

# Cognitive Predictors of Symptom Return Following Depression Treatment

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This study tested whether poor cognitive change during depression treatment predicted time to return of depressive symptoms. Depressed participants ( $N = 121$ ) completed assessments of dysfunctional attitudes and extreme thinking (i.e., number of *totally agree* and *totally disagree* responses) during hospitalization and again after 6 months of outpatient treatment. Participants then completed monthly depression assessments for 1 year. Survival analyses for time to symptom recurrence during follow-up were conducted among participants who reported 50% improvement in their depressive symptoms and were at least partially asymptomatic at the end of treatment ( $n = 53$ ). Poor change in dysfunctional attitudes and poor change in extreme thinking both predicted shorter time to return of depressive symptoms.

Depression is the most common psychiatric disorder found in the general public (Blazer, Kessler, McGonagle, & Swartz, 1994). At any given time, approximately 5% of Americans are depressed (30-day point prevalence); over 17% report at least one episode of depression in their lifetime (Blazer et al., 1994). Depression is also a recurrent disorder. The 2-month relapse rate for unipolar depression is 20% to 24% (Belsher & Costello, 1988). Six months after recovery, relapse rates range from 27% (Keller, Lavori, Lewis, & Klerman, 1983; Simons, Murphy, Levine, & Wetzel, 1986) to 34% (Keller, Shapiro, Lavori, & Wolfe, 1982). Two years after recovery, over 50% report a depressive relapse (Keller & Shapiro, 1981).

Several cognitive models have been proposed to explain these high rates of relapse (for a review, see Ingram, Miranda, & Segal, 1998). Beck, Rush, Shaw, and Emery (1979) suggested that stressful life events can activate dysfunctional attitudes, which, in turn, lead to the onset of depressive symptoms. More recent cognitive models emphasize the form, rather than the content, of dysfunctional thinking (Segal, Williams, & Teasdale, 2002). Teasdale et al. (2001) argued that extreme, absolutistic forms of thinking, either positive or negative in content, confers depressive suscep-

tibility. Although there are important theoretical differences between models, both posit that improved cognitive functioning (e.g., change in the content or form of dysfunctional thinking) should render a person less vulnerable to future episodes of depression.

Several studies have documented associations between depression risk and dysfunctional thought content. For instance, in the presence of dysphoric moods, people with a past history of depression report greater levels of dysfunctional attitudes than people who have never been depressed (e.g., Miranda, Persons, & Byers, 1990; Persons & Miranda, 1992; Roberts & Kassel, 1996; Solomon, Haaga, Brody, Kirk, & Friedman, 1998). Cognitive theories of depression vulnerability, however, posit that cognitive factors are causal agents in the disorder (Ingram et al., 1998). If so, cognitive functioning should also be associated with future depressive episodes. Segal, Gemar, and Williams (1999) found support for this idea. Among a sample of people recently remitted from depression, change in dysfunctional thought content (as assessed by the Dysfunctional Attitudes Scale; Weissman, 1979) from before to after a dysphoric mood induction was prospectively associated with an increased risk of relapse 1 to 4 years after initial testing.

Relatively less work has examined the connection between the form of negative thinking and depression susceptibility. One such study was conducted by Teasdale et al. (2001), who examined whether change in the form of depression-related thinking during preventive treatment predicted relapse among people partially remitted from a major depressive episode. In their sample of 158 individuals, Teasdale et al. (2001) found that extreme thinking (i.e., number of *totally agree* or *totally disagree* responses on measures of depressive cognition) reduced over the course of treatment. However, people with higher levels of posttreatment extreme thinking relapsed more quickly during the 60-week follow-up period. This finding remained intact even after controlling for severity of depression.

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In related work, across a series of studies, Teasdale et al. (2002) found that change in "metacognitive awareness" was associated with longer survival times to relapse following cognitive therapy (Beck et al., 1979) and mindfulness-based cognitive therapy (Segal et al., 2002). Teasdale et al. (2002, p. 275) defined metacognitive awareness as "a cognitive set in which negative thoughts/feelings are experienced as mental events, rather than as the self." They reported that change in metacognitive awareness was responsible for the reduction of relapse associated with cognitive therapy and mindfulness-based cognitive therapy. However, as noted by Teasdale et al. (2002), because relapse occurred before the posttreatment assessment of metacognitive awareness for some individuals, they were unable to demonstrate formally whether change in metacognitive awareness fully mediated the relapse prevention effects.

Finally, although several depression treatment studies have found that cognitive change is positively associated with treatment outcome (e.g., Blackburn & Bishop, 1983; DeRubeis et al., 1990; Seligman et al., 1988), relatively less research has examined whether it mitigates the return of symptoms following treatment. A study by Tang and DeRubeis (1999) provides some indirect evidence. They examined the session-by-session depression severity data for participants who received cognitive-behavioral treatment in the Elkin et al. (1989) and the Hollon et al. (1992) depression treatment efficacy studies. They found that cognitive change (e.g., belief change, accepting a new cognitive technique) was more likely to occur in sessions that preceded large decreases in depressive symptoms (sudden gains) than in sessions that were not followed by large symptom change. Interestingly, individuals who experienced sudden gains were less depressed at the end of treatment, and that difference was maintained at an 18-month follow-up assessment. However, that study did not examine whether cognitive change per se was associated with lower levels of depression at follow-up.

In summary, several recent longitudinal studies have linked cognitive change with depression vulnerability. Participants in those studies were either recently remitted (Segal et al., 1999) or partially remitted from a major depressive episode (Teasdale et al., 2002; Teasdale et al., 2001). Among people who were being treated for a major depressive episode, one study has documented that large, sudden decreases in depressive symptoms during treatment, which were often preceded by cognitive change, are associated with ensuing reductions in depression susceptibility (Tang & DeRubeis, 1999). However, there is relatively little evidence that directly tests whether cognitive change decreases subsequent vulnerability among people receiving treatment for a major depressive episode. We offer the study here as one such test.

This research was conducted within the context of a larger study examining the efficacy of combined treatment for severe depression (Miller, Keitner, Ryan, Solomon, & Cardemil, 2003). Dysfunctional attitudes and extreme response style were first assessed during psychiatric hospitalization for depression. After discharge, participants were randomized to 6 months of acute outpatient treatment. Following outpatient treatment, participants' dysfunctional attitudes and extreme thinking were reassessed, allowing for an assessment of change. Participants were then monitored for 1 year, including monthly assessments of depression.

Because we wanted to predict time to symptom return during the follow-up period, we selected a subsample of participants who

reported significant depression improvement and were at least partially asymptomatic following acute depression treatment. Among this subsample, we examined whether pretreatment to posttreatment change in dysfunctional attitudes and extreme thinking could predict time to recurrence of clinically significant depressive symptoms during the yearlong follow-up period. Based on cognitive models of vulnerability to depression, we expected that (a) poor change in dysfunctional attitudes would be associated with a shorter interval to the return of significant depressive symptoms in the year following acute treatment; (b) poor change in extreme thinking would also be associated with a shorter interval to the return of significant depressive symptoms in the year following acute treatment; and (c) cognitive change would be associated with the return of depressive symptoms even after controlling for pretreatment to posttreatment change in depression and other relevant participant variables.<sup>1</sup>

## Method

### Design

Participants were selected from a larger study examining the efficacy of combined pharmacological and psychosocial treatments for depression. Because the methodology of the treatment study was described in detail in Miller et al. (2003), we provide only a brief summary. Participants were recruited during an admission to a psychiatric hospital. Hospital treatment consisted of pharmacotherapy and milieu treatment. Following discharge, participants were randomized to 6 months of outpatient pharmacotherapy or pharmacotherapy plus psychosocial treatment. The psychosocial treatments provided were family (Epstein & Bishop, 1981), cognitive-behavioral (Beck et al., 1979), or combined cognitive-behavioral and family treatment.

The larger study was also designed to examine whether matching treatment type to participant characteristics improved treatment efficacy. Participants were provided outpatient treatment that either matched or mismatched their deficits. People with relatively high family impairment and low cognitive distortion could be randomized to receive either medication plus family therapy or medication plus cognitive therapy. The former treatment is a match for the participant's deficits, whereas the latter is a mismatch. Persons with high cognitive distortion and poor family functioning could be randomized to a combined treatment of medication, cognitive, and family therapy (match) or to medication alone (mismatch). (See Miller et al., 2003, for more information on the treatment-matching algorithm as well as the results of this treatment-matching approach.)

Immediately after this period of acute outpatient treatment, participants entered the follow-up phase of the study, which lasted 1 year. During this year, participants completed either (a) continuation of treatment offered during the outpatient phase but at a reduced frequency ( $n = 29$ ); (b) treatment based on clinical need provided by study personnel ( $n = 3$ ) or by community providers ( $n = 13$ ); (c) no treatment ( $n = 3$ ); or (d) unknown treatment ( $n = 5$ ). Throughout the follow-up period, all participants (regardless of treatment status) were contacted monthly and completed interviewer-based depression assessments.

<sup>1</sup> We acknowledge that the labels *poor* and *good* cognitive change are evaluative. These descriptors are intended to refer to both the direction and magnitude of change. For instance, the labels *less* or *more* cognitive change only describe the magnitude of change. That is, both large decreases and large increases in dysfunctional thinking reflect more cognitive change; however, in this example, only large decreases should be associated with a reduction in depression vulnerability.

## Participants

Upon admission to the psychiatric hospital, all participants: (a) met criteria for major depressive disorder according to the Structured Clinical Interview for *DSM-III-R* diagnoses (SCID; Spitzer & Williams, 1985); (b) had Modified Hamilton Rating Scale for Depression (MHRSD; Miller, Bishop, Norman, & Maddever, 1985) and Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) scores greater than 17; (c) were 18 to 65 years of age; and (d) had sufficient reading skills to complete questionnaires. Individuals were excluded from the study if they (a) met *Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R*; American Psychiatric Association, 1987) criteria for bipolar disorder, alcohol or drug dependence, somatization disorder, or schizophrenia; (b) met criteria for dementia or displayed significant cognitive impairment; or (c) presented with a medical illness severe enough to contraindicate antidepressant medication.

Because the main focus of this study was to predict the return of depressive symptoms, we retained for analyses a subsample of participants who reported significant improvement in their symptoms and were at least partially asymptomatic following treatment. For the purposes of this study, we identified this subsample with the following criteria: (a) 50% or greater reduction in MHRSD score from admission to post-outpatient treatment, and (b) a posttreatment MHRSD score of 12 or less. A 50% reduction in MHRSD score is a commonly used criterion in depression treatment outcome studies to define symptomatic improvement (e.g., Walsh, Seidman, Sysko, & Gould, 2002). Given the severity of depressive symptoms reported by participants upon admission, we also used an absolute MHRSD score criterion to exclude participants who were experiencing elevated symptoms despite meeting the improvement criterion. A similar absolute criterion has been previously used in depression vulnerability research (e.g., Segal et al., 1999).

Of the 121 participants recruited for the treatment study, 66 met criteria for treatment response. Of the 66, 13 were excluded from analyses due to missing data: 5 were missing demographic or questionnaire data used in analyses, and 8 had insufficient follow-up data (i.e., missing more than 50% of follow-up data). We thus had a total of 53 participants in this study. Retained participants did not significantly differ from excluded participants in years of education, socioeconomic standing, total number of previous episodes, duration of previous episodes, pretreatment depression, pretreatment dysfunctional attitudes, posttreatment depression, and posttreatment dysfunctional attitudes ( $ps > .08$ ). However, people excluded were significantly younger ( $M = 32.14$ ,  $SD = 10.05$  and  $M = 38.51$ ,  $SD = 10.28$ ),  $t(65) = -2.08$ ,  $p < .05$ , and had a younger age of onset for their first episode of depression ( $M = 24.29$ ,  $SD = 8.10$  and  $M = 30.77$ ,  $SD = 13.99$ ),  $t(65) = -2.24$ ,  $p < .05$ , compared to those retained.

## Measures

**Modified Hamilton Rating Scale for Depression.** The 17-item MHRSD (Miller et al., 1985) is a widely used interview-based measure of severity of depressive symptomatology with acceptable reliability and validity (Miller et al., 1985). The MHRSD has been demonstrated to have excellent interrater reliability and to correlate highly with the original Hamilton Rating Scale for Depression (Miller et al., 1985). Assessors were trained clinical raters. The MHRSD was used to determine pretreatment to posttreatment change in depression as well as to assess severity of depression during the follow-up phase.

**Dysfunctional Attitudes Scale (DAS; Weissman, 1979).** The DAS total score served as our index of dysfunctional attitudes. The DAS form A has 40 statements to which participants respond on a 7-point scale (*totally disagree to totally agree*). The DAS assesses dysfunctional beliefs that are thought to reflect a person's self-evaluation. DAS items measure concerns about approval from others, prerequisites for happiness, and perfectionistic standards. The DAS has been used widely in depressed and psychiatric control populations (Oliver & Baumgart, 1985). The short form of the DAS

has good internal consistency ( $\alpha = .85$ ; Oliver & Baumgart, 1985) and a test-retest correlation of .84 over an 8 week period (Weissman, 1979).

**Extreme response style.** To compute our index of extreme thinking, the number of extreme responses (i.e., *totally agree* or *totally disagree*) on the DAS were summed. Greater number of extreme responses on measures of depression-related thought content has been associated with a shorter time to relapse among individuals partially remitted from depression receiving preventative treatment (Teasdale et al., 2001).

## Return of Clinically Significant Symptoms

Return of clinically significant symptoms was defined as an MHRSD score of 17 or greater during the follow-up period. This is a commonly used criterion to indicate clinically significant depressive symptoms. An MHRSD score of 17 or greater has frequently been used as a criterion for entry into pharmacological treatment of depression (see Endicott, Cohen, Nee, Fleiss, & Sarantakos, 1981). Because the MHRSD was designed to measure severity of a depressive episode, it does not assess *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV*; American Psychiatric Association, 1994) diagnostic criteria. Although it is likely that people who report MHRSD scores of 17 or greater would meet *DSM-IV* criteria for a major depressive disorder, additional information is needed to establish a diagnosis. We thus use the phrase "return of clinically significant depressive symptoms" rather than "relapse."

## Timing of Assessments

After admission to the hospital, participants were administered the SCID, DAS, MHRSD, as well as other instruments not included in this report. The MHRSD and the DAS were readministered following 6 months of outpatient treatment. MHRSD assessments were also conducted on a monthly basis during the follow-up period. Among participants retained for this study, 47/53 (87%) completed at least 10 monthly MHRSD assessments.

## Data Analysis

To estimate cognitive change, we utilized a residualized change score (DuBois, 1957). Residualized change scores are generally preferred over raw score change because residualized change scores account for regression to the mean. This approach generates the linear association between pretreatment and posttreatment measurements and then estimates how far each observation deviates from the expected linear association. Residuals greater than the predicted linear association have positive values. These values indicate higher posttreatment scores than expected based upon scores at admission. Residuals below the expected linear association have negative values and indicate lower than expected posttreatment values given respective scores at admission. In this study, positive residuals reflect worse than expected cognitive change, and negative residuals indicate better than expected cognitive change. Residuals are then used to predict the return of depressive symptoms during follow-up.

Cox regression survival analysis tested whether cognitive change was associated with time to recurrence of depressive symptoms during the follow-up period. Time to recurrence was coded as the month in which the person first met criteria for the return of depressive symptoms (i.e.,  $MHRSD \geq 17$ ). We then estimated degree of increased rate of symptom recurrence for a one-unit increase in cognitive change (i.e., hazard ratio). In addition, we examined the increased rate of symptom recurrence for people with relatively poor change for each cognitive predictor. Poor change was defined as one standard deviation above the mean.

## Results

### Patient Characteristics

See Table 1 for descriptive statistics of participant characteristics. Participants were predominantly female, middle-aged, Cau-

Table 1  
*Sample Descriptive Statistics (N = 53)*

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	%
Female	36			67.9
Caucasian	49			92.5
Married	26			49.1
Age (years)	53	38.50	10.27	
Education (years)	53	13.47	2.59	
Age at first episode of depression (years)	53	30.77	13.98	
Duration of previous episode (months)	53	19.37	59.62	
No. of previous episodes, lifetime	53	3.21	3.28	
Hospital treatment				
Length of hospital stay (days)	53	13.96	7.20	
Outpatient treatment (Months 1–6)				
Pharmacological treatment only	10			18.9
Pharmacological plus psychosocial treatment	43			81.1
Completed full course of treatment	40			75.5

casian, and high school educated. On admission, participants exhibited high levels of depression (MHRSD  $M = 23.91$ ,  $SD = 4.36$ ) and reported an average of 3.21 previous episodes of lifetime depression ( $SD = 3.28$ ). Average length of hospital stay for the current episode was approximately 2 weeks. Following discharge from the hospital, the majority received combined pharmacological and psychosocial treatment. Most participants completed a full course (6 months) of outpatient treatment. Finally, 43% (23/53) reported the return of clinically significant depressive symptoms during the follow-up period.

#### *Change in Depression, Dysfunctional Attitudes, and Extreme Thinking During Acute Outpatient Treatment*

We expected that overall levels of dysfunctional attitudes and extreme thinking would be significantly lower at posttreatment than at pretreatment. We first confirmed that there was a significant reduction in depression by conducting a repeated measure  $t$  test with pretreatment and posttreatment depression as the dependent variable. Not surprisingly, MHRSD scores were significantly higher at hospital admission ( $M = 23.91$ ,  $SD = 4.36$ ) than at posttreatment ( $M = 4.94$ ,  $SD = 3.67$ ),  $t(52) = 29.33$ ,  $p < .001$ .

To assess overall cognitive change during outpatient treatment, identical analyses were conducted with pretreatment and posttreatment dysfunctional attitudes and extreme thinking. As expected, there was a significant reduction in dysfunctional attitudes,  $t(52) = 4.36$ ,  $p < .001$ ; dysfunctional attitudes were significantly higher at admission ( $M = 152.19$ ,  $SD = 41.48$ ) than posttreatment ( $M = 123.98$ ,  $SD = 36.94$ ). Overall levels of extreme thinking did not significantly decrease over the course of treatment ( $M = 12.41$ ,  $SD = 10.76$  and  $M = 12.85$ ,  $SD = 11.39$ ),  $t(52) = 0.30$ , *ns*. The correlation between pretreatment and posttreatment number of extreme responses was large ( $r = .56$ ), suggesting that the tendency to endorse extreme responses was relatively stable.

#### *Patient Predictors of Symptom Return*

Before testing whether cognitive change during outpatient treatment was associated with the return of depressive symptoms during follow-up, we first wanted to account for the influence of participant characteristics. Participant characteristics significantly

associated with time to the return of depressive symptoms were retained and used as covariates in subsequent analyses.

Participant variables were entered simultaneously into a Cox regression survival analysis predicting time to symptom return during the follow-up period. Variables entered were age, gender, marital status, age at onset of first depressive episode, duration of previous episode, number of lifetime previous episodes, whether a full course (6 months) of acute outpatient treatment was completed (no, yes), and whether acute outpatient treatment included cognitive therapy (yes, no).<sup>2</sup>

The overall model was statistically significant,  $-2 \log$  likelihood ( $-2LL$ ) = 156.97,  $\chi^2(8, N = 53) = 19.90$ ,  $p < .05$ . Of the variables entered, number of lifetime episodes of depression and participant age were significantly associated with time to recurrence of depressive symptoms. More previous depressive episodes and older participant age were both associated with a shorter time to symptom recurrence. These two participant predictors were thus used as covariates in subsequent survival analyses (see Table 2).

In addition to these participant variables, we also included pretreatment to posttreatment residualized change in depression as a covariate. Controlling for depression change during treatment is particularly important given the previously documented association between cognitive functioning and depressed mood states (e.g., Zuroff, Blatt, Sanislow, Bondi, & Pilkonis, 1999). Controlling for change in depression also determines whether cognitive change uniquely predicts time to symptom return beyond that predicted by change in depression.

#### *Change in Dysfunctional Attitudes and Extreme Thinking as Predictors of Symptom Return*

Prior to testing hypotheses, descriptive statistics were examined (see Table 3). Not surprisingly, DAS scores, number of extreme responses on the DAS, and MHRSD scores measured before

<sup>2</sup> Type of treatment (medication alone, medication plus family therapy, medication plus cognitive therapy, medication plus cognitive therapy and family therapy) was not associated with the return of depressive symptoms during maintenance period,  $\chi^2(3, N = 53) = 0.55$ ,  $p = .95$ . We thus collapsed across treatment type to increase statistical power.

Table 2  
Participant Predictors of Time to Symptom Return: Cox Regression Survival Analysis

Step and variable	b	SE	Wald $\chi^2$	p	e <sup>b</sup>	95% CI (e <sup>b</sup> )
Step 1						
Patient age	.07	.03	4.60	<.05	1.08	1.0063–1.1512
Gender	.40	.58	0.47	<.50	1.48	0.4787–4.6053
Marital status	-.02	.13	0.02	<.90	0.98	0.7654–1.2579
Age at first episode onset	-.05	.03	2.77	<.10	0.95	0.9038–1.0083
Duration of previous episode	.00	.00	0.42	<.55	1.00	0.9960–1.0080
Episodes of depression, lifetime	.14	.06	5.59	<.05	1.15	1.0243–1.2927
Treatment completion	-.92	.52	3.08	<.10	0.40	0.1436–1.1129
Cognitive therapy	.25	.53	0.22	<.65	1.28	0.4519–3.6383

Note. e<sup>b</sup> = hazard ratio; CI = confidence interval; df = 1 for all Wald statistics.

treatment were significantly correlated with their respective post-treatment measurements. DAS and MHRSD scores were significantly positively associated at posttreatment but not at pretreatment.

Notably, DAS total score and number of extreme DAS responses were significantly inversely associated at pretreatment and posttreatment. This counterintuitive association occurred because participants reported more extreme positive than negative responses at pretreatment (positive,  $M = 7.70$ ,  $SD = 8.54$ ; negative,  $M = 4.72$ ,  $SD = 6.06$ ) and posttreatment (positive,  $M = 10.51$ ,  $SD = 10.40$ ; negative,  $M = 2.33$ ,  $SD = 4.71$ ). That is, people were more likely to *totally agree* with functional DAS items or *totally disagree* with dysfunctional DAS items than *totally agree* with dysfunctional DAS items or *totally disagree* with functional DAS items. Considered independently, number of extreme positive responses was inversely associated with DAS total score at pretreatment and posttreatment assessments ( $r_s = -.71$  and  $-.74$ , respectively), whereas number of extreme negative responses was positively associated with total DAS score ( $r_s = .52$  and  $.51$ , respectively).

We next examined whether cognitive change was associated with time to symptom return. The covariates (number of lifetime episodes of depression, participant age, and residualized change in depression) were entered in the first step of the Cox regression survival analysis. These variables were significantly associated with symptom return,  $-2LL = 161.90$ ,  $\chi^2(3, N = 53) = 16.25$ ,  $p < .01$ . In the second step, residualized change scores for dysfunctional attitudes and for number of extreme responses were entered. These variables significantly improved the predictive ability of the model,  $-2LL = 153.83$ ,  $\Delta\chi^2(2, N = 53) = 8.07$ ,  $p < .05$ .<sup>3</sup>

As can be seen in Table 4, previous episodes of depression were associated with time to symptom recurrence. The hazard ratio indicated that for every additional previous episode of depression, the rate to symptom recurrence increases by 19%. Importantly, poor change in dysfunctional attitudes and poor change in number of extreme responses were also associated with shorter intervals to symptom recurrence. For every one-unit increase in change in dysfunctional attitudes, rate to symptom recurrence increases by approximately 2%. For an individual with relatively poor change in dysfunctional attitudes (i.e., one standard deviation above mean change), rate to symptom recurrence increases by 106% when other predictors in the model are set to their respective means.

Similarly, for every one-unit increase in number of extreme responses, rate to symptom recurrence increases by approximately 6%. For an individual with relatively poor change in number of extreme responses, rate to symptom recurrence increases by 81% when statistically adjusting for other predictors in the model. Figure 1 presents the adjusted survival probabilities across the follow-up period for both predictors.<sup>4</sup>

#### Absolute Levels of Cognitive Predictors of Symptom Return

The preceding analyses suggest that cognitive change is associated with time to the return of depressive symptoms. A related question is whether absolute levels of posttreatment dysfunctional attitudes and extreme thinking could predict symptom return. That is, regardless of levels at admission, we examined whether elevated levels of dysfunctional attitudes and number of extreme responses following acute outpatient treatment could predict the return of depressive symptoms during follow-up.

A Cox regression survival analysis tested this idea. In the first step, we entered the same control variables as before, number of previous episodes of depression and participant age. We wanted to control for depression severity following acute outpatient treatment, so we also included posttreatment MHRSD depression score as a covariate in the first step. These variables were significantly associated with symptom return,  $-2LL = 158.85$ ,  $\chi^2(3, N = 53) = 18.68$ ,  $p < .001$ . As before, number of previous episodes of depression significantly predicted time to return of depressive symptoms but participant age did not. Posttreatment depression

<sup>3</sup> Change in posttreatment dysfunctional attitudes and dichotomous thinking was also significantly associated with time to symptom return when entered in separate steps of a hierarchical Cox regression survival analysis. In this analysis, control variables were entered in the first step, pretreatment depression severity, pretreatment dysfunctional attitudes, and pretreatment extreme thinking were entered in the second step, and posttreatment depression severity, posttreatment dysfunctional attitudes, and posttreatment extreme thinking were entered in the third step.

<sup>4</sup> Residualized change scores for extreme responses to positive and negative DAS items were examined separately in subsequent analyses. Both predicted the return of depressive symptoms in the expected fashion when considered independently.

Table 3  
*Bivariate Correlations, Means, and Standard Deviations for Predictors of Symptom Return*

Variable	1	2	3	4	5	6	7	8
1. No. of episodes, lifetime		-.24	.07	.16	-.09	.24	-.03	-.05
2. Patient age			.03	-.33*	.12	-.05	-.07	.13
3. Pretreatment MHRSD				-.16	.11	.33*	-.03	.06
4. Pretreatment DAS total score					-.27*	.08	.28*	-.09
5. Pretreatment DAS extreme responses						-.05	.02	.56*
6. Posttreatment MHRSD							.42*	-.28*
7. Posttreatment DAS total score								-.47*
8. Posttreatment DAS extreme responses								
<i>M</i>	3.21	38.51	23.91	152.18	12.41	4.94	123.98	12.84
<i>SD</i>	3.28	10.28	4.36	41.48	10.76	3.69	36.94	11.39

Note. MHRSD = Modified Hamilton Rating Scale for Depression; DAS = Dysfunctional Attitudes Scale.

\*  $p < .05$ .

severity also predicted symptom return; higher depression severity predicted a shorter interval to symptom recurrence.

In the second step, posttreatment DAS total score and posttreatment number of extreme DAS responses were entered. These variables did not significantly improve the predictive ability of the model,  $\Delta\chi^2(2, N = 53) = 4.31, p = .12$ . The effects of posttreatment total DAS score and posttreatment number of DAS extreme responses on time to return of symptoms also did not reach statistical significance (see Table 5).

### Discussion

The overall goal was to examine whether cognitive change during acute outpatient depression treatment could prospectively predict the return of clinically significant symptoms of depression in the following year. Participants were drawn from a larger study examining the efficacy of combined treatments for depression (Miller et al., 2003). Participants who showed clinical improvement in their depression and were at least partially asymptomatic following treatment were retained for analyses.

The main findings were: (a) poor change in dysfunctional attitudes during treatment was associated with a shorter time to symptom return during the follow-up period; (b) poor change in extreme thinking during treatment was also associated with shorter time to symptom return during the follow-up period; and (c) absolute levels of dysfunctional attitudes and extreme thinking following outpatient treatment were not significantly associated with time to symptom return. Importantly, all analyses controlled for the influence of participant variables such as number of lifetime episodes of depression, participant age, and pretreatment to posttreatment change in depression.

Taken together, the results of this work support cognitive models of depression vulnerability (Beck et al., 1979; Teasdale et al., 2001). Interestingly, although all participants exhibited significant symptom reduction during acute outpatient treatment, participants varied in the direction and magnitude of cognitive change they experienced. Better than expected cognitive change (relative to scores at admission) predicted longer times to symptom recurrence, whereas worse than expected change predicted shorter times to symptom recurrence. The importance of this finding is bolstered by the fact that cognitive change predicted the return of depressive symptoms even after accounting for change in depression, thus

supporting the idea that cognitive change was not simply an epiphenomenon of depression change.

The finding that change in dysfunctional thought content prospectively predicted time to symptom recurrence is consistent with Beck et al.'s (1979) cognitive theory of depression, which suggests that people who possess overly rigid, negativistic beliefs about the self are at heightened risk for depression. Although this finding is consistent with theory, it must also be reconciled with several previous studies that did not find such a relationship between dysfunctional attitudes and future depression (e.g., Hart, Craighead, & Craighead, 2001; Lewinsohn, Steinmetz, Larson, & Franklin, 1981; Teasdale et al., 2001).

Sample composition may be one important difference between this study and previous work. The sample here consisted of people who were recruited during hospitalization for severe depression. Previous work has used community (Lewinsohn et al., 1981), partially remitted depressed (Teasdale et al., 2001), and fully remitted depressed (Hart et al., 2001) samples. As depression severity and dysfunctional attitudes are often positively associated (Miller & Norman, 1986; Norman, Miller, & Dow, 1988), highly depressed people may report significantly greater dysfunctional thinking than their moderately depressed counterparts. Reductions in dysfunctional thought content may thus have a more important role in mitigating depression susceptibility among severely depressed people compared to people with more mild forms of the disorder. Future work is needed to examine associations between cognitive change and depression vulnerability among individuals who range in depression severity.

A second potential explanation for why the findings diverge from some previous work is that we used a relative measure of dysfunctional thought content. That is, vulnerability was associated with poor change in dysfunctional thought content during treatment. People who were high in dysfunctional thought content at the end of treatment relative to their level at the beginning of treatment were especially vulnerable to symptom recurrence. This pattern of results is similar to other research that reported change in dysfunctional attitudes from before to after a dysphoric mood induction predicts subsequent depression vulnerability among remitted depressed people (Segal et al., 1999). It may be that certain cognitive vulnerabilities need to be assessed repeatedly, both in the absence and presence of dysphoric mood states (either induced or

Table 4  
Cognitive Change as Predictors of Time to Symptom Return: Cox Regression Survival Analysis

Step and variable	b	SE	Wald $\chi^2$	p	$e^b$	95% CI ( $e^b$ )
Step 1						
Episodes of depression, lifetime	.17	.06	9.52	<.01	1.19	1.0651–1.3268
Patient age	.01	.03	0.28	<.60	1.01	0.9635–1.0665
Change in MHRSD	.07	.07	0.83	<.40	1.07	0.9247–1.2393
Step 2						
Change in DAS	0.02	.01	7.81	<.01	1.02	1.0061–1.0354
Change in extreme responses	0.06	.03	4.02	<.05	1.06	1.0015–1.1322

Note.  $e^b$  = hazard ratio; CI = confidence interval; MHRSD = Modified Hamilton Rating Scale for Depression; DAS = Dysfunctional Attitudes Scale;  $df = 1$  for all Wald statistics.

naturally occurring), in order to assess fully their contribution to depression vulnerability. As suggested by findings from Segal et al., (1999) and the work here, the discrepancy between cognitive functioning when depressed mood is low versus high may yield a particularly sensitive measurement of cognitive vulnerability.

Dysfunctional thought content was not the only significant prospective predictor of depression vulnerability. Specifically, poor change in extreme thinking (operationalized as number of *totally agree* and *totally disagree* responses on the DAS) was also associated with time to symptom return during the yearlong follow-up period. Compared to dysfunctional thought content, this type of vulnerability may be particularly insidious; a remitted depressed person may report an extremely positive outlook, yet the form of his or her thinking belies a vulnerability to depression.

Teasdale et al. (2001) suggested that extreme thinking is associated with depression vulnerability because it reflects rapid, automatic information processing that is uncorrected by subsequent reappraisal. This type of uncorrected automatic information processing is thought to produce extreme or exaggerated cognitive products, which, in turn, lead to extreme emotional reactions and thus confer vulnerability (also, see Beck et al., 1979, chap. 1). Subtle changes in how depression vulnerable people process information, then, may substantially influence their course of recovery following treatment. Additional research is needed to further test this intriguing hypothesis.

Several strengths of this work are notable. The prospective design builds upon past research, which has relied primarily upon cross-sectional designs that compare cognitive functioning of remitted depressed people with people who have never been depressed (e.g., Hedlund & Rude, 1995; Ingram, Bernet, & McLaughlin, 1994; Miranda & Persons, 1988). The temporal sequencing of cause and effect here provides clues to causality, but, of course, third variable explanations cannot be ruled out. However, this work is consistent with several recent longitudinal studies among people treated for depression that indicate poor cognitive change predicts future intensification of depression (Segal et al., 1999; Teasdale et al., 2001).

To increase our confidence in the results, we tried to minimize competing explanations by statistically controlling for variables that were also associated with symptom return. Given the established state-dependent association between dysfunctional attitudes and depression (Zuroff et al., 1999), perhaps the most important alternative explanation we controlled for was change in depression during treatment. We also controlled for participant variables such as number of previous episodes of depression and participant age. Even after these statistical controls, relatively poor change in dysfunctional attitudes and extreme thinking predicted the return of depressive symptoms.

Several limitations of this work should also be noted. Additional research is needed to test the generalizability of these findings. Participants in this study were derived from a larger study examining the efficacy of combined treatments in the posthospital care of depression. Because depression severity was severe enough to warrant inpatient hospitalization and because the larger study examined combined treatment modalities, participants received relatively intensive treatment. Whether these findings apply to less severely depressed populations that receive less intensive treatment is unclear. However, it should also be noted that severely

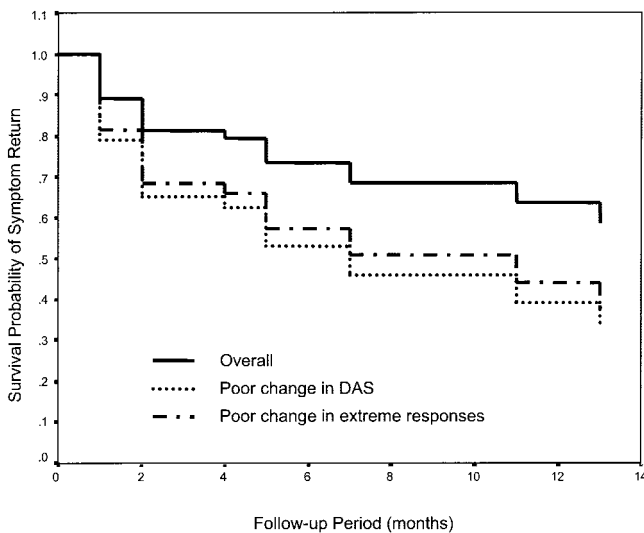


Figure 1. Adjusted survival probability of symptom return during follow-up period. Survival functions for each predictor are adjusted for the effects of other predictors in the model. Each predictor is set to its mean for the overall survival function. For Dysfunctional Attitudes Scale (DAS) survival function, change in DAS predictor is set to one standard deviation above the mean and all other predictors are set to their respective means. For extreme response survival function, change in extreme responses is set to one standard deviation above the mean, and all other predictors are set to their mean. Survival functions can be generated for any value along each predictor's continuum of values, but one standard deviation from the mean is an often-used convention to identify high and low values.

Table 5  
*Absolute Levels of Posttreatment Cognitive Vulnerabilities as Predictors of Time to Symptom Return: Cox Regression Survival Analysis*

Step and variable	b	SE	Wald $\chi^2$	p	e <sup>b</sup>	95% CI (e <sup>b</sup> )
Step 1						
Episodes of depression, lifetime	.14	.05	7.51	<.01	1.15	1.0408–1.2721
Patient age	.02	.03	0.41	<.60	1.02	0.9669–1.0690
Posttreatment MHRSD	.13	.07	3.95	<.05	1.14	1.0019–1.2990
Step 2						
Posttreatment DAS	.01	.01	3.79	<.10	1.01	0.9999–1.0247
Posttreatment extreme responses	.03	.02	2.32	<.15	1.03	0.9911–1.0743

Note. e<sup>b</sup> = hazard ratio; CI = confidence interval; MHRSD = Modified Hamilton Rating Scale for Depression; DAS = Dysfunctional Attitudes Scale; *df* = 1 for all Wald statistics.

depressed people are often excluded from studies of depression because of high levels of suicidality. Such a recruitment bias provides little data about a subgroup of depressed individuals who may be particularly vulnerable to future episodes of depression. Therefore, although the generalizability of the findings may be questioned, this study does provide important information about a subgroup of depressed individuals who are generally understudied.

Another potential limitation is that we used monthly MHRSD interviews to measure severity of depressive symptoms throughout the study. A constraint of this approach is that the MHRSD does not assess nosological criteria. Given this limitation, we defined the return of clinically significant symptoms with a MHRSD score commonly used for admission into depression treatment studies (Endicott et al., 1981). One benefit of using monthly MHRSD interviews, however, is that it limits bias associated with retrospective recall given the relatively short intervals between depression assessments.

Another possible limitation is our reliance on self-report assessments of cognitive functioning, which are susceptible to demand effects. However, given that depression assessments were conducted repeatedly throughout the follow-up period, it is less likely that demand effects are solely responsible for the findings. In addition, our measure of extreme thinking (i.e., number of extreme scores on the DAS) may be less susceptible to demand effects because of its unconventional approach to scoring. Nevertheless, future research should examine whether other approaches to the assessment of cognitive functioning (e.g., implicit processes; Gamar, Segal, Sagrati, & Kennedy, 2001), as well change in other noncognitive features of depression (e.g., physiological functioning; Charles, Schittecatte, Rush, Panzer, & Wilmotte, 1989; Coryell, 1990), also predict return of depressive symptoms.

A final limitation is that this work did not fully test the diathesis–stress component of cognitive models of depression vulnerability, as negative life events were not measured during follow-up. We are thus left to assume that poor change in dysfunctional attitudes and dichotomous thinking put participants at risk for future depression because of its interaction with life stress. Future research should measure life events during the follow-up period in order to test fully the diathesis–stress component of cognitive theories of depression.

In conclusion, this work suggests that poor change in dysfunctional thought content and poor change in extreme thinking during treatment were both associated with time to recurrence of depres-

sive symptoms. Important work remains to be done to identify specific interventions that reliably produce cognitive change (e.g., Tang & DeRubeis, 1999). Perhaps with such research, future depression treatments can be developed to lower the exceedingly high relapse rates. Data from this study indicate that treatments designed to change both the content and form of dysfunctional thinking may be particularly likely to achieve this important goal.

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