Does Weight Matter?
Prospective Examination of Premorbid Body Weight as a Predictor of Eating Pathology

A Thesis

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by

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Abstract

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Despite years of research on eating disorders, their persisting prevalence suggests limited efficacy of current prevention efforts and establishes the need for the identification of additional risk and maintenance factors. Predominant conceptualizations of eating disorders have placed continued emphasis on psychosocial factors associated with eating pathology. As such, the role of actual body weight has received limited attention, and relevant relationships between these factors and eating disorder development remain elusive. Preliminary evidence has identified elevated premorbid body weights in eating disorder patients, suggesting that these individuals may be biologically predisposed to higher weights and that this predisposition may be a factor relevant to subsequent eating disorder development. The current study sought to examine whether higher premorbid body weights prospectively predict subsequent clinical status in a sample of female eating disorder inpatients. By examining body weight in relation to subsequent symptom severity, the current study sought to establish premorbid body weight as a factor relevant to the development of eating pathology. Results indicated a significant relationship between highest premorbid z-BMI and self-reported Eating Concerns at treatment intake, though this relationship was not significant when accounting for current BMI. There was also a marginally significant interaction between premorbid z-BMI and current weight, such that those with a higher current BMI and a history of relatively lower highest premorbid z-BMI exhibited the greatest eating and weight concerns. These finding

suggest a potential combined influence of current and historical weight in the role of Eating and Weight concerns.

Chapter 1: Introduction

1.1. Traditional Conceptualizations of Eating Disorders

Despite years of research and treatment development, eating disorders remain a fundamental problem that affects a significant number of individuals each year. A review of lifetime prevalence rates of eating disorder subtypes in the U.S. alone suggests anywhere from 0.9%-3.5% of females will develop an eating disorder, including anorexia nervosa (AN, 0.9%) bulimia nervosa (BN, 0.9-1.5%), binge-eating disorder (BED, 3.5%) and other specified feeding or eating disorders (OSFED) (Smink, van Hoeken, & Hoek, 2012). Importantly, these statistics do not include males, adolescents, or individuals in non-U.S. countries, among whom lifetime prevalence rates of eating disorders are also significant. Perhaps more concerning is the heightened mortality rates identified across all eating disorders, particularly AN (Smink et al., 2012). The persisting prevalence of eating disorders underscores the limited efficacy of current prevention efforts, and demonstrates the continuing need to identify etiological factors associated with disorder onset. Indeed, identifying predictors of eating disorder development will not only aid in prevention efforts but may also be integrated into current treatment protocols, which themselves remain in relatively rudimentary stages of development. As such, the mechanisms underlying eating disordered behaviors demand increased attention to assist in the development of more effective preventative efforts and treatment protocols.

Eating disorder symptoms span a range of dysfunctional behaviors which may include abnormal eating patterns, compensatory behaviors, or other weight and eating-related rituals. Though the symptom profile comprising an eating disorder varies based on diagnosis, there exists some overlap and crossover between disorders (Tozzi et al.,

2005; Vervaet, van Heeringen, & Audenaert, 2004). At present, the most widely-accepted model for conceptualization and treatment of eating disorders is the recent enhanced cognitive-behavioral therapy (CBT-E), as introduced by Fairburn and colleagues (Fairburn, Cooper, & Shafran, 2003). The CBT-E model stresses the trans-diagnostic cognitive mechanisms identified in a number of eating disorders, particularly the overvaluation of shape and weight as the core psychopathology of the disorder. While the original CBT model identified this trait in BN, the enhanced model extends this cognitive trait to all diagnostic subtypes, including AN, BED and OSFED. The CBT-E model holds that this overvaluation leads to extreme food restriction, checking-behaviors, and other related rituals driven by preoccupation with shape, weight, or control. These restrictive behaviors often lead to disinhibition of food intake and subsequent binge-eating behaviors, which are characterized by eating an objectively or subjectively large amount of food, combined with a strong feeling of loss of control over food consumption.

Cognitive-behavioral and other predominant models conceptualizing eating disorders have thus far focused primarily on the maintenance of eating disorders. Moreover, current research places continued emphasis on affective, cognitive and social mechanisms underlying eating disorder symptoms. Indeed, an extensive body of research has investigated the relationship between eating disorders and negative affect (Lingswiler, Crowther, & Stephens, 1989; Macht, 2008; Stein et al., 2007)(Lingswiler et al., 1989), inhibitory control (Herman & Polivy, 1988; Wu et al., 2013), interoceptive awareness (Engler, Crowther, Dalton, & Sanftner, 2006; Garner, Olmsted, Polivy, & Garfinkel, 1984) and cognitive flexibility, such as set-shifting (Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007). Other frequently examined constructs include body

dissatisfaction, drive for thinness, and self-esteem (Ramacciotti et al., 2001; Stice, South, & Shaw, 2012; Wade & Tiggemann, 2013; Williams et al., 1993).

While existing research has provided an extensive and critical foundation toward a better understanding of eating disorder development and maintenance, research over the past several decades has largely overlooked the significance of body size and its role in eating disorder development and trajectory. Interestingly, the role of body weight in eating disorder development is not a novel concept; it was initially introduced by Russell (1979), who addressed the potential role of elevated body weight and subsequent weight loss in the development of BN. Despite this, the overwhelming majority of both clinical and research work have continued to focus primarily on psychological constructs, such as body image, while overlooking actual body weight. However, actual body weight arguably influences one's body image, satisfaction, and subsequent behavior. For example, it is likely that an eating disorder patient who fears weight gain toward a previous high weight will experience increased distress at elevated weights and may alter behavior to initiate desired weight loss. Indeed, the absence of body weight as a construct in the literature hinders a more multi-dimensional understanding of the potential relationship between historical body weight and risk for eating disorder development.

1.2. The Role of Body Weight in Eating Disorders: A Review of Current Literature

Given that psychological constructs associated with body weight have been implicated in eating disorders, it becomes necessary to identify and clarify the distinction between the weight-related (e.g., actual body mass and change in body mass) and psychological (e.g., perception and negative evaluation of body mass) components of

body weight and their respective implications. Thus far, associations between body weight and eating disorders have been examined in terms of weight history (e.g., premorbid body weights) and weight after disorder development (e.g., current body weight at time an individual is assessed, and weight suppression, or the difference between highest historical weight and current body weight). A review of preliminary findings related to each construct is presented below.

1.2.1. Weight History

Evidence of a Predisposition to Higher Weights

Current literature suggests eating disorder patients may have a susceptibility to elevated weights before disorder development. Indeed, elevated body mass index (BMI) has been identified in both AN and BN patients premorbidly. For example, early research on AN suggests a substantial portion of female patients are premorbidly overweight or obese when compared with matched population mean weight (Crisp, Hsu, Harding, & Hartshorn, 1980; Crisp & Stonehill, 1971). In one sample, 46.2% of patients were premorbidly overweight (Crisp & Stonehill, 1971). A subsequent study of 102 patients reported that patients were on average slightly overweight premorbidly, and that 28% of these patients were premorbidly obese (Crisp et al., 1980). More recent research also provides support for the notion that a substantial proportion of eating disorder patients are premorbidly obese: a survey of eating disorder inpatients found a high prevalence of lifetime obesity, ranging from 4.6-28.8% across eating disorder subgroups (Villarejo et al., 2012). Similarly, another investigation of AN-BP, AN-R and OSFED patients reported premorbid obesity in 20% of patients (Wojciechowska, Garland, & Hergenroeder, 2013). Consistent with this notion, research on males with eating disorders

suggests these patients are more likely to be premorbidly obese, with 45-60% of patients being premorbidly overweight (Carlat, Camargo, & Herzog, 1997; Jasik, Breland, & Buckelew, 2010), further providing support for the notion that eating disorder patients may be inherently susceptible to higher weights.

It should be noted that the origins of elevated premorbid weights remain unclear. The prevalence of premorbid overweight and obesity in these samples could reflect a hereditary predisposition or deviation from normal weight trajectory during development occurring for non-genetic reasons. Current findings suggest both hereditary and developmental influences may be at play. Parental obesity has been associated with offspring eating disorder diagnoses (Fairburn, Cooper, Doll, & Welch, 1999; Garfinkel, Moldofsky, & Garner, 1980; Shaw et al., 2012); given that BMI is a highly heritable trait (Elks et al., 2012), offspring of obese parents may therefore be susceptible to elevated weights. An examination of premorbid weight in the years prior to eating disorder onset identified a pattern of weight gain prior to weight loss (Swenne, 2001). Moreover, the majority of patients (87%) gained weight quickly and at a higher than average rate before reaching their maximum premorbid weight and developing a disorder, suggesting that premorbid deviation from normative growth may also contribute to higher rates of overweight and obesity in these samples.

Taken together, these findings suggest that elevated weights in premorbid states may be relevant to eating disorder development. However, at present, the means by which these elevated premorbid weights may confer risk for eating disorder development and maintenance remain uncertain. Higher weights could contribute to different

symptoms such as increased psychological distress (e.g., higher body dissatisfaction), weight loss behaviors (e.g., more intensive dieting or food restriction), or both.

Premorbid Weight and Clinical Presentation

Research on the relationship between premorbid body weight and symptom development remains nascent, though preliminary research suggests associations between elevated premorbid weights and clinical outcome. Lifetime obesity across all eating disorder subtypes has been associated with increased eating disorder severity (Villarejo et al., 2012). Moreover, high premorbid weights may be relevant to the development of specific eating disordered symptoms, including binge/purge symptoms and restrictive pathologies. For example, initial research found binge-purge subtypes of AN patients (AN-BP) were more likely to report elevated premorbid BMIs than restrictive subtypes of AN (AN-R), suggesting that weights may be particularly elevated among patients with binge-purge symptoms (Garfinkel et al., 1980).

However, more recent research suggests that elevated premorbid weight may be associated with restrictive symptoms as well: an examination of both AN and atypical AN patients (those who exhibited AN symptoms and weight loss but remained in a normal or above average weight range) found both greater symptom severity, including Restraint, Eating, Shape and Weight concerns, and a greater rate of elevated premorbid BMIs among the atypical AN group (71%) than among the AN group (12%) (Sawyer, Whitelaw, Le Grange, Yeo, & Hughes, 2016). Because the atypical AN group was also higher in current BMI than the AN group, it is difficult to discern whether differences in symptom severity were due to current or premorbid weights. In another study examining male eating disorder patients, a history of overweight or obesity was associated

specifically with reduced dietary intake and increased compensatory behaviors (Jasik et al., 2010). Elevated weights may therefore be relevant to both symptom profile and clinical severity.

Among AN patients, elevated premorbid body weight levels also predict increased amount of weight lost prior to treatment referral and reduced energy intake throughout treatment (Coners, Remschmidt, & Hebebrand, 1999; Shinder & Shephard, 1993). The notion that AN patients with higher premorbid body weights consumed less during treatment suggests that even after such substantial weight loss, these individuals engaged in more active attempts to avoid returning to these higher weights. This finding suggests another reason why highest premorbid weight reached may be important to examine in relation to later eating disorder characteristics.

Taken together, a history of elevated weights among eating disorder patients suggests these individuals carry a biological predisposition toward higher weights. Consistent with the notion that higher weights could promote increased eating disorder behaviors, higher rates of premorbid elevated weight among certain eating disorder subgroups suggest that a predisposition toward higher weights may be associated with clinical presentation spanning a range of symptoms, including binge/purge and restrictive symptoms. These findings imply not only a significant relationship between elevated body weight and eating disorder risk and maintenance, but also suggest that premorbid body weight may be relevant to subsequent symptom development and clinical outcome. Indeed, continued body dissatisfaction and constant effort required to avoid returning to previous higher weights could serve to perpetuate an eating disorder. While certain eating disorder subgroups have exhibited a greater predisposition toward higher

premorbid weights, the extent to which premorbid body weight per se influences specific eating disorder symptoms has not yet been explored. As such, the relationship between premorbid predisposition toward higher weights and subsequent clinical presentation warrants additional consideration as a factor in eating disorder development and maintenance.

1.2.2. Weight After Disorder Development

In addition to potential relationships between premorbid BMI and eating disorder etiology, individuals' weight after development of an eating disorder (i.e., current weight status, including current body weight and weight relative to premorbid levels) may also carry clinical significance.

Current Weight

The relatively persistent nature of BMI suggests that a premorbid predisposition to higher weights may persist after eating disorder onset. Despite varying amounts of weight loss between individuals, there remains a strong correlation between an individual's premorbid weight and subsequent weights throughout an eating disorder; the correlation between body weights across time despite significant weight fluctuation has been referred to as BMI "tracking" (Carter, McIntosh, Joyce, Gendall, & Bulik, 2004; Coners et al., 1999; Föcker et al., 2014; Miyasaka et al., 2003; Steinhausen, Grigoroiu-Serbanescu, Boyadjieva, Neumarker, & Metzke, 2009). Indeed, premorbid body weights have been linked to subsequent weight throughout disorder development, maintenance and outcome. For example, Shaw et al. (2012) examined pre- and post-morbid BMI levels in BN patients and found that in addition to reporting elevated premorbid BMIs, the majority of patients reached their highest BMI after the onset of the disorder. This

finding further suggests that eating disorder patients may be initially predisposed to higher weights premorbidly and that this biological predisposition may continue to manifest itself after eating disorder onset. As such, one may continue to exhibit this predisposition throughout the lifespan, and may need to exert continued effort to counteract biological drives toward weight gain.

Current Weight and Clinical Presentation

Much of the research on weight after disorder development includes measurements of body weight before treatment (i.e., at referral), during treatment, and after treatment (i.e., at follow-up). Current findings suggest that one's current weight, as assessed at any of these time points, may be relevant to clinical presentation and outcome. For example, BN patients who gained weight at a 5-year follow-up were more likely to have higher BMIs and greater body dissatisfaction at pre-treatment, post-treatment, and follow-up (Carter, McIntosh, Joyce, Gendall, Frampton, et al., 2004). These findings not only support the notion of BMI tracking, but also indicate greater body dissatisfaction with higher weights across a series of time-points.

Similarly, among AN patients, lower BMIs at treatment referral were associated with lower depression scores (Miyasaka et al., 2003). The authors of this research suggest that lower depression scores among AN patients with lower BMIs may be attributed to greater satisfaction with low body weights, further implicating the interpretation of and satisfaction with current weight as a potential maintenance factor for eating disorders. This particular finding is interesting to consider in light of the fact that higher premorbid body weights have previously been associated with lower BMIs at treatment referral in AN patients (Coners et al., 1999; Shinder & Shephard, 1993). Specifically, it could

reasonably be theorized that the more weight AN patients lose, the greater their psychological investment in maintaining a lower weight. Taken together, these findings suggest that AN patients with higher premorbid BMIs not only lose more weight (presenting with lower BMIs at referral), but have increased satisfaction with their weight loss (indicated by lower depression scores) and subsequently increase their efforts to maintain their weight loss and prevent weight gain (as evidenced by reduced energy intake throughout treatment), thereby perpetuating the disorder.

Lower BMI at referral has also been associated with poorer long-term outcomes, including chronic AN and death by emaciation, while those with higher percentage of average body weight at intake have demonstrated faster time to both partial and full recovery (Chakraborty & Basu, 2010; Hebebrand et al., 1997). Consistent with these findings, AN patients with low BMIs at post-treatment follow up exhibited significantly poorer outcomes, characterized by greater eating pathology, lower psychosocial development, and lower total functioning (Steinhausen et al., 2009). These findings thereby implicate current body weight as a factor of importance in the symptoms associated with eating disorders as well.

Weight Suppression

Current weight relative to prior weights may also be relevant to consider: eating disorder development represents a departure from premorbid BMI levels, usually characterized by weight loss, which may carry biological and psychological implications. For example, major weight loss is known to reduce metabolic rate, thereby slowing one's energy expenditure and promoting the likelihood of weight gain (Stice, Durant, Burger, & Schoeller, 2011). Weight loss has also been associated with increased binge-eating

behavior, which is likely to have similar effects on weight (Lowe, Thomas, Safer, & Butryn, 2007). The biological and psychological correlates of weight loss may therefore serve to promote weight gain. This is undoubtedly distressing to eating disordered individuals and is likely met with increased attempts to control or prevent this weight gain, in turn perpetuating maintenance of one's disorder. Indeed, this cycle of symptom maintenance may in part explain the persistent nature of eating disorders. Moreover, one's opinion of their current body weight (e.g., if one's body weight falls within the normal weight range but is heavier than their ideal weight, they may deem their current weight unacceptable) may contribute to body dissatisfaction and subsequent weight loss behaviors, further influencing disorder trajectory. One construct frequently used to assess the discrepancy between premorbid body weights and current body weight is weight suppression (WS), or the difference between one's current weight and highest-ever adult weight (Lowe, 1993). A number of studies have identified heightened levels of WS in BN patients, and mean rates of WS in BN patients are reportedly higher than mean rates of healthy adults (Butryn, Juarascio, & Lowe, 2011; Butryn, Lowe, Safer, & Agras, 2006; Keel & Heatherton, 2010; Lowe, 1993; Lowe, Annunziato, et al., 2006; Lowe, Davis, Lucks, Annunziato, & Butryn, 2006). Studies of BN patients have reported WS means ranging from 9.4-12.0 kg, (20.7-26.4 lbs) compared to healthy college-aged individuals with a reported mean WS of approximately 2.7 kg (5.6 lbs) (Butryn et al., 2011; Butryn et al., 2006; Lowe, Annunziato, et al., 2006; Lowe, Davis, et al., 2006).

The notion that WS is higher in patient populations as compared to healthy controls suggests that current weight status relative to premorbid weight may be a clinically relevant construct. Consistent with this notion, current research suggests that

higher levels of WS could carry both weight and clinical implications among eating disorder patients.

Indeed, research suggests WS is associated with weight change in eating disordered individuals, and has been found to be a robust predictor of weight gain (Carter, McIntosh, Joyce, & Bulik, 2008; Wildes & Marcus, 2012). Among BN patients, higher levels of WS have been shown to predict weight gain over the course of treatment (Carter et al., 2008; Herzog et al., 2010; Lowe, Davis, et al., 2006). Similar findings have implicated WS as a predictor of weight gain in AN patients, with greater WS predicting both greater total weight gain and a faster rate of weight gain throughout treatment (Wildes & Marcus, 2012). Moreover, research indicates that WS and BMI may function independently of one another and may be uniquely associated with weight change. For example, WS has been shown to interact with BMI in AN patients to predict weight related outcomes: Witt et al. (2014) identified a relationship between WS and BMI at lowest weight, such that WS predicted the most weight gain among those with the lowest BMIs. These findings highlight an important interplay between current weight (e.g., BMI) and current weight relative to past weight (e.g., WS), and suggest that both the magnitude of weight lost throughout eating disorder development and current weight influence eating disorder patients' subsequent weight trajectory in both AN and BN patients.

Weight Suppression and Clinical Presentation

Given that the biological and behavioral effects of WS may intensify one's tendency to return to, if not exceed, their highest past weights, WS may also be relevant to eating pathology. Research on the relationship between WS and eating disorder

symptoms suggests WS may be associated with eating disorder development, maintenance and outcome.

Indeed, findings suggest WS may function as a predictor of eating pathology: one study examining WS in college samples found WS to predict the onset of a BN syndrome at 10-year follow-up (Keel & Heatherton, 2010; Lowe, 1993). WS has also been implicated as a maintenance factor for eating disorders. For example, Keel and Heatherton (2010) found greater WS to predict maintenance of symptoms at 10-year follow-up. WS has also been shown to predict bulimic symptoms throughout treatment in both BN and AN patients (Butryn et al., 2006; Wildes & Marcus, 2012). Associations between higher WS in AN patients and bulimic symptoms is consistent with findings of Garfinkel et al. (1980), and further suggest that predisposition to elevated body weight (as evidenced by higher premorbid BMI or greater WS) may be a risk factor for diagnostic cross-over (Wildes & Marcus, 2012).

Finally, WS has been implicated in treatment response and outcome. For example, WS has been shown to interact with BMI to predict eating pathology-related outcomes at treatment discharge, such that high WS at admission predicted increased symptom severity among those with high BMIs, and decreased symptom severity among those with lower BMIs (Berner, Shaw, Witt, & Lowe, 2013). These findings suggest that the further one is from their highest past weight, the stronger the tendency to return toward it, in turn promoting increased attempts to stave off weight gain with maladaptive eating patterns. Similarly, among BN patients receiving CBT, higher WS was associated with treatment drop-out and greater BN symptomatology among treatment completers (Butryn et al., 2006). However, a number of other studies designed to replicate these findings found no

associations between WS and CBT treatment completion, outcome, or change in BN symptoms (Carter et al., 2008; Dawkins, Watson, Egan, & Kane, 2013; Zunker et al., 2011), though these differences could be due to differential magnitude of WS across studies.

1.3. Summary

Because the majority of research on eating disorders emphasizes the cognitive, affective, and social mechanisms of eating pathology, the role of actual body weight has received relatively scant attention. However, findings suggest that eating disordered individuals may be in a constant and losing battle against biological drives toward weight gain. Evidence of such a predisposition may begin premorbidly, either through hereditary or developmental means, and may first be met with attempts to lose weight. The effects of weight loss and WS may call on biological drives to re-initiate weight gain, leaving eating disordered patients at the mercy of their eating disorder symptoms to counter these drives. Therefore, it is possible that weight history may place and hold eating disorder patients at pervasive risk for higher weights throughout the lifespan, and may thereby be relevant to eating disorder symptom development and outcome. However, the means by which premorbid weights may influence clinical outcome remains largely unexplored and is therefore an important target for future research.

1.4. The Proposed Study

While body weight has been established as a construct that warrants further attention, the research thus far has primarily examined highest body weights in adulthood or after disorder development, making it difficult to establish the origins of abnormal weight trajectories. Moreover, the collection of premorbid body weights thus far has

largely relied on retrospective self-report, and may therefore be prone to error. Furthermore, extant literature on body weight and symptom severity has examined only certain subgroups of eating disorder patients, such as AN or BN; research has yet to directly examine premorbid body weights and clinical presentation across all eating disorder diagnoses. As such, access to records of both premorbid body weight measurements throughout childhood and current measures of symptom severity is necessary to identify differential weight patterns and to facilitate a deeper understanding of the relevance of premorbid body weight to eating disorder development and symptom severity.

The Renfrew Center is a treatment facility equipped to treat female patients of all eating disorder diagnoses in both inpatient and outpatient settings. Importantly, Renfrew requests medical growth charts from all of its patients 23 year olds and younger, and from certain older patients to clarify individual weight goals; these growth charts therefore include a series of premorbid BMIs throughout childhood and adolescence. The opportunity to integrate these data with subsequent eating disordered symptomatology at treatment intake offers the potential for a prospective analysis of the relationship between premorbid weight variables and later clinical characteristics and outcome. Analysis of premorbid weight trajectory and its associations with symptoms at treatment presentation will afford us the opportunity to test premorbid body weight as a factor relevant to eating pathology. Findings from the current study may also substantiate the need for increased emphasis on body weight in future eating disorder research and conceptualizations.

The current study is part of a joint project, wherein another doctoral student and I are examining the relation between premorbid BMIs and eating disorder characteristics

but are focusing on different groups of these characteristics. As such, each project examines distinct outcomes. The current project is focused primarily on the relationship between premorbid body weights and restrictive eating symptoms and weight and shape concerns across patients of all diagnoses. Binge/purge related symptom severity has been examined separately in another project and as such will not be addressed henceforth.

Classification of psychological symptoms and disorders has historically followed a categorical framework, such that presenting symptoms have traditionally informed a decision to assign an individual to a specific diagnostic group (e.g., AN vs BN). Indeed, these categorical diagnoses allow for an efficient classification system which has guided treatment and research decisions for decades. However, in recent years, the field of psychology has witnessed increased emphasis on dimensional frameworks of psychological disorders, which identify domains or constructs relevant to functioning and assess these on a continuum from "normal" to "abnormal". Dimensional classification of domains thereby measures individual variability in functioning and can elucidate differential patterns of symptom severity. Dimensional frameworks of psychological disorders and symptoms have been espoused by the American Psychological Association, U.S. National Institutes of Health, and World Health Organization (Insel et al., 2010; Regier, 2007). An example of such a dimensional framework is the National Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC) initiative, which identifies constructs that can be measured continuously (e.g., attention) and that contribute to broader domains of functioning (e.g., cognitive systems).

Given the field's recommendations and because the current project is exploratory in nature, the current study sought to examine the relationship between premorbid body

weights and eating pathology using primarily dimensional constructs that are relevant across traditional categorical diagnoses (e.g., dietary restraint as an eating disordered behavior identified in both AN and BN patients) to identify specific symptom clusters associated with premorbid body weights. Although the framework for the current project was primarily dimensional, different eating disorder diagnoses have different ages of onset and may therefore be differentially associated with premorbid weight status and symptom severity. As such, diagnoses were considered in the secondary aims of the study, described below.

1.5. Aims and Hypotheses

As aforementioned, the current study was exploratory and designed to elucidate relatively uninvestigated relationships. Findings from current research suggest eating disorder patients may be susceptible to higher premorbid weights, and that premorbid weights may be relevant to eating disorder development and outcome. With respect to these findings, the current study sought to examine the relationship between premorbid body weights and clinical outcome, as measured by severity of eating pathology spanning a range of symptoms.

Primary Hypothesis: **Relationship between premorbid BMI and clinical outcomes**
Current findings with regard to relationships between premorbid BMIs and clinical outcomes suggest potentially positive associations between the two. For example, given the notion that eating disordered individuals have a strong desire to be thin, it stands to reason that the further away from an ideal body weight these individuals are (i.e., the higher their weight), the greater the pressure to engage in eating disordered behaviors.

Moreover, elevated weight is itself a predictor of accelerated weight gain in the future, leaving eating disordered individuals at the continued mercy of their eating disordered behaviors to counteract drives for weight gain. Higher premorbid body weights may therefore be relevant to symptom development (i.e., weight loss from an undesired higher weight) and maintenance (i.e., struggle to prevent a return to the higher weight). Preliminary research provides support for this notion: findings provide support for the potential relationship between elevated premorbid BMIs and increased eating-disorder related symptoms typically associated with AN symptoms, including Restrained Eating, Eating Concerns and Weight and Shape concerns (Garfinkel et al., 1980; Sawyer et al., 2016; Shinder & Shephard, 1993). As such, we expect elevated premorbid BMI measurements to predict greater symptom severity at intake.

> **Specific Primary Aim:** To investigate whether premorbid body weights are associated with subsequent severity of symptoms (including Restrained Eating, Eating Concerns, and Shape and Weight concerns) at treatment intake.

Relationship between premorbid weights and current weight status

Available evidence suggests potential relationships between premorbid BMI and subsequent clinical outcomes. However, we recognize that there may be an additional relationship between premorbid weights and current weight status (i.e., current BMI and current WS). For instance, as discussed above, research supports the notion that BMI "tracks" over time, implicating a relationship between premorbid body weights and current body weight. Moreover, given the pressures of avoiding weight gain, elevated premorbid weights may also motivate patients to keep their current weight far below premorbid levels. Indeed, this notion is consistent with findings that AN patients with

higher premorbid levels lost more weight before entering treatment (Coners et al., 1999). As such, premorbid weight may be associated with current BMI and levels of WS (i.e., sustained weight loss). BMI and WS were therefore considered as possible covariates to include in primary analyses.

Secondary Analysis: Relationship between premorbid weight, age of onset, and diagnosis

It is well known that there is a difference in the average age of onset between AN and BN patients, with AN patients developing their disorder at an earlier age (Favaro, Caregaro, Tenconi, Bosello, & Santonastaso, 2009). If AN patients do have an earlier age of onset than BN patients, it is plausible that disorder development at an earlier age may contribute to lower premorbid weights, and may therefore partly explain relationships between premorbid weight and clinical outcome. Given that both diagnosis and age of onset are collected by Renfrew, the current study sought to examine whether AN and BN patients differed in premorbid BMIs, and whether age of onset explained some of this difference.

- **Secondary Aim:** To 1) confirm that prior findings with regard to differential age of onset for AN and BN, and 2) determine whether premorbid BMIs differ in AN and BN patients (and if so, whether the difference is partly explained by different ages of onset).

Chapter 2: Methods

2.1 Participants

The current study included female adolescent and adult inpatients who received residential treatment services from The Renfrew Center Eating Disorder Treatment Facility Spring Lane (Philadelphia, PA) and Coconut Creek (Coconut Creek, FL) locations. All inpatients received treatment between February 2015 and August 2016 and were either currently receiving treatment or had recently discharged. De-identified data from patients' growth charts, intake assessments and weight data were collected from patients and included in analyses.

2.1.1 Recruitment

Growth charts were collected between September 2015 and December 2016. All participants were at least 14 years of age and were current or previous inpatients who received residential treatment from Renfrew's Spring Lane or Coconut Creek locations.

During this time, all patients at Renfrew were asked to complete three assessment surveys, including one at intake, one at discharge, and one at six-month follow-up. All assessment surveys were self-report measures administered either online or in paper format, and were comprised of questions designed to measure eating pathology, mood and anxiety, and individuals' experience in treatment at Renfrew. Patients who completed all three assessments (and responded to at least 90% of their follow-up assessment items) were given a $30 Amazon gift card to compensate them for their time. Because the current study utilized de-identified data that had already been collected by Renfrew, no additional compensation was offered for the current study.

2.1.2. Inclusion and exclusion criteria

To be included in analyses, participants were required to: 1) provide prior consent to use patient-provided data for external research purposes (consent provided by the patient, if adult, or by her parent(s) or legal guardian(s), if adolescent), and 2) have growth charts with a minimum of one BMI data point prior to the onset of the eating disorder.

2.2 Procedures

Growth chart data were requested by Renfrew for all patients 23 years old and younger at admission. If growth chart data were not collected from these patients at admission, follow-up requests for growth charts were made for patients who were 1) significantly low weight, 2) significantly high weight, or 3) highly weight suppressed (i.e., great than 15 lbs). Growth chart data were also requested for certain patients above 23 years in the event that the patient was insistent that she should be a lower weight.

Intake and discharge assessment and weight data were collected by the research team at both Renfrew Center locations for the majority of patients. Every 6-8 weeks, Renfrew research team members reviewed patient data for inpatients who had been admitted over the past month. Patients who consented to using data for research purposes received a unique identifying participant code, which was used to label their data. Data from these patients were then collected, de-identified, labeled and shared with Drexel study team members according to procedures detailed below.

2.2.1. Growth Charts

Growth charts for eligible inpatients were de-identified and labeled with participant code by Renfrew's research team members. De-identified growth charts were

then scanned and e-mailed to the current study team members. Data from growth charts were subsequently cleaned and entered according to the procedures described below (see Section 2.5, Data Analysis).

2.2.2. Assessment Data

The Renfrew research team maintains a research database with all intake assessment, discharge assessment, and weight data for all patients. Research team members selected eligible participants from this larger database and transferred their de-identified information to a new database. Data were then labeled using participant codes assigned to each patient, and the updated database was shared with study team members.

Data included in the updated, de-identified database were cleaned by study team members according to the procedures described below and were then combined with growth chart data for subsequent analyses (see Section 2.6, Data Analysis).

2.3 Measures

Because the current study sought to prospectively examine relationships using data already collected, specific instruments used to measure constructs of interest were not selected by study team members. Although there may be more ideal measures to consider for the purposes of the present study, the measures described below were selected based on study aims and data available to the research team.

2.3.1. Weight Measures

Growth Charts: Growth charts were provided by patients' pediatricians and primary care physicians. The growth charts included records of patients' measured height, weight, and occasionally BMI at a series of ages, and were used to calculate two different

measurements of premorbid BMI, described below (see Section 2.5.2, Data Cleaning). The data provided in the growth charts were subject to a number of inherent limitations (see Section 2.5.1, Limitations of Data). As such, a series of guidelines were developed to ensure consistent and reliable cleaning and coding of data to minimize this variability. See Section 2.5.2 (Data Cleaning) for specific cleaning and coding procedures.

To adjust BMI relative to age, all BMI values were converted to z-scores according to the Center for Disease Control and Prevention (CDC) growth charts. Because premorbid body weight is not an extensively researched or clearly defined construct, we calculated the following two measurements of premorbid BMI. See Figure 1 for an illustration of each of these values using a sample growth chart.

1. Premorbid BMI Measurement #1, Highest premorbid BMI: Highest premorbid z-BMI is of interest because it represents the extent of elevated BMI reached prior to disorder development and therefore reflects a general proneness toward reaching higher BMI levels. See Figure 1, point B for an illustration of this variable.
2. Premorbid BMI Measurement #2: Trajectory to highest BMI: Trajectory to highest premorbid z- BMI provides additional information on one's weight prior to reaching their highest premorbid BMI and may therefore be informative independently of highest premorbid BMI alone. This measurement is defined as the slope between the highest premorbid z-BMI and its penultimate premorbid z-BMI. In Figure 1, this variable would be measured by calculating the slope between points A and B.

Current BMI: Renfrew staff measured participants' height and weight at both intake and discharge.

Weight Suppression: Weight suppression was calculated as the difference between the patient's self-reported highest ever adult weight and patient's weight taken at admission.

2.3.2. Eating Disorder Diagnosis and Symptoms

Eating Disorders Examination Questionnaire Version 5.2 (Fairburn & Beglin, 2008) *(EDE-Q) 5.2:* The EDE-Q is a 31-item self-report version of the Eating Disorders Examination structured interview, and is used to measure the severity and frequency of symptoms associated with eating pathology over the previous 4 weeks (28 days). EDE-Q scores are calculated according to a global score (based on all items), and specific subscale scores (Restraint, Eating Concerns, Weight Concerns, and Shape Concerns). The EDE-Q has demonstrated reliability for the total scale and four subscales (Peterson et al., 2007).

Age of Onset: Renfrew's intake assessment questionnaire asks patients to self-report the age at which they developed eating disorder-related symptoms ("at what age did your eating disorder first begin?"). Patient response was used as age of eating disorder onset.

Diagnosis. Renfrew psychiatrists assessed and assigned an eating disorder diagnosis to all patients at intake.

2.4 Ethical Considerations

The current study fell under a blanket protocol approved by Drexel University's Institutional Review Board. All participants included in the study provided informed consent to have their data used for research purposes. As described above, all participant data were de-identified by Renfrew staff and labeled with a non-identifying participant code before being shared with Drexel study team members. Therefore, Drexel study team

members did not have access to participants' identifying information and were not be able to link collected data with names. The design of the study also precluded direct contact between participants and study team members, thereby significantly minimizing the likelihood of risks or adverse events. No risk or adverse events were noted.

2.5 Data Preparation

2.5.1 Limitations of Data

As mentioned above, growth chart data have certain inherent limitations. For one, data were recorded most frequently as points on a graph (i.e., a dot at a point representing height and weight) and were only rarely recorded as specific values (i.e., "121.3 lbs"). As such, in the absence of specific values, data required visual inspection and were estimated, leaving the possibility for some error inherent in this process. Moreover, changes in height and weight were recorded at variable time points both within and between participants, such that some patients had numerous data points (e.g., more than 10), while others had very few (e.g., 1 or 2). Additionally, within patients, the time between data points varied significantly depending on the frequency of their past medical visits. Finally, the number of data points collected prior to the onset of a patient's eating disorder was also highly variable. Because growth chart data were highly variable both within and between participants, efforts were made to clean and organize the data for analysis purposes. Study team members responsible for data cleaning and entry were trained according to study protocol. Specific procedures for each measure provided are included below.

2.5.2. Data Cleaning

Data Entry

The occasionally inconsistent nature of the growth chart data required standardized coding efforts to maximize accuracy and reliability of the data entered. Two Drexel study team members reviewed the initial batch of collected growth charts to identify all possible ambiguities and develop a standardized set of rules to guide data entry decisions, as detailed in Table 1. The first ten growth charts were entered jointly by both study team members according to these guidelines. The subsequent ten growth charts were entered independently by the same study team members and compared to determine inter-rater reliability.

Data Organization

Once all data were entered, study team members ensured that all participants had height and weight data in centimeters and kilograms, respectively. For participants whose growth charts did not include BMI data, height and weight values were used to determine BMI values in kg/m^2.

All growth chart data were then combined with all other variables (EDE-Q, age of onset, current BMI, WS) in a master database. Age of disorder onset was used to distinguish premorbid BMIs from BMIs recorded after disorder development. All BMI values were converted to z-scores according to the Center for Disease Control and Prevention (CDC) growth charts. We then calculated the two measurements of premorbid BMI listed above.

Chapter 3: Data Analysis

All analyses were conducted in SPSS v. 23.0. After establishing rules for data entry, 10 growth charts were entered independently by two raters and checked for reliability. Premorbid weight data between two raters demonstrated excellent inter-rater reliability across datapoints, *r*age = 1.0, *r*weight = .99, *r*stature = .98. Once reliability was confirmed, remaining premorbid weight data were entered by both raters.

Growth chart data were collected from a total of 101 female participants. Of the data collected, 41 participants with both viable weight data collected prior to self-reported eating disorder onset and completed intake assessment data were available for analyses. Approximately half of these participants (n =21) had enough weight data to calculate weight trajectory to highest premorbid z-BMI. Due to the small sample size, weight trajectory to highest premorbid z-BMI was not included in analyses.

Descriptive statistics were generated for all study variables. A one-way analysis of variance (ANOVA) was employed to compare weight data, eating disorder symptoms, and age of onset across different diagnoses. Relationships between highest premorbid z-BMI and current weight variables (WS and BMI at admission) were assessed using two-tailed Pearson correlation analyses. Across all weight variables, BMI measurement #1 (highest premorbid z-BMI) was significantly positively correlated with admission BMI, r = .570, p < .001. This relationship was expected due to prior findings with regard to BMI tracking over time (Steinhausen et al., 2009). There were no other significant associations between variables (see Table 2).

The effects of highest premorbid z-BMI on current eating disorder symptom severity were assessed using multiple linear regression. Assumptions for multiple linear

regression were tested, including the absence of influential outliers, normal error distribution, absence of multicollinearity, and homoscedasticity. Highest premorbid z-BMI was first individually entered into a regression as a measure of the independent effects on eating disorder symptoms.

Chapter 4: Results

Descriptive Statistics

The final sample consisted of 41 female participants aged 14 to 43 (M_{age} = 17.27, SD_{age} = 4.79). Across all participants, BMI at admission ranged from 11.8 to 44.1 (M_{BMI} = 19.74, SD_{BMI} = 6.69). Self-reported disorder onset occurred during adolescence for all participants (M_{age} = 13.61, SD_{age} = 1.76, $range_{age}$ = 10-18). Highest premorbid z-BMIs ranged from -1.6 to 2.5 across participants ($M_{z\text{-}BMI}$ = 0.66, $SD_{z\text{-}BMI}$ = 0.96). See Table 3 for descriptive statatistics for EDE-Q scores on Eating Concerns, Weight concerns, Shape Concerns, and Restrained Eating behaviors.

Eating Disorder Diagnoses

Eating disorder diagnosis was unavailable for one participant. Of the remaining 40 participants, 18 (45%) met criteria for a diagnosis of AN-R, 10 (25%) for BN, 7 (~18%) for atypical AN, and 5 (~13%) for AN-B/P. Results of the one-way ANOVA between these four groups indicated a significant difference in age of onset, $F(3,36) = 3.04$, $p = .04$, weight variables (highest premorbid z-BMI, $F(3,36) = 3.41$, $p = .03$, BMI at admission, $F(3,36) = 11.28$, $p < .001$), and one of the four eating disorder subscales measured by the EDE-Q (Eating Concern subscale scores, $F(3,36) = 2.94$, $p = .046$). There was also a trending difference in self-reported EDE-Q Weight Concern, $F(3,36) = 2.428$, $p = .08$. It is noted that the small sample sizes of each group limit the validity of

any apparently meaningful differences. Results from Tukey post hoc analyses are discussed in detail below.

Age of Onset

Tukey post hoc tests revealed that the atypical AN group had a significantly earlier age of onset (12.14 ± 1.68) than those with AN-R (14.23 ± 1.98). There were no statistically significant differences in age of onset between any other group.

Weight Variables

Tukey post hoc analyses revealed significantly higher premorbid z-BMI in patients diagnosed with BN (1.42 ± 1.99) as compared to those with AN-R (*mean* = 0. 19±.54, $p = .03$) and atypical AN (.26 ± .96, $p = .05$). Similarly, BMI at admission was significantly higher among BN patients (27.41 ± 9.49) than AN-R (16.14 ± 1.96, $p < .001$), AN-B/P (17.24 ± 1.18, $p = .004$) and atypical AN (20.54 ± 1.14, $p = .04$). There were no significant differences in either weight variable between AN subtypes.

Eating Disorder Symptoms

Post hoc tests revealed marginally greater self-reported Eating Concerns (EDE-Q Eating Concern subscale score) in BN patients (4.62 ± .93) than in AN-R patients (3.47 ± 1.29, $p = .06$). No additional significant group differences in Eating Concern or Weight Concern scores were detected between any groups.

Single-Variable Effects of Highest Premorbid Weight

Results of linear regression analyses indicated that when entered independently into the model, higher premorbid z-BMIs accounted for a significant amount of variability in EDE-Q Eating Concern scores at intake, R^2=.11, $F(1,39) = 4.69$, $p = .04$, suggesting that those with higher premorbid weights are susceptible to higher Eating

Concerns at admission. Highest premorbid z-BMI did not significantly predict EDE-Q Weight Concern, Shape Concern or Restraint. See Table 4 for paramaters of each regression.

Effect of Highest Premorbid Weight and Covariates

To examine the effects of highest premorbid z-BMI with relevant covariates, including age at highest premorbid z-BMI and BMI at admission, subsequent linear regression analyses were run to determine whether highest premorbid z-BMI explained a significant amount of variance in EDE-Q Eating Concern scores with inclusion of these covariates. Overall, the model explained a significant amount of variability in EDE-Q Eating Concern scores at intake, $F(3,37) = 3.94$, R^2=.20, p = .039. However, higher premorbid z-BMI no longer predicted Eating Concern scores, B = .13, SE(b) = .24. p = .58, $r2$ = .007. Instead, results suggest that BMI emerged as the only predictor of Eating Concerns, B = .07, SE(B) = .77. p = .046, r2 = .09. Age at highest premorbid z-BMI did not significantly predict Eating Concerns, B = -.02, $SE(B)$ = .05, p = .58, r2 = .004 (see Table 5).

Exploratory Analyses

Additional exploratory linear regression analyses were run to examine the possible interaction between highest premorbid z-BMI and BMI at admission. First, both variables were centered and their product term was computed; centered variables and their product term were then entered as predictors of each of the four eating disorder symptom severity scores.

Table 5 lists the parameters of these regression analyses. Overall, the model with both variables and their interaction term significantly predicted EDE-Q Eating Concerns, $F(3,37) = 4.35$, $R^2 = .26$, $p = .01$. There was a main effect of BMI at admission, $B = .155$, $SE(B) = .06$, $p = .01$, $r^2 = .14$, such that higher BMI was associated with higher Eating Concern at admission. There was no significant main effect of highest premorbid z-BMI, $B = .08$, $SE(B) = .23$, $p = .74$, $r^2 = .002$. There was a marginally significant interaction between highest premorbid z-BMI and BMI at admission, $B\ interaction = 1.07$, $SE(B) = .04$, $p = .08$, $r^2 = .06$. Specifically, this interaction indicated that a higher BMI at admission combined with a history of relatively lower highest premorbid weight predicted greatest Eating Concerns at admission. To better understand these interaction patterns, we divided all patients into 2 admission BMI groups (high vs. low) and 2 premorbid z-BMI groups (high vs. low) to compare EDE-Q Eating Concern scores. See Figure 2 for an illustration of this relationship.

Regression analyses yielded similar results for the EDE-Q Weight Concern scores, $F(3, 37) = 4.83$, $R^2 = .28$, $p = .006$. There was again a main effect of BMI at admission, such that higher BMI was associated with higher Weight Concern scores at admission, $B = .24$, $SE(B) = .07$, $p = .002$, $r^2 = .22$. There was no main effect of highest premorbid z-BMI, $B = -.45$, $SE(B) = .28$, $p = .116$, $r^2 = .05$. There was a marginally significant interaction between highest premorbid z-BMI and BMI at admission, B interaction = -.09, $SE(b) = .048$, $p = .056$, $r^2 = .07$. The pattern of these findings was consistent with Eating Concern results: Weight Concern was greatest among those with both higher BMI at admission and a history of relatively lower highest premorbid z-BMIs (see Figure 3).

To better understand the interaction between current and historical weight, we also compared mean weight variables between the four groups. See Table 7 for mean values of admission BMI and highest premorbid z-BMI across groups.

Regression analyses were run again to control for age at highest premorbid z-BMI as a possible covariate. These analyses did not change results; see Table 6 for parameter estimates from both regression analyses.

4.1. Power Analysis

An initial power analysis was conducted prior to data collection using G*power (Faul, Erdfelder, Lang, & Buchner, 2007). Input parameters included alpha level (.05), power (.80), and effect size (.15). Effect size was set to medium due to the primarily exploratory nature of the analyses. Results indicated that for the primary hypothesis, the current study required a sample size of 68 participants. The sample size of the current study largely depended on the number of patients who both provided sufficient data and consented to share these data for research purposes. With only 41 total participants included in the analyses, the current study was underpowered. Limited power is discussed and considered as a potential study limitation below.

Chapter 5: Discussion

In considering the results of the current study, it should be noted that several factors create interpretive challenges. These limitations will be detailed and addressed throughout the discussion.

Although there was a significant difference in premorbid z-BMIs between AN and BN patients, there was not a significant difference in the age of onset between these

patient groups, suggesting that earlier age of onset did not necessarily account for differences in premorbid weight. As such, all patients were included and considered in the same analyses.

To confirm the accuracy of growth chart weights, we ran a correlation between highest premorbid z-BMI and self-reported highest past weight prior to disorder onset. A significant correlation between the two variables (r = .47, p = .005) suggest the highest premorbid z-BMIs as collected by growth charts were correlated with patients' reported highest past weight, and are therefore further corroborated.

On average, highest premorbid z-BMI across participants was 0.66, suggesting higher than average weights premorbidly; this result replicates past findings about those with eating disorders having relatively elevated premorbid BMIs (Crisp et al., 1980). However, because the current analysis did not include a control group, it is not certain that elevated z-BMIs in the current sample were necessarily due to an eating disorder diagnosis. Consistent with study hypotheses, initial results indicated that among female eating disorder inpatients, highest premorbid z-BMI was associated with greater Eating Concerns at treatment admission. Moreover, there was a medium effect size of this relationship, suggesting a potentially significant relationship.

However, in the presence of other variables, including BMI at admission and age at highest premorbid z-BMI, highest premorbid z-BMI was no longer independently associated with any eating disorder symptoms. Instead, BMI at admission emerged as the strongest predictor of Eating Concerns. Taken together, these findings were inconsistent with our hypotheses that individual differneces in highest premorbid BMI have lingering effects that continue to influence body dissatisfaction and eating pathology. Findings

instead suggest that BMI at admission, or current BMI, better accounted for variability across eating disorder symptoms than highest premorbid BMI. It is noted that although multicollinearity does not affect overall model results, the substantial correlation between admission BMI and highest premorbid z-BMI suggests that coefficient estimates for individual predictors may be unstable or invalid. As such, it is difficult to draw definitive conclusions that highest premorbid z-BMI is not independently important when including admission BMI. Interestingly, exploratory analyses examining the combined influence of highest premorbid z-BMI and BMI at admission suggested a potential interaction between historical and current weight. Overall, individuals at higher current BMIs tended toward increased Eating and Weight concerns. However, among these individuals, those with an added history of relatively lower premorbid weights exhibited the greatest Eating and Weight concerns.

These initial findings run counter to our original hypotheses, in that lower premorbid weight was associated with greater eating disorder symptoms later on. However, these findings potentially capture an unexpected relationship between current and historical weight. Based on initial hypotheses, the original expectation would be that those with high historical and current weights would have the greatest Eating and Weight Concerns. However, the observed results suggest that a more powerful effect is associated with having a high current weight that is relatively elevated compared to premorbid weights, so that perhaps such individuals are mostly concerned with the prospect of reaching or exceeding their highest ever weight. On the other hand, individuals who had historically higher premorbid weights may evaluate their current weights relative to their historical weights. It is possible that despite being at a relatively

higher current BMI, they remain further below their highest premorbid weight and are, in turn, relatively less distressed. Moreover, despite being only marginally significant, these interactions both had small-medium effect sizes, indicating that lack of a significant interaction may have been due to the small sample size rather than a non-significant relationship. Perhaps, then, the role of historical weight and premorbid BMI is indeed an important measure of eating disorder severity, but one which should be carefully considered in light of current weight status, which undoubtedly influences current eating disorder symptom severity. Indeed, individuals with relatively lower premorbid weights could evaluate their current weight as too close to their historical highest weight and respond accordingly.

It is noted that these findings should be considered as highly tentative, given a number of limitations that are discussed below.

Limitations and Future Directions

In considering the findings of the current study, it is important to address a number of inherent limitations. For one, as aforementioned, there was significant variability in the premorbid weight data, with differences in both number of data points, time between data points, and age at which weights were taken. Importantly, a number of participants whose growth charts were available had switched physicians throughout adolescence and did not have weight data prior to disorder development. Therefore, despite collecting data from over 100 participants, only a limited number of individuals had data available for analyses, resulting in a small sample size that limited the power of the study and the likelihood of detecting true differences. Similarly, weight data needed to calculate other premorbid BMI variables were often not available, which reduced the

sample size for certain planned analyses too much for meaningful interpretation. Data used in analyses allowed for calculation of highest premorbid z-BMI, but were not thorough enough to examine the relationships between other premorbid weight measurements, such as trajectory to highest weight, first available, z-BMI, or earliest to highest weight change, and eating disorder symptom severity at admission.

Moreover, because follow-up requests for growth chart data were made for participants with specific weight concerns, it is possible that the data collected were more representative of a specific sub-sample of participants than the general inpatient population. As such, this method of collection should be considered as a potential study limitation.

We also acknowledge that because these analyses were exploratory, we did not correct for multiple comparisons. As such, it is possible that certain findings would lose significance by controlling for comparisons, and this should be taken into consideration when interpreting the current results. Moreover, as aforementioned, the strong correlation between admission BMI and highest premorbid z-BMI limits the interpretation of the coefficient estimates for the individual predictors.

Another limitation to consider is the lack of information about psychiatric history across patients. Despite having access to current diagnoses, history of prior eating disorders was not collected; as such, it is possible that certain BN patients had previously had an AN diagnosis, which could explain the unexpected earlier age of self-reported symptom onset among BN patients. Similarly, the current analyses did not examine comorbidities or current medication use, which could have plausibly influenced results.

Additionally, there are certain limitations to the measures employed in the study.

The current study's estimate of highest premorbid z-BMI was defined by the highest *available* z-BMI prior to eating disorder onset. As such, although this measurement was correlated with self-reported highest past weight and used as an index of highest premorbid z-BMI, it is plausible that higher premorbid weights were reached but not recorded or included in available growth charts. More specific and consistent growth chart data may have enabled us to better examine the role of historical weight. There are additional limitations to the outcome measures utilized. For example, the current gold standard for measurement of eating disorder psychopathology is the EDE clinician administered interview (Guest, 2000). However, the current measure of eating disorder symptom severity utilized a readily available version of the EDE-Q measure already distributed to patients at Renfrew; as such, study outcome variables were measured using subjective, self-reported severity rather than clinician calculations of severity. It is therefore possible that use of the self-report EDE-Q might have introduced error in the results.

Similarly, because age of onset is based on self-report in response to a written question, it is subject to different interpretation (e.g., time of symptom onset vs. weight loss vs. official disorder diagnosis). The ambiguity of this question limits our ability to confirm age of disorder onset, and is therefore noted as a potential study limitation.

It is also noted that certain eating disorder patients, particularly those who were low weight at admission, may have been content with their body weight, and may therefore not have endorsed high weight concerns. As such, Weight Concern subscale scores may not adequately reflect current symptom severity among these patients and should be considered as a limitation of the current study.

Finally, it is noted that the current study examined individuals in an inpatient setting. Given that a number of variables may differ between patients identified in inpatient settings versus patients in other outpatient or community settings, it is impossible to generalize our findings to all eating disorder participants.

Taken together, findings from the current study suggest a combined influence of historical *and* current weight on eating disorder symptom severity. Based on the available data, it appears that while the information gleaned from one's historical weight and current weight are independently important, an understanding of both variables together helps one to better understand one's evaluation of their current weight. However, as aforementioned, the current study presented a number of unexpected challenges which significantly limit interpretation of findings. Therefore, it is still plausible that a more methodologically adequate study could perhaps reveal a role for historical highest BMIs. For example, similar data could be pursued in other countries where high quality growth chart data are kept on citizens across the lifespan. Future research studies could use these data to better test the relevant hypothees. Moreover, these same data could also be employed to further examine the potential independent and interacting effects of historical and current BMI, which would allow for a better understanding of the role of body weight in eating disorder symptomatology.

List of References

1. Berner, L. A., Shaw, J. A., Witt, A. A., & Lowe, M. R. (2013). The relation of weight suppression and body mass index to symptomatology and treatment response in anorexia nervosa. *J Abnorm Psychol, 122*(3), 694-708. doi: 10.1037/a0033930

2. Butryn, M. L., Juarascio, A., & Lowe, M. R. (2011). The relation of weight suppression and BMI to bulimic symptoms. *International Journal of Eating Disorders, 44*(7), 612-617. doi: 10.1002/eat.20881

3. Butryn, M. L., Lowe, M. R., Safer, D. L., & Agras, W. S. (2006). Weight suppression is a robust predictor of outcome in the cognitive-behavioral treatment of bulimia nervosa. *J Abnorm Psychol, 115*(1), 62-67. doi: 10.1037/0021-843x.115.1.62

4. Carlat, D. J., Camargo, C. A., & Herzog, D. B. (1997). Eating disorders in males: a report on 135 patients. *Am J Psychiatry, 154*(8), 1127-1132. doi: 10.1176/ajp.154.8.1127

5. Carter, F. A., McIntosh, V. V., Joyce, P. R., & Bulik, C. M. (2008). Weight suppression predicts weight gain over treatment but not treatment completion or outcome in bulimia nervosa. *J Abnorm Psychol, 117*(4), 936-940. doi: 10.1037/a0013942

6. Carter, F. A., McIntosh, V. V. W., Joyce, P. R., Gendall, K. A., & Bulik, C. M. (2004). Impact of pre-treatment weight on weight trajectory in women treated for bulimia nervosa. *European Eating Disorders Review, 12*(6), 387-391. doi: 10.1002/erv.600

7. Carter, F. A., McIntosh, V. V. W., Joyce, P. R., Gendall, K. A., Frampton, C. M. A., & Bulik, C. M. (2004). Patterns of weight change after treatment for bulimia nervosa. *International Journal of Eating Disorders, 36*(1), 12-21. doi: 10.1002/eat.20021

8. Chakraborty, K., & Basu, D. (2010). Management of anorexia and bulimia nervosa: An evidence-based review. *Indian J Psychiatry, 52*(2), 174-186. doi: 10.4103/0019-5545.64596

9. Coners, H., Remschmidt, H., & Hebebrand, J. (1999). The relationship between premorbid body weight, weight loss, and weight at referral in adolescent patients with anorexia nervosa. *Int J Eat Disord, 26*(2), 171-178.

10. Crisp, A. H., Hsu, L. K., Harding, B., & Hartshorn, J. (1980). Clinical features of anorexia nervosa. A study of a consecutive series of 102 female patients. *J Psychosom Res, 24*(3-4), 179-191.

11. Crisp, A. H., & Stonehill, E. (1971). Relation between Aspects of Nutritional Disturbance and Menstrual Activity in Primary Anorexia Nervosa. *British Medical Journal, 3*(5767), 149-151.

12. Dawkins, H., Watson, H. J., Egan, S. J., & Kane, R. T. (2013). Weight Suppression in Bulimia Nervosa: Relationship with Cognitive Behavioral Therapy Outcome. *International Journal of Eating Disorders, 46*(6), 586-593. doi: 10.1002/eat.22137

13. Elks, C. E., den Hoed, M., Zhao, J. H., Sharp, S. J., Wareham, N. J., Loos, R. J., & Ong, K. K. (2012). Variability in the heritability of body mass index: a systematic review and meta-regression. *Front Endocrinol (Lausanne), 3*, 29. doi: 10.3389/fendo.2012.00029

14. Engler, P. A., Crowther, J. H., Dalton, G., & Sanftner, J. L. (2006). Predicting eating disorder group membership: an examination and extension of the sociocultural model. *Behav Ther, 37*(1), 69-79. doi: 10.1016/j.beth.2005.04.003

15. Fairburn, C. G., & Beglin, S. J. (2008). Eating Disorder Examination Questionnaire (6.0) *Cognitive Behavior Therapy and Eating Disorders*. New York: Guilford Press.

16. Fairburn, C. G., Cooper, Z., Doll, H. A., & Welch, S. L. (1999). Risk factors for anorexia nervosa: three integrated case-control comparisons. *Arch Gen Psychiatry, 56*(5), 468-476.

17. Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. *Beh Res Therapy, 41*. doi: 10.1016/s0005-7967(02)00088-8

18. Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior research methods, 39*(2), 175-191.

19. Favaro, A., Caregaro, L., Tenconi, E., Bosello, R., & Santonastaso, P. (2009). Time trends in age at onset of anorexia nervosa and bulimia nervosa. *J Clin Psychiatry, 70*(12), 1715-1721. doi: 10.4088/JCP.09m05176blu

20. Föcker, M., Bühren, K., Timmesfeld, N., Dempfle, A., Knoll, S., Schwarte, R., . . . Herpertz-Dahlmann, B. (2014). The relationship between premorbid body weight and weight at referral, at discharge and at 1-year follow-up in anorexia nervosa. *European Child & Adolescent Psychiatry, 24*(5), 537-544. doi: 10.1007/s00787-014-0605-0

21. Garfinkel, P. E., Moldofsky, H., & Garner, D. M. (1980). The heterogeneity of anorexia nervosa. Bulimia as a distinct subgroup. *Arch Gen Psychiatry, 37*(9), 1036-1040.

22. Garner, D. M., Olmsted, M. P., Polivy, J., & Garfinkel, P. E. (1984). Comparison between weight-preoccupied women and anorexia nervosa. *Psychosom Med, 46*(3), 255-266.

23. Guest, T. (2000). Using the eating disorder examination in the assessment of bulimia and anorexia: issues of reliability and validity. *Soc Work Health Care, 31*(4), 71-83. doi: 10.1300/J010v31n04_05

24. Hebebrand, J., Blum, W. F., Barth, N., Coners, H., Englaro, P., Juul, A., . . . Remschmidt, H. (1997). Leptin levels in patients with anorexia nervosa are reduced in the acute stage and elevated upon short-term weight restoration. *Mol Psychiatry, 2*(4), 330-334.

25. Herman, C. P., & Polivy, J. (1988). Psychological factors in the control of appetite. *Curr Concepts Nutr, 16*, 41-51.

26. Herzog, D. B., Thomas, J. G., Kass, A. E., Eddy, K. T., Franko, D. L., & Lowe, M. R. (2010). Weight suppression predicts weight change over 5 years in bulimia nervosa. *Psychiatry Res, 177*(3), 330-334. doi: 10.1016/j.psychres.2010.03.002

27. Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., . . . Wang, P. (2010). Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *Am J Psychiatry, 167*(7), 748-751. doi: 10.1176/appi.ajp.2010.09091379

28. Jasik, C., Breland, D., & Buckelew, S. (2010). The Association of Pre-Morbid Obesity with Severity of Eating Disorder Presentation in Males. *Journal of Adolescent Health, 46*(2, Supplement 1), S51-S52. doi: http://dx.doi.org/10.1016/j.jadohealth.2009.11.124

29. Keel, P. K., & Heatherton, T. F. (2010). Weight suppression predicts maintenance and onset of bulimic syndromes at 10-year follow-up. *J Abnorm Psychol, 119*(2), 268-275. doi: 10.1037/a0019190

30. Lingswiler, V. M., Crowther, J. H., & Stephens, M. A. P. (1989). Affective and cognitive antecedents to eating episodes in bulimia and binge eating. *International Journal of Eating Disorders, 8*(5), 533-539. doi: 10.1002/1098-108X(198909)8:5<533::AID-EAT2260080505>3.0.CO;2-O

31. Lowe, M. R. (1993). The effects of dieting on eating behavior: a three-factor model. *Psychol Bull, 114*(1), 100-121.

32. Lowe, M. R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., . . . Stice, E. (2006). Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite, 47*(1), 83-90. doi: 10.1016/j.appet.2006.03.160

33. Lowe, M. R., Davis, W., Lucks, D., Annunziato, R., & Butryn, M. (2006). Weight suppression predicts weight gain during inpatient treatment of bulimia nervosa. *Physiol Behav, 87*(3), 487-492. doi: 10.1016/j.physbeh.2005.11.011

34. Lowe, M. R., Thomas, J. G., Safer, D. L., & Butryn, M. L. (2007). The relationship of weight suppression and dietary restraint to binge eating in bulimia nervosa. *Int J Eat Disord, 40*(7), 640-644. doi: 10.1002/eat.20405

35. Macht, M. (2008). How emotions affect eating: a five-way model. *Appetite, 50*(1), 1-11. doi: 10.1016/j.appet.2007.07.002

36. Miyasaka, N., Yoshiuchi, K., Yamanaka, G., Sasaki, T., Kumano, H., & Kuboki, T. (2003). Relations among premorbid weight, referral weight, and psychological test scores for patients with anorexia nervosa. *Psychol Rep, 92*(1), 67-74. doi: 10.2466/pr0.2003.92.1.67

37. Peterson, C. B., Crosby, R. D., Wonderlich, S. A., Joiner, T., Crow, S. J., Mitchell, J. E., . . . le Grange, D. (2007). Psychometric properties of the eating disorder examination-questionnaire: factor structure and internal consistency. *Int J Eat Disord, 40*(4), 386-389. doi: 10.1002/eat.20373

38. Ramacciotti, A., Sorbello, M., Pazzagli, A., Vismara, L., Mancone, A., & Pallanti, S. (2001). Attachment processes in eating disorders. *Eat Weight Disord, 6*(3), 166-170.

39. Regier, D. A. (2007). Dimensional approaches to psychiatric classification: refining the research agenda for DSM-V: an introduction. *Int J Methods Psychiatr Res, 16 Suppl 1*, S1-5. doi: 10.1002/mpr.209

40. Roberts, M. E., Tchanturia, K., Stahl, D., Southgate, L., & Treasure, J. (2007). A systematic review and meta-analysis of set-shifting ability in eating disorders. *Psychol Med, 37*(8), 1075-1084. doi: 10.1017/s0033291707009877

41. Russell, G. (1979). Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychol Med, 9*(3), 429-448.

42. Sawyer, S. M., Whitelaw, M., Le Grange, D., Yeo, M., & Hughes, E. K. (2016). Physical and Psychological Morbidity in Adolescents With Atypical Anorexia Nervosa. *Pediatrics*. doi: 10.1542/peds.2015-4080

43. Shaw, J. A., Herzog, D. B., Clark, V. L., Berner, L. A., Eddy, K. T., Franko, D. L., & Lowe, M. R. (2012). Elevated pre-morbid weights in bulimic individuals are usually surpassed post-morbidly: implications for perpetuation of the disorder. *Int J Eat Disord, 45*(4), 512-523. doi: 10.1002/eat.20985

44. Shinder, S. E., & Shephard, R. J. (1993). Relationship of premorbid mass and energy intake to increase of body mass during the treatment of anorexia nervosa. *International Journal of Eating Disorders, 14*(1), 65-73. doi: 10.1002/1098-108X(199307)14:1<65::AID-EAT2260140109>3.0.CO;2-R

45. Smink, F. R., van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: incidence, prevalence and mortality rates. *Curr Psychiatry Rep, 14*(4), 406-414. doi: 10.1007/s11920-012-0282-y

46. Stein, R. I., Kenardy, J., Wiseman, C. V., Dounchis, J. Z., Arnow, B. A., & Wilfley, D. E. (2007). What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. *International Journal of Eating Disorders, 40*(3), 195-203. doi: 10.1002/eat.20352

47. Steinhausen, H. C., Grigoroiu-Serbanescu, M., Boyadjieva, S., Neumarker, K. J., & Metzke, C. W. (2009). The relevance of body weight in the medium-term to long-term course of adolescent anorexia nervosa. Findings from a multisite study. *Int J Eat Disord, 42*(1), 19-25. doi: 10.1002/eat.20577

48. Stice, E., Durant, S., Burger, K. S., & Schoeller, D. A. (2011). Weight suppression and risk of future increases in body mass: effects of suppressed resting metabolic rate and energy expenditure. *Am J Clin Nutr, 94*(1), 7-11. doi: 10.3945/ajcn.110.010025

49. Stice, E., South, K., & Shaw, H. (2012). Future directions in etiologic, prevention, and treatment research for eating disorders. *J Clin Child Adolesc Psychol, 41*(6), 845-855. doi: 10.1080/15374416.2012.728156

50. Swenne, I. (2001). Changes in body weight and body mass index (BMI) in teenage girls prior to the onset and diagnosis of an eating disorder. *Acta Paediatr, 90*(6), 677-681.

51. Tozzi, F., Thornton, L. M., Klump, K. L., Fichter, M. M., Halmi, K. A., Kaplan, A. S., . . . Kaye, W. H. (2005). Symptom fluctuation in eating disorders: correlates of diagnostic crossover. *Am J Psychiatry, 162*(4), 732-740. doi: 10.1176/appi.ajp.162.4.732

52. Vervaet, M., van Heeringen, C., & Audenaert, K. (2004). Personality-related characteristics in restricting versus binging and purging eating disordered patients. *Compr Psychiatry, 45*(1), 37-43. doi: 10.1016/j.comppsych.2003.09.008

53. Villarejo, C., Fernández-Aranda, F., Jiménez-Murcia, S., Peñas-Lledó, E., Granero, R., Penelo, E., . . . Menchón, J. M. (2012). Lifetime Obesity in Patients with Eating Disorders: Increasing Prevalence, Clinical and Personality Correlates. *European Eating Disorders Review, 20*(3), 250-254. doi: 10.1002/erv.2166

54. Wade, T. D., & Tiggemann, M. (2013). The role of perfectionism in body dissatisfaction. *Journal of Eating Disorders, 1*(1), 1-6. doi: 10.1186/2050-2974-1-2

55. Wildes, J. E., & Marcus, M. D. (2012). Weight suppression as a predictor of weight gain and response to intensive behavioral treatment in patients with anorexia nervosa. *Behav Res Ther, 50*(4), 266-274. doi: 10.1016/j.brat.2012.02.006

56. Williams, G. J., Power, K. G., Millar, H. R., Freeman, C. P., Yellowlees, A., Dowds, T., . . . Jackson, M. A. (1993). Comparison of eating disorders and other dietary/weight groups on measures of perceived control, assertiveness, self-esteem, and self-directed hostility. *Int J Eat Disord, 14*(1), 27-32.

57. Witt, A. A., Berkowitz, S. A., Gillberg, C., Lowe, M. R., Rastam, M., & Wentz, E. (2014). Weight suppression and body mass index interact to predict long-term weight outcomes in adolescent-onset anorexia nervosa. *J Consult Clin Psychol, 82*(6), 1207-1211. doi: 10.1037/a0037484

58. Wojciechowska, J., Garland, B. H., & Hergenroeder, A. (2013). 46. Is Premorbid Obesity Before the Development of Anorexia Nervosa or Eating Disorder NOS Associated With a Higher Occurrence of Anxiety, Depression or Binging Behavior? *Journal of Adolescent Health, 52*(2), S43. doi: 10.1016/j.jadohealth.2012.10.102

59. Wu, M., Giel, K. E., Skunde, M., Schag, K., Rudofsky, G., de Zwaan, M., . . . Friederich, H. C. (2013). Inhibitory control and decision making under risk in bulimia nervosa and binge-eating disorder. *Int J Eat Disord, 46*(7), 721-728. doi: 10.1002/eat.22143

60. Zunker, C., Crosby, R. D., Mitchell, J. E., Wonderlich, S. A., Peterson, C. B., & Crow, S. J. (2011). Weight suppression as a predictor variable in treatment trials of bulimia nervosa and binge eating disorder. *Int J Eat Disord, 44*(8), 727-730. doi: 10.1002/eat.20859

Appendix A: Self-Report Measures

EDE-Q5.2 Page 1

EATING QUESTIONNAIRE

EDE-Q5.2 Page 2

Questions 13-18: Please fill in the appropriate number in the boxes on the right. Remember that the questions only refer to the past four weeks (28 days).

Over the past four weeks (28 days) .....

13 Over the past 28 days, how many times have you eaten what other people would regard as an unusually large amount of food (given the circumstances)?	.....
14 ..... On how many of these times did you have a sense of having lost control over your eating (at the time that you were eating)?	.....
15 Over the past 28 days, on how many **DAYS** have such episodes of overeating occurred (i.e., you have eaten an unusually large amount of food and have had a sense of loss of control at the time)?	.....
16 Over the past 28 days, how many times have you made yourself sick (vomit) as a means of controlling your shape or weight?	.....
17 Over the past 28 days, how many times have you taken laxatives as a means of controlling your shape or weight?	.....
18 Over the past 28 days, how many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape or amount of fat, or to burn off calories?	.....

Questions 19 to 21: Please circle the appropriate number. Please note that for these questions the term "binge eating" means eating what others would regard as an unusually large amount of food for the circumstances, accompanied by a sense of having lost control over eating.

19 Over the past 28 days, on how many days have you eaten in secret (ie, furtively)? ..... Do not count episodes of binge eating	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
	0	1	2	3	4	5	6
20 On what proportion of the times that you have eaten have you felt guilty (felt that you've done wrong) because of its effect on your shape or weight? ..... Do not count episodes of binge eating	None of the times	A few of the times	Less than half	Half of the times	More than half	Most of the time	Every time
	0	1	2	3	4	5	6
21 Over the past 28 days, how concerned have you been about other people seeing you eat? ..... Do not count episodes of binge eating	Not at all		Slightly		Moderately		Markedly
	0	1	2	3	4	5	6

Questions 22 to 28: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days).

	Over the past 28 days	**Not at all**		**Slightly**		**Moderate -ly**		**Markedly**
22	Has your weight influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
23	Has your shape influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
24	How much would it have upset you if you had been asked to weigh yourself once a week (no more, or less, often) for the next four weeks?	0	1	2	3	4	5	6
25	How dissatisfied have you been with your weight?	0	1	2	3	4	5	6
26	How dissatisfied have you been with your shape?	0	1	2	3	4	5	6
27	How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a shop window reflection, while undressing or taking a bath or shower)?	0	1	2	3	4	5	6
28	How uncomfortable have you felt about others seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)?	0	1	2	3	4	5	6

What is your weight at present? (Please give your best estimate.) .....

What is your height? (Please give your best estimate.) .....

If female: Over the past three-to-four months have you missed any menstrual periods? .....

If so, how many? .....
Have you been taking the "pill"? .....

THANK YOU

Appendix B: Tables

Table 1. Guidelines for Entering Growth Chart Data.

1. When growth chart data include both points on a graph and numerical values representing weight, height or BMI at a given time-point, enter exact numerical values.

2. When growth chart data only include points on a graph, adhere to the following guidelines when deciding what numerical values the points represent:
    a. Round age to nearest quarter year (e.g., 15, 15.25, 15.5, 15.75, or 16).
    b. Round height to nearest whole centimeter.
    c. Round weight to nearest whole kilogram.
    d. Round BMI values to nearest tenth (e.g., 18.1, 18.2, 18.3)

2a) When nearest datapoint to round to is unclear or ambiguous:
    a) if dot is between two lines and touches or crosses one line but not the other, choose the value of the line it touches/crosses.
    b) if dot is in between two lines and touches or crosses **both** lines, determine what line the greater portion of the dot touches or crosses, and choose the value of that line.
    c) if dot is between two points and does not touch or cross either line, first round up to nearest quarter year, whole centimeter, whole kilogram, or tenth of a BMI unit.
        e. Note! If dot is not touching line, it is unlikely that the line's value was meant by the physician. Therefore, if rounding up means you will be entering the line's value, round down.

2b) When a number of datapoints are graphed for a given age, first enter data according to the following guidelines and then make note of the participant for later discussion with study team:
    a) Select height and weight datapoints that were recorded nearest to every quarter year (if multiple dots near quarter year, pick the dot **closest**).
    b) If datapoints corresponding to quarter years are too difficult to determine (e.g., too many datapoints, too close to one another), select height and weight datapoints that were recorded nearest to every **half** year
    c) If datapoints corresponding to half years are too difficult to determine, select height and weight datapoints that were recorded nearest to every **whole** year.

Table 2. Correlations Between Premorbid and Current Weight Variables.

Variable	Mean (SD)	N	Highest premorbid z-BMI	Trajectory to highest premorbid z-BMI	Admission BMI	Admission WS
Highest premorbid z-BMI	.66 (.96)	41	--			
Trajectory to highest premorbid z-BMI	.31 (.45)	27	-.226	--		
Admission BMI	19.74 (6.70)	41	.570**	-.235	--	
Admission WS	18.70 (6.21)	34	-.044	-.244	-.265	--

**$p < .001$

Table 3. EDE-Q Subscale Scores.

Subscale	***Mean***	***SD***	***Range***
Eating Concerns	3.80	1.24	.60-5.80
Weight Concerns	4.29	1.53	.20-6.00
Shape Concerns	4.90	1.33	.88-6.00
Restraint	3.67	1.66	.00-6.00

Table 4. Highest Premorbid z-BMI Parameter Estimates Predicting EDE Subscale Scores.

	Eating Concerns				Weight Concerns				Shape Concerns				Restraint			
	B	*SE(B)*	r^2	*p*	*B*	*SE(B)*	r^2	*P*	*B*	*SE(B)*	r^2	*p*	*B*	*SE(B)*	r^2	*P*
Intercept	3.52	.23		<.001	4.19	.29	--	<.001	4.79	.26		--	3.86	.32		<.001
Highest premorbid z-BMI	.43	.20	.107	.037	.15	.26	.01	.56	.163	.22	.01	.47	-.29	.27	.03	.30

Table 5. Parameter Estimates Predicting EDE-Q Eating Concern Scores Using Highest Premorbid z-BMI and Covariates.

	Eating Concerns			
	B	*SE(B)*	r^2	*p*
Intercept	2.49	.68		.001
Highest premorbid z-BMI	.13	.24	.01	.58
Age at highest premorbid z-BMI	-.02	.05	.003	..68
BMI at admission	.07	.04	.09	.046

Table 6. Parameter Estimates Predicting EDE-Q Subscale Scores Using Interaction between Highest Premorbid z-BMI and BMI at Admission. (a) Without Controlling for Age at Highest Premorbid z-BMI. (b) While Controlling for Age at Highest Premorbid z-BMI.

Table 6(a)

Variable	Eating Concerns				Weight Concerns				Shape Concerns				Restraint			
	B	*SE*	r^2	*p*	*B*	*SE*	r^2	*p*	*B*	*SE*	r^2	*p*	*B*	*SE*	r^2	*P*
Intercept	4.04	.22		<.001	4.62	.27		<.001	5.14	.26		<.001	3.90	.34		<.001
Highest premorbid z-BMI	.08	.23	.002	.74	-.45	.28	.05	.116	-.17	.26	.02	.533	-.28	.34	.02	.42
BMI at admission	.16	.06	.14	.01	.07	.07	.23	.002	.15	.07	.11	.04	.06	.09	.01	.51
Highest premorbid x BMI admission	-.07	.04	.06	.08	-.09	.05	.09	.056	-.07	.05	.05	.14	-.06	.06	.03	.30

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Table 6(b)

Variable	Eating Concerns				Weight Concerns				Shape Concerns				Restraint			
	B	*SE*	r^2	*p*	*B*	*SE*	r^2	*p*	*B*	*SE*	r^2	*P*	*B*	*SE*	r^2	*P*
Intercept	4.04	.23		<.001	4.60	.26		<.001	5.13	.26		<.001	3.883	.34		<.001
Highest premorbid z-BMI	.06	.24	.001	.80	-.56	.28	.07	.05	-.21	.27	.01	.46	-.366	.35	.03	.30
BMI at admission	.16	.06	.14	.01	.26	.07	.25	.001	.15	.07	.12	.03	.07	.09	.02	.43

Table 6(b) continued

Age at highest premorbid z-BMI	-.02	.05	.002	.744	-.11	.06	.06	.08	-.04	.06	.01	.52	-.08	.08	.03	.28
Highest premorbid x BMI admission	-.07	.04	.06	.088	-.09	.05	.07	.06	-.07	.05	.05	.16	-.06	.06	.03	.32

Table 7. Mean Weight Values Across Groups.

	Low-Low[1]	**Low-High**[2]	**High-Low**[3]	**High-High**[4]
BMI admission	14.93	16.03	19.27	25.06
Highest premorbid z-BMI	-0.19	1.16	-0.09	1.55

[1]Low-Low = low admission BMI and lower highest premorbid z-BMI
[2]Low-High = low admission BMI and higher highest premorbid z-BMI
[3]High-Low = high admission BMI and lower highest premorbid z-BMI
[4]High-High = high admission BMI and higher highest premorbid z-BMI

Appendix C: Figures

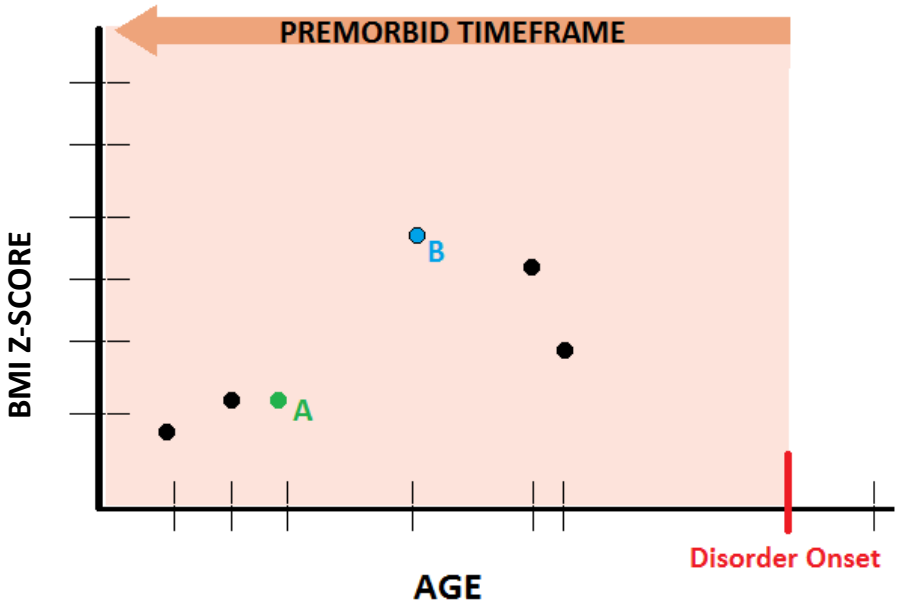

Figure 1. Sample Growth Chart Data. Premorbid BMI measurements calculated as: 1) Highest premorbid z-BMI **(B)**; 2) Trajectory to highest premorbid z-BMI **(slope of point A to point B).** For initial analyses, regressions testing hypothesis 1 included measurements 1 + 2 as predictors.

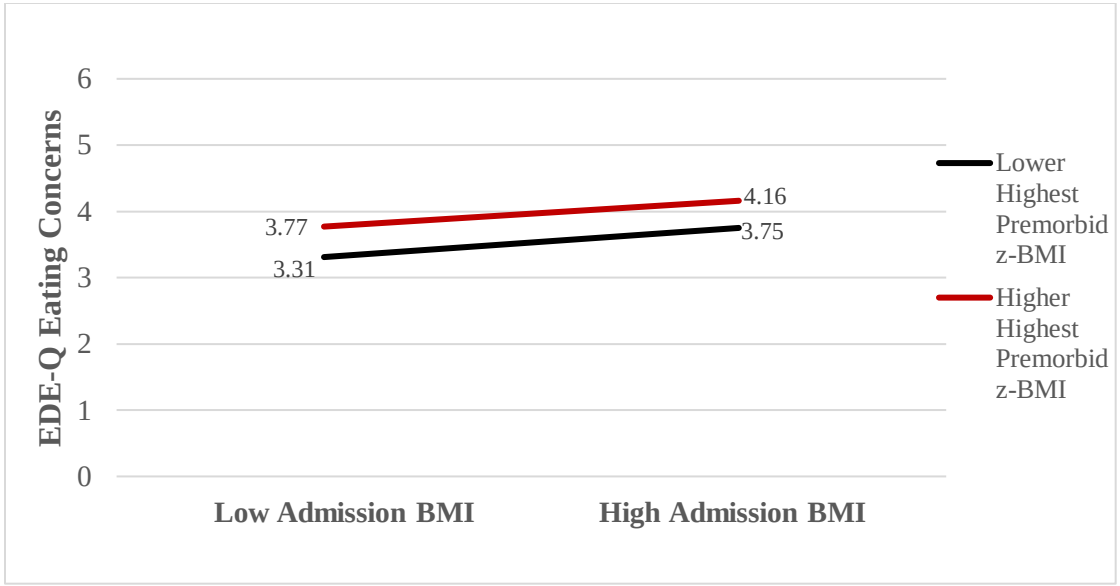

Figure 2. EDE-Q Eating Concerns Predicted by Highest Premorbid z-BMI x BMI at Admission.

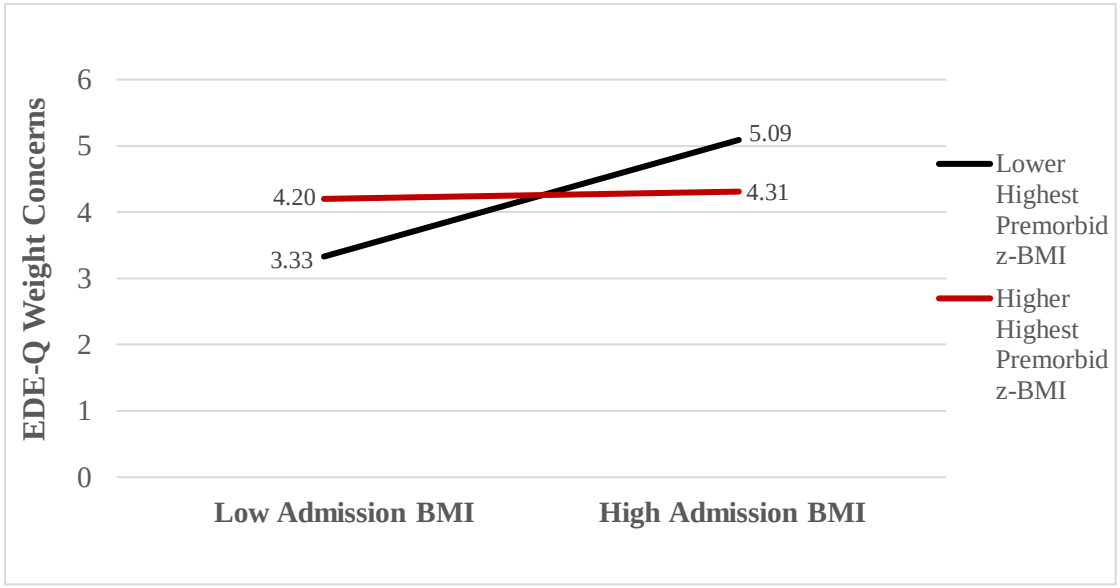

Figure 3. EDE-Q Weight Concerns Predicted by Highest Premorbid z-BMI x BMI at Admission.