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Testing a Gender Additive Model: The Role of Body Image in Adolescent Depression

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Abstract

Despite consistent evidence that adolescent girls are at greater risk of developing depression than adolescent boys, risk factor models that account for this difference have been elusive. The objective of this research was to examine risk factors proposed by the *gender additive* model of depression that attempts to partially explain the increased prevalence of depression in adolescent girls. The theory suggests that body image and eating related variables predict depression for girls, but not for boys, above and beyond the variance accounted for by other well-known risk factors, some of which were examined in the current study. The sample was 247 adolescent girls and 181 adolescent boys studied over a 24-month duration. Results suggest that body dissatisfaction is a potent predictor of depression for girls, but not for boys, above and beyond the predictive effects of other established risk factors. Results provide insight into the etiology of adolescent depression and the disparate rate of depression among adolescent girls and provide direction for identifying high-risk individuals and developing effective prevention programs.

Keywords

Adolescent depression; Sex differences; Risk factors; Body dissatisfaction

Risk for onset of major depression increases dramatically during adolescence, particularly for adolescent girls relative to adolescent boys (Ge et al. 1994; Hankin et al. 1998; Nolen-Hoeksema 1990). The marked sex difference in depression, wherein girls are twice as likely to show onset of depression than boys, emerges between ages 13 and 16 (Hankin and Abramson 2001; Hankin et al. 1998; Ge et al. 1994). Unfortunately, the factors that increase the risk for this disorder, and for girls in particular, are less well understood. Thus, the purpose of this study was to examine risk factors proposed by the *gender additive* model of depression that attempts to partially explain the increased prevalence of depressive symptoms in adolescent girls. Identifying variables that operate differentially for girls and boys, and clarifying the role of other, well-established risk factors in the development of adolescent depression, would inform etiologic theory, the design of depression prevention programs, and the identification of high-risk populations for targeted interventions.

Prospective studies have identified several variables that predict future onset of depression or future increases in depressive symptoms among samples of female and male adolescents. Stressful life events, negative affectivity, deficits in social support, and externalizing

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symptoms are among the most robust predictors (Fergusson et al. 2003; Gjone and Stevenson 1997; Leadbeater et al. 1999; Lewinsohn et al. 1994; Windle 1992); however, their role in the development of the sex disparity in adolescent depression is unclear. Whereas some studies have found sex differences in the predictive value of these risk factors (Hankin et al. 2001; Rudolph 2002), others have not found that sex moderates the relation between these risk factors and adolescent depression (Gjone and Stevenson 1997; Pelkonen et al. 2003; Spence et al. 2002; Wiesner 2003), or examined the relations separately without testing whether sex moderates the predictive effects of the risk factors (e.g., Kaltiala-Heino et al. 2001; Slavin and Rainer 1990). An explicit test of the sex-by-risk factor interaction is required to demonstrate that the effects of the risk factor are significantly stronger for adolescent girls versus boys (i.e., that sex moderates the predictive effects).

In response to the fact that individual risk factors do not appear to explain the sex differences in depression during adolescence, theorists have offered sex-specific models of depression as a way of explaining the greater increases for adolescent girls relative to boys. One such model is the *gender additive* model of depression, which posits that as girls move further from the thin ideal during puberty, they subsequently develop body image and eating related risk factors for depression that operate *in addition* to the risk factors they share with boys (Stice et al. 2000). Specifically, this model suggests that body dissatisfaction, thin-ideal internalization, body mass, dieting, and eating disorder symptoms are more potent predictors of depression for adolescent girls than for boys, as indicated by significant interactions between the proposed risk factors and sex, and that this constellation of risk factors operates above and beyond other risk factors that predict depression for both sexes. Although some of these risk factors may also predict depression for boys, central to this theory is the notion that these sex-specific variables exert additional risk for girls, thus explaining the increase in depression for girls relative to boys during adolescence.

Theoretically, internalization of the socially sanctioned thin-ideal promotes body dissatisfaction and depression during adolescence for girls, as pubertal weight gain increases the discrepancy between their own bodies and the ideal during this stage of development (Stice et al. 2000). Because appearance is a central evaluative concern for girls in Western culture, this dissatisfaction with their shape may have an adverse impact on mood. In addition, because the thin-ideal is difficult to attain, adolescent girls may embark on a frustrating endeavor to change their shape through dieting, which in turn may contribute to depression because of emotional distress caused by repeated dietary failures. Bulimic symptoms, theoretically resulting from body dissatisfaction and dieting, may further contribute to increases in depressive symptoms among adolescent girls because of the guilt and shame associated with losing control of one's eating behaviors.

In support of this model, thin-ideal internalization, but not body mass index, predicted future increases in depressive symptoms (Stice and Bearman 2001), and body dissatisfaction predicted future increases in depressive symptoms (Cole et al. 1998; Rierdon et al. 1989; Siegal 2002) and onset of depression (Stice et al. 2000) for adolescent girls. Furthermore, a randomized prevention trial indicated that a program that reduced body dissatisfaction produced marked reductions in girls' depressive symptoms relative to waitlist controls (Bearman et al. 2003). In addition, self-reported dietary restraint predicted subsequent increases in depressive symptoms (Stice and Bearman 2001) and onset of major depression (Stice et al. 2000) for adolescent girls. Bulimic symptoms also predicted future increases in depressive symptoms (Stice and Bearman 2001) and onset of depression (Stice et al. 2000) for girls.

Boys, by comparison, experience significantly lower levels of body dissatisfaction relative to girls, and become more satisfied as they progress through adolescence (Bearman et al.

2006; Hargreaves and Tiggemann 2002; Presnell et al. 2004). Results from one study suggested that body dissatisfaction did predict depressive symptoms for boys (Siegal 2002), but to a lesser extent than for girls. Because many boys endeavor to gain weight and increase muscle tone to approximate a lean, muscular male ideal (McCabe et al. 2001), it has been suggested that they may turn to exercise, rather than dieting (Ricciardelli and McCabe 2004), thereby decreasing boys' risk for depression relative to girls. Given that boys are less likely to experience bulimic symptoms, this may represent a unique risk factor for depression for girls. To our knowledge, however, no prospective studies have tested whether internalization of the ideal body, dieting, or bulimic symptoms predict depression among adolescent boys.

Thus, there is mounting support that thin-ideal internalization, body dissatisfaction, dieting, and bulimic pathology predict increases in depression for adolescent girls. Indeed, there is also evidence that these risk factors predict depressive symptoms for girls above and beyond the predictive value of some of the above-mentioned well-known risk factors, such as negative affectivity and social support deficits (Stice and Bearman 2001; Stice et al. 2000). However, this model has not yet been tested in a sample that compares girls and boys. Moreover, some of the most robust hypothesized shared risk factors, such as stressful life events, were not investigated previously. In order to perform a more complete test of the gender additive model, the aims of the present study were to (1) test whether sex moderates the relation of the gender additive risk factors, such that these risk factors show stronger predictive effects for girls relative to boys and (2) test whether these gender additive risk factors predict depressive symptoms for girls above and beyond the predictive value of other well-established risk factors (stressful life events, negative affectivity, social support deficits, and externalizing behaviors) found to predict depression for both boys and girls. Because a test of the effects of shared risk factors is a necessary step in the examination of the gender additive model, an additional aim will be to (3) replicate the finding that stressful life events, social support deficits, negative affectivity, and externalizing behaviors increase risk for future onset of depression in adolescents, and (4) test the hypothesis that sex will not moderate these predictive effects. We consider these latter two aims exploratory because of the inconsistent support each has received previously. Although BMI failed to predict increases in girls' depressive symptoms in the one prior study that tested this hypothesis (Stice and Bearman 2001), it was retained in the current study to provide a complete test of the *gender additive* model described by Stice et al. (2000).

Methods

Participants

Participants were 247 adolescent girls and 181 adolescent boys from four public (89%) and four private (11%) middle schools in a large metropolitan area of the Southwestern US. Adolescents ranged in age from 12 to 16 (mean=13.6) at baseline. The sample was composed of 2% Asian/Pacific Islanders, 4% African Americans, 64% Caucasians, 18% Latino, 1% Native Americans, and 5% who specified "other," which was representative of the ethnic composition of the schools from which we sampled (2% Asian/Pacific Islanders; 8% African Americans, 65% Caucasians, 21% Hispanics; 4% "other or mixed"). Six percent of the sample did not report their ethnicity. Parental educational attainment ranged from grade school graduate (2%) to graduate degree (19%) with a mode of college graduate (42%), which was representative of the city from which we sampled (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree).

Procedures

The study was presented to parents and participants as an investigation of adolescent mental and physical health behaviors. Parents of all eighth grade girls and boys from the participating schools were sent a description of the study along with an informed consent letter, and active parental consent and adolescent assent was obtained from all participants. This resulted in an average participation rate of 53% of eligible students across schools. This participation rate was similar to that observed in other school-recruited samples that used active consent procedures and involved structured interviews (e.g., 61% for Lewinsohn et al. 1994).

Participants completed a self-report questionnaire, participated in a structured psychiatric interview, and had their weight and height measured by research assistants at baseline (T1) and at 1 and 2-year follow-up (T2 and T3). All measures below were assessed at each of the three time points for all participants. Assessments took place during elective courses during regular school hours or immediately after school on the school campus or in the participants' homes. Interviews were conducted by clinical assessors with a bachelors, masters, or doctoral degree in psychology. Clinical assessors initially attended 24 hrs. of training over several weeks, wherein structured interview skills and diagnostic criteria were reviewed, and interviews were simulated and observed. They next reviewed and scored 12 tape-recorded interviews of individuals with and without major depression, and were required to show a minimum (kappa) agreement with expert raters of 0.80 before starting data collection. Assessors also attended refresher interviewer training workshops twice a year throughout the study, and had to maintain test–retest kappa values of 0.80 or greater for a randomly selected 5% of the interviews that were re-conducted by the same assessor and to maintain inter-rater kappa values of 0.80 or greater for a randomly selected 5% of the interviews that were re-conducted with independent assessors during the study. Participants received a $15 gift certificate to a local book and music store as compensation for participating in the study. The University of Texas Institutional Review Board approved this project.

Measures

Stressful Life Events—The Major Life Events scale (Lewinsohn et al. 1994) assessed the occurrence of nine stressful events in the past year (e.g., *Did your parents get divorced or separated?*). Response options ranged from *no*= 1 to *at least twice*=3 and items were summed to form a composite score. It has been noted (Cleary 1981) that internal consistency is not an appropriate index of the reliability for stressful life events measures because experiencing one stressful event (e.g., having a possession stolen) should not increase the odds of experiencing others (e.g., experiencing an illness). This scale has shown 1-week test–retest reliability (r=0.90) and predictive validity for future onset of major depression (Lewinsohn et al. 1994). As expected, this scale had an α=0.48 at T1, which was similar to other stressful life events measures (αs ranged from 0.41 to 0.53; Hurst et al. 1978).

Negative Affectivity—Twelve items from Buss and Plomin's (1984) Negative Affect Scale were used to assess a propensity toward becoming emotionally distressed. Items were averaged for analyses. Research has found this scale to possess acceptable internal consistency (α=0.82), test–retest reliability (r=0.80), and predictive validity for onset of depression (Buss and Plomin 1984; Hayward et al. 1998). This scale had a α=0.79 at T1.

Social Support—Perceived social support was measured with 12 items adapted from the Network of Relationships Inventory (Furman and Buhrmester 1985) assessing companionship, guidance, intimacy, affection, admiration, and reliable alliance with parents and peers. Items are averaged for analyses to form separate scales of parental support and peer support. The internal consistency (M α=0.89), test–retest reliability (M 1-month

r=0.69), and predictive validity of this measure have been documented for the original measure (Furman and Buhrmester 1985; Furman 1996), and the revised measure has been used in a number of studies and has reliably predicted depression, substance abuse, and eating disorders (Stice et al. 2001; Burton et al. 2004). At T1 parental support had a α=0.87 and peer support had a α=0.89.

Externalizing Behaviors—Adolescents reported on the frequency of 13 externalizing behaviors using items from the Child Behavior Checklist (CBCL; Achenbach and Edelbrock 1987) on a scale of 1=*never* to 5=*always*. The CBCL has demonstrated test–retest reliability (*r*=0.95; Barkley et al. 2001) and convergent validity (Fischer et al. 1993). This 13-item scale showed internal consistency (α=0.88), 1-year test–retest reliability (*r*=0.62), and predictive validity for future increases in substance abuse (Stice et al. 1998) and had a α=0.86 at T1.

Body Dissatisfaction—Body dissatisfaction was assessed with a nine-item adapted form of the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid et al. 1973), which asks participants to indicate their level of satisfaction with nine body parts. Items are summed for analyses. This scale has shown internal consistency (α=0.94), test–retest reliability (*r*=0.90), and predictive validity for future increases in bulimic symptoms (Stice et al. 2006). This scale had a α=0.92 at T1.

Dieting—The Dutch Restrained Eating Scale (DRES; van Strein et al. 1986b) was used to assess dieting. Participants indicated the frequency of nine dieting behaviors using 5-point scales (1=*never* to 5=*always*) and items were averaged for analyses. This scale has shown internal consistency (α=0.95), test–retest reliability (*r*=0.82), convergent validity with self-reported caloric intake (but shows weaker relations to objectively measured intake), and predictive validity for future increases in bulimic symptoms (Stice et al. 2004b, 2006; van Strein et al. 1986a). This scale had an α=0.92 at T1.

Body Mass—The body mass index (BMI=kg/m^2) was used to reflect adiposity (Pietrobelli et al. 1998). Height was measured to the nearest millimeter using stadiometers and weight was measured with digital scales. Two measures of height and weight were obtained and averaged. The BMI shows convergent validity (*r* 0.80–0.90) with direct measures of total body fat such as dual energy X-ray absorptiometry (Pietrobelli et al. 1998).

Ideal-Body Internalization—The Thinness and Restricting Expectancy Inventory (TREI; Hohlstein et al. 1998) assessed ideal-body internalization for the girls and boys. Participants indicated their level of agreement with six statements concerning expected social and psychological benefits from achieving thinness using a 5-point response format ranging from 1=*strongly disagree* to 5=*strongly agree*. The TREI has adequate internal consistency (α=0.98), test–retest reliability (*r*=0.80), and predictive validity (Hohlstein et al. 1998; Stice and Bearman 2001). Because it has been demonstrated that the ideal body type for boys differs from that of girls, (Smolak et al. 2001) items were appended to reflect the expected benefits from achieving leanness and muscularity as well as thinness for males, and these items were administered to the boys. This scale had a α=0.80 for the combined sample at T1 (α=85 at T1 for boys and a α=0.80 at T1 for girls).

Bulimic Symptoms—Seventeen items from the Eating Disorder Diagnostic Interview, a semi-structured interview for diagnosing eating disorders among adolescents (Stice et al. 2004a) was used to assess symptoms of bulimia nervosa. The EDDI focuses solely on diagnosing anorexia nervosa, bulimia nervosa, and binge eating disorder over the past year and provides an overall eating disorder symptom composite. Research with adolescents has

found that the symptom composite showed adequate internal consistency ($M\alpha$=0.91) and 1-month test–retest reliability (r=0.88; Stice et al. 2004a, 2006). This scale also showed acceptable inter-rater agreement (κ=0.86), as assessed by completely independent interviews (rather than ratings of recorded interviews), and 3–5 day test–retest reliability in an adolescent sample (κ=0.96; Stice et al. 2006). This continuous scale showed predictive validity for future onset of obesity, depression, and substance abuse (Stice et al. 2004a), as well as sufficient sensitivity to detecting intervention effects from randomized prevention and treatment trials with adolescents and young adults (Burton and Stice 2006; Stice et al. 2006).

Depression Diagnoses—A structured interview adapted from the DSM-IV criteria for major depression from the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich and Chambers 1983) was used to diagnose major depression. Our version of the K-SADS combined features of the epidemiological version with the present episode version. The K-SADS has been found to have acceptable test–retest reliability (κs=0.60–1.00), inter-rater reliability (κs=0.60–1.00) and internal consistency (αs=0.68–0.84), and to discriminate between disordered and non-disordered adolescents (Ambrosini 2000; Lewinsohn et al. 1993). For diagnoses of depression, DSM-IV criteria were applied, requiring at least five symptoms, of which one must be either depressed mood or anhedonia, and interviewer-rated clinical impairment overlapping for duration of 2 weeks or longer (for full depression). Participants who reported the presence of at least five of the necessary overlapping symptoms and were rated by expert assessors as experiencing significant impairment, but who endorsed a subthreshold level on at least one of these symptoms, were given a diagnosis of minor depressive disorder (DSM-IV-TR). We included a focus on minor depressive disorder because subthreshold levels of depression are associated with current and future subjective distress and functional impairment (Gotlib et al. 1995). In support of this assertion, only about half of adolescents referred for clinical services because of depressed mood meet complete diagnostic criteria for major depressive disorder (Compas et al. 1993). In the current sample, a randomly selected subset of participants (5%) were re-interviewed by another interviewer within 3 days to establish high inter-rater agreement (κ=1.0) or by the same interviewer within 1 week to establish test–retest reliability (κ=1.0). Annually, an additional 5% of recorded interviews were reviewed and scored by the project director in order to ensure that assessors continued to show acceptable inter-rater agreement (κ>0.80). Furthermore, depressive diagnoses derived from this interview in the current data set have been found to have predictive validity for future onset of bulimia nervosa and substance abuse (Stice et al. 2004a).

Analytic Overview

Preliminary analyses tested for differences between girls and boys on all study variables and demographic factors. Attrition analyses tested whether participants who dropped from the study differed significantly from those who did not. Next, hierarchical linear models (HLM; Bryk et al. 2000) tested the prospective relations between T1 stressful life events, social support deficits, negative affectivity, externalizing behaviors, body dissatisfaction, ideal-body internalization, body mass index, dietary restraint, eating disorder symptoms, and age and risk for major depression onset over the 2-year follow-up (T2 and T3). Time was anchored at baseline and all risk factor T1 values were centered around the sample mean, producing an average of zero. In HLM, the T1 values for the outcome (initial depression status) are used to estimate the parameters of change over time for each individual, thereby ensuring a prospective test (Raudenbush and Byrk 2002). For all models, baseline levels of the risk factors were entered as level 2 time-invariant covariates. We utilized a Bernoulli sampling model and a logit-link to examine the relation of initial levels of each risk factor to future depression onset over the course of the study. Level 2 random effects were excluded

from these models because the number of iterations required in order for the analyses to converge exceeded the recommended value, suggesting that the estimated variances are likely to be close to zero (Raudenbush and Byrk 2002). To examine the univariate relation of each T1 risk factor to depression onset, the following equation was tested:

$$\text{Prob}(Y=1|B)=P$$
$$\log[P/(1-P)]=B_{0i}+B_{1i} \times (\text{TIME}) \quad \text{Level 1}$$

$$B_0=G_{00}+U_{0i}$$
$$B_1=G_{10}+G_{11} \times (\text{RISK FACTOR})+U_1 \quad \text{Level 2}$$

where P is the log of the odds of depression onset, B_{0i} represents depression status for individual i at the first data collection (T1); B_{1i} represents the probability of depression onset across the three data collections for individual i and TIME_i represents the linear trend for time across the three data collections for individual i. At level 2, G_{00} represents mean status of depression onset for all participants at first data collection; U_{0i} represents random error in B_{0i} for individual i; G_{01} represents mean linear change in probability for depression onset for all participants, G_{11} represents mean linear change in probability for depression onset as predicted by baseline levels of each risk factor and U_{1i} represents random error in B_{1i} for individual i.

To assess whether sex moderated the relation between T1 risk factors and future risk for onset of depression, HLM models were generated to assess the effect of level 2 time-invariant covariates of sex (0 or 1), T1 risk factor, and the interaction of sex and the T1 risk factor on the slope of the level 1 unconditional model of probability of depression onset over time:

$$\text{Prob}(Y=1|B)=P$$
$$\log[P/(1-P)]=B_{0i}+B_{1i} \times (\text{TIME}) \quad \text{Level 1}$$

$$B_0=G_{00}+U_{0i}$$
$$B_1=G_{10}+G_{11} \times (\text{SEX})+G_{12} \times (\text{RISK FACTOR})+G_{11} \times (\text{SEX} \times \text{RISK FACTOR})+U_{1i} \quad \text{Level 2}$$

In the event of a significant interaction, follow-up analyses of the simple effects of the T1 risk factor on risk for depression onset were conducted separately for boys and girls. To test whether the T1 gender additive risk factors predicted depression onset above and beyond the predictive value of the T1 shared risk factors, probability of depression onset was modeled over time at level 1 separately for girls and boys. T1 values of gender additive risk factors that prospectively predicted the outcomes were entered as level 2 time-invariant covariates along with the shared risk factors that predicted onset of depression in multivariate models.

Results

Preliminary Analyses

Of the original 428 participants, 15 did not provide data at T2 (3%), and 19 did not provide data at T3 (4%), although only ten participants did not provide data at both T2 and T3 (2%). Participants who dropped out of the study did not differ from those who provided complete data on any of the variables considered in this study at T1. Because HLM uses full-information maximum likelihood estimation for missing data, the effective N for analyses was 428. Independent t tests indicated that girls reported greater body dissatisfaction, peer social support, negative affectivity, and dietary restraint at T1 and boys reported greater

stressful life events, externalizing behaviors, and depressive symptoms at T1. Of the 428 participants, 33 met criteria for major or minor depression (24 girls, 9 boys) at T1. Among participants who were nondepressed at the initial assessment, an additional 13 participants met criteria for a depressive disorder by T2 (seven girls, six boys), and another 25 participants met criteria for a depressive disorder by T3 (20 girls, 5 boys). Means and standard deviations for all baseline variables, and the correlations among them, are provided in Table 1. Means and standard deviations for all baseline measures by sex are reported in Table 2.

Descriptive Analyses

The univariate relation between age and depressive symptoms was first investigated in an individual model to test whether depressive onset increased from age 13 to 16. As expected, increases in age were significantly associated with increases in depression onset (β=0.77, t=4.74, p<0.001), and results indicated that the cross-level interaction of age and sex increased the probability of depression onset, although it was only a trend, increasing the probability for girls more than for boys (β=−0.63, t=−1.92, p<0.06). Chi-square analyses compared the rate of depression onset for girls versus boys aggregated by age rather than by measurement periods at ages 13, 14, 15 and 16 to determine the age at which this sex difference emerged. Although there were no significant differences in onset at age 13 or 14, the sex difference was evident by age 15 for the current sample [χ^2 (1, N=411)=9.380, p=0.002] and at age 16 [χ^2 (1, N=217)=9.594, p=0.002]. Figure 1 illustrates the absolute number of cases of depression for girls and boys at each age represented in the study, across all measurement periods.

Prospective Analyses

Univariate HLM models examined the relation of stressful life events, negative affectivity, deficits in peer support, deficits in parental support, externalizing behaviors, body mass index, thin-ideal internalization, body dissatisfaction, dietary restraint, and eating pathology on future risk for depression onset. The following T1 risk factors were significant predictors of depression onset: negative affectivity (β=0.43, t=4.11, p<0.001, OR=1.54), deficits in parental social support (β=− 0.35, t=−4.66, p<0.001, OR=−1.42), externalizing behaviors (β=0.30, t=3.24, p<0.005 OR=1.35), body dissatisfaction (β=0.25, t=3.17, p<0.005, OR=1.28), dietary restraint (β=0.18, t=3.11, p<0.005, OR=1.20), and eating pathology (β=0.30, t=3.54, p<0.005, OR=1.35). Stressful life events did not significantly predict onset of depression, but results approached significance (β=0.07, t=1.82, p=0.07, OR=1.06). These models, as well as non-significant predictor models, are reported in Table 3.

Tests of Sex Moderation

We hypothesized that sex would moderate the relations of the gender additive, but not the shared, risk factors with risk for depression onset. To test whether sex moderated any of the univariate effects of the risk factors, T1 risk factors, sex, and the interaction of sex and all T1 risk factors were entered as level 2 variables to the models described above. Results indicated that the interaction of sex and T1 risk factors significantly predicted future depression onset for the following: body dissatisfaction (β=−0.40, t=−1.98, p<0.05, OR=1.49) and dietary restraint (β=−0.39, t=−2.79, p<0.01, OR=1.48). Post-hoc analyses revealed that body dissatisfaction and dietary restraint predicted onset of girls', but not boys', depression. The interaction of sex and all other risk factors failed to significantly predict depression onset for the coed sample.

Test of the Gender Additive Model

The baseline values of the T1 shared risk factors and the significant gender additive risk factors were included in separate models of depressive onset to test whether the gender additive risk factors showed incremental predictive utility for girls, above and beyond the shared risk factors. Deficits in peer social support, ideal-body internalization, and BMI were excluded because of non-significant univariate effects. Body dissatisfaction remained a significant predictor of future onset of depression for girls, even when stressful life events, negative affectivity, deficits in parental social support, and externalizing behaviors were included in the model ($\beta=-0.21$, $t=2.29$, $p<0.05$, OR=1.22). Dietary restraint and eating pathology did not demonstrate a predictive effect on future onset of depression for girls when the shared risk factors were included in the model. Results are reported in Table 4.

Discussion

The aims of this study were to test whether a set of body and eating related risk factors were stronger predictors of depression for girls than boys and to test whether the gender additive variables predicted depression for girls above and beyond the predictive value of empirically established risk factors. Body dissatisfaction and dietary restraint predicted depression for girls, but not for boys, and body dissatisfaction remained a strong predictor of girls' depression even when other risk factors were included in the model, providing some support for the gender additive model. The majority of risk factors consistently found to predict depression in prior research also predicted depression onset in the current study, but did not predict differentially for girls and boys. Each of these results, and their implications, will be discussed.

Consistent with previous findings, the present study demonstrated that depression increased from ages 13 to 16 (e.g., Ge et al. 1994; Hankin et al. 1998). Among adolescents who did not initially meet criteria for depression, 13 adolescents showed onset of major or minor depression by T2 (3%) and 25 adolescents showed onset of major or minor depression by T3 (6%). Age did not exert the same effect on both boys and girls in the sample, however. For girls, increases in age were associated with increased depressive onset, while for boys the reverse was true. This parallels the study by Ge et al. (2001) that documented girls' depressive symptoms increased from 7th to 12th grade while boys' depressive symptoms were highest in 7th grade. For the current sample, the difference in rates of depression between girls and boys became significant at age 15. The increases in depression overall, and for girls in particular, underscores the importance of identifying risk factors that give rise to this pernicious disorder.

Gender Additive Risk Factors

Two variables put forth by the gender additive model showed greater predictive effects for depression among girls than boys. As theorized, the relation of body dissatisfaction to future depression onset was significant for girls, but not boys. Because physical appearance is a central evaluative concern for girls in Western culture, feelings of displeasure with their physique may have a consequent impact on their mood. In comparison, boys may place more emphasis on other domains, such as sports ability or academic achievement (Hankin and Abramson 2001). While average-weight girls have been shown to experience high levels of body dissatisfaction, boys are more vulnerable to body dissatisfaction in response to being either under or overweight (Bearman et al. 2006; Presnell et al. 2004; Richards et al. 1990). Thus, boys and girls alike may encounter the physical transformations of adolescence, but with different consequences.

The interaction of dietary restraint and sex also significantly predicted future depression onset, emerging as a significant predictor of girls' future depression only. In theory, dieting may represent an attempt to alter one's size and shape in response to feelings of body dissatisfaction. However, because self-reported dietary restraint has been found to predict weight gain rather than weight loss, as well as bulimic symptoms such as bingeing and purging (Field et al. 1999; Stice et al. 2002), it may set in motion a cycle of disordered eating, increases in weight, and feelings of shame and guilt. Caution should be used when interpreting these latter findings, however. Individuals with elevated dietary restraint scores do not eat significantly fewer calories than those with lower scores when objective measures of intake are used (Bathalon et al. 2000; Stice et al. 2004c; Sysko et al. 2005), suggesting that these scales identify people with overeating tendencies who try, unsuccessfully, to curb these tendencies via dieting. Boys may be less likely to restrict in response to body dissatisfaction since they may strive to increase muscle mass, more effectively accomplished via increases in exercise (McCabe and Ricciardelli 2001). Exercise has been shown to have a positive impact on mood (Calfas and Taylor 1994), whereas the frustration associated with unsuccessful dieting may leave girls more susceptible to depression than boys.

Bulimic pathology predicted future depression onset equally for girls and boys in the current study, in contrast to hypotheses. According to the model put forth by McCarthy (1990), individuals may binge eat to provide comfort and distraction from adverse emotions. People may also use radical compensatory behaviors, such as fasting, to reduce anxiety about impending weight gain or because they believe it serves as an emotional catharsis. Girls are more likely to experience bulimic symptoms than boys (Ricciardelli and McCabe 2004); however, results of this study suggest that boys who do engage in bulimic behaviors likewise face an increased risk for depression, which is a novel finding.

In the current study, BMI did not predict future depression onset for girls or boys and this relation was not moderated by sex. The absence of this effect is noteworthy, given the well-documented prejudice directed at obese individuals and the grim health outcomes that are associated with obesity (Wadden et al. 2002). It may be that girls' impressions of their size and shape, rather than physical weight per se, are related to increases in depression. These findings suggest that sex differences in depression may stem from attitudinal, rather than physical, differences.

Ideal-body internalization did not predict future depression onset for either sex. Although ideal-body internalization has predicted increases in depressive symptoms among adolescent girls (Stice and Bearman 2001), to our knowledge this is the first study to test this relation among adolescent boys. One possibility for the null results is that thin-ideal internalization interacts with other variables to increase vulnerability to depression. Stice et al. (2002) found that very slender girls' overvaluation of appearance did not increase their risk for binge eating, while for their heavier counterparts it did; perhaps a similar interaction exists for the prediction of depression. It may be that the cost of "buying into" the importance of an ideal body is only relevant when an individual is deviant from this ideal.

Shared Risk Factors

Most established risk factors were also significant predictors of depression in the current study, with the exception of stressful life events and peer support deficits. Among the remaining variables—negative affectivity, deficits in parental social support, and externalizing behaviors—all were significant predictors, but sex failed to moderate the relation between these established risk factors and depression onset.

It was surprising that stressful life events did not significantly predict depression onset in the present study, because this has been a consistent predictor of depression in prior research

(Cohen et al. 1984; Goodyer et al. 2000; Lewinsohn et al. 1994). There is evidence that adolescents experience more negative life events than their younger counterparts (Larson and Ham 1993), which may clarify why depression increases during adolescence. It is possible that our sample, which had an average age of 13.6 at baseline assessment, had yet to experience the increase in stressful events that occurs during adolescence.

It is also worth noting that deficits in peer support did not predict onset of depression for either boys or girls. This null effect dovetails with results of Lewinsohn et al. (1994), where parental but not peer support deficits were a significant predictor of adolescent depression onset in a coed sample. Although adolescents spend an increasing amount of time with friends outside the home, there is evidence that family relationships retain their salience throughout adolescence and remain more reliable predictors of depressive symptoms than peer relationships (Gore et al. 1993; Stice et al. 2004d). Whereas adequate support from parental relationships is thought to promote feelings of self-esteem and efficacy that may buffer the impact of life stressors and negative mood, both girls and boys who lack this support appear to be at heightened risk for depression. Most previous studies have not separated perceived support from peers versus parents; our results suggest that there may be important differences in the impact of these two types of support on depression in adolescents.

Multivariate Models

The gender additive model also posits that the body-image and eating related risk factors will predict depression outcomes for girls above and beyond the effect of shared risk factors that predict depression. As anticipated, body dissatisfaction continued to be a significant predictor for onset of future depression among girls, even when stressful life events, negative affectivity, deficits in parental social support, and externalizing behaviors were included in the models. However, dietary restraint and eating pathology no longer remained significant when the shared risk factors were included. This suggests that body dissatisfaction may be the central risk factor in this theoretical model, exerting unique effects for girls beyond the predictive value of other risk factors that generally increase adolescents' risk for depression.

With the exception of body dissatisfaction, the variables from the gender additive model did not show unique effects when the shared risk factors were included in the multivariate models of girls' future depression onset. The reverse was also true: when body dissatisfaction, dietary restraint, and eating pathology were included in the multivariate models with the shared risk factors, only deficits in parental social support consistently remained a robust predictor of girls' depression onset; the effects for stressful life events, negative affectivity and externalizing symptoms became non-significant. It is possible that the null effects for the majority of the risk factors is an indication that mediational relations exist among many of the risk factors, in which case one would not expect to see unique predictive effects since mediators account for the variance of the independent variable on the dependent variable. For example, it has been suggested that individuals exhibit dietary and eating disordered behaviors in response to negative affectivity, but that these behaviors in turn increase feelings of shame, guilt, and low self-worth (Stice et al. 2000). Thus, "downstream" variables in the model failed to demonstrate unique predictive effects when other implicated variables were included.

Ultimately, a revised version of the gender additive model should reflect the results of the current study by highlighting the importance of body dissatisfaction as a risk factor that uniquely interacts with sex to predict depression for adolescent girls, but not for boys, even when the impact of other significant risk factors has been partialed from the model. The current study is the third in a series (Stice et al. 2000; Stice and Bearman 2001) to suggest

that body dissatisfaction, dietary restraint, and eating pathology increase risk for depression for adolescent girls, and the first to demonstrate that body dissatisfaction and dietary restraint exert unique risk for adolescent girls relative to boys. Given that well-established risk factors appear unable to account for the troubling sex differences in adolescent depression that continue throughout adulthood, the promising support for the potent role of body dissatisfaction, and to a lesser extent, dietary restraint, suggest it would be advantageous to continue to conduct prospective studies to investigate the processes that lead to the sex differences in depression. We believe that it would be useful for studies to test whether sex moderates the predictive effects of other theorized risk factors, in order to provide a direct test of whether the predictive effects are stronger for adolescent girls versus boys. Nolen-Hoeksema and Girgus (1994) have asserted that girls enter adolescence with a ruminative style of responding to stressors that is less efficacious than boys' more active coping style, and that this interacts with stressors to place girls at greater risk for depression (Nolen-Hoeksema 1994). Although there is evidence that girls may be more likely to ruminate than boys (Broderick 1998; Schwartz and Koenig 1996), the interaction of sex and rumination has not been tested in a sample of adolescents. Because this is a central component of this model, it will be important to test this relation.

Although we attempted to improve upon previous studies by using a prospective design, diagnostic interviews, direct assessment of body mass, and direct tests of whether sex moderated predictive effects, this study had limitations. First, our moderate recruitment rate and limited ethnic diversity of our sample suggest that care should be taken in the generalization of our findings. Second, the reliance on self-report data—particularly the exclusion of both parent and teacher report—implies that shared reporter bias may inflate the magnitude of effects (although this would make it more difficult to detect the interactive and unique effects that were the focus of the present analyses). Third, we were not able to include all of the empirically established risk factors for depression (e.g., competence, coping styles, and parental depression), or all of the candidate variables suggested by other models of the sex differences in depression (e.g., emotion regulation, rumination, sex role socialization) because of respondent burden concerns. Furthermore, many of the risk factors assessed in this study (e.g., body dissatisfaction and ideal-body internalization) were assessed using measures developed for females. Some of these forms have been revised to make them more sensitive for use with males, but have not yet been tested in this population —suggesting a possible measurement bias. Finally, whereas longitudinal data provide information regarding temporal precedence, third-variable explanations cannot be ruled out with a non-experimental design. Therefore, it is possible that some shared causal variable increases both the risk factors and depression.

With regard to future directions, a study that incorporates assessment of parents and friends would be useful, in particular to disentangle constructs that are necessarily subjective, such as number of stressful life events or parental or peer support. Furthermore, future studies should revisit the possibility that the sex differences in depression result in part from the qualitatively different hormonal experience of adolescent girls relative to adolescent boys and whether hormonal factors interact with other risk factors, such as negative affectivity. Research that examines body image and eating related factors that may have more salience for males—such as exercise or steroid use—would also be useful for clarifying the role these factors may play in the development of depression for boys. Continuing to explore eating and body image variables in coed samples will also require ongoing development of measures that consider the differences and similarities of boys and girls. Results of the current study suggest that peer support deficits should be considered separately from parental support deficits in future examinations of the onset of adolescent depression, in order to determine what role, if any, peer support deficits play in predicting adolescent depression. Finally, as it is likely that risk factors are interrelated, a study that examined

mediational pathways based on the proposed etiologic models would clarify how these variables interact to promote depression.

The results of the present study also offer several clinical implications. First, this study identified a number of malleable risk factors for adolescent depression that are not typically included in extant depression prevention programs, including deficits in parental support, externalizing behaviors, body dissatisfaction, dietary restraint, and eating pathology. Consistent with the suggestion that prevention programs that reduce body image and eating disturbances may impact depressive symptoms, we have found that interventions that decrease these variables also reduced negative affect in randomized trials (Bearman et al. 2003; Stice et al. 2006). Second, this study also identified risk factors that increase boys' and girls' probability of becoming depressed, which may help identify novel high-risk subgroups for targeted and indicated prevention programs. Research suggests that preventive interventions for depression are typically stronger for high-risk youth (Horowitz and Garber 2006). Finally, the current study also adds to the wealth of evidence that girls are at higher risk for depression during adolescence than boys and identified potentially influential variables that may play a role in this sex difference. Thus, girls should be a particular focus of preventive efforts for depressive disorders, with an emphasis on increasing self and body esteem, and encouraging beneficial weight management behaviors.

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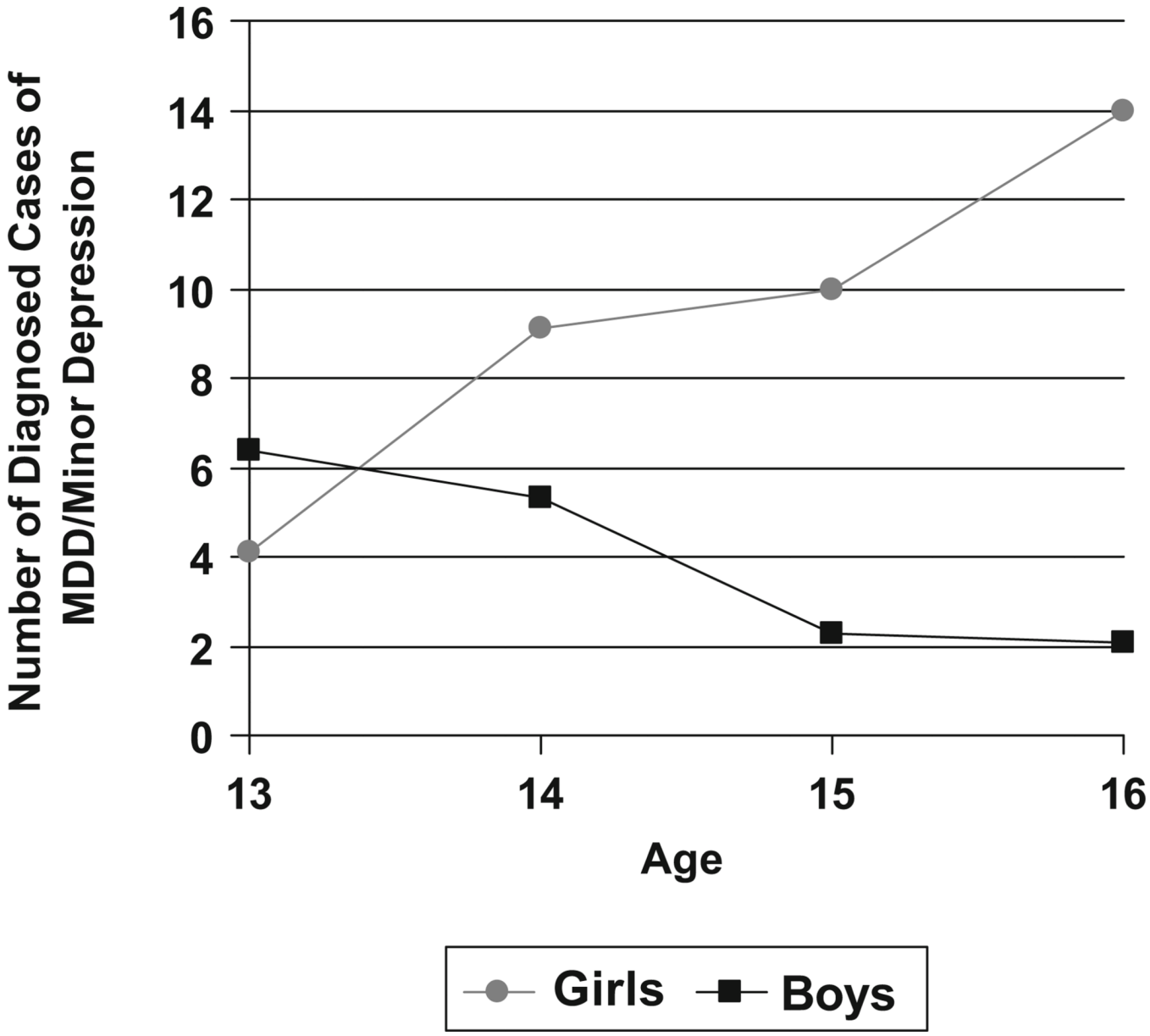

Fig. 1.
Absolute numbers of diagnosed cases of major depressive disorder (MDD) and minor depression at each age, for girls and boys

Table 1

Correlations among the T1 Putative Risk Factors and T1 Depression, along with Means and Standard Deviation for Boys and Girls

	Negative affectivity	Support-parent	Support-peer	Externalizing	Body mass index	Ideal-body internalization	Body dissatisfaction	Dietary restraint	Eating pathology	Depression diagnosis	Sex	Age	*M*	SD
Stressful life events	0.23	−0.25	−0.06	0.44	0.06	0.08	0.11	0.07	0.10	0.22	0.23	0.03	1.40	1.43
Negative affectivity		−0.19	−0.02	0.48	0.09	0.12	0.32	0.25	0.26	0.31	−0.18	−0.08	2.69	0.61
Support-parent			0.10	−0.39	−0.06	0.01	−0.32	−0.08	−0.25	−0.29	0.05	−0.01	3.96	0.84
Support-peer				−0.08	−0.04	−0.00	−0.10	−0.08	−0.03	−0.11	−0.29	0.05	4.16	0.77
Externalizing					0.10	0.13	0.10	0.14	0.19	0.30	0.17	0.06	1.77	0.61
Body mass index						−0.06	0.30	0.42	0.29	−0.03	0.02	0.07	21.66	4.69
Ideal-body internalization							0.11	0.16	0.17	0.08	0.00	0.00	3.22	0.75
Body dissatisfaction								0.38	0.42	0.13	−0.11	−0.02	2.66	0.88
Dietary restraint									0.38	0.02	−0.15	0.01	2.02	0.87
Eating pathology										0.20	−0.09	0.08	0.32	0.50
Depression diagnosis											−0.09	0.09	0.78	0.27
Sex												0.09	0.43	0.50
Age													13.56	0.55

Absolute correlations greater than 0.09 are significant at $p<0.05$.

Table 2

Means and Standard Deviation for each T1 Variable by Sex

	Girls		Boys	
Variable	***M***	**SD**	***M***	**SD**
Stressful life events	1.12_a	1.22	1.77_b	1.59
Negative affectivity	2.78_a	0.60	2.57_b	0.59
Support-parent	3.92	0.90	4.00	0.74
Support-peer	4.35_a	0.72	3.91_b	0.75
Externalizing	1.68_a	0.61	1.88_b	0.61
Body mass index	21.57	4.74	21.78	4.64
Ideal-body internalization	3.22	0.67	3.22	0.84
Body dissatisfaction	2.74_a	0.95	2.55_b	0.76
Dietary restraint	2.13_a	0.91	1.87_b	0.80
Eating pathology	0.35	0.53	0.27	0.44
Depression diagnosis	0.10	0.30	0.05	0.22
Age	13.52	0.53	13.62	0.57

Means having different subscripts are significantly different at $p<0.05$.

Table 3

Univariate Relations of each Risk Factor to Adolescent Boys' and Girls' Depressive Onset

Effect	Parameter	Coefficient	se	*t*	*p* value
Stressful life events	β_0	−2.44	0.105	−23.18	<0.001
	β_1	0.07	0.036	1.82	<0.070
Negative affectivity	β_0	−2.45	0.110	−22.19	<0.001
	β_1	0.43	0.106	4.11	<0.001
Parental social support	β_0	−2.45	0.110	−22.33	<0.001
	β_1	−0.35	0.074	−4.66	<0.001
Peer social support	β_0	−2.43	0.103	−23.46	<0.001
	β_1	−0.06	0.081	−0.80	ns
Externalizing behaviors	β_0	−2.44	0.108	−22.73	<0.001
	β_1	0.30	0.092	3.24	<0.005
Body-mass index	β_0	−2.43	0.103	−23.63	<0.001
	β_1	0.01	0.012	0.97	ns
Thin-ideal internalization	β_0	−2.43	0.103	−23.46	<0.001
	β_1	0.08	0.075	1.11	ns
Body dissatisfaction	β_0	−2.44	0.106	−23.04	<0.001
	β_1	0.25	0.080	3.17	<0.005
Dietary restraint	β_0	−2.42	0.103	−23.47	<0.001
	β_1	0.18	0.058	3.11	<0.005
Bulimic pathology	β_0	−2.45	0.107	−22.81	<0.001
	β_1	0.30	0.085	3.54	<0.005

Table 4

Multivariate Models of Sex Additive and Shared Risk Factors: Girls' Depression Onset

Effect	Parameter	Coefficient	se	*t*	*p* value
Depression	B_0	−2.27	0.141	−16.05	<0.001
Body dissatisfaction	G_1	0.21	0.091	2.29	<0.05
Stressful life events	G_2	−0.04	0.074	−0.52	ns
Negative affectivity	G_3	−0.01	0.179	−0.08	ns
Externalizing behavior	G_4	0.35	0.166	2.12	<0.05
Parental social support	G_5	−0.21	0.090	−2.39	<0.05
Depression	B_0	−2.27	0.141	−16.13	<0.001
Dietary restraint	G_1	0.13	0.085	1.55	ns
Stressful life events	G_2	−0.03	0.068	−0.43	ns
Negative affectivity	G_3	0.07	0.174	0.42	ns
Externalizing behavior	G_4	0.21	0.159	1.33	ns
Parental social support	G_5	−0.28	0.088	−3.24	<0.005
Depression	B_0	−2.27	0.141	−16.12	<0.001
Eating disorder pathology	G_1	0.07	0.124	0.56	ns
Stressful life events	G_2	−0.03	0.070	−0.41	ns
Negative affectivity	G_3	0.11	0.172	0.62	ns
Externalizing behavior	G_4	0.23	0.154	1.51	ns
Parental social support	G_5	−0.28	0.086	−3.30	<0.005

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