Cognitive Change as a Predictor of Session-to-Session Symptom Change in Cognitive Therapy for Depression

Undergraduate Honors Research Thesis

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Olivia Fitzpatrick

The Ohio State University
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Project Advisor: Dr. Daniel R. Strunk, Department of Psychology

Abstract

Studies of cognitive therapy for depression (CT) suggest that decreases in negative cognitions coincide with reductions in depressive symptoms over the course of treatment. Although these results are consistent with the theory that cognitive change is responsible for therapeutic gains, the timing of such assessments has precluded establishing cognitive change as a predictor of subsequent symptom reduction. To test cognitive change as a predictor of symptom change, we examined patient-reported cognitive change observed during (immediate cognitive change; CC-I) and between (delayed cognitive change; CC-D) therapy sessions as predictors of symptom reduction across sessions 1 through 5 in CT. Additionally, we explored if these potential predictive relations vary according to patients' pretreatment maladaptive personality traits and interpersonal problems and functioning. To further understand the function of cognitive change in CT, we also assessed CC-I as a predictor of session-to-session CC-D across these sessions of interest. A total of 126 adults with major depressive disorder participated in 16 weeks of CT. CC-I was evaluated immediately after each session, and CC-D and depressive symptoms were assessed before each session. To rule out stable patient characteristics as potential confounds, we disaggregated the within- and between-patient effects of cognitive change scores and focused on the within-patient effects as predictors. Within-patient CC-I significantly predicted subsequent CC-D, and within-patient CC-D significantly predicted subsequent symptom change. Within-patient CC-I did not significantly predict session-to-session symptom change. Interestingly, the relation of within-patient CC-I and symptom change was significantly moderated by patient maladaptive personality traits and interpersonal problems, whereas interpersonal functioning significantly moderated the relation of within-patient CC-D and symptom change. These results suggest that cognitive changes observed during therapy sessions predict additional cognitive change between sessions, which ultimately produce subsequent depressive symptom reduction.

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Chapter 1: Introduction

Cognitive therapy (CT) is a structured, collaborative psychotherapy that has been shown to be an efficacious treatment for depression in a number of randomized trials (Strunk & DeRubeis, 2001). These trials have often relied on medication as a comparison condition and have demonstrated that CT produces outcomes comparable to those of pharmacotherapy during the acute phase of treatment (DeRubeis et al., 2005; Elkin et al., 1995; Hollon & Dimidjian, 2008; Robinson et al., 1990). Evidence also suggests that CT generates enduring effects similar to those of continued medication (DeRubeis, Gelfand, Tang, & Simons, 1999; Strunk & DeRubeis, 2001) and that patients treated with CT are less likely to relapse when compared to patients for whom medication was discontinued following successful treatment (Hollon et al., 2005; Hollon, Stewart, & Strunk, 2006).

A key principle of CT posits that depressed patients have negative thoughts and beliefs that adversely impact their emotions and behaviors (Beck & Dozois, 2011; Beck, Rush & Shaw, 1979). These distorted cognitions span from automatic negative thoughts to maladaptive schemas. Schemas are structured belief systems that facilitate the storage of information, such as perceptions of self, and may contribute to psychological disorders if they contain rigid, negative biases (Beck & Dozois, 2011). With this in mind, the aim of CT is to provide patients with cognitive strategies that promote the independent recognition, assessment, and modification of negative thoughts and beliefs. According to cognitive theories, this process of modification reflects the essential therapeutic mechanism of cognitive change that ultimately promotes reductions in depressive symptoms during CT (Beck & Dozois, 2011).

The Role of Cognitive Change in Treatment for Depression

Although cognitive change is generally considered to be a factor in CT, its precise role in reducing symptoms during treatment is a point of contention. The original cognitive model suggests that changes in depressive cognitive content, such as dysfunctional attitudes and negative automatic thoughts, are responsible for symptom reductions across all forms of treatment for depression, including CT (Beck, Rush & Shaw, 1979). In line with this theory, numerous studies have shown that various therapies, such as CT and interpersonal psychotherapy, reduce both depressive cognitive content and symptoms, and that cognitive change is associated with reduced depressive symptoms over the course of treatment (Barber & DeRubeis, 2001; Bieling, Beck, & Brown, 2004; Christopher, Jacob, Neuhaus, Neary, & Fiola, 2009; Cristea et al., 2015; Quilty, McBride, & Bagby, 2008; Vittengl, Clark, Thase, & Jarrett, 2014; Westra, Dozois, & Boardman, 2002).

The original cognitive model has been questioned in part because it has been inaccurately interpreted to posit that cognitive change can *only* impact symptoms during CT, rather than across treatments (Lorenzo-Luaces, German, & DeRubeis, 2015). Researchers making this assumption have argued that if the impact of cognitive change on symptoms were unique to those treatments that rely heavily on cognitive procedures, CT would likely produce cognitive change and subsequent symptom change more than non-cognitive treatments (Lorenzo-Luaces, German, & DeRubeis, 2015). As research has not consistently demonstrated statistically significant differences between treatment outcome in cognitive and non-cognitive therapies (Cristea et al., 2015; Beevers & Miller, 2004; Elkin, et al., 1989; Teasdale et al., 2001; Warmerdam et al., 2008), it has been inferred that cognitive change is not the therapeutic mechanism by which CT achieves its effects (Hayes, 2004; Kazdin, 2009; Longmore & Worrell,

2007). However, according to Lorenzo-Luaces, German, & DeRubeis (2015), such conclusions are not warranted based on current evidence. Although the lack of statistically significant differences between treatments could indicate that different treatments work through different mechanisms that are equally effective in reducing symptoms, these findings could also be accounted for by the original cognitive model, showing that treatments focusing on changing cognitions and those addressing other constructs both achieve their effects through the same mechanism of cognitive change (Lorenzo-Luaces, German, & DeRubeis, 2015).

This evidence, in conjunction with research illustrating the covariance between cognitive change and symptom change, provides important information on the role of cognitive change in CT. However, the designs of previous studies do not allow researchers to rule out potential temporal confounds. Specifically, investigating cognitive and symptom change from pre-treatment to post-treatment leaves open the possibility that an observed relation could be attributed to the presumed causal effect (i.e., cognitive change produces symptom change) or a reverse causal effect (i.e., symptom change produces cognitive change). For instance, Burns and Spangler (2000) found a correlation between changes in depressive symptoms and dysfunctional attitudes in CT from pre-treatment to post-treatment but, given the timing of assessments, were unable to determine the directionality of the relation. Similarly, Vittengl, Clark, Thase, and Jarrett (2014) found that cognitive content and depressive symptoms concurrently improved over the course of treatment; however, evidence for cognitive change predicting subsequent symptom reduction was limited, perhaps because of the spacing of assessments. Given this timeline issue, such findings are necessary but not sufficient to conclusively establish cognitive change as a mechanism that causes depressive symptom reduction (Lorenzo-Luaces, German, & DeRubeis, 2015). In order to mitigate this problem and test cognitive change as a predictor of subsequent

symptom improvement, researchers must examine cognitive change during periods when this modification has occurred but the resulting impact on symptoms has yet to be observed.

One approach to finding such assessment points has been that of Tang and DeRubeis (1999). In this study, the authors identified sessions in which patients reported sudden gains, or large and lasting improvements in their depressive symptoms, and then examined which factors distinguished the sessions preceding these gains. Interestingly, they found that substantial cognitive changes observed during a given session predicted sudden gains, and that this substantial cognitive change was observed only during the session directly preceding sudden gains (i.e., pre-gain session) and not during the session prior to the pre-gain session, which acted as a within-patient control session (i.e., pre-pre-gain session). To determine if the identified relation of cognitive changes and sudden gains impacted the level of cognitive change observed during later sessions, Tang and DeRubeis then examined cognitive changes that occurred during the session directly following sessions in which patients reported sudden gains (i.e., after-gain sessions). This analysis revealed that, for patients who experienced sudden gains, the amount of cognitive change observed during after-gain sessions was higher than that in pre-pre-gain sessions. With this information in mind, Tang and DeRubeis theorized that cognitive changes occurring during pre-gain sessions generated symptom improvement in the form of sudden gains before the following session, which consequently produced more cognitive changes at the after-gain session. According to Tang and DeRubeis, this "upward spiral" ultimately led to greater treatment responsiveness. Consistent with this view, they found that patients who experienced sudden gains reported fewer depressive symptoms both at post-treatment and at 18-month follow-up compared to patients who did not experience sudden gains, and these results were replicated by Tang, DeRubeis, Beberman, and Pham (2005). Although the cognitive changes that

produce sudden gains appear to be important, they may be markedly distinct from those observed during a majority of therapy sessions. In order to clarify if cognitive change is responsible for treatment outcome, its role in producing symptom change should be examined continuously across a phase of treatment.

Methods of Assessing Cognitive Change

In previous research, the most common method of assessing cognitive change has been through self-report measures that prompt respondents to endorse specific depressive cognitions in general or the past week (e.g., Christopher, Jacob, Neuhaus, Neary, & Fiola, 2009; DeRubeis, Evans, Hollon, Garvey, Grove, & Tuason, 1990; Segal, Gemar, & Williams, 1999; Vittengl, Clark, Thase, & Jarrett, 2014). Important among these instruments are the Automatic Thought Questionnaire (ATQ; Hollon & Kendall, 1980), the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), and the Attributional Style Questionnaire (ASQ; Seligman, Abramson, Semmel, & von Baeyer, 1979). All three of these measures have been shown to be reliable, valid evaluations of depressive cognitions that distinguish clinically depressed from non-depressed individuals (Hollon & Kendall, 1980; Seligman, Abramson, Semmel, & von Baeyer, 1979; Weissman & Beck, 1978).

Despite the advantages of these instruments, they are limited to assessing specific forms of cognitive change. In addition, these measures are long enough to become burdensome to patients if administered frequently (i.e., during each therapy session). For instance, the ATQ is a 30-item questionnaire that is restricted to measuring the frequency of the occurrence of automatic negative thoughts that commonly accompany depression over the past week (Hollon & Kendall, 1980). The DAS is a collection of 40 statements that specifically aims to tap into the fundamental attitudes and assumptions of people with depression (Weissman & Beck, 1978).

Similarly, the ASQ was devised to exclusively measure changes in a construct known as attributional style, which involves the manner in which patients explain certain situations in their lives (Seligman, Abramson, Semmel, & von Baeyer, 1979).

Relatedly, previous evaluations of cognitive change have focused on either changes observed during therapy sessions (e.g., Tang & DeRubeis, 1999) or those experienced outside of therapy sessions (e.g., Christopher, Jacob, Neuhaus, Neary, & Fiola, 2009; Vittengl, Clark, Thase, & Jarrett, 2014). Consequently, these approaches have precluded examinations of the potential connection between cognitive changes observed during and between therapy sessions. More specifically, cognitive changes experienced during and after CT sessions, as well as independent CT skill use outside of sessions, may be interconnected and play key roles in promoting positive treatment outcome. In line with this theory, evidence suggests that understanding and independent rehearsal of the strategies taught during sessions predict lower risk of relapse after successful treatment (Strunk, DeRubeis, Chiu, & Alvarez, 2007). Relatedly, research indicates that cognitive changes experienced during CT can produce symptom reductions (Tang & DeRubeis, 1999). With this evidence in mind, it is possible that the cognitive changes a patient experiences during a therapy session are related to that patient's independent use of CT skills and experience of additional cognitive changes following the session.

To test this possibility and address the limitations of commonly used assessments of cognitive change, the current study involves two measures intended to capture a wide variety of cognitive changes observed during and between therapy sessions. The Assessment of Immediate Cognitive Change (CC-I) is a five-item self-report instrument designed to measure cognitive changes observed during a given therapy session. The Assessment of Delayed Cognitive Change (CC-D) is a nine-item self-report instrument designed to measure cognitive changes experienced

by patients between sessions. In addition, items of the CC-D were also intended to capture patients' independent rehearsal of cognitive skills, which are taught in CT sessions. Rather than assessing specific thoughts and beliefs endorsed by patients, these questionnaires are designed to evaluate a wide variety of cognitive change and therefore may assess many forms of this process variable. Although such an approach may preclude the examination of specific cognitive changes, it can be administered more often and may capture various kinds of cognitive change at key assessment points in treatment. Importantly, administering both measures enabled us to conduct a fine-grained assessment of cognitive change, in which immediate cognitive changes were examined as predictors of subsequent independent CT skill rehearsal and ongoing cognitive changes.

Disaggregation of Within-Patient & Between-Patient Effects of Cognitive Change

To our knowledge, previous research examining the relations of interest has focused solely on the impact of raw cognitive change on treatment outcome and has thus failed to decompose the within-patient and between-patient variation in this process variable. According to Curran and Bauer (2011), studies involving a repeated measures design allow for the collection of rich data that enable the use of several advanced statistical methods, including the disaggregation of within- and between-patient effects in a variable that changes over time (i.e., cognitive change) on an outcome (i.e., treatment outcome). Disaggregating within- and between-patient variability is important because the relation of between-patient variation and outcome could be accounted for by confounding stable patient characteristics, rather than a true causal effect (Sasso, Strunk, Braun, DeRubeis, & Brotman, 2015). For instance, Sasso and colleagues (2015) suggest that between-patient differences are responsible for the identified predictive relation of a form of therapist adherence (i.e., ability to set and negotiate session agendas, as well

as collaborate with patients) and subsequent symptom change. Relatedly, cognitive change could be shown to predict symptom change because of stable patient characteristics, rather than a true causal effect.

Conversely, models in which between-patient differences have been removed and only the within-patient effects of a process variable are examined rule out the possibility that any identified relation could be attributed to confounding patient characteristics. If high levels of cognitive change truly predict subsequent symptom improvement during treatment, then it is likely that the within-patient variation in cognitive change will significantly predict outcome (Curran & Bauer, 2011). Therefore, concentrating on the predictive relation of the within-patient variability in a process variable and treatment outcome could provide more meaningful information regarding psychological treatment and the mechanisms by which it achieves its effects (Sasso, Strunk, Braun, DeRubeis, & Brotman, 2015). With this in mind, the current study examines the effects of between- and within-patient variability in cognitive change on symptom change, with a primary focus on the within-patient effects of this process variable.

Moderators of the Relation of Cognitive & Symptom Change

In recent years, researchers have become increasingly interested in identifying for which patients particular interventions or clinical strategies are most likely to be useful. It has been proposed that researchers may be able to determine the optimal treatment option for a given patient through the identification of variables that predict differential treatment response (DeRubeis, Gelfand, German, Fournier, & Forand, 2014). As patients with comorbid personality disorders (PDs) and interpersonal vulnerabilities tend to have complex symptom presentations that are difficult to treat (Beck, Broder & Hindman, 2016; Gunderson et al., 2008), personality

pathology has been of interest to researchers examining how patients' course of and responsiveness to treatments for depression differ according to certain characteristics.

The results of these studies have largely suggested that interpersonal vulnerabilities and maladaptive personality traits predict suboptimal outcomes in treatment for depression. Several researchers have found that patient-reported interpersonal problems predict greater depressive symptoms following treatment, while controlling for pretreatment symptom severity (Hardy, Cahill, Shapiro, Barkham, Rees, & Macaskill, 2001; McEvoy, Burgess, & Nathan, 2013). Relatedly, Renner et al. (2012) found that heightened interpersonal distress at pretreatment predicted less robust reductions in depressive symptoms across treatment. High levels of interpersonal problems have also been identified as significant predictors of increased risk for early attrition during CT (McEvoy, Burgess, & Nathan, 2013; 2014). Consistent with these findings, evidence indicates that maladaptive personality traits adversely impact outcomes across treatments for depression (Fournier, DeRubeis, Shelton, Gallop, Amsterdam, & Hollon, 2008; Gorwood, Rouillon, Even, Falissard, Corruble, & Moran, 2010; Joyce, McKenzie, Carter, Rae, Luty, Frampton, & Mulder, 2007; Levenson, Wallace, Fournier, Rucci, & Frank, 2012; Wardenaar, Conradi, Bos, & de Jonge, 2014). A meta-analysis by Newton-Howes, Tyrer, and Johnson (2006), which indicated that comorbid PD with depression was linked to an increased risk for poor treatment outcome, has further supported these findings.

In line with these assessments, it is likely that treatment mechanisms, perhaps including cognitive change, also vary according to individual patient differences. Through their examination of pretreatment patient characteristics as moderators of the relation of three features of therapist adherence (i.e., cognitive methods, negotiating/structuring, behavioral methods/homework) and treatment outcome in CT for depression, Sasso, Strunk, Braun,

DeRubeis, and Brotman (2015) found that patient gender was a significant moderator of the relation between cognitive methods and outcome, and that both anxiety severity and number of prior depressive episodes moderated the relation of behavioral methods/homework and symptom change. Researchers have also found that the association between therapist competence (i.e., interpersonal effectiveness, application of cognitive-behavioral techniques, pacing, etc.) and depressive symptom change in CT is moderated by several patient characteristics, including anxiety, age of disease onset, and chronicity of depression (Strunk, Brotman, DeRubeis, & Hollon, 2010). Consistent with this idea, the strength of the relation of cognitive change and depressive symptom change in CT may also differ systematically across patients with different characteristics. Finding such variability would enable the identification of patients for whom cognitive change is more, and less, important in promoting effective courses of treatment in CT.

The Current Study

The primary aim of the current study is to examine the potential predictive relation of immediate with delayed cognitive changes, the role of these cognitive changes in predicting symptom change, and the extent to which these latter effects vary according to specific patient characteristics across a series of sessions in the early portion of treatment. To our knowledge, this will be the first study to examine the potential relation of immediate cognitive changes (i.e., those observed during a therapy session) and delayed cognitive changes (i.e., those experienced between two sessions) with subsequent symptom change across multiple early CT sessions.

We first examined immediate cognitive change as a predictor of subsequent delayed cognitive change across a series of early sessions. We then investigated the role of these cognitive changes in predicting subsequent symptom changes across sessions 1 through 5. We aimed to understand the function of cognitive change throughout the early phase of CT, during

which symptom improvements tend to be large and variable across patients and sessions (Strunk, Brotman, & DeRubeis, 2010). Furthermore, disaggregating the within- and between-patient variability in immediate and delayed cognitive change and focusing on within-patient effects allowed us to rule out stable patient characteristics as potentially confounding variables. To explore how these relations may vary across patients, we then examined several pretreatment variables as moderators of the predictive relations of interest. These potential moderators included patients' pretreatment maladaptive personality traits, interpersonal problems, and interpersonal functioning.

Our analyses focused on the following main hypotheses: **(1)** within-patient immediate cognitive change will predict subsequent delayed cognitive change before the next session; **(2)** within-patient immediate cognitive change will predict subsequent symptom change at the following session; and **(3)** within-patient delayed cognitive change will predict subsequent symptom change. In analyses for which we did not have specific hypotheses, we explored whether the effects of within-patient immediate and within-patient delayed cognitive change on subsequent symptom change varied as a function of three potential moderating variables (i.e., maladaptive personality traits, interpersonal problems, and interpersonal functioning). Please see Figure 1 for a visual representation of the study design on which these hypotheses are based.

Chapter 2: Methods

Participants

The sample consisted of 126 adults from the Columbus area who agreed to participate in 16 weeks of CT for depression as part of a naturalistic research study. This study was approved by the local Institutional Review Board and, according to DSM-IV-TR (American Psychiatric Association, 2000) criteria, all participants qualified for a primary Axis I diagnosis of Major

Depressive Disorder (MDD). Participants were recruited through online classified ads, social media, flyers, and referrals from health providers. After expressing interest in the study, participants completed an initial phone screen using the DSM-IV-TR criteria for MDD (APA, 2000). In order to be eligible for the study, participants had to meet the following inclusion criteria: (a) diagnosis of MDD, according to DSM-IV-TR criteria (APA, 2000); (b) 18 years of age or older; and (c) able and willing to give informed consent. Exclusion criteria included: (a) any history of bipolar affective disorder (type I or type II), or history of any psychotic disorder; (b) current Axis I disorder other than MDD if it constituted the predominant aspect of the clinical presentation and if it required treatment other than that being offered; (c) subnormal intellectual potential (IQ < 80); (d) evidence of any medical disorder or condition (including pregnancy or risk of pregnancy) that could cause depression; (e) clear indication of secondary gain (e.g., court ordered treatment or compensation issues); (f) current suicide risk or significant intentional self-harm in the last six months sufficient to preclude treatment on an outpatient basis; and (g) history of substance dependence in the past six months. In addition to meeting these criteria, patients previously on medication were asked to maintain a stable dosage over the course of the study. Individuals who appeared to meet these criteria were asked to complete an intake evaluation.

Demographics. The sample consisted predominately of female participants (60%), and 83% of patients identified as Caucasian. Of the remaining participants, 8% were Asian-American, 7% were African-American, and 2% were Hispanic-American. Participant ages ranged from 18-70 years (M = 31.72, SD = 13.35).

Assessors & Therapists

All assessments were administered by five advanced graduate students (3 male, 2 female). Under the supervision of Dr. Daniel Strunk, these assessors also served as the therapists

who provided CT to participants. However, the therapist to whom a patient was ultimately assigned for treatment was always different than his or her intake assessor. Therapists were randomly assigned to patients and followed the procedures outlined in standard manuals of CT for depression (Beck, Rush, Shaw, & Emery, 1979).

Measures

Depressive symptoms.

Major Depressive Disorder. The Structured Clinical Interview for the DSM-IV-TR (SCID-I; First et al., 2002) was utilized during an intake assessment to determine if potential participants met the criteria required for a diagnosis of MDD.

Self-reported depressive symptoms. The Beck Depression Inventory-II, an established and widely used assessment of symptom severity, served as the measure of patient-reported depressive symptoms (BDI-II; Beck, Steer, & Brown, 1996). When completing the BDI-II, participants were prompted to describe how they have been feeling during the past week by rating 21 items (i.e., sadness, agitation, worthlessness, etc.) on a scale from 0 to 3. Possible total scores range from 0 (minimal depression) to 63 (severe depression). The BDI-II was administered prior to the beginning of each therapy session and assessment (i.e., intake, week 4, and post-treatment).

Cognitive change.

Immediate cognitive change. Immediate cognitive change was evaluated following each therapy session through the Assessment of Immediate Cognitive Change (CC-I), a five-item self-report measure. Items included “I caught myself thinking negatively, recognized the negative bias, and reevaluated the situation” and “I noticed myself thinking less negatively” [during this session]. These items were intentionally broad so that various forms of cognitive changes could

be assessed. Patients were asked to indicate how strongly they agreed with each statement in regards to the session that just ended. The rating scale ranged from 0 (Not at all) to 6 (Completely). Potential scores on this measure could range from 0 to 35, with higher scores indicating greater immediate cognitive change. The aim of this instrument was to easily capture the total amount of cognitive change experienced by the patient during the therapy session he or she just completed.

Delayed cognitive change. Delayed cognitive change was assessed prior to the beginning of each therapy session through the Assessment of Delayed Cognitive Change (CC-D), a nine-item self-report instrument. The aim of the CC-D was to capture the cognitive skills utilized and the cognitive change experienced by participants since the previous session. This measure shares three items with the CC-I and includes six other items that are intended to capture cognitive skills that were rehearsed and cognitive changes that occurred during a single between-session interval (e.g., "When I got upset, I took time to step back from a situation and consider that my negative thoughts might be inaccurate"). As with the CC-I, these items were intentionally broad so that various forms of cognitive changes and skill use could be assessed. Patients were asked to indicate how strongly they agreed with each statement in regards to the past week. The rating scale ranged from 0 (Not at all) to 6 (Completely). Potential scores on this measure ranged from 0 to 54, with a higher score indicating greater delayed cognitive change.

Interpersonal vulnerabilities.

Interpersonal problems. Interpersonal problems were assessed during the intake assessment using the brief version of the Inventory of Interpersonal Problems (IIP-32; Barkham, Hardy & Startup, 1996), which aims to capture a range of difficulties that people experience in interpersonal relationships. This measure includes 32 items that are separated into two

subgroups: “things you find hard to do with other people” (e.g., “make friends”) and “things you do too much” (e.g., “I fight with other people too much”). Additionally, there are 8 subscales that capture a variety of interpersonal problems: *hard to be sociable, hard to be assertive, too aggressive, too open, too caring, hard to be supportive, hard to be involved*, and *too dependent*. When completing this measure, participants were asked to consider whether each item has been a problem in any of their interpersonal relationships and, if so, the extent to which it has been distressing to them. Scores for each item range from 0 (Not at all) to 4 (Extremely) and total scores range from 0 to 128, with higher scores reflecting more interpersonal problems.

Maladaptive personality traits. The Personality Inventory for DSM-5 brief form (PID-5-BF; Krueger, Derringer, Markon, Watson, & Skodol, 2013), which was administered during the intake assessment, was used to evaluate maladaptive personality traits. The PID-5-BF includes 25 items that aim to capture five personality domains (i.e., *negative affect, detachment, antagonism, disinhibition* and *psychoticism*), with five items focusing on each domain. Participants were asked to indicate the degree to which each item aligns with their perception of their personality. The rating scale ranges from 0 (very false or often false) to 3 (very true or often true) and total scores span from 0 to 75, with higher total scores reflecting more maladaptive personality traits.

Interpersonal functioning. Interpersonal functioning was measured through patient-reported Standard Interaction Tasks (SIT-SR; Leising, Krause, Köhler, Hinsen, & Clifton, 2011), which consist of 17 dyadic role-plays that aim to provide information on patients’ personality characteristics and interpersonal problems and skills. At each assessment (i.e., intake, week 4 and post-treatment), patients completed the SIT-SR with their assigned assessor. During these scenarios, assessors were required to interact with the patient through a set of standardized

responses. Role-play scenarios included initiating a conversation with a stranger at a party and reprimanding someone for being inconsiderate. Participants were asked to maintain the interaction for approximately 90 seconds and were given the opportunity to skip or postpone tasks if necessary. After completing each scenario, participants were asked to provide ratings of their performance on a scale from 1 (bad) to 8 (good).

Chapter 3: Analytic Strategy

Hypothesis 1: Immediate & Delayed Cognitive Change

To test our first hypothesis, we first ensured that any identified relation could not be attributed to differences in stable patient characteristics by disaggregating the within-patient and between-patient effects of both CC-I and CC-D scores (Curran and Bauer, 2011; Falkenström, Granström, & Holmqvist, 2013; Sasso, Strunk, Braun, DeRubeis, & Brotman, 2015). This procedure involved using a separate ordinary least squares regression for each patient, in which session, centered on the mean of each patient's average number of sessions, was input as the predictor of either immediate or delayed cognitive change. To obtain an estimate of the within-patient CC-I or CC-D scores at each session, we then retained session-specific residuals from these patient-specific regression models. Using these same models, we also retained the patient-specific intercepts, which reflect the between-patient CC-I and CC-D scores.

To test the relation of immediate cognitive change with delayed cognitive change, we utilized SAS proc mixed to implement a repeated measures model in which within-patient variation in CC-I scores was entered as a predictor of session-to-session change in within-patient CC-D. More specifically, within-patient CC-I scores across sessions 1 through 4 were input as predictors of within-patient CC-D scores reported across sessions 2 through 5 (Strunk, Brotman, & DeRubeis, 2010; Strunk, Cooper, Ryan, DeRubeis, & Hollon, 2012). In order to address the

potential confound of patients' initial symptom severity, depressive symptoms evaluated at the intake assessment were controlled for. For example, the total amount of immediate cognitive change observed during session 1 was tested as the predictor of delayed cognitive change experienced between sessions 1 and 2 and reported prior to the beginning of session 2, while controlling for depressive symptoms assessed at intake[1].

Hypotheses 2 & 3: Cognitive & Symptom Change

To examine CC-I as a predictor of session-to-session symptom change, we used the approach outlined in the previous section to disaggregate within- and between-patient variation in CC-I scores and simultaneously examined these effects as predictors of session-to-session symptom change, while controlling for current symptom severity. In particular, we utilized SAS proc mixed to implement a repeated measures model in which within-patient and between-patient CC-I scores across sessions 1 through 4 were input as predictors of next-session depressive symptoms across sessions 2 through 5 (Strunk, Brotman, & DeRubeis, 2010; Strunk, Cooper, Ryan, DeRubeis, & Hollon, 2012). In order to address the potential confound of patients' current symptom severity, depressive symptoms assessed prior to sessions 1 through 4 were controlled for at each respective session. This was achieved by including current BDI-II score as a predictor of symptoms at the following session. For example, the total amount of cognitive change experienced by a patient during session 1 was investigated as the predictor of that patient's

[1] It is important to clarify that these models are not characterizing delayed cognitive changes occurring during a specific between-session interval, but rather, are reflecting average delayed cognitive changes across all sessions of interest. However, for the purposes of facilitating the understanding of our models, we will provide concrete examples with the wording used in this example throughout the document.

symptoms assessed prior to the beginning of session 2, while controlling for the patient's symptoms assessed prior to the beginning of session 1.

To simultaneously assess the within- and between-patient effects of CC-D as predictors of session-to-session symptom change, we utilized the disaggregation framework described above and input within- and between-patient effects of CC-D scores experienced across sessions 1 through 5 and reported across sessions 2 through 5 as predictors of subsequent symptom change across sessions 2 through 5. Given the timing of the assessments of delayed cognitive change, we controlled for symptom severity at the previous session, rather than the current session, in models involving the CC-D.

Exploratory Moderation Analyses

To examine patient-reported PID-5-BF, IIP-32, and SIT-SR scores as moderators of the relation of immediate and delayed cognitive change with subsequent symptom change, we assessed each variable in three separate repeated measures regression models. For each of the models concerning immediate cognitive change, the predictors entered were: current BDI-II score, within-patient CC-I score, between-patient CC-I score, the moderator of interest, the interaction of within-patient CC-I and the potential moderator, and the interaction of between-patient CC-I and the potential moderator. For each of the models concerning delayed cognitive change, the predictors entered were: BDI-II score at the previous session, within-patient CC-D score, between-patient CC-D score, the moderator of interest, the interaction of within-patient CC-D and the potential moderator, and the interaction of between-patient CC-D and the potential moderator.

Chapter 5: Results

Psychometric Properties of Cognitive Change Measures

Immediate Cognitive Change.

As detailed in the Measures section, the CC-I was created specifically for this study in order to capture broad cognitive changes that patients experienced during a given session. Given that this was the first time this measure was utilized, we examined the properties of it through several analyses. First, we conducted a parallel analysis to determine the number of factors underlying the measure, which suggested one factor. We then ran an exploratory factor analysis (EFA) to augment this finding. As this study used a repeated measures design, we conducted an EFA of the measure at each session of interest. Across the first four sessions, these EFAs revealed that all of the items on the measure loaded onto one factor, with factor loadings ranging from 0.55 to 0.89. The eigenvalues from each session also indicated one factor, with the eigenvalues for the first factor ranging from 6.87 to 12.67 across the sessions. These results suggest that all of the items assessed the same construct of immediate cognitive change. Relatedly, we also evaluated the internal consistency of this measure at each session. These analyses yielded standardized Cronbach's α coefficients ranging from 0.82 to 0.91 across each of the first four therapy sessions, indicating good to excellent internal consistency for these items.

Delayed Cognitive Change.

As with the CC-I, the CC-D was created specifically for this study in order to capture broad cognitive changes that patients experienced and CT skills that patients independently used during the interval between two sessions. Given that this was the first use of this measure, we examined its properties through several analyses. First, we conducted a parallel analysis to determine the number of factors underlying the measure, which suggested one factor. We then

ran an EFA to supplement this finding through the same session-specific approach outlined above. Across sessions 2 through 5, these EFAs revealed that all of the items on the measure loaded onto one factor, with factor loadings ranging from 0.43 to 0.85. The eigenvalues from each session also indicated one factor, with the eigenvalues for the first factor ranging from 6.57 to 13.10 across the sessions of interest. Considered together, these results indicate that all of the items assessed the same construct of delayed cognitive change. Relatedly, we also examined the internal consistency of this measure at each session. These analyses yielded standardized Cronbach's α coefficients ranging from 0.82 to 0.91 across each of the therapy sessions of interest, indicating good to excellent internal consistency for these items.

Intraclass Correlation Coefficients of Cognitive Change

To determine if there was enough within-patient variation in immediate and delayed cognitive change to detect an effect, we first calculated intraclass correlation coefficients (ICCs) to estimate the proportion of total variation in these process variables that was accounted for by within- and between-patient variability. It should be noted that, given the nature of within-patient effects of process scores, within-patient variation might also include variation attributable to error. The analyses involving immediate cognitive change indicated that 60% of the variance in these scores was between-patient variability, with the remaining variation being within-patient (41%). The analyses involving delayed cognitive changed revealed that within-patient variation accounted for 50% of the variability in scores, with the remaining percentage of variance accounted for by within-patient variability. This pattern suggests considerable within-patient variability, allowing for meaningful tests of this variation as a predictor.

Correlations among Variables

Prior to investigating the predictive relations of interest, we examined correlations between the moderators of interest. We found that all moderating variables were significantly correlated, with the strongest correlation between PID-5-BF and IIP-32 (Table 3). We also investigated the associations between immediate cognitive change scores, which were averaged across sessions 1 through 4 for each patient, and each moderator of interest. As shown in Table 3, immediate cognitive change was not significantly correlated with either PID-5-BF or IIP-32. However, this analysis revealed a significant correlation between immediate cognitive change and SIT-SR. Using this same approach, we then calculated the correlations between delayed cognitive change scores and each moderator of interest. As shown in Table 3, delayed cognitive change was not significantly correlated with PID-5-BF, IIP-32, or SIT-SR. Finally, we examined the association between immediate and delayed cognitive change scores, which were averaged across the sessions of interest for each patient. As expected, this analysis revealed that these two variables were significantly correlated (see Table 3).

Degree of Symptom Change

In addition to these correlational analyses, we also examined the magnitude of symptom change across sessions 1 through 5 before testing the predictive relations of interest. In order to assess the variability in symptom change, we used a paired samples *t*-test to compare BDI-II scores at sessions 1 and 5, which indicated an average improvement of 4.47 points $t(119) = 5.60$, $p < .01$. As shown in Table 4, across the four intervals that we examined, the average change in session-to-session BDI-II ranged from 0.10 to 2.10, with the standard deviations around those means ranging from 5.30 to 6.90. It should be noted that these values for session-to-session symptom change were found by subtracting the BDI-II score at the later of the two sessions in

each interval from that at the earlier session (i.e., BDI-II score at session 1 – BDI-II score at session 2) and that a positive value reflects a decrease in patient-reported symptom severity. Given that all values from this study were positive, we can conclude that, on average, patients' symptoms improved across each session-to-session interval, with the greatest average symptom change occurring between sessions 1 and 2. Our models aim to capture this variability in BDI-II scores across the first five therapy sessions, as well as the role that immediate and delayed cognitive changes and the interaction of these cognitive changes with specific patient characteristics play in predicting this variability.

Primary Analyses

Hypothesis 1: Immediate & delayed cognitive change.

As outlined in the Analytic Strategy section, we aimed determine the relation between CC-I and CC-D through a repeated measures regression model. We first examined within-patient CC-I as a predictor of subsequent within-patient CC-D. Consistent with our hypothesis, within-person CC-I scores across sessions 1 through 4 significantly predicted within-person CC-D scores across sessions 2 through 5, while controlling for BDI-II at intake ($b = 0.24$, $SE = 0.05$, $t = 4.62$, $p < .01$). For instance, cognitive change observed during session 2 significantly predicted cognitive changes experienced between sessions 2 and 3 and reported prior to the beginning of session 3, while controlling for initial symptom severity. This finding suggests that the identified predictive relation of immediate cognitive change and subsequent delayed cognitive change is likely not attributable to between-patient differences and is consistent with a causal effect.

We then examined the relation of between-patient CC-I and between-patient CC-D. Given the nature of between-patient effects of process variables, we examined the correlation of between-patient CC-I scores and between-patient CC-D scores, rather than a potential predictive

relation of these two variables. This analysis yielded a Pearson correlation coefficient (r) of 0.66 ($p < .01$), suggesting a statistically significant association.

Hypothesis 2: Immediate cognitive change & symptom change.

All descriptive and inferential statistics from our models involving immediate cognitive change can be found in Table 1. We utilized the repeated measures regression model outlined in the Analytic Strategy section to simultaneously examine within-patient and between-patient CC-I as predictors of session-to-session symptom change across the sessions of interest. Between-patient CC-I scores significantly predicted next-session symptom changes (b = -0.12, SE = 0.04, t = -3.08, $p < .01$). However, contrary to our hypotheses, within-patient CC-I scores did not predict next-session symptom changes (b = -0.13, SE = 0.10, t = -1.24, p = .22). Given these findings, in conjunction with the results from our ICC analyses, it is possible that CC-I scores are largely driven by between-patient variability, suggesting that any identified predictive relation of CC-I and symptom change may be accounted for by stable patient characteristics.

Hypothesis 3: Delayed cognitive change & symptom change.

We then tested the within- and between-patient effects of delayed cognitive change as a predictor of session-to-session symptom change across early sessions in CT. In line with our hypothesis, both within- and between-patient CC-D scores significantly predicted session-to-session symptom changes (within-patient CC-D: b = -0.50, SE = 0.08, t = -6.04, $p < .01$; between-patient CC-D: b = -0.11, SE = 0.03, t = -4.16, $p < .01$). Given these findings, in concert with the results from our ICC analyses, it is likely that the identified relation of delayed cognitive change and subsequent symptom change cannot solely be accounted for by stable patient characteristics and is consistent with a causal effect.

Moderation Analyses

Immediate cognitive change.

In order to determine if the predictive relation of within-patient immediate cognitive change with session-to-session symptom change varies according to patient's pretreatment characteristics, we then examined SIT-SR, PID-5-BF, and IIP-32 scores as moderators of this effect. Total, patient-reported SIT-SR scores did not significantly interact with within-patient CC-I scores (b = -0.01, SE = 0.09, t = -0.13, p = .90) in predicting session-to-session symptom reduction. Conversely, PID-5-BF scores significantly interacted with within-patient CC-I scores in predicting session-to-session symptom change (b = 0.02, SE = 0.01, t = 2.46, p = .01). As Figure 2 illustrates, within-patient CC-I scores significantly predicted greater symptom change among patients with fewer maladaptive personality traits, (i.e., lower PID-5-BF scores). IIP-32 scores also significantly moderated the effect of within-patient CC-I scores on session-to-session symptom reduction (b = 0.01, SE = 0.01, t = 2.54, p = .01). As Figure 3 shows, among patients who reported few interpersonal problems (i.e., low IIP-32 score), within-patient CC-I scores significantly predicted greater symptom change.

It is possible that both PID-5-BF and IIP-32 scores significantly interacted with within-patient CC-I scores in predicting session-to-session symptom change because the instruments measure related constructs. To test this possibility, we then ran a repeated-measures regression model that included both PID-5-BF and IIP-32 as moderators of the main effect. Neither PID-5-BF nor IIP-32 significantly interacted with within-patient CC-I scores in predicting session-to-session symptom change (PID-5-BF × within-patient CC-I: b = 0.01, SE = 0.01, t = 1.21, p = .23; IIP-32 × within-patient CC-I: b = 0.01, SE = 0.01, t = 1.50, p = .13). This finding suggests that PID-5-BF and IIP-32 likely capture related, not independent, constructs.

Supplementary Analyses.

In order to examine if the significant interaction of within-patient CC-I and PID-5-BF was driven by specific maladaptive personality traits, we then investigated the effect of each PID-5-BF subscale (i.e., *negative affect, detachment, antagonism, disinhibition*, and *psychoticism*) as a moderator of the main effect. The *detachment* (e.g., "I steer clear of romantic relationships" and "I'm not interested in making friends"), *disinhibition* (e.g., "People would describe me as reckless" and "I feel like I act totally on impulse"), and *psychoticism* (e.g., "My thoughts often don't make sense to others" and "I have seen things that weren't really there") subscales significantly moderated the relation of within-patient CC-I and session-to-session symptom change. Specifically, within-patient CC-I scores significantly predicted greater symptom change among patients who endorsed less detachment (b = 0.00, SE = 0.01, t = 1.96, p = .05). Disinhibition significantly interacted with within-patient CC-I scores in predicting session-to-session symptom reduction (b = 0.09, SE = 0.04, t = 2.49, p = .01), such that for people presenting less disinhibition, within-patient CC-I scores significantly predicted greater symptom change. Lastly, within-patient CC-I scores significantly predicted greater symptom change among patients who endorsed less psychoticism (b = 0.06, SE = 0.03, t = 2.01, p = .05). It is important to note that the levels of psychoticism in this study were quite low, as individuals with psychotic disorders did not qualify for this study. Thus, it is possible that the traits of psychoticism endorsed on this subscale are not truly reflective of psychotic delusions. Total scores for the *negative affect* and *antagonism* subscales did not significantly interact with within-patient CC-I scores in predicting session-to-session symptom reduction (*negative affect*: b = 0.02, SE = 0.03, t = 0.56, p = .58; *antagonism*: b = 0.05, SE = 0.04, t = 1.30, p = .19).

In order to examine if the significant interaction of within-patient CC-I and IIP-32 was driven by specific interpersonal constructs, we then investigated the total score of each IIP-32 subscale (i.e., *hard to be sociable, hard to be assertive, too aggressive, too open, too caring, hard to be supportive, hard to be involved*, and *too dependent*) as a moderator of the main effect. The *hard to be sociable* (i.e., "hard to socialize with other people" and "hard to make friends") and *hard to be involved* (i.e., "hard to make a long-term commitment to another person" and "hard to experience a feeling of love for another person") subscales significantly moderated the relation of within-patient CC-I and session-to-session symptom change. Specifically, within-patient CC-I scores significantly predicted greater symptom change among patients who endorsed fewer problems with being sociable (i.e., low scores on *hard to be sociable* subscale; $b = 0.05$, $SE = 0.02$, $t = 12.07$, $p = .04$). Difficulty being involved significantly interacted with within-patient CC-I scores in predicting session-to-session symptom change (i.e., low scores on *hard to be involved* subscale; $b = 0.04$, $SE = 0.04$, $t = 1.97$, $p = .05$), such that for people presenting few difficulties in being involved, within-patient CC-I scores significantly predicted greater symptom change. Total scores for the *hard to be assertive, too aggressive, too open, too caring, hard to be supportive*, and *too dependent* subscales did not significantly interact with within-patient CC-I scores in predicting session-to-session symptom reduction (*hard to be assertive:* $b = 0.04$, $SE = .029$, $t = 1.49$, $p = .14$; *too aggressive:* $b = 0.03$, $SE = 0.03$, $t = 1.07$, $p = .29$; *too open:* $b = 0.03$, $SE = 0.50$, $t = .60$, $p = .55$; *too caring:* $b = 0.04$, $SE = 0.027$, $t = 1.65$, $p = .10$; *hard to be supportive:* $b = 0.06$, $SE = 0.030$, $t = 1.82$, $p = .07$; *too dependent:* $b = 0.01$, $SE = 0.03$, $t = 0.17$, $p = .86$).

Delayed cognitive change.

In order to determine if the relation of within-patient delayed cognitive change and session-to-session symptom change varies according to patient's pretreatment characteristics, we then examined PID-5-BF, IIP-32, and SIT-SR scores as moderators of this effect. PID-5-BF scores and IIP-32 scores did not significantly interact with within-patient CC-D scores (PID-5-BF: $b = 0.00$, $SE = 0.01$, $t = -1.16$, $p = .25$; IIP-32: $b = -0.01$, $SE = 0.01$, $t = -1.76$, $p = .08$) in predicting session-to-session symptom change. Conversely, total SIT-SR scores significantly interacted with within-patient CC-D scores in predicting session-to-session symptom change ($b = 0.01$, $SE = 0.01$, $t = 2.23$, $p = .03$). As Figure 4 illustrates, this interaction was significant for both patients with low and high levels of interpersonal functioning in the same direction, with a stronger interaction for patients with poor interpersonal functioning. For all patients, the predictive relation of CC-D and session-to-session symptom change aligned with expectations, such that high CC-D scores predicted larger reductions in subsequent depressive symptoms and low CC-D scores predicted less robust reductions in subsequent depressive symptoms.

Chapter 6: Discussion

In this study, we investigated (1) immediate cognitive change as a predictor of subsequent delayed cognitive change; (2) both immediate and delayed cognitive change as predictors of subsequent symptom change; and (3) maladaptive personality characteristics, interpersonal problems, and interpersonal functioning as potential moderators of the relation of within-patient cognitive change and subsequent symptom change. To rule out stable patient characteristics as potential confounding variables, we disaggregated the within- and between-patient effects in the predictors of interest for all analyses. In the first of these examinations, our results support a predictive relation of within-patient CC-I and subsequent, within-patient CC-D.

These findings suggest that when patients experience a particularly high level of cognitive change during a therapy session, they are likely to experience subsequent delayed cognitive change following that session.

The research design and analytic strategy used in our assessments of cognitive and symptom change allowed us to test whether immediate and delayed cognitive changes predicted *subsequent* symptom change. After disaggregating the within- and between-patient variability in cognitive change scores, between-patient but not within-patient CC-I scores were significant predictors of session-to-session symptom change. If immediate cognitive changes produce symptom change during treatment, it would be expected that this relation would be reflected in within-patient, immediate cognitive change predicting subsequent symptom change. As this was not the case, it is possible that the identified predictive relation of between-patient immediate cognitive change and symptom change is better accounted for by stable patient characteristics, rather than the possible causal effect of interest. Interestingly, both the between- and within-patient effects of CC-D scores emerged as significant predictors of subsequent symptom change across a series of early sessions in CT. The identified relation of delayed cognitive change and subsequent symptom change is consistent with a causal effect, as within-patient, delayed cognitive change emerged as a significant predictor of outcome.

To determine whether the effect of within-patient variability in CC-I on symptom changes varies as a function of pretreatment characteristics, we examined moderators of the main effects of interest using within-patient effects of cognitive change scores. In examining potential moderators of the relation of within-patient CC-I and session-to-session symptom change, we found that this relation was stronger for patients who endorsed few maladaptive personality traits (PID-5-BF) and interpersonal problems (IIP-32). More specifically, for patients with low levels

of maladaptive personality traits and interpersonal problems, high scores on the CC-I significantly predicted larger reductions in depressive symptoms at the following session, and low scores on the CC-I significantly predicted less robust reductions in depressive symptoms at the following session. Fine-grained analyses of these measures revealed that the interaction of PID-5-BF and within-patient CC-I scores was largely driven by the *disinhibition, detachment* and *psychoticism* subscales, and the interaction of IIP-32 and within-patient CC-I scores was mostly accounted for by the *hard to be sociable* and *hard to be involved* subscales. In particular, these results suggest that immediate cognitive change may not be crucial for patients who endorse problems being sociable and/or involved with others and who present traits of disinhibition, detachment and/or psychoticism.

Given that the PID-5-BF and IIP-32 are highly correlated, it is possible that these interpersonal problems and maladaptive personality traits reflect a shared vulnerability that should be considered in research and clinical contexts. For instance, the *detachment* subscale on the PID-5-BF and the *hard to be involved* and *hard to sociable* subscales on the IIP-32 all describe difficulties forming and maintaining interpersonal relationships. Relatedly, the *disinhibition* subscale on the PID-5-BF, which captures the trait of impulsivity, and the *hard to be involved* subscale on the IIP-32, which encapsulates the ability to commit to others, may be reflective of related issues. More specifically, exhibiting high levels of impulsivity may hinder individuals from committing to others. In the context of therapy, patients who present with high levels of interpersonal problems and maladaptive personality traits may be unable to effectively interact with therapists, preventing the establishment of a committed alliance. Relatedly, these patients may be less open to collaborating with therapists to reevaluate negative cognitions, which may hinder the process of immediate cognitive change. With this in mind, patients who

scored highly on both the PID-5-BF and IIP-32 may be quite similar and require personalized strategies during therapy sessions to foster immediate cognitive change.

Interestingly, interpersonal functioning, as measured by the SIT-SR, did not significantly moderate the relation of CC-I and symptom change. It is possible that this measure did not emerge as a significant moderator because it does not tap into the construct that is driving the significant interactions of within-person CC-I with the other two moderating variables. In line with this possibility, the SIT-SR was not highly correlated with either the PID-5-BF or IIP-32 (Table 3). These results suggest that the PID-5-BF and IIP-32 reflect a shared construct (i.e., interpersonal vulnerability), which influences the process of immediate cognitive change, that the SIT-SR does not capture.

In our assessment of moderators of the relation of within-patient CC-D and symptom change, the SIT-SR emerged as the only significant moderator. Interestingly, the effect of within-patient, delayed cognitive change on symptom change was significant for both individuals with high and low levels of interpersonal functioning in the same direction, with a stronger effect for patients with lower interpersonal functioning. This evidence indicates that delayed cognitive change facilitates symptom change across patients with varying levels of interpersonal functioning and especially for patients with poor interpersonal functioning. It is possible that practicing cognitive skills is particularly important for these individuals because it enables them to reevaluate their application of other skills (i.e., social skills) and address the vulnerabilities that contribute to their poor interpersonal functioning and depressive symptoms. For patients with relatively higher levels of interpersonal functioning, it is possible that cognitive change has a relatively weaker effect on symptom change because these patients are using both social and cognitive skills to reduce their levels of depression. Considered together, these results suggest

that independently utilizing CT skills and experiencing delayed cognitive change is important for all patients with varying levels of interpersonal functioning.

Limitations.

Although we found some evidence that is consistent with immediate cognitive change predicting delayed cognitive change and both cognitive changes predicting session-to-session symptom change, it is important to address several limitations. Firstly, given the naturalistic design of this study, we cannot conclusively establish causal relationships from these data. In order to remove the possibility of a reverse predictive relationship (i.e., symptom change producing cognitive change), the study and analyses were designed so that examinations of independent variables always temporally preceded those of dependent variables. Additionally, we aimed to rule out stable patient characteristics as potential confounds through our disaggregation approach. It is important to note that immediate cognitive change did not emerge as a significant predictor of subsequent symptom change on the within-patient level, with the exception of patients with few maladaptive personality traits and interpersonal problems. Therefore, it is possible that the identified relation of between-patient immediate cognitive change and subsequent symptom change may be better ascribed to unmeasured between-patient differences or therapeutic processes (i.e., therapeutic alliance).

Secondly, all of the measures used to collect data during this study were self-report instruments. Although such assessments provide rich information on patients' psychological and interpersonal factors, self-report measures have been shown to be vulnerable to reporting biases, including social desirability and recall biases (Furnham, 1986). Given that research has shown social desirability bias as a factor in questionnaires related to personality traits (Pedregon, Farley, Davis, Wood, & Clark, 2012; Soubelet & Salthouse, 2011), it is possible that this bias is present

in this study. However, it is unlikely that recall bias existed, as the measures asked patients about stable characteristics or events, feelings, and thoughts that occurred recently (i.e., within the hour). In order to address the disadvantages of self-report instruments, future studies could examine the relation between cognitive change and symptom change using a multi-modal approach.

Relatedly, this is the first study in which these assessments of immediate and delayed cognitive changes have been used. Given that the reliability and validity of these measures have yet to be fully examined, it is possible that they did not fully capture the construct of cognitive change as intended. However, EFAs and parallel analyses suggest that all items on the CC-I and CC-D assess the same construct of immediate and delayed cognitive change, respectively. Additionally, analyses of internal consistency yielded standardized Cronbach's α coefficients in the good to excellent range for both measures.

Finally, the current sample consisted largely of Caucasian females. Therefore, the results of this study may not be generalizable to more diverse populations. Future researchers should assess the predictive relation of immediate with delayed cognitive change, as well as these cognitive changes as predictors of symptom change, in a sample that is more representative of the broader population.

Chapter 7: Conclusions

The results of this study offer additional support for the theory that cognitive change contributes to symptom reduction in CT for depression. Additionally, these findings indicate that cognitive changes observed during therapy sessions predict cognitive skills utilized and cognitive changes experienced directly following the session on a within-patient level. As the within-patient effects of delayed cognitive change significantly predicted session-to-session symptom

change across the early portion of treatment, this form of cognitive change may play a key role in producing positive treatment outcome. Relatedly, our results suggest that immediate cognitive change may be especially important for patients with low levels of maladaptive personality traits and interpersonal problems, whereas all patients with varying levels of interpersonal functioning may benefit from delayed cognitive change.

Future research should aim to replicate these findings and further investigate the therapeutic experience for patients with high levels of maladaptive traits and interpersonal problems. For instance, it is possible that these individuals benefit from a mechanism other than cognitive change, such as the therapeutic alliance, in CT for depression. If replicated, these findings could have considerable implications for clinicians. As immediate cognitive change was shown to produce delayed cognitive change, which then predicted subsequent session-to-session symptom change on a within-patient level, clinicians may aim to facilitate cognitive change in patients during therapy sessions to instigate this chain of therapeutic gains. Relatedly, clinicians may be able to optimize treatment by personalizing therapy sessions according to patient characteristics, including presence of a personality disorder and level of interpersonal skills and functioning. Considered together, these findings emphasize that cognitive change plays an essential role in producing subsequent symptom changes, indicating that this process of change should be a major focus of future research and during therapy sessions in CT for depression.

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Appendix A: Tables & Figures

Table 1. *Descriptive and Inferential Statistics for Model with CC-I as a Predictor of Session-to-Session Symptom Change*.

Predictors	***M* (*SD*)**	***b***	***SE***	***p***
Current BDI-II	26.0 (11.7)	0.96	0.02	**< .01*** **
Within-Patient CC-I	0.0 (2.7)	-0.13	0.10	.22
Between-Patient CC-I	15.3 (5.2)	-0.12	0.04	**< .01** **
	***M* (*SD*)**	***b***	***SE***	***p***
PID-5-BF	30.7 (10.0)	0.04	0.06	.54
IIP-32	53.0 (16.4)	-0.01	0.04	.77
SIT-SR	82.9 (19.3)	0.14	0.48	.78
		b	***SE***	***p***
Within-Patient CC-I × PID-5-BF		0.02	0.01	**.01***
Within-Patient CC-I × IIP-32		0.01	0.01	**.01***
Within-Patient CC-I × SIT-SR		-0.01	0.09	.90

***Note.* *** $p < .0001$, ** $p < .01$, * $p < .05$.**

Table 2. *Descriptive and Inferential Statistics for Model with CC-D as a Predictor of Session-to-Session Symptom Change*.

Predictors	***M* (*SD*)**	***b***	***SE***	***p***
Current BDI-II	26.0 (11.7)	0.94	0.02	**< .01*** **
Within-Patient CC-D	0.0 (3.2)	-0.50	0.08	**< .01*** **
Between-Patient CC-D	19.9 (8.4)	-0.11	0.03	**< .01*** **
	***M* (*SD*)**	***b***	***SE***	***p***
PID-5-BF	30.7 (10.0)	0.06	0.05	.21
IIP-32	53.0 (16.4)	0.00	0.03	.90
SIT-SR	82.9 (19.3)	0.02	0.02	.26
		b	***SE***	***p***
Within-Patient CC-D × PID-5-BF		-0.00	0.01	.25
Within-Patient CC-D × IIP-32		-0.01	0.01	.08
Within-Patient CC-D × SIT-SR		0.01	0.01	**.03***

Note. *** $p < .0001$, ** $p < .01$, * $p < .05$.

Table 3. *Correlations among Variables.*

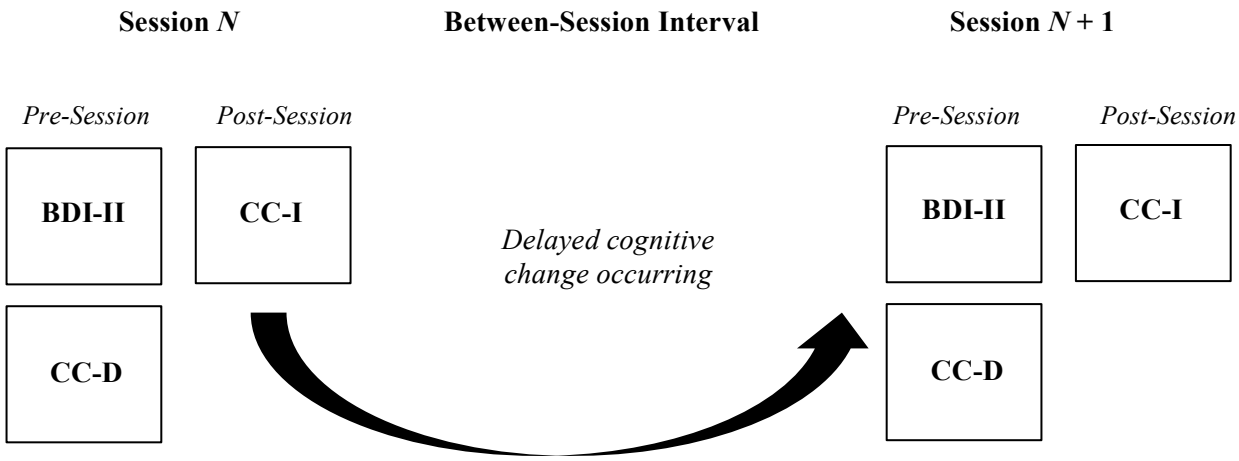
	CC-I	CC-D	PID-5-BF	IIP-32	SIT-SR
CC-I	1.00	---	---	---	---
CC-D	**0.73*** **	1.00	---	---	---
PID-5-BF	0.04	-0.02	1.00	---	---
IIP-32	-0.11	-0.09	**0.57*** **	1.00	---
SIT-SR	**0.24****	0.13	**-0.11***	**-0.14****	1.00

***Note.* *** $p < .0001$, ** $p < .01$, * $p < .05$**; scores on cognitive change measures were averaged across all sessions of interest for each patient.

Table 4. *Descriptive Statistics for BDI-II Scores across Sessions 1 through 5.*

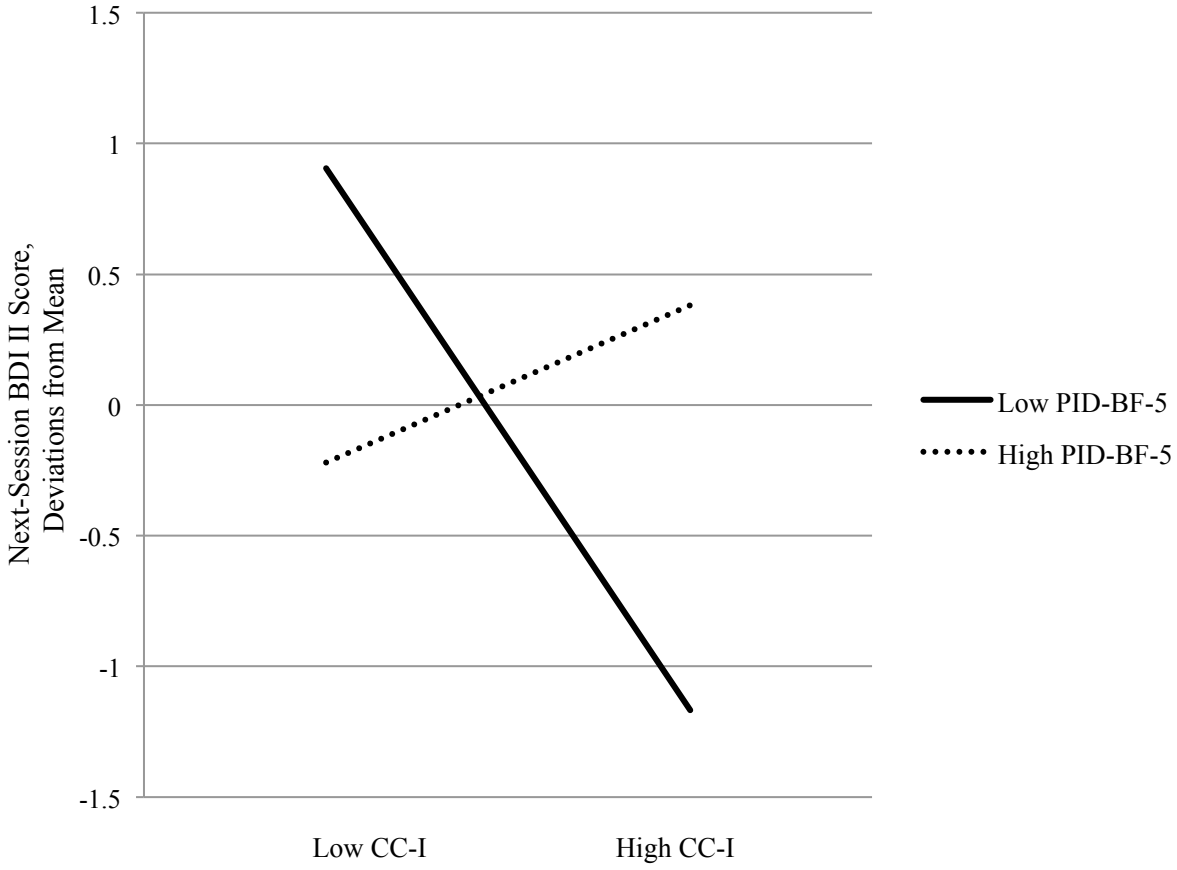
BDI-II scores at each session		**Change in BDI-II scores for each session-to-session interval, *M* (*SD*) of differences**	
Session	***M* (*SD*)**		
1	28.4 (10.5)	Session 1 - 2	2.1 (6.9)
2	26.6 (11.5)	Session 2 - 3	1.6 (5.9)
3	24.9 (12.1)	Session 3 - 4	0.8 (5.3)
4	24.0 (12.4)	Session 4 - 5	0.1 (6.5)
5	24.1 (13.1)		

Figure 1. *Visual representation of study design.*



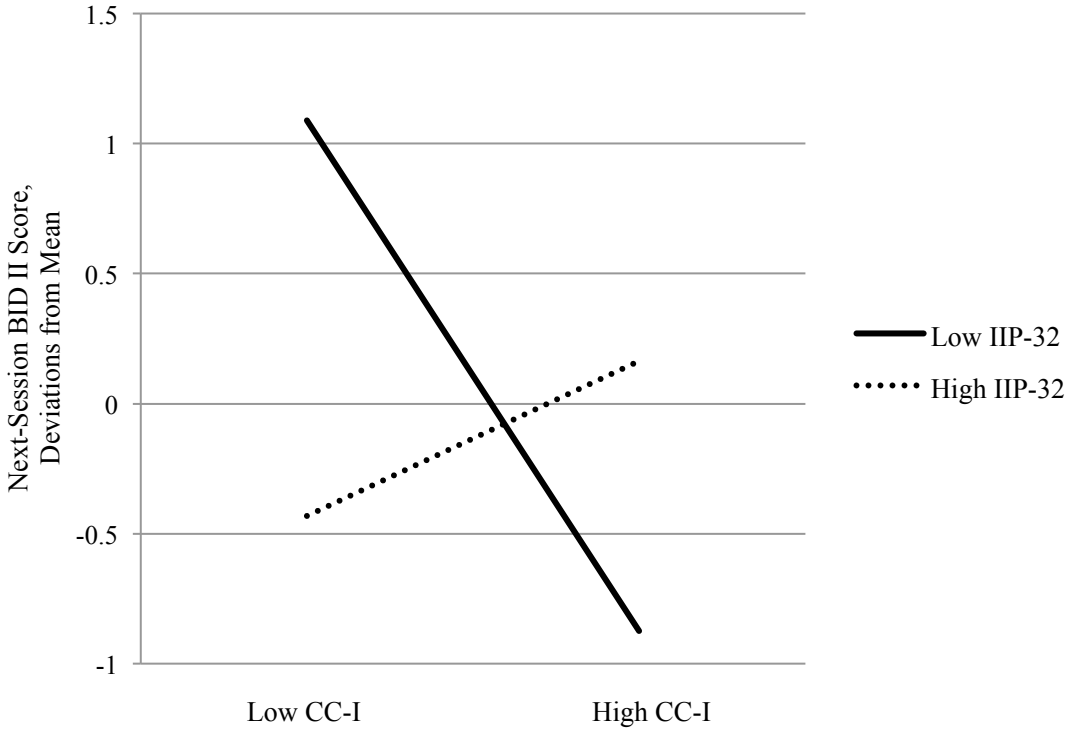
Note. BDI-II = Beck Depression Inventory; CC-I = Assessment of Immediate Cognitive Change; CC-D = Assessment of Delayed Cognitive Change; the BDI-II and CC-D assessed depressive symptoms and delayed cognitive changes, respectively, experienced over the past week; the CC-I assessed immediate cognitive changes observed during the session that just ended.

Figure 2. *Predictive relation of within-patient CC-I and session-to-session symptom change across overall patient maladaptive personality traits (PID-5-BF).*



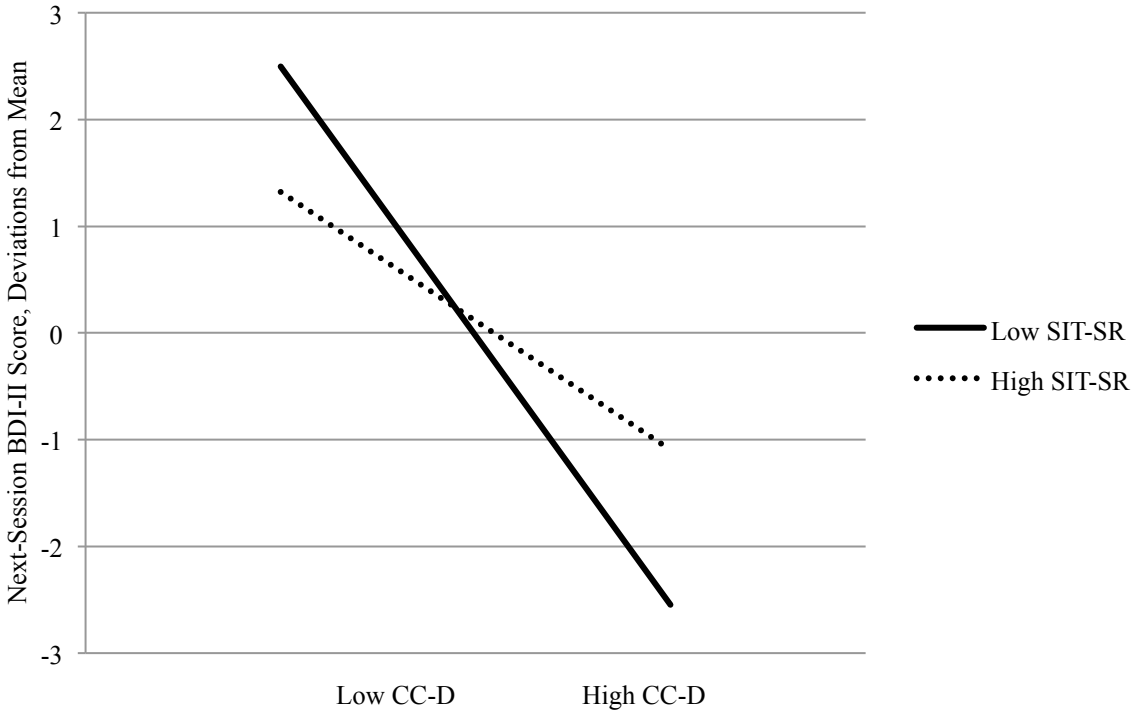
Note. High and low values of within-patient, immediate cognitive change scores are ±1 *SD* from the mean ($M = 0$, $SD = 2.73$). Patients' next-session *Beck Depression Inventory-II* (BDI-II) scores ($M = 26.02$) were centered at zero. Plotted values represent deviations from the mean in *BDI-II* units.

Figure 3. *Predictive relation of within-patient CC-I and session-to-session symptom change across patient interpersonal problems (IIP-32).*



Note. High and low values of within-patient, immediate cognitive change scores are ±1 *SD* from the mean ($M = 0$, $SD = 2.73$). Patients' next-session *Beck Depression Inventory-II* (BDI-II) scores ($M = 26.02$) were centered at zero. Plotted values represent deviations from the mean in *BDI-II* units.

Figure 4. *Predictive relation of within-patient CC-D and session-to-session symptom change across patient interpersonal functioning (SIT-SR).*



Note. High and low values of within-patient, delayed cognitive change scores are ±1 *SD* from the mean ($M = 0$, $SD = 3.19$). Patients' next-session *Beck Depression Inventory-II* (BDI-II) scores ($M = 26.02$) were centered at zero. Plotted values represent deviations from the mean in *BDI-II* units.