

The Effects of Treatments for Depression On Perceived Failure in Self-Regulation

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Two studies examined the effect of treatments for depression on perceived failure in self-regulation, operationalized as within-self discrepancy. In Study 1, patients received group cognitive-behavioral therapy (CBT); in Study 2, patients received either individual CBT, interpersonal psychotherapy (IPT), or medication. Treatments showed equivalent efficacy, but only psychotherapy was associated with decreased self-discrepancy and priming reactivity. Highly self-discrepant patients showed less improvement than other patients in all treatments, even after controlling for initial severity. The findings suggest that treatments differ in their impact on self-regulatory cognition, and that highly self-discrepant patients may require longer or alternative treatment.

KEY WORDS: depression; cognitive-behavioral therapy; interpersonal psychotherapy; self-regulation; self-discrepancy; antidepressant medication.

Depression is associated with a number of hypothetical contributory factors (Kendler, Kessler, Neale, Heath, & Eaves, 1993), and treatments for depression target particular hypothetical causes of dysfunction (Kupfer, 1993). The challenge for clinical science is to determine the common and specific changes induced by different treatments and the mechanisms responsible for such changes, in order to match patients with the most appropriate treatments (Beutler, 1991).

Depression is a recurrent disorder, and symptom management alone does not prevent relapse and recurrence (Hollon, DeRubeis, & Seligman, 1992). To provide treatments that both alleviate symptoms and reduce vulnerability to subsequent episodes, the impact of treatments on factors that cause and maintain depression must be determined for each individual patient (Simons, Gordon, Monroe, & Thase, 1995; Teasdale, Segal, & Williams, 1995).

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This article reports two studies examining the impact of existing treatments for depression on self-regulatory cognition, a hypothetical contributory factor for the onset and maintenance of depressive episodes. Specifically, we sought to determine whether current treatment modalities altered two different manifestations of *perceived failure in self-regulation*, viewed from the perspective of self-discrepancy theory (Higgins, 1987). In addition, because several studies of treatment-related cognitive change observed that patients with high levels of depressogenic cognition may not respond optimally to treatment, we also examined how depressed patients with high levels of perceived failure in self-regulation fared in different treatments.

Self-regulation can be defined as the process of evaluating one's behaviors and attributes in reference to important goals or standards, and modifying those behaviors and attributes if needed (Carver & Scheier, 1990). Self-regulation is an ongoing process that occurs both consciously and unconsciously, involving a variety of goals (e.g., impressing someone, succeeding at a task, avoiding mistakes) and standards (e.g., how I compare with peers, whether I am being the kind of person my spouse/parents/children would like). As a cognitive process, self-regulation is linked with the brain/behavior systems mediating approach and avoidance and their associated motivational and affective states (Cicchetti & Tucker, 1994; Depue & Collins, 1999).

Whenever an individual's explicit or implicit efforts to meet a goal or standard are perceived as unsuccessful, a negative affective state results (Duval & Wicklund, 1972). If a person perceives their efforts at self-regulation as chronically unsuccessful, chronic negative affect will result, decreasing the likelihood of successfully meeting those goals or standards. We and others have hypothesized that chronic perceived failure in self-regulation is a contributory factor for depression in certain individuals (Carver & Scheier, 1990; Strauman, 1996a, 1999).

Self-discrepancy theory (SDT; Higgins, 1987) is a model of self-regulation and affect which focuses on two important types of goals or desired outcomes (referred to as *self-guides*) associated with different emotional states. *Ideal* self-guides are the individual's hopes and aspirations. According to SDT, ideal standards are associated with positive outcomes such as happiness and joy when they are achieved and negative outcomes such as sadness and disappointment when they are not achieved. That is, a perceived actual-self:ideal-self discrepancy (AI) represents a *failure to attain a personally significant positive outcome*, and hence, a failure of self-regulation. In contrast, *ought* self-guides are the individual's responsibilities and obligations. Ought standards are associated with negative outcomes such as fear, anxiety, and worry when they are not achieved and positive outcomes such as calmness and security when they are achieved. A perceived actual-self:ought-self discrepancy (AO) represents a *failure to avoid a personally significant negative outcome*, and hence, a different kind of failure in self-regulation. AI and AO discrepancy are discriminantly associated with depressive versus anxious symptoms (Scott & O'Hara, 1993).

Consistent with a hypothetical contributory role in depression, perceived failure in self-regulation in the form of self-discrepancy is stable over time (Strauman, 1996b). In a recent longitudinal study (Strauman, under review), AI and AO discrepancy were discriminantly stable across a 2-year period even when the other discrepancy was partialled out (zero-order cross-time correlations, $r = .46$ and $.40$ respectively; discriminant cross-time correlations, $pr = .32$ and $.30$ respectively), as

were affective reactions to priming self-discrepancies. Moreover, people who perceived themselves to be failing chronically in their pursuit of ideal goals (i.e., chronic AI discrepancy) manifested significant increases in depressive symptoms across the study period.

Cognitive-behavioral therapy (CBT), interpersonal psychotherapy (IPT), and antidepressant medications all are effective treatments for depression (Antonuccio, Danton, & DeNelsky, 1995; Persons, Thase, & Crits-Christoph, 1996). Nonetheless, it is unclear whether treatments differ in their effects on cognitive processes associated with depression. Findings across studies have been inconsistent, and investigators using different definitions of cognitive change have obtained different results (Whisman, 1993). Moreover, patients manifesting high levels of depressogenic cognitive diatheses often respond more poorly to treatment than other patients (e.g., Blatt, 1995).

In recent reviews, Persons and Miranda (1995) and Segal and Ingram (1994) argued that the failure to *activate* depressogenic cognitions during assessment hampers testing of hypotheses about cognitive change and that such failure could account for the inconsistent findings in the cognitive change literature. Both reviews recommended the use of cognitive “priming” techniques to obtain more reliable discrimination of symptomatic improvement from change in underlying cognitive processes. In the studies reported below, self-report (interview responses) and priming measures of self-regulatory cognition were used—methods validated in clinical samples (Strauman, 1989, 1992).

Brewin (1989) proposed that cognitive change in psychotherapy can occur on one or both of two levels: conscious (changes in expressed beliefs, values, and attitudes, all of which patients can describe), and unconscious (changes in underlying representations or pathways of information processing, which patients cannot describe). Accordingly, we examined the effects of different treatments on two different manifestations of self-regulatory cognition: *magnitude of self-discrepancy*, i.e., the patient’s consciously accessible perceptions of consistency or inconsistency between her/his actual self-attributes and her/his self-guides, and *affective responses to priming of self-guides*, i.e., momentary affective reactions to cues which activate self-discrepancies.

STUDY 1: EFFECTS OF GROUP COGNITIVE-BEHAVIORAL THERAPY FOR DEPRESSION ON PERCEIVED FAILURE IN SELF-REGULATION

This study considered whether group CBT for depression reduced self-discrepancy or reactivity to priming of ideal self-guides or both. Patients participated in a self-belief interview and a cognitive priming task before and after a course of group CBT. We addressed the following questions: (1) Did CBT lead to reduction in self-discrepancy (conscious self-regulatory cognition)? (2) Did CBT change patients’ responses to priming of their ideal self-guides (unconscious self-regulatory cognition)? (3) Were either baseline level or degree of change of perceived failure in self-regulation correlated with clinical improvement?

Method

Overview

Patients meeting criteria for major depressive episode who were not currently receiving any other treatment participated in 12 weeks of group CBT. Measures of depressive symptoms and self-regulatory cognition were obtained at pre- and posttreatment.

Participants

Participants were outpatients presenting to the Psychology Research and Training Clinic at the University of Wisconsin—Madison for evaluation and treatment in response to newspaper advertisements offering low-cost group therapy. Applicants first were assessed by two doctoral students in clinical psychology under the supervision of a licensed PhD psychologist. The initial screening included the Structured Clinical Interview for *DSM-III-R*, Axis I (Spitzer, Williams, Gibon, & First, 1992) with reference to *DSM-III-R* (*Diagnostic and Statistical Manual of Mental Disorders* [3rd ed. Rev.], American Psychiatric Association, 1987) criteria for major depressive disorder (nonpsychotic, nonbipolar), along with exclusionary criteria (see below). Subsequently, eligible patients were referred to two of the coauthors (ND and VS) for a second assessment that included the 17-item version of the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960).

Patients were considered eligible if they met *DSM-III-R* criteria for major depressive disorder at both assessments (approximately 10 days apart); did not suffer from untreated or poorly controlled medical conditions that may cause depressive symptoms (e.g., hypothyroidism); were not taking antidepressant or anxiolytic medication at the time of the study; and had no history of psychotic symptoms, antisocial personality disorder, or alcohol/drug abuse or dependence (as determined by clinical interview). A total of 78 applicants were screened; 45 were found to meet eligibility criteria, 36 began treatment, and 32 completed the first four sessions. An additional three dropped out between the fourth and the seventh sessions; the remaining 29 individuals (18 female) completed courses of treatment to a minimum 80% attendance criterion. Dropouts did not show statistically significant differences from completers on any of the pretreatment measures with the exception of number of previous episodes (mean = 3.2 for dropouts vs. 1.8 for completers, $p < .05$).

Treatment

Participants providing informed consent received a 12-session course of group cognitive-behavioral therapy consisting of 90-min sessions once per week. Therapy was conducted by pairs of therapists from a pool of four doctoral candidates in clinical psychology at the Psychology Research and Training Clinic of the University of Wisconsin—Madison. Four groups were conducted, with an average of 7.25 patients completing each group. Therapists were blind to study hypotheses and did not have access to data from the assessments of self-regulatory cognition.

The groups were conducted using the Beck, Rush, Shaw, and Emery (1979) manual and the modifications for group-based treatment contained therein (Beck et al., 1979, chap. 16, "Group Cognitive Therapy for Depressed Patients"), with the first author serving as therapy supervisor via weekly group supervision sessions. Videotapes of four randomly-selected sessions from each group were assessed using the Collaborative Study Psychotherapy Rating Scale (Evans, Piasecki, Kriss, & Hollon, 1984) to ensure adherence and to evaluate the competence of each therapist. All sessions scored above the minimum overall criterion for CBT adherence adopted in the NIMH Treatment of Depression Collaborative Research Project (Shaw, 1984).

Measures

Each patient was scheduled for two pretreatment research sessions conducted a minimum of 1 week apart. In the first (held prior to the start of the treatment group), a semistructured interview was conducted in which self-beliefs and self-discrepancies were assessed. In the second (held during the week between the first and second group therapy sessions), the self-guide priming task was conducted. All of the measures were administered again upon completion of treatment.

Symptom Measures. The 17-item HRSD (Hamilton, 1960) and the 21-item Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) were used to assess depressive symptoms. The HRSD was completed by a clinical evaluator at intake; the BDI was completed by the patient at intake and at each treatment session. Each patient met again with a clinical evaluator one week after the final group session, whereupon the HRSD was readministered and the patient completed the final BDI. Overall interrater reliability for the HRSD (intraclass correlation, using the 2/1 formula provided by Shrout & Fleiss, 1979) as determined by comparing the scores of two independent raters on 10 of the patients' initial interviews was .84.

Self-Discrepancy. An interview version of the Selves Questionnaire (Higgins, Bond, Klein, & Strauman, 1986), a free-response measure that asks participants to describe three domains of self-beliefs (actual, ideal, and ought), was administered individually during the first of the two pretreatment and posttreatment research sessions. The interview consisted of a series of questions, each pertaining to a domain of self/standpoint on the self combination (e.g., the question "What are the attributes of the type of person *you* believe you *actually are*?" pertains to the patient's actual/own self-beliefs). The interview included both the participant's own standpoint (i.e., her/his own beliefs) and the participant's beliefs concerning the standpoint of her/his parents (living or deceased).

Two raters blind to study hypotheses independently classified each attribute in each participant's ideal/own, ideal/other, ought/own, and ought/other self-states according to its relation to the attributes in the actual/own self-state. Self-guide attributes could be classified as *matches* (synonymous with an actual/own attribute), *mismatches* (antonymous with an actual/own attribute), or *nonmatches* (neither synonymous nor antonymous with any actual/own attribute). For the ideal/own, ideal/other, ought/own, and ought/other self-states, self-discrepancy was quantified as the number of mismatches, weighted by a factor of 2, minus the number of matches

(Higgins et al., 1986).³ Actual/own:ideal/own and actual/own:ideal/other discrepancy were averaged to obtain an overall actual:ideal (AI) discrepancy score; similarly, an overall actual:ought (AO) discrepancy score was obtained. Scores could range from 20 (maximum self-discrepancy) to -10 (maximum self-congruency). Overall interrater reliability (intraclass correlation) was .89.

Self-Guide Priming Procedure. We assessed participants' responses to priming of self-standards using an autobiographical memory paradigm (Strauman, 1992) conducted during the second pretreatment and posttreatment research sessions. Participants were presented with a series of words and were instructed to use each word to verbalize the first childhood memory that comes to mind. Unknown to the participant, a set of cues was prepared based in part on her/his responses to the Selves Interview. For this study, two types of cues were included within each participant's cue list. *Self-guide* cues were selected from among the ideal attributes (a total of six selected from across the participant's ideal/own and ideal/other interview responses) that the participant had generated during the pretreatment Selves Interview. These attributes were chosen so as to be unique to the ideal domain and to include as many self-discrepant attributes as possible. *Yoked-control* cues were selected from the ideal self-guide attributes of other participants (and so were positively valenced as well). These cues were not semantically related to any attribute generated by the participant in the pretreatment Selves Interview. Each set of control cues was taken from the ideal self-guide cues of approximately three other participants. The control cues provided a test of the alternative hypothesis that the literal content of cues accounted for their impact following priming.

The participant was told that she/he would be presented with words to help them remember experiences from before junior high school. For each word, she/he was to describe out loud the first childhood memory that came to mind, in enough detail that the experimenter could understand the gist of the experience. They had a maximum of 30 s to retrieve a memory, after which the trial was considered a memory failure and they were given the next cue. Two practice trials using the cues *pleasant* and *content* were conducted; then the experimenter informed the participant that the experimental trials would begin. Cues were presented in random order. Memories of recent events were not scored as memory failures, but participants were reminded to report memories from before junior high school. No participant had more than six responses that were not sufficiently remote in time (average = 1.2). We did not focus on memory failures in the present study; however, they occurred at approximately the same incidence (5.5% for self-guide cues and 11% for control cues) as in previous studies using this childhood memory priming task. At the conclusion of the second posttreatment research session, each participant was asked whether she/he had any guesses about the hypotheses being tested, received debriefings, and were paid for participating. No participant identified their self-guide cues or identified the purpose of the memory paradigm.

Two measures were obtained from the priming task. The first, *time to retrieval*, concerned the efficiency with which cues promoted memory retrieval (an index of

³The weights for calculating discrepancy scores are based on the scoring algorithm of Higgins et al. (1986), which were obtained from unstandardized regression coefficients derived from analyses of analog and clinical samples.

Table I. Demographic Characteristics and Pretreatment Symptom Severity and Self-Discrepancy Scores for Depressed Patients in Study 1

Patient characteristic	Mean score
Gender (# female)	21
Age	38.1 (8.7)
Number of previous episodes	1.8 (0.8)
Duration of current episode (months)	9.9 (6.1)
Number definite/probable endogenous	16
HRSD	18.8 (5.6)
BDI	22.9 (6.8)
Actual:ideal self-discrepancy	3.8 (2.2)
Actual:ought self-discrepancy	0.9 (3.1)

Note. $N = 29$. Standard deviations are in parentheses. HRSD: Hamilton Rating Scale for Depression; BDI: Beck Depression Inventory.

self-guide accessibility). Time to retrieval was scored as elapsed time from cue presentation to the start of the memory verbalization (disregarding irrelevant utterances). Interrater reliability for this measure, computed by comparing data from two independent raters, was .95 (intraclass correlation). The second dependent measure was the *affective content* of the memories.⁴ Memories were content analyzed for evidence of dysphoric affect using the depressive content scale of Gottschalk and Hoigaard-Martin (1986) and for evidence of anxious affect using the anxiety content scale of Gottschalk and Gleser (1969). Two raters independently scored the presence or absence of affective content in each memory on a phrase-by-phrase basis. Using the two scales (designed for joint use), each phrase was coded as *dysphoric*, *anxious*, or *none/other*. Overall interrater reliability (intraclass correlation) for the affect coding, based on two independent raters, was .89.

Results

Patient Characteristics

Table I lists demographics as well as pretreatment symptom severity and self-discrepancy for the 29 patients who completed the protocol (16 of the 29, or 55%, were characterized as definite or probable endogenous subtype by Research Diagnostic Criteria; Spitzer, Endicott, & Robins, 1978). The sample distributions of AI and AO discrepancy were consistent with those reported in previous studies of clinically diagnosed samples (e.g., Scott & O'Hara, 1993; Strauman, 1989). As predicted, patients manifested higher levels of AI (mean = 3.8) than AO (mean = 0.9) discrepancy. Analysis of variance (ANOVA) with Type of Discrepancy as a within-subject factor revealed a significant difference in magnitude of self-discrepancy, $F(1, 27) = 5.52$, $p < .05$. Twelve of the 29 patients (41%) manifested at least three instances of

⁴Only negative affective content of the memories was examined, because of the nature of self-guides as (almost exclusively) positively valenced attributes. Positive attributes would tend to lead to retrieval of negatively valenced memories *only* if such memories were retrieved based on the affective significance of the cue rather than its literal valence (Strauman, 1992, 1996a, 1996b).

actual–self:ideal–self mismatch on the Selves Interview, a magnitude of discrepancy associated with risk for depression (Strauman, 2000). Self-discrepancy was modestly correlated with pretreatment BDI and HRSD scores: for AI, $r(28) = .24$ and $.21$ respectively; for AO, $r(28) = .20$ and $.12$ respectively.

Symptomatic Improvement

Completers on average showed clinically significant improvement in symptoms. A multivariate repeated-measures ANOVA (Huberty & Morris, 1989) examining change on the two symptom measures indicated a significant effect of treatment, $F(1, 28) = 10.48$, $p < .001$. The mean HRSD score fell from 18.8 ($SD = 5.6$) at pretreatment to 9.07 ($SD = 5.8$) at termination, $F(1, 27) = 11.52$, $p < .001$, and the BDI from 22.9 ($SD = 6.8$) to 9.8 ($SD = 6.6$), $F(1, 27) = 12.98$, $p < .001$. The posttreatment mean BDI and HRSD scores were below the range of scores for clinically depressed samples but above the normative range for nondepressed adults. Using the criteria for clinically significant change specified by Ogles, Sawyer, and Lambert (1995, p. 323), 19 of the 29 patients (66%) manifested a clinically significant reduction of depressive symptoms on the BDI and 18 (62%) manifested clinically significant improvement on the HRSD.

Change in Magnitude of Self-Discrepancy

How did group CBT affect patients' type and magnitude of self-discrepancy? A repeated-measures ANOVA was conducted with Time (pretreatment, posttreatment) and Type of Discrepancy (AI, AO) as within-subject factors. A main effect for Time was observed, $F(1, 25) = 5.52$, $p < .01$, in which posttreatment self-discrepancy was significantly less than pretreatment self-discrepancy, as well as a main effect for Type of Discrepancy, $F(1, 25) = 6.02$, $p < .01$, in which magnitude of AI discrepancy was significantly greater overall than magnitude of AO discrepancy. More importantly, we observed a significant Time \times Type of Discrepancy interaction, $F(1, 25) = 4.87$, $p < .05$. There was a significant decrease in AI discrepancy, from a mean of 3.8 ($SD = 2.2$) pretreatment to 0.8 ($SD = 2.8$) posttreatment, $F(1, 25) = 4.92$, $p < .05$ (effect size as $R^2 = .18$), but a nonsignificant decrease in AO discrepancy, from 0.9 ($SD = 3.1$) to -0.6 ($SD = 2.7$), $F(1, 25) = 1.47$ (effect size as $R^2 = .035$).

Change in Response to Priming

How did group CBT affect patients' reactions to priming with their ideal self-guides? A repeated-measures ANOVA was conducted with time to retrieval as the dependent measure, using Time (pretreatment, posttreatment) and Type of Priming (self-guide, yoked-control) as within-subject factors. A significant Type of Priming effect was observed, $F(1, 25) = 7.89$, $p < .01$; across both time points, patients showed significantly faster retrieval following self-guide cues (overall mean = 9.56 s) than following yoked-control cues (overall mean = 13.48 s). Neither the Time effect nor the Time \times Type of Priming interaction even approached statistical significance.

Therefore, memories cued by self-guides were more highly accessible than memories cued by yoked control primes both before and after group CBT.

Taking dysphoric memory content as the dependent measure, a repeated-measures ANOVA was conducted including Time (pretreatment, posttreatment) and Type of Priming (self-guide, yoked-control) as within-subject factors. Neither the main effect for Time nor the main effect for Type of Priming approached statistical significance. However, a significant Time \times Type of Priming interaction was observed, $F(1, 25) = 5.52, p < .01$. There was a significant decrease in dysphoric memory content following self-guide cues (mean incidence 0.8 per verbalized memory at pretreatment vs. 0.25 per verbalized memory at posttreatment), $F(1, 25) = 4.48, p < .05$ (effect size as $R^2 = .16$), but only trivial change in dysphoric memory content in response to yoked-control cues (from 0.6 to 0.5, effect size as $R^2 = .01$). Overall, patients manifested decreased affective responses to priming with self-guides following a course of group CBT.

Correlation Between Improvement and Cognitive Change

Was there an association between clinical improvement and change in self-discrepancy or priming responses or both? To answer this question, a hierarchical regression analysis was conducted to determine whether symptom reduction and change in AI discrepancy were correlated using posttreatment BDI as the criterion variable. In the first step, pretreatment BDI and AI self-discrepancy were entered; this step accounted for a nonsignificant portion of variance ($R^2 = .038$). In the second step, posttreatment AI was added but accounted for a nonsignificant increment in variance of posttreatment BDI (increment in $R^2 = .05$).

A second hierarchical regression analysis was conducted to determine whether symptom reduction and change in reactivity to priming with self-guides were correlated; posttreatment BDI again was the criterion variable. In the first step, pretreatment BDI and dysphoric content following self-guide priming were entered; this step accounted for a nonsignificant portion of variance ($R^2 = .025$). In the second step, posttreatment dysphoric content following self-guide priming accounted for a nonsignificant increment in variance of posttreatment BDI (increment in $R^2 = .03$). Thus, clinical improvement and change in self-regulatory cognition were not significantly correlated.

Correlation Between Changes in Self-Discrepancy and Response to Priming

Although cognitive change was not associated with clinical improvement, were the two measures of cognitive change associated? A hierarchical regression analysis was conducted in which posttreatment dysphoric content following self-guide priming was the criterion variable. Pretreatment AI discrepancy and dysphoric content following self-guide priming were entered first, accounting for a nonsignificant portion of variance ($R^2 = .045$). In the second step, posttreatment AI discrepancy accounted for a significant increment in variance in posttreatment dysphoric memory content, $F(1, 25) = 6.92$, increment in $R^2 = .10, p < .01, \beta = .39$. Thus, there was evidence that conscious and unconscious cognitive change via group CBT were significantly correlated.

Pretreatment Perceived Failure in Self-Regulation and Clinical Improvement

Did patients with high levels of self-discrepancy, or strong affective responses to priming of self-guides or both, respond as well to group CBT as other patients? To examine this question, patients were divided into two groups, high-discrepant ($n = 12$) and low-discrepant ($n = 17$), based on whether they manifested at least three mismatches between actual-self and ideal-self attributes (Strauman, 2000). A repeated-measures MANOVA was conducted with BDI and HRSD as criterion variables and Time (pretreatment, posttreatment) and Level of Discrepancy as within-subject factors. The main effect for Time was significant (reflecting overall improvement), $F(1, 25) = 10.48, p < .001$ (effect size as $R^2 = .39$). More importantly, we observed a significant Time \times Level of Discrepancy interaction, $F(1, 25) = 4.87, p < .05$ (effect size as $R^2 = .09$), in which high-discrepant patients had significantly higher posttreatment BDI and HRSD scores (means = 13.1 and 11.5 respectively) than low-discrepant patients (mean = 8.2 and 7.5 respectively) but no difference in pretreatment BDI and HRSD scores (23.8 vs. 22.1 on the BDI and 18.4 vs. 19.0 for the HRSD). The difference in posttreatment BDI and HRSD scores remained statistically significant after controlling for age, gender, endogenous/nonendogenous subtype, and number of previous episodes (with all covariates failing to account for significant variance in posttreatment BDI scores in subsequent analyses).

Overall, Study 1 yielded the following results: (1) As predicted, the sample of depressed patients manifested higher actual:ideal (AI) discrepancy than actual:ought (AO) discrepancy. (2) Completers on average showed a clinically significant degree of symptomatic improvement at the end of 12 weeks of group CBT. (3) The course of group CBT was associated on average with a significant reduction in AI discrepancy, but not AO discrepancy. (4) The treatment was associated on average with a significant reduction in dysphoric memory content following priming with self-guides. (5) Change in self-discrepancy and change in priming reactivity were correlated, but neither was associated significantly with clinical improvement. (6) Highly self-discrepant patients showed less improvement than other patients, even though the two groups did not differ in pretreatment symptom severity.

STUDY 2: THE IMPACT OF INDIVIDUAL PSYCHOTHERAPY VERSUS MEDICATION FOR DEPRESSION ON PERCEIVED FAILURE IN SELF-REGULATION

Study 1 indicated that group CBT for depression was associated with change in self-regulatory cognition but that highly self-discrepant patients were less improved following 12 sessions than low-self-discrepant patients. In Study 2 we sought to determine whether changes in self-discrepancy and priming reactivity would be associated with specific treatments or with recovery per se, as well as whether highly-self-discrepant patients would be less responsive than low-self-discrepant patients to treatments other than group CBT. Study 2 compared three standard treatments: two types of individual psychotherapy and antidepressant medication.

Method

Overview

Patients meeting criteria for major depressive episode participated in one of three treatments over a 4-month course : individual CBT (Beck et al., 1979), interpersonal psychotherapy (IPT; Klerman, Weissman, Rounsaville, & Chevron, 1984), or medication (sertraline or fluoxetine). Symptoms and self-regulatory cognition were assessed before and after the course of treatment.

Participants

Participants were outpatients presenting to the Depression Treatment Program at the University of Wisconsin, Department of Psychiatry, for treatment. Patients were assessed by a psychology intern or psychiatry resident and a faculty member (psychologist or psychiatrist). The intake included the Structured Clinical Interview for *DSM-IV* Axis I Disorders (First, Spitzer, Gibbon, & Williams, 1995). Patients meeting criteria for major depressive disorder were referred for additional assessment. The screenings were conducted at least 7 days apart; patients were not accepted unless they met criteria for depression at both times.

Patients were eligible if they met *DSM-IV* criteria for a diagnosis of major depressive disorder; did not suffer from poorly controlled medical conditions that may cause depressive symptoms; were not taking antidepressant or anxiolytic medication; and had no history of psychotic symptoms, antisocial personality disorder, or alcohol/drug abuse or dependence (assessed via clinical interview). A total of 94 outpatients were screened; 52 met entry criteria, 41 began treatment, and 39 completed the first three sessions (one dropped from IPT and one from medication). An additional four (2 CBT, 1 IPT, 1 medication) dropped out between the 4th and 6th sessions; the remaining 35 individuals (23 female) completed treatment as specified below. The dropouts did not show statistically significant differences from completers on any pretreatment measures.

Treatment

Participants were given the choice of medication or psychotherapy.⁵ Patients choosing medication (completers = 13) were assigned to a faculty psychiatrist/psychiatry resident pair, who prescribed either sertraline or fluoxetine. To be classified as a medication completer, patients had to meet with the faculty member at least twice during the first 4 weeks of treatment, and then at least every 4 weeks for the remainder of the 4-month study period; reach a dose level deemed adequate by the faculty psychiatrist by the 8th week of treatment and maintain that dose level for

⁵It was not possible to randomly assign patients to treatment condition because of ethical concerns raised by the local Institutional Review Board. Specifically, some members of the IRB (not mental health professionals) argued that medication was the treatment of choice for depression and therefore participants should have the option of receiving medication. Despite the lack of data supporting this argument, the IRB recommended against random assignment. Interestingly, however, the majority of participants chose psychotherapy.

the subsequent 2 months; and refrain from taking any other psychiatric medication and from receiving psychotherapy or counseling during the study period. Patients receiving sertraline ($n = 7$) maintained final dose levels of either 50 or 100 mg/day; completers receiving fluoxetine ($n = 6$) maintained final dose levels of either 20 or 40 mg/day. A completed course was declared once the patient had been taking antidepressant medication for 16 weeks.

Patients choosing psychotherapy were assigned to a predoctoral intern or postdoctoral fellow in clinical psychology (from a total available pool of five therapists), who chose either CBT (completers = 16) or IPT (completers = 15) based on their initial assessment of the patient's presenting problems. Therapists did not have access to data from assessments of self-regulatory cognition. All of the therapists completed at least two CBT and two IPT treatments within the study. To be classified as a psychotherapy completer, patients had to meet with their therapist weekly during the first 8 weeks of treatment, and then at least four more times over the next 2 months; and refrain from taking any psychiatric medication or receiving any other counseling or psychotherapy during the study period. A completed course of treatment was declared either upon the completion of the 16th session or after a minimum of 12 sessions and agreement between patient and therapist that the depression had been in remission for at least four weeks and the therapy was completed. Thirteen of the 16 CBT patients and 11 of the 15 IPT patients completed courses of treatment within the 4-month period; the others participated in the posttreatment data collection after 16 weeks of treatment and then completed their courses of treatment within the 4-week period that followed.

CBT was provided following the Beck et al. (1979) manual; IPT was provided according to the Klerman et al. (1984) manual. All study therapists had previous training and research-protocol experience with both modalities. Weekly supervision meetings were held in which active cases were presented, directed by the first two authors (TJS and GGK). Ratings of audiotapes from four randomly selected sessions using the Collaborative Study Psychotherapy Rating Scale—Version 6 (Evans et al., 1984) were performed by a different therapist. Ninety-two percent of the sessions scored demonstrated acceptable adherence and competence using the criteria from Study 1.

Measures and Procedures

The identical measures and procedures from Study 1 were used. Interrater reliability (intraclass correlations) for scoring of the Selves Interview, the HRSD, and the affect coding from patients' responses on the memory task were .87, .82, and .85 respectively.

Results

Patient Characteristics

Table II lists demographic variables, pretreatment symptom severity, and pretreatment self-discrepancy for the 44 completers (24 of whom were characterized as definite or probable RDC endogenous subtype). The only significant difference

Table II. Demographic Characteristics and Pretreatment Symptom Severity and Self-Discrepancy Scores for Depressed Patients by Treatment Condition in Study 2

Patient characteristic	Medication (<i>N</i> = 13)	CBT (<i>N</i> = 16)	IPT (<i>N</i> = 15)
Gender (# female)	7	9	9
Age	37.1 (8.2)	41.2 (7.6)	43.9 (9.1)
Number of previous episodes	2.2 (0.6)	1.7 (1.0)	2.0 (1.3)
Duration of episode (months)	6.4 (7.9)	7.2 (7.7)	7.8 (6.9)
Definite/probable endogenous	7	8	9
HRSD	20.4 (3.5)	21.9 (5.2)	22.0 (6.6)
BDI	27.1 (7.3)	25.1 (7.5)	27.0 (8.0)
Actual:ideal self-discrepancy	4.2 (2.6)	4.5 (2.9)	4.1 (3.1)
Actual:ought self-discrepancy	1.4 (3.1)	1.1 (2.6)	1.6 (3.2)

Note. Standard deviations are in parentheses. HRSD: Hamilton Rating Scale for Depression; BDI: Beck Depression Inventory.

observed among the groups was in age, $F(2, 41) = 4.87, p < .05$; patients in the two psychotherapy conditions combined (mean age = 42.5 year) were significantly older than the patients in the medication condition (mean age = 37.1 year).⁶

Patients again manifested greater AI discrepancy than AO discrepancy. An ANOVA with self-discrepancy as the dependent variable and using Treatment Condition (medication, CBT, IPT) as a between-subjects factor and Type of Discrepancy as a within-subject factor revealed a significant effect for Type of Discrepancy, $F(1, 42) = 7.01, p < .01$. Patients manifested higher levels of AI (mean = 4.3) than AO (mean = 1.3) discrepancy. Twenty of the 44 patients (45%) manifested at least three instances of actual–self:ideal–self mismatch on the Selves Interview. Self-discrepancy was modestly correlated with pretreatment BDI and HRSD scores: for AI, $r(42) = .29$ and $.28$ respectively; for AO, $r(42) = .20$ and $.26$ respectively.

Symptomatic Improvement

Completers on average showed clinically significant improvement in symptoms. A repeated-measures MANOVA using BDI and HRSD scores as criterion variables was conducted with Treatment Condition as a between-subjects factor and Time (pretreatment, posttreatment) as a within-subject factor. A significant effect was found only for Time, $F(1, 42) = 13.66, p < .001$; neither the main effect for Treatment Condition nor the Time \times Treatment interaction were statistically significant. For the medication condition, the mean BDI score fell from 27.1 ($SD = 7.3$) pretreatment to 12.2 ($SD = 5.4$) posttreatment, and the HRSD from 20.4 ($SD = 3.5$) to 8.4 ($SD = 4.6$). For the CBT condition, the mean BDI score fell from 25.1 ($SD = 7.5$) pretreatment to 12.0 ($SD = 5.7$) posttreatment, and the HRSD from 21.9 ($SD = 5.2$) to 9.6 ($SD = 4.6$). For the IPT condition, the mean BDI score fell from 27.0 ($SD = 8.0$) pretreatment to 11.9 ($SD = 6.9$) posttreatment, and the HRSD from 22.0 ($SD = 6.6$) to 10.0

⁶Supplemental analyses indicated that this significant difference in age did not affect the outcome of any of the analyses reported below. Thus, for convenience we present the results without including age as a covariate.

($SD = 5.9$). Using the criteria for reliable clinically significant change specified by Ogles et al. (1995), we observed that 30 of the 44 patients (68%) manifested a clinically significant reduction of depressive symptoms on the BDI and 27 of the 44 (61%) manifested clinically significant improvement on the HRSD.

Change in Magnitude of Self-Discrepancy

How did the treatments affect type and magnitude of self-discrepancy? A repeated-measures ANOVA was conducted for self-discrepancy with Treatment Condition as a between-subjects factor and Time (pretreatment, posttreatment) and Type of Discrepancy (AI, AO) as within-subject factors. A significant main effect for Time was observed, $F(1, 40) = 8.85, p < .01$, in which overall posttreatment self-discrepancy was significantly less than pretreatment self-discrepancy, as well as a significant main effect for Type of Discrepancy, $F(1, 40) = 7.71, p < .01$, with AI scores significantly higher across the study period than AO scores. The effect for Treatment Condition was nonsignificant. In addition, we observed a significant Time \times Type of Discrepancy interaction, $F(1, 40) = 6.49, p < .05$, indicating that there was greater reduction in AI discrepancy than AO discrepancy.

Most importantly, we observed a significant Treatment Condition \times Time \times Type of Discrepancy interaction, $F(2, 40) = 5.87, p < .05$. Patients in the CBT and IPT conditions manifested substantial decreases in AI discrepancy, from means of 4.5 and 4.1 respectively pretreatment to 0.8 and 1.1 respectively posttreatment (combined effect size as $R^2 = .19$); patients in the medication condition manifested almost no change in AI discrepancy, with means of 4.2 pretreatment and 4.0 posttreatment (effect size as $R^2 = .01$). All treatments showed trivial decreases in AO discrepancy: medication, from 1.4 to 0.9; CBT, from 1.1 to -0.2 ; IPT, from 1.6 to 0.7.

Change in Response to Priming

How did the treatments affect reactions to priming with ideal self-guides? A repeated-measures ANOVA was conducted with time to retrieval as the dependent measure, using Treatment Condition as a between-subjects factor and Time (pretreatment, posttreatment) and Type of Priming (self-guide, yoked-control) as within-subject factors. Only a significant Type of Priming effect was observed, $F(1, 40) = 12.24, p < .001$. Combining across the pretreatment and posttreatment assessments and the three treatment conditions, patients showed significantly faster memory retrieval following presentation of self-guide cues (overall mean = 9.51 s) than following yoked-control cues (overall mean = 12.98 s). That is, memories cued by self-guides were more accessible than memories cued by yoked control primes both before and after each of the treatments.

Taking dysphoric memory content as the dependent measure, a repeated-measures ANOVA was conducted with Treatment Condition as a between-subjects factor and Time (pretreatment, posttreatment) and Type of Priming (self-guide, yoked-control) as within-subject factors. A main effect for Time was observed, $F(1, 40) = 6.18, p < .05$, as well as a main effect for Type of Priming, $F(1, 40) = 7.79, p < .01$, with incidence of dysphoric content following self-guide primes higher than incidence following yoked-control primes. We also observed a significant

Time \times Type of Priming interaction, $F(1, 40) = 5.51, p < .05$; dysphoric content following self-guide primes tended to decrease following treatment whereas dysphoric content following yoked-control primes did not.

Most importantly, we observed a significant Treatment Condition \times Time \times Type of Priming interaction, $F(2, 40) = 6.98, p < .05$. Patients in the CBT and IPT conditions manifested significant decreases in incidence of dysphoric content following self-guide cues, from mean incidence of 2.1 and 2.4 per memory respectively pretreatment to 0.7 and 0.9 respectively posttreatment (combined effect size as $R^2 = .27$). Patients in the medication condition showed a slight increase in incidence of dysphoric content, with a mean of 2.4 pretreatment and 2.7 posttreatment (effect size as $R^2 = .025$).

Correlation Between Improvement and Cognitive Change

Was there an association between clinical improvement and change in self-discrepancy or priming responses in any of the treatment conditions or both? To answer this question, we conducted a hierarchical regression analysis using posttreatment AI discrepancy as the criterion variable. In the first step, treatment condition, pretreatment BDI score, and pretreatment AI discrepancy were entered; this step accounted for a significant portion of variance, $R^2 = .089, F(3, 40) = 7.75, p < .05$. In the second step, posttreatment BDI score was entered, accounting for a significant increment in variance, increment in $R^2 = .065, F(1, 40) = 5.12, p > .05, \beta = .32$. That is, decrease in BDI scores was significantly but modestly associated with decrease in AI discrepancy. No interaction involving treatment condition was found, suggesting that the association between clinical improvement and change in AI discrepancy did not vary across treatment.

We then conducted a hierarchical regression analysis to determine whether symptom reduction and change in reactivity to priming with self-guides were correlated, using posttreatment dysphoric content in response to self-guide cues as the criterion variable. In the first step, treatment condition, pretreatment BDI, and pretreatment dysphoric content following self-guide cues were entered; this step accounted for a nonsignificant portion of variance ($R^2 = .036$). In the second step, posttreatment BDI was added but did not account for a significant increment in variance of posttreatment priming reactivity (increment in $R^2 = .02$). That is, decrease in BDI scores was not correlated with decrease in reactivity to priming with self-guide cues. In addition, we did not observe an interaction with treatment condition in this analysis.

Correlation Between Changes in Self-Discrepancy and in Response to Priming

Although cognitive change was at best only modestly associated with clinical improvement, were the two indices of cognitive change associated within any of the treatments? A hierarchical regression analysis was conducted in which posttreatment dysphoric content following self-guide priming was the criterion variable. In the first step, pretreatment AI discrepancy, dysphoric content following self-guide priming, and treatment condition were entered; this step accounted for a nonsignificant portion of variance ($R^2 = .034$). Posttreatment AI discrepancy was added as a

second step and accounted for a significant increment in variance of posttreatment priming reactivity, increment in $R^2 = .08$, $F(1, 40) = 6.78$, $p < .01$, $\beta = .35$. That is, change in AI discrepancy (conscious self-regulatory cognition) was correlated with change in priming reactivity (unconscious self-regulatory cognition). No interaction involving treatment condition was found, suggesting that the association between the two measures of cognitive change did not vary across treatment.

Did Pretreatment Perceived Failure in Self-Regulation Predict Improvement?

Did patients with high levels of self-discrepancy, or strong affective responses to priming of self-guides or both, respond as well to treatment as other patients? To examine this question, we again divided patients into two groups, high-discrepant ($n = 20$) and low-discrepant ($n = 24$), using the same criterion as in Study 1 (three or more instances of actual:ideal mismatch). A repeated-measures MANOVA was conducted with BDI and HRSD scores as criterion variables, Treatment Condition as a between-subjects variable, and Time (pretreatment, posttreatment) and Level of Discrepancy as within-subject factors. A significant main effect for Time was observed, $F(1, 42) = 13.66$, $p < .001$. We also observed a significant Time \times Level of Discrepancy interaction, $F(1, 40) = 5.01$, $p < .05$. High-self-discrepant patients manifested significantly less improvement (mean BDI scores were 26.2 pretreatment and 14.1 posttreatment) than their low-discrepant counterparts (mean BDI scores were 27.9 pretreatment and 8.2 posttreatment) across treatments (effect size as $R^2 = .10$). There was no evidence that high-self-discrepant patients responded differently to a particular treatment.

Overall, Study 2 yielded the following results: (1) As in Study 1, the sample of depressed patients manifested higher actual:ideal (AI) discrepancy than actual:ought (AO) discrepancy. (2) Completers on average showed a clinically significant degree of symptomatic improvement at the end of their treatment course (approximately 4 months); the three treatments were equivalently effective. (3) CBT and IPT were associated on average with a significant reduction in AI discrepancy (but not AO discrepancy); the medication condition was not. (4) CBT and IPT were associated on average with a significant reduction in dysphoric memory content following priming with self-guides; the medication condition was not. (5) Change in self-discrepancy and change in priming reactivity were correlated, but neither was strongly associated with clinical improvement. (6) Highly self-discrepant patients again showed less clinical improvement than other patients, this time across all three treatment conditions.

DISCUSSION

All three psychotherapy conditions examined (group CBT, individual CBT, and IPT) resulted on average in a statistically significant decrease in AI discrepancy, as well as a significant decrease in dysphoric memory content following priming with self-guides. In contrast, there was no reduction in AI discrepancy for the patients in Study 2 receiving medication, even though they showed clinical improvement equivalent to patients receiving psychotherapy. These findings indicate that reduction in

self-discrepancy is neither an epiphenomenon of clinical improvement nor necessarily mediated by change in mood state (cf. Strauman, 1992).

Because all three treatments in Study 2 were efficacious, why did patients receiving medication not manifest less self-discrepancy after successful treatment? We suggest several possibilities. It is possible that the lack of random assignment to treatment may have influenced this outcome, even though no differences were observed among the groups prior to treatment on any of the study variables. Second, it could be argued that patients in cognitive or interpersonal therapy merely had learned the socially desirable (non-self-discrepant) way to respond to the Selves Interview; that is, they became aware of their tendency to self-denigrate and by the end of the study were able to present themselves in a more positive light. However, because therapists did not have access to patients' interview responses, and since patients themselves were not able to identify the purpose of the interview, we see this possibility as unlikely.

Our preferred interpretation is that both CBT and IPT encourage patients to identify their goals, standards, and beliefs about themselves and (more importantly) to modify those aspects of self-regulation which are not effective. Although CBT and IPT are substantially different in technique as well as underlying theory, each emphasizes having patients examine *who they are* in relation to their individual "worlds"—a functional definition of motivationally significant self-knowledge (Higgins, 1998). Of course, we cannot rule out the possibility that other psychotherapies also would have reduced self-discrepancy; for example, client-centered therapy focuses in part on reducing the disparity between actual-self and ideal standards (Rogers, 1961). Nonetheless, the logic and interventions of CBT as well as IPT are clearly applicable to problems in self-regulation.

Likewise, why did patients receiving medication not manifest reduced priming reactivity? Again, it is possible that the lack of random assignment to treatment condition may have influenced the outcome. Due to the design of the priming task and logic of the dysphoric content measure, it is extremely unlikely that patients receiving psychotherapy were *intentionally* responding on the priming task in a socially desirable manner. Also, therapists did not have access to patients' priming-task responses, and patients were not able to identify the nature of the priming task at debriefing. Thus, we do not believe that the differential impact of psychotherapy versus medication on priming reactivity is an artifact.

Because there was no treatment-induced change in time to memory retrieval, the observed decrease in affective response to self-guide priming was not due to a change in the *accessibility* of self-evaluative standards. Instead, we suggest that the decrease in priming reactivity resulted from a change in the "distress potency" of self-evaluative standards. Self-guide cues were associated with the same ease of processing before and after treatment (measured via time to retrieval), indicating that they were no less likely to come to mind after a course of psychotherapy. The difference was that self-evaluative standards were simply less likely to trigger memories of unpleasant experiences—that is, less likely to signify *the psychological experience of failure*. A 4-month course of medication led to clinically significant improvement in depressive symptoms but evidently did not alter the psychological situation that was created for patients when they were indirectly exposed to their own ideal guides.

We view the present findings as both theoretically and clinically significant in light of research suggesting that perceived failure in self-regulation can contribute to the onset and maintenance of depressive episodes (Scott & O'Hara, 1993; Strauman, 1989, 1992). Both studies indicated that AI self-discrepancy and reactivity to priming with ideal guides are common features of depression. In addition, highly AI-discrepant patients in both studies did not respond as fully and quickly to existing treatments as other patients, even though such patients did not differ from others in severity, number of previous episodes, age, or gender. Thus, in addition to being a potential contributor to the onset of depression, chronic perceived failure in self-regulation also may predict treatment outcome. Patients for whom problems in self-regulation are prominent features of depression may require interventions which are targeted more specifically at the process of self-regulation as well as the availability, accessibility, and emotional significance of self-guides.

It should be acknowledged that the two studies were limited in a number of respects. The lack of a no-treatment control condition necessarily restricted the possible conclusions that could be drawn. Of course, this decision reflected our ethical concerns regarding withholding treatment; nonetheless, it is not possible to determine from this research the impact of spontaneous remission of depressive symptoms on self-discrepancy and priming reactivity. The lack of random assignment in Study 2 dictates caution in interpreting those findings, even though analyses of covariance did not detect group differences in any of the potential confounding factors that were assessed. In Campbell and Stanley's terminology (Campbell & Stanley, 1963), both studies used a nonequivalent control group design, a quasi-experimental design which controls for some (but not all) of the potential sources of invalidity in behavioral research. Also, therapists in Study 2 provided both CBT and IPT. There has been some controversy regarding the advantages and disadvantages of such a design element in outcome research; although the adherence data indicated that treatments were delivered adequately, there may be limits to the comparability of our findings with treatment studies conducted under different conditions. Fourth, in order to avoid complications with repeated assessment of self-discrepancies and responses to the memory task, those measures were only administered twice—not frequently enough to determine whether changes occurred at the same rate in different treatments. This decision reduced the likelihood that patients would identify our hypotheses or discuss interview or priming task responses during treatment sessions, but it necessarily limited the inferences that can be made regarding cognitive change.

There are a host of unanswered questions regarding self-regulatory cognition in depression. Foremost is the issue of relapse and recurrence: will those patients whose self-discrepancies and reactivity to priming with self-guide cues were altered be at reduced risk for subsequent episodes? In addition, will highly self-discrepant depressed patients benefit from longer treatment with conventional therapies, or will different therapeutic strategies be required? We look forward to continuing investigation of self-regulation in depression and to the opportunity to integrate this perspective with more established theories of cognitive vulnerability to depression.

ACKNOWLEDGMENTS

This research was supported in part by grants from the National Institute of Mental Health (#45800 and 52281). We thank Patricia Furlan, Tory Higgins, Marjorie Klein, Thomas Kwapil, Jodi Nordmann, John Wimberly, and two anonymous reviewers for their thoughtful comments on an earlier version of this manuscript.

REFERENCES

- Antonuccio, D. O., Danton, W. G., & DeNelsky, G. Y. (1995). Psychotherapy versus medication for depression: Challenging the conventional wisdom with data. *Professional Psychology: Research and Practice, 26*, 574–585.
- Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford.
- Beck, A. T., Ward, C., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 44*, 561–571.
- Beutler, L. (1991). Have all won and must all have prizes? Revisiting Luborsky et al.'s verdict. *Journal of Consulting and Clinical Psychology, 59*, 226–232.
- Blatt, S. J. (1995). The destructiveness of perfectionism: Implications for the treatment of depression. *American Psychologist, 50*, 1003–1020.
- Brewin, C. R. (1989). Cognitive change processes in psychotherapy. *Psychological Review, 96*, 379–394.
- Campbell, D. T., & Stanley, J. C. (1963). *Experimental and quasi-experimental designs for research*. Chicago: Rand McNally.
- Carver, C. S., & Scheier, M. F. (1990). Principles of self-regulation: Action and emotion. In E. T. Higgins & R. M. Sorrentino (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 3–52). New York: Guilford.
- Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology, 6*, 533–549.
- Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences, 22*, 491–569.
- Duval, S., & Wicklund, R. A. (1972). *A theory of objective self-awareness*. New York: Academic Press.
- Evans, M., Piasecki, J., Kriss, M., & Hollon, S. (1984). *Raters' manual for the Collaborative Study Psychotherapy Rating Scale*. Minneapolis: University of Minnesota.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured Clinical Interview for DSM-IV Axis I Disorders* (Patient ed., SCID-I/P, Version 2.0). New York: Biometrics Department, New York State Psychiatric Institute.
- Gottschalk, L. A., & Gleser, G. C. (1969). *The measurement of psychological states through the content analysis of verbal behavior*. Berkeley: University of California Press.
- Gottschalk, L. A., & Hoigaard-Martin, J. (1986). A depression scale applicable to verbal samples. *Psychiatry Research, 17*, 213–227.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry, 23*, 56–62.
- Higgins, E. T. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review, 94* (3), 319–340.
- Higgins, E. T. (1998). Promotion and prevention: Regulatory focus as a motivational principle. In M. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 30, pp. 1–46).
- Higgins, E. T., Bond, R., Klein, R., & Strauman, T. J. (1986). Self-discrepancies and emotional vulnerability: How magnitude, type and accessibility of discrepancy influence affect. *Journal of Personality and Social Psychology, 51*, 5–15.
- Hollon, S. D., DeRubeis, R. J., & Seligman, M. E. P. (1992). Cognitive therapy and the prevention of depression. *Applied and Preventive Psychology, 1*, 89–95.
- Huberty, C. J., & Morris, J. D. (1989). Multivariate analysis versus multiple univariate analyses. *Psychological Bulletin, 105*, 302–308.
- Kendler, K. S., Kessler, R. D., Neale, M. C., Heath, A. C., & Eaves, L. J. (1993). The prediction of major depression in women: Toward an integrated etiologic model. *Archives of General Psychiatry, 150*, 1139–1148.

- Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York: Basic Books.
- Kupfer, D. (1993). The mission of therapeutics. *Neuropsychopharmacology*, *9*, 169–180.
- Ogles, B. M., Sawyer, J. D., & Lambert, M. J. (1995). Clinical significance of the National Institute of Mental Health Treatment of Depression Collaborative Research Program data. *Journal of Consulting and Clinical Psychology*, *63*, 321–326.
- Persons, J. B., & Miranda, J. (1995). The search for mode-specific effects of cognitive and other therapies: A methodological suggestion. *Psychotherapy Research*, *5*, 102–112.
- Persons, J. B., Thase, M. E., & Crits-Christoph, P. (1996). The role of psychotherapy in the treatment of depression: A review of two practice guidelines. *Archives of General Psychiatry*, *53*, 283–290.
- Rogers, C. R. (1961). *On becoming a person*. Boston: Houghton Mifflin.
- Scott, L., & O'Hara, M. (1993). Self-discrepancies in clinically anxious and depressed university students. *Journal of Abnormal Psychology*, *102*, 282–287.
- Segal, Z. V., & Ingram, R. E. (1994). Mood priming and construct activation in tests of cognitive vulnerability to unipolar depression. *Clinical Psychology Review*, *14*, 663–695.
- Shaw, B. (1984). Specification of the training and evaluation of cognitive therapists for outcome studies. In J. B. W. Williams & R. L. Spitzer (Eds.), *Psychotherapy research: Where we are and where we should go* (pp. 173–188). New York: Guilford Press.
- Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin*, *86*, 420–428.
- Simons, A. D., Gordon, J. S., Monroe, S. M., & Thase, M. E. (1995). Toward an integration of psychological, social, and biologic factors in depression: Effects on outcome and course of cognitive therapy. *Journal of Consulting and Clinical Psychology*, *63*, 369–377.
- Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry*, *35*, 773–782.
- Spitzer, R. L., Williams, J. B., Gibbon, M., & First, M. B. (1992). The Structured Clinical Interview for DSM-III-R (SCID): I. History, rationale, and description. *Archives of General Psychiatry*, *49*, 624–629.
- Strauman, T. J. (1989). Self-discrepancies in clinical depression and social phobia: Cognitive structures that underlie affective disorders? *Journal of Abnormal Psychology*, *98*, 1–22.
- Strauman, T. J. (1992). Self-guides, autobiographical memory, and anxiety and dysphoria: Toward a cognitive model of vulnerability to emotional distress. *Journal of Abnormal Psychology*, *101*, 87–95.
- Strauman, T. J. (1996a). Self-beliefs, self-evaluation, and depression: A perspective on emotional vulnerability. In L. Martin & A. Tesser (Eds.), *Striving and feeling: Interactions among goals, affect, and self-regulation* (pp. 175–201). Hillsdale, NJ: Erlbaum.
- Strauman, T. J. (1996b). Stability within the self: A longitudinal study of the implications of self-discrepancy theory. *Journal of Personality and Social Psychology*, *71*, 1142–1153.
- Strauman, T. J. (1999). Is depression a dysfunction in self-regulating the brain/behavior system for approach? *Behavioral and Brain Sciences*, *22*(3), 571–572.
- Strauman, T. J. (2000). *Self-discrepancy and major depressive disorder: Cross-sectional and predictive associations*. Manuscript in preparation.
- Strauman, T. J. (2001). *Self-discrepancy as a predictor of subsequent depressive symptoms*. Manuscript under review, *Personality and Social Psychology Bulletin*.
- Teasdale, J. D., & Barnard, P. J. (1993). *Affect, cognition, and change: Re-modeling depressive thought*. Hillsdale, NJ: Erlbaum.
- Whisman, M. A. (1993). Mediators and moderators of change in cognitive therapy for depression. *Psychological Bulletin*, *114*, 248–265.