Cognitive Therapy and Research, Vol. 21, No. 5, 1997, pp. 569-589

Labile Self-Esteem, Life Stress, and Depressive Symptoms: Prospective Data Testing A Model of Vulnerability[1]

John E. Roberts[2,4] and Jon D. Kassel[3]

Recent theory and research suggests that labile self-esteem (SE) is an important dimension of vulnerability to depression (Butler, Hokanson, & Flynn, 1994; Roberts & Monroe, 1992, 1994, in press). In the present study, participants completed seven daily ratings of SE from which we derived a measure of lability. The interaction between labile SE and life stress predicted increases in depressive symptoms across a two-month prospective interval, particularly in participants who were initially low in depression and who had more severe worst lifetime episodes of depressive symptomatology. Interactions between life stress and labile SE were stronger for life stress measures that were based on the subjective appraisal of stress than for those that were based on raw life event counts. In contrast to predictions, depletions in self-esteem failed to mediate the synergistic effects of labile SE and life stress.

KEY WORDS: self-esteem; life stress; depression.

Recent theory and research suggests that daily fluctuation or lability in self-esteem (SE) plays a stronger role in vulnerability to depression than does trait level of SE (Roberts & Monroe, 1994, in press). This perspective is based on work suggesting that persons exhibit both a characteristic trait

[1]We would like to thank Stephen Russo for his help in data collection, as well as express appreciation for the many thoughtful suggestions made by several anonymous reviewers on earlier versions of this paper. Jon D. Kassel is now at the Department of Clinical and Health Psychology at the University of Florida.
[2]Northwestern University, Evanston, Illinois 60208.
[3]University of Pittsburgh, Pittsburgh, Pennsylvania 15260.
[4]Address all correspondence concerning this article to John E. Roberts, who is now at State University of New York at Buffalo, Department of Psychology, Park Hall, Box 604110, Buffalo, New York 14260-4110.

0147-5916/97/1000-0569$12.50/0

level of SE and a characteristic degree of fluctuation around that daily mean (see Kernis, Grannemann, & Barclay, 1989, 1992). Although low levels of SE are present during episodes of depression, a number of studies have found that low trait SE does not persist subsequent to remission or predict the onset of depression (Roberts & Monroe, in press). In contrast, *labile* SE might be a risk factor that can be found outside of episodes of depression in persons who are vulnerable. Theoretically, labile SE acts as a diathesis. That is, individuals with labile SE are thought to respond more adversely to life stress than those whose SE is relatively stable.

The idea that vulnerability to depression is partially reflected in labile SE is drawn from diverse theoretical and empirical literatures. For example, based on their review of psychosocial functioning in depression, Barnett and Gotlib (1988) concluded that depression-prone persons are highly dependent on external sources of self-worth. Similarly, a number of psychodynamic theorists have suggested that depression-prone persons are overly dependent on the love, praise, and affection of important others in maintaining SE (e.g., Arieti & Bemporad, 1978; Chodoff, 1972; Rado, 1928; see also Kuiper, Olinger, & MacDonald, 1988). As a result, depression-prone persons are held hostage to these few sources of positive self-evaluation and suffer depletions in self-worth with the loss, or threatened loss, of those sources. Their SE would act as a highly sensitive barometer of perturbations in their social worlds (Butler, Hokanson, & Flynn, 1994). Given the vicissitudes of life, depression-prone persons' SE would appear labile over the course of time.

From a somewhat different perspective, there is evidence that negative self-cognition (e.g., low SE) becomes more highly accessible during dysphoric mood states in depression-prone persons than in nonvulnerable individuals (Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990; Roberts & Kassel, 1996; Teasdale & Dent, 1987). Negative evaluations of the self, such as low SE, might be manifest only during dysphoric mood states in persons vulnerable to more serious depressions. According to recent theory, depression-prone persons synthesize relatively positive views of themselves (high SE) when nondepressed, but create negative self constructions (low SE) when dysphoric. In contrast, nonvulnerable persons' SE is relatively unaffected by mood state (Teasdale & Barnard, 1993). Consequently, we would expect that the depression-prone person's SE would act as a highly sensitive barometer to changes in internal, affective, experiences. According to the formulation discussed above, persons at risk for depression would experience labile SE, as opposed to chronically low SE. However, rather than being highly sensitive to environmental events, their SE would be overly responsive to mood. Interestingly, there is empirical support for both perspectives, suggesting that labile SE could be the result of reactivity to both exogenous (daily events) and endogenous (fluctuations in mood) sources (Roberts & Monroe, 1994).

A recent investigation tested the hypothesis that labile SE reflects heightened risk for depressive reactions following life stressors (Roberts & Monroe, 1992). Participants (192 college students) were followed over a 3-week baseline during which they completed nine assessments of global SE. Within-participant variance scores in SE were used as a measure of lability, whereas disappointment on an important exam was defined as a life stressor. Depressive symptomatology was measured 2 days before this exam, on the evening grades were known, and 2 days later. The results indicated that temporal instability of SE was associated with increased risk for depressive symptoms following this life stressor, whereas low SE conferred no special sensitivity to stress. However, this effect only was found for participants who were initially asymptomatic. Therefore, it is possible that labile SE is of little clinical relevance and merely pertains to mild shifts in symptomatology in those who are relatively nonvulnerable to major depression. On the other hand, labile SE might play an important role in the *onset* of more severe forms of depression, but not be involved after a certain threshold of symptomatology is passed. Finding that the effects of labile SE are limited to individuals who are *initially* low in symptomatology is consistent with either possibility.

One means of evaluating these two possibilities would involve investigating whether or not interactions between labile SE and life stress occur in individuals with relatively severe worst lifetime episodes of depressive symptomatology. Given the high probability of recurrence of depressive episodes (Belsher & Costello, 1988), as well as evidence that higher levels of subclinical symptoms of depression increase the risk for subsequent episodes of clinical depression (Coryell, Endicott, & Keller, 1991; Horwath, Johnson, Klerman, & Weissman, 1992; Lewinsohn, Hoberman, & Rosenbaum, 1988; Zonderman, Herbst, Schmidt, Costa, & McCrae, 1993), individuals with a relatively severe previous episode of depressive symptomatology can be seen as being more depression-prone (vulnerable) than those without such a history. Demonstrating that labile SE moderates the impact of stress in these depression-prone participants would suggest that the effects of labile SE are not limited to relatively nonvulnerable persons and might have clinical relevance. Such results would indicate that labile SE operates as a diathesis in persons who seem to be at risk for more serious depression. On the other hand, if labile SE moderates the impact of life stress *only* among initially asymptomatic persons whose worst lifetime episodes are relatively mild, the potential clinical relevance of labile SE would be dubious.

Although the previous study (Roberts & Monroe, 1992) employed a measure of stress (academic disappointment) that was relatively well defined, objective, and temporally discrete, it was a very narrow assessment of the total stress experienced in individuals' lives. Further, it is not known whether or not the subjective appraisal of stress is critical in igniting the labile SE diathesis. A number of theorists (e.g., Brown & Harris, 1978; Lazarus & Folkman, 1984) would argue that the subjective appraisal of stress and differences in the meaning and significance of life events are crucial. These theorists would note that there are tremendous individual differences in the objective circumstances and severity of the actual experiences included within a given event category (e.g., events such a relationship breakup vary in their severity depending on the particular circumstances of the relationship) and that the individual's appraisal of the event in part captures these differences in severity. Additionally, in the previous study, brief prospective intervals were used in predicting changes in depressive symptoms (7 and 9 days). It is unclear whether or not labile SE confers vulnerability to depressive symptoms over longer time periods. Finally, the mechanism underlying the relation between labile SE and depressive symptoms has not been empirically investigated. Conceptually, we might expect that depletions in experienced self-worth mediate the relation between labile SE and depression subsequent to life stressors (Roberts & Monroe, 1994). That is, persons with labile SE might be particularly prone to plunges in SE following life stress. These drops in SE, in turn, would be the proximal cause of increases in depressive symptoms.

The purpose of the current study was to replicate and extend our earlier work (Roberts & Monroe, 1992), using more comprehensive measures of both perceived and objective stress, as well as a longer prospective interval (2 months), than the earlier study. We hypothesized that: (1) life stress would have a more depressogenic impact on participants with relatively labile SE (the diathesis-stress hypothesis), particularly among those who were initially relatively asymptomatic; (2) interactions between labile SE and life stress would be apparent among individuals whose worst lifetime episodes of depressive symptomatology were relatively severe; and (3) depletions in SE would mediate the relation between Labile SE $\times$ Life Stress interactions and changes in depressive symptoms. We also examined whether findings would vary according to the degree of subjective appraisal in the life stress measure on an exploratory basis.

METHOD

Participants

The participants were 225 university undergraduate students (141 female) enrolled in introductory psychology courses at the University of Pittsburgh. A total of 12 individuals were not included in subsequent analyses due to incomplete data, leaving a final sample of 213. The individuals participated in exchange for course credit.

Overview

The participants were administered a battery of questionnaires early in the semester (Session 1) and were instructed to complete six daily measures of SE and depressive mood over the next 6 days. Daily measures were returned through campus mail each day after completion. The participants were told that they needed to complete all daily ratings on time in order to receive experimental credit. These ratings, combined with ratings on identical measures at Session 1, yielded a total of seven daily assessments. The participants returned for a final session (Session 2) 2 months after Session 1. At this time they were administered another packet of questionnaires and were debriefed.

Measures

Self-Esteem. The Rosenberg Self-Esteem Scale (Rosenberg, 1979) is a 10-item inventory designed to measure global self-regard (e.g., "On the whole, I am satisfied with myself"). The items were scored on a 5-point Likert-type scale (1 = *strongly agree;* 5 = *strongly disagree*). In the present sample, average coefficient alpha was .89 across the eight assessments. Higher scores reflect greater self-worth. Consistent with previous research (e.g., Kernis et al., 1989), labile SE was operationalized as within-participant standard deviation scores of SE over the seven daily assessments. Five participants who completed fewer then four daily assessments on time were assigned missing values on this variable. All but 14 of the remaining participants completed all daily assessments. Higher scores reflect greater lability in SE.

Depressive Mood. A modified version of the Multiple Affect Adjective Checklist (MAACL; Zuckerman, Lubin, & Rinck, 1983) was used to measure transient depressed mood during each assessment. Twelve depressed-content adjectives were selected from the full MAACL on the basis of their factor loadings (Zuckerman et al., 1983). The participants indicated which of these adjectives described how they felt during the day. Similar to our measure of labile SE, we constructed a measure of affective lability as within-participant standard deviation scores on the MAACL over the seven daily assessments. The average coefficient alpha over the eight assessments was .74.

Depressive Symptoms. The Inventory to Diagnose Depression (IDD; Zimmerman, Coryell, Corenthal, & Wilson, 1986) was used to measure depressive symptoms. Although this 22-item instrument was designed to classify individuals categorically in terms of diagnostic criteria for major depression, it also provides an index of severity of depressive symptomatology. For each symptom, the participants choose which of five statements best describes how they have been feeling during the past week. All major content areas of depression (cognitive, affective, somatic/vegetative) are covered on this instrument. When used as a continuous measure of severity, the IDD correlates highly with the Beck Depression Inventory (r = .87) and the Hamilton Rating Scale (r = .80; Zimmerman et al., 1986). In the present sample, the coefficient alpha was .85 at Session 1 and .87 at Session 2. The Inventory to Diagnose Depression — Lifetime (IDD-L; Zimmerman & Coryell, 1987) evaluates the same 22 symptoms as the IDD, but these symptoms are rated in terms of the most depressed week of the individual's life. Coefficient alpha was .92.

Life Stress. A modified version of the List of Threatening Events Questionnaire (LTE-Q; Brugha, Bebbington, Tennant, & Hurry, 1985; Brugha & Cragg, 1990) was used to measure stressful life events. This inventory was developed to assess the types of life experiences found to be critical in triggering depression in George Brown's interview-based approach (Brown & Harris, 1978). The LTE-Q has good test-retest reliability (kappa = .78 to 1.0 on all categories except "something you valued was lost or stolen," where kappa = .24), high agreement between participant and informant ratings (kappa = .7 to .9), as well as good agreement with Brown's interview-based ratings (sensitivity = .89; specificity = .74; Brugha & Cragg, 1990).

We added four events believed to be particularly relevant to our sample of college students (e.g., failed an important exam; parents got divorced or separated). The participants were instructed to indicate which events occurred during the 2-month interval between Session 1 and Session 2 and then to rate the degree to which the event was "upsetting" (1 = *did not happen;* 2 = *happened but was not upsetting;* 3 = *happened and was somewhat upsetting;* 4 = *happened and was moderately upsetting;* 5 = *happened and was extremely upsetting*). Two scales were derived from this measure. The first scale was the

raw number of negative events reported (rated 3 or higher; Life Events), whereas the second scale included subjective appraisal by weighting events by each individual's upsettingness ratings (Weighted Life Events).

Diffuse subjective stress was measured with the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). The PSS consists of 14 items such as "felt that things were going your way" (reverse scored) and "felt that difficulties were piling up so high that you could not overcome them." These items were rated according the frequency that they were thought or felt during the previous month on a 5-point scale (1 = *never;* 5 = *very often*). Coefficient alpha was .87 in the current study.

Analyses

Setwise hierarchical regression analysis (Cohen & Cohen, 1983) was used to test the major hypotheses. First, we examined whether the two-way interaction between labile SE and life stress, and whether the triple interaction between labile SE, life stress, and initial depression, predicted changes in depressive symptoms. Second, we examined whether or not these relations could be demonstrated in participants with relatively severe worst lifetime episodes of depressive symptomatology by conducting similar regression analyses separately in individuals with relatively severe or mild previous depression. Finally, we followed the strategy outlined by Baron and Kenny (1986) to test whether depletions in SE mediate the relation between Labile SE × Life Stress interactions and residual change in depressive symptoms.

In each primary regression analysis, we used Session 2 IDD scores as the criterion variable and entered initial depressive symptomatology at Step 1. Thus, the remaining variables predict *residual change* in depressive symptoms, that is, the variance in Session 2 depression that cannot be explained by earlier depression scores. We report the magnitude of effects with partial correlations (*pr*'s). For each equation, we conducted three separate regression analyses corresponding to the three measures of stress: Life Events, Weighted Life Events, and Perceived Stress.

RESULTS

Descriptive Statistics

The participants' ages ranged from 17 to 49 years old with a mean of 20.3 (SD = 5.1). As can be seen in Table I, the average IDD score was 13.0 (SD = 8.5) at Session 1 and 10.2 (SD = 8.4) at Session 2. Although

these scores were highly correlated, $r = .57$, $p < .001$, they were significantly different, $t(212) = 5.15$, $p < .001$, indicating that the sample as a whole was somewhat less depressed at the follow-up session. Gender differences were apparent on several variables of interest in this sample. Females showed a trend towards greater IDD scores at Session 2 (11.0 vs. 8.9), $t(211) = 1.78$, $p < .08$, and reported more severe worst lifetime depression scores (29.4 vs. 24.4), $t(199) = 2.49$, $p < .05$, (separate variances test), greater affective lability (1.42 vs. 1.19), $t(191.6) = 1.99$, $p < .05$, (separate variances test), and more perceived stress (42.3 vs. 39.6), $t(211) = 2.54$, $p < .05$, compared to males.

As can be seen in Table I, labile SE was not highly correlated with affective lability ($r = .38$), SE at Session 1 ($r = -.20$), SE at Session 2 ($r = -.17$), or aggregate SE ($r = -.24$). These correlations suggest that labile SE is related to, but certainly not identical to, affective lability or level of SE. In contrast, affective lability was highly correlated with aggregate depressive affect ($r = .80$), indicating that these two variables were measuring the same underlying construct.

Diathesis-Stress Analyses: Entire Sample

Setwise hierarchical regression analyses were conducted to determine whether or not labile SE acted as a diathesis to depressive reactions subsequent to life stress. In particular, these analyses tested whether or not life stress, labile SE, initial depressive symptoms, and their interactions predicted residual change in depressive symptoms in the sample as a whole. The results from these analyses are presented in Table II.

Life Events. Although life events significantly predicted residual change in depressive symptoms, the main effect of labile SE was nonsignificant. Individuals who experienced a greater number of life events reported higher levels of depressive symptoms over the prospective interval. Inconsistent with the diathesis-stress hypothesis, the Labile SE × Life Events interaction was not significant, and the three-way interaction between labile SE, life events, and initial depressive symptoms only showed a nonsignificant trend ($p < .09$).[5]

[5]Although the triple interaction only showed a trend toward significance, follow-up analyses indicated that the Life Events × Labile SE interaction was stronger among individuals who were initially relatively asymptomatic, $t(103) = 3.15$, $p < .01$, $pr = .30$, than among those who were initially higher in depressive symptoms, $t(100) = 0.03$, n.s., $pr = .00$. Consistent with the diathesis-stress hypothesis, among initially asymptomatic individuals, labile SE had a more depressogenic impact on those experiencing more life events, $t(51) = 1.72$, $p < .1$, $pr = .23$, than on those with relatively few life events, $t(51) = 0.67$, $p > .1$, $pr = .09$.

Table I. Correlation Matrix, Means, and Standard Deviations of Central Variables[a]

Variable	1	2	3	4	5	6	7	8	9	10	11	12	*M*	*SD*
1. SE at Session 1													38.50	6.11
2. SE at Session 2	.73[d]												39.32	6.32
3. Aggregate SE	.85[d]	.81[d]											40.04	5.67
4. Labile SE	-.20[c]	-.17[b]	-.24[d]										3.03	1.72
5. IDD-L	-.34[d]	-.34[d]	-.37[d]	.16[c]									27.51	15.23
6. IDD at Session 1	-.58[d]	-.51[d]	-.59[d]	.19[c]	.53[d]								13.00	8.46
7. IDD at Session 2	-.39[d]	-.48[d]	-.40[d]	.17[b]	.46[d]	.57[d]							10.23	8.41
8. Aggregate depressive affect	-.35[d]	-.37[d]	-.50[d]	.21[c]	.24[d]	.44[d]	.38[d]						1.52	1.23
9. Affective lability	-.28[d]	-.33[d]	-.45[d]	.38[d]	.29[d]	.38[d]	.31[d]	.80[d]					1.33	0.85
10. Life events	-.23[c]	-.21[c]	-.25[d]	.19[c]	.13	.29[d]	.31[d]	.19[c]	.18[b]				2.16	1.92
11. Weighted life events	-.24[d]	-.22[c]	-.24[d]	.18[c]	.13	.30[d]	.36[d]	.19[c]	.16[b]	.97[d]			22.75	5.87
12. Perceived stress	-.50[d]	-.62[d]	-.51[d]	.25[d]	.35[d]	.50[d]	.64[d]	.39[d]	.38[d]	.30[d]	.34[d]		41.28	7.69

[a] SE = Self-esteem; IDD = Inventory to Diagnose Depression; IDD-L = Inventory to Diagnose Depression — Lifetime.
[b] $p < .05$, two-tailed.
[c] $p < .01$, two-tailed.
[d] $p < 0.001$, two-tailed.

Table II. Regression Analysis Predicting IDD Severity Scores at Session 2: Entire Sample (N = 213)[a]

	Life events		Weighted life events		Perceived stress	
Order of entry of set/predictors	Test of significance	*pr*	Test of significance	*pr*	Test of significance	*pr*
Step 1: Main effects	$F(1, 211) = 99.81^{d}$		$F(1, 211) = 99.81^{d}$		$F(1, 211) = 99.81^{d}$	
Session 1 IDD	$t(211) = 9.99^{d}$	.57	$t(211) = 9.99^{d}$	.57	$t(211) = 9.99^{d}$	.57
Step 2: Main effects	$F(2, 209) = 4.03^{b}$		$F(2, 209) = 6.38^{c}$		$F(2, 209) = 34.83^{d}$	
Labile SE	$t(209) = .68$	.05	$t(209) = .61$	.04	$t(209) = .31$	−.02
Stress	$t(209) = 2.63^{c}$	.18	$t(209) = 3.41^{d}$	.23	$t(209) = 8.26^{d}$	.50
Step 3: Two-way interactions	$F(3, 206) = 2.20$, $p < .09$		$F(3, 206) = 2.79^{b}$		$F(3, 206) = 9.75^{d}$	
Labile SE × Stress	$t(206) = 1.23$	.09	$t(206) = 1.93$, $p < .06$	.13	$t(206) = 4.21^{d}$	.28
Stress × Session 1 IDD	$t(206) = 1.04$	.07	$t(206) = .99$	.07	$t(206) = 1.46$	.10
Labile SE × Session 1 IDD	$t(206) = 2.06^{b}$	−.14	$t(206) = 1.99^{b}$	−.14	$t(206) = 4.90^{d}$	−.32
Step 4: Three-way interaction	$F(1, 205) = 2.99$, $p < .09$		$F(1, 205) = 5.75^{b}$		$F(1, 205) = 1.06$	
Labile SE × Stress × Session 1 IDD	$t(206) = 1.73$, $p < .09$	−.12	$t(205) = 2.40^{b}$	−.17	$t(205) = 1.03$	−.07

[a] SE = self-esteem; IDD = Inventory to Diagnose Depression.
[b] $p < .05$.
[c] $p < .01$.
[d] $p < .001$.

Weighted Life Events. Although weighted life events prospectively predicted residual change in depressive symptoms, the main effect of labile SE again was nonsignificant. Individuals who appraised the life events that they experienced as more upsetting reported higher levels of depressive symptoms over the prospective interval. Of greater relevance, the theoretically important interaction between labile SE and life stress showed a trend toward significance, $p < .06$, and the three-way interaction between labile SE, weighted life events, and Session 1 IDD attained conventional levels significance.[6] Follow-up analyses on this three-way interaction were conducted by dividing the sample into high and low initial depression groups based on a median split of the IDD at Session 1 and testing the two-way interaction between labile SE and life stress in each. Significant two-way interactions were then decomposed by conducting a median split on labile SE and examining the effect of life stress in each group. In addition to this statistical description, the form of this three-way interaction is graphically portrayed in Figs. 1 and 2.[7]

For the high initial depression group, the Labile SE × Weighted Life Events interaction was a nonsignificant predictor of changes in depressive symptoms, $t(100) = 0.21$, $pr = .02$. Labile SE failed to moderate the relation between weighted life events and symptom changes in individuals who already were experiencing depressive symptomatology. In contrast, for the low initial depression group, the Labile SE × Weighted Life Events interaction was a significant predictor of residual change in depression, $t(103) = 3.63$,

[6]Additional analyses were conducted to determine whether labile SE continued to interact with weighted life events after statistically controlling for related variables (affective lability; level of SE) and nonlinear trends in each of the interaction's component terms. Session 1 IDD score was entered first, followed by the set of SE at Session 1, SE squared (controls for nonlinear relations between SE and labile SE), affective lability, and weighted life events. Labile SE was entered next. To control for nonlinear trends potentially associated with interaction terms, Session 1 IDD, weighted life events, and labile SE, each squared (see Cohen & Cohen, 1983; Lubinski & Humphreys, 1990), were entered simultaneously, followed by the set of all relevant two-way interactions. The Labile SE × Life Events × Initial Depression interaction was entered as the last step. Importantly, the two-way interaction between labile SE and life events was a significant predictor of changes in depressive symptoms, $t(200) = 2.71$, $p < .01$, $pr = .19$, as was the three-way interaction between labile SE, life events, and initial depression, $t(199) = 2.95$, $p < .01$, $pr = -.20$.

[7]To construct these figures, residual change scores in depressive symptoms were calculated for the sample as a whole. In particular, IDD scores at Session 2 were regressed on the IDD at Session 1 and the residuals were saved. Subsequently, these residuals were plotted according to median splits on each variable composing the interaction. In contrast, Cohen and Cohen (1983) recommended solving the regression equation after setting each component variable of the interaction to 1 *SD* above and below the mean. We believe that this latter approach — because it is based on hypothetical points on the regression line, rather than the data of actual participants — likely inflates the magnitude of effects. In the case of a triple interaction, very few actual participants (if any) will have scores greater than 1 *SD* of the mean on *each* of the three variables. Median splits provide a more conservative portrait of the interaction that more fairly represents the actual data.

$p < .001$, $pr = .34$. Follow-up analyses on this two-way interaction indicated that, for the initially nondepressed, weighted life events predicted changes in depression more strongly in participants with relatively labile SE, $t(40) = 2.30$, $p < .05$, $pr = .34$, than in those with relatively stable SE, $t(62) = 0.20$, $pr = .03$. These results suggest that, for the initially nondepressed individuals, labile SE acted as a diathesis: Among the individuals with labile SE, higher levels of weighted life events predicted increases in depressive symptoms over time, whereas for those with stable SE, weighted life events did not contribute to the development of depressive symptoms.

Perceived Stress. Although labile SE again failed to show a significant main effect, perceived stress was a strong prospective predictor of changes in depressive symptoms ($pr = .50$). Individuals who perceived experienced greater amounts of stress in their lives reported higher levels of depressive symptoms over the prospective interval. Of greater theoretical interest, the Labile SE × Perceived Stress interaction was significant,[8] but the triple interaction between labile SE, perceived stress, and initial depression was a nonsignificant predictor of residual change in depressive symptoms. Follow-up analyses of the two-way interaction between labile SE and perceived stress were consistent with the diathesis-stress hypothesis: Perceived stress was a stronger predictor of residual change in depressive symptoms among individuals with labile SE, $t(106) = 5.47$, $p < .001$, $pr = .59$, than among individuals with stable SE, $t(101) = 3.93$, $p < .001$, $pr = .36$.

Diathesis-Stress Analyses: Distinctions Based on Severity of Previous Depressive Symptomatology

We next explored whether the effects of labile SE were dependent on the severity of individuals' worst lifetime episode of depressive symptomatology. In these analyses, we conducted identical regression analyses as above separately among participants with and without relatively severe worst lifetime episodes of depressive symptomatology (based on a median split of the IDD-L). These analyses are reported in Table III. In presenting these results, we focus solely on the interactions between labile SE, life stress, and initial symptomatology. There were no significant labile SE main effects in any of these analyses.

[8]A conservative regression analysis was conducted similar to that presented in footnote 6. After statistically controlling for affective lability, level of SE, and nonlinear trends in each of the interaction's component terms (as well as in level of SE), the Labile SE × Perceived Stress interaction continued to show a strong trend toward significance, $t(200) = 1.91$, $p < .06$, $pr = .13$.

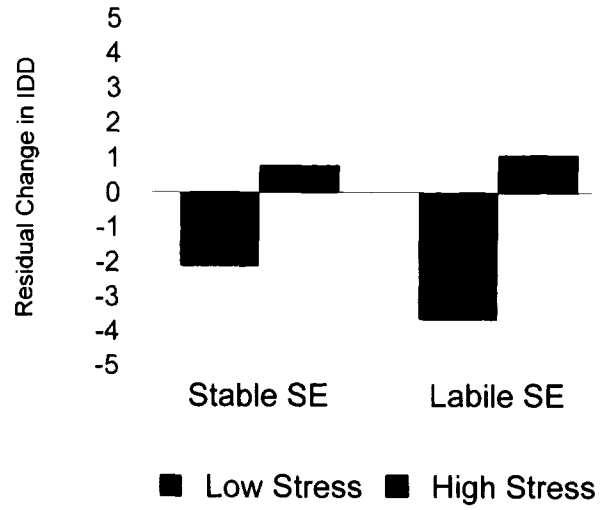

Fig. 1. Residual changes in Inventory to Diagnose Depression (IDD) score over a 2-month prospective interval as a function of labile self-esteem (SE) and life stressors in participants initially high in depressive symptoms.

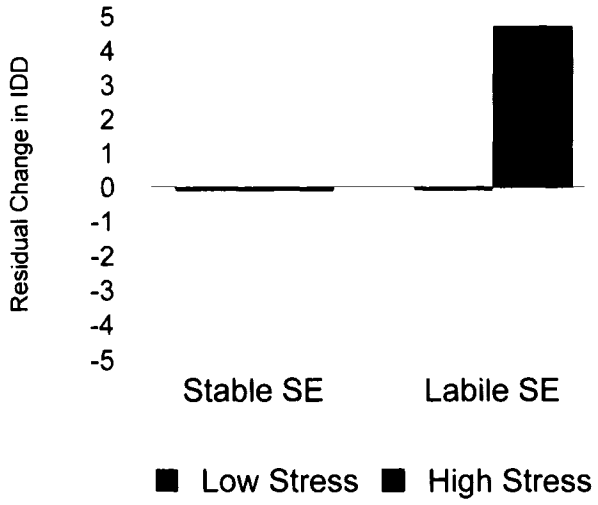

Fig. 2. Residual changes in Inventory to Diagnose Depression (IDD) score over a 2-month prospective interval as a function of labile self-esteem (SE) and life stressors in participants initially low in depressive symptoms.

Table III. Regression Analysis Predicting IDD Severity Scores at Session 2: Distinctions Based on Severity of Previous Depression[a]

Order of entry of set/predictors	Life events		Weighted life events		Perceived stress	
	Test of significance	*pr*	Test of significance	*pr*	Test of significance	*pr*
	Relatively severe previous depression (n = 110)					
Step 3: Two-way interactions	$F(3, 103) = 1.60$		$F(3, 103) = 1.80$		$F(3, 103) = 5.12^c$	
Labile SE × Stress	$t(103) = .78$	.08	$t(103) = 1.43$	.14	$t(103) = 2.83^c$	.27
Stress × Session 1 IDD	$t(103) = .20$	.02	$t(103) = .18$	−.02	$t(103) = 1.18$	.12
Labile SE × Session 1 IDD	$t(103) = 2.14^b$	−.21	$t(103) = 2.07^b$	−.20	$t(103) = 3.17^c$	−.30
Step 4: Three-way interaction	$F(1, 102) = 4.27^b$		$F(1, 102) = 10.02^c$		$F(1, 102) = 2.13$	
Labile SE × Stress × Session 1 IDD	$t(102) = 2.07^b$	−.20	$t(102) = 3.17^c$	−.30	$t(102) = 1.46$	−.14
	Relatively mild previous depression (n = 103)					
Step 3: Two-way interactions	$F(3, 96) = 2.73^b$		$F(3, 96) = 2.85^b$		$F(3, 96) = .67$	
Labile SE × Stress	$t(96) = .16$	−.02	$t(96) = .20$	−.02	$t(96) = .30$	−.03
Stress × Session 1 IDD	$t(96) = 2.40^b$	.24	$t(96) = 2.53^b$	.25	$t(96) = .19$	.02
Labile SE × Session 1 IDD	$t(96) = 1.57$	−.16	$t(96) = 1.63$	−.16	$t(96) = .77$	−.08
Step 4: Three-way interaction	$F(1, 95) = .56$		$F(1, 95) = .15$		$F(1, 95) = 8.16^c$	
Labile SE × Stress × Session 1 IDD	$t(95) = .75$	.08	$t(95) = .39$	.04	$t(95) = 2.86^c$	−.28

[a] SE = self-esteem; IDD = Inventory to Diagnose Depression.
[b] $p < .05$.
[c] $p < .01$.

Severe Previous Depression. In the case of individuals who reported a relatively severe previous episode of depressive symptomatology, the results from the regression analyses were similar to those of the sample as a whole, albeit stronger. As can be seen in the upper portion of Table III, analyses based on both life events and weighted life events indicated that the two-way interactions between labile SE and life stress were nonsignificant, but that the triple interactions between labile SE, life stress, and initial depressive symptoms were significant predictors of residual change in depressive symptoms. Also consistent with the sample as a whole, the Labile SE × Perceived Stress interaction was significant, whereas the Labile SE × Perceived Stress × Initial Depression triple interaction failed to significantly predict residual change in depressive symptoms. Overall, these findings suggest that labile SE acts as a diathesis for depressive reactions to life stress among persons who are relatively depression-prone.

Mild Previous Depression. Among individuals who reported relatively mild worst lifetime episodes of depressive symptoms, the results from regression analyses indicated that labile SE generally was a weaker diathesis than in the sample as a whole. As can be seen in the lower portion of Table III, the Labile SE × Weighted Life Events × Initial Depression triple interaction (which was found in the sample as a whole) was a nonsignificant predictor of residual change in depressive symptoms. Also inconsistent with the sample as a whole, the Labile SE × Perceived Stress interaction was not significant. However, there was a significant triple interaction between labile SE, perceived stress, and initial depression. Follow-up analyses of this triple interaction indicated that the Labile SE × Perceived Stress interaction was significant for initially nondepressed individuals, $t(42) = 2.07$, $p < .05$, $pr = .30$, but not for individuals who were mildly depressed at the start of the study, $t(51) = 1.62$, $p > .1$, $pr = -.22$. Among the initially asymptomatic, perceived stress had a more depressogenic impact on those with labile SE, $t(21) = 3.00$, $p < .01$, $pr = .55$, than among those with stable SE, $t(20) = 0.57$, $pr = .13$.

Mediation Analyses

Analyses were conducted in order to determine whether depletions in level of SE mediated the relations between the Labile SE × Weighted Life Events × Initial Depressive Symptoms triple interaction, as well as the Labile SE × Perceived Stress two-way interaction, and residual change in depressive symptoms. To demonstrate such mediation, the following conditions must be met (see Baron & Kenny, 1986): (1) These interactions must significantly predict residual change in SE; (2) residual change in SE must

significantly predict residual change in depressive symptoms after statistically controlling for the effects of the interactions; and (3) finally, the relation between these interactions and residual change in depressive symptoms must be either attenuated (in the case of partial mediation) or rendered nonsignificant (in the case of full mediation) after statistically controlling for the effect of residual change in SE.

Inconsistent with the mediation hypothesis, the triple interaction between weighted life events, labile SE, and initial depressive symptoms failed to significantly predict residual change in SE, $t(204) = 0.86$, $pr = .06$. Furthermore, although residual change in SE predicted residual change in depressive symptoms beyond the contribution made by the Labile SE × Weighted Life Events × Initial Depression interaction, $t(203) = 3.81$, $p < .001$, $pr = -.26$, the magnitude of the triple interaction was not appreciably diminished after changes in SE were statistically controlled ($pr = -.16$). Although the Perceived Stress × Labile SE interaction predicted residual change in SE, $t(205) = 2.26$, $p < .05$, $pr = -.16$, residual change in SE failed to significantly predict residual change in depressive symptoms beyond the contribution made by the Labile SE × Perceived Stress interaction. Furthermore, the magnitude of this two-way interaction was not appreciably diminished after changes in SE were controlled ($pr = .27$). Overall, these results suggest that depletions in SE do not mediate the relation between labile SE and residual change in depressive symptoms.[9]

DISCUSSION

The purpose of this study was to provide a conceptual replication and extension of our earlier investigation that demonstrated that labile SE was associated with increased depressive impact of a life stressor among initially asymptomatic individuals (Roberts & Monroe, 1992). Consistent with our previous results, temporal instability in SE was associated with the development of depressive symptoms subsequent to life stress, particularly in persons who were initially low in symptomatology and in those with relatively severe worst lifetime episodes of depressive symptomatology. Among these individuals, life stress had a greater depressogenic impact on persons with labile SE compared to those with stable SE. Importantly, labile SE moderated the impact of life stress when related constructs, such as affective lability and level

[9]As suggested by an anonymous reviewer, we also conducted mediation analyses separately on the subgroup of participants in which labile SE effects were strongest — those with relatively severe worst lifetime depressive episodes. These analyses also were inconsistent with the mediation hypothesis.

of SE, and nonlinear components of the interaction terms were statistically controlled (see footnotes 6 and 8). In contrast to our previous study (Roberts & Monroe, 1992), but consistent with two other investigations (Kernis, Grannemann, & Mathis, 1991; Roberts & Gotlib, in press), we found no evidence for a main effect of labile SE. Absence of a main effect suggests that temporal instability in SE primarily acts as a diathesis. That is, labile SE is associated with more adverse reactions to life stress, but is unrelated to changes in depressive symptoms when stress is not present. It is noteworthy that we replicated major findings with more comprehensive measures of life stress and over a longer prospective interval.

Another major goal of the current study was to determine whether labile SE has potential clinical relevance. We reasoned that demonstrating lability effects in persons who were relatively prone to depression would argue in favor of clinical importance. Such findings would suggest that lability does not merely pertain to mild shifts in symptomatology in relatively nonvulnerable healthy persons, but also operates in individuals who are at risk for more serious depression. In fact, our data suggest that labile SE might have more pernicious effects in persons who are prone to depression than in those who have a history of only mild worst lifetime episodes of depressive symptomatology. Labile SE effects that were demonstrated in the sample as a whole were nonsignificant in participants who reported relatively mild worst lifetime depressions. In contrast, labile SE acted as a powerful risk factor in participants with relatively severe worst lifetime episodes of depressive symptomatology. Among these depression-prone individuals, life stress had a more severe impact on those with relatively labile SE. Given these results, labile SE could indicate vulnerability to the recurrence of depressive episodes in the face of environmental adversity and perceptions of stress.

Of course examining whether labile SE acts as diathesis in nonclinical individuals with previous episodes of self-reported depressive symptomatology is a relatively weak test of clinical relevance. In order to increase confidence in and the generalizability of our findings, it would be critical to replicate this research with more severe, clinical samples, diagnosed through structured interviews. In particular, it would be important to examine remitted clinical depressives to determine if labile SE predicts relapse and recurrence of episodes of major depression. Such research also could investigate whether the effects of labile SE are specific to depression or are associated with risk for more general psychological distress and disorder. Interestingly, results from a more recently completed nonclinical study suggest that labile SE acts as diathesis for symptoms of depression, but not for anxiety (Roberts & Gotlib, in press).

Although the idea that depletions in SE mediate the relation between labile SE and depressive symptoms is conceptually appealing, the current data generally do not support this hypothesis. Likewise, our more recent study (Roberts & Gotlib, in press), as well as an unpublished reanalysis of our earlier data (Roberts & Monroe, 1992), are inconsistent with the hypothesis that depletions in SE mediate the effects of labile SE. What then is the mechanism by which labile SE operates? Interestingly, other theory and research suggests that hopelessness might be the driving factor (Brown & Harris, 1978; Metalsky, Joiner, Hardin, & Abramson, 1993; Whisman & Kwon, 1993). Individuals with relatively labile SE, particularly those who are formerly depressed, might have greater difficulty maintaining a sense of optimism about their life situations following negative life events and feelings of stress. They would be less able to use a secure and stable sense of self in warding off the pessimism generated by life stress (Janoff-Bulman & Hecker, 1988). Loss of hope then might act as the proximal cause of depression (Abramson, Metalsky, & Alloy, 1989). In stark contrast, it also is possible that labile self-esteem reflects more general cognitive and affective instability that results from dysregulation in underlying biological systems (see Depue & Iacono, 1989; Depue, Krauss, & Spoont, 1987). In this case, vulnerability to depression might be conferred independently of higher-order psychological mechanisms and processes. In order to better understand the significance and underlying basis of labile SE, it is crucial that future research examine these possibilities.

Interestingly, the results of the current study varied according to whether or not the stress measure involved subjective appraisal. In general, measures of perceived stress and subjectively weighted life events were stronger triggers of the labile SE diathesis than was the raw number of events that occurred during the prospective interval. In fact, the interaction between labile SE and the number of events reported was nonsignificant and the triple interaction between labile SE, number of life events, and initial symptoms only showed a trend toward significance in the sample as a whole — though it was significant among those with relatively severe previous episodes of depressive symptomatology. Consistent with models that emphasize the importance of the meaning and appraisal of life stressors (e.g., Brown & Harris, 1978; Lazarus & Folkman, 1984), the current findings suggest that the subjective appraisal of stress might be critical in igniting the labile SE diathesis. On the other hand, such life stress measures potentially are problematic because they can be confounded with depression (Monroe & Roberts, 1990). To some extent our prospective design should mitigate these concerns. Nonetheless, future research should determine whether or not Brown and Harris' more objective life events interview and ratings provide results similar to those of our initial investigations. This method uses the individual's unique environmental context in rating the severity of events.

In designs similar to ours biases from tautology can arise when measures of depression contain items reflecting poor SE and when measures of stressful life events include symptoms of depression (e.g., changes in sleep patterns). In other words, findings could be driven by overlapping item content in predictor and outcome measures. To guard against these potential biases, we reran the primary regression analyses after deleting the one item that reflected low SE on the depression measure (IDD Item 9). Importantly, the results were virtually identical to those obtained with the full measure. In particular, the Labile SE × Weighted Life Events × Initial Depression triple interaction was significant ($pr = -.16$, $p < .05$) and the Labile SE × Perceived Stress interaction also was significant ($pr = .29$, $p < .001$). In terms of biases between measures of life stress and depression, the life event and perceived stress instruments used in the current study contained no items that were direct symptoms of depression. Consequently, our findings do not appear to be driven by biases from tautology — direct item overlap between measures. However, we should note again that measures of perceived stress can be confounded with depression and that future work needs to more cleanly disentangle those aspects of stress perception that reflect true differences in the circumstances and meaning of the stressor from those that reflect the dysphoric world view of the depressed individual.

In summary, our data suggest that labile SE is associated with risk for developing depressive symptoms following life stress, particularly among persons who have initially low levels of symptomatology and who have more severe worst lifetime episodes of depressive symptoms. Labile SE appears to be particularly associated with the development of depressive symptoms when recent events are appraised as upsetting and diffuse feelings of stress are experienced. However, interactions between labile SE and life stress do not appear to be mediated by depletions in SE. Future research needs to investigate other possible mechanisms by which labile SE confers vulnerability to depression, as well as determine whether or not labile SE acts as a diathesis to clinical depression and interacts with more objective measures of life stress.

REFERENCES

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96,* 358-372.

Arieti, S., & Bemporad, J. (1978). *Severe and mild depression.* New York: Basic Books.

Barnett, P. A., & Gotlib, I. H. (1988). Psychosocial functioning and depression: Distinguishing among antecedents, concomitants, and consequences. *Psychological Bulletin, 104,* 97-126.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173-1182.

Belsher, G., & Costello, C. G. (1988). Relapse after recovery from unipolar depression: A critical review. *Psychological Bulletin, 104,* 84-96.

Brown, G. W., & Harris, T. O. (1978). *Social origins of depression.* New York: The Free Press.

Brugha, T., Bebbington, P., Tennant, C., & Hurry, J. (1985). The list of threatening experiences: A subset of 12 life event categories with considerable long-term contextual threat. *Psychological Medicine, 15,* 189-194.

Brugha, T. S., & Cragg, D. (1990). The List of Threatening Experiences: The reliability and validity of a brief life events questionnaire. *Acta Psychiatrica Scandinavica, 82,* 77-81.

Butler, A. C., Hokanson, J. E., & Flynn, H. A. (1994). A comparison of self-esteem lability and low trait self-esteem as vulnerability factors for depression. *Journal of Personality and Social Psychology, 66,* 166-177.

Chodoff, P. (1972). The depressive personality. *Archives of General Psychiatry, 27,* 666-677.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences.* Hillsdale, NJ: Erlbaum.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24,* 385-396.

Coryell, W., Endicott, J., & Keller, M. B. (1991). Predictors of relapse into major depressive disorder in a nonclinical population. *American Journal of Psychiatry, 148,* 1353-1358.

Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology, 40,* 457-492.

Depue, R. A., Krauss, S., & Spoont, M. R. (1987). A two-dimensional threshold model of seasonal bipolar affective disorder. In D. Magnusson & A. Ohman (Eds.), *Psychopathology: An integrative perspective* (pp. 95-123). Orlando: Academic Press.

Horwath, E., Johnson, J., Klerman, G. L., & Weissman, M. M. (1992). Depressive symptoms as relative and attributable risk factors to first onset major depression. *Archives of General Psychiatry, 49,* 817-823.

Janoff-Bulman, R., & Hecker, B. (1988). Depression, vulnerability, and world assumptions. In L. B. Alloy (Ed.), *Cognitive processes in depression* (pp. 177-192). New York: Guilford Press.

Kernis, M. H., Grannemann, B. D., & Barclay, L. C. (1989). Stability and level of self-esteem as predictors of anger arousal and hostility. *Journal of Personality and Social Psychology, 56,* 1013-1022.

Kernis M. H. Grannemann, B. D., & Barclay, L. C. (1992). Stability of self-esteem: Assessment, correlates, and excuse making. *Journal of Personality, 60,* 621-644.

Kernis, M. H., Grannemann, B. D., & Mathis, L. C. (1991). Stability of self-esteem as a moderator of the relation between level of self-esteem and depression. *Journal of Personality and Social Psychology, 61,* 80-84.

Kuiper, N. A., Olinger, L. J., & MacDonald, M. R. (1988). Vulnerability and episodic cognitions in a self-worth contingency model of depression. In L. B. Alloy (Ed.), *Cognitive processes in depression* (pp. 289-309). New York: Guilford Press.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping.* New York: Springer.

Lewinsohn, P. M., Hoberman, H. M., & Rosenbaum, M. (1988). A prospective study of risk factors for unipolar depression. *Journal of Abnormal Psychology, 97,* 251-264.

Lubinski, D., & Humphreys, L. G. (1990). Assessing spurious "moderator effects": Illustrated substantively with the hypothesized ("synergistic") relation between spatial and mathematical ability. *Psychological Bulletin, 107,* 385-393.

Metalsky, G. I., Joiner, T. E., Hardin, T. S., & Abramson, L. Y. (1993). Depressive reactions to failure in a naturalistic setting: A test of the hopelessness and self-esteem theories of depression. *Journal of Abnormal Psychology, 102,* 101-109.

Miranda, J., & Persons, J. B. (1988). Dysfunctional attitudes are mood-state dependent. *Journal of Abnormal Psychology, 97,* 76-79.

Miranda, J., Persons, J. B., & Byers, C. N. (1990). Endorsement of dysfunctional beliefs depends on current mood state. *Journal of Abnormal Psychology, 99,* 237-241.

Monroe, S. M., & Roberts, J. E. (1990). Conceptualizing and measuring lifestress: Problems, principles, procedures, progress. *Stress Medicine, 6,* 209-216.

Rado, S. (1928). The problem of melancholia. *International Journal of Psychoanalysis, 9,* 420-438.

Roberts, J. E., & Gotlib, I. H. (in press). Temporal variability in global self-esteem and specific self-evaluation as prospective predictors of emotional distress: Specificity in predictors and outcome. *Journal of Abnormal Psychology.*

Roberts, J. E., & Kassel, J. D. (1996). Mood-state dependence in cognitive vulnerability to depression: The roles of positive and negative affect. *Cognitive Therapy and Research, 20,* 1-12.

Roberts, J. E., & Monroe, S. M. (1992). Vulnerable self-esteem and depressive symptoms: Prospective findings comparing three alternative conceptualizations. *Journal of Personality and Social Psychology, 62,* 804-812.

Roberts, J. E., & Monroe, S. M. (1994). A multidimensional model of self-esteem in depression. *Clinical Psychology Review, 14,* 161-181.

Roberts, J. E., & Monroe, S. M. (in press). Vulnerable self-esteem and social processes in depression: Toward an interpersonal model of self-esteem regulation. In T. Joiner & J. Coyne (Eds.), *Recent advances in interpersonal approaches to depression.* Washington, DC: American Psychological Association.

Rosenberg, M. (1979). *Conceiving the self.* New York: Basic Books.

Teasdale, J. D., & Dent, J. (1987). Cognitive vulnerability to depression: An investigation of two hypotheses. *British Journal of Clinical Psychology, 26,* 113-126.

Teasdale, J. D., & Barnard, P. J. (1993). *Affect cognition and change.* Hillsdale, NJ: Erlbaum.

Whisman, M. A., & Kwon, P. (1993). Life stress and dysphoria: The role of self-esteem and hopelessness. *Journal of Personality and Social Psychology, 65,* 1054-1060.

Zimmerman, M., & Coryell, W. (1987). The inventory to diagnose depression, lifetime version. *Acta Psychiatrica Scandinavica, 75,* 495-499.

Zimmerman, M., Coryell, W., Corenthal, C., & Wilson, S. (1986). A self-report scale to diagnose major depressive disorder. *Archives of General Psychiatry, 43,* 1076-1081.

Zonderman, A. B., Herbst, J. H., Schmidt, C., Costa, P. T., & McCrae, R. R. (1993). Depressive symptoms as a nonspecific, graded risk for psychiatric diagnoses. *Journal of Abnormal Psychology, 102,* 544-552.

Zuckerman, M., Lubin, B., & Rinck, C. M. (1983). Construction of new scales for the multiple affect adjective check list. *Journal of Behavioral Assessment, 5,* 119-129.