A Prospective Test of the Dual-Pathway Model of Bulimic Pathology: Mediating Effects of Dieting and Negative Affect

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Because there have been few longitudinal investigations of integrative etiological theories of bulimia nervosa, this study prospectively tested the dual-pathway model using random regression growth curve models and data from a 3-wave community sample of adolescent girls (N = 231). Initial pressure to be thin and thin-ideal internalization predicted subsequent growth in body dissatisfaction, initial body dissatisfaction predicted growth in dieting and negative affect, and initial dieting and negative affect predicted growth in bulimic symptoms. There was prospective evidence for most of the hypothesized meditational effects. Results are consistent with the assertion that pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and negative affect are risk factors for bulimic pathology and provide support for the dual-pathway model.

Approximately 4% of adolescent girls will suffer from bulimia nervosa (Rand & Kulda, 1992; Whitaker et al., 1990), making it one of the more prevalent psychiatric problems faced by this population. Bulimia nervosa is characterized by a persistent course, can result in serious medical complications, and is associated with comorbid psychopathology, including affective disorders, anxiety disorders, and substance abuse (Fairburn, Cooper, Doll, Norman, & O’Connor, 2000; Garfinkel et al., 1995; Wilson, Heffernan, & Black, 1996). Additionally, bulimic pathology predicts future onset of obesity (Stice, Cameron, Killen, Hayward, & Taylor, 1999), which results in elevated morbidity and mortality (Dietz, 1998). Eating pathology also predicts onset of depression (Stice, Hayward, Cameron, Killen, & Taylor, 2000), which foretells school dropout, interpersonal problems, unemployment, substance abuse, delinquency, and legal problems (Goldib, Lewinsohn, & Seeley, 1998). An improved understanding of the factors that promote eating pathology would facilitate the design of more effective preventive and treatment interventions for this serious mental health problem.

Although several longitudinal studies identified risk factors that predict bulimic pathology (e.g., Graber, Brooks-Gunn, Paikoff, & Warren, 1994; Killen et al., 1996; Leon, Fulkerson, Perry, & Early-Zald, 1995; Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990), few tested integrative models of the mediational mechanisms by which these risk factors might work together to promote bulimic symptoms. Longitudinal studies that investigated mediational models have not been able to provide evidence of temporal precedence for each of the constituent pathways in the multivariate models. This latter omission probably resulted because there is no accepted test of mediation that generates prospective evidence for each of the criteria proposed by Baron and Kenny (1986), specifically that (a) the independent variable predicts the mediator, (b) the mediator predicts the dependent variable, and (c) a significant relation between the independent and dependent variables becomes weaker when the effect of the mediator is controlled statistically. Accordingly, the aims of this study were to test a multivariate etiological model of bulimic pathology and to introduce a new approach for investigating mediation that can generate prospective evidence for each link in a mediational chain.

This study tested the dual-pathway model of bulimic pathology (Stice & Agras, 1998), which represents a synthesis of the sociocultural (Striegel-Moore, Silberstein, & Rodin, 1986), dietary (Polivy & Herman, 1985), and affect regulation (McCarthy, 1990) accounts of eating disorder. The dual-pathway model posits that internalization of the thin ideal espoused for women contributes to body dissatisfaction because this ideal is virtually unattainable (see Figure 1). It also hypothesizes that elevated pressure to be thin from family, peers, and the media fosters body dissatisfaction, because repeated messages that one is not thin enough likely promote discontent with one’s body. Theoretically, this increased body dissatisfaction, in turn, fosters dieting and negative affect, which consequently increase the risk for bulimic pathology.1

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1 It should be noted that dieting is not synonymous with dietary restraint. Dieting refers to intentional efforts to achieve a desired weight by effecting a negative energy balance between caloric intake and expenditure (Dahlgren, Callahan, & Linton, 1979). In contrast, dietary restraint has been used to refer to a tendency to oscillate between periods of caloric restriction and overeating (Heatherton, Herman, Polivy, King, & McGree, 1988). Because the first definition corresponds to the theoretical construct invoked in the dual-pathway model, the term dieting rather than dietary restraint was used throughout this article.
DUAL-PATHWAY MODEL OF BULIMIC PATHOLOGY

Figure 1. Theoretical components of the dual-pathway model of bulimic pathology.

Dissatisfaction is thought to lead to dieting because of the common belief that this is an effective weight control technique. Body dissatisfaction may also contribute to negative affect because appearance is a central evaluative dimension for women in our culture. Dieting, in turn, is thought to foster negative affect because of the failures that are often associated with weight control efforts and the impact of caloric deprivation on mood. Dieting is also theorized to result in a greater risk for bulimic pathology, because individuals may binge eat to counteract the effects of caloric deprivation. Further, dieting might promote binge eating because breaking strict dietary rules can result in disinhibited eating (the abstinence-violation effect). Negative affect might foster bulimic symptoms because it is commonly believed that eating provides comfort and distraction from negative emotions. Thus, this model posits that individuals may initiate bulimic behavior because of either extreme dieting or chronic negative affect or a combination of these factors. That is, either one of these two pathways may be sufficient to promote onset of bulimic pathology (both are not necessary). It might be noted that the dual-pathway model shares similarities with the spiral model (Heatherton & Polivy, 1992), which focuses on body dissatisfaction, dieting, and negative affect, although the latter also asserts that low self-esteem promotes dieting and that there are reciprocal relations among dieting, negative affect, and bulimic behaviors.

There is mounting evidence that pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and negative affect are risk factors for bulimic pathology. Following Kraemer et al. (1997), the term risk factor is used exclusively to refer to variables that have been found to predict prospectively some subsequent pathological outcome. Because cross-sectional studies do not permit a clear interpretation regarding the direction of effects, this review focuses on longitudinal and experimental studies. Although no known longitudinal studies have provided evidence that thin-ideal internalization predicts body dissatisfaction, negative affect, or dieting, this factor was found to predict onset of bulimic pathology (Stice & Agras, 1998). In addition, pressure to be thin predicted growth in body dissatisfaction and dieting (Cattarini & Thompson, 1994; Stice, Mazotti, Krebs, & Martin, 1998) as well as bulimic symptom onset (Stice & Agras, 1998). There is also evidence that body dissatisfaction predicted growth in dieting (Stice, Mazotti, et al., 1998) and depressive symptoms (Allgood-Merten, Lewinsohn, & Hops, 1990; Cole, Martin, Peeke, Seroczyński, & Hoffman, 1998; Rieder, Koff, & Stubbs, 1989) and onset of major depression (Stice, Hayward, et al., 2000). Moreover, body dissatisfaction predicted bulimic symptom onset (Graber et al., 1994; Killen et al., 1996; Stice & Agras, 1998). Also consistent with the dual-pathway model, dieting predicted onset of bulimic symptoms (Killen et al., 1994; Patton et al., 1990; Stice & Agras, 1998; Stice, Killen, Hayward, & Taylor, 1998a), and randomized experiments indicate that caloric deprivation resulted in disinhibited eating in the laboratory (Telch & Agras, 1996; Wardle & Beales, 1988). Finally, negative affect predicted bulimic pathology onset (Stice & Agras, 1998; Stice et al., 1998a), and experimentally induced negative affect triggered disinhibited eating among restrained eaters (Cools, Schotte, & McNally, 1992; Ruderman, 1985).

Although these studies collectively suggest that pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and negative affect are risk factors for bulimic pathology, little research has explored the mediational processes by which these risk factors may work together to promote this outcome. Thus, the first aim of this study was to test the dual-pathway model, which represents an integrative etiological theory of bulimic pathology. This study focused on bulimic symptoms because investigators have called for an examination of the full spectrum of eating disturbances rather than just diagnostic levels of pathology (Shisslak, Crago, & Estes, 1995). Indeed, half of individuals presenting at eating disorder treatment clinics are diagnosed with partial-rather than full-syndrome eating disorders (Herzog, Hopkins, & Burns, 1993; Williamson, Gleaves, & Savin, 1992). Data also suggest that approximately 12% of adolescent girls suffer from subthreshold bulimia nervosa, and that it is associated with emotional distress (Stice, Killen, Hayward, & Taylor, 1998b). Because studies indicate that the average age of onset for bulimic pathology is 16 to 18 years (Mitchell, Hatsukami, Pyle, & Eckert, 1986; Stice et al., 1998a), this study followed adolescent females from approximately age 14 to 18 years to gain a better understanding of the development of these behaviors.

As noted, the second aim was to introduce a new approach for investigating mediation that provides a prospective test for each of

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2 Some prospective studies reported the correlations between predictors and future bulimic symptoms, without controlling for baseline variation in bulimic symptoms. Because these “cross-time” correlations may simply reflect baseline associations between the predictors and the criterion (Monroe, 1983), the findings from such studies cannot be unambiguously interpreted and are, therefore, not reviewed here.
the three criteria proposed by Baron and Kenny (1986). Obviously, generating prospective support for each of Baron and Kenny’s three criteria would make the strongest case for mediation. Unfortunately, no accepted test of mediation exists that accomplishes this objective. It seems that random regression growth curve models that incorporate time-varying covariates might be particularly well suited for this task. This analytic framework can potentially demonstrate that (a) the independent variable prospectively predicts growth in the mediator, (b) the mediator prospectively predicts growth in the dependent variable, and (c) the prospective relation between the independent variable and growth in the dependent variable becomes weaker when the effect of growth in the mediator is statistically partialed (as reflected by a time-varying covariate). This last demonstration is key because it is change in the mediator, rather than the initial level of the mediator, that theoretically explains the relation between the initial level of the independent variable and change in the dependent variable. Accordingly, the mediational relations specified in the dual-pathway model were evaluated using this new technique for providing prospective support for each of Baron and Kenny’s three criteria for mediation.

Method

Participants

Participants were 231 female students from two northern California private high schools. At baseline, participants were in 9th or 10th grade and ranged in age from 13 to 17 years (M = 14.9 years). Because bulimic pathology is rare among boys (Garfinkel et al., 1995), the present study included only girls. Twenty percent of the sample were Asian, 4% Black, 65% Caucasian, 2% Hispanic, and 1% Native Americans; 8% specified “other.” Average parental education ranged from grade school graduate (1%) to advanced degree (38%), which was also the mode.

Procedures

A passive-consent procedure was used wherein a letter describing the study was mailed to parents, and they were asked to return a signed letter if they did not want their daughter to participate in the study. This resulted in a 94% participation rate. The study was described as an investigation of the development of adolescent physical and mental health. Participants completed a seven-page survey in groups of approximately 30 to 50 students at baseline (T1), 10 months after baseline (T2), and 20 months after baseline (T3). A numeric code was used to identify students to ensure confidentiality. To compensate participants for their time, raffles were held for $15 gift certificates to a local book and music store or magazine subscriptions.3 This project was approved by the Stanford University Committee for the Protection of Human Subjects.

Measures

Perceived pressure to be thin. Participants reported the amount of pressure to be thin they perceived from family, friends, dating partners, and the media on the eight-item Perceived Sociocultural Pressure Scale (Stice & Agras, 1998; sample item: “I’ve perceived a strong message from my family to have a slender figure”). Items used a 7-point response format ranging from none to a lot, and items were averaged for analyses. This scale possessed adequate internal consistency (α = .88), 2-week test–retest reliability (r = .93), and predictive validity (Stice & Agras, 1998). This scale had a Cronbach’s α of .81 at T1 and a 10-month test–retest coefficient of .68 in this sample.

Thin-ideal internalization. Internalization of the thin ideal was assessed with the Ideal-Body Stereotype Scale-Revised (Stice & Agras, 1998). This scale asks participants to indicate their level of agreement with 10 statements concerning what attractive women look like (e.g., “slender women are more attractive”) on 5-point scales ranging from strongly disagree to strongly agree. Items were averaged for analyses. This scale had acceptable internal consistency (α = .89) as well as convergent, discriminant, and predictive validity (Stice & Agras, 1998). This scale had a Cronbach’s α of .83 at T1 and a 10-month test–retest coefficient of .63.

Body dissatisfaction. An adapted form of the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid, Walster, & Bohnstedt, 1973) was used to assess body dissatisfaction. Specifically, only items reflecting satisfaction with body dimensions were used in this study (e.g., items concerning hair and eyes were omitted). This scale asks participants to indicate their level of satisfaction with nine body parts (waist, thighs, buttocks, hips, legs, weight, figure, appearance of stomach, and body build) on 6-point scales ranging from extremely satisfied to extremely dissatisfied. Items were averaged for analyses. Research has found that this scale has acceptable internal consistency (α = .94), temporal reliability (3-week test–retest coefficient = .90), and predictive validity (Stice & Agras, 1998). This scale had a Cronbach’s α of .91 at T1 and a 10-month test–retest coefficient of .74.

Dieting. The Dutch Restrained Eating Scale (DRES; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) was used to assess dieting. Participants indicate the frequency of dieting behaviors using 5-point scales ranging from never to always. Items were averaged for analyses. In terms of reliability, this scale has been found to have acceptable internal consistency (α = .95; Stice & Agras, 1998; van Strien et al., 1986), and an unpublished pilot study (N = 50) revealed a 2-week test–retest coefficient of .82. In support of criterion validity, the DRES (along with the Three-Factor Eating Questionnaire Cognitive Restraint Scale; TFEQ-R; Stunkard & Messick, 1985) predicted reduced caloric intake in the natural environment (Laesls, Tuschl, Kotthaus, & Pirke, 1989; van Strien et al., 1986; Wardle, 1987; Wardle & Beales, 1987).4

3 Another article that used data from this project tested whether there were any effects of receiving the magazine subscription (which was to a fashion magazine) that was randomly distributed to participants by means of a raffle (Stice, Spangler, & Agras, in press). However, there were no main effects of this manipulation on change in body dissatisfaction, dieting, negative affect, or bulimic symptoms, and the results of the current analyses were identical when models controlled for magazine subscription condition.

4 It should be noted that the DRES and the TFEQ-R appear to measure something different than the Restraint Scale (Herman & Polivy, 1980). First, although the DRES and TFEQ-R show the expected correlations with reduced caloric intake in the natural environment (Laesls et al., 1989; van Strien et al., 1986; Wardle, 1987; Wardle & Beales, 1988), the Restraint Scale does not (Kirkley, Burge, & Ammerman, 1988; Laesls et al., 1989). Second, the Restraint Scale is associated with overeating in the laboratory (Herman & Mack, 1975; Polivy, Herman, & McFarlane, 1994), whereas the DRES and TFEQ-R are not (Jansen, Oosterlaan, Merckelbach, & van den Hout, 1988; Lowe & Maycock, 1988; Wardle & Beales, 1987). These findings collectively suggest that the Restraint Scale reflects an inability to restrain one’s eating. Thus, although the DRES and TFEQ-R possess criterion validity, the Restraint Scale does not because it is associated with overconsumption rather than underconsumption. The most obvious explanation as to why the Restraint Scale predicts overeating in the laboratory and other dieting measures do not is because the former contains items directly assessing overeating. This criterion confound (content overlap) renders this scale an inappropriate predictor of overeating or binge eating unless these items are omitted. Because the DRES appears to be the most reliable and valid measure of dieting, it was selected for use in this study.
second unpublished pilot study (N = 59) demonstrated that the DRES predicted unobtrusively observed caloric intake (r = - .28) in the natural environment. In support of the predictive validity of this scale, as would be expected from the dietary restraint model, the DRES predicted future onset of binge eating (Stice & Agras, 1998). The DRES had a Cronbach's α of .93 at T1 and a 10-month test–retest coefficient of .74.

**Negative affect.** Buss and Plomin's (1984) Emotionality Scale was used to assess negative affect. Participants indicate their level of agreement with statements regarding their tendency to become affectively distressed (sample item: “I frequently get distressed”) on 5-point response scale ranging from 1 (never true of me) to 5 (always true of me). Items were summed for analyses. This scale has acceptable internal consistency (α = .82), convergent validity (with other measures of negative affect), and predictive validity (Buss & Plomin, 1984; Stice et al., 1998a, 1998b). This scale had a Cronbach’s α of .79 at T1 and a 10-month test–retest coefficient of .69.

**Body mass.** The body mass index (BMI = kg/m²) was used to reflect adiposity. The BMI divides weight by height (squared) to control for variations in weight because of height and is thus a measure of “relative weight.” Self-reported weight has been found to correlate well with confederate measured weight; the correlations typically range from .96 to .99 (Attie & Brooks-Gunn, 1989; U.S. Public Health Service, 1988). Research has documented that the BMI is a reliable and valid index of adiposity (Pietrobelli et al., 1998; Stice et al., 1999).

**Bulimic symptoms.** Items assessing the diagnostic criteria of bulimia nervosa from the Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994) were used to reflect bulimic symptoms. These items measure overvaluation of weight and shape, frequency of binge eating, and frequency of compensatory behaviors over the past 28 days. These items were standardized (to accommodate the different response formats) and averaged to create an overall bulimic symptom index. The additional subscales assessing weight, shape, and eating concerns were not administered because they are not used to make eating disorder diagnoses. The EDE-Q was derived directly from the Eating Disorder Examination interview (EDE; Fairburn & Cooper, 1993), a validated measure of eating pathology. The internal consistency (α = .84), test–retest reliability (mean r = .80), and convergent validity (with the interview version of the EDE; mean r = .81) have been documented (Black & Wilson, 1996; Fairburn & Beglin, 1994; Luce & Crowther, 1999). A third pilot study with an adolescent sample (N = 26) revealed a 3-week test–retest coefficient of .89. The EDE-Q had a Cronbach's α of .84 at T1 and a 10-month test–retest coefficient of .72.

**Results**

**Preliminary Analyses**

Preliminary analyses tested for attrition biases that might compromise the generalizability of the findings. Nine of the initial participants did not provide data at T2 (4%) and 8 did not provide data at T3 (3%), although only 4 participants did not provide data at both T2 and T3 (2%). Participants who failed to provide data at one or more assessments did not differ significantly from those who provided complete data on any of the variables considered in this study at T1. Because the random regression growth curve models used here can accommodate cases with only two of three waves of data, the effective attrition rate was 2%.

**Test of Dual-Pathway Model Using Random Regression Growth Curve Analyses**

Random regression growth curve analyses (Rogosa, Brandt, & Zimowski, 1982; Rogosa & Willett, 1985) were used to test the dual-pathway model. This was accomplished by first generating individual slope and intercept parameters for each participant for pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms. The slopes represented the average linear growth (change) in the construct across each of the two 10-month intervals for each participant. The intercept parameters are conceptually equivalent to y-intercepts from ordinary least squares analyses and were coded to represent the value of the linear growth trajectory for each participant at T1.

Consistent with other community studies (e.g., Stice & Agras, 1998), there were fairly high rates of bulimic symptoms in this adolescent sample. Specifically, at T1 15% of participants reported at least one binge eating episode and 4% reported engaging in at least one compensatory behavior (e.g., vomiting, laxative use, or diuretic use) in the past 28 days. Moreover, there were marked increases in bulimic symptoms for many participants. Among the 20 students who showed the greatest growth in bulimic symptoms over the study, the percentage who reported any binge eating in the past 28 days increased from 17% to 44%, and the percentage who reported any compensatory behaviors increased from 0% to 22%. As such, the increases in bulimic symptoms observed in this sample over time appear to represent clinically meaningful growth in eating-disordered behaviors.

The dual-pathway model proposes that pressure to be thin and thin-ideal internalization will predict growth in body dissatisfaction over the study period, and that body dissatisfaction will predict growth in dieting and negative affect. This model hypothesizes that dieting, in turn, will predict growth in negative affect and bulimic symptoms, and that negative affect will predict growth in bulimic symptoms (see Figure 1). Thus, eight three-variable mediational chains are contained in the dual-pathway model: (a) body dissatisfaction should mediate the relation between pressure to be thin and dieting, (b) body dissatisfaction should mediate the relation between pressure to be thin and negative affect, (c) body dissatisfaction should mediate the relation between thin-ideal internalization and dieting, (d) body dissatisfaction should mediate the relation between thin-ideal internalization and negative affect, (e) dieting should mediate the relation between body dissatisfaction and bulimic symptoms, (f) negative affect should mediate the relation between body dissatisfaction and bulimic symptoms, (g) dieting should mediate the relation between body dissatisfaction and negative affect, and (h) negative affect should mediate the relation between dieting and bulimic symptoms. These three-variable mediational chains are also listed in Table 1. A series of four regression models were used to individually test each of these three-variable mediational chains. The results of these analyses are summarized in Table 1 and are described below in detail.

**Does the independent variable predict growth in the mediator?**

To test Baron and Kenny's (1986) first criterion for mediation—that the independent variable predicts the mediator—random regression models were conducted wherein the slope parameter for the mediator was regressed on the intercept version of the independent variable and the intercept of the mediator. The intercept of the outcome variable was included in all models to control for the effects of initial level of the variable on subsequent growth over time. As is the case in all longitudinal analyses, it is necessary to control for baseline levels of the outcome to ensure that models are predicting change over time rather than simply reflecting initial differences in the level of the outcome. This is equivalent to
controlling for baseline versions of the outcome variable in prospective regression models.

As predicted, initial pressure to be thin and thin-ideal internalization showed significant positive relations to subsequent growth in body dissatisfaction over the 2-year study period (Column 1 in Table 1). Also as hypothesized, initial body dissatisfaction showed significant positive relations to subsequent growth in dieting and negative affect. However, initial dieting only showed a marginally significant relation to subsequent growth in negative affect. Thus, there was prospective evidence for Baron and Kenny’s (1986) first criterion for mediation for seven of the three-variable mediational chains hypothesized in the dual-pathway model, but the effect for the eighth mediational chain only reached marginal significance. It should be noted that there is overlap among the linkages in the eight three-variable mediational chains contained in the dual-pathway model (e.g., the relation between body dissatisfaction and dieting is contained in both the body dissatisfaction-dieting-bulimic symptom and the body dissatisfaction-dieting-negative affect mediational pathways). Therefore, fewer than eight independent tests are necessary to test each of Baron and Kenny’s criteria for the eight three-variable mediational chains proposed in this model.

For heuristic purposes, the parameter estimates representing the main effects between each of the variables in the dual-pathway model are presented following path diagram conventions (see Figure 2). However, it should be noted that this model was not tested using standard path analysis or simultaneous estimation structural equation modeling but rather with a series of overlapping random regression growth curve models so that temporal precedence could be demonstrated for each constituent linkage in the multivariate model.

**Does the mediator predict growth in the dependent variable?** To assess Baron and Kenny’s (1986) second criterion for mediation—that the mediator predicts the dependent variable—random regression models were conducted wherein the slope parameter for the dependent variable was regressed on the intercept versions of the mediator and the dependent variable.

Consistent with hypotheses, initial body dissatisfaction predicted subsequent growth in dieting and negative affect over the study period (Column 2 in Table 1). Also as expected, initial dieting and negative affect showed a significant prospective relation to subsequent growth in bulimic symptoms. As indicated, however, initial dieting only showed a marginally significant relation to subsequent growth in negative affect. Thus, findings
provided prospective support for Baron and Kenny’s second criterion for mediation for seven of the mediational chains contained in the dual-pathway model, but support for one mediational chain reached only marginal significance.

**Is a prospective relation between the independent and dependent variable reduced when change in the mediator is controlled?**

To assess Baron and Kenny’s (1986) third criterion for mediation—that a significant relation between the independent and dependent variables becomes weaker when growth in the mediator is statistically controlled—a series of paired random regression models were estimated for each three-variable mediational chain. In the first model, the slope parameter for the dependent variable was regressed on the intercept versions of the independent and dependent variables. The second model was identical, except that it also included the intercept and slope parameter for the mediator (as a time-varying covariate) to control for the effects of change in the mediator over time. As suggested by Baron and Kenny (1986), the significance of the mediated effect was also directly assessed using the formula derived by Sobel (1982). Moreover, following MacKinnon (1994), the percentage of the direct effect accounted for by the mediated effect is reported.

Consistent with hypotheses, initial pressure to be thin predicted subsequent growth in dieting, and this relation became weaker when the effects of growth in body dissatisfaction were controlled (Columns 3 and 4 in Table 1, respectively). In this case, the mediated effect was statistically significant (\( \beta = .07, p < .05 \)) and accounted for 25% of the total direct effect. Thus, there was support for the hypothesis that change in body dissatisfaction would mediate the prospective relation between pressure to be thin and growth in dieting.

Results indicated that the relation between initial pressure to be thin and subsequent growth in negative affect was only marginally significant; however, this effect did become nonsignificant when the effects of change in body dissatisfaction were controlled. This mediated effect was only marginally significant (\( \beta = .04, p < .10 \)) but accounted for 50% of the total direct effect. Collectively, there was only marginal support for the assertion that change in body dissatisfaction would mediate the prospective relation between pressure to be thin and growth in negative affect.

Also as expected, initial thin-ideal internalization predicted subsequent growth in dieting, and this relation became weaker when the effects of growth in body dissatisfaction were partialed. The mediated effect was statistically significant (\( \beta = .10, p < .001 \)) and accounted for 23% of the total direct effect. Therefore, there was support for the position that change in body dissatisfaction would mediate the prospective relation between thin-ideal internalization and growth in dieting.

Consistent with predictions, initial thin-ideal internalization predicted growth in negative affect, and this relation became nonsignificant when the effects of change in body dissatisfaction were statistically controlled. This mediated effect was statistically significant (\( \beta = .06, p < .01 \)) and accounted for 35% of the total direct effect. Collectively, results provided support for the hypothesis that change in body dissatisfaction would mediate the prospective relation between thin-ideal internalization and growth in negative affect.

There was also evidence that initial body dissatisfaction predicted growth in negative affect, and that this relation became weaker when the effects of growth in dieting were statistically partialed. However, this mediated effect was only marginally significant (\( \beta = .05, p < .10 \)) and accounted for only 14% of the total direct effect. Because both the relation between initial dieting and growth in negative affect and the mediated effect were non-significant trends, findings collectively provide only marginal support for the hypothesis that change in dieting mediated the prospective relation between body dissatisfaction and growth in negative affect.

Whereas initial dieting was significantly related to growth in bulimic symptoms, this effect did not become weaker when the effects of change in negative affect were partialed. This mediated effect was not statistically significant (\( \beta = .03, p < .05 \)) and did not account for any of the direct effect. Because the relation between dieting and growth in bulimic symptoms did not become weaker when growth in the putative mediator was controlled and the mediated effect was nonsignificant, results did not support the hypothesis that change in negative affect mediated the prospective relation between dieting and growth in bulimic symptoms.

Central to the dual-pathway model, the significant relation between initial body dissatisfaction and subsequent growth in bulimic symptoms became nonsignificant when the effects of change in dieting were statistically partialed. This mediated effect was statistically significant (\( \beta = .19, p < .001 \)) and accounted for 68% of the total direct effect. Also central to this model, the significant relation between initial body dissatisfaction and subsequent growth in bulimic symptoms became nonsignificant when the effects of change in negative affect were controlled. This mediated effect was statistically significant (\( \beta = .05, p < .01 \)) and accounted for 96% of the total direct effect. Although not reported in Table 1, results also indicated that when the effects of change in both dieting and negative affect were partialed, initial body dissatisfaction was nonsignificantly related to growth in bulimic symptoms, \( \beta(1, 218) = .01, p = .30 \). Findings, therefore, provided support for the position that changes in dieting and negative affect mediate the prospective relation between body dissatisfaction and growth in bulimic symptoms.

In sum, results provided prospective support for Baron and Kenny’s (1986) third criterion for mediation for seven of the eight three-variable mediational chains contained in the dual-pathway model, although support for two of these chains involved marginal trends. Notably, the full model accounted for 23% of the variance in growth in bulimic symptoms after controlling for the effects of baseline levels of bulimic symptoms, suggesting that this model explained a substantively meaningful proportion of the variance in the outcome.5

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5 Although the EDE-Q assesses all of the Diagnostic and Statistical Manual of Mental Disorders (fourth edition; DSM-IV; American Psychiatric Association, 1994) symptoms of bulimia nervosa, including overvaluation of weight and shape, binge eating, and use of compensatory behaviors, it is possible that this etiological model is primarily predicting growth in overvaluation of weight and shape, which would be of less clinical significance. To test this possibility, the model was re-estimated, excluding items assessing overvaluation of weight and shape. Results indicated that the relations between the risk factors and growth in bulimic symptoms became slightly stronger when the outcome solely reflected growth in the frequency of binge eating and compensatory behaviors. These results suggest that this model is not simply predicting change in overvaluation of weight and shape but rather is predicting growth in all of the diagnostic symptoms of bulimia nervosa.
Test of a Competing Model

An important step in evaluating a theoretical model is to test alternative accounts that may better explain the relations among the variables. A closely related model involving the same key constructs proposes that negative affect may foster both body dissatisfaction and dieting, which, in turn, promote bulimic pathology (Thompson, Heinberg, Altbe, & Tantleff-Dunn, 1999). Accordingly, this model was estimated using the same techniques as those outlined previously. However, this alternative model accounted for significantly less variance in growth in bulimic symptoms, controlling for baseline levels of bulimic symptoms (19% compared with 23% for the original model), $F(4, 212) = 3.01, p < .05$. Moreover, the individual pathways were also smaller in this alternative model, explaining an average of 5% of the variance (compared with 8% in the original model). Thus, this alternative model did not do as well as the dual-pathway model in accounting for the relations among these variables and in predicting growth in bulimic symptoms.

Specificity of Dual-Pathway Model

Another key step in evaluating an etiological model is determining whether it is specific to the outcome of interest rather than predicting conceptually distinct but related outcomes. It seems particularly important to ensure that the dual-pathway model is not simply explaining the development of overeating and consequent obesity. Thus, the dual-pathway model was estimated with growth in body mass over the study as the ultimate outcome. Although initial dieting predicted subsequent growth in body mass, $\beta(1, 218) = .27, p < .001$, none of the other factors significantly predicted this outcome, and this model only accounted for 8% of the variance in growth in body mass (compared with 23% of the variance in growth in bulimic symptoms). Thus, the dual-pathway model was relatively specific to bulimic symptoms rather than predicting the development of obesity. As an aside, several other studies found that dieting predicted increased body mass over time among adolescents (Stice, 1998; Stice et al., 1999) and adults (French, Jeffery, & Wing, 1994; Klesges, Isbell, & Klesges, 1992; Klesges, Klem, & Bene, 1989).

Discussion

Test of the Dual-Pathway Model

The first aim was to provide a test of the dual-pathway model of bulimic pathology. Consistent with hypotheses, there was evidence that initial pressure to be thin and thin-ideal internalization predicted subsequent growth in body dissatisfaction, initial body dissatisfaction predicted growth in dieting and negative affect, and initial dieting and negative affect predicted growth in bulimic symptoms. The expected relation between initial dieting and growth in negative affect was only marginally significant. There was also prospective support for most of the hypothesized mediational relations.

The evidence that pressure to be thin and thin-ideal internalization prospectively predicted growth in body dissatisfaction was novel in that few studies have investigated the risk factors for this outcome with longitudinal data. However, the first result dovetails with the finding that weight-related teasing prospectively predicted increased body image disturbances (Cattarin & Thompson, 1994). These results provide support for the assertion that pressure to be thin from the proximal social environment plays a role in the genesis of body dissatisfaction as well as for the claim that a subscription to the thin ideal may contribute to body image disturbances.

The finding that body dissatisfaction prospectively predicted growth in dieting was also relatively novel, although this effect is consistent with one prior study (Stice, Mazotti, et al., 1998). Individuals with body image problems are thought to engage in dieting because of the common belief that this is an effective route to weight reduction. The evidence that thin-ideal internalization prospectively predicted growth in dieting was also unique. Theoretically, thin-ideal internalization promotes body dissatisfaction, which in turn leads to dieting efforts that are intended to alter body shape. Consistent with this assertion, there was evidence that change in body dissatisfaction mediated the relation between initial thin-ideal internalization and growth in dieting. However, the mediated effect only accounted for a quarter of the direct effect, suggesting that thin-ideal internalization may directly promote dieting even in the absence of body dissatisfaction. The finding that pressure to be thin predicted growth in dieting was also novel. It is thought that pressure to be thin fosters body dissatisfaction, which, in turn, gives rise to dieting. Consonant with this, findings suggested that change in body dissatisfaction mediated the relation between initial pressure to be thin and subsequent growth in dieting. Again, however, because the mediated effect was relatively small and only accounted for one quarter of the direct effect, it seems that pressure to be thin directly promotes dieting even when it does not result in body dissatisfaction. Collectively, these results suggest that both sociocultural pressure to be thin and subscription to the thin ideal play direct and indirect roles in promoting dieting.

The evidence that body dissatisfaction predicted growth in subsequent negative affect converges with prior studies (e.g., Cole et al., 1998). Theoretically, body dissatisfaction contributes to negative affect because appearance is one the most important evaluative dimensions for females in our culture. The relation between pressure to be thin and growth in negative affect was only marginally significant but does provide some support for the suggestion that negative messages about one’s appearance from the proximal social environment contributes to affective disturbances. Pressure to be thin is thought to result in body dissatisfaction, which in turn contributes to affective distress. However, there was only marginal support for the position that change in body dissatisfaction mediated the relation between initial pressure to be thin and growth in negative affect. The finding that initial thin-ideal internalization predicted subsequent growth in negative affect was also novel. It was postulated that thin-ideal internalization gives rise to body dissatisfaction, which, in turn, contributes to negative affect. Consistent with this model, data provided evidence that change in body dissatisfaction mediated the relation between initial thin-ideal internalization and growth in negative affect, although this mediated effect was not large.

Also consistent with hypotheses, dieting predicted subsequent growth in bulimic symptoms. This result converges with several past studies that found that dieting predicted onset of binge eating and bulimic pathology (e.g., Killen et al., 1994; Patton et al., 1990). That these studies covered different developmental periods.
and used various measures suggests that this is a robust relation. Dieting is thought to increase the chances that an individual will binge eat in response to acute caloric deprivation. Further, transgressions of strict dietary rules may result in disinhibited eating because of the abstinence-violation effect. Interestingly, the evidence that dieting prospectively predicted bulimic pathology appears to be at odds with cross-sectional studies suggesting that dieting intensity was uncorrelated with binge frequency in a college sample (Lowe et al., 1996) and negatively correlated with binge frequency in a sample of bulimic individuals (Lowe, Gleave, & Murphy-Eberenz, 1998). Reanalysis of data from a sample of 265 bulimic women (Stice & Agras, 1999) similarly suggested that dieting intensity was inversely related to frequency of binge eating \( (r = -0.13, p < 0.05) \). One explanation for this pattern of findings is that dieting plays a more important role in development rather than maintenance of binge eating. This explanation could account for the null findings in the college sample, because binge eating typically emerges in middle adolescence (Stice et al., 1998a). Consistent with this interpretation, there was a significant positive correlation between dieting and the frequency of uncontrollable binge eating (assessed by the EDE-Q diagnostic item) at baseline in the current sample \( (r = 0.28, p < 0.001) \), which captures the developmental period in which binge eating typically emerges. The realization that one can use radical compensatory behaviors to control weight may result in a decreased reliance on dieting for this purpose. Alternatively, these inconsistent findings might reflect the difficulty of measuring dieting within the context of regular binge eating. All extant dieting scales reflect chronic dieting rather than assess dieting behaviors that occur exclusively between binge-eating episodes.

There was also support for the hypothesis that negative affect would predict growth in bulimic symptoms. This finding contributes to a growing number of studies suggesting that affective disturbance is a risk factor for future bulimic pathology (e.g., Stice et al., 1998a). Theoretically, individuals engage in binge eating in an effort to distract themselves from their emotional distress or to provide comfort. It is also possible that negative affect serves to disinhibit dietary restriction, which results in binge eating. This finding suggests that it might be useful for greater attention to be directed at the possible role of affect in the etiology of bulimic pathology.

Data also supported the expectation that initial body dissatisfaction would predict subsequent growth in bulimic symptoms, which converges with prior findings (e.g., Graber et al., 1994). Body dissatisfaction theoretically promotes dieting and negative affect, which, in turn, increase the chances of bulimic pathology. Consistent with this notion, there was prospective evidence that the relation between initial body dissatisfaction and subsequent growth in bulimic symptoms was mediated by a change in dieting and negative affect. The relation between body dissatisfaction and future bulimic pathology appeared to be completely mediated by growth in dieting and negative affect in that the direct effect was no longer significant when the mediational pathways were in the model. These results were unique in that prior tests of these relations have not generated prospective support for each constituent path of this mediational model. The evidence that, when change in either one of these mediational variables was entered separately into the model, the relation between initial body dissatisfaction and growth in bulimic symptoms became nonsignificant suggests that either one of these pathways may be sufficient to promote bulimic pathology. That caloric deprivation resulted in elevated binge eating among psychiatrically nondisturbed individuals (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950) is consistent with the notion that only one of these pathways may be sufficient to produce bulimic behavior. Interestingly, though, in this sample, change in these two mediational variables was overlapping (i.e., they changed in tandem). This implies that for most individuals who experienced increased bulimic pathology in this study, both the dietary and negative affect pathways were operating. It would be useful for future studies with larger samples to attempt subgrouping analyses to test more directly whether some individuals develop bulimic pathology solely because of either the dieting or negative affect pathways. It is also worth noting that the dietary pathway was much larger than the negative affect pathway, suggesting that dieting is still the most potent risk factor for this pathology.

The hypothesized relation between initial dieting and subsequent growth in negative affect was only marginally significant. Although this effect was weak, it dovetails with the evidence that the Restraint Scale predicted onset of depression (Stice, Hayward, et al., 2000). Theoretically, the failures that are often associated with dieting contribute to negative affect (Heatherton & Polivy, 1992). However, this relation was weak and, consequently, the indirect effects involving this pathway were not supported. Interestingly, the pattern of findings wherein the Restraint Scale showed stronger relations to future affective disturbance than the DRES suggests that it may be the inability to restrain one's eating, rather than actual caloric restriction, that contributes to negative affect. This interpretation raises questions about the presumed negative emotional effects of dieting. Perhaps dieting sometimes succeeds well enough to give the dieter an increased sense of control over eating and weight and thereby improves affect.

All told, results provided considerable support for the dual-pathway model. This study generated prospective evidence for six of the seven main effects proposed in this model, and the seventh was marginally significant. There was also support for most of the hypothesized mediational pathways, although several of these effects were relatively small. These results appear to be clinically significant in that this model collectively accounted for 23% of the variance in growth of DSM-IV symptoms of bulimia nervosa over the 2-year study. The analyses indicating that this model was not simply predicting growth in weight and shape overvaluation (see Footnote 5) but rather growth in all of the diagnostic symptoms of bulimia nervosa provide further evidence of the clinical utility of this account. That this study produced prospective evidence for these relations is noteworthy, because past tests of multivariate etiological models primarily relied on cross-sectional data or did not provide prospective tests for all of the constituent pathways in the models. The prospective design of this study provides some assurance that the direction of effects were as hypothesized.

Support for the dual-pathway model was further bolstered by the fact that it explained significantly more variance in bulimic symptom growth than an alternative model involving several of the same key constructs. However, the difference in variance accounted for was not large (23% vs. 19%), which roughly corresponds to a medium effect size according to Cohen's (1988) criteria. This pattern of findings may suggest that there are reciprocal relations among some of the constructs, such as body dis-
satisfaction and negative affect. Such a position would be consistent with the assertion from the cognitive-behavioral model of the maintenance of bulimic pathology proposed by Fairburn (1997). It might, therefore, be useful to consider adapting the dual-pathway model to incorporate some of these feedback loops.

That this model accounted for far less variance in growth in body mass relative to bulimic symptoms (8% vs. 23%) provides some assurance that this model is specific to bulimic pathology rather than simply predicting the development of overeating and consequent obesity. Nonetheless, it was interesting that dieting predicted weight gain. Whereas this finding is consistent with five past studies (e.g., French et al., 1994; Klesges et al., 1992), it might seem at odds with the evidence that individuals with bulimia nervosa from clinical samples often report histories of a low body weight (e.g., Garner & Fairburn, 1988). However, because no prospective community studies have demonstrated that a period of low weight predicts subsequent onset of bulimic pathology, this latter finding may be an artifact of unreliable retrospective reports or the use of unrepresentative clinical samples. Indeed, the evidence that body mass was positively related to risk for onset of future bulimic pathology (Kellen et al., 1994) raises questions about the veracity of the findings from these retrospective clinical studies.

That the dual-pathway model was positively related to future weight gain also suggests that this model is specific to bulimic rather than anorexic symptoms. Nonetheless, it is interesting to speculate how the dual-pathway model might be adapted to explain the etiology of anorexia nervosa. It seems that pressure to be thin, thin-ideal internalization, and body dissatisfaction may be shared risk factors for the two disorders, because these variables are thought to promote dieting. However, a key question is what allows some individuals to be highly successful dieters (anorexia nervosa) rather than fail at their dietary attempts and initiate episodes of binge eating (bulimia nervosa)? Clinical experience suggests that anorexic individuals develop a powerful cognitive reinforcement system within their mind wherein caloric restriction is associated with positive feelings (e.g., goodness, willpower, and moral superiority) and eating with negative feelings (e.g., disgust, gluttony, and moral weakness). This cognitive reinforcement system may allow anorexic individuals to achieve prolonged and severe caloric deprivation. It is also probable that anorexic individuals value thinness more than eating, whereas it may be the opposite for bulimic individuals. Thus, a difference in motives or expected benefits of eating versus thinness might partially explain the different trajectories for the two disorders. There may also be individual differences in the reinforcing value of eating, with bulimic individuals simply reaping more benefits from eating (perhaps mediated by differences in the dopaminergic and serotonergic systems). Another possible explanation for why intensive dieting culminates in binge eating for bulimic individuals but not anorexic individuals is that the former may have elevated temperamental impulsivity and the latter might have a greater need for control of impulses. It would be useful for future iterations of the dual-pathway model to incorporate these additional putative risk factors for bulimic pathology. Moreover, it is hoped that investigators will explore whether the proposed adapted model for anorexia nervosa has predictive utility.

**Prospective Test of Mediation**

The second aim was to introduce a new approach for testing mediation that generates prospective evidence for each of the criteria outlined by Baron and Kenny (1986). Random regression growth curve models with time-varying covariates appear well suited for this task, because they provide a prospective test for each linkage, including a test of whether a prospective relation between the independent variable and growth in the dependent variable is reduced when one controls for change in the mediator. This represents a more rigorous test of mediation than previous cross-sectional or autoregressive approaches (e.g., structural equation modeling), because it requires temporal precedence for each of Baron and Kenny’s criteria. Moreover, these analyses can be paired with direct tests of the significance of the mediated effects using Sobel’s (1982) formula. This approach to testing mediation can be easily extended to randomized experiments that manipulate the independent variable. This would provide even greater inferential confidence than is the case with exclusively prospective data, which are always open to third-variable confounds.

However, the prospective test of mediation proposed here does not capture the second meaning implied by the concept of mediation that involves the temporal sequencing of change in the variables. This second meaning implies that the independent variable causes subsequent change in the mediator, which then causes subsequent change in the dependent variable. Two possible extensions to the proposed approach may permit the documentation of the implied temporal sequencing of change. First, if more frequent measures of the mediator and dependent variables were collected, it would be possible to test whether the change in the mediator precedes change in the dependent variable. For example, in a randomized prevention trial, one could test whether the mean time at which a clinically significant reduction in the mediator (e.g., a 0.5 SD reduction) occurs is significantly earlier than the mean time at which such a reduction occurs in the dependent variable. Alternatively, one could dichotomize both the mediator and the dependent variable, collect data on the timing of these events, and use survival analysis to test whether the peak risk of occurrence for the mediator occurs significantly earlier than the peak risk of occurrence for the dependent variable. Fortunately, the framework proposed for random regression growth curve analyses could be extended easily to survival analyses, because these latter models also permit the incorporation of time-varying covariates. It is hoped that this method of prospectively testing mediation will find application, both within the eating disorder field and in other substantive areas, because it seems to represent a more methodologically rigorous test than prior approaches.

**Limitations**

It is important to consider the limitations of this study when interpreting the findings. First, the reliance on self-report data likely biased-high the magnitude of the relations. Future research should attempt to incorporate multiple reporter data (e.g., peer reports of dieting). Second, the nonexperimental nature of this study limits the confidence that can be placed in the causal inferences. It is possible that some third variable accounts for these relations, such as a genetic propensity toward obesity. Third, it would have been preferable to have assessed bulimic pathology
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with psychiatric interviews to better ensure that participants understood the concepts of binge-eating and compensatory behaviors. Fourth, a more complete understanding of the processes that may contribute to the development of bulimic pathology would have been provided if the adolescents were followed over a longer period of time. Finally, it is not known whether these findings will generalize to the prediction of clinically meaningful bulimic pathology, because the functional impairment associated with the bulimic behaviors observed in this sample was not assessed (e.g., role impairment).

Research, Prevention, and Treatment Implications

In terms of research implications, because the dual-pathway model only accounted for 23% of the variance in growth in bulimic symptoms, future research should search for additional risk factors and explore how they might work together to promote bulimic pathology. For example, it will be important to examine other factors that contribute to the variables in the dual-pathway model (such as negative affect). Part of this search for new explanatory variables should include factors that potentiate or mitigate the effects of established risk factors for bulimic symptoms. Because moderational effects are difficult to detect in field research, studies should use larger samples, optimally reliable measures, more frequent assessments, greater participant incentives, and structured interviews to maximize reliability and statistical power. Future prospective studies should also include measures of functional impairment to allow a determination of whether growth in bulimic pathology is clinically meaningful (e.g., role impairment, subjective distress, and treatment-seeking behavior). This is vitally important to help establish the emotional and functional costs of eating pathology.

Because nonexperimental research cannot rule out third-variable confounds, future research should conduct randomized prevention trials that individually reduce these risk factors to provide a more rigorous experimental test of the hypothesized etiological processes. If this model is correct, interventions that reduce any of the risk factors should result in a consequent reduction in all downstream risk factors and bulimic pathology. The first in a series of randomized prevention trials that are targeting each of the risk factors in the dual-pathway model showed that an intervention that reduced thin-ideal internalization produced subsequent decreases in body dissatisfaction, dieting, negative affect, and bulimic pathology (Stice, Dare, & Inzlicht, in press). The combination of prospective risk factor research and randomized prevention trials offers a particularly strong method of generating triangulating evidence regarding etiological processes and would also provide information on how to prevent this serious mental health problem.

These findings may also have useful clinical implications, because many of the factors in the dual-pathway model appear to play a role in the maintenance of bulimic pathology (Stice & Agras, 1998). Results collectively suggest that it might be profitable to assess the variables in this model to aid in treatment planning. For example, research suggests that bulimic individuals with elevated dieting and negative affect show a much poorer response to treatment relative to those with only elevated dieting (Stice & Agras, 1999). An assessment of the factors in the dual-pathway model might, therefore, be useful in determining the optimal focus of treatment. Such an approach would help tailor interventions to symptom presentation and has the potential to improve treatment.

References


Received November 23, 1999
Revision received June 13, 2000
Accepted August 2, 2000