

Distribution Agreement

In presenting this thesis or dissertation as a partial fulfillment of the requirements for an advanced degree from Emory University, I hereby grant to Emory University and its agents the non-exclusive license to archive, make accessible, and display my thesis or dissertation in whole or in part in all forms of media, now or hereafter known, including display on the world wide web. I understand that I may select some access restrictions as part of the online submission of this thesis or dissertation. I retain all ownership rights to the copyright of the thesis or dissertation. I also retain the right to use in future works (such as articles or books) all or part of this thesis or dissertation.

Signature:

Lisa M. Smith

Date

Eating Disorder Prevention in Sororities: Testing
Mediators of Intervention Effects

By

Lisa M. Smith
Master of Arts

Psychology

Linda W. Craighead, Ph.D.
Advisor

Lawrence Barsalou, Ph.D.
Committee Member

Marshall Duke, Ph.D.
Committee Member

Accepted:

Lisa A. Tedesco, Ph.D.
Dean of the Graduate School

Date

Eating Disorder Prevention in Sororities: Testing
Mediators of Intervention Effects

By

Lisa M. Smith
Bachelor of Arts

Advisor: Linda W. Craighead, Ph.D.

An abstract of
A thesis submitted to the Faculty of the Graduate School of Emory University
in partial fulfillment of the requirements for the degree of
Master of Arts
in Psychology
2009

Abstract

Eating Disorder Prevention in Sororities: Testing Mediators of Intervention Effects

By Lisa M. Smith

This study evaluated mechanisms through which intervention effects were achieved in a cognitive dissonance-based program (CD) that significantly reduced eating disorder risk factors among sorority women. CD is hypothesized to reduce body dissatisfaction, and thus risk for disordered eating. More importantly CD is hypothesized to reduce body dissatisfaction primarily through reducing the degree of thin-ideal internalization (Becker, Smith & Ciao, 2005). Thus, the current study evaluated the degree to which thin-ideal internalization and body dissatisfaction each serve as specific mechanisms of change within CD. CD produced significant reductions in the outcome measures (dietary restraint and eating disorder pathology) and in reductions in both of the mediators (thin-ideal internalization and body dissatisfaction). Our results provide some support for partial and complete mediation in outcome measures during intervention and in change sustained from pre-intervention to one-month follow-up. Our data suggest, however, that ultimately reduced body dissatisfaction is the primary mediator of sustained reductions in eating disordered behaviors.

Eating Disorder Prevention in Sororities: Testing
Mediators of Intervention Effects

By

Lisa M. Smith
Bachelor of Arts

Advisor: Linda W. Craighead, Ph.D.

A thesis submitted to the Faculty of the Graduate School of Emory University
in partial fulfillment of the requirements for the degree of
Master of Arts
in Psychology
2009

Table of Contents

Introduction.....	1
Current Study.....	18
Participants.....	19
Procedure.....	19
Measures.....	22
Constructs.....	25
Statistical Analyses.....	25
Results.....	29
Discussion.....	45
Limitations.....	51
Future Directions.....	51
References.....	53
Table 1.....	61
Table 2.....	62
Table 3.....	63
Table 4.....	64
Figure 1.....	65
Figure 2.....	66
Figure 3.....	67
Figure 4.....	68

Eating disorder prevention in sororities: Testing
mediators of intervention effects

Eating disorder prevention is important because of the early average age of onset and because acute symptoms often become chronic conditions that are associated with significant medical complications including bone density depreciation or osteoporosis, cardiac problems, and even death (Fairburn & Harrison, 2003; Sullivan, 1995; Treasure & Szmukler, 1995). Additionally, eating disorders have a high rate of comorbid psychopathology, including anxiety disorders, depression, and substance use disorders (Johnson, Cohen, Kasen, & Brook, 2002). Most individuals with eating disorders suffer a variable course, even if receiving treatment (Wilson, Becker, & Heffernan, 2002). Young adult women are an easily identifiable high-risk group for developing disordered eating habits. Women comprise approximately 90% of all individuals diagnosed with an eating disorder (American Psychiatric Association, 1995) and the typical age of onset of disordered eating falls during middle to late adolescence. Recent research suggests that approximately 11-12% of young women have clinically significant disordered eating symptoms, including the diagnoses of bulimia nervosa (BN) and partial BN, binge eating disorder (BED) and partial BED, purging disorder, and anorexia nervosa (AN) and partial AN (Keel, Heatherton, Dorer, Joiner, and Zalta, 2006). Epidemiological research estimates that 3% of adolescent and college-aged females meet full criteria for eating disorders (Becker, Grinspoon, Klibanski, & Herzog, 1999; Hoek, 1995; Hoek & VanHoeken, 2003).

Although full syndrome AN and BN occur in a minority of college women (3%), research indicates that sub-threshold eating pathology is much more common in this

population (Mintz & Betz, 1988). For example, up to 61% of undergraduate women engage in disordered or problematic eating (Mintz & Betz, 1988), suggesting that prevention of eating disordered behaviors is an important goal among college women. Furthermore, many students who experience disordered eating doubt that their symptoms require treatment and/or do not pursue therapy (Becker, Franko, Nussbaum, & Herzog, 2004; Meyer, 2005). Because treatment of severe and/or chronic eating disorders is only modestly effective and because there are substantial comorbid medical and psychological complications associated with eating disorders (Wilson et al., 2002), successful prevention of eating disorders is warranted.

Additionally, college women are a population of particular interest with regards to the etiology, prevention, and treatment of eating disorders because a woman's college tenure often involves major life changes and stressors, which are risk factors for engaging in eating disordered behaviors (Cooley & Toray, 2001). In conjunction with major life changes and stressors encountered by undergraduate women, the American culture values thinness as the standard of beauty and this is especially evident among college women. Cultural attitudes valuing thinness are contagious, and social pressures to conform exacerbate women's negative feelings about their appearance (Crandall, 1988). This cultural concept of thinness as the standard of beauty is known as the "thin ideal" and the media perpetuates this ideal. Through exposure to magazines, television, and advertising in general, women are constantly told they are not beautiful enough, and this is particularly the case if they are not thin. In addition, higher body mass is posited to exacerbate the usual social pressures to be thin, therefore heightening body dissatisfaction (Stice, Chase, Stormer, & Appel, 2001). Furthermore, media exposure directly affects

eating pathology through factors such as internalization of the thin ideal and body dissatisfaction, (Stice, Schupak-Neuber, Shaw, & Stein, 1994).

In a society endorsing thinness as the standard of beauty, the drive for thinness perseveres. The drive for thinness is particularly problematic, as this mentality has been acknowledged as a risk factor for developing eating disorders (Stice & Agras, 1998). Body image, or the way one subjectively experiences one's body weight and shape, is a construct strongly related to drive for thinness. Poor body image has been well established as a risk factor for developing maladaptive eating behaviors and dieting (Cooley & Toray, 2001). Encompassed within the notion of body image is the concept of body dissatisfaction. Women experiencing higher levels of dissatisfaction about their bodies are at a greater risk for developing eating disorders, as body dissatisfaction has been repeatedly linked to disordered eating habits (Attie & Brooks-Gunn, 1989; Heatherton, Mahamedi, Striepe, Field, & Keel, 1997; Killen, et al., 1996; Stice & Agras, 1998; Striegel-Moore, Silberstein, Frensch, & Rodin, 1989). Therefore, with contagious social pressures to pursue the thin ideal (Crandall, 1988), leading to increased drive for thinness (Stice & Agras, 1998), and major life changes (Cooley & Toray, 2001), college women are an important population to target in the reduction of eating disorders risk factors.

Within the undergraduate population, women in sororities were initially believed to be at a heightened risk for developing eating disordered behaviors. The assumption of higher risk was based on evidence that women who are exceptionally concerned with social perception and expectations are at greater risk to develop eating disorders in order to meet such expectations (Striegel-Moore, Silberstein, and Rodin, 1993). Furthermore,

research has indicated that on average women in sororities are very invested in physical appearance, weight, and body shape, (Rose, 1985), which are established risk factors for developing bulimia (Striegel-Moore, Silberstein, and Rodin, 1986). While the perception that sorority women are at increased risk for eating pathology, surprisingly little research has concluded that sorority women do in fact engage in significantly more disordered eating behaviors (Alexander, 1998).

Evidence for this hypothesis remains inconclusive, and limited data suggest that members may be at somewhat increased risk for eating disorders. For example, Crandall (1988) found that, within a sorority, the most popular sorority members were more likely to binge eat compared to non-sorority women. However, research has not generally supported the hypothesis that sorority members engage in significantly greater eating disordered behaviors when compared to non-sorority students (Cashel, Cunningham, Landeros, Cokley, & Muhammad, 2003) or compared to control college women as well as to a group of women with higher levels of activity, such as women on athletic and dance teams (Alexander, 1998). Recent research indicates that sorority women endorse more risk factors for developing eating disorders, such as problematic eating attitudes and perceived social pressure compared to non-sorority women, and that sororities attract women who endorse such risk factors (Basow, Foran, & Bookwala, 2007). Allison and Park (2004) found that sorority women did not differ on eating disordered behaviors from non-sorority women at the time of joining a sorority. Over time, however, sorority women endorsed greater drive for thinness, although not more eating disordered behaviors, than non-sorority members.

These recent studies suggest that membership in sororities may well increase risk factors for developing eating disorders, but rates of engaging in eating disordered behaviors do not differ between sorority and non-sorority undergraduate women. Despite the paucity of research documenting that sorority women engage in more eating disordered behaviors than other college women, sorority members do appear to endorse more problematic attitudes towards eating and body image compared to non-sorority students so they represent an important population to target. Additionally, as explained later, there are other compelling reasons to utilize the highly organized social structure of sorority systems as an effective and efficient way to reach a substantial proportion of the female college population.

In order to combat external influences leading to poor body image, such as social pressure and media exposure, researchers have attempted to develop preventative interventions. Significant effort has been devoted to preventing eating disorders at a variety of age levels. Until recently, however, many programs yielded few positive results, particularly with respect to behavior change (Pearson, Goldklang, & Striegel-Moore, 2002; Stice & Shaw, 2004). These early prevention programs targeted eating disordered behaviors, which in at least one study resulted in an increase in eating disordered behaviors (Killen et al., 1993, cited in Franko & Orosan-Weine, 1998). Killen et al. (1993) implemented an 18-week eating disorders prevention program in junior high school health classes, focusing on the problems of extreme weight regulation. After this program, the participants reported an increase in knowledge of eating disordered behaviors however they did not decrease practicing eating disordered behaviors. As a result, Killen et al. (1993) proposed that eating disorders prevention might need to target

only at-risk individuals and to utilize a smaller group so intervention might be more effective in changing behaviors (cited in Franko & Orosan-Weine, 1998).

In another effort, Mann and colleagues (Mann, Nolen-Hoeksema, & Huang, 1997) conducted an eating disorders prevention program including both healthy participants (primary prevention) and high-risk participants (secondary prevention). In this program, Mann and colleagues (1997) utilized an informational approach including facts about eating disordered behaviors and testimonials from recovered eating disorders patients. Results again indicated that healthy participants actually increased eating disordered behaviors after participation in the prevention program.

Mann et al. (1997) hypothesized that their effort to intervene with combined primary and secondary prevention did not work well because the strategies for eating disorders prevention for primary and secondary prevention need to be different. For primary prevention, research suggests emphasizing that eating disordered behaviors are abnormal and not simply a step beyond dieting, in order to discourage participants from trying problem behaviors. In secondary prevention, eating disorders are presented as common, treatable, and as a small step beyond normal dieting, to encourage participants to believe they can scale back to more normal levels of dieting and prevent clinical problems. Because the foci of these two strategies counteract each other, initially healthy participants may be harmed by the strategies of a secondary prevention method (Mann et al., 1997).

Secondly, Mann et al. (1997) suggested that an informational approach including patient testimonials could have provided information on how to engage in disordered eating for those participants who were unaware of such behaviors. Participants who were

not previously concerned with eating behaviors witnessed a patient testimonial, which served as a model for eating disordered behaviors. The informational approach unintentionally gave the initially healthy participants greater directions on how to have an eating disorder, so eating disordered behaviors increased (Mann et al., 1997). Mann et al. (1997) concluded that both factors (information and testimonials) of the prevention program undermined the strategy needed for primary prevention. Therefore, Mann and colleagues argued that primary and secondary prevention efforts should be kept separate, and suggested that novel approaches to eating disorders prevention were needed (Mann et al., 1997).

After this call for a new focus in eating disorders prevention, Stice and colleagues (Stice, Mazotti, Weibal, & Agras, 2000; Stice, Chase, Stormer, & Appel, 2001) created an eating disorders prevention program utilizing a quite different approach, following the suggestions of Mann and colleagues (Mann et al., 1997). Stice and colleagues (2000) targeted improving body image (reducing a risk factor) rather than “reducing” actual eating disorders. According to the dual-pathway model for the development of bulimia (Figure 1; Stice, Ziemba, Margolis, & Flick, 1996), social pressures to be thin result in greater internalization of the thin ideal, greater body dissatisfaction, and more dieting behaviors in women. As explained earlier, elevations in thin-ideal internalization and body dissatisfaction are well-established risk factors for developing disordered eating habits (Stice & Agras, 1998, Attie & Brooks-Gunn, 1989; Heatherton, Mahamedi, Striepe, Field, & Keel, 1997; Killen, et al., 1996; Stice & Agras, 1998; Striegel-Moore, Silberstein, Frensch, & Rodin, 1989). In the dual-pathway model, dieting is one of the two final proximal predictors for developing bulimia (the other being negative affect).

These two final predictors mediate the effects of the earlier risk factors, thin-ideal internalization and body dissatisfaction (Stice et al., 1996). Based on this dual-pathway model for developing bulimia, Stice and colleagues (2000) decided to target body image risk factors, most specifically thin-ideal internalization, in the prevention of eating disorders (Stice et al., 2001). Stice and colleagues targeted thin-ideal internalization because that risk factor occurs earliest in the dual-pathway causal model for developing bulimia (Stice et al., 2001).

Targeting risk factors early in a causal chain for developing a pathological behavior is a commonly used strategy to initiate behavior change within cognitive-behavioral approaches to intervention (Craighead, Kazdin, & Mahoney, 1976). From a theoretical behavioral perspective, the rationale for this strategy is that more established behaviors or thought patterns are more difficult to extinguish or to counteract with a competing behavior or thought. Intervention is more likely to be successful when implemented either early in the causal chain for engaging in a problematic behavior or thought pattern or early in the development of a problematic behavior or thought pattern before that pattern has become a well-established response (Craighead et al., 1976).

Stice and colleagues conceptualized thin-ideal internalization as an “attitude” that needed to be changed. That conceptualization led Stice to develop a program based on the theory of cognitive dissonance (Festinger, 1957). In order to retain the efficiency and cost-effectiveness of using a group approach that could reach a larger number of women, Stice and colleagues developed a primary prevention cognitive dissonance-based approach to promote attitudinal change in groups of participants (Stice et al., 2000; 2001). This approach was derived from the social psychology principle of cognitive

dissonance, a phenomena that occurs when people experience a discrepancy between their stated beliefs and their actions (Festinger, 1957). Such a discrepancy leads to psychological distress, which then motivates the individual to reduce the inconsistency. The theory suggests that this is typically done by bringing the discordant beliefs more in line with their behavior. Thus, for an intervention one would attempt to elicit counter-attitudinal behaviors as the mechanism to achieve attitude change. Experimental studies suggest that the degree of dissonance experienced by the individual is impacted both by the importance of the issue at hand and the degree of incongruity between the belief and the behavior, such that the greater the degree of dissonance experienced, the more likely the individual is to attempt to reconcile it (Festinger, 1957).

Cognitive dissonance theory has been predominantly studied in social psychology, with respect to attitude change, stimulus devaluation, and perception of experiences. First explicated by Festinger (1957), this theory has been studied extensively in research examining attitude change both individually and in groups of participants. Groups have been studied because an individual incorporates the values of the group into his/her self-concept and beliefs, and in turn the level of dissonance experienced may be influenced by group behavior (Tajfel & Turner, 1979). Within this theory, group behavior may impact dissonance in two different ways. One example is the case study by Festinger and colleagues (1956), where group behavior prevented cognitive dissonance in the individual (Festinger, Reicken, & Schachter, 1956). This case study examined how religious groups responded when religious prophecies did not occur and yet the faith of the group persisted (Festinger et al., 1956). One explanation for this specific phenomenon was that cognitive dissonance and therefore attitude change occurs when a member of the

individual's ingroup behaves counter to the individual's attitude, but not when a member of an outgroup acts counter to the individual's attitude (Glasford, Pratto, & Dovidio, 2008). In the case study by Festinger and colleagues (1956), the ingroup did not behave counter to their attitudes, which therefore prevented dissonance.

The other impact of group behavior on dissonance is that group behavior can promote cognitive dissonance (Matz & Wood, 2005; Norton, Monin, Cooper, & Hogg, 2003; Glasford et al., 2008). For example, individual group members experience cognitive dissonance when they learn that other members of their ingroup hold opinions contrary to their own beliefs. In this case, dissonance is promoted when members of an ingroup are shown as having opposing beliefs to an individual member. Dissonance is promoted because the individual member defines themselves partly as being part of the group (Matz & Wood, 2005). Furthermore, individuals observing a fellow ingroup member give a speech contrary to the speaker's belief may also experience vicarious cognitive dissonance. In this case, the observer knows the attitude of the fellow ingroup speaker, and is aware that the speaker is presenting an attitude counter to his/her own beliefs. In observing this situation, the individual experiences vicarious dissonance by witnessing another ingroup member experience dissonance (Norton et al., 2003).

However, group behavior does not promote cognitive dissonance in an individual when it is an outgroup member behaving in a manner contrary to the individual's attitude. Glasford and colleagues (2008) demonstrated that an individual experienced cognitive dissonance when a member of his/her own ingroup violated a personal value, but not when a member of an outgroup violated a personal value. Dissonance findings are relevant to the current intervention because we utilize both the theory of cognitive

dissonance as well as a social systems approach. In the social systems approach, we target a social system, or ingroup, and attempt to promote cognitive dissonance by engaging participants in speaking out and behaving in a manner counter to both her individual attitude and the attitude of the ingroup.

In the Cognitive Dissonance eating disorders prevention intervention (CD), the goal is to induce cognitive dissonance by having participants make statements and engage in behaviors that are contrary to the thin-ideal standard of beauty. For example, participants commit to participating with an open mind, generate and verbalize their own understanding of the negative consequences and costs of pursuing the thin ideal, and practice persuading others to reconsider pursuing the thin ideal through role play exercises. Participants must then reconcile these actions with their previously held beliefs endorsing the thin ideal. Theoretically, this reconciliation would lead to changes in their original attitudes or beliefs. In this case, reconciliation results in a decrease in thin-ideal internalization, and consequently decreases in body dissatisfaction, negative affect, and dieting, which are the more proximal risk factors according to the dual-pathway of developing bulimia (Stice et al., 1996).

The original CD program consisted of three 1-hour group sessions, where participants were asked to make statements and commit to acts contrary to their previously held beliefs regarding body image. By moving the focus away from eating disorders and instead focusing on body image, Stice et al. (2000) eliminated the informational and testimonial approach of previous research (Mann et al., 1997), which had been shown to be problematic. Moreover, in a series of studies (Stice, Chase, Stormer, & Appel, 2001; Stice, Mazotti, Weibel, & Agras, 2000; Stice, Trost, & Chase,

2003; Stice, Shaw, Burton, & Wade, 2006), Stice and colleagues demonstrated that CD clearly did reduce both distal and more proximal eating disorder risk factors: thin-ideal internalization, dieting, body dissatisfaction, and negative affect.

In building on the positive findings of Stice and colleagues over the years (2000, 2001, 2003, 2006), Becker and colleagues (Becker, Jilke, & Polvere, 2002) decided to utilize a social systems approach to enhance the effects of CD. In an undergraduate setting, one of the most accessible social systems is the sorority system. Although research indicating that sorority women may be at heightened risk for clinical eating disorders is equivocal (Rose, 1985; Cashel, Cunningham, Landeros, Cokley, & Muhammad, 2003; Basow, Foran, & Bookwala, 2007; Allison & Park, 2004), the sorority system still provides an efficient way to reach a large percentage of the female undergraduate population. Sorority women have the ability to harness the power of their sorority system as a large undergraduate social network, and therefore have the potential to take the lead in promoting undergraduate cultural change more generally. The nature of sorority structure lends itself well to providing a primary type of intervention. As noted by Levine and Piran (1999), most prior prevention research, including most CD research, targeted individuals. In other words, most programs attempt to create change in the individual, not a larger social system. Yet, prevention efforts with individuals are likely to be most effective when accompanied by simultaneous efforts to change and engage the social systems that influence those individuals (Levine & Piran, 1999). Additionally, by inducing cognitive dissonance and therefore attitude change in the ingroup of sorority women, the individuals are more likely to experience cognitive dissonance both directly (Glasford et al., 2008) and vicariously (Norton et al., 2003).

The engagement of social systems also may facilitate the long-term implementation of prevention programs because social systems employ significant resources to maintain a program if it is deemed useful. Furthermore, research indicates that peers play an integral role in identifying disordered eating habits and in advocating that a friend with disordered eating seek professional help (Suls, Martin, & Leventhal, 1997). For example, Price and Desmond (1990) found that referrals by fellow students, parents, or teachers are a common way that students with eating disorders are identified. By implementing a prevention program that includes all members of a group, awareness of all is increased as well as the chance that a healthier peer would facilitate referrals for less healthy peers.

In an extension of the early work by Stice and colleagues' work (2000, 2001, 2003, 2006), Becker and colleagues at Trinity University (2002) compared CD, Media Psychoeducation (MP), and waitlist control (WL) in sorority members specifically identified as high risk, based on elevated scores on the Body Shape Questionnaire (Cooper, Taylor, Cooper, Z., & Fairburn, 1987). Becker and colleagues (2002) modified the CD program to consist of two, two-hour group sessions and found that both CD and MP reduced dietary restraint, eating pathology, and body dissatisfaction. CD also resulted in decreased thin-ideal internalization, and yielded significantly greater reduction in body dissatisfaction compared to MP. It was notable that sorority participants who participated then requested that all sorority members be allowed to participate, regardless of risk status. After opening the program to all sorority members, regardless of risk for developing eating disorders, Becker, Smith, and Ciao (2005) found that both CD and MP interventions reduced body dissatisfaction, dietary restraint, and overall eating disorder

pathology at one-month follow-up. CD also reduced thin-ideal internalization compared to waitlist. Finally, analyses indicated that both lower- and higher-risk members benefited from both CD and MP, which in fact supported the conclusion that universal prevention programs may not increase risk for developing disordered eating in low-risk participants, as earlier studies using an educational approach had suggested. In a follow-up study comparing CD to MP in new sorority members, Becker, Smith, and Ciao (2006) found CD to be superior at 8-month follow-up in reducing dietary restraint, thin-ideal internalization, body dissatisfaction, and eating pathology.

Although prevention programs now appear more promising (Stice et al., 2000, 2001, 2003, 2006; Becker et al., 2002, 2005, 2006), additional work is still needed. In a meta-analytic review, Stice and Shaw (2004) concluded that eating disorder prevention programs need enhanced methodologies and more theoretical foundation. Identification of mediators or moderators of intervention was suggested as an important way to enhance understanding of the processes that were involved (Stice, Presnell, Gau & Shaw, 2007).

To our knowledge, only two studies of eating disorder prevention have examined mediators of prevention intervention effects (Bearman, Stice & Chase, 2003; Stice et al., 2007). Bearman and colleagues investigated body dissatisfaction as a potential mediator of intervention effects within a body acceptance prevention program (Bearman et al., 2003). They found body dissatisfaction partially mediated intervention effects, as assessed by measures of bulimic and depressive symptoms (Bearman et al., 2003). In 2007, Stice and colleagues examined thin-ideal internalization as a mediator of a cognitive dissonance-based eating disorder prevention program (CD) in high-risk participants. Stice and colleagues (2007) predicted that reductions in internalization of the

thin-ideal, as measured by the Ideal Body Stereotype Scale (IBSS-R; Stice, Ziemba, Margolis, & Flick, 1996), would mediate the effects of CD results as assessed by four outcome measures: body dissatisfaction, negative affect, bulimic symptoms, and dieting. To test this hypothesis very specifically, Stice and colleagues (2007) included two comparison groups that were not hypothesized to work through reductions in thin-ideal internalization. Stice and colleagues (2007) predicted that reduction in cognitive dissonance would mediate change in the CD program but that positive changes in healthy eating and physical activity would mediate the effects of a healthy weight management program.

Using a rigorous, 5-tier system to identify mediators, Stice and colleagues investigated thin-ideal internalization as a mediator and found that it partially mediated intervention effects of CD on measures of body dissatisfaction, negative affect, bulimic symptoms, and dieting (Stice et al., 2007). In an effort to explain why the mediation was not more complete, Stice and colleagues (2007) pointed out that a more global measure of the thin-ideal internalization, such as the Social Attitudes Toward Appearance Questionnaire (Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004), might show a more significant mediational effect than the measure they used (Ideal Body Stereotypes Scale-Revised; Stice, Fisher, & Martinez, 2004). Most importantly, Stice and colleagues (2007) found that thin-ideal internalization was a specific mediator for the CD approach, (i.e. thin-ideal internalization showed partial mediation for CD but not for Healthy Weight Management).

In the current study, these two previously studied mediators (thin-ideal and body dissatisfaction) were evaluated within the context of a CD replication study (Becker et

al., 2005). In the Stice et al. 2007 study, body dissatisfaction had been conceptualized as an outcome measure since thin-ideal internalization viewed as a precursor to body dissatisfaction. However, we choose to consider body dissatisfaction a potential mediator since body dissatisfaction precedes dietary restraint, which we consider the first outcome since it is the first behavioral sign of “disordered eating”. Thus, we hypothesized that thin-ideal internalization and body dissatisfaction would each distinctly mediate reduction in the outcomes, dietary restraint and global eating pathology.

These two risk factors (identified in the dual-pathway model) can be added to the standard cognitive-behavioral model (Fairburn, Marcus, & Wilson, 1993, Figure 2) to enhance our understanding of the more distal factors increasing vulnerability to eating pathology. Stice’s construct of thin-ideal internalization is quite specific. It refers to the degree to which an individual is aware that the current societal standard for beauty/attractiveness is “thinness” as well as the degree to which that individual has taken on that standard as their own. However, it does not directly assess the degree to which an individual believes that beauty/attractiveness (thinness) is the (or one of the) most important determinants of self-worth. Fairburn’s model emphasizes that aspect of attitudes about weight. That construct, which is labeled “over-evaluation of self worth based on weight and shape,” is considered the first step (i.e. risk-factor) increasing vulnerability to dietary restraint and subsequent eating pathology (binge eating and compensatory behaviors).

Individuals evaluate their self worth utilizing numerous facets of their lives in addition to weight or appearance, but Fairburn’s model specifies that those who believe that self worth is heavily dependent upon shape or weight and their ability to control

eating are most likely to resort to excessive restriction and/or dieting and/or compensatory behaviors in their efforts to achieve their goals, whether those goals are extreme thinness, or just prevention of excessively feared weight gain (Fairburn, 1997). Thus, such individuals are vulnerable whether or not they endorse an excessively thin idea and whether or not they are currently highly dissatisfied with their weight or shape. Stice's work suggests that thin-ideal internalization and body dissatisfaction might occur even earlier in the pathway and contribute to over-evaluation, so we have placed them prior to that construct in our extension of Fairburn's model (see Figure 3). However, it is difficult to separate the effects of those three constructs. It may turn out to be more useful to simply consider all three of them as highly interrelated risk factors, all leading to dietary restraint and subsequent global eating pathology.

Stice's model also differs from Fairburn's model in identifying a second, "negative affect," pathway. Negative affect (whether from body dissatisfaction or from other causes), or perhaps the lack of more adaptive ways to cope with negative affect is hypothesized to lead directly to bulimic symptoms separately from the dietary restraint path. This additional path is noted in our extended model but it is not directly relevant to the current study.

Thus, integrating the cognitive-behavioral model for the development of eating disorder pathology with research on risk factors for the development of eating disorders, (Attie & Brooks-Gunn, 1989; Heatherton, Mahamedi, Striepe, Field, & Keel, 1997; Killen, et al., 1996; Stice & Agras, 1998; Striegel-Moore, Silberstein, Frensch, & Rodin, 1989), we propose that body dissatisfaction (the perceived discrepancy between actual body mass index and the individual's standard) and thin-ideal internalization (endorsing

societal standards of thinness as attractive) will both contribute (independently or together) to over-evaluation of self worth based on weight, shape, and eating. All of these risk factors likely work together to increase vulnerability to dietary restraint and subsequent eating pathology, which are the outcomes that prevention interventions are designed to reduce.

The Current Research Study

Given the paucity of research on mediators of intervention effects in eating disorder prevention programs, the current study serves to areas where further investigation is imperative. In line with suggestions for future research by Stice et al. (2007), our first aim was to investigate thin-ideal internalization as a mediator in an eating disorder prevention program implemented in undergraduate sorority utilizing a broader measure of thin-ideal internalization than Stice and colleagues tested in their 2007 study. We predicted that reduction in thin-ideal internalization from pre- to post-intervention would mediate the effects of CD on outcomes measures (dietary restraint and eating disorder pathology) as compared to a waitlist control (WL), but that thin-ideal internalization would not mediate reduction in these outcome measures in Media Psychoeducation (MP) as compared to WL. Our second aim was to investigate body dissatisfaction as a mediator. We predicted that reduction in body dissatisfaction from pre- to post-intervention would also mediate the effects of CD on the outcome measures. The effects of these two mediators were evaluated at two time points, “immediate change” (pre- to post change) and “sustained change” (pre-intervention to one-month follow-up).

Participants

At Trinity University, we recruited 161 sorority members from the six university sororities, which are all local (i.e., not affiliated with national sororities) and none of the sororities have residential housing for their members. Participants ranged in age from 18 to 22 ($M=19.95$, $SD=.90$), and had a mean body mass index (BMI), which was based on self-report height and weight, of 22.01 ($SD=2.65$). A total of 89% of participants self-identified their ethnicity as Caucasian, while 5% endorsed Hispanic, 4% endorsed Asian, and 1% selected “other” (Becker et al., 2005). Data collected from the participants in this study will be used for the proposed research study.

Procedure

Prior to beginning the research program, we attained approval from the Trinity University Institutional Review Board, Greek Council, and Student Affairs.

Undergraduate researchers recruited participants at each sorority’s weekly meeting. A total of 245 members completed consent forms for randomization. We were unable to randomize members primarily due to either scheduling conflicts or graduation prior to study entry.

Participants were randomized into CD, MP, or WL. There were 63 participants in CD, 59 participants in MP, and 39 participants in WL. It is important to note a specific element in the randomization process: in the first year of the study, members were randomized across all three conditions. After this year, sorority leaders requested that we eliminate the WL and that members who were previously randomized to WL then be re-randomized into either CD or MP, in order to avoid frustrating those members initially randomized to WL but who had desired to participate in the active interventions. Due to

the cooperative nature of this study with the sororities, we complied. This resulted in fewer total members having being randomized into WL.

Participants completed a consent form and a baseline questionnaire packet at the start of all conditions. Both CD and MP consisted of two 2-hour sessions scheduled one week apart. Groups were led by a licensed clinical psychologist and two undergraduate student co-leaders, and ranged in size from six to sixteen participants. Participants completed a consent form and a baseline questionnaire packet at the start of all conditions. In the CD and MP condition, participants completed the assessment packet after session two. In the WL condition, participants completed the packet one week after the first assessment. During the second assessment for all groups, we instructed participants to only respond based on the past week at the second assessment, even though instructions on the measure requested information over a longer period of time, because they had completed the same measures only one week prior. One-month follow-up measures were taken in all conditions.

Intervention: Cognitive Dissonance Group

Session 1. We began the first session by explaining the study overview and history. We then clearly articulated that this study was not targeting sororities because we think they are at greater risk for eating disorders, but because they have an organizational structure ideal for facilitating change as well as a large number of women to accomplish this change. Next, participants defined the thin-ideal, and then discussed how it is maintained and who in society benefits from the maintenance of the thin-ideal standard of beauty. After this collective discussion, members then participated in an individual writing exercise where each woman detailed the costs of pursuing thin-ideal. Each

member then shared her list with the group, and members then collectively analyzed how attainable thin-ideal is, given the costs in pursuing it. The first session ended with a counter-attitudinal exercise as homework to be completed before session two. We instructed participants to stand in front of a mirror in as little clothing as they felt comfortable. While in front of the mirror, participants recorded only positive qualities about herself, including physical, mental, and emotional attributes.

Session 2. The second session began with each member sharing one positive physical quality about herself (i.e. “I really like my calves), and one positive quality that was not considered to be a physical quality (i.e. “I have a good sense of humor”). Members then discussed the pressures on first-year college women to pursue the thin-ideal and developed strategies that the sororities could employ to improve the body image of sorority members. Next, we collectively discussed the time and effort needed to make celebrities appear ideal. We then divided the members into three subgroups to participate in a role-play exercise. In the role-play, the participants’ goal was to convince each of the group leaders to abandon pursuit of the thin-ideal. One leader role-played a student with anorexia nervosa, one a compulsive exerciser, and one an excessive and unhealthy dieter. The subgroups first brainstormed reasons why each leader should stop pursuing the thin-ideal and then rotated to each leader, trying to persuade each leader to cease her pursuit of the thin-ideal. We then reconvened as a whole group and discussed the role-play, and lastly participants each gave a final remark on her experience.

Intervention: Media Psychoeducation Group

Session 1. As in CD, we provided a study overview, and reassured sorority members regarding the rationale for utilizing sororities. We then began by collectively

generating a description of the thin-ideal. We then transitioned into a discussion on the media's influence on the thin-ideal, highlighting the role of advertising. Participants discussed ways in which advertisements perpetuate the thin-ideal. Next, participants watched a 35-minute video aimed at the influence of advertising on body image and the perpetuation of the thin-ideal. Finally, participants shared their reactions to the video and brainstormed strategies to counteract media pressures to pursue the thin-ideal.

Session 2. We began session two by discussing the attainability of the thin-ideal, and broadened the discussion from advertising to all forms of media. Participants discussed the difference between media figures (i.e. celebrities) and themselves. The members collectively assessed if achieving the thin-ideal is realistic and costs associated with the pursuing the thin-ideal. We then discussed the time and effort needed to make celebrities appear ideal, as in CD. Participants then watched a 20-minute edited version of a video on eating disorders. The video contained testimonials from individuals who had recovered from an eating disorder and examples of women who ceased pursuit of the thin-ideal. The video also provided information about the long-term effects of eating disorders. We edited the video to remove all details about the specific behaviors associated with eating disorders in order to minimize the likelihood of normalizing specific behaviors. Lastly, the group reflected on the video and each participant provided a final comment.

Measures

Participants across conditions completed a questionnaire packet at all three time points, and the measures used in the current investigation are described below: the Eating Attitudes Test (Garner, Olmsted, Bohr, & Garfinkel, 1982), the Eating Disorders

Examination-Questionnaire (Fairburn & Beglin, 1994), the Dutch Restrained Eating Scale (van Strein, Frijters, van Staveren, Defares, & Deurenberg, 1986), the Body Shape Questionnaire (Cooper, Taylor, Cooper, Z., & Fairburn, 1987), the Social Attitudes Toward Appearance Scale-3 (Thompson et al., 2004).

Eating Attitudes Test-26. The Eating Attitudes Test-26 (EAT-26; Garner, Olmsted, Bohr, & Garfinkel, 1982) is a questionnaire that determines participants' attitudes toward food and their behaviors with regards to food and eating. This survey uses the Likert scale which allows participants to assess their attitudes and behaviors regarding food. Scores are revised from the raw score on a scale of 0 to 5 to a score of 0 to 3. The scores are revised in order to separate unhealthy eating behaviors from normative behaviors, where these behaviors do not become unhealthy until practiced frequently. The revised scores accounts for very moderate levels of concern that are not considered pathological. For the current study, we used the revised scores.

Eating Disorder Examination Questionnaire. The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a survey taken from the Eating Disorder Examination clinical interview. The EDE-Q is a validated measure of eating pathology, and questions are aimed at behaviors and attitudes experienced within the past 4 weeks, including behaviors such as bingeing and purging (Fairburn & Beglin, 1994). The total scale is broken into 4 subscales: restraint, weight concern, eating concern, and shape concern. The subscales have acceptable internal consistency and two week test-retest reliability, with Chronbach alphas and Pearson coefficients for all scales consistently exceeding 0.80 (Luce & Crowther, 1999).

Dutch Restrained Eating Scale. The Dutch Restrained Eating Scale assesses eating restraint and dieting behavior (DRES; van Strein, Frijters, van Staveren, Defares, & Deurenberg, 1986). The Likert scale is used in order to determine how often the participant engages in restrained eating behaviors. Past research suggests internal consistency ($\alpha = .95$) and indicates that scores on the DRES correlate with actual caloric intake (Stice & Agras, 1998; Stice et al., 2000; van Strien et al., 1986; Wardle & Beales, 1987).

Body Shape Questionnaire. The Body Shape Questionnaire (BSQ; Cooper, Taylor, Cooper, Z., & Fairburn, 1987) uses a Likert scale to assess the degree to which the participant has felt body dissatisfaction or preoccupation in regards to their body shape over the past 28 days. The BSQ was originally developed to differentiate body dissatisfaction in non-patients from concerns of eating disordered patients. Scores on the BSQ were significantly different for eating disordered patients in comparison to non-patients ($t = 11.7$, $df = 571$, $p < .000$). Also, scores on the BSQ correlate significantly with scores on the EAT ($r = .35$) thus indicating concurrent validity (Cooper et al., 1987).

Social Attitudes Toward Appearance Scale-3. The SATAQ-3 (Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004) uses a Likert scale in order for the participant to rank the importance of media influence. The SATAQ-3 consists of statements regarding media and thinness. Subscales include: television/magazine, social comparisons, and athletics. Individually, internalization for television/magazine (.92), internalization-athlete (.89), and internalization-comparison (.96) all have internal consistency. We combined all internalization scales, rather than evaluate the subscales separately (Cusumano & Thompson, 2001).

Constructs

Due to the lack of trained assessors and the utilization of undergraduate researchers, we only used self-report measures. Because we were limited to self-report data, and because each individual measure elicits information from different aspects of a construct, we opted to create composite constructs for our outcome variables using multiple measures in order to increase measurement reliability (Kazdin, 2003).

Dietary Restraint. This construct was created by averaging the EDE-Q restraint subscale (Fairbrun & Beglin, 1994) score and the DRES score (van Strein et al., 1986). We averaged scores on the EDE-Q and DRES to result in a more reliable measure of the participants' eating and dietary behaviors (Fairburn & Beglin, 1994; van Strein et al., 1986). Dietary restraint measures the degree to which participants regulate, monitor, and try to control food intake. This construct evidenced good internal consistency, ($\alpha = .91$).

Eating disorder pathology. This construct was created by averaging EAT-26 revised scores (Garner et al., 1982) and the EDE-Q (all subscales used) scores (Fairburn & Beglin, 1994). The eating pathology construct provides a reliable way to measure the eating disordered or non-disordered behaviors of the participants. Although this construct includes the restraint subscale, used in the Dietary Restraint construct, this does not impact statistical analyses because we do not include both constructs in a single test. This construct evidenced good internal consistency, ($\alpha = .90$).

Statistical Analyses

Aim 1 Analyses: To explore thin-ideal internalization as measured by the SATAQ-3 (Thompson et al., 2004) as a mediator of intervention effects. Specifically, we hypothesized that reduction in thin-ideal internalization as measured by the SATAQ-3

(Thompson et al., 2004) would act as a mediator for intervention effects in the CD intervention as compared to WL, but not for MP as compared to WL. Decrease in scores within thin-ideal internalization from pre- to post-intervention would predict a decrease in our outcome constructs of dietary restraint and eating disorder pathology from pre- to post-intervention (Aim 1a) and in sustained change from pre-intervention to one-month follow-up (Aim 1b).

In order to investigate this primary aim, we followed the procedure for identifying a mediator developed by Baron and Kenny (1986). Per this model, we used a series of regression analyses in order to determine whether thin-ideal internalization is a mediator of intervention effects as evidenced by change in our outcome variables, dietary restraint and eating disorder pathology, from pre- to post-intervention (Aim 1a) and from pre-intervention to one-month follow-up (Aim 1b). In this model, each regression analysis must meet three criteria (Baron & Kenny, 1986).

Criterion 1. The independent variable (intervention group) significantly affects the mediator variable (change in thin-ideal internalization from pre- to post-intervention). To test this criterion, we regressed the mediator variable, which is the difference between pre-intervention and post-intervention scores on the SATAQ-3 (Thompson et al., 2004), on the independent variable, which is intervention group. In the first contrast, our independent variables were CD compared to WL. Our second contrast consisted of MP and WL as our independent variables. This follows the contrast model used by Stice and colleagues (2007) for examining a potential mediator with specificity to intervention type. They compared CD to a control group and an alternate active intervention to a control group, in order to evaluate the specificity of a potential mediator.

Criterion 2. The independent variable (intervention group) must significantly affect the outcome variable. Because we have two outcome variables in Aim 1a (change in the dietary restraint construct and change in the eating disorder pathology construct from pre- to post-intervention), we must conduct two separate simple linear regression analyses for each outcome variable: contrast CD versus WL and contrast MP versus WL.

Criterion 3. The mediator (change in thin-ideal internalization from pre- to post-intervention) must significantly affect the outcome variable. In order to test this third criterion, we conducted multiple regression analyses in which we regressed the outcome variable on the mediator in Block 1 and on the independent variable in Block 2. We ran one multiple regression analysis for each outcome variable (dietary restraint and eating disorder pathology) and each contrast (CD versus WL and MP versus WL) that was significant in the second criterion.

Criterion 4. The effect of the independent variable (intervention group) on the outcome variable must be attenuated or non-existent when controlling for the mediator than it was when the mediator was not entered into the regression analysis as a covariate (in criterion 2). If the effect of the independent variable (intervention group) becomes non-significant when the mediator is controlled for in Block 1 of the regression, then the mediator is considered a complete mediator. If the effect of the independent variable becomes smaller but remains significant, then the mediator is a partial mediator. We assessed this criterion with the multiple regression analyses we ran for criterion 3.

In sum, we ran a series of three regression analyses for each outcome variable (dietary restraint and eating disorder pathology) and for each contrast (CD versus WL and MP versus WL), using the mediator (thin-ideal internalization change from pre- to

post-intervention). When analyzing thin-ideal internalization as a mediator for sustained change (Aim 1b), we used the same contrasts of independent variables (CD versus WL and MP versus WL) as we did for change during intervention (Aim 1a), as described above.

Aim 2 Analyses: To explore body dissatisfaction as measured by the BSQ (Cooper et al., 1987) as a mediator of intervention effects. We hypothesized that reduction in body dissatisfaction as measured by the BSQ (Cooper et al., 1987) would act as a mediator for intervention effects in the CD intervention as compared to WL, but not for MP as compared to WL. Decrease in scores within body dissatisfaction from pre- to post-intervention would predict a decrease in our outcome measures of dietary restraint and eating disorder pathology from pre- to post-intervention (Aim 2a) and in sustained change from pre-intervention to one-month follow-up (Aim 2b).

In order to investigate this second aim, we utilized the same Baron and Kenny (1986) model for identifying a mediator as we used in the primary aim. We ran the same contrasts comparing CD to WL and then MP to WL to examine a potential mediator specific to intervention type. Therefore, we ran a series of three regression analyses for each outcome variable in Aim 2a (change in dietary restraint and eating pathology from pre- to post-intervention) and for each contrast (CD versus WL and MP versus WL), using the mediator (body dissatisfaction change from pre- to post-intervention). We used this same procedure to investigate body dissatisfaction as a potential mediator for sustained change in each outcome variable, i.e. change from pre-intervention to one-month follow-up (Aim 2b).

Results

Aim 1a: To explore thin-ideal internalization as measured by the SATAQ-3 (Thompson et al., 2004) as a mediator of intervention effects during intervention (Table 3).

Criterion 1. To test whether the independent variable significantly affected the mediator variable, we regressed the mediator (reduction in thin-ideal internalization from pre- to post-intervention) on the independent variable (intervention group). In the first contrast, our independent variables were CD compared to WL. We found that intervention group significantly affected the reduction in thin-ideal internalization from pre- to post-intervention ($r(90) = .313, p = .003, R^2 = .098$). Further investigation of this relationship indicated that participants in CD showed significantly greater reductions in thin-ideal internalization from pre- to post-intervention (Table 1), which was found in a previous study on intervention outcomes using this sample (Becker et al., 2005).

In our second contrast, we compared MP and WL as our independent variables with the same mediator. We found that intervention group did not significantly affect reduction in thin-ideal internalization from pre- to post-intervention ($r(91) = .060, p = ns, R^2 = .004$). Because this relationship was not statistically significant, indicating that group membership had no effect on the change in thin-ideal internalization from pre- to post-intervention, it did not satisfy criterion 1 of a mediational relationship and therefore change in thin-ideal internalization from pre- to post-intervention is not a mediator of intervention effects in MP compared to WL. Hence, we did not run further analyses to test criteria 2 through 4 in MP compared to WL.

Criterion 2. To test whether the independent variable (intervention group) significantly affected each outcome variable, we conducted two simple linear regression

analyses. Because we have two outcome variables in Aim 1a (reduction in the dietary restraint construct and reduction in the eating disorder pathology construct from pre- to post-intervention, Table 2), we conducted one simple linear regression analysis for each outcome variable. When we regressed reduction in the dietary restraint construct from pre- to post-intervention, we found a statistical trend for intervention group (CD or WL) affecting the outcome variable ($r(92) = .183, p = .08, R^2 = .034$). Although this relationship was not statistically significant at a $p = .05$ level, we proceeded to test criterion 3. We proceeded because we are aware that we have a small sample size, which very likely impacted our power to detect a true difference between the two intervention groups as they predict reduction in the outcome variable, reduction in the dietary restraint construct from pre- to post-intervention. We also recognize that we maintained strict criteria for determining a meditational relationship throughout the series of analyses. In spite of this pattern, we proceeded to test subsequent criteria in this case because we found a statistical trend very close to a statistically significant level. In other analyses, our non-significant findings did not indicate statistical trends nor were they approaching statistical significance. Therefore, we did not maintain our strict statistical procedure in this criterion because this p value fell so close to statistical significance, while other analyses with non-significant results did not fall close to our statistical cutoff.

To test whether membership in intervention group (CD or WL, independent variable) significantly affected reduction in the eating disorder pathology construct from pre- to post-intervention (outcome variable), we conducted one simple linear regression analysis. When we regressed reduction in the eating disorder pathology construct from pre- to post-intervention, we found that intervention group membership (CD or WL)

significantly affected the reduction in the eating disorder pathology construct from pre- to post-intervention ($r(92) = .337, p = .001, R^2 = .114$). Since this relationship between group membership and reduction in the eating disorder pathology construct from pre- to post-intervention was statistically significant, we proceeded to test criterion 3.

Criterion 3. To test if the mediator (reduction in thin-ideal internalization from pre- to post-intervention) significantly affected the outcome variable, we conducted two multiple regression analyses in which we regressed each outcome variable on the mediator in Block 1 and on the independent variable in Block 2. We ran one multiple regression analysis for each outcome variable, reduction in dietary restraint and in eating disorder pathology from pre- to post-intervention. When we regressed reduction in the dietary restraint construct from pre- to post-intervention on reduction in thin-ideal internalization from pre- to post-intervention in Block 1 and intervention group in Block 2, we found that the impact of the mediator (reduction in thin-ideal internalization) on the outcome variable (reduction in the dietary restraint construct from pre- to post-intervention) was not statistically significant ($r(90) = .180, p = ns, R^2 = .032$).

To test if reduction in thin-ideal internalization from pre- to post-intervention (mediator) significantly affected reduction in the eating disorder pathology construct from pre- to post-intervention (outcome variable), we conducted one multiple regression analysis. When we regressed reduction in the eating disorder pathology construct on reduction in thin-ideal internalization in Block 1 and intervention group in Block 2, we found that the mediator (reduction in thin-ideal internalization) significantly predicted the outcome variable, reduction in the eating disorder pathology construct from pre- to post-

intervention ($r(90) = .264, p = .012, R^2 = .070$). Because this relationship was statistically significant, we proceeded to assess criterion 4.

Criterion 4. In order to satisfy the final criterion for a mediational relationship, the effect of the independent variable (intervention group) on the outcome variable must be attenuated or non-existent when controlling for the mediator than it was when regressing the independent variable on the outcome variable alone, in criterion 2. If the effect of the independent variable (intervention group) becomes non-significant when the mediator is controlled for in Block 1 of the regression, then the mediator is considered a complete mediator. If the effect of the independent variable becomes smaller but remains significant, then the mediator is a partial mediator. We assessed this criterion by comparing the effect of intervention group on the outcome variable in the regression analysis in criterion 2 with the effect of intervention group on the outcome variable in multiple regression analyses we ran for criterion 3.

To test criterion 4, we compared the amount of variance in the outcome variable (reduction in the eating disorder pathology from pre- to post-intervention) accounted for by intervention group when we controlled for the effect of the mediator (regression analysis from criterion 3) to the amount of variance accounted for by intervention group without controlling for the effect of the mediator (regression analysis from criterion 2). In criterion 2, intervention group accounted for 11.4% of the variance in the outcome variable of change in the eating disorder pathology construct from pre- to post-intervention ($r(92) = .337, p = .001, R^2 = .114$). When we controlled for the effect of the mediator, reduction in thin-ideal internalization from pre- to post-intervention, the effect of intervention group was attenuated, accounting for 7.5% of the variance in the outcome

variable ($r(90) = .380, p = .007, R^2 = .075$). Although this relationship attenuated when controlling for the mediator, it is still statistically significant. Thus indicating that reduction in thin-ideal internalization from pre- to post-intervention is a partial mediator of intervention effects for reduction in the eating disorder pathology construct from pre- to post-intervention, during intervention.

Aim 1b: To explore thin-ideal internalization as measured by the SATAQ-3 (Thompson et al., 2004) as a mediator of sustained intervention effects from pre-intervention to one-month follow-up (Table 3).

Criterion 1. Because the mediator is the same for Aim 1b as it was in Aim 1a, the results of the simple linear regression analysis from criterion 1 in Aim 1a apply to satisfy criterion 1 for Aim 1b. In this regression analysis, we compared CD compared to WL and found that intervention group significantly affected the reduction in thin-ideal internalization from pre- to post-intervention ($r(90) = .313, p = .003, R^2 = .098$). In our second contrast, we compared MP and WL and found that intervention group did not significantly affect change in thin-ideal internalization from pre- to post-intervention ($r(91) = .060, p = ns, R^2 = .004$). Because this relationship was not statistically significant, indicating that group membership had no effect on the change in thin-ideal internalization from pre- to post-intervention, it did not satisfy criterion 1 of a mediational relationship and therefore change in thin-ideal internalization from pre- to post-intervention is not a mediator of intervention effects in MP compared to WL. Hence, we did not run further analyses to test criteria 2 through 4 in MP compared to WL in Aim 1b.

Criterion 2. To test whether the independent variable (intervention group) significantly affected each outcome variable, we conducted two simple linear regression

analyses. Because we have two outcome variables in Aim 1b (sustained change in the dietary restraint construct and sustained change in the eating disorder pathology construct, from pre-intervention to one-month follow-up, Table 2), we conducted one simple linear regression analyses for each outcome variable. When we regressed sustained change in the dietary restraint construct, we found that intervention group (CD or WL) significantly affected the outcome variable ($r(91) = .263, p = .012, R^2 = .069$). Because this relationship was statistically significant, we proceeded to test criterion 3.

To test whether membership in intervention group (CD or WL, independent variable) significantly affected sustained reduction in the eating disorder pathology construct from pre-intervention to one-month follow-up (outcome variable), we conducted one simple linear regression analysis. When we regressed sustained reduction in the eating disorder pathology construct, we found that intervention group membership (CD or WL) significantly affected sustained reduction in the eating disorder pathology construct ($r(91) = .416, p < .001, R^2 = .173$). Since this relationship between group membership and sustained change in the eating disorder pathology construct was statistically significant, we proceeded to test criterion 3.

Criterion 3. To test if the mediator (change in thin-ideal internalization from pre- to post-intervention) significantly affected the outcome variable, we conducted two multiple regression analyses in which we regressed each outcome variable on the mediator in Block 1 and on the independent variable in Block 2. We ran one multiple regression analysis for each outcome variable, sustained change in dietary restraint and in eating disorder pathology. When we regressed sustained change in the dietary restraint construct on change in thin-ideal internalization from pre- to post-intervention in Block 1

and intervention group in Block 2, we found that the impact of the mediator (reduction in thin-ideal internalization from pre- to post-intervention) on the outcome variable (sustained change in the dietary restraint construct) was not statistically significant ($r(89) = .155, p = ns, R^2 = .024$).

To test if reduction in thin-ideal internalization from pre- to post-intervention (mediator) significantly affected sustained change in the eating disorder pathology construct (outcome variable), we conducted one multiple regression analysis. When we regressed sustained change in the eating disorder pathology construct on reduction in thin-ideal internalization in Block 1 and intervention group in Block 2, we found that the mediator (reduction in thin-ideal internalization) significantly predicted the outcome variable, sustained reduction in the eating disorder pathology construct ($r(89) = .212, p = .047, R^2 = .045$). Because this relationship was statistically significant, we proceeded to assess criterion 4.

Criterion 4. In order to satisfy the final criterion for a mediational relationship, the effect of the independent variable (intervention group) on the outcome variable must be attenuated or non-existent when controlling for the mediator than it was when regressing the independent variable on the outcome variable alone, in criterion 2. If the effect of the independent variable (intervention group) becomes non-significant when the mediator is controlled for in Block 1 of the regression, then the mediator is considered a complete mediator. If the effect of the independent variable becomes smaller but remains significant, then the mediator is a partial mediator. We assessed this criterion by comparing the affect of intervention group on the outcome variable in the regression

analysis in criterion 2 with the affect of intervention group on the outcome variable in multiple regression analyses we ran for criterion 3.

To test criterion 4, we compared the amount of variance in the outcome variable (sustained change in the eating disorder pathology) accounted for by intervention group when we controlled for the affect of the mediator (regression analysis from criterion 3) to the amount of variance accounted for by intervention group without controlling for the affect of the mediator (regression analysis from criterion 2). In criterion 2, intervention group accounted for 17.3% of the variance in the outcome variable of sustained reduction in the eating disorder pathology construct ($r(91) = .416, p < .001, R^2 = .173$). When we controlled for the effect of the mediator, reduction in thin-ideal internalization from pre- to post-intervention, the effect of intervention group was attenuated, accounting for 12.8% of the variance in the outcome variable ($r(89) = .416, p < .001, R^2 = .128$). Although this relationship attenuated when controlling for the mediator, it is still statistically significant. Thus indicating that reduction in thin-ideal internalization from pre- to post-intervention is a partial mediator of intervention effects for sustained reduction in the eating disorder pathology construct from pre-intervention to one-month follow-up.

Aim 2a: To explore body dissatisfaction as measured by the BSQ (Cooper et al., 1987) as a mediator of intervention effects during intervention (Table 4).

Criterion 1. To test whether the independent variable significantly affected the mediator variable, we regressed the mediator (change in body dissatisfaction from pre- to post-intervention) on the independent variable (intervention group). In the first contrast, our independent variables were CD compared to WL. We found that intervention group

significantly affected the change in body dissatisfaction from pre- to post-intervention ($r(92) = .349, p = .001, R^2 = .122$). Further investigation of this relationship indicated that participants in CD showed significantly greater reductions in body dissatisfaction from pre- to post-intervention (Table 1), which was found in a previous study on intervention outcomes using this sample (Becker et al., 2005).

In our second contrast, we compared MP and WL as our independent variables with the same mediator. We found that intervention group did not significantly affect change in body dissatisfaction from pre- to post-intervention ($r(92) = .162, p = ns, R^2 = .026$). Because this relationship was not statistically significant, indicating that group membership had no affect on the change in body dissatisfaction from pre- to post-intervention, it did not satisfy criterion 1 of a mediational relationship and therefore change in body dissatisfaction from pre- to post-intervention is not a mediator of intervention effects in MP compared to WL. Hence, we did not run further analyses to test criteria 2 through 4 in MP compared to WL.

Criterion 2. To test whether the independent variable (intervention group) significantly affected each outcome variable, we conducted two simple linear regression analyses. Because we have the same two outcome variables in Aim 2a (change in the dietary restraint construct and change in the eating disorder pathology construct from pre- to post-intervention, Table 2) as we did in Aim 1a, the results of the simple regression analyses were the same. When we regressed change in the dietary restraint construct from pre- to post-intervention, we found a statistical trend for intervention group (CD or WL) affecting the outcome variable ($r(92) = .183, p = .08, R^2 = .034$). Although this relationship was not statistically significant at a $p = .05$ level, we proceeded to test

criterion 3. Again we proceeded because we are aware that we have a small sample size, which very likely impacted our power to detect a true difference between the two intervention groups as they predict change in the outcome variable, change in the dietary restraint construct from pre- to post-intervention.

When we regressed change in the eating disorder pathology construct from pre- to post-intervention, we found that intervention group membership (CD or WL) significantly affected the change in the eating disorder pathology construct from pre- to post intervention ($r(92) = .337, p = .001, R^2 = .114$). Since this relationship between group membership and reduction in the eating disorder pathology construct from pre- to post-intervention was statistically significant, we proceeded to test criterion 3.

Criterion 3. To test if the mediator (reduction in body dissatisfaction from pre- to post-intervention) significantly affected the outcome variable, we conducted two multiple regression analyses in which we regressed each outcome variable on the mediator in Block 1 and on the independent variable in Block 2. We ran one multiple regression analysis for each outcome variable, reduction in dietary restraint and in eating disorder pathology from pre- to post-intervention. When we regressed reduction in the dietary restraint construct from pre- to post-intervention on reduction in body dissatisfaction from pre- to post-intervention in Block 1 and intervention group in Block 2, we found that the mediator (reduction in body dissatisfaction) significantly affected reduction in the dietary restraint construct from pre- to post-intervention ($r(92) = .258, p = .013, R^2 = .067$). Because this relationship was significant, we proceeded to assess criterion 4.

To test if reduction in body dissatisfaction from pre- to post-intervention (mediator) significantly affected reduction in the eating disorder pathology construct

from pre- to post-intervention (outcome variable), we conducted one multiple regression analysis. When we regressed reduction in the eating disorder pathology construct on reduction in body dissatisfaction in Block 1 and intervention group in Block 2, we found that the mediator (reduction in body dissatisfaction) significantly predicted the outcome variable, reduction in the eating disorder pathology construct from pre- to post-intervention ($r(92) = .610, p < .001, R^2 = .372$). Because this relationship was statistically significant, we proceeded to assess criterion 4.

Criterion 4. In order to satisfy the final criterion for a mediational relationship, the effect of the independent variable (intervention group) on the outcome variable must be attenuated or non-existent when controlling for the mediator than it was when regressing the independent variable on the outcome variable alone, in criterion 2. If the effect of the independent variable (intervention group) becomes non-significant when the mediator is controlled for in Block 1 of the regression, then the mediator is considered a complete mediator. If the effect of the independent variable becomes smaller but remains significant, then the mediator is a partial mediator. We assessed this criterion by comparing the affect of intervention group on the outcome variable in the regression analysis in criterion 2 with the affect of intervention group on the outcome variable in multiple regression analyses we ran for criterion 3.

To assess criterion 4 using reduction in the dietary restraint construct from pre- to post-intervention, we compared the amount of variance in the outcome variable accounted for by intervention group when we controlled for the affect of the mediator (regression analysis from criterion 3) to the amount of variance accounted for by intervention group without controlling for the affect of the mediator (regression analysis

from criterion 2). In criterion 2, intervention group accounted for 3.4% of the variance in the outcome variable of change in dietary restraint from pre- to post-intervention ($r(92) = .183, p = .08, R^2 = .034$). When we controlled for the effect of the mediator, reduction in body dissatisfaction from pre- to post-intervention, the effect of intervention group was attenuated and accounted for 1.0% of the variance in the outcome variable ($r(92) = .277, p = ns, R^2 = .010$). When controlling for the mediator, the effect of intervention group becomes non-significant.

Although this indicates that reduction in body dissatisfaction from pre-to post-intervention completely mediates the relationship between the independent variable (intervention group) and the outcome variable (reduction in the dietary restraint construct from pre- to post-intervention), the simple linear regression in criterion 2 showed that the relationship between intervention group and the outcome variable was only a statistical trend. Therefore, this mediational relationship should be interpreted with caution. This finding does suggest, however, that with a larger sample size leading to increased power to detect a true relationship, reduction in body dissatisfaction from pre- to post-intervention would completely mediate the relationship between intervention group and reduction in the dietary restraint construct from pre- to post-intervention.

To assess criterion 4 using reduction in the eating disorder pathology construct, we compared the result of the simple linear regression analysis in criterion 2 to that of the multiple regression analysis in criterion 3. In criterion 2, intervention group accounted for 11.4% of the variance in the outcome variable of reduction in the eating disorder pathology construct from pre- to post-intervention ($r(92) = .337, p = .001, R^2 = .114$). When we controlled for the effect of the mediator, reduction in body dissatisfaction from

pre- to post-intervention, the effect of intervention group was attenuated, accounting for 1.8% of the variance in the outcome variable ($r(92) = .624, p = ns, R^2 = .018$). When controlling for the mediator, the effect of intervention group becomes non-significant. Thus indicating that reduction in body dissatisfaction from pre- to post-intervention is a complete mediator of intervention effects for reduction in the eating disorder pathology construct from pre- to post-intervention, during intervention.

Aim 2b: To explore body dissatisfaction as measured by the BSQ (Cooper et al., 1987) as a mediator of sustained intervention effects from pre-intervention to one-month follow-up (Table 4).

Criterion 1. Because the mediator is the same for Aim 2b as it was in Aim 2a, the results of the simple linear regression analysis from criterion 1 in Aim 2a apply to satisfy criterion 1 for Aim 2b. In this regression analysis, we compared CD compared to WL and found that intervention group significantly affected the reduction in body dissatisfaction from pre- to post-intervention ($r(90) = .313, p = .003, R^2 = .098$). In our second contrast, we compared MP and WL and found that intervention group did not significantly affect change in body dissatisfaction from pre- to post-intervention ($r(91) = .060, p = ns, R^2 = .004$). Because this relationship was not statistically significant, indicating that group membership had no affect on the change in body dissatisfaction from pre- to post-intervention, it did not satisfy criterion 1 of a mediational relationship and therefore change in body dissatisfaction from pre- to post-intervention is not a mediator of intervention effects in MP compared to WL. Hence, we did not run further analyses to test criteria 2 through 4 in MP compared to WL in Aim 2b.

Criterion 2. To test whether the independent variable (intervention group) significantly affected each outcome variable, we conducted two simple linear regression analyses. Because we have the same two outcome variables in Aim 2b (sustained change in the dietary restraint construct and change in the eating disorder pathology construct, from pre-intervention to one-month follow-up, Table 2) as we did in Aim 1b, the results of the simple regression analyses were the same. When we regressed sustained reduction in the dietary restraint construct, we found that intervention group (CD or WL) significantly affected the outcome variable ($r(91) = .263, p = .012, R^2 = .069$). Because this relationship was statistically significant, we proceeded to test criterion 3.

When we regressed sustained change in the eating disorder pathology construct, we found that intervention group membership (CD or WL) significantly affected sustained change in the eating disorder pathology construct ($r(91) = .416, p < .001, R^2 = .173$). Since this relationship between group membership and sustained reduction in the eating disorder pathology construct was statistically significant, we proceeded to test criterion 3.

Criterion 3. To test if the mediator (reduction in body dissatisfaction from pre- to post-intervention) significantly affected the outcome variable, we conducted two multiple regression analyses in which we regressed each outcome variable on the mediator in Block 1 and on the independent variable in Block 2. We ran one multiple regression analysis for each outcome variable, sustained change in dietary restraint and in eating disorder pathology. When we regressed sustained change in the dietary restraint construct on change in body dissatisfaction from pre- to post-intervention in Block 1 and intervention group in Block 2, we found a statistical trend for the impact of the mediator

(reduction in body dissatisfaction from pre- to post-intervention) on sustained reduction in the dietary restraint construct ($r(91) = .190, p = .071, R^2 = .036$). Although this relationship was not statistically significant at a $p = .05$ level, we proceeded to assess criterion 4. Again we proceeded because we are aware that we have a small sample size, which very likely impacted our power to detect a true relationship between the mediator and the outcome variable, reduction in the dietary restraint construct from pre- to post-intervention.

To test if reduction in body dissatisfaction from pre- to post-intervention (mediator) significantly affected sustained change in the eating disorder pathology construct (outcome variable), we conducted one multiple regression analysis. When we regressed sustained change in the eating disorder pathology construct on change in body dissatisfaction in Block 1 and intervention group in Block 2, we found that the mediator (reduction in body dissatisfaction) significantly predicted the outcome variable, sustained reduction in the eating disorder pathology construct ($r(91) = .472, p < .001, R^2 = .223$). Because this relationship was statistically significant, we proceeded to assess criterion 4.

Criterion 4. In order to satisfy the final criterion for a mediational relationship, the effect of the independent variable (intervention group) on the outcome variable must be attenuated or non-existent when controlling for the mediator than it was when regressing the independent variable on the outcome variable alone, in criterion 2. If the effect of the independent variable (intervention group) becomes non-significant when the mediator is controlled for in Block 1 of the regression, then the mediator is considered a complete mediator. If the effect of the independent variable becomes smaller but remains significant, then the mediator is a partial mediator. We assessed this criterion by

comparing the affect of intervention group on the outcome variable in the regression analysis in criterion 2 with the affect of intervention group on the outcome variable in multiple regression analyses we ran for criterion 3.

To assess criterion 4 using sustained reduction in the dietary restraint construct from pre-intervention to one-month follow-up, we compared the amount of variance in the outcome variable accounted for by intervention group when we controlled for the effect of the mediator (regression analysis from criterion 3) to the amount of variance accounted for by intervention group without controlling for the effect of the mediator (regression analysis from criterion 2). In criterion 2, intervention group accounted for 6.9% of the variance in the outcome variable of sustained change in dietary restraint ($r(91) = .263, p = .012, R^2 = .069$). When we controlled for the effect of the mediator, reduction in body dissatisfaction from pre- to post-intervention, the effect of intervention group was attenuated and accounted for 4.4% of the variance in the outcome variable ($r(91) = .283, p = .044, R^2 = .044$). When controlling for the mediator, the effect of intervention group is attenuated but remains statistically significant.

Although this indicates that reduction in body dissatisfaction from pre-to post-intervention partially mediates the relationship between the independent variable (intervention group) and the outcome variable (sustained reduction in the dietary restraint construct), the result of the multiple linear regression in criterion 3 showed that the relationship between mediator and the outcome variable was only a statistical trend. Therefore, this mediational relationship should be interpreted with caution. This finding does suggest, however, that with a larger sample size leading to increased power to detect a true relationship, reduction in body dissatisfaction from pre- to post-intervention would

partially mediate the relationship between intervention group and sustained reduction in the dietary restraint construct from pre-intervention to one-month follow-up.

To assess criterion 4 using sustained reduction in the eating disorder pathology construct, we compared the result of the simple linear regression analysis in criterion 2 to that of the multiple regression analysis in criterion 3. In criterion 2, intervention group accounted for 17.3% of the variance in the outcome variable of sustained reduction in the eating disorder pathology construct ($r(91) = .416, p < .001, R^2 = .173$). When we controlled for the effect of the mediator, reduction in body dissatisfaction from pre- to post-intervention, the effect of intervention group was attenuated, accounting for 7.1% of the variance in the outcome variable ($r(91) = .542, p = .004, R^2 = .071$). Although this relationship attenuated when controlling for the mediator, it is still statistically significant. Thus indicating that reduction in body dissatisfaction from pre- to post-intervention is a partial mediator of intervention effects for sustained reduction in the eating disorder pathology construct from pre-intervention to one-month follow-up.

Discussion

Previous models conceptualizing the development of eating pathology have identified several risk factors within a causal chain that ultimately leads to pathological eating attitudes and behaviors. According to Stice's dual-pathway model for the development of bulimia, thin-ideal internalization falls causally prior to body dissatisfaction, which falls causally prior to dietary restraint. Thus, dietary restraint can be considered either a very early sign of disordered eating or a more proximal risk factor for serious bulimic symptomatology/eating disorder pathology (Stice et al., 1996).

Based on the dual-pathway model, Stice and colleagues (2000) designed an eating disorders prevention program to reduce thin-ideal internalization through inducing cognitive dissonance. Stice and colleagues (2000) targeted this particular risk factor because in their model it falls causally prior to other risk factors, such as body dissatisfaction, dietary restraint, and negative affect (Stice et al., 1996). Stice and colleagues (2007) considered body dissatisfaction and dietary restraint to be outcome measures because these are more proximal risk factors for the development of bulimic symptomatology (Stice et al., 1996). They found that reductions in thin-ideal internalization partially mediated intervention effects of CD for all outcomes, i.e. body dissatisfaction, negative affect, bulimic symptoms, and dieting (Stice et al., 2007). Although in that study, thin-ideal internalization was the only mediator tested, Stice suggested that body dissatisfaction could be conceptualized as a mediator of other outcomes rather than as an outcome itself (Stice et al., 2007).

In contrast, the original Fairburn model (1997) which conceptualized the inception of eating disorder pathology from the cognitive-behavioral perspective identified over-evaluation of self worth based on weight, shape, and eating behaviors as the most causally distal risk factor (Fairburn, 1997) leading to eating pathology.

In an effort to integrate conceptual constructs from both of these models, we proposed an enhanced model in which two risk factors from the Stice model were viewed as causally prior to Fairburn's construct, over-evaluation of weight, shape, and eating. Individuals may begin to base their self worth on weight, shape, and/or eating if they buy into societal standards of thinness as attractive. Or, individuals may develop the over-evaluation of self worth based on weight, shape, and/or eating if they perceive their actual

body mass index to be different than their individual standard and are dissatisfied with this discrepancy (i.e. an individual is unhappy with her own body and wants to lose weight to meet the social standard). Moreover, we proposed that body dissatisfaction and thin-ideal internalization likely enhance each other and therefore many individuals develop over-evaluation of self worth based on weight, shape, and eating because they both internalize the thin-ideal and are dissatisfied with their shape (i.e. an individual is thin but still dissatisfied and is preoccupied with maintaining her weight and/or trying to change her shape).

Thus, we conceptualized thin-ideal internalization and body dissatisfaction as distal risk factors and we conceptualized dietary restraint as an early outcome, the first step towards eating disorder pathology more generally. Dietary restraint is thus a separate outcome that warrants being tested separately even though dietary restraint is also a component of “global eating pathology,” which includes a wider range of disordered attitudes and behaviors including binge eating and compensatory behaviors.

Our results provide moderate support for the role of thin-ideal internalization as a mediator of the effects of a CD intervention. Our findings were in the same direction as, but were weaker than those of Stice and colleagues (2007), who reported that thin-ideal internalization showed partial mediation on a number of outcomes. However, Stice had a much larger sample size and thus greater power. Unfortunately, our use of a broader measure did not enhance our ability to detect mediation as Stice had suggested it might.

We did find that thin-ideal internalization was an intervention-specific partial mediator, replicating Stice’s finding. Thin-ideal internalization was significant for the CD program but not the MP program. However, mediation was only significant for global

eating pathology, while we found a trend for mediation of reduction in dietary restraint during intervention. In contrast, Stice and colleagues (2007) found partial mediation at and the end of the intervention for all outcome measures, including dietary restraint.

The effects noted were similar whether the outcome was specifically dietary restraint or the more global measure of eating pathology. However, results were stronger for the global than the specific measure. The women in our sample were a nonclinical sample so the dietary restraint construct may not have been sufficiently sensitive to reliably measure change in the outcome. Eating disorder pathology is a broader measure of eating attitudes and behaviors and therefore may have been more sensitive to change (Stice et al., 2007). Our results indicate that intervention group membership accounted for greater variance in sustained reduction of eating disorder pathology which suggests that intervention group had a more significant impact on the outcome measure than did the mediator, and therefore more factors are contributing to the effectiveness of CD than reduction in thin-ideal internalization alone.

Reduction in thin-ideal internalization may not be the most effective mechanism of reducing or sustaining reduction in dietary restraint in a nonclinical sample of undergraduate women. Although posited to reduce eating disorders risk factors, thin-ideal internalization might not be the primary mechanism of change since it did not seem to be responsible for the sustained change created by participating in the CD program. The CD program showed a significantly greater reduction in thin-ideal internalization from pre- to post-intervention than the MP program, but both programs showed similar change on the outcomes of interest, dietary restraint and global eating pathology (as evidenced in Becker et al., 2005). Thus, while CD reduced dietary restraint and there was a trend that

reduction of thin-ideal internalization was a partial mediator of the immediate effects of the CD intervention, it was not a significant mediator of sustained reduction of dietary restraint. These results suggest that reducing thin-ideal internalization is initially effective in reducing dietary restraint and other eating pathology, but that some other mechanisms are clearly involved and those seem to be more responsible for insuring that those changes are maintained.

We found much stronger support for reduction in body dissatisfaction as a specific mediator of intervention effects within the CD program. Body dissatisfaction was not a mediator within the MP program. This finding extends the findings of Stice and colleagues (2007) which did not evaluate body dissatisfaction as a mediator. Although again it was only a trend when the specific measure of dietary restraint was used, reduction in body dissatisfaction completely mediated reduction in dietary restraint during intervention and partially mediated sustained reduction. Results were clearly significant for the global measure of eating pathology. Reduction in body dissatisfaction completely mediated reduction in eating pathology during intervention, and partially mediated sustained reduction in eating pathology. Reduction in body dissatisfaction demonstrated stronger mediational effects than did reduction in thin-ideal internalization, and it accounted for more variance in each of the two outcome constructs.

In comparison to reduction in thin-ideal internalization, reduction in body dissatisfaction was a stronger mediator, accounting for more variance in the outcome constructs and with either partial or complete mediation for all outcomes. According to the dual-pathway model (Stice et al., 1996), body dissatisfaction is a more proximal risk factor for developing bulimic symptomatology than thin-ideal internalization. Within this

model, our findings suggest that reduction of thin-ideal internalization is at least initially helpful in reducing body dissatisfaction, but reduction in body dissatisfaction appears to be the critical mechanism for reducing eating disorder pathology. It also appears that mechanisms other than cognitive dissonance (and reducing thin-ideal internalization) must be involved in reducing body dissatisfaction, so future work will need to clarify what those might be.

According to our proposed model, body dissatisfaction and thin-ideal internalization are causally prior to dietary restraint as well as the ultimate outcome of eating disorder pathology. Our results provide stronger support for the reduction of body dissatisfaction as a mediator of intervention effects than for reduction of thin-ideal internalization. In the context of our proposed model, body dissatisfaction may be the more critical risk factor for developing dietary restraint and eating disorder pathology. Our results suggest that reduction of thin-ideal internalization alone is not sufficient to reduce dietary restraint or eating disorder pathology. Instead, reduction of body dissatisfaction produces the most sustained reduction in eating disordered behaviors. To the extent that reduction in thin-ideal internalization is associated with reduction in body dissatisfaction during an intervention, reduction in thin-ideal internalization appears to be a useful part of the process. Fortunately the CD program that was studied here did lead to reductions in both thin-ideal internalization and body dissatisfaction, and thus was effective in reducing dietary restraint as well as global eating pathology. Our findings suggest that exploring additional ways to reduce body dissatisfaction would likely enhance the effectiveness of the CD intervention.

Limitations

There were several limitations of the current study. First, the reliance on self-report data limits the quality and generalizability of our findings. Self-report data may have biased the magnitude of the relations we found, and the use of observational and/or interview data may have balanced effects of using self-report data. Second, this study had a small sample size and therefore our ability to detect true change at a statistically significant level was attenuated. Furthermore, we had too few time points of data collection to run chronological mediational analyses (Stice et al., 2007) and therefore have limited ability to interpret whether the mediator or outcome variables changed earlier in the intervention. Additionally, we utilized a mediational design that did not include the ability to detect bidirectional effects between the change in the mediators and the change in the outcome measures. We also did not assess other nonspecific factors that may impact the intervention effects in addition to, or stronger than, the mediators we assessed.

Future Directions

The current findings provide reasonable support that reduction in thin-ideal internalization partially mediates the outcome measures post-intervention. Findings also provide support that body dissatisfaction both partially and completely outcome measures both post-intervention and at one-month follow-up. A potential direction for future research in this area is replicating this program with a larger sample size to increase the power to detect differences in the mediators and outcomes. Additionally, future research could investigate if the two mediators mediate each other and test bidirectional effects between change in the mediators and change in the outcome measures. Another potential

direction for future research is including mid-intervention assessment in order to assess chronological change in the mediator as compared to change in the outcome measures. Because we found intervention-specific mediation, future research could test for statistical moderated mediation in eating disorders prevention programs. Future research could also test for additional mediators of intervention effects, including the over-evaluation of self-worth based on weight, shape, and eating, which we did not assess in the current study. By investigating alternative mediators, future research may explicate mechanisms accounting for change in eating disordered behaviors and therefore refine and improve eating disorders prevention programs.

References

- Alexander, L. A. (1998). The prevalence of eating disorders and eating disordered behaviors in sororities. *College Student Journal, 32*(1), 66-75.
- Allison, K. C, Park, C. L. (2004). A prospective study of disordered eating among sorority and nonsorority women. *International Journal of Eating Disorders, 35*(3), 354-358.
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders: Fourth Edition*. Washington, D.C.: American Psychiatric Association.
- Attie, I., & Brooks-Gunn, J. (1989). Development of eating problems in adolescent girls: A longitudinal study. *Developmental Psychology, 25*, 70–79.
- Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173–1182.
- Basow, S. A., Foran, K. A., Bookwala, J. (2007). Body objectification, social pressure, and disordered eating behavior in college women: The role of sorority membership. *Psychology of Women Quarterly, 31*(4), 394-400.
- Bearman, S. K., Stice, E., & Chase, A. (2003). Effects of body dissatisfaction on depressive and bulimic symptoms: A longitudinal experiment. *Behavior Therapy, 34*, 277–293.
- Becker, A. E., Franko, D. L., Nussbaum, K., & Herzog, D. B. (2004). Secondary prevention of eating disorders: The impace of education, screening, and referral in a college-based screening program. *International Journal of Eating Disorders, 36*(2), 157-162.

- Becker, A. E., Grinspoon, S. K., Klibanski, A., & Herzog, D. B. (1999). Eating disorders. *The New England Journal of Medicine*, *340*(14), 1092-1098.
- Becker, C. B., Jilka, K., & Polvere, L. (2002). *Cognitive dissonance vs. media psychoeducation: A pilot study of eating disorder prevention in sorority members*. Paper presented at the Association for Advancement of Behavior Therapy, Reno, NV.
- Becker, C. B., Smith, L. M., & Ciao, A. C. (2005). Reducing eating disorder risk factors in sorority members: A randomized trial. *Behavior Therapy*, *36*, 245-254.
- Becker, C. B., Smith, L. M., & Ciao, A. C. (2006). Peer facilitated eating disorder prevention: A randomized effectiveness trial of cognitive dissonance and media advocacy. *Journal of Counseling Psychology*.
- Cashel, M. L., Cunningham, D., Landeros, C., Cokley, K. O., & Muhammad, G. (2003). Sociocultural attitudes and symptoms of bulimia: Evaluating the SATAQ with diverse college groups. *Journal of Counseling Psychology*, *50*(3), 287-296.
- Cooley, E., & Toray, T. (2001). Body image and personality predictors of eating disorder symptoms during the college years. *Body Image and Eating*, 28-36.
- Cooper, P. J., Taylor, M. J., Cooper, Z., Fairburn, C. G. (1987). The development and validation of the Body Shape Questionnaire. *International Journal of Eating Disorders*, *6*, 485-494.
- Craighead, W. E., Kazdin, A. E., Mahoney, M. J. (1976). *Behavior modification: Principles, Issues, and Applications*. Oxford, England: Houghton Mifflin.
- Crandall, C. S. (1988). Social contagion of binge eating. *Journal of Personality and Social Psychology*, *55*(4), 588-598.

- Fairburn, C. G. (1997a). Eating disorders. In D. M. Clark, & C. G. Fairburn (Eds.), *Science and practice of cognitive behaviour therapy* (pp. 209–241). Oxford: Oxford University Press.
- Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*, 363-370.
- Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A “transdiagnostic” theory and treatment. *Behaviour Research and Therapy*, *41*, 509–528.
- Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *Lancet*, *361*, 407–416.
- Fairburn, C. G., Marcus, M. D., & Wilson, G. T. (1993). Cognitive-behavioral therapy for binge eating and bulimia nervosa: a comprehensive treatment manual. In C. G. Fairburn, & G. T. Wilson (Eds.), *Binge eating: nature, assessment and treatment* (pp. 361–404). New York: Guilford Press.
- Festinger, L. (1957). *A Theory of Cognitive Dissonance*. Stanford, CA: Stanford University Press.
- Festinger, L., Riecken, H. W., & Schachter, S. (1956). *When prophecy fails*. Minneapolis: University of Minnesota Press.
- Franko, D. L., & Orosan-Weine, P. (1998). The prevention of eating disorders: Empirical, methodological, and conceptual considerations. *Clinical Psychology: Science and Practice*, *5*(4), 459-477.
- Garner, D. M., Olmsted, M. P., Bohr, Y., Garfinkel, P. E. (1982). The Eating Attitudes Test: Psychometric features and clinical correlates. *Psychological Medicine*, *12*, 871-878.

- Glasford, D. E., Pratto, F., & Dovidio, J. F. (2008). Intragroup dissonance: Responses to ingroup violation of personal values. *Journal of Experimental Social Psychology, 44*, 1057–1064.
- Heatherton, T. F., Mahamedi, F., Striepe, M., Field, A. E., & Keel, P. (1997). A 10-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of Abnormal Psychology, 106*, 117–125.
- Hoek, H. W. (1995). The distribution of eating disorders. In K. D. Brownell & C. G. Fairburn (Eds.), *Eating disorders and obesity: A comprehensive handbook* (pp. 207-211). New York: Guilford Press.
- Hoek, H. W., & VanHoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders, 34*, 383-396.
- Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2002). Eating disorders during adolescence and the risk for physical and mental disorders during early adulthood. *Archives of General Psychiatry, 59*, 545–552.
- Kazdin, A. E. (2003). *Research Design in Clinical Psychology* (4th ed.). Boston, MA: Allyn and Bacon.
- Keel, P. K., Heatherton, T. F., Dorer, D. J., Joiner, T. E., & Zalta, A. K. (2006). Point prevalence of bulimia nervosa in 1982, 1992, and 2002. *Psychological Medicine, 36*, 119–127.
- Killen, J. D., Taylor, C. B., Hayward, C., Haydel, K. F., Wilson, D. M., Hammer, L., Kraemer, H., Blair-Greiner, A., & Strachowski, D. (1996). Weight concerns influence the development of eating disorders: A 4-year prospective study. *Journal of Consulting and Clinical Psychology, 64*, 936–940.

- Levine, M. P., & Piran, N. (1999). Reflections, conclusions, and future directions. In N. Piran, M. P. Levine, & C. Steiner-Adair (Eds.), *Preventing Eating Disorders: A Handbook of Interventions and Special Challenges* (pp. 319-330). Philadelphia, PA: Brunner/Mazel.
- Luce, K. H., & Crowther, J. H. (1999). The reliability of the eating disorder examination-self-report questionnaire version (EDE-Q). *International Journal of Eating Disorders, 25*, 349-351.
- Mann, T., Nolen-Hoeksema, S., Huang, K., Burgard, D., Wright, A., & Hanson, K. (1997). Are two interventions worse than none? Joint primary and secondary prevention of eating disorders in college females. *Health Psychology, 16*(3), 215-255.
- Matz, D. C., & Wood, W. (2005). Cognitive dissonance in groups: The consequences of disagreement. *Journal of Personality and Social Psychology, 88*, 22-37.
- Mintz, L. B., & Betz, N. E. (1988). Prevalence and Correlates of Eating Disordered Behaviors Among Undergraduate Women. *Journal of Counseling Psychology, 35*(4), 463-471.
- Norton, M. I., Monin, B., Cooper, J., & Hogg, M. A. (2003). Vicarious dissonance: Attitude change from the inconsistency of others. *Journal of Personality and Social Psychology, 85*, 47-62.
- Pearson, J., Goldklang, D., & Striegel-Moore, R. H. (2002). Prevention of eating disorders: Challenges and opportunities. *International Journal of Eating Disorders, 31*(3), 233-239.
- Price, J. A., & Desmond, S. M. (1990). School counselors' knowledge of eating

- disorders. *Adolescence*, 25, 945–957.
- Rose, M. A. (1985). *Rush: The girl's guide to sorority success*. New York: Villard.
- Stice, E., & Agras, W. S. (1998). Predicting onset and cessation bulimic behaviors during adolescence: A longitudinal grouping analysis. *Behavior Therapy*, 29(2), 257-276.
- Stice, E., Chase, A., Stormer, S., & Appel, A. (2001). A randomized trial of a dissonance-based eating disorder prevention program. *International Journal of Eating Disorders*, 29(3), 247-262.
- 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., & Shaw, H. (2004). Eating disorder prevention programs: A meta-analytic review. *Psychological Bulletin*, 130(2), 206-227.
- Stice, E., Shaw, H., Burton, E., & Wade, E. (2006). Dissonance and healthy weight eating disorders prevention programs: A randomized efficacy trial. *Journal of Consulting and Clinical Psychology*.
- Stice, E., Schupak-Neuberg, E., Shaw, H. E., Stein, R. I. (1994). Relation of media exposure to eating disorder symptomatology: An examination of mediating mechanisms. *Journal of Abnormal Psychology*, 103(4), 836-840.
- Stice, E., Trost, A., & Chase, A. (2003). Healthy weight control and dissonance-based eating disorder prevention programs: Results from a controlled trial. *International Journal of Eating Disorders*, 33, 10–21.
- Stice, E., Ziemba, C., Margolis, J., & Flick, P. (1996). The dual pathway model

differentiates bulimics, subclinical bulimics, and controls: Testing the continuity hypothesis. *Behavior Therapy*, 27, 531-549.

Stice, E., Presnell, K., Gau, J., & Shaw, H. (2007). 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.

Striegel-Moore, R., Silberstein, L. R., Frensch, P., & Rodin, J. (1989). A prospective study of disordered eating among college students. *International Journal of Eating Disorders*, 8, 499-509.

Striegel-Moore, R. H., Silberstein, L. R., & Rodin, J. (1986). Toward an understanding of risk factors for bulimia. *American Psychologist*, 41(3), 246-263.

Striegel-Moore, R. H., Silberstein, L. R., & Rodin, J. (1993). The social self in bulimia nervosa: Public self-consciousness, social anxiety, and perceived fraudulence. *Journal of Abnormal Psychology*, 102, 297-303.

Sullivan, P. F. (1995). Mortality in anorexia nervosa. *American Journal of Psychiatry*, 152, 1073-1074.

Suls, J., Martin, R., & Leventhal, H. (1997). Social comparison, lay referral, and the decision to seek medical care. In B. P. Buunk, & F. X. Gibbons (Eds.), *Health, coping, and wellbeing: Perspectives from social comparison theory* (pp. 195-226). Mahwah, NJ: Erlbaum.

Tajfel, H., & Turner, J. C. (1979). *An integrative theory of intergroup conflict*. In W. G. Austin & S. Worchel (Eds.), *The social psychology of intergroup relations* (pp. 33-47). Monterey, CA: Brooks-Cole.

- Thompson, J. K., van den Berg, P., Roehrig, M., Guarda, A. S., & Heinberg, L. J. (2004). The Sociocultural Attitudes Towards Appearance Scale-3 (SATAQ-3): Development and validation. *International Journal of Eating Disorders, 35*(3), 293-304.
- Treasure, J., & Szukler, G. (1995). Medical complications of chronic anorexia nervosa. In G. Szukler, C. Dare, & J. Treasure (Eds.). *Handbook of eating disorders: Theory, treatment, and research*. New York: Wiley.
- van Strien, T., Frijters, J. E., van Staverson, W. A., Defares, P. B., & Deurenberg, P. (1986). The predictive validity of the Dutch Restrained Eating Scale. *International Journal of Eating Disorders, 5*, 747-755.
- Wardle, J., & Beales, S. (1987). Restraint and food intake: An experimental study of eating patterns in the laboratory and in normal life. *Behaviour Research and Therapy, 25*(3), 179-185.
- Wilson, G. T., Becker, C. B., & Heffernan, K. (2002). Eating Disorders. In E. J. Mash & A. R. Barkley (Eds.), *Child Psychopathology* (2nd Edition ed., pp. 687-715). New York: Guilford Press.

Table 1

Means and Standard Deviations for Total Sample

Change During Intervention	
Mediator	<i>M (SD)</i>
Internalization	
CD	.303 (.519)
MP	.063 (.572)
WL	.004 (.313)
Dissatisfaction	
CD	.357 (.449)
MP	.207 (.516)
WL	.061 (.281)

Note: Internalization = Thin-ideal internalization. Dissatisfaction = Body Dissatisfaction.

Table 2

Means and Standard Deviations for Total Sample

Outcome Construct	Change During Intervention	Sustained Change
	<i>M (SD)</i>	<i>M (SD)</i>
Restraint		
CD	.364 (.644)	.325 (.600)
MP	.213 (.422)	.292 (.491)
WL	.145 (.461)	-.009 (.616)
ED Pathology		
CD	.265 (.346)	.253 (.355)
MP	.124 (.295)	.180 (.326)
WL	.045 (.221)	-.038 (.234)

Note: Restraint = Dietary Restraint. ED Pathology = Eating Disorder Pathology.

Table 3

Tests of Thin-ideal Internalization as Hypothesized Mediator of Intervention Effects

Criteria	Effects
1. Effect of group on mediator:	
Group on Δ TII	$r(90) = .313, p = .003, R^2 = .098$
2. Effect of group on outcome:	
Group on Δ DR during intervention	$r(92) = .183, p = .08, R^2 = .034$
Group on Δ DR sustained	$r(91) = .263, p = .012, R^2 = .069$
Group on Δ EPath during intervention	$r(92) = .337, p = .001, R^2 = .114$
Group on Δ EPath sustained	$r(91) = .416, p < .001, R^2 = .173$
3. Effect of mediator on outcome:	
Δ TII on Δ DR during intervention	$r(90) = .180, p = .089, R^2 = .032$
Δ TII on Δ DR sustained	$r(89) = .155, p = ns, R^2 = .024$
Δ TII on Δ EPath during intervention	$r(90) = .264, p = .012, R^2 = .070$
Δ TII on Δ EPath sustained	$r(89) = .212, p = .047, R^2 = .045$
4. Effect of group with control of mediator:	
Δ TII on Δ DR during intervention	$r(90) = .222, p = ns, R^2 = .017$
Δ TII on Δ DR sustained	Not assessed
Δ TII on Δ EPath during intervention	$r(90) = .380, p = .007, R^2 = .075$
Δ TII on Δ EPath sustained	$r(89) = .416, p < .001, R^2 = .128$

Note: Δ TII = change in thin-ideal internalization; Δ DR = change in dietary restraint; Δ EPath = change in eating disorder pathology

Table 4

Tests of Body Dissatisfaction as Hypothesized Mediator of Intervention Effects

Criteria	Effects
1. Effect of group on mediator:	
Group on Δ BD	$r(92) = .349, p = .001, R^2 = .122$
2. Effect of group on outcome:	
Group on Δ DR during intervention	$r(92) = .183, p = .08, R^2 = .034$
Group on Δ DR sustained	$r(91) = .263, p = .012, R^2 = .069$
Group on Δ EPath during intervention	$r(92) = .337, p = .001, R^2 = .114$
Group on Δ EPath sustained	$r(91) = .416, p < .001, R^2 = .173$
3. Effect of mediator on outcome:	
Δ BD on Δ DR during intervention	$r(92) = .258, p = .013, R^2 = .067$
Δ BD on Δ DR sustained	$r(91) = .190, p = .071, R^2 = .036$
Δ BD on Δ EPath during intervention	$r(92) = .610, p < .001, R^2 = .372$
Δ BD on Δ EPath sustained	$r(91) = .472, p < .001, R^2 = .223$
4. Effect of group with control of mediator:	
Δ TII on Δ DR during intervention	$r(92) = .277, p = \text{ns}, R^2 = .010$
Δ TII on Δ DR sustained	$r(91) = .283, p = .044, R^2 = .044$
Δ TII on Δ EPath during intervention	$r(92) = .624, p = \text{ns}, R^2 = .018$
Δ TII on Δ EPath sustained	$r(91) = .542, p = .004, R^2 = .071$

Note: Δ BD = change in body dissatisfaction; Δ DR = change in dietary restraint; Δ EPath = change in eating disorder pathology

Figure 1

Dual-pathway model for the development of bulimia nervosa from Stice et al., 1996

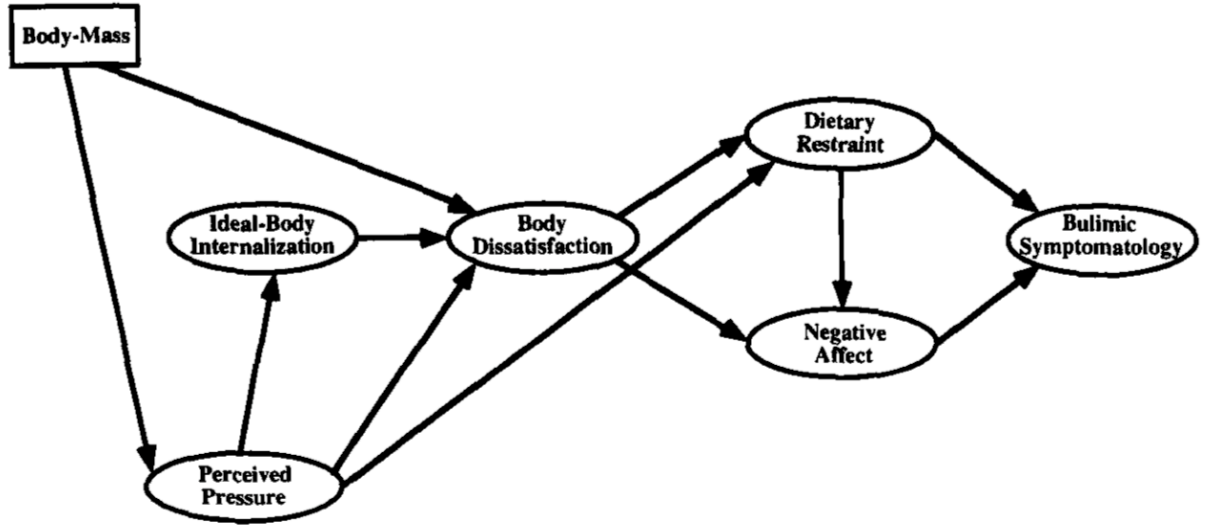
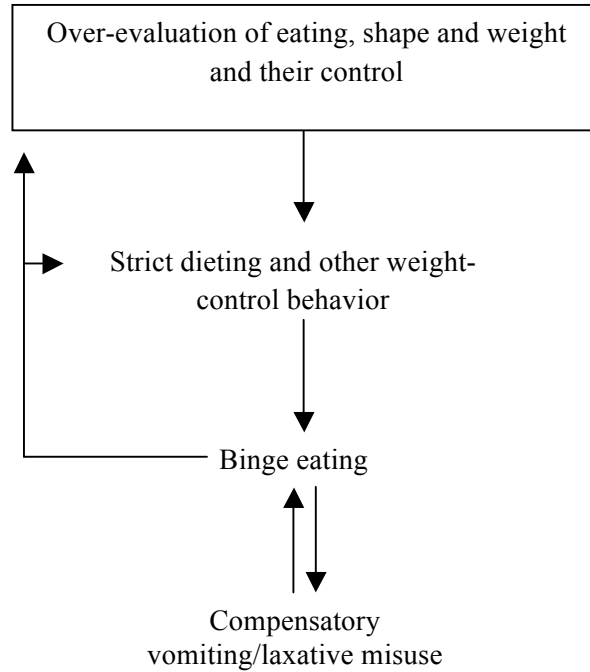


Figure 2

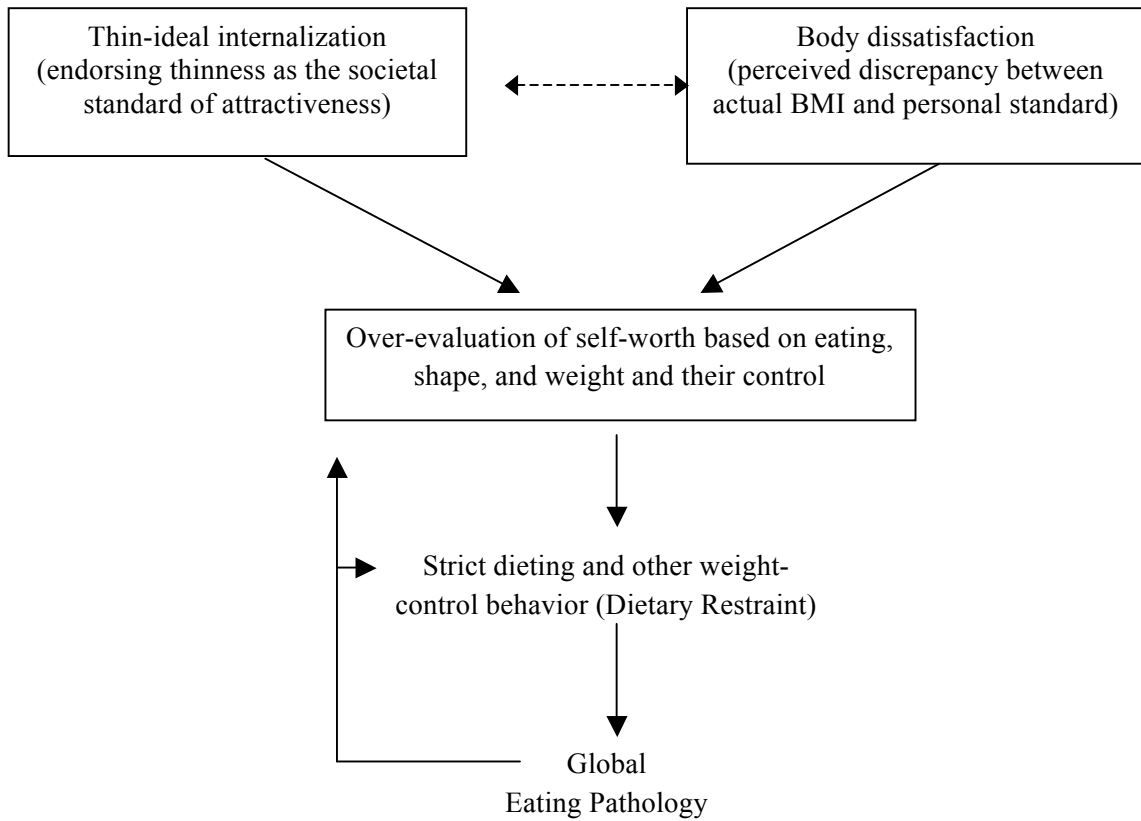
Cognitive-behavioral model for the development of bulimia nervosa



Note: A schematic representation of the cognitive behavioral theory of the development of bulimia nervosa (modified from Fairburn, Marcus, & Wilson, 1993)

Figure 3

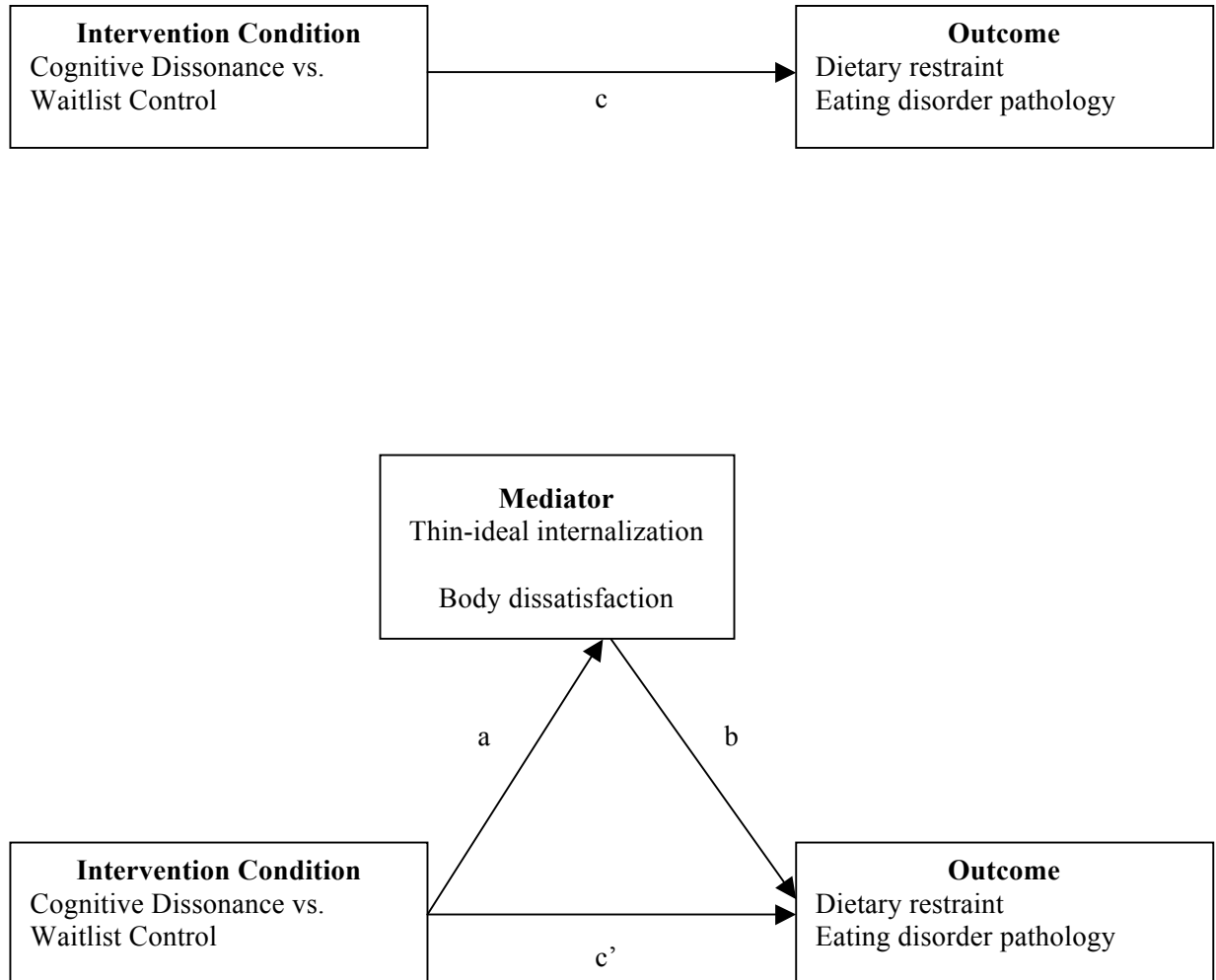
Extended cognitive-behavioral model for the development of eating disorder pathology (adapted from Fairburn et al., 1993)



Note: BMI = Body Mass Index

Figure 4

Graphical model of hypothesized mediators of intervention effects for thin-ideal internalization



Note: Path a = intervention condition predicts change in the mediator; path b = change in the mediator is significantly correlated with change in the outcome; path c = intervention condition predicts change in the outcome; path c' = effect of the intervention condition on change in the outcome when controlling for change in the mediator (in complete mediation, c' is eliminated).