Patterns of Change in Body Weight Among Individuals During Inpatient Treatment for Anorexia Nervosa

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PATTERNS OF CHANGE IN BODY WEIGHT AMONG INDIVIDUALS DURING INPATIENT TREATMENT FOR ANOREXIA NERVOSA

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Abstract

Patterns of Change in Body Weight during Inpatient Treatment for Anorexia Nervosa

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Despite the chronicity and less than optimal outcomes of inpatient treatment (IPT) for anorexia nervosa (AN), treatment guidelines continue to reflect the common notion of one-size-fits-all and the process of weight restoration continues to be poorly understood. Weight restoration, a primary goal of IPT for AN, does not occur in isolation but rather reflects an adaptation process within internal and external environments. It is unknown whether or not there are unique patterns of change in body weight that are associated with factors identified in the existing literature as being predictors of weight gain. The purpose of this study was to explore the extent to which patterns of change in body weight existed among individuals during IPT for AN, and the relationship with factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of ideal body weight [IBW] at time of admission, body weight at time of discharge, body mass index [BMI] at time of discharge). Individuals who were diagnosed with AN and admitted to the inpatient unit of an eating disorder treatment facility in the Northeast between January 1, 2012 to December 31, 2015 were included in this retrospective, exploratory study (N = 500). Group-based trajectory modeling (GBTM) was used to identify distinct trajectories of change in body weight, and to determine the risk of being in a particular trajectory. Four distinct trajectories were identified: weight gain (n = 197), weight loss (n = 177), weight plateau (n = 82), and weight fluctuate (n = 44) groups. Significant predictors of trajectories were age, history of prior IPT for AN, admission caloric intake, body weight at

time of admission and discharge, and length of stay. Results from this study suggest that a further understanding of patterns of change in body weight among individuals with AN, will help guide assessment and treatment interventions and consequently influence outcomes. Additionally, there is an opportunity to update treatment guidelines and recommendations for AN.

Keywords: anorexia nervosa, body weight, eating disorders, group-based trajectory model, inpatient treatment, patterns, Roy Adaptation Model

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Chapter 1

Introduction

This chapter provides an overview of the problem of anorexia nervosa (AN) with a specific focus on weight restoration during inpatient treatment (IPT) for individuals diagnosed with AN. The background of the problem and the significance of the study are outlined including the potential implications. A statement of the purpose of the study and the research questions are provided. The chapter concludes with definitions of terms pertinent to the study and assumptions of the study.

Statement of the Problem

Anorexia nervosa is a psychiatric disorder with lifetime prevalence rates ranging from 0.2% to 0.9% among adolescents and adults in the United States (Hudson, Hiripi, Pope, & Kessler, 2007; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association [APA], 2013) identifies the cardinal feature of AN as the "restriction of energy intake relative to requirements, leading to significantly low body weight in the context of age, sex, developmental trajectory, and physical health" (APA, 2013, p. 338). To meet diagnostic criteria, individuals must have an intense fear of gaining weight or becoming fat, or engage in behaviors that interfere with weight gain despite having a significantly low weight; and a disturbance in the way in which body weight or shape is experienced, self-evaluation disproportionately influenced by body weight or shape, or lacking recognition of the seriousness of significantly low body weight (APA, 2013).

In the United States, the *Practice Guideline for the Treatment of Eating Disorders* (APA, 2006) provides recommendations for all levels of treatment. Inpatient treatment for AN is often required to intervene with cognitive impairment and medical consequences resulting from severe deficiency in caloric energy intake and malnutrition. Weight restoration

is one of the primary goals of IPT, and is defined as restoring body weight to more than 85% of ideal body weight (IBW; APA, 2006). Achievement of weight restoration has been shown to be associated with better prognosis (Guarda, 2008), but it is not guaranteed with some patients initially losing weight and others having an overall weight loss during the inpatient admission (Garber, Michihata, Hetnel, Shafer, & Moscicki, 2012; Hart, Abraham, Franklin, & Russell, 2011). Furthermore, patients choose to leave IPT prematurely, ranging from 20.2% to 56.2% (Fassino, Pierò, Tomba, & Abbate-Daga, 2009; Huas et al., 2011; Sly & Bamford, 2011), and approximately 40% of weight-restored patients relapse within a year of discharge from IPT (Carter et al., 2012). Despite the chronicity and less than optimal outcomes of IPT for AN, treatment guidelines continue to reflect the common notion of one-size-fits-all and the process of weight restoration is poorly understood.

Existing literature highlights that body weight gain and body mass index (BMI) have often been used as a proxy for weight restoration in adults, and have been assessed using absolute measures, means, and parameters of weight curves. Researchers have examined the relationship between treatment outcomes and body weight gain during IPT for AN (Baran, Weltzin, & Kaye, 1995; Kaplan et al., 2009; Lock & Litt, 2003; Zipfel, Lowe, Reas, Deter, & Herzog, 2000). Studies have reported that predictors of treatment outcomes (e.g., weight maintenance at one year follow-up) include absolute weight gain (Zipfel et al., 2000) and body weight or BMI at time of discharge (Baran et al., 1995; Kaplan et al., 2009; Lock & Litt, 2003).

Other researchers have investigated rate of weight gain during IPT for AN (Davies & Jaffa, 2005; Garber et al., 2012; Hart et al., 2011; Neuberger, Rao, Weltzin, Greeno, & Kaye, 1995). They have reported variability in mean weekly rates of weight gain, with some individuals having minimal weight gain and/or losing weight during the admission. These studies also revealed that predictors of rate of weight gain include: age at time of admission

(Neuberger et al., 1995), admission caloric intake (Garber et al., 2012), and percent of IBW at time of admission (Neuberger et al., 1995). Unfortunately, generalizability of these results is limited because of small sample sizes, non–U.S. samples, and treatment provided at specific facility.

More recently, researchers have used statistical techniques usually applied for the analysis of longitudinal growth curves to examine parameters of patients' weight curves (Hartmann, Wirth, & Zeeck, 2007; Lay, Jennen-Steinmetz, Reinhard, & Schmidt, 2002; Mewes, Tagay, & Senf, 2008). These studies have reported that predictors of treatment outcome (i.e., BMI up to discharge, weight loss after discharge from IPT) include average weekly BMI gain (Mewes et al., 2008), average magnitude of weight loss (Mewes et al., 2008), BMI at admission (Hartmann et al., 2007; Mewes et al., 2008), duration of the first half of treatment (Mewes et al., 2008), tempo of weight increase (Lay et al., 2002), and variation of weight curve (Lay et al., 2002). Unfortunately, these studies did not investigate or identify predictors of changes in body weight during an admission. Such information would better inform individualized treatment plans and consequently influence treatment outcomes.

Over the past 20 years, research has begun to take into consideration the importance of capturing changes in body weight over a period of time by using statistical analyses that more appropriately fit the data. Unfortunately, researchers do not consider that weight restoration does not occur in isolation but rather reflects an adaptive process within environments. It remains unknown whether or not there are unique patterns of change in body weight that are associated with factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge). Such knowledge would potentially lead to the development of treatment assessment tools and

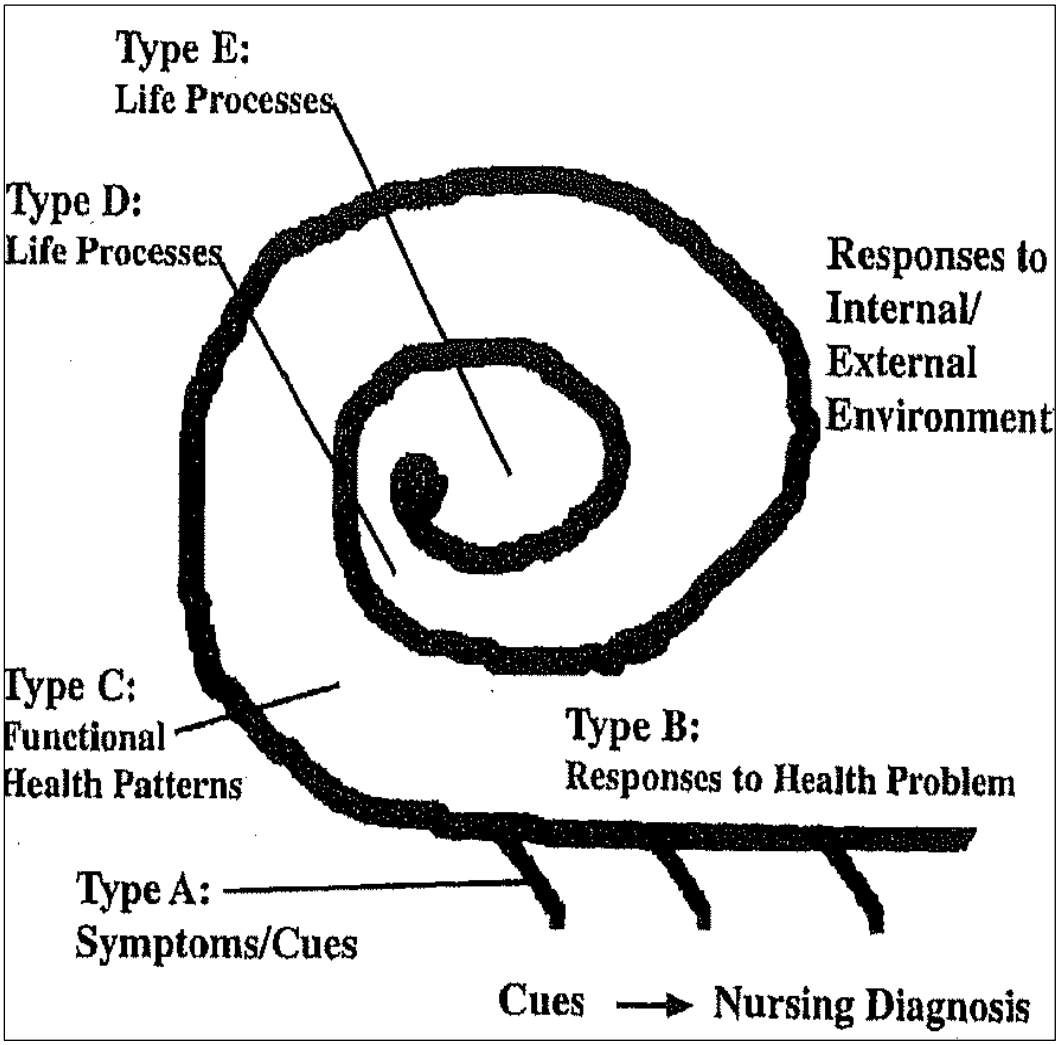
interventions to inform individualized treatment plans and consequently influence treatment outcomes. Thus, this dissertation research examined daily body weights to explore the extent to which patterns of change in body weight exist among individuals during IPT for AN, and the relationship with factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge).

Significance of the Problem

A study exploring patterns of change in body weight during IPT of AN is important for several reasons. First, identifying patterns of change in body weight will help to gain a better understanding of the process of weight restoration, including differences and similarities within this population. Second, this study will determine optimal body weight response patterns among individuals with AN who are at-risk for not achieving weight restoration or leaving treatment prematurely, and such information will inform treatment interventions and influence treatment outcome. Third, a fundamental issue confronting healthcare systems and mental health is financial cost. Knowledge of potential predictors of patterns of change in body weight can serve as feedback to clinical decision making and interventions, as well as influence financial costs.

First, the identification of patterns of change in body weight will provide new knowledge about the process of weight restoration. According to Roy (2009), functional life patterns are a person's response to health problems and are derived from life processes. In order to alter the health-illness trajectory of the person, nursing must have knowledge about life and functional health patterns and responses (Eisenhauer, 1994; Roy, 2009). Thus, nursing interventions should be directed towards altering human responses, patterns, and life processes (Eisenhauer, 1994). Eisenhauer (1994) offers a model of a typology of nursing therapeutics in which nursing interventions aim to alter patterns and processes (see Figure 1).

Figure 1. Typologies of Nursing Therapeutics



Note: From "A Typology of Nursing Therapeutics," by L. Eisenhauer, 1994, *Journal of Nursing Scholarship*, 26, p. 262. Copyright 2007 by John Wiley & Sons. Reprinted with permission.

Gaining knowledge about patterns of change in body weight during IPT for AN, will better inform the development of Type C and D interventions to alter the health–illness trajectory of a person and life processes, leading to adaptation and the promotion of health. Table 1 provides an explanation of the different types of typologies and examples of interventions.

Table 1.

Intervention Typology and Examples

Type/Level of Alteration	Examples
A: Altering and managing cues, symptoms, or patient-environment interface	Applications of heat and cold, oxygen therapy wound care, infection control, positioning, incontinence care, massage, visual imagery, progressive muscle relaxation. music therapy
B: Altering responses to actual or potential health problems	Biofeedback, dysreflexia management, stress management
C: Altering functional health patterns (FHP)	Sleep enhancement, bowel management, social support, assertiveness training, problem solving, cognitive reappraisal, or restructuring
D: Altering life patterns	Therapeutic touch, repatteming
E: Altering life processes	Counseling, spiritual counseling, use of Type C and D interventions over extended period of time
F: Facilitative interventions	Mutual goal setting, negotiation, contracting, active listening

Note: From "A Typology of Nursing Therapeutics," by L. Eisenhauer, 1994, *Journal of Nursing Scholarship*, 26, p. 262. Copyright 2007 by John Wiley & Sons. Reprinted with permission.

In the past few decades, research has examined epidemiological trends, classification, measurement, mechanisms of disease, medical and psychiatric comorbidities, and optimal treatments (Lipsman, Woodside, & Lozano, 2014). Numerous risk factors have been identified including biological (e.g., genetics, neurobiology), environmental (e.g., culture, parenting, family dynamics) and psychological (e.g., body image disturbance, personality traits; Rikani et al., 2013; Treasure, Claudino, & Zucker, 2010). Yet, despite these advances, AN continues to have the highest mortality rate among all psychiatric disorders (Arcelus, Mitchell, Wales, & Nielsen, 2011), elevated risk for suicidal ideation, suicidal gestures, suicide attempts (Kostro, Lerman, & Attia, 2014; Swanson et al., 2011), and a 40-fold higher

risk of suicide compared to the general population (Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). Not only are individuals refractory to treatment, but psychiatric comorbidities and medical consequences are commonplace with this population (Gaudio & Di Ciommo, 2011; Hudson et al., 2007; Swanson et al., 2011; Zanarini, Reichman, Frankenberg, Reich, & Fitzmaurice, 2010). Group-based trajectory modeling (GBTM) allows for taxonomic theorizing about distinct health-illness trajectories for individuals during IPT for AN via patterns of changes in body weight, and for an examination of potential factors that may account for the distinctiveness. Thus, utilization of the RAM as a theoretical framework and GBTM as the method of analyses will facilitate knowledge development about the process of weight restoration, and will better inform clinical decision making and interventions and eventually influence treatment outcomes.

Second, the identification of patterns of change in body weight will lead to exploration of optimal body weight response patterns among individuals with AN who are at-risk for not achieving weight restoration. Weight restoration to more than 85% IBW is an established integral component of inpatient treatment for AN (APA, 2006), with the standards of care and practice guidelines being primarily based on clinical expertise and consensus (APA, 2006). The recommended average weight gain is 0.9–1.4 kg per week (2–3 pounds) with caloric intake typically starting at 1,000–1,600 calories per day (APA, 2006). Unfortunately, there is a paucity of empirical data to support such recommendations, which only contributes to the ambiguous and generalized treatment guidelines with regard to weight restoration. Research suggests that a subset of individuals will lose weight or have minimal weight gain during an admission (Garber et al., 2012; Hart et al., 2011), and there is debate as to the risks and benefits of starting at a higher admission caloric intake (Katzman, 2012). Furthermore, studies suggest that at least 20% of patients choose to leave IPT prematurely (Fassino et al., 2009; Huas et al., 2011; Sly & Bamford, 2011) and almost half of weight-

restored patients relapse within a year of discharge from IPT (Carter et al., 2012). Thus, this study will examine the optimal body weight response patterns among individuals with AN who are at-risk for not achieving weight restoration, and thus better inform clinical decision making and interventions regarding the process of weight restoration.

Third, a fundamental issue confronting healthcare systems and mental health in the U.S. is financial cost. In recent years, despite reduced lengths of stays for treatment of AN, costs have increased. For example, between 1999 and 2009, the average cost per hospital stay for eating disorders increased 29% from $7,300 to $9,400 and the national estimates for hospitalizations with AN (as the primary or secondary diagnosis) increased 13% (Zhao & Encinosa, 2011). The increased number of hospitalizations and costs associated with AN may be explained, in part, by patients who are prematurely discharged and fail to achieve weight restoration (Baran et al., 1995; Lock & Litt, 2003; Sly & Bamford, 2011). This study will attempt to identify predictors of unique patterns of change in weight during IPT for AN, which will help guide clinical treatment decisions that improve outcomes such as re-hospitalizations and thus decrease financial costs for IPT of AN.

Purpose of the Study

The purpose of this study was to explore the extent to which patterns of change in body weight exist among individuals during IPT for AN, and the relationship with factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge). Patterns of change in body weight were determined by identifying homogenous groups of individuals who had similar changes in body weight during their inpatient admission. The identification of patterns of change in body weight was important to this study because it recognized how weight restoration is an adaptive process within environments, and does not occur in isolation. For the second part of

the study, an examination of the relationship between factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge) and patterns of change in body weight was important because such knowledge will lead to the development of models to predict patterns of change in body weight among this population and potentially improve treatment and outcomes.

Research Questions

1. To what extent do patterns of change in body weight exist among individuals during IPT for AN?
2. What is the relationship between factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge), and patterns of change in body weight among individuals during IPT for AN?

Definition of Terms

Anorexia nervosa: as defined by the fifth edition of the *DSM* criteria for AN (APA, 2013). The criteria includes the restriction of energy intake leading to significantly low body weight; an intense fear of gaining weight or becoming fat, or engaging in behaviors that interfere with weight gain despite having a significantly low weight; and a disturbance in the way in which body weight or shape is experienced, self-evaluation disproportionately influenced by body weight or shape, or lacking recognition of the seriousness of significantly low body weight (APA, 2013). Severity of illness ranges from mild (BMI $\geq$ 17 kg/m^2) to extreme (BMI < 15 kg/m^2), and reflects the clinical symptoms and the functional impairment of the individual (APA, 2013)

Inpatient treatment: a 24-hour service that is delivered in a licensed hospital setting and provides clinical intervention for mental health and/or substance abuse diagnoses (www.mass.gov). The following factors may indicate the need for this level of care: rapid or persistent decline in oral intake, decline in weight despite other interventions (e.g., intensive outpatient treatment or partial hospitalization), knowledge of weight at which instability previously occurred, co-occurring psychiatric issues, and the degree of denial and resistance to participate in less intensive supervised setting (APA, 2006).

Weight restoration: restoring or increasing body weight to healthy weight estimates based on numerous factors including weight range prior to onset of AN, age, growth charts, maturity, and stages of sexual development. According to the *Practice Guideline for the Treatment of Eating Disorders* (APA, 2006), weight restoration is defined as restoring body weight to more than 85% of ideal body weight, and is one of the primary goals of IPT for AN.

Body mass index: an index of weight-for-height, and is defined as weight in kilograms divided by the square of height in meters (kg/m^2). It is commonly used to classify weight status and provides a reliable indicator of body fatness according to body weight, age, and height (Centers for Disease Control and Prevention, 2013).

Body weight: the product of the force of gravity. For a human being, it is the anthropometric measurement of the heaviness of a person.

Ideal body weight: an anthropometric measurement of the heaviness of a person that is considered to be maximally healthful based on height, gender, age, build, and muscular development (APA, 2006). According to the *Practice Guideline for the Treatment of Eating Disorders* (APA, 2006), IBW may be based on historical considerations (e.g., growth charts, age, maturity, stage of sexual development), and for women, resuming of menstruation and ovulation.

Body weight gain: the increase in body weight over a period of time, and has been used as a proxy to weight restoration in the existing literature on AN.

Age of AN onset: the age at which AN presents, and usually occurs during adolescence and young adulthood (APA, 2006).

Pattern of change in body weight: a group of individuals who follow similar changes of body weight (weight gains and losses) over the length of their admission.

Assumptions Based on Existing Knowledge

- Individuals with AN engage in behaviors to prevent weight gain and/or promote weight loss.
- Individuals who require IPT are usually less than 85% of IBW as well as medically compromised and/or cognitively impaired.
- Inpatient treatment for AN is required for acute stabilization of psychiatric and/or medical complications.
- Weight restoration is one of the primary goals for IPT of AN.
- There is no known cure for AN.

Chapter 2

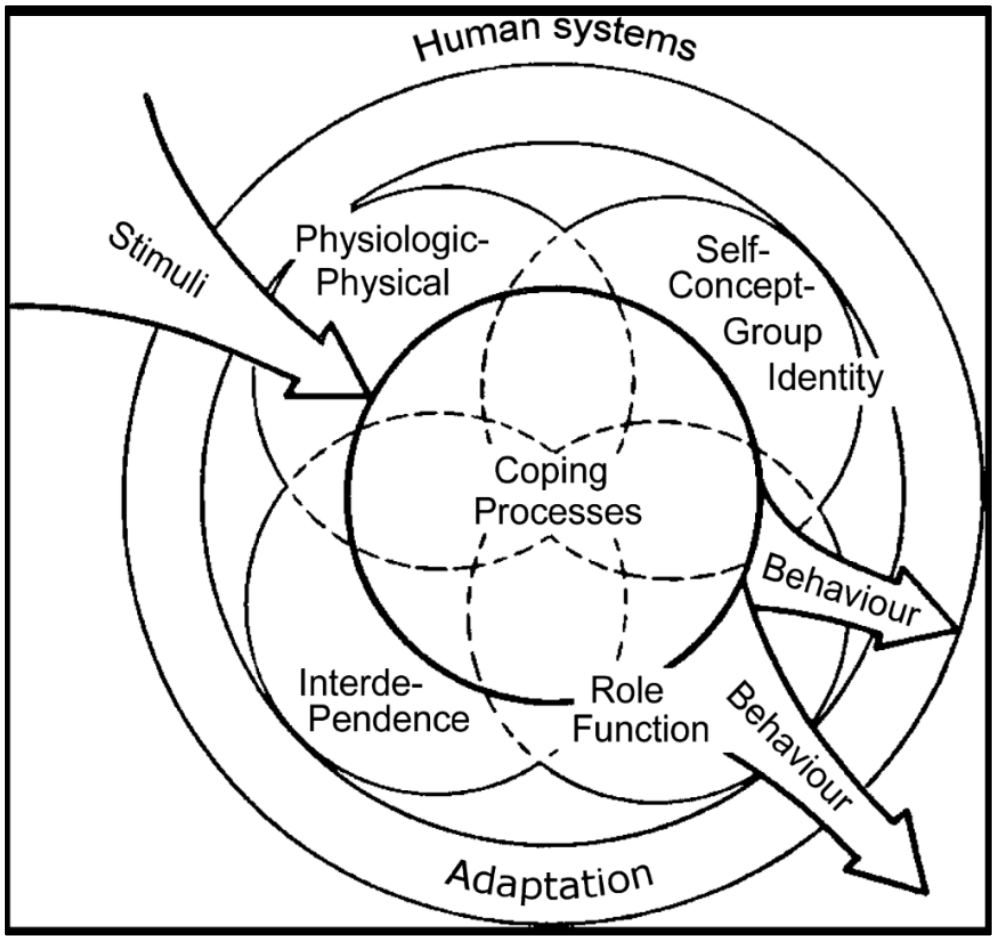
Literature Review

This chapter provides an overview of the Roy Adaptation Model (RAM) as the theoretical framework for the current study and a review of the existing literature regarding anorexia nervosa, inpatient treatment, and weight restoration. The review of the literature discusses scientific literature as well as important documents that guide treatment for individuals with AN within the U.S.

Theoretical Framework: The Roy Adaptation Model

This descriptive study used the RAM (Roy, 2009) as an overarching theoretical framework to explore the extent to which weight patterns exist in individuals receiving IPT for AN and the relationship with factors identified in the existing literature as being predictors of weight gain during IPT for AN. Roy views the person as a complex adaptive system interacting with environments and striving towards adaptation and health. According to Roy, an individual experiences stimuli which activate coping processes resulting in behavioral responses that are observed in four critical adaptive modes: interdependence, physiological, role-function, and self-concept (see Figure 2). The ability of a person to effectively adapt to stimuli is contingent upon the adaptation level, the situational demands, and pre-existing life processes. According to the RAM, the four complex and interrelated modes serve as a framework for assessment of interactions with the environment, behavioral responses, and adaptation.

Figure 2. Representation of Human Adaptive Systems



Note: From C. Roy, 2009, *The Roy Adaptation Model, Third Edition,* p. 45. Copyright 2009 by Prentice Hall Health. Reprinted with permission.

Assumptions

The theoretical assertion of the RAM is based on philosophic, scientific, and cultural assumptions derived from systems theory, adaptation-level theory, and cultural challenges of the 21st century (Roy, 2009; see Table 2). The philosophical assumptions are characterized by the general principles of humanism, veritivity and cosmic unity (Roy, 2009). Humanism recognizes the individual and subjective dimensions of the human experience as central to knowing and valuing. Veritivity is a principle of human nature that affirms a common purposefulness of human existence. Cosmic unity is a philosophical view of reality which

stresses the principle that people and the earth have common patterns and integral relationships. The scientific assumptions are based on the phenomena of living systems having complex processes of interaction and acting to maintain the purposefulness of existence in a universe (Roy, 2009). The cultural assumptions are an integration of cross-cultural experiences, cultural needs and the necessity to eliminate culture-bound analysis of key concepts (Roy, 2009). These underlying assumptions are evident in the four major concepts of the RAM: (1) an individual as adaptive system (Roy also describes groups as adaptive systems, but the individual is the focus of this study); (2) the environment; (3) health; and (4) the goal of nursing (Roy, 2009). As an adaptive system, an individual is defined as a whole with parts that function as a unity for a purpose. The environment is defined as all conditions, circumstances, and influences that surround and affect the development and behavior of humans as adaptive systems. Health is a state and process of being and becoming integrated and whole.

Table 2.

Assumptions of the Roy Adaptation Model

Philosophical Assumptions
▪ Persons have mutual relationships with the world and a God-figure ▪ Human meaning is rooted in an omega point convergence of the universe ▪ God is intimately revealed in the diversity of creation and is the common destiny of creation ▪ Persons use human creative abilities of awareness, enlightenment, and faith ▪ Persons are accountable for entering the process of deriving, sustaining, and transforming the universe
Scientific Assumptions
▪ Systems of matter and energy progress to higher levels of complex self-organization ▪ Consciousness and meaning are constitutive of person and environment integration ▪ Awareness of self and environment is rooted in thinking and feeling ▪ Human decisions are accountable for the integration of creative processes ▪ Thinking and feeling mediate human action ▪ System relationships include acceptance, protection, and fostering interdependence

- Persons and the earth have common patterns and integral relations
- Person and environment transformations are created in human consciousness
- Integration of human and environment meanings results in adaptation

Cultural Assumptions
▪ Experiences within a specific culture will influence how each element of the RAM model is expressed ▪ Within a culture there may be a concept that is central to the culture and will influence some or all of the elements of the RAM to a greater or less extent ▪ Cultural expressions of the elements of the RAM may lead to changes in practice activities such as nursing assessment ▪ As RAM elements evolve within a cultural perspective, implications for education and research may differ from experience in the original culture

Note: From C. Roy, 2009, *The Roy Adaptation Model, Third Edition,* p. 31. Copyright 2009 by Prentice Hall Health. Reprinted with permission.

Complex Adaptive Systems

A system is a set of interdependent parts that are connected to function as a whole for a purpose (Roy, 2009). Similar to any complex adaptive system, people experience stimuli which activate coping processes resulting in behavioral responses. Points of interaction with the environment are considered stimuli. Coping processes are ways of responding to the changing environment and consists of two subsystems, regulator and cognator. Since it is not possible to observe directly the functioning of the regulator-cognator subsystems, behavioral responses resulting from control processes are observed in four critical adaptive modes: (1) interdependence, (2) physiological, (3) role-function, and self-concept (Roy, 2009). Behavioral responses are internal or external actions and reactions to stimuli, and act as feedback or further input to determine whether or not people should continue with current coping processes (Roy, 2009).

Stimuli

Stimuli describe the environment, and are constantly shifting in response to human and environment interactions (Roy, 2009). The three classes of stimuli that form the environment are contextual, focal and residual. Focal stimulus is the internal or external stimulus that involves the immediate awareness of the individual and requires the use of energy and resources. As mentioned above, AN is characterized by significantly low body weight secondary to severe restriction of energy intake as well as a fear of or persistent behavior that interferes with body weight gain. Weight restoration is a primary goal of IPT, but the person with AN tends to focus on thoughts, feelings, and behaviors related to limiting body weight gain resulting in the activation of defense mechanisms and behaviors to prevent such changes (i.e., restricting caloric intake, engaging in compensatory behaviors). As the person continually struggles for control over his or her body weight, patterns of behaviors to prevent weight gain and/or promote weight loss become even more deeply rooted. For example, research has shown that during IPT, individuals with AN will engage in more caloric intake restriction to feel more empowered during periods of "enforced" treatment or negative interaction with "pessimistic" staff (Bezance & Holliday, 2013; Fox & Diab, 2015).

Contextual stimuli are all factors that influence the ability to respond to the focal stimulus and contribute directly to adaptation (Roy, 2009). These contributing factors are internal or external stimuli that are not the focus of attention and energy. The diagnosis of AN, related biological vulnerabilities, and impaired defense mechanisms can be characterized as contextual stimuli. For example, predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge) may be considered contextual stimuli because they influence the individual's ability to adapt to the process of weight restoration.

Residual stimuli are internal or external environmental factors that may affect the current situation, but the influence is unclear or unknown (Roy, 2009). For example, a

potential residual stimulus may be a person's perception of body weight and shape. Identification of residual stimuli is based on the individual's situation at time of the inpatient admission, and constantly shifts in response to the person's interaction with the changing environment. Individuals with AN may alter their perception of body weight and shape as they progress through IPT, and such changes may shift their patterns of behavior to actually promote body weight gain.

Coping Processes

Coping processes are divided into two subsystems, regulator and cognator. The regulator subsystem refers to genetically pre–determined defense mechanisms that occur without human intervention, and is concerned with the individual's innate and automatic signals from neural, chemical, and endocrine system channels (Roy, 2009). This subsystem responds to stimuli within the environment with a complex integrative central and peripheral signaling network of positive and negative feedback mechanisms to maintain energy homeostasis. The cognator subsystem involves learned responses to stimuli which occur through four cognitive emotive channels: perceptual and informational processing, learning, judgment, and emotion (Roy, 2009). Perceptual and informational processing includes selective attention, coding and memory. Learning involves imitation, reinforcement and insight. Judgment involves problem-solving and decision making. Emotion involves defenses used to seek relief from anxiety and make affective appraisals and attachments.

Individuals with AN receive environmental input (e.g., relief from negative emotions, appraisal and/or increased attention from others) to support the belief that restriction of caloric intake is an effective way to manage internal emotional states and resolve challenges to personal control. As time passes, restricting caloric intake becomes an acquired defense mechanism that is deeply rooted as the learned response to control internal emotional states and challenges to sense of self. During IPT, the cognator subsystem of an individual with AN

perceives the process of weight restoration as a threat; uses prior learning to manage (e.g., limit or prevent) this process; makes judgment to engage in restricting caloric intake to manage internal emotional status (e.g., anxiety, depression) and challenges to personal control. As the intensity of the internal emotional state exceeds a person's ability to control the emotion, caloric restriction is perceived as necessary and an essential coping strategy for survival. Such behavioral response influences the regulator subsystem, and this subsystem is altered and unable to maintain energy homeostasis. Physiological indicators of such an imbalance include malnutrition, cardiac complications (e.g., bradycardia, orthostatic hypotension, tachycardia), endocrine abnormalities (e.g., hypokalemia, hypoglycemia), and cognitive impairment.

Behavioral Responses

Behavioral responses are internal or external actions and reactions and demonstrate how well an individual is adapting to stimuli (Roy, 2009). Behavioral responses reflect the abilities of coping processes to be adaptive or ineffective, but also act as feedback to the human adaptive system. Adaptive behavioral responses promote the integrity of the person and the goals of adaptation: survival, growth, reproduction, mastery, and human and environment transformations (Roy, 2009). Ineffective behavioral responses do not contribute to the integrity of the person and the goals of the human system. Based on the effectiveness of behavioral responses, an individual will alter or continue to use specific behaviors to cope with stimuli. Behavioral responses can be observed, measured, and/or subjectively reported and include capacities, assets, knowledge, skills, abilities and commitments (Roy, 2009). For individuals with AN, restricting caloric intake tends to be the learned behavior to reflect the deeply rooted defense mechanisms to manage emotional states and resolve challenges to personal control. Unfortunately, for the overall human body system, such behavioral responses are ineffective and compromise health and overall adaptation.

Adaptive Modes

Since it is not possible to observe the processes of the regulator and cognator subsystems, behavioral responses are manifested in four modes of adaptation: (1) interdependence, (2) physiological, (3) role function, and (4) self-concept (Roy, 2009). The interdependence mode focuses on social integrity including the development and maintenance of affectionate and supportive relationships. The physiological mode emphasizes the maintenance of physiological integrity. The role function mode relates to social integrity associated with various roles throughout one's lifetime. The self-concept mode focuses on psychic integrity manifested through perceptions of the physical and personal self. Individuals with AN, tend to exhibit ineffective behavioral responses in at least one of the four modes of adaptation, and a disruption in one significantly influences the other three modes. Table 3 provides the definition and application of each adaptive mode to individuals diagnosed with AN.

Table 3.

Application of Adaptive Modes to Persons with Anorexia Nervosa

Adaptive mode	Definition	Application
Interdependence	Need is relational integrity. To feel secure in nurturing relationships through the giving & receiving of love, respect, & value.	Struggle with emotion dysregulation & trusting others. Struggle to establish & maintain friendships or intimate relationships. Social withdrawal & isolation.
Physiologic	Need is physiologic integrity. Manifestation of all physiologic activities comprising the human body.	Human body downregulates to preserve homeostasis, leading to significant & potentially life-threatening medical consequences & cognitive deficits.

	Five basic needs: activity and rest, elimination, nutrition, oxygenation, & protection. Four complex processes: electrolytes & acid-bases balance, endocrine function, fluid, & neurologic function.	Such consequences & deficits contribute to treatment obstacles including reluctance to change eating disorder behaviors & resistance to weight gain.
Role function	Need is social integrity. Pertains to roles & expectations of the person in society in relationship to others.	Struggle with trusting & relinquishing control of eating disorder to healthcare providers.
Self–concept	Need is psychic & spiritual integrity. Composite of beliefs and feelings about oneself that directs all behavior.	Poor self-esteem. Poor self-worth. Poor body image.

Interdependence mode. The interdependence mode focuses on interactions related to the giving and receiving of love, respect, and value, with the underlying need of relational integrity (Roy, 2009). The need to feel secure in nurturing relationships is specific to two types of relationships: significant others and support systems. For example, individuals with AN struggle with establishing and maintaining friendships and other relationships (Fox & Diab, 2015). Additionally, this population has a higher likelihood of experiencing childhood abuse and trauma, and thus struggle with trusting others (Harrison, Tchanturia, & Treasure, 2010; Racine & Wildes, 2015).

Physiologic mode. The physiologic mode is the manifestation of all physiologic activities comprising the human body, and the underlying need is physiologic integrity (Roy, 2009). This mode has nine components. The five basic needs: (1) oxygenation, (2) nutrition, (3) elimination, (4) activity and rest, and (5) protection. The four complex processes are the senses: (1) fluid, (2) electrolytes and acid-bases balance, (3) neurologic function, and (4) endocrine function. As individuals with AN restrict caloric intake and lose body weight, the

human body downregulates to preserve homeostasis and medical consequences may develop prior to and during nutrient rehabilitation. For example, during the process of weight restoration, patients may experience significant biochemical changes, specifically in fluid and electrolyte balance as well as micronutrient status (Mehanna, Moledina, & Travis, 2008; Solomon & Kirby, 1990).

Role-function mode. The role-function mode pertains to the roles of the person in society, and the underlying need is social integrity. A role within a society is defined as the expectations about how an individual occupying one position behaves toward an individual occupying another position (Roy, 2009). During IPT for AN, patients tend to struggle with relinquishing control to the interdisciplinary treatment team, which influences the therapeutic alliance and the process of weight restoration (Bezance & Holliday, 2013; Fox & Diab, 2015). Additionally, the non-individualized treatment approach (i.e., standardized care), the focus on physiological versus psychological recovery, and the assumptions about AN conveyed by staff contribute to the struggle for control for patients (Bezance & Holliday, 2013).

Self-concept mode. The self-concept mode involves the personal aspects of human beings, and the underlying need is psychic and spiritual integrity. Self-concept directs behavior and is defined as the beliefs and feelings about oneself at a given time (Roy, 2009). This mode includes the physical self (i.e., body sensations, body image) and the personal self (i.e., self-consistency, self-ideal, moral-ethical-spiritual self). For individuals with AN, body image is a significant component of self-concept. Body image influences the maintenance and relapse of the disorder, and plays a complex role in everyday perceptions, attitudes, and feelings of individuals with AN. During IPT for AN, the notion of one-size-fits-all and the process of weight restoration significantly impacts body image and hence affects self-

concept. During IPT for AN, individuals may experience a profound sense of lost identity and struggle to redefine body image, and thus struggle with redefining the self.

Levels of Functioning

The ability of an individual with AN to effectively adapt to stimuli is contingent upon the person's adaptation level, the situational demands, and pre-existing life processes (Roy, 2009). These life processes are conceptualized as three levels of functioning: integrated, compensatory, and compromised. Integrated is an adaptation level at which the structures and functions of a life process are working as a whole to meet human needs. Compensatory is an adaptation level at which the cognator and regulator have been activated by a challenge to the integrated life processes. Compromised results from inadequate integrated and compensatory life processes, and is an adaptation problem. If an integrated life process changes to a compensatory level of functioning, then the person will attempt to reestablish an integrated adaptation level. If an integrated adaptation level is not reestablished, then a compromised level of functioning will result. At a significantly low body weight, individuals with AN are not functioning at an integrated adaptation level because human needs are not being met (e.g., cognitive impairment, malnourishment). During IPT for AN, weight restoration is vital to reestablishing an integrated level of functioning in which all components of the individual function in unison to maintain health, or adaptation. The examination of patterns of change in body weight will help to identify individuals with AN who do not have the ability to effectively reestablish an integrated level of functioning, and are at risk of leaving treatment prematurely or not achieving weight restoration.

Healthy body weight is an anthropometric measurement that indicates adequate nutrition and caloric energy intake (nutritional need of physiological mode) to maintain energy homeostasis. Body weight in healthy persons is normal, and healthy persons eat normal amounts of food and maintain a healthy body weight. Persons with AN restricts caloric intake

to manage emotional states and resolve challenges to personal control, and this behavior becomes a deeply rooted defense mechanism. As a result, individuals with AN develop a severe deficiency in caloric energy intake and malnutrition indicated by significantly low body weight. The complex integrative central and peripheral signaling network of positive and negative feedback mechanisms of the innate and automatic signals from neural, chemical, and endocrine system channels are altered and unable to maintain energy homeostasis. Significant restriction of caloric intake is ineffective and compromises health. During IPT for AN, the process of weight restoration, or restoring body weight to a healthy weight, is perceived as a threat to the person with AN's ability to control and cope, resulting in their need to engage in the learned response of restricting caloric intake to regain control and manage negative emotional states.

According to the RAM (Roy, 2009), the goal of nursing is to identify the patient's adaptation level and coping capacity, identify behaviors and stimuli, and provide interventions to facilitate adaptation and interactions with the environment, thereby promoting health. The identification of patterns of change in body weight, and an examination of the relationship between factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge) and patterns of change in body weight, will provide information about patients' adaptation level and coping capacity and facilitate the development of interventions and treatment guidelines to promote weight restoration and adaptation.

Synthesized Review of the Literature

Anorexia Nervosa: Definition & Classification

In the 1870s, William Gull and Charles Lasègue described the clinical characteristics of AN as a mental disorder associated with severe emaciation and amenorrhea with unknown

etiology, and coined the terms “anorexia nervosa” and “l’anorexie hystérique,” respectively (Russell, 1995). Throughout history, the traits of AN have remained constant (i.e., denial of illness, extreme, life-threatening emaciation, restricting caloric intake, tendency towards recovery, young females; Theander, 1995). As noted earlier, the *DSM-5* (APA, 2013) identifies the cardinal feature of AN as the “restriction of energy intake relative to requirements, leading to significantly low body weight in the context of age, sex, developmental trajectory, and physical health” (see Table 4). To meet diagnostic criteria, individuals must have an intense fear of gaining weight or becoming fat, or engage in behaviors that interfere with weight gain despite having a significantly low weight; and a disturbance in the way in which body weight or shape is experienced, self-evaluation disproportionately influenced by body weight or shape, or lacking recognition of the seriousness of significantly low body weight (APA, 2013). Among children and adolescents, instead of weight loss, they may fail to meet expected weight gain or maintain normal developmental trajectory (Rosen & American Academy of Pediatrics Committee on Adolescence, 2010).

Table 4.

Criteria for Anorexia Nervosa

A. Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. Significantly low weight is defined as a weight that is less than minimally normal or, for children and adolescents, less than that minimally expected
B. Intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight
C. Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight
Specify:

A. Restricting type: During the last 3 months, the individual has not engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, and/or excessive exercise

B. Binge-eating/purging type: During the last 3 months, the individual has engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)

Severity:
Mild: BMI $\geq$ 17 kg/m^2
Moderate: BMI 16–16.99 kg/m^2
Severe: BMI 15–15.99 kg/m^2
Extreme: BMI < 15 kg/m^2

Note: From American Psychiatric Association (APA), 2013, *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition,* p. 338-339. Copyright 2013 by APA. Reprinted with permission.

Besides significantly low body weight, the *DSM-V* criteria includes subtypes and severity of AN as well as associated features supporting the diagnosis (APA, 2013). The two subtype classifications describe the most current symptoms in the past three months: binge eating/purging – individuals who regularly engage in binge eating and purging behaviors including self-induced vomiting and misuse of laxatives, diuretics, or enemas, and restricting – individuals who do not regularly engage in binge eating or purging behaviors and primarily engage in dieting, fasting, and/or excessive exercising (APA, 2013). Some individuals with binge-eating/purging subtype do not engage in binge eating but regularly purge after any caloric intake, especially solid foods. Severity of illness must also be specified and includes: mild (BMI $\geq$ 17 kg/m^2), moderate (BMI 16–16.99 kg/m^2), severe (BMI 15–15.99 kg/m^2) and extreme (BMI < 15 kg/m^2). The minimum level of severity is based on BMI for adults and BMI percentile for adolescents and children, but may be increased to "reflect clinical symptoms, the degree of functional disability, and the need for supervision" (APA, 2013).

Associated features include: depressed mood, disinterest in sexual activity (as age appropriate), fear of eating in public, insomnia, irritability, obsessive-compulsive behaviors related to food (e.g., collecting recipes, preoccupation with television food networks) and social withdrawal (APA, 2013).

Epidemiology

Anorexia nervosa is a rare disorder among the general population (Karg et al., 2014), with lifetime prevalence between 0.3–0.9% among U.S. adults (age 18 years or older) and 0.3% among adolescents (ages 13–18; Hudson et al., 2007; Swanson et al., 2011). The lifetime prevalence of AN is higher in adult females compared to adult males (0.9% versus 0.3%; Hudson et al., 2007; Stice, Marti, & Rohde, 2013), and prevalence rates are similar for adolescent males and females (0.3%; Swanson et al., 2011). For adults and adolescents, non-Hispanic whites were more likely to report AN compared to others. Among adults, non–Hispanic whites have a higher lifetime prevalence of AN compared to Asians (0.3–0.9% versus 0.08%), non-Hispanic blacks (0.3–0.9% versus 0.17%), and Latinos (0.3–0.9% versus 0.08%; Alegria et al., 2007; Nicdao, Hong, & Takeuchi, 2007; Taylor, Caldwell, Baser, Faison, & Jackson, 2007). The average age of AN onset is 18.9 years for adults (Hudson et al, 2007) and 12.3 years for adolescents (Swanson et al., 2011). However, the disorder and prodromal symptoms may present earlier.

Etiology

The precise etiology of AN is unknown because of the difficulties deciphering between the causes and consequences of malnutrition. However, it is clear that the cause is multifaceted and includes biological (e.g., genetic effects, neurobiology), developmental, familial, and sociocultural.

Biological. This section will discuss biological etiology of AN including genetic effects and neurobiology.

Genetic effects. Since monozygotic twins share almost 100% of their genome and dizygotic twin share about 50%, twin studies allow researchers to elucidate genetic and environmental factors that may contribute to the development of AN. Studies suggest heritability estimates ranging from 31% to 76% with the remaining variance being attributed to environmental factors (Bulik et al., 2006; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik, 2001). Strong evidence for familial aggregation also exists in that relatives of individuals with AN being 7–12 times more likely to have a lifetime of an eating disorder compared to relatives of healthy controls (Lilenfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000). Regarding the role of molecular genes in AN (e.g., regulating appetite genes, genes influencing food intake), a recent review by Trace and colleagues (2014) reported that findings are inconsistent because of inadequate statistical power, non-replications, small sample sizes and varying diagnostic definitions of AN. However, ongoing research continues to examine potential chromosomes, genes, neuropeptides, and proteins that may play a specific role in the development of AN (Bulik, Landt, van Furth, & Sullivan, 2007; Clarke, Weiss, & Berrettini, 2012; Trace, Baker, Peñas–Lledó, & Bulik, 2014).

Neurobiology. The regulation of feeding behavior and body weight is a complicated relationship between peripheral and central nervous systems. Studies in animals show that neuropeptides (e.g., CRH, leptin, the endogenous opioids, neuropeptide-Y) modulate feeding behaviors and energy metabolism (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Recent reviews of the scientific literature suggest numerous structural and functional deficits in the central nervous system and peripheral systems of individuals with AN both underweight and weight–restored (Phillipou, Rossell, & Castle, 2013; Trace et al., 2014). Reduced gray matter, increased cerebrospinal fluid (CSF) volume, altered white matter, and disturbances of metabolism, electrolytes, and endocrine functioning appears to be consistently associated

with underweight individuals with AN (Mainz, Schulte-Ruther, Fink, Herpertz-Dahlmann, & Konrad, 2012). However, findings among weight–recovered individuals are not consistent. For example, some researchers report that weight–recovered individuals have normalized CSF volumes and grey matter compared to healthy controls (Mainz et al., 2012), while others suggest elevated CSF volume but persistent grey matter deficits (Roberto et al., 2011).

Regarding monoamine systems and functioning, research has focused on alterations of dopamine (DA), norepinephrine (NE), and serotonin (5–HT) systems among underweight and weight-restored individuals with AN (for a review, see Phillipou et al., 2013). Monoamine systems are complex pathways (e.g., receptors, transporters, enzymes, intracellular cascade) that modulate appetite, mood, and neuroendocrine functioning. Findings suggest that underweight individuals have lower CSF concentrations of 5–HIAA (major metabolite of 5–HT) and weight-restored individuals have higher concentration levels compared to healthy controls (Kaye, Ebert, Raleigh, & Lake, 1984; Kaye, Gwirtsman, George, Jimerson, & Ebert, 1988; Kaye, Gwirtsman, George, & Ebert, 1991). Additionally, underweight individuals with AN and weight–restored individuals with restricting AN subtype have lower CFS concentrations of homovanillic acid, a metabolite of DA, compared to individuals with binge–purge subtype, bulimia nervosa (BN) and healthy controls (Kaye et al., 1984; Kaye, Frank, & McConaha, 1999). However, abnormalities of monoamine systems are not necessarily unique to AN but play a significant role in the pathophysiology of other psychiatric disorders such as depression and anxiety. Thus, it is unclear as to whether such alterations are a cause or consequence of AN, and/or a psychiatric comorbidity.

Developmental. Some theorists argues that AN is a function of an impaired child-maternal environment in the early years of childhood (Schwartz, Thompson, & Johnson, 1985). More specifically, the child splits off the inner representation of the mother because of chaotic and lack of empathetic mothering (Schwartz et al., 1985). As the child experiences

puberty and the body changes, the inner representation of the mother returns and at the expense of the self. At this time, individuals tend to have increased feelings of helplessness, confusion, dependency, and self-loathing. The development of AN is a maladaptive effort to cope with these feelings and to regain control of self and personhood (Schwartz et al., 1985).

Familial. Family systems theories about AN posit that families of individuals who struggle with AN are deeply enmeshed, overprotective, rigid, and unable to tolerate conflict (Schwartz et al., 1985). The development of AN is a manifestation of the child's inability to develop autonomy and self-realization because other family members (e.g., mothers, fathers) are preoccupied with and need to control the child's psychological and physical functioning (Schwartz et al., 1985).

Sociocultural. Thinness as an ideal female body shape and muscularity and leanness as the ideal male body shape permeate main culture in post–industrialized, high–income countries (Bazzini, Pepper, Swofford, & Cochran, 2015). However, the incidence of eating disorders in low– and middle–income countries may be growing because of increased media exposure to the Western ideal body shapes (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002; Becker et al., 2011; Eddy, Hennessey, & Thompson–Brenner, 2007). Although the clinical presentation of AN appears to be similar for both genders (Woodside, Garfinkle, Lin, Goering, & Kaplan, 2001), males are less likely to be diagnosed and seek treatment for an eating disorder (Merikangas et al., 2011). With regards to sexual orientation, gay men were more likely to report body dissatisfaction and eating pathology compared to heterosexual men; whereas, such differences were not observed among females (Morrison, Morrison, & Sager, 2004). Furthermore, researchers suggest that internalization of sociocultural appearance standards among men may mediate the relationship between conformity to masculine norms and drive for muscularity and leanness (Franko et al., 2015).

In the U.S., numerous sociocultural factors may contribute to the development of AN among ethnic and racial minority groups including acculturation, depictions of white females as the standard of beauty, membership in a devalued group, and stress associated with discrimination (Cachelin, Phinney, Schug, & Striegel–Moore, 2006; Gordon, Castro, Sitnikov, & Holm–Denoma, 2010). However, the prevalence of AN among non–white populations continues to be significantly lower compared to whites (Alegria et al., 2007; Hudson et al., 2007; Nicdao et al., 2007; Swanson et al., 2011; Taylor et al., 2007), and white females are more likely to select a thinner body shape ideal compared to non–white females (Gordon et al., 2010). It is possible that such differences continue to exist because mental health service utilization is significantly lower among ethnic and racial minority groups compared to whites (Merikangas et al., 2011), and diagnostic criteria are primarily based on white females and do not take into consideration cultural differences.

Prognosis and Course of Illness

As mentioned, AN has the highest mortality rate among all psychiatric disorders (Arcelus et al., 2011). In a recent meta–analysis of 36 studies of mortality in AN, the weighted annual mortality was 5.10, 95% confidence intervals [CI] [3.99, 6.14], per 1,000 persons per year, or 5.1% per decade (Arcelus et al., 2011). The standard mortality ratio was 5.86, 95% CI [4.17, 8.26] with a mean follow–up period of 14.2 years (Arcelus et al., 2011). In other words, the number of deaths among individuals with AN occurred at 5.86 times the rate expected in the general population. The majority of deaths were related to medical complications including cardiac and organ failure; and approximately 20% of deaths were the result of suicide (Arcelus et al., 2011).

The course of illness for AN is approximately 5 years in duration (Keski-Rahkonen et al., 2007; van Son, van Hoeken, van Furth, Donker, & Hoek, 2010). Remission rates vary based on time between discharge from IPT and follow-up, with a longer period of time

associated with better outcomes (i.e., higher recovery and improvement rates, lower mortality rates) but also higher mortality rates (Keel & Brown, 2010). Studies suggested that approximately 50% of adults recover from AN, 30% demonstrate improvement with residual symptoms, and 20% remained diagnosed with AN or had died at follow-up (Fichter, Quadflieg, & Hedlund, 2006; Steinhausen, 2002). Studies with adolescents suggested that about 75% of individuals recovered within 10 years and had a lower risk of relapse (Herpertz-Dahlmann et al., 2001). Prognostic indicators for outcome are associated with severity and duration of AN and the absence of unipolar depression (Castellini et al., 2011). Additionally, early detection and intervention are associated with better prognosis, and family involvement tends to limit the chronicity of AN (Fitzpatrick & Lock, 2011; Steinhausen, 2002).

As stated above, up to half of patients drop out of IPT prematurely (Fassino et al., 2009; Huas et al., 2011) and approximately 40% of weight-restored patients relapse within a year of discharge from IP level of care (Carter et al., 2012; Steinhausen, Grigoroiu-Serbanescu, Boyadjieva, Neumärker, & Metzke, 2008). Studies have suggested that binge-purging subtype, borderline personality disorder or traits, and level of motivation to recover are significantly related to dropout from IPT (Carter et al., 2012; Fassino et al., 2009). Patients have identified numerous factors that may contribute to their relapse after discharge from IPT such as ambivalence about recovery, being viewed as "dishonest," attention-seeking, and self-sabotaging, lack of control during hospitalization, and treatment focused on weight restoration and not psychological aspects (Bezance & Holliday, 2013; Carter et al., 2012; Westwood & Kendall, 2012).

In a six year follow–up study, Castellini and colleagues (2011) reported that diagnostic crossover rates were as follows: 23–27% of individuals developed BN, almost no one developed binge eating disorder (BED), and about 60% of individuals who relapse

developed BN. Crossover from AN to BN was associated with the presence of the following during AN diagnosis: unipolar depression, substance abuse, panic disorder, and the absence of obsessive compulsive disorder (Castellini et al., 2011). Furthermore, 30–43% of individuals with the restricting subtype of AN will develop bingeing and/or purging behaviors, which is associated with unipolar depression (Castellini et al., 2011).

Medical Sequelae of Anorexia Nervosa

During the course of AN, individuals may experience short–term and long–term medical complications related to the disorder. As mentioned above, as individuals restrict caloric intake, the human body downregulates to function in a decreased energy state and such a persistent hypo–metabolic state may lead to significant and potentially life-threatening consequences. The most common consequences of AN are detailed below and provided in Table 5. The majority of these medical consequences are reversible with nutritional rehabilitation.

Cardiovascular. Cardiovascular complications occur frequently among individuals with AN. Bradycardia (< 60 beats per minute), hypotension (< 90/60 mm Hg) and significant postural changes in heart rate and blood pressure are the most common (Misra et al., 2004). Arrhythmias, hypotension, mitral valve prolapse and tachycardia occur more frequently in individuals who struggle with chronic self–induced vomiting as well as misuse of diet pills, diuretics, laxatives, or supplements (Brown & Mehler, 2013). Additionally, individuals with AN may have structural and functional abnormalities (e.g., atrophy, decreased contractility, pericardial effusion, reduced mass), but these irregularities are usually reversible with weight restoration, nourishment, and decreased compensatory behaviors (Katzman, 2005).

Individuals with AN are frequently asymptomatic even with significant abnormalities in heart rate and blood pressure, and it is usually during refeeding and weight restoration that cardiovascular complications become more apparent. Notably, hypotension and significant

postural changes in vital signs do not necessarily indicate dehydration, and thus aggressive volume replenishment may lead to cardiac overload or congestive heart failure (Katzman, 2005).

Digestive/Gastrointestinal. Gastrointestinal symptoms are common among individuals with AN. Although it remains unclear as to whether gastric abnormalities are a cause or consequence of the disorder, individuals with AN frequently complain of constipation, early satiety, postprandial discomfort, acute intestinal occlusion, and swollen salivary glands (Zipfel et al., 2006). Not only can decreased food intake contribute to delayed gastric emptying, dysmotility, constipation, dyspepsia, and reflux (Hadley & Walsh, 2003), but chronic/severe self–induced vomiting may lead to esophagitis, esophageal rupture, hematemesis, parotid swelling, reflux, and spontaneous emesis (Brown & Mehler, 2013). Chronic and/or severe binge eating also increases the risk of gastric dilation and gastric rupture (Hadley & Walsh, 2003). Additionally, significant delays in gastric emptying and constipation may play a role in other medical complications, and contribute to difficulties, both psychological and physiological, with refeeding and weight restoration.

Endocrine/Metabolic. Individuals with AN tend to have hypothalamic suppression including low gonadotropin and sex hormone levels as well as low insulin-like growth factor-I, thyroxine, and triiodothyronine levels (Katzman, 2005; Misra et al., 2004; Warren, 2011). Adolescents may present with decelerated linear growth, pubertal delay or regression, and menstrual dysfunction for females (Misra et al., 2004). Unfortunately, even with weight restoration and nutritional rehabilitation, individuals may not achieve full growth potential (Lantzouni, Frank, Golden, Shenker, 2002).

Immunologic/Hematologic. In a recent review of hematology and AN, approximately one third of patients had anemia and leukopenia, 5–10% had thrombocytopenia, and about 50% showed signs of bone marrow atrophy (Hűtter, Ganepola,

& Hofmann, 2009). However, alterations returned to normal with refeeding and weight restoration (Hűtter et al., 2009).

Musculoskeletal. Osteoporosis and osteopenia are common and serious complications of AN, and may not be reversible with weight restoration. Research suggest that adolescents and adults with AN have lower bone mineral density, decreased bone microarchitecture and strength estimates (e.g., cortical and trabecular thickness), and impaired bone metabolism compared to healthy controls (Katzman, 2005; Misra & Kilbanski, 2014). Additionally, females with AN have a higher risk for fractures and joint problems compared to the general population (Faje et al., 2014; Mehler, Cleary, & Gaudiani, 2011). The cause of reduced bone mineral density in this population appears to be multifaceted and factors may include abnormal secretion of leptin, growth hormone resistance, hypogonadism, hypercortisolism, poor nutritional intake, and reduced fat mass (Katzman, 2005; Warren, 2011; Winston, 2012). Mehler and MacKenzie (2009) conducted a systematic review on controlled clinical studies of interventions for low bone mass in AN, and concluded that there is a lack of rigorous evidence to guide medical intervention once bone loss has occurred. However, early detection and weight restoration reduces the development of osteoporosis or other permanent bone damage.

Fluids and electrolytes. Malnutrition and compensatory behaviors (e.g., misuse of diet pills, diuretics, laxatives, and ipecacs, self-induced vomiting) contribute to both acute and chronic biochemical disturbances (Brown & Mehler, 2013; Winston, 2012). Additionally, fluid restriction and self–induced vomiting may lead to dehydration and renal insufficiency as well as other renal complications (e.g., pyuria, proteinuria, and hematuria). Hypokalemia and hypophosphatemia are the most common electrolyte abnormalities in individuals with AN (Winston, 2012). Other electrolyte disturbances include hypocalcemia, hypomagnesemia, hyponatremia, and low urea and creatinine. During treatment and nutrient rehabilitation,

biochemical changes may occur, specifically in fluid and electrolyte balance as well as micronutrient status (Mehanna et al., 2008; Solomon & Kirby, 1990). Individuals must be monitored closely to prevent cardiac and respiratory failure secondary to rapid shift from a catabolic to an anabolic state (e.g., depleted thiamine, hypophosphatemia, hypokalemia, and hypomagnesemia; Mehanna et al., 2008; Winston, 2012).

Table 5.

Medical Sequelae of Anorexia Nervosa

System	Signs & Symptoms
Cardiovascular	Bradycardia (< 60 beats/minute), cardiomyopathy, chest pain, congestive heart failure, dysrhythmias, electrocardiogram irregularities, hypotension (< 90/60 mm Mg), mitral valve prolapse, orthostatic hypotension, pericardial effusion, prolonged QT interval, shortness of breath, sudden death
Digestive Gastrointestinal	Abdominal discomfort/pain, abnormal liver function, acute pancreatitis, benign parotid hyperplasia, bloating, constipation, delayed gastric emptying, dental caries, diarrhea, esophagitis, esophageal or gastric rupture, gastric distention, gingivitis, reflux, vomiting
Endocrine Metabolic	Amenorrhea, delayed puberty, enlarged parotid glands, growth retardation, hypoglycemia, hypothermia, hyperamylasemia, hypercortisolism Decreased estrogen, luteinizing hormone, follicle-stimulating hormone, testosterone, and thyroxine; increased growth hormone, cortisol, & cholesterol
Immunologic Hematologic	Anemia, bone marrow suppression, bruising/clotting abnormalities, cold intolerance, decreased albumin, fatigue, impaired cell-mediated immunity, leukopenia, low sedimentation rate, thrombocytopenia
Integumentary	Acrocyanosis, dry & brittle hair, hair loss, lanugo, loss of subcutaneous fat, pitting edema, Russell's sign, yellowing of skin (related to hypercarotenemia)
Musculoskeletal	Arrested skeletal growth, fractures, muscle weakness, muscle aches and cramps, muscle wasting, osteopenia, osteoporosis
Neurological	Anxiety, depression, cognitive impairment, cortical atrophy, fatigue, irritability, myopathy, peripheral neuropathy, seizures, vertigo
Fluids & electrolytes	Dehydration, hypokalemia, hypomagnesemia, hyponatremia, hypophosphatemia, ketonuria, metabolic alkalosis, metabolic acidosis, peripheral edema, renal impairment
Reproductive	Arrested sexual development, fertility & pregnancy problems, neonatal complications

Note: Adapted from "Caring for the hospitalized patient with an eating disorder," by B. E. Wolfe & L. B. Gimby, 2003, *The Nursing Clinics of North America*, 38*,* p. 81; adapted from "The clinical biochemistry of anorexia nervosa," by A. P. Winston, 2012, *Annals of Clinical Biochemistry*, 49*,* p. 132-143.

Psychiatric Comorbidities

Prevalence rates of at least one psychiatric comorbidity among individuals with AN range from 45% to 97% (Blinder, Cumella, & Sanathara, 2006; Swanson et al., 2011), and the presence of psychiatric comorbidities is associated with the severity of eating disorder symptomatology (Brand-Gothelf, Leor, Apter, & Fennig, 2014; Fernández-Aranda et al., 2008; Spindler & Milos, 2007). Additionally, individuals with AN who experience childhood emotional abuse tend to have higher reports of emotion dysregulation and symptom severity (Racine & Wildes, 2015). Prior to the onset of AN, individuals tend to display specific characteristics in childhood including anxiety, depression, obsessive traits, and perfectionism (Bardone-Cone et al., 2007; Halmi et al., 2012). The literature has consistently shown the association between substance use and bulimic behavior (Herpertz–Dahlmann, 2015), AN and anxiety disorders (O'Brien & Vincent, 2003), and AN and obsessive-compulsive disorder (OCD) (Blinder et al., 2006; Halmi et al., 2005). Although 40.0–77.6% of individuals with AN seek treatment for any emotional problem, only 12.8–33.8% reported service utilization for the treatment of AN (Hudson et al., 2007; Merikangas et al., 2011; Swanson et al., 2011).

Anxiety disorders. About one-fourth of patients with AN report at least one anxiety disorder, and the most frequent anxiety disorders are separation anxiety disorders, social phobia, and specific phobias (Salbach–Andrae et al., 2009; Swanson et al., 2011).

Bipolar, depressive, and related disorders. There is a high frequency of mood disorders (up to 95%) among individuals with AN (Blinder et al., 2006; Godart et al., 2015).

Individuals often endorse depressive signs and symptoms, such as depressed mood, difficulties with concentration, insomnia, irritability, low energy, and social isolation (Attia & Walsh, 2007). This population also has an elevated risk for suicidal ideation, suicidal gestures, suicide attempts compared to the general population (Kostro et al., 2014; Swanson et al., 2011). Individuals with binge-eating/purging AN subtype have higher rates of impulsivity, suicidality and self-harming behaviors compared to restricting AN subtype (Kostro et al., 2014). Additionally, manic and hypomanic episodes are more frequent among persons with binge-eating/purging AN subtype compared to individuals with restricting subtype (Godart et al., 2015).

Trauma and stress-related disorders. Individuals with AN have a higher likelihood of experiencing childhood abuse and trauma (Harrison et al., 2010; Racine & Wildes, 2015). Individuals with binge-purging AN subtype are twice as likely to have post-traumatic stress disorder compared to persons with restricting AN subtype (Blinder et al., 2006).

Obesessive–compulsive and related disorders. This population tends to describe the presence of anxiety, perfectionism, or obsessional traits during childhood or prior to onset of AN (Bardone-Cone et al., 2007; Godart et al., 2015; Halmi et al., 2012). Compared to individuals with other eating disorders, individuals with AN are twice as likely to have OCD (Blinder et al., 2006), and symptoms do not include eating disorder–related obsessions and compulsions and may be related to personality disorders. Not only is OCD associated with the chronicity and severity of AN (especially among restricting AN subtype; Blinder et al., 2006; Halmi et al., 2012), but obsessive–compulsive traits are related to poorer treatment outcomes (Crane, Roberts, & Treasure, 2007; Nilsson, Sundbom, & Hagglof, 2008).

Substance–related and addictive disorders. Approximately one-fourth of individuals with AN struggle with substance-related and addictive disorders. Persons with binge-eating/purging AN subtype are twice as likely to have such a disorder compared to

individuals with restricting AN subtype (Baker et al., 2013; Blinder et al., 2006; Bühren et al., 2014; Root et al., 2010).

Personality disorders. There are high prevalence rates for Cluster B personality disorders (i.e., borderline, histrionic, narcissistic) among individuals with binge–eating/purging AN subtype. Whereas, Cluster C personality disorders occur more frequently among individuals with restricting AN subtype (i.e., avoidant, dependent, obsessive-compulsive; Herpertz–Dahlmann et al., 2001; O'Brien & Vincent, 2003).

Stigma & Anorexia Nervosa

Individuals with AN experience stigma associated with mental illness and eating disorders. This population is viewed as attention–seeking, conceited, emotional, insecure, manipulative, and weak (Crisp, 2005; Crisp, Gelder, Rix, Metzler, & Rowlands, 2000; Overton & Medina, 2008), as well as responsible for the disorder (Crisp, 2005; Johnstone & Rickard, 2006; Mond, Robertson-Smith, & Vetere, 2006; Stewart, Schiavo, Herzog, & Franko, 2008). Not only does AN have devastating psychological and physical consequences, but the stigma related to mental health and eating disorders has social and psychological consequences including decreased employment and education opportunities, decreased long–term relationships, and decreased self–esteem (Corrigan, 1998; Corrigan & Watson, 2002; Wahl, 1999). Additionally, stigma reduces the likelihood of individuals to seek treatment for this life–threatening disorder (Hinshaw, 2006).

Anorexia Nervosa and Inpatient Treatment

Standards of care and practice guidelines for IPT of AN are primarily based on clinical expertise and consensus (APA, 2006; Anderson, 1985). Although evidence–based practices with empirical support are not well-established for IPT, this level of care is more effective at facilitating weight gain compared to others (APA, 2006). Inpatient treatment is often required to intervene with medical consequences of AN, starvation-induced cognitive

impairment, and psychiatric concerns (e.g., suicidal ideation; Garber et al., 2012; Katzman, 2012). This level of treatment is recommended under the following circumstances: (1) severe malnutrition and weight less than 75% of ideal body weight (IBW), (2) inability to interrupt weight loss on an outpatient basis, (3) medical complications (e.g., dehydration, electrolyte abnormalities, cardiac complications), (4) suicidal ideation or psychiatric comorbidity, and (5) environmental barriers blocking progress on an outpatient basis (e.g., family conflict, financial issues; APA, 2006).

The inpatient setting provides structure and support for the normalization of eating, but may also foster dependence and a sense of detachment from the outside as well as a loss of control and sense of identity for patients (Colton & Pistrang, 2004; Offord, Turner, & Cooper, 2006). Collaboration between treatment team and patients as well as discharge planning (e.g., education about cognitive, emotional, and behavioral changes, medical stability, and meal planning) provide patients with the opportunity to feel empowered, reconnect with normality outside of the inpatient setting, and feel prepared for discharge (Bezance & Holliday, 2013; Turrell, Davies, Graham, & Weiss, 2005).

Inpatient Treatment and Weight Restoration

Nutritional rehabilitation and weight restoration (i.e., ideally more than 85% IBW) are the primary goals of IPT for AN, and other treatment goals include achieving normal perception of hunger and satiety, correcting biological and psychological effects of malnutrition, and normalizing eating patterns (APA, 2006). It is recommended that healthy weight estimates be individualized and based on various measurements and clinical factors (e.g., the return of menses and normal ovulation in females; normal sexual drive and hormone levels in males; normal physical and sexual growth and development in children and adolescents; APA, 2006). During IPT for AN, integrated treatment modalities (e.g., art therapy, cognitive and dialectical behavioral therapies, individual, family and group

psychotherapies, motivational interviewing, psychopharmacology) and an interdisciplinary approach (e.g., medical, nursing, nutritional, psychiatric, social work) are recommended (APA, 2006).

As noted above, a primary goal of IPT for AN is weight restoration to more than 85% IBW (APA, 2006), and the recommended average weekly weight gain is 0.9–1.4 kg per week (2–3 pounds; APA, 2006). The admission caloric intake typically starts at 1,000–1,600 calories per day and then increases as much as 70–100 calories per kilogram per day (APA, 2006). Individuals are encouraged to gain weight by ingesting liquids and/or solid foods during supervised meals and are expected to consume 100% the prescribed calories. If an individual is unable to or unwilling to consume the prescribed calories, then a liquid nutrition supplement or nasogastric feeding is provided (APA, 2006). Individuals with AN may require 5,000–10,000 excess calories to gain one kilogram due to the variability in physical activity, energy efficiency, thermoregulatory response, fluid shifts, age, and treatment phase (Marzola, Nasser, Hashim, Shih, & Kaye, 2013; Mehler, Winkelman, Andersen, & Gaudiani, 2010). Hypermetabolism and diet-induced thermogenesis may influence weight changes because patients may have reduced efficiency in converting calories into tissue and may experience elevated body temperatures (Garber et al., 2012; Reiter & Graves, 2010; Yamashita et al., 2010).

Although weekly body weight changes tend to be used to evaluate progress (APA, 2006), daily body weights are monitored to avoid refeeding syndrome (APA, 2006). Refeeding syndrome is characterized by a rapid shift from a catabolic to an anabolic state, and biochemically includes hypophosphatemia, hypomagnesium, hypokalemia, glucose intolerance, fluid overload, and thiamine deficiencies (Mehanna et al., 2008; Winston, 2012). Clinical characteristics include: congestive heart failure, hypotension, respiratory failure, rhabdomyolysis, coma, seizures, skeletal and muscular weakness, and metabolic acidosis

(Winston, 2012). Assessment of refeeding syndrome includes vital signs, laboratory results, electrocardiogram, monitoring for rapid weight gain, edema of extremities, chest pain, and gastrointestinal symptoms (APA, 2006; Mehanna et al., 2008).

Existing recommendations state that patients should be weighed after voiding at the same time of day (i.e., morning) and under the same circumstances (e.g., amount/type of garments worn). According to the APA guidelines (2006), weigh-ins should be completed in the morning after voiding and supervised by nursing staff on calibrated scales, with the patient wearing only underwear and/or a hospital gown. Anthropometric devices are usually inspected and calibrated per the protocol of the treatment facility, and detailed records of weights are maintained as part of the standard of care. Unfortunately, standards of care and practice guidelines for IPT do not provide recommendations about whether or not to inform individuals with AN of their body weight. Thus, institutional protocols and practices during IPT for AN are either informing or not informing patients of body weight, and very few choose a more individualized approach.

Body Weight Gain: A Proxy of Weight Restoration

An overview of the existing literature on body weight gain, a proxy of weight restoration during IPT for AN, will be discussed. Research about body weight gain as a predictor of treatment outcomes will be reviewed. Details about descriptive studies investigating rate of weight gain during IPT for AN and research about predictors of such rates will be discussed, followed by a summary of the research examining the parameters (e.g., intercepts, slope) of weight gain.

Weight gain and treatment outcomes. Research indicates that weight restoration during IPT may be a promising clinical indicator of treatment outcomes. For example, inadequate weight gain during the first admission (Zipfel et al., 2000), and low weight at time of discharge (Baran et al., 1995) predicted poor outcomes (e.g., re-hospitalization, psychiatric

status rating score). Other studies reported that higher weight or BMI at discharge predicted weight maintenance at one year follow-up (Kaplan et al., 2009; Lock & Litt, 2003), and individuals who gained at least 0.8 kg per week were less likely to experience clinical deterioration (based on Clinical Global Impression Severity scores; Lund et al., 2009).

Rate of weight gain during IPT for AN. Three studies have provided descriptions of actual versus recommended weight gain among individuals during IPT for AN (Davies & Jaffa, 2005; Garber et al., 2012; Hart et al., 2011). Davies and Jaffa (2005) reported a mean weekly weight gain of 0.82 kg (*SD* = 0.29) per week among 53 adolescents which is less than the expected rate of weight gain (1.0–1.5 kg). Results indicated that weight gain tended to be "poor" in week 1, varied during weeks 2–14 (0.7–1.0 kg/week), and then tended to plateau. Only 69.8% (*n* = 37) reached target weight range, 11.3% (*n* = 6) did not reach target weight range, and 18.7% (*n* = 10) left treatment before a target weight range was set. Another group of researchers examined weight gain among 35 adolescents, and reported mean daily weight gain of 0.15 kg (*SD* = 0.10) and mean average total weight gain of 2.42 kg (*SD* = 1.85) from day 1 until day of discharge (Garber et al., 2012). Similar to Davies and Jaffa (2005), Garber and colleagues found that 83% of patients initially lost weight with peak weight loss of 0.60 (*SD* = .57) kg on day 2.9 (*SD* = 1.1), and significant weight gain tended to occur starting on Day 8 and continued until discharge. Hart and colleagues (2011) examined weight change in 247 female patients representing 414 inpatient admissions, and reported that approximately 21.3% of admissions (*n* = 88) did not meet the expected rate of weight gain of 1.0 kg per week. More specifically, 4.6% of admissions (*n* = 19) lost weight at a rate of –0.17 kg per week (a total loss of –0.83 kg over 37.3 days) and 16.7% of admissions (*n* = 69) gained less than expected per week (mean rate of 0.33 kg/week and a total gain of 4.5 kg over 88.9 days). Overall, research indicates variation in rate of weight gain during an inpatient admission for AN. A subset of individuals will initially lose weight secondary to fluid retention or have

minimal weight gain during the first week of treatment, followed by significant weight gain and/or weight fluctuations for the remainder of their admission (Garber et al., 2012). A smaller subset of patients will not gain, but lose weight during their admission (Hart et al., 2011).

Two studies have compared rates of weight gain among individuals with different expected weight gain criteria (Herzog, Zeeck, Hartmann, & Nickell, 2004; Solanto, Hertz, Jacobsen, Heller, Golden, 1994). Solanto and colleagues (1994) examined 53 adolescents during first inpatient admission, and suggested that a higher expected weight gain criterion was associated with greater rate of weight gain without increasing complications of refeeding. Another group of researchers compared different targets for minimum weekly weight gain (MWWG) among 32 male and female adults (Herzog et al., 2004). They reported no difference in rates of weight gain among the two groups, but a trend for the overall rate of weight gain to be greater in the low MWWG target group (achievers and non-achievers of target weight) compared to the high MWWG target group. Additionally, the low MWWG target group (0.5 kg) was more likely to reach target weight compared to the high MWWG target group (0.75 kg). For those who achieved target weight, individuals in the high MWWG target group were less likely to maintain weight during their admission and were more likely to discharge below target weight compared to individuals in the lower MWWG target group. Inconsistent results of the two studies regarding the impact of expected weight gain criterion on rate of weight gain may be explained with difference in sample (female adolescents on a general adolescent medicine unit vs. male and female adults on an inpatient eating disorder unit).

Overall, there were numerous limitations among these studies including small sample sizes, non-U.S. samples, treatment provided at specific facility, nonrandomized design, and patients appearing more than once in dataset. Hence, it is difficult to compare studies and

generalize results to other treatment facilities and populations of individuals diagnosed with AN.

Predictors of rate of weight gain. Two studies identified predictors of rate of weight gain (Garber et al., 2012; Neuberger et al., 1995). More specifically, a higher caloric starting diet predicted a greater rate of weight gain (Garber et al., 2012), and a younger age at admission and a higher percent of IBW on day 1 predicted a greater rate of weight gain for restricting subtype of AN (Neuberger et al., 1995)

Parameters of weight gain. More recently, researchers have used statistical techniques usually applied for the analysis of longitudinal growth curves to deduce longitudinal parameters of patients' weight. Below are three studies that have used such statistical techniques to examine nonlinearity of weight changes among individuals diagnosed with AN during IPT. Mewes and colleagues (2008) examined parameters of weight curves as predictors of drop-out and completion of treatment among 100 females. Among treatment dropouts (n = 50), predictors of BMI up to discharge were higher average weekly BMI gain, lower average magnitude of weight loss, and longer duration of the first half of treatment. Among treatment completers (n = 50), predictors of BMI up to discharge were lower BMI at admission and higher average weekly BMI gain in the first half of treatment.

Another study investigated 40 female adolescents, and the course of changes in body weight during admission, analyzed the time to onset of weight loss following discharge, time to readmission, and the relationship between inpatient weight curve characteristics and post-hospital relapse criteria (Lay et al., 2002). Results showed that the average overall weight increase was 9.4 kg or 2.5 kg per month, and 77.5% of subjects (n = 31) achieved target weight but only 22.5% (n = 9) maintained it for at least 10 days while in treatment. Additionally, about 72% of patients had substantial weight loss within 8 weeks of discharge, and 77.5% were readmitted within 12 months following discharge. Finally, patients with

greater negative variation of their weight curve and greater tempo of their weight increase were more likely to lose weight after discharge compared to others.

Hartmann and other researchers (2007) examined predictors of failure in the early course of first admission for IPT for AN among 85 patients using growth curve analysis (to model individual weight curves), regression with methods of optimal scaling (to model nonlinear relationships between outcome and curve parameters), and receiver operating characteristics curve analysis (to determine the best balance of sensitivity and specificity based on regression results). Results showed that BMI at intake was negatively correlated to outcome with a higher mean BMI at intake (0.8 points higher) of those who did not respond to treatment compared to those who did respond to treatment. The more advanced statistical analyses revealed the following results. Patients who gained weight in weeks 1–2 and then proceeded to have a significant weight gain during weeks 3–4 without much fluctuation, had a higher probability of at least moderate treatment success. In contrast, patients who lost weight in weeks 1–2 and even more weight loss in weeks 3-4 with fluctuation, had a higher probability of treatment failure. Results also indicated that having a constant but slow weight gain during weeks 1–2 does not indicate treatment failure.

Researchers have begun to take into consideration the importance of capturing changes in body weight over a period of time using statistical analyses that more appropriately fit the data. Unfortunately, there are numerous limitations of these studies such as small sample size, non-U.S. sample, exclusion of admissions less than 6 weeks, exclusion of data on early dropouts and early treatment failures, and arbitrary definitions of treatment success (e.g., greater than 2 BMI weight gain). Thus, generalizability of results is limited.

Summary

While researchers have examined the nonlinearity of weight changes during IPT for AN and identified predictors of weight gain (i.e., age at time of admission, admission caloric

intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge), research has yet to consider that weight restoration is an adaptive process with internal and external environments. The identification of patterns of change in body weight may point to predictors of the process of weight restoration, as well as provide knowledge that will allow providers to better anticipate care needs and tailor interventions for this population.

Chapter 3

Methodology

This chapter provides an overview of the design, sample, setting, and procedures that were used in the study. First, details of the pilot study for accessibility and feasibility are presented. Next, the dissertation study design, sample, setting, and procedures. Variables of interest and data collection instruments are provided, followed by a statement regarding protection of human subjects. The chapter concludes with a discussion of data analyses and sample size.

Pilot Study for Accessibility and Feasibility

Prior to this full–scale study, a descriptive pilot was conducted to determine the availability of relevant diagnostic, clinical, and anthropometric items and accessibility of patients' charts. Data of interest included identified predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge), demographic variables (e.g., age, gender, race/ethnicity), and clinical factors (e.g., duration of AN, history of prior IP treatment for AN, readmission status, psychiatric comorbidities).

The specific aims of the pilot study were (1) to determine the percent of patients' charts with missing data for each item, and (2) to determine how variables of interest will be studied in a full–scale study based on percent of missing data. Random sampling was used to select charts from an existing list of medical record numbers (MRNs) of all individuals with AN admitted for IPT. The patients' charts were retrieved from the Medical Records Room, reviewed for relevant data, and data of interest were transcribed onto an electronic data abstraction form. The feasibility criteria for determining the utility of each item was based on percent of missing data (Banks, 1998): (1) an item with < 6% of missing data was deemed most appropriate for inclusion; (2) an item with 6–15% of missing data was examined to

determine whether or not the range options needed to be changed; (3) an item with 16–25% of missing data was examined to determine whether or not alternative data needed to be abstracted; and (4) an item with >25% of missing data was unsuitable for inclusion.

Of the 50 charts, six (12%) were excluded because the patient was not diagnosed with AN (n = 4) or was diagnosed with a psychotic disorder (n = 2). Nineteen of the 64 items (29.7%) had missing data for at least one participant. Based on the percent of missing data, each item was deemed to be included or excluded (see Table 6). An additional three items that did not have missing data, were modified to ensure that the most appropriate information were obtained (see Table 7).

Table 6.

Variables, Missing Data, and Outcome

Variable	# Missing	% Missing	Outcome
Admission caloric intake (kcal per day)	2	4.5	Included
EDE-Q subscales & global scores at admission and discharge*	20	45.5	Included*
Suicidal ideation at time of admission	1	2.3	Included
Self-injurious behaviors at time of admission	1	2.3	Included
Substance abuse at time of admission	1	2.3	Included
Body weight (kg) at start of eating disorder	23	52.3	Excluded
Lowest weight (kg)	6	13.6	Included no change
Age (in years) at lowest weight	9	20.5	Excluded
Highest weight (kg)	4	9.1	Included no change
Age (in years) at highest weight	9	20.4	Excluded

Note: EDE-Q = Eating Disorder Examination Questionnaire; kcal = calorie; kg = kilogram.

*variables (EDE–Q four subscales and global scores) were not excluded because only

established measure of eating psychopathology.

Table 7.

Altered Items of Data Abstraction Form

Original Item	**Modifications**
What are the endorsed eating disorder behaviors at admission? Check all that apply.	ADDED ITEM: "Specify AMOUNT and FREQUENCY for each behavior endorsed in the previous question. (For example, restricting < 500 calories per day; self-induced vomiting up to 5 times per day)"
Does patient report medical complications at admission? Check all that apply.	CHANGED ITEM: "What are the medical diagnoses (if any) at admission?"
What is the patient's disposition at discharge?	ADDED OPTION: "Administrative"

Regarding daily weights, 6 individuals (13.6%) had at least one missing data point because of refusal to be weighed on one day (n = 2); refusal to be weighed on 3 consecutive days (n = 1); no documented weight for one day (n = 2); and undocumented weight for entire admission except Day 0 (n =1). For the three individuals who refused to be weighed, one person refused on day 3 of 3, another refused on day 14 of 20, and one person refused on days 3, 4, and 5 of 30. For the two charts with no documented weight for one day, one person did not have a documented weight on day 11 of 45, and the other person did not have a documented weight on day 8 of 9. The length of stay for the person with undocumented weights for the entire admission except Day 0 was four days.

The overall time required to retrieve and return 50 charts was approximately 3 hours. Each abstraction form required between 10 and 20 minutes to complete, with earlier admissions requiring more time because of outdated standardized forms and the need to look in different locations in the chart for data.

Study Design

This retrospective, exploratory dissertation study used a time-series design to provide a description of patterns of change in body weight for individuals diagnosed with AN during IPT. Time-series is a design in which multiple observations are made longitudinally, and is useful in exploring changes over time (Abraham & Neundorfer, 1990).

Subjects

The study sample was individuals diagnosed with AN who were admitted to the inpatient unit at an eating disorder treatment facility in the Northeast. Based on the inclusion criteria for admission to the inpatient unit, all genders and races and/or ethnicities were included in this study.

Inclusion criteria was: 1) diagnosis of AN at admission, 2) BMI < 18.5 kg/m^2, 3) age ≥ 10 years old (based on minimal unit admission age), 4) gender, male or female (based on all genders being admitted), 5) any race and/or ethnicity (based on all races and/or ethnicities being admitted), and 6) hospitalized on an inpatient unit between January 1, 2012 and December 31, 2015. Assessment and diagnosis of AN were based on both the *DSM-IV* and *DSM-5* (APA, 2000; 2013) and determined by an unstructured clinical interview with a unit psychopharmacologist (i.e., psychiatrist, advanced practice nurse practitioner). Diagnostic criteria for AN have undergone several changes in the latest version of the *DSM*. For Criterion A, *DSM-IV* required a refusal to maintain body weight at or above minimal expectations (e.g. < 85% expected body weight), and *DSM-5* no longer specifies a numerical standard to define low. Instead, the *DSM-5* requires specification of severity level which is based on BMI for adults and BMI percentile for children and adolescents. For Criterion B, *DSM-IV* required an explicit fear of gaining weight or becoming fat; and *DSM-5* permits individuals to *either* endorse this fear *or* engage in persistent behavior that interferes with weight gain despite being at low weight. Criterion D (amenorrhea) has been removed from

the *DSM-5.* The diagnosis of AN was based on the diagnostic criteria for AN in the 4th and 5th editions of the *DSM.* The specific version used for a diagnostic purpose was contingent on admission date with the *DSM-5* being used exclusively on the inpatient unit since October 1, 2015. A recent study comparing operational definitions of *DSM-5* for research contexts suggested using an operational definition of BMI < 18.5 kg/m^2 to balance inclusivity, disorder homogeneity or clinical significance, and validity (Brown, Holland, & Keel, 2014). Based on availability of charts and taking into consideration seasonal effects (e.g., summer versus winter) and time of year (e.g., holidays), inpatient admissions considered for inclusion in this study ranged from January 1, 2012 to December 31, 2015.

Exclusion criteria were: 1) meets *DSM-IV* or *DSM-5* criteria for BN, binge eating disorder, avoidant/restrictive food intake disorder, unspecified eating disorder, or eating disorder not otherwise specified at admission, 2) meets *DSM-IV* or *DSM-5* criteria for psychotic disorder at admission (based on cognitive impairment), and 3) prior participation in the study (omitted repeat admissions). The purpose of this study was to examine individuals with AN, and thus individuals with an admission diagnosis of a different eating disorder were not included.

Setting

The setting was an organization providing a full continuum of care to individuals and families suffering from eating and psychiatric disorders in the Northeast. The eating disorders inpatient unit has a bed capacity of 23 with a readmission rate of 13.4% and an average length of stay 13.8 days. According to the organization's Admission Weight Guidelines and Interventions," potential admissions must be at least 60% of ideal body weight (without consultation) and are expected to complete 100% of meals and snacks which may include the use of a nasogastric feeding tube. The primary goals of the eating disorder

inpatient program include weight restoration and monitoring, nutrition education, and milieu therapy to address abnormal eating attitudes and behaviors.

Upon admission to the inpatient unit, medical, psychiatric, psychosocial, psychological and nutritional evaluations were completed. The medical examination includes anthropometric measurements (i.e., height, weight), review of systems, and laboratory blood work. The psychiatric evaluation includes assessment and clarification of the eating disorder diagnosis and psychiatric comorbidities, safety or potential harm to self or others, and incoming medication regimen. Patients were weighed on the day of admission and then daily over the course of treatment. All weigh-ins were supervised by nursing staff and occurred on calibrated scales with the patient wearing underwear and/or hospital gown. Per protocol, patients were blindly weighed (i.e., not able to view weight). The anthropometric devices were inspected and calibrated per the treatment facility's protocol. Detailed records of daily body weights were kept as part of the standard of care.

Procedures

The treatment facility provided a list of all admissions diagnosed with AN for IPT including MRNs and date of admission. To select participants, simple random sampling was used which is a version of probability sampling or "'the gold standard' for ensuring generalizability and rigorous basis for estimating the fidelity with which phenomena observed in the sample represent those in the population" (Hulley, Newman, & Cummings, 2013, p. 28). Random sampling ensures that each individual in the population has an equal probability of being selected, which then allows for more generalizability of the results (Creswell, 2014). For the pilot study, 50 MRNs were randomly selected, and 6 patients (12%) were excluded because they did not meet inclusion criteria (i.e., diagnosis of AN) or met exclusion criteria (i.e., diagnosis of psychotic disorder at admission). For this study, the target sample size was 500, and MRNs were randomly selected from the list of all admissions with

a diagnosis of AN. Medical record numbers were stratified by quarters (i.e., January–March, April–June, July –September, October–December) to account for seasonal and academic variability.

Since individuals may have had more than one admission, a MRN may have repeated on the list of all admissions. A MRN was included in the study up to one time, and subsequent selections of the number were discarded. This method allowed for 500 unique MRNs. The admission date associated with the selected MRNs was used to retrieve the patients' charts of the specific admission from the Medical Records Room. Thus, the randomly selected admission was not necessarily the first admission for the patient.

Once the charts of the specific admission were retrieved from the Medical Records Room, the principal investigator (PI) reviewed the chart for initial data on relevant demographic, clinical, and diagnostic variables. Screening assessment procedures involved collecting data on psychiatric diagnoses and eating disorder presentation at time of admission. If the individual did not meet criteria to participate in the study, then the chart was returned to its appropriate place in the Medical Records Room and another MRN was selected. For individuals who met criteria for this study, the PI continued to review patients' charts to retrieve the remaining data on diagnostic and clinical variables for the entire admission.

Data Collection Instruments

This section provides an overview of the instruments used in the study. The development and formatting of the electronic data abstraction, and details about the Eating Disorder Examination Questionnaire (EDE-Q; © 2008 by Christopher G. Fairburn and Sarah Beglin), an established measurement tool of eating psychopathology, are discussed.

Data Abstraction Form. An electronic data abstraction form was developed and formatted with Research Electronic Data Capture (REDCap; © 2015 by Vanderbilt

University; see Appendix A). REDCap is a secure web-based application for building and managing online surveys and databases, and automated export procedures to directly transfer to SAS/STAT® (Harris et al., 2009). A descriptive pilot study was conducted to determine the accessibility and availability of relevant diagnostic, clinical, and anthropometric variables in patients' charts. Based on the results, some questions and/or response options were reformatted to ensure that the most appropriate measurements of the variables of interest are utilized. Table 8 provides a list of variables of interest, the operational definitions, and the best source in the medical record to obtain desired data (Banks, 1998; Gearing, Mian, Barber, Ickowicz, 2006; Gregory & Radovinsky, 2012). An electronic abstraction form was completed for each participant.

Table 8.

Variables, Operational Definitions, Location of Information

Variable	Operational definition	Location of information
Age at time of admission	Defined as age in years and is the difference in date of admission and date of birth	Psychiatric Evaluation Form
Gender	Response options: 1) female; 2) male	Psychiatric Evaluation Form
Race/Ethnicity	Choose all categories that apply listed below. Responses will be coded as "0" = no (unchecked), "1" = yes. 1) White, non-Hispanic; 2) White, Hispanic; 3) Black, non-Hispanic; 4) Black, Hispanic; 5) Asian/Pacific Islander; 6) Native American; 7) other, specify	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Years of education	Response options: 1) ≤ 9 years; 2) > 9 and ≤ 12 years, high school or some high school; 3) > 12 and ≤ 16 years, college or some college; 4) > 16 years,	Psychosocial Assessment Form

	post-college education	
Marital status	Response options: 1) never married, single; 2) never married, in a relationship; 3) married; 4) separated; 5) divorced; 6) other, specify	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Living situation at time of admission	Response options: 1) alone; 2) with significant other; 3) with roommate/friend; 4) with family; 5) other, specify	Psychosocial Assessment Form Other locations: Nutrition Assessment Form
Readmission status	Response to whether or not readmission to unit Response options: "yes" or "no." Responses will be coded as "0" = no, "1" = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Prior IPT admissions for AN	Response options: "yes" or "no." Responses will be coded as "0" = no, "1" = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form; Nutrition Assessment Form
AN subtype	Response options: 1) restricting type; 2) binge-eating/purging subtype	Psychiatric Evaluation Form Other locations: Nutrition Assessment Form
Body weight at time of admission	Anthropometric measurement in kilograms on date of admission	Nutrition Assessment Form Other locations: Nutritional Discharge Summary Report Form
Body height at time of admission	Anthropometric measurement in meters on date of admission	Nutrition Assessment Form

BMI at time of admission	An integer based on the calculation kg/m^2 with "kg" being body weight in kilograms and "m" being body height in meters on date of admission.	Nutrition Assessment Form Other forms: Nutritional Discharge Summary Report Form
Eating disorder behaviors time of admission	Choose all behaviors that apply listed below. Responses will be coded as "0" = no (unchecked), "1" = yes. If response is "yes" then open-ended question inquiring about amount and/or frequency. Categories include: 1) restricting; 2) binge eating; 3) self-induced vomiting; 4) diuretics; 5) diet pills; 6) laxatives; 7) ipecac; 8) caffeine; 9) exercise	Nutrition Assessment Form Other locations: Psychiatric Evaluation Form
Admission caloric intake	Defined as kilocalories, or calories, per day assigned at time of admission	Nutrition Assessment Form
Age of AN onset	Defined as age in years of the first occurrence of AN diagnosis	Nutrition Assessment Form
Lowest weight	Anthropometric measurement in kilograms	Nutrition Assessment Form
Highest weight	Anthropometric measurement in kilograms	Nutrition Assessment Form
Duration of illness	Number of years based on the calculation: age at admission minus age of AN onset	N/A
Suicidal ideation at time of admission	Response options: "yes" or "no." Responses will be coded as "0" = no, "1" = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Self-injurious behaviors at time of admission	Response options: "yes" or "no." Responses will be coded as "0" = no, "1" = yes.	Psychiatric Evaluation Form

		Other locations: Psychosocial Assessment Form
Substance misuse at time of admission	Choose all substances that apply listed below. Responses will be coded as "0" = no (unchecked), "1" = yes. Categories include: 1) alcohol, 2) cocaine, 3) marijuana, 4) opiates, 5) sedatives, 6) others	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Psychiatric comorbidities at time of admission	Choose all comorbidities that apply listed below. Responses will be coded as "0" = no (unchecked), "1" = yes. Categories include: 1) Anxiety disorders; 2) Bipolar and related disorders; 3) Depressive disorders; 4) Neurodevelopmental disorders; 5) Obsessive-compulsive and related disorders; 6) Personality disorders; 7) Substance-related and addictive disorders; 8) Trauma- and stressor-related disorders; 9) Others	Psychiatric Evaluation Form
Number of psychiatric comorbidities at time of admission	Calculation will be the sum of endorsed categories of "types of psychiatric comorbidities" Categories will include: 1) None; 2) one; 3) two; 4) three; 5) 4+	Calculation based on response to "Types of psychiatric comorbidities"
Family history of psychiatric disorders and/or substance misuse	Response options: "yes" or "no." Responses will be coded as "0" = no, "1" = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Family history of eating disorders	Response options: "yes" or "no." Responses will be coded as "0" = no, "1" = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form

History of traumatic experiences	Response options: “yes” or “no.” Responses will be coded as “0” = no, “1” = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
Type of traumatic experiences	Choose all types of trauma that apply listed below. Responses will be coded as “0” = no (unchecked), “1” = yes. Categories include: 1) emotional/psychological, 2) physical, 3) sexual	Psychiatric Evaluation Form
History of self-injurious behaviors	Response options: “yes” or “no.” Responses will be coded as “0” = no, “1” = yes.	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form
History of suicidal ideation	Response options: “yes” or “no.” Responses will be coded as “0” = no, “1” = yes.	Psychiatric Evaluation Form Other locations: Psychiatric Discharge Note Form Psychosocial Assessment Form
History of suicide attempts/gestures	Response options: “yes” or “no.” Responses will be coded as “0” = no, “1” = yes.	Psychiatric Evaluation Form Other locations: Psychiatric Discharge Note Form Psychosocial Assessment Form
History of substance misuse	Response options: “yes” or “no.” Responses will be coded as “0” = no, “1” = yes. If yes, then choose all substances that apply listed below. Responses will be coded as “0” = no (unchecked), “1” =	Psychiatric Evaluation Form Other locations: Psychosocial Assessment Form

	yes. Categories: 1) alcohol, 2) cocaine, 3) marijuana, 4) opiates, 5) sedatives, 6) others	
Discharge disposition	Choose one of the options listed below. Responses will be coded as "0" = no (unchecked), "1" = yes. Categories: 1) residential within facility, 2) partial hospitalization within facility, 3) intensive outpatient treatment within facility, 4) against medical advice, 5) administrative, 6) transfer to other facility at lower level of care, 7) other, specify	Psychiatric Discharge Note Form Other locations: Nursing Discharge Summary Form
Length of stay	The number of days based on the calculation: discharge date minus admission date	Nursing Discharge Summary Form
Body weight at time of discharge	Anthropometric measurement in kilograms on date of discharge	Psychiatric Discharge Note Form Other locations: Nutritional Discharge Summary Report Form
BMI at time of discharge	An integer based on the calculation kg/m^2 with "kg" being body weight in kilograms and "m" being body height in meters on date of admission.	Nutritional Discharge Summary Report Form
Eating psychopathology at time of admission	Global score and subscale scores on 4 subscales (Restraint, Shape Concern, Weight Concern, and Eating Concern)	Eating Disorder Examination Questionnaire (1)
Eating psychopathology at time of discharge	Global score and subscale scores on 4 subscales (Restraint, Shape Concern, Weight Concern, and Eating Concern)	Eating Disorder Examination Questionnaire (2)

Note: AN = anorexia nervosa; BMI = body mass index; IPT = inpatient treatment

Eating Disorder Examination Questionnaire (EDE-Q 6.0). The EDE-Q 6.0 was administered to each patient on day of admission and discharge. For this study, the four subscales (Restraint, Eating Concern, Shape Concern, and Weight Concern) and global scores were used as a descriptive measure of eating disorder psychopathology. The EDE-Q 6.0 is a 28-item measure (© 2008 by Christopher G. Fairburn and Sarah Beglin) derived from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; see Appendix B). The EDE is a well-established semi-structured interview designed to assess eating disorder psychopathology primarily occurring over the preceding 28 days (Fairburn & Cooper, 1993). Studies of the validity of the EDE-Q have confirmed a high level of agreement between the EDE-Q and EDE in measuring core attitudinal features of eating disorder psychopathology in the general population, bariatric surgery candidates, individuals diagnosed with BN and BED, and adolescents diagnosed with BN and AN (Anderson, De Young, & Walker, 2009). Among studies included in a recent systemic review of the psychometric properties of the EDE-Q and EDE, internal consistency was acceptable with the following range of alpha coefficients: Restraint (.70–.85), Eating Concern (.73–.86), Shape Concern (.83–.93), and Weight Concern (.72–.89; Berg, Peterson, Frazier, & Crow, 2012). The EDE-Q tended to yield higher scores than the EDE for all subscales.

Psychometric data pertaining to the EDE-Q have confirmed its reliability and validity in assessing eating disorder psychopathology. Internal consistency in the EDE-Q has been shown to be good, with Cronbach's alpha coefficients ranging from .70 to .83 in a clinical sample and from .78 to .93 in a general population sample (Luce & Crowther, 1999; Anderson et al., 2009). Mond and colleagues (2004) reported an alpha coefficient of .93 for the global scale. Two studies examined test-retest reliability of EDE-Q scores over 1 to 14 days, and reported the test-retest correlations ranged from .66 to .94 for subscale scores (Reas, Grilo, & Mashab, 2006; Luce & Crowther, 1999) and .40 to .78 on the individualized

items used to create the four subscales (Reas et al., 2006). One of the studies examined a community sample of undergraduate students (Luce & Crowther, 1999), and the other study examined women seeking treatment for BED (Reas et al., 2006). Four studies examined and support the ability of the EDE-Q to differentiate between eating disorder and control groups (Berg et al., 2012), with one of the four studies examining adolescent females with and without AN (Engelsen & Laberg, 2001).

The EDE-Q is scored using a 7-point, forced-choice rating scale (0–6) with scores of 4 or higher indicative of clinical range. The subscale and global scores reflect the severity of aspects of the psychopathology of eating disorders. To obtain subscale scores, the ratings for the relevant items are added together and the sum divided by the total number of items forming the subscale. If ratings are only available on some items but more than half, then a score is obtained by dividing the resulting total by the number of rated items. A "global" score is the sum of the four subscale scores divided by the number of subscales (i.e., four).

Protection of Human Subjects

Prior to the onset of this study, approval was obtained with an inter-institutional agreement between Boston College's Institutional Review Board and the organization (see Appendix C). De-identified or non-identifiable data was used and recorded on an electronic data abstraction form. To maintain confidentiality of private and protected health information, unique sequential study numbers were created using four digits, starting with "0001." The code chart linking medical record and study numbers was stored separately in an electronic file only accessible to the PI and dissertation committee. The computer used for data analysis was password protected, and an external hard drive provided a back-up copy of the data. Only CITI-trained members of the research team and dissertation committee had access to the data files. Data will be maintained for a maximum of five years after the completion of the study.

Data Analyses

The data was analyzed using STATA®, statistical software. Statistical significance was set at $p<.05$, two-tailed. Normal distribution (e.g., skewness, kurtosis) was evaluated. Descriptive analyses compared the demographics of the sample to the target population, using means (*M*) and standard deviation (*SD*) for continuous variables and frequencies and percentages for categorical variables.

Missing data was determined based on the percentage of complete cases and percentage of missing cells. Approximately 96% ($n = 482$) of the data were completed cases, and 3.6% ($n = 18$) were missing data for level of education due to not being documented in the charts. No changes or imputations were made.

To address question 1, the data was analyzed using GBTM with STATA software, version 9.3 (StataCorp, 2015) and the PROC TRAJ macro (http://www.andrew.cmu.edu/user/bjones), a closed-source module developed specifically for use with STATA software. The purpose of GBTM is to group individuals who follow similar growth trends and identify differences in the causes and consequences of different trajectories (Nagin & Odgers, 2010). Thus, GBTM is suitable for research questions that pertain to understanding group differences in trajectories. This method of finite mixture modeling assumes that the population is composed of a mixture of distinct groups defined by trajectories (Nagin & Odgers, 2010). Unlike hierarchical and latent curve modeling (two main approaches for analyzing developmental trajectories) which provides modeling of a single average trajectory, GBTM identifies relatively homogenous clusters of trajectories (Loughran & Nagin, 2006). Another advantage of GBTM is that models are estimated using maximum likelihood estimation which has two important properties. The first property is that the maximum likelihood-based parameter estimates are asymptotically unbiased, and implies that such estimates accurately measure population parameters that they are intended to

estimate (Kiefer & Wolfowitz, 1959). The second property is that the maximum likelihood-based parameter estimates are asymptotically normally distributed, and establishes theoretical basis for using statistical tests of parameter estimates (Kiefer & Wolfowitz, 1959).

For this study, patterns of change in body weight were based on the identification of relatively homogenous clusters of individuals who followed similar trends of change in body weight, and derived from daily body weights from admission to discharge. Initially, determination of the maximum number of clusters was based on visual inspection of trajectories from the pilot data. Next, Bayesian Information Criterion (BIC) was used with a stepwise approach to increase the number of groups in the model specification to the identified maximum solutions. Shapes for the trajectories (e.g., cubic, linear) and a censored normal probability distribution were compared to identify the number of groups that best characterized the data. The BIC approach selects the number of groups that best represent the heterogeneity among the trajectories, and corrects for the complexity of the models (i.e., quadratic trend; Nagin & Odgers, 2010). Smaller BICs indicate a better fit but theory and parsimony must also be considered in model selection (Nagin & Odgers, 2010).

To address question 2, the extensions of GBTM was used to include time-stable covariates. Time-stable covariates function as risk factors and are modeled into the likelihood of being in a particular trajectory using the logistic link functions. GBTM estimates a set of parameters that maximizes the probability of an individual belonging in a specific trajectory by assuming conditional independence which specifies the likelihood function as a mixing distribution at the level of group and not individual (Nagin, 2014). In other words, this statistical method identifies “causes and consequences” of different trajectories, and allows for the capacity to distinguish between chance and actual variation across individuals (Nagin & Odgers, 2010).

Sample Size

The target sample size of 500 was determined based on an article indicating that the maximum likelihood estimates obtained in GBTM provide close estimate of the true population values and have normal distribution with a minimum of this sample size (Loughran & Nagin, 2006). As an exploratory and descriptive study, a two-tailed test was used to test statistical significance of probability in both directions.

Chapter Four

Results

This chapter describes the results of the data analyses. To review, the following questions were posed: (1) To what extent do patterns of change in body weight exist among individuals during IPT for AN? and (2) What is the relationship between factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge), and patterns of change in body weight among individuals during IPT for AN?

First, demographic information on the sample will be reviewed. This section will include personal and clinical factors. Next, results will be discussed based on research questions followed by a summary of the findings.

Demographic Characteristics of the Sample

Personal Characteristics

The final sample (N = 500) was predominantly Caucasian (91.4%) and female (90.4%). Only 1.6% of the sample identified as Black, 2.4% Asian, 2.6% Hispanic, 1.2% Bi/ Multiracial, and 0.6% "Other" (i.e., Egyptian, Indian, Lebanese). The mean age was 26.8 years (SD = 13.9) with a range of 11–69 years old. Approximately 32% of the sample were children/adolescent (< 18 years old), 18.8% young adults (≥ 18 and ≤ 21 years old), and 48.8% older adults (> 21 years old). All children, adolescents, and young adults reported never being married. About 12% of all adults (≥ 18 years old) had completed graduate school, 61.9% completed some or all of college, and 26.3% completed some or all of high school. Additional sample characteristics are presented in Table 9.

Table 9.

Demographic Characteristics of the Sample

Characteristic	*M* (*SD*)	Range
Age (in years)	26.8 (13.9)	11–69
	n	%
Gender		
Female	452	90.4
Male	48	9.6
Race/ethnicity		
White	458	91.6
Black	8	1.6
Asian	12	2.4
Hispanic	13	2.6
Bi/Multiracial	6	1.2
Other	3	0.6
Education (*n* = 482)*		
≤ 9th grade	61	12.6
> 9th grade & ≤ 12th grade	183	37.9
> 12th grade & ≤ 16 years	201	41.7
> 16 years	37	7.7
Marital status		
Never married	401	80.2
Married	68	13.6
Separated	23	4.6
Divorced	8	1.6
Living situation		
Alone	63	12.6
With significant other	38	7.6
With roommate(s)/friend(s)	36	7.2
With family	351	70.2
Other (e.g., foster care)	12	2.4

Note: *Sample sizes were smaller than N = 500 due to missing data.

Clinical Characteristics

The median duration of illness was 3 years, 46.6% of the sample (n = 234) had at least one prior inpatient treatment for AN, 33.2% (n = 166) were readmissions to this specific unit, and 70.4% (n = 352) of the sample were restricting subtype of AN. Regarding psychiatric comorbidities at time of admission, 43.6% (n = 218) of the sample were diagnosed with one psychiatric comorbidity, 25% (n = 125) diagnosed with two, and 6.0% (n = 30) diagnosed with three or more. 69.4% (n = 347) of individuals were prescribed at least one psychotropic medication at time of admission, and 28.2% (n = 141) were prescribed an atypical anti-psychotic (e.g., olanzapine, quetiapine, risperidone).

There were statistically significant differences between males and females in that males were less likely to have had prior IPT for AN, $x^2(1, 500) = 6.63$, $p = .01$, and less likely to have psychotropic medication prescribed at time of admission, $x^2(1, 5000 = 7.50$, $p = .006$, compared to females. A difference in readmission to unit was trending towards significance with males being less likely to have had previous admission to the unit, $x^2(1, 500) = 3.66$, $p = .06$.

At time of admission, the mean body weight was 42.6 kg (SD = 6.2) and the average BMI was 15.9 kg/m^2 (SD = 1.5). The average admission caloric intake was 1911 kcal (SD = 401). At time of discharge, the average change in body weight was 2.5 kg (SD = 2.3), and the mean change in BMI was 1.0 kg/m^2 (SD = 0.9). The average length of stay was 16 days (SD = 12.6). Discharge disposition included: residential (27%), partial hospitalization (16.8%), intensive outpatient treatment (6.8%), home to outpatient team (18.6), lower level of care at different organization (4.8%), home (12.6%), medical hospital transfer (3.6%), and against medical advice (7.4%). Details about clinical characteristics are provided in Table 10.

Table 10.

Statistics for Clinical Characteristics of the Sample

Characteristic	*n*	Percent
Subtype of AN		
Binge/purge	148	29.6
Restricting	352	70.4
	M (*SD*)	Range
Change in body weight (kg)	2.5 (2.3)	–4.3–14.5
Change in BMI (kg/m^2)	1.0 (0.9)	–1.5–5.7
Admission caloric intake (kcal)	1911 (401)	1275–3815
Length of stay (in days)	16.0 (12.6)	2–106
Duration of illness (in years)	8.8 (11.1)	0–50
Admission		
Body weight (kg)	42.6 (6.3)	24.3–63.3
BMI (kg/m^2)	15.9 (1.5)	10.1–18.5
Percent of IBW	77.0 (7.8)	48.1–96.2
Discharge		
Body weight (kg)	45.1 (6.3)	25.3–70.2
BMI at discharge (kg/m^2)	16.9 (1.5)	11.8–22.8
Percent of IBW	81.6 (7.8)	56.3–108.2
	n	%
Disposition		
Residential	135	27.0
Partial Hospitalization	84	16.8
Intensive Outpatient	34	6.8
Lower level of care at different organization	24	4.8
Home to outpatient team	93	18.6
Home	63	12.6
Against medical advice	37	7.4
Transfer to medical facility	18	3.6
Other	12	2.4

Note: AN = anorexia nervosa; BMI = body mass index; IBW = ideal body weight; kcal = calorie; kg = kilogram; m = meter; SD = standard deviation

Patterns of Change in Body Weight (Question 1)

Based on visual inspection of trajectories from the pilot data, the maximum number of clusters was five. Next, BIC was used with a stepwise approach to increase the number of groups in the model specification 3-, 4-, 5-, and 6-group solutions. BIC values and predicted group proportions for solutions are shown in Table 11. Based on a combination of the BIC

(the value closest to 0 indicates the best-fitting model), fit statistics, and the size of the groups, the 4-group solution was selected as the final model.

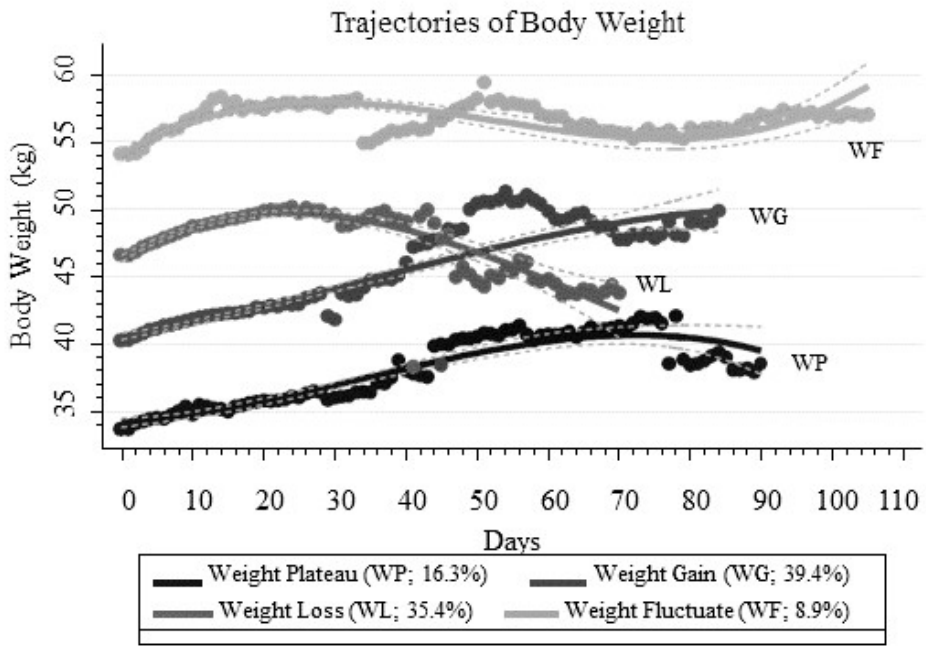
Table 11.

Bayesian Information Criterion (BIC) Values, Predicted Group Proportions for Solutions

# of groups	BIC	Group 1	Group 2	Group 3	Group 4	Group 5	Group 6
3	–22193.55	.21	.50	.29			
4	**–20730.33**	**.16**	**.39**	**.35**	**.09**		
5	–19600.91	.10	.27	.34	.24	.05	
6	–22017.02	.17	.17	.17	.17	.17	.17

Model selection. The final group-based trajectory model for body weight (kg) over time (days) was the 4-group solution, and included weight gain (WG; *n* = 197; 39.4%), weight loss (WL; *n* = 177; 35.4%), weight plateau (WP; *n* = 82; 16.4%), and weight fluctuate (WF; n = 44; 8.8%) groups. Figure 3 provides the trajectories for the 4 groups. The solid lines in Figure 3 are based on the parameter estimates of the model, and the dashed lines form the 95% CIs on the estimated probabilities of group membership. The dots are calculated with the actual data in which individual's responses are weighted based on posterior probabilities of group membership. The confidence intervals on the graph have minimal overlap indicating that the model captures distinctive features of the population distribution of trajectories.

Figure 3. Trajectories of Body Weight Groups



For the WG group, the average body weight and BMI at time of admission were 40.2 kg and 15.5 kg/m^2. At time of discharge, mean body weight was 42.8 kg and mean BMI was 16.5 kg/m^2. The average rate of change in body weight was 1.26 kg per week, with an average total gain of 2.6 kg. Based on the trajectory models, individuals assigned to the WG group were more likely to have a constant increase in body weight over time (see Figure 3).

The WL group was the second largest group of individuals. At time of admission, the average body weight was 46.5 kg and the mean BMI was 16.9 kg/m^2. Mean body weight and BMI at time of discharge were 48.9 kg and 17.8 kg/m^2. The average rate of change in body weight was 1.2 kg per week, with a total gain of 2.3 kg. The group-based trajectory model showed that individuals assigned to this group were more likely to have had an initial increase in body weight, followed by a decrease. Additionally, individuals were more likely

to have a lower body weight at time of discharge compared to body weight at time of admission (see Figure 3).

Individuals assigned to the WP group were more likely to have an initial increase in body weight and then eventually plateau (see Figure 3). In the WP group, the average body weight at time of admission was 33.6 kg and the mean BMI at time of admission was 14.2 kg/m^2. At time of discharge, the mean BMI and body weight were 15.4 kg/m^2 and 36.4 kg. The WP group had an average rate of change in body weight of 1.3 kg per week, and the mean total change was 2.8 kg.

The WF group had an average BMI and body weight at time of admission of 17.4 kg/m^2 and 54.2 kg. At time of discharge, the mean BMI and body weight were 18.2 kg/m^2 and 56.8 kg. The average rate of change in body weight was 1.3 kg per week, and the mean total weight gain was 2.6 kg. Individuals assigned to the WF group were more likely to have an initial increase in body weight followed by periods of fluctuation (see Figure 3).

Differences in characteristics. There were statistically significant group differences in demographic and clinical characteristics. A chi square test of independence revealed that males were significantly more likely to be assigned to WF group compared to any other groups, $\chi^2(3, n = 500) = 85.34, p < .001$. Individuals assigned to the WF group were significantly less likely to be readmissions to the unit compared to other groups, $\chi^2(3, n = 500) = 7.87, p < .05$. As determined by one-way ANOVA, there were statistically significant differences between groups in age, $F(3, 496) = 3.71, p = .01$, gender, $F(3, 496) = 34.03, p < .001$, readmission to unit, $F(3, 496) = 2.64, p = .05$, duration of illness, $F(3, 373) = 5.25, p = .002$, admission caloric intake, $F(3, 496) = 5.98, p = .001$, body weight at time of admission, $F(3, 496) = 883.98, p < .001$, body weight at time of discharge, $F(3, 496) = 707.67, p < .001$, and length of stay, $F(3, 496) = 3.47, p = .02$. Means, standard deviations, and percentages are presented in Table 12.

Post hoc comparisons using Tukey HSD revealed that individuals assigned to the WF group were significantly younger compared to individuals assigned to the WG (–7.12 ± 2.30, $p = .01$), WL (–6.17 ± 2.32, $p = .04$), and the WP (–7.97 ± 2.58, $p = .01$) groups. Admission caloric intake was significantly lower in the WP group compared to the WL (–184.71 ± 52.76, $p = .003$) and WF (–240.65 ± 73.81, $p = .007$) groups. Duration of illness was significantly lower in the WF compared to the WG (–6.24 ± 1.98, $p = .01$), WL (–5.22 ± 2 $p = .05$), and WP (–8.93 ± 2.31, $p = .001$) groups.

Regarding body weight at time of admission, the WP group had a significantly lower body weight compared to the WG (–6.62 ± .33, $p < .001$), WL (–12.93 ± .33, $p < .001$), and the WF (–20.61 ± .46, $p < .001$) groups. The WG group had a significantly lower body weight compared to the WL (–6.31 ± .26, $p < .001$) and WF (–13.98 ± .41, $p < .001$) groups. The WL group had a significantly lower body weight at time of admission compared to the WF group (–7.67 ± .42, $p < .001$). At time of discharge, body weight was significantly lower in the WP group compared to the WG (–6.40 ± .36, $p < .001$), WL (–12.46 ± .36, $p < .001$), and WF (–20.42 ± .51, $p < .001$) groups. The WG group had a significantly lower body weight at time of discharge compared to the WL (–6.06 ± .28, $p < .001$) and WF (–14.02 ± .45, $p < .001$) groups. The WL group also had significantly lower body weight at time of discharge compared to the WF group (–7.96 ± .46, $p < .001$). Length of stay was significantly lower in the WL group compared to the WP group (–5.15 ± 1.67, $p = .01$). Although there were statistically significant group differences in body weight at time of admission and discharge, there were not group differences in weekly change in body weight and BMI, and overall change in body weight.

Table 12.

Summary of Percentages, Means, and Standard Deviations for Demographic and Clinical Characteristics by Trajectory Groups

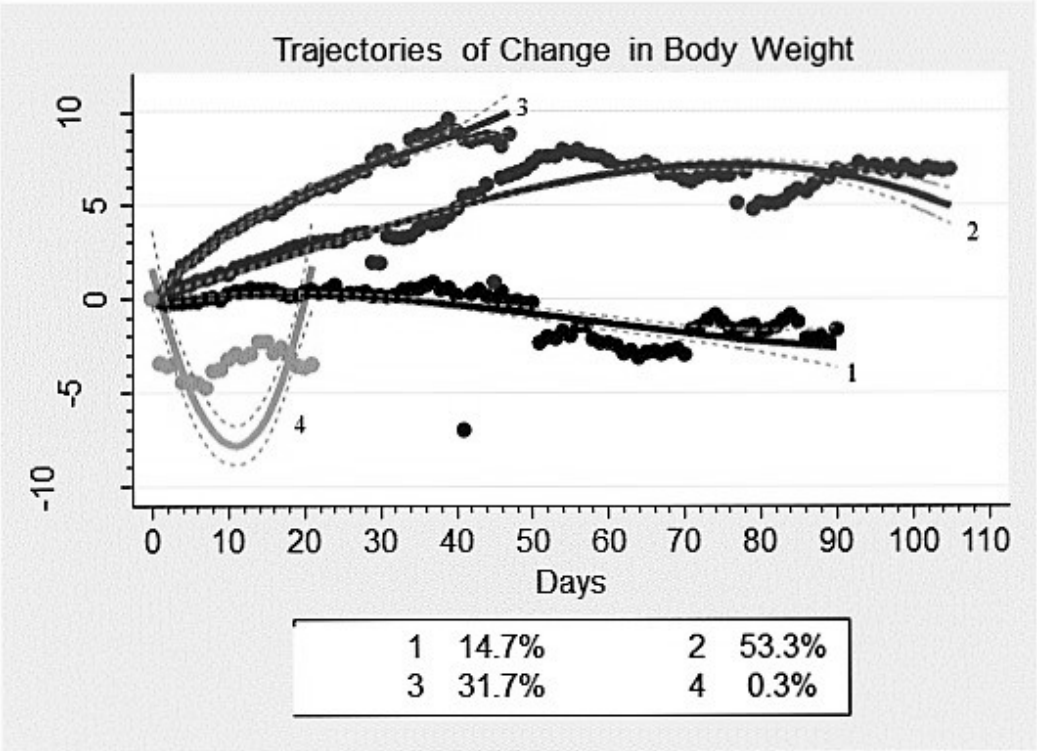
Characteristic	Weight Gain (*n* = 197)	Weight Loss (*n* = 177)	Weight Plateau (*n* = 82)	Weight Fluctuate (*n* = 44)
		n (%)		
Gender				
Female	190 (96.4)	160 (90.4)	79 (96.3)	21 (47.7)
Male	7 (3.6)	17 (9.6)	3 (3.7)	23 (52.3)
Subtype of AN				
Binge/purge	58 (29.4)	58 (32.8)	18 (22.0)	14 (31.8)
Restricting	139 (70.6)	119 (67.2)	64 (78.0)	30 (68.2)
		M (SD)		
History of prior inpatient treatment for AN	98 (49.7)	75 (42.4)	44 (53.7)	17 (38.6)
Readmission to Unit	73 (37.1)	56 (31.6)	30 (36.6)	7 (15.9)
Age (in years)	27.6 (14.2)	26.7 (14.0)	28.4 (14.8)	20.5 (7.8)
Admission				
Body weight (kg)	40.2 (2.4)	46.5 (2.2)	33.6 (2.7)	54.2 (3.6)
BMI (kg/m^2)	15.5 (1.2)	16.9 (1.0)	14.2 (1.1)	17.4 (.8)
Percent of IBW	74.7 (6.3)	81.4 (5.7)	69.1 (6.8)	83.7 (5.5)
Discharge				
Body weight (kg)	42.8 (2.3)	48.9 (2.5)	36.4 (3.1)	56.8 (4.3)
BMI at discharge (kg/m^2)	16.5 (1.2)	17.8 (1.2)	15.4 (1.4)	18.2 (1.1)
Percent of IBW	79.5 (6.3)	85.6 (6.6)	74.8 (7.3)	87.8 (6.5)
Admission caloric intake (kcal)	1877 (373)	1976 (441)	1791 (334)	2032 (398)
Length of stay (days)	16.3 (12.3)	13.8 (9.7)	19.0 (14.6)	17.1 (18.3)
Average rate of change in body weight (kg/week)	1.3 (1.2)	1.2 (1.0)	1.3 (1.1)	1.3 (1.2)
Average rate of change in BMI ($[kg/m^2]$/week)	0.5 (0.5)	0.5 (0.4)	0.5 (0.4)	0.4 (0.4)
Change in body weight (kg)	2.6 (2.2)	2.3 (2.2)	2.8 (2.6)	2.6 (2.2)
Change in BMI (kg/m^2)	1.0 (0.9)	0.9 (0.8)	1.2 (1.1)	0.8 (0.7)

Note: AN = anorexia nervosa BMI = body mass index; IBW = ideal body weight; kcal = calories; kg = kilogram; m = meter

Body weight at time of admission. Figure 3 indicates that body weight at admission influences trajectory group membership. Thus, a group-based trajectory model for change in body weight over time (i.e., body weight at admission was subtracted from all weights) was

tested. Figure 4 provides the trajectories, and three of the four trajectories (i.e., WG, WL, WP) were similar to the original model.

Figure 4. Trajectories of Change in Body Weight Groups

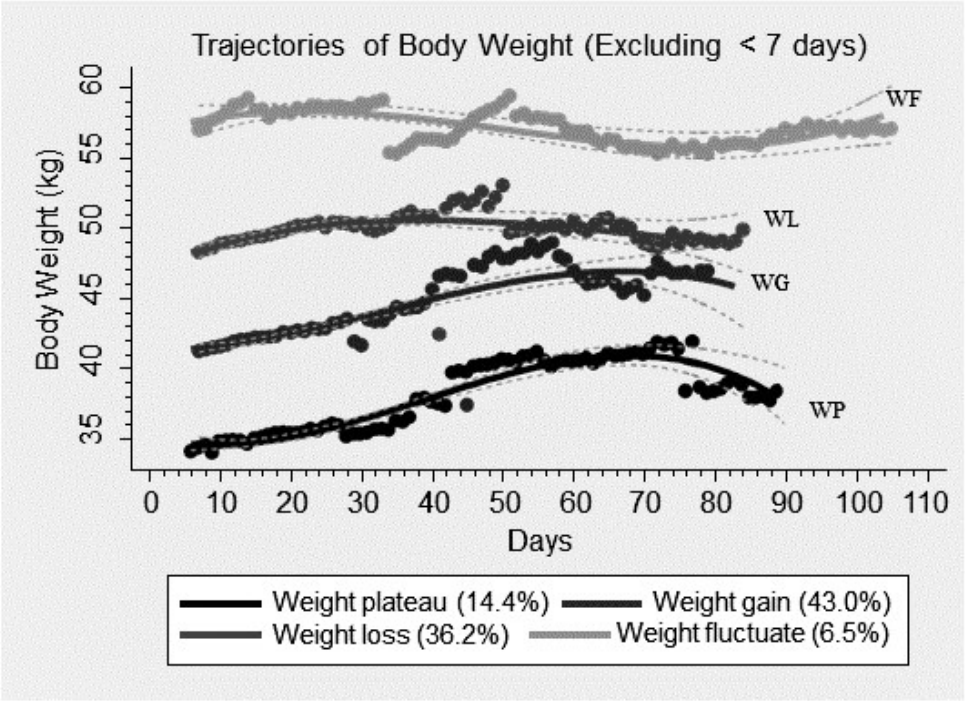


The fourth group (WF) was different from the original model, but the proportion of individuals assigned to the WF group in the new model was less than the original model (n = 1 versus n = 44). Thus, the new group model was essentially a 3-group solution. Based on these results, the original 4-group solution for body weight over time remained the final model because it captures more distinctive features of the population distribution of trajectories

Length of stay. One-way ANOVA was conducted to determine if length of stay was different based on discharge dispositions. There was a statistically significant difference, $F(8, 491) = 4.31$, $p < .001$. A Bonferroni post-hoc test revealed that length of stay was statistically significantly lower for individuals who dropped out of treatment (7.6 ± 6.5) compared to

those discharged to residential treatment (16.9 ± 15.0, $p < .005$), partial hospitalization (17.8 ± 11.2, $p < .001$), lower level of care at different facility (18.4 ± 9.8 $p < .05$), home to outpatient team (16.7 ± 10.8, $p < .01$), and other disposition (27.2 ± 27.3, $p < .001$). The 4-group model was tested with and without individuals who discharged within 7 days of admission. Figure 5 shows that the removal of this subsample ($n = 83$) changed the significance of the slopes.

Figure 5. Trajectories of Body Weight Groups (excludes LOS <7 days)



Based on this result, the initial 4-group model remained the final model because it appears to capture the influence of discharge disposition on trajectory groups.

Predictors of Patterns of Change in Body Weight (Question 2)

To examine research question 2, GBTM specifies the relationship of time-stable covariates to the probability of group membership as a multinomial logit model. Factors that researchers have identified as predictors of weight gain were included, such as age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at

time of discharge, and BMI at time of discharge. Individual, psychiatric, and eating disorder factors were also analyzed in respect to the trajectory group membership. Since the WG trajectory was the largest group and the "ideal" trajectory of change in body weight, it was used as the reference group. Table 13 provides the coefficient estimates and confidence intervals.

Table 13.

Predictors of Change in Body Weight Trajectory Group Membership: Logit Estimates[a]

Characteristics	Weight Plateau[b]		Weight Loss[b]		Weight Fluctuate[b]	
	Coefficient Estimate	*SE*	Coefficient Estimate	*SE*	*Coefficient Estimate*	*SE*
Age	–0.02	0.02	0.04	0.02 □	–0.03	0.11
History of prior IPT for AN	–0.40	1.03	–2.41	1.27 □	–1.02	2.24
Admission caloric intake	0.08	8.59	0.01	0.00**	–0.01	10.34
Body weight at time of admission	–1.82	0.30***	2.99	0.55***	9.42	2.59***
Body weight at time of discharge	–1.94	0.35***	5.20	1.47***	6.99	1.52***
Length of stay	0.15	0.04***	–0.33	0.09***	0.42	0.10***

NOTE: AN = anorexia nervosa; IPT = inpatient treatment; □ = approaching significance.

[a]Trajectory Group 1 (Weight Gain) is the reference group.

[b]The coefficient estimates correspond to the parameters of a multinomial logit function.

*$p < .05$. **$p < .01$. ***$p < .001$.

Estimates in Table 13 suggest that a longer length of stay, 95% CI [–0.24, 0.15], and higher body weight at time of admission, 95% CI [19.32, 29.47], and discharge, 95% CI [7.65, 13.60], were risk factors associated with being assigned to the WF groups. Risk factors associated with being assigned to the WL group included being older, 95% CI [–0.04, 0.04], having no history of prior IPT for AN, 95% CI [–5.55, –0.57], having a higher admission caloric intake, 95% CI [–0.0002, 0.0002], a shorter length of stay, 95% CI [–0.21, –0.15], and a higher body weight at time of admission, 95% CI [0.57, 2.72] and discharge, 95% CI [4.76, 10.53]. A longer length of stay, 95% CI [–0.07, 0.08] and lower body weight at admission, 95% CI [–1.13, 0.04] and discharge, 95% CI [–1.37, 0.01] were associated with being assigned to the WP group.

Summary

The findings of this study sought to answer two questions: (1) To what extent do patterns of change in body weight exist among individuals during IPT for AN? and (2) What is the relationship between factors identified in the existing literature as being predictors of weight gain and patterns of change in body weight among individuals during IPT for AN?

For question 1, four trajectories, or patterns, of change in body weight emerged from the data. The groups were described as weight gain (WG), weight loss (WL), weight plateau (WP), and weight fluctuate (WF). Individuals assigned to the WG group were more likely to have a constant increase in body weight, whereas individuals assigned to the WL group were more likely to have an initial increase in body weight followed by a decrease in body. Individuals assigned to the WP group were more likely to have an increase in body weight and then plateau with a slight decrease in body weight prior to discharge. Finally, individuals assigned to the WF group were more likely to have an initial increase in body weight followed by periods of fluctuation.

For question 2, the statistically significant predictors of the patterns of change in body weight were (a) age, (b) history of prior IPT for AN, (c) admission caloric intake, (d) body weight at time of admission, (e) body weight at time of discharge, and (f) length of stay. Individuals assigned to the WL group on average (1) were older, (2) had no history of prior IPT for AN, (3) had a higher starting caloric diet, (4) had higher body weight at time of admission and time of discharge, and (5) had a shorter length of stay. Individuals assigned to the WP group on average had a longer length of stay as well as a lower body weight at time of admission and discharge. Individuals assigned to the WF group on average had higher body weight at time of admission and discharge, and had a longer length of stay.

Chapter Five

Conclusion

To this researcher's knowledge, this was the first study to examine patterns of change in body weight among a relatively large sample ($N = 500$) of individuals who were admitted to IPT for AN, and examine the relationship between factors identified in the existing literature as being predictors of weight gain and patterns of change in body weight. This study provides unique data because individuals appear only once in the dataset (omitted repeat admissions) and all participants were included in the analyses regardless of length of stay or treatment failure. Additionally, sophisticated data analyses were used (i.e., GBTM, nonlinear). Previous studies have indicated that weight restoration during IPT may be a promising clinical indicator of treatment outcomes (Kaplan et al., 2009; Lock & Litt, 2003; Lund et al., 2009; Zipfel et al., 2000). More recently, researchers have examined the nonlinearity of weight changes during IPT for AN using statistical analyses that more appropriately fit the data, and identified predictors of weight gain (i.e., age at time of admission; admission caloric intake; percent of IBW at time of admission; body weight at time of discharge; BMI at time of discharge; Hartmann et al., 2007; Lay et al., 2002; Mewes et al., 2008). Unfortunately, generalizability of results is limited because these previous studies had small sample sizes ($N = 40–100$), excluded admissions less than 6 weeks, and/or excluded data on early dropouts and early treatment failures.

The purpose of this study was to explore the extent to which patterns of change in body weight exist among individuals during IPT for AN, and the relationship with factors identified in the existing literature as being predictors of weight gain (i.e., age at time of admission, admission caloric intake, percent of IBW at time of admission, body weight at time of discharge, BMI at time of discharge). Based on the results of this study, four patterns (trajectories) of change in body weight emerged from the data: weight gain, weight loss,

weight plateau, and weight fluctuate. The predictors of the four patterns of change in body weight were: (a) age, (b) history of prior IPT for AN, (c) admission caloric intake, (d) body weight at time of admission, (e) body weight at time of discharge, and (f) length of stay.

In this chapter, the results will be discussed. Then, limitations will be addressed followed by implications for practice, education, research, and policy.

Demographics

Similar to prevalence rates among U.S. adults and adolescents (Alegria et al., 2007; Hudson et al., 2007; Nicdao et al., 2007; Swanson et al., 2011; Taylor et al., 2007), the current study sample was primarily non-Hispanic white and female. The majority of participants were never married and living with family. In regards to clinical presentation, most participants were diagnosed with the restricting subtype of AN, and had at least one psychiatric comorbidity diagnosed at time of admission. These findings are similar to studies that reported prevalence rates of at least one psychiatric comorbid diagnoses among individuals with AN (Blinder et al., 2006; Swanson et al., 2011). Research has suggested that psychiatric comorbidities are commonplace among individuals with AN, and is associated with the severity and duration of the eating disorder (Brand-Gothelf et al., 2014; Swanson et al., 2011).

There were statistically significant differences between males and females in clinical characteristics. Specifically, males were less likely to have had prior IPT, readmission to unit, psychotropic medications at time of admission compared to females. Although research suggests that there is not a difference in core eating disorder symptoms between sexes (Raevuori, Keshi-Rahkonen, & Hoek, 2014), males are less likely to recognize eating disorder symptoms and even less likely to seek treatment for an eating disorder (Merikangas et al., 2011; Räisänen & Hunt, 2014). Results from the current study are consistent with the existing research. It is possible that males do not associate their symptoms with an eating

disorder; and are less likely to seek treatment for AN because they experience an additional stigma of having a psychiatric disorder that has been commonly perceived as a female-specific disorder.

Patterns of Change in Body Weight (Question 1)

The data indicated that the final group-based trajectory model for body weight over time was the 4-group solution, including: weight gain, weight loss, weight plateau, and weight fluctuate. Findings from this study are similar to other studies that have found variation in changes of weight during IPT for AN. Researchers have suggested that some individuals with AN will initially lose weight followed by significant weight gain and/or weight fluctuations for the remainder of the inpatient admission (Garber et al., 2012); and a smaller subset of patients will lose weight or have minimal weight gain during IPT for AN (Garber et al., 2012; Hart et al., 2011).

The WG group was the largest subset of the sample (n = 197; 39.4%), and had the second to lowest body weight and BMI at time of admission. Thus, individuals assigned to this group were expected to gain more weight compared to two of the three other groups (WL and WF). Since individuals assigned to the WG group had a faster rate of change in BMI per week compared to the other groups, it is possible that individuals in this group had a higher level of self-esteem and/or lower levels of perfectionism at time of admission. Studies have indicated that self-esteem and perfectionism predict time to reach 85% of IBW (Phillips et al., 2010; Vansteelandt, Pieters, Vanderlinden, & Probst, 2010). Individuals with higher self-esteem may derive it from other sources and not primarily from body weight and their eating disorder. Unlike individuals with lower self-esteem and higher levels of perfectionism, this group may have fewer difficulties with consuming calories because their sense of control is not dependent on stringent standards and rules surrounding body weight and body image.

Thus, they may gain weight at a faster rate compared to others, even though they may have a lower body weight at time of admission.

Surprisingly, the WL group was the second largest subset of the sample, and individuals assigned to this group were more likely to display an initial increase in body weight, followed by a decrease that may end with a lower body weight at time of discharge compared to time at admission. It is possible that individuals assigned to the WL group who experienced this trajectory of change in body weight, did not have the necessary caloric requirements to maximize weight gain during IPT for AN. According to Mehler and colleagues (2010), individuals with AN tend to have lower resting energy expenditure at time of admission compared to later in the admission. It is commonplace for individuals with AN to require very high levels of caloric intake for abbreviated period of time to promote weight gain (Mehler et al., 2010).

Another explanation for the unique trajectory is that individuals assigned to the WL group struggled with greater feelings of shame (negative views of oneself and the negative view one imagines others hold about one-self) compared the other groups. Researchers have suggested that individuals with AN with higher levels of global self-criticism have more severe eating disorder pathology due to elevated global shame (not specific to eating disorders like food and body; Gilbert, 2009; Kelly & Carter, 2013). Individuals assigned to the WL group may have been more critical of oneself which triggered and intensified feelings of shame leading to behaviors focused on altering weight and shape, or leaving treatment prematurely. Upon admission to IPT for AN, individuals in this group may have had an initial increase in pride and decrease in shame, but it was temporary. As feelings of shame returned, individuals may have sought relief by engaging in eating disorder behaviors to manage feelings of shame, or discharging earlier than recommended. Hence individuals assigned to this group were likely to display a decrease in body weight after a period of weight gain.

Individuals assigned to the WP group displayed a constant increase in body weight and then plateaued followed by a slight decrease. This group had the lowest body weight and BMI at time of admission as well as the lowest admission caloric intake. Individuals assigned to this group also had the most change in body weight. The WP group followed the most common weight curve for individuals with AN during IPT in that individuals displayed a rapid weight gain at the beginning and then leveled off near the end of treatment (Davies & Jaffa, 2005; Lay et al., 2002; Vanstelandt et al., 2010). The WP group had a BMI at time of admission was 14.2 kg/m^2 and BMI at time of discharge was 15.4 kg/m^2. Since this group had a lower BMI and body weight at time of admission,, it is possible that they experienced fluid loss during refeeding leading to weight loss compared to other groups that had higher BMIs at time of admission and discharge. Fluid loss during refeeding in AN, results from an expanded extracellular water compartment and tends to occur at BMI 15–16 threshold (Rigaud, Boulier, Tallonneau, Brindisi, & Rozen, 2010), which is similar to the average BMI at time of admission for the WP group.

The WF displayed fluctuations of body weight over the length of stay. This group had the highest admitting body weight, and individuals assigned to this group were more likely to have a longer length of stay compared to others. It is possible that this group struggled with emotion regulation, and as they gained weight, they had to confront negative emotions and use alternative coping techniques (Phillips et al., 2010). However, they had a difficult time with identifying and struggling with negative emotions, and resorted to eating disorder behaviors to alleviate the feelings. The weight fluctuations during treatment were indicative of their struggles with emotion regulation and using different coping strategies besides eating disorder behaviors.

Predictors of Patterns of Change in Body Weight (Question 2)

Age and Prior Inpatient Treatment.

Compared to the WG group, individuals assigned to the WL group were more likely to be older and less likely to have had prior IPT for AN. Previous research has suggested that past IPT for AN was associated with severity of eating disorder symptomatology (Milos et al., 2004), but a younger age at admission predicted a greater rate of weight gain (Neuberger et al., 1995). Similar to prior studies, individuals in the WG group may have had more severe eating disorder symptomatology and hence lower body weight at time of admission. However, since they tended to be a younger age at time of admission, they may have greater changes in body weight.

Admission Caloric Intake

According to Mehler and colleagues (2010), individuals with AN who are in a state of starvation are metabolically inefficient and may require more than 3,500 kcal beyond maintenance caloric needs to restore a pound of weight (Mehler et al., 2010). The needed caloric requirement to cause weight gain can vary between 1,800–4,500 kcal per day, and males tend to peak at 4,000 kcal per day and females at 3,500 kcal per day (Mehler et al., 2010). Optimal dietary interventions for promoting weight gain during IPT for AN are unknown, but avoiding refeeding syndrome is desired. Individuals assigned to the WL group were more likely to have a higher admission caloric intake compared to the WG group. It is possible that clinicians were attempting to maximize weight gain for individuals assigned to the WL group based on clinical assessment (i.e., decreased likelihood of prior inpatient treatment). Since individuals assigned to the WL group tended to have a higher body weight at time of admission and no prior IPT for AN, clinicians may have been slightly less concerned with refeeding syndrome and initiated a higher admission caloric intake.

Body Weight at Time of Admission

Although the existing literature has not identified body weight at time of admission as a predictor of weight gain, this factor was statistically significant in this study. Results

suggest individuals assigned to the WG group were more likely to have a higher body weight at time of admission compared to the WP group. The WG and WP had lower body weight at time of admission compared to the WF or WL groups. Research has suggested that low body weight at time of admission was associated with treatment outcome (Herzog et al., 2004), and higher BMI at time of admission predicts positive outcomes (Zipfel et al., 2000). Sly and Bamford (2011) argue that individuals with severe eating disorders are frequently admitted to lower levels of care (e.g., residential, partial hospitalization) to reduce healthcare costs, and more recently IPT has been postponed until individuals have lost a substantial amount of weight and are medically unstable. Findings from their study indicate that patients admitted at a lower weight were subsequently discharged at a lower weight, and those who admitted with BMI less than 15 kg/m^2 were more likely to be readmitted within a year (Sly & Bamford, 2011). In the current study, individuals assigned to the WG group were more likely to have had prior inpatient treatment, and thus it is possible that this group struggled with low body weight for a longer period of time, and had an admission within the past year in which they were discharged at a lower body weight and are now re-presenting for IPT for AN. Whereas individuals in the WL were more likely to be presenting for their first inpatient admission at a higher body weight at time of admission and possibly even less severity of eating disorder symptomatology.

Body Weight at Time of Discharge

Research has suggested that inadequate weight gain (Zipfel et al., 2000), and low weight at time of discharge (Baran et al., 1995) predicted poor outcomes (e.g., re-hospitalization, psychiatric status rating score). Other studies have reported that individuals who had a higher weight at time of discharge (Kaplan et al., 2009; Lock & Litt, 2003) or gained at least 0.8 kg per week (Lund et al., 2009) were less likely to have clinical deterioration compared to others.

Individuals assigned to the WG group were more likely to have a lower body weight at time of discharge compared to the WL group, and individuals assigned to the WP group were more likely to have a lower body weight at time of discharge compared to the WL and WF groups. One explanation is that individuals admitted to the WG and WP groups tended to have lower body weight at time of admission, and thus they were more likely to gain weight compared to the WL and WF groups. Sly and Bamford (2011) suggested that individuals who are admitted at a lower body weight are subsequently being discharged at a lower body weight.

Although the WL group had a higher body weight at time of admission compared to the WG and WP groups, individuals assigned to the WL group were more likely to lose weight and had the lowest total change in body weight over length of stay. These findings are similar to Hartmann and colleagues (2007) who examined predictors of failure to restore weight during IPT for AN, and used sophisticated statistical analyses (i.e., parameters of weight curves, nonlinear analyses). They found that higher BMI at intake was negatively correlated to weight restoration. Unfortunately, the sample selected was limited and only included first admissions, admission durations of at least 6 weeks, and BMI at time of admission less than 16 kg/m^2. However, in the current study, the WG and WP had mean BMIs at time of admission of less than 16 kg/m^2 and the mean BMI for the WL group was slightly above at 16.9 kg/m^2.

An explanation for individuals assigned to the WL group being more likely to lose weight and having the lowest total change in body weight over length of stay, is that they did not have the necessary caloric requirements to maximize weight gain during IPT for AN. Individuals assigned to this group were more likely to have a higher admission caloric intake, but it is possible that they were not monitored as closely as the WG and WP groups who had lower body weights at time of admission and lower admission caloric intakes. Thus,

individuals assigned to the WL group may not have had the appropriate dietary requirements to continue to gain weight, and hence experienced a decrease in body weight or minimal increase.

Individuals assigned to the WP group also may not have had the necessary dietary interventions to sustain weight gain, and hence displayed a plateau prior to discharge. According to Mehler and colleagues (2010), changes in resting metabolism, spontaneous activity, thermic effect of exercise, or thermic effect of food may contribute to difficulties with sustaining weight gain during the admission. Plateaus may be observed because of the change in the resting energy expenditure value and underestimation of caloric needs (Mehler et al., 2010). Weight loss during IPT is not catastrophic, but research suggests that it is important to limit the period of weight loss because a long period of weight loss may be a negative predictor of outcomes (Lay et al., 2002). Additionally, individuals who do not achieve and maintain weight stabilization during IPT for AN, are more likely to have readmissions and lose weight at a faster rate after discharge (Lay et al., 2002).

Length of Stay

Individuals assigned to the WG and WP groups were more likely to have longer length of stay compared to individuals assigned to the WL group. Findings from this study were similar to Mewes and colleagues (2008) in that the gaining group had lower body weight at time of admission and higher average weekly body weight gain in the first half of treatment. A key implication may be that slower weight gain may enable individuals with AN to acclimate to the physical changes and be more amenable to increasing weight. However, some researchers suggest that longer length of stay is a negative prognostic indicator (Keel & Brown, 2010). Sly and Bamford (2011) did not find any difference in length of stay based on BMI at time of admission. Similar to Sly and Bamford, results from the current study indicate that there may not be difference in total weight gain across length of stay, and individuals

who are admitted at a lower body weight are subsequently being discharged at a lower body weight.

Overall, research suggests a balance between the physiological and psychological changes during IPT for AN (Hart et al., 2011; Herzog et al., 2004). However, since numerous stakeholders are involved in determining length of stay (e.g., insurance companies, the treatment team, family members, and the patient), decisions regarding length of stay are complex and may not be dependent on body weight alone. It is also possible that individuals assigned to different trajectories have different metabolic states throughout treatment, which may impact weight gain and loss as well as body weight at time of discharge.

Limitations

There are several limitations of the study to consider. First, as a retrospective chart review, the research design lacked a rigorously standardized prospective real-time approach but was representative of real-world treatment. Second, diagnoses of AN were based on unstructured interviews conducted by a number of different clinicians. It is possible that the current study does not have a representative sample of males because clinicians were more likely to diagnose males as unspecified eating disorder versus AN. Research suggests that eating disorders have a highly skewed sex distribution, with the proportion of unspecified eating disorder cases being significantly higher among males compared to females, and most likely a reflection of under diagnosis and under treatment in males (Raevuori et al., 2014). Although the *DSM-5* provides a more sex-neutral diagnostic criteria for AN (e.g., eliminating amenorrhea criterion), clinician bias may still have contributed to the under-diagnosis of AN in males (Merikangas et al., 2011). Third, personal information was assessed retrospectively, and individuals may have minimized or denied symptoms due to embarrassment and other psychological barriers.

Another consideration is that the sample may not be fully representative of the target population. The setting was a proprietary health care company, and thus the treatment provided may not be representative of other treatment facilities with a different business or treatment model. Likewise, the homogeneity of the sample is a limitation. The majority of the participants were non-Hispanic, white females. Although the current study was similar to studies that examined prevalence rates of eating disorders (Hudson et al., 2007; Swanson et al., 2011), the sample was not diverse with respect to race and sex. Research suggests that males are less likely to be diagnosed and seek treatment for an eating disorder, and mental health service utilization is significantly lower among ethnic and racial minority groups compared to whites (Merikangas et al., 2011). Since the majority of studies recruit participants from treatment facilities, samples will inherently underrepresent certain subpopulations. Despite these limitations, this is the largest dataset to date to have looked at patterns of change in body weight among individuals during IPT for AN.

Implications for Nursing Practice

This study highlighted the heterogeneous trajectories of changes in body weight among individuals admitted to IPT for AN, and predictors of the course of weight restoration versus at a single time point. Having such knowledge, will allow nurses to be better informed in the decision making process during a client's admission. Nurses are an integral component of the interdisciplinary team, and as an advocate, educator, and coach, nurses are well-positioned to identify early in treatment subsets of the population who may be more susceptible to weight loss or minimal gain. Such knowledge will better inform the interdisciplinary team of potential risk factors for poor trajectory of weight restoration at the start of treatment, especially for individuals who are presenting for their first admission for IPT of AN.

With identifiable risk factors for unique patterns of change in body weight, nurses can develop new assessments and interventions that may lead to improvements in outcomes, quality of life and patient satisfaction. For example, nurses can develop and administer an assessment tool at time of admission that identifies individuals with AN who may be at-risk for weight loss or minimal gain. Additionally, a similar but potentially shorter assessment tool can be administered throughout the admission to measure any shift in risk based on specific changes of body weight over a period of time (e.g., consistent loss of body weight over the past three days) versus daily fluctuations. Nurses will then be able to inform treatment team of individuals with AN who have an increased risk for following a poor trajectory of change in body weight. Based on this information, nurses and the interdisciplinary treatment team will be able to modify treatment plans (e.g., increase caloric intake; focus on emotion dysregulation, self-concept and shame). Such patient-centered assessment and treatment will promote achievement of healthy weight prior to discharge and consequently reduce the risk of readmission, reduce long-term healthcare costs, and improve outcomes.

Implications for Research

A next step for this research topic will be to replicate this study in different treatment settings and more diverse population (e.g., gender, race, ethnicity, and sexuality). Replication of this study across different settings is necessary to capture the similarities and differences in treatment across the continuum of care (e.g., medical or general psychiatric unit, residential, partial hospitalization). Future studies will provide more knowledge about patterns of change in body weight, and help to identify individuals with AN who may be at a higher risk of not achieving weight restoration or dropping out of treatment prematurely. Additionally, the examination of time-varying covariates (e.g., motivation to change) as predictors of patterns of change in body weight is needed because motivation and desire to recover fluctuates over

time and may influence the process of weight restoration during IPT for AN. Such knowledge will help to define and develop more effective clinical assessments and interventions that are more individualized and based on assessment at time of admission as well as throughout the length of the admission.

Results from the present study suggest that there are opportunities for intervention studies to tailor interventions for optimizing weight restoration in the weight gain group and having crossover into the weight gain group. For example, a future randomized controlled study involving randomized assignment to different rates of change in caloric intake among individuals who have been identified as being at risk of following a trajectory with weight loss or minimal change in body weight. Hart and colleagues (2011) argue that best practice in the process of weight restoration involves the identification of patterns of change in body weight and is one of the most important aspect of IPT for AN. Further identification of differences between individuals during IPT for AN will help to develop assessments and interventions to alter trajectories towards the weight gain group and optimize weight restoration in the weight gain group. Such assessments and interventions would be tailored to the individual rather than the one-size-fits-all approach.

Another step for this research topic is to examine the phenomenon of weight restoration during IPT for the patient as well as the clinicians' experience. A qualitative study would provide insight about how the process of weight restoration is perceived by both the client and the provider, and also help to depict individual factors that may influence the promotion or prevention of health. Once this information is obtained, then nursing will be able to examine how interventions are framed and effective among individuals admitted to IPT for AN.

Implications for Education

Results from this study suggest that there is a need for tailored assessments and interventions for individuals during IPT for AN. The overall knowledge related to the process of weight restoration for AN is severely limited. This study provides a shift in the level of understanding of patterns of change in body weight which impact the process. If researchers are able to unravel the uniqueness of specific patterns of change in body weight, then there would be a better understanding of the process of weight restoration including factors contributing to the success and failure of this process. This study has the potential to better inform treatment guidelines, and provide insight to interdisciplinary teams regarding the importance of gathering pertinent information at time of admission and utilizing such information to inform assessment and treatment.

Educational efforts can capitalize on helping stakeholders (e.g., insurance companies, nurses, treatment teams, patients) become aware of the different patterns of change in body weight, and individual factors that may be potential risk factors for following a particular trajectory. Professional journals and conferences are significant ways to disseminate information as well as incorporating such knowledge into inter-professional arenas (e.g., medical, social work, nursing, and psychology).

Implications for Policy

A key theme of the National Institute of Nursing Research's (NINR) Strategic Plan is symptom science (NINR, 2011). Symptom science focuses on understanding chronic illness and improving quality of life, with one area of scientific focus being symptom clusters. Symptom clusters promotes treatment designed to target a group of symptoms versus individual symptoms, which may provide better outcomes (Grady & Gough, 2015; NINR, 2011). Eisenhauer (1994) argued that nursing should focus on human patterns and responses and life processes rather than a singular sign and/or symptom. Based on symptom science, current research has identified factors that influence young adults with sickle cell disease

seeking care for pain (Jenerette, Brewer, & Atage, 2014), and examined changes in cluster of five symptoms among patients with breast cancer over the course of chemotherapy and radiation treatment (Kim, McDermott, & Barsevick, 2014). Similarly, this study identified and compared clusters of individuals with different patterns of change in body weight during IPT for AN. Such knowledge will provide a better understanding of the mechanisms contributing to such unique patterns and the process of weight restoration, and can improve patient outcomes and guide healthcare providers in the development of more individualized interventions. Additionally, there is an opportunity to inform the national and global treatment guidelines and recommendations for treatment of AN, to include new knowledge about the best practice and how to meet the specific needs of this population and sub-groups.

Summary

The results of the current study suggest that there are four unique trajectories, or patterns, of change in body weight among individuals during IPT for AN. Moreover, there are statistically significant predictors of trajectory group membership which provides new knowledge about the process of weight restoration for AN. Additionally, the identification of such patterns and predictors provide much needed insight about individuals with AN who are at-risk for not achieving weight restoration during IPT (i.e., higher body weight at time of admission, no previous admission, shorter length of stay). Results point to new directions for intervention opportunities (e.g., randomized controlled studies that alter caloric intake based on assessment of risk factors for weight loss or minimal gain trajectories) as well as research (e.g., replication of current study with larger sample and different treatment or business models; further explication of predictors of patterns of change in body weight) to improve the process of weight restoration for individuals during IPT for AN and consequently influence outcomes.

References

Abraham, I. L., & Neundorfer, M. M. (1990). The design and analysis of time-series studies. In L.E. Moody (Ed.), *Advancing nursing science through research: Volume 2.* (pp.145-176). London: Sage Publications.

Alegria, M., Woo, M., Cao, Z., Torres, M., Meng, X., & Striegel-Moore, R. (2007). Prevalence and correlates of eating disorders in Latinos in the United States. *International Journal of Eating Disorders*, *40*, S15–S21. doi:10.1002/eat

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, fourth edition (DSM-IV)*. Washington, DC: American Psychiatric Press.

American Psychiatric Association. (2006). *Practice guideline for the treatment of patients with eating disorders* (3rd Ed). Arlington: American Psychiatric Publishing.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders, fifth edition (DSM-5)*. Washington, DC: American Psychiatric Press.

Anderson, E. A. (1985). *Practical comprehensive treatment of anorexia nervosa and bulimia.* Baltimore, MD: Johns Hopkins University Press.

Anderson, D. A., De Young, K. P., & Walker, D.C. (2009). *Handbook of assessment methods for eating behaviors and weight-related problems* (2nd Ed.). Newbury Park, CA: Sage.

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*, 724–731. doi:10.1001/archgenpsychiatry.2011.74

Attia, E., & Walsh, B. T. (2007). Anorexia Nervosa. *American Journal of Psychiatry*, *164*, 1805–1810. doi:10.1176/appi.ajp.2007.07071151

Baker, J. H., Thornton, L. M., Strober, M., Brandt, H., Crawford, S., Fichter, M. M., … Bulik, C. M. (2013). Temporal sequence of comorbid alcohol use disorder and

anorexia nervosa. *Addictive Behaviors*, *38*, 1704–1709. doi:10.1016/j.addbeh.2012.10.005

Banks, N. J. (1998). Designing medical record abstraction forms. *International Journal for Quality in Health Care*, *10*, 163–7. doi: 10.1093/intqhc/10.2.163

Baran, S. A., Weltzin, T. E., & Kaye, W. H. (1995). Low discharge weight and outcome in anorexia nervosa. *American Journal of Psychiatry, 15,* 1070–1072. doi: 10.1176/ajp.152.7.1070

Bardone-Cone, A. M., Wonderlich, S. A., Frost, R. O., Bulik, C. M., Mitchell, J. E., Uppala, S.,…& Simonich, H. (2007). Perfectionism and eating disorders: Current status and future directions. *Clinical Psychology Review*, *27*, 384–405. doi:10.1016/j.cpr.2006.12.005

Bazzini, D. G., Pepper, A., Swofford, R., & Cochran, K. (2015). How healthy are health magazine? A comparative analysis of cover captions and images of Women's and Men's Health magazine. *Sex Roles, 72*, 198–210. doi: 10.1007/s11199-015-0456-2

Becker, A. E., Burwell, R. A., Gilman, S. E., Herzog, D. B., & Hamburg, P. (2002). Eating behaviours and attitudes following prolonged television exposure among ethnic Fijian adolescent girls. *British Journal of Psychiatry, 180*, 509–514. doi: 10.1192/bjp.180.6.509

Becker, A. E., Fay, K. E., Agnew–Blais, J., Khan, A. N., Striegel–Moore, R. H., & Gilman, S. E. (2011). Social network media exposure and adolescent eating pathology in Figi. *British Journal of Psychiatry, 198,* 43–50. doi: 10.1192/bjp.bp.110.078675

Berg, K.C., Peterson, C.B., Frazier, P., & Crow, S.J. (2012). Psychometric evaluation of the Eating Disorder Examination and Eating Disorder Examination-Questionnaire: A

systematic review of the literature. *International Journal of Eating Disorders, 45,* 428–438. doi: 10.1002/eat.20931

Berridge, K. C., Ho, C., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Research, 1350,* 43–64. doi: 10.1016/j.brainres.2010.04.003

Bezance, J., & Holliday, J. (2013). Adolescents with anorexia nervosa have their say: A review of qualitative studies on treatment and recovery from anorexia nervosa. *European Eating Disorders Review*, *21*, 352–360. doi:10.1002/erv.2239

Blinder, B. J., Cumella, E. J., & Sanathara, V. A. (2006). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine*, *68*, 454–462. doi:10.1097/01.psy.0000221254.77675.f5

Brand-Gothelf, A., Leor, S., Apter, A., & Fennig, S. (2014). The impact of comorbid depressive and anxiety disorders on severity of anorexia nervosa in adolescent girls. *The Journal of Nervous and Mental Disease*, *202*, 759–762. doi:10.1097/NMD.0000000000000194

Brown, T. A., Holland, L. A., & Keel, P. K. (2014). Comparing operational definitions of *DSM-5* anorexia nervosa for research contexts. *International Journal of Eating Disorders, 47*, 76–84. doi: 10.1002/eat.22184

Brown, J. F., & Mehler, P. S. (2013). Medical complications of self-induced vomiting. *Eating Disorders: The Journal of Treatment & Prevention, 21*, 287–294. doi: 10.1080/10640266.2013.797317

Bühren, K., Schwarte, R., Fluck, F., Timmesfeld, N., Krei, M., Egberts, K., … Herpertz-Dahlmann, B. (2014). Comorbid psychiatric disorders in female adolescents with first-onset anorexia nervosa. *European Eating Disorders Review*, *22*, 39–44. doi:10.1002/erv.2254

Bulik, C. M., Landt, M., van Furth, E. F., & Sullivan, P. F. (2007). The genetics of anorexia nervosa. *Annual Reviews in Nursing, 27*, 263–275. doi: 10.1146/annurev.nutr.27.061406.093713

Bulik, C. M., Sullivan, P.F., Tozzi, F., Furberg, H., Lichtenstein, P., & Pederson, N. L. (2006). Prevalence, heritability, and prospective risk factors for anorexia nervosa. *Archives of General Psychiatry, 63,* 305–312. doi:10.1001/archpsyc.63.3.305

Cachelin, F. M., Phinney, J. S., Schug, R. A., & Striegel–Moore, R. H. (2006). Acculturation and eating disorders in a Mexican American community sample. *Psychology of Women Quarterly, 30*, 340–347. doi: 10.1111/j.1471-6402.2006.00309.x

Carter, J. C., Mercer-Lynn, K. B., Norwood, S. J., Bewell-Weiss, C. V., Crosby, R. D., Woodside, D. B., & Olmsted, M. P. (2012). A prospective study of predictors of relapse in anorexia nervosa: Implications for relapse prevention. *Psychiatry Research*, *200*, 518–523. doi:10.1016/j.psychres.2012.04.037

Castellini, G., Sauro, C. L., Mannucci, E., Ravaldi, C., Rotella, C. M. Faravelli, C.,…Ricca, V. (2011). Diagnostic crossover and outcome predictors in eating disorders according to *DSM-IV* and *DSM-V* proposed criteria: A 6-year follow-up study. *Psychosomatic Medicine, 73*, 270–279. doi: 10.1097/PSY.0b013e31820a1838

Centers for Disease Control and Prevention. (2013, December 6). *Body mass index.* Retrieved from http://www.cdc.gov/healthyweight/assessing/bmi/index.html

Clarke, T. K., Weiss, A. R. D., & Berrettini, W. H. (2012). The genetics of anorexia nervosa. *Clinical Pharmacology & Therapeutics, 91*, 181–188. doi: 10.1038/clpt.2011.253

Colton, A., & Pistrang, N. (2004). Adolescents' experiences of inpatient treatment for anorexia nervosa. *European Eating Disorders Review, 12*, 307–316. doi: 10.1002/erv.587

Corrigan, P. (1998). The impact of stigma on severe mental illness. *Cognitive and Behavioral Practice, 5,* 201–222. doi: 10.1016/S1077-7229(98)80006-0

Corrigan, P. W., & Watson, A. C. (2002). Understanding the impact of stigma on people with mental illnesses. *Journal of World Psychiatry, 1*, 6-19.

Crane, A. M., Roberts, M. E., & Treasure, J. (2007). Are obsessive-compulsive personality traits associated with a poor outcome in anorexia nervosa? A systematic review of randomized controlled trials and naturalistic outcome studies. *International Journal of Eating Disorders, 40*, 581–588. doi: 10.1002/eat.20419

Creswell, J. W. (2014). *Research design: Qualitative, quantitative, and mixed methods approaches* (4th Ed.). Thousand Oaks, CA: SAGE Publications, Inc.

Crisp, A. (2005). Stigmatization of and discrimination against people with eating disorders including a report of two nationwide surveys. *European Eating Disorders Review, 13*, 147–152. doi: 10.1002/erv.648

Crisp, A., Gelder, M. G., Rix, S., Metzler, H. J., & Rowlands, O. J. (2000). Stigmatization of people with mental illness. *British Journal of Psychiatry, 144*, 4–7. doi: 10.1192/bjp.177.1.4

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. doi:10.1002/erv.652

Eddy, K. T., Hennessey, M., & Thompson–Brenner, H. (2007). Eating pathology in east African women: The role of media exposure and globalization. *Journal of Nervous Mental Disorders, 195,* 196–202. doi:

Eisenhauer, L. A. (1994). A typology of nursing therapeutics. *Journal of Nursing Scholarship*, *26*, 261–264. doi: 10.1111/j.1547-5069.1994.tb00331.x

Engelsen, B.K., & Laberg, J.C. (2001). A comparison of three questionnaires (EAT-12, EDI, and EDE-Q) for assessment of eating problems in health female adolescents. *Nordic Journal of Psychiatry, 55,* 129–135. doi: 10.1080/08039480121342

Fairburn, C. G., & Beglin, S. (2008). Eating Disorder Examination Questionnaire. In C.G. Fairburn (Ed.), *Cognitive behavior therapy and eating disorders* (pp. 309–313). New York, NY: Guilford Press.

Fairburn, C.G., & Cooper, Z. (1993). The eating disorder examination (12th Ed.). In C. Fairburn & Wilson, G.T. (Eds.) *Binge eating: Nature assessment and treatment* (pp. 317–360). New York, NY: Guilford Press.

Faje, A. T., Fazeli, P. K., Miller, K. K., Katzman, D. K.., Ebrahimi, S., Mendes, N.,…Klibanski, A. (2014). Fracture risk and areal bone mineral density in adolescent females with anorexia nervosa. *International Journal of Eating Disorders, 47*, 458–466. doi: 10..1002/eat.22248

Fassino, S., Pierò, A., Tomba, E., & Abbate-Daga, G. (2009). Factors associated with dropout from treatment for eating disorders: A comprehensive literature review. *BMC Psychiatry*, *9*, 67. doi:10.1186/1471-244X-9-67

Fernández-Aranda, F., Pinheiro, A. P., Thornton, L. M., Berrettini, W. H., Crow, S., Fichter, M. M., … Bulik, C. M. (2008). Impulse control disorders in women with eating disorders. *Psychiatry Research*, *157*, 147–157. doi:10.1016/j.psychres.2007.02.011

Fichter, M. M., Quadflieg, N., & Hedlund, S. (2006). Twelve-year course and outcome predictors of anorexia nervosa. *International Journal of Eating Disorders, 39*, 87–100. doi: 10.1002/eat.20215

Fitzpatrick, K. K., & Lock, J. (2011). Anorexia nervosa. Systematic review 1011. *BMJ Clinical Evidence*. Retrieved from http://clinicalevidence.bmj.com/x/systematic-review/1011/overview.html

Fox, J., & Diab, P. (2015). An exploration of the perceptions and experiences of living with chronic anorexia nervosa (cAN) whilst an inpatient on an eating disorders unit (EDU): An interpretative phenomenological analysis (IPA) study. *Journal of Health Psychology*, *20*, 27–36. doi:10.1177/1359105313497526

Franko, D. L., Fuller-Tyszkiewicz, M., Rodgers, R. F., Gattario, K. H., Frisen, A., Diedrichs, P. C.,…Shingleton, R. M. (2015). Internalization as a mediator of the relationship between conformity to masculine norms and body image attitudes and behaviors among young men in Sweden, US, UK, and Australia. *Body Image, 15*, 54–60. doi: 10.1016/j.bodyim.2015.05.002

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. *The Journal of Adolescent Health*, *50*, 24–9. doi:10.1016/j.jadohealth.2011.06.011

Gaudio, S., & Di Ciommo, V. (2011). Prevalence of personality disorders and their clinical correlates in outpatient adolescents with anorexia nervosa. *Psychosomatic Medicine*, *73*, 769–74. doi:10.1097/PSY.0b013e318235b9b5

Gearing, R. E., Mian, I. A., Barber, J., & Ickowicz, A. (2006). A methodology for conducting retrospective chart review research in child and adolescent psychiatry. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *15*, 126–34.

Gilbert, P. (2009). Introducing compassion-focused therapy. *Advances in Psychiatric Treatment*, *15*, 199–208. doi: 10.1192/apt.bp.107.005264

Godart, N., Radon, L., Curt, F., Duclos, J., Perdereau, F., Lang, F.,…Flament, M. F. (2015). Mood disorders in eating disorder patients: Prevalence and chronology of ONSET. *Journal of Affective Disorders, 185*, 115–122. doi:10.1016/j.jad.2015.06.039

Gordon, K. H., Castro, Y., Sitnikov, L., & Holm–Denoma, J. M. (2010). Cultural body shape ideals and eating disorder symptoms among White, Latina, and Black college women, *Cultural Diversity and Ethnic Minority Psychology, 16*, 135–143. doi: 10.1037/a0018671

Grady, P. A., & Gough, L. L. (2015). Nursing science: Claiming the future. *Journal of Nursing Scholarship, 47*, 512–521. doi: 10.1111/jnu.12170

Gregory, K., & Radovinsky, L. (2012). Research strategies that result in optimal data collection from the patient medical record. *Applied Nursing Research*, *25*, 108–116. doi:10.1016/j.apnr.2010.02.004.Research

Guarda, A. S. (2008). Treatment of anorexia nervosa: Insights and obstacles. *Physiology and Behavior*, *94*, 113–120. doi:10.1016/j.physbeh.2007.11.020

Hadley, S. J., & Walsh, T. (2003). Gastrointestinal disturbances in anorexia nervosa and bulimia nervosa. *CNS and Neuroloigcal Disorders, 2,* 1–9. doi: 10.2174/1568007033338715

Halmi, K. A., Bellace, D. L., Berthod, S., Ghosh, S., Berrettini, W. H., Brandt, H. A., …Strober, M. (2012). Early childhood perfectionism. *International Journal of Eating Disorders*, *45*, 800–807. doi:10.1002/eat.22019

Halmi, K. A., Tozzi, F., Thornton, L. M., Crow, S., Fichter, M. M., Kaplan, A. S.,…Bulik, C. M. (2005). The relation among perfectionism, obsessive-compulsive personality disorder and obsessive-compulsive disorder in individuals with eating disorders. *International Journal of Eating Diorders, 38*, 371–374. doi: 10.1002/eat.20190

Harris, P. A., Taylor, R., Thielke, R., Payne, J., Gonzalez, N., & Conde, J. G. (2009). Research electronic data capture (REDCap) – A metadata-driven methodology and workflow process for providing translational research informatics support. *Journal of Biomedical Informatics*, *42*, 377–381. doi: 10.1016/j.jbi.2008.08.010

Harrison, A., Tchanturia, K., & Treasure, J. (2010). Attentional bias, emotion recognition, and emotion regulation in anorexia: State or trait? *Biological Psychiatry, 15*, 755–761. doi: 10.1016/j.biopsych.2010.04.037

Hart, S., Abraham, S., Franklin, R., & Russell, J. (2011). Weight changes during inpatient refeeding of underweight eating disorder patients. *European Eating Disorders Review*, *19*, 390–397. doi:10.1002/erv.1052

Hartmann, A., Wirth, C., & Zeeck, A. (2007). Prediction of failure of inpatient treatment of anorexia nervosa from early weight gain. *Psychotherapy Research, 17*, 218–229. doi: 10.1080/10503300600702315

Herpertz–Dahlmann, B. (2015). Adolescent eating disorders: Update on definitions, symptomatology, epidemiology, and comorbidity. *Child & Adolescent Psychiatric Clinics in North America, 24*, 177–196. doi: 10.1016/j.chc.2014.08.003

Herpertz-Dahlmann, B., Muller, B., Herpetz, S., Heussen, N. Hebebrand, J., & Remschmidt, H. (2001). Prospective 10-year follow-up in adolescent anorexia nervosa: Course, outcome, psychiatric comorbidity, and psychosocial adaptation. *Journal of Clinical Psychology and Psychiatric, 42*, 603–612. doi: 10.1111/1469-7610.00756

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. doi: 10.1002/erv.424

Hinshaw, S. (2006). The mark of shame: Stigma and mental illness and an agenda for change. Oxford Publishing: New York, New York.

Huas, C., Godart, N., Foulon, C., Pham-Scottez, A., Divac, S., Fedorowicz, V., … Rouillon, F. (2011). Predictors of dropout from inpatient treatment for anorexia nervosa: Data from a large French sample. *Psychiatry Research*, *185*, 421–426. doi:10.1016/j.psychres.2009.12.004

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biological Psychiatry*, *61*, 348–358. doi:10.1016/j.biopsych.2006.03.040

Hulley, S.B., Newman, T.B., & Cummings, S.R. (2013). Choosing the study subjects: Specification, sampling, and recruitment. In S.B. Hulley, S.B., S.R. Cummings, W.S., Browner, D.G., Grady, & T.B. Newman (Eds.), *Designing clinical research, 4th edition* (pp. 23–31). Philadelphia, PA: Lippincott, Williams & Wilkins.

Hűtter, G., Ganepola, S., & Hofmann, W. K. (2009). The hematology of anorexia nervosa. *International Journal of Eating Disorders, 42*, 293–300. doi: 10/1002/eat.20610

Jenerette, C. M., Brewer, C. A., & Atage, K. I. (2014). Care seeking for pain in young adults with sickle cell disease. *Pain Management Nursing, 15*, 324–330. doi: 10.1016/j.pmn.2012.10.007

Johnstone, J., & Rickard, K. (2006). Perceptions of college women with disordered eating and exercise patterns. *Social Behavior and Personality, 34*, 1035–1050. doi: 10.2224/sbp.2006.34.9.1035

Kaplan, A. S., Walsh, B. T., Olmstead, M., Attia, E., Devlin, M. J., Pike, K. M.,…Parides, M. (2009). The slippery slope: Prediction of successful weight maintenance in anorexia nervosa. *Psychological Medicine, 39*, 1037–1045. doi: 10.1017/S003329170800442X

Karg, R. S., Bose, J., Batts, K. R., Forman-Hoffman, V. L., Liao, D., Hirsch, E., … Hedden, S. L. (2014). Past year mental disorders among adults in the United States: Results from the 2008–2012 mental health surveillance study. Retrieved from http://www.samhsa.gov

Katzman, D. K. (2005). Medical complications in adolescents with anorexia nervosa: A review of the literature. *International Journal of Eating Disorders, 37*, S52–S59. doi: 10.1002/eat.20118

Katzman, D. K. (2012). Refeeding hospitalized adolescents with anorexia nervosa: Is "start low, advance slow" urban legend or evidence based? *Journal of Adolescent Health*, *50*, 1–2. doi:10.1016/j.jadohealth.2011.10.003

Kaye, W. H., Ebert, M. H., Raleigh, M., & Lake, R. (1984). Abnormalities in CNS monoamine metabolism in anorexia nervosa. *Archives of General Psychiatry, 41*, 350–355. doi: 10.1001/archpsyc.1984.01790150040007

Kaye, W. H., Frank, G. K. W., & McConaha, C. (1999). Altered dopamine activity after recovery from restricting–type anorexia nervosa. *Neuropsychopharmacology, 21*, 503–506. doi: 10.1016/S0893-133X(99)00053-6

Kaye, W. H., Gwirtsman, H. E., George, D. T., & Ebert, M. H. (1991). Altered serotonin activity in anorexia nervosa after long–term weight restoration. *Archives of General Psychiatry, 48*, 556–562. doi: 10.1001/archpsyc.1991.01810300068010

Kaye, W. H., Gwirtsman, H. E., George, D. T., Jimerson, D. C., & Ebert, M. H. (1988). CSF 5–HIAA concentrations in anorexia reduced values in underweight subjects after weight gain. *Biological Psychiatry, 23,* 102–105. doi: 10.1016/0006-3223(88)90113-8

Keel, P. K., & Brown, T. A. (2010). Update on course and outcome in eating disorders. *International Journal of Eating Disorders*, *43*, 195–204. doi:10.1002/eat.20810

Kelly, A. C., & Carter, J. C. (2013). Why self-critical patients present with more severe eating disorder pathology: The mediating role of shame. *British Journal of Clinical Psychology, 52*, 148–161. doi: 10.1111/bjc.12006

Keski-Rahkonen, A., Raevuori, A., Bulik, C. M., Hoek, H. W., Rissanen, A., & Kaprio, J. (2007). Factors associated with recovery from anorexia nervosa: A population-based study. *International Journal of Eating Disorders*, *47*, 117–123. doi:10.1002/eat.22168

Kiefer, J., & Wolfowitz, J. (1959). Optimum designs in regression problems. *Annuals of Mathematical Statistics, 30,* 271–294. doi:10.1214/aoms/1177706252

Kim, H.J., McDermott, P. A., & Barsevick, A. M. (2014). Comparison of groups with different patterns of symptom cluster intensity across the breast cancer treatment trajectory. *Cancer Nursing, 37*, 88–96. doi: 10.1097/NCC.0b013e31828293e0

Klump, K. L., Miller, K. B., Keel, P. K., McGue, M., & Iacono, W. G. (2001). Genetic and environmental influences on anorexia nervosa syndromes in a population-based twin study. *Psychological Medicine, 31,* 737–740. doi: 10.1017/s0033291701003725

Kortegaard, L. S., Hoerder, K., Joergensen, J., Gillberg, C., & Kyvik, K. O. (2001). A preliminary population-based twin study of self-reported eating disorder. *Psychological Medicine, 31*, 361–365. doi: 10.1017/S0033291701003087

Kostro, K., Lerman, J. B., & Attia, E. (2014). The current status of suicide and self-injury in eating disorders: A narrative review. *Journal of Eating Disorders*, *2*, 1–9. doi:10.1186/s40337-014-0019-x

Lantzouni, E., Frank, G. R., Golden, N. H., & Shenker, R. I. (2002). Reversibility of growth stunting in early onset anorexia nervosa: A prospective study. *Journal of Adolescent Health, 31*, 162–165. doi: 10.1016/S1054-139X(02)00342-7

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. doi: 10.1002/erv.432

Lilenfeld, L. R., Kaye, W. H., Greeno, C. G., Merikangas, K. R., Plotnicov, K., Pollice, C. P., Rao, R., Strober, M., Bulk, C. M., & Nagy, L. (1998). A controlled family study of anorexia nervosa and bulimia nervosa: Psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Archives of General Psychiatry, 55,* 603–610. doi: 10.1001/archpsyc.55.7.603

Lipsman, N., Woodside, D. B., & Lozano, A. M. (2014). Trends in anorexia nervosa research: An analysis of the top 100 most cited works. *European Eating Disorders Review, 22*, 9–14. doi: 10.1002/erv.2270

Lock, J., & Litt, I. (2003). What predicts maintenance of weight for adolescents medically hospitalized for anorexia nervosa? *Eating Disorders, 11*, 1–7. doi:10.1002/erv.496

Loughran, T., & Nagin, D.S. (2006). Finite sample effects in group-based trajectory models. *Sociological Methods and Research, 35,* 250–278. doi: 10.1177/0049124106292292

Luce, K., & Crowther, J.H. (1999). The reliability of the Eating Disorder Examination-Self Report Questionnaire Version (EDE-Q). *International Journal of Eating Disorders, 25,* 349–351. doi: 10.1002/(SICI)1098-108X(199904)25:3<349::AID-EAT15>3.0.CO;2-M

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. doi: 10.1002/eat.20634

Mainz, V., Schulte-Ruther, M., Fink, G. R., Herpertz-Dahlmann, B., & Konrad, K. (2012). Structural brain abnormalities in adolescent anorexia nervosa before and after weight recovery and associated hormonal changes. *Psychosomatic Medicine*, *74*, 574–582. doi: 10.1097/PSY.0b013e31824ef10e

Marzola, E., Nasser, J. A, Hashim, S. A, Shih, P. A., & Kaye, W. H. (2013). Nutritional rehabilitation in anorexia nervosa: Review of the literature and implications for treatment. *BMC Psychiatry*, *13*, 1–26. doi:10.1186/1471-244X-13-290

Mehanna, H. M., Moledina, J., & Travis, J. (2008). Refeeding syndrome: What it is, and how to prevent and treat it. *British Medical Journal, 336*, 1495–1498. doi: 10.1136/bmj.a301

Mehler, P. S., Cleary, B. C., & Gaudiani, J. L. (2011). Osteoporosis in anorexia nervosa. *Eating Disorders, 19,* 194–202. doi: 10.1080/10640266.2011.551636

Mehler, P. S., & MacKenzie, T. D. (2009). Treatment of osteopenia and osteoporosis in anorexia nervosa: A systematic review of the literature. *International Journal of Eating Disorders, 42*, 195–201. doi: 10.1002/eat.20593

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, 2010*, 1–7. doi:10.1155/2010/625782

Merikangas, K. R., He, J. P., Burstein, M., Swendsen, J., Avenevoli, S., Case, B.,…Olfson, M. (2011). Service utilization for lifetime mental disorders in U.S. adolescents: Results of the National Comorbidity Survey-Adolescent Supplement (NCS-A). *Journal of American Child & Adolescent Psychiatry, 50*, 32–45. doi: 10.1016/j.jaac.2010.10.006

Mewes, R., Tagay, S., & Senf, W. (2008). Weight curves as predictors of short-term outcome in anorexia nervosa inpatients. *European Eating Disorders Review*, *16*, 37–43. doi:10.1002/erv.807

Milos, G., Spindler, A., Schnyder, U., Martz, J., Hoek, H. W., & Willi, J. (2004). Incidence of severe anorexia nervosa in Switzerland: 40 years of development. *International Journal of Eating Disorders, 35*, 250–258. doi: 10.1002/eat.10244

Misra, M., Aggarwal, A., Miller, K. K., Almazan, C., Worley, M., Soyka, L. A…Klibanski, A. (2004). Effects of anorexia nervosa on clinical, hematologic, biochemical, and bone density parameters in community-dwelling adolescent girls. *Pediatrics, 114*, 1574–1583. doi: 10.1542/peds.2004-0540

Misra, M., & Kilbanski, A. (2014). Endocrine consequences of anorexia nervosa. *Lancet Diabetes Endocrinology, 2*, 581–592. doi: 10.1016/S2213-8587(13)70180-3

Mond, J.M., Hay, P.J., Rodgers, B., Owen, C., & Beumont, P.J.V. (2004). Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy, 42,* 551–567. doi:10.1016/S0005-7967(03)00161-X

Mond, J. M., Robertson-Smith, G., & Vetere, A. (2006). Stigma and eating disorders: Is there evidence of negative attitudes towards anorexia nervosa among women in the community? *Journal of Mental Health, 15*, 519–532. doi: 10.1080/09638230600902559

Morrison, M. A., Morrison, T. G., & Sager, C. (2004). Does body satisfaction differ between gay men and lesbian women and heterosexual men and women? A meta-analytic review. *Body Image, 1*, 127–138. doi: 10.1016/j.bodyim.2004.01.002

Nagin, D. S. (2014). Group-based trajectory modeling: An overview. *Annals of Nutrition and Metabolism, 65*, 205–210. doi: 10.1159/00360229

Nagin, D.S., & Odgers, C.L. (2010). Group-based trajectory modeling in clinical research. *Annual Reviews in Clinical Psychology, 6,* 109–138. doi: 10.1146/annurev/clinpsy.121208.131413

National Institute of Nursing Research (2011). *Mission and strategic plan.* Retrieved from http://www.ninr.nih.gov

Neuberger, S. K., Rao, R., Weltzin, T. E., Greeno, C., & Kaye, W. H. (1995). Differences in weight gain between restrictor and bulimic anorectics. *International Journal of Eating Disorders, 17*, 331–335. doi: 10.1002/1098-108X(199505)17:4<331::AID-EAT2260170404>3.0.CO;2-Z

Nicdao, E. G., Hong, S., & Takeuchi, D. T. (2007). Prevalence and correlates of eating disorders among Asian Americans: Results from the National Latino and Asian

American Study. *International Journal of Eating Disorders*, *40*, S22–S26. doi:10.1002/eat.20450

Nilsson, K., Sundbom, E., & Hagglof, B. (2008). A longitudinal study of perfectionism in adolescent onset anorexia nervosa-restricting type. *European Eating Disorders Review, 16*, 386–394. doi: 10.1002/erv.850

O'Brien, K. M., & Vincent, N. K. (2003). Psychiatric comorbidity in anorexia and bulimia nervosa: Nature, prevalence, and causal relationships. *Clinical Psychology Review, 23*, 57–74. doi: 10.1016/S0272-7358(02)00201-5

Offord, A., Turner, H., & Cooper, M. (2006). Adolescent inpatient treatment for anorexia nervosa: A qualitative study exploring young adults' retrospective views of treatment and discharge. *European Eating Disorders Review, 14*, 377–387. doi: 10.1002/erv.687

Overton, S. L., & Medina, S. L. (2008). The stigma of mental illness. *Journal of Counseling and Development, 86*, 143–151. doi: 10.1002/j.1556-6678.2008.tb00491.x

Phillipou, A., Rossell, S. L., & Castle, D. (2013). The neurobiology of anorexia nervosa: A systemic review. *Australian and New Zealand Journal of Psychiatry,* 00, 1–25. doi: 10.1177/0004867413509693

Phillips, R., Stewart, S. M., Presnell, K., Simmons, A., Kennard, B. D., Liss, D. & Setliff, S. (2010). Psychological variables impacting weight gain rapidity in adolescents hospitalized for eating disorders. *European Eating Disorders Review, 18,* 376–384. doi: 10.1002/erv.998

Preti, A., Rocchi, M. B. L., Sisti, D., Camboni, M. V., & Miotto, P. (2011). A comprehensive meta-analysis of the risk of suicide in eating disorders. *Acta Psychiatrica Scandinavica*, *124*, 6–17. doi:10.1111/j.1600-0447.2010.01641.x

Racine, S. E., & Wildes, J. E. (2015). Emotion dysregulation and anorexia nervosa : An exploration of the role of childhood abuse. *International Journal of Eating Disorders*, *48*, 55–58. doi:10.1002/eat.22364

Raevuori, A., Keski-Rahkonen, A., Hoek, H. W. (2014). A review of eating disorders in males. *Current Opinion in Psychiatry, 6*, 426–30. doi: 10.1097/YCO.0000000000000113

Räisänen, U., & Hunt, K. (2014). The role of gendered constructions of eating disorders in delayed help-seeking in men: A qualitative interview study. *BMJ Open, 4,* 1–8. doi:10.1136/bmjopen-2013- 004342

Reas, D.L., Grilo, C.M., & Mashab, R.M. (2006). Reliability of the Eating Disorder Examination-Questionnaire in patients with binge eating disorder. *Behaviour Research and Therapy, 44,* 43–51. doi:10.1016/j.brat.2005.01.004

Reiter, C. S., & Graves, L. (2010). Nutrition therapy for eating disorders. *Nutrition in Clinical Practice*, *25*, 122–136. doi:10.1177/0884533610361606

Rigaud. D., Boulier, A., Tallonneau, I., Brindisi, M. C., & Rozen, R. (2010). Body fluid retention and body weight change in anorexia nervosa patients during refeeding. *Clinical Nutrition, 29*, 749–755. doi: 10.1016/j.clnu.2010.05.007

Rikani, A. A., Choudhry, Z., Choudhry, A. M., Ikram, H., Asghar, M. W., Kajal, D., … Mobassarah, N. J. (2013). A critique of the literature on etiology of eating disorders. *Annals of Neurosciences*, *20*, 157–161. doi:10.5214/ans.0972.7531.200409

Roberto, C. A., Mayer, L. E. S., Brickman, A. M., Barnes, A., Muraskin, J., Yeung, L…Walsh, B. T. (2011). Brain tissue volume changes following weight gain in adults with anorexia nervosa. *International Journal of Eating Disorders, 44*, 406–411. doi: 10.1002/eat.20840

Root, T. L., Pinheiro, A. P., Thornton, L., Strober, M., Fernández -Aranda, F., Brandt, H., … Bulik, C. M. (2010). Substance use disorders in women with anorexia nervosa. *International Journal of Eating Disorders*, *43*, 14–21. doi:10.1002/eat.20670

Rosen, D. S., & The Committee of Adolescence. (2010). Identification and management of eating disorders in children and adolescents. *Pediatrics*, *126*, 1240–1253. doi:10.1542/peds.2010-2821

Roy, C. (2009). *The Roy Adaptation Model (3rd Ed.).* Upper Saddle River, NJ: Prentice Hall Health.

Russell, G. (1995). Anorexia nervosa through time. In G. Szmukler, C. Dare, & J. Treasure (Eds.), *Handbook of Eating Disorders: Theory, Treatment, and Research* (pp. 5–17). Chichester, England: John Wiley & Sons, Ltd.

Salbach-Andrae, H., Schneider, N., Seifert, K., Pfeiffer, E., Lenz, K., Lehmkuhl, U.,…Korte, A. (2009). Short-term outcome of anorexia nervosa in adolescents after inpatient treatment: A prospective study. *European Child & Adolescent Psychiatry, 18*, 701–704. doi: 10.1007/s00787-009-0024-9

Schwartz, D. M., Thompson, M. G., & Johnson, C. L. (1985). Anorexia nervosa and bulimia: The sociocultural context. In S. W. Emmett (Ed.), *Theory and treatment of anorexia nervosa and bulimia: Biomedical, sociocultural, and psychological perspectives* (pp. 95–112). New York, NY: Brunner/Mazel, Inc.

Sly, R., & Bamford, B. (2011). Why are we waiting? The relationship between low admission weight and end of treatment weight outcomes. *European Eating Disorders Review*, *19*, 407–410. doi:10.1002/erv.1061

Solanto, M. V., Hertz, S., Jacobson, M. S., Golden, N. H., & Heller, L. (1994). Rate of weight gain of inpatients with anorexia nervosa under two behavioral contracts. *Pediatrics, 93*, 989–991.

Solomon, S. M., & Kirby, D. F. (1990). The refeeding syndrome: A review. *Journal of Parenteral and Enteral Nutrition, 14*, 90–97. doi: 10.1177/014860719001400190

Spindler, A., & Milos, G. (2007). Links between eating disorder symptom severity and psychiatric comorbidity. *Eating Behaviors, 8*, 364–373. doi:10.1016/j.eatbeh.2006.11.012

StataCorp. 2015. *Stata Statistical Software: Release 14*. College Station, TX: StataCorp LP.

Steinhausen, H. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry, 159*, 1284–1293. doi: 10.1176/appi.ajp.159.8.1284

Steinhausen, H. C., Grigoroiu-Serbanescu, M., Boyadjieva, S., Neumärker, K. J., & Metzke, C. W. (2008). Course and predictors of rehospitalization in adolescent anorexia nervosa in a multisite study. *International Journal of Eating Disorders*, *41*, 29–36. doi:10.1002/eat.20414

Stewart, M. C., Schiavo, R. S., Herzog, D. B., & Franko, D. L. (2008). Stereotypes, prejudice, and discrimination of women with anorexia nervosa. *European Eating Disorder Review, 16*, 311–318. doi: 10.1002/erv.849

Stice, E., Marti, N., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed *DSM-5* eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology*, *122*, 445–457. doi:10.1037/a0030679

Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry, 157*, 393–401. doi: 10.1176/appi.ajp.157.3.393

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents. Results from the national

comorbidity survey replication adolescent supplement. *Archives of General Psychiatry*, *68*, 714–723. doi:10.1001/archgenpsychiatry.2011.22

Taylor, J. Y., Caldwell, C. H., Baser, R. E., Faison, N., & Jackson, J. S. (2007). Prevalence of eating disorders among Blacks in the National Survey of American Life. *International Journal of Eating Disorders*, *40,* S10–S14. doi:10.1002/eat.20451

Theander, S. (1995). The essence of anorexia nervosa: Comment on Gerald Russell's "Anorexia nervosa through time." In G. Szmukler, C. Dare, & J. Treasure (Eds.), *Handbook of Eating Disorders: Theory, Treatment, and Research* (pp. 19–24). Chichester, England: John Wiley & Sons, Ltd.

Trace, S. E., Baker, J. H., Penas–Lledo, E., & Bulik, C. M. (2014). The genetics of eating disorders. *Annual Review of Clinical Psychology, 9*, 589–620. doi: 10.1146/annurev-clinpsy-050212-185546

Treasure, J., Claudino, A. M., & Zucker, N. (2010). Eating disorders. *The Lancet*, *375*, 583–593. doi:10.1016/S0140-6736(09)61748-7

Turrell, S. L., Davies, R., Graham, H., & Weiss, J. (2005). Adolescents with anorexia nervosa: Multiple perspectives of discharge readiness. *Journal of Child & Adolescent Psychiatric Nursing, 18*, 116–126. doi: 10.1111/j.1744-6171.2005.00021.x

Van Son, G. E., Van Hoeken, D., Van Furth, E. F., Donker, G. A., & Hoek, H. W. (2010). Course and outcome of eating disorders in a primary care-based cohort. *International Journal of Eating Disorders, 43,* 130–138. doi: 10.1002/eat.20676

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. doi: 10.1002/eat.20763

Wahl, O. F. (1999). Mental health consumers' experience of stigma. *Schizophrenia Bulletin, 25*, 467–478.

Warren, M. P. (2011). Endocrine manifestations of eating disorders. *Journal of Clinical Endocrinology Metabolism, 96*, 333–343. doi: 10.1210/jc.2009-2304

Westwood, L. M., & Kendall, S. E. (2012). Adolescent client views towards the treatment of anorexia nervosa: A review of the literature. *Journal of Psychiatric and Mental Health Nursing*, *19*, 500–508. doi:10.1111/j.1365-2850.2011.01819.x; 10.1111/j.1365-2850.2011.01819.x

Winston, A. P. (2012). The clinical biochemistry of anorexia nervosa. *Annals of Clinical Biochemistry*, *49*, 132–143. doi:10.1258/acb.2011.011185

Wolfe, B. E., & Gimby, L. B. (2003). Caring for the hospitalized patient with an eating disorder. *Nursing Clinics of North America*, *38*, 75–99. doi:10.1016/S0029-6465(02)00064-6

Woodside, D. B., Garfinkle, P. E., Lin, E., Goering, P., & Kaplan, A. S. (2001). Comparisons of men with full or partial eating disorders, men without eating disorders, and women with eating disorders within the community. *American Journal of Psychiatry, 158*, 570–574. doi: 10.1176/appi.ajp.158.4.570

Yamashita, S., Kawai, K., Yamanaka, T., Inoo, T., Yokoyama, H., Morita, C., … Kubo, C. (2010). BMI, body composition, and the energy requirement for body weight gain in patients with anorexia nervosa. *The International Journal of Eating Disorders*, *43*, 365–71. doi:10.1002/eat.20700

Zanarini, M. C., Reichman, C. a., Frankenburg, F. R., Reich, D. B., & Fitzmaurice, G. (2010). The course of eating disorders in patients with borderline personality disorder: A 10-year follow-up study. *International Journal of Eating Disorders*, *43*, 226–232. doi:10.1002/eat.20689

Zhao, Y., & Encinosa, W. (2011). *An Update on Hospitalizations for Eating Disorders, 1999 to 2009* (pp. 1–10). Rockville: Agency for Healthcare Research and Quality. Retrieved from http://www.hcup-us.ahrg.gov/reports/statbriefs/sb120.pdf

Zipfel, S., Lowe, B., Reas, D. L., Deter, H. C., & Herzog, W. (2000). Long-term prognosis in anorexia nervosa: Lessons from a 21-year follow-up study. *Lancet, 26*, 721–722. doi: 10.1016/S0140-6736(99)05363-5

Zipfel, S., Sammet, I., Rapps, N., Herzog, W., Herpertz, S., & Martens, U. (2006). Gastrointestinal disturbances in eating disorders: Clinical and neurobiological aspects. *Autonomic Neuroscience Basic and Clinical, 129*, 99–106. doi: 10.1016/j.autneu.2006.07.023

Appendix A. Data Abstraction Form

Study_ID _____

Demographics

1. What is the patient's age (years) at admission?

2. What is thepatient's gender?
   Female
   Male

3. What is the patient's marital status?
   Never married, single
   Never married, in a relationship married
   Separated divorced other, specify
   Not documented
   If "other," then specify

4. What is the patient's race/ethnicity? Check all that apply.
   White, non-Hispanic
   White, Hispanic
   Black, non-Hispanic
   Black, Hispanic
   Asian/Pacific Islander
   Native American
   Other, specify
   Not documented

5. What are the patient's years of education?
   Less than or equal to 9 years
   More than 9 years and less than or equal to 12 years; high school or some high school
   More than 12 and less than or equal to 16; college or some college
   16 years; post college education
   Not documented

6. What is the patient's living situation at admission?
   Alone
   With significantother
   With roommate(s)/friend(s)
   With family (e.g., parents, grandparents, children)
   Not documented
   Other, specify

7. Is this a readmission to inpatient treatment at Walden?
   No
   Yes

   Not documented

8. Does the patient have prior inpatient treatment admissions for anorexia nervosa?
   No
   Yes
   Not documented

Eating Disorder Presentation at Admission

9. What is the patient's height (inches) at admission?

10. Patient's calculated height (meters) at admission

11. What is the patient's weight (lb) at admission?

12. Patient's calculated weight (kg) at admission

13. Patient's calculated BMI at admission.

14. What is the patient's ideal body weight (lb)?

15. What is the patient's % IBW at admission?

16. What are the initial calories at admission determined for patient?

17. What are the endorsed eating disorder behaviors at admission? (Check all that apply)
    Restricting
    Bingeing
    Self-induced vomiting
    Diuretics
    Diet pills
    Laxatives
    Ipecac
    Caffeine misuse
    Excessive/over-exercising
    Other(s), specify
    Not documented

18. Specify AMOUNT and FREQUENCY for each behavior endorsed in the previous question.

Eating Disorder History

19. What is the patient's age at the start of their eating disorder?

20. What is the patient's weight (lb) at the start of the eating disorder?

21. What is the patient's lowest weight (lb)?

22. What is the patient's age (years) at lowest weight?

23. What is the patient's highest weight (lb)?

24. What is the patient's age (years) of highest weight?

Psychiatric and Medical Presentation at Admission

25. List psychiatric diagnoses at admission?

26. What are the psychotropic medications at admission? List medication names ONLY.

27. Does the patient report suicidal ideation at admission?
    No
    Yes
    Not documented

28. Does the patient report self-injurious behaviors at admission?
    No
    Yes
    Not documented

29. Does the patient report substance abuse at admission?
    No
    Yes
    Not documented

30. If yes, then check all that apply.
    Alcohol
    Cocaine
    Marijuana
    Opiates (e.g., heroin, oxycodone, percocets)
    Sedatives
    Other(s), specify

31. What are the medical diagnoses (if any) at admission?

Psychiatric History

32. Does the patient report a family history of psychiatric disorders and/or substance abuse?
    No
    Yes
    Unknown/I don't know
    Not documented

33. If yes, then check all that apply
    Anxiety disorders

Completed suicides/accidental overdoses
Eating disorders
Mood disorders (i.e., depression, bipolar)
Psychotic disorders
Substance abuse
Unknown/I don't know
Not documented

34. If patient endorsed a family history of eating disorders, then check all that apply.
Anorexia nervosa
Bulimia nervosa
Binge eating disorder
Eating disorder NOS
Not documented

35. Does the patient report a history of traumatic experience(s)?
No
Yes
Not documented

36. If yes, then check all that apply.
Emotional/psychological
Physical
Sexual
Other, specify Not documented
If "other," please specify

37. Does the patient report a history of self-injurious behaviors?
No
Yes
Not documented

38. Does the patient report a history of suicidal ideation?
No
Yes
Not documented

39. Does the patient report a history of suicide attempts/gestures?
No
Yes
Not documented

40. Does the patient endorse a history of substance misuse?
No
Yes

Not documented

41. If yes, then check all that apply
Alcohol

Cocaine
Marijuana
Opiates (e.g., heroin, oxycodone, percocets)
Sedatives
Other(s), specify

Discharge Information

42. What are the patient's psychiatric diagnoses at discharge?

43. What are the psychotropic medications at discharge? List medication names ONLY.

44. What is the patient's disposition at discharge?
Residential within facility
Partial hospitalization within facility
IOP within facility
Against medical advice (AMA)
Administrative
Transfer to other facility for lower level of care
Other, specify
Not documented

45. What is the patient's length of stay (days)?

46. What is patient's weight (lb) at discharge?

47. Patient's calculated weight (kg) at discharge.

48. Calculated patient's BMI at discharge.

49. Calculated patient's % IBW at discharge.

Eating Disorder Examination Questionnaire (EDE-Q) Scores

50. What is the Restraint subscale score at admission?

51. What is the Eating Concern subscale score at admission?

52. What is the Shape Concern subscale score at admission?

53. What is the Weight Concern subscale score at admission?

54. What is the Global score at admission?

55. What is the Restraint subscale score at discharge?

56. What is the Eating Concern subscale score at discharge?

57. What is the Shape Concern subscale score at discharge?

58. What is the Weight Concern subscale score at discharge?

59. What is the Global score at discharge?