APPROVAL SHEET

Title of Dissertation: Predictors and outcomes of different growth curve trajectories

of weight gain in hospital based treatment for anorexia nervosa

Name of Candidate: Saniha Hassan Makhzoumi Doctor of Philosophy, 2016

Dissertation and Abstract Approved:

Steven C. Pitts, Ph.D. Associate Professor Department of Psychology

Date Approved: April 22, 2016

Abstract

Anorexia Nervosa (AN) is primarily characterized by extremely low body weight and can result in medical, psychological, and social complications. Weight restoration, or returning to a medically normal weight, is a primary goal of treatment and is associated with positive outcomes. Though treatment programs strive for fast and consistent weight gain to increase the likelihood of achieving weight restoration prior to discharge, research using growth curve trajectories indicates there is variation in weight gain during inpatient treatment. Further, trajectories of weight gain may be predictive of long-term outcomes. However, research on the heterogeneity of weight gain during treatment as well as precursors and outcomes of differing trajectories is limited, and distinct patterns of weight gain are not clear. This study aimed to identify subgroups of patients with differing patterns of weight gain during hospital-based treatment of AN using mixture modeling, and identify predictors and treatment outcomes of estimated trajectory groups.

Participants were female inpatients with AN or subthreshold AN (N = 219) who were admitted to the Johns Hopkins Eating Disorders Program between 2003 and 2012. Participants completed self-report measures, including eating disorder behaviors and psychosocial constructs, at admission and a subset (n = 123) were measured again six-months after treatment discharge. Clinical outcome variables were assessed via chart review following discharge.

Three distinct patterns of weight gain were found; a negative quadratic trajectory (Optimal group), a negative quadratic trajectory with a faster initial rate of weight gain (Accelerated group), and a positive linear trajectory with slower rate of

weight gain (Sub-optimal group). Previous hospitalization, highest lifetime BMI, behavioral subtype, regularly restricting, bingeing, vomiting, and using laxatives at admission, and extraversion were predictors of group membership. Group membership predicted BMI and weight restoration at both discharge and follow-up, and regularly bingeing, and drive for thinness at follow-up. The Accelerated group emerged as the most distinct relative to the Optimal and Sub-optimal groups at admission, discharge, and follow-up whereas the Optimal and Sub-optimal groups appeared relatively similar. The Accelerated group was associated with increased disorder severity at admission and was primarily composed of women with bingeing and purging behaviors. Further, following treatment, the Accelerated group was associated with more positive weight outcomes; however they were also more likely to return to pre-treatment behaviors and had the least amount of improvement on psychological aspects of AN. Results suggest variation in weight gain with certain patterns associated with increased illness severity and risk of relapse. Further, results highlight the importance of psychological factors when defining recovery in addition to weight markers.

PREDICTORS AND OUTCOMES OF DIFFERENT GROWTH CURVE TRAJECTORIES OF WEIGHT GAIN IN HOSPITAL-BASED TREATMENT FOR ANOREXIA NERVOSA

Saniha Hassan Makhzoumi, M.A.

Dissertation proposal submitted to the Faculty of the Graduate School of the University Of Maryland, Baltimore County in partial fulfillment of the requirements for the degree of Doctor of Philosophy 2016

© Copyright by Saniha Hassan Makhzoumi 2016

Dedication

This dissertation is undoubtedly dedicated to my constant and unconditional sources of support, encouragement, guidance, and love: my family.

To my parents, who have been there for me from the beginning and who believed in me when I didn't believe in myself. Papa, thank you for teaching me one of the most important and pivotal lessons in my career and life: "Hold tight to your heart's desires, never ever let it go. Let nobody fool you into giving it up too soon." Thank you for showing me the joys of patient care and for being the healthcare provider I strive to be like everyday. Mama, although I always knew how much you sacrificed, it wasn't until the completion of this dissertation that I truly appreciated just how much you gave up so that we could achieve our dreams. And that dream is to be just like you. Thank you for being my Ph.D. role model. Thank you for always listening, supporting, and challenging me. Thank you for being my voice and confidence when mine were wavering.

To my bristers, Hamudi and Zaineb: you both have been right by my side cheering me on through every milestone, hurdle, win, and loss. Thank you for being my loudest fans, quietest critics, strongest protectors, and perfect role models. Seeing you both develop into intelligent, caring, and incredibly successful adults has helped shape my dreams and goals. Thank you for paving the way, showing me the benefits of hard work, and for always believing in me even when I was full of doubt. I hope to make you both half as proud of me as I am of you.

And to Ayman, Katie, and Ameena, thank you for the love and support you have given me during this dissertation process.

Without all of you, none of this would be possible. Your faith and encouragement is what allowed me to pursue this doctorate and I am eternally grateful for all you have done to support me.

Acknowledgements

I would like to express my sincere gratitude to the many people who provided me with invaluable support over the course of my graduate career. First and foremost, I would like to thank my advisor and mentor, Dr. Steven Pitts, for his incredible guidance throughout graduate school. His knowledge, encouragement, and enthusiasm have not only inspired and fostered my research, but have been crucial to my intellectual and professional development. Thank you for your patience, endless support, and most of all your confidence in me. I am so grateful to have joined your lab and thankful to call you my mentor.

I would like to thank my committee members, Dr. Robin Barry, Dr. Janelle Coughlin, Dr. Jason Schiffman, and Dr. Shuyan Sun, who were more than generous with their expertise, instrumental advice, and time as I developed and refined my program of research. Thank you for your feedback, assistance, and support. I would also like to thank the UMBC faculty for providing me with amazing educational, research, and clinical opportunities, and for all that they taught.

I would like to thank the Johns Hopkins Eating Disorders Team who has played a large and critical role in my eating disorders training. Dr. Janelle Coughlin, Dr. Angela Guarda, and Dr. Graham Redgrave took me under their wings, always found time in their busy schedules to answer my many questions, and exposed me to the incredible world of eating disorders research. Thank you for pushing and encouraging me to continually expand my knowledge base and develop new skills. I want to thank Dr. Colleen Schreyer for her unwavering support and mentorship throughout this process. I cannot express how much your friendship and belief in me has meant. This dissertation would not have been possible without the support and guidance of the entire team. I am eternally grateful to have been a part of this team and feel lucky to be able to call you all advisors, mentors, and most importantly friends.

I would like to thank Dr. Harry Brandt who was my first psychology mentor, and who introduced me to the clinical world of eating disorders and thus started me on this journey. I would also like to thank Dr. Laura Mufson, my advisor from Columbia University, who first introduced me to research and showed me the powerful impact it could have on patient care. Thank you both for inspiring and encouraging me to pursue a doctorate in clinical psychology.

I would like to especially thank my GW family, Linda, Anyah, Farbish, Ross, Heidi, and Bardo for being unending sources of support and encouragement over the years. From GW to Stanford, thank you for your supportive shoulders, listening ears, and understanding of my busy schedule. I would also like to thank my UMBC family, especially my Core Four, for their moral support, encouragement, and most importantly friendship.

Table of	Contents
----------	----------

Dedication	ii
Acknowledgements	iii
Table of Contents	iv
List of Tables	vi
List of Figures	vii
Overview	1
Chapter 1: Introduction	5
Diagnostic Criteria, Background, and Prevalence	5
Treatment	8
Treatment Outcomes	11
Role of Ambivalence in Treatment	14
Weight Restoration	15
Weight Gain Rate	18
Prognostic Significance of Weight Gain Trajectories	19
Predictors of Variance in Weight Gain Parameters	23
Current Study	25
Proposed Hypotheses	
Research Ouestions	
Chapter 2: Method	
Participants	
Procedure	
Measures	35
Demographic Variables	35
Clinical Outcome Variables	35
Eating Disorder Behaviors	36
Fating Disorder Symptomatology	36
Eating Disorder Bymptomatology	36
Personality	36
Weight Restoration	36
Flansed Time	
Chapter 3: Results	38
Aim 1: Mixture Modeling	38
Aim 2: Predictors of Trajectory Group Membership	
Research Question 1	
Aim 3: Sequelse of Trajectory Group Membership	02
Research Question 2	
Chapter 4: Discussion	02
Dattern of Weight Coin	25 رن
Predictors of Weight Gain Pattern	
Program Effects	
Flogran Energy	90

Outcomes of Weight Gain Patterns	
Limitations	<u> </u>
Future Directions	

References103

Appendix A: Additional Literature on Optimal Weight Gain Rate	
Appendix B: Self-Report Measures	
Eating Disorder Behaviors Questionnaire	
Eating Disorder Inventory (EDI-2)	
Eating Disorder Recovery Self-Efficacy Questionnaire (EDRSQ)	

List of Tables

Table 1: Clinical Variables at Admission, Discharge, and Six-months After Treatment	
Discharge	.33
Table 2: Results from Modeling Using Weight Gained (pounds) as Growth Variable	10
	.40
Table 3: Percentage of Participants with Probability of Primary Group Membership	
Greater than .90	.44
Table 4: Descriptives for Modeling Groups Based on Weight Gained in Pounds	.48
Table 5: Group Means for Excluded Participants and the Analysis Sample on	
Continuous Variables	.57
Table 6: Group Proportions for Excluded Participants and the Analysis Sample on	
Categorical Variables	.58
Table 7: Uncontrolled Multinomial Logistic Regressions Estimating Overall Bivariate	
Relations between Group Membership and Predictors	.60
Table 8: Pairwise Comparisons for Predictors with Significant Overall Bivariate	
Relations from Listwise Deletion	.62
Table 9: Multiple Multinomial Logistic Regression between Group Membership and	
Significant Predictors	.64
Table 10: Pairwise Comparisons for Significant Predictors from the Multiple	
Multinomial Logistic Regression	.65
Table 11: Correlations among Significant Predictors from Logistic Regression	.66
Table 12: Group Means for Excluded Participants and Analysis Sample on	
Continuous Variables	.72
Table 13: Group Proportions for Excluded Participants and the Analysis Sample on	
Categorical Variables	.73
Table 14: Overall Effects of Treatment	.75
Table 15: Table of Means and ANOVA Results Comparing Groups on Outcome	
Variables	.77
Table 16: Logistic Regressions Estimating Overall Relation between Group	
Membership and Outcomes	.78
Table 17: Pairwise Comparisons for Outcomes with Significant or Trending Bivariate	
Relations	.79
Table 18: Results for Covariates from ANCOVA and Logistic Regressions	.81
Table 19: Table of Means and ANCOVA Results Comparing Groups on Outcome	
Variables	.83
Table 20: Multiple Logistic Regression Predicting Outcomes from Group	
Membership Controlling for Covariates	.84
Table 21: Unique Effects of Predictors with Significant Unique Relations	.85

List of Figures

Figure 1: Predicted growth curve trajectories of weight gain	26
Figure 2: Growth curve trajectories of weight gain in pounds while on weight gain	
protocol	46
Figure 3: Simple effects of group on six-month BMI separately for participation in	
day hospital	87
Figure 4: Simple effects of group on six-month drive for thinness separately for	
participation in day hospital	88
Figure 5: Simple effects of group on six-month normative eating self-efficacy	
separately for participation in day hospital	88

Predictors and outcomes of different growth curve trajectories of weight gain in hospital-

based treatment for anorexia nervosa

Anorexia Nervosa (AN) is a complex mental disorder involving extremely low body weight resulting from disordered eating behaviors, such as restricting food intake and/or purging (Diagnostic and Statistic Manual of Mental Disorders, Fifth Edition, 2013). These risky eating behaviors are driven, at least in part, by high levels of body dissatisfaction and drive for thinness (Cash, 2002; Garner, 2004). Individuals with AN often develop physical, medical, and psychosocial complications from the disorder, and AN has a documented 20-year mortality rate of up to 20% (Ratnasuriya, Eisler, Szmukler & Russell, 1991; Thompson, 2011). In spite of AN's high mortality rate and numerous side effects, individuals with AN often have high ambivalence and resistance toward treatment and behavior change (Guarda & Coughlin, 2009). This contributes to the disorder's high relapse rate of up to 63% (McFarlane, Olmsted, & Trottier, 2008; Pike, 1998).

Overview

Because low body weight is a hallmark of AN and one of the main contributors of the disorder's life-threatening medical complications, weight restoration (returning to a medically normal weight) is a primary goal of treatment. Full weight restoration at program discharge has been shown to positively impact all areas of long-term recovery, such as reduced likelihood of readmissions, increased likelihood of maintaining a normal weight, and engaging in fewer eating disorder (ED) behaviors at follow-up (Accurso, Ciao, Fitzsimmons-Craft, & Lock, 2014; Haynos, Roberto, Martinez, Attia, & Fruzzetti, 2014; Rigaud, Pennacchio, Bizeul, Reveillard, & Verges, 2011). In addition, weight

restoration is often used as a marker of readiness to progress to a lower level of treatment (Accurso et al., 2014; Haynos et al., 2014). Given the chronicity and severity of AN, as well as the large amount of weight needed to be gained to achieve weight restoration in many cases, prolonged treatment stays are required. However, due to various obstacles such as lack of insurance coverage; ambivalence toward treatment, behavior change, and weight gain; and high treatment drop-out, length of treatment stays are relatively short. In the past 25 years, average length of stay for AN treatment has reduced from approximately 140 to 40 days (Willer, Thuras & Crow, 2005). Although weight restoration prior to discharge is associated with better outcomes, as a result of reduced treatment length, patients are much less likely to achieve it (Rigaud et al., 2011).

One way to increase the likelihood of achieving weight restoration prior to treatment discharge is to increase the rate at which patients gain weight while in treatment. Though there is debate in the literature as per the optimal rate of weight gain, relatively faster rates have been shown to be more effective at weight restoration and are associated with better outcomes than slower rates (Keel, Dorer, Franko, Jackson, & Herzog, 2005; Lund et al., 2009). Researchers and clinicians in the field recommend weight gain rates of two to four pounds (0.91 to 1.81 kilograms) per week (Woodside, 2002).

Despite striving for faster weight gain rates, variation in weight gain during inpatient and partial hospital treatment is observed across both programs and patients. Three main variables shown to impact weight gain during treatment are behavioral subtype, level of ED pathology, and illness severity. Behavioral subtype (restricting vs. purging) has been shown to be a factor in weight gain rate such that individuals with AN

restricting subtype (AN-R) tend to gain weight slower than AN purging subtype (AN-P) (Neuberger, Rao, Weltzin, Greeno, & Kaye, 1994). ED pathology, including body dissatisfaction and drive for thinness, impacts weight gain rate during treatment such that those with higher levels of pathology gain weight slower (Garner, 2004; Thompson, 2004). Finally, increased illness severity as measured by lower admission BMI, longer duration of illness, and older age, is associated with slower rates of weight gain (Fairburn & Brownell, 2002; Thompson, 2011).

Preliminary research suggests that variation in weight gain during AN treatment may be predictive of long-term outcomes (Accurso et al., 2014; Le Grange, Accurso, Lock, Agras, & Bryson, 2014; Vansteelandt, Pieters, Vanderlinden, & Probst, 2010). However, the majority of research on the effects of weight gain on treatment outcomes relies on simplistic measures, such as overall weight gained or average rate of weight gain while in treatment, which overlook individual differences in, and clinical course of, weight gain. One method of investigating individual differences in weight gain that may be of practical value is through mixture modeling. Mixture modeling is an analytical technique that allows for estimation of individual growth curve trajectories while simultaneously classing (grouping) individuals with similar trajectories; this allows for the identification of subgroups with similar growth trajectories.

Recent research on trajectories of weight gain suggests pattern of weight gain is predictive of treatment outcomes (Hartmann et al., 2007; Lay et al., 2004). However, studies have been limited by small sample size, lack of long-term follow-up, and failure to include psychosocial measures. Further, it is not clear which factors contribute to variation in weight gain and how those differences in weight gain impact long-term

outcomes (Hartmann et al., 2007; Lay et al., 2004; Le Grange et al., 2014). Weight gain trajectories, though research is limited, have the potential to provide valuable information about short and long-term outcomes of AN treatment (Hartmann, Wirth, & Zeeck, 2007; Lay, Jennen-Steinmetz, Reinhard, & Schmidt, 2004; Le Grange et al., 2014). Pattern of weight gain may have the potential to serve as an in-treatment estimate of treatment response, compliance, and risk of relapse. This would allow clinicians to identify patients with increased risk of relapse and address the increased risk prior to discharge, which may help reduce AN's alarmingly high relapse rate and increase patients' chances at recovery.

This study aimed to estimate weight gain trajectories and identify subgroups of individuals who differ in their pattern of weight gain in an inpatient hospital sample using mixture modeling. Based on observed trajectories, admission level variables were used to predict group membership to identify which types of patients were more likely to follow each trajectory. Finally, the relation between weight gain trajectory group and outcomes (at treatment discharge and six-months after discharge) were explored to see which trajectories were associated with positive and negative outcomes.

First, I will review relevant background information on AN, including diagnostic criterion, prevalence rates, and types of treatment. Then I will focus on treatment outcomes, mainly focusing on weight restoration as a treatment goal and outcome measure. The implications of achieving weight restoration prior to treatment discharge will be provided. I will then focus on weight gain parameters, such as rate of weight gain, that play an important role in weight restoration, mainly focusing on recent research investigating weight gain trajectories. I will then discuss the prognostic significance of

weight gain trajectories in short and long-term outcomes and provide predictors of variance in weight gain parameters. Finally, I will conclude by discussing the aims of the current study.

Diagnostic Criteria, Background, and Prevalence

Anorexia Nervosa (AN) is characterized by body weight less than minimally normal as a result of a restriction of energy intake (DSM, 2013). This diagnostic criterion typically requires an individual to weigh less than 85% of the lower bound of medically ideal weight. In addition, individuals with AN have an intense fear of gaining weight and / or engage in behaviors that prevent weight gain, such as restricting food intake. AN also requires one or more of the following symptoms: disturbance in the experience of body image or appearance, undue influence of body weight or shape on self-evaluation, and / or a persistent failure to recognize the severity of one's low body weight.

There are two subtypes of AN based on behaviors to restrict energy intake: restricting (AN-R) and purging (AN-P). Restricting behaviors include restricting food portions, eating meals low in fat and calories, and narrowing the food repertoire. Purging behaviors include self-induced vomiting, diet pill use, laxative use, diuretic use, enemas, and ipecac. Additional behaviors often observed in association with AN include bingeing, chewing and spitting, excessive exercise, and rumination. Subthreshold AN (EDNOS AN) is when all criteria for AN are met except that, despite significant weight loss, the individual's weight is within or above the normal range. In addition to behavioral symptoms, AN is associated with physical and cognitive symptoms (Maguire et al., 2008). Most typically observed physical symptoms include delayed gastric emptying and early satiety, and cognitive symptoms include body dissatisfaction and drive for thinness (Garner, 2004). The behavioral, cognitive, and physical symptoms of AN are interrelated and contribute to the persistence and perpetuating nature of this disorder.

Body dissatisfaction has been shown to be a risk factor for various eating related negative outcomes, such as disordered eating, eating disorders, and obesity, and is a motivating factor in ED symptoms (Cash, 2002; Stice & Shaw, 2002; Thompson, 2011). Body dissatisfaction is defined as a negative, subjective evaluation of one's overall weight and shape (Gardner, 2004; Stice & Shaw, 2002). Body dissatisfaction often arises from a discrepancy between an individual's perceived current and ideal weight and shape, which are often based on sociocultural norms, attitudes, and beliefs (Gardner, 2004). In addition, body dissatisfaction stems from an increased internalization of sociocultural pressures to be thin, often referred to as the "thin-ideal," coupled with perceived deviations from the thin-ideal (Heinberg et al., 2008; Thompson, 2011). Increased and persistent body dissatisfaction often predicts various disordered eating and negative health behaviors, such as extreme dieting, with the goal of achieving one's ideal body image. In addition to eating disorders, increased body dissatisfaction can result in decreased self-esteem and increased depression (Cash, 2002; Stice & Shaw, 2002; Thompson et al., 1999).

AN is often described as a motivated behavioral disorder where overvalued fear of fatness and drive for thinness result in disordered eating behaviors. Overvalued fear of fatness is defined as an irrational belief about, or morbid fear, of weight gain and becoming overweight (Morgan & Russell, 1975) and often results in drastic behaviors to avoid weight gain (Cash, Counts, & Huffine, 1990). Fear of fatness is a strong motivator in, and diagnostic criteria of, AN (DSM, 2013). Drive for thinness, defined as an

excessive concern with dieting and weight gain, preoccupation with weight/shape, and perseveration on achieving thinness, plays a central role in the development and maintenance of AN (Garner, 2004). Drive for thinness is often accompanied by motivated behaviors, such as restricting food intake and excessive exercise, with the goals of reducing body dissatisfaction, achieving the thin-ideal, and/or losing weight (Fox, 1990).

ED pathology, such as body dissatisfaction and drive for thinness, is often used as markers of illness severity in AN. However, illness severity can be defined in many ways. Many researchers use years of illness duration as a marker (Maguire et al., 2008) whereas others use cognitive measures of ED pathology, admission body mass index (BMI), and frequency of ED behaviors (Casanovas et al., 2007; McHugh, 2007; Maguire et al., 2008). Strengths and weaknesses of differing definitions of severity will be discussed below.

There are many medical complications associated with AN. As a result of starvation, individuals with AN can suffer from various gastrointestinal, cardiac, metabolic, and endocrine complications. The most common include heart failure, amenorrhea, kidney failure, osteoporosis, cognitive impairment, electrolyte imbalance, teeth erosion, esophageal irritation/tear, and death. Many of these medical complications can be reversed with normalization of eating and weight restoration, and do not require pharmacological interventions. In addition to medical side effects, AN can lead to various social and developmental complications, including interpersonal problems and social isolation, and academic and occupational impairments due to multiple hospitalizations and the physical side effects of starvation (Thompson, 2011).

Unfortunately, AN is the psychiatric disorder with the highest mortality rate, with 8-20% passing away within 20-years of treatment discharge (Keel et al., 2003;

Ratnasuriya et al., 1991; Thompson, 2011). Approximately 50% of the deaths in AN are due to suicide and the remaining are due to the medical complications of starvation, such as cardiac abnormalities (Arcelus, Mitchell, Wales, & Nielsen, 2011; Crow et al., 2009; Keel et el., 2003).

It is estimated that approximately 1% of females and 0.3% of males have AN (Bulik et al., 2006; Hoek, 2006; Hoek & van Hoeken, 2003). However, prevalence estimates tend to underestimate the true occurrence of AN due to difficulties in epidemiologic research (Keski-Rahkonen et al., 2007). Specifically, individuals with ED, particularly AN, have high rates of denial and illness concealment. Many individuals with ED do not seek treatment and as many epidemiologic studies gather information from medical records and psychiatric case registries, rates tend to underestimate the true prevalence (Currin, Schmidt, Treasure, & Jick, 2005; Lucas, Crowson, O'Fallon, & Melton, 1999)

Treatment

There are four types of treatment for AN that differ based on level of intensity. Outpatient and intensive outpatient treatment are the least intense and involve daily or weekly therapeutic individual or group therapy. More intensive types of treatment are partial, or day, hospitalization, and inpatient hospitalization, which are appropriate for more severe cases of AN as indicated by a dangerously low weight, being medically unstable, or of harm to oneself (Keel & McCormick, 2010). Partial hospital is an all-day treatment program which individuals typically attend seven days a week where they

participate in group therapy during the day and consume all meals with trained staff. Inpatient care also involves daily group therapy and monitored meals but requires 24hour care on a locked unit. Inpatient and partial hospital treatment provide greater focus on refeeding, nutritional rehabilitation, and medical monitoring than outpatient treatment (Attia & Walsh, 2009; Lock & Le Grange, 2013). Underweight patients unable to gain weight in a less restrictive level of care, such as outpatient, may require more intensive treatment. An intensive level of care is common in this population; approximately 50% of individuals with AN require inpatient or partial hospital treatment during the course of their illness (Agras et al., 2004). Typical partial hospital and inpatient treatment each last approximately 20 to 30 days, though length of stay varies depending on admission BMI, rate of weight gain, illness severity, and insurance coverage (Willer, Thuras & Crow, 2005). Treatment for eating disorders, in particular AN, can be very expensive and complex due to the disorder's chronic course and life-threatening complications (Kreipe & Dukarm, 1996; Lock & Le Grange, 2013). In addition, having a comorbid disorder is common in AN, particularly depression, anxiety, personality disorders, and/or subtstance use, which further complicates treatment (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Keel et al., 2005; Lock & Le Grange, 2013).

Inpatient and partial hospital treatment for AN usually requires a multidisciplinary approach and includes nutritional rehabilitation for underweight patients, psychosocial interventions, and medication (Grilo & Mitchell, 2010; Kaplan & Howlett, 2010; Mehler, Winkelman, Andersen, & Gaudiani, 2010). A large component of nutritional rehabilitation, which focuses on normalizing eating behaviors and weight restoration, is medical monitoring in which trained staff monitor and assess physical complications of refeeding. Monitoring includes assessing vital signs, electrolytes, and food/fluid intake and output as well as monitoring for edema, rapid weight gain, cardiac failure/arrhythmia, gastrointestinal complications, seizures, and hypophosphatemia. Psychosocial interventions, such as cognitive behavioral therapy, dialectical behavior therapy, and interpersonal therapy, all focus on changing maladaptive thoughts and behaviors related to AN. They are the most frequently used psychosocial interventions for AN treatment (Agras, 2010; Fairburn & Brownell, 2005). Medication, such as antidepressants and antianxiety, are often used to treat comorbid disorders to aid in treatment and recovery (Agras, 2010).

Goals of inpatient and partial hospital treatment for AN include restoring underweight patients to a medically healthy weight (weight restoration), normalizing eating patterns, enhancing patient's motivation to change their ED behaviors and cognitions, correcting maladaptive thoughts and feelings related to the ED, and preventing relapse (American Psychiatric Association, 2004). Individuals who enter treatment underweight are placed on a weight gain protocol, which includes high calorie diets and exercise restrictions, with a primary goal of weight restoration.

Treatment research in AN is difficult due to high dropout rates and resistance to seek treatment. Premature termination of treatment (dropout) is a well-documented and common occurrence in AN treatment with rates as high as 57% (Mewes, Tagay & Senf, 2008; Sly, Mountford, Morgan, & Lacey, 2013; Whisenhunt & Srikameswaran, 2004). Geller and colleagues (2004) found dropout to be related to increased illness severity, lower level of social support, and reduced readiness to change. As a result of treatment dropout and resistance, studies are left with small sample sizes that do not represent the

full population. Despite the limitations in treatment research, behavioral interventions focusing on weight restoration and normalizing eating behaviors are one of the most validated and accepted treatments for AN (Attia & Walsh, 2009). Research suggests that structured inpatient behavioral treatment is an effective treatment for AN in achieving weight restoration (Attia & Walsh, 2009).

Treatment Outcomes

It is important to recognize that specific definitions of treatment outcome vary greatly across studies. Generally, recovery and relapse are the two post-treatment outcomes of particular importance to treatment research. Recovery is typically with respect to both weight and psychological recovery and includes aspects of physical, psychological, and social functioning (Morgan & Hayward, 1988; Russell, Szmukler, Dare, & Eisler, 1987). Weight recovery is defined as an individual having restored and maintained his or her weight within medically normal levels; however, there is not a clear consensus within the field on the actual values. Weight recovery is one aspect of the Expanded Morgan-Russell Outcomes Criteria (Eckert, Halmi, Marchi, & Grove, 1995; Morgan & Russell, 1987), a common outcome measure that defines full weight recovery as "At least 90% of one's ideal body weight (IBW)," a good outcome as "Between 85% and 90% of one's IBW," a fair outcome or partial recovery as "Between 80 and 85% of IBW," and a poor outcome/relapse as "Less than 80% of IBW." Ideal body weight is defined as medically ideal body weight based on an individual's age and height according to the 1959 Metropolitan Life Insurance Tables (Morgan & Russell, 1987). It does vary, but most commonly a BMI (Body Mass Index) of 22 would be thought to be IBW for an eating disordered population. A second, somewhat more objective, measure of weight

restoration is BMI. BMI is calculated as weight (in kilograms) divided by height squared (in meters)¹. An additional advantage of BMI is the measure is continuous and offers quantitative criteria. Though there appears to be agreement that weight restoration requires a BMI of at least 17.5, many clinicians use higher BMI cutoffs to define recovery (Hartmann et al., 2007; Rigaud et al., 2011). Both weight restoration and weight gain receive additional attention below.

Psychological recovery involves ED pathology, such as drive for thinness and body dissatisfaction, as well as the quantity and frequency of ED behaviors engaged in. Individuals are typically described as recovered if they have low to absence of ED pathology and behaviors for a minimum of eight weeks (Eckert et al., 1995; Maguire et al., 2008; Pike, Walsh, Vitousek, Wilson, & Bauer, 2003; Strober, Freeman, & Morrell, 1997). Currently, there is little to no consensus on what *low* pathology and behaviors entails. Moreover, one may speculate that definitions of *low* and *reduced* ED pathology should be tailored to the individual. The formal operational definitions to define psychological recovery in this population can actually be quite disparate making it difficult, if even possible, to effectively compare treatments.

Relapse, in contrast to recovery, is generally defined as a return to full syndrome criteria after a period (typically less than 6 months) of minimal or absence of symptoms and diagnostic criteria (Field, Herzog, Keller, West, Nussbaum, & Colditz, 1997; Frank et al., 1991; Keel et al., 2005). Many researchers distinguish between relapse and recurrence. Recurrence, though similar to relapse in that both involve the reappearance of symptoms and diagnostic criteria, is distinct in that it involves at least a 6-month period

¹ This can easily be calculated using the US System by calculating weight in pounds

of recovery prior to the resurgence of symptoms. ED researchers tend to use relapse and recurrence interchangeably. Most researchers use the term "relapse" to indicate the presence of symptoms or diagnostic criteria following treatment and a period without symptoms; however I will distinguish between these terms when possible. With respect to weight gain, a relapse episode is most typically defined when an individual's BMI drops to less than criteria (e.g., 17.5) post-treatment. However, unless the patient is actually in partial or inpatient hospitals, much of the research studying relapse relies on individual self-report. Given bias in weight reporting observed within this population (Fairburn & Beglin, 1994; Grilo, Masheb, & Wilson, 2001), it is possible that relapse (and relapse rates) is under-reported.

Psychological relapse involves the presence of eating disordered behaviors and higher ED pathology than healthy controls (Eckert et al., 1995; Maguire et al., 2008; Pike et al., 2003). According to the expanded Morgan-Russell criteria (1995), psychological relapse is characterized by ED pathology, such as drive for thinness and body dissatisfaction, that is more than one standard deviation above healthy controls and / or engaging in ED behaviors. Outcomes research typically measures ED behaviors and diagnostic criteria through self-report; therefore as discussed above, the true rate of relapse related to cognitive and behavioral factors may be under-reported.

Unfortunately, relapse is a common outcome of treatment for AN with relapse rates ranging from 22 to 63% with up to 78% relapsing within one-year of treatment discharge (Lay et al., 2000; McFarlane et al., 2008; Pike, 1998). Across numerous studies, research suggests that approximately 45-59% maintain recovery, 33%

divided by the square of height in inches and multiplying this ratio by 703

demonstrate partial recovery, and 20% are chronically ill (Keel et al., 2003; Steinhausen, 2002; Steinhausen & Weber, 2009). In a nine-year longitudinal study, Keel and colleagues (2005) found that over the course of the study, only 31% (42 / 136) of individuals with AN ever achieved remission, defined as 8 consecutive weeks with minimal or no symptoms, as compared to the 75% (83 / 110) rate found in individuals with bulimia nervosa. Moreover, considering the 42 individuals with AN who achieved remission at one point, 36% of them reported a weight and psychological relapse within the nine year follow-up period. Longitudinal studies suggest less than half the patients treated for AN maintain recovery (Keel & McCormick, 2010; Pike, 1998); one study found that 17% of patients did not experience any aspect of recovery in a seven-year follow-up period (Herzog et al., 1999). Correlational predictors of relapse following treatment for AN that have been identified include: low BMI at both admission and discharge, younger age, longer duration of disorder, and higher ED attitudes at admission (Castro, Gila, Puig, Rodriguez, & Toro, 2004; Kee et al., 2005; Wiseman, Sunday, Klapper, Harris, & Halmi, 2001). Treatment compliance and personality characteristics have also been identified as predictors of treatment outcomes (Steiner, Mazer & Litt, 1990), with less compliance and higher psychopathology associated with poorer outcomes and relapse.

Role of Ambivalence in treatment

One factor that plays an integral role in the treatment of AN is patient ambivalence towards behavior change. Despite the risky eating behaviors, dangerously low weight, and numerous medical and psychosocial complications of the disorder, individuals with AN are often unwilling to acknowledge the need for treatment or change these behaviors. As such, they are ambivalent toward both behavior and psychological change regarding behaviors and attitudes they do not feel are problematic. Ambivalence towards treatment and behavior change stems from the disorder's ego-syntonic nature as typical ED behaviors and outcomes are reinforcing to the individual and become habitual and difficult to stop (Guarda & Coughlin, 2009). Ambivalence is so typical, particularly regarding AN, it is often thought of as a core feature of ED and, though not in the DSM, many researchers have argued that it should be a diagnostic criteria (Cockell, Geller, & Linden, 2002; Guarda, 2008; Guarda & Heinberg, 2003).

High levels of ambivalence and denial of illness often result in an avoidance of treatment, low motivation to change while in treatment, and relapse following treatment (Guarda & Coughlin, 2009). Ambivalence can also prevent individuals from engaging in treatment, leading to premature treatment termination, and impact treatment progress, such as rate of weight gain and weight restoration (Guarda & Coughlin, 2009). Due to the resistance and ambivalence to treatment, family members and clinicians often use physical/psychological coercion, such as involuntary commitment to treatment, as part of the treatment process.

Weight Restoration

Achieving weight restoration prior to treatment discharge is associated with positive outcomes, such as weight maintenance, improved ED pathology and comorbidities, and increased rate of recovery at both short and long-term follow-ups (Baran, Weltzin, & Kaye, 1995; Steinhausen, Grigoroiu-Serbanescu, Boyadjieva, Neumarker, & Metzke, 2008). Individuals who are discharged while still underweight tend to have a worse clinical course, including ongoing disordered eating behaviors,

increased risk of relapse, and increased psychiatric comorbidities (Accurso et al., 2014; Haynos et al., 2014). Although weight restoration is not the sole focus of treatment, due to the life-threatening complications of malnutrition as well as increased likelihood of recovery and positive outcomes, it should be a primary goal early in treatment. It is important to note that weight recovery tends to occur before psychological recovery (Coutrier & Lock, 2006); therefore it may be that psychological outcomes improve more slowly and current longitudinal studies may not follow participants long enough to observe changes in psychological functioning.

At its most simplistic, weight restoration is a dichotomous construct in which an individual either: 1) does or 2) does not achieve target weight. As individuals with AN are underweight by definition, weight restoration is a primary goal in treatment (APA, 2006; NICE, 2004). Although measures of treatment outcome are not limited to weight restoration, it remains one of the primary definitions of recovery (Coutrier & Lock, 2006). Treatment programs set specific target weights for patients that serve as an indicator of weight restoration and readiness to progress to the next stage of treatment (Accurso et al., 2014; Haynos et al., 2014). As discussed, though there is no consensus on exact BMI levels that correspond to weight restoration in AN, the minimum BMI appears to be 17.5 (Hartmann et al., 2007; Rigaud et al., 2011). This is likely due to the increasing severity of medical symptoms as BMI drops below this mark.

Research has examined the impact of achieving target weight in treatment on a number of clinical outcomes. Baran and colleagues (1995) found that in a sample of 22 women with AN, patients who were discharged while underweight were more likely to be rehospitalized, engage in unhealthy eating behaviors, and have ongoing mood

disturbances two-years after discharge compared to patients who were discharged after target weight had been achieved. Patients who achieve partial recovery per Morgan-Russell criteria (between 80 and 85% of IBW) are, relative to full recovery patients (at least 90% of IBW), at increased risk of relapse two-years after treatment (Accurso et al., 2014).

In addition to weight restoration, discharge BMI has been shown to be one of the strongest predictors of long-term outcome (Bean et al., 2004; Castro-Fornieles et al., 2007; Howard, Evans, Quintero-Howard, Bowers, & Andersen, 1999; Steinhausen et al., 2008). In a large sample of 484 adults, Rigaud and colleagues (2011) investigated longterm prognosis of AN treatment. Participants were adult inpatients with AN who had been treated between 1985 and 1995 and were followed for 13 years following discharge. They were assessed at admission, discharge, and at least once a year during the 13-year follow-up period. Recovery was defined as having a normal and stable body weight (BMI 18.5 - 25) and engaging in normal eating behaviors. Relapse was defined as a distinct drop in weight associated with elevated ED pathology and behaviors. They found that 80% of the sample was rehospitalized at least once during the 13-year period, with a large portion of the full sample (33%) requiring more than one readmission. Of the full sample, 52% relapsed within one-year and 16% did so within two years of treatment discharge. The strongest predictor of relapse at two-year follow-up was discharge BMI, followed by excessive exercise and restricting behaviors within one month of discharge. Weight at discharge negatively predicted relapse such that participants with lower BMI were more likely to relapse, with the highest relapse rate occurring in participants with a discharge BMI between 15.5 and 16.5. Importantly, this relation extends beyond simply whether

participants achieved goal weight. Even considering participants below goal weight (BMI = 18.5), differences in relapse rate were present. These findings suggest that low discharge BMI and even partial weight restoration are risk factors for relapse and that full weight restoration may serve as a protective factor in relapse after treatment.

Research on the effects of weight restoration on psychological outcomes following treatment is mixed. Whereas some studies suggest weight restoration is associated with increased body image anxiety and dissatisfaction (Bamford, Attoe, Mountford, Morgan, & Sly, 2014), others have found weight restoration is associated with a decrease in depression, anxiety, and ED symptoms, and overall clinical improvement in adolescents and adults (Accurso et al., 2014; Haynos et al., 2014). Weight recovery prior to discharge has also been associated with reduced dietary restraint, weight/shape concerns, and body dissatisfaction post-treatment (Accurso et al., 2014). Mixed results may be due to the differences in speed between weight and psychological recovery as mentioned above; changes in psychological functioning may be not be captured by current research methods.

Weight Gain Rate

Though weight restoration and discharge BMI have been shown to be strong predictors of short and long-term outcomes following AN treatment, rate of weight gain appears to be a significant predictor as well. An example of weight gain rate might simply be the number of pounds gained in a week. For patients with AN, weight gain rate has demonstrated clinical utility in determining early-stage compliance as well as posttreatment response (Lund, Hernandez, Yates, Mitchell, McKee, & Johnson, 2009). Weight gain rate is often used as a marker of readiness to progress to a less restrictive

level of treatment (Accurso et al., 2014; Haynos et al., 2014). Finally, weight gain rate can be used as a measure of program effectiveness and is associated with a program's ability to weight restore their patients.

Although there is a consensus that weight restoration is a pivotal aspect of AN treatment, there has been debate in the literature about the optimal rate of weight gain. Some researchers suggest that a slower rate of weight gain may be more beneficial than a faster rate. In certain studies, faster weight gain rates were associated with increased anxiety, faster weight loss after discharge, and higher rates of rehospitalization (Davies & Jaffa, 2005; Hartmann & Nickel, 2004; Lay et al., 2002; Willer, Thuras & Crow, 2005). Conversely, other researchers suggest a faster weight gain rate may be optimal. Faster weight gain rates during inpatient treatment have been associated with better outcomes at program discharge and long-term follow-up, including a decreased likelihood of deterioration, relapse, and readmission (Castro et al., 2004; Lund et al., 2009; Mewes et al., 2008).

Weight gain rate appears to be an important treatment parameter that is predictive of short and long-term outcomes; however due to study limitations and methodological biases, research recommendations remain mixed on the optimal rate of weight gain. *Prognostic significance of weight gain trajectories*

Many studies have focused on the achievement of weight restoration, overall number of pounds gained during treatment, average rate of weight gain, or categorization of overall speed of weight gain (fast versus slow). These weight gain parameters describe the discrepancy between admission and discharge weights and the post-hoc aggregate weight gain during treatment. Although these parameters are necessary and informative,

they lack descriptive information about how patients gain weight during their treatment course. The rate at which patients gain weight may vary during treatment, with rates fluctuating week to week. Solely focusing on overall single weight gain parameters may lack important variation in weight gain over the course of treatment.

Research has suggested that weight gain during inpatient and partial hospital treatment can progress in different patterns, or trajectories, and these patterns may be predictive of long term outcome (Accurso et al., 2014; Le Grange, Accurso, Lock, Agras, & Bryson, 2014; Lay et al., 2002). One pattern frequently studied is early versus later weight gain. In a randomized control trial comparing family-based treatment and individually focused therapy, Accurso and colleagues (2014) investigated weight gain in 121 adolescent outpatients with AN. They found weight gain early in treatment predictive of recovery. More weight gain earlier rather than later in both treatments was the strongest predictor of weight restoration at the end of treatment, defined as 95% of their target weight. However, early weight gain was not associated with remission at the 12month follow-up for either treatment. Conversely, Mewes et al. (2008) found that faster weekly weight gain in the second half of treatment was associated with better outcomes than faster rates in the beginning of treatment. Discrepancies between these studies may be due to differences in samples, including illness severity, inpatient versus outpatient settings, treatment program, and definitions of outcome.

In a similar study focusing on outpatient adolescents with AN, Le Grange et al. (2014) found that compared to weight gain later in treatment, earlier weight gain was associated with weight restoration at discharge and reduced ED psychopathology at follow-up. This suggests perhaps early weight gain is associated with weight restoration

at discharge while weight restoration at discharge is predictive of long-term remission. It may be that fast weight gain early in treatment is an indicator of treatment compliance and reduced ambivalence towards behavior change. Trajectories of weight gain during treatment appear to be related to treatment outcomes; however current research is mixed and the relation is not clear.

Weight gain trajectories are thought to be an intermediate outcome that can provide relevant information about clinical outcomes. Vansteelandt and colleagues (2010) investigated weight gain trajectories in inpatient AN treatment and potential moderators of trajectories using multilevel modeling. They found average weight gain trajectory was characterized by an overall linear trajectory with an initially steep weight increase that began to plateau towards the end of treatment as patients approached weight restoration. After probing for individual differences, they found patients with higher body dissatisfaction at admission had an overall slower rate of weight gain throughout treatment. Neither drive for thinness nor behavioral subtype (restricting vs. purging) predicted changes in weight gain trajectory. Of note, no patient characteristics predicted the plateau at the end of treatment. Interestingly, admission BMI did not predict trajectory. This suggests that although patients follow the same treatment protocol, they gain weight differently and that certain ED-related variables, such as body dissatisfaction, are related to different patterns. However, the link between weight gain trajectory and long-term outcomes as well as prognostic significance of trajectories was not addressed in the study and researchers identified this area an important future research area.

In a similar study focusing on individuals with AN who relapse, Lay and colleagues (2000) investigated patterns of weight gain in a sample of adolescents (N =

40) who were in their second admission for AN. They found that patients who had a slower rate of weight gain with periods of weight loss during their first admission had faster weight loss following treatment discharge. Authors concluded that more rapid weight gain followed by weight stabilization may reduce the risk of relapse after discharge. This suggests pattern of weight gain during treatment can provide valuable prognostic information, such that increased variability in weight gain may be an indicator of increased risk of weight loss after discharge. However, this study utilized a small sample limited to adolescents who had relapsed at least once. It is not clear how these patterns relate to individuals who have maintained recovery.

Hartmann and colleagues (2007) modeled weight gain trajectories using growth curve analysis to predict relapse following inpatient treatment for AN. Treatment failure, or nonresponse, was defined as having a discharge BMI less than 17.5 or a weight gain of less than 2 kg (4.4 pounds). Treatment successes, or responders, were defined as achieving weight restoration (BMI of 17.5 or greater) or having adequate weight gain. Of the full sample (N = 85), 20% were considered failures. For weight gain trajectories, researchers distinguished weeks one and two (early weeks) from weeks three and four (later weeks) by running separate models to allow for prediction of later trajectories from early trajectories. Comparing the models, there was greater variability in slope during early weeks and later weeks were more predictive of treatment success. Increased risk of failure was associated with weight loss in either model as well as increased fluctuations (periods of weight loss) in later weeks. Conversely, increased likelihood of success was associated with a combination of adequate weight gain in early weeks and increased weight gain without many fluctuations in later weeks. However, weight was only

collected twice a week; therefore weekly weight gain didn't take into account daily fluctuations. In addition, patients were only included in the study if they were in treatment for at least 6 weeks, which eliminated a large proportion of their sample. As a result, their sample may not be representative of typical AN samples, as average length of stay is 40 days.

Research on weight restoration has predominately focused on single weight estimates, such as discharge BMI or overall rate of weight gain. Although research on the course of weight restoration during treatment is limited, weight gain curves appear to be relevant as they can offer important information about both short and long term outcomes of AN. Further, they have the potential to serve as an in-treatment marker of treatment response and indicator of relapse risk, which would allow clinicians to address that risk prior to discharge, thereby increasing likelihood of recovery.

Predictors of variance in weight gain parameters

As discussed above, potential predictors of weight gain trajectories include behavioral subtype, body dissatisfaction, drive for thinness, and weight suppression. Research has shown that behavioral subtype of AN has an influence on rate of weight gain, with individuals of the restricting subtype gaining weight slower than purging type (Neuberge et al., 1994). Slower weight gain rate with AN-R was associated with increased age, longer duration of illness, and lower weight; all indicators of increased illness severity, suggesting that increased illness severity is associated with slower weight gain.

Level of self-efficacy related to changing ED behaviors plays a role in treatment response and outcome. Self-efficacy, defined as an individual's perceived ability to

successfully perform a behavior (Bandura, 1977), has been shown to impact behavior change with lower levels of efficacy related to a decreased likelihood of change (Bandura, 2000). Normative eating self-efficacy, such as the perceived confidence to refrain from ED behaviors and acceptance of body shape, appears to be an important factor in treatment outcomes. Research suggests patients with lower levels of efficacy endorse increased ED pathology (Pinto, Guarda, Heinberg, & DiClemente, 2006; Rieger et al., 2002; Schneider et al., 1987). Individuals with higher levels of self-efficacy may have greater confidence in their ability to change their behaviors and work towards recovery, and are more likely to engage in treatment.

Further, weight suppression, or the difference between an individual's highest lifetime weight and current weight, is related to rate and amount of weight gain (Butryn, Lowe, Safer, & Agras, 2006). Weight suppression has been show to predict amount of weight gained by individuals with bulimia nervosa during hospitalization. Research suggests individuals with eating disorders with higher weight suppression gain more weight than those with lower weight suppression (Lowe, Davis, Lucks, Annuziato, & Butryn, 2006). In addition, higher weight suppression has been associated with faster weight gain during treatment (Herzog, Thomas, Kass, Eddy, & Lowe, 2010).

Research indicates variation in weight gain during treatment and recent studies have begun to investigate distinct patterns of weight gain. However, the majority of research on patterns of weight gain focuses on early versus later weight gain and more specific trajectories of weight gain remain unclear. Further, although there are wellestablished predictors of simplistic measures of weight gain (i.e., weight gain rate and
weight restoration), research on predictors of *patterns* of weight gain during treatment is limited.

Current Study

This study explored patterns of weight gain during inpatient treatment for AN and the effect of patterns on treatment outcomes. Specifically, this study aimed to identify subgroups of patients with differing trajectories of weight gain in a sample of underweight female adolescents and adults in an inpatient ED treatment program. Predictors and treatment outcomes of the estimated trajectory group membership were investigated. Preliminary research suggests weight gain patterns are predictive of posttreatment functioning and therefore may serve as an in-treatment marker of relapse (Hartmann et al., 2007; Lay et al., 2004; Le Grange et al., 2014). However, recent studies on weight gain patterns have been limited to small sample sizes, have failed to include psychological variables, and/or only focused on outpatient treatment (Accurso et al., 2014; Lay et al., 2004; Vansteelandt et al., 2010). This study aimed to extend previous research and help determine if weight gain trajectories provide valuable information about both short and long-term outcomes following a behaviorally based AN treatment in an inpatient hospital setting.

The primary goal of this study was to investigate individual differences in weight gain in a hospitalized sample with AN using mixture modeling. Mixture modeling is a statistical approach that allows for estimation of individual variation in growth and identification of differences in outcome in naturally occurring groups. Using mixture modeling, weight gain trajectories during the inpatient phase of the program were identified and described. For simplicity and to maximize sample size, weight gain

trajectories were only estimated for the inpatient phase of the program. The growth variable of interest was weight gained and modeling was conducted using three differing operationalizations of weight gain. Weight gained was defined as: change in weight in pounds; change in weight in BMI; and BMI. The time referent was number of days on weight gain protocol. It was anticipated that there would be four distinct weight gain trajectories; a steady linear trajectory (Optimal group), a linear trajectory with a slower rate of weight gain (Sub-optimal group), a negative quadratic trajectory (Accelerated group), and a positive quadratic trajectory (Risk-Slow group); see Figure 1.



Figure 1: Predicted growth curve trajectories of weight gain.

A secondary goal of this study was to identify predictors of weight gain trajectory group membership. Using multinomial logistic regression, clinically relevant admissionlevel variables were used to predict membership in the estimated weight gain trajectory groups. This would allow for comparisons between trajectory groups and identify correlates of group membership. Hypothesized predictors of membership were: age, length of illness, previous hospitalizations, behavioral subtype (restricting vs. purging), admission BMI, highest lifetime BMI, frequency of ED behaviors in the 8 weeks prior to admission, level of ED pathology (drive for thinness and body dissatisfaction), level of normative eating self-efficacy, and personality factors. It was expected that indicators of illness severity, such as older age, longer illness duration, lower admission BMI, higher ED pathology, and previous hospital admission, would discriminate the trajectory groups with slower rates of weight gain due to decreased motivation to change and engagement in treatment. Patients with higher levels of self-efficacy and lower frequency of ED behaviors at admission were expected to be in those groups with average rates of weight gain.

A third and final goal of this study was to determine the predictive ability of weight gain trajectory group membership on short and long-term treatment outcomes. Using analysis of variance (ANOVA), analysis of covariance (ANCOVA), and logistic regression, the clinical significance of trajectory groups and their relation with discharge and six-month outcomes were analyzed. Effects of trajectory group on BMI (at discharge and six-month), weight restoration (at discharge and six-month), length of time on weight gain protocol, ED behavior relapse, readmission to an ED program, drive for thinness, body dissatisfaction, and normative eating self-efficacy were estimated. Probing the relation between trajectory group and outcomes would indicate if certain trajectory groups were associated with worse outcomes and thereby identify patients who are at risk for relapse following treatment.

Proposed Hypotheses

- It was hypothesized that women on weight gain protocol would be classified into one of four distinct groups based on predicted trajectories of weight gain, as depicted in Figure 1. Considering Figure 1, it was estimated there would be a group following a constant rate of weight gain (Optimal), a group with a slower, though still constant, rate of weight gain (Sub-optimal), a group following a negative quadratic trajectory that initially gained rapidly and then plateaued towards the end of treatment (Accelerated), and a group following a positive quadratic trajectory that had an initially slow weight gain followed by faster, though still relatively slow, gains as treatment progressed (Risk-Slow).
- 2) Clinically relevant variables were expected to predict trajectory group membership in a series of bivariate relations (e.g., uncontrolled logistic regressions). Though group is the outcome, the following bivariate relations are more easily described through consideration of hypothesized group differences. It was expected that age, length of illness, previous hospitalization, admission BMI, ED behavioral subtype (restricting vs. purging), frequency of ED behaviors, level of ED pathology (drive for thinness and body dissatisfaction), and level of normative eating self-efficacy would predict trajectory group membership.

2a. The Optimal group was expected to be comprised of relatively younger women, who engaged in fewer eating disordered behaviors, were more likely to have a purging subtype, higher admission BMI, shorter length of illness, lower drive for thinness and body dissatisfaction, and greater normative eating self-efficacy.
2b. The Risk-Slow group was expected to be comprised of relatively older women, who engaged in more eating disordered behaviors, were more likely to have a

restricting subtype, lower admission BMI, longer length of illness, higher drive for thinness and body dissatisfaction, and lower normative eating self-efficacy.

2c. The Sub-optimal group was hypothesized to be between the Optimal and Risk-Slow groups on admission level variables that are markers of illness severity. It was not clear where the Sub-optimal group would fall relative to the Optimal and Risk-Slow groups on other predictors of group membership, though I predicted the Suboptimal group would be more similar to the Risk-Slow than the Optimal group. 2d. The Accelerated group was also hypothesized to be between the Optimal and Risk-Slow groups on admission-level variables that are markers of illness severity. As with the Sub-Optimal group, I did not make as explicit hypotheses on other variables, but I did hypothesize the Accelerated group would appear more similar to the Optimal than the Risk-Slow group.

3) It was hypothesized that trajectory group membership would predict differences in discharge and six-month follow-up outcomes in a series of bivariate relations.
3a. The Optimal group was hypothesized to be associated with more positive outcomes. I predicted the Optimal group would be more likely to achieve target weight, have higher BMI, and be more likely to stay in the program until recommended discharge. Considering six-month outcomes, I expected the Optimal group would be more likely to relapse on ED behaviors, less likely to require rehospitalization, have lower drive for thinness and body dissatisfaction, and higher normative eating self-efficacy than all other groups.

3b. The Risk-Slow group was hypothesized to be associated with more negative outcomes. I predicted the Risk-Slow group would be less likely to achieve target weight, have lower BMI, and less likely to stay in the program until recommended discharge. Considering six-month outcomes, I expected the Risk-Slow group would be less likely to maintain target weight, have lower BMI, be more likely to relapse on ED behaviors, more likely to require rehospitalization, have higher drive for thinness and body dissatisfaction, and lower normative eating self-efficacy than all other groups.

3c. I had less specific hypotheses regarding the Sub-optimal group. I predicted the Sub-optimal group would be less at risk for negative outcomes than the Risk-Slow group but more at risk than the Optimal group. Considering discharge and six-month outcomes, I expected the Sub-optimal group would typically be associated with positive outcomes and appear more similar to the Optimal than the Risk-Slow group. 3d. Overall, I expected the Accelerated group to be less at risk for negative outcomes than the Risk-Slow group but more than the Optimal group. At discharge, I predicted the Accelerated group to be associated with more positive than negative outcomes and appear more similar to the Optimal group. However, considering six-month outcomes, I expected the Accelerated group to be associated with more negative outcomes and be more similar to the Risk-Slow than the Optimal group.

Research Question:

1. From hypothesis 2, any predictors that show a bivariate relation at p < .10 with group membership were included in a single multiple logistic regression. In brief,

research question 1 sought to determine whether any given variable that showed an overall bivariate relation continued to predict group membership after controlling for other predictors.

 From hypothesis 3, it was expected that ED-related variables may impact discharge and six-month outcomes, such as age, behavioral subtype, prior hospitalizations, and participation in partial hospital treatment. Research question 2 sought to address the following two related questions:

2a. Do the hypothesized relations between trajectory group membership and discharge/six-month outcomes remain after controlling for relevant covariates?2b. Does participation in partial hospital treatment act as a moderator on the effects of trajectory group membership on six-month outcomes?

Method

Participants

Data collection took place over the span of approximately 10 years (from 2003 through 2012). At the time of admission, 419 patients consented to the study and completed admission questionnaires. Individuals with bulimia nervosa and subthreshold bulimia nervosa (n = 104), binge eating disorder (n = 8), and atypical eating disorder (n = 22) were dropped from the sample as they were not underweight and weight restoration was not an emphasis of treatment. Of the 285 patients with AN or subthreshold AN, those who were not on weight gain protocol for a minimum of seven days and/or were not at least four pounds below their target weight (n = 47) were dropped. Finally, males (n = 19) were dropped due to differences in weight gain rates and the sparsity of them in the

program. This resulted in the current sample (N = 219) of whom 76% transitioned to partial hospital and 56% (n = 123) completed six-month questionnaires.

Participants were 219 female patients diagnosed with AN or subthreshold AN who were admitted to an integrated inpatient-partial hospital ED treatment program. All participants consented to participate in a longitudinal outcomes study approved by the Johns Hopkins Institutional Review Board. The sample was 88% Caucasian with a mean age of 28.52 years (SD = 12.41); 18% were minors. Thirty-three percent of participants had AN restricting type (AN-R) and 52% had AN purging type (AN-P); the remainder were diagnosed with eating disorder not otherwise specified (EDNOS; n = 33). Considering the EDNOS sub-sample, n = 12 had subthreshold AN-R and n = 21 had subthreshold AN-P. Considering just restricting versus purging subtypes (i.e., collapsing across AN and EDNOS), n = 85 (39%) were of restricting subtype and n = 134 were of purging subtype. In terms of indicators of illness severity, average BMI upon admission was 16.14 (SD = 2.05), average duration of self-reported dieting behaviors was 13.66 years (SD = 12.39), average duration that ED behaviors had interfered with the individual's functioning was 10.67 years (SD = 11.46), and 50% of the participants reported at least one previous ED inpatient admission. Participants needed to gain an average of 26.58 pounds (SD = 12.79) to achieve weight restoration (defined as BMI = 20). Finally, overall length of stay was 55.64 days (SD = 31.81) with an average of 28.96 days (SD = 21.41) in inpatient and 33.58 days (SD = 18.73) in partial hospital. Inpatient treatment was successful for the majority of the sample with 64% (n = 141) reaching their target weight prior to discharge and an average discharge BMI of 19.86 (SD = 1.82).

Table 1 provides additional information about the sample at admission, discharge, and

six-month follow-up.

Table 1

Clinical Variables at Admission, Discharge, and Six-months after Treatment Discharge

	Admission ($N = 219$)	Discharge ($N = 219$)	Six-month ($N = 123$)
BMI	16.14 (<i>SD</i> = 2.05)	19.86 (<i>SD</i> = 1.82)	18.87 (<i>SD</i> = 2.32)
Percent of target weight	78.11 (<i>SD</i> = 10.00)	96.08 (<i>SD</i> = 8.49)	90.99 (<i>SD</i> = 11.42)
Days on weight gain protocol	26.81 (<i>SD</i> = 19.38)		
Restricting ¹	85%		52%
Bingeing ¹	26%		17%
Vomiting ¹	50%		25%
Laxative Use ¹	18%		10%
Drive for thinness ²	1.81 (<i>SD</i> = 0.99)		1.32 (SD = 1.00)
Body dissatisfaction ²	1.73 (SD = 0.93)		1.52 (SD = 0.96)
Normative eating self-efficacy ³	2.14 (<i>SD</i> = 1.13)		2.64 (SD = 1.25)

¹ Percent of sample who engage in the behavior regularly (once a week or more)

 2 Scale ranges from 0 to 3, with higher values indicating higher levels of variable

³Scale ranges from 1 to 5, with higher values indicating higher levels of variable

Procedure

For the current study, self-report measures were completed at two time points: inpatient admission and six months following discharge from the program. The first time point utilized paper/pencil surveys whereas the six-month follow-up was administered with Surveymonkey Audience. If participants did not complete the six-month survey within 2 weeks, a paper copy was sent in the mail. If the survey packet was not returned within two weeks, research staff phoned participants. If participants had not completed the packet after two phone calls, they were considered a drop-out. The time between the measurement waves varied across patient depending on length of stay in the program. Additionally, not all patients transitioned to partial hospital, in which case the six-month survey was administered six months following discharge from inpatient hospital. Compensation for participation was entry into a raffle for a \$50 Amazon gift card.

Eating disorder diagnoses were determined via Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997) administered by trained interviewers. However, consistent with DSM-5 criteria, amenorrhea was not required for AN diagnosis. Once in treatment, the clinical team set a target weight with a range of \pm four pounds (1.8 kilograms). Target weight was not disclosed to the patient to avoid preoccupation with weight. Target weight was defined based on patient age and height, with special consideration for patients younger than 16 years of age. For patients less than 16, target weight was set using growth charts and/or the 25th percentile of weight for her height. For patients from 16 through 24 years of age, target weight was set based on a BMI of 20, minus one pound (0.45 kilogram) per year of age below age 25. For patients 25 years of age and older, target weight was set based on a BMI of 20.

While in treatment, daily weights were collected by nursing staff every morning before breakfast and after voiding. Between 2003 and 2009, weights were recorded in the patient's paper chart, and after 2009, they were entered into their electronic medical record. For this study, daily weights were obtained via chart review after discharge and double entered to ensure accuracy. Duplicate datasets were cross-checked and resulted in 11 mistakes, which were then corrected. Rate of weight gain was calculated weekly and the current sample had an average weight gain rate of 4.39 pounds per week (SD = 1.92) throughout the inpatient program.

Measures

Demographic Variables At admission, participants were asked to complete a self-report questionnaire battery and provide basic demographic information including age, race, marital status, and level of education. Participants were also asked to report various ED-related variables, including lowest and highest weight (at current height), current weight, and history of obesity. Participant weight was measured daily by staff and recorded on the patient chart.

Clinical Outcome Variables Additional clinical data were collected at two time points: 1) proximal outcomes were collected at discharge from inpatient program and 2) distal outcomes were self-reported six-months following discharge from the full program. For 24% of the sample who did not participate in partial hospital, distal outcomes were measured six months following discharge from in-patient; for all others, distal outcomes were measured six months following discharge from partial hospital. Proximal outcomes, as assessed by chart review, include: discharge weight, days on weight gain protocol, target weight and range, behavioral subtype (restricting vs. purging diagnoses), number of previous ED hospitalizations, years of illness duration, highest lifetime BMI, and weight gain rate. Distal outcomes, as self-reported by the participant, include: current weight and ED-related rehospitalization.

Eating Disorder Behaviors Frequency of six current (within eight weeks) eating disordered behaviors were assessed at admission and six-month follow-up. The ED behaviors included binge eating; vomiting; use of laxatives; skipping meals; restricting food portions; and eating meals low in fat or calories. Frequency was measured on a Likert-type scale ranging from *never* (1) to *several times a day* (7). For the current study,

three of the ED behaviors were defined as measured; these were, binging, purging, and laxative use. A fourth behavior measure, restricting, was created by computing the mean of skipping meals, restricting food portions, and eating meals low in fat or calories.

Eating Disorder Symptomatology The Eating Disorders Inventory (EDI-2; Garner, 1991) is a 91-item, self-report questionnaire that measures the severity of ED disturbance on 11 subscales; participants rate each item on scale from 1 (never) to 6 (always). The EDI-2 was measured at both admission and six-month follow-up. For this study, only Drive for Thinness and Body Dissatisfaction subscales will be used. The Drive for Thinness (DT) subscale is comprised of seven items assessing preoccupation with dieting and weight, desire to be thinner, and fear of weight gain; with higher scores indicating greater drive for thinness. The Body Dissatisfaction (BD) subscale is comprised of nine items that assess satisfaction with one's overall shape and specific body regions, with higher scores indicating greater body dissatisfaction. DT and BD subscales' reliability in this population is well established with estimates ranging from .75 to .95 (Cumella, 2006; Garner, 1991). These subscales have also demonstrated strong concurrent validity discriminating up to 97% of individuals with AN versus healthy controls (Shoemaker, Verbraak, Breteler, & van der Staak, 1997). Cronbach's α for DT and BD in the current study were .91 and .93 at admission and .90 and .94 at follow-up, respectively.

Eating Disorder Recovery Self-Efficacy Questionnaire (EDRSQ). The EDRSQ is a 23-item questionnaire that measures level of confidence to engage in normative eating behaviors and maintain a healthy body image (Pinto et al., 2006). Participants were asked to rate items on a scale ranging from 1 (*not at all confident*) to 5

(*extremely confident*), with higher scores indicating higher levels of self-efficacy. The normative eating subscale is composed of 14 items that assess perceived ability to eat without engaging in eating disordered behaviors such as, "*Eating from a buffet without feeling anxious*." Only the normative eating subscale was used for the current study. The overall EDRSQ has been shown to predict treatment outcomes including length of stay, body dissatisfaction, and weight gain rates in partial hospital (Pinto et al., 2008). Cronbach's α for normative eating in the current study is quite high, .97 and .95 at admission and follow-up, respectively.

Personality The NEO Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992) is a 60-item, self-report questionnaire that measures five personality domains (neuroticism, extraversion, openness, agreeableness, and conscientiousness); each domain is measured by 12 items. Participants were asked to rate items on a scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*), with higher scores indicating higher levels of personality traits. In this study, Cronbach's α for subscales were .85, .81, .76, .77, and .87 for the domains as listed above, respectively.

Measures to estimate and define weight gain trajectories

Weight Restoration For this study, target weight was defined as above. Weight restoration was merely a dichotomous variable measuring whether the weight at discharge was at, or exceeded, target weight. Weight restoration, at both discharge and six-month, served as dichotomous outcomes for Aim 3.

Days on weight gain protocol The time referent for the weight gain trajectory analyses was the number of days on weight gain protocol (WGP). Patients remained on WGP until they have reached their target weight; at which time, they transitioned from inpatient to day hospital and treatment focus turned from weight restoration to psychological recovery.

Results

Aim 1. Individual growth trajectories were estimated using mixture modeling with the SAS Trajectory procedure (Jones & Nagin, 2007). Mixture modeling is a statistical approach that estimates growth trajectories based on a semiparametric, group-based modeling strategy (Jones, Nagin, & Roeder, 2001). This approach allows for the examination of overall change in a population and individual differences in observed trajectories. The trajectory procedure program was beneficial as it accommodated both irregular spacing of measurements and differential length of study involvement. Through this modeling, I was able to estimate and describe differing trajectories of change throughout inpatient treatment. The time referent used for modeling was defined as days on weight gain protocol.

Consistent with an over-arching goal to identify the most parsimonious solution, I began with the simplest model (two groups, each characterized by linear growth) and incrementally increased complexity (i.e., allowing growth to be non-linear in one group and/or specifying additional groups). I used the BIC (Bayesian Information Criterion) and the AIC (Akaike Information Criterion) as well as distinct trajectory patterns, parsimony of the solution, and a minimum sample size to compare any two models; each trajectory group must contain at least 10% of the sample (approximately 22 participants) in order to be retained in the analysis.

Mixture modeling was conducted using three differing operationalizations of weight gain (though not at the same time). The three ways in which "weight gain" was

defined were: 1) change in weight in pounds, 2) change in BMI, and 3) BMI. Ultimately, given primary interest is in program efficacy, modeling *change* (in pounds or BMI) makes more conceptual sense as it does not confound starting weight with weight gain. Modeling BMI (results not presented) may be more appropriate for researchers seeking a richer understanding of the development and etiology of eating disorders and weight change over time and results in somewhat differing patterns of growth over time. Considering results from modeling both change variables (raw weight and BMI), it is the case that with either outcome the best solution suggested three groups wherein one group was characterized by positive linear growth over time and the other two groups were characterized by positive linear growth and a negative quadratic (i.e., the rate of growth slowed); the difference between the two groups with a downturn was primarily the initial rate of positive growth. Table 2 qualitatively summarizes model comparisons (i.e., how models differed; what criteria were considering choosing between the given pair of models; etc.) resulting in the final recommended model. For efficiency, Table 2 only summarizes the decision for models based on change in pounds as no substantial differences were observed when modeling change in BMI.

Table 2

Results from Modeling Using Weight Gained (pounds) as Growth Variable of Interest

Model	Number	Growth	BIC	Interpretation of modeling criteria
	of groups	pattern	AIC	
1	2	Both	-15156.21	Baseline model. Most participants have probabilities of primary group
		linear	-15136.70	membership $> .90$. Growth trajectory patterns appear distinct.
2	2	Lincor and	14042 27	Improvement over model 1 based on PIC/AIC. No major change in
Z	Z	cuedrotio	-14943.37	nuprovement over model 1 based on DIC/ARC. No major change m
		quadratic	-14931.97	remain distinct.
3	2	Both	-14726.32	Improvement over model 2 based on BIC/AIC. Similar probabilities of
		quadratic	-14719.90	primary group membership as model 2. Trajectories appear similar in
		1		pattern as model 2.
	2		1 4 5 9 9 9 1	
4	3	All linear	-14523.21	Improvement over model 3 based on BIC/AIC. Most participants have probabilities of primary group membership ≥ 00 . Betterns visually
			-14308.94	distinct
				distinct.
5	3	2 linear	-14068.94	Improvement over model 4 based on BIC/AIC. Similar probabilities as
		and 1	-14035.92	model 4 (majority above .90) with slight improvement (larger
		quadratic		discrepancies between groups). Patterns appear visually distinct.
<u>(</u> *	2	1 1	12040 57	Classic DIC/AIC Issuer (in
0.	3	1 innear	-13942.57	Clear improvement over model 5 based on BIC/AIC. Improvement in
		anu 2 quadratic	-13900.23	$M_{aiority}$ of the participants have probabilities > 90 with 01% above
		quadratic		.95. Patterns appear visually distinct.

7	3	All quadratic	-14281.10 -14241.47	No obvious improvement in model fit over model 6 based on BIC/AIC and worse probabilities as previous model. Patterns appear visually distinct.
8	3	Linear, quadratic, and cubic		False convergence.
9	4			False convergence for all variations of growth using 4 groups.
10	5			False convergence for all variations of growth using 5 groups.

Note. For models estimating 3, 4, and 5 groups, all possible combinations of growth were estimated; however for brevity not all results are provided. All models resulted in false convergence.

*Retained model with best fit and most parsimonious solution.

¹ with the exception of the models with failed convergence, all models returned solutions in which minimal sample size of group membership was met

² Probabilities > .90 for primary group membership classification were chosen as such magnitudes are consistent with low ambiguity of predicted group membership for a given participant

Evaluating change in weight versus change in BMI. As noted, the retained solutions for both change in weight and change in BMI yielded the same three group solution; this finding may be relevant to researchers attempting to choose between modeling change in either of the variables. The identical form in solution is also helpful in the current study as it allows direct comparison of the solutions with respect to predicted group membership. In the final model with three groups, there is over 95% agreement between the change in weight in pounds and change in BMI groups with 200 of the 209 participants being classified into the same group. Of the nine women who were misclassified across models, five women were categorized as Optimal group members in the pounds model and as Accelerated members in the BMI model. Further, two women were classified as Accelerated members in the pounds model and subsequently classified as Optimal members in the BMI model. It is not surprising these women alternated between the Optimal and Accelerated groups due to these groups' similar pattern of growth (negative quadratic). Of the final two misclassified women, one was classified in the Optimal group in the pounds model and classified in Sub-optimal in the BMI model whereas the other was classified as Sub-optimal in the pounds model and classified as Optimal in the BMI model.

Given the functional similarity in solutions between change in weight and change in BMI, it can be argued to be arbitrary as per which metric to present. I elected to focus on change in weight (pounds) as this is conceptually an easier metric to understand and is more consistent with the goals established for the woman for their weight gain protocol (WGP). Though the goals are *determined* based on target BMI, the goals themselves are expressed as a target gain in pounds. For the remainder of analyses in Aims 1, 2, and 3 I

conducted all analyses based on both change in pounds and change in BMI. However, for the sake of brevity I only present results based on change in weight unless there were inferential differences between the solutions in which case such differences were noted.

Another indirect method of evaluating success of the mixture modeling is to examine the probability of group membership. Functionally, each participant has a specific probability of being assigned to each of the identified groups. As there are a finite number of groups, the sum of the probabilities is always equal to 1.00. Large discrepancies in the probabilities (e.g., .97, .02, and .01 from a hypothetical three group solution) are indicative of a "good solution" *for that participant*. Whereas smaller differences (e.g., .35, .33, .32) may reflect a "poorer solution" that was not unambiguously able to classify *that participant*. Group probabilities (see Table 3) for the final model generally had large discrepancies between the three groups with high average probabilities of group membership for each group (above .97 for each group), which is above the minimum recommended guideline of .70 (Nagin, 2005; Nagin & Odgers, 2010). Based on the high average probabilities, this suggests that the final model was a good solution overall.

Table 3

Percentage of Participants with Probability of Primary Group Membership Greater than

Model	Number of groups	Growth pattern	Percentage
1	2	Both linear	90.52% (191/211)
2	2	Linear and quadratic	90.52% (191/211)
3	2	Both quadratic	91.47% (193/211)
4	3	All linear	92.42% (195/211)
5	3	2 linear and 1 quadratic	91.94% (194/211)
6*	3	1 linear and 2 quadratic	93.36% (197/211)
7	3	All quadratic	86.73% (183/211)

.90

*Retained model with best fit and most parsimonious solution.

Observed Groups

Figure two provides the actual and predicted change in weight as estimated using mixture modeling for the retained solution. Considering the three empirically derived groups relative to the hypothesized groups, the Risk-Slow group was not found. However, there was evidence for the Optimal group (characterized by a negative quadratic trajectory in which a steady rate of weight gain was followed by a slight plateau toward the end of protocol), the Accelerated group (characterized by a negative quadratic trajectory with an initially faster rate of weight gain and moderate plateau towards the end of protocol) and the Sub-optimal group (characterized by a positive linear trajectory with an overall consistently slower rate of weight gain). The primary difference between the Accelerated and Optimal groups is the Accelerated group has an initially faster rate of weight gain and slightly steeper plateau than the Optimal group (see Figure 2). The obvious distinction of the Sub-optimal group was the lack of plateau, likely due to the slower rate of weight gain over the course of treatment. From figure 2 it may be noted the

groups had differing lengths of trajectories. Given that time referent was days on weight gain protocol, these differences are reflective of differing lengths of time on the protocol and therefore differing amounts of weight needed to be gained to reach target weight. Differences in protocol length are due to the women meeting goal weight and subsequently being discharged.



Figure 2. Growth curve trajectories of weight gain in pounds while on weight gain protocol. Thick lines with symbols represent predicted means for each day on weight gain protocol. Thin lines without symbols represent observed means for each day on weight gain protocol.

Table four provides descriptives for each of the three groups on a number of baseline measures and treatment-protocol related measures. Note that Aim 2 will more formally examine relations between many of the baseline measures and group membership, thus at this point evaluation is simply descriptive in nature.

Table 4

Variable	Optimal (<i>n</i> = 114; 54%)	Sub-optimal (<i>n</i> = 59; 28%)	Accelerated (<i>n</i> = 38; 18%)
Age	27.32 (12.34)	29.00 (12.92)	31.08 (12.53)
Length of diet	12.14 (12.42)	15.12 (13.03)	15.73 (11.87)
Length of interference	9.00 (10.66)	12.06 (13.19)	12.95 (11.29)
Duaviana haamitalizatian	45 00/	50.00/	(2.)0/
Highest lifetime DMI	43.0%	50.9% 22.22 (6.51)	03.2%
Highest methic blvn	22.31 (4.00) 5 60/	23.22 (0.31)	24.94 (0.33)
(BMI $30\pm$)	5.0%	7.0%	15.5%
(BMI 50+) History of being overweight	11 1%	17 5%	21.6%
(BMI 25-29.9)	11.170	17.570	21.070
(Divit 25 2).))			
Purging Subtype	54.4%	57.6%	86.8%
Current weight control methods util	lized 1+ times/wee	^r k	
Restricting	90.7%	83.3%	67.7%
Bingeing	21.8%	23.2%	44.1%
Vomiting	35.5%	35.1%	65.7%
Laxative Use	14.5%	14.5%	34.3%
Engineer of unight control moths	da		
Prequency of weight control method Destricting	602(154)	5.85(2.01)	5 20 (2.06)
Dingoing	0.02(1.34) 2.32(2.10)	3.63(2.01)	3.20(2.00)
Vomiting	2.33(2.19)	2.27(1.91)	3.02(2.03)
	2.94(2.30)	2.93(2.57)	4.65(2.30)
Laxauve Use	1.00 (1.90)	1.70 (1.08)	2.77 (2.49)
Drive for thinness ^a	1 86 (1 00)	1 81 (0 93)	1 80 (0 98)
Body dissatisfaction ^a	1.72 (0.96)	1.78 (0.92)	1.82 (0.87)
Normative eating self-efficacy ^b	2.18(1.18)	2.08 (1.04)	1.98 (1.10)
- · · · · · · · · · · · · · · · · · · ·			
Admission BMI	16.11 (1.98)	16.58 (2.06)	15.68 (2.10)
Admission weight in pounds	94.02 (13.88)	98.17 (14.47)	92.12 (14.49)
Weekly Rate of weight gain	4.61 (1.37)	2.81 (1.30)	6.43 (2.02)
Days on WGP	25.61 (17.41)	28.61 (22.46)	25.57 (14.30)
(range)	(7 - 92)	(7 - 105)	(7 - 64)
Pounds gained while on WGP	14.52 (8.74)	8.89 (8.41)	21.41 (8.62)
Discharge BMI	18.76 (1.60)	18.42 (1.77)	19.22 (1.58)

Descriptives for Modeling Groups Based on Weight Gained in Pounds

^a Scale ranges from 0 to 3, with higher values indicating higher levels of variable ^b Scale ranges from 1 to 5, with higher values indicating higher levels of variable

The *Optimal group* comprised the majority of the sample (n = 114; 54.0%) and was characterized by positive linear growth with a negative quadratic component (evident as the slight plateau toward the end of protocol). About one-sixth of this group had a history of being overweight and/or obese. The group was comprised of roughly equal restricting and purging sub-types, though a large majority (more than 90%) regularly used restricting behaviors. Members of this group spent an average of 25.61 days on WGP and gained an average of 4.61 pounds a week (consistent with treatment expectations in this population). The average BMI at discharge for this group was nearly at treatment goal (M = 18.76, SD = 1.60).

The *Sub-optimal group* comprised nearly a third of the sample (n = 59; 28.0%) and was characterized by a positive linear growth with an overall steady rate of weight gain. About one quarter of this group had a history of being overweight and/or obese. The group was comprised of roughly equal restricting and purging subtypes, though a large majority (more than 80%) regularly used restricting behaviors. Members of this group spent an average of four weeks on WGP and gained an average of 2.81 pounds a week, which is well below treatment expectations. The average BMI at discharge for this group was nearly at treatment goal (M = 18.42, SD = 1.77). This apparent discrepancy (spending about 3 more days on WGP than the Optimal group, but with weight gain at about 60% of the Optimal group) is likely due to the variability in days on WGP in the Sub-optimal group.

The *Accelerated group* comprised the smallest percentage of the sample (n = 38; 18.0%) and was characterized by positive linear growth with a negative quadratic component (evident as the plateau toward the end of treatment). About one third of this

group had a history being overweight and/or obese. The group was primarily comprised of purging subtype (86.8%) and more than half regularly engaged in purging behaviors. Members of this group spent an average of 25.57 days on WGP and gained an average of 6.43 pounds a week, which is above treatment expectations. The average BMI at discharge for this group was at treatment goal (M = 19.22, SD = 1.57).

Comparing these three groups based on descriptive statistics, they differed primarily on ED subtype and behaviors, degree of illness severity, and treatment response. In terms of behavioral subtype of AN, the Optimal and Sub-optimal groups both had roughly equal restricting and purging subtypes whereas the majority of the Accelerated group was of the purging subtype. As well, those in the Accelerated group had higher reports of purging behaviors than both the Optimal and Sub-optimal groups, who tended to primarily engaging in restricting behaviors. That said, all three groups were highest, both in frequency and behavior, in the restricting behaviors, with the Optimal and Sub-optimal groups engaged in purging behaviors at noticeably lower frequencies. In terms of illness severity, members of the Optimal group had the least severe ED whereas members of the Accelerated group had the most severe ED as indicated by longer length of illness (both years of dieting and illness interference), previous hospitalizations, and lower admission BMI. Though not as severe as the Accelerated group, members of the Sub-optimal group also appeared to have higher illness severity and were closer in level of severity to the Accelerated than Optimal group. In terms of treatment response, the Accelerated group had the best response and the Sub-optimal group had the worst response and seemed to be the most treatment resistant, as indicated by slowest weekly rate of weight gain, longest length of treatment,

and least pounds gained while on WGP. Of note, women in the Accelerated group had the highest lifetime BMI and were more likely to have a history of being overweight or obese than the other groups.

Brief Discussion

Research on the heterogeneity of weight gain during treatment and identification of differing trajectories is limited. Aim 1 aimed to investigate individual differences in weight gain in a hospitalized sample with anorexia nervosa using mixture modeling. It was hypothesized there would be four distinct weight gain trajectories; a steady linear trajectory (Optimal group), a linear trajectory with a slower rate of weight gain (Suboptimal group), a negative quadratic trajectory (Accelerated group), and a positive quadratic trajectory (Risk-slow group). Results of this aim suggest three distinct patterns of weight gain during treatment; a negative quadratic trajectory (Optimal group), a negative quadratic trajectory with a faster initial rate of weight gain (Accelerated group), and a positive linear trajectory (Sub-optimal group). The fourth (Risk-slow) group was not found as hypothesized. These participants were hypothesized to be women who were resistant to treatment and gain minimal, if any, weight during treatment. It may be that because this hospital-based treatment program utilizes a unified behavioral protocol to which all patients must adhere, these individuals would have left treatment prematurely or transferred to another program. Alternatively, given the unified protocol and program requirements, these women may have been subsumed within the Sub-optimal group based on altered behaviors. The retained solution of three groups provided the best model fit based on multiple modeling criteria discussed above; this solution held across metric (change in pounds and change in BMI). Results suggest women with AN differ in pattern

of weight gain during treatment. Although all women followed the same treatment protocol, there was variability in how they gained weight.

In addition to pattern of weight gain, it was hypothesized that groups would differ in history and course of illness. Though not formally examined in this aim, descriptive statistics of admission level and treatment-protocol measures suggest the groups differed in type of ED behaviors, level of illness severity, and treatment response. While women in the Optimal and Sub-optimal groups primarily engaged in restricting behaviors to lose or prevent weight gain, women in the Accelerated group engaged in more purging behaviors. Interestingly, women in the Accelerated group had the most severe ED along with the best treatment response. Although women in the Sub-optimal group had a relatively worse treatment response, women in each group on average were successful in the program as indicated by average discharge BMI near target. These preliminary group differences will be further explored in Aim 2.

Aim 2. Multinomial logistic regression was used to identify predictors of weight gain trajectory group membership. Multinomial logistic regression is often used to predict outcomes with two or more unordered categories from one or more categorical or continuous predictor variables. It is a statistical approach used to estimate the relation between variables and group membership, and compares multiple groups on predictor variables. It also provides estimates of the probability of belonging to a specific population due to changes in predictor variables. This approach is advantageous as it provides the estimated probability of belonging to a specific group. The estimates used to evaluate these results are the likelihood ratio test, beta coefficients, Wald's chi-square, and odds ratio. The likelihood ratio test evaluates the bivariate relation between a

predictor variable and group membership controlling for other predictors in the model if relevant. With a single predictor, this test measures the improvement in model fit of the model with the predictor variable over the null model using a Chi-square distribution. If a predictor variable has an overall relation with the outcome, it is important to determine the specific relation between groups and between which groups the predictor significantly differentiates. This is evaluated using estimates specific to the individual predictors (beta coefficient, Wald's chi-square, and odds ratio).

The beta coefficients provide an estimate of the predicted change in the logit (natural log of the odds) of being in a specific group due to a one unit increase in the predictor. Wald's χ^2 is an approximate test of the statistical significance of the coefficient. The odds ratio (OR) is a convenient estimate for quantifying the impact of the predictor on group membership. Conceptually, it can be envisioned as the increased "odds" of being in the comparison group (over the referent group) due to a one unit increase in the predictor. Technically, ORs range from zero to infinity and center around one. ORs equal to one (equal odds) suggest no relation between the predictor and the two groups; ORs greater than one suggest increased odds (increased likelihood) of being in the comparison group rather than the referent group; ORs less than one suggest increased odds of being in the referent group rather than the group and less than one indicating decreased odds of being in the comparison group over the referent group. Multinomial logistic regression can be used with single (uncontrolled multinomial logistic regression) or sets (multiple multinomial logistic regression) of predictor variables. Multiple multinomial logistic regression is used when evaluating the predictive ability of a single predictor after controlling for other related variables.

For this aim, 19 uncontrolled multinomial logistic regressions were estimated to examine the bivariate relations between trajectory group and predictor variables. Variables expected to predict weight gain trajectory group included: age, length of illness (both length of dieting and length of illness interference), previous hospitalization, admission BMI, highest lifetime BMI, ED behavioral subtype (restricting vs. purging), frequency of ED behaviors (restricting, bingeing, vomiting, and laxative use), level of ED pathology (drive for thinness and body dissatisfaction), level of normative eating selfefficacy, and five personality characteristics. This statistical approach allowed for estimation of bivariate relations of group with possible predictors (analogous to a series of correlations of a continuous outcome with a series of continuous predictors). As group has three levels, for each predictor variable two multinomial logistic regressions models were necessary to allow examination of pairwise comparisons between the three groups. Models were also estimated controlling for admission BMI. Continuous measures were standardized based on the analysis sample to allow for easier interpretation given these variables used different metrics and were scaled differently. Dichotomous predictors were coded as 0, 1 (0 indicating "No" and 1 indicating "Yes") for all weight control methods and previous hospitalization.

Missing Data. Due to missing data on individual predictor variables, sample sizes ranged from N = 186 to N = 211. As one of the goals for the uncontrolled regressions was to identify predictors to include in the multiple regression it is important to recognize listwise missing data patterns (i.e., valid data on multiple variables). Based on bivariate relations with pairwise deletion, eight variables were observed to have overall relations with group; suggesting they would be included in the final model. When considering

group and these eight predictors, only N = 155 participants had valid data on all nine variables. Such a difference in sample size (from N = 211 to N = 155) makes understanding the impact of controlling for other variables (i.e., going from the uncontrolled to the controlled analyses) difficult. Specifically, if a change in statistical inference is observed it could be due to: a) a "different" sample being used, b) a change in statistical power from using a smaller sample, or c) because the predictor in question did not have a unique effect controlling for the other predictors. Though possible to simply conduct all analyses on the reduced sample (N = 155), this is not without problem as well. Primarily, such a strategy may obscure bivariate relations that could be potentially meaningful to future research and interventions. Mean imputation is an effective approach for addressing missing data and was considered as a strategy. However, imputation requires data to be missing at random (Little & Rubin, 1987), which could not be easily assessed for this study. Thus for simplicity I elected to consider results from uncontrolled regressions estimated using pairwise deletion and "listwise" deletion (N = 155). Though this does not confirm alternative explanations for possible changes in effect, it can be used to partially address such changes.

Prior to presentation of results, I evaluate patterns of missing data as well as differences between the excluded and retained participants on each predictor. When considering missing data, the single variable with the largest amount of missing data was extraversion (n = 20) followed by regularly restricting (n = 8). Participants with valid data on the set of eight predictors (n = 155) were compared to those participants who were excluded due to missing data (n = 56) and results are presented in Tables 5 and 6. T-tests were used to evaluate continuous variables and Chi-square tests of independence

were used to evaluate categorical variables. Excluded participants were older and had higher illness severity as indicated by longer length of interference and years of dieting and were more likely to regularly use laxatives and have a previous hospitalization. Further, excluded participants reported lower body dissatisfaction, extraversion, openness, and conscientiousness. A smaller proportion of excluded participants participated in day hospital. These excluded participants may have missing values due to survey format or participant oversight. Another explanation may be that these participants had more severe eating disorders and had increased shame/embarrassment related to question content and intentionally did not complete all items. Limitations on the results due to these differences will be discussed.

Table 5

	Excluded Participants	Analysis Sample	Statistical	Test
	$(Ns = 33-56)^{a}$	$(Ns = 137 - 155)^{a}$		
	M (SD)	M (SD)	<i>t</i> (df)	Cohen's d
Admission level variables				
Age	31.88 (13.60)	27.24 (11.97)	t(209) = 2.39*	0.37
Admission BMI	16.01 (2.38)	16.23 (1.90)	t(209) = -0.66	0.10
Highest lifetime BMI	23.00 (4.78)	23.20 (5.58)	t(200) = -0.22	0.04
Length of interference	14.12 (12.70)	9.43 (11.01)	t(193) = 2.50*	0.41
Length of diet	17.87 (13.52)	12.19 (11.91)	t(203) = 2.87 * *	0.46
Drive for thinness	1.76 (0.95)	1.86 (0.99)	t(184) = -0.57	0.10
Body Dissatisfaction	1.61 (0.91)	1.81 (0.93)	t(184) = -1.31	0.22
Normative eating self-	2.02 (1.10)	2.15 (1.14)	t(189) = -0.65	0.11
efficacy				
Neuroticism	2.61 (0.77)	2.61 (0.69)	t(193) = -0.02	0.00
Extraversion	1.92 (0.62)	2.08 (0.63)	t(186) = -1.33	0.26
Openness	2.27 (0.60)	2.39 (0.57)	t(190) = -1.15	0.21
Agreeableness	2.60 (0.55)	2.69 (0.49)	t(186) = -0.91	0.17
Conscientiousness	2.49 (0.64)	2.74 (0.70)	t(185) = -1.98*	0.36
Discharge level variables				
Davs on WGP	27.93 (17.05)	25.79 (18.98)	t(209) = 0.74	0.12
Pounds gained on WGP	14.39 (9.57)	14.01 (9.55)	t(209) = 0.25	0.04
Weekly weight gain rate	3.83 (1.65)	4.03 (1.84)	t(209) = -0.71	0.11
Discharge BMI	18.63 (1.82)	18.80 (1.60)	t(209) = -0.66	0.10

Group Means for Excluded Participants and the Analysis Sample on Continuous Variables.

Note.

^a N varied due to missing data. * p < .05** p < .01

Table 6

	Excluded Participants	Analysis Sample	Statistical Test	
	$(Ns = 37-56)^{a}$	(N = 155)	Statistical	
	Proportion	Proportion	χ^2 (df, N)	Phi coefficient
Admission level variables				
Previous hospitalization	.63	.46	$\chi^2(1, N = 206) = 4.40^*$.15
Purging subtype	.63	.61	$\chi^2(1, N = 211) = 0.06$.02
Regular restricting	.84	.85	$\chi^2(1, N = 192) = 0.04$.02
Regular bingeing	.20	.28	$\chi^2(1, N = 200) = 1.09$.07
Regular vomiting	.38	.41	$\chi^2(1, N = 202) = 0.13$.03
Regular laxative use	.27	.15	$\chi^2(1, N = 200) = 2.96$.12
Clinical Variables				Cramer's V
Weight restoration at	.27	.26	$\chi^2(1, N = 211) = 0.01$.00
discharge				
Participated in Day	.71	.83	$\chi^2(1, N = 211) = 3.15$.12
Hospital				
Weight Gain Group	.46 Optimal	.57 Optimal	$\chi^2(2, N = 211) = 2.45$.11
	.18 Accelerated	.18 Accelerated		
	.36 Sub-optimal	.25 Sub-optimal		

Group Proportions for Excluded Participants and the Analysis Sample on Categorical Variables.

Note.

^a N varied due to missing data. * p < .05

** *p* < .01

Overall bivariate relations

As discussed above, each uncontrolled multinomial logistic regression was estimated using both pairwise and listwise deletions. Table 7 presents overall bivariate relations based on pairwise deletion (first column) and listwise deletion (n = 155; second column). Bivariate relations predominantly remained the same between the pairwise and listwise analyses with the exception of two variables reflecting illness severity. The individual relations between group membership with previous hospitalization and highest lifetime BMI were statistically significant in the reduced sample whereas they had been non-statistically significant with pairwise deletion.

Table 7

Uncontrolled Multinomial Logistic Regressions Estimating Overall Bivariate Relation

Predictor	Likelihood Ratio Test ¹	Likelihood Ratio Test ²
Measures of illness severity		
Age	$\chi^2(2, N = 211) = 2.65$	$\chi^2(2, N = 155) = 4.46$
Length of diet	$\chi^2(2, N = 205) = 3.42$	$\chi^2(2, N = 155) = 2.95$
Length of interference	$\chi^2(2, N = 195) = 4.41$	$\chi^2(2, N = 145) = 4.32$
Previous hospitalization ³	$\chi^2(2, N = 206) = 3.77$	$\chi^2(2, N = 155) = 6.91^*$
Admission BMI	$\chi^2(2, N = 211) = 4.81$	$\chi^2(2, N = 155) = 1.79$
Highest lifetime BMI	$\chi^2(2, N=202) = 5.18$	$\chi^2(2, N = 155) = 10.29 **$
FD behaviors and symptomatol	າດນ	
Behavioral subtype ⁴	$\gamma^2 (2, N = 2.11) = 14.79 **$	$\gamma^2 (2, N = 155) = 18.15^{**}$
Regular restricting ³	$\chi^2(2, N = 192) = 8.93^*$	χ^2 (2, N = 155) = 7.90*
Regular bingeing ³	$\chi^2(2, N = 200) = 6.46^*$	$\chi^2(2, N = 155) = 5.53^*$
Regular vomiting ³	$\chi^2(2, N = 202) = 10.92^{**}$	χ^2 (2, N = 155) = 12.80**
Regular laxative use ³	$\chi^2(2, N = 200) = 6.69^*$	$\chi^2(2, N = 155) = 12.21^{**}$
Drive for thinness	$\chi^2(2, N = 186) = 0.16$	$\chi^2(2, N = 140) = 0.17$
Body dissatisfaction	$\chi^2(2, N = 186) = 0.33$	$\chi^2(2, N = 137) = 1.49$
Normative eating self-efficacy	$\chi^2(2, N = 191) = 0.97$	$\chi^2(2, N = 145) = 2.43$
Personality measures		
Agreeableness	$\gamma^2 (2, N = 188) = 1.51$	$\gamma^2 (2, N = 151) = 1.84$
Conscientiousness	$\chi^2(2, N = 187) = 1.14$	$\chi^2(2, N = 150) = 2.17$
Openness	$\chi^2(2, N = 192) = 1.08$	$\chi^2(2, N = 153) = 4.06$
Extraversion	$\chi^2(2, N = 188) = 9.68^{**}$	$\chi^2(2, N = 155) = 6.48^*$
Neuroticism	$\chi^2(2, N = 195) = 1.82$	$\chi^2(2, N = 154) = 0.42$
Neuroticism	$\chi^2(2, N = 195) = 1.82$	$\chi^2(2, N = 154) = 0.42$

between Group Membership and Predictors

¹ Using pairwise deletion for analyses

 2 Using listwise deletion for analyses for the variables that showed an overall relation from pairwise deletion

³ Previous hospitalization and all weight control methods, coded as 0 = No, 1 = Yes.

⁴ For behavioral subtype, 0 =Restricting, 1 =Purging.

* *p* < .05

** p <.01

Ultimately, the sample considered for the remaining analyses for Aim 2 was comprised of the N = 155 women with valid data on all variables defined above. As suggested above (see Table 6), the primary differences are the reduced sample is
somewhat younger, reporting less interference and length of diet, fewer previous hospitalizations, and higher conscientiousness. With respect to remaining variables, including trajectory group membership, there did not appear to be any pronounced differences.

Pairwise group differences. Table 8 provides the specific pairwise group differences for each of the statistically significant predictors from the uncontrolled regressions (Table 7; column 2).

	Accelerate Optima	ed vs. 11 ¹	Accelerate Sub-optin	ed vs. mal ¹	Sub-optima Optima1	l_{1} vs.
Predictor	β (SE)	OR	β (SE)	OR	β (SE)	OR
Previous hospitalization ²	1.16 (0.46)	3.20*	1.01 (0.52)	2.73†	0.16 (0.39)	1.17
Highest lifetime BMI	0.65 (0.22)	1.91**	0.36 (0.21)	1.43 [†]	0.29 (0.22)	1.33
Behavioral subtype ³	2.47 (0.76)	11.87**	2.31 (0.80)	10.05**	0.17 (0.39)	1.18
Regular restricting ²	-1.56 (0.55)	0.21**	-0.96 (0.60)	0.38	-0.60 (0.58)	0.55
Regular bingeing ²	1.02 (0.45)	2.77*	1.06 (0.54)	2.89*	-0.04 (0.46)	0.96
Regular vomiting ²	1.58 (0.48)	4.83**	1.50 (0.54)	4.46**	0.08 (0.40)	1.08
Regular laxative use ²	1.74 (0.52)	5.68**	1.73 (0.65)	5.66**	0.00 (0.63)	1.00
Extraversion	-0.56 (0.22)	0.57*	-0.46 (0.25)	0.63^{+}	-0.10 (0.20)	0.91

Pairwise Comparisons for Predictors with Significant Overall Bivariate Relations from Listwise Deletion

Note:

OR = Odds ratio

¹Referent group in comparison
² For previous hospitalization and all weight control methods, coded as 0 = No, 1 = Yes.
³ For behavioral subtype, 0 = Restricting, 1 = Purging.

[†] *p* <.10

* *p* < .05

** *p* <.01

Considering the effects of previous hospitalization, highest lifetime BMI, behavioral subtype, regularly restricting, bingeing, vomiting, and using laxatives, and extraversion in prediction of group, the primary effects arise in discriminating the Accelerated group from either the Optimal and/or Sub-optimal groups; none of the predictors discriminated between the Optimal and Sub-optimal groups. From the first panel of Table 8, it is observed that women in the Accelerated group were more likely than women in the Optimal group to: have been hospitalized, utilize bingeing, vomiting, and laxatives, and, consistent with the reported weight control methods more likely to be of the purging subtype. As well, women in the Accelerated group had a higher lifetime BMI. Finally, women in the Accelerated group were less likely to use restricting methods and were less extraverted than women in the Optimal group. These findings remained after controlling for admission BMI.

Differences between women in the Accelerated and Sub-optimal groups paralleled the differences with the Optimal group with subtle differences. Notably, effects of previous hospitalization, highest lifetime BMI and extraversion in discriminating the Accelerated and Sub-optimal groups were only trending (p < .10) and the effect of restricting was not statistically significant in discriminating these groups. These findings also remained after controlling for admission BMI.

In sum, women in the Accelerated group were more likely to be of the purging subtype and engage in these compensatory weight control methods than the rest of the sample. They were also more likely to have a higher lifetime BMI, have been hospitalized in the past, and be less extraverted than the rest of the sample.

Research question 1 Research question 1 investigated whether variables that showed an overall bivariate relation with group membership continued to predict group membership when controlling for other predictors with an overall relation. From above, a model in which previous hospitalization, highest lifetime BMI, behavioral subtype, regularly restricting, bingeing, vomiting, laxative use, and extraversion were included in a model predicting trajectory group. Table 9 provides the unique effect of each predictor in discriminating among the three groups and Table 10 provides pairwise group comparisons for each of the statistically significant predictors from the controlled model. As can be observed, only restricting and laxative use remained statistically significant predictors of group (i.e., displayed unique effects).

Table 9

Multiple Multinomial Logistic Regression between Group Membership and Significant Predictors

Predictor	Likelihood Ratio Test
Full model	$\chi^2(16, N = 155) = 54.92^{**}$
Previous hospitalization ¹	$\chi^2(2, N = 155) = 4.79$
Highest lifetime BMI	$\chi^2(2, N = 155) = 2.73$
Behavioral subtype ²	$\chi^2(2, N = 155) = 3.10$
Regular restricting ¹	$\chi^2(2, N = 155) = 14.25^{**}$
Regular bingeing ¹	$\chi^2(2, N = 155) = 0.18$
Regular vomiting ¹	$\chi^2(2, N = 155) = 0.90$
Regular laxative use ¹	$\chi^2(2, N = 155) = 8.94*$
Extraversion	$\chi^2(2, N = 155) = 4.12$

¹ For previous hospitalization and all weight control methods, coded as 0 = No, 1 = Yes.

² For behavioral subtype, 0 =Restricting, 1 =Purging.

^{*} *p* <.05

^{**} *p* <.01

Pairwise Comparisons for Significant Predictors from the Multiple Multinomial Logistic

Regression

	Accelerated vs.		Accelerated	d vs.	Sub-optimal vs.		
	Optima	al ¹	Sub-optin	nal	Optimal ¹		
Predictor	β (SE)	OR	β (SE)	OR	β (SE)	OR	
Regular restricting ²	-2.85 (0.80)	0.06**	-2.06 (0.81)	0.13*	-0.78 (0.64)	0.46	
Regular laxative use ²	1.80 (0.68)	6.05**	1.91 (0.78)	6.74*	-0.11 (0.69)	0.90	

Note.

OR = Odds ratio

¹Referent group in comparison

 2 For previous hospitalization and all weight control methods, coded as 0 = No, 1 = Yes.

³ For behavioral subtype, 0 =Restricting, 1 =Purging.

- † *p* <.10
- * *p* <.05

** p <.01

For the predictors that had unique effects on group, examination of the pairwise comparisons from the multiple regression provides a parallel understanding as from the series of uncontrolled models. Consistent with the explanation that women in the Accelerated group are more likely to be of the purging subtype, these women were more likely to report less restricting and more laxative use than women in either the Optimal or Sub-optimal groups; no apparent differences on either variable were observed between women in the Optimal and Sub-optimal groups on these predictors.

Another reason the uncontrolled effect of a predictor may no longer remain statistically significant when controlling for other predictors may be due to collinearity among the predictor variables. Table 11 presents the correlations, point-biserial correlations, phi-coefficients, and Cramer's V coefficients among the eight predictors and group membership. Examination of the relations involving restricting behaviors highlights why this predictor may have *retained* a unique effect on group when controlling for other predictors in the model; restricting does not appear related to any of the other predictors in the model whereas vomiting showed moderate to strong relations with three of the other predictors in the model.

Table 11

(Correlations	s among S	Significant I	Predictors fr	rom Logist	ic Regressic	n
			0,0	./	0	0	

	1	2	3	4	5	6	7	8
1. Previous hospitalization ¹								
2. Highest lifetime BMI	.09							
3. Behavioral subtype	.08	.15						
 (0 = Restricting, 1 = Purging) 4. Regularly restricting¹ 	.06	09	.04					
5. Regularly bingeing ¹	02	.08	.47**	07				
6. Regularly vomiting ¹	.07	.23**	.68**	.06	.53**			
7. Regular laxative use ¹	.00	.17*	.35**	.13	.01	.15		
8. Extraversion	19*	08	05	16	11	09	04	
9. Group membership ²	.21*	.27**	.31**	.24*	.20†	.29**	.31**	.20*

$^{+}p < .10$

* *p* <.05

** *p* <.01

¹ For previous hospitalization and all weight control methods, coded as 0 = No, 1 = Yes.

² Relations with dichotomous variables (1, 3, 4, 5, 6, and 7) are Cramer's V; relations with continuous variables (2 and 8) are multiple R in which group is the sole predictor

Brief Discussion

Aim 2 and research question 1 aimed to identify predictors of weight gain

trajectory group membership using multinomial logistic regression. Overall, it was

expected that indicators of illness severity would be predictive of group membership. Further, it was anticipated that increased illness severity, such as older age, longer illness duration, lower admission BMI, higher ED pathology, and previous hospital admission, would be associated with trajectories with slower rates of weight gain, such as the Suboptimal group, due to decreased motivation to change and engagement in treatment. More specifically, it was hypothesized that:

- a) The Optimal group would be comprised of relatively younger women, who engaged in fewer eating disordered behaviors, were more likely to have a purging subtype, higher admission BMI, shorter length of illness, lower drive for thinness and body dissatisfaction, and greater normative eating self-efficacy. It was expected that this group would have the least severe eating disorder.
- b) The Sub-optimal group was hypothesized to have increased illness severity relative to the Optimal group. This group was hypothesized to be comprised of relatively older women, who engaged in more eating disordered behaviors, were more likely to have a restricting subtype, lower admission BMI, longer length of illness, higher drive for thinness and body dissatisfaction, and lower normative eating self-efficacy. It was expected that this group would have the most severe eating disorder (after the Risk-Slow group, which was not observed in the previous aim).
- c) The Accelerated group was hypothesized to be between the Optimal and Sub-optimal groups on admission-level variables which are markers of

illness severity. It was expected that the Accelerated group would appear more similar to the Optimal than the Sub-optimal group.

Results of the analyses suggest previous hospitalization, highest lifetime BMI, behavioral subtype, regularly restricting, bingeing, vomiting, and using laxatives, and extraversion were all related to group membership. With the exception of extraversion, these variables are often used as predictors of treatment response; therefore it is not surprising they are predictive of different patterns of weight gain. The majority of predictor variables (five of the eight predictors) were eating disorder behaviors as opposed to the more cognitive aspects of the disorder. This reflects the influence and impact of behaviors on eating disorder severity and treatment response. The lack of relation between group membership and other predicted variables, such as level of eating disorder pathology (drive for thinness and body dissatisfaction), might be due to women underreporting their level of impairment and cognitive symptoms due to shame and embarrassment.

Contrary to hypotheses, the Optimal and Sub-optimal groups appeared to be similar in terms of eating disorder severity at admission and personality as none of the examined predictors appeared to discriminate between these groups. The Optimal and Sub-optimal groups were both comprised of women with similar admission BMI who primarily restrict. Although these groups appeared similar at admission, given their different pattern of weight gain, they seem to differ in terms of their treatment response. It may be that women in the Optimal and Sub-optimal groups differ with respect to their motivation and engagement in treatment, which was not measured in this study. It may be that women in the Optimal group were more engaged and motivated to recover and

therefore were more compliant with treatment. In contrast, perhaps women in the Suboptimal group were less motivated and engaged, resulting in slower weight gain relative to the Optimal group.

The Accelerated group emerged as the most distinguished among the three trajectory groups. The Accelerated group was generally comprised of women who were more likely to engage in binge/purge behaviors and be of the purging subtype. They were also more likely to have a higher lifetime BMI and have had a previous eating disorders hospitalization. Given this group primarily engaged in purging behaviors as a way to lose or prevent weight gain, it may be their relatively faster weight gain early in treatment is due to gaining water weight and edema (typically reduced in individuals with regular vomiting and/or laxative use). Although the research is mixed, purging behaviors are often associated with increased illness severity when observed in more chronic eating disorders. Further, the Accelerated group had similar admission BMI but significantly higher lifetime BMI suggesting the Accelerated group had the highest weight suppression; defined as the difference between highest premorbid and current treatment weight (Lowe, 1993). As discussed earlier, increased weight suppression is often associated with increased illness severity and increased frequency of behaviors in order to achieve similar weight loss as individuals with lower premorbid weight. It may be that women in the Accelerated group had a higher premorbid weight and therefore had to engage in more eating disorder behaviors, such as bingeing and purging, in order to achieve the same low admission BMI as women with lower weight suppression. These findings suggest the Accelerated group had the highest level of illness severity of the three trajectory groups, which had not been hypothesized.

After controlling for other predictors, regularly restricting and using laxatives were the only significant predictors of group membership; notably, discriminating women in the Accelerated group from the remainder of the sample. Interestingly, certain variables that had been strong predictors of group were no longer predictive after controlling for other variables. Based on the correlations, behavioral subtype, bingeing, vomiting, and laxative use were highly correlated with one another whereas restricting was not related to other predictors. It may be that the effect of subtype in the uncontrolled analyses was driven by purging behaviors. Overall, the Accelerated group emerged as the most distinct relative to the rest of the sample.

Aim 3. Analysis of Variance (ANOVA) and logistic regressions were used to investigate the sequelae of trajectory group membership. These analyses allowed for the estimation of the clinical significance of trajectory groups and ultimately indicate whether certain trajectory groups are associated with better or worse outcomes. ANOVA was used to estimate the effects of trajectory group membership on six differing continuous outcomes; outcomes at discharge were time on weight gain protocol and BMI, and six-month outcomes were BMI, drive for thinness, body dissatisfaction, and level of normative eating self-efficacy. Due to reduced sample size discussed below, partial eta² (η^2), a typical measure of effect size with ANOVA, was used in addition to *p*-level to infer group effects on a given outcome. If results indicated a significant difference between group means at *p* < .10 and/or η^2 > .03, follow-up analyses were conducted to explore all possible pairwise comparisons of means. Logistic regression was used to estimate the effects of trajectory group on nine differing dichotomous outcomes; outcomes at discharge were weight restoration and participation in day hospital, and six-

month outcomes were completion of six-month packet, weight restoration, readmission to an eating disorders unit, and regularly restricting, bingeing, vomiting, and laxative use. Bivariate relations predicting each outcome from group membership were initially estimated. Due to group differences in frequency of engagement in eating disorder behaviors at admission discussed in Aim 2, models predicting eating disorder behaviors at follow-up were also estimated controlling for each respective behavior at admission.

Missing Data. The sample used for this aim was the subsample of 155 participants used in Aim 2. As discussed above, the second time point for data collection in the current study was six-months following discharge from the program. A subset of women (n = 93) who completed the admission packet also completed the six-month packet. These women were compared to those who did not complete the six-month follow-up packet (n = 62) (drop-outs) on admission and discharge level variables and results are presented in Tables 12 and 13. Independent samples t-tests were used to compare continuous variables and Chi-square tests of independence were used to compare categorical variables. Participants who were excluded from analyses due to attrition had lower illness severity at admission as indicated by lower eating disorder pathology, higher normative eating self-efficacy, and were less likely to have had a previous hospitalization. Further, excluded participants reported lower neuroticism and higher extraversion. In terms of discharge-level variables, excluded participants had a faster weight gain rate. Of note, there were no differences in attrition between weight gain groups suggesting groups had equal rates of attrition. Attrition may be due to lack of interest or forgetting to complete the packet of measures. It may also be related to illness severity at follow-up, such that individuals who relapsed and were engaging in eating

disorder behaviors at follow-up were too ill or unwilling to complete the packet. An alternative explanation may be that individuals who were recovered or had decreased illness severity did not complete the packet to avoid focusing on their disorder. Limitations on the results due to these differences will be discussed.

Table 12

Group Means for Excluded Participants and Analysis Sample on Continuous Variables

-	Evoluded	Analysia	Test	
	Dorticipanta	Sampla	Test	
	$(N_a - 54.62)^a$	Sample $(M_2 - 82, 02)^a$		
	(1VS = 34-02)	$(IVS = \delta 3 - 93)$	4(df)	Cohon'a d
	M(SD)	M(SD)	<i>l</i> (d1)	Conen's a
Admission level variables				
Age	27.55 (12.68)	27.03 (11.54)	t(153) = 0.26	0.04
Admission BMI	16.12 (2.07)	16.30 (1.78)	t(153) = -0.57	0.09
Highest lifetime BMI	23.37 (5.94)	23.09 (5.34)	t(153) = 0.31	0.05
Length of illness	10.03 (12.54)	9.04 (9.95)	t(143) = 0.53	0.09
Length of diet	12.48 (13.05)	12.00 (11.16)	t(151) = 0.25	0.04
Drive for thinness	1.73 (1.11)	1.94 (0.90)	t(138) = -1.23	0.21
Body Dissatisfaction	1.67 (1.02)	1.90 (0.87)	t(135) = -1.37	0.24
Eating self-efficacy	2.32 (1.23)	2.04 (1.07)	t(143) = 1.44	0.24
Neuroticism	2.50 (0.71)	2.69 (0.68)	t(152) = -1.71	0.28
Extraversion	2.28 (0.52)	1.94 (0.70)	t(153) = 3.37 * *	0.55
Openness	2.35 (0.56)	2.41 (0.59)	t(151) = -0.64	0.11
Agreeableness	2.74 (0.52)	2.65 (0.46)	t(149) = 1.14	0.19
Conscientiousness	2.79 (0.70)	2.71 (0.71)	t(148) = 0.70	0.12
Discharge level variables				
Days on WGP	24.71 (19.95)	26.51 (18.38)	t(153) = -0.58	0.09
Pounds gained on WGP	14.35 (10.25)	13.78 (9.11)	t(153) = 0.36	0.06
Weekly weight gain rate	4.27 (1.96)	3.88 (1.75)	t(153) = 1.30	0.21
Discharge BMI	18.67 (1.81)	18.88 (1.44)	t(153) = -0.78	0.13
Nata				

Note.

^a N varied due to missing data.

** *p* < .01

	Excluded Participants	Analysis Sample	Statistical	Test
	(N = 62)	(N = 93)	2 (10))	
	Proportion	Proportion	χ^2 (df, N)	Phi-coefficient
Admission level variables				
Previous hospital	.35	.53	$\chi^2(1, N = 155) = 4.44^*$.17
Purging subtype	.57	.63	$\chi^2(1, N = 155) = 0.76$.07
Regular restricting	.79	.89	$\chi^2(1, N = 155) = 3.07$.14
Regular bingeing	.21	.32	$\chi^2(1, N = 155) = 2.37$.12
Regular vomiting	.37	.44	$\chi^2(1, N = 155) = 0.75$.07
Regular laxative use	.16	.15	$\chi^2(1, N = 155) = 0.03$.02
Participated in Day Hospital	.79	.85	$\chi^2(1, N = 155) = 0.90$.08
Weight restored at discharge	.26	.27	$\chi^2(1, N = 155) = 0.02$.01
				Cramer's V
Weight Gain Group	.60 Optimal	.55 Optimal	$\chi^2(2, N = 155) = 0.97$.08
	.19 Accelerated	.17 Accelerated		
	.21 Sub-optimal	.28 Sub-optimal		

Group Proportions for Excluded Participants and the Analysis Sample on Categorical Variables.

Note. * *p* < .05 ** *p* < .01

Overall effect of treatment. Prior to examining group differences on treatment outcomes, analyses were conducted to explore the overall effect of treatment (see Table 14). A series of paired samples t-test for continuous variables and McNemar's test for paired dichotomous variables were used to compare admission and six-month outcomes on the women who completed the six-month packet. Results suggest a reduction in proportion of women who regularly: restrict, binge, vomit, and use laxatives. Further, compared to admission level, women reported higher BMI, lower drive for thinness and body dissatisfaction, and higher normative eating self-efficacy. These results suggest an overall positive effect of treatment on eating disorders and reduction of illness severity.

Overall Effects of Treatment

Variable	Admission	Six-months		Test	
	M(SD)	M(SD)	r	<i>t</i> (df)	Cohen's d
Continuous variables					
BMI	16.24 (1.83)	19.04 (2.33)	.50**	t(84) = 12.09 * *	1.31
Drive for thinness	1.94 (0.90)	1.33 (1.02)	.52**	$t(74) = -5.62^{**}$	0.65
Body Dissatisfaction	1.92 (0.85)	1.51 (0.92)	.69**	$t(72) = -4.96^{**}$	0.58
Eating self-efficacy	2.05 (1.08)	2.67 (1.24)	.55**	$t(81) = 5.02^{**}$	0.55
Dichotomous variables	Proportion	Proportion		χ^2 (df, N)	Phi-coefficient
Regular restricting	.89	.52		$\chi^2(1, N = 88) = 27.68^{**}$.56
Regular bingeing	.32	.19		$\chi^2(1, N = 90) = 6.72^{**}$.27
Regular vomiting	.44	.30		$\chi^2(1, N = 91) = 8.47 **$.31
Regular laxative use	.15	.10		$\chi^2(1, N = 91) = 1.07$.11

* p < .05 ** p <.01

Group differences. Tables 15 through 17 provide the results of the ANOVA and logistic regressions used to determine the overall predictive ability of trajectory groups on treatment outcomes. Trajectory group membership predicted BMI at both discharge and follow-up as well as drive for thinness (see Table 15). Examination of the pairwise group comparisons suggest women in the Accelerated group had higher BMI at both discharge and follow-up; no apparent differences in BMI between the Optimal and Sub-optimal groups were observed at either time point. With respect to drive for thinness, women in the Sub-optimal group were lower on this measure than the rest of the sample (Table 15). Group membership also predicted weight restoration at both discharge and follow-up, as well as regular bingeing (Table 16). Further examination revealed that women in the Accelerated group were more likely to be weight restored at both time points though also more likely to report regular bingeing; no apparent differences between women in the Optimal and Sub-optimal groups were observed on these outcome measures.

Table of Means and ANOVA Results Comparing Groups on Outcome Variables

Variable	Accelerated	Optimal	Sub-optimal	Test		Pairwis	se
	$(Ns = 13-28)^{a}$	$(Ns = 47-88)^{a}$	$(Ns = 22-39)^{a}$			Comparis	sons
	M (SE)	M (SE)	M (SE)	F(df)	η²	<i>p</i> < .10	η²
Discharge Outcomes							
Days on WGP	22.75 (3.60)	25.75 (2.03)	28.05 (3.05)	F(2, 152) = 0.63	.01		
Discharge BMI	19.77 (0.29)	18.71 (0.16)	18.29 (0.25)	F(2, 152) = 7.98 **	.10	Acc > Opt	.06
-						Acc > Sub	.09
Six month follow-up O	outcomes						
BMI	20.75 (0.57)	18.61 (0.32)	18.82 (0.46)	$F(2, 82) = 5.46^{**}$.12	Acc > Opt	.11
						Acc > Sub	.08
Drive for thinness	1.40 (0.28)	1.44 (0.15)	0.95 (0.21)	F(2, 79) = 1.91	.05	Acc > Sub	.02
						Opt > Sub	.05
Body dissatisfaction	1.56 (0.25)	1.53 (0.13)	1.29 (0.20)	F(2, 80) = 0.61	.02		
Normative eating self-efficacy	2.63 (0.33)	2.59 (0.18)	2.82 (0.27)	F(2, 84) = 0.25	.01		

Note.

^a N varied due to missing data. * p < .05** p < .01

Logistic Regressions Estimating Overall Relation between Group Membership and Outcomes

Outcome	Likelikeed Detie Test
Outcome	Likelihood Ratio Test
Discharge outcomes	
Weight restored ¹	$\chi^2(2, N = 155) = 15.55 **$
Participation in day hospital ¹	$\chi^2(2, N = 155) = 0.46$
Six month follow-up outcomes	
Weight restored ¹	$\chi^2(2, N = 85) = 11.31^{**}$
Completion of six-month packet ¹	$\chi^2(2, N = 155) = 0.99$
Readmission to eating disorder unit ¹	$\chi^2(2, N = 91) = 1.54$
Regular restricting ^{1, 2}	$\chi^2(2, N=88)=0.34$
Regular bingeing ^{1, 2}	$\chi^2(2, N=90) = 4.63^{\dagger}$
Regular vomiting ^{1, 2}	$\chi^2(2, N=91) = 1.89$
Regular laxative use ^{1, 2}	$\chi^2(2, N=91) = 3.87$
¹ Coded as $0 = No, 1 = Yes.$	
² Provided estimates control for respective behavior at admission	
$^{\dagger} p < .10$	
* <i>p</i> < .05	
** <i>p</i> <.01	

Pairwise Comparisons for Outcomes with Significant or Trending Bivariate Relations

	Accelerat Optim	ed vs. al ¹	Accelerated optima	vs. Sub- d ¹	Sub-optima Optima	al vs. 1 ¹
Outcome	β (SE)	OR	β (SE)	OR	β (SE)	OR
Weight restored at discharge ²	1.58 (0.46)	4.84**	1.99 (0.59)	7.33**	-0.42 (0.51)	0.66
Weight restored at follow-up ²	2.08 (0.67)	8.02**	1.84 (0.74)	6.29*	0.24 (0.56)	1.28
Regular bingeing ²	1.40 (0.80)	4.03^{\dagger}	1.84 (0.94)	6.30^{\dagger}	-0.45 (0.83)	0.64

Note:

OR = Odds ratio

¹Referent group in comparison ² All variables coded as 0 = No, 1 = Yes.

p < .10* p < .05** p < .01

In sum, women in the Accelerated group had better weight outcomes at discharge and follow-up (both BMI and weight restoration); however these women were more likely to binge. Finally, women in the Sub-optimal group reported lower drive for thinness than women in either of the other groups.

Research Question 2. Research question 2 investigated whether relations between trajectory group membership and discharge/six-month outcomes remain after controlling for relevant covariates. Covariates included age, behavioral subtype, previous hospitalization, and (for six-month outcomes) participation in day hospital. These covariates were selected based on theoretical and statistical relations with treatment outcomes (see Table 18). For eating disorder behaviors, baseline level of the respective behavior was also included as a covariate. Additionally, for regularly bingeing and vomiting at follow-up, behavioral subtype was removed as a covariate due to collinearity between subtype and both bingeing and vomiting (r = .47 and .68 respectively, ps < .01). Finally as part of this research question, day hospital participation was examined as a possible moderator on the effects of trajectory group membership on each of the sixmonth outcomes.

		Age	5	Previous	Hospitali	zation	Behav	vioral Subt	уре	Da	y Hospita	ıl
Outcome Variables	β	χ^2	OR	β	χ^2	OR	β	χ^2	OR	β	χ^2	OR
Discharge ($N = 155$)												
Weight restored ¹	0.41	5.78*	1.51	0.79	3.69	2.21	0.88	3.53	2.40			
Participation in day hospital ¹	-0.48	8.21**	0.62	0.24	0.25	1.26	1.00	4.45*	2.71			
Six-month ($N = 85$)												
Weight restored ¹	0.04	0.03	1.04	-0.15	0.08	0.86	0.03	0.00	1.03	1.24	2.52	3.45
Completion of six- month packet ¹	-0.06	0.17	0.94	0.79	5.06*	2.20	0.30	0.66	1.35	0.32	0.49	1.38
Readmission to eating disorder unit ¹	-0.19	0.53	0.83	0.39	0.46	1.48	-0.20	0.12	0.82	0.46	0.32	1.58
Regular restricting ¹	0.20	0.99	1.22	0.46	0.97	1.58	0.75	2.41	2.13	-0.14	0.05	0.87
Regular bingeing ¹	-1.36	9.02**	0.26	-0.04	0.00	0.96				0.32	0.07	1.37
Regular vomiting ¹	-0.20	0.45	0.82	-0.13	0.05	0.88				-0.14	0.03	0.87
Regular laxative use ¹	0.12	0.12	1.12	0.24	0.10	1.27	0.92	1.25	2.51	-0.43	0.22	0.65
Continuous outcomes	F	β	η²	F	β	η^2	F	β	η^2	F	β	η²
Discharge ($N = 155$)												
Days on WGP	1.26	1.36	.01	26.40**	15.03	.15	0.86	-2.81	.01			
BMI	10.10**	0.31	.06	2.68	0.39	.02	14.03**	0.93	.09			
Six-month $(N = 85)$												
BMI	0.12	0.07	.00	0.07	-0.13	.00	0.00	-0.02	.00	13.53**	2.32	.15
Drive for thinness	0.34	-0.06	.00	3.14	0.40	.04	7.67**	0.64	.01	0.05	-0.07	.00
Body dissatisfaction	0.21	0.04	.00	7.41**	0.51	.09	17.01**	0.81	.18	1.76	-0.12	.04
Normative eating self- efficacy	0.64	-0.10	.01	0.45	-0.19	.01	5.05	-0.66	.06	0.65	0.48	.02

Table 18 Results for Covariates from ANCOVA and Logistic Regressions

¹All variables coded as 0 = No, 1 = Yes.

Group membership and outcomes with potential covariates. Analysis of covariance (ANCOVA) was used to estimate the unique effects of group membership on continuous outcomes. Multiple logistic regressions were used to estimate unique effects of group on dichotomous outcomes. Tables 19 through 21 provide the results of the ANCOVA and logistic regressions. In terms of continuous outcomes, trajectory group membership uniquely predicted days on weight gain protocol, BMI at both discharge and follow-up, drive for thinness, and body dissatisfaction (see Table 19). Examination of the pairwise group comparisons suggests women in the Accelerated group spent fewer days on weight gain protocol relative to the rest of the sample. Woman in the Accelerated group had higher BMI at discharge relative to the Suboptimal group and at follow-up relative to the rest of the sample; no differences between the Optimal and Sub-optimal groups at either time point were observed. With respect to drive for thinness, women in the Optimal group were higher on this measure than the rest of the sample. With respect to body dissatisfaction, women in the Accelerated group were lower on this measure than the Sub-optimal group. In terms of dichotomous outcomes, group membership predicted weight restoration at both discharge and follow-up (Table 20). Further examination revealed that women in the Accelerated group were more likely to be weight restored at both time points; no differences in weight between the Optimal and Sub-optimal groups at either time point were observed.

Table of Means and ANCOVA Results Comparing Groups on Outcome Variables

Outcome	Accelerated $(N_{\rm S} = 13-28)^{\rm a}$	Optimal $(N_{\rm S} = 47-88)^{\rm a}$	Sub-optimal $(N_{\rm S} = 22-39)^{\rm a}$	Test		Pairwise	
	$\frac{(118 - 13 20)}{M(SE)^{b}}$	$M(SE)^{\rm b}$	$\frac{(1+S)^2}{M(SE)^b}$	$F(\mathrm{df})$	η_p^2	p < .10	η^2
Discharge outcomes							
Days on WGP	19.75 (3.52)	26.60 (1.89)	28.28 (2.80)	F(2, 149) = 1.89	.03	Acc < Opt	.02
						Acc < Sub	.02
Discharge BMI	19.25 (0.29)	18.86 (0.15)	18.34 (0.23)	$F(2, 149) = 3.40^{*}$.05	Acc > Sub	.04
Six month follow-up or	utcomes						
BMI	20.53 (0.60)	18.62 (0.31)	18.93 (0.44)	$F(2, 78) = 3.81^*$.09	Acc > Opt	.09
		(111)				Acc > Sub	.05
Drive for thinness	1.08 (0.29)	1.50 (0.14)	1.03 (0.21)	F(2, 75) = 2.12	.05	Acc < Opt	.02
						Opt > Sub	.05
Body dissatisfaction	1.11 (0.25)	1.60 (0.12)	1.40 (0.18)	F(2, 76) = 1.76	.04	Acc < Opt	.04
Normative eating self-efficacy	2.94 (0.35)	2.53 (0.18)	2.75 (0.27)	F(2, 80) = 0.65	.01		

Note.

^a N varied due to missing data. ^b Adjusted means provided * p < .05** p < .01

Multiple Lo	ogistic	Regression	Predicting	Outcomes from	Group Memb	bership	Controlling fo	r Covariates
-------------	---------	------------	------------	---------------	------------	---------	----------------	--------------

Outcomes	Likelihood Ratio Test				
	Full Model	Unique effect of group			
Discharge outcomes					
Weight restored ¹	$\chi^2(5, N = 155) = 29.58^{**}$	$\chi^2(2, N = 155) = 7.04*$			
Participation in day hospital ¹	$\chi^2(5, N = 155) = 13.91^*$	$\chi^2(2, N = 155) = 0.32$			
Six month follow-up outcomes					
Weight restored ¹	$\chi^2(6, N = 85) = 14.01^*$	$\chi^2(2, N=85) = 8.27*$			
Completion of six-month packet ¹	$\chi^2(6, N = 155) = 7.73$	$\chi^2(2, N = 155) = 1.79$			
Readmission to eating disorder unit ¹	$\chi^2(6, N = 91) = 2.95$	$\chi^2(2, N = 91) = 1.84$			
Regular restricting ¹	$\chi^2(7, N = 88) = 10.35$	$\chi^2(2, N = 88) = 1.63$			
Regular bingeing ^{1, 2}	$\chi^2(6, N = 90) = 39.83^{**}$	$\chi^2(2, N=90) = 4.49$			
Regular vomiting ^{1, 2}	$\chi^2(6, N = 91) = 43.36^{**}$	$\chi^2(2, N = 91) = 1.71$			
Regular laxative use ¹	$\chi^2(7, N=91) = 10.43$	$\chi^2(2, N=91) = 4.47$			

Note.

For eating disorder behaviors, estimates provided control for respective behavior at admission. ¹ Coded as 0 = No, 1 = Yes. ² Model estimated without behavioral subtype as a covariate.

* p < .05** p < .01

Unique Effects of Predictors with Significant Unique Relations

	Accelerated vs. Optimal ¹		Accelerated vs. Sub- optimal ¹		Sub-optimal vs. Optima1 ¹	
Outcomes	β (SE)	OR	β (SE)	OR	β (SE)	OR
Weight restored at discharge ²	0.97 (0.51)	2.62^{\dagger}	1.57 (0.62)	4.79*	-0.61 (0.55)	0.55
Weight restored at follow-up ²	2.04 (0.76)	7.67**	1.75 (0.81)	5.74*	0.29 (0.58)	1.34

Note.

OR = Odds ratio ¹ Referent group in comparison ² All variables coded as 0 = No, 1 = Yes.

 $^{\dagger} p < .10$ * p < .05** p < .01

Moderating effect of day hospital. Potential differences in the effect of group membership on six-month outcomes due to participation in day hospital were investigated. Distributions of women in trajectory groups across levels of participation in day hospital were initially examined. For the Accelerated group, all but one woman participated in day hospital. Based on this distribution and lack of valid data for followup outcomes, the Accelerated group was omitted from moderation analyses. Moderating effects of day hospital participation on six-month continuous outcomes (BMI, drive for thinness, body dissatisfaction, and normative eating self-efficacy) were estimated using a series of 2 x 2 ANOVAs or ANCOVAs; as relevant, models included the respective baseline behavior or pathology. Moderating effects of day hospital participation on sixmonth dichotomous outcomes (weight restoration, completion of six-month packet, readmission to eating disorders unit, and regular restricting, bingeing, vomiting, and laxative use) were estimated using a series of logistic regressions; as relevant, models included the respective baseline behavior or pathology. For models with significant interactions, simple effects of participation in day hospital at levels of group were evaluated to further understand the moderating effect of day hospital following guidelines suggested by Aiken and West (1991).

There was a significant interaction between the effects of group membership and participation in day hospital on BMI (F[1, 65] = 9.27, p = .003, $f^2 = .13$), drive for thinness (F[1, 58] = 3.15, p = .08, $f^2 = .05$), and normative eating self-efficacy (F[1, 63] = 4.75, p = .03, $f^2 = .07$) (see Figures 3-5). Analysis of simple effects indicated the effect of day hospital for the Sub-optimal group was significant for BMI (F[1, 20] = 7.15, p = .02, $f^2 = .26$), drive for thinness (F[1, 17] = 4.68, p = .04, $f^2 = .22$), and normative eating self-

efficacy (*F*[1, 18] = 7.42, p = .01, f^2 = .29). These simple effects were not found in the Optimal group (*p*s > .05).



Figure 3. Simple effects of group on six-month BMI separately for participation in day hospital.



Figure 4. Simple effects of group on six-month drive for thinness separately for participation in day hospital.



Figure 5. Simple effects of group on six-month normative eating self-efficacy separately for participation in day hospital.

Results suggest there was no apparent benefit of day hospital participation for women in the Optimal group. However, for women in the Sub-optimal group, relative to women who did not attend day hospital, those who attended day hospital had higher BMI and normative eating self-efficacy and lower drive for thinness

Brief Discussion

Aim 3 and research question 2 estimated the clinical significance of trajectory groups on treatment outcomes. Overall, it was expected that group membership would be predictive of differences in discharge and six-month follow-up outcomes and that certain groups would be associated with better, or worse, treatment outcomes. More specifically, it was hypothesized that:

- a) The Optimal group would be associated with relatively positive treatment outcomes. I predicted the Optimal group would be more likely to achieve target weight, have higher BMI, and be more likely to attend day hospital. Considering six-month outcomes, I expected the Optimal group would be more likely to have maintained target weight, have higher BMI, be less likely to relapse on ED behaviors, less likely to require rehospitalization, have lower drive for thinness and body dissatisfaction, and higher normative eating self-efficacy than all other groups.
- b) The Sub-optimal group was hypothesized to be associated with more negative outcomes than the Optimal group. It was expected that this group would have the worst discharge and six-month outcomes (after the Risk-slow group, which was not observed).

c) The Accelerated group was hypothesized to be associated with both positive and negative outcomes. It was expected that at discharge, this group would be associated with more positive outcomes and appear more similar to the Optimal group (than the Risk-slow group). However, at follow-up it was expected that this group would be associated with more negative outcomes and be more similar to the Risk-slow (not observed) than the Optimal group.

Prior to examining group differences, the overall effect of treatment was investigated. Results suggest a positive effect of treatment in reducing eating disorder severity as evidenced by higher BMI, reduction in eating disorder behaviors, lower levels of eating disorder pathology (drive for thinness and body dissatisfaction), and higher normative eating self-efficacy. However, these analyses were completed on a reduced sample as about 40% (n = 62) of the women were excluded due to attrition. Limitations of this will be discussed later in the discussion section.

Results of this aim suggest group membership was predictive of BMI at discharge and follow-up, weight restoration at discharge and follow-up, regularly bingeing, and drive for thinness. Contrary to hypotheses, overall the Optimal and Sub-optimal groups generally appeared to be similar in terms of treatment outcomes with the exception of women in the Sub-optimal group endorsing lower drive for thinness at follow-up. Given these groups were similar on weight and behavioral outcomes at follow-up, women in the Sub-optimal group, characterized by slower weight gain, may have required more time in order to treat their disorder. Though clinicians often think slow weight gain is indicative of relapse, it appears these women are capable of recovery but simply require more time to do so as evidenced by the benefit of day hospital participation for these women.

The Accelerated group emerged as the most distinguished among the three trajectory groups and was associated with both positive and negative outcomes. Women in the Accelerated group had higher BMI and were more likely to be weight restored at discharge and follow-up relative to the rest of the sample. However, the Accelerated group was more likely to regularly binge relative to the rest of the sample. Results suggest the Accelerated group are women who entered treatment frequently bingeing who then returned to this behavior after treatment discharge. In addition, the Accelerated group endorsed higher drive for thinness relative to the Sub-optimal group, suggesting women in the Accelerated group did not improve as much on this psychological aspect of the disorder. Though preliminary, these results suggest clinicians should not rely solely on weight as markers of treatment response and recovery given the group with the most successful weight outcomes may be more likely to relapse.

After controlling for relevant covariates, the Accelerated group remained the most distinguished group between the three trajectory groups, with similar relations between group and outcomes as the uncontrolled analyses. In addition, group membership predicted days on weight gain protocol. The Accelerated group was on weight gain protocol for a shorter period of time than the rest of the sample. The shorter length of stay is likely a function this group's faster rate of weight gain.

Moderation analyses indicated that day hospital helped to increase BMI and normative eating self-efficacy and reduce drive for thinness in the Sub-optimal group and did not have an apparent effect on the Optimal group. Day hospital may have provided additional opportunities to achieve and maintain recovery for women in the Sub-optimal group who had a slower rate of weight gain and thus slower treatment response while

inpatient. It may be that women in the Optimal group entered treatment motivated and reduced their illness severity during the inpatient phase. These results suggest certain women require additional, though less intensive, treatment to achieve recovery. However given sample size was small for these analyses results should be interpreted with caution.

Discussion

The current study examined patterns of weight gain during inpatient treatment for anorexia nervosa and the effect of patterns on treatment outcomes. Recent studies suggest weight gain patterns may be predictive of post-treatment functioning and serve as an intreatment marker of relapse (Hartmann et al., 2007; Lay et al., 2004; Le Grange et al., 2014). However, research on weight gain patterns have been limited to small sample sizes, have failed to include psychological variables, and/or only focused on outpatient treatment (Accurso et al., 2014; Lay et al., 2004; Vansteelandt et al., 2010). This study aimed to extend previous research and determine if weight gain trajectories provide valuable information about both short and long-term outcomes following inpatient treatment. Identifying and describing weight gain trajectory groups associated with poor outcomes may allow treatment programs to identify those patients at increased risk of negative outcomes and address the increased risk prior to discharge, thereby possibly reducing the likelihood of relapse and rehospitalization.

Pattern of Weight Gain. Mixture modeling was used to investigate individual differences in weight gain in a hospitalized sample of women with AN or subthreshold AN. Three distinct patterns of weight gain were identified; a negative quadratic trajectory (Optimal group), a negative quadratic trajectory with a faster initial rate of weight gain

(Accelerated group), and a positive linear trajectory (Sub-optimal group). The Optimal group had an average weight gain rate that was within treatment expectations (4 to 5 pounds per week) whereas the Accelerated and Sub-optimal groups had average weight gain rates that were well above and below treatment expectations, respectively. Further, weight gain groups differed in their length of stay, with the Accelerated group being on weight gain protocol for the shortest amount of time while the Sub-optimal group spent the longest amount of time. Differing lengths of stay reflect women in each group reaching their target weight and transitioning to day hospital or being discharged at different rates. These distinct patterns, weight gain rates, and lengths of stay suggest groups have varying responses to treatment. Based on prior studies that found faster rates of weight gain were an indicator of better response (Castro et al., 2004; Lund et al., 2009; Mewes et al., 2008), descriptive statistics suggest the Accelerated group had the best response whereas the Sub-optimal group had the worst response and appeared to be the most treatment resistant, as indicated by slowest weekly rate of weight gain, longest length of treatment, and least pounds gained while on weight gain protocol. Although the treatment program utilized a unified behavioral protocol in which all patients must adhere to the same rules, requirements, and expectations, women recovering from AN appear to gain weight differently.

Predictors of Weight Gain Pattern. Age, length of dieting, length of illness interference, agreeableness, conscientiousness, openness, neuroticism, and BMI, drive for thinness, body dissatisfaction, at normative eating self-efficacy at admission, were not predictive of trajectory group membership. The lack of relation between pattern of weight gain and eating disorder pathology (drive for thinness and body dissatisfaction) may be

due to participants underreporting their level of impairment and cognitive symptoms related to their disorder due to shame or embarrassment. An alternative explanation may be that since the treatment program's primary focus is on changing the behavioral aspects of the disorder, the more cognitive aspects, such as drive for thinness, body dissatisfaction, and normative eating self-efficacy may not impact treatment response as measured by weight gain. Although eating disorder pathology has been predictive of relapse (Garner, 2004; Stice & Shaw, 2002; Thompson, 2004), this result is in line with previous studies that found drive for thinness did not predict changes in weight gain trajectory (Vansteelandt et al., 2010).

Previous hospitalization, highest lifetime BMI, eating disorder behavioral subtype, regularly restricting, bingeing, vomiting, and using laxatives, and extraversion were all related to group membership. With the exception of extraversion, these predictors are often used as markers of illness severity. These findings are consistent with prior research demonstrating the relation between behavioral subtype and illness severity and weight gain during treatment (Accurso et al., 2014; Lay et al., 2002). Comparing groups, the Optimal and Sub-optimal groups appeared to be relatively similar at admission in terms of ED severity and personality. Although these women appear similar at admission, they had different treatment responses as reflected in their distinct patterns of weight gain. This discrepancy in treatment response may be due to differing levels of motivation and engagement in treatment. Women in the Optimal group may have entered treatment with higher levels of motivation and were more compliant with treatment and as a result gained weight faster and had shorter lengths of stay.

The Accelerated group was the most distinct among the three groups at admission. Women in the Accelerated group were more likely to be of the purging subtype, engage in binge/purge behaviors, and have had a previous hospitalization. The Accelerated group appeared to have higher illness severity at admission relative to the rest of the sample; however this group also appeared to have the best treatment response in terms of their rate of weight gain and shorter length of stay. This group's negative quadratic trajectory with faster weight gain may in part be due to individuals with AN who frequently engage in purging behaviors gaining weight faster than those who do not engage in purging behaviors (Neuberger, Rao, Weltzin, Greeno, & Kaye, 1994). Another explanation may be more biological. Individuals in the Accelerated group also had a higher average lifetime BMI. Higher lifetime BMI, coupled with similar admission BMI, suggests increased weight suppression, another indicator of increased illness severity (Lowe, 1993). Thus, rapid weight gain early in treatment may be due to these women's increased weight suppression, as reflected by their higher maximum weight prior to the onset of their eating disorder; therefore these individuals may have gained weight faster because they had a higher premorbid weight.

An unexpected finding was that individuals in the Accelerated group appeared to have lower extraversion than the rest of the sample. This may be an unanticipated artifact based on the measurement of personality. Individuals who engage in bingeing and purging behaviors often have increased shame, embarrassment, and guilt around these behaviors (Cash & Deagle, 1997; Cash & Pruzinsky, 2004). As a result, they tend to isolate more and withdraw from social situations and relationships due to these behaviors. The specific items in the NEO measuring extraversion focus on social activities centered

on food and social gatherings, such as "I like to have a lot of people around me," "I like to try new and foreign foods," and "I really enjoy talking to people." Due to the shame and guilt associated with regularly bingeing, vomiting, and using laxatives as well as avoidance of food, individuals with AN who engage in these behaviors may be more withdrawn and isolative than individuals with AN who primarily restrict.

After controlling for other predictors, regularly restricting and laxative use were the only significant predictors of group membership. The Accelerated group remained the most distinct and was more likely to have engaged in laxative use and less likely to restrict relative to the rest of the sample. The effects of other predictors may have dropped due to collinearity among predictors; behavioral subtype, bingeing, vomiting and laxative use were highly correlated with one another whereas restricting was not correlated with other predictors.

Program effects. Prior to examining group differences in treatment outcomes, the overall effect of treatment was investigated. Results suggest an overall positive effect of treatment in reducing eating disorder severity. Women endorsed a reduction in illness severity as evidenced by a higher BMI at both discharge and follow-up, reduction in restricting, bingeing, vomiting, and laxative use, lower levels of eating disorder pathology (drive for thinness and body dissatisfaction), and higher normative eating self-efficacy. The sample's follow-up BMI (M = 19.04, SD = 2.33) was close to the program's target weight (BMI = 20) and above the commonly used marker of anorexia (BMI = 18.5). These results suggest overall treatment response was positive and that the program had a significant effect on treating women's eating disorders.
Outcomes of Weight Gain Pattern. Weight gain groups appeared to differ in their treatment outcomes and group membership was predictive of BMI at discharge and follow-up, weight restoration at discharge and follow-up, regularly bingeing, and drive for thinness. Similar to the previous aim, the Optimal and Sub-optimal groups generally appeared to be similar in terms of treatment outcomes though there were some differences, including women in the Sub-optimal group endorsed lower drive for thinness at follow-up. Though women in the Sub-optimal and Optimal groups differed in average weight gain, at six-month follow-up, the Optimal and Sub-optimal groups did not differ on any weight or behavioral outcomes. Moreover, moderation analyses indicated women in the Sub-optimal had a stronger positive response to day hospital relative to the Optimal group. Day hospital helped to increase BMI and normative eating self-efficacy and reduce drive for thinness in the Sub-optimal group with no apparent effects in the Optimal group. Thus, day hospital may have provided additional opportunities to achieve and maintain recovery for women in the Sub-optimal group, allowing them to catch up to the Optimal group following their slower inpatient treatment response. Results suggest day hospital is a necessary and crucial treatment option for women in the Sub-optimal group who have an initially slower treatment response, allowing them make the necessary gains in recovery to reduce relapse prior to discharge. Though clinicians often think slow weight gain is indicative of resistance to treatment, possibly relapse, and chronicity of the disorder, it appears these women are capable of recovery and positive treatment response but simply require more time to do so.

As at admission, the Accelerated group emerged as the most distinct among the three trajectory groups at discharge and follow-up. The Accelerated group was associated

with both positive and negative outcomes. Women in the Accelerated group had higher BMI and were more likely to achieve weight restoration at discharge and follow-up; however they were also more likely to engage in regular bingeing at follow-up. As discussed above, weight restoration is a pivotal and necessary factor in recovery and thus higher BMI and weight restoration are considered positive outcomes. The Accelerated group's increased likelihood of behavioral relapse was unexpected given they had the strongest treatment response. Results suggest the Accelerated group are women who entered treatment frequently bingeing who then relapsed after treatment discharge. It may be the Accelerated group had a higher BMI at follow-up due to regular bingeing, as this behavior is an ineffective weight loss strategy and can cause weight gain and/or minimal weight loss. Additionally, the Accelerated group was on weight gain protocol for a shorter period of time than the rest of the sample. It may also be that the Accelerated group, who had increased illness severity at admission relative to both groups, focused on the biological aspects of treatment (weight gain) without internalizing treatment objectives, such as normalized eating patterns and blocking urges to act on one's eating disorder, as much as other groups.

Overall, it appears women in the Accelerated group responded positively to treatment and achieved weight recovery but continued to struggle with some behavioral aspects. Given weight recovery typically occurs before behavioral/psychological recovery (Coutrier & Lock, 2006), it may also be Accelerated group's behavioral functioning is in the process of recovering and occurred slower than the Optimal and Sub-optimal groups. Importantly, given the Accelerated group reported better weight outcomes but were more likely to relapse, results suggest weight outcomes should not be

the sole measure of recovery and risk of relapse. In sum, it appears weight gain trajectory groups respond to treatment differently for different aspects of the disorder. The Optimal group overall responded well to treatment and were able to maintain those improvements at follow-up. The Sub-optimal group had slower treatment response but was able to make the same gains as the Optimal group and maintain them at follow-up with the help of day hospital. The Accelerated group had the most positive response in terms of weight outcomes; however continued to struggle with bingeing at follow-up. These preliminary findings suggest pattern of weight gain is an important in-treatment marker as not every woman recovers and improves at the same rate and certain subgroups may need more intensive treatment. These results though preliminary suggest clinicians should not rely solely on weight as markers of treatment response and recovery given the group with the most successful weight outcomes was also more likely to return to pre-treatment behaviors.

Limitations. This study had several limitations worth noting. The majority of variables assessed via self-report were ones that are typically under or over reported within this population, such as weight, frequency and intensity of disordered eating, and ED pathology. Although clinical outcomes at discharge (weight, reaching target weight, days on weight gain protocol) were documented by nursing staff and collected via chart review, all outcomes at follow-up were collected via self-report. It is possible that individuals underreport behaviors and pathology and over report weight. As a result, these estimates may not be an accurate assessment of participants functioning and recovery/relapse at follow-up.

Another limitation was missing data and attrition. A substantial percentage (26%) of the original sample was excluded from Aim 2 analyses due to missing data at admission. It is unclear why missing data were present, though it was found that those excluded endorsed lower levels of openness and conscientiousness relative to the analysis sample. Additionally in Aim 3, a significant proportion of the subsample (40%) was excluded from analysis due to attrition at six-month follow-up. Again, it is unknown exactly why participants did not complete the six-month assessment, though analyses found that the drop-out sample had lower illness severity at admission and a faster weight gain rate in treatment relative to the retained participants. Furthermore, in research question 2, the Accelerated group was excluded from moderation analyses due to nearly unanimous participation in day hospital by women in this group. Missing data, and differences between the women who were missing or included, warrant caution in generalization of some of the findings of the study.

The longitudinal outcomes study did not include measures of motivation nor of treatment engagement. Additionally, the current study did not include a measure of perceived coercion. As pattern of weight gain has been hypothesized to be a marker of treatment response, motivation for recovery, and engagement in treatment, inclusion of these constructs may have been useful to explain and/or qualify some of the observed effects. Finally, it is noted the sample was restricted only to women and findings should not be generalized to a male sample.

Future Directions. I consider three broad themes for suggested future research: 1) clarifying the role of psychological variables in treatment evaluation, 2) understanding the role of day hospital, and 3) further investigation of the role of weight suppression as a

predictor of treatment response and recovery. The current study highlighted the apparent conflict that arises with respect to focus on either medical outcomes (e.g., weight) as opposed to behavioral/psychological outcomes (e.g., body dissatisfaction). Notably, women in the Accelerated and Optimal groups appeared to be a success with respect to medical outcomes yet endorsed higher levels of drive for thinness relative to the Suboptimal group. It would obviously be of importance to replicate this finding in an independent (different) sample. Moreover, future research could conduct longer term follow-up (e.g., two years) and utilize clinical information rather than self-report. Moreover, other measures of psychological well-being such as self-esteem, life satisfaction, depression, and anxiety could be evaluated to determine the nature of both physical and psychological recovery. It is of theoretical and clinical importance to determine the importance of traditional psychological markers of ED risk (e.g., drive for thinness) and whether their importance is moderated by ED subtype.

This study focused solely on weight gain during the inpatient phase. Probing pattern of weight gain during day hospital and comparing inpatient and day hospital weight gain would be beneficial. Day hospital's main goal is to practice healthy eating behaviors learned in the inpatient phase and as a result, patients have less restrictions and are not monitored as heavily as during inpatient. Although the inpatient phase of treatment focuses on weight restoration more so than day hospital, it would be interesting to see if certain groups are able to maintain their weight while in day hospital.

Further, results on the importance of history of obesity and weight suppression suggest a need for further exploration on the influence of these factors on treatment response and recovery. The majority of research on weight suppression has been

conducted with bulimia in outpatient samples and has not focused on weight outcomes. Probing the relation between obesity, weight suppression, weight gain, and treatment outcomes in AN would inform treatment protocols and expectations in regards to individuals with weight suppression and/or history of obesity.

Additionally, given the strong influence of cultural norms and expectations on eating disorders and disordered eating behaviors, investigating trajectory group's level of sociocultural attitudes at admission and follow-up would be interesting. It may be certain subgroups have higher levels of sociocultural influences, which may be a factor in increased risk of relapse. If so, this would allow treatment to address those cultural influences thereby possibly reducing risk of relapse. Given the severity of AN and high mortality rate, conducting randomized controlled trials are difficult. However, pending results of previously posited future research, subsequently conducting a randomized study comparing slower weight gain with faster weight gain and investigating treatment response and outcomes would provide rich information about optimal rate of weight gain. Lastly, future research should include the physiological nature of weight gain in treatment course and outcomes.

References

- Accurso, E.C,. Ciao, A.C., Fitzsimmons-Craft, E.E., Lock, J.D., & Le Grange, D. (2014).
 Is weight gain really a catalyst for broader recovery?: The impact of weight gain on psychological symptoms in the treatment of adolescent anorexia nervosa. *Behavior Research and Therapy*, 56, 1-6.
- Agras, W.S., Brandt, H.A., Bulik, C.M., Dolan-Sewell, R., Fairburn, C.G., Halmi,
 K.A.,...,Wilfley, D.E. (2004). Report of the National Institutes of Health
 workshop on overcoming barriers to treatment research in anorexia nervosa. *International Journal of Eating Disorders*, 509-521.
- Aiken, L. S. & West, S. G. (1991). Multiple regression: Testing and interpreting interactions. Thousand Oaks: Sage.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Arcelus, J., Mitchell, A.J., Wales, J, & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry*, 68(7), 724-731.
- Attia, E. & Walsh, B. T. (2009). Behavioral management for anorexia nervosa. New England Journal of Medicine, 360, 500-506.
- Baran, S.A., Weltzin, T.E., & Kaye, W.H. (1995). Low discharge weight and outcome in anorexia nervosa. American Journal of Psychiatry, 152(7), 1070-1072.
- 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. *Journal of Abnormal Psychology*, 115(1), 62-67.

- Casanovas, C., Fernandez-Aranda, F., Granero, R., Krug, I., Jimenez-Murcia, S., Bulik,
 C., & Vallejo-Ruiloba, J. (2007). Motivation to change in eating disorders:
 Clinical and therapeutic implications. *European Eating Disorders Review*, 15, 449-456.
- Cash, T.F. (1994). Body–image attitudes: Evaluation, investment and affect. *Perceptual and Motor Skills*, 78, 1168-1170.
- Cash, T.F., & Deagle, A. (1997). The nature and extend of body-image disturbances in Anorexia Nervosa and Bulimia Nervosa: A meta-analysis. *International Journal of Eating Disorders*, 22, 107-125.
- Cash, T.F., & Pruzinsky, T. (Eds). (2004). Body image: A handbook of theory, research, and clinical practice. New York: Guilford.
- Castro, J., Gila, A., Puig, J., Rodriguez, S., & Toro, J. (2004). Predictors of rehospitalization after total weight recovery in adolescents with anorexia nervosa. *International Journal of Eating Disorders.*, *36*, 22-30.
- Castro-Fornieles, J., Casula, V., Saura, B., Martinez, E., Lazaro, L., Vila, M. ... Toro, J. (2007). Predictors of weight maintenance after hospital discharge in adolescent anorexia nervosa. *International Journal of Eating Disorders*, 40, 129-135.
- Cockell, S.J., Geller, J., & Linden, W. (2002). Decisional balance in anorexia nervosa: Capitalizing on ambivalence. *European Eating Disorders Review*, *11*, 75-89.
- Currin, L., Schmidt, U., Treasure, J., & Jick, H. (2005). Time trends in eating disorder incidence. *British Journal of Psychiatry*, *186*, 132-135.

- Davies, S. & Jaffa, T. (2005). Patterns of weekly weight gain during inpatient treatment for adolescents with anorexia nervosa. *European Eating Disorders Review*, 13, 273-277.
- Eckert, E., Halmi, K., Marchi, P., & Grove, W. (1995). Ten year follow-up of anorexia nervosa: Clinical course and outcome. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences*, 25, 143-156.
- Fairburn, C., & Brownell, K. (2002). *Eating disorders and obesity* (2nd ed.). New York: The Guilford Press.
- Fairburn, C.G. & Beglin, S.J. (1994). Assessment of eating disorders: Interview or selfreport questionnaire? *International Journal of Eating Disorders*, *16*(4), 363-370.
- Garber, A.K., Michihata, N, Hetnal, K, Shafer, M.A., & Moscicki, A.B. (2012). A prospective examination of weight gain in hospitalized adolescents with anorexia nervosa on a recommended refeeding protocol. *Journal of Adolescent Health*, 50(1), 24-29.
- Garner, D. (1991). *Eating Disorder Inventory-2 Professional Manual*. Odessa, FL: Psychological Assessment Resources, Inc.
- Garner D.M., & Garfinkel, P.E. (1997). *Handbook of treatment for eating disorders*. (2nd ed). New York: Guilford Press; 1997.
- Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia.
 International Journal of Eating Disorders, 2, 15-34.
- Grilo, C., & Mitchell, J. (2010). *The treatment of eating disorders: A clinical handbook*.New York: The Guilford Press.

- Grilo, C.M., Masheb, R.M., & Wilson, G.T. (2001). A comparison of different methods for assessing the features of eating disorder patients with binge eating disorder. *Journal of Consulting and Clinical Psychology*, 69(2), 317-322.
- Guarda, A. (2008). Treatment of anorexia nervosa: Insight and obstacles. *Physiology and Behavior, 94,* 113-120.
- Guarda, A., & Coughlin, J. (2009). Treatment resistance: Persuasion, perceived coercion, and compulsion. In I. Dancyger & V. Fornari (Eds.), *Evidence based treatments for eating disorders: Children, adolescents and adults*. (pp. 171-186). Hauppauge, NY: Nova Science Publishers.
- Guarda A.S. & Heinberg, L. (2003). Inpatient and partial hospital approaches to the treatment of eating disorders. In: Thompson JK, ed. *Handbook of Eating Disorders and Obesity* (pp. 297-320). New Jersey: John Wiley & Sons Inc.
- Guarda, A., Pinto, A., Coughlin, J., Hussain, S., Huag, N., & Heinberg, L. (2007).
 Perceived coercion and change in perceived need for admission in patients hospitalized for eating disorders. *American Journal of Psychiatry*, 164, 108-114.
- Hartmann, A., Wirth, C., & Zeeck, A. (2007). Prediction of failure of inpatient treatment of anorexia nervosa from early weight gain. *Psychotherapy Research*, 17(2), 226-238.
- Haynos, A.F., Roberto, C.A., Martinez, M.A., Attia, E., & Fruzzetti, A.E. (2014).
 Emotion regulation difficulties in anorexia nervosa before and after inpatient weight restoration. *International Journal of Eating Disorders*, 1-4.

- Herzog, T. (1993). Patterns and predictors of recovery in anorexia nervosa and bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 835-842.
- Herzog, D., Thomas, J., 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 Research*, 177(3), 330-334.
- Herzog, T., Zeeck, A., Hartmann, A., & Nickel, T. (2004). Lower targets for weekly weight gain lead to better results in inpatient treatment of anorexia nervosa: a pilot study. *European Eating Disorders Review*, 12, 164-168.
- Hoek, H. (2006). Incidence, prevalence, and mortality of anorexia nervosa and other eating disorders. *Current Opinions in Psychiatry*, *19*, 389-394.
- Howard, W.T., Evans, K.K., Quintero-Howard, C.V., Bowers, W.A., & Andersen, A.E. (1999). Predictors of success or failure of transition to day hospital treatment for inpatients with anorexia nervosa. *American Journal of Psychiatry*, 156, 1697-1702.
- Jones, B.L, & Nagin, D.S. (2007). Advances in group-based trajectory modeling and an SAS procedure for estimating them. *Sociological Methods and Research*, 35(4), 542-571.
- Jones, B.L, Nagin, D.S., & Roeder, K. (2001). A SAS procedure based on mixture models for estimating developmental trajectories. *Sociological Methods and Research*, 29(3), 374-393.

- Kaplan, A., & Howlett, A. (2010). Pharmacotherapy for anorexia nervosa. In C. Grilo &
 J. Mitchell (Eds.), *The treatment of eating disorders: A clinical handbook*. (pp. 175-186). New York: The Guilford Press.
- Kaye, W.H., Bulik, C.M., Thornton, L., Barbarich, N., & Masters, K. (2004).
 Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *American Journal of Psychiatry*, 161, 2215-2221.
- Keel, P., Dorer, D., Eddy, K., Franko, D., Charatan, D., & Herzog, D. (2003) Predictors of mortality in eating disorders. *Archives of General Psychiatry*, 60, 179-183.
- Keel, P., Dorer, D., Franko, D., Jackson, S., Herzog, D. (2005). Postremission predictors of relapse in patients with eating disorders. *The American Journal of Psychiatry*, 162, 2263-2268.
- Keski-Rahkonen, A., Hoek, H.W., Susser, E.S., Linna, M.S., Sihvola, E., Raevuori, A.,...,Rissanen, A. (2007). Epidemiology and course of anorexia nervosa in the community. *American Journal of Psychiatry*, 164, 1259-1265.
- Lay, B., Jennen-Steinmetz, C., Reinhard, I., & Schmidt, M. H. (2002). Characteristics of inpatient weight gain in adolescent anorexia nervosa: relation to speed of relapse and re-admission. *European Eating Disorders Review*, 10, 22-40.
- Le Grange, D., Accurso, E.C., Lock, J., Agras, S., & Bryson, S.W. (2014). Early weight gain predicts outcome in two treatments for adolescent anorexia nervosa. *International Journal of Eating Disorders*, 47, 124-129.
- Little, R.J.A., & Rubin, D.B. (1987). Statistical Analysis with Missing Data. New York: John Wiley & Sons.

- Lock, J., Le Grange, D., Agras, W.S, & Dare, C. (2001). *Treatment Manual for Anorexia Nervosa: A Family Based Approach*. New York: The Guilford Press; 2001.
- Lowe, M.R., Davis, W.N., Annunziato, R.A., & Lucks, D.L. (2003). Inpatient treatment for eating disorders: Outcome at discharge and 3-month follow-up. *Eating Behaviors*, 4, 385-397.
- Lowe, M.R., Davis, W.N., Lucks, D., Annunziato, R.A., & Butryn, M.L. (2006). Weight suppression predicts weight gain during inpatient treatment of Bulimia Nervosa. *Physiology and Behavior*, 87, 487-492.
- Lucas, A.R., Crowson, C.S., O'Fallon, W.M., & Melton, L.J. (1999). The ups and downs of anorexia nervosa. *International Journal of Eating Disorders*, *26*, 397-405.
- Lund, B. C., Hernandez, E. R., Yates, W. R., Mitchell, J. R., McKee, P. A., & Johnson,
 C. L. (2009). Rate of inpatient weight restoration predicts outcome in anorexia nervosa. *International Journal of Eating Disorders*, 42, 301-305.
- Maguire, S., Le Grange, D., Surgenor, L., Marks, P., Lacey, H., & Touyz, S. (2008).
 Staging anorexia nervosa: Conceptualizing illness severity. *Early Intervention in Psychiatry*, 2, 3-10.
- Marinilli, A.P., Guarda, A.S., Heinberg, L.J., & DiClemente, C.C. (2006). Development of the eating disorder recovery self-efficacy questionnaire. *International Journal of Eating Disorders, 39*, 376-384.
- McFarlane, T., MacDonald, D.E., Royal, S., & Olmsted, M.P. (2013). Rapid and slow responders to eating disorder treatment: a comparison on clinically relevant variables. *International Journal of Eating Disorders*, 1-4.

- McFarlane, T., Olmstead, M., & Trottier, K. (2008). Timing and prediction of relapse in a transdiagnostic eating disorder sample. *International Journal of Eating Disorders*, 41, 587-593.
- McKnight, I. (2003). Risk factors for the onset of eating disorders in adolescent girls: Results of the McKnight longitudinal risk factor study. *American Journal of Psychiatry*, 160, 248-254.
- Mehler, P.S., Winkelman, A.B., Andersen, D.M., & Gaudiani, J.L. (2010). Nutritional rehabilitation: Practical guidelines for refeeding the anorectic patient. *Journal of Nutrition and Metabolism*, 1-7.
- Mewes, R., Tagay, S., & Senf, W. (2008). Weight curves as predictors of short-term outcome in anorexia nervosa inpatients. *European Eating Disorders Review*, 37-43.
- Morgan, H.G. & Russell, G.F. (1975). Value of family background and clinical features as predictors of long-term outcome in anorexia nervosa: Four-year follow-up study of 41 patients. *Psychological Medicine*, *5*(4), 355-371.
- National Institute for Clinical Excellence (2004). *Eating Disorders: Core Interventions in the Treatment and Management of Anorexia Nervosa, Bulimia Nervosa, and Related Eating Disorders: Clinical Guideline 9.* London: National Institute for Clinical Excellence.
- Neuberger, S.K., Rao, R., Weltzin, T.E., Greeno, C., & Kaye, W.H. (1994). Differences in weight gain between restictor and bulimic anorectics. *International Journal of Eating Disorders*, 17(4), 331-335.

- Olmstead, M., McFarlane, T., Carter, J., Trottier, K., Woodside, D., & Dimitropoulos, G. (2010). Inpatient and day hospital treatment for anorexia nervosa. In C. Grilo & J. Mitchell (Eds.), *The treatment of eating disorders: A clinical handbook*. (pp. 198-211). New York: The Guilford Press.
- Pinto, A., Guarda, A., Heinberg, L., DiClemente, C. (2006). Development of the Eating Disorder Recovery Self-Efficacy Questionnaire. *International Journal of Eating Disorders*, 39, 376-384.
- Pinto, A.M., Heinberg, L.J., Coughlin, J.W., Fava, J.L., & Guarda, A.S. (2008). The Eating Disorder Recovery Self-Efficacy Questionnaire (EDRSQ): change with treatment and prediction of outcome. *Eating Behaviors*, 9, 143-153.
- Ratnasuriya, I., Eisler, I., Szmukler, G., & Russell, G. (1991). Anorexia nervosa:
 Outcome and prognostic factors after 20 years. *British Journal of Psychiatry*, 158, 495-502.
- Rigaud, D., Pennacchio, H, Bizeul, C, Reveillard, V, & Verges, B. (2011). Outcome in anorexia nervosa adult patients: a 13-year follow-up in 484 patients. *Diabetes & metabolism*, 37(4), 305-311.
- Russell, G., Szmukler, G., Dare, C., & Eisler, I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, *44*, 1047-1056.
- Sly, R., Mountford, V.A., Morgan, J.F., & Lacey, J.H. (2014). Premature termination of treatment for anorexia nervosa: differences between patient-initiated and staffinitiated discharge. *International Journal of Eating Disorders*, 47, 40-46.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic

review. Psychological Bulletin, 128, 825-848.

- Stice, E., & Shaw, H.E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings. *Journal of Psychosomatic Research*, 53, 985-993.
- Spitzer, R., Williams, J., Gibbons, M., & First, M. (1992). Structured Clinical Interview for DSM-III-R (SCID). History, rational, and description. *Archives of General Psychiatry*,49, 624–629.
- Steinhausen, H. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, 159, 1284-1293.
- Steinhausen, H.C., Grigoroiu-Serbanescu, M., Boyadjieva, S., Neumarker, K.J., & Winkler, M. C. (2008). Course and predictors of rehospitalization in adolescent anorexia nervosa in a multisite study. *International Journal of Eating Disorders*, 41, 29-36.
- Thompson, J.K. (Ed.) (2001). Body image, eating disorders and obesity: An integrative guide for assessment and treatment. Washington, D.C.: American Psychological Association.
- Vansteelandt, K., Pieters, G., Vanderlinden, J., & Probst, M. (2010). Body dissatisfaction moderates weight curves in the inpatient treatment of anorexia nervosa.
 International Journal of Eating Disorders, 43, 694-700.
- Willer, M.G., Thuras, P., & Crow, S.J. (2005). Implications of changing use of hospitalization to treat anorexia nervosa. *American Journal of Psychiatry*, 162, 2374-2376.

- Wilson, G., Grilo, C., & Vitousek, K. (2007). Psychological treatment of eating disorders. *American Psychologist*, 62, 199-216.
- Wiseman, C.V., Sunday, S.R., Klapper, F., Harris, W.A., & Halmi, K.A. (2001).Changing patterns of hospitalization in eating disorder patients. *International Journal of Eating Disorders*, 30, 69-74.
- Woodside, D. (2002). Inpatient treatment and medical management of anorexia nervosa and bulimia nervosa. In C. Fairburn & K. Brownell (Eds.), *Eating disorders and obesity* (2nd ed.). (pp. 335-339). New York: The Guilford Press.

Appendix A: Additional Literature on Optimal Weight Gain Rate

Though weight restoration and discharge BMI have been shown to be strong predictors of short and long-term outcomes following AN treatment, rate of weight gain appears to be a significant predictor as well. An example of weight gain rate might simply be the number of pounds gained in a week. For patients with AN, weight gain rate has demonstrated clinical utility in determining early-stage compliance as well as posttreatment response (Lund et al., 2009). Weight gain rate is often used as a marker of readiness to progress to a less restrictive level of treatment (Accurso, Ciao, Fitzsimmons-Craft, & Lock, 2014; Haynos et al., 2014). Finally, weight gain rate can be used as a measure of program effectiveness and is associated with a program's ability to weight restore their patients.

As discussed above, a critical area of both inpatient and partial hospital treatment is monitoring weight gain. The National Institute for Clinical Excellence (NICE) guidelines (2004) recommend an average weekly weight gain rate of 0.5 to 1.0 kilograms (kg; 1.10 - 2.20 pounds) in an inpatient setting. However, Woodside (2002) posits that an ideal rate of weight gain is two to four pounds (0.91 to 1.81 kg) per week.

Although there is a consensus that weight restoration is a pivotal aspect of AN treatment, there has been debate in the literature about the optimal rate of weight gain. Some researchers suggest that a slower rate of weight gain may be more beneficial than a faster rate. In certain studies, faster weight gain rates were associated with increased anxiety and decreased perceived ability to complete treatment and weight restoration (Davies & Jaffa, 2005). It may be that faster weight gain rates intensify fear of fatness, which may then lead to treatment noncompliance and increased ED behaviors after

discharge. In addition, some research suggests that faster weight gain during treatment is associated with faster weight loss after discharge and higher rates of rehospitalization (Hartmann & Nickel, 2004; Lay et al., 2002; Willer, Thuras & Crow, 2005). One of the main limitations of these studies is that they did not measure long-term outcomes. It may be that faster weight gain rates temporarily increase anxiety and body dissatisfaction, and that these increased pathologies subside after a period of time.

Though some clinicians recommend a slower weight gain rate, recent research suggests that a faster rate may be optimal. Faster weight gain rates during inpatient treatment are associated with better outcomes at program discharge and long-term follow-up, including a decreased likelihood of deterioration and relapse (Castro, Gila, Puig, Rodriguez, & Toro, 2004; Lund et al., 2009; Mewes et al., 2008). Faster weight gain rates have been found to be associated with a greater reduction in depressive symptoms than average or slower rates (Accurso, Ciao, Fitzsimmons-Craft, & Lock, 2014). This may be explained by a regression to the mean in that individuals with faster weight gain rates often have lower admission BMI, and as such more severe AN; therefore they may benefit more from treatment than those with less severe AN.

In a study focusing on inpatient treatment, Lund and colleagues (2009) examined various weight restoration parameters as predictors of clinical outcome, including admission BMI, discharge BMI, length of stay, total weight gained, and rate of weight gain, in a sample of 79 adolesscent and adult female patients. They found that rate of weight gain was the only parameter that predicted functioning at the 12-month follow-up. Patients who gained at least 0.8 kg per week (1.76 lbs) were significantly less likely to experience clinical deterioration and endorse ED behaviors at follow-up after controlling

for age, ED history, and comorbidities. This suggests that a faster weight gain rate during treatment may be associated with improved long-term outcomes. Conversely, a slower weight gain rate may be associated with a greater risk of deterioration and relapse following treatment, and indicative of resistance to treatment.

Though requiring multiple hospitalizations is a norm in AN, a faster weight gain rate may be a protective factor against readmission. Castro and colleagues (2003) analyzed predictors of rehospitalization following treatment for AN in adolescents and young adults. They found that of the sample of 101 patients who were weight restored prior to discharge at their first admission, only 25% required readmission within 12months of discharge, which is lower than the average relapse rate (Keel et al., 2005; Steinhausen, 2002). Comparing patients who required readmission to those who did not, those who relapsed had a slower rate of weight gain while in treatment, suggesting that a faster weight gain rate may be predictive of long-term clinical outcomes, such as the need for readmission. It may be that a faster weight gain rate is an indicator of reduced ambivalence and increased motivation to change and thereby a measure of increased treatment compliance. However, this study did not assess psychological variables, such as body dissatisfaction, which may have impacted readmission and post-discharge functioning.

Advocates of slower weight gain rates often argue that faster rates are detrimental to the patient's physical and psychological functioning. Although treatment guidelines recommend a weight gain rate of two to four pounds per week, recent research has suggested that faster weight gain rates are both safe and effective (Redgrave, Coughlin, Schreyer, Martin, Leonpacher, Seide,...,Guarda, 2013). Redgrave and colleagues found

that a faster weekly weight gain rate of 4.37 pounds (1.98 kg) in inpatient and 3.00 pounds (1.36 kg) in partial hospital treatment was safe and that patients were no more likely to encounter medical complications of rapid refeeding than slower rates. Of note, approximately 72% of adults and 76% of minor inpatients with AN were weight restored before treatment discharge. These findings suggest that a faster weight gain rate may be advantageous as patients are more likely to reach their target weight before discharge without an increased risk of physical complications.

Due to insurance limitations, average length of stay for AN treatment has reduced from approximately 140 to 40 days in the past 25 years (Willer, Thuras & Crow, 2005). Lack of insurance coverage and, as a result, reduced lengths of stay result in a decreased likelihood of achieving target weight. Sick patients may be discharged prior to weight restoration, which negatively impacts prognosis and increases the risk of relapse and rehospitalization. Discharging patients prior to weight restoration may be contributing to the "revolving door of eating disorders treatment" phenomenon, which is increasing health risk and cost of future treatment (Neuberger, Rao, Weltzin, Greeno, & Kaye, 1994). If programs employ a slower weight gain rate, patients may be less likely to reach weight restoration prior to discharge, which is associated with increased negative short and long-term outcomes. Faster weight gain rates may be a cost-effective approach to reduce hospital lengths of stay and cost and increase the likelihood of achieving target weight and recovery (Davies & Jaffa, 2005).

Appendix B: Self-Report Measures

Current Eating Disorder Behaviors Questionnaire

During the <u>PAST EIGHT WEEKS</u>, what is the average frequency that you have engaged in the following behaviors:

	Never	Once a	Several	Once a	Several	Once a	More
		month or	times a	week	times a	day	than once
		less	month		week		a day
Bingeing	1	2	3	4	5	6	7
Vomiting	1	2	3	4	5	6	7
Laxative use to control	1	2	3	4	5	6	7
weight							
Use of diet pills	1	2	3	4	5	6	7
Use of diuretics	1	2	3	4	5	6	7
Use of enema	1	2	3	4	5	6	7
Use of ipecac syrup	1	2	3	4	5	6	7
Exercise to control weight	1	2	3	4	5	6	7
Skipping meals	1	2	3	4	5	6	7
Restricting food portions	1	2	3	4	5	6	7
Restricting food choices to	1	2	3	4	5	6	7
low fat/low calorie items							
Chewing and spitting out	1	2	3	4	5	6	7
food							
Rumination	1	2	3	4	5	6	7

Note: Binge-eating is defined as eating a very large amount of food and feeling out of control about eating. It does not refer to simple over-eating or guilt after eating more than planned.

Eating Disorder Inventory, Drive for Thinness Subscale (EDI-DT)

For each item, describe how frequently the statement is true of you by selecting from the following options:

Always------Usually------Often-----Sometimes------Rarely------Never

- 1. I eat sweets and carbohydrates without feeling nervous.
- 2. I think about dieting.
- 3. I feel extremely guilty after overeating.
- 4. I am terrified of gaining weight.
- 5. I exaggerate or magnify the importance of weight.
- 6. I am preoccupied with the desire to be thinner.
- 7. If I gain a pound, I worry that I will keep gaining.

Eating Disorder Inventory: Body Dissatisfaction Subscale (EDI-BD)

- 1. I think that my stomach is too big.
- 2. I think that my thighs are too large.
- 3. I think that my stomach is just the right size.
- 4. I feel satisfied with the shape of my body.
- 5. I like the shape of my buttocks.
- 6. I think that my hips are too big.
- 7. I feel bloated after eating a normal meal.
- 8. I think that my thighs are just the right size.
- 9. I think that my buttocks are too large.
- 10. I think that my hips are just the right size.

Eating Disorder Recovery Self-Efficacy Questionnaire (EDRSQ), Normative Eating

Subscale

Select the option that best describes your confidence level: Not at all confident, Somewhat confident, Moderately confident, Very confident, Extremely confident

- 1. I can eat a family meal at a normal rate.
- 2. I can try new foods without feeling anxious.
- 3. I can eat a cheeseburger without compensating by restricting, exercising excessively, or purging.
- 4. I can eat when I feel hungry and stop eating when I feel satisfied.
- 5. I can eat holiday desserts this year and not compensate by purging, exercising excessively, or restricting.
- 6. I can eat one serving of ice cream without feeling guilty or anxious.
- 7. I can eat from a buffet without feeling anxious.
- I can buy food based on what I feel like eating, not because it is low fat and/or low calorie.
- 9. I can eat high fat/high calorie foods without worrying that I will gain weight.
- 10. I can eat lunch without thinking about how many calories I'm consuming.
- 11. I can eat 3 balanced meals a day without bingeing, purging, exercising excessively, or taking diuretics or laxatives.
- I can accept a dinner invitation to somebody's house and eat without restricting, bingeing, or purging.
- 13. I can eat high fat/high calorie foods in moderation without bingeing, purging, taking laxatives or diuretics, or exercising excessively.

14. I can go to a restaurant with friends who are not dieters and eat a normal,

balanced meal.