

# Unlinking Negative Cognition and Symptoms of Depression: Evidence of a Specific Treatment Effect for Cognitive Therapy

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In this study, the authors examined whether cognitive therapy alters the association between negative cognition and symptoms of depression. Participants were recruited during psychiatric hospitalization for depression. Following discharge, they were randomly assigned to 6 months of outpatient treatment. Treatment consisted of pharmacotherapy either alone or in combination with cognitive therapy and/or family therapy. Following this 6-month treatment period, negative cognition and symptoms of depression were assessed monthly for 1 year. Hierarchical linear modeling indicated that the association between negative cognition and depression during follow-up was weaker for patients randomized to cognitive therapy than for patients who did not receive cognitive therapy. Cognitive therapy appeared to unlink negative cognition and symptoms of depression to a greater extent than other forms of treatment.

Recent advances in cognitive models of depression suggest that how a person thinks when dysphoric may reveal a vulnerability to depression (Ingram, Miranda, & Segal, 1998). That is, although vulnerable and less vulnerable people may report relatively low levels of negative thinking when euthymic, increases in negative thinking as dysphoria increases (often referred to as cognitive reactivity) may reflect depression risk. Interventions that influence the association between negative cognition and symptoms of depression, such as cognitive therapy (Beck, Rush, Shaw, & Emery, 1979), may alter this vulnerability to depression. However, to our knowledge few studies have examined whether cognitive treatment changes how an individual thinks as symptoms of depression become more pronounced.

The majority of research has examined whether cognitive therapy reduces dysfunctional thinking at the end of treatment more so than other forms of treatment. In these studies, participants typically report their levels of negative cognition before and after depression treatments. In general, successful treatment is consistently associated with reductions in dysfunctional thinking. However, cognitive therapy does not appear to differentially modify dysfunctional thinking. At the end of treatment, remitted depressed people reported similar levels of dysfunctional thinking regardless of treatment modality (e.g., Fava, Bless, Otto, Pava, & Rosenbaum, 1994; Kovacs, Rush, Beck, & Hollon, 1981; Simons, Garfield, & Murphy, 1984).

These null treatment findings are also consistent with descriptive psychopathology research. In this research, dysfunctional thinking appeared to remit along with the symptoms of depression (e.g., Dohr, Rush, & Bernstein, 1989; Imber et al., 1990; Silverman, Silverman, & Eardley, 1984). Dysfunctional thinking of remitted depressed people was often no different from (e.g., E. W. Hamilton & Abramson, 1983) or was slightly higher than nondepressed controls (e.g., Peselow, Robins, Block, Barouche, & Fieve, 1990). Therefore, dysfunctional thinking appeared to wax and wane with the onset and remission of depression. Taken together, this pattern of results understandably led some to question the etiological significance of dysfunctional thinking for depression (Simons et al., 1984).

More recent research indicates that dysfunctional thinking may indeed have a significant role for depression. Although negative cognition appears to remit along with symptoms of depression, people with a history of depression are more likely to report dysfunctional thinking in the presence of negative mood states than people with no such history (Segal & Ingram, 1994). This cognitive reactivity to dysphoria has been observed for naturally occurring symptoms of depression (Miranda, Persons, & Byers, 1990), as well as for negative mood states induced in the laboratory (Miranda, Gross, Persons, & Hahn, 1998; Miranda & Persons, 1988; Roberts & Kassel, 1996; Solomon, Haaga, Brody, Kirk, & Friedman, 1998). Perhaps most important, greater increases in negative thinking following a dysphoric mood induction were associated with an increased risk of depressive relapse among remitted depressed individuals (Segal, Gemar, & Williams, 1999).

Given these more recent findings, additional research is needed to determine whether depression treatments alter the relationship between negative cognition and depression. That is, do present treatments change the rate at which dysfunctional thinking increases as dysphoria increases? Cognitive therapy seems particularly likely to alter this putative diathesis, as treatment focuses on changing patterns of negative thinking activated by life stress (Persons & Miranda, 2002). To date, only one published study to our knowledge has examined the impact of cognitive therapy on negative thinking following increases in dysphoria. In Segal et

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al.'s (1999) study, a questionnaire that assessed dysfunctional attitudes was administered before and after a dysphoric mood induction among people recently remitted from depression after cognitive therapy or pharmacotherapy. Cognitive reactivity was defined as change in dysfunctional attitudes from before to after the dysphoric mood induction. Consistent with previous research, participants did not differ in their dysfunctional attitudes prior to the mood induction regardless of treatment modality. However, people who received cognitive therapy reported significantly less cognitive reactivity than those who received pharmacotherapy.

These findings provide substantial support for the hypothesis that cognitive therapy specifically changes how a person thinks in the presence of dysphoria. However, a potential limitation of that study, noted by its authors, is that treatment was not randomly assigned. Therefore, the factors that self-selected people into cognitive therapy may have been partly responsible for the decreased cognitive reactivity observed in the cognitive therapy condition. Research that examines the ability of other psychosocial treatments (e.g., family therapy) to alter the relationship between cognition and depression is also needed. Doing so would help determine whether factors specific to cognitive therapy are responsible for altering the link between cognition and depression.

In the present study, we examine whether cognitive therapy alters the association between negative cognition and symptoms of depression. That is, as depressive symptoms escalate, are people who received cognitive therapy less likely to report negative cognition than those who did not receive cognitive therapy? To address this question, we assessed negative thinking and noncognitive symptoms of depression monthly for 1 year following treatment completion. We then estimated the rate at which negative cognition increased as depression increased during the yearlong follow-up period. We expected that cognitive therapy, but not family therapy, would weaken the association between negative cognition and depression.

## Method

### Design

All study procedures complied with the protocol approved by the Internal Review Board at Butler Hospital (Providence, Rhode Island). Participants were recruited for a study that examined the efficacy of combined pharmacological and psychosocial treatments for the posthospital care of severe depression. Because the methodology of the primary outcome study has been described in more detail elsewhere (Miller et al., in press), we provide only a brief summary. Patients were recruited during an admission

to a private psychiatric hospital. Treatment during hospitalization 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 cognitive (Beck et al., 1979), family (Epstein & Bishop, 1981), or combined cognitive and family treatment.

The larger treatment study was also designed to examine whether matching treatment type to patient characteristics improved treatment efficacy. Patients were provided outpatient treatment that either matched or mismatched their deficits. For example, people with relatively poor family functioning and low cognitive bias were randomized to receive either medication plus family therapy or medication plus cognitive therapy. The former treatment is a match for patients' deficits, whereas the latter is a mismatch. People with poor family functioning and high cognitive bias were randomized to a combined treatment of medication, cognitive, and family therapy (match) or to medication alone (mismatch; see Table 1). For the results of this treatment matching approach, see Miller et al.'s (in press) study.

Within the context of this treatment matching design, it was possible to examine the effects of treatment approaches by comparing those patients who received (a) cognitive therapy versus no cognitive therapy (Cells 1, 3, 5, 7 vs. Cells 2, 4, 6, 8 in Table 1) and (b) family therapy versus no family therapy (Cells 1, 4, 6, 7 vs. Cells 2, 3, 5, 8 in Table 1). These comparisons were neither confounded by patient characteristics used to match patients to treatment nor with treatment matching (i.e., matched vs. mismatched treatment). That is, regardless of cognitive bias and family functioning, patients were randomized to treatment that either included or did not include cognitive therapy. The same was true for the family therapy versus no family therapy comparison.

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 one of the following treatments: (a) continuation of treatment offered during the outpatient phase but at a reduced frequency ( $n = 46$ ), (b) treatment based on clinical need and provided by study personnel ( $n = 12$ ) or by community providers ( $n = 33$ ), (c) no treatment ( $n = 12$ ), or (d) unknown treatment ( $n = 18$ ). An important finding was that the probability of continued treatment during follow-up (either with study personnel or community providers) versus no or unknown treatment did not differ for those who received cognitive therapy compared with those who did not receive cognitive therapy,  $\chi^2(1, N = 121) = 1.23, ns$ . Throughout the follow-up period, all participants (regardless of treatment status) were contacted to complete monthly assessments of negative cognition and depression symptoms.

### Participants

Participants consisted of 121 patients recently admitted to inpatient and partial hospitalization units at a private psychiatric hospital. On admission, all participants (a) met criteria for major depressive disorder according to

Table 1  
*Matching Design Used to Allocate Patient Groups to Treatment*

Patient groups		Treatments			
Cognitive distortion	Family functioning	Combined cognitive plus family therapy	Combined cognitive therapy	Combined family therapy	Pharmacotherapy
High	Poor	1. Match	—	—	2. Mismatch
High	Good	—	3. Match	4. Mismatch	—
Low	Poor	—	5. Mismatch	6. Match	—
Low	Good	7. Mismatch	—	—	8. Match

Note. A dash indicates that the cell was not included in the study.

Structured Clinical Interview for *DSM-III-R*—Patient Version diagnoses (First, Spitzer, Williams, & Gibbon, 1995), (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 between 18 and 65 years of age, (d) had sufficient reading skills to complete questionnaires, (e) were presently living with one or more family members, and (f) provided informed consent to participate in the project (both patient and family). Patients were excluded from the study if they (a) met *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *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. All participants received a complete explanation of the study, and they provided signed informed consent.

Of the 121 participants who entered the study, 22 were not included in the present analyses because they did not complete any monthly assessments following outpatient treatment. The remaining 99 were retained for analyses. A comparison of the included and excluded participants revealed no differences in age, duration of previous episodes, age of onset for first episode, length of inpatient stay, and inpatient depression severity ( $p > .15$ ). However, the people who were retained had significantly more years of education than those dropped from analyses ( $M = 13.56$ ,  $SD = 2.73$ , and  $M = 12.00$ ,  $SD = 2.12$ , respectively),  $F(1, 119) = 6.35$ ,  $p < .05$ . Of those dropped from analyses, 7 were randomized to medication only, 4 to medication plus cognitive therapy, 9 to medication plus family therapy, and 2 to medication plus cognitive therapy plus family therapy. There were no significant differences for treatment assignment between people removed from analyses compared with those retained,  $\chi^2(3, N = 121) = 6.01$ ,  $ns$ .

To determine depression remission, we identified a subgroup of participants that were experiencing relatively low levels of depression at the end of acute outpatient treatment. We refer to this subgroup as remitted depressed. Consistent with our previous work (Miller et al., in press), we defined remission as a 17-item MHRSD of  $<7$ , a BDI total score of  $<9$ , and completion of outpatient treatment. Of the 121 participants randomized to treatment, 29 met remission criteria. Of the remitted depressed, 18 received cognitive therapy, and 11 did not receive cognitive therapy,  $\chi^2(1, N = 29) = 2.25$ ,  $ns$ . Of these 29, 1 person was not included in analyses because all follow-up data were missing.

### Acute Outpatient Treatment Conditions

All treatments began after discharge from the hospital and continued for a 24-week period. The specific components of each treatment condition are described below.

**Pharmacotherapy.** The pharmacotherapy condition consisted of a semistructured medication protocol and clinical management sessions with one of two board-certified psychiatrists. The medication protocol required a prescription of a U.S. Food and Drug Administration-approved antidepressant at recommended therapeutic dosages for at least a 4-week trial, although choice of the specific antidepressant was left to the clinical judgment of the psychiatrist. Antipsychotic and antianxiety agents could also be used as clinically indicated. If the patient did not respond to the first trial of medication, then subsequent trials with different antidepressants were initiated. Appointment scheduling was determined by the clinical judgment of the psychiatrist, with a maximum of 20 visits during the 24-week treatment period. Clinical management sessions followed guidelines described by Fawcett, Epstein, Fiester, Elkin, and Autry (1980).

**Combined cognitive therapy.** The combined cognitive therapy condition consisted of pharmacotherapy as described above plus individual cognitive therapy, as outlined by Beck et al. (1979). Cognitive therapy was provided by one of three doctoral-level clinical psychologists, each with at least 5 years of clinical experience, who had been trained as cognitive

therapists. Before treating patients in the study, all therapists were certified as competent by an external cognitive therapy expert (Marjorie Weishaar). In addition, all therapists received ongoing supervision from another highly experienced cognitive therapist (Stephen Bishop). Scheduling of cognitive therapy visits was determined by the clinical judgment of the cognitive therapist, with a maximum of 24 cognitive therapy sessions during the treatment period.

**Combined family therapy.** The combined family therapy condition consisted of pharmacotherapy plus family therapy based on problem centered systems therapy of the family (Epstein & Bishop, 1981). Family therapy was provided by one of two family therapists with a master's degree in social work and at least 5 years of clinical experience who were certified as competent in problem centered systems therapy of the family by one of the developers of the model (Duane Bishop). In addition, both family therapists received ongoing supervision from this same family therapy expert. The scheduling of sessions was at the discretion of the family therapist, with a maximum of 20 sessions.

**Combined cognitive plus family therapy.** The combined cognitive plus family therapy condition consisted of pharmacotherapy, combined cognitive therapy, and combined family therapy as described above.

### Assessments

**MHRSD.** The MHRSD (Miller et al., 1985) is a 25-item, interview-based assessment of depression severity with acceptable reliability and validity. The MHRSD contains 17 items that assess the same symptoms as the original Hamilton Rating Scale for Depression (M. Hamilton, 1960). These include depressed mood, guilt, suicidal ideation, insomnia, appetite, weight loss, energy level, anxiety, hypochondriasis, patient insight, and psychomotor retardation and agitation. Ratings for these items are summed and typically used to determine depression severity (i.e., 17-item MHRSD score). The MHRSD also contains an additional 8 items that assess other aspects of depression. Specifically, these additional items assess worthlessness, helplessness, hopelessness, quality of mood, mood reactivity, and diurnal variation in symptoms. The MHRSD has excellent interrater reliability, and the 17-item MHRSD correlates highly with the original Hamilton Rating Scale for Depression (Miller et al., 1985).

**Depression severity.** Monthly follow-up MHRSD assessments were completed via telephone or in person. Telephone assessments were used to reduce participant burden and to minimize missing data given the frequency of assessments. Research has suggested that depression assessments conducted via the phone produce very similar results to those conducted in person (Simon, Revicki, & VonKorff, 1993). Depression severity during follow-up was assessed with the 17-item MHRSD, with the following 3 items removed. We removed the guilt item because we did not want item overlap with our assessment of negative cognition (see below). Psychomotor retardation and agitation were also not included, as an in-person interview is required to observe these symptoms.

The resulting 14-item MHRSD depression severity score ranged from 0 to 42. At the end of acute outpatient treatment, the internal reliability (coefficient alpha) of the 14-item depression scale was .89. Coefficient alpha ranged from .86 to .89 for the 12 subsequent assessments during the follow-up period. The 14-item and 17-item versions of the MHRSD obtained at the end of acute outpatient treatment were highly correlated ( $r = .99$ ).

**Negative cognition.** Negative cognition was assessed with 4 MHRSD items that assessed guilt, worthlessness, helplessness, and hopelessness. Each item was assessed on a 4-point scale, allowing total scores to range from 0 to 16. These items were not used to determine depression severity in the 14-item MHRSD. Although guilt often refers to an affective state, this item was included in the negative cognition scale because it inquires about guilty cognitions, such as self-criticism. For instance, "Are you critical of yourself for your weaknesses or mistakes?" The coefficient alpha for this scale immediately after completion of acute outpatient treatment

was .90. Coefficient alphas ranged from .85 to .90 during the 12-month follow-up period.

Although this measure is brief, evidence has suggested that short measures of straightforward constructs are often as valid as longer ones (Burisch, 1984). To assess construct validity, we examined correlations between our measure of negative cognition and longer, well-established assessments of similar constructs. At the end of outpatient treatment, correlations between this four-item measure of negative cognition and the Dysfunctional Attitudes Scale (DAS; Weissman, 1979), Cognitive Bias Questionnaire (Krantz & Hammen, 1979), and Hopelessness Scale (Beck, Weissman, Lester, & Trexler, 1974) were .49, .44, and .70 ( $ps < .05$ ), respectively. The measure of negative cognition used for the present study, thus, had considerable overlap with other self-report questionnaires of negative cognition.

### Timing of Assessments

Thirteen monthly MHRSD assessments were conducted following completion of acute outpatient treatment (Months 6–18). Month 6 coincided with the end of outpatient treatment, and 12 monthly MHRSD assessments then ensued. Of 1,287 possible MHRSD assessments from 99 participants, 1,133 interviews were completed (88%). A total of 69 participants completed all 13 MHRSD assessments, 76 completed at least 90%, 82 completed at least 75%, and 88 completed at least 50%. The remaining 11 participants completed at least 1 assessment but less than 50%.

### Statistical Model

Hierarchical linear modeling (HLM; Bryk & Raudenbush, 1992) was used to analyze the data. HLM can estimate variation among within-subjects and between-subjects variables simultaneously. It is ideal for longitudinal data, as it is able to accommodate missing data at the within-subjects level with empirical Bayesian estimates. Conceptually, HLM estimates a regression model for each individual with all available data at the within-subjects level. A second regression model at the between-subjects level estimates associations among between-subjects and within-subjects parameters. Within- and between-subjects parameters are tested simultaneously with maximum likelihood methods of estimation.

Our goal was to determine whether variance in the association between negative cognition and depression, a within-subjects factor, was associated with between-subjects factors, such as treatment assignment. To do so, we tested a model of negative cognition that was based on the statistical model of hopelessness presented by Young et al. (1996). This model assumes that negative cognition has two components: amount of negative cognition in the absence of depression (referred to here as basal negative cognition) plus change in negative cognition as a function of depression severity.<sup>1</sup> In regression terms, basal negative cognition is the intercept (i.e., level of negative cognition when other variables are set to 0), and the slope represents change in negative cognition for a 1-unit increase in depression. These within-subjects parameters are expected to be normally distributed and to vary between individuals.

Between-subjects analyses can then determine whether variance in within-subjects parameters is associated with between-subjects factors. For instance, we can test whether the association between negative cognition and depression is weaker for people who received cognitive therapy compared with those who did not receive cognitive therapy. In addition, we can examine whether different clinical predictors (e.g., age of onset) are associated with each within-subjects parameter. Finally, we can account for the effects of time by including a variable that reflects when the assessment occurred during follow-up.

Such a multilevel model can be conveyed in algebraic terms. The within-subjects model is as follows:

$$NC_{ij} = \beta_{0j} + \beta_{1j}(Dep_i) + \beta_{2j}(Month_i) + r_{ij} \quad (1)$$

where NC represents negative cognition;  $\beta_{0j}$  represents participant  $j$ 's predicted negative cognition score when depression severity and time equal zero (basal negative cognition);  $\beta_{1j}$  represents the linear change in negative cognition per unit increase in depression severity;  $\beta_{2j}$  represents the linear change in negative cognition per unit increase of time; Dep represents depression severity; Month represents time (Months 6–18) when the assessment was conducted, with Month 6 coded as 0 and each subsequent month incremented by 1; and  $r_{ij}$  is the residual term associated with observation  $i$  and person  $j$ .

Simultaneously, the following between-subjects model can also be tested:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Treat}_j) + \mu_{0j} \quad (2)$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Treat}_j) + \mu_{1j} \quad (3)$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(\text{Treat}_j) + \mu_{2j} \quad (4)$$

where  $\gamma_{01}$  is the cross-level interaction term representing the effect of treatment on basal negative cognition;  $\gamma_{11}$  is the cross-level interaction term representing the effect of treatment on the relationship between negative cognition and depression;  $\gamma_{21}$  is the cross-level interaction term representing the effect of treatment on linear change in cognition over time;  $\gamma_{00}$  represents basal negative cognition when treatment is coded as 0;  $\gamma_{10}$  represents negative cognition and depression slope when treatment is coded as 0;  $\gamma_{20}$  represents linear change in negative cognition over time when treatment is coded as 0; and  $\mu_{0j}$ ,  $\mu_{1j}$ , and  $\mu_{2j}$  represent between-subjects residual terms.

## Results

### Patient Characteristics

Participants were predominantly women (73%), middle aged ( $M = 38.52$  years,  $SD = 11.12$ ), and high school educated ( $M = 13.57$  years of education,  $SD = 2.73$ ). Of the participants, 93% were Caucasian, 4% were African American, and 3% were Hispanic. On admission, patients exhibited high levels of depression (17-item MHRSD  $M = 24.18$ ,  $SD = 4.64$ ). Average length of hospital stay for the present episode was 14.13 days ( $SD = 8.25$ ). Following discharge from the hospital, 53 received cognitive therapy (24 received pharmacotherapy plus cognitive therapy; 29 received pharmacotherapy plus cognitive therapy plus family therapy) and 46 did not receive cognitive therapy (24 received pharmacotherapy alone; 22 received pharmacotherapy plus family therapy) as part of their treatment. Depression and negative cognition scores at hospitalization, posttreatment (6 months), 12 months, and 18 months are presented in Table 2.

### Within-Subjects Null Model

We first examined the within-subjects model without estimating between-subjects parameters. The mean, variance, and covariance among within-subjects parameters are presented in Table 3. The parameters for basal negative cognition, the negative cognition and depression slope, and time were significantly different from zero. Furthermore, the time parameter indicated that negative cognition significantly decreased during the course of the follow-up assessments. These parameters also had significant variance, indicating that

<sup>1</sup> In our description, we use the term *basal* instead of *baseline* to avoid confusion about when assessments were conducted, as baseline is often used to refer to an initial assessment.

Table 2

*Descriptive Statistics for Depression and Negative Cognition Scores at Baseline, 6 Months, 12 Months, and 18 Months by Treatment Condition*

Time	Received CT treatment		No CT treatment		Received FT treatment		No FT treatment	
	Dep	NC	Dep	NC	Dep	NC	Dep	NC
Hospital	21.39 (4.17)	9.34 (2.45)	21.57 (4.64)	9.11 (2.58)	21.61 (4.15)	8.71 (2.74)	21.33 (4.63)	9.79 (2.09)
6 months	9.17 (7.87)	4.04 (3.93)	10.79 (8.14)	5.19 (4.10)	9.75 (8.51)	4.20 (3.73)	10.20 (7.44)	5.04 (4.35)
12 months	8.89 (8.11)	3.97 (4.08)	10.00 (7.29)	3.57 (3.60)	10.06 (8.54)	4.02 (4.04)	8.70 (6.70)	3.52 (3.65)
18 months	8.02 (8.11)	2.93 (3.72)	7.75 (7.67)	3.50 (3.60)	7.06 (7.31)	2.70 (3.61)	8.80 (8.42)	3.75 (3.66)

*Note.* Depression and negative cognition assessments were also conducted at Months 7, 8, 9, 10, 11, 13, 14, 15, 16, and 17 but are not reported for sake of table clarity. Values in the table represent means and standard deviations (in parentheses). CT = cognitive therapy; FT = family therapy; Dep = 14-item Modified Hamilton Rating Scale for Depression (MHRSD) total score; NC = four-item MHRSD negative cognition scale.

the magnitude of these parameters differed across individuals. In addition, the within-subjects parameters were somewhat distinct, as they did not significantly covary. That is, basal negative cognition at the end of treatment ( $\beta_0$ ) was not related to the association between negative cognition and depression ( $\beta_1$ ). Similarly, the association between negative cognition and depression ( $\beta_1$ ) was only modestly correlated with change in negative cognition over time ( $\beta_2$ ).

#### *Patient Predictors of Within-Subjects Parameters*

Next, we examined whether patient predictors could explain variability in basal negative cognition, the strength of association between negative cognition and depression, and change in negative cognition over time. We did not make specific hypotheses about which clinical predictors would be associated with each within-subjects parameter; instead, we considered these analyses to be exploratory. Before entering variables into analyses, we confirmed that patient variables were not strongly associated with each other

to avoid multicollinearity. Correlations among patient variables ranged from |.01| to |.22|.

Results of these analyses are presented in Table 4. Age of onset was the only patient predictor significantly associated with basal negative cognition. Younger age of onset for the first depressive episode was significantly associated with higher basal negative cognition. In contrast, patient education, patient gender, and duration of index episode significantly predicted strength of association between negative cognition and depression. People with lower patient education, shorter duration of index episode, and female gender had stronger associations between negative cognition and depression than people with higher patient education, longer index episodes, and male gender, respectively. Furthermore, longer length of hospital stay and shorter duration of index episode were significantly associated with decreases in negative cognition over time. An interesting finding was that basal negative cognition and the negative cognition–depression slope were associated with different patient predictors.

Table 3

*Parameter Estimates for the Null Within-Subjects Model*

Random effect	Random effect model parameter		
	Coefficient	SE	<i>t</i> (98)
Basal ( $\beta_0$ )	1.16	0.21	5.63*
Association between NC and depression ( $\beta_1$ )	0.32	0.01	25.72*
Change in NC over time ( $\beta_2$ )	−0.05	0.02	−2.97*
	<i>SD</i>	Var	$\chi^2(98)$
Variance of basal NC ( $\mu_0$ )	1.63	2.65	259.55*
Variance of association between NC and depression ( $\mu_1$ )	0.07	0.01	164.96*
Variance in change in NC over time ( $\mu_2$ )	0.10	0.01	156.63*
	$\beta_0$	$\beta_1$	$\beta_2$
	Correlations among random effects parameters		
Basal NC ( $\beta_0$ )			
Association between NC and depression ( $\beta_1$ )	.01		
Change in NC over time ( $\beta_2$ )	−.19	−.21	

*Note.* NC = negative cognition; Var = variance component.

\*  $p < .05$ .

Table 4

Parameter Estimates for Patient Predictors of Basal Negative Cognition ( $\beta_0$ ), the Association Between Negative Cognition and Depression ( $\beta_1$ ), and Change in Negative Cognition Over Time ( $\beta_2$ )

Fixed effect model parameter	Coefficient	SE	<i>t</i> (92)	ES
Basal NC ( $\beta_0$ )				
Intercept ( $\gamma_{00}$ )	1.10	0.28	3.81*	.37
Patient education (years; $\gamma_{01}$ )	-0.02	0.08	-0.26	.03
Patient gender ( $\gamma_{02}$ )	0.49	0.52	0.93	.10
Length of inpatient stay ( $\gamma_{03}$ )	-0.01	0.02	-0.52	.05
Duration of index episode ( $\gamma_{04}$ )	$-2.42 \times 10^{-3}$	$2.21 \times 10^{-3}$	-1.10	.11
Age at time of first depression onset ( $\gamma_{05}$ )	-0.04	0.01	-2.86*	.29
Association between NC and depression ( $\beta_1$ )				
Intercept ( $\gamma_{10}$ )	0.32	0.02	18.84*	.89
Patient education (years; $\gamma_{11}$ )	-0.01	$3.97 \times 10^{-3}$	-2.76*	.28
Patient gender ( $\gamma_{12}$ )	0.07	0.03	2.21*	.22
Length of inpatient stay ( $\gamma_{13}$ )	$2.07 \times 10^{-3}$	$1.25 \times 10^{-3}$	1.65	.17
Duration of index episode ( $\gamma_{14}$ )	$-4.50 \times 10^{-4}$	$1.56 \times 10^{-4}$	-2.89*	.29
Age at time of first depression onset ( $\gamma_{15}$ )	$3.00 \times 10^{-4}$	$9.95 \times 10^{-4}$	0.31	.03
Change in NC over time ( $\beta_2$ )				
Intercept ( $\gamma_{20}$ )	-0.05	0.02	-2.56*	.26
Patient education (years; $\gamma_{21}$ )	0.01	0.01	1.21	.13
Patient gender ( $\gamma_{22}$ )	-0.04	0.03	-1.28	.13
Length of inpatient stay ( $\gamma_{23}$ )	$-3.45 \times 10^{-3}$	$1.58 \times 10^{-3}$	-2.18*	.22
Duration of index episode ( $\gamma_{24}$ )	$3.47 \times 10^{-4}$	$1.63 \times 10^{-4}$	2.13*	.22
Age at time of first depression onset ( $\gamma_{25}$ )	$1.64 \times 10^{-3}$	$1.38 \times 10^{-3}$	1.19	.12

Note. Each nondichotomous variable was centered around its mean; gender was coded as men = 0 and women = 1. ES = effect size correlation; NC = negative cognition.

\*  $p < .05$ .

### Treatment Effects on Within-Subjects Parameters

**Cognitive therapy.** We next added cognitive therapy as a between-subjects variable to the previous analysis. Thus, patient characteristics served as covariates. The cognitive therapy variable was dummy coded, with 0 assigned to patients who did not receive cognitive therapy and 1 assigned to those who did receive cognitive therapy. Cognitive therapy was not a significant predictor of basal negative cognition,  $\gamma_{06} = 0.07$  ( $SE = 0.39$ ),  $t(92) = 0.20$ , *ns* (effect size  $r = .01$ ). In contrast, the association between negative cognition and depression was significantly weaker for people who received cognitive therapy than for people who did not receive cognitive therapy,  $\gamma_{16} = -0.05$  ( $SE = 0.02$ ),  $t(92) = 1.99$ ,  $p < .05$  (effect size  $r = .20$ ). For every 1 point increase on the MHRSD Depression scale, negative cognition was .05 points lower for people who received cognitive therapy than for people who did not receive cognitive therapy. Furthermore, assignment to cognitive therapy was not significantly associated with linear change in negative cognition over time,  $\gamma_{26} = 0.04$  ( $SE = 0.03$ ),  $t(92) = 1.13$ , *ns* (effect size  $r = .12$ ).<sup>2</sup>

**Family therapy.** We next tested the specificity of these findings. To do so, we created a dummy-coded family therapy variable, with 0 assigned to patients who did not receive family therapy as part of their treatment and 1 assigned to those who did receive family therapy. Family therapy was not significantly associated with basal negative cognition,  $\gamma_{06} = -0.15$  ( $SE = 0.42$ ),  $t(92) = -0.35$ , *ns*. Similarly, the association between negative cognition and depression was not significantly different for people who did or did not receive family therapy,  $\gamma_{06} = 0.00$  ( $SE = 0.02$ ),  $t(92) = -0.18$ , *ns*. Finally, family therapy was not significantly associated with change over time,  $\gamma_{26} = 0.03$  ( $SE = 0.04$ ),  $t(92) = 0.86$ , *ns* (effect size  $r = .09$ ).<sup>3</sup>

### Accounting for Course of Depressive Symptoms

We next conducted analyses to examine whether differential course of depressive symptoms accounted for these findings. For instance, if people who received cognitive therapy reported fewer depressive symptoms at the end of treatment or during follow-up, then this restricted range could explain why cognitive therapy

<sup>2</sup> As suggested by one of the anonymous reviewers, we also examined whether cognitive therapy was associated with a within-subjects interaction between depression and time. Results of analyses that included the interaction were consistent with previous analyses. Cognitive therapy was not significantly associated with the interaction between depression and time,  $\gamma_{36} = 0.01$  ( $SE = 0.00$ ),  $t(92) = 1.44$ , *ns* (effect size  $r = .15$ ). However, cognitive therapy remained a significant predictor of the association between negative cognition and depression,  $\gamma_{16} = -0.08$  ( $SE = 0.04$ ),  $t(92) = -2.38$ ,  $p < .05$  (effect size  $r = .24$ ).

<sup>3</sup> We also examined whether cognitive therapy influenced change in dysfunctional attitudes over the course of follow-up. In this hierarchical linear model, the DAS (Weissman, 1979) was the dependent variable. Month of assessment was the within-subjects independent variable. DAS assessments were completed at posttreatment, 6 months, and 12 months after treatment. After controlling for patient characteristics, cognitive therapy was not associated with posttreatment DAS. Furthermore, although there was an overall tendency for DAS total scores to decrease over time, it did not reach statistical significance ( $p = .12$ ). More important, cognitive therapy was not associated with change in DAS during follow-up. Consistent with our primary findings with our negative cognition scale, cognitive therapy did not influence post-treatment DAS or change in DAS over time. Unfortunately, because of the infrequent assessment schedule, we were unable to adequately test whether cognitive therapy unlinked DAS and symptoms of depression. Three assessments did not provide enough variability in depressive symptoms within each individual to conduct such analyses.

predicted a weaker association between negative cognition and depressive symptoms. To address this issue, we examined simultaneously whether treatment groups differed in depression severity at the end of treatment and whether groups had differential symptom change during follow-up. For this multilevel analysis, depression severity was the outcome variable, time (i.e., month of assessment) was the within-subjects variable, and treatment assignment (cognitive treatment vs. noncognitive treatment) was the between-subjects factor.

Results from this analysis revealed that, not surprisingly, post-treatment 14-item MHRSD was significantly different from 0,  $\gamma_{00} = 10.79$  ( $SE = 1.02$ ),  $t(97) = 10.51$ ,  $p < .05$  (effect size  $r = .73$ ). However, posttreatment depression was not significantly different for those who received cognitive therapy than for those who did not receive cognitive therapy,  $\gamma_{01} = -2.19$  ( $SE = 1.41$ ),  $t(97) = -1.55$ ,  $ns$  (effect size  $r = .14$ ). Furthermore, depression scores significantly decreased over time,  $\gamma_{10} = -0.22$  ( $SE = 0.07$ ),  $t(97) = -2.89$ ,  $p < .05$  (effect size  $r = .28$ ). However, change over time did not significantly differ between those who received cognitive therapy and those who did not,  $\gamma_{11} = 0.13$  ( $SE = 0.10$ ),  $t(97) = 1.25$ ,  $ns$  (effect size  $r = .13$ ).

Although groups did not differ in posttreatment depression or change in depression over time, this was not due to a lack of variability across individuals. Both the intercept (posttreatment depression),  $\mu_0 = 6.44$  (variance component = 41.59),  $\chi^2(96, N = 99) = 652.41$ ,  $p < .001$ , and the slope (change in depression across time),  $\mu_0 = 0.35$  (variance component = 0.12),  $\chi^2(96, N = 99) = 177.20$ ,  $p < .001$ , had significant variability across individuals, even after accounting for treatment assignment.

To further examine the influence of treatment response on our prior findings, we repeated analyses that examined the effect of treatment on the association between negative cognition and depression, this time selecting individuals who had remitted from depression at the end of acute treatment. For these analyses, we selected individuals who completed treatment with an MHRSD of  $< 7$  and a BDI of  $< 9$ . A total of 29 people met these criteria, but 1 person was dropped from analyses because all follow-up data were missing.

To conserve degrees of freedom, we only included patient characteristic covariates that were significant in the previous analyses. In this subsample, patient covariates were not significantly associated with variance in basal negative cognition or the association between negative cognition and depression ( $ts < 1$ ). An important finding, which was consistent with the findings from the full sample, was that cognitive therapy was associated with a significantly weaker association between negative cognition and depression compared with treatment that did not include cognitive therapy,  $\gamma_{14} = -0.08$  ( $SE = 0.04$ ),  $t(23) = -2.21$ ,  $p < .05$  (effect size  $r = .42$ ). Furthermore, cognitive therapy did not differentially influence basal negative cognition,  $\gamma_{02} = -0.14$  ( $SE = 0.14$ ),  $t(25) = 1.00$ ,  $ns$  (effect size  $r = .20$ ).

We also examined whether family treatment reduced basal negative cognition and the strength of association between negative cognition and depression among remitted depressed people. As before, patients who did not receive family therapy as part of their treatment were coded as 0, and those who did receive family therapy were coded as 1. Consistent with our previous results, family therapy did not explain variance in the relationship between negative cognition and depression,  $\gamma_{14} = -0.01$  ( $SE = 0.05$ ),

$t(23) < 1.00$ ,  $ns$ , or in basal negative cognition,  $\gamma_{02} = 0.13$  ( $SE = 0.17$ ),  $t(25) < 1.00$ ,  $ns$ .

## Discussion

In this study, we tested whether cognitive therapy specifically reduces the rate at which negative cognition increases as depression severity increases—one mechanism thought to underlie vulnerability to depression (Segal & Ingram, 1994). Participants were recruited for a study that examined the efficacy of combined pharmacological and psychosocial treatments (i.e., cognitive and family therapy) for the posthospital care of severe depression (Miller et al., in press). Following 6 months of outpatient treatment, negative cognition and noncognitive symptoms of depression were assessed monthly for 1 year. We then examined whether cognitive therapy reduced the rate at which negative cognition increases as a function of increasing depression severity.

Even after accounting for clinical characteristics such as episode duration, age at first episode onset, and patient age, and after controlling for change in negative cognition over time, the association between negative cognition and depression severity was weaker for people who received cognitive therapy than for those who did not receive cognitive therapy. That is, as symptoms of depression increased, negative cognition increased more slowly for people who received cognitive therapy. This was observed in the full sample, and the effect was even stronger for people whose depression had remitted at the end of treatment. This effect also appeared to be somewhat specific. That is, family therapy did not influence the association between negative cognition and depression. Furthermore, cognitive and family therapy did not influence basal negative cognition or the decrease in negative cognition over time. Thus, cognitive therapy appeared to specifically alter the rate at which negative thinking increased as symptoms of depression increased.

These findings suggest that cognitive therapy unlinked negative cognition (e.g., worthlessness, self-criticism, hopelessness) from other symptoms of depression. This is consistent with an account proposed by Teasdale et al. (2002). Specifically, they suggested that cognitive therapy may help patients relate more functionally to their negative thoughts. For instance, cognitive therapy may help patients treat negative thoughts as mental events that may or may not have elements of truth. Therefore, when depressive symptoms increase, a person with this mind-set is less likely to endorse negative thoughts, such as worthlessness, than a person who believes that such thoughts represent intrapersonal deficiencies.

Findings from the present work, as well as Teasdale et al.'s (2002) study, suggest that cognitive therapy may help people detach negative cognition from other symptoms of depression (such as dysphoric mood). Other treatments specifically designed to achieve this goal have been developed (Hayes, Strosahl, & Wilson, 1999; Segal, Williams, & Teasdale, 2002). We find it interesting that among people with three or more previous episodes of depression, the addition of mindfulness-based cognitive therapy (Segal et al., 2002) to treatment as usual reduces depressive relapse to a greater extent than treatment as usual alone (Teasdale et al., 2000). Whether treatments specifically designed to unlink negative cognition from depression mitigate cognitive reactivity and reduce relapse to a greater extent than cognitive therapy remains to be investigated.

Consistent with Young et al.'s (1996) study, results from the present study suggest that depression-related negative cognition has two separable and relatively distinct components. The first component is a basal level of negative cognition that a person experiences when not depressed. The second component is the rate at which negative cognition increases as a function of depression severity, starting from the basal level of negative cognition. Thus, a person may report differing levels of negative cognition at various levels of depression severity, which may explain earlier conclusions that negative cognition is an epiphenomenon of depression (Coyne & Gotlib, 1983). However, rather than an epiphenomenon, the present conceptualization suggests that the rate at which negative cognition increases as a function of depression severity differs among individuals and may be an important contributor to depression susceptibility.

This conceptualization complements other present models of cognitive vulnerability to depression. For instance, Zuroff, Blatt, Sanislow, Bondi, and Pilkonis (1999) proposed a state-trait model of cognitive vulnerability to depression. They suggested that dysfunctional attitudes consist of two components: an aspect that fluctuates with negative mood (and other state dependent influences) and a trait-like component that is consistent over time. Similarly, the mood-state hypothesis (Miranda & Persons, 1988; Segal & Ingram, 1994) suggests that dysfunctional cognition is most likely to be observed in the presence of depressed mood among people who are vulnerable to depression. The present study builds on this work by suggesting that cognitive therapy reduces negative thinking as symptoms of depression increase.

It is important to note that the present model does not make causal assumptions about the effect of negative cognition on depressive symptoms and vice versa, as assessments of negative cognition and depressive symptoms were obtained concurrently. In our model, we simply examine the covariation between repeated assessments of negative cognition and depressive symptoms. This conceptualization, however, parallels recent formulations of depression vulnerability. Specifically, in an update of his cognitive theory, Beck (1996) suggested that depressive symptoms are manifestations of underlying modes that are "a composite of cognitive, affective, motivational, and behavioral systems" (p. 19). Similarly, Teasdale and Barnard (1993) suggested that negative cognition and depressive symptoms can form self-perpetuating cycles, in which their mutual entrainment leads to persistent episodes of depression. Our focus on the covariation between symptoms of depression and negative cognition is consistent with these more recent conceptualizations.

In the present study, we examined associations between negative cognition and naturally occurring symptoms of depression. Such an approach may have greater ecological validity than laboratory-based dysphoric mood inductions (e.g., Beevers & Carver, 2003). Repeated measurement over time can also increase reliability of assessments and reduce recall bias (Affleck, Zautra, Tennen, & Armeli, 1999). However, collecting intensive assessments of naturally occurring depressive symptoms sacrifices an element of control that can only be attained in the laboratory. Despite this lack of experimental control, we are encouraged that our attempt to capture life as it is lived (Bolger, Davis, & Rafaeli, 2003) appears to converge with findings derived from well-controlled laboratory work (Segal et al., 1999; Teasdale et al., 2002).

The present study has several limitations. First, we used a generic and idiosyncratic assessment of negative cognition. This measure did not assess a full range of negative cognition often associated with depression. Although our negative cognition scale was positively associated with more traditional self-report measurements of negative cognition, future work would benefit from researchers using a more theoretically derived assessment of cognition, such as the Automatic Thoughts Questionnaire (Hollon & Kendall, 1980). However, briefer versions of existing questionnaires may need to be developed if repeated measurements with relatively short intervals between assessments are to be used to minimize participant burden (Affleck et al., 1999).

A second limitation is that the treatment matching algorithm did not allow for direct comparisons between specific treatment modalities (e.g., cognitive therapy vs. family therapy), as patient characteristics (cognitive bias and family functioning) were used to assign people to specific treatment combinations (see Table 1). However, the treatment matching design did allow for comparisons between groups who received cognitive therapy as part of a treatment package and those who did not receive cognitive therapy. Related to this issue, analyses could not determine whether the unlinking of negative cognition and depression mediated the effect of cognitive treatment. Additional work that directly compares the effect of cognitive and family treatment on negative cognition and depression, and which also identifies whether change in this relationship mediates the effect of cognitive treatment, is clearly needed.

Another study limitation involves treatment during the follow-up period. Ideally, all participants would have either received no treatment or the same treatment during follow-up. Instead, follow-up treatment was not strictly controlled, with most patients receiving treatment from study or community providers. Despite this limitation, it is notable that random assignment to cognitive therapy during the acute treatment phase significantly weakened the association between negative cognition and depression during follow-up—a time when treatment was not closely controlled.

Additional research is needed to test the generalizability of these findings. Participants in the present study were recruited during hospitalization for severe depression. Because depression severity warranted hospitalization, participants received relatively intensive hospital and outpatient treatment. Whether these findings apply to less severely depressed populations, such as depressed outpatients, who often receive less intensive treatment, is unclear. However, it should also be noted that severely depressed people are often excluded from treatment studies of depression, often because of high levels of suicidality (Hollon et al., 2002). Therefore, although the generalizability of the findings may be questioned, this study does provide important information about a subgroup of depressed patients who are generally understudied and often systematically excluded from depression research.

Related to this issue, remission rates at the end of treatment were notably low. At the end of 6 months of intensive treatment, only 29 of the 121 initially recruited completed treatment with a BDI of  $<9$  and an MHRSD of  $<7$ . Although an in-depth discussion of these findings is beyond the scope of the present article, we briefly speculate why rates of remission were low. First, cognitive therapy was not initiated until after hospital discharge. In our previous work (Miller, Norman, & Keitner, 1989), patients received an

average of 10 inpatient cognitive therapy sessions. It is possible that beginning cognitive therapy while in the hospital is more beneficial than delaying initiation until discharge. Second, despite the fact that our protocol specified 20–24 sessions of cognitive therapy, the mean number of cognitive therapy sessions received was substantially fewer ( $M = 13$ ,  $SD = 6$ ). Poor patient compliance was the major reason for this reduced number of sessions. Finally, although all therapists were experienced, well qualified, and closely supervised, we have not yet conducted treatment adherence or competence ratings. Such analyses may help to further explain these low rates of remission.

Despite these limitations, the present work makes an important contribution to understanding whether cognitive therapy alters the association between negative cognition and depression. Findings suggest that cognitive therapy, but not family therapy, unlinks negative thoughts with symptoms of depression following treatment. Given that cognitive therapy consists of multiple components (Beck et al., 1979), additional research is needed to identify which techniques specifically unlink negative cognition and symptoms of depression. Research that translates basic research into clinical practice may be particularly helpful in identifying innovative techniques that modify this relationship (Hollon et al., 2002). Such research may help to improve existing treatments and further reduce the high rates of relapse associated with unipolar depression.

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