Published in final edited form as:
J Res Adolesc. 2010 June ; 20(2): 389–419. doi:10.1111/j.1532-7795.2010.00646.x.

Gender Differences in Depressive Symptoms During Adolescence: The Contributions of Weight-Related Concerns and Behaviors

Christine A. Vaughan and
Center for Developmental Science, University of North Carolina at Chapel Hill

Carolyn T. Halpern
Department of Maternal and Child Health, University of North Carolina at Chapel Hill

Abstract

A theoretical model of gender differences in depressive symptoms during adolescence was evaluated using data from Waves I and II of the National Longitudinal Study of Adolescent Health. The theoretical model under examination was primarily informed by the gender-additive model of gender differences in depressive symptoms during adolescence proposed by Stice and Bearman (2001). In the model, it was posited that BMI would be associated with perceiving oneself as overweight, which would then lead to a higher probability of dieting, which would be associated with greater depressive symptoms. Participants were 10,864 male and female adolescents. Gender did not moderate any of the model pathways, but mediation analysis indicated that gender differences in changes in depressive symptoms were mediated by perceived weight status and dieting. Findings suggest that one explanation for girls' higher depressive symptoms in adolescence relative to boys is girls' greater tendencies to perceive themselves as overweight and to diet.

Depression affects women at a rate twice as high as that observed among men (Nolen-Hoeksema, 1987, 1994). Before adolescence, depressed males may actually outnumber their female peers, but at approximately age 13 or 14, depression becomes more prevalent among females than males (Hankin et al., 1998; Hankin & Abramson, 2001; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Studies using continuous, self-report measures of depressive symptoms have found that girls begin to evidence significantly higher mean levels of depressive symptoms relative to boys at the age of 13 or 14 (Wade, Cairney, & Pevalin, 2002; Wichstrom, 1999). This pattern has been supported by meta-analysis (Twenge & Nolen-Hoeksema, 2002).

Efforts to explain gender differences in depression during adolescence typically have examined suspected risk factors for depression (e.g., rumination, communion, body dissatisfaction, stressful events) individually as mediators of gender differences in depression or have examined gender as a moderator of the relationship between the risk factor and depression (Hankin & Abramson, 1999). Conceptually, mediation of the relationship between two variables occurs when the effect of one variable on the other is exerted through an intervening variable (Baron & Kenny, 1986). Mediation models of gender differences in depression therefore capture the extent to which the effect of gender on depression is transmitted through a given variable or mediator. According to moderator

Correspondence concerning this article should be addressed to Christine A. Vaughan, 1776 Main St, P.O. Box 2138, Santa Monica, CA 90407-2138. Electronic mail may be sent to cvaughan@rand.org.
Christine A. Vaughan is now at the RAND Corporation.

models, girls may manifest greater sensitivity to risk factors for depression, and therefore are more vulnerable than boys in the face of a particular risk factor (Hankin & Abramson, 1999). Mediator and moderator models of gender differences in depression are not mutually exclusive (Hankin & Abramson, 1999).

In recent years, more elaborate theories of gender differences in depression have been articulated in which models comprised of multiple paths or sequences of mediating variables, rather than a single variable, have been delineated to account for the greater propensity of girls to develop depression in adolescence (e.g., Cyranowski, Frank, Young, & Shear, 2000; Hankin & Abramson, 2001; Hyde, Mezulis, & Abramson, 2008; Stice & Bearman, 2001). One of the main recurrent themes in the search for explanations of gender differences in depression in adolescence has been the importance of weight-related concerns (Hankin & Abramson, 2001; Hyde et al., 2008; Nolen-Hoeksema & Girgus, 1994; Stice & Bearman, 2001). In one study, a theory of how these weight-related concerns and attendant weight control behaviors are temporally linked to produce higher depressive symptoms in girls relative to boys during adolescence was proposed and empirically evaluated (Stice & Bearman, 2001). According to this *gender-additive* model, weight-related concerns and behaviors are conceived of as risk factors for depression that are specific to females, thereby conferring additional risk for depression on females above and beyond other, established risk factors for depression that may be equally encountered by male and female adolescents (e.g., social support deficits) (Stice & Bearman, 2001). Stice and Bearman's (2001) test of the gender-additive model, in which most aspects of the theory garnered empirical support, was conducted on an exclusively female sample, thereby leaving unanswered the empirical question of the extent to which weight-related concerns and behaviors contribute to males' depressive symptoms. Acknowledging this limitation of their work, Stice and Bearman (2001) issued a call for future investigations to examine the gender-additive model in a mixed-sex sample and offered the prediction that gender would moderate the model pathways, such that the pathways would be significant for girls but not boys. Borrowing from the theoretical framework outlined in the gender-additive model, the purpose of the current study is to replicate and extend work in this area by examining both moderator and mediator models of the contributions of weight-related concerns and behaviors to gender differences in depressive symptoms during adolescence using a mixed-sex sample. We first describe briefly the model to be tested in the current study, then elaborate on the theoretical foundation and empirical evidence brought to bear on it.

In the current study, the following theoretical sequence of variables is subjected to empirical scrutiny: Body mass, which increases during adolescence for both boys and girls as a result of pubertal development, is posited to predict the perception of being overweight, which may then promote dieting in an attempt to lose weight. Dieting, in turn, may then propagate depressive symptoms, as dieting efforts are frequently ineffective at achieving weight loss (Stice & Bearman, 2001). In addition, the perception of being overweight is proposed to exert a direct effect on depressive symptoms. This model is depicted in Figure 1.

Theoretical Background

Physical appearance is a salient aspect of one's identity throughout the life span, but it is a more powerful predictor of global self-concept during adolescence than during any other time in life (Harter, 1999). The ultra-thin ideal for females' physical attractiveness presented by the media is nearly impossible to achieve (Fredrickson & Roberts, 1997), thereby creating a sociocultural context that is propitious for the propagation of body dissatisfaction among girls and women. In addition, research has demonstrated that lower popularity among one's peers, assessed by peer nominations, is associated with heavier body sizes and lower levels of dieting behavior among both adolescent boys and girls (Wang, Houshyar, &

Prinstein, 2006). Although the cross-sectional, correlational nature of the Wang et al. study precludes causal inferences, this finding is consistent with the notion that the contingencies in the social environment may operate to reward the attainment of sociocultural ideals of physical attractiveness in which being overweight is considered undesirable. Given that peers assume an increasingly important role in the social realm of adolescents' lives (Furman & Buhrmester, 1992), then, the social rewards of adhering to sociocultural ideals of physical attractiveness and, conversely, social punishments of failing to satisfy these ideals, may serve to amplify the influence of body dissatisfaction on adolescents' self-concepts. The central importance of physical attractiveness as a predictor of global self-worth (Harter, 1999), combined with increases in body dissatisfaction among girls and decreases in body dissatisfaction among boys during adolescence (Bearman, Presnell, Martinez, & Stice, 2006; Rosenblum & Lewis, 1999), strongly implicates weight concerns in gender differences in depression. Indeed, gender differences in body dissatisfaction appear to emerge in early adolescence between the ages of 11 and 13 (Girgus, Nolen-Hoeksema, & Seligman, 1989; Marcotte, Fortin, Potvin, & Papillon, 2002; Rosenblum & Lewis, 1999; Wichstrom, 1999), evidencing temporal propinquity to the emergence of gender differences in depressive symptoms, which occurs at approximately age 13 (Twenge & Nolen-Hoeksema, 2002). Moreover, body dissatisfaction has been shown to operate as a mediator of the relationship between gender and depressive symptoms during adolescence in several studies (Allgood-Merten, Lewinsohn, & Hops, 1990; Marcotte et al., 2002; Seiffge-Krenke & Stemmler, 2002; Wichstrom, 1999).

According to the gender-additive model (Stice & Bearman, 2001), body dissatisfaction intensifies among girls during the transition to adolescence because of the increase in adiposity they experience as they undergo pubertal maturation. This physical change takes girls further from the thin ideal at the same time that physical appearance is increasing in terms of its importance to girls' self-concept (Harter, 1999). In contrast, the physical changes that boys undergo during puberty facilitate their approximation of sociocultural ideals for the male physique, as they acquire more lean muscle mass. The proposed link between greater physical maturity and greater body mass has been demonstrated in both girls (Barker & Galambos, 2003; Ge, Elder, Regnerus, & Cox, 2001; Halpern, Udry, Campbell, & Suchindran, 1999) and boys (Barker & Galambos, 2003; Ge et al., 2001).

Research on adolescent females (Field et al., 2001; Halpern et al., 1999; Paxton, Eisenberg, & Neumark-Sztainer, 2006; Presnell, Bearman, & Stice, 2004; Rosenblum & Lewis, 1999; Stice, 2002) and males (Field et al., 2001; Paxton et al., 2006; Presnell et al., 2004; Rosenblum & Lewis, 1999) has generally confirmed that elevated body mass prospectively predicts increases in body dissatisfaction or weight concerns. However, research that has specifically tested gender as a moderator of the relationship between body mass and weight concerns has yielded mixed findings. For example, in one study, body mass prospectively predicted body dissatisfaction to a similar extent in early adolescent boys and girls (Rosenblum & Lewis, 1999). In contrast, another study found that body mass prospectively predicted weight concerns to a greater extent in early adolescent boys than early adolescent girls (Field et al., 2001). However, the pattern reflected the tendency for boys to express weight concerns primarily when they were actually overweight, compared with girls' propensity to express dissatisfaction with weight whether they were objectively overweight or not (Field et al., 2001). In another longitudinal study (Presnell et al., 2004), the relationship between body mass and body dissatisfaction was best characterized by a positive, linear function among adolescent girls and a quadratic function among boys, such that boys were dissatisfied with their bodies at both lower and higher levels of body mass. In sum, each of these studies provides a slightly different picture of the moderating role of gender, with discrepancies in their findings likely accounted for by variation in sampling and measurement strategies. Nonetheless, all converge in demonstrating that greater body

mass prospectively predicts greater body dissatisfaction and weight concerns in both sexes during adolescence. However, while both boys and girls may be vulnerable to weight concerns, the physical changes of puberty appear to amplify girls' risk of body image disturbance by increasing adiposity while dampening boys' risk of body image disturbance by increasing muscularity.

While a direct, prospective relationship between weight concerns (i.e., body dissatisfaction) and both onset of depression (Stice, Cameron, Hayward, Killen, & Taylor, 2000) and depressive symptoms (Stice & Bearman, 2001) has been demonstrated, an indirect effect of weight concerns on depressive symptoms via dieting has also been posited as part of the gender-additive model (Stice & Bearman, 2001). Discontentment with one's body may motivate dieting in an effort to reduce the discrepancy between one's ideal and actual physical selves. There is empirical support for the proposed link between body dissatisfaction and dietary restriction (Keery, van den Berg, & Thompson, 2004; Stice, 2002; Stice & Bearman, 2001). Dieting, in turn, may increase depressive symptoms because of failure to achieve the desired physical result (Stice & Bearman, 2001), as naturalistic efforts at dietary restriction tend to predict increases, rather than decreases, in body mass (Field et al., 2007; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, Shaw, & Rohde, 2005). Support for this link is tentatively provided by a meta-analysis documenting that dieting prospectively predicts increases in negative affect (Stice, 2002), which is one symptom of depression. In addition, mediation of the relationship between body dissatisfaction and depressive symptoms by dieting has been confirmed in a past test of the gender-additive model conducted on adolescent females (Stice & Bearman, 2001).

The single, most comprehensive investigation of the gender-additive model yielded support for most of its predictions in an adolescent female sample (Stice & Bearman, 2001). The contributions of weight-relevant variables to depressive symptoms remained significant even while controlling for social support and emotional reactivity. However, while the gender-additive model accounted for increases in depressive symptoms over time among adolescent girls, its utility in the explanation of gender differences in depressive symptoms during adolescence has not yet been empirically established, as an appropriate test of this model would require assessment of both boys and girls.

The Current Study

The primary purpose of the current study is to assess the utility of a model of weight-related concerns and behaviors as a contributor to gender differences in depressive symptoms during adolescence. The model is evaluated using data collected at two time points (separated by approximately 18 months) from the National Longitudinal Study of Adolescent Health (Add Health), a large, nationally representative sample of adolescents. Ideally, the ability of the model to explain the emergence of gender differences in depressive symptoms in early adolescence would be assessed in the current study. However, such a test would require that the first wave of assessment antecede the emergence of gender differences in depressive symptoms (Nolen-Hoeksema & Girgus, 1994). This test was precluded by the existence of gender differences in depressive symptoms in every age group at Add Health Wave I (Joyner & Udry, 2000). Therefore, the principal goal of the current analysis is to test the ability of the model to explain gender differences in depressive symptoms during adolescence. To this end, both moderator and mediator models, explicated in greater detail below, are evaluated.

Moderator Model

According to Stice & Bearman (2001), evaluation of the gender-additive model in a mixed-sex sample should reveal that gender moderates the pathways in the model, such that the

pathways are significant among girls but not boys. Thus, support for this model would be obtained if, for girls but not boys, BMI predicts increases in perceived weight status, which in turn predicts increases in dieting behavior, which in turn predicts increases in depressive symptoms. In addition, support for the model's exclusive applicability to girls would be further buttressed by demonstration of longitudinal mediation of the indirect effect of BMI on depressive symptoms through perceived weight status and dieting for girls but not boys.

Mediational Model

Alternatively, it is possible that the slopes for these pathways are homogeneous for boys and girls (i.e., model pathways do not interact with gender), and that girls are simply higher on variables reflecting weight-related concerns and behavior, which accounts for their higher levels of depressive symptoms relative to boys. This is a mediational model of gender differences in depressive symptoms, i.e., the notion that gender differences in depressive symptoms are explained by gender differences in risk factors for depressive symptoms. Applying a mediational framework to the gender-additive model, girls' higher levels of depressive symptoms relative to boys can be explained by girls experiencing greater weight concerns than boys, which would increase their probability of dieting relative to boys', which would then lead girls to experience greater levels of depressive symptoms. Thus, if boys experienced weight concerns to the same extent as girls do, they would engage in similar levels of weight control behavior and experience similar levels of depressive symptoms. Although BMI is expected to be higher among boys than girls, the model of mediation of gender differences in depressive symptoms by model variables would be supported if, holding boys and girls equal on (i.e., controlling for) BMI, girls perceive themselves as more overweight than boys, which would then be predictive of a higher probability of dieting for girls than boys, which would then be predictive of higher depressive symptoms among girls than boys. To evaluate this model, the mediation of gender differences in changes in depressive symptoms by perceived weight status and dieting was investigated.

Method

Data Source

Data used in the current analyses were collected as part of the National Longitudinal Study of Adolescent Health (Add Health). The purpose of Add Health is to improve understanding of the predictors of health behavior and health outcomes in adolescence and emerging adulthood. To date, data have been collected at three different waves, beginning with a cohort of adolescents whose academic standing ranged from the 7^{th} to the 12^{th} grades during the 1994–1995 academic year (Bearman, Jones, & Udry, 1997); data used in the current study were limited to Waves I and II.[1]

Recruitment for participation in Add Health was initiated in schools selected to represent a stratified probability sample of all U.S. high schools. Questionnaires were completed in school by more than 90,000 adolescents in 134 high schools stratified by region. Based on completion of an in-school questionnaire or inclusion in a school roster, students were rendered eligible for participation in a follow-up interview conducted in the home. A representative sample was invited to undergo an in-home interview, resulting in the completion of more than 20,000 Wave I in-home interviews in 1995 (roughly 79% of

[1]Although three waves of data from Add Health are available, the third wave of data was not included in the present analyses because it was collected roughly five or six years after the collection of Wave II data. Thus, at Wave III respondents were between the ages of 18 and 26 and more appropriately labeled "emerging or young adults." Because the primary goal of the current investigation was to examine a theoretical model of adolescent depression, then, the inclusion of Wave III data would not facilitate the fulfillment of this goal.

eligible respondents). Wave II in-home interviews were generally conducted 12 to 18 months following the Wave I interview, with nearly 14,000 respondents or 88% of eligible respondents who completed in-home Wave I interviews also completing interviews at Wave II.

Participants recorded their responses to in-home interview questionnaires on laptop computers at both Wave I and Wave II. Questions about sensitive topics, such as sexual activity, were administered with audio-CASI technology.

Participants

The current analyses were limited to youth who reported not dieting at Wave I, who were no older than 19 at Wave II, and who had an available sampling weight. The age restriction was imposed to limit the sample to those participants who were still adolescents at the second wave of data collection, as the goal of the study was to explain gender differences in depressive symptoms during adolescence.

Listwise deletion was used, thereby reducing the initial pool of 11,642 participants who satisfied the inclusion criteria to a final sample size of 10,864. Thus, a total of 778 participants were deleted due to missing data on one or more study variables. To ascertain the extent to which the final sample may have been biased as a result of this item non-response, means and standard errors of all study variables were compared across respondents in the final sample and a larger, more inclusive sample comprised of all respondents who met eligibility criteria. Group differences on means and standard errors for all study variables were very small, with all differences in means .10 or less and all differences in standard errors less than .01; 95% confidence intervals for means demonstrated considerable overlap across groups. Given the negligible impact of attrition on univariate summary statistics for study variables, then, it appears that the final sample was minimally biased by item non-response.

The final sample, whose composition is described here with weighted percentages and unweighted frequencies (*n*s), was characterized by a slight male majority (females: 45%, n = 5,071; males: 55%, n = 5,793), which is most likely attributable to the fact that respondents who reported dieting at Wave I were excluded from analyses, and more girls than boys reported dieting at Wave I. Reflecting the racial/ethnic breakdown of the general population, the sample was characterized by a preponderance of Caucasian adolescents (67%, n = 5,752) relative to African American (15%, n = 2,341), Hispanic (11%, n = 1,730), Asian (4%, n = 756), Native American (2%, n = 188), and other adolescents who did not identify with any of the allotted racial/ethnic categories (1%, n = 97). The majority of adolescents reported that the highest level of education attained by their parents was less than a college degree (66%, n = 6,810). Finally, the mean (weighted) age of adolescents in the current sample at Wave I was 14.99 years (SE = .11).

Measures

Age—Age was estimated as the respondent's date of birth subtracted from the date of the Wave I interview.

Gender—Gender was recorded by the interviewer. In analyses, males were assigned a value of 1, and females were assigned a value of 0.

Race/ethnicity—Race/ethnicity was self-reported by the participant. Although Add Health's assessment of race/ethnicity allowed participants to identify multiple racial/ethnic origins, to ensure that each participant was assigned to only one racial/ethnic category in the

current study, Hispanic race/ethnicity superseded African American race/ethnicity, which in turn superseded Caucasian race/ethnicity in the assignment of racial/ethnic categories. Participants who fell into a racial/ethnic category other than Hispanic, African American, or Caucasian were classified into an "other" category. The variable race/ethnicity was dummy coded in analyses, with Caucasians serving as the reference category; there were four categories of race/ethnicity, resulting in three dummy codes to be included as covariates in analyses.

Highest parental education—The higher level of education attained by either parent served as a proxy for socioeconomic status and was included in the model as a covariate. This variable was computed by taking the higher level of maternal and paternal education reported by the adolescent. If data were not available for one parent, the education level of the other parent was used. This variable was scored continuously on a scale ranging from 0 (*she/he never went to school*) to 9 (*professional training beyond a four-year college or university*); higher scores therefore represent higher parental educational attainment.

Body mass index—BMI was determined by dividing weight in pounds by squared height in inches and multiplying by 703 (Centers for Disease Control and Prevention, 2003). At Wave I, self-reported height and weight were used to compute BMI. At Wave II, both self-reported and measured height and weight were recorded, and so measured height and weight, which are the more objective indicators of height and weight, were used to compute BMI. For some respondents ($n = 101$), however, measured height and weight were missing, and so Wave II BMI was computed using self-reported height and weight instead when available. For individuals who had available data for both measured and self-reported height and weight at Wave II, correlations between self-reported and measured weight ($r = .96$), height ($r = .94$), and BMI ($r = .92$) were all very high, indicating that self-reported height and weight, although less desirable than more objective estimates, correspond closely to measured height and weight. Past research has demonstrated the validity of BMI as a measure of adiposity in children and adolescents, as evidenced by its convergence with other methods of measuring body fat, such as dual energy X-ray absorptiometry (Pietrobelli et al., 1998), and with indicators of physical health, including blood pressure (Moussa, Skaik, Selwanes, Yaghy, & Bin-Othman, 1994) and serum insulin levels (Travers, Jeffers, Bloch, Hill, & Eckel, 1995).

Perceived weight status—Respondents indicated their perception of the extent to which they were underweight or overweight at Waves I and II by answering the question, "How do you think of yourself in terms of weight?" Response options included 1 (*very underweight*), 2 (*slightly underweight*), 3 (*about the right weight*), 4 (*slightly overweight*), and 5 (*very overweight*). Higher scores on this variable therefore represent higher perceived weight status.

Dieting—Dieting behavior at Waves I and II was assessed dichotomously with the question, "In the past 7 days, which of the following things did you do in order to lose weight or to keep from gaining weight?" Respondents who selected the "dieted" option were assigned a 1, and those who did not were assigned a 0. Our limited measure does not capture a dieting continuum and its duration/chronicity as would measures such as the Dietary Intent Scale (DIS; Stice, Mazotti, Krebs, & Martin, 1998). However, dichotomous measures of self-labeled dieting demonstrate high test-retest reliability over a one-month period ($r = .67$, Franzoi & Shields, 1984), and moderate-to-large correlations, per Cohen's (1992) categorization of effect sizes, with continuous measures of dieting that reflect dieting chronicity (Stice, 1998). Moreover, past Add Health research using the present dieting

measure supports the measure's convergent and predictive validity in longitudinal analyses (Field et al., 2007; Halpern, King, Oslak, & Udry, 2005).

Depressive symptoms—Depressive symptoms at Waves I and II were assessed with a 19-item, slightly modified subset of the original 20-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). Respondents indicate how frequently they experienced a wide range of depressive symptoms during the past week on a scale ranging from 0 (*never or rarely*) to 3 (*most of the time or all of the time*). Although the CES-D was originally developed and validated on an adult sample (Radloff, 1977), the psychometric properties of the CES-D have since been investigated and documented in adolescents (Radloff, 1991). A recent confirmatory factor analysis of the CES-D conducted on a large, ethnically diverse sample of young adolescents supported the utility of a single composite score to represent the CES-D (Phillips et al., 2006). Therefore, item responses were averaged to obtain a composite score, with possible scores ranging from 0 to 3; higher scores indicate greater levels of depressive symptoms. Internal consistency reliabilities for depression scores in the current study at both Waves I and II were acceptable (Cronbach's alpha: Wave I α = .86; Wave II α = .87).

Study Design

Study hypotheses were evaluated with a prospective, longitudinal design. The recommendations of Cole and Maxwell (2003) for conducting tests of longitudinal mediation were taken into account in the study's design. According to Cole and Maxwell (2003), at least three waves of data are optimal for evaluating longitudinal mediation. In the estimation of each pathway of the mediational model, prior levels of the outcome variable should be controlled for in the regression of the outcome variable at a later time point on the predictor at a preceding time point in order to control for this "almost ubiquitous 'third variable' confound" (Cole & Maxwell, 2003, p. 560). However, although at least three waves of data are optimal, a test of longitudinal mediation can be conducted using only two waves of data under the assumption of stationarity of the causal processes posited in the model (Cole & Maxwell, 2003). Stationarity is present if "the degree to which one set of variables produces change in another set remains the same over time." As only two waves of data were available for evaluating the hypotheses of the current study, the strategy recommended by Cole and Maxwell (2003) for evaluating longitudinal mediation with two waves of data was applied in the present analyses.[2] For every pair of variables in the gender-additive model pathway except for dieting, the Wave II outcome variable was regressed on the Wave I predictor that immediately precedes it in the mediational chain and Wave I levels of the outcome variable. Thus, BMI, perceived weight status, and depressive symptoms measured at both Waves I and II were included in the current analysis.[3]

To conduct a prospective test of dieting, Wave I levels of dieting were controlled by design by eliminating participants who reported dieting at Wave I, so that all participants were effectively equal on dieting (i.e., not dieting) at baseline. The path coefficients representing the effect of the Wave I predictor on the Wave II outcome variable while controlling for the Wave I outcome variable for every pair of proximal variables in the model were then examined in a joint test of significance to assess longitudinal mediation. Two sets of tests of

[2]Although the assumption of stationarity is said to be testable with three waves of data, the third wave of data from Add Health would not have allowed for the evaluation of this assumption because of the different lengths of time that characterize the interval between Wave I and II (i.e., 18 months) and the interval between Wave II and Wave III (i.e., 5 or 6 years). Thus, the assumption of stationarity was untestable and so remains uncertain.

[3]If there are three variables, the independent variable X, a mediator Y, and the dependent variable Z, such that path a represents the regression of Y at time t-1 on X at time t-2 while controlling for Y at time t-2, and path b represents the regression of Z at time t on Y at time t-1 while controlling for Z at time t-1, then longitudinal mediation is evaluated by examining the significance of the product ab (i.e., the null hypothesis that $ab = 0$).

longitudinal mediation were conducted for the overall sample: 1) mediation of the indirect effect of Wave I BMI on Wave II depressive symptoms through perceived weight status and dieting, and 2) mediation of gender differences in depressive symptoms through perceived weight status and dieting. Although causal relationships are clearly implied in the depiction and description of the theoretical model, it should be noted that the correlational design of the current study does not permit causal inferences to be drawn. Wave I age, parent education, and race/ethnicity were included as covariates in the prediction of all dependent variables in the model.

Results

Plan of Analysis

Given the design of Add Health, all data analysis incorporated information about sampling weights, clusters or primary sampling units (i.e., schools), and stratification in the estimation of variances to ensure valid tests of significance. Stata survey analysis software (StataCorp, 2005) was used to compute univariate statistics, and Mplus version 4.2 (Muthen & Muthen, 2006) was used for model estimation.

Prior to examining study hypotheses, descriptive statistics, including means and standard errors for study variables, were computed by gender. Evaluation of study hypotheses was then accomplished through path analysis, a variant of structural equation modeling with manifest rather than latent variables. As the proposed path model contains both continuous and categorical dependent variables, thereby precluding multivariate normality, a weighted least squares (WLS) estimator, which is robust to violations of multivariate normality, was used (Jaccard & Wan, 1996). Specifically, the particular estimator employed here (WLSMV) uses a diagonal weight matrix with standard errors and a mean- and-variance-adjusted chi-square test statistic that uses a full weight matrix (Muthen & Muthen, 2006).

To interpret the results of the path analyses, three global fit indices were used to assess overall model fit: a scaled chi-square test (χ^2), root mean square error of approximation (RMSEA), and comparative fit index (CFI). Because chi-square is very sensitive to sample size and likely to be significant (indicating rejection of the model) when sample sizes are large, as in the current study, other fit indices less influenced by sample size, such as the CFI and RMSEA, were weighted more heavily in judging the adequacy of model fit, per the recommendation of Jaccard and Wan (1996). The adequacy of overall model fit must first be confirmed before interpreting the local fit indices or path coefficients in the model. The significance of the difference in fit of two models, one of which is nested in the other, was evaluated using an alternative chi-square difference test[4] computed with the DIFFTEST option in Mplus (Asparouhov & Muthen, 2006). Because the chi-square value generated with WLSMV estimation is a scaled chi-square (mean-and variance-adjusted), the traditional method of testing chi-square differences in nested models is not appropriate, as the difference between two scaled chi-square values is not distributed as chi-square (Muthen & Muthen, 2006). As recommended by Cole and Maxwell (2003), in each model estimated, the disturbances for all endogenous variables were allowed to correlate with one another to account for unmeasured influences on model variables.

[4]For all model comparisons conducted using the alternative chi-square difference test, the change in scaled chi-square ($\Delta\chi^2$) is reported. It should be noted that a deterioration in model fit is indicated by an increase in chi-square, as larger values of chi-square suggest poorer model fit. Thus, if it is stated that a deterioration in model fit resulted from changes to the baseline model, the number reported as the change in chi-square ($\Delta\chi^2$) reflects an *increase* in chi-square. Conversely, if it is stated that an improvement in model fit resulted from changes to the baseline model, the number reported as the change in chi-square corresponds to a *decrease* in chi-square.

All path coefficients reported are standardized path coefficients. Given the large sample size and therefore high power to detect even very minor effects, the magnitude of effect for each predictor of a continuous dependent variable was also evaluated in terms of the effect size (f^2) associated with its unique R^2. According to Cohen (1992), $f^2 = R^2/(1 - R^2)$, and small, medium, and large effects are indicated by an f^2 of .02, .15, and .35, respectively. To convey the magnitude of paths for which Wave II dieting, a categorical variable, was the outcome, predicted probabilities of dieting at Wave II given a particular value of the predictor(s) were generated from probit analysis.

Moderator Model—The moderator model was evaluated by first examining the model separately in boys and girls to determine whether the model fit was superior in girls relative to boys and whether the individual linkages were significant for girls but not boys. In addition, to provide a formal test of moderation of model pathways by gender, a multiple group analysis with gender as the grouping variable was conducted to assess the structural invariance of the model (Byrne, 1998). Because the alternative chi-square difference test is a test of significance that is heavily influenced by sample size, the change in the CFI is also examined to inform judgments of the meaningfulness of the group differences under examination; in the context of a multiple group analysis, a change in CFI greater than .01 in absolute value is indicative of a meaningful difference between two groups (Cheung & Rensvold, 2002). In the current model, support for a moderator model would be demonstrated by finding that, for girls but not boys (or to a stronger extent in girls relative to boys), Wave I BMI prospectively predicts Wave II perceived weight status while controlling for Wave I perceived weight status, Wave I perceived weight status predicts Wave II dieting behavior, and Wave II dieting behavior prospectively predicts Wave II depressive symptoms while controlling for Wave I depressive symptoms.

Mediational Model—After confirming the expected gender differences in perceived weight status, dieting, and depressive symptoms through the Wald tests described above, the gender-additive model was estimated in the entire group of respondents (i.e., single group analysis) using path analysis. For the proposed mediational hypothesis, the following effects were of primary interest: 1) the direct effect of gender on Wave II depression while controlling for baseline (i.e., Wave I) depressive symptoms, 2) the direct effect of gender on Wave I perceived weight status while controlling for Wave I BMI, 2) the direct effect of Wave I perceived weight status on Wave II dieting (where Wave I dieting was controlled by including only participants who were not dieting at Wave I), 3) the direct effect of Wave II dieting on Wave II depression while controlling for baseline depressive symptoms, and 4) the mediated effect of gender on Wave II depression through Wave I weight status and Wave II dieting, whose significance was determined by evaluating the null hypothesis that the paths connecting gender to Wave I weight status, Wave I weight status to Wave II dieting, and Wave II dieting to Wave II depression are simultaneously equal to zero. This test was accomplished by evaluating a baseline model in which all paths were freely estimated and comparing it to a nested model in which the three aforementioned pathways were simultaneously constrained to zero. A significant deterioration in model fit resulting from the imposition of these model constraints would indicate rejection of the null hypothesis and provide evidence of mediation.

Tests of Other Forward Effects and Backward Effects—In addition to conducting a test of longitudinal mediation, two other types of tests were performed: 1) A test of omitted paths, i.e., the direct effects of upstream variables on “downstream” endogenous variables not specified in the theoretical model of longitudinal mediation (Cole & Maxwell, 2003), which included the direct effect of Wave I BMI on Wave II dieting and Wave I BMI on Wave II depression, and 2) A test of “backward,” or reverse, effects that included evaluation

of the direct effect of Wave I depression on Wave II dieting, Wave II perceived weight status, and Wave II BMI; Wave II dieting on Wave II perceived weight status and Wave II BMI; and Wave I perceived weight status on Wave II BMI. Cole and Maxwell (2003) recommend performing these additional tests to maximize the model's predictive utility.

The first set of tests was intended to determine whether there were other "forward" direct effects not outlined in the gender-additive model that warranted inclusion in the model. After determining which, if any, additional direct effects should be included, this model then served as a baseline model in which the longitudinal mediational model was nested, thereby allowing for a chi-square difference test to evaluate longitudinal mediation through the proposed pathways. The evaluation of the additional direct effects was conducted separately within each gender in the context of the evaluation of the moderator model.

The possibility of "backward", or reverse, effects was considered by evaluating direct effects in the direction opposite that proposed by the gender-additive model. After evaluating the moderator and mediational models that were of primary interest in the current study, the aforementioned direct effects in the reverse direction were estimated in the context of a single group analysis (i.e., the entire group of respondents).

Sample Descriptives

Descriptive statistics, including means and standard errors for model variables, are presented in Table 1 by gender, along with Wald significance tests of gender differences in model variables. Consistent with expectation for a nonclinical sample, adolescents of both genders reported relatively low levels of depressive symptoms at Waves I and II, and there was no average change in depressive symptoms in between waves for the whole sample ($M = -.002$, $SE = .005$, 95% CI [−.01, .01]). For both males and females, adolescents' BMI and perceptions of their weight converged in suggesting a healthy weight for participants as a group. Wave II dieting behavior was reported by a minority of youth of both genders. Gender differences in model variables were statistically significant for all variables at Waves I and II, with girls reporting lower BMI at both waves, higher perceived weight status, and higher levels of depressive symptoms at Waves I and II than boys. In addition, a significantly greater percentage of girls relative to boys endorsed dieting behavior at Wave II.

Bivariate correlations among model variables are depicted in Table 2 by gender. Within both boys and girls, correlations among each pair of proximal variables in the model were inspected to confirm their significance, as a nonsignificant correlation would not bode well for the tenability of the model, given that the strength of relationship between two variables is typically attenuated after controlling for other variables in the estimation of the full model. For both boys and girls, Wave I BMI was significantly and positively correlated with Wave I and Wave II perceived weight status, Wave I perceived weight status was significantly and positively correlated with Wave II dieting, and Wave II dieting was significantly and positively correlated with depressive symptoms at Waves I and II. In addition, the correlations between each variable at Wave I and Wave II were large in both genders according to Cohen's (1992) categorization of effect sizes (small: $r = .10$, medium: $r = .30$, large: $r = .50$), indicating high temporal stability of BMI, perceived weight status, and depressive symptoms over the 18-month interval between Waves I and II.

Moderator Hypothesis: Multiple Group Analysis

Prior to evaluating gender as a moderator of pathways in the gender-additive model, a test of direct effects not specified in the gender-additive model was conducted within each gender to determine whether these effects warranted inclusion in the model. The additional direct

effects under consideration were from Wave I BMI to Wave II dieting and from Wave I BMI to Wave II depressive symptoms. All pathways outlined in the gender-additive model, as well as the two additional direct effects described above, were estimated. Although the model was rejected in both boys (χ^2[10, N = 5,793] = 102.67, p < .001) and girls (χ^2[8, N = 5,071) = 119.88, p < .001) by the scaled chi-square, other global fit indices less influenced by sample size suggested an excellent fit to the data in both boys (CFI = .97, RMSEA = .03) and girls (CFI = .96, RMSEA = .03). Given the good model fit suggested by the CFI and RMSEA, local fit indices (i.e., path coefficients) were then interpreted; all path coefficients reported in text are standardized path coefficients.

All pathways outlined in the gender-additive model were significant and in the expected direction in both boys and girls, with the sole exception of the direct effect of Wave I perceived weight status on Wave II depressive symptoms, which was not significant in either boys (γ = −.02, *ns*) or girls (γ = −.03, *ns*), and so, in the interest of parsimony, was excluded from subsequent model runs. The direct effect of Wave I BMI on Wave II dieting was positive and significant in both boys and girls, indicating that higher Wave I BMI was predictive of a greater probability of dieting at Wave II, and so was included in all subsequent model runs. However, the direct effect of Wave I BMI on Wave II depressive symptoms was not significant in either boys (γ = −.01, *ns*) or girls (γ = −.02, *ns*), and so was deemed unnecessary and excluded from subsequent model runs. For both boys and girls, then, it appeared that higher Wave I BMI predicted the perception of being overweight at Wave II (boys: γ = .26, p < .01; girls: γ = .33, p < .01) and an increase in dieting from Wave I to Wave II (boys: γ = .16, p < .01; girls: γ = .13, p < .01) while controlling for Wave I perceived weight status, age, parent education, and race/ethnicity; Wave I perceived weight status predicted an increase in dieting from Wave I to Wave II (boys: γ = .27, p < .01; girls: γ = .31, p < .01) while controlling for Wave I BMI, age, parent education, and race/ethnicity; and Wave II dieting predicted higher depressive symptoms at Wave II (boys: β = .08, p < .05; girls: β = .12, p < .01) after controlling for Wave I depressive symptoms, age, parent education, and race/ethnicity.

Although the prediction that the pathways in the gender-additive model would be significant for girls and not boys was contradicted by the above findings, a formal test of gender as a moderator was conducted to determine whether the magnitude of the model pathways might be greater for girls than boys. To this end, a multiple group analysis with gender as the grouping variable was conducted to evaluate the multigroup structural invariance of the model (i.e., whether the structural pathways are invariant or not different in boys and girls). First, a baseline, stacked model in which all paths were freely estimated across gender was estimated. The model was rejected by the scaled chi-square (χ^2[20, N = 10,864] = 239.30, p < .001), but other fit indices less influenced by sample size indicated good (CFI = .96) or adequate model fit (RMSEA = .05). Next, a second, nested model in which the structural paths of the gender-additive model (and the direct effect of Wave I BMI on Wave II dieting) were constrained to be equal for boys and girls was estimated. The scaled chi-square did not change significantly ($\Delta\chi^2$[3, N = 10,864] = 6.07, p = .11), and the CFI remained the same (.96) after imposing these equality constraints. According to this analysis, then, there was no support for the notion that gender moderates the pathways of the gender-additive model.

Longitudinal Mediation: Indirect Effect of BMI on Depressive Symptoms

Given that the gender-additive model evidenced structural invariance across boys and girls, data from both genders were pooled to form a single group.[5] Then, a test of longitudinal

[5]Because the structural model was invariant across gender, i.e., model pathways were not different for males and females, and to conserve space, the effect sizes and probit analyses for model pathways were reported and described only for the single group analysis in which data from males and females were combined.

mediation[6] of the indirect effect of Wave I BMI on Wave II depression through perceived weight status and dieting was performed within the overall sample, with gender included in the model as an exogenous variable for which a direct effect on each endogenous variable was estimated to control for gender. To conduct this test, a baseline model that consisted of the significant direct effects identified above in the multiple group analysis was estimated and compared to a subsequent, nested model in which the paths connecting Wave I BMI to Wave II perceived weight status (*a*), Wave I perceived weight status to Wave II dieting (*b*), and Wave II dieting to Wave II depression (*c*) were simultaneously constrained to zero. The significance of the degradation in model fit resulting from imposing these constraints was then examined, which effectively constitutes a test of the null hypothesis $abc = 0$. A significant change in chi-square would indicate rejection of this null hypothesis, thereby providing evidence of longitudinal mediation. Although the baseline model was rejected by the scaled chi-square ($\chi^2[13, N = 10{,}864] = 307.28$, $p < .001$), other fit indices less influenced by sample size suggested that the baseline model fit the data excellently (CFI = .94, RMSEA = .04). Setting the aforementioned pathways that comprise the longitudinal mediated effect specified by the gender-additive model to zero significantly degraded the fit of the model ($\Delta\chi^2[2, N = 10{,}864] = 163.69$, $p < .001$). Thus, under the assumption of stationarity, the current analyses provide evidence of longitudinal mediation of the effect of Wave I BMI on Wave II depression through perceived weight status and dieting within the entire, mixed-sex group.

Mediational Hypothesis

A second single group analysis was conducted to ascertain whether the linkages specified in the gender-additive model mediate gender differences in changes in depressive symptoms over time. The baseline model for this single group analysis was identical to the model examined in the preceding longitudinal mediation analysis with the following exceptions: To estimate gender differences in Wave I perceived weight status while controlling for Wave I BMI, direct effects of gender and Wave I BMI on Wave I perceived weight status were added to the model. The baseline model, including standardized path coefficients, is depicted in Figure 2.

Although the baseline model was rejected by the scaled chi-square ($\chi^2[11, N = 10{,}864] = 235.36$, $p < .001$), other fit indices less influenced by sample size suggested an excellent fit of the baseline model to the data (CFI = .95, RMSEA = .04). As depicted in Figure 2, the direct effect of gender on Wave II depressive symptoms after controlling for Wave I depressive symptoms, age, parent education, race/ethnicity, and Wave II dieting was significant, indicating that girls continued to report higher levels of Wave II depressive symptoms than boys even in the presence of the aforementioned covariates. The direct effect of gender on Wave I perceived weight status remained significant even after controlling for Wave I BMI, age, parent education, and race/ethnicity, indicating that, even when boys and girls are held equal on BMI at Wave I, girls perceive themselves as more overweight than their male peers; however, the unique effect of gender on Wave I perceived weight status ($f^2 = .01$) (i.e., after partialling out the effects of the other variables) was very small. Wave I perceived weight status and Wave I BMI made significant, independent contributions above and beyond those made by the aforementioned covariates to Wave II dieting, and Wave II dieting was significantly predictive of higher levels of depressive symptoms at Wave II even after controlling for age, parent education, race/ethnicity, and gender. A probit analysis of the effects of Wave I perceived weight status and Wave I BMI on Wave II dieting was

[6]It is possible to have a significant indirect effect of an independent variable on a dependent variable through a chain of intervening (i.e., mediating) variables even if the direct effect of the independent variable on the dependent variable is not significant (e.g., see Patock-Peckham & Morgan-Lopez, 2006, for an example of mediation demonstrated in the absence of a direct effect of the independent variable on the dependent variable).

conducted to make sense of the magnitude of paths leading to this categorical dependent variable. Predicted probabilities of dieting were estimated for different levels of Wave I BMI, revealing that the predicted probability of dieting at Wave II for an individual with a Wave I BMI of 20 was .34%; at a BMI of 30, which is the minimum threshold of obesity, the predicted probability of dieting was 1.02%. Estimation of the predicted probabilities of dieting as a function of different levels of Wave I perceived weight status demonstrated that the perception of being normal weight was associated with a 1.32% probability of dieting, whereas the perception of being very overweight was associated with an 8.49% probability of dieting. Wave II dieting, in turn, predicted Wave II depressive symptoms after controlling for Wave I depressive symptoms, age, parent education, gender, and race/ethnicity, but the size of this effect was very small ($f^2 = .01$).

To evaluate the hypothesis of mediation of gender differences in Wave II depressive symptoms by perceived weight status and dieting, the paths connecting gender to Wave I perceived weight status[7], Wave I perceived weight status to Wave II dieting, and Wave II dieting to Wave II depressive symptoms were simultaneously constrained to zero to assess the resultant degradation of model fit. The degradation of model fit was significant ($\Delta\chi^2[3, N = 10{,}864] = 256.65$, $p < .001$), thereby indicating that the null hypothesis of all three paths simultaneously being equivalent to zero was rejected. When the three aforementioned pathways were constrained to zero, the direct effect of gender on Wave II depressive symptoms was $\gamma = -.12$, $p < .01$; as noted above, the direct effect of gender on Wave II depressive symptoms in the model that included a direct effect of Wave II dieting on Wave II depressive symptoms was significant at $\gamma = -.10$, $p < .01$. Because the direct effect of gender on Wave II depressive symptoms was significant in the model that included a direct effect of Wave II dieting on Wave II depressive symptoms, the mediation observed here can be described only as partial mediation, as the direct effect of gender on Wave II depressive symptoms in the presence of the variable preceding it in the mediational chain, Wave II dieting, would have to have been zero for full mediation to have been demonstrated. Thus, changes in gender differences in depressive symptoms are partly explained by gender differences in perceived weight status and dieting.

Reverse Effects

Per the recommendation of Cole and Maxwell (2003), a test of reverse or "backward" effects was conducted to determine whether the effects specified in the gender-additive model are bidirectional. To this end, a formal comparison (i.e., chi-square difference test) was made between the baseline model used in the single group mediation analysis (shown in Figure 2), which consisted of all of the significant forward effects, and an expanded version of the model that included all possible reverse effects. Although the model that included both forward[8] and backward effects was rejected by the scaled chi-square, ($\chi^2[10, N =$

[7]In the test of mediation of gender differences in Wave II depressive symptoms through perceived weight status and dieting, the first linkage in this mediational chain was the direct effect of gender on Wave I perceived weight status, which differs from the longitudinal mediational model under examination in the multiple group analysis, in which the first linkage of the mediational chain was the direct effect of Wave I BMI on Wave II perceived weight status while controlling for Wave I perceived weight status. However, because the chain of variables expected to mediate the effect of gender on Wave II depressive symptoms begins with perceived weight status (because it does not make sense to begin this chain with BMI, for which boys are higher than girls), Wave I perceived weight status served as the initial dependent variable of interest. As an alternative, it would have been possible to begin this chain with a direct effect of gender on Wave II perceived weight status while controlling for both Wave I BMI and Wave I perceived weight status, but treating the direct effect of gender on Wave I perceived weight status as the first linkage in the mediational chain seemed more intuitively sensible. However, the mediated effect of gender on Wave II depression through perceived weight status and dieting was also assessed by simultaneously constraining to zero the direct effect of gender on Wave II perceived weight status, the direct effect of Wave I perceived weight status on Wave II dieting, and the direct effect of Wave II dieting on depressive symptoms, and then comparing this model to the same baseline model. A significant degradation of model fit as a result of imposing these model constraints was revealed, $\Delta\chi^2[3, N = 10{,}864] = 265.97$, $p < .001$, indicating that the mediated effect of gender on Wave II depressive symptoms through perceived weight status and dieting was significant regardless of whether the effect of gender on Wave I or Wave II perceived weight status was included in the mediational pathway.

10,864] = 217.13, $p < .001$), other fit indices less influenced by sample size suggested good model fit (CFI = .96, RMSEA = .04). Moreover, the chi-square difference test indicated that model fit was significantly improved by adding the reverse effects to the baseline model ($\Delta\chi^2$[4, N = 10,864] = 131.78, $p < .001$). Thus, the model that included reverse effects was evaluated further. Of the six reverse effects that were estimated, four were significant, including the direct effect of Wave I depressive symptoms on Wave II dieting ($\gamma = .08$, $p < .01$), the direct effect of Wave II dieting on Wave II perceived weight status ($\beta = .22$, $p < .01$; $f^2 = .04$), the direct effect of Wave II dieting on Wave II BMI ($\beta = .03$, $p < .05$; $f^2 = .001$), and the direct effect of Wave I perceived weight status on Wave II BMI ($\beta = .09$, $p < .01$; $f^2 = .01$). Inspection of the values of f^2 suggested that the latter three effects were of small magnitude. Conducting a probit analysis to assess the magnitude of the effect of Wave I depressive symptoms on Wave II dieting revealed a very weak effect, as the probability of endorsing dieting behavior at Wave II for an individual with the highest possible score on Wave I depressive symptoms (3) was only .23%. The direct effect of Wave I depressive symptoms on Wave II perceived weight status was not significant ($\gamma = -.01$, *ns*), and the effect of Wave I depressive symptoms on Wave II BMI was also not significant ($\gamma = -.01$, *ns*). In conclusion, then, for all of the pathways delineated in the gender-additive model examined here except for the pathway from perceived weight status to depressive symptoms, there is evidence of reciprocal influences of model variables on each other.

Discussion

The current findings replicate past research that has demonstrated the contributions of weight-related concerns and behaviors to adolescent girls' depressive symptoms (Stice & Bearman, 2001) and extend it by documenting the contributions of the same variables to boys' depressive symptoms. Contrary to the prediction of Stice and Bearman (2001), there was no evidence of moderation of model pathways by gender; however, it appeared that adolescent girls were higher than their male peers on perceived weight status and dieting, which mediated the relationship between gender and Wave II depressive symptoms while controlling for Wave I depressive symptoms. Thus, with regard to gender differences in depressive symptoms, the current results buttress support for a mediational model and contradict a moderator model of the role of weight-related concerns and behaviors in gender differences in depressive symptoms during adolescence. The current pattern of results converges with previous research demonstrating that gender differences in depressive symptoms in adolescence are at least partially attributable to weight-related concerns (Allgood-Merten et al., 1990; Marcotte et al., 2002; Seiffge-Krenke & Stemmler, 2002; Wichstrom, 1999).

Three findings indicated that girls' greater vulnerability to depressive symptoms during adolescence is conferred by their tendency to exhibit higher levels of the contributing factors to depressive symptoms examined here than boys, rather than greater susceptibility to depressive symptoms in the face of a particular contributing factor. First, girls' levels on all model variables except for BMI were in the direction associated with higher depressive symptoms (i.e., greater perceived weight status, greater probability of dieting). Second, gender differences in changes in depressive symptoms are significantly mediated by the proposed pathways. Third, there is no moderator effect of gender. Echoing the conclusions of other investigators (e.g., Allgood-Merten et al., 1990), it may therefore be the case that if adolescent boys attained comparable levels of these factors, they would evidence a level of

[8]The forward effects were minimally impacted by the inclusion of the reverse effects in the model, with all forward effects maintaining the same level of significance that was demonstrated in the baseline model (shown in Figure 2) that did not include reverse effects.

depressive symptoms commensurate with that observed in adolescent girls (Allgood-Merten et al., 1990).

Resonating with past research on the prospective prediction of body dissatisfaction from body mass (Field et al., 2001; Paxton et al., 2006; Presnell et al., 2004; Rosenblum & Lewis, 1999), greater body mass was longitudinally associated with greater perceived weight status in both boys and girls in the current study. In addition, gender differences in perceived weight status persisted after controlling for concurrent body mass, indicating that even when body mass was held constant across gender, girls perceived themselves as more overweight than boys. This finding might be construed as evidence that girls consider themselves overweight at a lower threshold than boys, consistent with the notion that girls and women are more frequently subjected to pressure to be thin from the media and other sources of influence (Fredrickson & Roberts, 1997). However, this finding might alternatively reflect girls' reaction to the increase in adiposity that accompanies pubertal development, whereas boys with the same BMI may have greater lean muscle mass, which would be less likely to activate concerns about excess weight. Thus, even if boys experienced similar pressures to be thin, the physical changes wrought by pubertal maturation would pose a greater psychological challenge to the well-being of girls than boys. Although past research has documented the validity of BMI as a measure of adiposity in adolescents (Pietrobelli et al., 1998), BMI nonetheless does not distinguish between adiposity and lean muscle mass. In future research, more objective assessments of adiposity (i.e., percentage of body fat assessed via skin caliper) would be useful for clarifying gender differences in weight concerns at different levels of adiposity.

Although not explicitly delineated in the gender-additive model, an additional direct path from body mass to dieting was identified in the current study. That BMI predicts dieting is to be expected: Given the sociocultural ideal of thinness, individuals who have higher body mass would presumably be more inclined to attempt to reduce the discrepancy between their actual body mass and their ideal body mass. Not surprisingly, then, past research has demonstrated that BMI predicts dieting among adolescent females (Halpern et al., 2005), and the robustness of this relationship among adolescent females has been underscored by a meta-analysis of the prospective prediction of dieting by body mass (Stice, 2002). In addition, a prospective effect of BMI on dieting while controlling for baseline levels of dieting and weight concerns in both boys and girls has been established in other research (Field et al., 2001). One explanation for the significance of the direct effect of BMI on dieting in the presence of the effect of perceived weight status on dieting is that individuals may seek to diet even if they do not perceive themselves as overweight, given that standards of thinness portrayed in the media as ideals are considered so thin as to be unattainable by the vast majority of women and therefore much smaller than the body size associated with a healthy weight (Fredrickson & Roberts, 1997). Perhaps, then, if actual body dissatisfaction, rather than perceived weight status, had been measured in the current study, the hypothesized complete mediation of the effect of BMI on dieting through body dissatisfaction implicit in the gender-additive model (Stice & Bearman, 2001) would have been demonstrated. However, it might be noted that, although the expectation of complete mediation of the BMI-dieting relationship through body dissatisfaction is intuitively appealing, it is possible that other mediators of this relationship, such as health concerns, might be operative, particularly for individuals who are less prone to experience sociocultural pressures to be thin.

Dovetailing with the previous test of the gender-additive model of depressive symptoms (Stice & Bearman, 2001), in which dieting behavior significantly predicted increases in depressive symptoms among adolescent girls, dieting behavior significantly predicted increases in depressive symptoms in the entire mixed-sex sample examined in the current

study. Although the relationship between dieting behavior and depressive symptoms was significant, it should be noted that this relationship constituted a very weak effect, in contrast to previous studies of dieting and depressive symptoms (Stice & Bearman, 2001) in which the effect was in the small-to-medium range. A meta-analysis in which the prospective effects of dieting on negative affect were evaluated revealed an average effect size that was small (Stice, 2002), however, suggesting that, although the effect in our study is weaker than the precedent established by Stice and Bearman (2001), the results of the current study are not anomalous compared to those of past research. It seems probable that the single-item, categorical measure of dieting used in the current study may have attenuated the relationship between dieting and depressive symptoms. In contrast, Stice and Bearman (2001) used a continuous measure of dieting behavior assessed over a greater duration (i.e., past six months) than the interval (i.e., the past week) assessed by the categorical measure of dieting used in the current study. Chronicity of dieting would presumably be more detrimental to one's psychological well-being than only brief intervals of dieting. In addition, continuous measures may be better at detecting variation in the construct over time than categorical measures. However, given that the effect obtained by Stice and Bearman (2001) was not very large, and the effect of this relationship suggested by meta-analysis was small (Stice, 2002), a search for moderators of the relationship between dieting and depressive symptoms may prove fruitful, as it stands to reason that the effect of dieting on depressive symptoms may depend on other factors (e.g., the type of diet undertaken, the success of dieting efforts, social support for dieting behavior).

Finally, an additional, more peripheral contribution of the current analysis is the identification of reciprocal influences in the gender-additive model. That is, although the temporal relationships specified in the gender-additive model were supported, temporal relationships in the opposite direction were also supported, indicating bidirectional influences between BMI and perceived weight status, BMI and dieting, perceived weight status and dieting, and dieting and depressive symptoms. That dieting predicts depressive symptoms may indicate that higher levels of depressive symptoms, to the extent that they are associated with higher body dissatisfaction, promote dieting in an attempt to reduce the discrepancy between one's actual and ideal body sizes. The finding that dieting prospectively predicts BMI has been suggested by other research using Add Health data (Field et al., 2007) and research conducted with other samples (Klesges, Isbell, & Klesges, 1992; Neumark-Sztainer et al., 2006), indicating that dieting is often unsuccessful at achieving the goal of weight loss or maintenance that originally motivated its initiation. The finding that dieting predicts weight status is also consistent with this research, as, to the extent that dieting is associated with BMI, it would reasonably be anticipated that dieting would also predict the perception of being overweight. That perceived weight status, i.e., perceiving oneself as overweight, is longitudinally associated with BMI might indicate that individuals who perceive themselves as more overweight engage in behaviors that enhance weight management (or, conversely neglect to engage in behaviors that would effectively enhance weight management).

Limitations

The primary weakness of the current study is the relative paucity of information on the psychometric properties of several of the measures used in Add Health. While the measures used in the current study appear to have face validity, extensive investigation of the reliability and validity of these measures has not been undertaken. In addition, several of the measures employed in the current analysis consisted of a single item, which suggests lower reliability and greater measurement error, thereby likely diminishing the magnitude of some of the observed relationships. However, although it is suspected that many of the path coefficients represent underestimates of the true relationships between model variables due

to measurement error, it is also possible that path coefficients may have been inflated by common method variance resulting from exclusive reliance on self-report measures. In a related vein, the clinical relevance of the proposed model is left ambiguous by the sole use of self-reported depressive symptomatology to the exclusion of clinician-rated diagnoses of depression, as it is unknown whether the current model can account for gender differences in diagnosable depression in early adolescence.

A caveat to consider in interpreting these results is that some of the effects obtained were of a small magnitude and may have attained statistical significance in spite of this only because of the large sample size. In addition, the findings reported here, while consistent with the proposed theoretical model, do not allow for inferences of causation due to the study's correlational design.

Future Research

Identifying pathways implicated in the emergence of gender differences in depressive symptoms during early adolescence would help to inform efforts at preventing the gender gap in depressive symptoms from unfolding during this vulnerable period. Because gender differences in depressive symptoms were already present in the current sample at the first wave of assessment, it was not possible to examine factors responsible for the emergence of gender differences in depressive symptoms in the current study. As the age of onset of puberty has declined over the last several decades (Herman-Giddens, 2006), and pubertal development is implicated in the genesis of gender differences in depression (Angold, Costello, & Worthman, 1998), it seems that research seeking to examine the emergence of gender differences in depression should begin assessing relevant developmental processes and risk factors for depression no later than middle childhood. In addition, conducting multiple waves of assessment with shorter lags in between would allow for a more precise explication of the processes that confer greater vulnerability of depression on girls than boys.

While the focus of the current study was confined to weight-related concerns and behavior, the small effects obtained in the proposed model potentially suggest that several other contributing factors to depression not examined in the current study warrant inclusion in future tests of theoretical models of gender differences in depression. Past research has implicated cognitive styles (e.g., attribution style, schematic processing, tendency to ruminate) (Hankin & Abramson, 2001; Nolen-Hoeksema, 1994), stressful life events (Hankin & Abramson, 2001; Hankin, Mermelstein, & Roesch, 2007), and an interpersonal orientation (Cyranowski et al., 2000) as factors that differentially predispose girls to increases in depressive symptoms in early adolescence, thus indicating that future theoretical models of depression may be enhanced by the incorporation of these variables. Finally, given the higher levels of depressive symptoms observed among minority youth relative to Caucasian youth here and elsewhere (e.g., Twenge & Nolen-Hoeksema, 2002), a refinement of extant theoretical models of adolescent depression to account for racial/ethnic disparities in depression should be an important priority for future research.

Conclusion

The primary aim of this study was to provide a test of the contributions of weight-related concerns and behaviors to gender differences in depressive symptoms in a mixed-sex, nationally representative sample of adolescents. In sum, the current findings are consistent with the notion that gender differences in changes in depressive symptoms during adolescence occur through the greater perceived weight status of girls relative to their same-aged male peers, even controlling for BMI. Differences in perceived weight status then result in greater dieting among girls relative to boys, which translates into greater change in

depressive symptoms among girls than boys. Thus, with regard to weight-related concerns and behaviors, the results of the current study are consistent with a mediator model, rather than a moderator model, of gender differences in depressive symptoms during adolescence. The mediator model indicates that girls' greater vulnerability to depressive symptoms during adolescence is at least partly accounted for by their greater levels of these contributing factors to depressive symptoms rather than greater susceptibility to depressive symptoms in response to these weight-related factors. The current results have particular import in light of recent increases in weight gain and obesity among both male and female U.S. adolescents (Ogden et al., 2006), indicating that increases in weight gain may be accompanied by increases in depressive symptoms among adolescents. In a similar vein, the current results potentially suggest that efforts to curtail overweight and obesity in adolescents, which have increasingly become the targets of public health interventions, may have salutary effects on depressive symptoms.

The current study had several strengths, including the generalizability of study findings to United States youth by virtue of the nationally representative sample of Add Health; the use of a mixed-sex sample to assess specifically gender differences in changes in depressive symptoms, as opposed to changes in depressive symptoms in an exclusively female sample (e.g., Stice & Bearman, 2001); prospective, longitudinal tests of the theoretical relationships among model variables; and the large sample size, which allowed for the evaluation of models of longitudinal mediation in which baseline levels of outcome variables were controlled in the estimation of all mediational linkages. These analyses typically require a sizeable sample due to the high stability of depressive symptoms and other psychological constructs over time (i.e., the limited amount of variance in the outcome that remains to be explained after controlling for baseline levels necessitates a sizeable sample to assess the significance of a predictor of change in the outcome variable). Overall, the current results attest to the viability of a theoretical framework built around weight-related concerns and behaviors in the elucidation of gender differences in depressive symptoms during adolescence.

Acknowledgments

Portions of this research were presented at the 2007 annual meeting of the Society for the Study of Human Development in State College, PA. This research was supported by a National Research Service Award (5T32HD007376) from the National Institute of Child Health and Human Development to the Carolina Consortium on Human Development at the Center for Developmental Science, University of North Carolina at Chapel Hill. The authors would like to thank Jason Beckstead of the University of South Florida and Cathy Zimmer and Chris Wiesen of the Odum Institute at the University of North Carolina at Chapel Hill for their thoughtful input on statistical analyses.

References

Allgood-Merten B, Lewinsohn PM, Hops H. Sex differences and adolescent depression. Journal of Abnormal Psychology. 1990; 99:55–63. [PubMed: 2307767]

Angold A, Costello EJ, Worthman CM. Puberty and depression: the roles of age, pubertal status and pubertal timing. Psychological Medicine. 1998; 28:51–61. [PubMed: 9483683]

Asparouhov T, Muthen B. Robust chi-square difference testing with mean and variance adjusted test statistics. Mplus Web Notes: No. 10. 2006 May.26:2006.

Barker ET, Galambos NL. Body dissatisfaction of adolescent girls and boys: Risk and resource factors. Journal of Early Adolescence. 2003; 23:141–165.

Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology. 1986; 51:1173–1182. [PubMed: 3806354]

Bearman, PS.; Jones, J.; Udry, JR. The National Study of Adolescent Health: Research Design. 1997. Retrieved from www.cpc.unc.edu/projects/addhealth/design.html

Bearman SK, Presnell K, Martinez E, Stice E. The skinny on body dissatisfaction: A longitudinal study of adolescent girls and boys. Journal of Youth and Adolescence. 2006; 35:229–241.

Byrne, BM. Structural equation modeling with LISREL, PRELIS, and SIMPLIS: Basic concepts, applications, and programming. Mahwah, NJ: Erlbaum; 1998.

Centers for Disease Control and Prevention. 2003. Retrieved from http://www.cdc.gov/nccdphp/dnpa/bmi/bmi-adult-formula.htm

Cheung GW, Rensvold RB. Evaluating goodness-of-fit indexes for testing measurement invariance. Structural Equation Modeling. 2002; 9:233–255.

Cohen J. A power primer. Psychological Bulletin. 1992; 112:155–159. [PubMed: 19565683]

Cole DA, Maxwell SE. Testing mediational models with longitudinal data: Questions and tips in the use of structural equation modeling. Journal of Abnormal Psychology. 2003; 112:558–577. [PubMed: 14674869]

Cyranowski JM, Frank E, Young E, Shear K. Adolescent onset of the gender difference in lifetime rates of major depression: A theoretical model. Archives of General Psychiatry. 2000; 57:21–27. [PubMed: 10632229]

Field AE, Aneja P, Austin SB, Shrier LA, de Moor C, Gordon-Larsen P. Race and gender differences in the association of dieting and gains in BMI among young adults. Obesity. 2007; 15:456–464. [PubMed: 17299119]

Field AE, Camargo CA, Taylor CB, Berkey CS, Roberts SB, Colditz GA. Peer, parent, and media influences on the development of weight concerns and frequent dieting among preadolescent and adolescent girls and boys. Pediatrics. 2001; 107:54–60. [PubMed: 11134434]

Franzoi SL, Shields SA. The body esteem scale: Multidimensional structure and sex differences in a college population. Journal of Personality Assessment. 1984; 48:173–178. [PubMed: 6726603]

Fredrickson BL, Roberts TA. Objectification theory: Toward understanding women's lived experiences and mental health risks. Psychology of Women Quarterly. 1997; 21:173–206.

Furman W, Buhrmester D. Age and sex differences in perceptions of networks of personal relationships. Child Development. 1992; 63:103–115. [PubMed: 1551320]

Ge X, Elder GH Jr, Regnerus M, Cox C. Pubertal transitions, perceptions of being overweight, and adolescents' psychological maladjustment: Gender and ethnic differences. Social Psychology Quarterly. 2001; 64:363–375.

Girgus, JS.; Nolen-Hoeksema, S.; Seligman, MEP. Why do sex differences in depression emerge during adolescence?. Paper presented at the 97th annual convention of the American Psychological Association; New Orleans, LA. 1989 Aug.

Halpern CT, King RB, Oslak SG, Udry JR. Body mass index, dieting, romance, and sexual activity in adolescent girls: Relationships over time. Journal of Research on Adolescence. 2005; 15:535–559.

Halpern CT, Udry JR, Campbell B, Suchindran C. Effects of body fat on weight concerns, dating, and sexual activity: A longitudinal analysis of black and white adolescent girls. Developmental Psychology. 1999; 35:721–736. [PubMed: 10380863]

Hankin BL, Abramson LY, Moffitt TE, Angell KE, Silva PA, McGee R. Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. Journal of Abnormal Psychology. 1998; 107:128–140. [PubMed: 9505045]

Hankin BL, Abramson LY. Development of gender differences in depression: description and possible explanations. Annals of Medicine. 1999; 31:372–379. [PubMed: 10680851]

Hankin BL, Abramson LY. Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. Psychological Bulletin. 2001; 12:773–796. [PubMed: 11726071]

Hankin BL, Mermelstein R, Roesch L. Sex differences in adolescent depression: Stress exposure and reactivity models. Child Development. 2007; 78:279–295. [PubMed: 17328705]

Harter, S. The construction of the self: A developmental perspective. New York: Guilford Press; 1999.

Herman-Giddens ME. Recent data on pubertal milestones in United States children: the secular trend toward earlier development. International Journal of Andrology. 2006; 29:241–246. [PubMed: 16466545]

Hyde JS, Mezulis AH, Abramson LY. The ABCs of depression: Integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. Psychological Bulletin. 2008; 115:291–313.

Jaccard, J.; Wan, CK. LISREL approaches to interaction effects in multiple regression. Thousand Oaks, CA: Sage; 1996.

Joyner K, Udry JR. You don't bring me anything but down: Adolescent romance and depression. Journal of Health and Social Behavior. 2000; 41:369–391. [PubMed: 11198563]

Keery H, van den Berg P, Thompson JK. An evaluation of the tripartite influence model of body dissatisfaction and eating disturbance with adolescent girls. Body Image. 2004; 1:237–251. [PubMed: 18089156]

Klesges RC, Isbell TR, Klesges LM. Relationship between restraint, energy intake, physical activity, and body weight: A prospective analysis. Journal of Abnormal Psychology. 1992; 101:668–674. [PubMed: 1430606]

Lewinsohn PM, Hops H, Roberts R, Seeley JR, Andrews JA. Adolescent psychopathology: I. Prevalence and incidence of depression and other DSM-III-R disorders in high school students. Journal of Abnormal Psychology. 1993; 102:133–144. [PubMed: 8436689]

Marcotte D, Fortin L, Potvin P, Papillon M. Gender differences in depressive symptoms during adolescence: Role of gender-typed characteristics, self-esteem, body image, stressful life events, and pubertal status. Journal of Emotional and Behavioral Disorders. 2002; 10:29–42.

Moussa MA, Skaik MB, Selwanes SB, Yaghy OY, Bin-Othman SA. Factors associated with obesity in school children. International Journal of Obesity and Related Metabolic Disorders. 1994; 18:513–515. [PubMed: 7920878]

Muthen, LK.; Muthen, BO. Mplus User's Guide. 4. Los Angeles, CA: Author; 2006.

Neumark-Sztainer D, Wall M, Guo J, Story M, Haines J, Eisenberg M. Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: How do dieters fare five years later? Journal of the American Dietetic Association. 2006; 106:559–568. [PubMed: 16567152]

Nolen-Hoeksema S. Sex differences in unipolar depression: Evidence and theory. Psychological Bulletin. 1987; 101:259–282. [PubMed: 3562707]

Nolen-Hoeksema S. An interactive model for the emergence of gender differences in depression in adolescence. Journal of Research on Adolescence. 1994; 4:519–534.

Nolen-Hoeksema S, Girgus JS. The emergence of gender differences in depression during adolescence. Psychological Bulletin. 1994; 115:424–443. [PubMed: 8016286]

Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. Journal of the American Medical Association. 2006; 295:1549–1555. [PubMed: 16595758]

Patock-Peckham JA, Morgan-Lopez AA. College drinking behaviors: Mediational links between parenting styles, impulse control, and alcohol-related outcomes. Psychology of Addictive Behaviors. 2006; 2:117–125. [PubMed: 16784353]

Paxton SJ, Eisenberg ME, Neumark-Sztainer D. Prospective predictors of body dissatisfaction in adolescent girls and boys: A five-year longitudinal study. Developmental Psychology. 2006; 42:888–899. [PubMed: 16953694]

Phillips GA, Shadish WR, Murray DM, Kubik M, Lytle LA, Birnbaum AS. The Center for Epidemiologic Studies Depression Scale with a young adolescent population: A confirmatory factor analysis. Multivariate Behavioral Research. 2006; 41:147–163.

Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB. Body mass index as a measure of adiposity among children and adolescents: A validation study. Journal of Pediatrics. 1998; 132:204–210. [PubMed: 9506629]

Presnell K, Bearman SK, Stice E. Risk factors for body dissatisfaction in adolescent boys and girls: A prospective study. International Journal of Eating Disorders. 2004; 36:389–401. [PubMed: 15558645]

Radloff LS. The CES-D Scale: A self-reported depression scale for research on the general population. Applied Psychological Measurement. 1977; 1:385–401.

Radloff LS. The use of the Center for Epidemiology Studies Depression Scale in adolescents and young adults. Journal of Youth and Adolescence. 1991; 20:149–166.

Rosenblum GD, Lewis M. The relations among body image, physical attractiveness, and body mass in adolescence. Child Development. 1999; 70:50–64. [PubMed: 10191514]

Seiffge-Krenke I, Stemmler M. Factors contributing to gender differences in depressive symptoms: A test of three developmental models. Journal of Youth and Adolescence. 2002; 31:405–417.

StataCorp. Stata statistical software: Release 9.2. College Station, TX: StataCorp LP; 2005.

Stice E. Prospective relation of dieting behaviors to weight change in a community sample of adolescents. Behavior Therapy. 1998; 29:277–297.

Stice E. Risk and maintenance factors for eating pathology: A meta-analytic review. Psychological Bulletin. 2002; 128:825–848. [PubMed: 12206196]

Stice E, Bearman SK. Body-image and eating disturbances prospectively predict increases in depressive symptoms in adolescent girls: A growth curve analysis. Developmental Psychology. 2001; 37:597–607. [PubMed: 11552756]

Stice E, Cameron RP, Hayward C, Killen JD, Taylor CB. Body-image and eating disturbances predict onset of depression among female adolescents: A longitudinal study. Journal of Abnormal Psychology. 2000; 109:438–444. [PubMed: 11016113]

Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. Journal of Consulting and Clinical Psychology. 1999; 67:967–974. [PubMed: 10596518]

Stice E, Mazotti L, Krebs M, Martin S. Predictors of adolescent dieting behaviors: A longitudinal study. Psychology of Addictive Behaviors. 1998; 12:195–205.

Stice E, Presnell K, Shaw H, Rohde P. Psychological and behavioral risk factors for obesity onset in adolescent girls: A prospective study. Journal of Consulting and Clinical Psychology. 2005; 73:195–202. [PubMed: 15796626]

Travers SH, Jeffers BW, Bloch CA, Hill JO, Eckel RH. Gender and Tanner stage differences in body composition and insulin sensitivity in early pubertal children. Journal of Clinical and Endocrinological Metabolism. 1995; 80:172–178.

Twenge JM, Nolen-Hoeksema S. Age, gender, race, socioeconomic status, and birth cohort differences on the children's depression inventory: A meta-analysis. Journal of Abnormal Psychology. 2002; 111:578–588. [PubMed: 12428771]

Wade TJ, Cairney J, Pevalin DJ. Emergence of gender differences in depression during adolescence: National panel results from three countries. Journal of the American Academy of Child and Adolescent Psychiatry. 2002; 41:190–198. [PubMed: 11837409]

Wang SS, Houshyar S, Prinstein MJ. Adolescent girls' and boys' weight-related health behaviors and cognitions: Associations with reputation- and preference-based peer status. Health Psychology. 2006; 25:658–663. [PubMed: 17014285]

Wichstrom L. The emergence of gender difference in depressed mood during adolescence: The role of intensified gender socialization. Developmental Psychology. 1999; 35:232–245. [PubMed: 9923478]

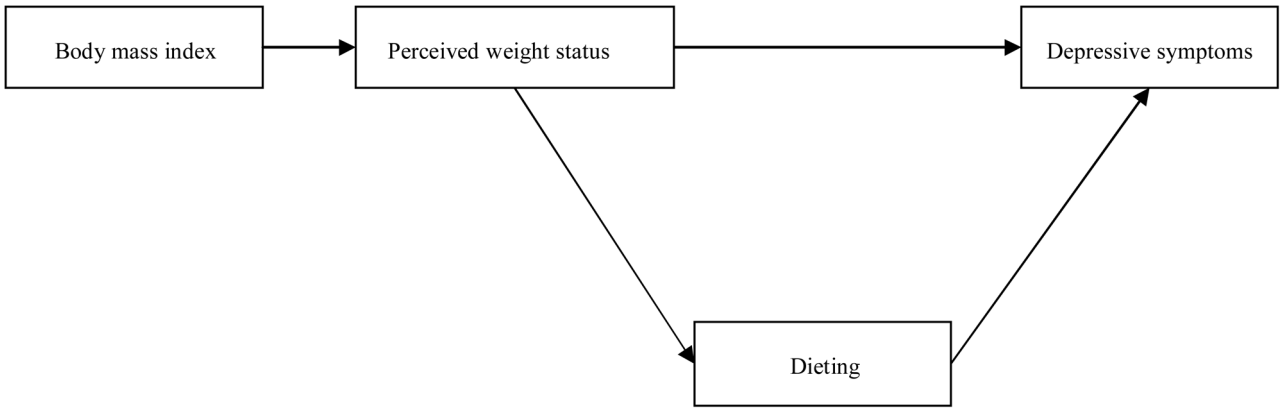

Figure 1.
Based on the gender-additive model, the basic model to be evaluated in the current investigation.

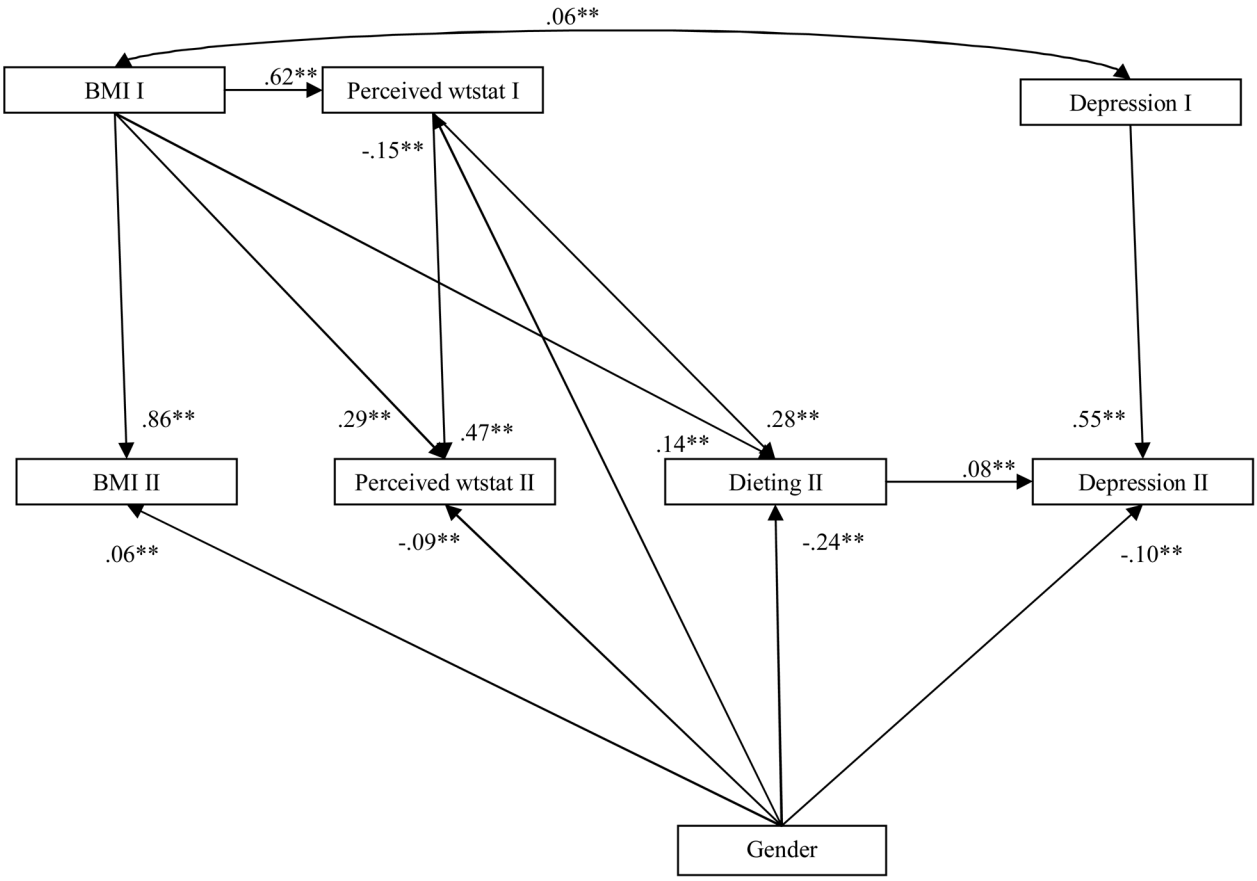

Figure 2.

Path diagram depicting the hypothesized model with standardized path coefficients (N = 10,864). Perceived wtstat I = perceived weight status at Wave I; perceived wtstat II = perceived weight status at Wave II. Gender is coded such that 0 = female, 1 = male. Absent from the figure are the covariates age, parent education, and race/ethnicity, which were controlled for in the prediction of all endogenous variables, and correlations between the disturbances of all endogenous variables not already connected by a direct effect.
*$p < .05$; **$p < .01$.

Table 1

Means, Standard Errors, and Wald Tests of Significance for Gender Differences on Model Variables at Waves I and II

	Males (*n* = 5,793)		Females (*n* = 5,071)		
	M	**SE**	**M**	**SE**	**F**
Wave I					
Body mass index	22.12	.12	21.40	.11	42.44
Weight status	2.96	.01	3.18	.01	134.48
Depressive symptoms	.52	.01	.59	.01	64.44
Wave II					
Body mass index	22.68	.11	22.14	.13	16.75
Weight status	2.98	.01	3.22	.01	148.10
Dieting	.05	.00	.15	.01	127.02
Depressive symptoms	.51	.01	.60	.01	89.08

Note. For all Wald tests of significance, $df_{num} = 1$, $df_{denom} = 128$; all gender differences were statistically significant at $p < .01$. *M* = mean, *SE* = standard error. The mean for dieting represents the percentage of respondents who reported dieting. All means and standard errors listed are weighted estimates, but *n* represents the unweighted number of participants in each group.

Table 2

Bivariate Correlations Among Model Variables by Gender

	Age	Pared	BMI1	Wtst1	Dep1	BMI2	Wtst2	Diet2	Dep2
Age	--	−.05**	.20**	−.07**	.15**	.15**	−.07**	−.02	.13**
Pared	−.03	--	−.06**	−.04**	−.16**	−.05**	−.03*	−.01	−.17**
BMI1	.16**	−.12**	--	.61**	.03*	.87**	.53**	.32**	.03*
Wtst1	.02	−.04**	.60**	--	−.03*	.59**	.64**	.37**	−.01
Dep1	.10**	−.18**	.07**	.11**	--	.01	−.03*	.07**	.58**
BMI2	.10**	−.11**	.85**	.57**	.07**	--	.56**	.32**	.03*
Wtst2	.02	−.04**	.60**	.65**	.08**	.62**	--	.43**	.00
Diet2	.06**	.00	.32**	.38**	.11**	.32**	.46**	--	.07**
Dep2	.07**	−.17**	.06**	.08**	.58**	.04**	.09**	.13**	--

Note. Age = Wave I age; Pared = parent education; BMI1 = Wave I BMI; Wtst1 = Wave I perceived weight status; Dep1 = Wave I depressive symptoms; BMI2 = Wave II BMI; Wtst2 = Wave II perceived weight status; Diet2 = Wave II dieting; Dep2 = Wave II depressive symptoms. Bivariate correlations for girls appear in the lower left triangle, and bivariate correlations for boys appear in the upper right triangle.

* $p < .05$;

** $p < .01$.