Baseline BED and BN remained non-significant, while baseline suicidality remained a significant predictor of the likelihood of suicidality two years later,  $B = 2.05$ ,  $SE = 0.29$ ,  $p < .001$ ,  $OR = 7.72$ ,  $95\% CI [4.19, 13.28]$ . Neither race/ethnicity nor age was a significant covariate; however, sex significantly predicted the likelihood of suicidality two years later,  $B = -0.51$ ,  $SE = 0.16$ ,  $p = .001$ ,  $OR = 0.60$ ,  $95\% CI [0.43, 0.83]$ , with females being significantly more at risk than males.

### Discussion

The present study examined longitudinal associations between self-harm, suicidality, and eating disorders in children. Self-harm predicted BN two-years later, while suicidality did not. In contrast, suicidality predicted BED two years later, while self-harm did not. Both BN and BED were associated with later onset of self-harm, but neither were predictive of suicidality. All four variables of interest were strong predictors of themselves two years later, suggesting continuity over time. Notably, the findings highlight unique risks associated with the onset of binge-type eating disorders. Given the small proportion of self-harm endorsement, suicidality, and binge-related EDs in the present sample (particularly BN), the current findings should be interpreted with caution.

Self-harm among 9-10 year olds predicted a BN diagnosis between ages 11-12, controlling for the effects of covariates. The longitudinal association detected in the present study of preadolescents is consistent with previous literature among college students (Riley et al., 2015) and older adolescents (Kiekens et al., 2019). Notably, the effect of suicidality on later BN was non-significant. This may indicate that self-harm and purging serve a similar underlying

function. According to the experiential avoidance model, both self-harm behavior and purging behavior can be used as an escape or distraction from unwanted emotional states (Chapman et al., 2006). These behaviors are often reinforced by a sense of relief, increasing the likelihood of repeated use (Gratz et al., 2018; Bekiaris & Koletski, 2019). Individuals seeking to distract themselves from negative emotions and who are then subsequently reinforced by a sense of relief or comfort may learn to use several different behavioral strategies, in part explaining the relationship between self-harm and purging behavior, even from a young age. There are few studies that specifically test this model longitudinally, which could have important clinical implications. Self-harm and EDs typically develop during adolescence, and the present study shows that their temporal association can be detected even earlier, in middle childhood and pre-adolescence. Underlying traits and cognitive/biological differences should be further probed to better understand risk factors associated with these harmful behaviors. This could potentially inform the experiential avoidance model by incorporating early risk factors that lead to increased emotion dysregulation and a better understanding of the temporality of a range of avoidance behaviors.

While self-harm was not associated with later BED, suicidality at baseline predicted increased likelihood of BED two years later, controlling for baseline BED. This finding expands upon two previous systematic analysis found that BED was associated with a marked increase in suicidal behaviors and suicidal ideation among adults and adolescents (Conti et al. 2017; Smith et al., 2018), and suggests that suicidality may precede BED in younger populations. Among adolescent girls, suicidal ideation has been linked to binge eating severity (Ackard et al., 2011), higher body mass index (Ackard et al., 2011; Iwatate et al., 2023) and perceived overweight (Daly et al., 2020).

These associations may be partly explained by weight stigma, which is frequently experienced by children with overweight or obesity (Hagg et al., 2021). This stigma may contribute to the higher rates of depression and suicidality among children with overweight or obesity (Puhl & Lessard, 2020), who are also more likely to meet criteria for BED than their non-overweight counterparts (Decaluwe & Braet, 2003). While weight stigma has been acknowledged as an important contributor to health in recent literature, continued outreach, education, and interventions are needed to ensure it is adequately addressed among children and adolescents (i.e., school-based interventions).

Although suicidality predicted the later onset of BED, the reverse was not observed- BED at baseline did not predict later suicidality. This temporal pattern may indicate that binge eating serves as a coping strategy following the onset of suicidality and associated depressive symptoms. Indeed, prior work has found that both clinical and non-clinical samples of adolescents who use avoidant coping strategies are also more likely to binge eat (Sierra-Bagrie et al., 2012), and emotional eating is associated with depressive symptoms among children and adolescents both cross-sectionally and longitudinally (Muha et al., 2024). Early intervention for children with depression and suicidality can be effective at reducing symptoms, such as within the context of pediatric primary care and mental health integration programs (Schweitzer et al., 2023). Particularly, children with depressive symptoms and associated suicidality may benefit from learning skills and healthy coping strategies to address their mood concerns in efforts to prevent later onset of maladaptive coping behaviors, such as binge eating.

The present study is not without limitations. Parent-report measures were used for assessment of suicidality, self-harm, and eating disorder behaviors. While parental guardians are

typically considered reliable reporters of disordered eating among children (Barch et al., 2018), binge eating and purging behavior are often concealed due to guilt, embarrassment, and concern from others (Gupta et al., 2008). Additionally, cognitive and emotional symptoms of eating disorders such as body image disturbance, and shame or fear surrounding eating may be more difficult to parents to detect. This could lead to unknown false negatives in present sample. Further, NSSI was measured with one item. This one item assessed current parent endorsement of NSSI, though there is a range of severity of NSSI behavior that is missed in addition to a potential lack of knowledge by parental guardians.

Additionally, the present study relied on clinician-administered reports that were not supplemented with anthropometric data. Assessing body mass index percentiles as covariates could have been helpful to understand the impact of overweight/obesity status on EDs and other mental health outcomes such as self-harm and suicidality. Nevertheless, notable strengths of the current study are the utilization of a large and diverse sample of children that mirrors the U.S. population, as well as longitudinal follow-up of key psychiatric outcomes during a critical developmental window.

In summary, the present study revealed distinct longitudinal associations among a large and diverse sample of children. Particularly, self-harm endorsement was predictive of a BN diagnosis two years later, while suicidality was predictive of a BED diagnosis two years later. These findings suggest that while self-harm and suicidality often co-occur, they may differentially predict the onset of specific eating disorders. Future research should continue to parse apart potentially unique associations and better understand the cognitive, personality, and neurobiological mechanisms driving these patterns. The current findings should be used to

inform detection and prevention efforts among young children who experience suicidality and engage in self-harm, as these behaviors may signal elevated risk of the later onset of eating disorders.

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**Table 1***Sample Characteristics at Baseline*

Demographic and Psychological Variables	Range or <i>n</i>	<i>M(SD)</i> or %
Age, range	8.9-11.08	9.9 (0.62)
Race/ethnicity		
White	5602	53.79%
Black	1422	13.65%
Asian	216	2.07%
Other	1088	10.45%
Hispanic	2086	20.03%
Study Measures		
BN	17	0.16%
BED	111	1.08%
Self-harm	159	1.54%
Any suicidality	147	1.41%
Passive suicidal ideation	63	0.54%
Suicidal ideation, non-specific, active	132	1.14%
Suicidal ideation, active, method established	20	0.17%
Suicidal ideation, active, plan established	4	0.03%
Suicidal behavior, preparatory action present	12	0.10%
Aborted suicide attempt	1	0.009%
Suicide attempt	7	0.06%

*Note.* BN = bulimia nervosa; BED=binge eating disorder; M= mean; SD=standard deviation.

**Table 2***Longitudinal Logistic Regression Results*

Outcome at Year Two Follow Up	Predictor	$\beta$	<i>SE</i>	<i>p</i>	Odds Ratio	95% CI
<b>Bulimia nervosa</b>						
	BL self-harm	1.89	0.74	.039*	6.62	1.11, 25.51
	BL suicidality	1.36	0.91	.21	--	--
	BL BN	4.50	0.74	<.001**	84.85	15.05, 320.69
<b>Binge eating disorder</b>						
	BL self-harm	0.76	0.41	.08	--	--
	BL suicidality	1.64	0.34	<.001**	5.17	2.54, 9.72
	BL BED	3.39	0.23	<.001**	29.80	18.61, 46.79
<b>Self-harm</b>						
	BL BN	1.97	0.71	.020*	7.18	1.36, 24.33
	BL BED	1.48	0.35	<.001**	4.39	2.06, 8.36
	BL self-harm	1.78	0.27	<.001**	5.93	3.42, 9.87
<b>Suicidality</b>						
	BL BN	0.69	1.44	.66	--	--
	BL BED	0.82	0.50	.14	--	--
	BL suicidality	1.99	0.30	<.001**	7.33	3.97, 12.42

*Note.* BL=baseline; BN = bulimia nervosa; BED=binge eating disorder; SE= standard error; CI= confidence interval. \*  $p < .05$ , \*\*  $p < .001$ .

## Overall Discussion

Eating Disorders (EDs) are mental disorders characterized by engagement in a range of harmful eating behaviors, such as restrictive eating, binge eating, and compensatory behaviors such as purging and overexercise (APA, 2013). These behaviors often coincide with body image disturbance, fear of gaining weight, and guilt or shame surrounding food. EDs are highly comorbid with other mental disorders, commonly anxiety disorders (Rojo-Moreno et al., 2015; Convertino & Blashill, 2022), and obsessive compulsive disorder (Udo & Grilo, 2019). EDs have some of the highest rates of mortality (Fitcher & Quadflieg, 2016), often via suicide (Huas et al., 2013), in addition to high rates of NSSI endorsement (Cucchi et al., 2016). Notably, co-occurring mental disorders present more often in some ED subtypes than in others, for example, OCD and anorexia (Micali et al., 2011), or NSSI and purge subtype EDs (Cuchi et al., 2016). A central purpose of this dissertation was to examine the implications of comorbid diagnoses on the development of eating disorder onset in early adolescence. Specifically, these papers set out to further assess possible similarities and differences in longitudinal risk factors across ED subtypes and characterize the sample across time.

These research questions were examined across three related studies. Study one examined the prevalence of binge-related eating disorders across three timepoints, and assessed demographic risk factors such as race/ethnicity and biological sex at each of these timepoints. Results indicated that the prevalence of binge-related EDs are relatively stable during preadolescence, and are equally prevalent among boys and girls. Additionally, Black, Hispanic, and mixed race children are at increased risk for a binge-related ED at one or more timepoints. Study two examined the temporal relationships between binge-related EDs, anxiety disorders, and OCD. The results showed a distinct temporal relationship between BED and preceding

anxiety and depression, while there was a bidirectional relationship between OCD and BN. Further, exploratory analyses revealed that roughly one third of children with BED at baseline also met criteria two years later. Lastly, study three examined the longitudinal associations between binge-related EDs, NSSI, and suicidality. Similar to study two, distinct relationships were revealed between BED and BN. Specifically, suicidality predicted the onset of BED and not BN, while NSSI predicted the onset of BN and not BED. The results of these studies provide insight into several issues, including better understanding of risk factors involved in the development of binge-related eating pathology. When considered together, the studies highlight temporal associations that distinguish between BED and BN among a large nationally representative sample of children.

It is important to highlight the limitations of these three studies in discussion of their possible implications. Notably, parental responses on the KSADS were used to determine all diagnoses. While parents are often reliable reporters of psychiatric symptoms among children (Barch, 2018), some internalizing presentations (body image concerns, negative emotions surrounding binge eating, general concern for the future) and secretive behaviors such as purging or developing a suicide plan may not always be apparent to adult guardians. Additionally, despite the large sample of families that have taken part in the ABCD study, the prevalence of binge-related EDs, particularly BN, is low (0.18%). Given the small subsample of children who meet criteria for BN, findings in the present study may not be applicable more broadly. Rates of OCD in the present sample were higher (7.7%) than indicated by other prevalence studies (Mohammadi et al., 2021; Heyman et al., 2001), and it is possible that the rates in the present study are inflated and may not align with true diagnostic criteria.

Results from Study 1 indicate an increased risk of binge-related EDs among Black, Hispanic, and mixed race children. However, more clarity is needed to best understand the complex dynamics that impact racial/ethnic disparities in the current sample. Importantly, small racial/ethnic subsamples (particularly of Asian children) that were underrepresented compared to the general population in the ABCD sample impacted the present findings, and limit generalizability. Another limitation of the three studies was use of a five-option categorical variable to measure race/ethnicity, without consideration of other factors that largely impact racial/ethnic minority groups. There are likely socioeconomic and acculturative influences that interact with race/ethnicity status and increase the likelihood of binge-related EDs. Additional studies should assess disordered eating behaviors across the various locations within the ABCD study and the unique demographic makeup of the locations, and assess possible within group differences.

There are several limitations to the analytic strategies used across the three studies that warrant further discussion. All variables used to indicate diagnoses were binary in nature, preventing the means to look at variability within the constructs of interest. While a Firth's penalized approach adjusted estimation errors of dependent variables across statistical models and allowed for convergence, it failed to address the rare outcomes used as predictor variables. The rare outcomes used as indicator variables were highly skewed, potentially leading to inaccurate results due to a mismatch of the data and statistical assumptions. It is possible that using transformations to address the high skewness of these variables could have altered the current findings and made them more trustworthy. These considerations all suggest that the results and their implications should be interpreted with caution, and that additional research is needed to replicate these findings. Given that the objectives of Study 3 included assessing

reciprocal relationships across time, a cross-lagged model would have been a preferred analytic strategy. A cross-lagged model can control for relationships among variables within timepoints as well as the stability in symptoms over time. Given the high rates of comorbidity at both timepoints and the high rates of BED maintenance over two years, a cross-lagged approach is a preferred strategy to assess self-harm, suicidality, and binge-related EDs among the current sample, and could also provide preliminary evidence for causality rather than temporality alone.

Future studies on EDs in the ABCD sample should consider several alternative approaches instead of longitudinal logistic regression. Use of count variables (such as frequency of binge eating or purging behavior) as outcomes of interest would allow for more variance than simple binary variables, and would likely be less skewed due to their higher prevalence as compared to full threshold EDs. Further analyses at the symptom level rather than the diagnostic level could be useful in identifying patterns that cut across diagnoses, such as avoidance behaviors or ruminative cognitions (Christian & Levinson, 2022; McEvoy et al., 2013). A better understanding of symptom level dynamics over time could help inform transdiagnostic treatment approaches that target broad maintenance factors, as shown in several randomized controlled trials of the Unified Protocol (Kennedy et al., 2019; Carlucci et al., 2021), or in positive affect interventions (Craske et al., 2023).

However, despite these limitations, the results of this dissertation do suggest that there are differences in the development of BN and BED among the present sample. A strength of the current studies is their longitudinal nature, allowing temporal inference and the ability to test associations bi-directionally. Several findings highlight the temporality of binge-related EDs and other co-occurring diagnoses, allowing for an understanding of when these diagnoses commonly develop over time, in reference to one another. This could potentially inform prevention and

detection efforts and allow for a personalized approach to care. For example, self-harm was associated with the later onset of BN and not BED, indicating a need to more closely monitor for the development of purging behavior among children who self-harm. Another strength of the present studies is the large and diverse sample utilized. While a focus on high socioeconomic status, white women impeded the field of eating disorder research for decades (Gordon et al., 2002; Gard & Freeman, 1996), study one had the statistical power to detect differences across five race/ethnicity groups that were also diverse in location, urbanicity, and socioeconomic status. Given that findings indicate an increased risk for binge-related EDs among Black, Hispanic, and mixed children, which is consistent with past ABCD studies (Makowski et al., 2023), further research should seek to better understand causal factors that explain these differences.

Furthermore, the results of this dissertation speak to the need to develop more effective treatment for EDs in children, particularly for non-white subgroups, and those who have BED. There are several possible avenues to address the high lack of treatment response for BED. First, there are several first line treatments for BED that are efficacious. These include cognitive behavioral therapy (CBT) for BED and interpersonal therapy (McElroy et al., 2015). CBT for BED has been adapted for children to account for developmental differences and the importance of parental involvement, which showed significant improvement post-treatment (Hilbert et al., 2020). While current treatments offered may reduce symptomatology, later recurrence of binge eating and relapse is common, with one meta-analysis showing that about 50% of individuals with BED do not respond to psychological treatment (Linardon, 2018). There are some novel treatment approaches that have recently been studied among individuals with other longstanding disorders, such as ketamine therapy and psilocybin assisted treatment. Some proof of concept

and pilot studies have found these to be effective in reducing disordered eating and facilitating weight gain among individuals with anorexia (Ragnhildstveit et al., 2022; Koning & Brietzke, 2024). These therapies still need to be further researched, however, pilot testing should also be considered among individuals with BED. Recent research has also studied GLP-1 agonists as treatment for binge eating. GLP-1s have shown to be effective in reducing binge eating frequency and distressing food-related cognitions (Richards et al., 2023). While GLP-1s have been approved for use among children as young as twelve years old with obesity, there are no studies that have assessed their utility as treatment for BED. Caution is needed as GLP-1s are explored as treatment options for EDs, as they could pose a risk of drug misuse and an increase of weight-centric culture that promotes restrictive eating.

In addition to novel treatment, new prevention efforts are highly needed among children. While detection efforts and early intervention programs have increased in their availability across the country (McGorry & Mei, 2018), the present studies show that a range of psychiatric symptoms can develop into other types of psychopathology later in life, highlighting the relationships within a broad spectrum of psychopathology. Prevention efforts should take a targeted approach, based on prior literature of causal factors and co-occurring problems. For example, one recent call to action regarding NSSI prevention programs focused on preadolescent girls with ADHD and a history of maltreatment, who are at significantly greater risk of NSSI engagement (Beauchaine et al., 2019). Similarly, recent findings from genome wide association studies consistently point to a strong genetic component of anorexia (de Jorge Martinez et al., 2022), highlighting the need for a targeted approach to ED prevention efforts. Further interventions may wish to focus on underlying factors of psychopathology, such as executive function deficits, impulsivity, or clinical perfectionism. These trait differences may be evident in

neurobiological markers, such as in fMRI or endocrinological studies of EDs (Olivo et al., 2019; Eddy et al., 2014). Additional research that is informed by fields of psychology, neuroscience, and bioinformatics will be highly important to develop a better understanding of psychiatric disorders, risk factors, and possible treatment avenues.

In sum, this dissertation identified several risk factors of early onset EDs in a large and diverse sample of children. The causality inferred by the longitudinal analyses help to bolster these findings, though replication is still necessary. The temporal risk factors revealed in two of the present manuscripts identify unique risk factors that differentiate BED and BN onset, which could inform prevention and detection efforts for children. Characterization of the present sample showed that the treatment resistant nature of BED begins early in life, as does increased risk of BN and BED based on race/ethnicity group. However, biological sex did not differentiate the likelihood of an ED at any point in time, indicating that this later sex difference may be related to risk factors that develop during and after puberty. Findings from the current three studies should inform continued research on EDs in children, with the intention to develop improved and more nuanced theories of eating pathology and exploration of more effective treatment possibilities.

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**Supplemental Table 1***KSADS-PL-5 Semi-Structured Interview Example Questions*

Supplement	Example Clinical Interview Questions
Eating Disorders and Substance Related Disorders Supplement: Eating Disorder Module	<ul style="list-style-type: none"> <li>- How much time does your child spend thinking about food and worrying about getting fat?</li> <li>- If your child saw that they had gained a pound or two, do they change their eating habits?</li> <li>- Does your child sometimes make themselves throw up?</li> <li>- Has there ever been a time when your child ate so much they felt sick? How often does it happen?</li> <li>- How did they feel after they binged?</li> </ul>
Anxiety, Obsessive-Compulsive, and Trauma Related Disorders Supplement: Obsessive-Compulsive Disorder Module	<ul style="list-style-type: none"> <li>- Does your child worry about having dirt or germs on their hands?</li> <li>- What about thoughts that something bad might happen, or that they did something terrible, even if they knew it wasn't true?</li> <li>- Has your child ever repeated actions over and over? Did they have to do things in the exact right way, or in a special way?</li> <li>- Have other people ever commented on your child's repetitive habits?</li> </ul>
Anxiety, Obsessive-Compulsive, and Trauma Related Disorders Supplement: Panic Disorder, Social Anxiety Disorder, Separation Anxiety Disorder, General Anxiety Disorder, Specific Phobia, and Agoraphobia Modules	<ul style="list-style-type: none"> <li>- Has there been a time where your child worried about things before they happened?</li> <li>- Is your child very, very, afraid of anything?</li> <li>- Is it difficult for your child to be with other kids, even kids they know?</li> <li>- Was there a time, after the age of 4, that your child followed you everywhere you went?</li> <li>- Has your child ever had a time when, out of the blue, for no reason at all, suddenly felt anxious, nervous, or frightened?</li> <li>- Has your child ever been very afraid of being in a crowded place or going outside in public?</li> </ul>

<p>Depressive and Bipolar Related Disorders Supplement: Suicide Module</p>	<ul style="list-style-type: none"> <li>- Sometimes children who get upset or feel bad wish they were dead or feel they'd be better off dead. Has your child had these thoughts? When?</li> <li>- Sometimes children who get upset or feel bad think about dying or even killing themselves. Has your child had these thoughts? When?</li> <li>- Has your child attempted to kill themselves? When? What did they do? Did they tell anyone in advance?</li> </ul>
<p>Depressive and Bipolar Related Disorders Supplement: Non-suicidal, Self-Injurious Behavior (single item)</p>	<ul style="list-style-type: none"> <li>- Has your child ever tried to hurt themselves?</li> <li>- Have they ever burned themselves with matches/candles?</li> <li>- Or scratched themselves with needles/a knife?</li> <li>- How often do they do this?</li> <li>- Some children do these types of things because they want to kill themselves, and other children do them because it makes them feel better afterwards. Why do they do these things?</li> </ul>