The Relation Among Stress, Psychological Symptoms, and Eating Disorder Symptoms: A Prospective Analysis

James C. Rosen
Bruce E. Compas
Barbara Tacy

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Prior research has shown that psychopathology and stress are associated with eating disorder symptoms, but the relations among these variables were confounded by concurrent measurement at a single point in time. The present study examined the relations among stress, psychological, and eating disorder symptoms prospectively over 4 months in 143 adolescent girls. In cross-sectional analyses at follow-up, controlling for baseline levels, stress and eating disorder symptoms each predicted changes in the other. Eating disorder symptoms were not predicted by psychological symptoms nor vice versa. Stress was predicted by eating disorder symptoms from the first assessment to follow-up. On balance, the results are somewhat stronger for the argument that eating disorder symptoms are predictive of subsequent psychological stress over short periods of time. © 1993 by John Wiley & Sons, Inc.

Stressful daily and major events are related prospectively to psychological and behavioral problems in adolescents (Compas, Howell, Phares, Williams, & Giunta, 1989; Wagnner, Compas, & Howell, 1988), the population most at risk for eating disorders. Stress is believed to be an important antecedent to eating disorders, in particular, because it may disrupt normal appetite regulation, intensify body image preoccupation, or provoke maladaptive coping responses such as binge eating (Cattanach & Rodin, 1988; Shatford & Evans, 1986). Indeed, studies of these two variables show that stress and eating disorder symptoms are positively correlated (Shatford & Evans, 1986; Wolf & Crowther, 1983). More clinically oriented samples of anorexia and bulimia nervosa subjects also show stress to be significant: it distinguishes cases from noneating disorder controls (Cattanach & Rodin, 1988; Greenberg, 1986; Kagan & Squires, 1984; Strober, 1984) and predicts relapse following treatment (Mitchell, Davis, & Goff, 1985). For similar reasons, other psychopathology, such as depressive disorder, is believed to be an important trig-
gering condition for eating disorders (Hudson, Pope, & Jonas, 1984). In fact, the co-
ocurrence of these disorders is high (Hinz & Williamson, 1987; Strober & Katz, 1988).

Although there appears to be a consistent association of stress, psychopathology, and eating disorder symptoms, these studies all employed cross-sectional designs that do not permit a positive conclusion about the chronology of these variables. Only a pros-
spective, longitudinal design is appropriate for testing hypotheses of causality. A few studies of eating disorders have taken steps in this direction. Garner, Garfinkel, Rock-
er, and Olmstead (1987) measured personality and body image at baseline to predict anorexia or bulimia nervosa 2 to 4 years later in female adolescent ballet students, however they did not control for initial eating disorder status. Patton, Johnson-Sabine, Wood, Mann, and Wakeling (1990) and Striegel-Moore, Silberstein, Frencsch, and Ro-
din (1989) studied adolescent girls who became eating disordered or showed a worsening of eating disorder symptoms by Time 2, as compared with subjects who remained the same. Having used change scores on psychological risk factors from the beginning to end of the year as their predictors, these studies essentially were cross-sectional in-
vestigations at Time 2.

The only truly prospective risk study to date was by Attie and Brooks-Gunn (1989). They attempted to predict eating disorder symptoms in adolescent girls 2 years after an initial assessment by controlling first for Time 1 symptoms and then entering a battery of Time 1 predictor variables, including measures of psychopathology at baseline. This study was more appropriate for questions of prediction over time.

In our opinion, however, there still are two important questions not examined pro-
spectively that this study was designed to address. First, what is the influence of both stress and psychopathology in triggering eating disorder symptoms? These are the two psychological risk factors most frequently cited in theories of eating disorder etiology and they should be tested together in a predictive study. Based on the literature, we hypothesize that each will add uniquely to the prediction of eating disorder symptoms when the stability of eating disorder symptoms over time is controlled.

The second question needing some attention is what is the temporal relation among these variables? Increases in psychological maladjustment are believed to trigger eating disorder symptoms. However, clearly, some maladjustment and stress in anorexia and bulimia nervosa patients are secondary to their eating disorders. Heretofore, no study has examined changes in psychological adjustment subsequent to eating disorder. We hypothesize that eating disorder symptoms will predict stress and psychological symp-
toms when the stability of psychological adjustment over time is controlled.

**METHOD**

**Subjects**

Subjects were girls recruited from three independent secondary boarding (preparato-
ry) schools located in the northeast of the United States. The study was explained in the regular weekly dormitory meetings and 162 out of the 248 students who were pre-
sent returned a signed consent form and completed questionnaire; 143 or 88% of these individuals also returned the Time 2 questionnaire. To analyze possible effects of attri-
tion, the scores on each of the measures at Time 1 were compared for those who par-
ticipated at both times and those who only participated at Time 1. These groups did not differ on age, socioeconomic status (SES), weight, or any of the questionnaires com-
pleted by the subjects.
The mean age of the participants was 15.92 years (SD = 1.12) and the grade distribution was: 9th = 15%, 10th = 26%, 11th = 36%, 12th = 23%. The sample was geographically varied in that the participants originated from 33 different states prior to enrolling in the schools. SES of the subjects’ families according to the Hollingshead procedure (1975) were: 27.7% in Class V (major business and professional), 45.4% in Class IV (medium business, minor professional), 20.3% in Class III (skilled craftsperson, clerical), 6.6% in Class II (semiskilled work), and 0% in Class I (unskilled laborer). Race distribution was 85% white, 10% black, 3% Asian, 1% Hispanic, and 1% other.

Measures

The subjects completed the questionnaires under supervision in their dormitories in January (Time 1) and May (Time 2). Four months between testing was selected so that the data collections would coincide with the beginning and end of the school semester when there were no exams or holidays.

Eating Disorder Symptoms

Subjects completed the 26-item version of the Eating Attitudes Test (EAT; Garner, Olmsted, Bohr, & Garfinkel, 1982) that taps into eating disorder symptoms of desire to lose weight, fear of fatness, avoidance of eating, binge eating, guilt over eating, preoccupation with eating and weight, finicky eating, and purging.

Stress

The subjects completed the middle adolescent version of the Adolescent Perceived Events Scale (APES), designed for high school ages (Compas, Davis, Forsythe, & Wagner, 1987), which asks the individual to indicate which of 205 major and daily life events occurred during the last 3 months. Each event that is endorsed is then rated on a scale of desirability ranging from very undesirable to very desirable and a scale for impact of the event, ranging from no impact to extremely high impact. The variable used here was the weighted negative events score that was calculated as the sum of the products of desirability and impact ratings for only those events that were rated in the undesirable direction. Test-retest reliability and concurrent validity have been shown to be adequate (Compas et al., 1987).

Psychological Symptoms

The Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982) was completed. This is a 53-item multidimensional scale on which subjects rate the degree to which a broad array of psychological symptoms had been distressing. The analyses used the Global Severity Index, an overall index of psychological distress that is computed by taking the mean of the severity ratings for all items. Adequate reliability and validity have been established (Derogatis & Spencer, 1982).

RESULTS

Descriptive Statistics and Simple Correlations

The mean Global Severity Index on the BSI corresponded to a T score of 56 at Time 1 (M = .97, SD = .62), based on norms for a nonclinical sample of female adolescents (Derogatis & Spencer, 1982). Using a T score of 63 or greater as the cutoff for clinically
significant level of symptoms (Derogatis & Spencer, 1982). 12.7% of the sample fell into the clinical range at Time 1. The mean EAT score at Time 1 corresponds to the 67th percentile ($M = 13.22, SD = 11.92$) for female adolescent norms (Rosen, Silberg, & Gross, 1988). Using the recommended cutoff score for the clinical range (extrapolated for the 26-item version; Garner & Garfinkel, 1979), 24.6% fell above this level at Time 1. Thus, the distributions for this sample on the BSI and the EAT were similar to or higher than the normative data presented for these measures.

The adolescents' Time 1 stress on the APES was not significantly related to the EAT at Time 1, $r = .13, p = .08$, however the correlation between stress at Time 2 and eating disorder symptoms at Time 2 was significant, $r = .33, p < .001$, indicating that more stressful events were associated with more severe eating disorder symptoms at that time. Psychological symptoms at Time 1 were significantly related to the EAT at Time 1, $r = .29, p < .001$; the more severe the psychological symptoms, the more intense the eating disorder symptoms. At Time 2, psychological symptoms and eating disorder symptoms were modestly but significantly correlated, $r = .19, p = .02$. Prospectively, simple correlations showed that neither Time 1 stress nor psychological symptoms predicted Time 2 eating disorder symptoms. However, Time 1 eating disorder symptoms predicted Time 2 stress and psychological symptoms, $r_s = .21$ and $r_s = .17, p < .05$.

**Effects of Stress and Psychological Symptoms on Eating Disorder Symptoms at Time 2 Controlling for Time 1 Eating Disorder Symptoms**

We examined stress (APES) and psychological symptoms (BSI) at Time 2 as predictors of eating disorder symptoms (EAT) at Time 2 after controlling statistically for Time 1 scores (4 months earlier) on the EAT. We entered the Time 1 EAT scores in the first step of a hierarchical multiple regression analysis. Stress and psychological symptoms at Time 2 were entered next, but no order was specified owing to the lack of data or theory with which to make this determination. This design examined the contribution of recent stress and psychological symptoms to changes in eating disorder symptoms over time. The unique variance accounted for by each predictor variable was tested when entered last, in order to make the most conservative estimate of the relations among these variables. These three predictors yielded an $R^2$ of .26, $F(3,117) = 13.34, p < .0001$. Both the baseline level of eating disorder symptoms, $s^2 = .151, p < .0001$, and Time 2 stress, $s^2 = .042, p < .01$, emerged as significant unique predictors of eating disorder symptoms at Time 2 (see Table 1).

**Effects of Eating Disorder Symptoms on Stress and Psychological Symptoms at Time 2, Controlling for Time 1 Stress and Psychological Symptoms**

We next examined eating disorder symptoms (EAT) at Time 2 as a predictor of stress (APES) and psychological symptoms (BSI) at Time 2 after controlling statistically for Time 1 scores on the APES and BSI. The overall $R^2$ for the prediction of psychological symptoms at Time 2 was .44, $F(3,117) = 29.29, p < .0001$, with the order of entry being psychological symptoms at Time 1, stress at Time 2, and eating disorder symptoms at Time 2. Stress was entered in the second step due to its established relation with psychological symptoms. Both the baseline level of psychological symptoms, $s^2 = .130, p < .0001$, and Time 2 stress, $s^2 = .283, p < .0001$, emerged as significant unique predictors of psychological symptoms at Time 2.

The concurrent relation of psychological symptoms and eating disorder symptoms at
Psychological Adjustment

Table 1. Cross-sectional analysis: prediction of adjustment at Time 2 controlling for Time 1 adjustment

<table>
<thead>
<tr>
<th>Variable (Time 2)</th>
<th>Incremental $R^2$ when Controlling for all Other Variables$^a$</th>
<th>Overall $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating disorder symptoms (Time 1)</td>
<td>Stress (Time 2)</td>
<td>Psychological symptoms (Time 2)</td>
</tr>
<tr>
<td>Psychological symptoms (Time 2)</td>
<td>Stress (Time 2)</td>
<td>Psychological symptoms (Time 1)</td>
</tr>
<tr>
<td>Stress (Time 2)</td>
<td>Stress (Time 1)</td>
<td>Psychological symptoms (Time 2)</td>
</tr>
</tbody>
</table>

$^a$Only statistically significant increments once other variables were controlled for \((squared \ semipartial \ correlations)\) are noted.

Time 2 (entered at second and third steps) to stress at Time 2 after controlling statistically for Time 1 stress (entered first in the regression) yielded an $R^2$ of .39, $F(3,117) = 21.38, p < .0001$. Eating disorder symptoms at Time 2 accounted for a significant amount of unique variance in stress at Time 2, $s_r^2 = .047, p = .006$, even beyond the incremental contribution of baseline stress, $s_r^2 = .028, p = .03$, and psychological symptoms at Time 2, $s_r^2 = .186, p < .0001$.

**Prospective Analysis: Effects of Stress and Psychological Symptoms at Time 1 on Eating Disorder Symptoms at Time 2**

This analysis tested the prediction of eating disorder symptoms (EAT) at Time 2 from stress (APES) and psychological symptoms (BSI) at Time 1, controlling for prior levels of eating disorder symptoms. This design is less affected by possible confounds between the dependent and independent variables than when they are assessed concurrently.

In order to control for prior levels of eating disorder symptoms (4 months earlier), we entered the Time 1 EAT scores in the first step of the regression. Stress and psychological symptoms at Time 1 were entered next with no order specified (see Table 2). These three predictors yielded an overall $R^2$ of .22, $F(3,117) = 10.80, p < .0001$. Only

Table 2. Prospective analysis: effects of adjustment at Time 1 on adjustment at Time 2

<table>
<thead>
<tr>
<th>Variable (Time 2)</th>
<th>Incremental $R^2$ when Controlling for all Other Variables$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating disorder symptoms</td>
<td>Eating Disorder Symptoms</td>
</tr>
<tr>
<td>Psychological symptoms</td>
<td>.213</td>
</tr>
<tr>
<td>Stress</td>
<td>.053</td>
</tr>
</tbody>
</table>

$^a$Only statistically significant increments once other variables were controlled for \((squared \ semipartial \ correlations)\) are noted.

$p = .06$. 
the baseline level of eating disorder symptoms was a significant unique predictor, $s^2 = .213$, $p < .0001$. Thus, stress and psychological symptoms did not contribute significantly to the prediction of future eating disorder symptoms after the stability of eating disorder symptoms over time was taken into account.

**Prospective Analyses: Effects of Eating Disorder Symptoms at Time 1 on Stress and Psychological Symptoms at Time 2**

Next, we examined Time 1 eating disorder symptoms (EAT) and stress (APES) as predictors of psychological symptoms (BSI) 4 months later (Time 2) with Time 1 psychological symptoms controlled (entered in Step 1 of the regression). Stress and eating disorder symptoms were entered in the second and third steps, respectively. These three predictors yielded an $R^2$ of .15, $F (3,117) = 6.84$, $p = .0003$. The baseline level of psychological symptoms was a significant predictor, $s^2 = .045$, $p = .01$, and the unique contribution of Time 1 stress approached significance, $s^2 = .026$, $p = .06$.

Finally, we examined the Time 1 eating disorder symptoms (EAT) and psychological symptoms (BSI) as predictors of stress (APES) 4 months later (Time 2) with Time 1 stress controlled (entered in the first step). Psychological symptoms and eating disorder symptoms at Time 1 were entered in the second and third steps, respectively. These three predictors yielded an $R^2$ of .20, $F (3,117) = 8.66$, $p < .0001$. Eating disorder symptoms at Time 1 accounted for a significant amount of unique variance in stress at Time 2, $s^2 = .053$, $p = .01$, even beyond the incremental contribution of baseline stress, $s^2 = .150$, $p < .001$, and psychological symptoms, $s^2 = .061$, $p = .006$.

In sum, after controlling for the baseline level of the dependent variable, the results of the prospective analyses indicate that stress and psychological symptoms do not contribute uniquely to the prediction of future levels of eating disorder symptoms. Eating disorder symptoms, on the other hand, contribute uniquely to the prediction of future levels of stress, but not psychological symptoms.

**Supplementary Analyses**

Although the simple correlations showed a concurrent and prospective relation between eating disorder and psychological symptoms, these relations failed to emerge in the regression analyses when the other predictor variables were controlled. This raises the possibility that these relations were accounted for in the regressions by the shared variance of one or both of these variables with stress. To further explore the relation between eating disorder and psychological symptoms, we conducted some additional regression analyses with the predictor variables entered in different orders. In each instance, the prior level of the dependent variable was controlled by entering that variable first in the equation.

With regard to the prediction of eating disorder symptoms, the results were essentially the same as reported above. That is, stress was related to changes in eating disorder symptoms concurrently, but not prospectively, regardless of the order in which the variables were entered. Psychological symptoms were not predictive of eating disorder symptoms, even when entered in the equation before stress. Thus, the variance in eating disorder symptoms explained by stress was not shared with psychological symptoms.

With regard to the prediction of psychological symptoms, the results for the prospective analysis were essentially the same as reported above. That is, stress was modestly
predictive of subsequent levels of psychological symptoms, but eating disorder symptoms were not. However, in the cross-sectional analysis at Time 2, eating disorder symptoms were predictive of changes in psychological symptoms when eating disorder symptoms were entered before stress (see Table 3). Eating disorder symptoms were not predictive when they were entered last. Thus, eating disorder symptoms were related to changes in psychological symptoms, but the variance explained by eating disorder symptoms was completely shared with stress.

**DISCUSSION**

The results of this study support prior observations that stress and psychological symptoms are associated with eating disorder symptoms concurrently according to simple correlations. However, the results indicate that some of the prospective relations are not as strong as had been suggested and that the directions of the relations are complex.

With regard to psychological symptoms and eating disorder symptoms, the results of both the cross-sectional and prospective regression analyses showed that neither variable was consistent in uniquely adding to the prediction of the other. (Attie and Brooks-Gunn [1989] also found that psychological symptoms were not predictive prospectively.) Only when eating disorder symptoms were entered in the cross-sectional regression equation prior to stress did a significant predictive relation between the two types of symptoms emerge. Thus, it does not appear that increases in psychological maladjustment will subsequently lead to more symptoms of eating disorders or vice versa. It seems that the two types of symptomatology may be more autonomous than is generally believed and that other psychological or biological mechanisms come into play to sustain these symptoms. In other words, a third factor might be responsible for the relation rather than either type of symptom contributing directly to the other. This possibility is suggested by the finding that stress added uniquely to the prediction of both variables and that the variance in psychological symptoms explained by eating disorder symptoms was completely shared with stress.

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Incremental $R^2$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross-sectional analysis</td>
<td>Dependent Variable: Psychological Symptoms (Time 2)</td>
<td></td>
</tr>
<tr>
<td>Stress entered last</td>
<td>Psychological symptoms (Time 1)</td>
<td>.120</td>
</tr>
<tr>
<td></td>
<td>Eating disorder symptoms (Time 2)</td>
<td>.033</td>
</tr>
<tr>
<td></td>
<td>Stress (Time 2)</td>
<td>.283</td>
</tr>
<tr>
<td>Eating disorder symptoms entered last</td>
<td>Psychological symptoms (Time 1)</td>
<td>.120</td>
</tr>
<tr>
<td></td>
<td>Stress (Time 2)</td>
<td>.316</td>
</tr>
<tr>
<td></td>
<td>Eating disorder symptoms (Time 2)</td>
<td>.001</td>
</tr>
</tbody>
</table>
symptoms was completely shared with stress. These findings are consistent with prior research that has suggested that symptoms of psychopathology are secondary to eating disorder symptoms (Cooper & Fairburn, 1986; Hinz & Williamson, 1987; Kaplan & Woodside, 1987; Wilson & Smith, 1987).

Although the data seem to indicate that eating disorder symptoms and psychological symptoms are linked to stress, rather than directly influencing each other, a reciprocal model of causation also should be considered. As others have argued, psychological maladjustment might increase vulnerability to initial eating disturbances and overconcern with appearance (Striegel-Moore, Silberstein, & Rodin, 1986). Subsequent changes in cognitive, affective, and behavioral functioning could precipitate from eating disorder symptoms. Over time the two types of symptomatology might become tightly intertwined and mutually sustaining. A repeated assessment of the two types of symptomatology over time with more than two measurement points would be necessary to establish such a reciprocal model of causation.

With regard to stress and eating disorder symptoms, these findings clearly indicate that eating disorder symptoms contribute to increased stress over both short and relatively longer periods of time, whereas the contribution of stress to eating disorder symptoms is more immediate. At Time 2, when the relation was examined cross-sectionally controlling for baseline levels of the dependent variable, the relation between stress and eating disorder symptoms was bidirectional. That is, stress predicted increased eating disorder symptoms, and eating disorder symptoms predicted increased stress. However, when the relation was examined over a period of 4 months, stress was more a consequence of eating disorder symptoms than vice versa. Specifically, past (Time 1) and more recent (Time 2) symptoms of eating disorders were uniquely predictive of stress. However, only the most recent level of stress (Time 2) was predictive of eating disorder symptoms; more remote stressors (Time 1) were not predictive of subsequent eating disorder symptoms. Therefore, on balance, the results are somewhat stronger for the argument that eating disorder symptoms are predictive of psychological stress. Although stressful events may not serve as an early marker of increased risk for eating disorder symptoms, recent stressors are clearly associated with changes in eating disorder symptoms over time. Given that stress was uniquely related to symptoms of both psychopathology and eating disorders, it may very well be a third factor that underlies the relation between the two types of symptoms. For example, eating disorder symptoms might predict psychological symptoms through the mediation of their stressful effects. A prospective, longitudinal design that measures these variables at three points in time would be necessary to test this hypothesis.

Some shortcomings of the present study deserve mention. The sample was restricted to adolescents residing in preparatory schools and this presented a set of unique circumstances including that the subjects shared a common living and eating situation, they were living away from home, and they may have been more than generally academically motivated. The importance of these factors with respect to generalizing the findings to other adolescents is uncertain. It should be reiterated at this point, however, that in spite of these differences, the sample exhibited normal levels of psychological and eating disorder symptoms. A second issue is that measurement of psychological symptoms, eating disorder symptoms, and stress was conducted using self-report questionnaires. Questionnaires of eating disorder symptoms, in particular, have been criticized as being superficial and limited in their ability to distinguish the extreme eating, weight, and dieting attitudes and behaviors of women with eating disorders from those that are more widely practiced in our weight conscious society (Ros-
levels of disorder. Finally, we have noted that some concepts of causality in eating disorders among these variables would be the same for people with clinically significant distant predictors of eating disorder symptoms. Fourth, because we studied a group of subjects with moderate levels of stress and symptoms, we cannot be certain that the relations among these variables would be the same for people with clinically significant levels of disorder. Finally, we have noted that some concepts of causality in eating disorders require a research design that utilizes multiple, concurrent assessments of variables over a longer time. These models deserve further attention in future efforts to understand pathological processes in these serious disorders.

REFERENCES


