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Modeling Relapse in Unipolar Depression: The Effects of Dysfunctional Cognitions and Personality Disorders

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Survival analytic models were used to determine the effects of Axis II pathology and dysfunctional cognitions on depressive relapse in a sample of 50 depressed inpatients followed 33 to 84 months (M = 49.9) postdischarge. In analyses based on follow-up interview measures, expected remission duration among patients without personality disorders was approximately 7.4 times longer than among patients with Axis II comorbidity. Attributional style also accounted for unique variance in the relapse model, with adaptive positive event attributions inversely related to relapse probability. Neither dysfunctional attitudes nor negative event attributions were significantly related to relapse. Dimensional Axis II Cluster B and C pathology ratings were associated with decreased survival time, whereas Cluster A pathology was associated with increased survival. Among measures obtained during index hospitalization, only the dimensional rating of Axis II pathology was significantly predictive, with a cumulative 8% decrease in expected survival for each Axis II criterion item met.

Depression relapse has become a primary focus of affective disorders research over the past decade, as it has become increasingly clear that a large proportion of successfully treated depressed individuals relapse within several months of clinical remission (Klerman & Weissman, 1992). For example, by 12 months postremission between 35% and 55% of formerly depressed patients experience a relapse episode (see Belsher & Costello, 1988). The consistent finding of such heightened relapse risk has helped catalyze research efforts to discover patient characteristics that render certain individuals especially vulnerable to relapse. The identification of such characteristics could serve to illuminate likely mechanisms through which the relapse process occurs, as well as to identify those patients for whom prophylactic treatment is most needed.

Cognitive Processes in Depression Relapse

Both Beck's cognitive model (Beck, 1976) and the reformulated learned helplessness model (Abramson, Seligman, & Teasdale, 1978) of depression propose the existence of a specific cognitive diathesis to depression. This hypothesized cognitive vulnerability applies both to the initial onset of the depressive syndrome and to subsequent experiences of relapse. Interestingly, in several longitudinal tests of Beck's cognitive model, there has been little empirical support for the hypothesized relationship between depressotypic cognitions and the initial onset of depression (see Haaga, Dyck, & Ernst, 1991); the evidence, however, regarding a cognitive vulnerability to depressive relapse is more compelling. In fact, there have been four reported investigations of the relationship between dysfunctional attitudes—negativistic beliefs hypothesized by Beck (1976) to be depressogenic—and subsequent depressive relapse (Rush, Weissenburger, & Eaves, 1986; Segal, Shaw, Vella, & Katz, 1992; Simons, Murphy, Levine, & Wetzel, 1986; Thase et al., 1992), each of which found a significant relapse risk associated with elevations on the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978).

The reformulated learned helplessness model hypothesizes that a person's explanatory style—specifically, the tendency to attribute negative events to internal, stable, and global causes—may serve as a cognitive diathesis for depression. In a subsequent extension of the model, Seligman, Abramson, Semmel, and von Baeyer (1979) suggested that a characteristic style of external, specific, and unstable attributions for positive events may also prove to be depressogenic. The reformulated learned helplessness model, however, has received little empirical support vis-à-vis the prediction of depression relapse. This may be due, in part, to the surprisingly small number of studies that have reported data on attributional style in clinically depressed patient samples (see Robins & Hayes, 1995). Alloy, Lipman, and Abramson (1992) found that a depressive attributional style among nondepressed college students was associated with a greater likelihood of such students having experienced prior depressive episodes (retrospectively assessed), many of which, presumably, were relapse episodes (although the ratio of relapses to first-onset cases in this sample was not reported).

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However, in the only reported prospective study with a population diagnosed with major depression, Rush et al. (1986) found that patients' attributions for failure events did not predict their scores on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at a 6-month follow-up. Furthermore, the widely cited study of Lewinsohn and colleagues (Lewinsohn, Seinmetz, Larson, & Franklin, 1981) found no relationship between attributional style and future depression onset in a large community sample. Similarly, the only investigation of the interaction of attributional style and stressful life events in a clinical sample detected no significant interaction between attributional style and stressful events in the prediction of depression onset (Persons & Rao, 1985). Therefore, there is only equivocal support for the hypothesized role of negative event attributions with respect to depression onset or relapse, although the hypothesis has not yet been adequately tested in a clinically depressed sample. Furthermore, no study has yet examined the role of positive event attributions with respect to relapse risk, although such attributions have been linked to recovery from dysphoric mood in two recent investigations (Edelman, Ahrens, & Haaga, 1994; Needles & Abramson, 1990).

Axis II Personality Disorders and Depression Relapse

There are several reasons for hypothesizing that Axis II personality disorders may predispose individuals to the experience of depression (including depression relapse), for example, (a) Axis II disorders, by definition, engender "clinically significant distress" (American Psychiatric Association, 1994, p. 633); (b) Axis II disorders predispose individuals to the experience of negative life events (American Psychiatric Association, 1994; Pfohl, Coryell, Zimmerman, & Stangl, 1984), which have been causally implicated in the onset of some depressive episodes (Brown & Harris, 1989); and (c) individuals with Axis II pathology tend to have very high levels of depressotypic cognitions (Evans & Craighead, 1995; O'Leary et al., 1991).

There have been four empirical investigations of the relationship between Axis II personality pathology and depression relapse (Peselow, Fieve, & DiFiglia, 1992; Pfohl, Coryell, Zimmerman, & Stangl, 1987; Thompson, Gallagher, & Czirr, 1988; Zimmerman, Coryell, Pfohl, Corenthal, & Stangl, 1986). These studies suggest that Axis II comorbidity confers an increased relapse risk after several forms of therapeutic intervention, including pharmacotherapy (Peselow et al., 1992; Pfohl et al., 1987), electroconvulsive therapy (ECT; Zimmerman et al., 1986), and short-term psychotherapy (Thompson et al., 1988).

Two methodological caveats, however, are worth noting. First, none of the previous studies controlled for differences in posttreatment residual depression symptomatology that may exist between Axis II and non-Axis II patients. There is evidence that increased depression severity, especially at treatment termination, is a predictor of subsequent relapse (e.g., Shea et al., 1992); therefore, to the extent that Axis II pathology is positively correlated with residual depression severity—as it was, for example, in the Pfohl et al. study—the reported relationship between Axis II pathology and depression relapse may be artifactual. It is also important to note that, with one exception (Thompson et al., 1988), each of the aforementioned studies assessed depressed patients for Axis II pathology at pretreatment, that is, while patients were fully syndromal. However, a number of investigators have documented the potential bias inherent in personality assessment that occurs during a depressive episode, with depressed patients often reporting significantly higher levels of personality pathology than they do after achieving clinical remission (e.g., Stuart, Simons, Thase, & Pilkonis, 1992). Therefore, it is possible that the observed relationship between Axis II pathology and depression relapse may be confounded, to some extent, by the assessment of personality at pretreatment. To address this possibility, the present investigation has included an assessment of Axis II pathology during a follow-up interview, at which time the majority of patients were no longer syndromal for major depression.

There is substantial evidence that dysfunctional cognitive patterns and Axis II personality disorders each comprise risk factors for depressive relapse. However, because Axis II pathology and dysfunctional cognitions appear to covary (Ilardi & Craighead, 1997; O'Leary et al., 1991), it is quite possible that these two factors overlap considerably with respect to the prediction of depression relapse; that is, depressotypic cognitions and Axis II pathology may actually account for the same portion of the variance in relapse prevalence. No studies germane to this hypothesis have heretofore been reported. The present study, therefore, tests this hypothesis by including measures of depressotypic cognitions and Axis II pathology simultaneously in a survival-analytic model of depression relapse (Lavori, Keller, & Klerman, 1984) among 50 depressed inpatients. Because both pretreatment and follow-up measures of cognitive dysfunction were obtained for most participants in the study, this investigation also includes separate analyses for both pretreatment and follow-up measures in an effort to determine whether differences exist in the predictive power of these cognitive indices as a function of the time of assessment. In addition, the present study has controlled statistically for several variables that have been reported to contribute to the likelihood of depression relapse: residual depressive symptomatology at treatment termination (Shea et al., 1992), so-called "double depression" (Coryell, Endicott, & Keller, 1991), and number of prior depressive episodes (Keller et al., 1987).

Method

Patient Selection

All patients included in the present investigation were inpatients on an affective disorders unit at the Duke University Medical Center. Study patients were selected on the basis of their meeting the following inclusion criteria: (a) a diagnosis of unipolar, nonpsychotic major depression according to the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., revised; *DSM–III–R* American Psychiatric Association, 1987) on the basis of a structured clinical interview; (b) age, at the time of intake, between 18 and 65; (c) completion, during hospitalization, of either at least one cognitive questionnaire measure (the DAS or the Attributional Style Questionnaire [ASQ; Seligman et al., 1979]) or a structured Axis II interview; and (d) achievement of full (n = 45) or partial (n = 5) remission[1] of the index depressive

[1] *Partial remission* is defined by Frank et al. (1991) as "a period during which an improvement of sufficient magnitude is observed that the individual is no longer fully symptomatic . . . but continues to evidence more than minimal symptoms" (p. 852).

episode (cf. Frank et al., 1991) during index hospitalization, and extending for at least 2 weeks postdischarge. Furthermore, patients who met *DSM–III–R* criteria for schizophrenia, bipolar disorder, organic brain syndrome, or any dementing illness were excluded from the patient sample. A total of 134 patients, admitted between 1987 and 1991, met the inclusion criteria. Of this group, there were 98 patients for whom current addresses could be located through medical center records. This group was then contacted by phone and recruited for participation in the follow-up evaluation portion of the present study. A total of 50 patients agreed to enlist in the present follow-up investigation. As a means of evaluating potential selection biases in this investigation, the final sample of 50 patients was compared on a number of clinical and demographic measures[2], with the 84 nonparticipating patients who met initial inclusion criteria. No significant differences ($p > .10$) were observed on any measure.

Materials

Interviews. Study patients were interviewed during their initial hospitalization using relevant portions of the National Institute of Mental Health (NIMH) Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981)[3], a structured interview designed to elicit patient information germane to the diagnosis of Axis I disorders according to *DSM–III–R* criteria. Patient diagnoses of unipolar major depression were made on the basis of DIS interview data obtained during hospitalization[4].

The Personality Disorder Examination (PDE; Loranger, Susman, Oldham, & Russakoff, 1987) was used to obtain information concerning the presence or absence of Axis II disorders for study participants. The PDE is a structured clinical interview that elicits information relevant to the diagnostic criteria associated with each of the 11 Axis II disorders enumerated under the *DSM–III–R*. PDE data were also used to construct a dimensional Axis II score for each patient; this score was computed as the sum of all criteria met for each of the 11 Axis II disorders. Follow-up PDE interviews for 10 study patients were evaluated by a second rater (an advanced psychology graduate student) for the purpose of assessing interrater reliability. An estimate of intraclass correlation (ICC) for PDE dimensional scores was calculated using Shrout and Fleiss's (1979) method for so-called Case 2 reliability models (Shrout & Fleiss, 1979, p. 423). The resultant ICC (2,1) of .955 is indicative of very high interrater reliability.

Patients were assessed at follow-up using the Longitudinal Interval Follow-Up Evaluation (LIFE), a semistructured interview developed to assess the longitudinal course of psychiatric disorders (Keller et al., 1987). Although the LIFE was originally designed for use over a 6-month follow-up period, it is capable of adaptation to "any length or number of follow-up intervals" (Keller et al., 1987, p. 540). For the purposes of the present investigation, the LIFE interview was modified to query for any relapse or recurrence of affective disorder (including rehospitalization) throughout the 33- to 84-month follow-up period. To assess the interrrater reliability of the modified LIFE interview used in the present investigation, interviews for 8 study patients were observed and coded by a second rater (a first-year psychology graduate student). Excellent reliability was achieved, with ICC (2,1) equal to .978. Furthermore, there was 100% agreement between raters regarding the presence or absence of major depressive episode at the time of the follow-up interview.

Weekly ratings of depression symptomatology were made during hospitalization by unit nursing staff using the Montgomery–Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979), which ranges in score from 0 to 60. Ratings were made on the basis of information obtained from interviews and patient observation. Nursing staff MADRS ratings have demonstrated good interrater reliability (Craighead et al., 1997), with an estimated nurse–nurse ICC of .77 and nurse–physician ICC of .60.

Questionnaires. Depressive cognitions associated with Beck's cognitive model (Beck, 1976) were assessed by means of the DAS (Form A; Weissman & Beck, 1978), a 40-item self-report questionnaire. The DAS is designed to measure the presence of depressotypic underlying assumptions, or dysfunctional beliefs; it is the most widely used instrument for this purpose.

We assessed attributional style by means of the ASQ (Seligman et al., 1979), which requires patients to "vividly imagine" 12 separate hypothetical events (6 positive and 6 negative events), and to make attributions about the occurrence of each event. The instrument yields separate composite subscale scores for positive and negative events (ASQ-P and ASQ-N, respectively). The negative composite score represents the sum of "internal," "stable," and "global" subscales based on negative event items; likewise with the positive composite score and positive event items. Negative and positive composite scores, rather than individual ASQ subscale scores, were used because of the significantly higher reliability estimates associated with the composite measures (Sweeney, Anderson, & Bailey, 1986). Both positive and negative ASQ composite scores were found to have satisfactory internal consistency in this patient sample, as evidenced by coefficient alphas (Cronbach, 1951) of .84 and .87, respectively.

Patients also completed the BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at the follow-up evaluation, which provided a self-report, 21-item measure of current depressive symptomatology. The BDI is the most widely used self-report depression measure. Its reliability and validity as a measure of major depression severity have been demonstrated (Beck, Steer, & Garbin, 1988; Bumberry, Oliver, & McClure, 1978).

Procedure

Hospitalization phase—assessment and treatment. All patients included in the present investigation were treated as inpatients on a 21-bed affective disorders unit at Duke University Medical Center. Routine clinical assessment methods completed during the first week of hospitalization include psychiatric and nursing interviews. In addition, patients were referred for psychological evaluation, which was completed in all cases during the first or second week of hospitalization; at the time of this evaluation, all patients met diagnostic criteria for major depression. The evaluation included completion of the NIMH DIS; during the evaluation, 44 patients also completed the DAS, and 37 completed the ASQ. Twenty-three of the 50 study patients were also evaluated for the pres-

[2] Demographic measures included age, gender, race, and marital status. Clinical measures were as follows: number of prior depressive episodes, age of first depression onset, hospitalization discharge depression severity rating (on the Montgomery–Asberg Depression Rating Scale), Axis II comorbidity, and dysthymia comorbidity.

[3] Twenty-three of the DIS Axis I interviews on study patients were conducted during index hospitalization under the auspices of the CRC/PE for the Study of Depression, supported by Grant MH40159 from the National Institute of Mental Health to the Department of Psychiatry at Duke University, Durham, NC (principal investigator, Dan G. Blazer II). The relevant portions of the DIS interview for such patients were incorporated within the more extensive Duke Diagnostic Evaluation Schedule (DDES; see Blazer, Hughes, & George, 1992, for a more detailed discussion).

[4] For those 23 subjects who were interviewed as a result of their participation in the CRC study, Axis I diagnoses were made on the basis of case conference consensus (see Blazer et al., 1992). For the other 27 patients, diagnoses were made by a clinical psychologist based on data obtained from the affective disorders portion of the DIS interview.

Table 1
Sample Characteristics at Index Hospitalization

Variable	Group[a]		
	Axis II (n = 22)	Non-Axis II (n = 28)	Total (n = 50)
M (and *SD*)			
Age[b]	34.9 (9.7)	41.0 (8.0)	38.3 (9.2)
No. of prior episodes	4.7 (4.1)	2.8 (3.5)	3.6 (3.8)
Age of onset	23.8 (10.7)	27.3 (9.8)	25.8 (10.2)
MADRS (discharge)[c]	18.3 (7.1)	10.9 (6.7)	14.2 (7.7)
n (and %)			
Gender			
Male	7 (31.8)	4 (14.3)	11 (22.0)
Female	15 (68.2)	24 (85.7)	39 (78.0)
Marital status			
Single	5 (22.7)	4 (14.3)	9 (18.0)
Married	10 (45.5)	18 (64.3)	28 (56.0)
Separated–divorced	7 (31.8)	6 (21.4)	13 (26.0
Race			
White	20 (90.9)	25 (89.3)	45 (90.0)
African American	2 (9.1)	3 (10.7)	5 (10.0)
Dysthymia comorbidity	12 (54.6)	11 (39.3)	23 (46.0)

Note. MADRS = Montgomery–Asberg Depression Rating Scale.
[a] Axis II category based on follow-up interview. [b] Axis II group different from non-Axis II ($p < .05$). [c] Axis II group different from non-Axis II ($p < .001$).

ence of Axis II pathology at this time using the PDE[5]. Weekly ratings of depression symptomatology were made by unit nursing staff using the MADRS.

During their hospitalization, 49 patients received antidepressant medication according to the clinical judgment of the attending psychiatrist and 1 patient received ECT. Forty-nine patients continued to receive pharmacotherapy postdischarge, and 1 patient received maintenance ECT. Ninety-four percent (47 of 50) of the patients reported receiving continuation pharmacotherapy for at least 6 months postdischarge; of the 3 patients who did not, 1 received maintenance ECT, 1 terminated tricyclic therapy after 4 months, and 1 terminated tricyclic therapy after 1 month.

Follow-up phase. Study participants were recruited by telephone beginning in early 1994, 33 to 84 months (M = 49.9, SD = 15.0) after their initial hospitalization. Patients were assessed at the follow-up evaluation using the Longitudinal Interval Follow-Up Evaluation (LIFE). Patients also completed the BDI at the follow-up evaluation, which provided a self-report measure of the severity of current depressive symptomatology. The follow-up protocol also included administration of the PDE interview and patient completion of the DAS and ASQ self-report questionnaires[6]. All patients received $20 in remuneration for their participation in the follow-up phase of the study.

Results

Sample Characteristics

Demographic and clinical characteristics of the patient sample are presented in Table 1. Patients were grouped for presentation purposes according to Axis II diagnosis, as determined by the follow-up interview. As shown, Axis II and non-Axis II groups differed significantly with respect to age (M = 34.9 vs. 41.0, respectively), $t(48) = 2.48$, $p < .05$, and discharge MADRS rating (M = 18.3 vs. 10.9), $t(48) = 3.74$, $p < .001$. No other significant between-groups differences were observed. Table 2 displays summary statistics on self-report measures for the patient sample at follow-up. Axis II patients were observed to have significantly higher scores than non-Axis II patients on the follow-up BDI (M = 18.6 vs. 6.5), $t(48) = 5.42$, $p < .0001$; follow-up DAS (M = 147.2 vs. 105.5), $t(48) = 4.67$, $p < .0001$; and follow-up ASQ-N (M = 84.2 vs. 71.3), $t(44) = 2.78$, $p < .01$. Axis II patients also had significantly lower scores than non-Axis II patients on the follow-up ASQ-P (M = 81.8 vs. 89.3), $t(44) = 2.20$, $p < .05$. Additionally, the length of time between the index hospitalization and follow-up interview (follow-up interval) was significantly shorter for Axis II patients (for Axis II, M = 43.4 months; for non-Axis II, M = 54.9 months), $t(48) = 2.44$, $p < .02$. Finally, a significantly higher proportion of Axis II patients met diagnostic criteria for major depression at the follow-up assessment (36.4% vs. 7.1%), $\chi^2(1, N = 50) = 6.58$, $p < .01$.

Statistical Analyses

To explore the effect of personality pathology and depressotypic cognitions on the risk of relapse after recovery from depression, we used a series of survival-analytic models (see Cox, 1972; Cox & Oakes, 1984). Such models are especially well suited to analyzing the effect of predictor variables on event probabilities in data sets with right-censoring (i.e., data in which

[5] There were six additional study patients who were interviewed at the pre-treatment assessment with the Structured Interview for the *DSM–III* Personality Disorders (SIDP; Stangl, Pfohl, Zimmerman, Bowers, & Corenthal, 1985). However, because Axis II diagnoses derived from the SIDP are not fully compatible with those based on the PDE (inasmuch as the former uses *DSM–III*, and the latter *DSM–III–R*, criteria), it was decided to exclude data from these six SIDP interviews in any study analyses.

[6] Patients also completed a visual analog mood scale (Luria, 1975) at follow-up. This instrument was used in a set of secondary analyses not reported herein.

Table 2
Summary Statistics

Variable	Group[a]					
	Axis II (n = 22)		Non-Axis II (n = 28)		Total (n = 50)	
	M	*SD*	*M*	*SD*	*M*	*SD*
Follow-up						
BDI[b]	18.6	10.3	6.5	5.1	11.9	9.8
DAS[b]	147.2	34.6	105.5	28.6	123.8	37.4
ASQ-N[c]	84.2	17.4	71.3	14.0	76.9	16.7
ASQ-P[d]	81.8	12.1	89.3	11.2	86.0	12.1
Change scores: hospitalization to follow-up						
DAS	−6.2	24.7	−10.5	39.3	−8.5	33.2
ASQ-N	−3.1	9.9	−10.4***	13.1	−6.9***	12.1
ASQ-P	−5.6	16.1	−9.6***	13.4	−7.7***	14.7

Note. BDI = Beck Depression Inventory; DAS = Dysfunctional Attitudes Scale; ASQ-N = Attributional Style Questionnaire composite subscale for negative events; ASQ-P = Attributional Style Questionnaire composite subscale for positive events.
[a] Axis II category based on follow-up interview. [b] Axis II group different from non-Axis II at $p < .0001$. [c] Axis II group different from non-Axis II at $p < .01$. [d] Axis II group different from non-Axis II at $p < .05$.
*** $p < .01$.

1 or more patients has not experienced the target event—in this case, relapse—by the end of the observation period); thus, survival analysis is particularly appropriate for use with the type of longitudinal follow-up data reported in the present study (see Singer & Willett, 1991). Accordingly, parametric accelerated "failure-time" models were analyzed using SAS's LIFEREG procedure (SAS Institute, 1987), with parameters estimated by maximum likelihood using a Newton–Raphson algorithm. The models' baseline survival function was initially hypothesized to correspond to a Weibull distribution—a variant of the exponential distribution in which the entire distribution is transformed by an estimated scale parameter (in the exponential distribution, the scale parameter is constrained to 1.0). However, a chi-square test of the baseline function demonstrated no significant deviation of the scale parameter from 1.0; Lagrange multiplier $\chi^2(1, N = 50) = 1.35$, $p > .20$. Thus, an exponential distribution was used for all survival models.

Depression Relapse: Survival Analyses

The baseline survival function for the 50 study patients is shown in Figure 1. The survival function models the cumulative risk of relapse as a function of time since recovery. Although the distinction is frequently made between relapse and recurrence (see Frank et al., 1991), with the former referring to the return of the full depressive syndrome after remission and the latter designating the occurrence of new episode after recovery[7], for the purposes of the present investigation no such distinction is made: The term *relapse* is used to refer to the return of syndrome depression after a period of full or partial remission of at least 8 weeks' duration (see Spitzer, Endicott, & Robins, 1978). As shown, the risk of relapse in this former inpatient sample was quite substantial; only 57% of the sample remained relapse-free at 6 months postrecovery, 45% remained relapse-free at 12 months, and 32% remained relapse-free at 24 months. Sixteen study patients had not experienced a relapse by the time of follow-up assessment; that is, the model included 16 right-censored failure-time values.

Because the number of prior depressive episodes, the presence of double depression (i.e., major depression and dysthymia), and depression severity rating at posttreatment have all been found by previous investigators to predict relapse risk, measures of these three variables (obtained at index hospitalization) were included as covariates. In addition, because the mean length of time between index hospitalization and follow-up (follow-up interval) was found to be significantly shorter for Axis II, compared with non-Axis II patients ($p < .01$) in this sample, the follow-up interval length was also included as a covariate in each survival model that used follow-up measures. Finally, the follow-up BDI score was added as a covariate as a means of controlling for the potentially confounding effect of depressive symptomatology at the time of the follow-up assessment, including dysphoric mood (Coyne, 1994), which has been specifically linked to mood-congruent biases in recall of prior depressive episodes (Goodwin & Sher, 1993) at the time of the follow-up assessment. The initial survival analysis included only the aforementioned five covariates as independent variables. Only the follow-up interval, likelihood ratio $\chi^2(1, N = 50) = 14.49$, $p < .0001$; and BDI, likelihood ratio $\chi^2(1, N = 50) = 26.54$, $p < .0001$, were found to be significantly predictive of relapse; longer follow-up intervals were associated with decreased relapse risk, whereas BDI was related to increased risk. The num-

[7] Frank et al. (1991), in a recent review, noted that the term *recovery* is usually applied to a remission that lasts for some prescribed duration; research diagnostic criteria (RDC; Spitzer, Endicott, & Robins, 1978), for example, has defined recovery as a symptom-free period that lasts a minimum of 8 weeks.

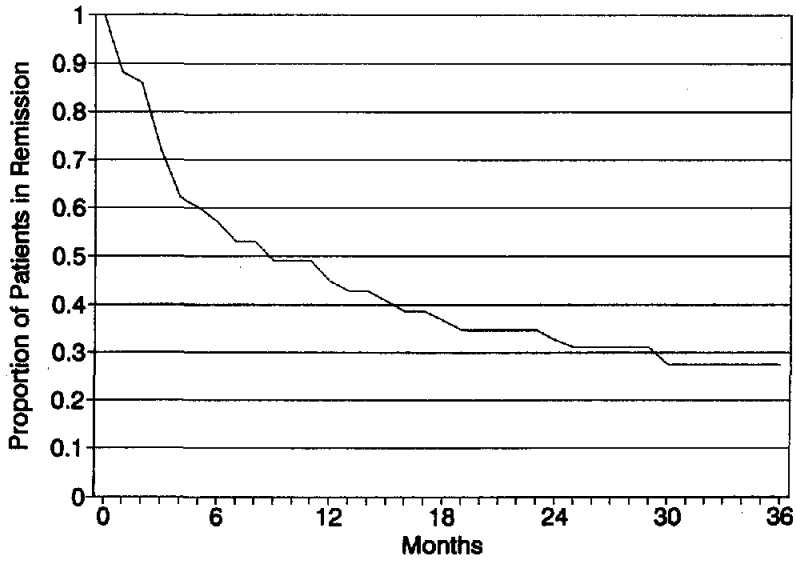

Figure 1. Baseline survival function.

ber of prior episodes, posttreatment MADRS rating, and dysthymia comorbidity were not significantly predictive ($p > .40$). Accordingly, the latter three variables were omitted as covariates in subsequent survival models.

As shown in Table 3, Models 1 through 7 used various follow-up cognitive and Axis II measures as independent variables. All models included follow-up BDI score and follow-up interval length as covariates. Model 1 included follow-up DAS as the independent variable. The effect of DAS on relapse was not significant ($p > .20$). Model 2 included both attributional measures, ASQ-N and ASQ-P, as independent variables. Only ASQ-P (attribution for positive events) had a significant effect on relapse, likelihood ratio $\chi^2(1, N = 44) = 10.73, p < .001$, with a coefficient estimate of 0.057. Because of the nature of the log-linear model used in this analysis, each coefficient estimate may be transformed into a risk multiplier, which represents the proportional increase–decrease in expected survival time due to each unit increment for a given independent variable. The multiplier for ASQ-P score may, thus, be represented as $e^{0.057}$, which equals approximately 1.06; this indicates an approximate 6% cumulative increase in expected survival time for each incremental point on a patient's ASQ-P score. In order to understand the relative influence on expected survival time indicated by the aforementioned ASQ-P multiplier, it may be instructive to consider the effect of a 12-point increase (approximately 1 *SD*) in ASQ-P score: The resulting multiplier would be $e^{0.057*12}$, or 1.98, which would indicate an approximate doubling of expected survival time. Model 3 included the dimensional score of Axis II pathology as a lone independent variable. The coefficient estimate was −0.081, likelihood ratio $\chi^2(1, N = 50) = 39.80$, $p < .0001$, with an indicated survival multiplier of 0.92.

All three cognitive measures, in addition to the Axis II dimensional rating, were included simultaneously as independent variables in Model 4. Only the Axis II rating was significantly associated with relapse, likelihood ratio $\chi^2(1, N = 44) = 6.01$, $p < .02$, with a coefficient estimate of −0.091; no significant effects were observed for DAS, ASQ-N, or ASQ-P (all $ps > .20$). Model 5 used the same independent variables as Model 4 but used a categorical, rather than dimensional, rating of Axis II (dummy coded as 1 for Axis II patients and 0 for non-Axis II patients). The only significant predictor in Model 5 was Axis II category, likelihood ratio $\chi^2(1, N = 44) = 8.35, p < .004$,

Table 3
Survival Models: Follow-Up Measures

Model and predictors	Coefficient estimate	χ^2	Survival multiplier
Model 1			
DAS	*ns*		
Model 2			
ASQ-N	*ns*		
ASQ-P	0.057	10.73****	1.06
Model 3			
Axis II (dimensional)	−0.081	14.87****	0.92
Model 4			
DAS	*ns*		
ASQ-N	*ns*		
ASQ-P	*ns*		
Axis II (dimensional)	−0.091	6.01****	0.91
Model 5			
DAS	*ns*		
ASQ-N	*ns*		
ASQ-P	*ns*		
Axis II (categorical)	−2.006	8.35***	0.13
Model 6			
Axis II (dimensional)	−0.070	8.59***	0.93
ASQ-P	0.031	3.44**	1.03
Model 7			
Cluster A	0.746	20.35****	2.11
Cluster B	−0.339	21.93****	0.71
Cluster C	−0.155	8.85***	0.86

Note. For all chi-square values, the degree of freedom was 1. DAS = Dysfunctional Attitudes Scale; ASQ-N = Attributional Style Questionnaire composite subscale for negative events; ASQ-P = Attributional Style Questionnaire composite subscale for positive events.
** $p < .05$. *** $p < .01$. **** $p < .001$.

with a coefficient estimate of −2.01. This indicates an expected survival multiplier of 0.13 (or $e^{-2.01}$) associated with the presence of an Axis II disorder (i.e., the expected survival time for Axis II patients is only 13% that of non-Axis II patients). Figure 2 illustrates the unadjusted baseline survival curves for Axis II and non-Axis II patients. As noted, there was no significant effect on relapse observed for DAS, ASQ-N, or ASQ-P. Because the three cognitive measures are believed to serve as indicators for similar cognitive constructs, the simultaneous inclusion of all three measures in a predictive model introduces the possibility of substantial multicollinearity. This could, in turn, attenuate the model's ability to detect significant effects of the cognitive variables on relapse risk. Therefore, in Model 6, it was decided to include only ASQ-P, the only cognitive measure with a significant effect in Models 1 and 2, along with the Axis II dimensional score and covariates. The Axis II score continued to be significantly associated with relapse, likelihood ratio $\chi^2(1, N = 46) = 8.59$, $p < .004$. In addition, there was a significant effect for ASQ-P, likelihood ratio $\chi^2(1, N = 46) = 4.03$, $p < .05$; as in Model 2, higher levels of positive attribution were associated with longer survival times.

In our test for possible differential effects of Axis II clusters on depressive relapse, Model 7 included cluster-specific dimensional ratings of Cluster A, Cluster B, and Cluster C pathology entered simultaneously, along with the two covariates (discharge MADRS and follow-up interval). Significant effects were found for Cluster A, likelihood ratio $\chi^2(1, N = 46) = 20.35$, $p < .0001$; Cluster B, likelihood ratio $\chi^2(1, N = 46) = 21.94$, $p < .0001$; and Cluster C, likelihood ratio $\chi^2(1, N = 46) = 8.85$, $p < .003$, with coefficient estimates of 0.746, −0.339, and −0.155, respectively. These coefficients correspond to survival time multipliers of 2.11, 0.71, and 0.86 for each unit increase in Cluster A, Cluster B, and Cluster C dimensional scores, respectively. Thus, each incremental increase in dimensional scores on Clusters B and C indicate decreases in expected survival of 29% and 16%, respectively; surprisingly, Cluster A pathology was associated with significantly increased survival times.

Prospective Analyses

Each of the aforementioned models used measures of Axis II pathology, DAS, ASQ-N, and ASQ-P obtained at the follow-up assessment, 33 to 78 months after the index hospitalization. However, there were 44 study patients who completed the DAS and 37 who completed the ASQ during hospitalization (pretreatment); 23 patients also completed the PDE Axis II interview at pretreatment. Although, as noted previously, the presence of the depressive episode may substantially bias assessment of these personality and cognitive constructs, use of such in-episode ratings provides a stronger prospective test of the effect of these constructs on subsequent risk of relapse. Accordingly, Models 1–7 were each replicated using index hospitalization (in-episode) measures of cognitive and Axis II measures (see Table 4), including all patients for whom such measures were available in each analysis. The follow-up BDI measure was dropped as a covariate in Models 8–14, inasmuch as it would not be expected to have any confounding influence upon the in-episode measures. The effect of the other four covariates on relapse was reexamined in the absence of the BDI effect. The length of follow-up interval remained significantly predictive ($p < .001$). Because there was also a trend for a significant effect of posttreatment MADRS score ($p = .08$), it was added as a covariate in Models 8–14.

Model 8 included the in-episode DAS score, in addition to the two covariates. No significant effect was observed for the DAS ($p > .80$). Model 9 included in-episode ASQ-N and ASQ-P. Both measures exhibited a nonsignificant trend toward relapse prediction: ASQ-N likelihood ratio $\chi^2(1, N = 37) = 2.76$, $p < .10$; ASQ-P likelihood ratio $\chi^2(1, N = 37) = 3.41$, $p < .10$. As in Model 2, higher scores on ASQ-P were associated with longer survival times (estimated multiplier = 1.03), whereas ASQ-N scores were negatively related to survival (estimated multiplier = 0.97). In Model 10, the in-episode dimensional Axis II rating was significantly predictive of relapse, likelihood ratio $\chi^2(1, N = 23) = 5.77$, $p < .02$, with a coefficient estimate of −0.079 and estimated multiplier of 0.92. Model 11 incorpo-

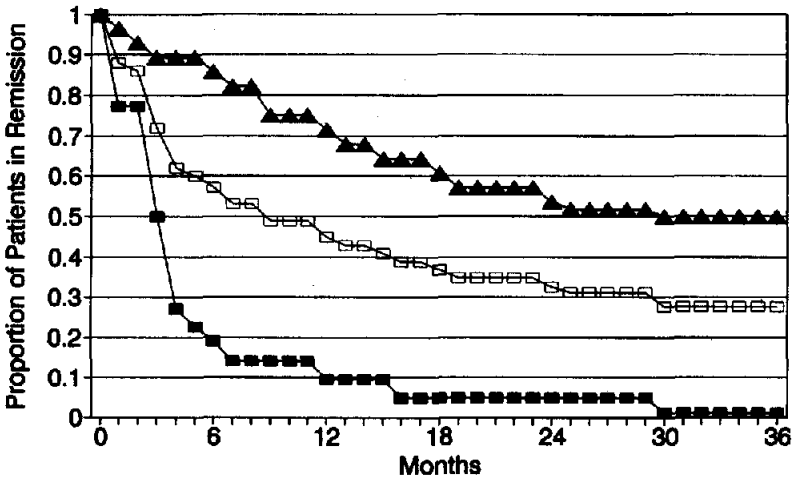

Figure 2. Survival function by Axis II category. Open squares indicate all patients; closed squares indicate Axis II patients; closed triangles indicate non-Axis II patients.

Table 4
Survival Models: In-Episode Measures

Model and Predictors	Coefficient estimate	χ^2	Survival multiplier
Model 8[a]			
DAS	*ns*		
Model 9[b]			
ASQ-N	−0.026	2.76*	0.97
ASQ-P	0.031	3.41*	1.03
Model 10[c]			
Axis II (dimensional)	−0.079	5.77***	0.92
Model 11[d]			
DAS	*ns*		
ASQ-N	*ns*		
ASQ-P	*ns*		
Axis II (dimensional)	−0.11	4.76**	0.89
Model 12[d]			
DAS	*ns*		
ASQ-N	*ns*		
ASQ-P	*ns*		
Axis II (categorical)	*ns*		
Model 13[d]			
Axis II (dimensional)	−0.072	3.80**	0.93
ASQ-P	*ns*		
Model 14[c]			
Cluster A	*ns*		
Cluster B	−0.216	2.97*	0.81
Cluster C	*ns*		

Note. For all chi-square values, the degree of freedom was 1. DAS = Dysfunctional Attitudes Scale; ASQ-N = Attributional Style Questionnaire composite subscale for negative events; ASQ-P = Attributional Style Questionnaire composite subscale for positive events.
[a] $n = 44$. [b] $n = 37$. [c] $n = 23$. [d] $n = 20$.
* $p < .10$. ** $p < .05$. *** $p < .01$.

rated all three in-episode cognitive measures, in addition to the Axis II dimensional rating. As in Model 4, only the Axis II rating was significantly predictive, likelihood ratio $\chi^2(1, N = 20) = 4.76$, $p < .05$. The Axis II coefficient estimate was −0.111, with an estimated multiplier of 0.89. In Model 12, the Axis II categorical measure was used instead of the dimensional Axis II rating. No significant effects were observed. As in Model 6 (above), Model 13 included both the Axis II dimensional rating and the ASQ-P composite score as predictors. The in-episode Axis II measure remained significantly predictive, likelihood ratio $\chi^2(1, N = 20) = 3.80$, $p = .05$, but no significant prediction was observed with the ASQ-P score ($p > .40$). Finally, Model 14 tested the effect of in-episode Axis II cluster scores on relapse. Neither Cluster A nor Cluster C scores were significantly predictive. The Cluster B score was marginally significant, likelihood ratio, $\chi^2(1, N = 23) = 2.97$, $p < .10$. The estimated coefficient was −0.216, with an indicated multiplier of 0.81.

Discussion

Axis II Pathology and Depressive Relapse

This investigation's principal finding concerns the effect of Axis II personality pathology on depressive relapse. A dimensionalized measure of Axis II pathology (calculated as the total number of criteria met for all 11 Axis II disorders on the basis of the follow-up interview) was found to be significantly and substantially associated with relapse, with an approximate 8% cumulative decrease in expected remission duration for each Axis II criterion item met. In light of the strong association that has been observed between personality disorders and depressotypic cognitions (e.g., O'Leary et al., 1991), we also decided to include measures of Axis II pathology, dysfunctional attitudes, and attributional style simultaneously in a survival model, as a means of determining the degree of relapse risk uniquely associated with each of these constructs. Disordered personality continued to exert a robust effect on relapse, regardless of whether such pathology was measured as a dimensional (Model 4) or categorical (Model 5) construct. While controlling for the effects of all cognitive measures and covariates, the survival model indicated that patients without a personality disorder have an expected survival (i.e., remission) period approximately 7.4 times longer than that of patients who met *DSM–III–R* criteria for at least one Axis II disorder. The greatest risk disparity between Axis II and non-Axis II patients was observed during the first 6 months after recovery, during which time 77% (17 of 22) of patients with personality disorders, but only 14% (4 of 28) of patients without personality disorders, had relapsed. It is important to note that these survival models included each patient's follow-up BDI score as a covariate to control statistically for the potentially confounding influence of state depression[8] on both the reporting of Axis II pathology (e.g., Stuart et al., 1992) and retrospective recall of relapse episodes (Goodwin & Sher, 1993). Thus, the observed effect of Axis II pathology on relapse does not appear to be a mere artifact of differential depressive symptomatology among patients at the follow-up assessment.

Because the aforementioned models rely solely on follow-up (as opposed to prospective) measures, they do not permit an unequivocal interpretation of the observed relationship between Axis II and depressive relapse risk. There appear to be two distinct interpretations that could account for this study's finding: (a) Axis II pathology may have served as a genuine risk factor that rendered certain patients more vulnerable to the experience of depressive relapse, or (b) the occurrence of chronic or repeated episodes of depression over the follow-up period may have had a deleterious effect on the personality functioning of a subset of study patients, thereby causing them to report elevated levels of Axis II pathology at follow-up. To address the latter possibility, we conducted additional survival analyses (Models 10–14) using data obtained from that subset of study patients (46%) for whom truly prospective, in-episode interviews of Axis II pathology were available. Not only did Axis II pathology emerge as a significant predictor in these prospective models, but the estimated risk multiplier for the Axis II dimensional variable, calculated as an exponential function of survival model coefficients, remained relatively constant across assess-

[8] It remains to be clarified whether it is specifically the presence of syndromal depression or simply a dysphoric mood state that is primarily responsible for the artifactual reporting bias frequently observed when assessment of personality or cognitive constructs is conducted while patients are depressed. Inasmuch as the BDI measures dysphoria (see Coyne, 1994), as well as a host of cardinal depressive symptoms, inclusion of the BDI as a covariate in this investigation appears to control for both potential sources of reporting bias.

ment periods (Model 4 vs. Model 11). Specifically, expected patient survival time decreased by approximately 8% (compounded) for each additional Axis II criterion met, regardless of whether Axis II pathology was assessed during hospitalization or at follow-up. These prospective findings lend support to the aforementioned hypothesis that Axis II pathology substantially increases a patient's risk for the experience of subsequent relapse episodes.

The differential effects of the three Axis II clusters with respect to relapse were also examined. Dimensional ratings of Clusters A, B, and C (on the basis of follow-up interviews) were entered simultaneously in a survival model; all three ratings were found to add significantly and independently to the relapse model. Intriguingly, however, only Cluster B and Cluster C pathology had a deleterious effect on relapse probability; increased Cluster A pathology was actually found to increase the likelihood of remaining relapse-free. This rather perplexing finding—that variance "unique" to Cluster A (i.e., controlling simultaneously for Clusters B and C) is associated with prolonged remission—is somewhat difficult to explain, although it may be noted that the three disorders that constitute Cluster A (schizoid, schizotypal, and paranoid) are each characterized by some degree of social detachment, a quality that may render such individuals less vulnerable to depressogenic social rejection experiences (Greenberg, Craighead, & Evans, 1996).

Dysfunctional Cognitions and Depressive Relapse

This investigation also examined the effect on relapse of three cognitive constructs: dysfunctional attitudes, attributions for negative events, and attributions for positive events. Neither dysfunctional attitudes nor negative event attributions were found to be significantly associated with depressive relapse, regardless of whether in-episode or follow-up measures of these constructs were used. A significant and substantial effect, however, was observed for the follow-up positive attribution composite measure, ASQ-P. Specifically, the tendency to make internal, stable, and global attributions for positive events served as a type of "buffer" against depressive relapse, with a 6% cumulative increase in expected survival time for each 1-point increase in ASQ-P score; by extension, a 1-*SD* increase in ASQ-P score indicated an approximate doubling of expected survival time. Furthermore, ASQ-P accounted for unique variance in the relapse model, beyond that accounted for by personality pathology. The prospective ASQ-P measure (assessed during patients' index hospitalization) did not appear to be as strongly associated with relapse, though its effect was found to be marginally significant.

Although the reformulated learned helplessness theory has hypothesized a preeminent role for negative event attributions in the onset of depression, the results of this investigation indicate that positive event attributions may, in fact, constitute a more important determinant of relapse risk. Such a finding appears to be somewhat consistent with a model of recovery from depression articulated by Needles and Abramson (1990), which suggests that stable and global attributions for positive events may be central to the process of recovery from depressive affect (see Craighead, 1991; Edelman et al., 1994). Thus, patients who engage in such positive event attributions might be especially resilient in the face of the subclinical dysphoric states that frequently occur subsequent to remission of the depressive syndrome. Because this study represents the first reported investigation of positive event attributions and depression relapse, it would be quite valuable to see a replication.

Methodological Considerations and Future Directions

The failure of both the pretreatment and follow-up DAS measures to exert a significant effect on relapse risk in this investigation stands in stark contrast to the findings of four previous studies (Rush et al., 1986; Segal et al., 1992; Simons et al., 1986; Thase et al., 1992). The most obvious explanation for this discrepancy is the fact that these previous investigations all obtained DAS scores at posttreatment rather than at pretreatment or at follow-up. In fact, one might expect the continued presence of dysfunctional attitudes during the period immediately posttreatment to confer a greater relapse vulnerability than the presence of such dysfunctional cognitions either at pretreatment (when state depression serves as a notable confound) or at a follow-up several years posttreatment (by which time some dysfunctional attitudes may have changed). Thus, the present findings are not viewed as a strong disconfirmation of the hypothesized depressogenic role for dysfunctional attitudes. Nonetheless, because this is the only study of the DAS that has both controlled for depressive symptom severity at the time of DAS assessment and used *DSM* diagnostic criteria to determine patient relapse status, further investigation of the effect of posttreatment DAS on depressive relapse appears warranted. Because dysfunctional attitudes appear to be somewhat mood state dependent (i.e., there is evidence that they remain "latent" until they are primed by the occurrence of a negatively valenced mood state; Miranda & Persons, 1988; Miranda, Persons, & Byers, 1990), we believe that the optimal approach for future investigations would be to assess DAS immediately postremission but in tandem with some form of priming technique.

With a single exception—the dimensional rating of Axis II pathology—all cognitive and personality measures used in this investigation were more strongly associated with relapse when such measures were based on the follow-up, as opposed to the in-episode, assessment. This set of findings may perhaps best be explained as follows: (a) This study's personality assessments involved expert interviewers rather than self-report procedures and, as such, appear to have been less subject to possible artifactual mood-congruent reporting biases during the in-episode assessment than were the study's cognitive measures (Loranger, Lenzenwenger, Gartner, & Susman, 1991); and (b) the dimensional ratings of personality pathology were likely more robust than were categorical personality ratings to any trait–state artifacts that did exist, inasmuch as dimensional personality ratings appear to be more valid indicators of what are likely, in actuality, continuous latent variables (e.g., Trull, Widiger, & Guthrie, 1990).[9]

Several limitations of this study are worth noting. First, data on the longitudinal course of posthospitalization depression symptomatology were obtained from a single follow-up interview that was rather far removed in time (33 to 84 months; *M*

[9] We thank David A. Haaga for providing this explanation for the observed robustness of the dimensional Axis II measure.

= 49.9 months) from the index hospitalization. This lengthy follow-up period undoubtedly also contributed to the substantial loss of potential study participants (36 of whom could not be tracked and 48 of whom declined participation[10]); although there were no significant differences on demographic or pretreatment clinical measures between participants and nonparticipants, the existence of selection biases based on posttreatment patient characteristics cannot be ruled out. Furthermore, the structured interview used to obtain these data, the LIFE, was originally designed for use over a 6-month follow-up period (Keller et al., 1987). Although its designers note that the LIFE is capable of adaptation to "any length . . . of follow-up intervals" (Keller et al., 1987, p. 540), it is reasonable to suspect that accuracy of patient recall of depressive symptomatology may be somewhat attenuated by the large time intervals used in the present investigation; we are aware of no reported data with respect to the reliability and validity of LIFE interviews for major depression conducted beyond the recommended 6-month follow-up window. Accordingly, it would be valuable to see a replication of this study in which patients are assessed at regular 6-month intervals throughout the follow-up period. It should also be noted that the findings reported herein are based on a fairly small sample (n = 50), which affords statistical power sufficient to detect only medium-to-large effects. Finally, although Axis II pathology emerged in this study as an important risk factor for depression relapse, the causal mechanisms by which personality disorders contribute to relapse risk remain unknown (see Ilardi & Craighead, 1994–1995). The Axis II effect on relapse was not related, in this investigation, to differential pharmacotherapy treatment over the follow-up period; indeed, 94% of all study patients continued to receive pharmacotherapy for at least 6 months postremission, and there were no observed differences in pharmacotherapy between Axis II and non-Axis II groups (it remains possible, of course, that medication compliance was lower among Axis II patients—such a hypothesis was not addressed by this investigation). It does seem likely, however, on the basis of the findings discussed herein, that personality pathology contributes to the risk of relapse by means of some mechanism beyond the operation of dysfunctional cognitive processes. In light of the very large effect of Axis II pathology on relapse risk observed in this investigation, elucidation of the specific mechanisms through which personality disorders engender depressive relapse would appear to constitute an important goal for subsequent research endeavors in this area.

[10] Approximately 70% of those who declined participation cited a travel distance in excess of 100 miles (about 160.9 km) as their primary reason.

References

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87,* 49–74.

Alloy, L. B., Lipman, A. J., & Abramson, L. Y. (1992). Attributional style as a vulnerability factor for depression: Validation by past history of mood disorders. *Cognitive Therapy and Research, 16,* 391–407.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders.* New York: International Universities Press.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561–571.

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

Blazer, D., Hughes, D. C., & George, L. K. (1992). Age and impaired subjective support: Predictors of depressive symptoms at one-year follow-up. *Journal of Nervous and Mental Disease, 180,* 172–178.

Brown, G. W., & Harris, T. O. (Eds.). (1989). *Depression: Life events and illness* (pp. 49–93). New York: Guilford Press.

Bumberry, W., Oliver, J. M., & McClure, J. N. (1978). Validation of the Beck Depression Inventory in a university population using psychiatric estimates as the criterion. *Journal of Consulting and Clinical Psychology, 46,* 150–155.

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

Cox, D. R. (1972). Regression models and life tables. *Journal of the Royal Statistical Society, 34*(Series B), 187–220.

Cox, D. R., & Oakes, D. (1984). *Analysis of survival data.* London: Chapman & Hall.

Coyne, J. C. (1994). Self-reported distress: Analog or ersatz depression? *Psychological Bulletin, 116,* 29–45.

Craighead, W. E. (1991). Cognitive factors and classification issues in adolescent depression. *Journal of Youth and Adolescence, 20,* 311–326.

Craighead, W. E., Evans, D. D., Luten, A., Saur, C., McDonald, W. M., Krishnan, K. R. R., Blazer, D. G., & Nemeroff, C. B. (1996). *Montgomery–Asberg Depression Rating Scale: Reliability on an inpatient unit.* Manuscript submitted for publication.

Cronbach, L. F. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika, 15,* 297–334.

Edelman, R. E., Ahrens, A. H., & Haaga, D. A. F. (1994). Inferences about the self, attributions, and overgeneralization as predictors of recovery from dysphoria. *Cognitive Therapy and Research, 18,* 551–566.

Evans, D. D., & Craighead, W. E. (1995, November). *Relationships among cognitive distortions and personality disorders in severely depressed patients.* Paper presented at the 29th annual meetings of the Association for Advancement of Behavior Therapy, Washington, DC.

Frank, E., Prien, R. F., Jarrett, R. B., Keller, M. B., Kupfer, D. J., Lavori, P. W., Rush, A. J., & Weissman, M. M. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder: Remission, recovery, relapse, and recurrence. *Archives of General Psychiatry, 48,* 851–855.

Goodwin, A. H., & Sher, K. J. (1993). Effects of induced mood on diagnostic interviewing: Evidence for a mood and memory effect. *Psychological Assessment, 5,* 197–202.

Greenberg, M. D., Craighead, W. E., & Evans, D. D. (1996). *Personality disorder behaviors as predictors of poor response to inpatient treatment for unipolar depression.* Manuscript submitted for publication. (Available from W. E. Craighead, Department of Psychology, University of Colorado, Boulder, CO 80309-0345)

Haaga, D. A. F., Dyck, M. J., & Ernst, D. (1991). Empirical status of cognitive theory of depression. *Psychological Bulletin, 110,* 215–236.

Ilardi, S. S., & Craighead, W. E. (1994–1995). Personality pathology

and response to somatic treatments for major depression: A critical review. *Depression, 2,* 200–217.

Ilardi, S. S., & Craighead, W. E. (1997). *Personality disorders, depressive symptoms, and dysphoria as predictors of depressotypic cognitions.* Manuscript submitted for publication. (Available from S. S. Ilardi, Department of Psychology, University of Colorado, Boulder, CO 80309-0345)

Keller, M. B., Lavori, P. W., Friedman, B., Nieven, E., Endicott, J., McDonald-Scott, P., & Andreasen, N. C. (1987). The Longitudinal Interval Follow-Up Evaluation: A comprehensive method for assessing outcome in prospective longitudinal studies. *Archives of General Psychiatry, 44,* 540–548.

Klerman, G. L., & Weissman, M. M. (1992). The course, morbidity, and costs of depression. *Archives of General Psychiatry, 49,* 831–834.

Lavori, P. W., Keller, M. B., & Klerman, G. L. (1984). Relapse in affective disorders: A reanalysis of the literature using life table methods. *Journal of Psychiatric Research, 18,* 13–25.

Lewinsohn, P. M., Seinmetz, J. L., Larson, D. W., & Franklin, J. (1981). Depression-related cognitions: Antecedent or consequence? *Journal of Abnormal Psychology, 91,* 213–219.

Loranger, A. W., Lenzenweger, M. F., Gartner, A. F., & Susman, V. L. (1991). Trait–state artifacts and the diagnosis of personality disorders. *Archives of General Psychiatry, 48,* 720–728.

Loranger, A. W., Susman, V. L., Oldham, J. M., & Russakoff, L. (1987). The Personality Disorder Examination: A preliminary report. *Journal of Personality Disorders, 1,* 1–13.

Luria, R. E. (1975). The validity and reliability of the visual analogue mood scale. *Journal of Psychiatric Research, 12,* 51–57.

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.

Montgomery, S. A., & Asberg, M. (1979). A new depression scale designed to be sensitive to change. *British Journal of Psychiatry, 134,* 382–389.

Needles, D. J., & Abramson, L. Y. (1990). Positive life events, attributional style, and hopefulness: Testing a model of recovery from depression. *Journal of Abnormal Psychology, 99,* 156–165.

O'Leary, K. M., Cowdry, R. W., Gardner, D. L., Leibenluft, E., Lucas, P. B., & DeJong-Meyer, R. (1991). Dysfunctional attitudes in borderline personality disorder. *Journal of Personality Disorders, 5,* 233–242.

Persons, J. B., & Rao, P. A. (1985). Longitudinal study of cognitions, life events, and depression in psychiatric inpatients. *Journal of Abnormal Psychology, 94,* 51–63.

Peselow, E. D., Fieve, R. R., & DiFiglia, C. (1992). Personality traits and response to desipramine. *Journal of Affective Disorders, 24,* 209–216.

Pfohl, B., Coryell, W., Zimmerman, M., & Stangl, D. (1984). The implications of *DSM–III* personality disorders for patients with major depression. *Journal of Affective Disorders, 7,* 309–318.

Pfohl, B., Coryell, W., Zimmerman, M., & Stangl, D. (1987). Prognostic validity of self-report and interview measures of personality disorder in depressed inpatients. *Journal of Clinical Psychiatry, 48,* 468–472.

Robins, C. J., & Hayes, A. M. (1995). The role of causal attributions in the prediction of depression. In G. Buchanan & M. E. P. Seligman (Eds.), *Explanatory style* (pp. 71–97). Hillsdale, NJ: Erlbaum.

Robins, L. N., Helzer, J. E., Croughan, J. L., & Ratcliff, K. S. (1981). National Institute of Mental Health diagnostic interview schedule: Its history, characteristics, and validity. *Archives of General Psychiatry, 38,* 381–389.

Rush, A. J., Weissenburger, J., & Eaves, G. (1986). Do thinking patterns predict depressive symptoms? *Cognitive Therapy and Research, 10,* 225–236.

SAS Institute. (1987). *SAS/STAT guide for personal computers, version 6.* Cary, NC: Author.

Segal, Z. V., Shaw, B. F., Vella, D. D., & Katz, R. (1992). Cognitive and life stress predictors of relapse in remitted unipolar depressed patients: Test of the congruency hypothesis. *Journal of Abnormal Psychology, 101,* 26–36.

Seligman, M. E. P., Abramson, L. Y., Semmel, A., & von Baeyer, C. (1979). Depressive attributional style. *Journal of Abnormal Psychology, 88,* 242–248.

Shea, M. T., Elkin, I., Imber, S. D., Sotsky, S. M., Watkins, J. T., Collins, J. F., Pilkonis, P. A., Beckham, E., Glass, D. R., Dolan, R. G., & Parloff, M. B. (1992). Course of depressive symptoms over follow-up: Findings from the National Institute of Mental Health Treatment of Depression Collaborative Research Program. *Archives of General Psychiatry, 49,* 782–787.

Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin, 86,* 420–428.

Simons, A. D., Murphy, G. E., Levine, J. L., & Wetzel, R. D. (1986). Cognitive therapy and pharmacotherapy for depression: Sustained improvement over one year. *Archives of General Psychiatry, 43,* 43–48.

Singer, J. D., & Willett, J. B. (1991). Modeling the days of our lives: Using survival analysis when designing and analyzing longitudinal studies of duration and the timing of events. *Psychological Bulletin, 110,* 268–290.

Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry, 35,* 773–782.

Stangl, D., Pfohl, B., Zimmerman, M., Bowers, W., & Corenthal, C. (1985). A structured interview for the *DSM–III* personality disorders. A preliminary report. *Archives of General Psychiatry, 42,* 591–596.

Stuart, S., Simons, A. D., Thase, M. E., & Pilkonis, P. (1992). Are personality assessments valid in acute major depression. *Journal of Affective Disorders, 24,* 281–289.

Sweeney, P. D., Anderson, K., & Bailey, S. (1986). Attributional style in depression: A meta-analytic review. *Journal of Personality and Social Psychology, 50,* 974–991.

Thase, M. E., Simons, A. D., McGeary, J., Cahalane, J. F., Hughes, C., Harden, T., & Friedman, E. (1992). Relapse after cognitive behavior therapy of depression: Potential implications for longer courses of treatment. *American Journal of Psychiatry, 149,* 1046–1052.

Thompson, L. W., Gallagher, D., & Czirr, R. (1988). Personality disorder and outcome in the treatment of late-life depression. *Journal of Geriatric Psychiatry, 21,* 133–153.

Trull, T. J., Widiger, T. A., & Guthrie, P. (1990). Categorical versus dimensional status of borderline personality disorder. *Journal of Abnormal Psychology, 99,* 40–48.

Weissman, A. N., & Beck, A. J. (1978, November). *Development and validation of the Dysfunctional Attitude Scale.* Paper presented at the 12th Annual Meeting of the Association for Advancement of Behavior Therapy, Chicago.

Zimmerman, M., Coryell, W., Pfohl, B., Corenthal, C., & Stangl, D. (1986). ECT response in depressed patients with and without a *DSM–III* personality disorder. *American Journal of Psychiatry, 143,* 1030–1032.

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