THE INFLUENCE OF WEIGHT SUPPRESSION ON THE DEVELOPMENT AND MAINTENANCE OF EATING PSYCHOPATHOLOGY

A dissertation submitted
to Kent State University in partial fulfillment of the requirements for the degree of Doctor of Philosophy

by

Michelle D. Jones

August 2016

©Copyright
All rights reserved
Except for previously published materials
TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACKNOWLEDGMENTS

CHAPTERS

I. Introduction

Theoretical Basis of Weight Suppression

Weight Suppression and Eating Disorder Psychopathology

Proposed Model

Hypotheses

II. Method

Participants

Measures

Procedure

III. Results

Data Quality

Missing Data

Data Preparation

Correlational Analyses

Mediation Analyses

Moderation Analyses

Post Hoc Analyses
VI. Discussion.................................................................40

Weight Suppression, Dietary Restriction, and Eating Psychopathology........40

BMI, Impulsivity, Emotion Regulation, and Perfectionism as Moderators.......44

Limitations and Suggestions for Future Research.................................46

REFERENCES...........................................................................49

APPENDICES............................................................................74

A. Measures............................................................................74
LIST OF FIGURES

Figure 1. Proposed Model of the Relationship between Weight Suppression and Eating Disorder Psychopathology………………………………………………………………………66

Figure 2. Relationships Proposed in Hypotheses 1-4……………………………………………67

Figure 3. Proposed Relationships in Hypothesis 6……………………………………………..68
LIST OF TABLES

Table 1. Time 1 Differences between Participant Groups.............................................69
Table 2. Descriptive Statistics.........................................................................................70
Table 3. Results of Correlational Analyses.......................................................................71
Table 4. Results of Regression Analyses...........................................................................72
Table 5. Results of Post Hoc Analyses Predicting Time 3 EDDS.....................................73
Acknowledgements

Throughout my years of education, I have received support and encouragement from a number of individuals. First and foremost, I must thank my advisor, Dr. Jan Crowther, for her mentorship over the past six years. Thank you for everything, but most importantly for believing in me, holding me accountable for deadlines, and always making sure I was properly dressed for winter weather. I could not have asked for a better mentor and role model.

Thank you to my dissertation committee, Drs. Jeff Ciesla, John Updegraaff, Richard Adams, and Sara Newman, for your contributions to this project. I must extend a special thank you to Dr. Jeff Ciesla for his help with the statistics on this project, as well as many others throughout my tenure as a graduate student.

To the past and present members of the Crowther Lab, thank you for being incredible labmates, travel buddies, and friends. Thank you also to my many friends and colleagues at Kent for the advice, laughs, and adventures, and for being the primary consumers of my baked goods. You all helped me to keep going when the road was rough.

None of my accomplishments would have been possible without the love and support from my family. To my parents, thank you for showing me how to work hard for what I want and for never allowing me to give up on my dreams. Thank you to my brother, Chad, for always encouraging me. Thank you to my son, Sawyer, for inspiring me in everything I do. Finally, thank you to my husband, Kyle, for the incredible sacrifices you made in order for me to pursue my goals and for never wavering in your love, encouragement, and support.
The influence of weight suppression on the development and maintenance of eating psychopathology

Weight suppression is defined as the difference between an individual’s highest weight and his or her current weight (Lowe, 1984). It has also been described as significant weight loss achieved through dieting that is sustained for a significant period of time (e.g., several months, longer than one year; French & Jeffrey, 1997; Lowe, 1993). The definition of weight suppression considers highest weight not due to pregnancy or illness and, depending on the population, may be calculated based on highest weight at current height or highest weight after age 18. When considering individuals currently in treatment or who have recently completed treatment for an eating disorder, weight suppression is typically calculated as the difference between the individual’s highest weight and their pretreatment weight. This is to account for the fact that many eating disorder treatments encourage or require weight gain and an individual’s weight may change frequently throughout the course of treatment. The individual’s weight during or immediately following treatment may not accurately reflect the extent to which the individual suppressed his or her weight prior to treatment. Weight suppression represents a distinct construct from weight fluctuation (significant weight change over some predetermined period of time; e.g., greater than a 15 pound weight change over one year; or the difference between highest and lowest adult weight; Van Son, van der Meer, & Van Furth, 2013) and weight rebound (the difference between current weight and lowest previous weight at current height; Butryn, Lowe, Safer, & Agras, 2006) in both definition and potential impact on eating behaviors.
The purpose of this dissertation was to examine the relationships among weight suppression, restrictive eating, and eating disorder pathology and to evaluate a proposed model that incorporates weight suppression as a causal variable in the development of eating psychopathology. To fulfill these aims, the theoretical basis and conceptual basis of weight suppression, including restraint theory, will first be explored. Next, empirical research examining the characteristics, correlates, and consequences of weight suppression will be reviewed. The four primary existing models of eating disorder onset and maintenance will then be explained and critiqued. Finally, several relationships between weight suppression and variables related to eating pathology will be hypothesized, culminating in the proposal of a new theoretical model of weight suppression and eating psychopathology.

**Theoretical Basis of Weight Suppression**

Weight suppression was first formally identified in the literature as the third factor of Lowe’s three-factor model of dieting (Lowe, 1993). This model emerged as an attempt to expand and improve upon the unifactorial restraint theory (Herman & Polivy, 1984). Restraint theory originally developed from Nisbett’s (1972) set point theory, which states that obese individuals are prone to under-responding to hunger and satiety cues and over-responding to external cues when their weight is suppressed below its biologically appropriate level, or “set point.” Herman and Polivy (Herman & Mack, 1975; Herman & Polivy, 1975) applied this theory to individuals of normal weight, suggesting that normal weight individuals who suppress their weight below their natural set point experience the same cue response as obese individuals below their set point. Herman and Polivy extended their theory further by suggesting that when the dietary self-control of restrained individuals is disrupted, the individuals’ underlying hunger would behaviorally manifest itself through overeating (Herman & Polivy, 1980).
Herman and Polivy (1980) further developed restraint theory to incorporate a cognitive explanation of the behavior of restrained eaters over their previous set point explanation. Restraint was described as efforts made to achieve or maintain a lower weight regardless of whether this weight loss was achieved (Herman & Polivy, 1980). This shift in restraint theory was ultimately formalized as the “boundary” model of eating (Herman & Polivy, 1984). According to this model, restrained eaters differ from unrestrained eaters as a function of both physiological factors and social and psychological factors. Physiological factors influence the initiation and cessation of eating due to physiological sensations that cue sensations of hunger and satiety. However, much of the time individuals are in a state that is not significantly influenced by hunger or satiety cues—a state that Herman and Polivy termed the “zone of biological indifference” (Herman & Polivy, 1984). When individuals are in this zone, social and psychological factors determine when eating is initiated and ended. In the boundary model, restrained eaters (also referred to as “dieters”) create a “diet boundary” at some point within their zone of biological indifference, which influences cognitive rules that dictate food intake in order to lose or maintain weight. The dietary history of restrained eaters, which involves both dieting and overeating, causes habituation to hunger and satiety cues. As a result, restrained eaters have a larger zone of biological indifference compared to unrestrained eaters. When a dietary boundary in a restrained eater is violated, the individual’s dietary inhibitions are removed. Because the restrained eater’s zone of biological indifference is relatively large, a greater amount of food is required to reach satiety and the individual overeats as a result. Thus, violations of dietary boundaries in restrained individuals typically result in overeating (Herman & Polivy, 1984).
A substantial amount of empirical research provides evidence supporting restraint theory. The majority of research conducted to evaluate restraint theory has utilized either preload or induction of emotional distress methodologies. Preload studies involve the consumption of high-calorie preloads (e.g., milkshakes), which are assumed to violate a diet boundary, followed by examination of voluntary food consumption. Restrained eaters are less able to regulate their eating than non-restrained eaters and, as a result, consume more food following a preload compared to non-restrained eaters and compared to restrained eaters who do not consume a preload (e.g., Heatherton & Baumeister, 1991; Herman & Mack, 1975; Herman & Polivy, 1975; Lowe, 1987; Ruderman, 1986). Restrained eaters also consume greater amounts of food when they are emotionally distressed compared to when they are calm. This is the opposite of the response pattern seen in unrestrained eaters, who typically eat less when experiencing emotional distress compared to the amount of food they consume when experiencing a calm emotional state (Herman & Polivy, 1975). Studies that separately evaluate cognitive factors in preload studies have found that participants’ beliefs about caloric intake have a greater effect on overeating than the actual caloric intake (Polivy, 1976; Spencer & Fremouw, 1979; Woody, Costanzo, Liefer, & Conger, 1981). Restrained eaters report experiencing both psychological and physiological responses to food (Klajner, Herman, Polivy, & Chhabra, 1981) that may increase their vulnerability to overeating. Taken together, these results indicate that restrained eaters exhibit distinct patterns of consumption compared to their unrestrained counterparts that may be the result of not only their psychological, but their physiological response to food exposure and consumption.

Despite substantial empirical evidence supporting restraint theory, Lowe (1993) presented two primary criticisms of the research on restraint theory and the theory itself. First,
research using two different measures of restraint, one cognitive (i.e., cognitive restraint) and one behavioral (i.e., dietary restraint) has failed to explain why restrained eaters overeat in some situations (Laessle, Tuschl, Kotthaus, & Pirke, 1989a; Laessle, Tuschl, Kotthaus, & Pirke, 1989b). Second, Lowe also pointed out that restraint theories and studies evaluating restraint theory imply that the terms “restrained eating” and “dieting” are synonymous. Despite current dieting efforts and their likelihood to think dichotomously about their diets, restrained eaters who are currently dieting typically do not overeat following the breach of dietary boundaries. Thus, Lowe argued that dieting and restraint are more accurately conceptualized as unique constructs. Lowe (1993) concluded that restraint theory fails to account for restrained eaters’ overeating in some situations and fails to identify and explain differences between dieting and restraint.

In response to these criticisms, Lowe (1993) presented his three-factor model of dieting to explain dieting behavior. As the name suggests, the three-factor model includes three aspects of dieting to explain eating behaviors among restrained eaters. The first of these factors, frequency of dieting and overeating, reflects a consensus in the literature that restrained eaters experience repeated episodes of dieting and overeating that render them relatively insensitive to the effects of food consumption. This lack of sensitivity allows restrained eaters to consume excessive amounts of food once dietary control has been relinquished (Lowe, 1993). Current dieting represents the second factor in the three-factor model and refers to an individual’s ongoing efforts to restrict his or her caloric intake for the purposes of losing weight. This factor focuses on efforts to lose weight rather than success in weight loss and suggests that dieting individuals employ cognitive aspects of commitment to adhere to a diet. These individuals are also likely to view their diets in “rigid, all-or-none terms” (Lowe, 1993). Finally, the three-factor model suggests that the third factor, weight suppression, represents dieting success in the form of
sustained weight loss. As a result of dieting success and long-term weight loss, weight suppressors are more capable of continuous dietary restriction and eating control compared to individuals who do not suppress their weight (Lowe, 1993).

One issue raised by Lowe (1993) warrants clarification: the difference between dieting and restraint. Historically, dieting has been defined in the literature in terms of restrained eating (French & Jeffrey, 1997; Lowe, 1993; Ruderman, 1986). There is, in fact, some overlap between individuals who are dieting and individuals who are restrained eaters, but many individuals currently dieting are not restrained eaters and only a small percentage of restrained eaters are currently dieting (Lowe, 1993). Additionally, prior research yielded discrepant results that may be due to this type of definition of dieting, which prompted further clarification of these terms (Heatherton, Herman, Polivy, King, & McGree, 1988; Lowe, 1993). Lowe (1993) defined restrained eaters as those with a history of chronic dieting and dieting as current cognitive and behavioral efforts to lose weight. Weight suppression also represents a distinct construct from restraint, as women who suppress their weight have similar scores on measures of restraint as non-weight suppressors (French & Jeffrey, 1997). Thus, Lowe’s three-factor model (1993) not only serves to provide a more comprehensive explanation for dieting behavior, but also clarifies the distinction between restraint, dieting, and suppressed weight. Lowe’s (1993) differentiation of these three factors and their potential impact on eating and weight-control behaviors influenced a new area of research within the greater body of disordered eating literature that explored the characteristics and consequences of weight suppression.

**Weight Suppression and Eating Disorder Symptomatology**

The literature on weight suppression indicates a significant relationship between weight suppression and dieting and eating disorder-related variables in both population-based and eating
disorder samples. Correlational research using community samples has found that weight suppression is positively associated with number of years since maximum weight, number of intentional weight loss episodes, current dieting behaviors, low-fat eating behaviors, frequency and intensity of exercise (French & Jeffrey, 1997), age, dietary restraint, drive for thinness, disinhibition, history of dieting during childhood, history of dieting during adulthood, (Mitchell, Neale, Bulik, Lowe, Maes, Kendler, & Mazzeo, 2011), and overweight or obese status during childhood (Van Son et al., 2013). The presence of weight suppression appears to be, in part, influenced by genetics (Mitchell et al., 2011). Findings are mixed regarding the relationship between weight suppression and body dissatisfaction in population-based samples, with one study finding a negative relationship between these variables (Mitchell et al., 2011) and another finding a non-significant relationship (Van Son et al., 2013). In correlational studies using eating disorder samples, weight suppression is positively associated with the frequency of binge eating (Butryn et al., 2011; Lowe et al., 2007) and purging (Butryn et al., 2011).

In laboratory studies examining food consumption following a high-calorie preload, individuals high in weight suppression consumed smaller amounts of food compared to individuals low in weight suppression (Lowe & Kleifield, 1988) and experienced a reduced preference for sweet taste (Kleifield & Lowe, 1988). High weight suppression participants in these studies also reported experiencing less hunger and consumed a lower number of calories prior to participating in the study compared to low weight suppression participants (Lowe & Kleifield, 1988). Longitudinal studies indicate that higher weight suppression is associated with increased weight gain (Lowe, Annunziato, et al., 2006) and greater bulimic symptomatology, including onset and maintenance of BN over a 10-year period of time (Keel & Heatherton, 2010). In longitudinal studies using treatment samples of women with BN, weight suppression
was found to predict weight gain and time to remission, with individuals higher in weight suppression gaining more weight and having a longer time to remission of BN symptoms compared to individuals lower in weight suppression (Herzog, Thomas, Kass, Eddy, Franko, & Lowe, 2010; Lowe et al., 2011).

Research examining weight suppression as a predictor of treatment outcome in eating disorder samples has generally produced mixed findings. In one study, weight suppression significantly predicted premature termination and treatment outcome in BN-spectrum samples, such that individuals higher in weight suppression were more likely to terminate treatment prematurely and less likely to be abstinent from binging and purging following treatment completion (Butryn et al., 2006). Higher weight suppression has also been found to predict greater weight gain over the course of treatment in individuals diagnosed with BN-spectrum disorders in some studies (Carter, McIntosh, Joyce, & Bulik, 2008; Lowe, Davis, et al., 2006), but not others (Zunker, Crosby, Mitchell, Wonderlich, Peterson, & Crow, 2011). Other studies have found no relationship between weight suppression and treatment outcome (i.e., treatment completion and abstinence from binging and purging; Carter et al., 2008; Dawkins, Watson, Egan, & Kane, 2013; Zunker et al., 2011). Weight suppression does not seem to be a relevant variable for predicting treatment outcome among individuals with binge eating disorder (BED; Zunker et al., 2011), but higher weight suppression predicts greater weight gain, higher rate of weight gain, and presence of bulimic symptoms during treatment and at discharge among individuals diagnosed with AN (Berner, Shaw, Witt, & Lowe, 2013; Wildes & Marcus, 2012). The relationship between degree of weight suppression and treatment outcome also appears to be moderated by BMI among individuals diagnosed with AN (Berner et al., 2013).
Although previous research has provided important information regarding the nature and effects of weight suppression, several limitations of the existing literature raise additional questions about the relationship between weight suppression and eating disorder symptomatology. There is a general agreement in the literature that weight suppression is equivalent to the discrepancy between an individual’s highest historical weight and his or her current weight (Lowe, 1993); however, empirical studies slightly differ in their operational definition of weight suppression. For example, some studies consider the highest weight at the individual’s current height (Butryn et al., 2006; Butryn et al., 2011; Lowe, Annunziato, et al., 2006; Lowe et al., 2007; Wildes & Marcus, 2012) while others consider the individual’s highest weight since reaching his or her adult height (Berner et al., 2013; Herzog et al., 2010) or the individual’s highest weight since age 18 (Carter et al., 2008; Dawkins et al., 2013; Keel & Heatherton, 2010; Mitchell et al., 2011; Van Son et al., 2013; Zunker et al., 2011). A few studies (French & Jeffrey, 1997; Lowe, Davis, et al., 2006; Lowe & Kleifield, 1988) only considered highest reported weight without specifying age or height when this weight was achieved. Although the resulting values are likely similar, these small discrepancies in calculation may result in different findings.

Another criticism of the measurement of weight suppression in previous studies relates to the consideration of an individual’s height. With few exceptions (Kleifield & Lowe, 1991; Lowe & Kleifield, 1988), the reviewed studies typically did not consider height when calculating weight suppression and interpreting its relationships and effects. Although the absolute value of weight suppression may be equivalent for two individuals, the percentage of an individual’s weight that is suppressed will likely be greater for individuals of shorter stature compared to those who are taller (e.g., an individual who is five feet tall who suppresses his or her weight by
20 pounds will likely have suppressed a greater percentage of his or her weight than an individual who is six feet tall and also suppressed his or her weight by 20 pounds. Given that the amount of weight suppression is likely important in determining outcomes (Butryn et al., 2006; Butryn et al., 2011; Lowe et al., 2011; Van Son et al., 2013), a higher percentage of weight suppressed may also be associated with greater negative consequences compared to lower percentages.

The collection of weight suppression data is another area of inconsistency among previous empirical studies. Measurements of weight suppression often differ based on whether they rely on measured or self-reported weight, sometimes within the same study (e.g., weight suppression is calculated from self-reported highest weight and measured current weight; Berner et al., 2013; Herzog et al., 2010; Kleifield & Lowe, 1991; Lowe, Annunziato, et al., 2006; Lowe et al., 2007; Wildes & Marcus, 2012). Although previous research has identified self-report as a valid method of measurement for weight among both population-based and eating disorder samples (Swenne, Belfrage, Thurfjell, & Engstrom, 2005; Tamakoshi, Yatsuya, Kondo, Hirano, Hori, Yoshida, et al., 2003), recalled weights are subjective and may still be inaccurate. While using self-report data for both previous and current weights in calculating weight suppression may control for measurement error, using a combination of self-reported previous weight and measured current weight does not control for this error. Therefore, studies using a combination of self-reported previous weight and measured current weight may not accurately capture an individual’s true degree of weight suppression.

In addition to differences in how weight suppression data is collected, discrepancies in the analysis of weight suppression data (i.e., use of categorical or continuous weight suppression variables) may explain some inconsistencies in research findings. Previous studies are mixed in
their use of continuous (Berner et al., 2013; Butryn et al., 2006; Butryn et al., 2011; Carter et al., 2008; Dawkins et al., 2013; Herzog et al., 2010; Keel & Heatherton, 2010; Lowe, Annunziato, et al., 2006; Lowe, Davis, et al., 2006; Lowe et al., 2007; Lowe et al., 2011; Mitchell et al., 2011; Van Son et al., 2013; Wildes & Marcus, 2012; Zunker et al., 2011) and categorical weight suppression variables (Carter et al., 2008; French & Jeffrey, 1997; Kleifield & Lowe, 1988; Lowe, Annunziato, et al., 2006; Lowe & Kleifield, 1991; Wildes & Marcus, 2012; Zunker et al., 2011). Of these methods, using a categorical weight suppression variable is more likely to be problematic, given that the resulting range restriction could underestimate the effects of weight suppression (Carter et al., 2008). There are also inconsistencies with how individuals are assigned to groups (e.g., weight suppressors versus non-weight suppressors), with some studies using upper and lower quartiles (Carter et al., 2008; Kleifield & Lowe, 1991; Lowe & Kleifield, 1988) and others using median splits (Lowe, Annunziato, et al., 2006; Wildes & Marcus, 2012; Zunker et al., 2011).

Studies on weight suppression typically lack information on when the weight loss occurred, length of time between the weight loss episode and data collection, weight change since the weight loss, and whether the weight loss was intentional (French & Jeffrey, 1997). Additionally, some studies only investigated specific pathological behaviors (e.g., binge eating; Mitchell et al., 2011; Van Son et al., 2013). It is possible that weight suppression may influence the development and maintenance of the underlying pathology rather than specific behavioral symptoms. Given the discrepancy in some findings (i.e., frequency of binge eating; weight gain over time) among population-based and eating disorder samples (Carter et al, 2008; Dawkins et al., 2013; Lowe, Annunziato, et al., 2006; Mitchell et al., 2011; Zunker et al., 2011), the effects of weight suppression may differ for those with and without eating disorders. However, the
extent to which the effects of weight suppression differ between these groups and why these differences exist is currently unclear.

Previous research has been conducted with little consideration of existing models of eating pathology and has primarily examined the direct consequences of weight suppression without addressing predictors of weight suppression or the various mediators and moderators that may explain these identified relationships. Taken together, these limitations of existing research suggest the need to continue research examining the relationships among weight suppression and eating psychopathology and to consider new ways of conceptualizing weight suppression in the onset and maintenance of eating pathology. Existing models of eating disorder onset and maintenance lend insight to our current understanding of the factors that influence eating psychopathology, which may be useful in developing such conceptualizations.

**Existing Models of Eating Disorder Onset and Maintenance**

Despite evidence suggesting the involvement of weight suppression in eating pathology, none of the existing models of eating disorder development or maintenance consider the influence of weight suppression. Current research partially supports four separate models of eating psychopathology. The first of these, Stice’s dual-pathway model (Stice, 1994) posits that girls and women are exposed to sociocultural pressures to be thin from family, peers, and the media, which leads to the internalization of the thin ideal. Because the thin ideal promoted by Western society is unattainable for most women, the discrepancy between the high standards of the thin ideal and the individual’s actual shape and weight typically results in body dissatisfaction in individuals who internalize the thin ideal. In turn, body dissatisfaction leads to bulimic symptomatology through one of two mediating pathways: restrained eating or negative affect (Stice, 1994).
A number of studies have provided empirical support for the relationships contained within the dual-pathway model. Correlational and prospective relationships between thin-ideal internalization and body dissatisfaction are well-established (Cafri, Yamimya, Brannick, & Thompson, 2004; Stice, 2002; Stice, Shaw, & Nemeroff, 1998; Thompson & Stice, 2001). Research has also found that perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, restrained eating or dieting, and negative affect prospectively predict the onset of bulimic behaviors (Bradford & Petrie, 2008; Stice, 2001, 2002; Stice & Agras, 1998; Stice, Presnell, & Spangler, 2002). Additionally, body dissatisfaction, independently, and negative affect, in the presence of body dissatisfaction, significantly predict bulimic symptoms (Downey & Chang, 2007). Depressive symptoms, which include negative affect, have been found to predict the onset of bulimic pathology (Stice, Burton, & Shaw, 2004).

Vohs and her colleagues (1999) subsequently proposed an interactive model of the development of bulimic symptomatology. This model suggests that the three-way interaction of perfectionism, perceived weight status, and self-esteem predicts bulimic symptoms (Vohs, Bardone, Joiner, Abramson, & Heatherton, 1999). According to the model, bulimic symptoms arise when individuals who are high in perfectionism and consider themselves overweight also experience low self-esteem. The three-way interaction has been found to significantly predict increases in bulimic symptoms over the course of one year (Vohs et al., 1999). Additional research supporting this model has found that perfectionism and low self-esteem are significantly related to the exacerbation and maintenance of bulimic symptoms (Bardone, Vohs, Abramson, Heatherton, & Joiner, 2000; Bardone-Cone, Abramson, Vohs, Heatherton, & Joiner, 2006; Holm-Denoma, Gordon, Bardone-Cone, Vohs, Abramson, Heatherton, et al., 2005).
The third model, the transdiagnostic model, is a maintenance model of eating pathology that expands upon the earlier cognitive behavioral model proposed by the same authors (Fairburn, Cooper, & Shafran, 2003). The cognitive behavioral model posits that overvaluation of shape, weight, and control of eating behaviors triggers the individual to engage in weight control behaviors (i.e., strict dieting and food restriction). When these extreme behaviors cannot be maintained long-term, binge eating occurs. Binge eating is typically followed by compensatory behaviors (i.e., vomiting, laxative use, diuretic use, fasting, excessive exercise) and a return to strict weight control behaviors. The individual ultimately alternates between periods of strict dietary control and binge eating. The transdiagnostic model expands upon the cognitive behavioral model by incorporating four additional variables: interpersonal difficulties, mood intolerance, clinical perfectionism, and core low self-esteem. The individual may experience interpersonal difficulties and mood intolerance that contribute to binge eating episodes. Additionally, the individual may have an overall dysfunctional system of self-evaluation that includes clinical perfectionism and core low self-esteem. These characteristics contribute to dieting and weight control behavior (Fairburn et al., 2003). Although the transdiagnostic model has not been empirically evaluated in its entirety, some of its components have been examined. Specifically, perfectionism and low self-esteem have been found to be related to bulimic symptomatology (Engler, Crowther, Dalton, & Sanftner, 2006; Holm-Denoma et al., 2005; Shafran, Cooper, & Fairburn, 2002; Vohs et al., 1999).

Finally, the affect regulation model focuses on the function of binge eating. This model posits that binge episodes are triggered by negative emotions and that binge eating provides comfort and distraction in order to reduce negative affect (Hawkins & Clement, 1984). Over time, binge eating becomes a conditioned response to negative emotions that is maintained.
through negative reinforcement. Although binge eating is generally ineffective in reducing negative emotions (Haedt-Matt & Keel, 2011), substantial empirical support exists for the notion that increases in negative affect occur prior to binge episodes among individuals with BN and BED (e.g., Agras & Telch, 1998; Bruce & Agras, 1992; Chua, Touyz, & Hill, 2004; Mitchell, Hatsukami, Eckert, & Pyle, 1985; Davis & Jamieson, 2005; Vanderlinden, Grave, Fernandez, Vandereycken, Pieters, & Noorduin, 2004) and appear to contribute to the occurrence of this type of disordered eating.

Although existing models have been useful in conceptualizing and understanding the development and maintenance of eating pathology, they are not without fault. While there is some empirical support for each existing model of eating pathology, no model fully predicts the onset of disordered eating behaviors (Holm-Denoma et al., 2005; Stice et al., 1998). For example, the dual-pathway model only accounts for 23% of the variance in the prediction of bulimic symptomatology (Stice, 2001), suggesting the need for consideration of additional variables and explanatory relationships. There is also significant overlap across the four primary models. The sociocultural, transdiagnostic, and affect regulation models emphasize mood-related difficulties (i.e., negative affect, mood intolerance; Hawkins & Clement, 1984; Stice, 1994). Both the sociocultural and transdiagnostic models include dietary restraint and an imposed importance of meeting a certain standard for shape and weight (i.e., internalization of the thin ideal, overvaluation of shape and weight; Fairburn et al., 2003; Stice, 1994). The interactive and transdiagnostic models both consider the influence of perfectionism and self-esteem on the development of eating psychopathology (Fairburn et al., 2003; Vohs et al., 1999). Additionally, the perceived weight status variable in the interactive model appears to be conceptually similar to body dissatisfaction, which is also included in the sociocultural model.
(Stice, 1994; Vohs et al., 1999). The current models primarily examine the onset and maintenance of bulimic symptomatology; little consideration is given to the development and maintenance of AN. Only the transdiagnostic model considers anorexic symptoms, and even then only a brief discussion of potential relationships is provided (Fairburn et al., 2003).

Despite evidence for a prospective relationship between weight suppression and eating disorder symptomatology (e.g., Keel & Heatherton, 2010), no existing model considers weight suppression in the onset or maintenance of eating disorders. Moreover, only one published study has explored potential moderators of weight suppression and its relationship to eating pathology (Dawkins et al., 2013), no published studies have explored potential mediators of this relationship, and weight suppression has not yet been evaluated as part of a model of eating disorder onset or maintenance. These limitations of existing research point to the need to examine weight suppression in the context of an eating disorder model and to evaluate mediating and moderating relationships between weight suppression and eating disorder-related outcome variables.

**Proposed Model**

Several questions remain regarding the nature of weight suppression, its effects on eating disorder development and maintenance, and the antecedents and characteristics that might influence its impact. The proposed model aims to address both the limitations of prior research on weight suppression and the contributions of existing models of eating pathology. Previous research indicates that eating disorder pathology is multi-determined; however, weight suppression may be an important variable to examine in the causal pathway. The proposed model includes weight suppression as a causal variable in a pathway that may ultimately lead to
the manifestation of eating disorders and considers both mediating and moderating variables with respect to the relationship between weight suppression and eating psychopathology (Figure 1).

The existing literature on weight suppression and eating pathology appears to indicate a possible mediating effect of dieting and restraint on the relationship between weight suppression and eating disorder symptomatology. A number of dieting-related variables have been associated with weight suppression. Specifically, groups of weight suppressed individuals contain a higher percentage of current dieters compared to groups of non-weight suppressed individuals (Lowe & Kleifield, 1988; French & Jeffrey, 1997). Weight suppression is also positively associated with high intensity dieting (Van Son et al., 2013) and one might hypothesize that dieting frequency would be associated with higher weight suppression. Thus, dieting is likely an important variable to consider in constructing a model of eating pathology. Restraint, a similar, yet distinct, construct related to dieting, may also help to explain the relationship between weight suppression and its consequences. As previously explained, restraint has been prospectively associated with eating disorder symptoms (i.e., binge eating; Mitchell, Hatsukami, Pyle, & Eckert, 1986), which warrants inclusion in this model. Additionally, diet “boundaries,” or food rules, described by Herman and Polivy (1984), appear to play a role in the eating behaviors of some individuals. Thus, dieting, restraint, and food rules are considered in the proposed model as contributors to the latent construct of dietary restriction.

Only two studies thus far have evaluated moderators of the relationship between weight suppression and its outcomes (Butryn et al., 2011; Dawkins et al., 2013). One of these studies (Dawkins et al., 2013), which focused on treatment related variables and outcomes, found no significant moderating effects. These null findings may have been due to the selection of the moderating variables, which were largely comprised of variables related to individual and family
history (e.g., parental history of overweight, childhood body shape, pretreatment BMI). It may be that variables drawn from existing eating disorder theory better explain the relationship between weight suppression and eating disorder symptomatology. The other moderation study found that BMI moderated the relationship between weight suppression and frequency of binge eating (Butryn et al., 2011). Previous research that broadly assessed predictors of eating pathology also suggests that BMI may moderate relationships between eating disorder outcomes and their predictors (Stice, 1994). For this reason, BMI was included in the model as a moderator of the relationship between weight suppression and dietary restriction. Weight suppressed individuals may choose to employ dieting or weight loss efforts based on their BMI. In addition to BMI, proposed moderators in the model include emotion dysregulation, perfectionism, and impulsivity.

Existing research suggests that individuals with eating disorders are likely to experience difficulties in emotion regulation (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Harrison, Sullivan, Tchanturia, & Treasure, 2009; Whiteside, Chen, Neighbors, Hunter, Lo, & Larimer, 2007) and this risk appears to be transdiagnostic rather than associated with a specific eating disorder diagnosis (Brockmeyer, Skunde, Wu, Bresslein, Rudofsky, Herzog, et al., 2013; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012). Additionally, emotion dysregulation has been found to predict both disordered eating behaviors and severity of eating disorder symptoms (Cooper, O’Shea, Atkinson, & Wade, 2014). Emotion dysregulation, or mood intolerance, influences negative affect and depressive symptoms that have been associated with eating pathology in empirical research (Aldao, et al., 2010; Bradford & Petrie, 2008; Downey & Chang, 2007; Fairburn et al., 2003; Racine & Wildes, 2013; Stice, 2001, 2002; Stice & Agras, 1998; Stice, 1994; Stice et al., 1998) and affect or mood play a role in the dual-pathway (Stice, 1994),
transdiagnostic (Fairburn et al., 2003), and affect regulation (Hawkins & Clement, 1984) models. Individuals who experience mood intolerance or emotion dysregulation may lack appropriate skills to cope with negative affect or changes in mood and may therefore resort to more dysfunctional coping methods, including disordered eating behaviors. Thus, weight suppressed individuals who also experience emotion dysregulation may be more likely to develop eating psychopathology than those without emotion dysregulation.

Perfectionism is included in both the interactive model (Vohs et al., 1999) and the transdiagnostic model (Fairburn et al., 2003) and the research literature provides substantial evidence of a prospective relationship between perfectionism and symptoms of AN and BN (Bardone et al., 2000; Bardone-Cone et al., 2006; Bastiani, Rao, Weltzin, & Kaye, 1995; Engler, Crowther, Dalton, & Sanftner, 2006; Holm-Denoma et al., 2005; Halmi, Sunday, Strober, Kaplan, Woodside, Fichter, et al., 2000; Hewitt, Flett, & Ediger, 1995; Jones & Crowther, 2013; Shafran, Cooper, & Fairburn, 2002; Vohs et al., 1999). Additionally, perfectionism is associated with both the onset and maintenance of eating disorders (Holland, Bodell, & Keel, 2013). Perfectionism appears to be associated with both AN (Chang, Ivezaj, Downey, Kashima, & Morady, 2008; Halmi et al., 2000; Hewitt et al., 1995) and BN (Chang et al., 2008; Halmi et al., 2000; Hewitt et al., 1995; Lilenfeld, Stein, Bulik, Strober, Plotnicov, Pollice, et al., 2000) and is associated with increased likelihood of treatment dropout in treatment samples (Sutandar-Pinnock, Woodside, Carter, Olmsted, & Kaplan, 2003).

Although impulsivity has not previously been incorporated into an existing model of eating psychopathology, empirical evidence suggests that impulsivity is an important construct in the presentation of eating psychopathology, and it is implicated in the onset and maintenance of eating disorder symptoms. Previous research has found that individuals who binge eat report
greater impulsivity compared to controls (Kelly, Bulik, & Mazzeo, 2013), as do individuals with an eating disorder diagnosis (Boisseau, Thompson-Brenner, Caldwell-Harris, Pratt, Farchione, & Barlow, 2012; Vandereycken & Van Houdenhove, 1996; Waxman, 2009). The presence of impulsive behaviors in individuals with BN is associated with greater bulimic symptomatology, a greater lack of interoceptive awareness, earlier onset of bulimic symptomatology, and a longer duration of illness (Favaro & Santonastoso, 1998; Favaro, Zanetti, Tenconi, Degortes, Ronzan, Veronese, et al., 2005; Wiederman & Pryor, 1996). Research assessing coping strategies among individuals with BN has found that impulsive individuals with BN employ more maladaptive coping strategies, suggesting that impulsivity may function to maintain bulimic symptomatology (Nagata, Matsuyama, Kiriike, Iketani, & Oshima, 2000). High rates of comorbidity between BN and impulse control disorders (Fernandez-Aranda, Jimenez-Murcia, Alvarez-Moya, Granero, Vallejo, & Bulik, 2006) suggest an underlying risk-factor that influences the presentation of both types of disorders. While most research examining impulsivity in eating disorder samples has been conducted using samples of individuals with BN, impulsivity is also positively associated with eating disorder symptoms among individuals with binge-purge AN (Hoffman, Gagne, Thornton, Klump, Brandt, Crawford, et al., 2012; Vandereycken & Van Houdenhove, 1996).

Given that emotion dysregulation (Gratz & Roemer, 2004), perfectionism (Frost, Marten, Lahart, & Rosenblate, 1990; Hewitt et al., 1993), and impulsivity (Cassin & von Ranson, 2005) have been conceptualized as trait variables, they likely influence the nature of the relationship between weight suppression, dietary restriction, and eating pathology rather than causally explaining this relationship. Thus, they were included in the model as moderating variables. As previously conceptualized in the literature, emotion dysregulation was hypothesized to strengthen the relationship between dietary restriction and eating disorder symptomatology.
Individuals who engage in dietary restriction and experience emotion dysregulation may binge eat when dietary rules are violated and they lack effective and adaptive coping skills to manage their affect.

Perfectionism, which is largely conceptualized as a risk factor (Bardone-Cone, Wonderlich, Frost, Bulik, Mitchell, Uppala, et al., 2007), was included as a moderator of the relationship between body dissatisfaction and weight suppression. Given that previous research has identified perfectionism as a moderator of the relationship between body dissatisfaction and disordered eating (Welch, Miller, Ghaderi, & Vaillancourt, 2009), perfectionism was included in the proposed model as a moderator of the relationship between body dissatisfaction and weight suppression. Body dissatisfied individuals who are perfectionistic may be more likely to suppress their weight or may suppress their weight to a greater degree than individuals who are not perfectionistic.

Impulsivity is primarily associated with an exacerbation of eating disorder symptoms (Favaro & Santonastoso, 1998; Favaro et al., 2005; Wiederman & Pryor, 1996) and impulsive individuals with an eating disorder diagnosis may represent a sub-population of individuals with eating disorders (Wonderlich, Crosby, Joiner, Peterson, Bardone-Cone, Klein, et al., 2005). Because not all individuals with an eating disorder diagnosis display impulsive traits or behaviors and impulsivity is associated with an exacerbation of symptoms, it is conceptualized as a moderating variable in this model. Impulsivity was proposed to moderate the relationship between dietary restriction and eating disorder symptomatology such that individuals who engage in dietary restriction and are high in impulsivity experience greater eating psychopathology compared to individuals who are not high in impulsivity.
In addition to examining potential mediating and moderating variables of the relationship between weight suppression and eating psychopathology, several variables will be examined as potential correlates of weight suppression. One factor that appears to be important in the development of weight suppression is previous weight status and, in particular, a prior weight classification of overweight or obese (Keel & Heatherton, 2010). A substantial percentage of individuals diagnosed with full-threshold or sub-clinical BN have a history of being overweight or obese (Garner & Fairburn, 1988) and typically have higher premorbid weights compared to their peers (Butryn et al., 2011; Fairburn, Welch, Doll, Davies, & O’Conner, 1997; Swenne, 2005). Individuals with BN who were premorbidly overweight or obese are more likely to have a family history of overweight or obese (i.e., one or both parents is overweight or obese; Fairburn et al., 1997), suggesting that family weight status may important to consider among premorbidly overweight or obese individuals with BN.

Previous research has established a relationship between history of dieting and weight suppression that warrants the inclusion of dieting history in the proposed model. Specifically, a history of dieting is positively associated with weight suppression (Mitchell et al., 2011). Individuals high in weight suppression who also have a history of dieting gain more weight than such individuals who do not have a history of dieting (Lowe, Annunziato, et al., 2006), which suggests that dieting history can influence both the presence of weight suppression and the degree of influence that weight suppression has on outcome variables.

Although several studies have failed to find a significant relationship between weight suppression and body dissatisfaction (Berner et al., 2013; Van Son et al., 2013), body dissatisfaction has been consistently shown to predict both onset and maintenance of eating disorder symptomatology (Bradford & Petrie, 2008; Downey & Chang, 2007; Jones & Crowther,
Given the discrepant findings, additional research on the relationship between body dissatisfaction and weight suppression appears warranted.

**Hypotheses**

The purpose of this research was to examine the impact of weight suppression on eating disorder development and maintenance by testing pathways of the proposed model prospectively. For the purposes of this study, weight suppression was defined as the difference between an individual’s highest historical weight at her current height and her current weight. It was hypothesized that 1) weight suppression would significantly predict eating psychopathology (Figure 2), 2) the relationship between weight suppression and eating psychopathology would be mediated by dietary restriction (Figure 2), 3) the relationship between weight suppression and dietary restriction would be moderated by BMI (Figure 2), and 4) the relationship between dietary restriction and eating psychopathology would be moderated by impulsivity and emotion dysregulation (Figure 2). In addition to these primary hypotheses, this research also investigated two secondary hypotheses to further our understanding of weight suppression: 5) weight history, dieting history, and body dissatisfaction would be associated with weight suppression and 6) perfectionism would moderate the relationship between body dissatisfaction and weight suppression (Figure 3).
Method

Participants

Participants were 309 female undergraduate students who volunteered for study participation using the SONA website. Participants had a mean age of 19.55 (SD = 2.03) and a mean BMI of 24.35 (SD = 5.76). A majority of participants identified as Caucasian (87.4%), with the remaining participants identifying as African-American (2.6%), Asian (2.6%), Hispanic (2.6%), or another race or ethnicity (4.7%). Nearly half (44.5%) of participants were freshman, 18.3% were sophomores, 16.2% were juniors, and 20.9% were seniors.

Measures

Demographic questionnaire. Participants were asked to report their age, class year, race/ethnicity, and current height and weight. Current height and weight were used to calculate BMI using the following formula: \[\text{weight in pounds}/(\text{height in inches})^2\times 703\] (Center for Disease Control, 2011).

Dieting history, weight suppression, weight rebound, and current dieting. The Dieting and Weight History Questionnaire (DWHQ; Witt, Katterman, & Lowe, 2013) is a 16-item self-report questionnaire that measures each of the factors identified in the three-factor model of dieting: dieting history, current dieting, and weight suppression (Lowe, 1993). Participants provide information on current and past weight, reasons for weight loss, and stability of their present weight. They also provide information about current and past dieting, including whether they are currently dieting, length of current diet, motivation for dieting, age of first diet, number of times they have been on a diet, and weight lost during current and previous diets.
Eleven of these items are standard items used to assess dieting history, current dieting, and weight suppression in research studies and five items were included to allow researchers to gain knowledge of these constructs and expand upon previous research (Witt et al., 2013). Weight suppression was calculated using this measure by subtracting the individual’s current weight from their highest historical weight since reaching their current height. Weight rebound was calculated using this measure by subtracting the individual’s lowest historical weight at their current height from the individual’s current weight.

**Weight history.** Two questions were created for the purpose of this study to assess the weight history of participants and their parents. The first of these questions asked whether the participant was overweight or obese as a child or adolescent. The second of these questions asked whether the participant’s parents were overweight or obese when the participant was a child.

**Body dissatisfaction.** The Body Shape Questionnaire (BSQ; Cooper, Taylor, Cooper, & Fairburn, 1987) is a 34-item self-report questionnaire that measures an individual’s feelings of body dissatisfaction over the past four weeks. Participants respond to items using a 6-point Likert scale ranging from “never” to “always,” with higher scores indicating greater body dissatisfaction. The BSQ shows good psychometric properties, including internal consistency and test-retest reliability (Rosen, Jones, Ramirez, & Waxman, 1996). The BSQ is significantly correlated with similar measures that assess body dissatisfaction, indicating good concurrent validity (Cooper et al., 1987). It also demonstrates satisfactory discriminant validity, as individuals low in shape and weight concern score lower on this measure as compared to women who independently indicated concerns with shape and weight. Additionally, women with
symptoms of bulimia nervosa (BN) score higher on the BSQ than women without BN symptoms (Cooper et al., 1987). Internal consistency in this sample was excellent (α = .97).

**Perfectionism.** The Frost Multidimensional Perfectionism Scale (FMPS; Frost et al., 1990) is a 35-item self-report measure that assesses six domains of perfectionism. The FMPS contains six subscales: Concern over Mistakes, Personal Standards, Parental Expectations, Parental Criticism, Doubts about Actions, and Organization. The total score of the FMPS was used for this study. Participants respond to items using a 5-point Likert scale ranging from “strongly disagree” to “strongly agree,” with higher scores indicating greater perfectionism. The overall scale demonstrates good psychometric properties, including internal consistency (α = .90) and concurrent validity (Frost et al., 1990). Internal consistency in this sample was good (α = .92).

**Restriction and food rules.** The Multifactorial Assessment of Eating Disorder Symptoms (MAEDS; Anderson, Williamson, Duchmann, Gleaves, & Barbin, 1999) is a 56-item self-report measure that assesses six symptom clusters associated with eating disorders. These six clusters are measured using six subscales: Depression, Binge Eating, Purgative Behavior, Fear of Fatness, Restrictive Eating, and Avoidance of Forbidden Foods. Participants respond to items using a 7-point Likert scale ranging from “never” to “always,” with higher scores indicating greater symptomatology. The 9-item Restrictive Eating and 10-item Avoidance of Forbidden Foods subscales were selected for use in this study. The Restrictive Eating and Avoidance of Forbidden Foods subscales demonstrate good psychometric properties, including internal consistency (α = .85-87) and test-retest reliability (r = .94-.96), and both subscales are correlated with related measures (Anderson et al., 1999). Internal consistency in this sample was
good for both the Restrictive Eating ($\alpha = .91$) and Avoidance of Forbidden Foods ($\alpha = .90$) subscales.

**Impulsivity.** The UPPS Impulsive Behavior Scale (UPPS; Whiteside & Lynam, 2001) is a 46-item self-report measure assessing four facets of the personality construct of impulsivity. The UPPS contains four subscales that assess separate facets of impulsivity: Premeditation, Urgency, Sensation Seeking, and Perseverance. The 12-item Urgency subscale was selected for use in this study. Participants respond to items using a 4-point Likert scale with higher scores indicating greater impulsivity. The UPPS demonstrates good psychometric properties, including internal consistency and divergent, external, and concurrent validity (Whiteside & Lynam, 2001; Whiteside, Lynam, Miller, & Reynolds, 2005). Internal consistency for the Urgency subscale in this sample was good ($\alpha = .89$).

**Emotion dysregulation.** The Difficulties in Emotion Regulation Scale (DERS; Gratz, & Roemer, 2004) is a 36-item self-report measure that assesses emotion dysregulation. The DERS contains six subscales: Nonacceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behavior, Impulse Control Difficulties, Lack of Emotional Awareness, Limited Access to Emotion Regulation Strategies, and Lack of Emotional Clarity. The total score of the DERS was used for this study. Participants respond to items using a 5-point Likert scale from “almost never” to “almost always,” with higher scores indicating greater difficulties with regulation emotion. The DERS demonstrates good psychometric properties, including internal consistency ($\alpha = .93$) and test-retest reliability ($r = .88$). The DERS also demonstrates construct and predictive validity (Gratz & Roemer, 2004). Internal consistency in this sample was excellent ($\alpha = .96$).
**Eating psychopathology.** The Eating Disorder Diagnostic Scale (EDDS; Stice, Telch, & Rizvi, 2000) is a 22-item self-report measure that assesses eating disorder symptomatology. Participants report feelings of fatness, influence of shape and weight on self-evaluation, symptoms of binge eating episodes, height and weight, missed menstrual periods, and frequency of binge eating episodes and compensatory behaviors. Results of the EDDS can be used to diagnose anorexia nervosa, bulimia nervosa, and binge eating disorder. The symptom composite score was used for this study. The symptom composite scores of the EDDS demonstrate good internal consistency (α = .89) and test-retest reliability (r = .87). Eating disorder diagnoses derived from the EDDS have high rates of agreement with diagnoses derived from structured interviews (93-99%) and the EDDS correlates with related measures of eating psychopathology (Stice et al., 2000).

**Procedure**

Participants volunteered for study participation through the SONA website. Data collection took place over three time points spaced four weeks apart. After signing up to participate in the study, this author contacted participants via email to provide login information for the online survey. Following informed consent, participants used an online survey to complete an initial battery of self-report questionnaires, including the DWHQ, BSQ, FMPS, the Restrictive Eating and Food Rules subscales of the MEADS, the Urgency subscale of the UPPS, DERS, EDDS, and current height and weight. Participants were contacted four weeks after they completed the initial online survey to complete the second online survey. They were reminded of their login information and instructed to complete the second survey within seven days after receiving the email. Participants received a reminder email if they had not completed the survey after three days and received a second reminder email on the fifth day if the survey had not been
completed at that time. If participants did not complete the survey within seven days, they were no longer able to participate in the second survey. Participants were also contacted four weeks after they completed the second online survey to complete the third online survey. They again had seven days to complete the third questionnaire battery and received similar reminders if they had not completed the survey within 3-5 days. If participants did not complete the survey within seven days, they were no longer able to participate in the third survey. The second and third online surveys consisted of the following self-report questionnaires: DWHQ, BSQ, the Restrictive Eating and Food Rules subscales of the MEADS, EDDS, and current height and weight. Participants received three research points toward their psychology research requirements for completing the first online survey and two research points for each subsequent online survey they complete, for a total of seven possible research points. In addition, participants who completed the second online survey received two entries into a raffle for one of six gift cards worth $50 each and participants who completed the third online survey received four entries into the raffle.
Results

Data Quality

Of the 309 participants who signed up and received credit for participating in the study, 13 individuals provided multiple responses under the same participant number for one of the three time points. These duplicate responses were removed from the dataset. An additional 18 participants did not respond to any items on one or more questionnaires at Time 1, 11 of whom provided only their participant number and no other data. Thus, a total of 278 participants responded to all questionnaires at Time 1. Of the participants responding to all questionnaires at Time 1, 30 provided no data at Time 2, and 3 did not complete all questionnaires at Time 2, resulting in 245 participants who responded to all questionnaires at Time 1 and Time 2. Of the 245 participants who responded to all questionnaires at Time 1 and Time 2, 41 participants did not provide data at Time 3. Thus, a total of 204 participants responded to all questionnaires at all three time points.

Independent-samples t-tests were conducted to determine whether there were Time 1 differences between individuals who responded to all questionnaires at all three time points and those who did not. Results indicated that there were no significant differences between groups in age, BMI, or scores on the BSQ, FMPS, the Avoidance of Forbidden Foods and Restrictive Eating subscales of the MAEDS, the Urgency subscale of the UPPS, DERS, or EDDS at Time 1 (ps > .05; Table 1).
**Missing Data**

A total of 82 items out of 32,864 total items (0.25%) at Time 1 were missing. A total of 28 items out of 15,300 total items (0.18%) at Time 2 were missing. A total of 26 items out of 15,300 total items (0.17%) at Time 3 were missing. Due to the way in which the EDDS is scored, missing data on this measure could not be imputed. Therefore, it was not possible to calculate valid scores on the EDDS for participants who did not respond to all items on this measure. Participants who failed to provide responses to all items on the EDDS at Time 1 (N = 5), Time 2 (N = 5), or Time 3 (N = 3) were removed from the study, resulting in a total of 191 participants whose data were used in analyses. Given the small amount of total missing data (0.21%), mean imputation was selected as a conservative method of handling missing data on the remaining scales (Tabachnick & Fidell, 2013).

**Data Preparation**

In the proposed model, dietary restriction represents a latent construct comprised of the following variables: current dieting (CD), as measured by the DWHQ; food rules, as measured by the Avoidance of Forbidden Foods (AFF) subscale of the MAEDS; and restriction, as measured by the Restrictive Eating (RE) subscale of the MAEDS. AFF and RE were standardized to account for the fact that both of these subscales originate from the same measure and to account for a difference in metric compared to CD. In order to create a composite variable to represent dietary restriction, values for CD, AFF, and RE were equally weighted and summed. This composite variable was used in all analyses that include dietary restriction.

All independent variables were assessed for normality and determined to be normally distributed (Table 2), with the exception of Time 1 weight suppression. A square root transformation was used on the Time 1 weight suppression variable to normalize the distribution.
of this variable. The transformed Time 1 weight suppression variable, which was normally
distributed, was used in all relevant analyses. Any outliers present in these independent variables
were examined and retained. All dependent variables were found to be normally distributed
(Table 2) and free from outliers.

Bivariate correlational analyses were conducted to examine the association between the
independent and dependent variables. Large positive correlations exist between the Time 1 and
Time 2 dietary restriction and Time 1, Time 2, and Time 3 EDDS scores ($r_s = .582-.611$, $p$s $<
.01$). BSQ scores were also strongly and positively correlated with dietary restriction and EDDS
scores at all time points ($r_s = .569-.801$, $p$s $< .01$). All other correlations between the
independent variables used in the regression analyses were near or below .30 and were
determined to be at an acceptable level (Table 3).

According to Green (1991), 163 participants would be required given four predictors and
a medium effect size. The highest number of predictors in any regression analysis was four: one
control variable and three predictor variables. A medium effect size was assumed given the
limited research on the relationships between these variables (Cohen, 2003). Thus, the sample
size of 191 was considered sufficient to detect a medium effect size given the number of
predictors used in the regression analyses.

**Correlational Analyses**

Hypothesis 5 states that weight history, dieting history, and body dissatisfaction would be
associated with weight suppression. Bivariate correlations were conducted to examine the
relationships among Time 1 weight history, Time 1 dieting history, Time 1 body dissatisfaction,
and Time 1 weight suppression. Weight suppression was significantly positively correlated with
dieting history ($r = .174$, $p = .028$) and weight history ($r = .224$, $p = .002$), but was not
significantly correlated with body dissatisfaction ($r = .080$, $p = .274$). Results of these analyses are displayed in Table 3.

**Mediation Analyses**

Hypothesis 1 states that weight suppression at Time 1 would significantly predict eating psychopathology at Time 3. Hierarchical linear regression was employed to examine Hypothesis 1. For this analysis, Time 3 EDDS scores were entered as the dependent variable. Time 1 EDDS scores were entered in the first step as a control variable in order to account for the impact of baseline eating psychopathology on Time 3 EDDS scores. Time 1 weight suppression was entered as a predictor in step two. A nonsignificant coefficient for Time 1 weight suppression indicates that weight suppression was not a significant predictor of eating psychopathology ($\beta = .002$, SE $B = .325$, $t(190) = .053$, $p = .958$; Table 4).

Hypothesis 2 states that the relationship between weight suppression at Time 1 and eating psychopathology at Time 3 would be mediated by dietary restriction at Time 2. A bootstrapping procedure offering a direct test of mediation (Hayes, 2013; Preacher & Hayes, 2004, 2008) was used to examine Hypothesis 2. This bootstrapping procedure computes the mediating effect in each of a large number of samples ($n = 5,000$) from the original data and averages these mediating effects over the $n$ samples. This procedure overcomes limitations of the Baron and Kenny (1986) approach to mediation by yielding more accurate results that are less influenced by sample size (Hayes, 2013; Preacher & Hayes, 2004, 2008).

A macro developed by Hayes (2011) was used to test mediation. The Hayes (2011) macro includes a confidence interval to account for error variance. There is evidence of an indirect effect when this confidence interval does not contain zero. For this analysis, Time 1 weight suppression was entered as the predictor, Time 3 EDDS scores were entered as the
dependent variable, and Time 2 dietary restriction scores were entered as the mediator. Time 1 EDDS scores were also entered as a control variable. The model was significant, $F(3, 182) = 161.44, p < .001$, accounting for 72.7% of the variance in eating disorder symptomatology. The confidence interval indicated there was evidence of mediation ($CI_{90\%} = .02 \text{ - } .40$). These results indicate that dietary restriction mediates the relationship between weight suppression and eating disorder symptomatology.

**Moderation Analyses**

Hypothesis 3 states that the relationship between weight suppression and dietary restriction would be moderated by BMI. Hierarchical linear regression was used to evaluate Hypothesis 3 and the analysis was based on the recommendations of Baron and Kenny (1986). According to Baron and Kenny (1986), moderation should be tested using a model in which the independent variable, the moderator, and the interaction, or product, of the independent variable and moderator are entered as predictors of the dependent variable. Moderation is supported if the interaction term is significant (Baron & Kenny, 1986).

Prior to conducting analyses for Hypothesis 3, the Time 1 weight suppression and BMI variables were centered in order to reduce multicollinearity and an interaction term was created using the product of Time 1 weight suppression and Time 1 BMI. For this equation, Time 1 dietary restriction was entered in the first step as a control variable to account for the impact of baseline dietary restriction on Time 2 dietary restriction. Time 1 weight suppression and Time 1 BMI were entered in the second step as predictors, the interaction between Time 1 weight suppression and Time 1 BMI was entered in the third step as a predictor, and Time 2 dietary restriction was entered as the dependent variable. Results indicated that the main effects for weight suppression ($\beta = .148, SE B = .156, t(181) = .854, p = .394$) and BMI ($\beta = .007, SE B = ..$)
and the interaction term between weight suppression and BMI \((\beta = -.165, \text{SE } B = .006, t(181) = -.918, p = .360)\) were nonsignificant (Table 4). Thus, BMI did not significantly moderate the relationship between weight suppression and dietary restriction.

Hypothesis 4 states that the relationship between dietary restriction and eating psychopathology would be moderated by impulsivity and emotion dysregulation. As in Hypothesis 3, moderation analyses were based on the recommendations of Baron and Kenny (1986). Two separate hierarchical linear regression models were used to test this hypothesis. For both models, centered predictors were used to calculate interaction terms in order to reduce multicollinearity and centered predictors were used in the equations.

For the first model, Time 2 EDDS scores were entered as a control variable in the first step to account for the impact of Time 2 eating psychopathology on Time 3 EDDS scores, Time 2 dietary restriction and Time 1 UPPS Urgency scores were entered as predictors in the second step, the interaction term between Time 2 dietary restriction and Time 1 UPPS Urgency scores was entered as a predictor in the third step, and Time 3 EDDS scores were entered as the dependent variable. For the second model, Time 2 EDDS scores were entered as a control variable in the first step to account for the impact of Time 2 eating psychopathology on Time 3 EDDS scores, Time 2 dietary restriction and Time 1 DERS scores were entered as predictors in the second step, the interaction term between Time 2 dietary restriction and Time 1 DERS scores was entered as a predictor in the third step, and Time 3 EDDS scores were entered as the dependent variable.

For the first model, a significant coefficient for Time 2 dietary restriction \((\beta = .106, \text{SE } B = .386, t(185) = 2.512, p = .013)\) indicated a main effect for dietary restriction (Table 4). Nonsignificant coefficients for Time 1 UPPS Urgency scores \((\beta = .107, \text{SE } B = .106, t(185) = .017, t(181) = .106, p = .916)\) and the interaction term between weight suppression and BMI \((\beta = -.165, \text{SE } B = .006, t(181) = -.918, p = .360)\) were nonsignificant (Table 4). Thus, BMI did not significantly moderate the relationship between weight suppression and dietary restriction.

For the second model, a significant coefficient for Time 2 dietary restriction \((\beta = .106, \text{SE } B = .386, t(185) = 2.512, p = .013)\) indicated a main effect for dietary restriction (Table 4). Nonsignificant coefficients for Time 1 UPPS Urgency scores \((\beta = .107, \text{SE } B = .106, t(185) = .017, t(181) = .106, p = .916)\) and the interaction term between weight suppression and BMI \((\beta = -.165, \text{SE } B = .006, t(181) = -.918, p = .360)\) were nonsignificant (Table 4). Thus, BMI did not significantly moderate the relationship between weight suppression and dietary restriction.

Hypothesis 4 states that the relationship between dietary restriction and eating psychopathology would be moderated by impulsivity and emotion dysregulation. As in Hypothesis 3, moderation analyses were based on the recommendations of Baron and Kenny (1986). Two separate hierarchical linear regression models were used to test this hypothesis. For both models, centered predictors were used to calculate interaction terms in order to reduce multicollinearity and centered predictors were used in the equations.

For the first model, Time 2 EDDS scores were entered as a control variable in the first step to account for the impact of Time 2 eating psychopathology on Time 3 EDDS scores, Time 2 dietary restriction and Time 1 UPPS Urgency scores were entered as predictors in the second step, the interaction term between Time 2 dietary restriction and Time 1 UPPS Urgency scores was entered as a predictor in the third step, and Time 3 EDDS scores were entered as the dependent variable. For the second model, Time 2 EDDS scores were entered as a control variable in the first step to account for the impact of Time 2 eating psychopathology on Time 3 EDDS scores, Time 2 dietary restriction and Time 1 DERS scores were entered as predictors in the second step, the interaction term between Time 2 dietary restriction and Time 1 DERS scores was entered as a predictor in the third step, and Time 3 EDDS scores were entered as the dependent variable.

For the first model, a significant coefficient for Time 2 dietary restriction \((\beta = .106, \text{SE } B = .386, t(185) = 2.512, p = .013)\) indicated a main effect for dietary restriction (Table 4). Nonsignificant coefficients for Time 1 UPPS Urgency scores \((\beta = .107, \text{SE } B = .106, t(185) = .017, t(181) = .106, p = .916)\) and the interaction term between weight suppression and BMI \((\beta = -.165, \text{SE } B = .006, t(181) = -.918, p = .360)\) were nonsignificant (Table 4). Thus, BMI did not significantly moderate the relationship between weight suppression and dietary restriction.
1.926, \( p = .056 \)) and the interaction term between Time 2 dietary restriction and Time 1 UPPS Urgency scores (\( \beta = -.047, \ SE \ B = .267, \ t(185) = -.924, \ p = .357 \)) indicate there is no significant main effect for impulsivity and there are no moderating effects (Table 4). These results indicate that dietary restriction is a significant predictor of eating psychopathology such that greater dietary restriction predicts greater eating psychopathology, but impulsivity is not a significant predictor of eating psychopathology and impulsivity does not moderate the relationship between dietary restriction and eating psychopathology.

For the second model, a significant coefficient for Time 2 dietary restriction (\( \beta = .104, \ SE \ B = .392, \ t(185) = 2.423, \ p = .016 \)) indicated a main effect for dietary restriction (Table 4). Nonsignificant coefficients for Time 1 DERS scores (\( \beta = .040, \ SE \ B = .031, \ t(185) = .693, \ p = .489 \)) and the interaction term between Time 2 dietary restriction and Time 1 DERS scores (\( \beta = -.019, \ SE \ B = .276, \ t(185) = -.339, \ p = .735 \)) indicate there is no main effect for emotion dysregulation and there are no moderating effects (Table 4). These results indicate that dietary restriction is a significant predictor of eating psychopathology such that greater dietary restriction predicts greater eating psychopathology, but emotion dysregulation is not a significant predictor of eating psychopathology and emotion dysregulation does not moderate the relationship between dietary restriction and eating psychopathology.

Finally, hypothesis 6 states that perfectionism would moderate the relationship between body dissatisfaction and weight suppression. As in Hypotheses 3 and 4, moderation analyses were based on the recommendations of Baron and Kenny (1986) and hierarchical linear regression was used to test this hypothesis. Predictor variables were centered prior to conducting regression analyses in order to reduce multicollinearity. Time 1 BSQ scores and Time 1 FMPS scores were entered as predictors in the first step, the interaction term between Time 1 BSQ
scores and Time 1 FMPS scores was entered as a predictor in the second step, and Time 1 weight suppression was entered as the dependent variable. The coefficients for Time 1 BSQ ($\beta = .092$, SE B = .136, $t(190) = 1.180$, $p = .239$) and Time 1 FMPS scores ($\beta = -.009$, SE B = .146, $t(190) = -.113$, $p = .910$) were nonsignificant. A nonsignificant coefficient for the interaction term between Time 1 BSQ and Time 1 FMPS ($\beta = -.043$ SE B = .112, $t(190) = -.538$, $p = .592$) indicates there are no moderating effects (Table 4). These results indicate that body dissatisfaction and perfectionism are not significant predictors of weight suppression, nor does perfectionism moderate the relationship between body dissatisfaction and weight suppression.

**Post Hoc Analyses**

Given the lack of statistical support for the majority of the hypotheses, additional analyses were conducted to further explore the data. First, the change in EDDS scores over time were examined using paired samples t-tests. Results indicated a significant difference between Time 1 EDDS scores and Time 2 EDDS scores, $t(190) = 3.631$, $p < .001$, with higher EDDS scores at Time 1 ($M = 24.50$, $SD = 14.152$) compared to Time 2 ($M = 22.40$, $SD = 14.361$). There was no significant difference between EDDS scores at Time 2 and Time 3, $t(190) = 1.647$, $p = .101$. Overall, there was a significant difference between Time 1 EDDS scores and Time 3 EDDS scores, $t(190) = 5.033$, $p < .001$, with higher EDDS scores at Time 1 ($M = 24.50$, $SD = 14.152$) compared to Time 3 ($M = 21.57$, $SD = 14.234$).

Second, the relationship between Time 1 weight suppression and Time 1 length of weight suppression was explored. Length of weight suppression was defined as the number of months the individual’s current weight has been maintained. Length was rounded down to the nearest month in order to use a more conservative estimate. Bivariate correlation analysis indicated that Time 1 weight suppression was significantly negatively correlated with Time 1 length of weight suppression.
suppression \((r = -.153, p = .036)\) such that greater weight suppression was associated with a shorter length of weight suppression.

A hierarchical linear regression was then conducted to investigate whether length of weight suppression moderated the relationship between weight suppression and eating psychopathology such that individuals who were higher in weight suppression and had maintained their suppressed weight for longer periods of time would experience greater eating psychopathology. Time 1 EDDS scores were entered as a control variable in the first step in order to account for the impact of baseline eating psychopathology on the dependent variable, Time 1 weight suppression and Time 1 length of weight suppression were entered as predictors in the second step, the interaction term between Time 1 weight suppression and Time 1 length of weight suppression was entered as a predictor in the third step, and Time 3 EDDS scores were entered as the dependent variable. Results indicated that there were no main effects for weight suppression \((\beta = -.137, \text{SE } B = .493, t(187) = -.311, p = .756)\) or length of weight suppression \((\beta = -.021, \text{SE } B = .074, t(187) = -.021, p = .773)\) and length of weight suppression did not significantly moderate the relationship between weight suppression and eating psychopathology \((\beta = .015, \text{SE } B = .030, t(187) = .493, p = .622; \text{Table 5}).\)

Finally, the relationship between weight suppression and weight rebound was explored. Weight rebound was defined as the difference between an individual’s current weight and their lowest historical weight at their current height. Because weight rebound was not normally distributed \((M = 16.613, SD = 18.296, \text{Skewness } = 3.002, \text{Kurtosis } = 13.495)\), a square root transformation was used to normalize the distribution of this variable. The transformed weight rebound variable, which was normally distributed \((M = 16.613, SD = 18.296, \text{Skewness } = 3.002, \text{Kurtosis } = 13.495)\), was used in all subsequent analyses. Bivariate correlation analysis indicated
that weight suppression was significantly correlated with weight rebound ($r = -.169$, $p = .019$) such that greater weight suppression was associated with lower weight rebound.

A hierarchical linear regression was then conducted to investigate whether weight rebound moderated the relationship between weight suppression and eating psychopathology such that individuals with lower weight suppression and greater weight rebound would experience greater eating psychopathology. Time 1 EDDS scores were entered as a control variable in the first step to account for the impact of baseline eating psychopathology on the dependent variable, Time 1 weight suppression and Time 1 weight rebound were entered as predictors in the first step, the interaction term between Time 1 weight suppression and Time 1 weight suppression was entered as a predictor in the second step, and Time 3 EDDS scores were entered as the dependent variable. Results indicated that there were no main effects for weight suppression ($\beta = .232$, SE $B = .656$, $t(190) = .355$, $p = .723$) or weight rebound ($\beta = .824$, SE $B = .456$, $t(190) = 1.808$, $p = .072$), and weight rebound did not significantly moderate the relationship between weight suppression and eating psychopathology ($\beta = -.011$, SE $B = .160$, $t(190) = -.006$, $p = .945$; Table 5).
Discussion

This research sought to examine the predictive relationship between weight suppression and eating psychopathology by empirically evaluating portions of a proposed model of the development of and change in eating psychopathology. Results of this study suggest that dietary restriction is a proximal predictor of eating psychopathology and mediates the prospective relationship between weight suppression and eating psychopathology. Weight suppression does not directly predict dietary restriction or eating psychopathology over time and there were no significant moderating effects on the relationship between weight suppression and dietary restriction or the relationship between dietary restriction and eating psychopathology. Body dissatisfaction does not appear to be related to weight suppression, nor does perfectionism moderate the relationship between body dissatisfaction and weight suppression. However, higher weight suppression is associated with a history of overweight status and a history of dieting behavior.

Weight Suppression, Dietary Restriction, and Eating Psychopathology

As hypothesized, dietary restriction not only predicts eating psychopathology longitudinally, but also mediates the relationship between weight suppression and eating psychopathology among female college students. These results suggest that after losing a significant amount of weight and sustaining this weight loss for several months, weight suppressed individuals may find it difficult to continue to maintain their weight or lose additional weight. When this occurs, these individuals may experience a renewed or more intense focus on dietary restriction. Participants in this research, who had an average weight suppression of nine pounds and, on
average, maintained their suppressed weight for one year, may have experienced such an increase in focus on dietary restriction. When dietary restriction is unsuccessful or they desire to lose even greater amounts weight, the individual may begin to engage in disordered eating behaviors, such as fasting, binging, purging, and engaging in excessive exercise.

The original composite variable of dietary restriction, which incorporates several components of dieting behaviors and food restriction, was developed and operationalized for use in this study. While components of this variable, including dietary restraint (e.g., Lowe et al., 2007; Lowe, Gleaves, & Murphy-Eberenze, 1998; Mitchell et al., 1986; Presnell & Stice, 2003; Stice, 2002) and food rules (Brown, Parman, Rudat, & Craighead, 2012; Eiber, Mirabel-Sarron, & Urdapilleta, 2005; Herman & Polivy, 1984; Mahes, Brownley, Mo, & Bulik, 2009) have previously been identified in the literature as being associated with eating psychopathology, this is the first study to incorporate multiple facets of dieting and restriction into one latent variable that functions as a useful predictor of eating psychopathology. The development of the dietary restriction variable allowed for the exploration of the relationship between weight suppression, dietary restriction, and eating psychopathology in a novel way.

The bivariate correlational findings related to weight suppression and dietary restriction replicate previous cross-sectional and laboratory research that has found a relationship between weight suppression and current dieting (Lowe & Kleifield, 1988; French & Jeffrey, 1997) and high intensity dieting (Van Son et al., 2013). These results also expand upon previous research by incorporating other facets of dietary restriction (i.e., food rules, restraint) into the latent dietary restriction variable along with current dieting behaviors and examining the relationship between weight suppression and the composite dietary restriction variable. However, results of the regression analysis did not support a longitudinal relationship between weight suppression
and dietary restriction. It is possible that the use of a composite variable, which accounted for multiple facets of restriction, may have obscured the simple behavioral outcome of dieting. Additionally, controlling for Time 1 dietary restriction accounted for a large portion of the variance in the analysis (Adjusted R square = .701) and may have prevented the detection of a significant prospective relationship between weight suppression and dietary restriction.

As predicted, dieting history and weight history were associated with weight suppression. These results are consistent with previous research findings that dieting is predictive of weight suppression (Mitchell et al., 2011) and previous overweight or obese status may contribute to weight suppression (Keel & Heatherton, 2010). A study by Keel and Heatherton (2010) examined weight suppression in a two cohorts of college students, with data originally collected in 1982 and 1992, and found that perceived weight status was positively correlated with weight suppression. Results of this study represent a replication of the Keel and Heatherton (2010) findings in a sample of college students that represents a different generation. Thus, these results demonstrate that the association between weight suppression, dieting history, and weight history are cross-generational.

Contrary to Hypothesis 1, Time 1 weight suppression did not predict Time 3 eating psychopathology longitudinally. Although this finding was unexpected, there are several possible reasons why a significant relationship was not found between these two variables over time. First, the study took place over a relatively short time period. Although there were significant changes in eating psychopathology over the eight weeks between Time 1 and Time 3, the magnitude of the change in EDDS scores was relatively small. Over a longer time period, the changes in EDDS scores may be more substantial and a significant relationship between weight suppression and eating psychopathology may be detected.
Second, EDDS scores decreased from Time 1 to Time 3. Given the prevalence of disordered eating, including diagnosable eating disorders, among college women (Berg, Frazier, & Sherr, 2009; Crowther, Armey, Luce, Dalton, & Leahey, 2008; Eisenberg, Nicklett, Roeder, & Kirz, 2011), one might expect that eating disorder symptoms would increase over time. However, in both previous research (Berg et al., 2009) and in this study, eating psychopathology actually decreased over time. Individuals who successfully suppressed their weight may not have developed or increased disordered eating cognition and behaviors because they were satisfied with their suppressed weight. Over time, the methods these individuals used to suppress their weight may become less effective and it is possible that eating psychopathology may emerge in an attempt to maintain their low weight or lose additional weight. Third, controlling for EDDS scores at Time 1 accounted for a large proportion of the variance in Time 3 EDDS scores (Adjusted R square = .702). This may have prevented the detection of any significant predictive value of weight suppression on eating psychopathology over time.

Finally, weight suppression may not be a significant predictor of eating psychopathology in the population used in the study except through dietary restriction. The undergraduate female participants in this study represent a relatively homogeneous, high-functioning population. The population was also young, with an average age of only 19.6 years, suggesting that most participants have likely only been at their current height for a few years. Some individuals may require more time to experience significant changes in weight at their current height; thus, weight suppression may predict eating psychopathology in an older sample. Other research assessing the impact of weight suppression on eating disorder symptoms typically uses an older sample with a greater age range (e.g., Butryn et al., 2011; Lowe et al., 2007), providing further support for the notion that weight suppression may be more relevant in an older population.
Additionally, the sample was not particularly pathological with respect to eating disorder symptoms (Krabbenborg, Danner, Larsen, et al., 2012). Weight suppression may be a more useful predictor of eating psychopathology in a clinical sample.

Although there is a significant cross-sectional relationship between weight suppression and dietary restriction, weight suppression is not a significant predictor of dietary restriction over time. Similar to the relationship between weight suppression and eating psychopathology, the absence of a significant longitudinal relationship between weight suppression and dietary restriction may be the result of the short time between time points and a large amount of variance explained once control variables are considered. This is particularly likely given that there were only four weeks between the measurement of weight suppression at Time 1 and the measurement of dietary restriction at Time 2. Additionally, there was little variance left to be explained after controlling for Time 1 dietary restriction, which may have prevented the detection of a significant relationship between weight suppression and dietary restriction.

**BMI, Impulsivity, Emotion Regulation, and Perfectionism as Moderators**

Although there is some support in previous literature for BMI as a moderator of the relationship between weight suppression and dietary restriction, the findings are limited and mixed. Thus, it is not surprising that BMI did not emerge as a moderator of this relationship in this study. The study in which BMI was a significant moderator (Butryn et al., 2011) examined frequency of binge eating as an outcome variable. BMI may be more closely associated with binge eating behaviors rather than dietary restriction, particularly given the lack of a bivariate association between BMI and dietary restriction.

Neither impulsivity nor emotion regulation were significant predictors of eating psychopathology longitudinally, nor did either of these variables interact with dietary restriction.
to moderate the longitudinal relationship between dietary restriction and eating psychopathology. These findings are somewhat surprising, given the strong correlational relationships between impulsivity, emotion dysregulation, and eating psychopathology at all time points in this study and the support in the research literature associating emotion dysregulation (e.g., Aldao, et al., 2010; Harrison, et al., 2009; Whiteside, et al., 2007) and impulsivity (e.g., Boisseau, et al., 2012; Kelly, et al., 2013; Vandereycken & Van Houdenhove, 1996; Waxman, 2009) with eating disorder symptoms. However, given the lack of significant change in eating psychopathology between Time 2 and Time 3, there was little variance left to be explained, even by variables that were strongly associated with Time 3 eating psychopathology. Future studies might examine the predictive relationships between impulsivity and eating psychopathology and emotion dysregulation and eating psychopathology over a longer period of time.

Despite evidence of a strong predictive relationship between body dissatisfaction and eating psychopathology (Bradford & Petrie, 2008; Downey & Chang, 2007; Jones & Crowther, 2013; Stice, 2001, 2002; Stice & Agras, 1998), a significant relationship has yet to be found between body dissatisfaction and weight suppression (Berner et al., 2013; Van Son et al., 2013). Body dissatisfaction is common and may be considered somewhat normative, particularly among young women. Due to the prevalence of body dissatisfaction among women and the degree to which many women experience dissatisfied thoughts about their appearance (Fallon, Harris, & Johnson, 2014; Fiske, Fallon, Blissmer, & Redding, 2014), it may not be an experience that is unique enough to predict weight suppression. Weight suppression is likely driven by factors other than dissatisfaction with one’s weight or shape. Potential motivating factors of weight suppression should be explored in future research.
Although perfectionism is an important variable in the interactive model (Vohs et al., 1999), perfectionism was not associated with weight suppression in this sample, nor did it moderate the relationship between body dissatisfaction and weight suppression. The interactive model, which includes a three-way interaction between perfectionism, perceived weight status, and self-esteem, posits that individuals who consider themselves overweight and are also high in perfectionism are more likely to experience low self-esteem and subsequently develop bulimic symptoms. The model proposed in this study does not account for self-esteem. It may be that self-esteem is the crucial component of the interactive model, without which perfectionism does not predict certain behaviors associated with eating disorders. Additionally, the proposed model is not specific to bulimic symptoms, which may have reduced the influence of perfectionism. Given the large body of research supporting an association between perfectionism and eating disorder symptoms (e.g., Bardone-Cone et al., 2006; Engler et al., 2006; Holm-Denoma et al., 2005; Jones & Crowther, 2013; Shafran et al., 2002), it is likely that perfectionism is associated specifically with eating disorder symptomatology rather than an eating disorder risk factor, such as weight suppression. The high correlations between FMPS scores and EDDS scores at all three time points in this study support this notion. Future research should examine perfectionism in the context of self-esteem and explore a possible interaction between weight suppression and perfectionism in order to predict eating psychopathology.

**Limitations and Suggestions for Future Research**

Although this study expanded upon previous research investigating weight suppression, several limitations exist. First, this study utilized self-report measures, including self-reported height and weight. Inherent biases in the use of self-report measures may call into question the accuracy of the data presented, particularly height and weight. Additionally, the use of online
data collection did not allow for the measurement of height and weight. However, self-report is considered a valid method of measurement of weight in clinical and non-clinical samples (Swenne et al., 2005; Tamakoshi et al., 2003). Self-report is not only the most efficient and cost-effective method of assessing behavior and cognition, there are no other known methods of collecting information about participants’ cognitions, such as body dissatisfaction and emotion dysregulation.

A second limitation of this research is the homogenous population. The sample was largely young and Caucasian and likely has limited socioeconomic variability. By definition, all participants have achieved a certain level of education and are relatively high-functioning. Although eating disorder symptoms are common among a young, undergraduate female population (Berg et al., 2009; Eisenberg, et al., 2011, it may be that the relationship between weight suppression and eating psychopathology functions differently in a more diverse population. Future research should examine the proposed relationships in populations that are more diverse in age, race, and education.

Finally, the data collection took place over a relatively short frame. A span of eight weeks from Time 1 to Time 3 may have been insufficient to capture meaningful changes in the variables of interest, particularly eating psychopathology. Changes in outcome variables, whether or not they were statistically significant, may not have been representative of a meaningful change. Future research should examine these relationships over a longer period of time to replicate results from previous research (Keel & Heatherton, 2010) and expand upon the findings of this study.

Despite these limitations, this study expanded upon the existing weight suppression literature in several important ways. First, it introduced a unique method of examining thoughts and
behaviors related to dieting through the use of the latent dietary restriction variable. Second, it replicated previous findings that weight suppression is associated with dieting and weight history and expanded these findings to a new generation of college students. Finally, this research identified dietary restriction as a mediator of the longitudinal relationship between weight suppression and eating psychopathology and reinforced the importance of dietary restriction as a longitudinal predictor of eating psychopathology. Future research should continue to examine correlates of weight suppression and moderators of the relationship between weight suppression and eating psychopathology. Additionally, this study found high correlations between weight rebound and a number of moderating and dependent variables (i.e., BMI, impulsivity, emotion dysregulation, body dissatisfaction, weight history, dieting history, and eating psychopathology). It may be that, in some respects, weight rebound is a more salient, relevant variable than weight suppression in young, nonclinical populations. In these populations, weight gain, particularly in the form of weight rebound, may be a more significant risk factor for later development of eating psychopathology than sustained weight loss and this relationship may be moderated by trait variables, such as impulsivity and emotion dysregulation. Understanding the temporal relationships among these variables, including when individuals experienced their highest and lowest weights compared to their current weights, may shed additional light on the relevance of both weight suppression and weight rebound among college aged women. Future research might investigate the relationship between weight rebound and other variables of interest to determine the utility of this variable in the understanding of the development and maintenance of eating psychopathology.


Figure 1
Proposed Model of the Relationship between Weight Suppression and Eating Disorder Symptomatology
Figure 2
Relationships Proposed in Hypotheses 1-4

Weight Suppression → BMI → Dietary Restriction

Current Dieting → Restriction

Restriction → Food Rules

Eating Psychopathology → Impulsivity → Emotion Dysregulation
Figure 3
Proposed Relationship in Hypothesis 6
Table 1

*Time 1 differences between participant groups*

<table>
<thead>
<tr>
<th></th>
<th>Complete</th>
<th></th>
<th>Incomplete</th>
<th></th>
<th>t</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>19.49</td>
<td>2.01</td>
<td>19.23</td>
<td>1.49</td>
<td>1.019</td>
<td>276</td>
</tr>
<tr>
<td>BMI</td>
<td>24.39</td>
<td>5.77</td>
<td>25.42</td>
<td>6.25</td>
<td>-1.291</td>
<td>276</td>
</tr>
<tr>
<td>BSQ</td>
<td>100.82</td>
<td>35.45</td>
<td>100.91</td>
<td>33.70</td>
<td>-.018</td>
<td>276</td>
</tr>
<tr>
<td>FMPS</td>
<td>109.50</td>
<td>111.86</td>
<td>18.77</td>
<td>17.95</td>
<td>-.937</td>
<td>276</td>
</tr>
<tr>
<td>MAEDS - RE</td>
<td>25.04</td>
<td>9.27</td>
<td>25.00</td>
<td>8.60</td>
<td>.032</td>
<td>276</td>
</tr>
<tr>
<td>MAEDS - AFF</td>
<td>33.31</td>
<td>11.90</td>
<td>36.04</td>
<td>12.10</td>
<td>-1.681</td>
<td>276</td>
</tr>
<tr>
<td>UPPS</td>
<td>30.44</td>
<td>7.57</td>
<td>29.49</td>
<td>8.05</td>
<td>.914</td>
<td>276</td>
</tr>
<tr>
<td>DERS</td>
<td>90.23</td>
<td>27.08</td>
<td>92.39</td>
<td>25.40</td>
<td>-.598</td>
<td>276</td>
</tr>
<tr>
<td>EDDS</td>
<td>24.42</td>
<td>14.56</td>
<td>25.36</td>
<td>13.82</td>
<td>-.484</td>
<td>276</td>
</tr>
</tbody>
</table>

*Note:* Complete = participants who provided responses to all questionnaires at all three time points; Incomplete = participants who did not provide responses to all questionnaires at all three time points; BMI = body mass index; BSQ = Body Shape Questionnaire; FMPS = Frost Multidimensional Perfectionism Scale; MAEDS – RE = Multifactorial Assessment of Eating Disorder Symptoms – Restrictive Eating subscale; MAEDS – AFF = Multifactorial Assessment of Eating Disorder Symptoms – Avoidance of Forbidden Foods subscale; UPPS = UPPS Impulsive Behavior Scale – Urgency subscale; DERS = Difficulties in Emotion Regulation Scale; EDDS = Eating Disorder Diagnostic Scale
Table 2

Descriptive statistics

<table>
<thead>
<tr>
<th></th>
<th>M</th>
<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Square Root WS – Time 1</td>
<td>2.487</td>
<td>1.752</td>
<td>1.090</td>
<td>3.833</td>
</tr>
<tr>
<td>DR – Time 1</td>
<td>.198</td>
<td>1.898</td>
<td>.659</td>
<td>-.040</td>
</tr>
<tr>
<td>DR – Time 2</td>
<td>1.889</td>
<td>1.573</td>
<td>.541</td>
<td>.045</td>
</tr>
<tr>
<td>EDDS – Time 1</td>
<td>24.500</td>
<td>14.152</td>
<td>.403</td>
<td>-.248</td>
</tr>
<tr>
<td>EDDS – Time 2</td>
<td>22.400</td>
<td>14.361</td>
<td>.753</td>
<td>.510</td>
</tr>
<tr>
<td>Weight History</td>
<td>14.379</td>
<td>5.753</td>
<td>1.046</td>
<td>-.079</td>
</tr>
<tr>
<td>BSQ – Time 1</td>
<td>101.170</td>
<td>35.536</td>
<td>.168</td>
<td>-.509</td>
</tr>
<tr>
<td>FMPS</td>
<td>108.970</td>
<td>18.727</td>
<td>.455</td>
<td>.697</td>
</tr>
<tr>
<td>BMI – Time 1</td>
<td>24.349</td>
<td>5.757</td>
<td>1.733</td>
<td>3.827</td>
</tr>
<tr>
<td>UPPS</td>
<td>30.280</td>
<td>7.521</td>
<td>-.145</td>
<td>-.340</td>
</tr>
<tr>
<td>DERS</td>
<td>89.650</td>
<td>26.958</td>
<td>.699</td>
<td>.322</td>
</tr>
<tr>
<td>WS Length</td>
<td>12.680</td>
<td>13.974</td>
<td>1.692</td>
<td>2.457</td>
</tr>
<tr>
<td>WR – Time 1</td>
<td>16.613</td>
<td>18.296</td>
<td>3.002</td>
<td>13.495</td>
</tr>
<tr>
<td>Square Root WR – Time 1</td>
<td>3.607</td>
<td>1.902</td>
<td>.971</td>
<td>1.999</td>
</tr>
</tbody>
</table>

Note: WS = weight suppression, DR = dietary restriction, EDDS = eating psychopathology, BSQ = body dissatisfaction, FMPS = perfectionism, BMI = body mass index, UPPS = impulsivity, DERS = emotion dysregulation, WS Length = length of weight suppression, WR = weight rebound.
Table 3
Results of correlational analyses

<table>
<thead>
<tr>
<th></th>
<th>WS</th>
<th>T1</th>
<th>DR T1</th>
<th>DR T2</th>
<th>EDDS T1</th>
<th>EDDS T2</th>
<th>EDDS T3</th>
<th>WH</th>
<th>BSQ</th>
<th>FMPS</th>
<th>BMI T1</th>
<th>UPPS</th>
<th>DERS</th>
<th>DH</th>
<th>WS</th>
<th>Length</th>
<th>WR</th>
</tr>
</thead>
<tbody>
<tr>
<td>WS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DR T1</td>
<td>.235*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DR T2</td>
<td>.168*</td>
<td>.839**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDDS T1</td>
<td>.113</td>
<td>.604**</td>
<td>.597**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDDS T2</td>
<td>.147*</td>
<td>.590**</td>
<td>.611**</td>
<td>.842**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDDS T3</td>
<td>.097</td>
<td>.582**</td>
<td>.606**</td>
<td>.839**</td>
<td>.881**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WH</td>
<td>.224**</td>
<td>.152*</td>
<td>.127</td>
<td>.257**</td>
<td>.293**</td>
<td></td>
<td>.270**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BSQ</td>
<td>.080</td>
<td>.641**</td>
<td>.569**</td>
<td>.801**</td>
<td>.735**</td>
<td>.728**</td>
<td></td>
<td>.293**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FMPS</td>
<td>.004</td>
<td>.391**</td>
<td>.374**</td>
<td>.359**</td>
<td>.429**</td>
<td>.452**</td>
<td>.094</td>
<td></td>
<td>.341**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI T1</td>
<td>.024</td>
<td>.023</td>
<td>.003</td>
<td>.220**</td>
<td>.263**</td>
<td>.234**</td>
<td>.563**</td>
<td>.318**</td>
<td>.002</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UPPS</td>
<td>-.071</td>
<td>.340**</td>
<td>.256**</td>
<td>.543**</td>
<td>.474**</td>
<td>.469**</td>
<td>.170*</td>
<td>.534**</td>
<td>.437**</td>
<td>.140</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DERS</td>
<td>-.104</td>
<td>.391**</td>
<td>.293**</td>
<td>.483**</td>
<td>.463**</td>
<td>.443**</td>
<td>.023</td>
<td>.534**</td>
<td>.535**</td>
<td>.003</td>
<td>.676**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DH</td>
<td>.174*</td>
<td>.305**</td>
<td>.272**</td>
<td>.367**</td>
<td>.367**</td>
<td>.415**</td>
<td>.221**</td>
<td>.443**</td>
<td>.089</td>
<td>.259**</td>
<td>.147</td>
<td>.136</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WS</td>
<td>-.153*</td>
<td>.212**</td>
<td>-.123</td>
<td>-.234**</td>
<td>-.236**</td>
<td>-.190**</td>
<td>-.081</td>
<td>-.209**</td>
<td>-.081</td>
<td>.053</td>
<td>-.192**</td>
<td>-.106</td>
<td>-.053</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length</td>
<td>-.169*</td>
<td>.173*</td>
<td>.119</td>
<td>.265**</td>
<td>.302**</td>
<td>.317**</td>
<td>.307**</td>
<td>.427**</td>
<td>.108</td>
<td>.629**</td>
<td>.273**</td>
<td>.155*</td>
<td>.248**</td>
<td>-.039</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: * = p < .05, ** p < .01; T1 = Time 1, T2 = Time 2, 3 = Time 3, WS = weight suppression, DR = dietary restraint, EDDS = eating psychopathology, WH = weight history, BSQ = body dissatisfaction, FMPS = perfectionism, BMI = body mass index, UPPS = impulsivity, DERS = emotion dysregulation, DH = dieting history, WS Length = length of weight suppression, WR = weight rebound.
Table 4

*Results of regression analyses*

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypothesis 1 – Predicting Time 3 EDDS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>.862</td>
<td>1.328</td>
<td></td>
</tr>
<tr>
<td>EDDS – Time 1</td>
<td>.043</td>
<td>.040</td>
<td>.838*</td>
</tr>
<tr>
<td>WS – Time 1</td>
<td>.017</td>
<td>.325</td>
<td>.002</td>
</tr>
<tr>
<td><strong>Total R²</strong></td>
<td></td>
<td></td>
<td>.703</td>
</tr>
<tr>
<td><strong>Hypothesis 3 – Predicting Time 2 DR</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>1.683</td>
<td>.444</td>
<td></td>
</tr>
<tr>
<td>DR – Time 1</td>
<td>.693</td>
<td>.035</td>
<td>.838*</td>
</tr>
<tr>
<td>WS – Time 1</td>
<td>.133</td>
<td>.156</td>
<td>.148</td>
</tr>
<tr>
<td>BMI – Time 1</td>
<td>.002</td>
<td>.017</td>
<td>.007</td>
</tr>
<tr>
<td>WS*BMI</td>
<td>-.005</td>
<td>.006</td>
<td>-.165</td>
</tr>
<tr>
<td><strong>Total R²</strong></td>
<td></td>
<td></td>
<td>.706</td>
</tr>
<tr>
<td><strong>Hypothesis 4 – Model 1 – Predicting Time 3 EDDS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-4.042</td>
<td>2.927</td>
<td></td>
</tr>
<tr>
<td>EDDS – Time 2</td>
<td>.795</td>
<td>.047</td>
<td>.785*</td>
</tr>
<tr>
<td>DR – Time 2</td>
<td>.969</td>
<td>.386</td>
<td>.106*</td>
</tr>
<tr>
<td>UPPS</td>
<td>.203</td>
<td>.106</td>
<td>.107</td>
</tr>
<tr>
<td>DR*UPPS</td>
<td>-.247</td>
<td>.267</td>
<td>-.407</td>
</tr>
<tr>
<td><strong>Total R²</strong></td>
<td></td>
<td></td>
<td>.797</td>
</tr>
<tr>
<td><strong>Hypothesis 4 – Model 2 – Predicting Time 3 EDDS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-.395</td>
<td>2.578</td>
<td></td>
</tr>
<tr>
<td>EDDS – Time 2</td>
<td>-.821</td>
<td>.047</td>
<td>.811*</td>
</tr>
<tr>
<td>DR – Time 2</td>
<td>.949</td>
<td>.392</td>
<td>.104*</td>
</tr>
<tr>
<td>DERS</td>
<td>.021</td>
<td>.031</td>
<td>.040</td>
</tr>
<tr>
<td>DR*DERS</td>
<td>-.093</td>
<td>.276</td>
<td>-.019</td>
</tr>
<tr>
<td><strong>Total R²</strong></td>
<td></td>
<td></td>
<td>.793</td>
</tr>
<tr>
<td><strong>Hypothesis 6 – Predicting Time 1 WS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>2.508</td>
<td>.133</td>
<td></td>
</tr>
<tr>
<td>BSQ</td>
<td>.161</td>
<td>.136</td>
<td>.092</td>
</tr>
<tr>
<td>FMPS</td>
<td>-.017</td>
<td>.146</td>
<td>-.009</td>
</tr>
<tr>
<td>BSQ*FMPS</td>
<td>-.060</td>
<td>.112</td>
<td>-.043</td>
</tr>
<tr>
<td><strong>Total R²</strong></td>
<td></td>
<td></td>
<td>.008</td>
</tr>
</tbody>
</table>

*Note: *p < .05; WS = weight suppression, DR = dietary restraint, EDDS = eating psychopathology, BMI = body mass index, DR = dietary restriction, UPPS = impulsivity, DERS = emotion dysregulation, BSQ = body dissatisfaction, FMPS = perfectionism.*
Table 5

*Results of post hoc regression analyses predicting Time 3 EDDS*

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>.848</td>
<td>1.669</td>
<td></td>
</tr>
<tr>
<td>EDDS – Time 1</td>
<td>.855</td>
<td>.042</td>
<td>.848*</td>
</tr>
<tr>
<td>WS – Time 1</td>
<td>-.137</td>
<td>.439</td>
<td>-.017</td>
</tr>
<tr>
<td>LWS – Time 1</td>
<td>-.021</td>
<td>.074</td>
<td>-.021</td>
</tr>
<tr>
<td>WS*LWS</td>
<td>.015</td>
<td>.030</td>
<td>.038</td>
</tr>
<tr>
<td>Total R²</td>
<td>.713</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-1.789</td>
<td>2.044</td>
<td></td>
</tr>
<tr>
<td>DR – Time 1</td>
<td>.812</td>
<td>.042</td>
<td>.807</td>
</tr>
<tr>
<td>WS – Time 1</td>
<td>.232</td>
<td>.656</td>
<td>.029</td>
</tr>
<tr>
<td>WR – Time 1</td>
<td>.824</td>
<td>.456</td>
<td>.110</td>
</tr>
<tr>
<td>WS*WR</td>
<td>-.011</td>
<td>.160</td>
<td>-.006</td>
</tr>
<tr>
<td>Total R²</td>
<td>.713</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: *p < .001; EDDS = eating psychopathology, WS = weight suppression, LWS = length of weight suppression, WR = weight rebound.*
Appendix A
Measures

Demographic Questionnaire

Age: ___ years

Height: ___ ft ___ in

Weight: ___ lbs

Current year: __ Freshman __ Sophomore __ Junior __ Senior __ Post-Undergraduate
__ Other (please specify: _______________)

Race/Ethnicity: __ American Indian or Alaskan Native __ African American/Black __ Asian
__ Caucasian/White __ Hispanic/Latina __ Other (please specify: ___________)

74
The Dieting and Weight History Questionnaire
(Witt, Katterman, & Lowe, 2013)

1. What is the most you have ever weighed since reaching your current height? (do not count any weight gains due to medical conditions or medications). The most I have weighed since reaching my current height is:

_______ pounds

2. What is the least you have ever weighed since reaching your current height? (do not count any weight losses due to medical conditions or medications). The least I have weighed since reaching my current height is:

_______ pounds

3. What is your current weight?

_______ pounds

4. Please determine the difference between your answer to number 1 and number 3. If this difference is less than 5 lbs. skip this item and go on to item 5. If this difference is 5 lbs. or more, circle the letter of the statement below that best describes this difference:

A. The difference between my highest weight and my current weight is due to weight that I lost on purpose.

B. The difference between my highest weight and my current weight is due to weight I lost even though I wasn’t trying to.

C. I’m not sure why I weigh less than I once did.

5. For about how long have you been at or close (within 2 lbs.) to your present weight?

__________________
6. Which of these statements best describes what has happened to your weight during the past 6 months? (circle one)

A. My weight has stayed about the same.
B. I’ve been losing weight.
C. I’ve been gaining weight.
D. My weight has fluctuated a lot.

7. Are you currently on a diet? (circle one)             Yes              No (if no, go to #13).

8. Are you currently dieting to lose weight or to avoid gaining weight? (circle one)
   To lose weight               To avoid gaining weight

9. How long have you been on your current diet?              ______________

10. How much longer do you anticipate being on your diet?          ______________

11. How much weight (if any) have you lost on your current diet?          ______________

12. How much more weight do you intend to lose on your current diet?          ______________

13. Answer #13 only if you are not currently dieting:
   Have you ever been on a diet to lose weight?             Yes              No
   (if no, skip #14-16; you are done)

14. About how long ago were you last on a diet to lose weight?
   (if you are currently dieting, refer to the most recent diet prior to your current diet)____________

15. About how old were you when you went on your first diet?   __________years old.

16. Please estimate as best you can the number of times in your life you have dieted and purposely lost the amount of weight listed.
   How many times in your life have you dieted and lost:
   1-4 pounds? _________times
   5-10 pounds? _________times
   11-20 pounds? _________times
   21 or more pounds? _________times
Weight History Questions

1. Were you ever overweight or obese as a child or adolescent?
   A. Yes, as a child
   B. Yes, as an adolescent
   C. Yes, as both a child and an adolescent
   D. No, I was never overweight or obese as a child or adolescent

2. Were your parents overweight or obese when you were a child?
   A. Yes, my mother was overweight/obese
   B. Yes, my father was overweight/obese
   C. Yes, both my mother and father were overweight/obese
   D. No, neither of my parents were overweight/obese
**Body Shape Questionnaire**  
*(Cooper, Taylor, Cooper, & Fairburn, 1987)*

We would like to know how you have been feeling about your appearance over the PAST FOUR WEEKS. Please read each question and circle the appropriate number to the right. Please answer all the questions.

**OVER THE PAST FOUR WEEKS:**

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Has feeling bored made you brood about your shape?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2.</td>
<td>Have you been so worried about your shape that you have been feeling that you ought to diet?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3.</td>
<td>Have you thought that your thighs, hips or bottom are too large for the rest of you?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4.</td>
<td>Have you been afraid that you might become fat (or fatter)?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5.</td>
<td>Have you been worried about your flesh not being firm enough?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6.</td>
<td>Has feeling full (e.g., after eating a large meal) made you feel fat?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7.</td>
<td>Have you felt so bad about your shape that you have cried?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8.</td>
<td>Have you avoided running because your flesh might wobble?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9.</td>
<td>Has being with thin women made you feel self-conscious about your shape?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10.</td>
<td>Have you worried about your thighs spreading out when sitting down?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11.</td>
<td>Has eating even a small amount of food made you feel fat?</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Question</td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very Often</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------------------------</td>
<td>-------</td>
<td>--------</td>
<td>-----------</td>
<td>-------</td>
<td>------------</td>
</tr>
<tr>
<td>12. Have you noticed the shape of other women and felt that your own shape compared unfavorably?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>13. Has thinking about your shape interfered with your ability to concentrate (e.g., while watching, television, reading, listening to conversations)?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>14. Has being naked, such as when taking a bath, made you feel fat?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>15. Have you avoided wearing clothes which made you particularly aware of the shape of your body?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>16. Have you imagined cutting off fleshy areas of your body?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>17. Has eating sweets, cakes, or other high calorie food made you feel fat?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>18. Have you not gone out to social occasions (e.g., parties) because you have felt bad about your shape?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>19. Have you felt excessively large and rounded?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>20. Have you felt ashamed of your body?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>21. Has worry about your shape made you diet?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>22. Have you felt happiest about your shape when your stomach has been empty (e.g., in the morning)?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>23. Have you thought that you are the shape you are because you lack self-control?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>24. Have you worried about other people seeing rolls of flesh around your waist or stomach?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>25. Have you felt that it is not fair that other women are thinner than you?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>26. Have you vomited in order to feel thinner?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>27. When in company have you worried about taking up too much room (e.g., sitting on a sofa or a bus seat)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>28. Have you worried about your flesh being dimply?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>29. Has seeing your reflection (e.g., in a mirror or shop window) made you feel bad about your shape?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>30. Have you pinched areas of your body to see how much fat there is?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>31. Have you avoided situations where people could see your body (e.g., communal changing rooms or swimming baths)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>32. Have you taken laxatives in order to feel thinner?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>33. Have you been particularly self-conscious about your shape when in the company of other people?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>34. Has worry about your shape made you feel you ought to exercise?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Never</td>
<td>Rarely</td>
<td>Sometimes</td>
<td>Often</td>
<td>Very</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Frost Multidimensional Perfectionism Scale
(Frost, Marten, Lahart, & Rosenblate, 1990)

This material is copyrighted and is not presented in this document.
The Multifactorial Assessment of Eating Disorder Symptoms
Restrictive Eating and Avoidance of Forbidden Foods Subscales
(Anderson, Williamson, Duchmann, Gleaves, & Barbin, 1999)

This material is copyrighted and does not appear in this document.
UPPS Impulsive Behavior Scale – Urgency Subscale
(Whiteside & Lynam, 2001)

1. I have trouble controlling my impulses.
   Disagree  Disagree  Agree  Agree
   Strongly  Some  Some  Strongly
   1  2  3  4

2. I have trouble resisting my cravings (for food, cigarettes, etc.).
   1  2  3  4

3. I often get involved in things I later wish I could get out of.
   1  2  3  4

4. When I feel bad, I will often do things I later regret in order to make myself feel better now.
   1  2  3  4

5. Sometimes when I feel bad, I can’t seem to stop what I am doing even though it is making me feel worse.
   1  2  3  4

6. When I am upset I often act without thinking.
   1  2  3  4

7. When I feel rejected, I will often say things that I later regret.
   1  2  3  4

8. It is hard for me to resist acting on my feelings.
   1  2  3  4

9. I often make matters worse because I act without thinking when I am upset.
   1  2  3  4

10. In the heat of an argument, I will often say things that I later regret.
    1  2  3  4

11. I am always able to keep my feelings under control.
    1  2  3  4

12. Sometimes I do things on impulse that I later regret.
    1  2  3  4
**Difficulties in Emotion Regulation Scale**  
(Gratz & Roemer, 2004)

**Instructions:** Please indicate how often the following 36 statements apply to you by writing the appropriate number from the scale below (1-5) in the space alongside each item.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almost never (0-10%)</td>
<td>Sometimes (11-35%)</td>
<td>About half the time (36-65%)</td>
<td>Most of the time (66-90%)</td>
<td>Almost always (90-100%)</td>
</tr>
</tbody>
</table>

_____ 1. I am clear about my feelings.
_____ 2. I pay attention to how I feel.
_____ 3. I experience my emotions as overwhelming and out of control.
_____ 4. I have no idea how I am feeling.
_____ 5. I have difficulty making sense out of my feelings.
_____ 6. I am attentive to my feelings.
_____ 7. I know exactly how I am feeling.
_____ 8. I care about what I am feeling.
_____ 9. I am confused about how I feel.
_____ 10. When I’m upset, I acknowledge my emotions.
_____ 11. When I’m upset, I become angry with myself for feeling that way.
_____ 12. When I’m upset, I become embarrassed for feeling that way.
_____ 13. When I’m upset, I have difficulty getting work done.
_____ 14. When I’m upset, I become out of control.
_____ 15. When I’m upset, I believe that I will remain that way for a long time.
_____ 16. When I’m upset, I believe that I’ll end up feeling very depressed.
_____ 17. When I’m upset, I believe that my feelings are valid and important.
_____ 18. When I’m upset, I have difficulty focusing on other things.
_____ 19. When I’m upset, I feel out of control.
_____ 20. When I’m upset, I can still get things done.
_____ 21. When I’m upset, I feel ashamed with myself for feeling that way.
_____ 22. When I’m upset, I know that I can find a way to eventually feel better.
_____ 23. When I’m upset, I feel like I am weak.
<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Almost never</td>
<td>Sometimes</td>
<td>About half the time</td>
<td>Most of the time</td>
<td>Almost always</td>
</tr>
<tr>
<td></td>
<td>(0-10%)</td>
<td>(11-35%)</td>
<td>(36-65%)</td>
<td>(66-90%)</td>
<td>(90-100%)</td>
</tr>
</tbody>
</table>

24. When I’m upset, I feel like I can remain in control of my behaviors.
25. When I’m upset, I feel guilty for feeling that way.
26. When I’m upset, I have difficulty concentrating.
27. When I’m upset, I have difficulty controlling my behaviors.
28. When I’m upset, I believe that there is nothing I can do to make myself feel better.
29. When I’m upset, I become irritated with myself for feeling that way.
30. When I’m upset, I start to feel very bad about myself.
31. When I’m upset, I believe that wallowing in it is all I can do.
32. When I’m upset, I lose control over my behaviors.
33. When I’m upset, I have difficulty thinking about anything else.
34. When I’m upset, I take time to figure out what I’m really feeling.
35. When I’m upset, it takes me a long time to feel better.
36. When I’m upset, my emotions feel overwhelming.
Eating Disorder Diagnostic Scale
(Stice, Telch, & Rizvi, 2000)

OVER THE PAST THREE MONTHS:

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Not at all</td>
<td>Slightly</td>
<td>Moderately</td>
<td>Extremely</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

___ 1. Have you felt fat?
___ 2. Have you had a definite fear that you might gain weight or become fat?
___ 3. Has your weight influenced how you think about (judge) yourself as a person?
___ 4. Has your shape influenced how you think about (judge) yourself as a person?

5. During the past 6 months, have there been times when you felt you have eaten what other people would regard as an unusually large amount of food (i.e., a quart of ice cream) given the circumstance?
   YES   NO

6. During the times when you ate an unusually large amount of food, did you experience a loss of control (feel you couldn’t stop eating or control what or how much you were eating)?
   YES   NO

7. How many DAYS per week on average over the past 6 MONTHS have you eaten an unusually large amount of food and experienced a loss of control?
   0 1 2 3 4 5 6 7

8. How many TIMES per week on average over the past 3 MONTHS have you eaten an unusually large amount of food and experienced a loss of control?
   0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

During these episodes of overeating and loss of control did you…

9. Eat much more rapidly than normal?   YES   NO

10. Eat until you felt uncomfortably full?   YES   NO

11. Eat large amounts of food when you didn’t feel physically hungry?   YES   NO

12. Eat alone because you were embarrassed by how much you were eating?   YES   NO

13. Feel disgusted with yourself, depressed, or very guilty after overeating?   YES   NO

14. Feel very upset about your uncontrollable overeating or resulting weight gain?   YES   NO

15. How many times per week on average over the past 3 months have you made yourself vomit to prevent weight gain or counteract the effects of eating?
   0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

16. How many times per week on average over the past 3 months have you used laxatives or diuretics to prevent weight gain or counteract the effects of eating?
   0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

17. How many times per week on average over the past 3 months have you fasted (skipped at least 2 meals in a row) to prevent weight gain or counteract the effects of eating?
   0 1 2 3 4 5 6 7 8 9 10 11 12 13 14

18. How many times per week on average over the past 3 months have you engaged in excessive exercise specifically to counteract the effects of overeating episodes?
   0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
20. How tall are you? _____ ft. _____ in.
21. Over the past 3 months, how many periods have you missed? 1 2 3 4 n/a
22. Have you been taking birth control pills during the past 3 months? YES NO