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Prospective Relations between Bulimic Pathology, Depression, and Substance Abuse: Unpacking Comorbidity in Adolescent Girls

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Abstract

To elucidate the processes that contribute to the comorbidity between bulimic pathology, depression, and substance abuse, we tested the temporal relations between these disturbances with prospective data from adolescent girls (N = 496). Multivariate analyses indicated that depressive symptoms predicted onset of bulimic pathology, but not substance abuse, bulimic symptoms predicted onset of depression, but not substance abuse, and substance abuse symptoms predicted onset of depression, but not bulimic pathology. Results suggest that the comorbidity arises because certain disorders are risk factors for the other disorders. Findings also provide support for select etiologic theories and further establish the clinical significance of these conditions by showing that they increase risk for onset of other psychiatric disturbances.

Prospective Relations between Bulimic Pathology, Depression, and Substance Abuse: Unpacking Comorbidity in Adolescent Girls

Community-recruited studies have consistently indicated that bulimic pathology is associated with significant elevations in major depression and substance abuse during adolescence (Johnson, Cohen, Kasen, & Brook, 2002;Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993;Newman et al., 1996;Stice, Presnell, & Bearman, 2001) and adulthood (Garfinkel et al., 1995;Kessler et al., 1994). For example, among individuals with bulimia nervosa 20% meet criteria for major depression and 4% meet criteria for alcohol dependence, relative to 2% and 1%, respectively, among individuals without bulimia nervosa (Garfinkel et al., 1995). Given that the comorbidity between bulimia nervosa, depression, and substance abuse is clearly established, the next logical step is to investigate the temporal sequencing of the onset of these disorders with prospective data to illuminate the processes that give rise to this comorbidity (Wolfe & Maisto, 2000). As noted by Glantz (2002), surprisingly little is known regarding the temporal order of the development of these psychiatric conditions.

There are three general processes that could give rise to such comorbidity. One disorder may be a risk factor for the other comorbid disorder (unidirectional effect). A risk factor is a variable that has been shown to prospectively predict onset of a pathological condition among initially non-disordered individuals (Kreamer et al., 1997). Alternatively, each disorder could increase the risk for onset of the other disorder (reciprocal effects). It is also possible that elevated co-occurrence of two disorders occurs because they share common risk factors (Kraemer, Stice,

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Kazdin, & Kupfer, 2001). If this were the case, one might observe elevated comorbidity between the disorders (correlation), but no reliable evidence of temporal precedence between the conditions. It is important to investigate these alternative explanations of elevated comorbidity because each has distinct etiological, prevention, and treatment implications. For instance, if substance abuse is a risk factor for depression, but not vice versa, prevention programs would need to target the former in order to effectively reduce risk for both conditions. Accordingly, we tested six hypotheses concerning the nature of the temporal relations between these three classes of psychopathology in an effort to elucidate the processes that produce the observed comorbidity.

Bulimia Nervosa and Depression

Numerous theorists have hypothesized that depression increases the risk for development of bulimic pathology (Leon, Fulkerson, Perry, & Early-Zald, 1995;McCarthy, 1990). The negative affect model posits that depressed individuals binge eat because they believe it provides comfort and distraction from negative emotions. Individuals might also use radical compensatory behaviors, such as vomiting, to reduce anxiety about impending weight gain consequent to overeating or because they believe that purging serves as an emotional catharsis. In support of this assertion, negative affect and temperamental emotionality have been found to predict future onset of bulimic pathology (Killen et al., 1996;Stice & Agras, 1998) and increases in bulimic symptoms (Cooley & Toray, 2001;Stice, 2001). Depressive symptoms have also been found to predict onset of binge eating (Stice, Presnell, & Spangler, 2002) and general eating pathology (Johnson, Cohen, Kotler, Kasen, & Brook, 2002). In contrast, temperamental emotionality, negative affect, and depressive symptoms have not been found to predict future increases in general eating pathology in other studies (Attie & Brooks-Gunn, 1989;Keel, Fulkerson, & Leon, 1997;Leon et al., 1995;Leon, Fulkerson, Perry, Keel, & Klump, 1999). However, we were unable to locate a previous study that tested whether depressive symptoms predicted onset of bulimic pathology.

Conversely, it has been posited that bulimic pathology increases the risk for depression (Heatherton & Polivy, 1992;Stice, Hayward, Cameron, Killen, & Taylor, 2000). Theoretically, binge eating and purging foster feelings of shame, guilt, and dysphoria, which increase the risk for depression onset. It has also been suggested that caloric deprivation leads to tryptophan depletion (a precursor to serotonin), which may contribute to mood disturbances (Kaye, Gendall, & Strober, 1998). Consistent with hypotheses, bulimic symptoms have been found to predict future increases in depressive symptoms (Stice & Bearman, 2001) and onset of major depression (Stice et al., 2000).

Collectively, previous theory and prospective findings suggest that depression and bulimic pathology may be reciprocally related. Accordingly, we tested the *a priori* hypotheses that initial depressive symptoms will increase the risk for onset of bulimic pathology and that initial bulimic symptoms will increase the risk for onset of depressive pathology.

Depression and Substance Abuse

Theorists have also proposed that depression is a risk factor for the development of substance abuse (Newcomb & Bentler, 1988). Depressed individuals theoretically consume psychoactive substances to improve their mood and to provide distraction from adverse emotions. The functional nature of this substance use putatively results in frequent consumption, thereby increasing the risk of the use-related negative consequences that characterize substance abuse. The fact that depression increases markedly during adolescence for girls (Hankin, Abramson, Moffitt, Silva, McGee, & Angell, 1998) suggests that depressive symptoms might be a particularly strong predictor of substance abuse for this population. Surprisingly, only a few studies have prospectively tested whether affective disturbances predict onset of substance

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abuse or increases in substance abuse symptoms. One study found that a measure of internalizing pathology (reflecting both depressive and anxious symptoms) predicted future increases in alcohol abuse symptoms in a univariate model (Stice, Barrera, & Chassin, 1998). However, studies that estimated multivariate models found that depressive symptoms did not predict onset of problem alcohol use (Costa, Jessor, & Turbin, 1999) and that depressive pathology did not predict onset of alcohol abuse symptoms (Clark, Parker, & Lynch, 1999). This pattern of findings may suggest that only broadband measures of affective disturbances are related to substance abuse onset or that this effect is so small that it easily becomes non-significant when examined within the context of a multivariate model. This conclusion should be considered tentative because we were unable to locate any prospective research that tested whether depressive symptoms predicted onset of substance abuse.

Researchers have also theorized that substance abuse increases the risk for development of depression (Brook, Cohen, & Brook, 1998; Schuckit, 1994). Repeated administration of psychoactive substances may cause structural or functional alterations in neurological functioning that contribute to the development of depression. For example, recurrent use of substances may cause receptor down-regulation in the mesolimbic dopamine pathways of the reward system, resulting in attenuated positive affect. Recurrent negative consequences from substance use might also increase the risk for depression because they would be expected to contribute to dysphoric mood. In support of these assertions, substance abuse has been found to predict depression onset in adulthood (Rao, Daley, & Hammen, 2000;Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001). However, we could not locate any research that prospectively tested this relation during adolescence.

Collectively, past theory and prospective findings tentatively suggest that depression and substance abuse may also be reciprocally related. Therefore, we tested the *a priori* hypotheses that initial depressive symptoms will increase the risk for future onset of substance abuse and that initial substance abuse symptoms will increase the risk for onset of depressive pathology.

Bulimia Nervosa and Substance Abuse

Although less theoretical attention has been directed at the relation between bulimic pathology and substance abuse, there are several possible mechanisms by which bulimic pathology might increase the risk for substance abuse. First, the strong drive for thinness that typifies bulimic pathology may increase the risk for recurrent use of stimulant drugs for the purpose of weight loss, which would increase the risk for the use-related negative consequences. Stimulants activate the sympathetic nervous system, which results in less efficient routes of caloric storage, decreased rates of storage, and increased caloric utilization and excretion (because they increase metabolic rate and thermogenesis; Wack & Rodin, 1982). Second, binge eating and compensatory behaviors are thought to promote feelings of shame and guilt (Stice, et al., 2000), which might promote attempts to regulate affect through substance use. Third, deficits in impulse control for appetitive behaviors may constitute a general risk factor for the overconsumption of food and psychoactive substances (Wonderlich & Mitchell, 2001). A proclivity toward overconsumption might also be rooted in a dysregulation of the reward neural circuitry, given that both eating and substance use trigger this network (Robbins & Everitt, 1999). The fact that bulimic pathology increases markedly during adolescence for girls (Stice, Killen, Hayward, & Taylor, 1998) suggests that this factor might also be a particularly strong predictor of substance abuse for girls. Although we were unable to locate any prospective tests of whether bulimic pathology predicted substance abuse onset, there is indirect support for this hypothesized relation. Retrospective data suggests that binge eating tends to emerge earlier than substance abuse among individuals exhibiting both behaviors (Wilson, 1993). Eating pathology has also been found to increase the risk for onset of one form of substance abuse – cigarette smoking (French, Perry, Leon, & Fulkerson, 1994;Stice & Shaw, 2003).

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Little theoretical consideration has been given to the possibility that substance abuse increases the risk for bulimic pathology. The dietary restraint model (Polivy & Herman, 1985) posits that factors resulting in a relaxation of cognitive control over eating, such as intoxication, increase the risk for disinhibited eating that spirals into binge eating episodes. That is, dieters who use psychoactive substances may be at increased risk for onset of bulimic pathology. The theory that deficits in impulse control for appetitive behaviors increase the risk for overconsumption of food and psychoactive substances also suggests that there may be a prospective effect from initial substance abuse to risk for subsequent onset of bulimic pathology. We only located one study that prospectively investigated this relation. Johnson, Cohen, Kotler, and associates (2002) found no significant relation between substance abuse during early adolescence and risk for onset of eating pathology during late adolescence or adulthood. Retrospective data (Wilson, 1993) similarly suggest that the effect from substance abuse to onset of bulimic pathology may be weaker than the reverse relation between these constructs.

Past theory provides some support for the possibility that bulimic pathology and substance abuse are reciprocally related, but past findings suggest that there is no relation between substance abuse and future onset of bulimic pathology. Nonetheless we thought it useful to provide an exploratory test of these relations because empirical findings can provide an important impetus to future theoretical development (Tukey, 1977).

Overview of Study

In sum, the goal of this study was to elucidate the processes that may produce the observed comorbidity between bulimic pathology, depression, and substance abuse during adolescence by investigating the temporal relations between these three psychiatric conditions. Specifically, we tested whether initial symptoms of each disorder constituted a risk factor for subsequent onset of the other disorders with prospective data. To our knowledge, this is the first prospective investigation of the temporal relations between these three psychiatric constructs. Such analyses are important because they may suggest that the comorbidity between two disorders arises solely because one disorder is a risk factor for the other (unidirectional effect) or because each disorder is a risk factor for the other (reciprocal effects). More generally, these analyses provided tests for several etiologic theories concerning the risk factors for bulimic pathology, depression, and substance abuse. The results should also inform prevention and treatment efforts for these conditions by identifying factors that might be targeted in interventions and potential screening factors that might be used to generate high-risk samples for selected prevention programs. Finally, results might provide further evidence of the clinical significance of these disturbances by showing that they increase the risk for future onset of other psychiatric disorders.

These aims were addressed with data from a community-recruited longitudinal study of adolescent girls because this developmental period is a high-risk time for onset of substance abuse (Langerbucher & Chung, 1995), depression (Hankin et al., 1998), and eating pathology (Stice, Killen et al., 1998), and because bulimic pathology is rare among males.

Methods

Participants

Participants were 496 adolescent girls from four public (82%) and four private (18%) middle schools in a metropolitan area of the Southwestern United States. Adolescents ranged in age from 11 to 15 (M = 13) at baseline. The sample included 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Latina, 1% Native Americans, and 4% who specified *other or mixed* racial heritage. The ethnic composition of the sample was

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representative of the ethnic composition of the schools within the metropolitan area from which we sampled (2% Asian/Pacific Islanders; 8% African Americans, 65% Caucasians, 21% Hispanics; 4% *other or mixed*). Average parental education was as follows: 29% high school graduate or less; 23% some college; 33% college graduate; 15% graduate degree. Average educational attainment of parents (a proxy for socioeconomic status) was similar to census data for comparably aged adults in the metropolitan area from which we sampled (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree).

Procedures

The study was described to parents and participants as an investigation of adolescent mental and physical health. An active parental consent procedure was used to recruit participants. First, an informed consent letter describing the study and a stamped self-addressed return envelope were sent to parents of eligible girls. A second mailing was sent to non-responders after 2 weeks. Adolescent assent was secured immediately before data collection took place. This resulted in an average participation rate of 56% across schools. This participation rate was similar to that of other school-recruited samples that used active consent procedures and involved structured interviews (e.g., 61% for Lewinsohn et al., 1993). Moreover, the one-year prevalence rates of major depression (4.2%), bulimia nervosa (0.4%) and substance abuse (8.9%; Stice et al., 2001) were similar to the prevalence rates from other epidemiological studies (Lewinsohn et al., 1993;Newman et al., 1996).

Girls completed a questionnaire, participated in a structured interview, and had their weight and height measured by female research assistants at baseline (T1) and at one and two year follow-ups (T2 & T3). Female assessors with a bachelor, masters, or doctoral degree in psychology conducted all interviews. Assessors attended 24 hours of training, wherein they learned interview skills, reviewed diagnostic criteria for relevant DSM-IV disorders, observed simulated interviews, and role-played interviews. Assessors had to demonstrate an interrater agreement for diagnoses (kappa [κ] > .80) with experts using tape-recorded interviews before collecting data. Interviewers were recorded periodically throughout the study to ensure that assessors continued to demonstrate acceptable inter-rater agreement (κ > .80) with other clinical assessors. Assessments took place during regular school hours or immediately after school on the school campus or at participants' houses. Girls received a $15 gift certificate to a local book and music store to compensate them for participating in the study.

Measures

Bulimic pathology.—The Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a structured psychiatric interview, assessed diagnostic criteria for DSM-IV bulimia nervosa. Girls were diagnosed with past year threshold or subthreshold diagnostic criteria for bulimia nervosa at each of the three assessments. Girls who reported the presence of all symptoms of bulimia nervosa, but who endorsed a subthreshold level on at least one symptom, were given a subthreshold diagnosis (e.g., binge eating only once per week). We included a focus on subthreshold bulimic pathology because subdiagnostic eating pathology is associated with subjective distress and functional impairment (Lewinsohn, Streigel-Moore, & Seeley, 2000) and because the majority of adolescents seeking treatment for eating pathology do not meet full threshold diagnostic criteria for DSM-IV eating disorders (Fisher, Schneider, Burns, Symons, & Mandel, 2001). Items were also averaged to form an overall T1 bulimic symptom composite. Because the symptom composite was skewed, a square root transformation was used to normalize the distribution. The EDE has good internal consistency (α = .76 – .90) and inter-rater reliability (κ = .70 – .99), and discriminates between eating disordered individuals and controls (Fairburn & Cooper, 1993;Williamson, Anderson, Jackman, & Jackson, 1995). A randomly selected subset of girls (5%) completed a second diagnostic interview with an assessor who was blinded to the initial diagnosis, resulting in high inter-rater agreement (κ = .

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88). Another randomly selected subset of participants (5%) completed a second diagnostic interview with the same clinical assessor one week later, resulting in high test-retest reliability ($\kappa = 1.0$). The symptom composite had an $\alpha = .92$ at T1.

Depressive symptoms.—An adapted version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich & Chambers, 1983), a structured psychiatric interview, assessed diagnostic criteria for DSM-IV major depression. Girls were diagnosed with past year threshold or subthreshold major depression at each of the three assessments. We included a focus on subthreshold depression because it is associated with current and future subjective distress and functional impairment (Gotlib, Lewinsoh, & Seeley, 1995; Nolen-Hoeksema, Girgus, & Seligman, 1992). Girls who reported the presence of at least five of the required symptoms for major depression, but who endorsed a subthreshold level on at least one symptom, were given a subthreshold diagnosis (e.g., reporting a period of dysphoric mood that was shorter in duration than two weeks). Severity ratings for each symptom were averaged to form a T1 overall depressive symptom composite. The K-SADS has good test-retest reliability ($\kappa = .63 – 1.00$), interrater reliability ($\kappa = .73 – 1.00$) and internal consistency ($\alpha = .68 – .84$), and discriminates between depressed and non-depressed individuals (Ambrosini, 2000;Lewinsohn et al., 1993). The inter-rater agreement and 1-week test-retest reliability for the K-SADS diagnoses, for randomly selected subsets of participants (5% each), was excellent ($\kappa = 1.0$ for both). The symptom composite had an $\alpha = .85$ at T1.

Substance abuse.—Items adapted from Stice, Barrera et al. (1998) assessed DSM-IV substance abuse symptoms over the past year.[1] These items were specifically developed to assess substance use related negative consequences in adolescents. For example, items assessing obligation impairment inquired about negative consequences in both the school and work environment. Items focused on obligation impairment, health problems, physically hazardous behavior, legal problems, and social difficulties resulting from substance use (e.g., got arrested because of substance use, had an accident or injury because of substance use, lost a job or got kicked out of school because of substance use). Following DSM-IV criteria, girls reporting recurrent instances (at least two) of any of the symptoms were diagnosed with substance abuse. Separate diagnoses were made at each of the three assessments. Items were also averaged to create a T1 substance abuse symptom composite. Because this composite was skewed, a $\log_{10}$ transformation was used to normalize the distribution. These items possess adequate internal consistency ($\alpha = .85$) and convergent validity (Stice, Barrera et al., 1998). Pilot testing ($\underline{N} = 62$) revealed 1-month test-retest coefficient of .78 for the substance abuse symptom composite. More generally, self-reports of substance use appear to be the most valid measure of substance abuse (Winters, Stinchfield, Henly, & Schwartz, 1991).

Results

Preliminary Analyses

Of the initial 496 girls, 10 (2%) did not provide data at T2 and 10 (2%) did not provide data at T3, although only 4 did not provide data at either T2 or T3 (1%). Analyses verified that girls who dropped from the study did not differ significantly from the remaining girls in terms of demographic factors, school type (public vs. private), or bulimic, depressive, and substance abuse symptoms ($\underline{p}$-values > .10), suggesting that attrition should not bias parameter estimates.

[1]Two considerations led us to measure substance abuse symptoms for alcohol or illicit drug use in a single composite. First, research suggests that 86% to 90% of adolescent substance abusers meet abuse criteria for two or more substances (Brown, D'Amico, McCarthy, & Tapert, 2001;Maisto et al., 2001). Second, we felt that asking the adolescents to complete the substance abuse items separately for each type of substance represented too great of a respondent burden.

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Descriptive Statistics

Eight (1.6%) of the 496 adolescents met criteria for subthreshold or threshold bulimia nervosa at T1 (corresponding rates were 2.4% at T2 and 1.8% at T3). Of the 488 initial non-bulimic girls at T1, 16 (3.2%) showed onset of subthreshold or threshold bulimic pathology by T2 or T3 (4 of the girls dropped out of the study before completing the eating disorder items at T2 or T3). The fact that only 3 of the 10 girls who showed onset of bulimic pathology at T2 also met diagnostic criteria at T3 suggested that this eating pathology was episodic during this developmental period.

At T1, 40 (8.1%) of the 496 adolescents met criteria for subthreshold or threshold major depression (corresponding rates were 9.7% at T2 and 6.5% at T3). Of the 456 initial non-depressed girls at T1, 51 (11%) showed onset of subthreshold or threshold major depression by T2 or T3 (4 girls dropped out of the study before completing the depression items at T2 or T3). Only 7 of the 32 girls who showed onset of depressive pathology at T2 also met criteria at T3, which suggests that this affective pathology was also episodic.

At T1, 32 (6.5%) of the 496 adolescents met criteria for substance abuse (3 did not complete the substance abuse items at T1). The corresponding rates of substance abuse were 6.7% at T2 and 8.9% at T3. Of the 461 non-substance abusing girls at T1, 39 (8%) showed onset of substance use by T2 or T3 (3 girls dropped before completing the substance abuse items at T2 or T3). Ten out of the 22 girls who showed onset of substance abuse at T2 also met criteria at T3, suggesting that this pathology was more chronic relative to the other disturbances.

The correlations between the bulimic, depressive, and substance abuse symptom composites at each of the three assessments, along with the overall means, standard deviations, and skew coefficients, are presented in Table 1.

Predicting onset of Bulimic Pathology, Depressive Pathology, and Substance Abuse

We first estimated univariate logistic regression models that tested whether each T1 symptom dimension predicted onset of each psychopathology at T2 or T3. At this step, we verified the assumption of linearity in the logit function. Following Hosmer and Lemeshow (2000), we transformed each variable into a 4-level categorical variable (based on the quartiles) and re-estimated the model with the categorical variable (with 3 design variables, wherein the first quartile serves as the reference group). We then plotted the estimated coefficients versus the midpoint for each group (the first group is plotted at zero) to determine whether the coefficients approximated a linear form. We also verified that there were no significant effects for demographic factors (age, ethnicity, and parental education), which would have necessitated the inclusion of these factors as covariates. Next, we estimated multivariate models that examined the unique effects of each of the symptom dimensions while controlling for the effects of the other symptom dimensions. Because two of the six hypotheses that we tested were exploratory, we used a more conservative alpha level to decrease the risk of chance findings (.01).

Univariate logistic regression models tested whether depressive and substance abuse symptoms predicted bulimic pathology onset among initially non-bulimic girls, whether bulimic and substance abuse symptoms predicted depression onset among initially non-depressed girls, and whether depressive and bulimic symptoms predicted substance abuse onset among initially non-substance abusing girls. All predictors were assessed at T1 to ensure a prospective test of relations. Predictors were transformed to standardized scores to facilitate direct comparisons between the odds ratios. The odds ratios, 95% confidence intervals, and significance levels for the univariate relations between the T1 predictors and the risk for onset of bulimic pathology, depression, and substance abuse are presented in Table 2. Initial elevations in depressive

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symptoms, but not substance abuse symptoms, predicted onset of bulimic pathology during the two-year follow-up period.[2] Initial elevations in bulimic symptoms and substance abuse symptoms predicted onset of depressive pathology. Finally, initial elevations in depressive and bulimic symptoms predicted onset of substance abuse. The significant effects were small to medium in magnitude according to Cohen's criteria (1988).

Multivariate logistic regression models tested whether each of the symptom dimensions showed significant unique effects while controlling for the effects of the other symptom dimensions. These analyses were given more interpretational weight because they examine the unique effects of the predictors, while statistically controlling for the effects of the other predictors. The odds ratios, 95% confidence intervals, and significance levels for the multivariate relations between the T1 predictors and the risk for onset of bulimic pathology, depressive pathology, and substance abuse are presented in Table 3. In the multivariate model, depressive symptoms showed a significant unique relation to subsequent onset of bulimic pathology, but the effect for substance abuse symptoms remained non-significant. Both bulimic symptoms and substance abuse symptoms showed significant unique relations to the onset of depressive pathology in the multivariate models. Finally, both the effects of depressive symptoms and bulimic symptoms on risk for onset of substance abuse became non-significant in the multivariate model.[3] The significant unique effects ranged in magnitude from small to medium according to Cohen's criteria (1988).

Predicting Increases in Bulimic, Depressive, and Substance Abuse Symptoms

Because of concerns that reporter bias inflated the magnitude of the prospective effects, we estimated a series of models that more stringently controlled for the influence of reporter bias. We tested whether initial symptoms of each disorder predicted increases in the symptoms for each other disorder, while controlling for the correlation between initial levels of the two symptom dimensions – which would include any shared reporter bias. In such models, reporter bias would manifest itself in the initial correlation between symptom dimensions, and this effect is statistically controlled when testing for prospective effects.[4] Although these models do not allow inferences about the risk factors for onset of clinically meaningful psychopathology, they are useful for assessing the possibility that reporter-bias inflated the effect sizes.

We used random regression growth curve models for this purpose, rather than auto-regressive models, because the former model change in a more reliable fashion and accommodate missing data (Rogosa, Brandt, & Zimowski, 1982). We generated individual slope and intercept parameters for each participant for bulimic, depressive, and substance abuse symptoms. The slopes represent the average linear change (growth) in the construct across each of the 1-year intervals for each participant. The intercept parameters represent the value of the growth trajectory for each participant at T1. We then tested whether each T1 symptom dimension predicted growth in each other symptom domain controlling for initial levels of that symptom (the intercept for the outcome variable). The standardized regression coefficients (β), unstandardized regression coefficients (B), the 95% confidence interval, and significance levels for the univariate relations between the T1 predictors and future increases in bulimic, depressive, and substance abuse symptoms are presented in Table 4. Depressive symptoms,

[2]Post hoc analyses tested whether depressive symptoms showed differential relations to the onset of the two cardinal features of bulimia nervosa: binge eating and compensatory behaviors. Results confirmed that T1 depressive symptoms predicted onset of both twice-weekly binge eating (OR = 1.83, $p < .001$) and twice-weekly compensatory behaviors (OR = 1.65, $p < .001$).

[3]It might be noted that *post hoc* analyses indicated that there was no statistically significant two-way interactions between the symptoms dimensions in these three multivariate models, which would have necessitated the inclusion of these higher-order terms.

[4]It should be noted that this same logic suggests that the effects from the multivariate models should be less subject to inflation from reporter bias. This is because the reporter bias would be manifest in the correlation between symptom dimensions in the multivariate models and this correlation is statistically controlled (i.e., omitted from the unique effect estimates for each predictor).

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but not substance abuse symptoms, predicted future increases in bulimic symptoms. Bulimic symptoms and substance abuse symptoms predicted increases in depressive symptoms. Finally, bulimic symptoms, but not depressive symptoms, predicted increases in substance abuse symptoms. The fact that only one of the five effects that was significant in the univariate models predicting onset became non-significant in these conservative models that minimize the impact of reporter bias suggests that most of the effects are not likely to be a sole function of reporter bias.

Discussion

Bulimia Nervosa and Depression

Both the univariate and multivariate models provided support for the etiological hypothesis that elevated depressive symptoms increase the risk for onset of bulimic pathology. Individuals are thought to initiate binge eating to provide comfort and distraction from negative mood states (McCarthy, 1990). Although this appears to be the first study to specifically test whether depressive symptoms increase the risk for onset of bulimic pathology, it does converge with conceptually similar findings (e.g., that temperamental emotionality predicted onset of bulimic pathology; Killen et al., 1996). These results provide additional evidence that eating pathology may be partially rooted in efforts to regulate negative affect.

There was also support from the univariate and multivariate models for the hypothesis that bulimic pathology increases the risk for depression onset. Recurrent binge eating and compensatory behaviors putatively result in feelings of shame, guilt, and dysphoria, which increase the risk for depression (Heatherton & Polivy, 1992). Caloric deprivation may also promote depression because it leads to tryptophan depletion. The current findings converge with those from two independent prospective studies (Stice & Bearman, 2001;Stice et al., 2000) and suggest that bulimic pathology is a robust risk factor for depression among adolescent girls.

These results collectively imply that the comorbidity between bulimic pathology and depression in adolescent girls emerges because they are reciprocally related: each symptom dimension increases the risk for onset of the other disorder. This conclusion suggests that prevention and treatment interventions for one disorder might prove effective in decreasing the risk for the other psychiatric condition. Consistent with the assertion, effective eating disorder prevention programs have been found to produce reductions in negative affect (Stice, Trost, & Chase, 2003) and effective depression prevention programs have been found to produce reductions in bulimic pathology (Burton, Stice, Bearman, & Rohde, 2003).

Depression and Substance Abuse

There was mixed support for the etiological hypothesis that depressive symptoms increase the risk for onset of substance abuse; although this relation was significant in the univariate model, it was non-significant in the more stringent multivariate model. Whereas this appears to be the first study to specifically test whether depressive symptoms increase the risk for onset of substance abuse, past research that tested conceptually similar relations also produced mixed results, with studies generating significant (Stice, Barrera et al., 1998), marginally significant (Costa et al., 1999), and non-significant findings (Clark et al., 1999). Taken together, these findings provide only weak support for the affect-regulation theory of substance abuse.

In contrast, there was consistent support for the etiological hypothesis that substance abuse is a risk factor for onset of depression, in that this relation was significant in both the univariate and multivariate models. Whereas this appears to be the first prospective study to provide evidence that substance abuse predicts onset of depression during adolescence, it converges

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with the evidence that substance abuse predicted onset of depression during adulthood (Rao et al., 2000;Rohde et al., 2001). Theoretically, regular use of psychoactive substances causes neurological changes that result in attenuated positive affect and use-related negative consequences promote dysphoria. The magnitude of the relation between substance abuse and onset of depression was fairly large, suggesting that substance abuse is a potent risk factor for mood disturbances in this population.

These findings collectively imply that the comorbidity between depression and substance abuse arises primarily because substance use increases the risk for development of depression, rather than vice versa. The implication of this conclusion is that prevention and treatment interventions for substance use should result in decreased depression, but that interventions for depression may have little impact on substance abuse. Support for both of these assertions have been observed in the literature (e.g., Valentine, Gootlieb, Keel, Griffith, & Ruthhazer, 1998 and Burton et al., 2003 respectively).

Bulimia Nervosa and Substance Abuse

There was little support for the relation between bulimic symptoms and risk for onset of substance abuse; this effect was significant in the univariate model, but not in the multivariate model. Although, the univariate finding provides evidence of the clinical significance of eating pathology by linking it to onset of a serious psychiatric condition, the fact that this effect only emerged in the univariate model suggests that it is a relatively small effect that becomes non-significant when covariates are included in the multivariate model. That the comorbidity between bulimic pathology and substance abuse is relatively weaker than the comorbidity between bulimic pathology and depression and between substance abuse and depression may explain why the prospective effect found in the current study between the former pair of disorders was modest.

In contrast, there was a complete absence of support for the exploratory hypothesis that substance abuse might increase risk for onset of bulimic pathology in the univariate and multivariate models. These null findings converge with the evidence that substance abuse did not predict onset of eating pathology in adulthood (Johnson, Cohen, Kotler et al., 2002).

That nearly all of the prospective tests of the relations between bulimic pathology and substance abuse in the current study were nonsignificant suggests that the comorbidy between these two disorders may arise because they share common risk factors rather than because one increases the risk for onset of the other. More confidence could be placed in this conclusion if future randomized prevention and treatment trials experimentally test whether interventions that reduce one of these disorders results in decreases in the other disorder.

Limitations

The prospective design provides some assurance that the direction of effects were as hypothesized, the use of diagnostic interviews increases the confidence that can be placed in the findings, and the large community-recruited sample augments the generalizability of the results. Nonetheless, it is important to consider the limitations of this study. First, although adolescents appear to be the most valid reporters of their own emotional disturbances, eating disorder symptoms, and substance abuse behaviors (e.g., Cantwell, Lewinsohn, Rohde, & Seeley, 1997;Edelbrook, Costello, Dulcan, Kalas, & Conover, 1985), two problems may be created by the sole reliance on self-report data. On the one hand, reporter-bias may artificially inflate the magnitude of correlations between constructs (Cole, Truglio, & Peeke, 1997). Fortunately, post hoc analyses suggested that most of the effects remained in analyses that more stringently controlled for the effects of reporter bias. On the other hand, perceptual biases or demand characteristics associated with self-report data may artificially attenuate true

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relations that would be detected with more objective measures (Gotlib & Robinson, 1982). It would have been ideal if diagnostic data were collected from several sources, including the adolescents, parents, and teachers, so that consensus diagnoses could have been used.

Second, prospective effects are always open to third variable alternative explanations. Because randomized experiments that manipulate the independent variables are more immune to third variable alternative explanations, randomized prevention and treatment trials allow firmer inferences regarding causal relations. For example, a CBT depression prevention program that reduced depressive pathology was found to also produce decreases in bulimic symptoms, but not decreases in substance use (Burton et al., 2003). Such experiments are crucial because they offer the most rigorous way of differentiating whether comorbidity arises because two disorders simply share risk factors or because one disorder increases the risk for the onset of the other disorder. Future experimental tests regarding the nature of the relations between these symptom dimensions would be a useful complement to prospective studies such as this one.

Third, it would have been optimal if we could have examined the unique predictors of specific psychoactive substances, such as stimulant drugs, because they factor centrally in the hypothesized relations between bulimic pathology and substance abuse. Future studies should attempt to conduct separate assessments for alcohol, marijuana, stimulants, and hallucinogens, in an effort provide a more precise test of the hypothesized relations.

Finally, although this sample was representative of the ethnic composition of the population from which we sampled, these results should be generalized with caution to ethnic minority groups because the sample was not large enough for us to verify that the relations were invariant across ethnic groups.[5]

Directions for Future Research

Because of the relatively exploratory nature of some of the hypotheses tested here, it will be important to attempt to replicate the current findings in an independent study. Future research should also directly test for shared risk factors that might contribute to the comorbidity between the three disorders examined in this study (e.g., individual differences in impulse control). In addition, it would be useful if future studies began exploring the factors that mediate the relations examined in this report (e.g., the hypothesis that tryptophan depletion mediates the relation between bulimic pathology and depression). As noted above, whereas the prospective design is an improvement over cross-sectional data, it does not rule out third variable explanations for the relations. Randomized prevention and treatment trials that decrease each of these symptom dimensions represent one promising way to experimentally test the hypothesized relations.

Conclusions and Implications

In sum, results suggested that the comorbidity between bulimic pathology and depression in adolescent girls emerge because each disorder increases the risk for onset of the other disorder. In contrast, findings implied that the comorbidity between depression and substance abuse results because substance use increases the risk for the onset of depression, rather than vice versa. Finally, results suggest that the most likely explanation for the comoribidy between bulimic pathology and substance abuse is that they share common risk factors. These findings provide support for several etiologic hypotheses for these disturbances and serve to further establish the clinical significance of bulimic pathology, depressive pathology, and substance

[5]Post hoc analyses tested whether the univariate relations reported in Table 2 were significantly different for Caucasian versus ethnic minority participants (we collapsed across all ethnic minority groups to maximize power). However, there was no evidence that ethnic status moderated the relations (all p-values > .10 for the interactive effects).

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abuse by providing evidence that each increases the risk for development of other serious psychiatric conditions. Findings also imply that prevention programs that reduce the levels of one of these disorders (e.g., depression) may also reduce the rates of the other disorders (e.g., bulimic pathology). Such a state of affairs would increase the yield of prevention programs. It is hoped that these preliminary findings will stimulate other researchers to further investigate the processes that might produce the comorbidity between these pernicious psychiatric conditions.

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Table 1

Correlations between the Bulimic, Depressive, and Substance Abuse Symptom Composites, along with the Means, Standard Deviation, Skew Coefficients

	2.	3.	4.	5.	6.	7.	8.	9.	Mean	SD	Skew
1. T1 bulimic symptoms	.32	.21	.38	.30	.23	.34	.23	.21	0.58	1.22	1.49
2. T1 depressive symptoms		.33	.24	.62	.31	.23	.42	.21	1.34	0.37	1.68
3. T1 substance abuse symptoms			.12	.31	.52	.14	.25	.47	0.08	0.21	1.86
4. T2 bulimic symptoms				.38	.26	.36	.32	.22	0.43	0.62	0.18
5. T2 depressive symptoms					.32	.34	.59	.28	1.35	0.38	1.72
6. T2 substance abuse symptoms						.18	.27	.65	0.09	0.26	1.62
7. T3 bulimic symptoms							.40	.16	0.62	0.76	−0.26
8. T3 depressive symptoms								.33	1.38	0.37	1.74
9. T3 substance abuse symptoms									0.09	0.25	1.73

Note. Absolute correlations greater than .15 are significant at $p < .01$. The skew coefficients for the bulimic symptom and substance abuse symptom composites are from the transformed version of these variables used in the analyses.

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Table 2

Parameter Estimates and Confidence Intervals from the Univariate Logistic Regression Models Predicting Onset of Bulimic Pathology, Depressive Pathology, and Substance Abuse.

	Onset of bulimic pathology	
T1 predictors		(95% CI)
Depressive symptoms	1.86***	1.30 – 2.16
Substance abuse symptoms	1.59	1.05 – 2.31
	Onset of depressive pathology	
T1 predictors		(95% CI)
Bulimic symptoms	1.84****	1.38 – 2.46
Substance abuse symptoms	1.83****	1.44 – 2.38
	Onset of substance abuse	
T1 predictors		(95% CI)
Depressive symptoms	1.57**	1.18 – 2.10
Bulimic symptoms	1.62***	1.21 – 2.18

Note. CI = Confidence interval. All predictors were standardized so that the odds ratios could be directly compared. Alpha = .01.

** $p < .01$,

*** $p < .001$,

**** $p < .0001$

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Table 3

Parameter Estimates and Confidence Intervals from the Multivariate Logistic Regression Models Predicting Onset of Bulimic Pathology, Depressive Pathology, and Substance Abuse.

	Onset of bulimic pathology	
T1 predictors		(95% CI)
Depressive symptoms	1.74**	1.18 – 2.55
Substance abuse symptoms	1.29	0.85 – 1.96
	Onset of depressive pathology	
T1 predictors		(95% CI)
Bulimic symptoms	1.68***	1.24 – 2.27
Substance abuse symptoms	1.72****	1.34 – 2.20
	Onset of substance abuse	
T1 predictors		(95% CI)
Depressive symptoms	1.39	1.01 – 1.91
Bulimic symptoms	1.45	1.05 – 1.99

Note. CI = Confidence interval. All predictors were standardized so that the odds ratios could be directly compared. Alpha = .01.

** $p < .01$,

*** $p < .001$,

**** $p < .0001$

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Table 4

Parameter Estimates from the Univariate Growth Curve Models Predicting Increases in Bulimic, Depressive, and Substance Abuse Symptoms.

		Increases in bulimic symptoms	
T1 predictors	β	B	(95% CI for B)
Depressive symptoms	.10***	.16	0.078 – 0.246
Substance abuse symptoms	.04	.02	−0.003 – 0.034
		Increases in depressive symptoms	
T1 predictors	β	B	(95% CI for B)
Bulimic symptoms	.11**	.02	0.006 – 0.036
Substance abuse symptoms	.10**	.01	0.002 – 0.021
		Increases in substance abuse symptoms	
T1 predictors	β	B	(95% CI for B)
Depressive symptoms	.07	.21	−0.074 – 0.491
Bulimic symptoms	.11**	.12	0.024 – 0.211

Note. β = standardized regression coefficient. CI = Confidence interval. All predictors were standardized so that the odds ratios could be directly compared. Alpha = .01.

** $p < .01$,

*** $p < .001$,

**** $p < .0001$