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Depression and Eating Pathology: Prospective Reciprocal Relations in Adolescents

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Abstract

The association between disordered eating and depression has been established, but less is known about the temporal relations between these two disturbances. Accordingly, the current study examined the reciprocal relations between depressive and bulimic symptoms over an 8-year period with longitudinal data from a community sample of 496 female adolescents. Depressive symptoms predicted future increases in bulimic symptoms, and bulimic symptoms likewise predicted increases in depressive symptoms, controlling for earlier levels of symptoms for each outcome. These results provide evidence consistent with the hypothesis that the two disorders contribute reciprocally to each other, and indicate that successful prevention or treatment of one disorder may yield effects for the other. However, the relatively small predictive effect sizes imply that some third variable may contribute to both conditions (e.g., temperamental negative affectivity).

Research from both clinical and epidemiological studies have documented substantial comorbidity between eating disorders and depression among females (Lewinsohn, Striegel-Moore, & Seeley, 2000; Santos, Richards, & Bleckley, 2007; Zaider, Johnson, & Cockell, 2000). For example, one study found that 46% of female inpatients diagnosed with DSM-IV bulimia nervosa also met criteria for concurrent major depressive disorder (Blinder, Cumella, & Sanathara, 2006). Moreover, when other forms of unipolar depression were considered, 48% had a current comorbid diagnosis of depressive disorder NOS, and 8% were diagnosed with dysthymic disorder. Similarly, the concurrent prevalence of major depression among outpatients with bulimia nervosa has been estimated to range between 28% and 41% (Brewerton et al., 1995; Fornari, Kaplan, Sandberg, Matthews, Skolnick, & Katz, 1992). Fischer & le Grange (2007) found that nearly half of treatment-seeking adolescents with bulimia had a concurrent mood disorder. However, because clinical samples usually generate overestimates of psychiatric comorbidity (Berkson, 1946), it is also important to examine data from non-treatment seeking samples. Rates of current major depressive disorder among community samples with full syndrome or partial syndrome bulimia nervosa are estimated at 20% and 18%, respectively (Garfinkel, et al., 1995).

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Additionally, among a community-recruited sample of women with bulimia, 55% of those with an eating disorder at 5-year follow up also met current criteria for concurrent major depression (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000).

Despite consistent evidence regarding the association between bulimia and depression, the temporal nature of this relation remains unclear. For example, it is possible that depression gives rise to bulimia, that depression is a consequence of bulimia, or that depression and bulimia are reciprocally related to one another. Resolving the nature of this comorbid relationship would help inform whether treatment or prevention of one disorder may help resolve or circumvent the other, which could reduce unnecessary distress and mental health care expenses.

The escape theory (Heatherton & Baumeister, 1991) conceptualizes binge eating as a maladaptive coping method by which an individual attempts to escape and alleviate affective distress by focusing on external food cues. Purging may then be employed to reduce anxiety about weight gain, creating a self-perpetuating cycle of bulimic symptoms (Fairburn, 1995). Thus, this conceptualization suggests that elevated depressive symptoms may increase the risk for subsequent onset or exacerbation of bulimic symptoms. In support of this hypothesis, prospective research has found that negative affect predicts later increases in bulimic symptoms (Stice, 2001; Stice, Akutagawa, Gaggar, & Agras, 2000; Tyrka, Waldron, Graber, & Brooks-Gunn, 2002) and onset of binge eating and compensatory behaviors (Stice & Agras, 1998; Stice, Killen, Hayward, & Taylor, 1998) among female adolescents. Similarly, earlier depressive symptoms have been found to predict later onset of bulimic pathology (Dobmeyer & Stein, 2003; Gilbert & Meyer, 2005; Stice, Burton, & Shaw, 2004; Wertheim, Koerner, & Paxton, 2001) among adolescent and college aged females. Moreover, dysthymia predicted an increase in risk for bulimia nervosa among a sample of adolescent females, above and beyond prior psychiatric problems (Zaider, Johnson, & Cockell, 2002). One study found that depressive symptoms were not predictive of later bulimic symptoms (Procopio, Holm-Denoma, Gordon, & Joiner, 2006). However, this study involved middle-aged adult participants, and therefore the null findings may have simply reflected that this sample was beyond the peak period of risk for increases in eating pathology.

On the other hand, bulimic symptoms may also contribute to depressed mood since binge eating and purging may lead to feelings of shame and guilt, increased rumination about one's inability to control food intake, and impairment in interpersonal functioning (Heatherton & Polivy, 1992; Keel, Mitchell, Miller, Davis, & Crow, 2000; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Prospective research has also provided findings that are consistent with this hypothesis. Specifically, bulimic symptoms have been found to predict future increases in depressive symptoms (Stice & Bearman, 2001; Stice et al., 2004) and onset of depression (Stice et al., 2000) among female adolescents. Additionally, the presence of an eating disorder during adolescence was associated with a four-fold increase in risk for a depressive disorder in early adulthood (Johnson, Cohen, Kasen, & Brook, 2002). However, one study involving adults did not find a prospective effect of bulimic symptoms (Procopio et al., 2006) on subsequent increases in depressive symptoms.

As these studies suggest, there may be reciprocal relations among depressive and bulimic symptoms, and recent research has attempted to explore this possibility. Two studies have found that earlier levels of eating pathology predicted future increases in depressive symptoms, whereas the reverse relationship was supported in one study (Stice et al., 2004) but not the other (Marmorstein von Ranson, Iacono, & Malone, 2008). Resolving the temporal sequencing of these disorders is particularly important for guiding prevention and treatment recommendations. If depression increases risk for eating pathology, but not the

reverse, it would imply that focusing efforts on developing and refining effective depression prevention and treatment programs may reduce both conditions. Likewise, if bulimia nervosa is more likely to give rise to depression, then prevention and treatment efforts should focus on reducing bulimic pathology. Finally, if these two disorders are reciprocally related, it could be argued that it is more advisable to focus on the condition for which more effective prevention programs have been developed. Thus, the aim of the current study is to clarify the temporal relations between depressive and bulimic symptoms using 8-year prospective longitudinal data from a community sample of female adolescents. We focused on examining these relations over a longer developmental period than previous studies to capture both the peak risk for eating pathology and depressive symptoms, and utilized diagnostic interviews to assess the main outcomes.

Method

Participants

Participants were 496 female adolescents recruited from four public (82%) and four private (18%) middle schools in a metropolitan area of the Southwestern United States, ranging in age from 11 to 15 (mean = 13.5, SD = 0.67) at the time of study entry. The sample included 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% who specified *other or mixed* racial heritage, 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 racial heritage). Average parental education, a proxy for socioeconomic status, was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was representative of 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 presented to parents and participants as an investigation of adolescent mental and physical health. Active parental consent and adolescent assent was obtained prior to any data collection, resulting in an average participation rate of 56%, which was comparable to rates in other school-recruited samples that used active consent procedures and structured interviews (e.g., 61% for Lewinsohn et al., 1994). Participants completed a survey and participated in a structured interview at baseline (T1) and at seven annual follow-ups (T2, T3, T4, T5, T6, T7, and T8). Female clinical assessors with at least a bachelor's degree in psychology conducted all interviews. Assessors were required to demonstrate a high level of inter-rater agreement (kappa [k] > .80) with supervisors using tape-recorded interviews before collecting data. Interviews were recorded periodically during the study to ensure that assessors showed acceptable inter-rater agreement (k > .80). Assessments took place at the school during or immediately after school hours or at participants' houses. Participants were compensated at each assessment with a gift certificate or a cash payment. This study was approved by the Institutional Review Board of the University of Texas at Austin.

Measures

Bulimic Symptoms—The Eating Disorder Diagnostic Interview, a semi-structured interview that was adapted from the Eating Disorder Examination (EDE; Fairburn et al., 1993), assessed DSM-IV eating disorder symptoms. Items assessing symptoms during the past month were summed to create an overall bulimic symptom composite at each assessment, as done previously (Presnell & Stice, 2003; Stice et al., 2004). To adjust for skew in the bulimic symptom composite used in the current study, a normalizing square root transformation was applied. The symptom composite showed internal consistency (α = .92),

1-week test-retest reliability (r = .90), sensitivity to detecting intervention effects, and predictive validity for future onset of depression in past studies (Presnell & Stice, 2003; Stice et al., 2004). In the current trial the symptom composite showed internal consistency (α = .86 at pretest) and 1-month test-retest reliability for assessment-only controls (r = .81). To assess the test-retest reliability for eating disorder diagnoses for this adapted interview, a randomly selected subset of 137 participants who were interviewed by the assessors for this study and another study (Stice et al., 2004) were re-interviewed by the same assessor within a 1-week period, resulting in high test-retest reliability for threshold and subthreshold diagnoses of anorexia nervosa, bulimia nervosa, and binge eating disorder (κ = .96). To assess the inter-rater agreement for these threshold and subthreshold eating disorder diagnoses, a randomly selected subset of 149 participants who were interviewed by the assessors for this and another study were re-interviewed by a second blinded assessor, resulting in high inter-rater agreement (κ = .86).

Depressive symptoms—The Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich & Chambers, 1983), a semi-structured diagnostic interview, was used to assess DSM-IV symptoms of major depression. Participants reported on the peak severity of each symptom during the prior 12 months using expanded response options ranging from 1 = *not at all* to 4 = *severe symptoms*. The past year severity ratings for each symptom were averaged to form a continuous depressive symptom composite at each assessment. This version of the K-SADS depression interview has shown 1-week test-retest reliability (κ = 1.00) and inter-rater agreement (κ = 1.00) for depression diagnosis and internal consistency ($M\alpha$ = .81), 1-week test-retest reliability (r = .86) and inter-rater agreement (r = .87) for the symptom composite (Nolen-Hoeksema et al., 2007).

Results

Of the initial 496 participants, follow-up data was obtained for 98% at T2, 98% at T3, 99% at T4, 98% at T5, 96% at T6, 94% at T7, and 94% at T8. However, only 1% did not provide data at any of the follow-up assessments. Attrition analyses verified that participants who were missing data at any assessment point did not differ significantly from the remaining participants on demographic factors or any of the variables examined in the report, indicating that attrition did not introduce systematic bias. Intercorrelations among bulimic symptoms and depressive symptoms at each timepoint are presented in Table 1, along with the means and standard deviations. Overall, mean bulimic and depressive symptom composite scores indicate moderate stability at the group level over the 7-year study period. The correlations between earlier levels of each of the variables with subsequent levels indicate that they may be related over time; however, it is possible for individual scores to change while mean scores remain relatively constant. Thus, we examined individual level change across time.

To examine the prospective, reciprocal relations between depressive and bulimic symptoms, we estimated two N - 1 lagged growth models with time-varying covariates (Singer & Willett, 2003). Specifically, we tested whether depressive symptoms predicted future increases in bulimic symptoms and whether bulimic symptoms predicted future increases in depressive symptoms over the seven 1-year intervals, controlling for the 1-year lag of the dependent variable. Thus, the first model tested whether the average predictive effect of depressive symptoms assessed at T1, T2, T3, T4, T5, T6, and T7 predicted bulimic symptoms at T2, T3, T4, T5, T6, T7, and T8, respectively, controlling for the effects of bulimic symptoms at the prior assessment (i.e., to control for the autoregressive effects for the dependent variable). The second model tested these same relationships with the predictor

and outcome reversed. Models were estimated in HLM (Version 6; Bryk, Raudenbush, & Congdon, 2004).

We first tested the hypothesis that depressive symptoms would predict future increases in bulimic symptoms. The 1-year lag of depressive symptoms, controlling for the 1-year lag of bulimic symptoms, predicted higher levels of bulimic symptoms over the seven time points, $t(3181) = 7.02$, $B = 0.12$, $p < .001$, 2% variance explained. In addition, we tested the hypothesis that bulimic symptoms would predict future increases in depressive symptoms. The 1-year lag of bulimic symptoms, controlling for the 1-year lag of depressive symptoms, predicted higher levels of depressive symptoms over the seven time points, $t(3181) = 5.79$, $B = 0.10$, $p < .001$, 3% variance explained. Results from the lagged hierarchical linear models are presented in Table 2.

Discussion

The current study examined the reciprocal relations between depressive and bulimic symptoms in adolescent females. Results indicated that depressive symptoms prospectively predicted future increases in bulimic symptoms, and that bulimic symptoms likewise predicted future increases in depressive symptoms in adolescent females over this developmental period.

These findings converge with prior prospective studies which found that depressive symptoms predict increases in bulimic symptoms (Dobmeyer & Stein, 2003; Gilbert & Meyer, 2005; Stice, et al., 2004; Wertheim et al., 2001) among adolescent and college aged females. Additionally, our results are largely consistent with studies in which bulimic symptoms have been found to predict later increases in depressive symptoms (Stice & Bearman, 2001; Stice et al., 2004). Our small to moderate effect sizes also converge with a meta-analytic study examining risk and maintenance factors for eating pathology (Stice, 2002), which found that the average predictive effect of negative affect for both increases in eating pathology (r = .09) and for maintenance of eating pathology (r = .13) were both small in magnitude, according to Cohen's (1988) criteria. More generally, the average univariate effects for a number of other established risk factors for bulimic pathology (e.g., body dissatisfaction, thin-ideal internalization, modeling of disordered eating) examined in this meta-analysis were also small. This suggests that a single variable is unlikely to be responsible for a large proportion of the variance in changes in eating pathology, and implies that multivariate models may be required to increase explanatory power. The relatively small magnitude of effects may also suggest that other risk factors are operating to result in increases in bulimic and depressive symptoms.

Overall, these findings indicate that bulimic and depressive symptoms are reciprocally related, with each increasing the risk for the other. However, these results are contrary to a study by Marmorstein et al. (2008), which indicated that eating pathology predicted later depressive symptoms, but earlier depressive symptoms did not predict later eating pathology. This discrepancy may have resulted because the Marmorstein et al. (2008) study focused on a general measure of eating pathology while the other two studies that found effects focused solely on bulimic symptoms. Additionally, Marmorstein et al. used a 3-year lag whereas the current study used a 1-year lag between assessments, and it is possible that depressive symptoms exert their influence on increases in eating pathology over a relatively short period of time.

The current findings are consistent with the hypothesis that individuals may initiate binge eating as a means of coping with or avoiding negative affective states. Recent evidence indicating that a ruminative coping style, which entails repeatedly focusing on symptoms of

depression and its causes in the absence of active attempts at problem solving, may amplify feelings of depression and represent a response style that increases the likelihood that an adolescent may turn to binge eating for escape or distraction (Nolen-Hoeksema et al., 2007). Compensatory behaviors, such as self-induced vomiting or fasting, may then be employed to reduce anxiety about resulting weight gain, creating a cycle of self-perpetuating bulimic symptoms.

There was also support for the hypothesis that bulimic symptoms increase the risk for subsequent depressive symptoms. This may be due, in part, to the feelings of shame and guilt that often accompany recurrent binge eating and purging. Additionally, it is possible that dissatisfaction with body shape and weight may lead to more generalized negative mood because Western culture emphasizes appearance as a key evaluative dimension for women (Stice, Hayward et al., 2000). The extreme dietary restriction that is often employed between binge episodes may further contribute to depression due to reduced consumption of tryptophan-rich foods (an amino acid precursor to serotonin)(Kaye, Gendall, & Strober, 1998).

When considering the relatively small magnitude of the predictive effects observed in the current study, another possibility is that some third variable contributes to both conditions. Two alternative explanations for effects observed in longitudinal studies include reverse causation and third variable explanations. Although we accounted for reverse causation by examining reciprocal relations, it remains possible the observed relationship is a function of a joint association of the independent and dependent variables with a third variable. Potential third variables may include body dissatisfaction, temperamental negative affectivity, rumination, low distress tolerance and social support deficits, which have been linked to both bulimia (Anestis, Selby, Fink, & Joiner, 2007; Ghaderi, 2003; Nolen-Hoeksema et al., 2007; Stice & Agras, 1998; Stice et al., 2000; Stice, 2001; Tyrka et al., 2002) and depression (Gjone & Stevenson, 1997; Moulds & Kandris, 2007; Sheeber, Hops, Alpert, Davis, & Andrews, 1997; Nolen-Hoeksema et al., 2007; Stice et al., 2000; Stice & Bearman, 2001). For example, it is possible that a temperamental disposition towards negative affectivity increases vulnerability to depression because it leads individuals to focus on unpleasant aspects of situations and become distressed in the absence of any overt or objective stress. Negative affectivity may independently contribute to bulimic symptoms because individuals may binge eat to provide comfort or distraction from negative emotional states.

Gaining a greater understanding of the mechanisms that link bulimia and depression will be necessary to help guide intervention efforts. For example, findings from prospective studies suggesting that depression and bulimia are reciprocally related imply that prevention and treatment programs that reduce levels of one disorder should also impact the other. Consistent with this hypothesis, recent evidence suggests that a brief depression prevention program that significantly reduced depressive symptoms among high risk adolescents with elevated depressive symptoms, also produced reductions in bulimic symptoms (Burton, Stice, Bearman, & Rohde, 2007). However, the beneficial effects on bulimic symptoms did not emerge in a second trial, despite significant reductions in depressive symptoms (Stice, Rohde, Seeley, & Gau, 2008). Although the discrepant findings might have resulted because the latter trial included both males and females whereas the former included only females, it is also possible that an omitted third variable contributes to the comorbidity between bulimia and depression. Indeed, another study that focused on reducing body dissatisfaction among adolescent females found that reductions in body image concerns led to short-term reductions in both depressive and bulimic symptoms (Bearman, Stice, & Chase, 2003), which also implies that these disorders may be related because they have shared risk factors. If the association between bulimia and depression is due to third variables (such as body

dissatisfaction, temperamental negative affectivity, or low distress tolerance), then these variables would be the more appropriate targets for intervention.

Strengths and Limitations

Strengths of the current study include a large sample size, long follow-up period, and low attrition rate, as well as the use of continuous measures that allowed detection of subthreshold levels of symptom domains that may nonetheless represent clinically significant impairment. The confidence that can be placed in our findings is also strengthened by the fact that we used structured psychiatric interviews to assess the primary outcomes. Nonetheless, several limitations should be taken into consideration when interpreting the findings. Although we used prospective analyses to test our hypotheses, it is possible that some unmeasured third variable explains the observed prospective effects, as is the case with all longitudinal research. Second, we relied exclusively on adolescent report, and although adolescents are generally considered the best reporters of their own internalizing symptoms, reports from peers or parents would provide additional information. Third, our moderate participation rate and inclusion of relatively few individuals from certain ethnic groups suggest that these findings should be generalized with caution.

Conclusions

Overall, the present findings suggest that female adolescents with high levels of depressive symptoms may be at risk for future escalations in bulimic symptoms. Likewise, individuals suffering from bulimic symptoms are at elevated risk for future increases in depressive symptoms. However, independent replication is necessary to increase confidence in these relations. Although some preliminary evidence suggests that it may be possible to impact multiple mental health outcomes among adolescent females by targeting one disorder that is demonstrated to give rise to another, this possibility should be specifically addressed in future randomized prevention and treatment trials. If such interventions are demonstrated to impact multiple mental health problems, the greatest public health effects may be realized by focusing on the disorder that is more readily prevented or treated. Additionally, it would be useful for future research to attempt to identify possible mechanisms for these links between depressive and bulimic symptoms. For example, low distress tolerance, temperamental negative affectivity, or a ruminative coping style may confer increased risk for both depression and eating pathology. Such information would help further clarify the nature of the relationship between depressive and bulimic symptoms, and help guide prevention and treatment efforts.

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Table 1

Correlations Between Depressive and Bulimic Symptom Composites

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	M	SD
1. T1 bulimic sxs	-	.39	.45	.34	.36	.31	.25	.22	.29	.18	.21	.19	.20	.13	.26	.14	0.52	0.55
2. T1 depressive sxs		-	.25	.62	.24	.43	.12	.37	.20	.34	.17	.30	.06	.28	.11	.32	1.34	0.37
3. T2 bulimic sxs			-	.38	.41	.30	.30	.15	.27	.19	.20	.18	.21	.17	.23	.16	0.48	0.45
4. T2 depressive sxs				-	.34	.59	.18	.46	.23	.45	.20	.39	.19	.38	.22	.40	1.35	0.38
5. T3 bulimic sxs					-	.37	.37	.30	.33	.26	.28	.24	.28	.21	.20	.21	0.58	0.41
6. T3 depressive sxs						-	.27	.55	.23	.46	.18	.39	.24	.44	.23	.44	1.38	0.37
7. T4 bulimic sxs							-	.25	.38	.25	.31	.19	.28	.18	.24	.15	0.65	0.33
8. T4 depressive sxs								-	.22	.64	.21	.53	.20	.49	.14	.55	1.41	0.41
9. T5 bulimic sxs									-	.24	.33	.26	.32	.26	.30	.22	0.63	0.37
10. T5 depressive sxs										-	.20	.55	.21	.51	.12	.52	1.44	0.43
11. T6 bulimic sxs											-	.32	.39	.25	.34	.30	0.64	0.41
12. T6 depressive sxs												-	.26	.54	.18	.60	1.46	0.42
13. T7 bulimic sxs													-	.29	.48	.24	0.72	0.34
14. T7 depressive sxs														-	.17	.58	01.46	0.45
15. T8 bulimic sxs															-	.26	0.68	0.36
16. T8 depressive sxs																-	1.39	0.39

Note. Absolute correlations greater than .10 are significant at $p < .05$.

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Table 2
Results of *N* – 1 Lagged Hierarchical Linear Models

Predictor	Coefficient	*SE*	*p*
Effects of depressive symptoms on bulimic symptoms			
Bulimic symptoms	.25	.02	.000
Depressive symptoms	.12	.02	.000
Effects of bulimic symptoms on depressive symptoms			
Depressive symptoms	.31	.03	.000
Bulimic symptoms	.09	.02	.000

Note. Coefficient refers to unstandardized slope coefficient.