Vulnerable Self-Esteem and Depressive Symptoms: Prospective Findings Comparing Three Alternative Conceptualizations

John E. Roberts
University of Pittsburgh

Scott M. Monroe
University of Oregon

Across diverse theoretical orientations, vulnerable self-esteem (SE) is thought to act as a diathesis for depression after life stress. In the present study, the roles of trait-level SE, low SE primed by depressed mood, and labile SE in prospectively predicting changes in depressive symptoms in a nonclinical sample \( n = 192 \) were examined. Results indicated that labile SE predicted increases in symptoms. Furthermore, a 3-way interaction (Labile SE \( \times \) Academic Stress \( \times \) Initial Depression) suggested that in Ss who were initially asymptomatic, lability made Ss differentially vulnerable to increases in depressive symptoms after stress. In contrast to labile SE, trait-level SE and priming of low SE were relatively weak predictors of changes in depressive symptoms and did not interact with stress.

Within diverse theoretical orientations for depression, vulnerable self-esteem (SE) plays a pivotal role in the onset, maintenance, and recurrence of the disorder (e.g., Beck, 1967; Bibring, 1953; Brown & Harris, 1978; Rado, 1928). Such theories typically posit that depression is triggered by negative life events interacting with a vulnerable SE predisposition. The exact nature of vulnerable SE, however, has been controversial. Three major conceptualizations of the construct can be gleaned from the literature.

In the first, persons predisposed to depression are thought to experience chronically low trait SE. Most empirical investigations of SE (and related constructs; e.g., negative self-schemata) are based on such a viewpoint and, consequently, examine level of SE (e.g., high vs. low SE). Although many of these are prospective studies, SE is only measured at one point in time in predicting depression and is thus treated as a relatively stable trait (e.g., Brown, Bifulco, Harris, & Bridge, 1986; Hammen, Marks, deMayo, & Mayol, 1985; Miller, Kreitman, Ingham, & Sashidharan, 1989).

Teasdale’s (1983, 1988) differential activation theory represents the second conceptualization of vulnerable SE. It suggests that persons vulnerable to depression harbor negative views of themselves that lie dormant until “primed” by depressed mood. Persons are thought to vary in the extent to which low SE is activated by negative mood. This variability is believed to account for whether the negative mood spirals down into a clinical depression or simply fades away.

Like trait-level SE, differential activation of low SE could be measured at a single point in time. However, SE would be informative only in subjects primed by depressed mood (i.e., there is an SE effect only when subjects have already become somewhat depressed). There are two growing bodies of support for differential activation: first, studies that demonstrate that negative self-cognition predicts persistence of an already existing depressive disorder (Dent & Teasdale, 1988; Lewinsohn, Steinmetz, Larson, & Franklin, 1981; Williams, Healy, Teasdale, White, & Paykel, 1990); and second, studies that examine the relationship between cognition and mood in formally depressed persons (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990; Teasdale & Dent, 1987). The latter studies have found that negative self-cognition is correlated with mood, but only in persons who have a history of clinical depression.

Rado’s (1928) psychoanalytic theory of depression laid the framework for the final view of vulnerable SE, suggesting that premorbid depressives exhibit labile SE. According to this view, depressives rely to an inordinate degree on the love and approval of significant others to maintain their SE. When such external sources of self-worth are present, the future depressive will have a normal level of SE. Only when these “narcissistic objects” are lost does the depressive show abnormally low SE. More generally, future depressives are seen as basing their self-worth on fewer and less stable sources than nondepressives do (Arieti & Bemporad, 1978; Barnett & Gotlib, 1988; Otley & Bolton, 1985). Therefore, their SE is thought to be highly reactive to daily stressors.

Within this theory, vulnerable persons would not necessarily be characterized by low SE at any particular point in time. However, over time, future depressives’ SE would be highly reactive to life’s vicissitudes and would therefore appear labile. Temporal instability in SE, then, might be expected to mark future depressives relative to the psychologically healthy (Barnett & Gotlib, 1988).

In the present study we used a prospective design with multiple assessments of SE and depressive symptoms that provided a simultaneous test of these three alternative conceptualizations of vulnerable SE. Vulnerable SE and depression were investigated in an academic stress paradigm. Disappointment in performance on a college examination was used as a single, temporally well-defined stressor (Follette & Jacobson, 1987; Hunsley,

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Correspondence concerning this article should be addressed to John E. Roberts, Department of Psychology, 4013 O’Hara Street, University of Pittsburgh, Pittsburgh, Pennsylvania 15260.
1989; Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982; Metalsky, Halberstadt, & Abramson, 1987). We were interested in both the main effects of vulnerable SE, as well as in its interactions with this stressor, to predict prospectively depressive symptoms.

Method

Subjects

Subjects were 216 university undergraduate students enrolled in introductory psychology courses. To test this study’s major hypotheses, we needed a full range of depressive symptoms, and subjects were thus not screened for elevated levels of depression. The confidentiality of information was emphasized throughout the experiment.

Overview

Two hundred sixteen individuals were administered questionnaires (a) nine times during a 3-week baseline period before their midterm exams and (b) three times during a 1-week follow-up period. Subjects returned questionnaires each day their psychology classes met (three times a week). Of this initial pool, 24 subjects were dropped from subsequent analyses because of failure to comply with instructions, leaving a final sample of 192. During both the baseline and follow-up periods, measures of SE, depressive symptoms, and importance of academic achievement were taken (these are described more fully below). Figure 1 diagrams this procedure. Subjects were debriefed after the final assessment had been completed.

Measures

Self-esteem. The Rosenberg Self-Esteem Scale (RSE) is a measure of global self-regard consisting of 10 items with good face validity (Rosenberg, 1965). Consistent with other research, this scale was scored according to the Likert format (Rosenberg, 1979). Two-week test-retest reliabilities of .85 and .88 have been reported (Rosenberg, 1979). The RSE correlates well with other measures of SE (Demo, 1985), as well as with other theoretically related variables (Wylie, 1974). In the present sample, coefficient alphas ranged from .85 to .92 across the 12 assessments.

Beck Depression Inventory (BDI). The BDI is a widely used instrument and has been shown to be a valid measure of depressive symptoms among college students (Bumby, Olver, & McClare, 1978). It possesses a split-half reliability of .93 and has been judged to correlate satisfactorily with ratings of depression severity made by independent clinicians (Beck, 1967; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Academic stress. Three weeks before an important exam, subjects were asked, “On my upcoming Introductory Psychology exam, I hope / expect to get a/an .” They indicated one of 13 possible choices (A+, A, A−, . . . , F) and these were converted to a numerical scale. Subjects had already received grades from an earlier psychology exam and therefore had some realistic basis for making this judgment. Their actual performance (self-reported) was converted to the same numerical scale. Subjects were also asked how important academic achievement was relative to other life domains on a 4-point scale (1 = much less important; 4 = much more important). An index of stress was constructed to measure the degree of stress associated with the receipt of the exam grade by subtracting actual performance from expected or hoped for performance. This discrepancy score was further weighted by being multiplied by the importance of academic achievement (measured at the ninth assessment—T9). Although relatively uncommon in our sample (6.6%), this scale also captures “positive stress” (i.e., performance that exceeds expectations and hopes). More common were subjects who did as well (or as poorly) as expected (23.2%). This procedure is similar to those used by other researchers using academic stress paradigms (e.g., Follette & Jacobson, 1987; Hunsley, 1989; Metalsky et al., 1982, 1987).

This relatively objective procedure avoids some of the methodological pitfalls that have plagued research in life stress, particularly confounds between the perception of stress and symptom status (Depue & Monroe, 1986; Monroe & Peterman, 1988). In the current study, depressive symptoms or vulnerable SE might very well influence how subjects perceive the stress in their lives, blurring the boundaries between stress, vulnerability, and outcome. This procedure helps guard against such possibilities. At the same time, weighted discrepancy scores theoretically tap individual differences in the meaning of the stressful experience. For example, getting a C would be treated as more stressful if one had expected or hoped for an A than for a B−, especially if academic achievement was very important for the person. Finally, of all the dimensions of stress associated with taking college exams (e.g., time pressure, conflicting demands, and excessive responsibilities), academic disappointment might be one of the most salient in terms of threats to SE.

We were interested in examining the effects of a single, discrete, well-defined event. We wanted to assure that this stressful experience truly was an objective event that was comparable across subjects (see Monroe & Roberts, 1990, 1991) and that it antecedes depressive reactions. Such a measure can be criticized as providing a limited portrait of the stress in these subjects’ lives, yet we thought it valuable to complement research using more comprehensive stress assessment with work more focused on specific, well-defined stressors.

Design and Analyses

A multiassessments, prospective design was used to provide a simultaneous test of the three alternative conceptualizations of vulnerable SE. As indicated above, a 3-week baseline period consisting of nine assessments was used to score subjects on trait-level SE, differential activation of low SE under depressed mood, and labile SE. These variables were predictors of change in depressive symptoms on their own and in interaction with academic stress on the day exam grades were received (T11) and about 2 days later (range of 2–3 days; T12).

Trait-level SE was operationalized as subjects’ SE scores at T9 (about 2 days before exams—range of 2–3 days), as well as aggregated SE scores over the entire baseline (T1–T9), whereas differential activation was operationalized as an initial Depression × SE interaction, again using scores at T9 and aggregations across the baseline. Within-subject variance scores in baseline SE were used as a measure of lability (Kernis, Grannemann, & Barclay, 1989). See Table 1 for an outline of these conceptualizations and how each was operationalized.

Setwise hierarchical multiple regression analyses were conducted to
Table 1
Conceptualization and Operationalization of Vulnerable Self-Esteem (SE)

<table>
<thead>
<tr>
<th>Conceptualization</th>
<th>Operationalization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trait-level SE: Chronically low SE predisposes to depression.</td>
<td>Level of SE at T9 or SE aggregated over baseline</td>
</tr>
<tr>
<td>Differential activation:</td>
<td>Two-way interaction between initial BDI score and SE depressed ranges of mood.</td>
</tr>
<tr>
<td>Depressed mood &quot;primes&quot; low SE, SE is only predictive within already mildly depressed ranges of mood.</td>
<td></td>
</tr>
<tr>
<td>Labile SE: Temporally unstable SE predisposes to depression.</td>
<td>Within-subject variance scores on SE across the baseline</td>
</tr>
</tbody>
</table>

Note. T9 = the ninth assessment.

test major hypotheses (see Cohen & Cohen, 1983). Separate equations were used in predicting changes in depressive symptoms during each follow-up period (T11; the day subjects received their grades, and T12, about 2 days after they received their grades).

Most generally, initial depression (BDI at T9) was entered first, followed by the set of vulnerable SE and academic stress entered simultaneously. Next, the set of all relevant two-way interactions were entered simultaneously (including the interaction between vulnerable SE and stress). Because there is evidence that various predictor variables show different relationships to depression according to initial level of depression (Cutrona, 1983; Depue & Monroe, 1986; Hammen, Mayol, deMayo, & Marks, 1986; Monroe, 1982; Monroe, Bromet, Connell, & Steiner, 1986), the three-way interaction among vulnerable SE, stress, and initial depression was entered last.1

As recommended by Cohen and Cohen (1983), we represented residual change scores by partial correlations (prs) after controlling for initial depression. Such partial correlations reflect the contribution of predictor variables to the variance that cannot be accounted for by initial depression (see also Metalsky et al., 1987).

Results

Descriptive Statistics

The final sample consisted of 122 women and 70 men. There were no gender differences on lability of SE or academic stress. However, at T9 women tended to be more depressed (M = 6.45 vs. 3.86, t = 3.23, p < .01) and to have lower SE (M = 30.90 vs. 32.84, t = 2.62, p < .05). Women were also more labile over time (M = 18.24 vs. 9.43, t = 2.35, p < .05).2 Subjects' ages ranged from 17 to 51 years (M = 18.7, SD = 2.7), although the majority were 18 years old (63.8%). Age did not significantly correlate with depression or any of the predictor variables.

The means and standard deviations of central variables are presented in Table 2, and their correlation matrix can be found in Table 3. The mean BDI score over the entire baseline period (T1 through T9) was 6.48 (SD = 5.87). Twenty-eight percent (n = 52) of the sample had average BDI scores of 9 or above during the baseline period. Within the time periods of most interest, BDI scores remained stable. The zero-order correlation between T9 BDI and T11 BDI was .76, p < .001, and between T9 BDI and T12 BDI was .78, p < .001, for the sample as a whole. The mean BDI score was 5.50 (SD = 6.05) at T9, 5.48 (SD = 6.79) at T11, and 5.05 (SD = 5.69) at T12. Repeated measures analysis of variance (ANOVA) indicated that these scores did not statistically differ from each other, Wilk's A = 0.98, p < .16. As would be expected, SE correlated with depression (at T9, r = −.52, p < .001). Examination of the correlations between the index of academic stress, as well as its components, with SE and the BDI reveals that there is virtually no confounding in this measure of stress. Academic stress appears to be independent of initial psychological functioning.

Trait-Level Self-Esteem

As can be seen in Table 4, the set of SE at T9 and academic stress was a significant predictor of residual change in depressive symptoms from T9 to T11, p < .05. Within-set analyses revealed that SE was solely responsible for the significance of the set, uniquely accounting for 26% of the variance in residual change, R2(175) = 2.17, p < .05. Those who showed lower SE experienced greater depression the day they received their grades (T11). However, in contrast to T11 predictions, the set of SE at T9 and academic stress did not make a significant contribution to the prediction of T12 BDI scores (see Table 5).

Of most importance to the vulnerability hypothesis, the two-way interaction between academic stress and SE at T9 was not a significant predictor of depression at either T11 (see Table 4) or T12 (see Table 5). Although the set of all two-way interactions was a significant predictor of residual change in depressive symptoms from T9 to T11, F(3, 172) = 3.78, p < .05, within-set analyses revealed that the interaction between SE and academic stress was not a significant contributor, F(172) = 1.06, p < .30. Thus, low SE at T9 did not make subjects differentially vulnerable to depressive reactions in response to academic stress.

Aggregation of SE across the nine baseline assessments did not enhance these predictions at either T11 or T12 after average baseline depression was controlled. Aggregated SE, stress, and their interaction were not significant predictors of residual change in depressive symptoms.

Further investigation of subjects with relatively stable SE (scored below the median on labile SE) provided an even stronger test of chronic low SE. With these subjects, the set containing SE at T9 and academic stress failed to reach significance. Most important, both SE at T9 and aggregated SE failed to interact with academic stress.

In summary, as a main effect, SE at T9 made a significant, albeit small, contribution to the prediction of residual change in depressive symptoms from T9 to T11, but not from T9 to T12. It is important that none of the SE × Academic Stress interactions were significant in predicting either T11 or T12

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1 In the current study, this procedure is not feasible for the trait-level SE analyses. The three-way interaction among stress, SE, and initial depression actually tests differential activation.

2 To control for possible sex differences in changes in depressive symptoms, primary analyses were conducted partialing out gender. Results of these analyses were essentially identical to those that did not control for gender. In addition, separate analyses were conducted for male and female subjects. In brief, these analyses suggested that labile SE played a stronger role in women than in men.
Table 2
Means and Standard Deviations for Central Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI at T9</td>
<td>191</td>
<td>5.50</td>
<td>6.05</td>
</tr>
<tr>
<td>BDI at T11</td>
<td>188</td>
<td>5.48</td>
<td>6.79</td>
</tr>
<tr>
<td>BDI at T12</td>
<td>190</td>
<td>5.05</td>
<td>5.69</td>
</tr>
<tr>
<td>Aggregated BDI</td>
<td>189</td>
<td>6.48</td>
<td>5.87</td>
</tr>
<tr>
<td>Labile BDI</td>
<td>189</td>
<td>15.07</td>
<td>28.21</td>
</tr>
<tr>
<td>SE at T9</td>
<td>192</td>
<td>31.61</td>
<td>5.12</td>
</tr>
<tr>
<td>SE at T11</td>
<td>187</td>
<td>31.46</td>
<td>5.33</td>
</tr>
<tr>
<td>SE at T12</td>
<td>190</td>
<td>31.80</td>
<td>5.14</td>
</tr>
<tr>
<td>Aggregated SE</td>
<td>190</td>
<td>31.31</td>
<td>4.79</td>
</tr>
<tr>
<td>Labile SE</td>
<td>190</td>
<td>6.49</td>
<td>10.14</td>
</tr>
<tr>
<td>Expect-hope</td>
<td>187</td>
<td>10.79</td>
<td>1.62</td>
</tr>
<tr>
<td>Importance</td>
<td>192</td>
<td>2.91</td>
<td>0.69</td>
</tr>
<tr>
<td>Actual grade</td>
<td>185</td>
<td>8.34</td>
<td>2.78</td>
</tr>
<tr>
<td>Academic stress</td>
<td>181</td>
<td>7.02</td>
<td>8.09</td>
</tr>
</tbody>
</table>

Note. BDI = Beck Depression Inventory; T9, T11, and T12 = the 9th, 11th, and 12th assessments; SE = self-esteem; expect-hope = grade subject “expected/hoped” to receive on an upcoming exam; importance = value of academic achievement; actual grade = grade subject reported receiving. Academic stress = discrepancy score between expected–hoped for grade and grade actually received, weighted by importance of academic achievement.

depression, even when aggregated measures of SE were used and stable, low SE was considered.

Differential Activation

Support for this hypothesis would come from finding that (a) those who were initially relatively depressed and who showed lower than expected SE given their initial level of depression would show the greatest residual increases in depression during follow-up (SE × Initial Depression interaction); and (b) academic stress had its major impact on those who were initially relatively depressed and who showed lower than expected SE given their initial level of depression (SE × Initial Depression × Academic Stress interaction).

As can be seen in Table 4, the set of two-way interactions was a significant predictor of residual change in depressive symptoms from T9 to T11, p < .05. Within-set analyses revealed that the Initial Depression × SE interaction accounted for the significance of this set, t(172) = 3.24, p < .01, uniquely accounting for 5.8% of the variance in residual change in depressive symptoms between T9 and T11.

Follow-up analyses on this two-way interaction were conducted by dividing the sample into high and low initial depression subgroups based on a median split (BDI ≤ 4 and BDI > 4 at T9) and examining the main effects of SE in each. These analyses supported the differential activation hypothesis by finding a significant effect for SE in the high initial depression subgroup, F(1, 83) = 4.95, p < .05, pr = -.24, but not in the low initial depression subgroup, F(1, 98) = 0.00. SE exhibited a greater effect on changes in depressive symptoms in those who were already mildly depressed. Those with higher SE demonstrated greater reductions in depressive symptomatology.

As can be seen in Table 5, the set of two-way interactions did not significantly predict residual change from T9 to T12. Likewise, the three-way interaction among initial level of depression, SE, and academic stress was not statistically significant in predicting either T11 or T12 depressive symptoms.

As was the case in the low-trait SE analyses, aggregation of SE across the entire baseline period did not lead to stronger predictions after average baseline affective functioning was controlled. In fact, the Aggregated SE × Initial Depression interaction was nonsignificant.

In summary, there was no support for the idea that differential activation acted as a vulnerability factor for increases in depressive symptoms that followed academic stress. However, evidence was found that SE may be important in the recovery from (and conversely, exacerbation of) mild depression.

Labile Self-Esteem

Although the main effects of lability are of theoretical interest, the two-way interactions between lability and academic stress, and the three-way interactions among lability, academic stress, and initial depression provide stronger tests of this model.

As can be seen in Tables 6 and 7, the set of labile SE and academic stress made a significant contribution in predicting depressive symptoms at both T11 and T12 (p < .01 in each). Within-set analyses indicated that lability accounted for the significance of the set in each time period: T11 (4.9% of variance in residual change), t(173) = 2.98, p < .01, and T12 (4.7% of variance in residual change), t(173) = 2.93, p < .01. At both time periods, greater lability prospectively predicted increases in depressive symptoms.

Although the set of two-way interactions was nonsignificant at both time periods, the three-way interaction among labile SE, academic stress, and initial depression predicted depressive symptoms at both T11 (4.3% of variance in residual change, p < .01) and T12 (9.2% of variance in residual change, p < .001). Two levels of follow-up analyses were conducted to further investigate these three-way interactions. First, the two-way interaction between academic stress and labile SE was examined separately for high and low initial depression groups (based on a median split of the BDI at T9). Second, significant two-way interactions were followed up by further subdividing subjects into academic failure (performing worse than one's hopes and expectations) and academic nonfailure (performing as well as or better than one's hopes and expectations) groups, and the main effect of labile SE was examined in each.

Low initial depression group. For the low initial depression group, the Labile SE × Academic Stress interaction was a significant predictor of residual change in depression from T9 to T11. This effect seems to be accounted for primarily from the endorsement of the positive SE items, rather than from the denial of negative items. When the five-item positive SE subscale was entered into the equation, its interaction with initial depression predicted changes in symptomatology more strongly than the global scale's interaction (14.6% of variance in residual change), t(172) = 5.43, p < .001. See Brown, Bifulco, & Andrews (1990) for interesting parallels.

4 This effect was most prominent in the most highly depressed subjects as indicated by an Initial Depression × Initial SE interaction in these mildly depressed subjects, F(1, 82) = 10.36, p < .01, pr = -.34.
T11, $F(1, 83) = 16.38, p < .001, pr = .41$. Follow-ups on this two-way interaction suggested that for the initially nondepressed, labile SE predicts depression under conditions of academic failure, $F(1, 60) = 66.29, p < .001, pr = .73$, but not nonfailure, $F(1, 22) = 2.43, p < .14$. In addition, the Labile SE $\times$ Academic Stress interaction was a significant predictor of residual change in depression from T9 to T12, $F(1, 82) = 18.03, p < .001, pr = .43$. Follow-ups on this two-way interaction again suggested that, for the initially nondepressed, labile SE predicts depression under conditions of academic failure, $F(1, 58) = 24.78, p < .001, pr = .55$, but not nonfailure, $F(1, 23) = 0.10, p < .76$.

High initial depression group. For the high initial depression group, the Labile SE $\times$ Academic Stress interaction was a non-significant predictor of residual change from T9 to T11, $F(1, 84) = 0.90$. However, this interaction was a significant predictor of residual change from T9 to T12, $F(1, 85) = 9.60, p < .01, pr = −.32$. Unexpectedly, follow-ups on this two-way interaction suggested that labile SE predicts depression under conditions of academic nonfailure, $F(1, 25) = 8.95, p < .01, pr = .51$, but not under conditions of academic failure, $F(1, 59) = 2.01, p < .17$. Under conditions of academic nonfailure, labile SE predicted higher levels of depressive symptoms.

Conservative analyses. Additional analyses were conducted to examine whether labile SE showed effects after statistically controlling for related variables (e.g., lability in depressive symptoms), as well as whether interactions remained significant after controlling for nonlinear trends in each of their component variables (see Lubinski & Humphreys, 1990). In these conservative analyses, T9 depression was entered first, followed by the set of SE at T9, SE at T9 squared (controls for nonlinear relations between SE and labile SE), lability in depression (individual's variance score on the BDI across the baseline), and academic stress. Labile SE was entered next. To control for nonlinear trends potentially associated with interaction terms, the set of BDI at T9, academic stress, and labile SE, each squared (see Cohen & Cohen, 1983; Lubinski & Humphreys, 1990), was entered simultaneously, followed by the set of all relevant two-way interactions. The Labile SE $\times$ Academic Stress $\times$ Initial Depression interaction was entered last.

Although the main effect of labile SE was nonsignificant in predicting changes in depression at either follow-up period, labile SE continued to show significant three-way interactions with academic stress and initial depression, $F(1, 163) = 4.12, p < .05, pr = −.16$, at T11, and $F(1, 163) = 13.00, p < .001, pr = −.27$, at T12.

In summary, labile SE prospectively predicted changes in depressive symptoms alone and in interaction with academic stress and initial depression. However, the main effect of labile SE was not independent of its association with related variables (T9 SE, T9 SE squared, academic stress, and lability in depression). It is important that the interactions among labile SE, academic stress, and initial depression were not due to multicollinearity among the product terms (interactions) and the quadratic terms of their components (Lubinski & Humphreys, 1990) and held after related variables (e.g., lability in depression) were controlled. Labile SE, however, appeared to act as a diathesis primarily in the initially asymptomatic.
Table 4

Regression Analysis Predicting BDI Score at the 11th Assessment (Trait-Level Self-Esteem and Differential Activation Analyses)

<table>
<thead>
<tr>
<th>Order of entry of set</th>
<th>Predictors in set</th>
<th>(F) for increment</th>
<th>(t) for within-set predictors</th>
<th>(df)</th>
<th>Partial correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>T9 BDI (k = 1)</td>
<td>246.88***</td>
<td>1, 177</td>
<td>.76</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Entire set (k = 2), T9 SE, Academic Stress</td>
<td>3.20*</td>
<td>3, 175</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Entire set (k = 3), T9 SE × Academic Stress, T9 BDI × Academic Stress, T9 SE × T9 BDI</td>
<td>3.78*</td>
<td>3, 172</td>
<td>-.16</td>
<td>1.08</td>
</tr>
<tr>
<td>4</td>
<td>T9 SE × Academic Stress × T9 BDI (k = 1)</td>
<td>1.39</td>
<td>1, 171</td>
<td>-.09</td>
<td></td>
</tr>
</tbody>
</table>

Note. T9 = 9th assessment; BDI = Beck Depression Inventory; k = number of predictors in set; SE = self-esteem. Academic stress = discrepancy score between expected–hoped for grade and grade actually received, weighted by importance of academic achievement.
* \(p < .05\). ** \(p < .01\). *** \(p < .001\).

Discussion

This study was conducted to provide a simultaneous test of three alternative conceptualizations of vulnerable SE. It examined which qualities of SE (trait-level SE, differential activation of low SE, and labile SE) prospectively predicted changes in depression, either alone or in interaction with academic stress. Previous theory has portrayed vulnerable SE as a diathesis that makes one more vulnerable to depressive reactions in the face of environmental adversity. The interactions between SE variables and stress were thus most important. However, main effects of SE variables were also of interest because SE might lead to changes in depression on its own (or in concert with stressors that were not assessed in this study).

Overall, this study demonstrates the viability of labile SE as a vulnerability factor for depressive symptoms. Labile SE prospectively predicted increases in depressive symptoms in both follow-up periods after initial depression was partialed out. Of even greater interest, labile SE acted as a moderator between academic stress and depressive reactions as a function of initial depression (Labile SE × Academic Stress × Initial Depression interaction). In a conservative analysis, this interaction predicted depressive symptoms in both follow-up periods after initial depression, initial SE (both linear and curvilinear effects), lability in depression, and nonlinear components of the interaction term were all statistically controlled.

The form of these three-way interactions suggests that labile SE acted as a diathesis to stress reactions primarily in the initially asymptomatic. Among these subjects, labile SE predicted increases in depression only after academic stress. Without this stressor, labile SE was unrelated to changes in depression. Labile SE seemed to play a different role in those who were initially somewhat depressed. Under these conditions, labile SE predicted increases in depression under low academic stress, but not high. In contrast, the diathesis hypothesis predicts that labile SE would predict increases under high stress more strongly.

Although admittedly difficult to interpret, these latter results suggest that with the mildly depressed, labile SE represents relatively healthy functioning in response to stress. Interestingly, subjects who exhibit low lability compose a heterogeneous group consisting of subjects who range from consistently high SE to consistently low SE. Because of the negative correla-

Table 5

Regression Analysis Predicting BDI Score at the 12th Assessment (Trait-Level Self-Esteem and Differential Activation Analyses)

<table>
<thead>
<tr>
<th>Order of entry of set</th>
<th>Predictors in set</th>
<th>(F) for increment</th>
<th>(t) for within-set predictors</th>
<th>(df)</th>
<th>Partial correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>T9 BDI (k = 1)</td>
<td>267.38**</td>
<td>1, 177</td>
<td>.78</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Entire set (k = 2), T9 SE, Academic Stress</td>
<td>2.35</td>
<td>2, 175</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Entire set (k = 3), T9 SE × Academic Stress, T9 BDI × Academic Stress, T9 SE × T9 BDI</td>
<td>2.10</td>
<td>3, 172</td>
<td>-.11</td>
<td>.13</td>
</tr>
<tr>
<td>4</td>
<td>T9 SE × Academic Stress × T9 BDI (k = 1)</td>
<td>0.02</td>
<td>1, 171</td>
<td>.01</td>
<td></td>
</tr>
</tbody>
</table>

Note. T9 = 9th assessment; BDI = Beck Depression Inventory; k = number of predictors in set; SE = self-esteem. Academic stress = discrepancy score between expected–hoped for grade and grade actually received, weighted by importance of academic achievement.
* \(p < .05\). ** \(p < .001\).
Table 6
Regression Analysis Predicting BDI Score at the 11th Assessment (Labile Self-Esteem Analyses)

<table>
<thead>
<tr>
<th>Order of entry of set</th>
<th>Predictors in set</th>
<th>( F ) for increment</th>
<th>( t ) for within-set predictors</th>
<th>( df )</th>
<th>Partial correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>T9 BDI (k = 1)</td>
<td>244.09***</td>
<td>1, 175</td>
<td>.76</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Entire set (k = 2), Labile SE, Academic Stress</td>
<td>5.30**</td>
<td>2, 173</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Entire set (k = 3), Labile SE × Academic Stress, T9 BDI × Academic Stress, Labile SE × T9 BDI</td>
<td>0.67</td>
<td>3, 170</td>
<td>.07</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Labile SE × Academic Stress × T9 BDI (k = 1)</td>
<td>7.66**</td>
<td>1, 169</td>
<td>-.21</td>
<td></td>
</tr>
</tbody>
</table>

Note. T9 = 9th assessment; BDI = Beck Depression Inventory; k = number of predictors in set; SE = self-esteem. Academic stress = discrepancy score between expected–hoped for grade and grade actually received, weighted by importance of academic achievement.

* \( p < .05 \)  ** \( p < .01 \)  *** \( p < .001 \)

The correlation between SE and depression (\( r = -.52 \), stable SE in the mildly depressed would most likely consist of stable low SE, whereas stability in the asymptomatic would consist of stable high SE. Therefore, labile SE might represent a less ingrained negative self-image in the mildly depressed that may relate to better coping with stress.

Regardless of interpretation, this study again demonstrates the importance of attending to initial level of depression (see Barnett & Gotlib, 1988; Depue & Monroe, 1986; Monroe, 1982). Those who were already mildly depressed showed quite different results than those who were initially asymptomatic.

In contrast to labile SE, there was no evidence that trait-level SE or differential activation moderates the impact of academic stress. Neither of these variables acted as a diathesis for depressive reactions following academic stress, even when aggregated across the baseline. However, there was a main effect for SE. Thinking that one was good and worthy was related to decreases in depressive symptoms on the day exam grades were given, but not 2 days later. As would be expected, such decreases in symptoms were greater in those who were initially mildly depressed (Initial Depression × SE interaction).

The present study treated individuals’ variance in SE over time as a measure of liability. Such an operationalization assumes that this variance generally conforms to a sine wave. That is, high-variance individuals should fluctuate up and down around their average level of SE over time. However, other possibilities exist. For example, someone whose SE begins at a high point but gradually declines to a low by the end of the baseline period could conceivably have the same variance score as someone who shows mild shifts up and down over this time period (Larsen, 1987). Such a possibility would be pernicious to vulnerability interpretations. Rather than a predisposition to depression, such a variance score might better reflect a prodromal symptom. Someone with a high variance score may be experiencing a progressive decline in SE that may be an early sign of depression and have absolutely nothing to do with etiology (Coyne & Gotlib, 1986).

In the present study there are two major sources of evidence against such interpretations: (a) Labile SE contributed to the prediction of depression in interaction with stress. If lability were simply a prodromal sign of no causal importance, one would not expect to find such interactions; and (b) visual inspection of the data in high-liability subjects suggests no such patterns.

Future research needs to replicate our findings, particularly with clinical populations and with more severe life events of the kind thought to lead to depression (see Brown & Harris, 1978, 1986). The Labile SE × Academic Stress × Initial Depression interaction suggests that liability has different implications according to severity of depression. In those who were initially asymptomatic, labile SE made subjects vulnerable to increases in symptoms following stress. However, in those who were initially mildly depressed, labile SE was most detrimental in the absence of stress. Labile SE may therefore operate in a substantially different (and as of yet, unexplored) way in clinical depression.

It is equally important that this study be replicated with other, particularly more comprehensive, measures of stress. Although we have already discussed the advantages of discrepancy scores between actual and aspired performance (weighted by importance of achievement), we want to emphasize their limitations, as well. First, academic disappointment provides an extremely narrow window into the total stress experienced in our subjects’ lives. Second, academic disappointment, except in the most extreme cases, is a relatively minor life event when compared with the types of environmental circumstances known to trigger clinical depression (see Brown & Harris, 1978, 1986). Third, discrepancy scores (in general) are known to have questionable reliability, whereas multiplicative composites are likely to provide underestimates of actual effect sizes when correlated with other variables (Evans, 1991). These concerns all highlight the possibility that other measures of stress might

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5 Follow-up analyses were conducted using the BDI with SE items removed (item numbers 3, 5, 7, and 8) to guard against possible confounding between predictor and criterion variables. Although labile SE continued to predict depression in the same manner as with the full scale, the main effect of level of SE and its interaction with initial depression were no longer significant, further highlighting the relative importance of liability.
Table 7
Regression Analysis Predicting BDI Score at the 12th Assessment (Labile Self-Esteem Analyses)

<table>
<thead>
<tr>
<th>Order of entry of set</th>
<th>Predictors in set</th>
<th>F for increment</th>
<th>t for within-set predictors</th>
<th>df</th>
<th>Partial correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>T9 BDI (k = 1)</td>
<td>264.36**</td>
<td>1,175</td>
<td>.78</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Entire set (k = 2), Labile SE, Academic Stress</td>
<td>5.71*</td>
<td>2,173</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Entire set (k = 3), Labile SE × Academic Stress, T9 BDI × Academic Stress, Labile SE × T9 BDI</td>
<td>1.49</td>
<td>3,170</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Labile SE × Academic Stress × T9 BDI (k = 1)</td>
<td>17.02**</td>
<td>1,169</td>
<td>-.30</td>
<td></td>
</tr>
</tbody>
</table>

Note: T9 = 9th assessment; BDI = Beck Depression Inventory; k = number of predictors in set; SE = self-esteem. Academic stress = discrepancy score between expected—hoped for grade and grade actually received, weighted by importance of academic achievement.

* p < .01. ** p < .001.

show different (probably stronger) interactive effects with vulnerable SE in predicting depression. On the other hand, they also demonstrate the sensitivity of labile SE to relatively minor environmental occurrences.

Using a similar academic stress paradigm, Metalsky et al. (1987) found that attributional style acted as a diathesis to depressive mood reactions following disappointment on a midterm exam. Academic stress was the sole predictor on the day that grades were received, whereas the Stress × Cognition interaction was the sole predictor a few days later. Future research might examine the relationship between attributional style and SE (particularly labile SE). If both measures were included in the same study, their relative contributions to the prediction of depression could be determined.

In summary, relatively strong support was found for labile SE as a vulnerability factor for depressive symptoms. Labile SE predicted changes in depression over time, both on its own and in interaction with stress and initial depression. In contrast, no support was found for trait level SE or differential activation as diatheses for depression. Although neither of these SE variables moderated the impact of stress, positive SE predicted reductions in depressive symptoms over time and may therefore play a role in recovery (see Brown, Bifulco, & Andrews, 1990).

Another recent study found that academic disappointment interacted with attributional style in predicting changes in depressive affect on the day students took college exams, but not on the day grades were given (Hunsley, 1989). To make comparisons with this study, we conducted additional analyses predicting changes in depressive symptoms on the day exams were actually taken (T10). Although students had not yet received their grades, they did have a fairly good idea of how well they performed, as seen in the correlation between the grades they thought they would receive (measured after taking the exam) and their actual grades (r = .59). At this time, labile SE strongly predicted changes in depressive symptoms (6.5% of variance in residual change, p < .001). Importantly, there was a significant labile SE × Academic Stress × Initial Depression interaction (14.4% of variance in residual change, p < .001). Furthermore, both labile SE (4.1% of variance in residual change, p < .01) and the three-way interaction (11.8% of variance in residual change, p < .001) continued to predict depression in the conservative analysis described earlier.

References
Williams, J. M. G., Healy, D., Teasdale, J. D., White, W., & Paykel, E. S. (1990). Dysfunctional attitudes and vulnerability to persistent depression. Psychological Medicine, 20, 375–381.

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