Is Major Depressive Disorder or Dysthymia More Strongly Associated with Bulimia Nervosa?

Marisol Perez,1 Thomas E. Joiner, Jr.,1,* and Peter M. Lewinsohn2

1 Department of Psychology, Florida State University, Tallahassee, Florida
2 Oregon Research Institute, Eugene, Oregon

Accepted 8 September 2003

Abstract: Objective: Research on adult samples has found that the comorbidity between depression and eating disorders exceeds the comorbidity of any other Axis I disorder and eating disorders. Few studies have investigated the specific associations of major depression versus dysthymia with eating disorders. Method: This sample consisted of 937 adolescents who were repeatedly assessed until the age of 24. Results: Analyses revealed that dysthymia was a stronger correlate with bulimia than major depression, even while controlling for other mood disorders and a history of depression and dysthymia. Conclusions: The presence of dysthymia in adolescence might be a possible risk factor for the development of bulimia nervosa. © 2004 by Wiley Periodicals, Inc., Int J Eat Disord 36: 55–61, 2004.

Key words: bulimia; dysthymia; depression

INTRODUCTION

Most of the literature investigating the comorbidity between depression and eating disorders has focused on adult samples, and has found a consistent association between depression and eating disorders. Usually, the comorbidity of depression and eating disorders exceeds the comorbidity of any other Axis I disorder and eating disorders (Braun, Sunday, & Halmi, 1994; Brewerton et al., 1995; Zerbe, Marsh, & Coyne, 1993). These findings may not be specific to Western cultures, in that similar rates of comorbidity between depression and eating disorders can be found in Eastern cultures as well (Iwasaki, Matsunaga, Kiriike, Tanaka, & Matsui, 2000).

Fewer studies have investigated these issues among adolescent samples. One recent study conducted on a female adolescent sample reported findings consistent with those reported in the adult literature. Lewinsohn, Striegel-Moore, and Seeley (2000) investigated the association of Axis I disorders with full-syndrome and partial-syndrome eating disorders. They found that high comorbidity rates of eating disorders with Axis I disorders were not a phenomenon limited to adult samples. Approximately 70% of the
adolescents who met criteria for full and partial-syndrome eating disorders also met criteria for an Axis I disorder. Individuals with eating disorders had high rates of mood disorders in general. Rates were not analyzed separately regarding major depression versus dysthymia. In addition, Lewinsohn et al. found that full and partial-syndrome eating disorders did not differ in terms of psychiatric comorbidity. Partial-syndrome eating-disordered individuals were just as likely to have Axis I disorders as full-syndrome individuals.

Zaider, Johnson, and Cockell (2000) also investigated comorbidity of depression and eating disorders among a female adolescent sample, but specifically examined the relation of eating disorders to dysthymia versus major depression. Like the Lewinsohn et al. (2000) study, Zaider et al. found that mood disorders had high comorbidity rates with eating disorders, but they also found that dysthymia was more related to eating disorders than was major depressive disorder (MDD). More specifically, Zaider et al. found that several Axis I disorders, including MDD, panic disorder, and dysthymia, were associated with eating disorders. However, only dysthymia, and not major depression, predicted eating disorders after controlling for other Axis I and Axis II disorders, leading these authors to conclude that dysthymia may be associated more strongly with eating disorders than MDD among adolescents.

Given the relatively few studies investigating adolescent female samples with eating disorders in terms of comorbidity between depression and eating disorders, further research in this area is warranted. Additional studies need to be conducted to establish if MDD or dysthymia is associated more strongly with eating disorders in adolescence. This distinction is important because MDD and dysthymia differ in terms of chronicity, severity, and persistence of the mood disturbance, factors that can impact the course and treatment of eating disorders. For this reason, the main purpose of the current study is to investigate the comorbidity of MDD, dysthymia, and eating disorders among an adolescent female sample. In context of the findings of Zaider et al. (2000), we are interested in investigating whether MDD or dysthymia emerges as the stronger correlate with eating disorders, after controlling for the presence of other Axis I disorders.

**METHOD**

**Participants and Procedure**

Participants were selected randomly from nine high schools representative of urban and rural districts in western Oregon. A total of 1,709 adolescents completed the initial (T1) assessments (interview and questionnaires) between 1987 and 1989, with an overall participation rate of 61%. With minor exceptions, the adolescents in the T1 sample were representative of high school students in western Oregon. Approximately one half of the T1 sample was female (53.7%), with an average age of 16.6 years (SD = 1.2). A total of 8.9% were non-White; 71.3% were living with two parents and 53% were living with two biologic parents; and 12.3% had repeated a grade in school. Parental education level (maximum value for mother or father) was as follows: 1.9% did not complete high school, 16.1% completed high school, 35.1% had a partial college education, and 46.9% had an academic or professional degree.

Participants were interviewed with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) that combined features of the epidemiologic version (K-SADS-E) and the present episode version (K-SADS-P).
Additional items were also included to derive diagnoses of past and current psychiatric disorders as outlined in the 3rd Rev. ed. of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; American Psychiatric Association [APA], 1987).

At the second assessment (T2), 1,507 participants (88.2 %) returned for a readministration of the interview and questionnaire (mean T1 to T2 interval = 13.8 months, SD = 2.3). There was no relation between depressive status and attrition. Additional details regarding the sample are provided in Lewinsohn, Hops, Roberts, and Seeley (1993).

At age 24 (M = 24.22 years, SD = .57), all participants with a history of MDD and dysthymia at T2 (n = 336) or a history of non-mood disorders (n = 284), and an approximately equal number of young adults with no history of psychopathology by T2 (n = 457), were invited to participate in a T3 evaluation. Of the 1,077 T2 participants selected for a T3 interview, 941 (87.4 %) completed the age 24 evaluation. The various T2 diagnostic groups did not differ on the rate of participation at T3.

At T2 and T3, participants were interviewed using the Longitudinal Interval Follow-Up Evaluation (LIFE; Shapiro & Keller, 1981), which elicited detailed information about the course of psychiatric disorders since the previous evaluation. Diagnostic information was available regarding the occurrence, onset age, and duration of all disorders before and during the course of the study. All T3 diagnoses were made using criteria in the 4th ed. of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994). Based on a randomly selected subsample at T1 (n = 236), interrater reliability for a lifetime diagnosis of MDD was excellent (k = .93). Similarly, the interrater reliability for a lifetime diagnosis of dysthymia based on the same sample was excellent (k = .91). For the T2 to T3 period (n = 329), interrater reliability for a lifetime diagnosis of MDD was good (k = .88). For the T2 to T3 period, interrater reliability for dysthymia was also good (k = .84). The interrater reliability for other disorders, including eating disorders, was also good.

Participant interviews at T3 were conducted by telephone, which generally yields comparable results to face-to-face interviews (Rohde, Lewinsohn, & Seeley, 1997; Sobin, Weissman, Goldstein, & Adams, 1993; Wells, Burnam, Leake, & Robins, 1988). Most of the interviewers had advanced degrees in clinical or counseling psychology or social work and several years of clinical experience. All interviewers were trained in the use of the Structured Clinical Interview for DSM-IV (SCID) and LIFE and completed a minimum of two supervised training interviews.

The sample in the current study included individuals who completed all the relevant assessments for the three time periods. Therefore, this sample consists of 937 individuals. To be able to evaluate an adequate sample size of bulimic women, individuals who met criteria for bulimia after the T1 period (including those who met criteria for bulimia between the T2 and T3 assessments) were combined, totaling 17 bulimic women.

**RESULTS**

Intercorrelations, means, and standard deviations are presented in Table 1. In general, correlations were consistent with past research. The combined bulimia variable significantly correlated with a lifetime diagnosis of dysthymia and no other variable. As expected, the lifetime diagnosis of major depression significantly correlated with current major depression. Similarly, the lifetime diagnosis of dysthymia correlated with the current diagnosis of dysthymia. Finally, current major depression correlated positively with current dysthymia.

To further investigate whether dysthymia or major depression is more predictive of the onset of bulimia, a logistic regression was used where the dependent variable was
bulimia and the predictors were current and lifetime diagnosis of major depression and dysthymia at T1. The dependent variable, bulimia, was coded such that 1 = no diagnosis after T1 and T2 = bulimic diagnosis after T1. As shown in Table 2, there was only one significant predictor, lifetime diagnosis of dysthymia, that was associated with the presence of bulimia. Consistent with the findings of Zaider et al. (2000), dysthymia, not major depression, was associated more strongly with bulimia.

To address the possibility that confounding variables could account for the previous finding, the same logistic regression was conducted while covarying for depression and dysthymia after T1 and other mood disorders. The covariates that were entered into the first block of the equation were a history of bulimia before the study, current dysthymia, current major depression, current bipolar disorder, current depression not otherwise specified (NOS) at T2 and T3, in between T1 and T2, and in between T2 and T3. As shown in Table 3, even when covarying dysthymia and major depression after T1 and other mood disorders, the results were similar. A history of dysthymia at T1 was the only significant predictor of bulimia after T1.

### DISCUSSION

The main purpose of the current study was to assess whether major depression or dysthymia was associated more strongly with the onset of bulimia. Consistent with previous findings (Zaider et al., 2000), it was found that dysthymia was a stronger
correlate with bulimia than was major depression. This result held when controlling for current dysthymia, current diagnosis of major depression, and covariates as predictors of bulimia.

Table 3. Lifetime diagnosis of dysthymia, lifetime diagnosis of major depression, current diagnosis of dysthymia, current diagnosis of major depression, and covariates as predictors of bulimia

<table>
<thead>
<tr>
<th>Variables</th>
<th>$B$</th>
<th>$SE$</th>
<th>Wald Value</th>
<th>$df$</th>
<th>$p$</th>
<th>Exp(B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First block</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifetime bulimia</td>
<td>3.15</td>
<td>1.32</td>
<td>5.66</td>
<td>1,921</td>
<td>.02</td>
<td>23.3</td>
</tr>
<tr>
<td>$T_1$-$T_2$ dysthymia</td>
<td>$-2.26$</td>
<td>217.04</td>
<td>.00</td>
<td>1,921</td>
<td>.99</td>
<td>.77</td>
</tr>
<tr>
<td>$T_1$-$T_2$ MDD</td>
<td>.25</td>
<td>.80</td>
<td>.10</td>
<td>1,921</td>
<td>.75</td>
<td>1.29</td>
</tr>
<tr>
<td>$T_1$-$T_2$ bipolar</td>
<td>1.09</td>
<td>1.65</td>
<td>.43</td>
<td>1,921</td>
<td>.51</td>
<td>2.97</td>
</tr>
<tr>
<td>$T_1$-$T_2$ Dep NOS</td>
<td>$-7.38$</td>
<td>23.17</td>
<td>.10</td>
<td>1,921</td>
<td>.75</td>
<td>.00</td>
</tr>
<tr>
<td>$T_1$ MDD</td>
<td>.08</td>
<td>1.26</td>
<td>.00</td>
<td>1,921</td>
<td>.95</td>
<td>.92</td>
</tr>
<tr>
<td>$T_1$ Dep NOS</td>
<td>$-6.99$</td>
<td>44.28</td>
<td>.00</td>
<td>1,921</td>
<td>.99</td>
<td>.50</td>
</tr>
<tr>
<td>$T_1$-$T_2$ dysthymia</td>
<td>$-6.75$</td>
<td>109.70</td>
<td>.00</td>
<td>1,921</td>
<td>.95</td>
<td>.00</td>
</tr>
<tr>
<td>$T_1$-$T_2$ Dep NOS</td>
<td>1.44</td>
<td>.58</td>
<td>6.21</td>
<td>1,921</td>
<td>.01</td>
<td>4.23</td>
</tr>
<tr>
<td>$T_2$ MDD</td>
<td>2.65</td>
<td>1.25</td>
<td>4.51</td>
<td>1,921</td>
<td>.03</td>
<td>14.11</td>
</tr>
<tr>
<td>$T_2$ Dep NOS</td>
<td>1.31</td>
<td>.69</td>
<td>3.61</td>
<td>1,921</td>
<td>.06</td>
<td>3.70</td>
</tr>
<tr>
<td>$T_2$-$T_3$ dysthymia</td>
<td>$-3.34$</td>
<td>156.93</td>
<td>.00</td>
<td>1,921</td>
<td>.99</td>
<td>.71</td>
</tr>
<tr>
<td>$T_2$ MDD</td>
<td>$-7.43$</td>
<td>45.45</td>
<td>.00</td>
<td>1,921</td>
<td>.97</td>
<td>.00</td>
</tr>
<tr>
<td>$T_2$ bipolar</td>
<td>.53</td>
<td>1.77</td>
<td>.09</td>
<td>1,921</td>
<td>.77</td>
<td>1.70</td>
</tr>
<tr>
<td>$T_3$ Dep NOS</td>
<td>$-2.29$</td>
<td>1.22</td>
<td>.05</td>
<td>1,921</td>
<td>.82</td>
<td>.75</td>
</tr>
<tr>
<td>Second block</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifetime dysthymia</td>
<td>2.40</td>
<td>.78</td>
<td>9.43</td>
<td>1,917</td>
<td>.00</td>
<td>10.98</td>
</tr>
<tr>
<td>Lifetime MDD</td>
<td>$-0.01$</td>
<td>.63</td>
<td>.00</td>
<td>1,917</td>
<td>.99</td>
<td>.99</td>
</tr>
<tr>
<td>Current dysthymia</td>
<td>$-8.64$</td>
<td>131.32</td>
<td>.00</td>
<td>1,917</td>
<td>.95</td>
<td>.00</td>
</tr>
<tr>
<td>Current MDD</td>
<td>1.00</td>
<td>1.01</td>
<td>.83</td>
<td>1,917</td>
<td>.36</td>
<td>2.72</td>
</tr>
</tbody>
</table>

Note: Lifetime bulimia = history of bulimia at $T_1$; $T_1$-$T_2$ dysthymia = since $T_1$ Dysthymia at $T_2$; $T_1$-$T_2$ MDD = since $T_1$ major depressive disorder at $T_2$; $T_1$-$T_2$ bipolar = since $T_1$ bipolar disorder at $T_2$; $T_1$-$T_2$ Dep NOS = since $T_1$ depression not otherwise specified at $T_2$; $T_2$ MDD = current major depressive disorder at $T_2$; $T_2$ Dep NOS = current depression not otherwise specified at $T_2$; $T_2$-$T_3$ dysthymia = since $T_2$ dysthymia at $T_3$; $T_2$-$T_3$ MDD = since $T_2$ major depressive disorder at $T_3$; $T_2$-$T_3$ bipolar = since $T_2$ bipolar disorder at $T_3$; $T_2$-$T_3$ Dep NOS = since $T_2$ depression not otherwise specified at $T_3$; $T_3$ dysthymia = current dysthymia at $T_3$; $T_3$ MDD = current major depressive disorder at $T_3$; $T_3$ bipolar = current bipolar disorder at $T_3$; $T_3$ Dep NOS = current depression not otherwise specified at $T_3$; lifetime dysthymia = history of dysthymia at $T_3$; lifetime MDD = history of major depressive disorder at $T_3$; current dysthymia = current dysthymia at $T_3$; current MDD = current major depressive disorder at $T_3$.

In the literature regarding adolescents and eating disorders, this is the second study to find that bulimia is associated with dysthymia, more so than with major depression. In the adult literature, the correlation between eating disorders and depression exceeds the correlation of all other Axis I disorders and bulimia (Braun et al., 1994; Brewerton et al., 1995; Zerbe et al., 1993), but the relationship between bulimia and dysthymia has not been investigated extensively. It is possible that in adults too, the comorbidity between dysthymia and bulimia exceeds that between major depression and bulimia—this possibility awaits future research.

It is also conceivable that comorbidity patterns change from adolescence to adulthood, such that dysthymia is more associated with bulimia in adolescence, whereas major depression is more associated with bulimia in adulthood. Such a change in comorbidity patterns from adolescence to adulthood (if the change exists) might be related to serial diminution of resources over time and, as a consequence, heightened risk for more numerous and severe depressive episodes. As the course of bulimia progresses, the social support network and resources of a bulimic individual may start to diminish, making negative life events harder to overcome. In addition, the binges and purges that serve as a type of coping mechanism in the beginning of the disorder may, over time, lose...
any palliative qualities, and their pernicious qualities may amplify. For these reasons, the intensity of the depression may increase with time, making the co-occurrence of acute depression and bulimia more likely in adulthood than in adolescence. This possibility represents an interesting avenue for future research.

Why would dysthymia be more associated than major depression with bulimia? Bulimic behavior has been construed as an attempt to regulate painful emotion (e.g., Heatherton & Baumeister, 1991). As pernicious as major depression can be, it tends to remit, even if untreated. By contrast, dysthymia is unrelenting, often lasting decades (indeed, the average episode length for dysthymia is more than 10 years; Shelton, Davidson, Yonkers, & Koran, 1997). Perhaps, the unique relation between dysthymia and bulimia involves the struggle to regulate unrelenting negative moods and self-views.

Relatedly, the link between dysthymia and bulimia might also be partly explained by self-esteem. Research has shown that dysthymics tend to have more pervasive and chronic low self-esteem even as compared with depressed individuals (Angst, 1998), who tend to have fluctuations of self-esteem that correspond to their depressive episodes (Sakamoto, Tomoda, & Kijima, 2002; Wallis, 2002). In this context, a recent psychological model of bulimia is interesting to consider (Vohs, Bardone, Joiner, & Abramson, 1999; Vohs et al., 2001). The model proposed that high perfectionism, when thwarted by unmet appearance-related goals and when accompanied by low self-efficacy, is predictive of bulimia. Dysthymics often report symptoms of low self-efficacy along with feelings of hopelessness and self-criticism. The chronic and pervasive self-esteem problems associated with dysthymia may render dysthymic people particularly vulnerable to bulimia (especially, according to the Vohs et al. model, if perfectionism and body dissatisfaction are also present).

These findings may also have significant clinical implications. The presence of dysthymia in adolescence might be a possible risk factor for the development of bulimia. As Zaider et al. (2000) reported, an adolescent who has dysthymia and subclinical disordered eating might be at greater risk for developing an eating disorder than someone who only has subclinical disordered eating. In addition, individuals with comorbid disorders usually have a worse course and prognosis in treatment than those without comorbidity (Nathan & Gorman, 1998). These findings can also provide additional information to create more focused treatments for adolescents with bulimia. Dysthymia can, at times, be masked by another disorder, such as bulimia, and can go untreated. Knowledge of the comorbidity between bulimia and dysthymia in adolescence can help therapists to assess specifically for dysthymia in bulimic clients and choose a treatment that will combat both disorders.

Several limitations to the current study must be discussed. First, a larger sample of bulimics would have strengthened the findings. Second, due to the small sample of anorexics, they were not included in the study. Previous research has found a correlation between anorexia and mood disorders, thus future research is warranted in this area.

Further studies are needed to replicate these findings. Establishing the specific depressive disorders that correlate with bulimia is important because MDD and dysthymia differ in terms of chronicity, severity, and persistence of the mood disturbance, factors that can impact the course and treatment of eating disorders.

REFERENCES