

THE IMPACT OF A DISSONANCE-BASED PREVENTION PROGRAM ON
EATING DISORDER DEVELOPMENTAL TRAJECTORIES

by

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A DISSERTATION

Presented to the Department of Counseling Psychology and Human Services
and the Graduate School of the University of Oregon
in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy

September 2016

DISSERTATION APPROVAL PAGE

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DISSERTATION ABSTRACT

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Department of Counseling Psychology and Human Services

September 2016

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Randomized trials provide support for the *Body Project*, an eating disorder prevention program wherein young women with body image concerns critique the thin ideal, which putatively reduces pursuit of this unrealistic ideal as a result of dissonance-induction. Despite medium to large effects, some *Body Project* participants subsequently develop an eating disorder during 3-year study follow-up, suggesting intervention or recruitment procedures could be improved. This study was the first to delineate the heterogeneous pathways of eating disorder symptom trajectories among *Body Project* versus control group participants during 3-year study follow-up. This study also investigated the predictive role of baseline risk factors on qualitatively distinct developmental pathways of eating disorder symptomology, helping to explain contributing factors to suboptimal *Body Project* response. Existing data from three randomized controlled trials were combined to examine response trajectories of prevention intervention versus control participants through 3-year follow-up. Group-Based Trajectory Modeling distinguished distinct response trajectories and the impact of prevention on mitigating the developmental course of eating disorder symptoms. The three-group solution for control participants produced the strongest model fit. The resulting trajectories were those of low-stable, moderate-stable, or high-variable levels of

eating disorder symptom courses. Dietary restraint and negative affect predicted increased likelihood of membership in the high-risk trajectory. The optimal solution for *Body Project* participants was a two-group trajectory model with low-decreasing or high-decreasing trajectories, with the moderate-level risk group observed in the control group seemingly deflected by prevention effects. This study also determined the predictive role of risk factors on qualitatively distinct developmental pathways of eating disorder symptomology, confirming the hypothesized impact of thin-ideal internalization, negative affect, and dietary restraint on sub-optimal prevention response. The results of this novel study supplement developmental research regarding eating disorder symptom predictors and course, ultimately informing future design and adaptation of evidence-based eating disorder prevention programs.

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ACKNOWLEDGMENTS

I want to express my most sincere appreciation to my dissertation committee for their assistance in the preparation of this manuscript and their guidance throughout my dissertation journey. Special thanks are due to Drs. Beth Stormshak and Eric Stice, whose mentorship, collaboration, and thoughtful feedback has been invaluable to my development as a researcher. I would also like to thank my family, friends, and CPSY community for their everlasting support. To my parents, Briggs and Melanie, thank you for a lifetime of love and encouragement. I wouldn't have the drive and determination (i.e., stubbornness) I have today had it not been for your motivation that I could do anything I set my mind to. To my sister, Stacy, who never fails to ask about my research, brags about my academic accolades on social media, and has sent "just because" flowers and gifts more than once when I needed a motivational boost. To the amazing CPSY community, many of whom are my closest, life-long friends. Your validation, humor, and empathy fueled these dissertation pages. And finally but most importantly, to my best friend, Kellen, who has sat right beside me during this wild ride. You are my rock, and it doesn't hurt that you also happen to be a great cook and fantastic copy editor. Thank you for making me smile in the most necessary moments.

Dedicated to Briggs, the scientist, whose laugh I will never forget.

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CHAPTER I

LITERATURE REVIEW

Introduction

The lifetime course of eating disorders is often chronic and marked by symptom fluctuation and diagnostic changes over time. This change over time is indicative of the variable developmental course of eating disorders as individuals move to more or less severe positions of eating disorder pathology. Although cross-sectional and retrospective designed studies are informative, they lack the ability to make strong inferences regarding whether risk factors temporally precede the development of eating pathology. Research continues to expand upon our ability to predict eating disorder onset, however further research is needed to understand the influence of varied risk factors on eating disorder development over time (Stice, 2002).

Many longitudinal eating disorder studies utilize binary outcome categories such as "remission" and "relapse," or categorical eating disorder diagnoses (e.g., anorexia nervosa, bulimia nervosa), to examine eating disorder development (Lavender et al., 2011). However, this approach does not capture the broad range of eating disorder classifications and diverse developmental trajectories. Recently, longitudinal mixture modeling analyses have been recommended as a means of examining eating disorder developmental trajectories (Lavender et al., 2011). This statistical approach has the potential to uncover novel information about eating disorder symptom predictors and developmental course, ultimately informing prevention and intervention efforts.

Heterogeneity of the duration and course of eating disorders has made it difficult to identify stable longitudinal predictors as different developmental pathways may be

associated with different risk factors (Graber, Brooks-Gunn, Paikoff, & Warren, 1994; Tyrka, Graber, Brooks-Gunn, 2000). Although research has examined the developmental course of eating disorders (e.g., Stice et al., 2009; Stice, Marti, & Rohde, 2013), few studies have used a finite mixture modeling approach to examine heterogeneous eating disorder developmental trajectory models (Aimé, Craig, Pepler, Jiang, & Connolly, 2008; Pearson, 2014; Smith, Simmons, Flory, Annus, & Hill, 2007). Further, no research has utilized a finite mixture modeling approach to examine the impact of empirically-based eating disorder prevention intervention on the course of eating pathology development. The novel design of this study allowed for the examination of the evolution of disordered symptoms, the influence of individual risk factors, and the changes in eating pathology with and without the influence of an efficacious eating disorder prevention program.

Eating disorder incidence, prevalence, and development

Over ten percent of adolescent girls and young women in the United States meet criteria for *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV-TR]: (APA, 2000) anorexia nervosa (AN), bulimia nervosa (BN), or eating disorder not otherwise specified (EDNOS) (Hudson, Hiripi, Harrison, & Kessler, 2007; Stice, Marti, Shaw, & Jaconis, 2009; Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006). A recent study of eating disorder prevalence and incidence based on *DSM-5* (APA, 2013) eating disorder criteria, suggested prevalence rates of eating disorders by age 20 exceed 13 percent (Stice, Marti, & Rohde, 2013). *DSM-5* changes to eating disorder criteria consist of: the inclusion of binge eating disorder (BED), a reduction in the frequency and duration of certain symptoms for BN and BED, the elimination of symptoms with limited empirical support, and the inclusion of several Feeding and Eating Concerns Not

Elsewhere Classified (FEC-NEC) that include conditions such as atypical AN, subthreshold BN, subthreshold BED, and purging disorder (PD) (APA, 2013; Stice, Marti, & Rohde, 2013).

Peak age of eating disorder onset is 19-20 years old for AN, 16-20 for BN, and 18-20 for BED, PD, and FEC-NEC (Stice, Marti, & Rohde, 2013). Research indicates that more than half of individuals seeking treatment for eating or body image concerns receive a subthreshold eating disorder diagnosis (Eddy, Celio, Hoste, Herzog, & le Grange, 2008; Fairburn & Bohn, 2005; Fisher, Schneider, Burns, Symons, & Mandel, 2001). Although *DSM-5* diagnoses may differ, individuals with full versus subthreshold eating disorder tend not to differ significantly in terms of functional impairment, morbidity, psychiatric comorbidity, and risk for future physical and mental health problems (APA, 2000; Eddy et al., 2008; Fairburn & Bohn, 2005; Fisher et al., 2001; Keel, Brown, Holm-Denoma, & Bodell, 2011; Stice, Marti, et al., 2009).

Eating disorder developmental trajectories are variable and can lack stability over time (Stice, Presnell, & Spangler, 2002; Tyrka, Graber, & Brooks-Gunn, 2000). For example, some individuals will recover after initial elevations in eating disorder symptoms others may exhibit linear growth of symptoms over time, and still others may exhibit variable eating disorder symptoms with a developmental course marked with relapse and recovery (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). Research suggests eating disorder risk increases through adolescence, with eating pathology trajectories existing at different time points during adolescence and into adulthood (Aimé, Craig, Pepler, Jiang, & Connolly, 2008; Fay & Lerner, 2013; Smith, Simmons, Flory, Annus, & Hill, 2007; Tyrka, Graber, Brooks-Gunn, 2000). Similar group trajectories

have been found in treatment seeking adult women with AN or BN (Lavender et al., 2011). Most recently, Pearson (2014) conducted a study examining the development of binge eating and purging behaviors among pre-adolescent and early adolescent girls, the first examination of eating disorder developmental trajectories in an elementary-aged sample. The examination revealed the onset of at-risk developmental trajectories for even young girls (Pearson, 2014).

Eating disorder risk factors

Interdependent domains of risk factors have emerged in the eating disorder literature and include culture, family (e.g. socioeconomic status, parental psychopathology, family conflict, parental dieting), genetics, traumatic events (e.g. sexual and physical abuse), and social psychological factors (Vitiello & Lederhendler, 2000). Retrospective, case-control research suggests childhood internalizing factors (i.e., childhood obsessive compulsivity, neuroticism, and perfectionism) are associated with higher risk for both AN and BN (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003). Additionally, core negative beliefs about weight and body image, often a result of familial influence or media exposure, may further perpetuate the development of eating pathologies (Grabe, Ward, & Hyde, 2008; Linville, Stice, Gau, & O'Neil, 2011; Vitiello & Lederhendler, 2000). An explanatory model for the development of eating disorders created by Southgate and colleagues (2005) utilized neuroscience data and suggested eating disorder onset can be triggered by stressful life events that may hinder or disrupt typical cognitive maturational processes.

Social psychological risk factors are well-evidenced predictors of eating disorders onset (Fairburn, Cooper, Doll, & Davies, 2005; Killen et al., 1996; Patton et al., 1999;

The McKnight Investigators, 2003) and will therefore be the risk factors targeted in this study. Research suggests drive for thinness (Fabian & Thompson, 1989), body dissatisfaction (Paxton, Neumark-Sztainer, Hannan, & Eisenberg, 2006), and disordered eating behaviors (Meno, Hannum, Espelage, & Douglas Low, 2008; Suisman, Slane, Burt, & Klump, 2008) all contributed to the development of eating disorders. Individuals reporting weight concerns and elevated negative affectivity also exhibit increased prevalence of eating pathologies (Killen et al., 1996; Meno et al., 2008; Stice, Marti, & Durant, 2011).

The current societal ideals for attractiveness in Western culture overemphasize the importance of thinness, and contribute to thin-ideal internalization, or the extent to which one cognitively subscribes to societal beauty ideals (Thompson & Stice, 2001). Thin body preoccupation and internalization predicts body dissatisfaction which in turn promotes dietary restraint and negative affect, and increases the risk for eating pathology (Stice, 2002; Thompson & Stice, 2001). Self-reported social pressure to be thin and thin body preoccupation has been shown to predict onset of threshold or subthreshold BN and BED for adolescent females (The McKnight Investigators, 2003).

Body dissatisfaction is one of the most consistent and robust risk factors for eating disorder onset (Stice & Shaw, 2002; Stice, 2002). Body dissatisfaction often leads to dietary restraint and compensatory behaviors (e.g., vomiting, excessive exercise), motivated by a belief that weight loss will promote body satisfaction (Stice & Shaw, 2002). Body dissatisfaction may also increase negative affect as a result of individuals using appearance as an evaluative dimension of self-worth. Body dissatisfaction

predicted onset of binge eating and bulimic pathology for adolescent females (The McKnight Investigators, 2003).

The dietary restraint model supports the theory that dietary restraint fosters eating pathology because caloric deprivation increases the risk of binge eating behaviors over time (Hawkins & Clement, 1984; Stice, 2002). Prospective studies also suggest self-reported dieting may promote negative affect and lead to an increased likelihood of binge-eating and bulimic pathology (Ricciardelli & McCabe, 2001; Stice, 2002; The McKnight Investigators, 2003).

Mood and anxiety disorders have long been associated with the development of eating concerns (Leon, Fulkerson, Perry, Keel, & Klump, 1999; McCabe & Ricciardelli, 2006; Spoor et al., 2006), and are the most commonly comorbid disorders for individuals suffering from eating disorders (Touchette et al., 2011). Negative affect has been independently established as a strong predictor of future eating pathology (Measelle, Stice, & Hogansen, 2006; Wertheim, Koerner, & Paxton, 2001; Wichstrøm, 2000), acting as a causal maintenance factor for binge eating behaviors (Stice, 2002). A study aimed at determining where eating pathology fits within a structural model of diagnostic taxonomy concluded that eating disorders are a variant of internalizing disorders, rather than their own latent class of disorders (Forbush et al., 2010). This finding is consistent with literature which documents the high rates of comorbidity between depression, anxiety, and eating disorders (Hudson et al., 2007; Touchette et al., 2011).

Among adolescents, eating disorders are significantly more common among individuals with a mood or anxiety disorder, versus those without (Zaider, Johnson, & Cockell, 2000). Elevations of negative affect during early adolescence has also been

shown to predict eating disorder symptoms during early adulthood (Johnson, Cohen, Kotler, Kasen, & Brook, 2002). Individuals who struggle to modulate negative mood states may be at higher risk of eating disorder development (Whiteside et al., 2007). Difficulties making sense of emotional states, a basic but important skill, and maladaptive emotion regulation strategies have both been linked to binge eating. Binge-eating individuals report at least half of their binges are driven by affect, rather than hunger (Greeno, Wing, & Shiffman, 2000; Wilson, Fairburn, & Agras, 1997).

The aforementioned social psychological eating disorder risk factors have generally shown modest, univariate effects of predicting onset or maintenance of eating pathology. Multivariate models are stronger predictors of eating disorder onset because the cumulative, interactive effect of risk factors more adequately captures theorized models of eating disorder development and provides greater explanatory power (Stice, 2002), and these models have been found to predict eating disorder onset in prospective etiologic studies with community samples (e.g., Fairburn & Bohn, 2005; Killen et al., 1996; Stice, Akutagawa, Gaggan, & Agras, 2000; Stice, Marti, & Rohde, 2013; Striegel-Moore et al., 2007). A recent study of eating disorder prevention response concluded that these same risk processes appear to be operating among individuals who enroll in selective eating disorder prevention programs (Horney, Stice, & Rohde, 2015).

Eating disorder prevention

The implementation and dissemination of efficacious eating disorder prevention is critical to decrease eating disorder symptoms and reduce future onset of disorders. A number of eating disorder prevention programs have effectively reduced social psychological risk factors for participants (Fairburn, Cooper, Doll, & Davies, 2005;

Killen et al., 1996; Patton et al., 1999; Stice, Marti, & Durant, 2011; Striegel-Moore et al., 2007). Several prevention programs have produced significant reductions in eating disorder symptoms through at least 6-month follow-up in a single trial (e.g., Jones et al., 2008; McVey, Tweed, & Blackmore, 2007; Neumark-Sztainer, Butler, & Palti, 1995; Stewart, Carter, Drinkwater, Hainsworth, & Fairburn, 2001). However, considerably more empirical support has emerged for the *Body Project* (Stice & Presnell, 2007), a selective dissonance-based eating disorder prevention program in which young women with body image concerns voluntarily critique the thin ideal in verbal, written, and behavioral exercises (Stice, Mazotti, Weibel, & Agras, 2000).

Grounded in the theory that humans seek to maintain consistency between their words, thoughts and actions, criticizing the thin ideal during this group-based intervention is believed to produce a motivational drive for participants to reduce their subscription to this unrealistic beauty ideal (Stice et al., 2000). Decreased pursuit of the thin ideal theoretically reduces body dissatisfaction, dietary restraint, negative affect, eating disorder symptoms, and risk for future eating disorder onset for *Body Project* participants (Stice et al., 2000).

Efficacy trials show that the *Body Project* produces greater reductions in eating disorder risk factors (e.g., thin-ideal internalization, body dissatisfaction, dietary restraint, negative affect), eating disorder symptoms, functional impairment, and eating disorder onset over a 3-year follow-up relative to assessment-only control conditions and three alternative interventions (e.g., Stice et al., 2008, 2000; Stice, Rohde, Durant, & Shaw, 2012; Stice, Shaw, Burton, & Wade, 2006). Additionally, independent efficacy trials have found that, relative to assessment-only control conditions and alternative

interventions, dissonance-based eating disorder prevention programs produce significantly larger reductions in eating disorder risk factors and symptoms (Becker, Smith, & Ciao, 2005; Halliwell & Diedrichs, 2014; Matusek, Wendt, & Wiseman, 2004; Mitchell, Mazzeo, Rausch, & Cooke, 2007). The *Body Project*, the only eating disorder prevention program to produce positive intervention effects that have been both independently replicated and significantly outperformed credible alternative interventions, is the only eating disorder prevention program to meet the American Psychological Association's (1995) designation as an efficacious intervention. A study by Green and colleagues (2005), compared participants assigned to high-dissonance (with manipulations to reinforce high level of effort expenditure, public attitude expression, and voluntary participation) versus low-dissonance (low level effort, belief that attitudes would remain private, perception that participation was not entirely voluntary) versions of the *Body Project*. Participants in the high-level dissonance program showed significantly greater reductions in eating disorder symptoms, suggesting the level of dissonance induction correlates to the strength of intervention effects (Green, Scott, Diyanakova, & Gasser, 2005; McMillan, Stice, & Rohde, 2011).

Supporting the intervention theory for this prevention program, reductions in thin-ideal internalization appear to mediate the effects of the *Body Project* on changes in the other outcomes (Seidel, Presnell, & Rosenfield, 2009; Stice, Presnell, Gau, & Shaw, 2009). Classification tree analysis determined that *Body Project* participation reduced the risk conveyed by the most potent eating disorder risk factor in that trial - denial of the costs of pursuing the thin ideal (Stice, Rohde, Gau, & Shaw, 2012). Participants who denied the costs of pursuing the thin ideal but completed the *Body Project* showed an

eating disorder incidence of 0% over 3-year follow-up, versus 18% for those who completed two alternative interventions, and 50% for assessment-only controls. In addition, an independent study concluded that participation in the *Body Project* eliminated the negative effect of exposure to supermodels on body dissatisfaction in young adolescent girls observed in controls (Halliwell & Diedrichs, 2014).

Prevention program response

Muller and Stice (2013) examined factors hypothesized to moderate the effects of the *Body Project* including thin-ideal internalization, body dissatisfaction, eating disorder symptoms, and participant age. The moderation study concluded that the prevention program produced stronger effects for individuals who began with elevated thin-ideal internalization and eating disorder symptoms (Müller & Stice, 2013). In addition, participants in late adolescence or early adulthood appeared to have larger reductions in body dissatisfaction than younger participants after completing the *Body Project* (Müller & Stice, 2013). Despite this prevention program being effective for a range of individuals, these results suggested the prevention program may be most beneficial for particular subgroups.

A second *Body Project* moderation study investigated both general and program-specific factors hypothesized to influence the effects of the prevention program on bulimic pathology through 1-year study follow-up (Stice, Marti, Shaw, & O'Neil, 2008). Results suggested the *Body Project* effect was amplified for participants with increased baseline body dissatisfaction, bulimic symptoms, and thin-ideal internalization, again suggesting that the prevention intervention effects are strongest for higher risk individuals (Stice, Marti, Shaw, et al., 2008).

Although research has examined factors that moderate the effects of the *Body Project* (Müller & Stice, 2013; Stice et al., 2008), only one study to date has examined participants who received the *Body Project*, but later developed the psychiatric conditions the program was designed to prevent (Horney et al., 2015). Horney and colleagues examined factors that distinguished participants who completed the *Body Project* but still went on to develop a *DSM-5* eating disorder, from those who completed the intervention and remained eating disorder-free during follow-up. Despite the fact that the *Body Project* has produced medium to large effect sizes, it is important to investigate ways to improve the effects of this prevention program and this study uncovered factors that interfere with an effective prevention process.

Results of this study provided support for the hypothesis that the small subset of participants who complete the *Body Project*, but later develop an eating disorder, began the prevention program with elevated levels of three of the four examined risk factors (thin-ideal internalization, body dissatisfaction, and negative affect) and elevated eating disorder symptoms. The results of this study suggested that the same risk factors that have predicted eating disorder onset in community samples of young women also predicted eating disorder onset in this high-risk sample, whether participants receive the *Body Project* intervention or not.

It is important to note that the results of this first study, taken in conjunction with the earlier moderation studies (e.g., Müller & Stice, 2013; Stice et al., 2008), suggest that participants with the greatest initial eating disorder symptoms are in general more likely to show the strongest *reductions* in symptoms *and* be at *highest risk* for later eating disorder development. This is explained by high risk participants having a greater

opportunity to show larger reductions in symptoms while a subset of participants are simultaneously still at elevated risk of subsequently developing an eating disorder due to these individuals starting the program as more symptomatic at baseline.

Study purpose

The present study built upon results and implications of the first examination of *Body Project* response (Horney et al., 2015). This first study of suboptimal *Body Project* response provided unique evidence that individuals who began the *Body Project* with elevated negative affect were at greater risk for onset of eating disorders (Horney et al., 2015). This suggests it would be useful to refine this prevention program so that it produces larger reductions in this outcome; as, unlike the other examined risk factors, the prevention program does not currently address negative affect directly. However, it was difficult to interpret the degree to which negative affect contributed to increased eating disorder symptom presentation, given the dichotomous outcome variable used in the original study (e.g., “*DSM-5* eating disorder diagnosis” versus “no eating disorder diagnosis”).

For the present study, our first aim was to delineate the heterogeneous eating disorder symptom trajectories among *Body Project* versus control group participants during 3-year study follow-up by examining separate groups by study condition. Our research questions for this first aim were: a) Are there multiple patterns of change in the outcome variable? b) How many patterns of change are there in the outcome? And c) what is the shape of the change over time?

We hypothesized that control group participants would exhibit response patterns that replicated theorized eating disorder symptom development (i.e., differential groups

representing increasing, stable, and variable eating disorder symptom trajectories) (Fairburn et al., 2000; Stice et al., 2002). We hypothesized that the response trajectories of *Body Project* participants would be more likely to fall into stable and decreasing trajectory groups. We expected response patterns to replicate theorized responses to the *Body Project* with the majority of participants falling into groups characterized by reducing or low stable eating disorder symptom trajectories. Given the empirical evidence supporting the *Body Project* as an efficacious eating disorder prevention program, *Body Project* participants would be less likely to fall into trajectory groups characterized by increasing, high stable, or variable eating disorder symptomology over time.

Additionally, we examined contextual differences between trajectory groups because research suggests the membership in different developmental groups is correlated with different risk factor antecedents. The second aim of the study was to determine the predictive role of eating disorder risk factors on qualitatively distinct developmental pathways of eating disorder symptoms: specifically examining a) What predicts membership in each trajectory group? b) What are the characteristics that differ between different groups? And c) does the predictive impact of risk factors differ for *Body Project* versus control group participants?

We hypothesized that the predictor variables for high risk groups will be associated with high levels of theorized multivariate models of eating disorder risk. Expected healthy trajectories (decreasing, and low stable symptoms) would be associated with low baseline negative affect. We also hypothesized that adverse *Body Project*

trajectories (increasing, high and stable, or variable symptom trajectories) would be associated with high baseline negative affect and elevated thin-ideal internalization.

The third aim of the study was to examine how the probability of trajectory group membership could be impacted by completing the *Body Project*. We examined group-based trajectories of the full dataset, to determine an optimal model of developmental trajectories. We hypothesized that events that occurred during the course of trajectory (i.e., eating disorder prevention) would alter the developmental course of the outcome of interest (i.e., eating disorder symptoms). We hypothesized that the control group condition would predict high-risk trajectories, and the *Body Project* condition would predict decreasing, low-risk trajectories.

CHAPTER II

METHODS

Participants and procedure

To achieve an adequate sample of participants to aid in the identification of homogenous clusters of developmental trajectories, data was merged from three randomized controlled trials ($N = 960$) that used similar research designs and methodologies (*Efficacy Trial*, $n = 246$; *High School Effectiveness Trial*, $n = 406$, and *College Effectiveness Trial*, $n = 408$). Study participants were young women and adolescent girls with body image concerns recruited for selective prevention programs. Analyses included participants assigned to both the *Body Project* condition ($n = 471$, M age = 18.58, $SD = 4.61$, M BMI [kg/m²] = 24.21, $SD = 5.22$), and the educational control condition ($n = 489$, M age = 18.52, $SD = 4.67$, M BMI [kg/m²] = 24.18, $SD = 5.33$). The sample was 67% Caucasians, 9% Latinos, 12% Asian/Pacific Islanders, 4% African Americans, less than 1% American Indians/Alaska Natives, and 7% who specified other/mixed racial heritage.

Dissonance intervention. Participants voluntarily engaged in the *Body Project*, a 3-hour (*Efficacy Trial*) or 4-hour (*High School Effectiveness Trial*; *College Effectiveness Trial*) dissonance intervention, wherein women with body image concerns participate in verbal, written, and behavioral exercises in which they critique the thin ideal. In-session and homework activities ask participants to write essays and conduct role-plays that are counter-attitudinal and argue against the societal thin ideal. In the 4-hour intervention (*High School Effectiveness Trial*; *College Effectiveness Trial*), during session 1, participants collectively defined the thin ideal, discussed costs of pursuing this ideal, and

were assigned home exercises (e.g., writing an essay about the costs associated with pursuing the thin ideal). In session 2, participants were asked to debrief each home exercise, engaged in role-plays in which they attempted to discourage facilitators from pursuing the thin ideal, and were assigned additional home exercises (e.g., generate a top-10 list of things young women can do to challenge the thin ideal). In session 3, they debriefed home exercises, conducted a role-play in which they challenged thin ideal statements, discussed personal body image concerns, and were assigned more home exercises (e.g., engage in a behavior that challenges their body image concerns). In session 4, participants debriefed home exercises, challenged subtle “fat-talk” in role-plays, planned how to respond to future anticipated pressures to be thin, and were assigned exit home exercises (e.g., write a letter to a younger adolescent girl about avoiding body image concerns). Participants received emails and text messages between sessions to remind them of the next group session and encourage them to complete the homework. Participants in the *Efficacy Trial* received a 3-hour version of the *Body Project* which included similar, slightly abbreviated interventions as compared to the 4-hour intervention.

Educational control condition. Control participants in all three trials received a two-page brochure, produced by the National Eating Disorders Association in 2002, which described negative and positive body image, noted the correlation between negative body image and eating disorder risk, and provided 10 steps for developing a positive body image. Control participants were mailed the educational brochures after randomization, and following the baseline assessment.

Measures

Survey interview and questionnaires can be found in Appendix A.

Demographic variables. Participants provided demographic information about age, ethnicity, father's education, and mother's education.

Thin-ideal internalization. The Ideal-Body Stereotype Scale-Revised (IBSS-R; Stice, Fisher, & Martinez, 2004) assessed thin-ideal internalization. Items used a response format ranging from 1 = *strongly disagree* to 5 = *strongly agree*. Items were averaged for this scale and scales described below to create variable composite scores. This scale has shown internal consistency ($\alpha = .91$), 2-week test-retest reliability ($r = .80$), predictive validity for bulimic symptom onset, and sensitivity to detecting intervention effects (Stice et al., 2008).

Body dissatisfaction. Items from the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid, Walster, & Bohrnstedt, 1973) assessed dissatisfaction with 9 body parts using a response scale ranging from 1 = *extremely satisfied* to 6 = *extremely dissatisfied*. This scale has shown internal consistency ($\alpha = .94$), 3-week test-retest reliability ($r = .90$), predictive validity for bulimic symptom onset, and sensitivity to detecting intervention effects (Stice et al., 2008).

Dieting. The Dutch Restrained Eating Scale (DRES; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) assessed the frequency of dieting behaviors using a response scale ranging from 1 = *never* to 5 = *always*. The DRES has shown internal consistency ($\alpha = .95$), 2-week test-retest reliability ($r = .82$), convergent validity with self-reported caloric intake (but not objectively measured caloric intake), predictive

validity for bulimic symptom onset, and sensitivity to detecting intervention effects (Stice et al., 2006; Stice, Sysko, Roberto, & Allison, 2010; van Strien et al., 1986).

Negative affect. Negative affect was measured with the negative affect subscale from the Positive Affect and Negative Affect Scale—Revised (PANAS-X; Watson & Clark, 1992) for *Efficacy Trial*, the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977) for *High School Effectiveness Trial*, and the Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988) for *College Effectiveness Trial*. The Sadness, Guilt, and Fear/Anxiety subscales from the PANAS-X assessed the extent to which participants had felt negative emotional states using a response format ranging from 1 = *very slightly or not at all* to 5 = *extremely*; it has shown internal consistency ($\alpha = .95$), 3-week test-retest reliability ($r = .78$), convergent validity, and predictive validity for bulimic symptom onset (Stice et al., 2006). The CES-D asked participants to assess depressive symptoms such as “I have been feeling pretty down and unhappy this week” on a 4-point scale (0 = *never* to 3 = *most of the time*). The CES-D has shown internal constancy ($\alpha = .8$ to $.9$), and test-retest stability ($r = .50$ to $.60$; Roberts, Lewinsohn, & Seeley, 1991). The 21-item BDI asked participants to select among four responses reflecting the increasing levels of symptom severity (0 = *no symptom present* to 3 = *severe symptom present*). The BDI has shown internal consistency ($\alpha = .73$ to $.95$), test-retest reliability ($r = .60$ to $.90$), and convergent validity with clinician ratings of depressive symptoms ($r = .75$; Beck et al., 1988). To allow analyses of combined data from the three trials, z-transformed versions of PANAS-X, CES-D, and BDI variables were used, a justified examination given the substantial correlation ($r =$ between $.58$ and

.75) among these three measures of negative affect (Tellegen, Watson, & Clark, 1988; Watson & Clark, 1992).

Eating disorder symptoms. The semi-structured Eating Disorder Diagnostic Interview (EDDI) assessed eating disorder symptoms over the past 12 months, or since the last interview. Participants reported on eating disorder symptoms on a month-by-month basis, over the entire 3-year follow-up period. Items assessing symptoms in the past month were summed to form a symptom composite at each assessment. This symptom composite has shown internal consistency ($\alpha = .92$), inter-rater agreement ($r = .93$), 1-week test-retest reliability ($r = .90$), sensitivity to detecting effects from eating disorder prevention and treatment interventions, and predictive validity for future onset of depression (Burton & Stice, 2006; Stice, Rohde, Gau, & Shaw, 2009).

DSM-5 eating disorders. The EDDI was also used to assess diagnostic criteria for *DSM-5* eating disorders. Responses determined whether participants met criteria for AN, BN, BED, and feeding or eating conditions not elsewhere classified, which included atypical AN, subthreshold BN, subthreshold BED, and PD, at pretest and at any time during the 3-year follow-up (operationalized in Stice, Marti, & Rohde, 2013). EDDI *DSM-5* eating disorder diagnoses have shown 1-week test-retest reliability ($r = .79$) and inter-rater agreement ($r = .75$; Stice, Marti, & Rohde, 2013). EDDI eating disorder diagnoses have also shown sensitivity to detecting intervention effects and functional impairment, as well as predictive validity for future depression onset (Burton & Stice, 2006; Seeley, Stice, & Rohde, 2010; Stice et al., 2008; Stice, Marti, & Rohde, 2013).

Data collection

Participants completed assessments at pretest, posttest, 12, 24, and 36 months following the dissonance intervention or educational control.

Data analytic method

Descriptive statistics including the mean, standard deviation, and frequency distributions were examined for all study variables to evaluate normal distribution, skew, and univariate outliers using SPSS version 22 (IBM Corp, 2013). Standardized skew index values between -3.0 and $+3.0$ were considered to be within normal limits, and a standardized kurtosis index of -10.0 to $+10.0$ was used to evaluate normality. We tested the covariance of demographic variables (age, ethnicity, parental education) and study trial (*Efficacy Trial*, *High School Effectiveness Trial*, and *College Effectiveness Trial*) on eating disorder diagnosis during study follow-up (a transformed variable using eating disorder symptom scores) to determine whether baseline differences were present and should be controlled for in subsequent analyses. A meta-analysis of eating disorder risk and maintenance factors concluded that body mass should be considered a risk factor for perceived pressure to be thin, body dissatisfaction, and dietary restraint (Stice, 2002). However, there is not conclusive evidence to suggest body mass is a risk or maintenance factor for eating disorder symptoms (Wichstrom, 2000). As a result, body mass was included in preliminary analyses to test for covariance. Bivariate correlations using Pearson's r were examined to screen for collinearity among variables.

Patterns of missingness were analyzed with SPSS version 22.0 to identify the distribution of missingness, e.g., missing completely at random, missing at random, missing not at random (Schafer & Graham, 2002). In order to model these data despite

patterns of missingness, maximum likelihood (ML) estimation was used as it is robust to missingness and some degree of nonnormality (Enders, 2001).

Group-based trajectory modeling (GBTM), a specialized form of finite mixture modeling, was used to identify clusters of individuals following distinct developmental trajectories of eating disorder symptoms across 3-year study follow-up (Nagin et al., 1999). This statistical approach has been recommended for use with psychological processes, such as eating disorders, that do not vary regularly across the population (Nagin & Odgers, 2010). GBTM has become increasingly common in clinical research given the ability to assess the developmental course of psychological disorders as well as the heterogeneous response to clinical interventions (Nagin & Odgers, 2010). GBTM allows for the depiction of qualitatively distinct developmental progressions, or courses of an outcome, over time (Nagin & Odgers, 2010). GBTM provides an empirical means of identifying clusters of individuals whose trajectory of development may be typical or atypical. This is in contrast to growth curve modeling, which assumes all individuals follow a similar trajectory of development, or growth mixture modeling, which allows for two or more response patterns but assumes a mean pattern of development (Nagin & Odgers, 2010). GBTM also allowed for the identification of key characteristics and behaviors (i.e., presence or absence of eating disorder risk factors) that distinguish individuals within one developmental pathway from those in another.

GBTM tested study hypotheses using PROC TRAJ, a user-written SAS add-on software (Jones, Nagin, & Roeder, 2001; Jones & Nagin, 2007). Longitudinal data analysis with a group-based approach was appropriate for this study given theory of eating disorder development which suggests heterogeneous pathological development

(Tyrka, Graber, Brooks-Gunn, 2000). This study used eating disorder symptom composite score as the outcome variable. This is in contrast to the first study of *Body Project* response (Horney et al., 2015), which used a dichotomous, categorical variable (e.g., eating disorder yes or no) to assess outcome. The continuous outcome variable used in this study allowed for the examination of variability in individual change over time. Time in the GBTM was indexed as “time since baseline assessment.”

GBTM estimated the number of participants in different trajectory subgroups. When using this method, we assumed the target population could be accurately described as a mixture of distinct groups defined by unique developmental trajectories. A censored normal (CNORM) model identified the number of qualitatively distinct groups that best fit the continuous, normally distributed data (B. L. Jones et al., 2001). We specified a CNORM model because it is useful for psychometric data of a selected preventive intervention sample, where symptom counts may be clustered at the bottom, top, or both end of a scale. When using this method, one assumes that the target population can accurately be described as a mixture of distinct groups defined by their developmental trajectories.

GBTM trajectory groups are comprised of individuals following approximately the same developmental course of the outcome variable, and groups in GBTM are not immutable. Nagin and Odgers (2010) provide a set of general principles to encourage transparent and clear reporting of GBTM results and these principles informed the statistical reporting in this study. Formal statistical criteria, fit indices, and substantive usefulness of the model as it related to the study research questions supported the choice of the number of groups included in the final model. The BIC becomes increasingly less

negative as fit of the group structure improves. The BIC statistic was supplemented by additional recommended statistics for the GBTM. Specifically, a group structure was identified as having good fit if the average probability of group membership was greater than 0.70 for each group and when the Akaike Information Criteria (AIC) became increasingly less negative. Next, PROJ TRAJ was used to select the shape of each group's trajectory over time as GBTM can model both linear and non-linear trajectories within the same model. A combination of study hypotheses based from substantive knowledge regarding eating disorder symptom development, and statistical inference (difference in Bayesian information criteria BIC between two models), was used to decide the shape of each group's trajectory. The 'best' models were selected based on theory and to select the simplest model that best describes the data.

To test the second study hypothesis, GBTM was expanded to include eating disorder risk factor predictor variables (thin-ideal internalization, body dissatisfaction, dietary restraint, and negative affect). Using the strongest models determined by our first research aim, we tested whether baseline predictors influenced trajectory group membership. Demographic variables (age, ethnicity, parental education) were tested as covariates, but not included in the final models as there were no significant group differences. Predictive value of study trial (*Efficacy Trial*, *High School Effectiveness Trial*, and *College Effectiveness Trial*) was also examined for each group trajectory. *DSM-5* eating disorder diagnosis was utilized as a validation variable for the latent trajectory analyses, and assessed whether participants in increasing eating disorder symptom trajectory groups exhibited more prevalent eating disorder diagnoses.

CHAPTER III

RESULTS

Preliminary analyses

Participant characteristics. Descriptive statistics for all demographic, risk factor, and outcome variables were examined using SPSS version 22.0 (IBM Corp, 2013). See table 1 for the means, standard deviations, skew and kurtosis index for all study variables. There were no significant differences of demographic variables between study trial participants (*Efficacy Trial*, *High School Effectiveness Trial*, and *College Effectiveness Trial*). A univariate analysis of variance (ANOVA) was also conducted to examine whether there was a significant difference between study trials in relation to baseline eating disorder symptoms. All assumptions of the analysis were met with the exception of homogeneity of variances ($p < .05$, Levene's test). Weighted ANOVA results (Welch, 1951) were used to account for difference in variance. The test revealed a statistically significant difference between study trials, $F(2, 955) = 21.75, p = .001$. Post-hoc tests revealed *Efficacy Trial* participants ($M = 16.84, SD = 16.07$) had significantly higher eating disorder symptoms at baseline than *High School Effectiveness Trial* ($M = 10.04, SD = 11.92$) or *College Effectiveness Trial* ($M = 11.84, SD = 12.61$) participants. Preliminary analyses also determined a significant difference at pretest of eating disorder symptoms by condition, with *Body Project* participants exhibiting significantly higher eating disorder symptoms at baseline than their control group counterparts, $F(1, 956) = 8.79, p = .003$.

Analyses confirmed that demographic factors (i.e., age, ethnicity, parental education) and pretest BMI did not predict eating disorder onset during 36-month follow-up, and were not controlled for in subsequent analyses.

Among the 471 *Body Project* participants, 67 (14%) met criteria for a DSM-5 eating disorder at some point during 3-year follow-up. Among these 67 *Body Project* participants, the incidence of DSM-5 diagnoses was 3 for AN, 16 for BN, 19 for BED, 9 for atypical AN, 30 for subthreshold BN, 21 for subthreshold BED, and 14 for purging disorder. Thirty-four *Body Project* participants were diagnosed with comorbid eating disorder diagnoses during follow-up. Among the 489 control participants, 65 (13%) met criteria for a DSM-5 eating disorder onset during 3-year follow-up. The incidence of DSM-5 diagnoses for these 65 control participants was 2 for AN, 22 for BN, 22 for BED, 6 for atypical AN, 32 for subthreshold BN, 18 for subthreshold BED, and 15 for purging disorder. Thirty-two control participants had comorbid eating disorder diagnoses during follow-up.

Correlations. A bivariate correlation analysis examined the full dataset to determine relations between baseline risk factors and eating disorder symptoms across time points (table 2). Table 2 presents Pearson r bivariate correlations for eating disorder symptoms through 36-month follow-up, and baseline eating disorder risk factor variables. As expected, results indicated moderate to low correlations between eating disorder risk factors and moderate to strong positive correlations between the five waves of eating disorder symptoms.

Table 1.

Means, Standard Deviations, Reliability, and Skew of Study Variables by Condition

Variable	Sample	Skew	Kurtosis	Condition	
				Body Project	Control
1. Eating Disorder Symptoms W1	12.54(12.61)	2.34(.08)	7.21(.16)	13.40(13.69)	10.83(10.81)
2. Eating Disorder Symptoms W2	8.47(9.80)	3.01(.08)	12.41(.16)	7.52(9.27)	8.67(8.73)
3. Eating Disorder Symptoms W3	9.45(14.58)	7.96(.08)	113.83(.16)	8.75(11.93)	9.20(10.72)
4. Eating Disorder Symptoms W4	8.75(11.19)	4.32(.08)	34.62(.16)	8.17(11.75)	8.91(10.55)
5. Eating Disorder Symptoms W5	7.98(9.79)	3.32(.08)	18.06(.17)	7.69(10.08)	8.21(9.33)
6. Thin-ideal Internalization W1	3.70(0.57)	-0.43(.08)	1.12(.16)	3.70(0.60)	3.70(0.53)
7. Body Dissatisfaction W1	3.06(0.82)	-0.10(.08)	-0.32(.16)	3.09(0.85)	3.03(0.79)
8. Dietary Restraint W1	2.66(0.90)	0.11(.08)	-0.67(.16)	2.72(0.90)	2.60(0.89)
9. Negative Affect W1	0.01(1.00)	1.02(.08)	0.77(.16)	0.01(1.01)	0.03(0.99)

Note. Sample = total sample; Skew and Kurtosis Indices reported with standard errors; All means reported with standard deviations.

Table 2.

Correlation Matrix of Eating Disorder Symptoms W1 through W5 and Baseline Eating Disorder Risk Factors

Variables	1	2	3	4	5	6	7	8
1. Eating Disorder Symptoms W1								
2. Eating Disorder Symptoms W2	.64**							
3. Eating Disorder Symptoms W3	.44**	.59**						
4. Eating Disorder Symptoms W4	.46*	.52**	.60**					
5. Eating Disorder Symptoms W5	.46**	.49**	.48**	.68**				
6. Thin-Ideal Internalization	.25**	.21**	.18**	.21**	.22**			
7. Body Dissatisfaction	.15**	.07*	-.01	.00	.04	-.08**		
8. Dietary Restraint	.47**	.40**	.24**	.30**	.31**	.37**	-.02	
9. Negative Affect	.29**	.24**	.19**	.20**	.17**	.16**	.17**	.18**

Note. Correlations were calculated using Pearson's r . p -values are two-tailed. * $p < .05$. ** $p < .01$.

Attrition analysis and treatment of missing data. The 11% of participants who did not complete the assessments through 3-year follow-up did not differ from the remaining 89% of participants on any demographic factors, baseline study variables, or study trial. Attrition did not differ across study condition, suggesting attrition was not systematic and scores were missing completely at random (MCAR). Analyses used in this study were able to accommodate data MCAR by using maximum likelihood (ML) estimation. This approach made use of all available data, and provided unbiased parameter estimates that are more efficient and accurate than list-wise deletion or alternative imputation approaches (Nagin & Odgers, 2010).

Group-based trajectory modeling

We conducted three trajectory analyses to examine group-based differences with the control group, *Body Project* group, and combined, full dataset. We used SAS Version 9.3 PROC TRAJ (censored normal; Jones, Nagin, & Roeder, 2001) to model the developmental trajectories as a function of five measurement waves. The highest order polynomial we tested was quadratic. For each analysis, we first specified 2 groups and then tested a series of models in which we increased the number of groups and used the BIC, AIC, the average probability of group membership, and the group sample size to evaluate model fit (Nagin, 2005). We utilized a backward elimination strategy in which we removed whichever model had the largest p -value, prioritized the simplest model, and selected the model with the most optimized fit indices (Table 3). We assume that within each trajectory group, self-reported eating disorder symptoms at each time-point is Poisson distributed. We further assume that the logarithm of the expectation of this Poisson variable follows a linear function of time for each trajectory group. Model

estimation requires specification of the number of trajectory groups and we determined the optimal number of trajectories of eating disorder symptoms for the control group and *Body Project* group was 3 and 2 groups, respectively. The BIC and AIC values became progressively less negative from the optimal-group solutions compared to higher-number group solutions, and the optimal-group solutions did not include groups with very small sample sizes. Additionally, omitted, higher-number models did not include trajectory groups with substantively different trajectories from those apparent in the optimal-group solution.

Table 4 reports the estimates of group membership for the three-group solution for control participants. The three-group solution for control participants produced less negative BIC and AIC values, and had average group membership probabilities of .91 to .97. The resulting trajectories are displayed in Figure 1. As shown in Figure 1, 362 of the 489 total participants (74% of the sample) in Trajectory #1 reported low, stable levels of eating disorder symptoms at each of the five waves of data collection, and 13.5% of the participants in this group trajectory exhibited onset of an eating disorder during study follow-up. A group of 99 participants (Trajectory #2; 20.2% of the sample) reported moderate, stable levels of eating disorder symptoms during each of the five measurement waves with 12% of this group exhibiting onset of an eating disorder during study follow-up. And the smallest observed group of 28 participants (Trajectory #3; 5.7% of the sample) endorsed high levels of eating disorder symptoms at baseline. Symptom levels varied over time but remained high during the course of study follow-up. This

Table 3.

Bayesian Information Criteria (BIC) Scores, Changes in BIC, and Percentages of Participants in the Smallest Group for Trajectory Models

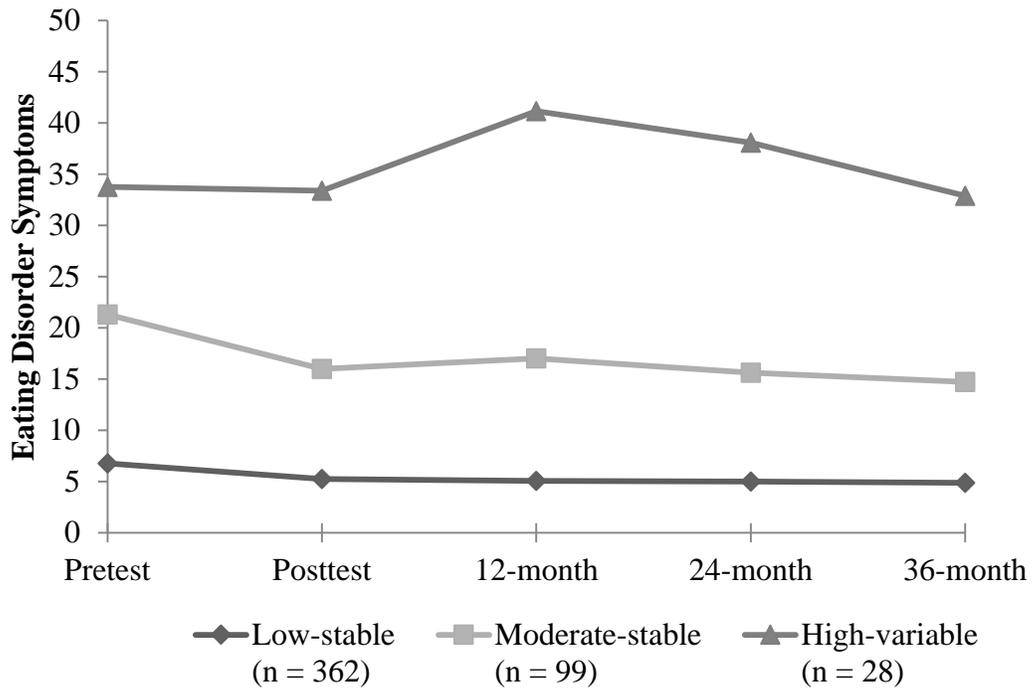
	BIC	Change in BIC	AIC	Size of smallest group (%)
Control Group				
One group	-8715.10	-	-8703.60	100.00
Two groups	-8274.57	-440.53	-8251.56	11.15
Three groups	-8143.39	-131.18	-8108.87	5.78
Four groups	-8158.90	15.51	-8112.87	< 0.00
Body Project				
One group	-8692.29	-	-8680.87	100.00
Two groups	-8430.53	-261.76	-8407.69	6.23
Three groups	-8445.96	15.43	-8411.69	< 0.00
Full Dataset				
One group	-17427.62	-	-17414.77	100.00
Two groups	-16760.60	-667.02	-16734.90	9.75
Three groups	-16456.83	-303.77	-16418.28	1.02
Four groups	-16473.69	16.86	-16422.28	< 0.00

Table 4.

Three-Group Control Trajectory Model Group Probability and Group Estimates

Variable	Trajectory 1 Low-Stable	Trajectory 2 Moderate- Stable	Trajectory 3 High- Variable
Group probability (π_j)	.7324912	.2096950	.0578138
Pretest eating disorder symptoms (λ^j_1)	7.22	20.59	32.39
Posttest eating disorder symptoms (λ^j_2)	6.40	17.83	36.95
12-month eating disorder symptoms (λ^j_3)	5.94	16.02	36.68
24-month eating disorder symptoms (λ^j_4)	5.79	15.17	37.58
36-month eating disorder symptoms (λ^j_5)	5.96	15.24	33.65

Figure 1. GBTM of eating disorder symptoms for control group



developmental course of high, variable eating disorder symptoms is one most reflective of chronic eating disorder pathology and 14.3% of this group exhibited onset of an eating disorder during study follow-up.

Table 5 reports the estimates of group membership for the two-group solution for *Body Project* participants. The optimal, two-group solution for *Body Project* participants had average group membership probabilities of .95 to .99, and large enough group sample sizes to ensure model fit and stability. As shown in Figure 2, 442 of the 471 total participants (Trajectory #1; 93.8% of the sample) reported low eating disorder symptoms at pretest that decreased gradually following *Body Project* participation. The magnitude of the change for this group can be understood in terms of time-specific scores. At pretest, the mean eating disorder symptom score for the low-stable group was $M = 11.70$, at posttest, the mean score was $M = 6.24$, and remained stable through 36-month follow-up, $M = 6.39$. Decrease in eating disorder symptoms from pretest to 36-month follow-up was statistically significant, $t(429) = 12.54, p < .001$. Only 11% of this group exhibited onset of an eating disorder during study follow-up.

The remaining 29 participants (Trajectory #2; 6.2%) reported high levels of eating disorder symptoms at pretest, a decrease in symptoms following the *Body Project* prevention intervention, and gradually decreasing eating disorder symptomology during the subsequent follow-up time-points. The decrease in eating disorder symptoms from pretest to posttest was statistically significant, $t(28) = 3.01, p = .005$. There was a statistically significant decrease in eating disorder symptoms from pretest to 36-month follow-up, $t(26) = 5.36, p < .001$, reflecting an expected decline in eating disorder symptoms for *Body Project* participants, despite scores remaining high. Slightly more

than half (51.7%) of the participants in this high-symptom trajectory group exhibited onset of an eating disorder during 36-month study follow-up.

Baseline risk factors associated with group membership are provided in Table 6. In each analysis, we selected a stable, moderate group from the model of interest as our basis for comparison. From the three-group control group model, we selected Trajectory #1 (low-stable) as the comparison group, and found that thin-ideal internalization, dietary restraint, and negative affect predicted increased likelihood of membership in Trajectory #2 (moderate-stable) and dietary restraint and negative affect predicted increased likelihood of membership in Trajectory #3 (high-variable). For *Body Project* participants, our comparison group was Trajectory #1 (low-decreasing) and found that thin-ideal internalization, dietary restraint, and negative affect predicted increased likelihood of membership in Trajectory #2 (high-variable). This finding is consistent with those of previous analyses that examined response to the Body Project (Horney et al., 2015).

Next, we examined group-based trajectories of the full dataset, to determine an optimal model for testing our third hypothesis that study condition would predict group-based trajectories. A three-group model emerged as having strongest model fit and stability, with group membership probabilities between .93 and .98., and the resulting trajectories are displayed in Figure 3. 800 of the 960 total participants (83% of the sample) reported low, stable levels of eating disorder symptoms at each of the five waves of data collection. Fifty-one percent of trajectory group was comprised of control participants, and 49% were *Body Project* participants. A group of 150 participants (16% of the sample) reported moderate, stable levels of eating disorder symptoms during each

Table 5.

Two-Group Body Project Trajectory Model Group Probability and Group Estimates

Variable	Trajectory 1 Low-Stable	Trajectory 2 High-Decreasing
Group probability (π_j)	.9376479	.2096950
Pretest eating disorder symptoms (λ_1)	11.77	41.32
Posttest eating disorder symptoms (λ_2)	9.29	39.67
12-month eating disorder symptoms (λ_3)	8.03	36.95
24-month eating disorder symptoms (λ_4)	7.78	33.16
36-month eating disorder symptoms (λ_5)	8.51	28.32

Figure 2. GBTM of eating disorder symptoms for *Body Project* group

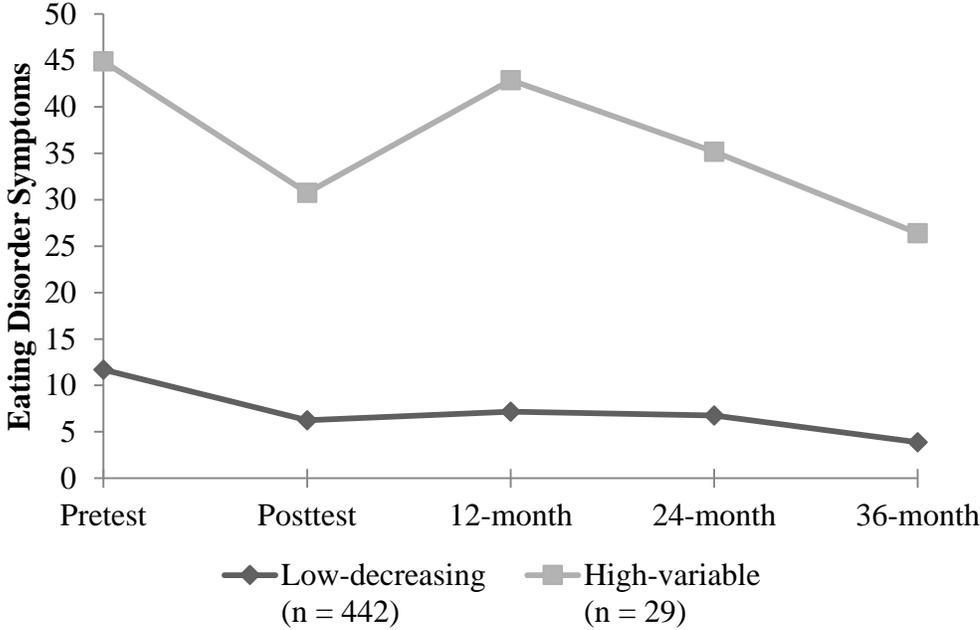


Table 6.

Risk Analyses for Trajectory Groups

Trajectory groups	Risk Factors			
	Thin-ideal internalization	Body dissatisfaction	Dietary restraint	Negative affect
Control				
1. Low-stable	-	-	-	-
2. Moderate-stable	1.18*** (0.33)	0.22 (0.18)	1.39*** (0.20)	0.32* (0.15)
3. High-variable	0.74 (0.46)	0.32 (0.26)	1.33*** (0.28)	0.45* (0.20)
Body Project				
1. Low-decreasing	-	-	-	-
2. High-variable	1.20** (0.40)	0.26 (0.21)	1.29*** (0.26)	0.61*** (0.16)

Note. Trajectory 1 (low-stable, and low-decreasing, respectively) was used as the comparison group for the risk analysis in each model.

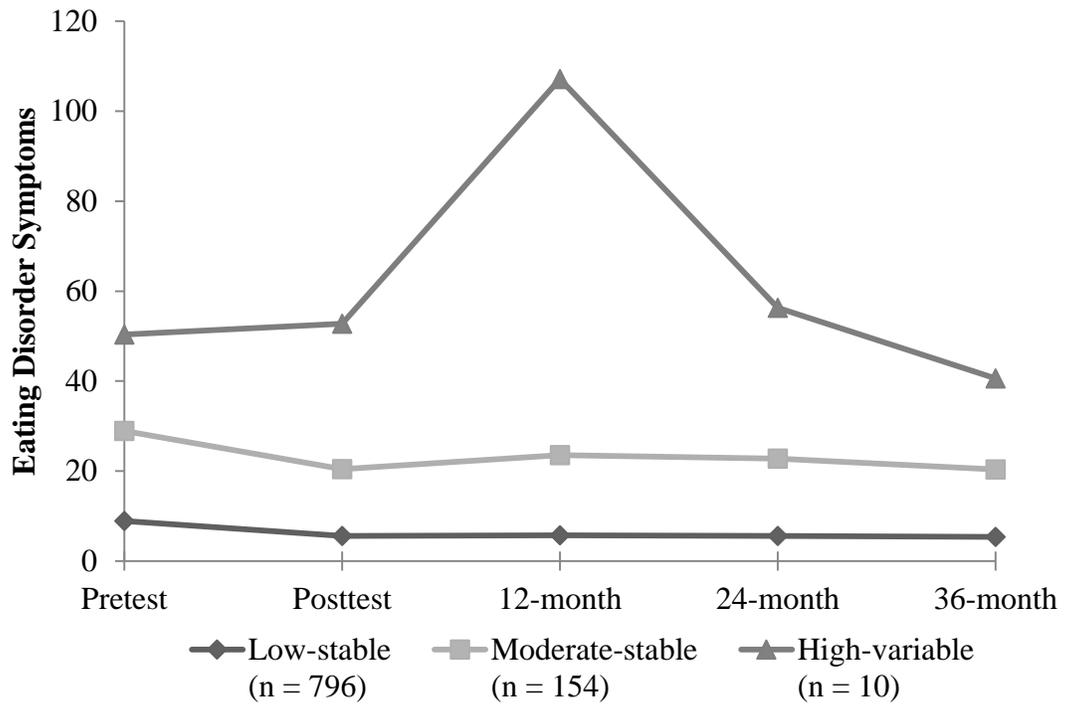
* $p < .05$, ** $p < .01$, *** $p < .001$

Table 7.

Three-Group Full Dataset Trajectory Model

Variable	Trajectory 1 Low-Stable	Trajectory 2 Moderate-Stable	Trajectory 3 High-Variable
Group probability (π_j)	.8291478	.1606259	.0102263
Pretest eating disorder symptoms (λ_1^j)	9.15	27.38	44.31
Posttest eating disorder symptoms (λ_2^j)	7.53	24.03	72.01
12-month eating disorder symptoms (λ_3^j)	6.67	21.95	80.41
24-month eating disorder symptoms (λ_4^j)	6.47	21.13	69.49
36-month eating disorder symptoms (λ_5^j)	6.90	21.58	39.27

Figure 3. GBTM of eating disorder symptoms for full dataset



of the five measurement waves. Again, the group was comprised of practically equal numbers of control versus intervention participants. And the smallest observed group of 10 participants (1.02% of the sample) endorsed high levels of eating disorder symptoms at baseline, and symptom levels varied over time but remained high during the course of study follow-up. This group included four control participants and 6 *Body Project* participants. Although the distribution of participants by study condition appeared equal based on frequencies, we conducted GBTM using this optimal full dataset model to confirm the potential presence of intervention effects on eating disorder symptom trajectory. As was expected, when examining the full dataset, we found no effects for the intervention condition on eating disorder symptom trajectory.

Summary

Group-based trajectory analyses was used to model eating disorder development as a function of five measurement waves, and to examine the impact of a dissonance-based prevention program on group-based differences. The three-group solution for control participants produced the strongest model fit. The resulting trajectories were those of low-stable, moderate-stable, or high-variable levels of eating disorder symptom courses. Dietary restraint and negative affect predicted increased likelihood of membership in the high-risk trajectory. The optimal solution for *Body Project* participants was a two-group trajectory model with low-decreasing or high-decreasing trajectories. For participants who completed the *Body Project*, their probability of membership in the high-risk trajectory was predicted by higher levels of thin-ideal internalization, dietary restraint, and negative affect. Contrary to study hypotheses, when

we examined the optimal, three-group model of the full dataset, study condition did not predict membership in high- versus low-risk trajectory groups.

CHAPTER IV

DISCUSSION

This innovative study expanded upon the first examination of *Body Project* non-response (Horney et al., 2015), and defined the qualitatively distinct trajectories of eating disorder symptom development in a selected prevention sample, as compared to an at-risk population control group. This study also examined the predictive role of established risk factors on eating disorder pathology over time, and the extent to which eating disorder prevention mitigated the potency of eating disorder risk factors. This study was the first longitudinal design to investigate response to an eating disorder prevention program through the identification of eating disorder developmental trajectories.

The study accomplished three main goals. First, despite the established success of the *Body Project*, this study provided information about factors that may predict sub-optimal prevention response and ultimately contribute to eating disorder onset. Second, this study identified qualitatively distinct trajectories of eating disorder pathology that vary with respect to baseline severity and change over time. Finally, this study responded to calls in the field to determine how established eating disorder prevention can be most effective, and to identify future steps to increase the yield on prevention success (Levine, 2015; Stice, South, & Shaw, 2012).

Eating disorder symptom trajectories without prevention

We found strong evidence to support the hypothesis that female development of disordered eating symptoms, among young women at high risk for eating disorders by virtue of body dissatisfaction, can best be described by three distinct, heterogeneous developmental trajectories that mirror those supported by eating disorder developmental

theory (Fairburn et al., 2000; Stice et al., 2002). Eating disorder symptom development was best described by multiple patterns of change, each pattern exhibiting a uniquely shaped trajectory over time. The most common eating disorder trajectory for a control group of at-risk individuals, was that of low, stable eating disorder symptoms. This trajectory of symptoms remained low over time, without indication of clinical or sub-clinical eating disorder onset. The second trajectory indicated moderate, stable levels of symptoms over time. These individuals exhibited a higher level of eating disorder symptoms, with very modest variation over time, but without a sharp increase in symptoms indicative of eating disorder onset. This trajectory is likely indicative of sub-clinical eating disorder symptomology. The third developmental trajectory portrayed a group of individuals for whom eating disorder symptoms remained high over time. This high-symptom trajectory shape portrayed slight increases and decreases in symptomology over time, with observed symptom levels indicating clinical-level eating disorder pathology.

Impact of prevention on eating disorder symptom trajectories

We found strong evidence to support the hypothesis that there are distinct developmental responses to the *Body Project* prevention intervention. This study identified two qualitatively separate longitudinal trajectories of response to an eating disorder prevention program; low-decreasing response and high-decreasing response. *Body Project* participants were most likely to fall into the low-decreasing trajectory group, absorbing the moderate-level trajectory group seen in the control group, an outcome supported by empirical evidence that the *Body Project* is an efficacious eating disorder prevention program. The trajectory of symptoms for participants in this

trajectory group began at a low level, and decreased consistently over time following receipt of the prevention intervention. The second, less common trajectory highlighted individuals with high, but gradually decreasing, eating disorder symptoms over time. This group of individuals began with high levels of eating disorder symptoms, showed a decrease in symptoms following the receipt of the prevention intervention, and exhibited a marginal decrease of eating disorder symptoms over time. Despite decreasing levels of eating disorder symptomology, symptoms remained at elevated, clinical levels keeping individuals at high-risk of eating disorder development. This trajectory captures the small percentage of individuals who exhibit sub-optimal responses to the *Body Project*, and who go on to develop an eating disorder despite receiving the efficacious prevention intervention.

We also examined the full dataset, to determine an optimal model of eating disorder symptom development across participants. Determining a best fit model for the full dataset allowed us to then test whether the prevention intervention predicted developmental trajectory. When the control and intervention groups were combined, a three-group developmental trajectory model emerged as the strongest model, with a low-stable, moderate-stable, and high-variable trajectory groups. However, study condition (educational control versus dissonance intervention) did not predict eating disorder symptom trajectory. Had additional, stable trajectory groups emerged, to delineate the distinct developmental pathways for intervention versus control participants, we may have observed the predictive impact of condition on trajectory. Future studies, with larger datasets, will allow for the emergence of additional distinct trajectories as the

GBTM model strength and stability increases as the trajectory group membership increases.

Predictive role of risk factors on risk trajectories

The second aim of the study was to determine whether the presence of established eating disorder risk factors at baseline impacted eating disorder symptom trajectory group membership. It was important to identify potential antecedents or correlates of the distinct developmental trajectories to understand contextual factors of eating disorder development, and to determine if the predictive impact of risk factors on developmental trajectories differs for individuals who participate in eating disorder prevention.

For control group participants, thin-ideal internalization, dietary restraint, and negative affect predicted increased likelihood of membership in a moderate-stable trajectory of eating disorder symptoms, and heightened dietary restraint and negative affect predicted increased likelihood of membership in a high-variable eating disorder symptom trajectory. The three study trials were all selected prevention studies, and participants with elevated body dissatisfaction were targeted for study recruitment. This explains the lack of significant difference for baseline body dissatisfaction across developmental trajectories, as we can reasonably conclude that elevated levels of body dissatisfaction was present for all study participants. These results confirmed our hypothesis that high risk trajectory groups would replicate theorized multivariate models of eating disorder risk and development, and therefore be associated with higher levels of eating disorder risk factors.

Body Project participants in the high-decreasing trajectory group were more likely to have increased thin-ideal internalization, dietary restraint, and negative affect at

baseline. This outcome highlighted the individual characteristics associated with higher risk of eating disorder development and decreased likelihood of optimal response to eating disorder prevention programs. This outcome confirmed our study hypotheses that atypical *Body Project* response would be associated with higher baseline risk factors. This conclusion also replicates findings from the first *Body Project* non-response examination that examined the mitigating effects of risk factors on long-term prevention efficacy (Horney et al., 2015).

Moderation studies have indicated that higher risk individuals showed greater reductions in eating disorder symptoms following participation in a dissonance-based eating disorder prevention program. Yet the first examination of *Body Project* suboptimal response revealed that elevated risk at baseline, particularly of thin-ideal internalization and negative affect, paradoxically increases risk for subsequent eating disorder onset despite completing the *Body Project*. The dichotomous outcome variables used in the original study of non-response (e.g., “*DSM-5* eating disorder diagnosis” versus “no eating disorder diagnosis”) did now allow for intricate examination of the impact of these risk factors.

An objective of the current study was to build upon earlier examinations, and interpret the degree at which negative affect contributed to increased eating disorder symptom presentation and sub-optimal prevention responses. The findings from the current study indicate the impact of pretest risk factors on a distinct developmental outcome. Specifically, these findings suggest that elevated risk at pretest predicts membership in a high-risk trajectory model, but the model indicates that symptoms *do* decrease following receipt of the prevention intervention. The trajectory suggests that

individuals with elevated risk factors are at great risk for onset of eating disorders, but also that these high-risk participants respond to the intervention with gradual (and marginal) decreased observed eating disorder symptoms over time.

These seemingly contrasting outcomes of the *Body Project* are similar to those found in depression prevention literature which has made a conceptual distinction between “treatment effects,” reductions in continuous outcomes from baseline levels, and “prophylactic effects,” reductions in the onset of psychiatric disorders (Horowitz & Garber, 2006). Many cognitive behavioral depression prevention programs have been shown to produce stronger prophylactic outcomes at longer time follow-up, with the treatment effects on initial depressive symptoms fading after posttest (Stice, Shaw, Bohon, Marti, & Rohde, 2009). These prophylactic effects suggest that the intervention effects are not simply occurring due to a decrease of baseline elevations in depressive symptoms (Stice, Shaw, Bohon, Marti, & Rohde, 2009). Outcomes of the current study suggested similar distinctive outcomes may occur for eating disorder prevention programs.

Clinical implications

Due to distinct eating disorder developmental pathways in the general population, and the heterogeneous response to prevention efforts, there is no “one-size-fits-all” approach to eating disorder prevention. The pernicious nature of eating disorders, severity across eating pathologies, and range of risk factors substantiate an urgent need for the improved understanding of these qualitatively distinct routes of eating disorder development and effective prevention intervention strategies. This study expanded upon the current understanding of how eating disorder prevention mitigates high- and

moderate-risk developmental trajectories of eating disorder symptoms. This study enhanced the eating disorder prevention literature by providing developmental context to the sub-optimal response to eating disorder prevention. Combining the results of the current study with those of the original *Body Project* response study (Horney et al 2015), provides us with a range of clinical implications to improve prevention programs and ultimately benefit individuals presenting with diverse eating disorder risk factors and symptom levels.

High-risk eating disorder symptom trajectories for *Body Project* participants appear to be driven by elevations of certain risk factors not directly targeted by the prevention intervention program (i.e., disordered eating behaviors, dietary restraint, and negative affect). This finding suggests the benefit of a qualitatively different, or more intensive, prevention program for high-risk individuals already demonstrating significant eating disorder symptoms and risk factors at pretest. Individuals in the high-decreasing eating disorder symptom trajectory group might benefit from an indicated variant of the *Body Project* that directly addresses the presence of eating disorder symptoms (Stice, Rohde, Butryn, Menki, & Marti, 2015). An intensive variant of the *Body Project* could incorporate direct interventions to increase feelings of cognitive dissonance regarding engaging in disordered eating, as this high-risk group is more likely to report dietary restraint and the developmental trajectory indicates an elevation in eating disorder symptoms at pretest. This discernible elevation in eating disorder symptoms at pretest would allow for the identification of individuals who could most benefit from an intensive intervention alternative.

The initial study of *Body Project* non-response recommended an adaptation of the prevention program that could produce larger reductions in negative affect, an eating disorder risk factor that the *Body Project* does not currently address directly (Horney et al., 2015). The findings from this current study provided additional evidence to support this benefit of refining the *Body Project* with interventions directly targeting this risk factor to improve the response of individuals who begin the program with elevated depression or anxiety. Horney and colleagues (2015) recommended adding exercises from cognitive behavioral therapy for depression (i.e., cognitive restructuring or behavioral activation), or having participants discuss the costs of negative affect and the benefits of positive, adaptive activities that help reduce negative affect (i.e., exercise), to help increase motivation for change and more directly intervene on affective.

In the initial study of *Body Project* response, a Cox proportional hazard model indicated that participants were most likely to show onset of an eating disorders between 10 and 30 months after pretest, rather than immediately after pretest (Horney et al., 2015). Our GBTM findings support this analysis, and provide a more nuanced understanding of sub-optimal response trajectories. This study of developmental trajectories indicated that high-risk participants who receive the *Body Project* do exhibit a response to the prevention program and a reduction in eating disorder symptoms. However the response appears to be marginal, and unfortunately, the eating disorder symptoms of these participants ultimately remain high and do not drastically decrease over time; the response to the intervention we would hope for and typically expect. These findings suggest higher risk *Body Project* recipients may benefit from prevention booster sessions, following initial intervention, for enhanced and continuous change over

time. This clinical implication may be equally as impactful as a refined, intensive program for high-risk participants. Additionally, it could be argued that the ease of studying a “double-dosage” *Body Project* trial, versus the time and money required for the development, implementation, and examination of a refined prevention program, provides a clear cost-benefit outcome. Comparing the efficacy of the 4-session *Body Project* to the 8-session indicated *Body Project* would require all participants endorse eating disorder symptoms at baseline for the exercises in the indicated intervention to be clinically indicated.

Despite the medium to large effects of the *Body Project*, a small percentage of individuals enter the efficacious prevention program with elevated risk high enough that the benefits of the prevention program are not strong enough to reduce their symptomology to a moderate or low level. These individuals tend to remain in the high-risk category of development over time, despite posttest improvements, and are at high risk of eating disorder onset. As such, higher risk *Body Project* recipients may require prevention booster sessions, or an indicated version of the prevention program, to ensure enhanced positive change, and response to intervention effects, over time.

Recommendations for future research and practice

These study implications can help improve the overall public health yield of eating disorder prevention by informing the future design of prevention programs and studies targeting individuals less likely to respond to current evidence-based prevention interventions. With a better understanding of what predicts suboptimal prevention response, we must now answer the question of what program changes or enhancements would be most effective in mitigating the effect of elevated baseline risk and boosting the

positive impact of prevention. This study points to two primary clinical implications; program adaption, and program enhancement. We must determine whether participants flagged as highest-risk would benefit most from a prevention program that more directly targets disordered eating and secondary risk factors (i.e., negative affect). Or, if these individuals would respond most optimally to an enhanced version of the current program, one in which they receive a second round, or booster session of sorts, weeks or even months following completion of the initial program.

It may also benefit participants to incorporate a social media follow-up component to the intervention, following intervention sessions. In a direct response to “thinspo” and “pro-ana” internet forums, individuals have taken to social media sites such as Facebook and Instagram to facilitate body acceptance and rejection of the thin ideal. The individual agency and potential for social connection found online could enhance the recovery process for individuals feeling isolated in their body image concerns. Future examinations will help determine whether this enhancement could produce stronger prophylactic effects among a group of individuals most at risk of eating disorder onset, and with risk levels high enough to be somewhat impermeable to the demonstrated effects of this intervention.

Strengths and limitations

The methodology utilized in this study determined the impact of a dissonance-based prevention intervention on subgroups characterized by different growth trajectories. Unobserved heterogeneity among participants is typical in prevention studies, for both intervention and control groups. This analysis benefited from the strength of a longitudinal design through the assessment of intervention effects on

trajectories, as opposed to effects at a single time point or of a dichotomous outcome (Muthén et al., 2002). Due to the frequency of heterogeneous response to prevention interventions, this approach also provided insight on potentially advantageous refinements to the design and implementation of future prevention efforts (Muthén et al., 2002).

Study limitations should also be considered when interpreting these findings. First, the use of this longitudinal research design allows for the representation of developmental processes, but prevents the assertion of casual inferences or clear directionality of associations among risk factors and developmental change. Despite this limitation, the longitudinal design was optimal to expand upon earlier examinations of *Body Project* response and to most accurately assess the developmental processes exhibited among study participants. Another potential limitation is the measure of negative affect in the study, as it was assessed by a different instrument in each of the three study trials and a z-transformed variables were used to examine data across trials. Despite the use of different measures, the three assessment instruments are highly correlated, and no differential effects emerged from the separate measures (Tellegen, Watson, & Clark, 1988; Watson & Clark, 1992).

The GBTM statistical approach utilized in this study imposes the assumption that the development of eating disorder symptoms among an at-risk sample can be described in terms of a finite number of heterogeneous groups. As such, those using PROC TRAJ are advised to remember that the developmental trajectories produced by the statistical modeling approach are not reified groups. The developmental trajectory groups presented in this study are estimations of distinct patterns of change within the population of

interest, and group membership, number of groups, and trajectory shape should not be perceived as absolute certainties. Relatedly, these findings are sample dependent and others should use caution when generalizing the developmental changes present in this study across eating disorder prevention and intervention programs.

A final limitation of the present study occurred as a result of merging data from three randomized controlled trials to achieve an adequate sample of participants. The implicit assumption behind merging these data sets was that the participants across studies would exhibit similar presentations at baseline. However preliminary analyses did reveal slight differences between study trials in that *Efficacy Trial* participants had significantly higher eating disorder symptoms at baseline than *High School Effectiveness Trial* or *College Effectiveness Trial* participants. This result may reflect regional differences in body image concerns and eating pathology as the study trial that was conducted solely in Oregon had the lowest eating disorder symptom scores, the study conducted solely in Texas exhibit the highest eating disorder scores, and the trial with moderate-level scores included participants recruited from Oregon, Texas, and Pennsylvania. These differences within groups, although slight, should be taken into consideration when interpreting study findings about the developmental trajectories of control group participants.

Another study limitation is the range of risk factors examined was narrow in focus, and did not include theoretically significant eating disorder risk factors such as biological variables or peer and familial influences.

Summary and conclusions

Randomized trials provide support for the *Body Project*, an eating disorder prevention program wherein young women with body image concerns critique the thin ideal. Grounded in the theory that humans seek to maintain consistency between their words, thoughts and actions, dissonance-induction produces a motivational drive for participants to reduce their pursuit of this unrealistic beauty ideal (Stice et al., 2000). Despite medium to large effects, some *Body Project* participants subsequently develop an eating disorder during long-term study follow-up, calling for the improvement to both recruitment and intervention procedures.

This study was the first to delineate the heterogeneous pathways of eating disorder symptom trajectories among *Body Project* versus control group participants. Existing data from three randomized controlled trials was combined to examine response trajectories of prevention intervention versus control participants through 3-year follow-up. GBTM methods distinguished distinct response trajectories and the impact of prevention on mitigating the developmental course of eating disorder symptoms. This study also determined the predictive role of risk factors on qualitatively distinct developmental pathways of eating disorder symptomology, confirming the hypothesized impact of thin-ideal internalization, negative affect, and dietary restraint on sub-optimal prevention response.

This novel study has the potential to improve the overall public health yield of eating disorder prevention by informing the future design and adaptation of programs targeting individuals less likely to respond to current evidence-based prevention interventions. These results and implications will help inform the design of more

effective variants of this prevention program and improve participant assignment to the appropriate level of intervention. If we look beyond the scope of eating disorder prevention, this study has significant heuristic value, and we recommend the application of this developmental trajectory approach to a broad range of prevention programs aimed to reduce physical and mental health problems. A more nuanced understanding of developmental processes gleaned from innovative statistical techniques has the potential to increase the overall success and future improvement of prevention efforts for a wider array of health outcomes.

APPENDIX

BODY PROJECT: INTERVIEW

ID: _____ Interviewer Name: _____ Date: _____

(Introduce yourself and establish rapport) I want to let you know that everything you say will be confidential, which means I won't tell anyone any of your answers and your name is not associated with your answers. We use an ID number, not your name on the assessments forms. We will not give information about you to anyone unless you provide a signed release or we have reason to suspect (1) abuse, neglect, or endangerment of a child or elder, or (2) or that anyone is in immediate danger of seriously hurting himself/herself or someone else. In these cases, we may have to break confidentiality and report this information to our supervisors and/or the appropriate authorities. There are no right or wrong answers to the questions I will be asking. Do you have any questions before we begin?

I would like to get a general picture of your eating habits over the past 4 weeks. Have your eating habits varied much from day to day? I am specifically interested in any overeating that you may have experienced over the past 4 weeks.

Different people mean different things by "overeating" so I would like to explain the term "binge episode" for you. This means eating an amount of food that most people would consider very large, and secondly feeling like you cannot control your eating.

This is different from when you feel your eating is out of control, but you don't eat what most people would consider a very large amount of food.

This is also different from simple overeating, when you do eat a large amount of food, but don't feel like your eating is out of your control. For example, at Thanksgiving, people tend to eat a lot of food, but most people feel as though they are in control during this time. Does that make sense?

Have you had a binge episode like that in the past month?

If they endorse bingeing, ask the following to get a sense of whether it was a true binge episode:
Can you give me an example of what you have eaten at these times? *If more than one episode, are the other binge episodes similar? Did you feel out of control during those times? Could you have stopped eating once you had started?*

1. For the past 4 weeks, on how many days did you have a binge episode? How many episodes did you have on each of those days? For the 2 months before that, how many days did you have a binge episode and how many total episodes were there? For the 9 months before that, how many days and episodes were there? [Rate best guess as to number of episodes, even if number is very large, so that data can be analyzed. Capture entire year's history. Rate 00 if none]

(Skip questions 2-7 ONLY if NO binges were endorsed over the past month)

2. During these episodes did you eat much more rapidly than normal? Yes No

3. Eat until you felt uncomfortably full? Yes No

4. Eat large amounts of food when you didn't feel physically hungry? Yes No

5. Eat alone because you were embarrassed by how much you were eating? Yes No

6. Feel depressed or very guilty after overeating? Yes No

7. Did you feel upset that you couldn't control your eating? Yes No

8. Over the past 4 weeks have you made yourself sick as a means of controlling your shape or weight? How many episodes of self-induced vomiting occurred over the last 4 weeks? 2 prior months? 9 months before that? [Accept student's definition of an episode. Rate best guess as to number of episodes.]

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

9. Over the past 4 weeks have you taken laxatives or diuretics (water pills) as a means of controlling your shape or weight? (e.g. medicines such as Ex-Lax, Correctol, Phenamin, Nature's Remedy, Sunril, Aqua-Ban, Pamprin, Midol-PMS.) How many episodes of laxative/diuretic use to control shape or weight occurred over the last 4 weeks? 2 prior months? 9 months before that?

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

10. Over the past 4 weeks have you fasted (skipped at least 2 meals in a row) as a means of controlling your shape or weight? How many episodes of fasting to control shape or weight occurred over the last 4 weeks? 2 prior months? 9 months before that? [The decision whether the fasting was "compensatory" should be made by the interviewer. If in doubt, the fasting should not be classified as compensatory]

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

11. Over the past 4 weeks have you engaged in exercise that was intended to burn calories to compensate for "overconsumption" of eating or drinking? How many days of compensatory exercise to occurred in the past 4 weeks? 2 prior months? 9 months before that? [Exercise must be excessive to count. The decision whether the exercising was "compensatory" should be made by the interviewer. If in doubt, the exercising should not be counted]

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

12. Typically, what form of exercise have you done?

(Write the most common form of exercise in the grey box. Write others next to the box.)

13. Typical duration (in minutes) per episode?

(Indicate the total amount of exercise above and beyond the individual's regular exercise routine)
 (Compensatory: general rule is 30+ min of intense exercise (sweating) or 60+ min of moderate-light exercise)

14. Over the past 4 weeks has your weight and/or shape been important in influencing how you feel about (judge, think, evaluate) yourself as a person?

- 0 no importance
- 1
- 2 some importance (definitely aspect of self-evaluation)
- 3
- 4 moderate importance (definitely one of the main aspects of self-evaluation)
- 5
- 6 supreme importance (nothing is more important in terms of self-evaluation)

15. Has it been similar for the 2 prior months? 9 months before that? [write in the rating (#) from above item (14) into the current month]

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

16. Over the past 4 weeks have you been afraid that you might gain weight (or become fat)? How many days?

- 0 no definite fear of fatness or weight gain
- 1 1-2 days
- 2 definite fear of fatness or weight gain present on less than half the days (2-3 days)
- 3 3-4 days
- 4 definite fear of fatness or weight gain present on more than half the days (4-5 days)
- 5 5-6 days
- 6 definite fear of fatness or weight gain present every day (6-7 days)

17. Has it been similar for the 2 prior months? 9 months before that? [write in the rating (#) from item 16 into the current month]

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

18. Over the past 4 weeks have you felt fat? [Omit item if the student is obviously overweight and rate 7.]

- 0 0 days/wk: has not felt fat
- 1 1-2 days/wk
- 2 2-3 days/wk: has felt fat on less than half the days
- 3 3-4 days/wk
- 4 4-5 days/wk: has felt fat on more than half the days

- 5 5-6 days/wk
- 6 6-7 days/wk: has felt fat every day
- 7 (Question not asked by interviewer)

19. Has it been similar for the 2 prior months? 9 months before that? [write in the rating (#) from above item (18) into the current month]

12	11	10	9	8	7	6	5	4	3	2	Month 1 (Current)

21. Have you missed your period in the past month? Yes No N/A

22. How many periods have you missed in the 11 months before that? N/A

23a. Have you been using any hormonal birth control this past year? Yes
No

23b. If yes, what kind?

23c. Have you been pregnant in the past year or had any medical problems that would have caused a significant change in your weight or eating habits? Yes
No

If yes, please describe:

24a. Have you participated in a structured weight loss treatment that cost money (e.g. Weight Watchers, Jenny Craig) in the past year? Yes
No

24b. If so, how long did you participate in this paid weight loss program?

24.c How much weight did you lose from participating in this program?

Measure Height and Weight:

25. Current Height: Record Below

26. Current Weight: Record Below

27. What has been your lowest weight in the past 12 months?

29. What has been your highest weight in the past 12 months?

Thank you for your participation. Do you have any questions about the interview?

PLEASE DON'T FORGET TO MEASURE HEIGHT AND WEIGHT!

25. Current Height (1) _____ cm (2) _____ cm

26. Current Weight (1) _____ kg (2) _____ kg

BODY PROJECT: QUESTIONNAIRE

Date: ____/____/____

Participant ID: _____

Height: _____ Weight: _____ Age: _____ Year in school:

How much do you agree with these statements?

	Strongly Disagree	Disagree	Neutral	Agree	Strongly Agree
1) Slender women are more attractive.	1	2	3	4	5
2) Women who are in shape are more attractive.	1	2	3	4	5
3) Tall women are more attractive.	1	2	3	4	5
4) Women with toned (lean) bodies are more attractive.	1	2	3	4	5
5) Shapely women are more attractive.	1	2	3	4	5
6) Women with long legs are more attractive.	1	2	3	4	5

Over the past MONTH, how satisfied were you with your...

Extremely Dissatisfied Moderately Dissatisfied Neutral Moderately Satisfied Extremely Satisfied

1) Weight	1	2	3	4	5
2) Figure	1	2	3	4	5
3) Stomach	1	2	3	4	5
4) Body Build	1	2	3	4	5
5) Waist	1	2	3	4	5
6) Thighs	1	2	3	4	5
7) Buttocks	1	2	3	4	5
8) Hips	1	2	3	4	5
9) Legs	1	2	3	4	5
10) Breasts	1	2	3	4	5

Circle the best response to describe your behavior over the last MONTH:

	Never	Seldom	Some- times	Often	Always
1) If you put on weight, did you eat less than you normally would?	1	2	3	4	5
2) Did you try to eat less at mealtimes than you would like to eat?	1	2	3	4	5
3) How often did you refuse food or drink because you were concerned about your weight?	1	2	3	4	5
4) Did you watch exactly what you ate?	1	2	3	4	5
5) Did you deliberately eat foods that were slimming (i.e. low fat or diet foods)?	1	2	3	4	5
6) When you ate too much, did you eat less than usual the next day?	1	2	3	4	5
7) Did you deliberately eat less in order not to become heavier?	1	2	3	4	5
8) How often did you try not to eat between meals because you were watching your weight?	1	2	3	4	5
9) How often in the evenings did you try not to eat because you were watching your weight?	1	2	3	4	5
10) Did you take into account your weight in deciding what to eat?	1	2	3	4	5

**Please circle the number next to the answer that describes how you have been feeling in the past
TWO WEEKS:**

1. [0] I do not feel sad.
[1] I feel sad much of the time.
[2] I am sad all of the time.
[3] I am so sad or unhappy that I can't stand it.
2. [0] I am not discouraged about my future.
[1] I feel more discouraged about my future than I used to.
[2] I do not expect things to work out for me.
[3] I feel my future is hopeless and will only get worse.
3. [0] I do not feel like a failure.
[1] I have failed more than I should have.
[2] As I look back, I see a lot of failures.
[3] I feel I am a total failure as a person.
4. [0] I get as much pleasure as I ever did from the things I enjoy.
[1] I don't enjoy things the way I used to.
[2] I get very little pleasure from the things I used to enjoy.
[3] I can't get any pleasure from the things I used to enjoy.
5. [0] I don't feel particularly guilty.
[1] I feel guilty about many things I have done or should have done.
[2] I feel quite guilty most of the time.
[3] I feel guilty all of the time.
6. [0] I don't feel that I am being punished.
[1] I feel that I may be punished.
[2] I expect to be punished.
[3] I feel that I am being punished.

Please circle the number next to the answer that describes how you have been feeling in the past

TWO WEEKS:

7. [0] I feel the same about myself as ever.
[1] I have lost confidence in myself.
[2] I am disappointed in myself.
[3] I dislike myself.
8. [0] I don't criticize or blame myself more than usual.
[1] I am more critical of myself than before.
[2] I criticize myself for all of my faults.
[3] I blame myself for everything bad that happens.
9. [0] I don't have any thoughts of killing myself.
[1] I have thoughts of killing myself, but would not carry them out.
[2] I would like to kill myself.
[3] I would like to kill myself if I had the chance.
10. [0] I don't cry any more than usual.
[1] I cry more than I used to.
[2] I cry over every little thing.
[3] I feel like crying, but I can't.
11. [0] I am no more restless than usual.
[1] I feel more restless or wound up than usual.
[2] I am so restless or agitated that it's hard to stay still.
[3] I am so restless or agitated that I have to keep moving or doing something.
12. [0] I have not lost interest in people or activities.
[1] I'm less interested in people or things than before.
[2] I've lost most of my interest in people or things.
[3] It's hard to get interested in anything.
13. [0] I make decisions about as well as ever.
[1] I find it more difficult to make decisions than usual.
[2] I have much greater difficulty in making decisions than I used to.
[3] I have trouble making any decisions.
14. [0] I do not feel I am worthless.
[1] I don't consider myself as worthwhile and useful as I used to.
[2] I feel more worthless as compared to other people.
[3] I feel utterly worthless.
15. [0] I have as much energy as ever.
[1] I have less energy than I used to have.

- [2] I don't have enough energy to do much.
- [3] I don't have enough energy to do anything.

16. [0] I have not experienced any change in my sleeping.
- [1a] I sleep somewhat more than usual.
 - [1b] I sleep somewhat less than usual.
 - [2a] I sleep a lot more than usual.
 - [2b] I sleep a lot less than usual.
 - [3a] I sleep most of the day.
 - [3b] I wake up 1-2 hours early and can't get back to sleep.

17. [0] I am no more irritable than usual.
- [1] I am more irritable than usual.
 - [2] I am much more irritable than usual.
 - [3] I am irritable all the time.

Please circle the number next to the answer that describes how you have been feeling in the past TWO WEEKS:

18. [0] I have not experienced any change in my appetite.
- [1a] My appetite is somewhat less than usual.
 - [1b] My appetite is somewhat greater than usual.
 - [2a] My appetite is much less than usual.
 - [2b] My appetite is much greater than usual.
 - [3a] I have no appetite at all.
 - [3b] I crave food all the time.

19. [0] I can concentrate as well as ever.
- [1] I can't concentrate as well as usual.
 - [2] It's hard to keep my mind on anything for very long.
 - [3] I find I can't concentrate on anything.

20. [0] I am no more tired or fatigued than usual.
- [1] I get more tired or fatigued more easily than usual.
 - [2] I am too tired or fatigued to do a lot of the things I used to do.
 - [3] I am too tired or fatigued to do most of the things I used to do.

21. [0] I have not noticed any recent change in my interest in sex.
- [1] I am less interested in sex than I used to be.
 - [2] I am much less interested in sex now.
 - [3] I have lost interest in sex completely.

Over the last SIX MONTHS, how often have you:

Never Seldom Some-
times Often Very
Often

1) Seen friends or spoken to friends on the telephone?	1	2	3	4	5
2) Gone out socially with other people, such as to a movie?	1	2	3	4	5
3) Had arguments with friends?	1	2	3	4	5
4) Had your feelings hurt by a friend?	1	2	3	4	5
5) Felt shy or uncomfortable with people?	1	2	3	4	5
6) Felt lonely and wished for more friends?	1	2	3	4	5
7) Dated someone or been in a long-term relationship?	1	2	3	4	5
8) Had arguments with your family?	1	2	3	4	5
9) Had your feelings hurt by a family member?	1	2	3	4	5
10) Missed school?	1	2	3	4	5
11) Felt upset at school?	1	2	3	4	5
12) Felt ashamed of how you do your school work?	1	2	3	4	5
13) Had arguments with people at school?	1	2	3	4	5
14) Missed work?	1	2	3	4	5
15) Felt upset at work?	1	2	3	4	5
16) Felt ashamed of how you do your work?	1	2	3	4	5
17) Had arguments with people at work?	1	2	3	4	5

We are interested in how often you talked with health providers about various concerns in your life.

How often did you speak to the following providers about these topics over the last MONTH?

<u>TOPIC</u>	A primary care doctor or other	A psychiatrist:	A nurse:	A therapist, psychologist, or other	A support group (excluding
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	physician:			counselor:	a group related to this study):
1) Physical health problem, injury, or illness	____ hours				
2) Mental health problem (depression, anxiety, etc.)	____ hours				
3) Weight problem:	____ hours				
4) Eating disorder or body image concern:	____ hours				
5) Other personal problem:	____ hours				

How often did you speak to the following providers about these topics over the last YEAR?

<u>TOPIC</u>	A primary care doctor or other physician:	A psychiatrist:	A nurse:	A therapist, psychologist, or other counselor:	A support group (excluding a group related to this study):
1) Physical health problem, injury, or illness	____ hours	____ hours	____ hours	____ hours	____ hours
2) Mental health problem (depression, anxiety, etc.)	____ hours	____ hours	____ hours	____ hours	____ hours
3) Weight problem:	____ hours	____ hours	____ hours	____ hours	____ hours
4) Eating disorder or body image concern:	____ hours	____ hours	____ hours	____ hours	____ hours
5) Other personal problem:	____ hours	____ hours	____ hours	____ hours	____ hours

Which condition were you assigned to for this study?

____ Group or Website Condition₁

Have you participated in any *additional* body acceptance classes since completing the group sessions for this study? Yes₁ No₀ If yes, when (term and year)? _____

Brochure or Video Condition₁

Have you participated in any body acceptance classes since enrolling in this study?

Yes₁ No₀ If yes, when (term and year)?

We are interested in medications you have taken for various problems.

Please list *all* of the medications (*excluding* birth control) you have been prescribed in the last MONTH:

How often did you take this medication in the last **MONTH**?

(check the appropriate box)

Medication Name (please list)	Purpose for Taking the Medication	Once a Day ₁	Once a Week ₂	Once a Month ₃	As Needed ₄
----------------------------------	--------------------------------------	----------------------------	-----------------------------	------------------------------	---------------------------

1)

2)

3)

4)

5)

6)

7)

8)

Please list *all* of the medications (*excluding* birth control) you have been prescribed in the last YEAR:

Medication Name (please list)	Purpose for Taking the Medication	How often did you take this medication in the last YEAR ? (check the appropriate box)				How many months in the last YEAR were you taking this medication? (if less than one month, enter 1)
		Once a Day ₁	Once a Week ₂	Once a Month ₃	As Needed ₄	
1)						
2)						
3)						
4)						
5)						
6)						
7)						
8)						

The following questions refer to the past 4 WEEKS (28 days) only. Please read each question carefully and circle the appropriate number on the right. Please answer *all* of the questions.

ON HOW MANY DAYS OUT OF THE PAST 28 DAYS...	0	1-5	6-12	13-15	16-22	23-27	Every
	Days	Days	Days	Days	Days	Days	Day
1) Has thinking about food or its calorie content made it much more difficult to concentrate on things you are interested in (e.g., reading, watching TV, or following a conversation)?	0	1	2	3	4	5	6
2) Have you been afraid of losing control and over eating?	0	1	2	3	4	5	6
3) Have you eaten in secret? (Do <i>not</i> count binges)	0	1	2	3	4	5	6
4) Have you definitely wanted	0	1	2	3	4	5	6

- your stomach to be flat?
- 5) Has thinking about shape or weight made it difficult to concentrate on things you are interested in (e.g., reading, watching TV, or following a conversation)?
- 6) Have you had a definite fear that you might gain weight or become fat?
- 7) Have you felt fat?
- 8) Have you had a strong desire to lose weight?
- | | | | | | | | |
|--|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
|--|---|---|---|---|---|---|---|

OVER THE PAST FOUR WEEKS (28 DAYS):

- 9) On what proportion of times that you have eaten have you felt guilty because of the effect on your shape or weight? (Do *not* count binges.) (Circle the number which applies.)

None of the times	A few of the times	Less than half the times	Half the times	More than half the times	Most of the time	Every time
0	1	2	3	4	5	6

Please circle the number that best describes your behavior.

- | | | | | | | | |
|--|------------------|--|----------|--|------------|--|----------|
| OVER THE PAST FOUR WEEKS (28 DAYS): | Not
at
All | | Slightly | | Moderately | | Markedly |
|--|------------------|--|----------|--|------------|--|----------|
- 10) Has your *weight* influenced how you think about (judge) yourself as a person?
- 11) Has your *shape* influenced how you think about (judge) yourself as a person?
- | | | | | | | | |
|--|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
|--|---|---|---|---|---|---|---|

- | | | | | | | | |
|--|------------------|--|----------|--|------------|--|----------|
| OVER THE PAST FOUR WEEKS (28 DAYS): | Not
at
All | | Slightly | | Moderately | | Markedly |
|--|------------------|--|----------|--|------------|--|----------|
- 12) How much would it
- | | | | | | | | |
|--|---|---|---|---|---|---|---|
| | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
|--|---|---|---|---|---|---|---|

	upset you if you had to weigh yourself once a week for the next four weeks?							
13)	How dissatisfied have you felt about your <i>weight</i> ?	0	1	2	3	4	5	6
14)	How dissatisfied have you felt about your <i>shape</i> ?	0	1	2	3	4	5	6
15)	How concerned have you been about other people seeing you eat?	0	1	2	3	4	5	6
16)	How uncomfortable have you felt seeing your body (e.g., in the mirror, in shop window reflections, while undressing or taking a bath or shower)?	0	1	2	3	4	5	6
17)	How uncomfortable have you felt about others seeing your body (e.g., in communal changing rooms, when swimming or wearing tight clothes)?	0	1	2	3	4	5	6

Please circle the response that reflects your agreement with the statements below. If you do not engage in the stated behavior, circle the response that would reflect your agreement if you did engage in the behavior.

1. I feel better about myself if I challenge my friends not to pursue the thin ideal for women glorified in the media
2. I don't feel good about myself when I spend lots of time looking at fashion magazines
3. I don't like it when I spend a lot of time talking with friends about how much weight we want to lose
4. I feel bad if I find myself obsessing about trying to look like the thin ideal

5. I regret it when I make a disapproving comment about someone's appearance
.....
6. I feel uncomfortable talking about the latest diet fads with friends
.....
7. I feel at peace when I notice the things that I like about my body
.....
8. I feel uneasy if I find myself obsessing about aspects of my appearance that don't conform to the thin ideal
9. I regret it when I engage in "fat-talk" with my Friends, talking about things we dislike about our bodies
10. I dislike it when I compare myself to other women regarding who looks the best
.....

EATING SCREEN

Please carefully complete all questions.

Over the past 12 MONTHS... Not at all Slightly
 Moderately Extremely

1. Have you felt fat?.....
0 1 2 3 4 5 6
2. Have you had a definite fear that you might gain weight or become fat?.....
0 1 2 3 4 5 6
3. Has your weight influenced how you think about (judge) yourself as a person?....
0 1 2 3 4 5 6
4. Has your shape influenced how you think about (judge) yourself as a person?....
0 1 2 3 4 5 6
5. During the past **12 months** have there been times when you felt you have eaten what other people would regard as an unusually large amount of food (e.g., a quart of ice cream) given the circumstances?.....
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 12 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 12 MONTHS** have you

eaten an unusually large amount of food and experienced a loss of control?

IF YOU CIRCLED “0” FOR Q7-Q8, SKIP TO QUESTION 15.

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 12 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 **12 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 **12 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 **12 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
- 19. Over the **past 12 MONTHS**, how many menstrual periods have you **missed**?
0 1 2 3 n/a
- 20. Have you been taking birth control pills during the past **past 12 MONTHS**?...
YES₁ NO₀

Please circle the best response.

1. My current cumulative college GPA is:

N/A₈ No C- or C₅ B- to B₃ A- to A₁
 GPA₇ lower₆ C+₄ B+₂

2. My current school status is:

- (a) Enrolled in school FULL time
- (b) Enrolled in school PART time
- (c) Graduated
- (d) Finished my degree, have been accepted into a graduate program or post-bac program
- (e) Did not finish my degree, no longer enrolled in school
- (f) Other (please describe:

_____)

		Excellent	Good	Fair	Poor
		1	2	3	4
3. How would you evaluate your entire education experience at the University where you participated in the Body Project Study?					
4. Please complete the following items if you are still attending college.	Never	Seldom	About half the time	Most of the time	All the time
a. Do you attend your scheduled classes regularly ?	1	2	3	4	5
.....					
b. Do you study for these classes each week, as needed?	1	2	3	4	5
.....					
c. Have you gone to class without homework completed?	1	2	3	4	5
.....					
d. Do you bring any required books and other materials to every class? .	1	2	3	4	5
.....					

REFERENCES CITED

- Aimé, A., Craig, W. M., Pepler, D., Jiang, D., & Connolly, J. (2008). Developmental pathways of eating problems in adolescents. *International Journal of Eating Disorders, 41*(8), 686–696. doi:10.1002/eat.20561
- American Psychiatric Association (APA). (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., t.). Washington, DC: Author.
- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- American Psychological Association Task Force on Psychological Intervention Guidelines. (1995). *Template for developing guidelines: Interventions for mental disorders and psychological aspects of physical disorders*. Washington, DC.
- Anderluh, M. B., Tchanturia, K., Rabe-Hesketh, S., & Treasure, J. (2003). Childhood obsessive-compulsive personality traits in adult women with eating disorders: Defining a broader eating disorder phenotype. *The American Journal of Psychiatry, 160*(2), 242–247. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/12562569>
- Beck, A. T., Steer, R. A., Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. *Clinical Psychology Review, 8*(1), 77–100.
- Becker, C. B., Smith, L. M., & Ciao, A. C. (2005). Reducing eating disorder risk factors in sorority members: A randomized trial. *Behavior Therapy, 36*, 245–253.
- Berscheid, E., Walster, E., Bohrnstedt, G. (1973). The happy American body: A survey report. *Psychology Today, 7*, 119–131.
- Burton, E., & Stice, E. (2006). Evaluation of a healthy-weight treatment program for bulimia nervosa: a preliminary randomized trial. *Behaviour Research and Therapy, 44*(12), 1727–1738. doi:10.1016/j.brat.2005.12.008
- Eddy, K., Celio, A., Hoste, R., Herzog, D., & le Grange, D. (2008). Eating disorder not otherwise specified in adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 47*(156-164).
- Enders, C. K. (2001). The impact of nonnormality on full information maximum-likelihood estimation for structural equation models with missing data. *Psychological Methods, 6*(4), 352–370. doi:10.1037//1082-989X.6.4.352
- Fabian, L. J., & Thompson, J. K. (1989). Body image and eating disturbance in young females. *International Journal of Eating Disorders, 74*(1), 63–74.

- Fairburn, C. G., & Bohn, K. (2005). Eating disorder NOS (EDNOS): An example of the troublesome “not otherwise specified” (NOS) category in DSM-IV. *Behaviour Research and Therapy*, *43*(6), 691–701. doi:10.1016/j.brat.2004.06.011
- Fairburn, C. G., Cooper, Z., Doll, H. a, & Davies, B. a. (2005). Identifying dieters who will develop an eating disorder: A prospective, population-based study. *The American Journal of Psychiatry*, *162*(12), 2249–2255. doi:10.1176/appi.ajp.162.12.2249
- Fairburn, C. G., Cooper, Z., Doll, H. a, Norman, P., & O’Connor, M. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry*, *57*(7), 659–665. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10891036>
- Fay, K., & Lerner, R. M. (2013). Weighing in on the Issue: A Longitudinal Analysis of the Influence of Selected Individual Factors and the Sports Context on the Developmental Trajectories of Eating Pathology Among Adolescents. *Journal of Youth and Adolescence*, *42*(1), 33–51. doi:10.1007/s10964-012-9844-x
- Fisher, M., Schneider, M., Burns, J., Symons, H., & Mandel, F. S. (2001). Differences between adolescents and young adults at presentation to an eating disorders program. *The Journal of Adolescent Health: Official Publication of the Society for Adolescent Medicine of the Society for Adolescent Medicine*, *28*(3), 222–227. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/11226845>
- Forbush, K. T., South, S. C., Krueger, R. F., Iacono, W. G., Clark, L. A., Keel, P. K., ... Watson, D. (2010). Locating eating pathology within an empirical diagnostic taxonomy: Evidence from a community-based sample. *Journal of Abnormal Psychology*, *119*(2), 282–292. doi:10.1037/a0019189
- Grabe, S., Ward, L. M., & Hyde, J. S. (2008). The role of the media in body image concerns among women: a meta-analysis of experimental and correlational studies. *Psychological Bulletin*, *134*(3), 460–76. doi:10.1037/0033-2909.134.3.460
- Graber, J. a., Brooks-Gunn, J., Paikoff, R. L., & Warren, M. P. (1994). Prediction of eating problems: An 8-year study of adolescent girls. *Developmental Psychology*, *30*(6), 823–834. doi:10.1037//0012-1649.30.6.823
- Green, M., Scott, N., Diyankova, I., & Gasser, C. (2005). Eating disorder prevention: An experimental comparison of high level dissonance, low level dissonance, and no-treatment control. *Eating Disorders*, *13*(2), 157–169. doi:10.1080/10640260590918955
- Greeno, C. G., Wing, R. R., & Shiffman, S. (2000). Binge antecedents in obese women with and without binge eating disorder. *Journal of Consulting and Clinical Psychology*, *68*(1), 95–102. doi:10.1037//0022-006X.68.1.95

- Halliwell, E., & Diedrichs, P. C. (2014). Testing a dissonance body image intervention among young girls. *Health Psychology, 33*(2), 201–204. doi:10.1037/a0032585
- Hawkins, R. C., & Clement, P. F. (1984). Binge eating: Measurement problems and a conceptual model. In R. C. Hawkins, W. J. Fremouw, & P. F. Clement (Eds.), *The binge-purge syndrome* (pp. 229–253). New York, NY: Springer.
- Horney, A. C., Stice, E., & Rohde, P. (2015). An examination of participants who develop an eating disorder despite completing an eating disorder prevention program: Implications for improving the yield of prevention efforts. *Prevention Science*.
- Horowitz, J. L., & Garber, J. (2006). The prevention of depressive symptoms in children and adolescents: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 74*(3), 401–415. doi:10.1037/0022-006X.74.3.401
- Hudson, J. I., Hiripi, E., Harrison, G. P. J., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biological Psychiatry, 61*(3), 348–358.
- IBM Corp. (2013). SPSS for Windows, version 22.0. Armonk, NY: IBM Corp.
- Johnson, J. G., Cohen, P., Kotler, L., Kasen, S., & Brook, J. S. (2002). Psychiatric disorders associated with risk for the development of eating disorders during adolescence and early adulthood. *Journal of Consulting and Clinical Psychology, 70*(5), 1119–1128. doi:10.1037//0022-006X.70.5.1119
- Jones, B. L., & Nagin, D. S. (2007). Advances in group-based trajectory modeling and an SAS procedure for estimating them. *Sociological Methods & Research, 35*, 542–571. doi:10.1177/0049124106292364
- Jones, B. L., Nagin, D. S., & Roeder, K. (2001). A SAS procedure based on mixture models for estimating developmental trajectories. *Sociological Methods & Research, 29*(3), 374–393. doi:10.1177/0049124101029003005
- Jones, M., Luce, K. H., Osborne, M. I., Taylor, K., Cunning, D., Doyle, A. C., ... Taylor, C. B. (2008). Randomized, controlled trial of an internet-facilitated intervention for reducing binge eating and overweight in adolescents. *Pediatrics, 121*(3), 453–462. doi:10.1542/peds.2007-1173
- Keel, P. K., Brown, T. a, Holm-Denoma, J., & Bodell, L. P. (2011). Comparison of DSM-IV versus proposed DSM-5 diagnostic criteria for eating disorders: Reduction of eating disorder not otherwise specified and validity. *The International Journal of Eating Disorders, 44*(6), 553–560. doi:10.1002/eat.20892

- Killen, J. D., Taylor, C. B., Hayward, C., Haydel, K. F., Wilson, D. M., Hammer, L., ... Strachowski, D. (1996). Weight concerns influence the development of eating disorders: A 4-year prospective study. *Journal of Consulting and Clinical Psychology, 64*(5), 936–940. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/8916622>
- Lavender, J. M., De Young, K. P., Franko, D. L., Eddy, K. T., Kass, A. E., Sears, M. S., & Herzog, D. B. (2011). An investigation of the joint longitudinal trajectories of low body weight, binge eating, and purging in women with anorexia nervosa and bulimia nervosa. *The International Journal of Eating Disorders, 44*(8), 679–686. doi:10.1002/eat.20880
- Leon, G. R., Fulkerson, J. A., Perry, C. L., Keel, P. K., & Klump, K. L. (1999). Three to four year prospective evaluation of personality and behavioral risk factors for later disordered eating in adolescent girls and boys. *Journal of Youth and Adolescence, 28*(2), 181–196.
- Levine, M. P. (2015). Does prevention work (and is this even a fair question)? *Eating Disorder Resource Catalogue*. Retrieved March 3, 2015, from <http://www.edcatalogue.com/does-prevention-work/>
- Linville, D., Stice, E., Gau, J., & O’Neil, M. (2011). Predictive effects of mother and peer influences on increases in adolescent eating disorder risk factors and symptoms: a 3-year longitudinal study. *The International Journal of Eating Disorders, 44*(8), 745–51. doi:10.1002/eat.20907
- Matusek, J. A., Wendt, S. J., & Wiseman, C. V. (2004). Dissonance thin-ideal and didactic healthy behavior eating disorder prevention programs: Results from a controlled trial. *The International Journal of Eating Disorders, 36*(4), 376–388. doi:10.1002/eat.20059
- McCabe, M. P., & Ricciardelli, L. a. (2006). A prospective study of extreme weight change behaviors among adolescent boys and girls. *Journal of Youth and Adolescence, 35*(3), 402–411. doi:10.1007/s10964-006-9062-5
- McMillan, W., Stice, E., & Rohde, P. (2011). High- and low-level dissonance-based eating disorder prevention programs with young women with body image concerns: an experimental trial. *Journal of Consulting and Clinical Psychology, 79*(1), 129–134. doi:10.1037/a0022143
- McPhee, L. (2000). *NOVA: Dying to be thin*. United States: PBS.
- McVey, G., Tweed, S., & Blackmore, E. (2007). Healthy Schools-Healthy Kids: A controlled evaluation of a comprehensive universal eating disorder prevention program. *Body Image, 4*(2), 115–136. doi:10.1016/j.bodyim.2007.01.004

- Measelle, J. R., Stice, E., & Hogansen, J. M. (2006). Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in adolescent girls. *Journal of Abnormal Psychology, 115*(3), 524–538.
- Meno, C. a, Hannum, J. W., Espelage, D. E., & Douglas Low, K. S. (2008). Familial and individual variables as predictors of dieting concerns and binge eating in college females. *Eating Behaviors, 9*(1), 91–101. doi:10.1016/j.eatbeh.2007.06.002
- Mitchell, K. S., Mazzeo, S. E., Rausch, S. M., & Cooke, K. L. (2007). Innovative interventions for disordered eating: Evaluating dissonance-based and yoga interventions. *International Journal of Eating Disorders, 40*(2), 120–128. doi:10.1002/eat
- Müller, S., & Stice, E. (2013). Moderators of the intervention effects for a dissonance-based eating disorder prevention program; results from an amalgam of three randomized trials. *Behaviour Research and Therapy, 51*(3), 128–33. doi:10.1016/j.brat.2012.12.001
- Muthén, B., Brown, C. H., Masyn, K., Jo, B., Khoo, S.-T., Yang, C.-C., ... Liao, J. (2002). General growth mixture modeling for randomized preventive interventions. *Biostatistics, 3*(4), 459–475. doi:10.1093/biostatistics/3.4.459
- Nagin, D. S., Dodge, K., Dugan, L., Garvin, A., Lynam, D., Raudenbush, S., & Roeder, K. (1999). Analyzing developmental trajectories: A semiparametric, group-based approach. *Psychological Methods, 4*(2), 139–157.
- Nagin, D. S., & Odgers, C. L. (2010). Group-based trajectory modeling in clinical research. *Annual Review of Clinical Psychology, 6*, 109–138. doi:10.1146/annurev.clinpsy.121208.131413
- Neumark-Sztainer, D., Butler, R., & Palti, H. (1995). Eating disturbances among adolescent girls: Evaluation of a school-based primary prevention program. *Journal of Nutrition Education, 27*(1), 24–31. doi:10.1016/S0022-3182(12)80262-7
- Patton, A. G. C., Selzer, R., Coffey, C., Carlin, J. B., Wolfe, R., Bmj, S., ... Coffey, C. (1999). Onset of adolescent eating disorders: Population based cohort study over 3 years. *British Medical Journal, 318*(7186), 765–768.
- Paxton, S. J., Neumark-Sztainer, D., Hannan, P. J., & Eisenberg, M. E. (2006). Body dissatisfaction prospectively predicts depressive mood and low self-esteem in adolescent girls and boys. *Journal of Clinical Child and Adolescent Psychology, 35*(4), 539–549. doi:10.1207/s15374424jccp3504_5
- Pearson, C. M. (2014). *Eating disorder onset in young girls : A longitudinal trajectory analysis*.
- Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychology Measurement, 1*(3), 385–401.

- Ricciardelli, L. A., & McCabe, M. P. (2001). Dietary restraint and negative affect as mediators of body dissatisfaction and bulimic behavior in adolescent girls and boys. *Behaviour Research and Therapy*, *39*(11), 1317–1328. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/11686266>
- Roberts, R. E., Lewinsohn, P. M., & Seeley, J. R. (1991). Screening for adolescent depression: A comparison of depression scales. *Journal of the American Academy of Child & Adolescent Psychiatry*, *30*(1), 58–66.
- Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, *7*(2), 147–177.
- Seeley, J. R., Stice, E., & Rohde, P. (2010). Screening for depression prevention: Identifying adolescent girls at high risk for future depression. *Journal of Abnormal Psychology*, *118*(1), 161–170. doi:10.1037/a0014741. Screening
- Seidel, A., Presnell, K., & Rosenfield, D. (2009). Mediators in the dissonance eating disorder prevention program. *Behaviour Research and Therapy*, *47*(8), 645–653. doi:10.1016/j.brat.2009.04.007
- Smith, G. T., Simmons, J. R., Flory, K., Annus, A. M., & Hill, K. K. (2007). Thinness and eating expectancies predict subsequent binge-eating and purging behavior among adolescent girls. *Journal of Abnormal Psychology*, *116*(1), 188–197. doi:10.1037/0021-843X.116.1.188
- Southgate, L., Tchanturia, K., & Treasure, J. (2005). Building a model of the aetiology of eating disorders by translating experimental neuroscience into clinical practice. *Journal of Mental Health*, *14*(6), 553–566. doi:10.1080/09638230500347541
- Spoor, S. T. P., Stice, E., Bekker, M. H. J., Strien, T. Van, Croon, M. A., & Heck, G. L. Van. (2006). Relations between dietary restraint, depressive symptoms, and binge eating: A longitudinal study. *International Journal of Eating Disorders*, *39*, 700–707. doi:10.1002/eat
- Stewart, D. a, Carter, J. C., Drinkwater, J., Hainsworth, J., & Fairburn, C. G. (2001). Modification of eating attitudes and behavior in adolescent girls: A controlled study. *The International Journal of Eating Disorders*, *29*(2), 107–118. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/11429973>
- Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect.
- Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, *128*(5), 825–848. doi:10.1037//0033-2909.128.5.825

- Stice, E., Akutagawa, D., Gaggar, A., & Agras, W. S. (2000). Negative affect moderates the relation between dieting and binge eating. *The International Journal of Eating Disorders*, *27*(2), 218–229. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10657895>
- Stice, E., Fisher, M., & Martinez, E. (2004). Eating disorder diagnostic scale: Additional evidence of reliability and validity. *Psychological Assessment*, *16*(1), 60–71. doi:10.1037/1040-3590.16.1.60
- Stice, E., Marti, C. N., & Durant, S. (2011). Risk factors for onset of eating disorders: Evidence of multiple risk pathways from an 8-year prospective study. *Behaviour Research and Therapy*, *49*(10), 622–627. doi:10.1016/j.brat.2011.06.009
- Stice, E., Marti, C. N., Shaw, H., & Jaconis, M. (2009). An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology*, *118*(3), 587–597. doi:10.1037/a0016481
- Stice, E., Marti, C. N., Spoor, S., Presnell, K., & Shaw, H. (2008). Dissonance and healthy weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology*, *76*(2), 329–340. doi:10.1037/0022-006X.76.2.329
- Stice, E., Marti, N., Shaw, H., & O’Neil, K. (2008). General and program-specific moderators of two eating disorder prevention programs. *The International Journal of Eating Disorders*, *41*(7), 611–7. doi:10.1002/eat.20524
- Stice, E., Mazotti, L., Weibel, D., & Agras, W. S. (2000). Dissonance prevention program decreases thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: A preliminary experiment. *International Journal of Eating Disorders*, *27*, 206–217.
- Stice, E., & Presnell, K. (2007). *The Body Project: Body acceptance and preventing eating disorders, Facilitators guide*. New York, NY: Oxford University Press.
- Stice, E., Presnell, K., Gau, J., & Shaw, H. (2009). Testing mediators of intervention effects in randomized controlled trials: An evaluation of two eating disorder prevention programs. *Journal of Consulting and Clinical Psychology*, *75*(1), 20–32. doi:10.1037/0022-006X.75.1.20
- Stice, E., Presnell, K., & Spangler, D. (2002). Risk factors for binge eating onset in adolescent girls: A 2-year prospective investigation. *Health Psychology*, *21*(2), 131–138. doi:10.1037//0278-6133.21.2.131
- Stice, E., Rohde, P., Durant, S., & Shaw, H. (2012). A preliminary trial of a prototype Internet dissonance-based eating disorder prevention program for young women with body image concerns. *Journal of Consulting and Clinical Psychology*, *80*(5), 907–16. doi:10.1037/a0028016

- Stice, E., Rohde, P., Gau, J., & Shaw, H. (2009). An effectiveness trial of a dissonance-based eating disorder prevention program for high-risk adolescent girls. *Journal of Consulting and Clinical Psychology, 77*(5), 825–34. doi:10.1037/a0016132
- Stice, E., Rohde, P., Gau, J., & Shaw, H. (2012). Effect of a dissonance-based prevention program on risk for eating disorder onset in the context of eating disorder risk factors. *Prevention Science, 13*(2), 129–139. doi:10.1007/s11121-011-0251-4
- Stice, E., Shaw, H., Bohon, C., Marti, C. N., & Rohde, P. (2009). A meta-analytic review of depression prevention programs for children and adolescents: Factors that predict magnitude of intervention effects. *Journal of Consulting and Clinical Psychology, 77*(3), 486–503. doi:10.1037/a0015168
- Stice, E., Shaw, H., Burton, E., & Wade, E. (2006). Dissonance and healthy weight eating disorder prevention programs: A randomized efficacy trial. *Journal of Consulting and Clinical Psychology, 74*(2), 263–275.
- Stice, E., & Shaw, H. E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings. *Journal of Psychosomatic Research, 53*(5), 985–993. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/12445588>
- Stice, E., South, K., & Shaw, H. (2012). Future directions in etiologic, prevention, and treatment research for eating disorders. *Journal of Clinical Child and Adolescent Psychology : The Official Journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53, 41*(6), 845–55. doi:10.1080/15374416.2012.728156
- Stice, E., Sysko, R., Roberto, C. a, & Allison, S. (2010). Are dietary restraint scales valid measures of dietary restriction? Additional objective behavioral and biological data suggest not. *Appetite, 54*(2), 331–339. doi:10.1016/j.appet.2009.12.009
- Stice, E, Marti, N.C., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective study of young women. *Journal of Abnormal Psychology, 0682*.
- Stice, E., Rohde, P., Butryn, M., Menki, K., & Marti, N. (2015). Randomized controlled pilot trial of a novel dissonance-based group treatment for eating disorders. *Behaviour Research and Therapy, 65*, 67–75.
- Stice, E., Yokum, S., & Waters, A. (n.d.). Dissonance-based eating disorder prevention program reduces reward region response to thin models: How actions shape valuation. *Plus ONE*.
- Striegel-Moore, R. H., Dohm, F., Kraemer, H. C., Schreiber, G. B., Taylor, C. B., & Daniels, S. R. (2007). Risk factors for binge-eating disorders: An exploratory study. *International Journal of Eating Disorders, 40*, 481–488. doi:10.1002/eat

- Strien, T., Frijters, J. E. R., van Staveren, W. A., Defares, P. B., & Deurenberg, P. (1986). The predictive validity of the dutch restrained eating scale. *International Journal of Eating Disorders*, 5(4), 747–755.
- Suisman, J. L., Slane, J. D., Burt, S. A., & Klump, K. L. (2008). Negative affect as a mediator of the relationship between weight-based teasing and binge eating in adolescent girls. *Eating Behaviors*, 9(4), 493–496. doi:10.1016/j.eatbeh.2008.04.001
- Tellegen, A., Watson, D., & Clark, L. A. (1988). Development and validation of brief measures of positive and negative affect. *Journal of Personality and Social Psychology*, 54, 1063–1070.
- The McKnight Investigators. (2003). Risk factors for the onset of eating disorders in adolescent girls: Results of the McKnight longitudinal risk factor study. *The American Journal of Psychiatry*, 160(2), 248–254. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/12562570>
- Thompson, J. K., & Stice, E. (2001). Thin-Ideal Internalization: Mounting Evidence for a New Risk Factor for Body-Image Disturbance and Eating Pathology. *Current Directions in Psychological Science*, 10(5), 181–183. doi:10.1111/1467-8721.00144
- Touchette, E., Henegar, A., Godart, N. T., Pryor, L., Falissard, B., Tremblay, R. E., & Côté, S. M. (2011). Subclinical eating disorders and their comorbidity with mood and anxiety disorders in adolescent girls. *Psychiatry Research*, 185, 185–192. doi:10.1016/j.psychres.2010.04.005
- Tyrka, A. R., Graber, J. A., Brooks-Gunn, J. (2000). The development of disordered eating: Correlates and predictors of eating problems in the context of adolescence. In S. M. Sameroff, A. J., Lewis, M., Miller (Ed.), *Handbook of Developmental Psychopathology* (2nd ed., pp. 607–624). New York, NY: Plenum Publishers.
- Vitiello, B., & Lederhendler, I. (2000). Research on eating disorders: Current status and future prospects. *Biological Psychiatry*, 47(9), 777–86. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10812036>
- Wade, T. D., Bergin, J. L., Tiggemann, M., Bulik, C. M., & Fairburn, C. G. (2006). Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *The Australian and New Zealand Journal of Psychiatry*, 40(2), 121–8. doi:10.1111/j.1440-1614.2006.01758.x
- Watson, D., & Clark, L. a. (1992). Affects separable and inseparable: On the hierarchical arrangement of the negative affects. *Journal of Personality and Social Psychology*, 62(3), 489–505. doi:10.1037//0022-3514.62.3.489
- Wertheim, E. H., Koerner, J., & Paxton, S. J. (2001). Longitudinal predictors of restrictive eating and bulimic tendencies in three different age groups of adolescent girls. *Journal of Youth and Adolescence*, 30(1), 69–81.

- Whiteside, U., Chen, E., Neighbors, C., Hunter, D., Lo, T., & Larimer, M. (2007). Difficulties regulating emotions: Do binge eaters have fewer strategies to modulate and tolerate negative affect? *Eating Behaviors*, 8(2), 162–169. doi:10.1016/j.eatbeh.2006.04.001
- Wichstrom, L. (2000). Psychological and behavioral factors unpredictable of disordered eating: A prospective study of the general adolescent population in Norway. *International Journal of Eating Disorders*, 28(1), 33–42.
- Wichstrøm, L. (2000). Psychological and behavioral factors unpredictable of disordered eating: A prospective study of the general adolescent population in Norway. *The International Journal of Eating Disorders*, 28(1), 33–42. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10800012>
- Wilson, G. T., Fairburn, C. G., & Agras, W. S. (1997). Cognitive-behavioral therapy for bulimia nervosa. In P. E. Garner, D. M., Garfinkel (Ed.), *Handbook of treatment for eating disorders* (2nd ed., pp. 67–93). New York, NY: Guilford Press.
- Zaider, T. I., Johnson, J. G., & Cockell, S. J. (2000). Psychiatric comorbidity associated with eating disorder symptomatology among adolescents in the community. *The International Journal of Eating Disorders*, 28(1), 58–67. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10800014>