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Motivational Deficits Differentially Predict Improvement in a Randomized Trial of Self-System Therapy for Depression

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Abstract

Objective—A randomized trial compared the time course and differential predictors of symptom improvement in two treatments for depression.

Method—Forty-nine adults (84% female) who were not taking antidepressant medications and met diagnostic criteria for major depressive disorder or dysthymia were randomly assigned either to cognitive-behavioral therapy (CBT) or self-system therapy (SST), a treatment that targets problems in self-regulation, the ongoing process of evaluating progress toward personal goals. Self-regulatory variables (promotion and prevention focus, and goal disengagement and reengagement) were assessed as potential moderators of efficacy. At intake, most participants reported depression in the moderate to severe range and had histories of recurrent episodes and previous treatment attempts. Self-reported symptoms of depression and anxiety were assessed at each therapy session. Multilevel modeling was used to examine (1) differences in change associated with the treatment conditions, and (2) moderation of treatment efficacy by pre-treatment measures of self-regulatory deficits.

Results—Both treatments were effective and did not show differences in the magnitude or rate of symptom change or in drop-out rates, suggesting that CBT and SST were equally effective in improving depression and anxiety. Patients with self-regulatory deficits, however, showed greater improvement in depressive symptoms with SST. Specifically, patients with low promotion focus and low goal reengagement responded better to SST, while patients with high prevention focus responded better to CBT.

Conclusions—Overall, these results corroborate previous research suggesting that SST is a viable short-term treatment for depression that is particularly effective in helping patients compensate for self-regulatory deficits.

Keywords

depression; self-system therapy; cognitive therapy; goal adjustment; regulatory focus

Engagement in, and successful accomplishment of, daily goal-directed activities is fundamental in supporting emotional well-being (Carver & Scheier, 1990; Deci & Ryan, 2000; Emmons, 1986). Evidence suggests that problems in self-regulation, or the internal

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and transactional processes that enable control of goal-directed activities (Karoly, 1999), are associated with emotional and physical problems (Eddington, 2013; Elliot & Sheldon, 1998; Strauman & Higgins, 1987; Strauman, Woods, Schneider, Kwapil, & Coe, 2004). Major depressive disorder (MDD) is defined in part by diminished motivation or loss of interest in daily activities, and behavioral conceptualizations propose that depression stems from a lack of engagement in goal-directed activities and positively-reinforcing behaviors (Jacobson, Martell, & Dimidjian, 2001; Lewinsohn & Graf, 1973).

Regulatory focus theory (RFT; Higgins, 1997) is a model of self-regulation that proposes two categories of goals, each associated with specific motivational states and strategies for goal pursuit. *Promotion goals* involve advancement, growth, and achievement; pursuing promotion goals means *making good things happen*, which is associated with either joyful or dysphoric mood, depending upon one's progress. *Prevention goals* involve security, safety, and responsibilities/obligations; pursuing prevention goals involves *keeping bad things from happening*, which is associated with either quiescent or anxious affect, depending upon one's progress. RFT stipulates that people whose socialization history (social/family environment during childhood) did not include an emphasis on rewarding activities and positive outcomes would have difficulty pursuing and attaining promotion goals during adolescence and adulthood and would be less oriented toward aspirations and accomplishments (Higgins, 1989; Higgins, Roney, Crowe, & Hymes, 1994). People with a low promotion focus thus have fewer opportunities to experience the positive motivational and affective states associated with moving toward promotion goals (Forster, Grant, Idson, & Higgins, 2001) and are at elevated risk for depression (Strauman, 2002). Exposure to stimuli representing one's unmet promotion goals elicits anxious and depressive feelings (Strauman, 1992) and activates areas of the cortex that have strong neural connections to areas involved in emotion and reward processing (Eddington, Dolcos, Cabeza, Krishnan, & Strauman, 2007; Eddington et al., 2009).

Strauman et al. (2001) found that three traditional, empirically supported treatments—cognitive-behavior therapy (CBT), interpersonal psychotherapy (IPT), and pharmacotherapy with selective serotonin reuptake inhibitors—were less effective for depressed adults with perceived failure in promotion goal pursuit than for other depressed patients. They concluded that interventions that directly target self-regulatory processes may be more effective for patients whose depression is associated with low promotion goal pursuit. An important question concerns whether these patients can learn skills to compensate for deficiencies in self-regulation. Self-system therapy (SST), a recent addition to the empirically-supported treatments for depression, aims to translate basic research on goal pursuit and self-regulation to the intervention context (Vieth et al., 2003), providing a means of improving patients' self-regulatory skills.

SST is a short-term therapy that targets problems in self-regulation and was conceptualized as a treatment that would be particularly effective for depressed people with problems in self-regulation, such as problems with pursuing and achieving promotion-type personal goals. The proposed mechanism of action in SST, therefore, is distinguishable in theory from that of other therapies such as CBT and IPT (Vieth et al., 2003), although it borrows

specific strategies (adapted for the purposes of improving self-regulation) from other, empirically-validated treatment approaches.

SST is hypothesized to produce symptom improvement through changes in goal representation (how people think about their goals) and goal pursuit (how people go about trying to achieve their goals). In CBT, the primary focus is on recognizing and challenging dysfunctional negative thoughts. In SST, the primary focus is on identifying the personal goals, standards, and expectations that guide the patient's behavior and on initiating changes that allow the patient to be more successful in goal pursuit, particularly for promotion-type goals. SST consists of three treatment phases: orientation (setting treatment goals, introducing concepts of promotion and prevention, and discussing the impact of the patient's own socialization history on her regulatory focus), exploration (closely examining one's self-standards as they come into play in daily situations and events), and adaptation (implementation of specific strategies to improve self-regulation).

Strauman et al. (2006) conducted a randomized clinical trial to determine whether SST was more effective than CBT for depressed patients with a poor promotion socialization history. Both CBT and SST were efficacious overall, but SST was more effective for patients whose self-reported socialization history lacked a promotion emphasis. Using a priming procedure in which the emotional content of responses to idiographic promotion goals was assessed before and after treatment, Strauman et al. (2006) found that the dysphoric content of responses was lower following treatment with SST compared to CBT. Together, these findings show that SST is effective for treating depression—especially for patients with a low promotion orientation—and that the two treatments may have different mechanisms of action. Furthermore, these results suggest that, rather than capitalizing on existing strengths in self-regulation, SST targets strategies that help patients compensate for deficits in self-regulation.

In the Strauman et al. (2006) clinical trial, treatment dose was unconstrained, the mean number of sessions was 22 (minimum 10, maximum 31), and symptom change was examined at only two time points (pre- and post-treatment). In addition, the initial trial included adults whose depression was, on average, at the low end of the moderate range. Since that initial trial, SST has been condensed into a manualized, 16-session protocol (see T. J. Strauman et al., 2001 for original research manual). The current study aimed to examine the efficacy of the shorter protocol in comparison with traditional cognitive-behavioral therapy (CBT) and to examine its efficacy in a sample of adults whose depression is in the moderate to severe range. Furthermore, we examined whether the two treatments showed differential efficacy for depressed adults with deficits in self-regulation. Specifically, we sought to replicate and extend previous results showing that SST is more effective for depressed patients low in promotion orientation. As such, we assessed promotion and prevention orientations with the more widely-used measures of success with promotion and prevention means of goal attainment (Higgins et al., 2001) rather than measures of socialization history.

We also included an additional measure of self-regulatory function: goal adjustment, the ability to respond flexibly to obstacles in goal pursuit (e.g., unexpected obstacles, stalled

progress). Goal adjustment is associated with higher well-being. Specifically, studies have shown that the ability to *disengage* from unattainable goals and *reengage* in new goals predicts higher levels of emotional and physical well-being (Rasmussen, Wrosch, Scheier, & Carver, 2006; Wrosch, Scheier, Carver, & Schulz, 2003; Wrosch, Scheier, Miller, Schulz, & Carver, 2003). Consistent with the notion that SST helps patients compensate for self-regulatory deficits, patients with poorer goal adjustment are expected to benefit more from SST than CBT.

In this study, symptoms of depression were assessed at each treatment session, rather than only at pre- and post-treatment, and trajectories of change over time were modeled to examine predictors of symptom change over the course of treatment. The present study thus offered a more comprehensive assessment of the process of change during the course of psychotherapy. We predicted that, consistent with the previous randomized trial, SST would be as effective as CBT overall for reducing depressive symptoms, even with a shorter treatment protocol and higher level of depression severity. Furthermore, we predicted that SST would be more effective with patients showing deficits in self-regulation, particularly people lower in promotion focus. We also examined the role of prevention focus but did not have a priori hypotheses about those results. We also conducted exploratory analyses on the relative efficacy of the two treatments for reducing anxiety symptoms. The original SST-CBT trial did not report outcomes for anxiety, but because depression and anxiety are often comorbid, it is important to determine whether SST is more or less effective in reducing anxiety symptoms.

Method

Participants

Participants were recruited from the greater Greensboro area primarily through advertisements in local health magazines, flyers placed in outpatient mental health clinics, and online recruitment. Inclusion criteria were as follows: ability to comply with the requirements of the study, primary DSM-IV-TR diagnosis of major depressive disorder (MDD) or dysthymia, BDI-II score above 14 (indicating more than minimal symptoms; A. T. Beck, Steer, & Brown, 1996), no antidepressant medication use (or herbal remedies for depression) for the past four months, no history of mania, no active substance dependence for the previous six months, no history of psychotic symptoms, no diagnosis of antisocial or borderline personality disorders, and no active suicidal intent or immediate threats of self-harm. Exclusionary symptoms and conditions were assessed using the SCID interview (see description below).

One hundred adults completed a phone screening and were invited to complete a more thorough assessment in the laboratory (see enrollment flow chart in Figure 1). Fifty-six adults met inclusion criteria and were assigned to either SST (n = 27) or CBT (n = 29) using a restricted randomization procedure. A random number generator was used to establish the initial assignments. Alterations to the sequence were made when a therapist for the assigned condition was not available or (near the end of the trial) in an attempt to balance sample sizes. Seven of the 56 participants did not return for the first treatment session and therefore are not included in the subsequent analyses. Thus, the final sample included 49 participants

in SST (n = 22) and CBT (n = 27). Details on the final sample demographics, symptom severity at intake, diagnostic status, and treatment history are shown in Table 1. Some data on sample race and ethnicity were missing due to experimenter error, but it should be noted that the majority of the sample consisted of non-Hispanic/Latino White participants. The only significant difference in demographic variables or pre-treatment symptom severity was a greater prevalence of dysthymia (primary or secondary) in the CBT condition. Across the two conditions the participants had a mean intake BDI-II score of 34.6 (SD = 8.5), which is at the low end of the severe range, and a mean intake BAI score of 23.0 (SD = 14.7), which is in the moderate range.

Measures

Semi-Structured Clinical Interview for DSM-IV-TR–Research Edition—The SCID-I and SCID-II (First, Gibbon, Spitzer, Williams, & Benjamin, 1997; First, Spitzer, Gibbon, & Williams, 2002) are widely used semistructured diagnostic interviews for DSM-IV-TR (American Psychiatric Association, 2000) Axis I and Axis II disorders, respectively. From the SCID-I, diagnosticians completed the overview and screening sections, mood modules, anxiety modules, and psychotic symptoms. Additional modules (e.g., substance abuse and dependence, eating disorders) were used as needed based on the participant's responses to the screening items. From the SCID-II, diagnosticians completed the antisocial and borderline personality disorders modules only.

Beck Depression Inventory—The BDI-II (A. T. Beck et al., 1996) is a widely used assessment of patient-reported depressive symptom severity of the past two weeks. There are 21 items and each is rated on a 4-point scale (0–3) scale, with total scores ranging from 0 to 63. The BDI-II has excellent psychometric properties (Dozois & Covin, 2004), and the internal consistency in this study was good (Cronbach's α = .84).

Beck Anxiety Inventory—The BAI (A. T. Beck, Epstein, Brown, & Steer, 1988) is a widely-used measure of anxiety symptom severity of the past two weeks with good psychometric properties (Fydrich, Dowdall, & Chambless, 1992). There are 21 items and each is rated on a 4-point scale (0–3) scale, with total scores ranging from 0 to 63. In this study, internal consistency was very good (Cronbach's α = .95)

Regulatory Focus Questionnaire—The RFQ (Higgins et al., 2001) is an 11-item measure that assesses subjective history of success using promotion- and prevention-oriented goal attainment. Each item is rated on a 5-point scale, and there are two subscales (Promotion Focus and Prevention Focus). An example item for Promotion is "*Compared to most people, are you typically unable to get what you want out of life?*" (reverse-coded) and for Prevention, "*How often did you obey rules and regulations that were established by your parents?*" Previous studies have reported good internal consistency values (Cronbach's α) of .73 and .80 for the Promotion and Prevention focus subscales, respectively (Higgins et al., 2001). In the current study, internal consistency for the Promotion was α = .73 and α = .82 for Prevention.

Goal Adjustment Scale—The GAS (Wrosch, Scheier, Miller, et al., 2003) consists of 10 items, four measuring goal disengagement (GAS-D) and six measuring goal reengagement (GAS-R). The questionnaire instructs the respondents to consider how they usually react when the have to stop pursuing important goals in their lives (e.g., "*I start working on other goals*" reflects reengagement; "*I find it difficult to stop trying to achieve the goal*" reflects disengagement). Item statements are rated on a 5-point scale of 1 to 5, with higher scores indicating better ability to disengage from goals (GAS-D) or to reengage in alternative goals (GAS-R) in the face of failure. Good internal consistency of the GAS-D (Cronbach's α = .84) and GAS-R (Cronbach's α = .86) has been reported (Wrosch, Scheier, Miller, et al., 2003); internal consistency in the current study was α = .82 for the GAS-D and .80 for the GAS-R.

Treatment Conditions

Self-system therapy (SST)—SST is a brief, structured therapy that draws on motivational theories of depression and integrates techniques from established depression treatment protocols. The treatment involves assessing the patient's goal pursuit history and style while identifying (and correcting) deficiencies in goal pursuit (especially promotion orientation). In the current study, a 16-session SST protocol was used that includes a clinician manual and patient worksheets; a detailed description of the development of SST is provided by Vieth et al. (2003).

Cognitive-behavioral therapy (CBT)—The CBT condition (also 16 sessions) was based on two widely-used treatment guides: *Cognitive Behavior Therapy: Basics and Beyond* (J. S. Beck, 2011), a fundamental therapist CBT guide, and *Mind Over Mood: Change How You Feel by Changing the Way You Think* (Greenberger & Padesky, 1995), a patient-oriented CBT guide with worksheets and patient-directed readings. The full protocol included psychoeducation about depression, behavioral activation, development of a cognitive conceptualization and identification of core beliefs, cognitive restructuring, and relapse prevention, and it was guided in sequence by worksheets from the *Mind Over Mood* workbook.

Therapists—Therapists were six master's-level trainees (including one of the co-authors, T.E.F.) enrolled in a clinical psychology doctoral program and one doctoral-level clinician who had just completed a predoctoral internship. All therapists were required to have at least 250 direct patient contact hours in order to serve as study therapists and were required to have prior supervised training with structured, empirically-based individual therapy. All the therapists had prior training in cognitive-behavioral treatment approaches with adult patients. Three of the seven therapists saw 3–4 patients each, and the remaining four therapists saw 9–11 patients each.

Therapist training and adherence monitoring—The study included three SST therapists and four CBT therapists; therapists provided treatment for only one of the two conditions. The primary rationale was that some of the therapists were only available to see a limited number of participants, and we anticipated that focused training in one condition would result in better adherence and competency. We used a preceptor training model in

which a primary supervisor (the first author, K.M.E., a certified Cognitive Therapy Trainer who is also trained in SST) provided intensive individual supervision and feedback to ensure treatment condition adherence. Didactic training for the SST therapists included assigned reading of the manual and worksheet packet followed by a half-day orientation and training session conducted by Timothy J. Strauman, one of the original developers of SST. Therapists in the CBT condition required relatively less training because the therapists were already well-versed in CBT techniques and strategies based on their coursework and practicum training. CBT training, which also followed the preceptor model, included assigned reading of the two treatment guides followed by a one-hour training provided by the first author.

In addition to didactic training, each therapist met weekly with the primary supervisor (K.M.E.) to monitor compliance with the assigned treatment condition. Every session was audiotaped, and tapes of every session for each therapist's first assigned patient were reviewed by the supervisor. Following the first assigned patient, if compliance was consistently good, random reviews of audiotaped sessions were conducted, although weekly supervision meetings continued throughout the course of treatment for all patients. Failure to adhere to the protocol would have resulted in a remediation process that included additional training and monitoring, although no adherence violations occurred.

A quasi-random selection of 105 session audio recordings was coded for adherence by independent coders. To ensure that adherence across the full course of therapy was assessed for each participant, recordings were divided into three treatment phases: sessions 2 – 6, 7 – 11, and 12 – 15. The first and final sessions were excluded from selection because the focus in those sessions (background and psychoeducation in session 1; relapse prevention and review in session 16) overlaps substantially in the two treatment conditions. Using a random number generator, one session per phase was selected for coding. Sessions from seven participants who completed fewer than 3 sessions total were not included in the adherence coding.

Sessions were coded using a scale similar to that described in Strauman et al. (2006), which was based on the Collaborative Study Psychotherapy Rating Scale (Evans, Piasecki, Kriss, & Hollon, 1984). The 54 adherence items consisted of 3 scales: common factors (CF; 21 items, Cronbach's $\alpha = .70$), SST adherence (18 items, $\alpha = .83$), and CBT adherence (15 items, $\alpha = .84$). Items were summed to yield a total score for each of the 3 scales. Eighty percent of the 105 sessions were independently coded by a second coder to evaluate inter-rater reliability of the coding scales.

Procedures

During the initial laboratory assessment, participants provided informed consent, completed the BDI-II and BAI, and (if the BDI-II score was above 14) completed relevant portions of the SCID-I and SCID-II. Diagnostic interviews were conducted by graduate students enrolled in a clinical psychology doctoral program who received training on the administration of the SCID-I and SCID-II. Consultation on diagnostic interviews was provided by licensed, doctoral-level psychologists (K.M.E. and T.R.K.) with extensive experience both with diagnostic assessment and with the administration of the SCID-I and

SCID-II. Following completion of the diagnostic interviews, participants completed a short battery of questionnaires on a computer and were given further instructions about the treatment portion of the study.

One week after the initial assessment, participants began the treatment phase of the study. Individual outpatient sessions were conducted at the UNCG Psychology Clinic. Prior to each therapy session, participants completed both the BDI-II and the BAI. Sessions were scheduled once weekly for 50 minutes, but the full course of treatment was not restricted to 16 weeks and often exceeded that length as a result of rescheduling due to illness, work conflicts, and other unexpected events. Participants received treatment at no cost and were compensated for the time required for data collection outside of the therapy sessions. Data entry and coding were done by research assistants who were unaware of the participants' treatment condition.

Results

Therapist Adherence to Treatment Protocols

Intraclass correlation coefficients were calculated for each of the three adherence scales (CF, SST, and CBT) to assess inter-rater reliability. Results showed good reliability for all three scales, CF = .89, SST = .74, and CBT = .78. Mean item ratings for all sessions that were coded by two raters were used in subsequent analyses.

SST sessions had significantly higher mean scores on the SST adherence scale (M = 35.76, SD = 9.61) compared to CBT sessions (M = 19.02, SD = 3.00; $t(58) = -11.18$; $p < .001$). Likewise, CBT sessions had significantly higher mean scores on the CBT adherence scale (M = 35.25, SD = 9.06) compared to SST sessions (M = 17.89, SD = 4.19; $t(78) = -11.18$; $p < .001$). For the CF adherence scale, SST sessions also had slightly higher mean scores (M = 69.74, SD = 12.82) compared to CBT sessions (M = 64.07, SD = 8.81; $t(86) = -2.62$; $p < .05$)[1].

Model Specification and Analytic Approach

Following an intent-to-treat framework, we included all available data in the analyses. The primary analytic approach was multilevel modeling, which accommodates between-person and within-person effects in light of missing data and unequal numbers of observations per participant (Raudenbush & Bryk, 2002; Singer & Willett, 2003). The models were estimated in HLM 7 using restricted maximum likelihood with robust standard errors, which performs better than full maximum likelihood when Level 2 sample sizes are smaller (Maas & Hox, 2005). At Level 1, the within-person level, time was specified according to sessions: the first session was scored as 0, yielding a range from 0 to 15 and making the intercept interpretable as BDI-II scores at the first session. A quadratic term was created by squaring the linear term. At Level 2, treatment condition was scored as 0 for CBT and 1 for SST. The potential

[1]Due to unequal variances between conditions, adjusted degrees of freedom were used in all 3 mean comparisons. CF scale scores are generally expected to be higher because (in addition to having more items) more of those items tap into therapist behaviors that occur throughout the course of therapy (e.g., collaboration, setting the session focus). By contrast, more of the SST and CBT items tap into specific strategies that may occur only in a certain phase of therapy (e.g., discussing the distinction between promotion and prevention in SST).

moderators (e.g., prevention and promotion focus) were standardized to create a meaningful 0 point. All intercepts and slopes were modeled as random.

Effect sizes in multilevel modeling are considerably more complex than in conventional single-level models. To estimate effect sizes, we computed the proportional reduction in unexplained variance (PRV), which evaluates the reduction in variance at a particular level when a predictor is added (Raudenbush & Bryk, 2002). One well-known issue with PRV is that it can occasionally yield small negative values (Snijders & Bosker, 1994), which is more likely to happen when "a truly nonsignificant predictor is entered into the equation" (Raudenbush & Bryk, 2002, p. 150).

A null model of BDI-II scores found an intraclass correlation (ICC) of .526, indicating that roughly equal amounts of variance in BDI-II scores were at Level 1 (47.4%) and Level 2 (52.6%). Table 2 shows the descriptive statistics and intercorrelations among the self-regulation measures and intake symptom scores.

Change in Depressive Symptoms and Treatment Efficacy

The first model estimated the within-person trajectories of change across sessions; Table 3 depicts the results. This model estimated linear and quadratic effects of time on BDI-II scores:

$$\text{Level1:BDI}-\text{II}_{ij}=\beta_{0j}+\beta_{1j}(\text{Session})_{ij}+\beta_{2j}(\text{Session}^2)_{ij}+r_{ij}$$

$$\text{Level2:}\beta_{0j}=\gamma_{00}+\mu_{0j}\ \ \beta_{1j}=\gamma_{10}+\mu_{1j}\ \ \beta_{2j}=\gamma_{20}+\mu_{2j}.$$

The model found significant linear ($\gamma = -1.75$, *SE* = .34, $p < .001$) and quadratic ($\gamma = .04$, *SE* = .02, $p = .028$) effects of time, reflecting the substantial drop in BDI-II scores across the sessions and the tendency for gains to be greatest in the earlier sessions. Regarding effect sizes, including the linear effect of time reduced the unexplained variance by 56.67%, and adding both the linear and quadratic effects reduced the unexplained variance by 60.66%, so the effect of sessions on BDI-II scores was substantial. Because subsequent analyses found very few moderating effects of the quadratic component, and because the quadratic component explained a fairly small amount of incremental variance, subsequent analyses simplified the model by omitting the quadratic component and estimating the moderators of linear change.[2]

Our next model examined the effect of condition to evaluate if CBT and SST had different rates of change across sessions:

[2]Specifically, treatment condition, promotion focus, goal disengagement, and goal reengagement had no significant main effects or interactions involving the quadratic component. The only significant effects were a main effect of prevention focus and a prevention-by-condition interaction for the quadratic component. The effect size of treatment condition on the quadratic component was small and negative.

$$\text{Level1:BDI}-\text{II}_{ij}=\beta_{0j}+\beta_{1j}(\text{Session})_{ij}+r_{ij}$$

$$\text{Level2:}\beta_{0j}=\gamma_{00}+\gamma_{01}(\text{Condition})_j+\mu_{0j}\ \ \beta_{1j}=\gamma_{10}+\gamma_{11}(\text{Condition})_j+\mu_{1j}.$$

Figure 2 depicts BDI-II scores across sessions for each condition. The multilevel model, reported in Table 4, revealed a non-significant effect of treatment condition on the rate of change ($\gamma = -.49$, *SE* = .35, $p < .164$), suggesting that CBT and SST were equally effective overall. Regarding the effect size, adding treatment condition as a predictor of the within-person slopes reduced the variance component by only 2.11%.

An additional metric of change is the percent of participants who ended participation with scores below 14, a common cut-off informed by clinical guidance and research on clinically significant change (Seggar, Lambert, & Hansen, 2002). The proportions of participants who ended with scores below 14 were similar for the CBT (11 of 27, 40.7%) and SST (12 of 22, 54.5%) conditions. A logistic regression (BDI-II ≥ 14 = 0, BDI-II < 14 = 1) model did not find a significant difference between the therapy conditions, *b* = .56, *SE* = .58, *p* = .37, *OR* = 1.75 (*OR* 95% CI = .56, 5.44).

Self-regulatory Moderators of Treatment Efficacy

Our next models examined whether self-regulatory factors moderated the efficacy of CBT and SST. These models included the main effect of a self-regulatory factor and, critically, its interaction with the treatment condition. Each model had the same specification. For example, the model that examined promotion focus as a moderator was specified as follows:

$$\text{Level1:BDI}-\text{II}_{ij}=\beta_{0j}+\beta_{1j}(\text{Session})_{ij}+r_{ij}$$

$$\text{Level2:}\beta_{0j}=\gamma_{00}+\gamma_{01}(\text{Condition})_j+\gamma_{02}(\text{Promotion})_j+\gamma_{03}(\text{Condition}\times\text{Promotion})_j+\mu_{0j}$$
$$\beta_{1j}=\gamma_{10}+\gamma_{11}(\text{Condition})_j+\gamma_{12}(\text{Promotion})_j+\gamma_{13}(\text{Condition}\times\text{Promotion})_j+\mu_{1j}.$$

Table 4 displays the results of these models.

Promotion focus and prevention focus—We first examined promotion focus and prevention focus, assessed using the RFQ, as moderators. For promotion, the multilevel model found a significant interaction between session, treatment, and promotion, $\gamma = .99$, *SE* = .31, $p = .002$. Figure 3 shows the model-estimated BDI-II scores for sessions 1 and 16 for people low and high in promotion focus.

As expected, people low in promotion focus showed greater improvement in SST than in CBT. We estimated the proportional reduction in variance by comparing the model to one in which treatment condition was the only predictor. Compared to this treatment-only baseline

model, including promotion's main effect and interaction with treatment condition reduced the unexplained variance in change slopes by 25.59%.

For prevention focus, a similar pattern appeared. The multilevel model found a significant interaction between session, treatment, and prevention, $\gamma = 1.11$, $SE = .26$, $p < .001$. Figure 4 shows the model-estimated BDI-II scores. People high in prevention focus benefitted more from CBT than SST. People high in prevention focus also had significantly lower intercepts, reflecting lower first-session BDI-II scores ($\gamma = -3.61$, $SE = 1.31$, $p = .008$). Compared to the treatment-only baseline model, including prevention's main effect and interaction with treatment condition reduced the unexplained variance in change slopes by 24.02%.

Goal disengagement and goal reengagement—Our next models examined the two facets of the Goal Adjustment Scale: goal disengagement and goal reengagement. Goal disengagement did not moderate the effect of treatment condition on change in BDI-II scores or have any other significant effects; the results are displayed in Table 4. This model yielded an example of a small negative proportion reduction in variance, which can occur in some cases (e.g., when Level 2 predictors are added that have essentially no effect; Raudenbush & Bryk, 2002, p. 150).

Goal reengagement, in contrast, yielded several important significant effects (see Table 5). People high in goal reengagement had significantly lower intercepts, reflecting lower first-session BDI-II scores ($\gamma = -3.44$, $SE = 1.48$, $p = .025$). Most relevant, however, is that goal reengagement significantly moderated the effect of treatment condition on change in BDI-II scores ($\gamma = .76$, $SE = .27$, $p = .007$). The pattern is depicted in Figure 5. As the figure shows, people low in goal reengagement benefitted more from SST than CBT. People high in goal reengagement, in contrast, benefitted equally from CBT and SST. Adding GAS-R and its interaction with treatment condition yielded a 29.89% reduction in the variance of the change slopes compared to the treatment-only baseline model.

Summary of the self-regulatory moderators—The prediction that participants with self-regulatory deficits would benefit more from SST than CBT was supported. Consistent with the previous trial (Strauman et al., 2006), low promotion focus was associated with a greater decline in depressive symptoms in the SST condition. Low prevention focus showed a similar pattern. Regarding goal adjustment, SST was more effective than CBT for patients with difficulties with goal reengagement, but no differences were found for disengagement.

Effects of CBT and SST on Anxiety

Multilevel models found that anxiety, measured at each session with the BAI, declined as well. The ICC for BAI scores was .676, so most of the variance (67.6%) was at the between-person level. A within-person model found significant linear ($\gamma = -1.55$, $SE = .25$, $p < .001$) and quadratic ($\gamma = .07$, $SE = .01$, $p < .001$) effects of time, reflecting an overall decline in BAI scores from the initial level ($\gamma = 16.74$, $SE = 1.40$, $p < .001$) across the sessions, particularly in the early weeks. Regarding effect size, including the linear and quadratic effects of session reduced the unexplained variance in BAI scores by 45.63%.

Subsequent models examined whether treatment condition and self-regulatory constructs moderated the linear change in BAI scores across sessions. As shown in Table 6, there was a non-significant effect of treatment condition on the rate of linear change ($\gamma = -.10$, *SE* = .23, $p < .648$), suggesting that CBT and SST were also equally effective in reducing anxiety. (The PRV effect size estimate was small and negative in this case.) Both promotion focus ($\gamma = .47$, *SE* = .21, $p = .034$, PRV = negative) and prevention focus ($\gamma = .66$, *SE* = .28, $p = .022$, PRV = 24.86%) significantly moderated the effect of treatment condition on rates of change (see Table 6). As Figure 6 shows, the patterns resembled the effects of promotion and prevention focus on BDI-II change. For both prevention focus and promotion focus, people low in the trait showed greater improvement in BAI scores in SST compared to CBT. People high, in contrast, showed less or no improvement in SST compared to CBT. No significant main effects or interactions were found for goal disengagement and reengagement.

Dropout and Length of Participation

Our final analyses explored predictors of dropout and length of participation. For dropout, each participant was classified as a "dropout" (0) or "completer" (1). Most completers attended all 16 sessions, but several were "early completers" who received a full "dose" of treatment in fewer than 16 sessions. Decisions to end early were based on a discussion between the therapist and supervisor and were permitted only if a full dose of therapy was received and stable improvement was evident. Cases in which participants unexpectedly left even after many therapy sessions were not considered "early completers." Completion rates were similar for CBT (15 of 27, 55.6%) and SST (16 of 22, 72.7%). A logistic regression model did not find a significant difference between the therapy conditions, $b = .758$, *SE* = .616, $p = .219$, *OR* = 2.13 (*OR* 95% CI = .64, 7.13).

To evaluate the duration of participation in more detail, a Cox regression survival analysis was used to estimate the duration of participation across the 16 sessions (see Corning & Malofeeva, 2004). Treatment condition did not significantly predict durations, $b = -.207$, *SE* = .419, $p = .621$, reflecting similar probabilities of remaining in treatment across sessions for CBT and SST (see Figure 7).

Discussion

The primary aims of this study were to examine the efficacy of a brief, 16-session protocol of SST in comparison to CBT treatment in a sample of moderately to severely depressed adults and to replicate and extend previous research suggesting that SST may compensate for the deficits in self-regulation that are associated with depression. In contrast to traditional outcome studies in which participants are assessed at pre- and post-treatment, the current study examined trajectories of change over time with repeated assessments of symptom severity. The results supported our hypotheses. Regarding overall efficacy, we found that both SST and CBT were effective treatments for depression and did not differ in terms of the rate or magnitude of symptom improvement (depression and anxiety) or in terms of drop-out rates. More than half of our sample had a history of ten or more previous depressive episodes, and nearly a third of our sample had been previously hospitalized for mental health concerns. This independent replication bolsters SST as a viable treatment

approach for depression and further suggests that its efficacy extends to higher levels of depression severity. Furthermore, the successful implementation of SST using relatively novice therapists suggests that the therapy could be feasibly disseminated into real-world treatment settings.

We should note that rates of full recovery, defined by an index of clinically significant change, in both conditions were modest. Roughly half of the participants in each condition had residual symptoms in at least the mild range upon terminating treatment. It is difficult to directly compare these results with the previous SST trial (Strauman et al., 2006) because slightly different symptom measures and definitions of clinical significance were used and the samples differed in pretreatment severity, but our rates fit with studies of CBT delivered under a similar preceptor supervision model (e.g., Merrill, Tolbert, & Wade, 2003). Given the association between residual post-treatment depressive symptoms and risk for relapse and poor long-term outcomes (e.g., Judd et al., 1998; Kennedy & Paykel, 2004), the proportion of patients who did not achieve full recovery underscores the importance of examining supplemental approaches to prevent relapse and maintain treatment gains.

Although the two treatments were equally effective in decreasing symptoms of depression and anxiety, our results are consistent with Strauman et al. (2006) in that participants with greater self-regulatory dysfunction fared better in SST compared to CBT. Specifically, those who reported less success with promotion-focused means of goal attainment showed greater symptom improvement with SST. Although depression is conceptualized as primarily involving a hypoactive promotion system (Strauman, 2002), we also examined the effects of prevention focus.

In our depressed sample, the two RFQ scales were uncorrelated, consistent with other studies (e.g., Eddington, Majestic, & Silvia, 2012), and higher intake depression (but not anxiety) was associated with lower promotion focus. Items on the prevention scale are distinctly more focused on recalling childhood experiences (regarding responsibilities and compliance with rules). By contrast, promotion items focus on a more current time frame. Exploratory analyses showed that higher prevention focus was associated with better symptom improvement in CBT compared to SST. Results from three studies by Liberman and colleagues (Liberman, Molden, Idson, & Higgins, 2001) suggested that a stronger prevention focus (assessed using a different measure) was associated with generating fewer alternative hypotheses and explanations in response to laboratory tasks. Thus, one possible explanation for this finding may be that patients with a stronger prevention focus may struggle with cognitions (e.g., catastrophizing and worrying) that underlie efforts to keep bad things from happening. CBT's focus on cognitive restructuring and on generating alternative explanations may be more effective for these patients, leading to greater symptom improvement. Although replication of these results is warranted, an interesting next step in this work would be to determine whether promotion and prevention focus could be used in a treatment matching context with SST and CBT.

Our second measure of self-regulatory dysfunction was goal adjustment, the ability to flexibly respond to problems in goal pursuit by disengaging from unsuccessful goals and reengaging in new ones. Our results showed that people with difficulties reengaging in new

goals when progress is stalled showed greater improvement in SST compared to CBT. Individual differences in disengagement, however, had no differential impact on treatment outcome. These findings likely reflect the primary focus in SST: encouraging patients to identify and make progress toward new, promotion-focused goals. While helping patients let go of unattainable goals may be a useful self-regulatory intervention, depressed patients often have already given up on many of their personal goals (adaptively or not), making it a less prominent feature of the treatment. In a large sample of college students, Eddington (2013) found mean scores on the GAS-D and GAS-R of 9.7 (*SD* = 3.0) and 21.7 (*SD* = 3.7), respectively. In the current clinical sample, GAS-D scores were somewhat higher and GAS-R scores were somewhat lower. The higher GAS-D scores in the current sample raise questions about whether goal disengagement, originally viewed as an adaptive aspect of self-regulation, may become more akin to anhedonia or premature "giving up" in the context of depression. Given the importance of goal adjustment in the dynamic process of self-regulation, future research should examine the possibility of both adaptive and maladaptive components of goal disengagement and reengagement, perhaps through the development of more comprehensive measures of these constructs.

A limitation of the study is the reliance on patients' self-reported symptoms. Although we used one of the most common measures of depressive symptom severity, the BDI-II, which enables easy comparison with other studies, concerns about the validity of frequent (weekly) administrations of the BDI-II at least in nonclinical populations have been noted (Longwell & Truax, 2005). Weekly clinician evaluations of symptoms would be helpful in this regard, but feasibility is a major concern given that such evaluations would have to be done by blind assessors. A second limitation concerns the generalizability of the results given that our sample was somewhat restricted in terms of education level and gender. A high proportion of female participants is not uncommon in depression treatment studies (Hollon, DeRubeis, Evans, & Wiemer, 1992; Merrill et al., 2003), but the effectiveness of SST for patients with less education remains to be examined.

In summary, this study has shown that SST is an effective treatment for depression, particularly for those patients whose depression prominently features problems in self-regulation, suggesting that matching patients to treatment based on self-regulatory features may enhance outcomes. The developers of SST maintain that the treatment is flexible, guided primarily by a set of core principles and overarching treatment goals related to basic theoretical and empirical work on self-regulation (Vieth et al., 2003). This integrative approach to treatment development is a much-needed shift in the field and parallels recent developments in the transdiagnostic conceptualization of psychopathology and treatment (e.g., Barlow, Allen, & Choate, 2004). Our data further suggest that SST was equally effective in reducing symptoms of depression and anxiety, suggesting that it may lead to the development of skills that are useful for a broader range of presenting problems. Although this study proposes that patients with self-regulatory deficits gained skills in SST to compensate for those deficits, this assertion was not directly tested.

A next important step in this line of research is to determine the extent to which the mechanisms of action underlying SST are different from those in other treatment approaches. Although the two treatments were clearly distinguishable in the behaviors of the

therapists delivering them, adherence data do not necessarily demonstrate differential mechanisms of action. More direct measurement of outcomes related to the treatment targets (i.e., cognitions in CBT and goal pursuit in SST), rather than symptoms per se, over the course of therapy are necessary to address that issue. In addition, studies that examine changes in neural processing of treatment-relevant material (e.g., changes in goal responsiveness) with therapy would be a useful next step in ascertaining differential mechanisms of change.

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Public Health Significance Statement: The results from this study provide corroborating evidence that self-system therapy is an effective short-term treatment for adult patients with depression at varying levels of severity. For those patients with deficits in self-regulation, self-system therapy was shown to be more effective than cognitive behavioral therapy.

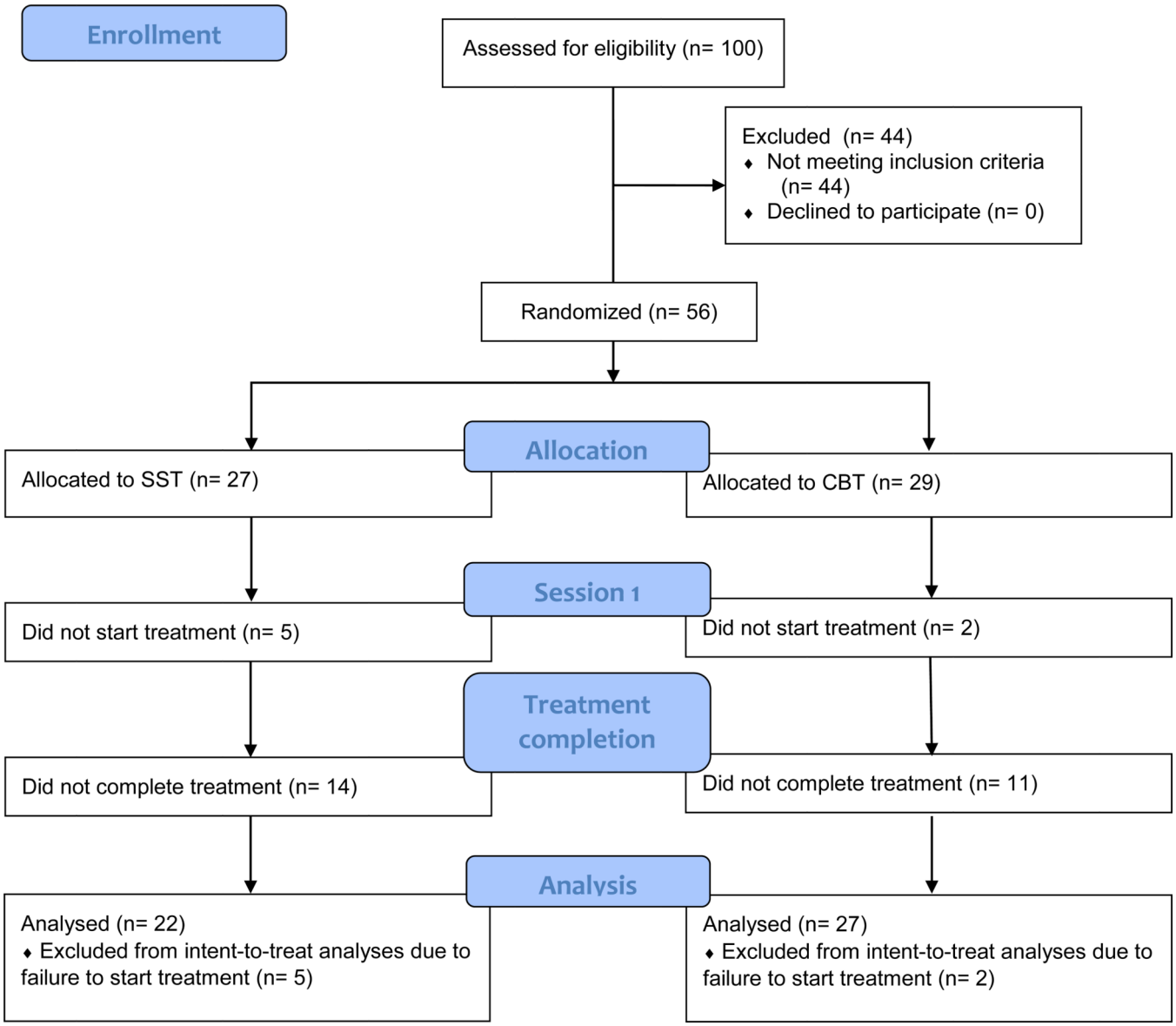

Figure 1.
Enrollment flow diagram

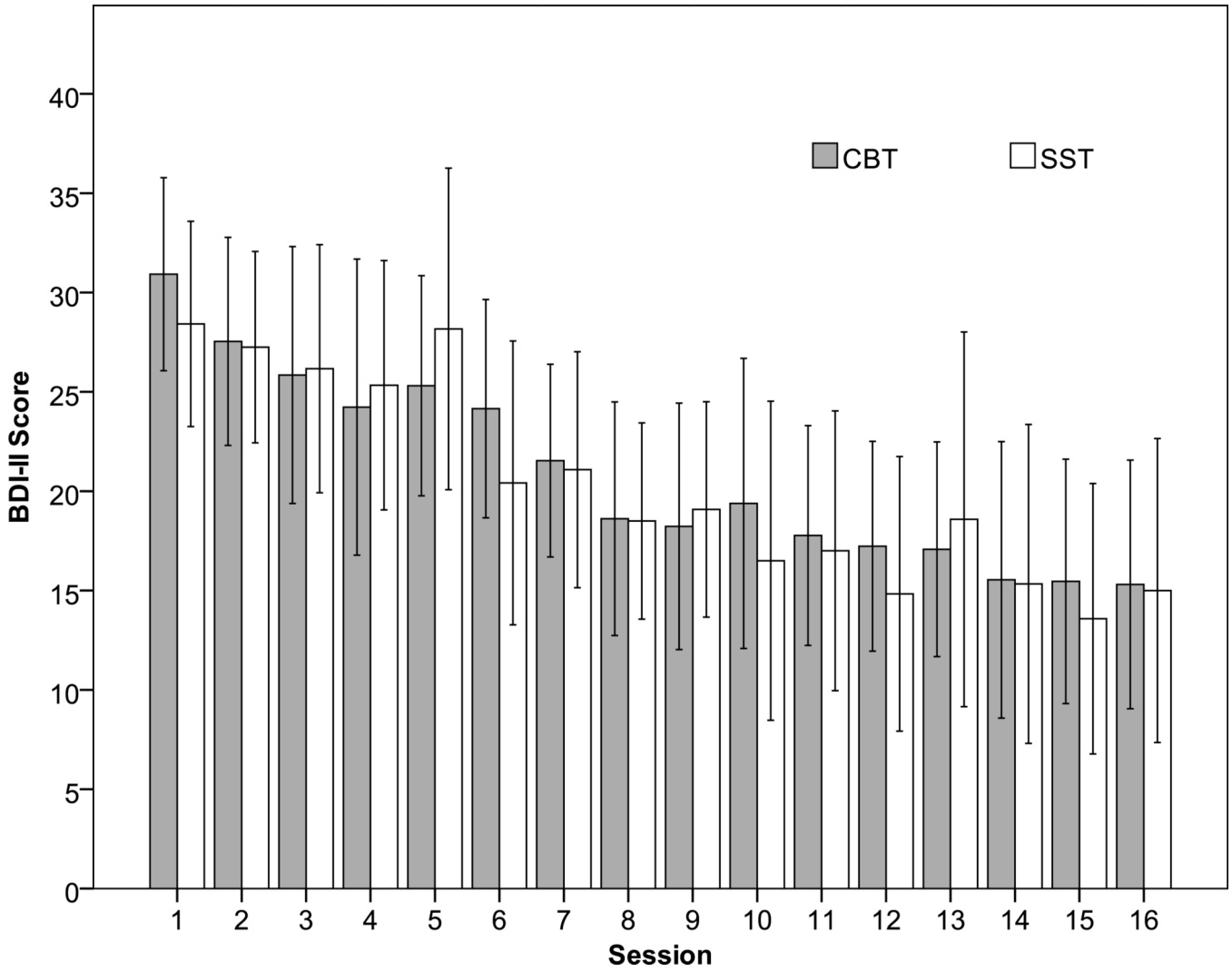

Figure 2.
Weekly BDI-II levels by treatment condition

Note. Error bars represent 95% between-person confidence intervals.

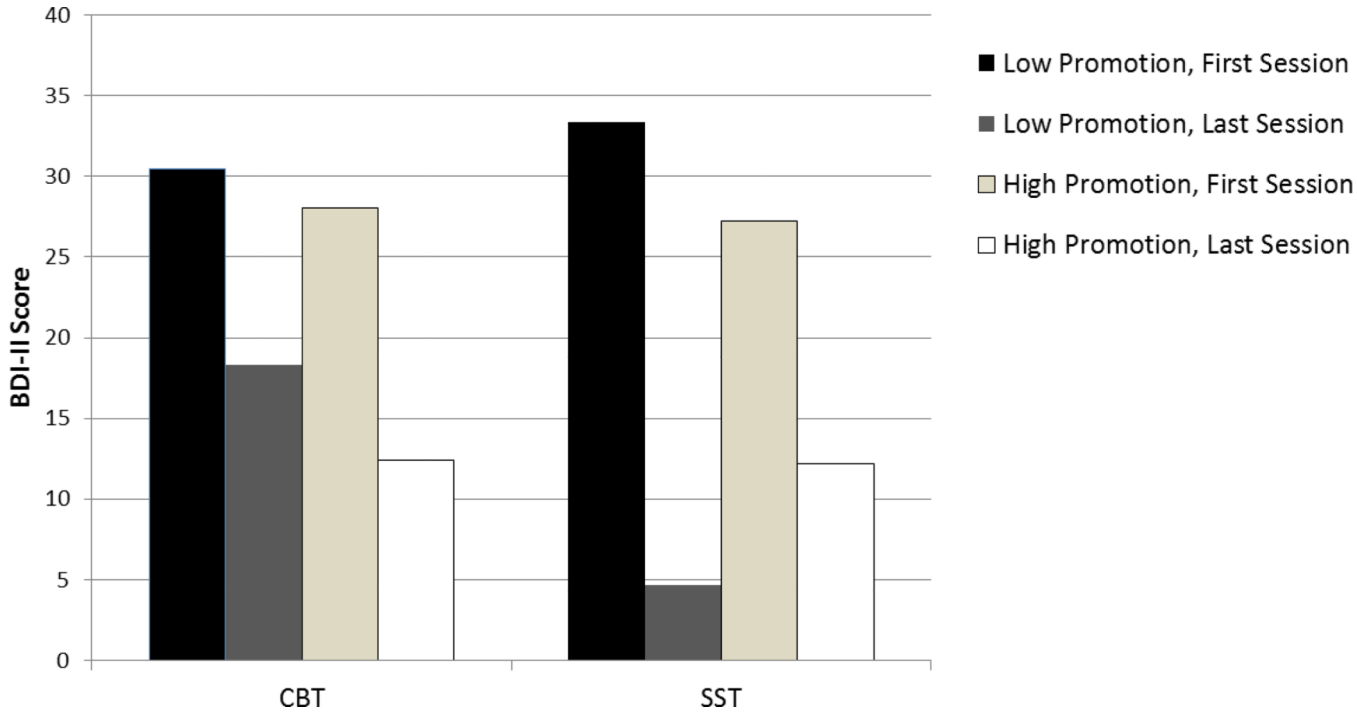

Figure 3.

Moderating effect of promotion focus on change in BDI-II scores for CBT and SST.

Note. The effects are estimated for sessions 1 and 16 for the 25% and 75% values of promotion focus.

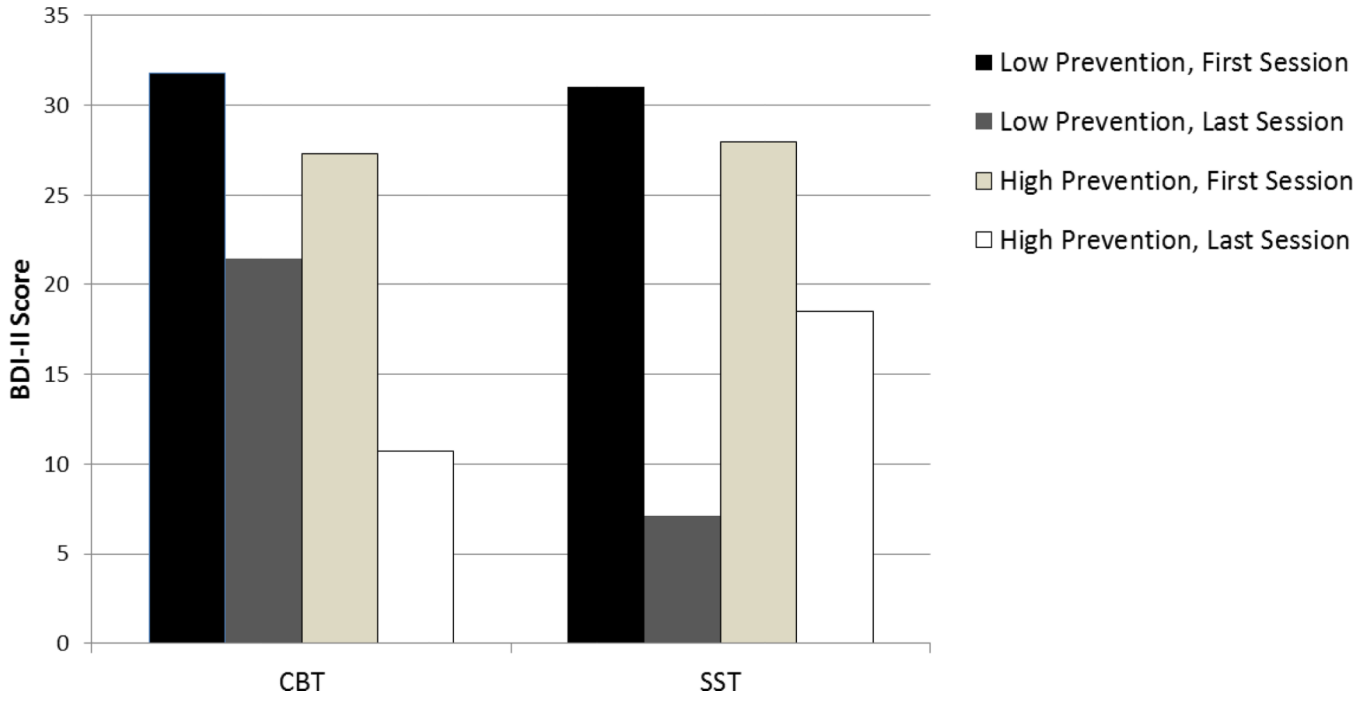

Figure 4.

Moderating effect of prevention focus on change in BDI-II scores for CBT and SST. **Note**. The effects are estimated for sessions 1 and 16 for the 25% and 75% values of prevention focus.

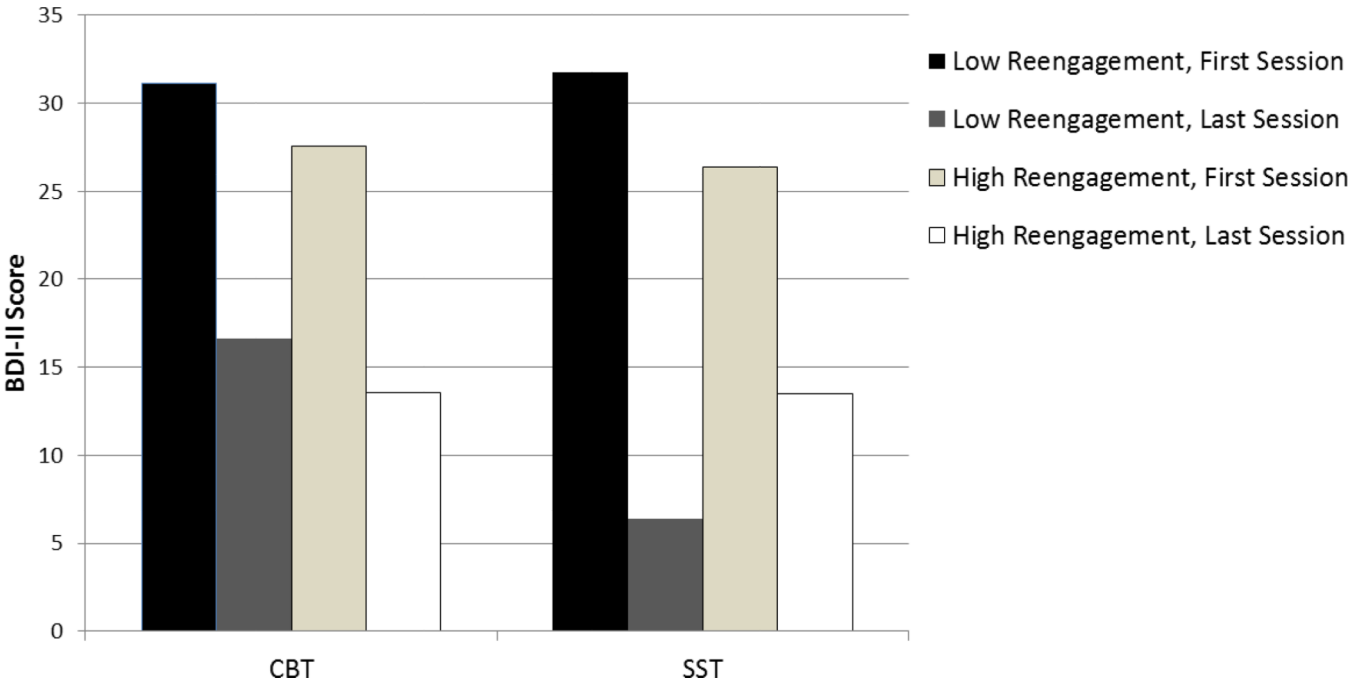

Figure 5.

Moderating effect of goal reengagement on change in BDI scores for CBT and SST.

Note. The effects are estimated for sessions 1 and 16 for the 25% and 75% values of goal reengagement.

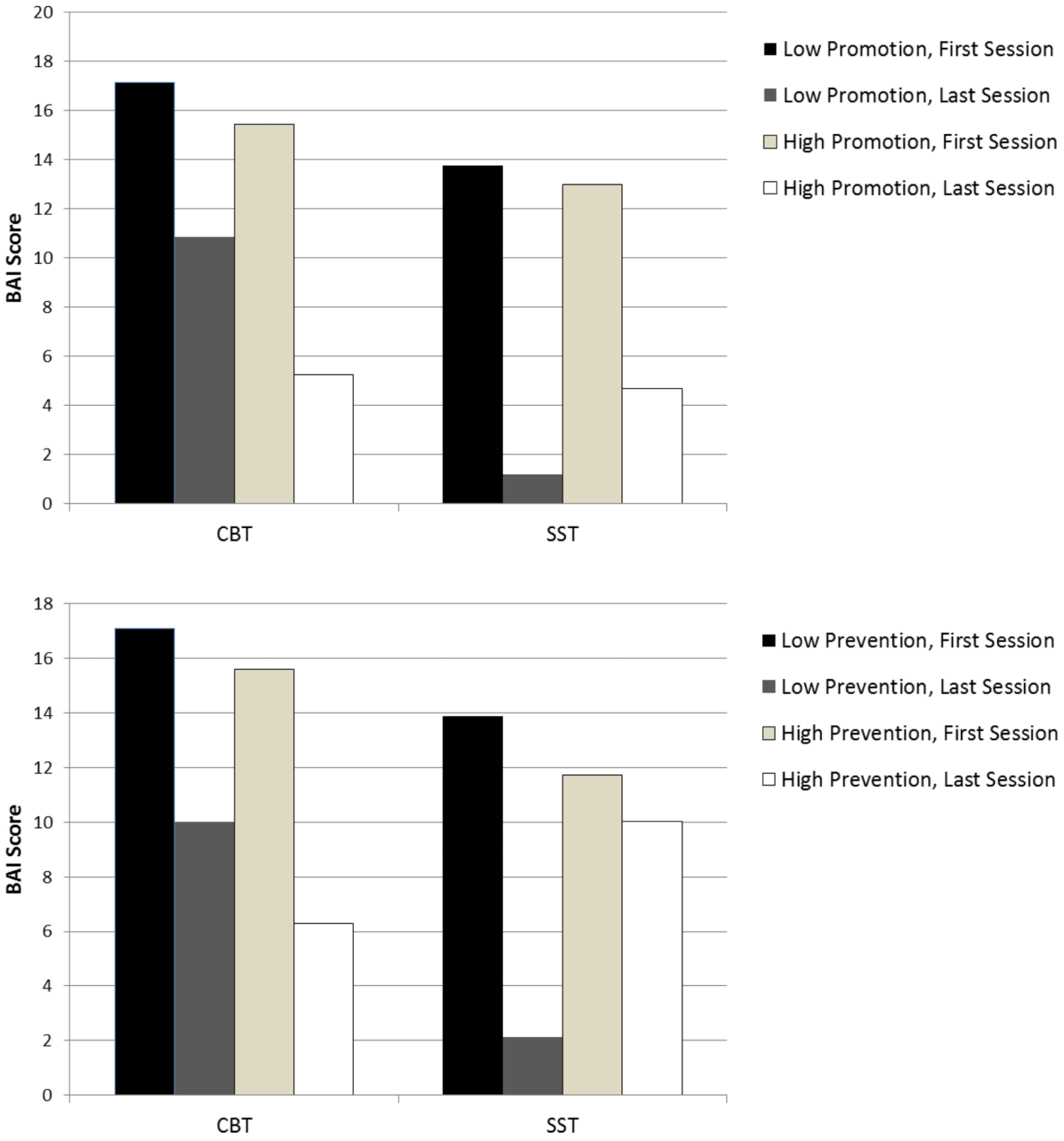

Figure 6.

Moderating effects of promotion and prevention orientations on anxiety (BAI) change.

Note. The effects are estimated for sessions 1 and 16 for the 25% and 75% values of promotion and prevention focus.

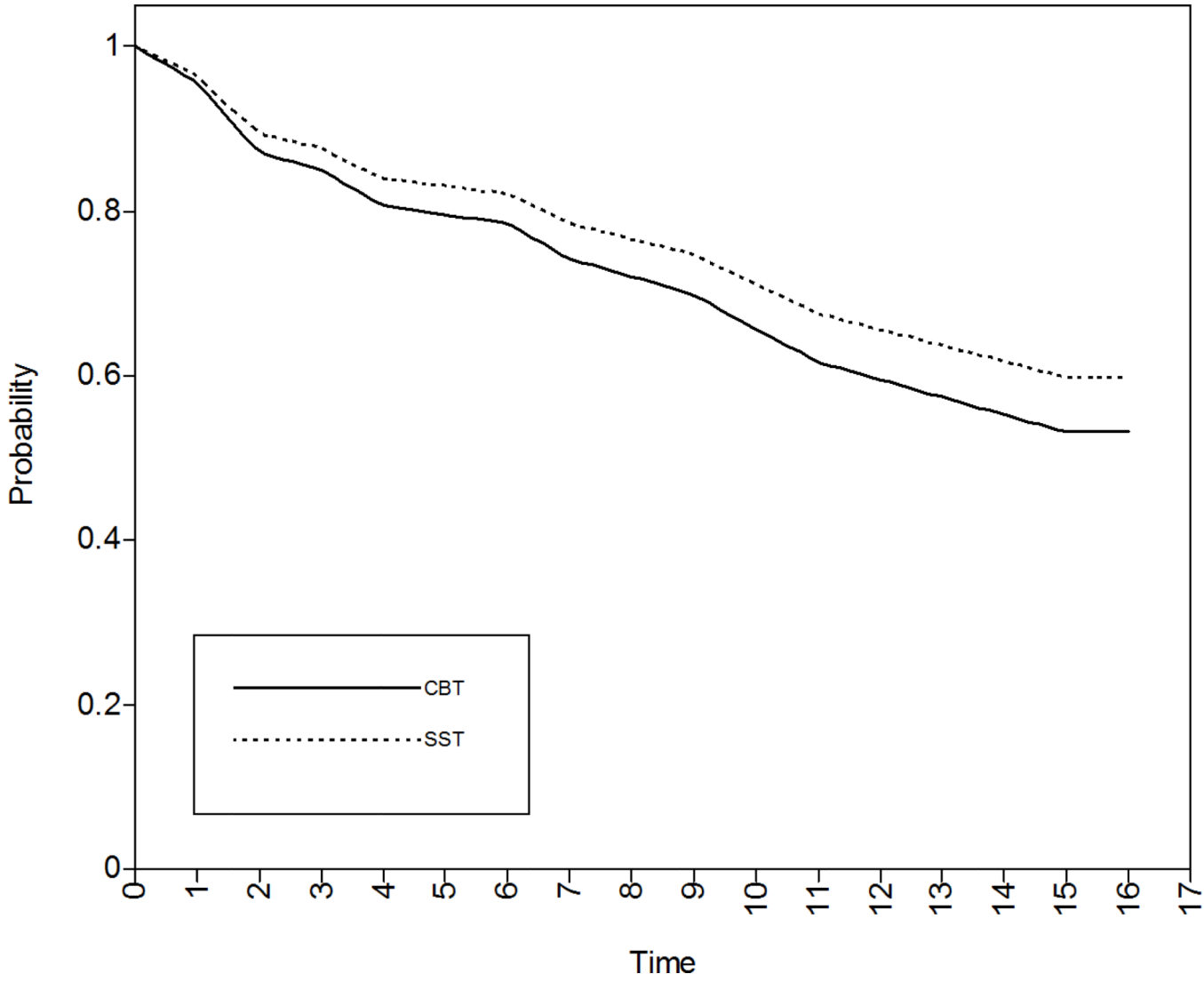

Figure 7.
Estimated probability of remaining in therapy for the CBT and SST groups.

Table 1

Sample Demographics and Intake Diagnostic and Severity Data By Condition.

	SST (n = 22)	CBT (n = 27)	Statistic	*p*-value
Age (*M, SD*)	37.86 (12.94)	37.89 (13.22)	$t(47) = 0.01$	*.995*
Sex	81.8% female	85.2% female	Fisher's exact test	*1.00*
Highest level of education			$X^2(2) = 0.53$	*.770*
High school or below	22.7%	14.8%		
Some college or college degree	59.1%	66.6%		
Some graduate school or advanced degree	18.1%	18.5%		
Employment status	68.2% employed	70.4% employed	$X^2(1) = 0.03$	*.869*
Marital status			$X^2(3) = 1.72$	*.633*
Married	27.3%	25.9%		
Divorced	22.7%	22.2%		
Separated	00.0%	7.4%		
Never married	50.0%	44.4%		
Diagnostic status				
Primary MDD	100%	92.6%	Fisher's exact test	*.495*
Dysthymia	13.6%	40.7%	$X^2(1) = 4.36$	*.037**
Secondary anxiety disorder	56.3%	72.7%	$X^2(1) = 1.16$	*.282*
Past substance abuse	13.6%	7.4%	Fisher's exact test	*.646*
Treatment history				
Past therapy	81.8%	92.6%	Fisher's exact test	*.388*
Past medications	36.4%	40.7%	$X^2(1) = 0.10$	*.754*
Past hospitalization	31.8%	29.6%	$X^2(1) = 0.03$	*.869*
BDI-II score at intake (*M, SD*)	35.68 (10.06)	33.70 (7.01)	$t(47) = 1.35$	*.185*
BAI score at intake (*M, SD*)	19.91 (13.35)	25.56 (15.53)	$t(47) = -0.81$	*.422*

* $p < .05$

Table 2

Descriptive Statistics and Correlations for Self-regulatory Variables and Symptom Severity at Intake

Variable	M	SD	Min, Max	1	2	3	4	5	6	7
1. Treatment condition	.45	.50	0, 1	1						
2. BDI-II (Intake)	34.59	8.48	22, 56	.12	1					
3. BAI (Intake)	23.02	14.72	1, 57	−.19	.44*	1				
4.Promotion Orientation	18.63	4.30	6, 27	−.04	−.30*	−.07	1			
5. Prevention Orientation	19.69	4.83	6, 30	−.21	−.27	−.22	.04	1		
6. Goal Disengagement	11.90	3.43	4, 20	.16	−.23	−.17	−.17	−.07	1	
7. Goal Reengagement	19.67	3.83	7, 25	−.16	−.28*	.17	.45*	.33*	−.17	1

Note. For the treatment condition variable, 0 = CBT and 1 = SST. Correlations with an asterisk are significant, $p < .05$.

Table 3

Multilevel Model for Change in BDI-II Scores Across Sessions and Treatment Conditions

Fixed Effects	**Estimate**	**Standard Error**	***t***	***P***
Intercept	30.82	1.39	22.10	< .001
Session: Linear	−1.75	.34	5.07	< .001
Session: Quadratic	.04	.02	2.26	.028
Random Effects	**Variance Component**	***df***	**Chi square**	***p***
L2: Intercept	82.95	42	270.27	< .001
L2: Linear Slope	3.70	42	117.41	< .001
L2: Quadratic Slope	.01	42	90.22	< .001
L1: Residual	27.70			
Model Fit	**Deviance**	**Parameters**	**AIC**	**BIC**
	3885.30	7	3899.30	3929.93

Note. *N* = 49 participants (Level 2), 587 observed sessions (Level 1; 16 maximum per participant). Session is coded 0 (first week) to 15 in increments of 1. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion.

Table 4

Multilevel Model for Change in Depression (BDI-II) Scores as Moderated by Treatment, Promotion, and Prevention Factors

	Treatment Condition Model				**Condition & Promotion Focus Model**				**Condition & Prevention Focus Model**			
Fixed Effects	**Estimate**	**Standard Error**	***t***	***p***	**Estimate**	**Standard Error**	***t***	***p***	**Estimate**	**Standard Error**	***t***	***p***
Intercept	29.15	1.72	16.93	< .001	29.18	1.67	17.51	< .001	29.77	1.62	18.41	< .001
Session: Linear	−.95	.20	4.83	< .001	−.93	.20	4.55	< .001	−.88	.23	3.87	< .001
Condition	1.16	2.76	.42	.676	.97	2.46	.40	.694	−.12	2.70	.04	.965
Condition x Session	−.49	.35	1.42	.164	−.50	.30	1.65	.105	−.28	.31	.91	.37
Promotion					−2.07	2.06	1.00	.321				
Promotion x Session					−.20	.20	.99	.324				
Promotion x Treatment					−3.22	2.68	1.20	.236				
Promotion x Session x Treatment					.99	.31	3.22	.002				
Prevention									−3.61	1.31	2.76	.008
Prevention x Session									−.33	.21	1.58	.122
Prevention x Treatment									1.16	2.59	.45	.657
Prevention x Session x Treatment									1.11	.26	4.21	< .001
Random Effects	**Variance Component**	***df***	**Chi square**	***p***	**Variance Component**	***df***	**Chi square**	***p***	**Variance Component**	***df***	**Chi square**	***p***
Intercept (μ_0)	84.99	45	472.17	< .001	72.92	43	401.78	< .001	77.66	43	406.19	< .001
Linear Slope (μ_1)	1.07	45	343.68	< .001	.79	43	280.04	< .001	.82	43	271.43	< .001
L1 Residual (r_{ij})	30.49				30.45				30.45			
Model Fit	**Deviance**	**Parameters**	**AIC**	**BIC**	**Deviance**	**Parameters**	**AIC**	**BIC**	**Deviance**	**Parameters**	**AIC**	**BIC**
	3905.21	4	3913.21	3930.71	3883.59	4	3891.59	3909.09	3883.17	4	3891.17	3908.67

Note. n = 49 participants (Level 2), 587 observed sessions (Level 1; 16 maximum per participant). Session is coded 0 (first week) to 15 in increments of 1. Condition is coded 0 = CBT, 1 = SST. Prevention and Promotion scores are standardized. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion.

Table 5

Multilevel Model for Change in Depression (BDI-II) Scores as Moderated by Treatment, Goal Disengagement, and Goal Reengagement Factors

	Condition & Goal Disengagement Model				**Condition & Goal Reengagement Model**			
Fixed Effects	**Estimate**	**Standard Error**	***t***	***p***	**Estimate**	**Standard Error**	***t***	***p***
Intercept	29.22	1.74	16.79	< .001	29.63	1.57	18.88	< .001
Session: Linear	−.96	.21	4.61	< .001	−.95	.20	4.75	<.001
Condition	.97	2.86	.34	.737	−.14	2.40	.06	.953
Condition x Session	−.48	.37	1.32	.193	−.39	.29	1.33	.191
Disengagement	.52	1.70	.31	.760				
Disengagement x Session	−.05	.24	.23	.822				
Disengagement x Treatment	.29	2.92	.10	.921				
Disengagement x Session x Treatment	.03	.40	.06	.951				
Reengagement					−3.44	1.48	2.32	.025
Reengagement x Session					.03	.20	.16	.874
Reengagement x Treatment					−1.69	2.46	.69	.496
Reengagement x Session x Treatment					.76	.27	2.84	.007
Random Effects	**Variance Component**	***df***	**Chi square**	***p***	**Variance Component**	***df***	**Chi square**	***p***
Intercept (μ_0)	88.74	43	468.55	< .001	69.98	43	383.10	< .001
Linear Slope (μ_1)	1.15	43	360.88	< .001	.75	43	259.84	< .001
L1 Residual (r_{ij})	30.47				30.49			
Model Fit	**Deviance**	**Parameters**	**AIC**	**BIC**	**Deviance**	**Parameters**	**AIC**	**BIC**
	3900.61	4	3908.61	3926.11	3882.48	4	3890.48	3907.98

Note. *n* = 49 participants (Level 2), 587 observed sessions (Level 1; 16 maximum per participant). Session is coded 0 (first week) to 15 in increments of 1. Condition is coded 0 = CBT, 1 = SST. Goal Disengagement and Goal Reengagement are standardized. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion.

Table 6

Multilevel Model for Change in Anxiety (BAI) Scores as Moderated by Treatment, Promotion, and Prevention Factors

	Treatment Condition Model				Condition & Promotion Focus Model				Condition & Prevention Focus Model			
Fixed Effects	**Estimate**	**Standard Error**	***t***	***p***	**Estimate**	**Standard Error**	***t***	***p***	**Estimate**	**Standard Error**	***t***	***p***
Intercept	16.24	1.85	8.79	< .001	16.24	1.87	8.70	< .001	16.43	1.80	9.13	< .001
Session: Linear	−.58	.14	4.01	< .001	−.55	.15	3.67	.001	−.53	.16	3.43	.001
Condition	−2.89	2.58	1.12	.268	−2.87	2.58	1.11	.271	−3.53	2.54	1.39	.171
Condition x Session	−.10	.23	.46	.648	−.13	.22	.60	.549	.06	.19	.30	.766
Promotion					−1.47	2.39	.62	.542				
Promotion x Session					−.22	.17	1.35	.185				
Promotion x Treatment					.79	2.96	.27	.789				
Promotion x Session x Treatment					.47	.21	2.18	.034				
Prevention									−1.22	1.85	.66	.512
Prevention x Session									−.12	.15	.77	.446
Prevention x Treatment									−.51	3.19	.16	.874
Prevention x Session x Treatment									.66	.28	2.38	.022
Random Effects	**Variance Component**	***df***	**Chi square**	***p***	**Variance Component**	***df***	**Chi square**	***p***	**Variance Component**	***df***	**Chi square**	***p***
Intercept (μ_0)	80.13	45	595.41	< .001	83.29	43	401.78	< .001	80.64	43	574.12	< .001
Linear Slope (μ_1)	.45	45	233.16	< .001	.46	43	280.04	< .001	.34	43	189.68	< .001
L1 Residual (r_{ij})	20.51				20.52				20.45			
Model Fit	**Deviance**	**Parameters**	**AIC**	**BIC**	**Deviance**	**Parameters**	**AIC**	**BIC**	**Deviance**	**Parameters**	**AIC**	**BIC**
	3671.89	4	3679.89	3697.39	3663.67	4	3671.08	3689.17	3657.81	4	3665.81	3683.31

Note. *n* = 49 participants (Level 2), 587 observed sessions (Level 1; 16 maximum per participant). Session is coded 0 (first week) to 15 in increments of 1. Condition is coded 0 = CBT, 1 = SST. Prevention and Promotion scores are standardized. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion.