Body-Image and Eating Disturbances Prospectively Predict Increases in Depressive Symptoms in Adolescent Girls: A Growth Curve Analysis

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Using data from a longitudinal community study (N = 231), the authors tested whether body-image and eating disturbances might partially explain the increase in depression observed in adolescent girls. Initial pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms, but not body mass, predicted subsequent increases in depressive symptoms, as did increases in these risk factors over the study. There was also prospective support for each of the hypothesized mediational relations linking these risk factors to increases in depressive symptoms. Effects remained significant when other established gender-nonspecific risk factors for depression (social support and emotionality) were statistically controlled. Results provide support for the assertion that body-image and eating disturbances, operating above and beyond gender-nonspecific risk factors, contribute to the elevated depression in adolescent girls.

Research suggests that depression is one of the most common psychological problems faced by adolescents (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993) and that the rates of depression have been increasing over the last several decades (Reinherz, Giacoma, Hauf, Wasserman, & Silverman, 1999). Adolescent depression is associated with suicide attempts and comorbid anxiety disorders, disruptive behavior disorders, and substance abuse and predicts future academic failure, marital difficulties, interpersonal problems, unemployment, substance abuse, delinquency, and legal problems (Birmaher et al., 1996; Gotlib, Lewinsohn, & Seeley, 1998; Reinherz et al., 1999). Depressive symptoms increase dramatically for girls relative to boys in early adolescence, and this trend continues through adulthood (Ge, Lorenz, Conger, Elder, & Simons, 1994; Hankin et al., 1998; Wichstrom, 1999). However, the explanation for this precipitous rise has been elusive (Nolen-Hoeksema & Girgus, 1994).

Established risk factors for adolescent depression, such as emotional reactivity, deficits in social support, and stressful life events (e.g., Gjone & Stevenson, 1997; Lewinsohn et al., 1994), cannot easily explain why girls show greater growth in depression than boys or the timing of the emergence of this gender difference. In response to the inability of traditional explanatory factors to account for the occurrence and timing of this gender difference, theorists have suggested that the increased depression observed among adolescent girls is rooted in an elevated subscription to the female gender role (Hill & Lynch, 1983; Petersen, Sarigiani, & Kennedy, 1991). Specifically, the gender intensification theory postulates that the physical differentiation between girls and boys that occurs with the development of secondary sexual characteristics that marks puberty triggers an increased differentiation and development of gender roles. This differential process is thought to socialize boys to value independence and exploration, and girls to value interpersonal relationships. This greater reliance on interpersonal relationships for self-worth theoretically renders girls more vulnerable to depression because people typically cannot control the emotional tone of these relationships (e.g., family discord; Davies & Windle, 1997). Moreover, this intensification of gender role putatively leads to a differentiation of coping styles between the sexes. It has been suggested that greater subscription to the male gender role results in more active coping strategies, such as planning, whereas greater subscription to the female gender role results in more passive coping strategies, such as rumination (Nolen-Hoeksema, 1994). These differences in coping are thought to leave girls more susceptible to depression.

Although theoretically appealing, the gender intensification model has received only weak and inconsistent empirical support. First, girls do not show the increases in femininity across pubertal development that are predicted by this model (e.g., Galambos, Almedia, & Petersen, 1990). Second, gender role internalization has not been found to be consistently correlated with depression during adolescence (Petersen et al., 1991; Wichstrom, 1999) or to prospectively predict increases in depressive symptoms (Allgood-Merten, Lewinsohn, & Hops, 1990; Petersen et al., 1991). Third, this account is difficult to reconcile with the evidence that it is early menarche, rather than pubertal development per se, that is correlated with depressive symptoms (Connelly, Paihoff, & Buchanan, 1996). Fourth, whereas pubertal timing is correlated with depression in early adolescence (Hayward, Gotlib, Schraedley, & Litt, 1999; Wichstrom, 1999), it has not been found to prospectively predict increases in depressive symptoms (e.g., Lewinsohn et al., 1994). Fifth, hormone levels associated with
pubertal development have not been found to correlate consistently with depressive symptoms (Buchanan, Eccles, & Becker, 1992; Nolen-Hoeksema & Girgus, 1994). Moreover, the assertion that girls are more interpersonally dependent than boys has also received inconsistent support (Ryan & Lynch, 1989; Steinberg & Silverberg, 1986). Finally, we were unable to locate any research that has found instrumental and ruminate coping to prospectively predict future increases in depression during adolescence.

A second explanation for the increase in depression for girls is that because puberty moves girls away from the thin ideal, it precipitates body-image concerns that contribute to the higher rates of depression (McCarthy, 1990; Nolen-Hoeksema, 1994; Petersen et al., 1991). In support of this explanation are findings that body dissatisfaction has emerged as a consistent predictor of future depression among girls (Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998; Rierdan, Koff, & Stubbs, 1989; Stice, Hayward, Cameron, Killen, & Taylor, 2000). Furthermore, the sex difference in depression is substantially reduced when the effects of body dissatisfaction are controlled (Allgood-Merten et al., 1990; Rierdan et al., 1989; Wichstrom, 1999). The aim of the current study was to extend this promising line of inquiry.

We propose that one possible account for the increase in depressive symptoms is that females face additional risk factors for depression above and beyond the ones they share with their male counterparts (e.g., deficits in social support) and that these extra risk factors escalate in early adolescence. We feel that such a gender-additive model may prove useful in explaining the marked increase in depressive symptoms among girls and the timing of this escalation. Extending the assertion that body dissatisfaction promotes depression among girls (McCarthy, 1990; Nolen-Hoeksema, 1994; Petersen et al., 1991), we submit that it is the broader constellation of body-image and eating-disturbance-related factors that escalate in adolescence for girls that contributes to their elevated depression (Stice et al., 2000). Drawing from an etiologic theory of bulimia nervosa (Stice & Agras, 1998), we propose that elevated body mass, perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, and eating work together in a mediational fashion to promote mood problems for adolescent girls (which in turn promote bulimic behaviors). The gender-additive model is depicted in Figure 1.

Theoretically, heightened body mass results in body dissatisfaction because being overweight is not currently socially desirable. Elevated body mass may also result in amplified perceived pressure to be thin from family, peers, and the media. Both of these effects should be strongest for girls who experience early menarche, because they are most deviant from their peers who have not yet reached puberty in terms of body dimensions. Perceived pressure to be thin in turn is thought to foster an internalization of the current thin-ideal body image for women. These socialization agents putatively reinforce this ideal through comments or actions that serve to support and perpetuate the thin-ideal body image for women (e.g., criticism regarding weight, encouragement to diet, and glorification of ultraslender models). Perceived pressure to be thin from these sources is also thought to directly result in body dissatisfaction, because repeated messages that one is not attractive would be expected to produce discontent with physical appearance. Internalization of the thin ideal putatively contributes to body dissatisfaction because this ideal is virtually unattainable for most individuals (the average fashion model is 5 feet 10 inches tall and weighs 115 pounds [1.78 m and 52 kg]). Body dissatisfaction in turn might promote depression, because appearance is one of the most central evaluative dimensions for females in our culture. Body dissatisfaction may be particularly likely to produce depression in our society because it is commonly accepted that one can attain an ultraslender body if one exercises more and consumes a lower fat diet (i.e., that body mass is under volitional control). Following from the accepted belief that caloric restriction is an effective means of weight control, body dissatisfaction is also thought to result in elevated dieting behaviors. Dieting is theorized to contribute to depression, because emotional distress may result from the failures associated with dietary efforts or could be a direct consequence of the adverse effects of caloric deprivation on mood. Bulimic symptoms, which theoretically result from body dissatisfaction and dieting, may contribute to the increases in depressive symptoms in adolescent girls because of the shame and guilt that can result from engaging in bulimic behaviors. Bulimic pathology might also contribute to depression because of the distress resulting from repeatedly losing control over eating. Thus, elevated body mass, pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic pathology are hypothesized to

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**Figure 1.** Mediational components of the gender-additive model of depression.
predict increases in depressive symptoms among girls during adolescence.

Although depressive symptoms have been shown to correlate positively with body mass, perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic pathology in samples of adolescent girls (Fabian & Thompson, 1989; Graber, Brooks-Gunn, Paikoff, & Warren, 1994; Stice, Killen, Hayward, & Taylor, 1998b; Wichstrom, 1999), few studies have investigated these relations longitudinally. A prospective design is necessary to ensure temporal precedence between the predictors and the criterion, which provides some assurance that the putative predictors are not simply concomitants or consequences of the criterion (Kraemer et al., 1997). Longitudinal studies have indicated that body dissatisfaction predicted depressive symptoms in adolescent girls (Cole et al., 1998; Rierdan et al., 1989), but there has been little research investigating a broader range of body image and eating-disturbance-related variables. In a preliminary test of the gender-additive model articulated above, elevated body dissatisfaction, dieting, and bulimic symptoms at baseline predicted subsequent onset of major depression among initially non-depressed girls over a 4-year period (Stice et al., 2000). Interestingly, body mass did not predict onset of depression, which suggests that attitudinal disturbances regarding body image may play a more important role in fostering depression than do actual body dimensions. This null finding also suggests that the assumption that elevated weight resulting from puberty is the catalyst for the body-image and eating disturbances may be questionable. One implication is that it might be necessary to revise a key tenet of the gender-additive model to focus on the attitudinal and behavioral disturbances associated with body image and eating, rather than on elevations in physical adiposity.

Whereas our preliminary study provided support for the assertion that body image and eating disturbances may contribute to the escalation in depression observed among adolescent girls, it suffered from certain methodological shortcomings (e.g., high attrition and only moderate reliability of the diagnoses of major depression). Accordingly, our first aim in this study was to attempt to replicate and extend the findings from our previous test of this model in an independent sample using an expanded set of body image and eating-related risk factors. Specifically, in this study we tested whether body mass, perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic pathology prospectively predicted increases in depressive symptoms in adolescent girls. We hypothesized that perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic pathology would prospectively predict increases in depressive symptoms. However, we expected that body mass would not predict increases in depressive symptoms because our revised model suggests that mood disturbances are more closely tied to perceived body-image problems than are actual elevations in adiposity. We focused on depressive symptoms, rather than on diagnoses of major depression, because subdiagnostic levels of depression persist over time, foretell the onset of psychiatric disorders, and are associated with comorbid psychopathology, academic problems, impaired social functioning and suicidal ideation (Gotlib, Lewinsohn, & Seeley, 1995; Nolen-Hoeksema, Girgis, & Seligman, 1991; Lewinsohn, Solomon, Seeley, & Zeiss, 2000).

Thus, the present study represents a "fuzzy" replication of our preliminary study because we used different measures of the putative risk factors and examined a slightly different outcome. We believe such fuzzy replications are important because they provide a test of whether the relations are robust enough to persist despite variations in measurement and methodology.

A second limitation of our preliminary investigation was that it did not explicitly test the hypothesized mediational relations among the risk factors in the gender-additive model. This model articulates a complex mediational sequence regarding how the risk factors work together to promote increased depression among girls. Specifically, we hypothesize that (a) pressure to be thin will predict increased thin-ideal internalization and body dissatisfaction; (b) thin-ideal internalization will predict increased body dissatisfaction; (c) body dissatisfaction will predict increased dieting, bulimic symptoms, and depression; (d) dieting will predict increased bulimic symptoms and depression; and (e) bulimic symptoms will predict increased depression. However, our revised model suggests that body mass will not show prospective effects on increased thin-ideal internalization and body dissatisfaction, because our preliminary study implied that body mass plays a trivial role in promoting depression. Because traditional tests of mediation (e.g., with structural equation modeling) do not provide evidence of temporal precedence for each of the constituent pathways, we developed a more rigorous analytic approach that generates a prospective test for each linkage in a complex mediational model. Thus, our second aim in the present study was to conduct a prospective test of each linkage in the gender-additive mediational model.

A third limitation of our previous test of the gender-additive model is that we did not test whether increases in the risk factors over the study predicted concomitant increases in theoretically consequent (i.e., "downstream") risk factors and depression. Although it is necessary to show that initial elevations in a putative risk factor predict subsequent increases in the outcome variable in order to provide evidence of temporal precedence, our theoretical model posits that increases in the risk factors should also predict increases in symptoms of depression. Other investigators have similarly argued for the importance of examining the effects of both initial levels of a risk factor and increases in the risk factor to provide a more dynamic analysis of change over time (Cumsille, Sayer, & Graham, 2000). Therefore, our third aim was to test whether increases in pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms, but not body mass, over the study correlated with increases in depressive symptoms. Similar analyses were also conducted for the hypothesized mediational relations, in which we tested whether increases in risk factors were correlated with increases in theoretically consequent risk factors. For example, we tested whether increases in body dissatisfaction correlated with increases in dieting. To our knowledge, past etiologic studies of adolescent depression have not addressed this question.

A final limitation of our preliminary study was that it provided only a partial test of the gender-additive model of depression because it focused solely on testing whether the body-image and eating-disturbance-related factors predicted depression in adolescent girls. The gender-additive model also postulates that these factors operate above and beyond the general risk factors for depression that males and females share. Therefore, our fourth aim was to test whether these body-image and eating-disturbance-related risk factors would predict increases in depression when two
established risk factors for depression were statistically controlled: deficits in social support (Greenberg, Lengua, Coie, & Pinder-hughes, 1999; Lewinsohn et al., 1994; Sheeber, Hops, Alpert, Davis, & Andrews, 1997; Windle, 1992) and elevations in temperamental emotionality (Gorman & Stevenson, 1997; Hayward, Killen, Kraemer, & Taylor, 1998). These analyses should serve as a test of the incremental predictive validity of this new explanatory model of the increases in depression observed among adolescent girls.

In sum, because the factors that contribute to the sharp increase in depression for adolescent girls are not well understood, we investigated whether body-image and eating disturbances predicted subsequent increases in depressive symptoms. Our first aim was to attempt a fuzzy replication of a preliminary study that had indicated that a subset of these factors predicted onset of major depression among adolescent girls; we did so by using different measures of all variables and examining an expanded set of risk factors. Our second aim was to prospectively test the hypothesized mediational relations among the risk factors articulated in the gender-additive model. Our third aim was to assess whether increases in risk factors correlated with increases in theoretically consequent risk factors and depressive symptoms. Our final aim was to explicitly test the assertion that these body-image and eating disturbances would predict increases in depressive symptoms for adolescent girls above and beyond the effects of two established risk factors for depression in both sexes: deficits in social support and temperamental emotionality.

Method

Participants

Participants were 231 female students from two northern California private high schools. At baseline, these students were in the 9th or 10th grade and ranged in age from 13 to 17 years ($M = 14.9$ years). Twenty percent of the sample were Asians, 4% were Blacks, 65% were Caucasians, 2% were Hispanics, 1% were Native Americans, and 8% specified "other." Average parental education (a proxy for socioeconomic status) ranged from grade school graduate (1%) to advanced degree (38%), which was also the mode.

Procedure

A passive consent procedure was used in which a letter was mailed home to parents that described the study and asked them to return a signed letter only if they did not want their daughter to participate in the study. Written assent was also obtained from adolescent participants. This procedure 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 7-page survey in groups of approximately 30–50 students at baseline (Time 1 = T1), 10 months following baseline (Time 2 = T2), and 20 months following baseline (Time 3 = T3). To ensure confidentiality, we used a numeric code to identify students. To compensate participants for completing the surveys, we held raffles for $15 gift certificates to a local book and music store or for magazine subscriptions. This project was approved by the Stanford University Committee for the Protection of Human Subjects.

Measures

Body mass. The body mass index (BMI = $kg/m^2$), based on self-report data, was used to reflect adiposity. Self-reported weight and height have been found to be highly correlated with direct measurements made by research assistants, with the correlations typically ranging from .94 to .99 (Attie & Brooks-Gunn, 1989; Galambos et al., 1990; United States Public Health Service, 1988). Research has documented that the BMI is a valid measure of adiposity (Garrow & Webster, 1985; Kraemer, Berkowitz, & Hamner, 1990).

Perceived pressure to be thin. Participants reported the amount of pressure to be thin they perceived from family, friends, dating partners, and the media using the 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. The internal consistency ($\alpha = .88$), test–retest reliability ($r = .93$), and predictive validity of this measure have been documented (Stice & Agras, 1998). This scale had a Cronbach’s alpha of .81 at T1 in the current 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 statements concerning what attractive women look like (sample item: "Slender women are more attractive") on 5-point scales ranging from strongly disagree to strongly agree. Items were averaged for analyses. This scale possesses acceptable internal consistency ($\alpha = .91$) and test–retest reliability ($r = .80$) as well as convergent and predictive validity (Stice & Agras, 1998). This scale had a Cronbach’s alpha of .83 at T1.

Body dissatisfaction. Body dissatisfaction was assessed with an adapted form of the Satisfaction and Dissatisfaction With Body Parts Scale (Berscheid, Walster, & Bohnstedt, 1973). This scale asks participants to indicate their level of satisfaction with eight body parts on 6-point scales ranging from extremely satisfied to extremely dissatisfied. Items were averaged for analyses. This scale has acceptable internal consistency ($\alpha = .94$), test–retest reliability ($r = .90$), and convergent and predictive validity (Stice & Agras, 1998). It had a Cronbach’s alpha of .91 at T1.

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. Research has documented the internal consistency ($\alpha = .95$) and criterion validity of this scale, including the fact that it correlates with self-reported caloric intake (Stice & Agras, 1998; Van Strien et al., 1986; Wardle & Beales, 1987). An unpublished pilot study ($N = 50$) revealed a 2-week test–retest reliability coefficient of .82 for this scale. The DRES had a Cronbach’s alpha of .93 at T1.

Bulimic symptoms. The diagnostic items from the Eating Disorder Examination–Questionnaire (EDE-Q; Fairburn & Beglin, 1994) were used to assess bulimic symptoms. The EDE-Q focuses on the past 28 days and assesses the main features of bulimia, including binge eating, compensatory behaviors, and overevaluation of weight and shape. The 17 diagnostic items for bulimia were standardized (to accommodate the different response formats) and averaged to create an overall bulimic symptom index. The internal consistency ($\alpha = .84$), criterion validity, and convergent validity of the EDE-Q have been documented (Black & Wilson, 1996; Fairburn & Beglin, 1994). An unpublished pilot study ($N = 20$) revealed a 3-week test–retest reliability coefficient of .86 for this scale. The EDE-Q had a Cronbach’s alpha of .84 at T1.

Social support. Perceived social support was measured with 12 items adapted from the Network of Relationships Inventory (Furman & Buhrmester, 1985) that assessed companionship, guidance, intimacy, affection,

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1 Although we acknowledge that there are additional established risk factors for adolescent depression, such as stressful life events (Lewinsohn et al., 1994), because there were limitations in the number of items that could be included in our questionnaire we were not able to measure additional risk factors.
admiration, and reliable alliance from parents and peers. These items asked participants to respond using a 5-point format ranging from strongly disagree to strongly agree, and all items were averaged. The internal consistency (α = .89), test–retest reliability (1-month test–retest r ranged from .66 to .70), and convergent and criterion validity of this measure have been documented (Furman, 1996; Furman & Buhrmester, 1985). This scale had a Cronbach’s alpha of .86 at T1.

Temperamental emotionality. Eight items from Buss and Plomin’s (1984) Emotionality Scale were used to assess a propensity toward becoming affectively distressed (sample item: “I get emotionally upset easily”). Items used a 5-point response format ranging from never true of me (1) to always true of me (5) and were summed for analyses. Research has found this scale to possess acceptable internal consistency (α = .82) and predictive validity (Buss & Plomin, 1984; Stice, Killen, Hayward, & Taylor, 1999a). This scale had a 10-month test–retest reliability coefficient of .66 in the current study and a Cronbach’s alpha of .79 at T1.

Depressive symptoms. The Burns Depression Checklist (BDC; Burns, 1997) was used to assess depressive symptoms. The BDC is a 25-item scale assessing depressive symptoms over the past few days; it uses a 5-point response format ranging from not at all (1) to extremely (5). Items were averaged for analyses. The BDC has been found to possess internal consistency (α = .95) and convergent validity (r = .92 with the Beck Depression Inventory; r = .87 with the Zung Depression Scale) and to accurately discriminate depressed patients from controls (Burns, 1997; Burns & Eidelson, 1998). An unpublished pilot study (N = 20) revealed an internal consistency coefficient of .87 and a 3-week test–retest reliability coefficient of .71 for this scale. This scale had a 10-month test–retest reliability coefficient of .66 in the current study and a Cronbach’s alpha of .94 at T1.

Results

Preliminary Analyses

In preliminary analyses we tested for attrition biases that might compromise the generalizability of the findings. Nine of the initial 231 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 out of three waves of data, the effective attrition rate was 2%.

Because preliminary analyses indicated that age, parental education, and ethnicity did not predict future increases in depressive symptoms (all p values > .10), these demographic variables were not used as covariates in the models. Finally, preliminary analyses also verified that the assumption of univariate normality was satisfied in that none of the skew coefficients for the measures examined here even approached 2.0 (Muthen & Kaplan, 1985).

Predictors of Increases in Depressive Symptoms

Random regression growth curve analyses (Rogosa, Brandt, & Zimowski, 1982; Rogosa & Willett, 1985) were used to investigate the study aims. We first generated individual slope and intercept parameters for each participant for body mass, perceived pressure, thin-ideal internalization, body dissatisfaction, dieting, bulimic symptoms, and depressive symptoms. The slopes are generated through ordinary least squares estimation techniques and therefore represent the average linear change (growth) in the construct across each of the two 10-month intervals for each participant. The intercept parameters were coded to represent the value of the linear growth trajectory for each participant at T1. Study aims were addressed by regressing the slope parameter for the criterion variable on the relevant intercept and slope parameters for the putative risk factors in a series of multiple regression models.

Univariate relations. We first tested whether the intercept parameters for each body-image and eating disturbance factor prospectively predicted increases in depressive symptoms, because such main effects are a necessary prerequisite for mediation (Baron & Kenny, 1986). By including the slope parameters for the risk factors in the models, these analyses also tested whether increases in each body-image and eating disturbance factor predicted increases in depressive symptoms. These aims were accomplished by regressing the slope parameter for depressive symptoms on the intercept and slope parameters for each putative risk factor in individual models. The intercept parameter for depressive symptoms was also included in all models to control for the effects of initial level of the outcome. As is the case in all longitudinal analyses, it is necessary to control for baseline levels of the criterion to ensure that the models are predicting change over time. This control ensures that any apparent effects of a risk factor on growth in depression were not simply due to the correlation between the risk factor and the criterion at baseline. This is equivalent to controlling for baseline versions of the criterion variable in prospective multiple regression models.

The unstandardized regression coefficients, 95% confidence intervals, standardized parameter estimates, percentages of variance explained, and significance levels are reported in Table 1. Consistent with expectations, neither initial body mass nor increases in body mass predicted increases in depressive symptoms over the study period. Also as hypothesized, initial levels of perceived pressure, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms predicted subsequent increases in depressive symptoms, providing evidence of temporal precedence for each of these risk factors. Furthermore, increases in perceived pressure, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms showed the expected correlations with increases in depressive symptoms, above and beyond the effects of

2 The ordinary least squares estimates of slopes and intercepts used herein are less efficient than those generated through Bayesian estimation procedures in hierarchical linear modeling (HLM) analyses (Bryk & Raudenbush, 1992). However, because HLM does not provide estimates of the unique effects (i.e., conditional effects) of multiple predictor variables on growth in a criterion variable, it was not possible to test the hypotheses of the current study with HLM. This limitation precluded us from controlling for the effects of initial levels of depressive symptoms when examining the predictors of growth in depressive symptoms over the study period with HLM. This would not allow us to rule out the possibility that any apparent effect of a risk factor on growth in depression was simply due to the correlation between the risk factor and depression at baseline. Stated differently, this type of analysis does not definitively establish temporal precedence. This limitation of HLM also prevented us from determining the effects of growth in a risk factor while controlling for initial levels of that risk factor, which was a central aim of this study. Nonetheless, it should be noted that the use of parameter estimation procedures that are less efficient should simply result in a more conservative test of the relations.
### Table 1

<table>
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<th>Risk factor</th>
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<td>Thin-ideal internalization slope</td>
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*Note. CI = confidence interval. All models included the intercept for depressive symptoms to control for the effects of initial level on change over time in this construct, but parameter estimates are not reported.*

The initial levels of these risk factors. The significant univariate effects explained between 2% and 10% of the variance in increases in depressive symptoms (above and beyond the effects of initial depressive symptoms).

Test of the mediational model. To test the hypothesized mediational relations in the gender-additive model, we estimated a series of random regression growth curve models that provided prospective tests for each of the individual linkages in this mediational account. Specifically, the slope parameters for each endogenous variable in the model were regressed on the intercept values of the "upstream" risk factors theorized to contribute to the endogenous variable. Again, the intercept parameters for criterion variables were used as covariates in all analyses to ensure that we were prospectively predicting change. These models also examined the effects of increases in the putative risk factors on increases in the criterion variables by including the slope parameters for the risk factors.

The unstandardized regression coefficients, 95% confidence intervals, standardized parameter estimates, percentages of variance explained, and significance levels from this series of models are reported in Table 2. In addition, the standardized parameter estimates from the prospective tests for each linkage are also presented in Figure 2. Consistent with expectations, initial body mass did not prospectively predict increases in pressure to be thin or body dissatisfaction. As hypothesized, initial pressure to be thin predicted subsequent increases in thin-ideal internalization and body dissatisfaction, and initial thin-ideal internalization predicted increases in body dissatisfaction. Furthermore, initial body dissatisfaction predicted increases in dieting, bulimic symptoms, and depressive symptoms. Also as expected, initial dieting predicted increases in bulimic symptoms and depressive symptoms, and initial bulimic symptoms predicted increases in depressive symptoms. Increases in these risk factors over the study also predicted increases in the theoretically consequent variables over the same time period. The significant effects explained between 2% and 23% of the variance in the criterion variables (above and beyond the effects of initial depressive symptoms).

Test of Incremental Predictive Validity

The final aim was to assess whether the body-image and eating disturbance risk factors predicted increases in depression above and beyond the effects of established gender-nonspecific risk factors for depression. We first regressed the slope parameter for depression on the initial social support and temperamentality parameters in individual models to verify that these effects were significant in the present sample. Consistent with past findings, initial social support showed a significant inverse relation with subsequent increases in depressive symptoms (β = -.15, p < .05, 2.6% of the variance explained), and initial emotionality showed a significant positive relation with increases in this outcome (β = .19, p < .01, 3.2% of the variance explained). As hypothesized, when the parameters representing initial levels of social support and emotionality were included in the univariate risk factor models, none of the significant effects became nonsignificant. However, the effect for initial thin-ideal internalization from the univariate risk factor model became a marginally significant trend (p = .06).

Discussion

The first aim of this study was to test whether the body-image and eating-disturbance-related factors that escalate in early adolescence for girls might partially account for the increases in depression that also emerge during this developmental period. Consistent with hypotheses, initial elevations in perceived pres-
Table 2
Results From the Random Regression Models Providing Prospective Tests of Each of the Mediation Linkages in the Gender-Additive Model

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Criterion variable</th>
<th>$B$</th>
<th>95% CI for $B$</th>
<th>$\beta$</th>
<th>% variance</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Pressure-to-be-thin slope</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass intercept</td>
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<td>-0.03</td>
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<td>Body mass slope</td>
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<td>-0.04</td>
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<td></td>
<td>Thin-ideal internalization slope</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>Pressure-to-be-thin intercept</td>
<td>0.16</td>
<td>0.08</td>
<td>0.24</td>
<td>.28</td>
<td>6.3***</td>
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<tr>
<td>Pressure-to-be-thin slope</td>
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<td>0.07</td>
<td>0.10</td>
<td>.24</td>
<td>5.1***</td>
</tr>
<tr>
<td></td>
<td>Body dissatisfaction slope</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass intercept</td>
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<td>-0.00</td>
<td>-0.03</td>
<td>.04</td>
<td>0.2</td>
</tr>
<tr>
<td>Body mass slope</td>
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<td>0.04</td>
<td>0.10</td>
<td>.31</td>
<td>11.6***</td>
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<td>0.01</td>
<td>0.20</td>
<td>.17</td>
<td>1.9*</td>
</tr>
<tr>
<td>Pressure-to-be-thin slope</td>
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<td>0.01</td>
<td>-0.30</td>
<td>.15</td>
<td>1.8*</td>
</tr>
<tr>
<td>Thin-ideal internalization intercept</td>
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<td>0.09</td>
<td>-0.28</td>
<td>.25</td>
<td>6.0***</td>
</tr>
<tr>
<td>Thin-ideal internalization slope</td>
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<td>-0.56</td>
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<td>14.2***</td>
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<tr>
<td></td>
<td>Dieting slope</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Body dissatisfaction intercept</td>
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<td>0.11</td>
<td>0.23</td>
<td>.38</td>
<td>12.5***</td>
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<tr>
<td>Body dissatisfaction slope</td>
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<td>0.52</td>
<td>.47</td>
<td>22.8***</td>
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<td></td>
<td>Bulimic symptom slope</td>
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<tr>
<td>Body dissatisfaction intercept</td>
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<tr>
<td>Body dissatisfaction slope</td>
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<td>1.01</td>
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<td>Dieting intercept</td>
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<td>4.03</td>
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<td></td>
<td>Depression symptom slope</td>
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<td>0.07</td>
<td>0.20</td>
<td>.27</td>
<td>6.7***</td>
</tr>
<tr>
<td>Body dissatisfaction slope</td>
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<td>0.07</td>
<td>0.29</td>
<td>.19</td>
<td>4.5*</td>
</tr>
<tr>
<td>Dieting intercept</td>
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<td>0.04</td>
<td>0.16</td>
<td>.22</td>
<td>5.0***</td>
</tr>
<tr>
<td>Dieting slope</td>
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<td>0.44</td>
<td>.29</td>
<td>9.9***</td>
</tr>
<tr>
<td>Bulimic symptom intercept</td>
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<td>0.01</td>
<td>0.03</td>
<td>.28</td>
<td>6.9***</td>
</tr>
<tr>
<td>Bulimic symptom slope</td>
<td>0.03</td>
<td>0.02</td>
<td>0.05</td>
<td>.26</td>
<td>7.5***</td>
</tr>
</tbody>
</table>

Note. CI = confidence interval.
* $p < .05$. ** $p < .01$. *** $p < .001$.

sure, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms, but not body mass, predicted subsequent increases in depressive symptoms. These findings replicate and extend the results from our preliminary test of the gender-additive model (Stice et al., 2000) in that the current study examined an expanded set of putative risk factors and used increases in depressive symptoms as the criterion rather than major depression onset. The concordance between the results of the two studies, which used different measures and methods, suggests that these relations are relatively robust. Moreover, the prospective nature of these analyses, in which initial levels of the risk factors predicted subsequent increases in depressive symptoms, rules out the possibility that the body-image and eating-disturbance-related risk factors are simply concomitants or consequences of depression. Nonetheless, because of the nonexperimental design, it is not possible to rule out third-variable explanations that could account for the relations.

The second aim of this study was to conduct a prospective test of each linkage in the gender-additive mediational model. In support of our hypotheses, initial perceived pressure to be thin prospectively predicted increases in thin-ideal internalization and body dissatisfaction over the study, and initial thin-ideal internalization predicted increased body dissatisfaction. Moreover, initial body dissatisfaction predicted increases in dieting, bulimic symptoms, and depressive symptoms. Initial dieting predicted increases in bulimic symptoms and depressive symptoms, and initial bulimic symptoms predicted increases in depressive symptoms. Also consistent with expectations was the fact that initial body mass did not show significant relations with increases in pressure to be thin and body dissatisfaction. Thus, there was prospective support for each of the hypothesized mediational linkages in the gender-additive model. We feel that the use of sequential random regression growth curve models to provide a prospective test of each mediational linkage provided a more rigorous test of this multivariate etiologic model than would other approaches to evaluating mediational accounts (e.g., structural equation modeling). Nonetheless, this technique does not currently provide a test of the temporal...
Figure 2. Standardized parameter estimates from the series of random regression growth curve models used to provide prospective tests of each linkage in the gender-additive model. * $p < .05$. *** $p < .001$.

sequencing of change implied by mediational models (i.e., whether the change in the mediator temporally precedes the change in the criterion). It may be possible for future studies to test hypotheses about the sequencing of change if more frequent assessments are conducted.

The third aim was to assess whether increases in the risk factors over the study period were associated with increases in theoretically consequent risk factors and depressive symptoms. As hypothesized, increases in perceived pressure, thin-ideal internalization, body dissatisfaction, dieting, and bulimic symptoms, but not body mass, were correlated with increases in depressive symptoms. Moreover, increases in pressure to be thin were correlated with increases in thin-ideal internalization and body dissatisfaction, and increases in thin-ideal internalization were correlated with increases in body dissatisfaction. Additionally, increases in body dissatisfaction were correlated with increases in dieting and bulimic symptoms, and increases in dieting were correlated with increases in bulimic symptoms. Interestingly, there was evidence that increases in body mass were correlated with increases in body dissatisfaction, which suggests that there is some dynamic interplay between these constructs. Although these tests do not establish temporal precedence like the above prospective effects do, these effects are consonant with the interrelations implied in the gender-additive model.

The evidence that perceived pressure to be thin and thin-ideal internalization predicted increases in depressive symptoms was novel in that we could not locate past research that examined these relations prospectively. The gender-additive model posits that pressure to be thin from socialization agents fosters an internalization of the thin-ideal and subsequent body dissatisfaction, which in turn promotes depression. The finding that body dissatisfaction predicted subsequent increases in depressive symptoms converges with past results (e.g., Riedan et al., 1989). Indeed, the evidence that body dissatisfaction predicted future depressive symptoms or onset of major depression in five independent studies suggests that this is a robust effect. Theoretically, body dissatisfaction contributes to depression because appearance is a critical evaluative dimension for females in our culture. Body dissatisfaction may also foster dieting, which in turn is postulated to lead to increased depression because of the failures that are often associated with weight loss efforts or the direct effects of caloric deprivation on mood. Consistent with this latter assertion, our findings also indicated that dieting predicted increases in depressive symptoms. Although this relation has not been evaluated in many prospective studies, this finding is consistent with previous evidence that dieting predicted onset of major depression among adolescent girls (Stice et al., 2000), and it provides support for the assertion that dieting has negative psychological consequences. Finally, data suggested that bulimic symptoms predicted increases in depressive symptoms. Bulimic pathology is thought to contribute to depression because of the shame and guilt that are often associated with binge eating and purging and because of the distress that results from repeatedly losing control over eating. Whereas this finding is relatively novel, it converges with past evidence that bulimic symptoms predicted major depression onset in adolescent girls (Stice et al., 2000).

Consistent with expectations and results from our preliminary study (Stice et al., 2000) and prior studies (Lewinsohn et al., 1994), elevated body mass did not predict increases in depression. There are several potential explanations for these null findings that should be considered. First, it is possible that the nonsignificant findings resulted because we lacked statistical power to detect an effect. However, power analysis verified that we had a power of greater than .99 to detect a medium effect in the random regression models (Cohen, 1988). A second possibility is that these effects were attenuated because there was insufficient variability in initial body mass or change in body mass over time. However, post hoc analyses verified that there was statistically significant variability in both baseline body mass and change in body mass over time ($p < .001$), making this explanation untenable. A more likely interpretation of the null findings for body mass from these two studies, taken in conjunction with the positive effects for body dissatisfaction, is that the additional risk factors for depression faced by adolescent girls are rooted in attitudinal rather than physical factors that emerge in early adolescence. Our results dovetail with the evidence that pubertal timing accounted for less
than 1% of the variance in body dissatisfaction (Wichstrom, 1999). These results seem somewhat incompatible with theories positing that the weight gain associated with puberty is the catalyst for the emergence of depression in adolescent girls because it promotes body dissatisfaction (e.g., Rierdan et al., 1989). Nonetheless, there is some possibility that puberty does fuel the increase in body-image and eating disturbances but that because most girls in this study had experienced puberty by ninth grade, body mass was no longer a risk factor for depression. Perhaps it is deviance from weight norms during a crucial developmental period that is important. Such an explanation fits well with a developmental psychopathology conception of risk. Alternatively, it may be the relative weight gain from prepubertal levels that places certain youth at risk. These interpretations suggest that the timing of puberty and the weight gain associated with puberty, rather than body mass during mid-adolescence, are potentially important risk factors for future studies to investigate.

The final aim of the study was to test the assertion that these body-image and eating-disturbance-related factors would predict increases in depression above and beyond two established gender-nonspecific risk factors for depression: deficits in social support and elevations in temperamental emotionality. Consistent with prior findings regarding these gender-nonspecific risk factors (e.g., Hayward et al., 1998; Sheeber et al., 1997), both deficits in social support and negative emotionality predicted increases in depressive symptoms. As hypothesized, none of the significant effects became nonsignificant when social support and emotionality were included as covariates, although one of the effects became marginally significant. Collectively, these findings are consistent with the assertion from the gender-additive model that the body-image and eating-disturbance-related risk factors operate above and beyond the effects of more established gender-nonspecific risk factors for depression. Nevertheless, to provide greater confidence in this conclusion it will be necessary to test whether these risk factors are predictive of future depression when other established risk factors for depression in both genders, such as stressful life events (Lewinsohn et al., 1994), are controlled.

Limitations of the Current Study

Several limitations of the current study should be taken into account when interpreting the findings. First, as in most longitudinal studies on the etiology of depression, we relied solely on self-report data. Although adolescents are considered the most valid reporters for their own depressive symptoms (Edelbrock, Costello, Dulcan, Kalas, & Conover, 1985), the use of mono-reporter data may have inflated the magnitude of the relations. Second, the measure of depression that we used is relatively new and possesses limited reliability and validity data. However, past research has provided evidence of its internal consistency, test–retest reliability, convergent validity, and discriminant validity. Third, the nonexperimental design limits the confidence that can be placed in the inferences. Randomized prevention trials that reduce these body-image and eating-disturbance-related risk factors would be useful in generating experimental evidence that these variables are causally related to the development of depression. Finally, because of the relatively small numbers of girls from specific ethnic minority groups, caution should be exercised when generalizing these findings.

Conclusions and Directions for Future Research

Collectively, the results provide support for the assertion that body-image and eating-disturbance-related variables, operating above and beyond gender-nonspecific risk factors, may partially account for the elevated rates of depression among adolescent girls. Moreover, the fact that these findings generally replicated the results from our preliminary study despite measurement and methodological differences provides increased confidence in the robustness of these effects. Nonetheless, because this study examined the risk factors for increased depression only among adolescent girls, it represents only a partial test of the gender-additive model. Future research will need to directly test whether these body-image and eating-disturbance-related risk factors prospectively predict increases in depression above and beyond the effects of gender-nonspecific risk factors for girls but not for boys. Specifically, the gender-additive model posits that in a mixed-sex sample, the gender-specific body-image and eating-disturbances factors would interact with sex in the prediction of increases in depression such that these effects would show significant relations with subsequent increases in depression for females but not for males. As detailed above, these effects should operate above and beyond the effects of the gender-nonspecific risk factors that should predict increases in future depression for both sexes. Research should also assess how the risk factors in the gender-additive model work in conjunction with other factors that have been advanced to explain the gender differences in depression, such as intensified gender socialization (Wichstrom, 1999) and coping style (Nolen-Hoeksema & Girgus, 1994). Future studies should also start at an earlier age so that the timing of the increases in risk factors can be clearly documented and linked to the emergence of the gender difference in depression. Finally, these results suggest several malleable risk factors that might be targeted in prevention efforts. Findings suggest that programs that reduce these body-image and eating disturbances might prove useful in decreasing the incidence of major depression among adolescent girls. Randomized prevention trials that target these factors would not only shed light on programs that might prove effective in reducing depression but also represent the most powerful way to test etiologic models.

References


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