UNDERSTANDING PATTERNS OF CHANGE: PREDICTORS OF RESPONSE PROFILES FOR CLIENTS TREATED IN A CBT TRAINING CLINIC

by

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Empirical support for the efficacy of CBT in treating depression suggests that the majority of clients will respond to this intervention. However, the more nuanced, and clinically relevant, question of "Which clients will respond to CBT for depression?" has been difficult to answer. Research efforts have focused on two different approaches to this question. One approach focuses on trajectories of symptom change within the first weeks of treatment to identify clients who are most likely to achieve response. A second approach looks to pretreatment client variables such as hopelessness and dysfunctional attitudes to identify clients who are more likely to respond. The current study is the first to simultaneously compare these two approaches to the prediction of treatment outcome. The sample consists of 222 clients (65.32% female, 92.79% Caucasian), ages 18 through 64 (M = 27.85, SD = 11.28), receiving treatment for mood and anxiety disorders (59%

met criteria for comorbid disorders) in a CBT oriented psychology training clinic. Results suggest that the rate of change in depressive symptoms over the first five treatment sessions significantly and consistently predicted outcome over and above the majority of pretreatment variables, except for precontemplation stages of change scores and initial severity of depression and anxiety symptoms. Similarly, rate of change in anxiety symptoms significantly predicted outcome on two of the three measures over and above the majority of pretreatment variables, except for hopelessness and initial severity of anxiety symptoms. Post hoc analyses revealed different predictors of outcome when trajectories of change and pretreatment variables were examined separately. Both rates of change *and* a number of pretreatment variables predicted outcome. Finally, pretreatment predictors of rate of early symptom change such as a contemplative orientation to change and therapist experience, were identified which may suggest that therapists should target these factors to potentially maximize rapid early symptom change, and in turn outcome. The findings are discussed in terms of their implications regarding methodological approaches to treatment outcome research and treatment planning for adults with comorbidities.

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CHAPTER I

INTRODUCTION

Rates of Response to Cognitive Behavioral Therapy

Over 325 Cognitive Behavioral Therapy (CBT) outcome studies reveal large effects for treating adult depression, panic disorder, generalized anxiety disorder, social phobia, and posttraumatic stress disorder (Butler, Chapman, Forman, & Beck, 2006). Meta-analyses have demonstrated that CBT is more effective than wait-lists, untreated controls, pharmacotherapy, behavior therapy, and a heterogeneous group of other therapies in treating over 16 different disorders (e.g., Dobson, 1989; Westen & Morrison, 2001). The adaptation of CBT protocols targeting a wide range of presenting problems has made CBT the most commonly cited empirically supported treatment (EST) included in the Best Practice Guidelines produced by the American Psychological Association (Chambless, Baker, Baucom, et al., 1998).

Although the above summary compellingly identifies CBT as an effective treatment for a number of disorders, a closer look at the literature suggests response rates are actually quite variable. For instance, rates of response to CBT in randomized clinical trials (RCTs) range from as low as 40% for moderate to severe depression (DeRubeis, Hollon, Amsterdam et al., 2005) to as high as 90% for panic disorder (Clark, Salkovskis, Hackmann, Middleton, Anastasiades, & Gelder, 1994). Inconsistent rates of response for

CBT targeting the *same* disorder have been observed even when the same treatment protocol is used. For example, two studies using the same protocol for group treatment of social phobia (Heimberg, 1991) reported response rates of 75% and 36% (Heimberg et al, 1990 and Hope et al, 1995 respectively) even though no obvious differences between the studies, in terms of sample characteristics, therapist experience, or outcome measure, were observed.

These very different response rates to the same treatment protocol suggest that although CBT has been identified as an EST, it is not effective for all clients and as such it may not be that informative to estimate a single overall response rate to CBT. This is certainly not a new idea. Four decades ago, Gordon Paul articulated the question, "What treatment, by *whom*, is most effective for this individual with that specific problem, *under* which set of circumstances?" (Paul, 1967, p. 111). This question has guided psychotherapy research in the hunt for predictors, moderators, and mediators of response, but it has not yet yielded especially constructive or consistent results.

Predictors of Response

For instance, numerous predictors of response to treatment for anxiety and depression have been identified. These predictors can be categorized into three broad domains: clinical, cognitive and contextual. Initial levels of symptom severity (Haby, Donnelly, Corry, & Vos, 2006; Merrill, Tolbert, & Wade, 2003) and therapist level of training (DeRubeis et al., 2005; Driscoll, Cukrowicz, Reitzel, Hernandez, Petty, & Joiner, 2003; Grey, Salkovskis, Quigley, Clark, & Ehlers, 2008; Lutz, Leon, Martinovich, Lyons, & Stiles, 2007; Stein & Lambert, 1995; Weertman & Arntz, 2007) have been identified

as predictors in clients with both depression and anxiety disorders. Nonspecific predictors of response for treatment of depression include dysfunctional attitudes, age, marital status, hopelessness (Barber & DeRubeis, 1992), severity of depression (Hamilton & Dobson, 2002), and readiness to change (Lewis, Simons, Silva, et al., 2009; Lichtenberg & Hummel, 2000). Fewer predictors have emerged from the anxiety literature; however, initial severity, comorbid depression, and motivation to change have been identified as predictors of response (e.g., Chambless, Tran, & Glass, 1997; Keijsers, Hoogduin, & Schaap, 1994).

Predictors of response are indeed useful to clinicians particularly in selecting treatment and treatment components. However, similar to the response rates reviewed above, predictors of response are more variable than would be desired. That is, dysfunctional attitudes, for example, have been identified as a predictor of response in some, but not all, depression treatment outcome studies leaving clinicians to decide to what extent pretreatment levels of dysfunctional attitudes should guide their treatment planning.

It may be that a different approach to answering Gordon Paul's question would be more illuminating. One such avenue for research involves taking a closer look at the rates, patterns and processes of change in symptoms over the course of psychotherapeutic treatment. That is, rather than restricting outcome analyses to pre-post group mean comparisons whereby the focus is on the relationship between pretreatment factors and posttreatment symptom scores, analysis of *within* treatment outcome data (session-by-session) may be more revealing. This level of analysis might provide the perspective

necessary to unpack the inconsistent and variable findings reported above. Said differently, although successful response to treatment would remain the primary target and focus of psychotherapy outcome research efforts, perhaps a better understanding of the different pathways to response would aid in efforts to identify factors predictive of outcome with greater consistency.

Indeed this line of research appears to be a fruitful endeavor. For instance, Ilardi and Craighead (1994) observed a rapid response to psychotherapy such that 60-80% of depressive symptom improvement occurred within the first four weeks of treatment. Numerous other studies found similar patterns of rapid early response upon reanalysis of the data (e.g., Beckham, 1989; Blackburn & Bishop, 1983; Fennell & Teasdale, 1987; Rush et al., 1977). These rapid rates of depressive symptom reduction observed during psychotherapeutic treatment have been found to predict outcome at follow-up more so than similar patterns observed in nonpsychological therapies (Gilboa-Schechtman & Shahar, 2006). Although rapid response to treatment was originally observed in depressed clients, this pattern has since been observed in clients treated for panic disorder (e.g., Penava, Otto, Maki, & Pollack, 1998), alcohol abuse (e.g., Breslin, Sobell, Sobell, Buchan, & Cuningham, 1997) and in mixed samples of clients (Crits-Christoph, Connolly, Gallop, et al., 2001) predicting success at follow-up. The predictive validity of rapid early rates of response in psychotherapy appears to be a robust finding in the literature. That is, rapid response has been consistently identified as a powerful predictor of successful outcomes, more so than any of the nonspecific pretreatment variables identified in the literature.

However, the rapid response and pretreatment predictor findings reported above were derived from analyses at the level of the group's mean. Implicit in Gordon Paul's question is the notion that individuals differ in important ways. Thus it is possible that analysis at the level of the individual might identify even more meaningful patterns and processes of change. That is, focus on group means without consideration of individual heterogeneity may obscure other patterns that could be identified at the individual level that might similarly be associated (or even more strongly associated) with good outcomes.

Sudden Gains Phenomenon

Tang and DeRubeis (1999) attempted to unpack the observed rapid early rates of response by taking the analysis down to the level of the individual in an investigation of patterns of response to treatment for Major Depressive Disorder (MDD). Their research was also motivated by the desire to identify mechanisms of change in CBT. Cognitive mediation was an obvious candidate given the theory upon which the therapy is based (Beck, Rush, Shaw, & Emery, 1979). Therapeutic alliance quickly became an opposing candidate, championed by those who support the notion of nonspecific mechanisms of change (Burns & Nolen-Hoeksema, 1992). In Tang and DeRubeis' (1999) reanalysis of session by session data from the Treatment for Depression Collaborative Research Program (TDCRP; Elkin et al., 1989), they observed a phenomenon whereby clients demonstrated sudden, dramatic, enduring decreases in depressive symptoms from one session to the next. They dubbed this phenomenon "sudden gains". Sudden gains were observed in approximately 50% of the clients, accounted for over 50% of the client's

total symptom reduction, typically occurred early in treatment (median = session 5), and were related to recovery (79% of clients who experienced sudden gains recovered). Tang and DeRubeis (1999) interpreted this data as support for the cognitive mediation hypothesis in that significantly greater cognitive change was observed in the session prior to the sudden gain. And, they observed significant increases in the therapeutic alliance in the session *after* the gain was made suggesting that symptom reduction led to reported increases in therapeutic alliance. These findings ignited a flurry of research efforts to investigate this phenomenon in different samples.

Since Tang and DeRubeis' (1999) original work, the number of studies examining sudden gains is well into the double digits (e.g., Gaynor et al., 2003; Hardy et al., 2005; Stiles et al., 2003; Tang et al., 2002). Importantly, sudden gains have been observed in a variety of settings: RCTs (e.g., Tang, DeRubeis, Beberman, & Pham, 2005; Vittengl, Clark, & Jarrett, 2005), "real world" settings (e.g., Stiles, Leach, Barkham, et al., 2003), and training clinics (e.g., Greenfield, 2009). The replication across sites and settings suggests that sudden gains is a reliable phenomenon that could inform our understanding of patterns of response.

Similar to the findings regarding rapid early rates of change, the sudden gains phenomenon was first identified in the depression literature and has since expanded to include a variety of other clinical disorders. Sudden gains have been observed in panic disorder (Clerkin, Teachman, & Smith-Janik, 2008), generalized anxiety disorder (Present, Crits-Christoph, Gibbons, et al., 2007), social phobia (Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006), bulimia nervosa (Grilo, Masheb, & Wilson, 2006),

alcohol abuse (Breslin, Sobell, Sobell, Buchan, & Cunningham, 1997), and in clients with comorbidities (Tschitsaz & Lutz, 2009). In addition, although the motivation for studying sudden gains developed in an effort to examine the cognitive mediation hypothesis in CBT--thus thinking sudden gains would be specific to CBT--researchers have observed sudden gains in Interpersonal Psychotherapy (Kelly, Cyranowski, & Frank, 2007), Supportive-Expressive Psychotherapy (Tang, Luborsky, & Andrusyna, 2002), Nondirective Supportive Therapy (Gaynor, Weersing, Kolko, Brimaher, Heo, & Brent, 2003), Psychoeducational group therapy (Kelly, Roberts, & Ciesla, 2005), Pharmacotherapy, and in Pill Placebo (Vittengl, Clark, & Jarrett, 2005). Sudden gains do *not* appear to be specific to any one treatment (e.g., CBT, IPT), or any one *kind* of treatment (e.g., psychotherapy, pharmacotherapy), nor do sudden gains appear to be specific to *treatment* but perhaps common to improvement however achieved. That is, Kelly, Roberts, and Bottonari (2007) observed 60% of depressed college students experienced sudden gains outside of the treatment context suggesting sudden gains may be a natural part of the course of depression and possibly other disorders given the diverse range of disorders in which sudden gains have been observed.

Despite numerous attempts, the literature has yet to identify *who* experiences sudden gains. Sudden gains do not appear to be related to age, ethnicity, marital status, employment status, gender, or socioeconomic status (Gaynor et al., 2003; Kelly et al., 2007). In addition, none of the following factors appear to be related to sudden gains: dysfunctional attitudes, attributional style, hopelessness, level of cognitive distortions, stress, or overall functioning (Gaynor et al., 2003; Hardy et al., 2005; Kelly et al., 2005;

Kelly et al., 2007). Without identifying predictors of sudden gains it remains difficult to anticipate which clients might go on to experience this privileged pathway to response.

Unfortunately, the lack of specificity is not the only limitation of the sudden gains literature. Researchers repeatedly acknowledge the somewhat arbitrary criteria on which the phenomenon is based (Tang & DeRubeis, 1999). Specifically, Tang and DeRubeis originally described this phenomenon in an exploratory investigation of the session-by-session data. They created the three-part criteria in an effort to characterize the seemingly meaningful pattern to facilitate replication. Although some effort has been made to maintain the original criteria, it is not uncommon for researchers to omit one of the three criteria (e.g., Gaynor, Weersing, Kolko, Birmaher, Heo, & Brent, 2003) or alter one or more of the criteria (e.g., Hardy et al., 2005; Kelly, Roberts, & Bottonari, 2007; Stiles, Leach, Barkham, et al., 2003; Vittengl, Clark, & Jarrett, 2005) thus limiting the ability to generalize findings across studies.

Recently, some researchers have reached consensus regarding the criteria for sudden gains but even so, the characteristics of the sudden gains differ dramatically from one sample to the next. For instance, the median session for a sudden gain to occur ranges from session 4 (Vittengl et al., 2005) to session 11 (Busch et al., 2006); the percent of total improvement captured by the gain ranges from 51 (Tang & DeRubeis, 1999) to 105 (i.e., the gain was greater than the symptom reduction maintained by termination; Stiles, et al., 2003); and, the percent of sudden gains reversed ranges from 17 (Tang & DeRubeis, 1999) to 57 (Gaynor et al., 2003). In addition to the abovementioned limitations, the sudden gains phenomenon represents only one of numerous possible

pathways to acute phase response that is experienced by as few as 17% of clients in routine clinical care (Stiles et al., 2003).

Taken together, the sudden gains literature may be less informative than originally perceived. That is, sudden gains were thought to be the product of a mechanism of change specific to CBT for depression, but instead it seems to lack specificity altogether. Rather than providing empirical support for the underpinnings of cognitive theory of depression, researchers seem to have identified a pattern of discontinuity common to symptom change in a small subset of individuals with mental illness.

Expanding on Sudden Gains

This is not to say that the sudden gains phenomenon is a worthless research pursuit, but rather it seems important to meaningfully characterize more of the sample beyond the subgroup who experience sudden gains. More recently researchers have expanded their investigation of the sudden gains phenomenon. For instance, Busch, Kanter, Landes, and Kohlenberg (2006) extended their analysis to include pretreatment and first-session gains in a small community sample ($N = 38$) receiving Cognitive Therapy for Major Depressive Disorder (MDD). They found that 100% of clients who experienced a first-session gain recovered—a significantly greater number of clients recovered as compared to those with only a pretreatment gain (67% recovery rate) and those with neither a pretreatment gain nor a first-session gain (46% recovery rate). Whereas typically this work has identified "successful" pathways to response, Tschitsaz and Lutz (2007) found that clients who experienced both sudden gains *and* losses fared markedly poorer than those who experienced either gains or losses--an effect size less

than .4 on the BDI as compared to large effect sizes (e.g., .8) for those who experienced sudden gains. Together, these findings build the case for considering gains *and* losses made by clients that cannot be captured by the sudden gains criteria alone.

The next logical step for this line of research might be to examine, in one study, the abovementioned gains and losses so as to possibly characterize the different aspects of the seemingly discontinuous trajectory of each client in the sample. Specifically, this would include examination of pretreatment gains, first session gains, sudden gains, sudden losses and those who experience both sudden gains *and* losses. Because previous research has observed a single client to experience one *or more* of these kinds of gains/losses, the unique predictive utility of these individual characterizations remains unclear because countless possible profile combinations of these gains/losses exist.

Growth Curve Modeling

As an alternative approach, Haas, Hill, Lambert, and Morrell (2002) used hierarchical linear modeling (HLM) to examine the relationship between rate of early symptom change and outcome. Indeed, consistent with earlier reports using group mean data, Haas and colleagues found that rapid rates of early response over the first 3 therapy sessions, modeled to include individual variability, predicted outcome. One advantage of this approach to analyses is that it utilizes the session-by-session, individual data (like the sudden gains literature), *and* it provides an informative picture of the sample's trajectory that is more comprehensive than the sudden gains literature affords.

Growth Curve Modeling (GCM), as employed in the work of Haas and colleagues, has a number of additional advantages over the sudden gains approach but

also over pre-post group mean analysis and the perhaps most typical repeated measures Analysis of Variance (ANOVA). Not only do ANOVA approaches require that the data adhere to difficult to meet assumptions (e.g., fully balanced data, equally spaced intervals between sessions) which GCM does not require, but also ANOVA approaches cannot accommodate both time-invariant and time-varying covariates, or continuous predictors of response as GCM allows (Laurenceau, Hayes, & Feldman, 2007). Perhaps an even greater advantage of GCM is that both inter-individual *and* intra-individual change can be modeled simultaneously (Collins & Sayer, 2000; Rogosa & Willett, 1985). That is, whereas ANOVA approaches treat the above-mentioned individual differences in change trajectory as measurement error, GCM considers these observations as meaningful heterogeneity in change pathways.

In GCM, trajectories of change can thus be characterized by replicable models that when compared across samples might bring the field closer to identifying a typical trajectory of response while considering individual variability. Laurenceau, Hayes, and Feldman (2007) reported that understanding the shape and rate of change through GCM is an important initial step if we are to improve psychotherapy outcomes. Specifically, they implied that understanding the difference in patterns of symptom change between responders and non-responders might provide insight into ways to improve rates of non-response. Indeed, Speer and Greenbaum (1995) compared five methods for computing significant individual client change in psychotherapy and found GCM to be the most sensitive, recommending it be used for treatment outcome research whenever possible. Perhaps surprisingly, despite the advantages and appropriateness of employing GCM

over more traditional approaches, until recently few studies have examined change in symptom severity over the course of treatment using GCM. By bridging the previously described approaches to outcome research we might be able to resolve the silent debate in the literature regarding which matters most in predicting therapy outcome: trajectories of early change or pretreatment variables.

Two studies, to our knowledge, have done exactly that; they have simultaneously examined the role of trajectories of change and pretreatment variables in an effort to determine predictors of relapse at follow-up. Santor and Segal (2002) compared initial depressive severity, rates of early change (weeks 1-3), rates of change in the first 10 weeks, and rates of change in the first 20 weeks. They found rates of change in the first 10 weeks predicted symptom return at 3-months over and above initial severity *and* rates of early or later change. More recently, Gilboa-Schechtman and Shahar (2006) found, in their comparative analysis, that rapid rates of change in the first 4 weeks of therapy predicted outcome at 12- and 18-months over and above initial severity and remoralizer status. Given the broad range in weeks since therapy commenced (weeks 4 – 10) in which rapid response significantly predicted outcomes at follow-up, it will be important for future research to identify, with some degree of consistency, by which session rapid change should be expected. In addition, these studies were somewhat limited in that they included only initial symptom severity as a candidate pretreatment predictor of response. Future studies examining a wider range of previously identified predictors of response would make an important contribution to the literature.

For instance, as the push to disseminate CBT into the community rises, concerns have been raised about the role of comorbidity. Specifically, although some studies have identified comorbidity as a predictor of outcome (e.g., Gelhart & King, 2001; Laberge, Gauthier, Cote, & Plamondon; Reich, Warshaw, Peterson, & White, 1995), others have not (e.g., Joormann, Kosfelder, & Schulte, 2005; McLean, Woody, Taylor, & Koch, 1998; Persons, Roberts, & Zalecki, 2003). As comorbidity is now referred to as "the rule rather than the exception" studies examining the extent to which comorbidity predicts rates of early change and outcomes are particularly important. Another understudied approach would be to simultaneously compare the rates of change in both depressive and anxiety symptoms. No studies, to our knowledge, have simultaneously modeled both symptom sets in the context of trying to understand the differential impact of rates of early symptom change on ultimate therapy outcomes.

The Present Study

In sum, predictors of outcome are thought to guide the clinician in treatment decision making to promote successful response. Unfortunately, the literature identifying pretreatment variables as predictors of distal outcomes has yielded inconsistent results making it difficult for practicing clinicians to inform their work in accord with the existing literature. On the other hand, the robust nature of rapid rates of early response as a predictor of outcome has guided the UK's approach to their widely disseminated psychotherapeutic treatment protocol: therapy sessions are delivered twice weekly for the first six weeks in an effort to maximize the potential for a "rapid response". Roz Shafran, "Chair of CBT" in the UK's national effort to "Improve Access to Psychological

Therapies", urged clinical psychologists at the most recent Association of Behavioral and Cognitive Therapies Convention (2009) to "experiment" with upping session dosage early in treatment in an effort to bring about this rapid response given its observed effect on outcome.

However, prior to disseminating an altered approach to session dose, it seems important to answer the following question in the same study: Which matters most in terms of successful response: rate of early symptom change or pretreatment factors? Therefore, the present study sought to move beyond the traditional pre-post group mean method of analysis approach to investigating predictors when examining outcome data from a Psychology training clinic. Instead, a bridging of methodologies was employed to advance the empirical work regarding patterns of symptom change in psychotherapy outcome studies. Specifically, a robust finding in the literature is that rapid early rates of response are predictive of outcome; however, the sudden gain phenomenon characterizes only one possible privileged pathway to recovery leaving much of the sample uncharacterized. Conversely, Latent Growth Curve Modeling (LGCM) fits a model to the sample as a whole that accommodates individual variability in both initial status and growth rate by first generating individual growth curves. Such modeling of symptoms from the early sessions of therapy allowed several hypotheses to be tested including the extent to which baseline factors predicted individual variability in symptom trajectories and whether and to what extent these early rates of change predicted treatment outcome. And, in an attempt to the merge these lines of inquiry, both rates of early symptom

change and previously identified pretreatment variables predictive of response were examined to determine which matters more in terms of achieving good outcomes.

Specific Aims

The aims of this study were five-fold.

Aim 1: To examine trajectories of depressive symptomatology during the first five treatment sessions and the extent to which pretreatment factors relate to these depressive trajectories.

Aim 2: To compare how well depressive symptom trajectories versus pretreatment variables predict ultimate therapy outcome.

Aim 3: To examine trajectories of anxiety symptomatology during the first five treatment sessions and the extent to which pretreatment factors relate to the anxiety trajectories.

Aim 4: To compare how well anxiety symptom trajectories versus pretreatment variables predict ultimate therapy outcome.

Aim 5: This study was also designed to test two hypotheses regarding the relationship between depressive and anxiety symptoms and their change over the course of CBT. Specifically, the following two hypotheses were tested: 1) depressive and anxiety symptoms are more distally related through latent constructs; (2) depressive and anxiety symptoms are more proximally related as evidenced by significant time-varying covariate relationships between symptom scores within each session.

CHAPTER II

METHOD

Participants

Participants were adults (ages 18-64) who voluntarily presented for therapy in a Psychology training clinic at the University of Oregon from the fall of 2005 to the spring of 2009. Of the 269 clients who sought treatment at the clinic (i.e., completed a phone screen), 236 went on to complete an intake. Of those who completed an intake, 14 clients were referred for services elsewhere leaving data from 222 clients in the current sample. Both completers (i.e., clients who reached a "natural" termination from therapy) and those who prematurely terminated were included in analyses.

Procedures

Client Assessment Procedure and Inclusion Criteria. The initial stage of the assessment included a standardized phone screen, administered by our clinic coordinator, to obtain the following information: age, referral source, medication usage, suicidality, counseling history, and presenting problems. Because our clinic does not offer 24-hour crisis support, clients with acute problems (e.g., those who endorsed suicidality and articulated a plan) were connected with a different community agency equipped with

crisis-ready resources. Acute suicidality, extreme psychosis and severe drug or alcohol dependence were essentially the only exclusion criteria for enrollment in therapy.

Clients who were deemed an appropriate fit with our clinic were assigned to a therapist who set up the initial intake appointment. The Semi-Structured Clinical Interview for the Diagnostics and Statistics Manual, Fourth Edition (SCID-I, First, Spitzer, Gibbon, & Williams, 1997) was administered at intake. The interview took 45 to 120 minutes (depending on the complexity of the clinical presentation) and yielded information necessary for making Axis I diagnoses. Clients who demonstrated symptoms consistent with our "exclusion" criteria during the intake were similarly referred out for more appropriate services. All other clients were offered services in the Psychology Training Clinic; these clients constitute the current study sample regardless of the length of time in treatment.

Measures

The Beck Depression Inventory (BDI-I; Beck, Ward, Medelsohn, Mock, & Erlbaugh, 1961; Beck et al, 1979) is a 21-item self-report measure that assesses severity of somatic, affective, cognitive, vegetative, and behavioral symptoms associated with depression. Each item is scored on a 0 (no symptoms) to 3 (very severe symptoms) scale. Psychometric properties of the BDI have been well documented (Beck, Steer & Garbin, 1988). Cronbach alphas from the present study demonstrated strong internal reliability over time (range in α: .91 - .94). The total score (ranging from 0 to 63) served as one of two primary symptom and outcome measures for this study as it was obtained each session the client met with the therapist. Higher total scores indicate greater depressive

symptom severity. Intake and the first five treatment session scores were included in the growth modeling. The following cutoffs were employed for interpretation of total scores (Beck et al., 1961): 0-9 = not depressed, 10-18 = mild-moderate depression, 19-29 = moderate-severe depression, 30-63 = severe depression.

The Beck Anxiety Inventory (BAI; Beck & Steer, 1993) is a 21-item self-report measure assessing severity of anxiety symptoms. Each item is scored on a 0 (no symptoms) to 3 (very severe symptoms) scale. The psychometric properties of the BAI have been well supported by the literature (Beck, Epstein, Brown, Steer, 1988). Cronbach alphas from the present study demonstrated strong internal reliability over time (range in α: .90 - .93). The total score (0 to 63) served as the other primary outcome for the study as it was obtained each session the client met with the therapist. Higher scores indicate greater anxiety symptom severity. Intake and the first five treatment session scores were included in the modeling. The abovementioned BDI cutoffs for assigning a descriptive label were used with the BAI scores as well.

The Stages of Change Schedule (SOCS; McConnaughy, Prochaska, & Velicer, 1983) is a 32-item self-report measure. Eight items correspond to each of the four subscales that represent the transtheoretical model thought to measure readiness to change: Precontemplation, Contemplation, Action, and Maintenance. A Likert scale from 0 (strongly disagree) to 5 (strongly agree) is used to respond to each item. The SOCS has demonstrated adequate internal consistency with adults (coefficient alphas: .75 to .87; McConnaughy, Prochaska, & Velicer, 1983). Multiple scoring procedures for the stages of change questionnaire (DiClemente, Schlundt, & Gemmell, 2004) are available in the

literature. McConnaughy et al., (1989) employed cluster analysis to produce client/patient profiles whereas DiClemente and colleagues (2004) calculated a "readiness score" as an alternative way to approximate stage status. Though scoring procedures such as cluster analysis and DiClemente's "readiness score" may utilize the information gleaned from the SOCQ in a manner consistent with the theoretical framework for the stages of change, these methods are limited in terms of: (1) interpretability, (2) ability for cross study comparisons, and (3) clinical utility. To address these limitations, the current study employed a third alternative approach to scale scoring (Dozois, Westra, Collins, Fung, & Garry, 2004; Lewis, Simons, Silva et al., 2009; Rogers, Martin, Anthongy, Massaro, Danley, Crean, & Penk, 2001) whereby four separate readiness to change scores were derived through simple summing subscale items.

The Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978) is a 40-item self report scale that measures patterns of maladaptive thinking or "depressogenic schema" thought to constitute a cognitive diathesis to depression. A Likert scale from 1 (totally disagree) to 7 (totally agree) is used to respond to each item. The scale includes two subscales: Achievement and Interpersonal; however, the psychometrics of the DAS suggest the measure is best used as a whole (coefficient alpha: .90; Oliver, & Baumgart, 1985). Higher scores indicate more dysfunctional attitudes. Total and subscale scores at intake were included in predictor analyses.

The Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974) is a 20-item T/F self report measure of pessimism; higher scores indicate greater hopelessness. The psychometric properties suggest high internal consistency (coefficient

alpha: .93). The scale has demonstrated sensitivity to changes in depression over time. Total scores at intake were included in predictor analyses.

The Global Assessment of Functioning (GAF; Spitzer R. L., Gibbon M., Williams J. B. W., & Endicott J. 1996) is a scale used by therapists to rate the social, occupational, and psychological functioning of the client from 1 to 100. Higher scores indicate greater functioning. Psychometric properties suggest moderate reliability of the GAF (coefficient alpha: .74; Hilsenroth, Ackerman, & Blagys, 2000). Intake GAF scores were included in predictor analyses.

Comorbidity. Clients were assigned to one of three comorbidity groups based on the number of diagnoses assigned from the SCID-I: (1) No Diagnosis; (2) Single Disorder; (3) Comorbid Disorders. That is, clients who did not meet criteria for an Axis I diagnosis (i.e., they demonstrated sub-threshold symptoms) were included in the "No Diagnosis" group. Clients who met criteria for a single disorder (e.g., Major Depressive Disorder, Social Phobia, Posttraumatic Stress Disorder) were included in the "Single Disorder" group. Finally, clients who met criteria for more than one DSM-IV disorder (e.g., Major Depressive Disorder and Social Phobia) were included in the "Comorbid Disorders" group. Comorbidity groupings were considered a potential covariate in subsequent analyses.

Medication Status. During the initial phone screen, clients were asked whether they were currently taking psychotropic medication to treat their condition. Client responses were coded either "yes" or "no". Medication status was considered a potential covariate in subsequent analyses.

Responders versus Non-responders. The criteria put forth by Jacobson, Roberts, Berns, and McGlinchey (1999) for clinically significant change as per the reliable change index were used to classify responders versus non-responders. As such, clients who experienced at least a 50% reduction in BDI scores or who moved to a BDI score of less than or equal to 10 were considered responders. Because the BAI is based upon the same scale, the same criteria were applied to determine responders versus non-responders. If clients recovered on one measure (e.g., BDI) but not the other (e.g., BAI) they were considered partial responders. This variable was coded to allow for inclusion in analyses as a continuous measure of outcome: 0 = Non-responder, 1 = Partial Responder, 2 = Responder.

Treatment

Therapy and Training. Therapists who participated in the practicum were trained to deliver CBT and to inform their therapy using a cognitive case conceptualization. Training consisted of weekly didactics and role play in addition to either individual or group supervision. Typically in their first year of practicum therapists received individual supervision beginning with one depression only case (or one anxiety only case) using a standard CBT protocol. Therapists' supervisors increased therapist caseloads based upon therapist readiness and skillfulness reaching a maximum of four individual cases within the first year. Therapists choosing to complete a second, third, fourth, etc. year of practicum could have been assigned to individual or group supervision and could have received additional training in CBT variants such as Acceptance and Commitment Therapy (ACT). Neither therapist competency nor fidelity to treatment was measured.

Therapeutic delivery was expected to be more flexible in terms of focus and also in terms of number of sessions as compared to RCTs following a manualized protocol.

Therapists

All therapists were clinical psychology doctoral students at the University of Oregon enrolled in the CBT practicum. Thirty-three therapists in their 2nd through 6th year of the doctoral program delivered CBT to an ongoing caseload of 4 clients. Therapists were predominantly female (90.04%) and the average number of months of CBT training at the initial session with the client was 14.63 (SD = 10.69). Unfortunately, therapists' previous and additional experiences data were not collected. *Therapist level of training* was measured in months since the therapist entered practicum and was included as a covariate in all subsequent analyses.

Data Analytic Plan

The five primary aims of the study were addressed through separate analytic steps. As stated, the first aim was to examine trajectories of depressive symptomatology during the first five treatment sessions and the extent to which pretreatment factors relate to these depressive trajectories. A second aim was to compare how well depressive symptom trajectories versus pretreatment variables predict ultimate therapy outcome. Aims 3 and 4 were identical to aims 1 and 2 with the exception that anxiety symptoms were the focus of investigation. Finally, a fifth aim of this study was to test two hypotheses regarding the relationship between depressive and anxiety symptoms and their change over the course of CBT.

Analytic Step 1 required baseline analyses to (a) broadly characterize the sample, (b) confirm/disconfirm suspect covariates (i.e., therapist level of training, comorbidity and medication status), (c) determine the extent to which collinearity existed among prospective predictors, and (d) assess the degree to which the symptom scores reflected multivariate normality. Analytic Step 2 involved Latent Growth Curve Modeling (LGCM) to identify the best fit model characterizing the early trajectory of client depressive and anxiety symptoms. Analytic Step 3 examined predictors of trajectories of early change in an attempt to explain any heterogeneity observed in initial status and growth rate of depressive and anxiety symptom severity. Analytic Step 4 included the combination of latent growth factors representing trajectories of early change *and* pretreatment factors to determine which factors "mattered most" in predicting outcome. Finally, Analytic Step 5 involved simultaneous modeling of depressive and anxiety symptom change through (a) parallel and (b) time-varying covariate models.

Statistical Analyses

Baseline analyses were run using SAS Version 9.1 (SAS Institute, Cary, NC). Specifically, general linear models (GLM) were used to compare the comorbidity groups (i.e., No Diagnosis, Single Disorder, and Comorbid Disorders) on depressive and anxiety symptom severity at baseline and across the first five treatment sessions, and *a posteriori* Student *t*-tests were conducted if an omnibus test was significant at the .05 level. Non-directional statistical tests were employed. Student *t*-tests were conducted to compare depressive and anxiety symptom severity across medication status. Bivariate Pearson product-moment correlations were run to examine the relation between baseline clinical,

cognitive, and contextual variables. Cohen's (1988) interpretation of correlation size was adopted: .5 = "large", .3 = "moderate", .1 = "small".

LGCM was run using MPlus 5.1 (Muthen & Muthen, 2007). The full information maximum likelihood algorithm was invoked to handle missing data in all analyses and expectation maximization served as the algorithm for maximization. This approach estimates missing data using all observed information available (e.g., outcomes, covariates) in the model. Client attrition, referral out, and variability in therapist behavior limited the completeness of the depressive and anxiety symptom assessments. Complete BDI and BAI data across all time points was available on 139 (62.2%) and 141 (63.5%) clients, respectively. The mean- and variance-adjusted Chi-square and degrees of freedom (χ^2, Bollen, 1989), as well as the Comparative Fit Index (CFI, Bentler, 1990) and Root Mean Squared Error of Approximation (RMSEA, Steiger, 1990), were used to assess goodness of model fit. Specifically, non-significant Chi-square values, and values greater than .95 for CFI and less than .06 for RMSEA (Hu & Bentler, 1999) served as benchmarks for adequate fitness. All regression models were run in MPlus and included in the overall growth curve modeling framework. Therefore, model fitness could be reassessed and compared to the best fitting baseline model using the Chi-square difference test.

CHAPTER III

RESULTS

Analytic Step 1.1: Baseline Analyses

Baseline Demographics. Clients ($N = 222$) receiving therapy in our training clinic were students (68.02%) and community members (31.98%). Clients were predominantly Caucasian (92.79%) ranging in age from 18 to 64 years ($M = 27.85$, $SD = 11.28$). Two-thirds of the sample were female (65.32%). Nearly 10% of clients did not meet criteria for an Axis I disorder ($n = 21$), 34.23% met criteria for a single diagnosis ($n = 76$), and 53.60% met criteria for comorbid diagnoses ($n = 119$). Twenty-two percent of clients endorsed suicidality and 40.28% reported taking medication at intake. Overall, mild-moderate depressive and anxiety symptom severity characterized symptomatology at intake. Global Assessment of Functioning scores suggested the sample was experiencing moderate impairment. Average levels of hopelessness were minimal: the mean score ($M = 7.33$, $SD = 4.92$) was below the clinical cutoff of 9. The highest subscale score of the Stages of Change Scale indicates the best description of the sample's readiness to change at intake. As such, baseline scores indicated the sample, on average, could be characterized as contemplative, suggesting that clients were aware of a distressing life

situation, but were not yet ready to fully take action toward change. See Table 1 for a complete list and breakdown of baseline demographics.

Preliminary Analyses with Suspect Covariates. GLMs assessing group differences in depressive and anxiety symptom severity for each of the first five treatment sessions were run to determine whether comorbidity status (i.e., No Diagnosis, Single Diagnosis, Comorbid Diagnosis) should be included as a covariate in subsequent analyses. The omnibus GLM confirmed that both the BDI and BAI differed between comorbidity groups. *A posteriori* Student *t*-tests revealed that clients with comorbid disorders demonstrated higher BDI scores each session from intake to session four with a trend (p = .069) in the same direction at session five (see Table 2). The same pattern was mirrored by the relationship between comorbidity status and BAI scores with the exception that clients with comorbid diagnoses continued to demonstrate significantly higher BAI scores at session five. These preliminary analyses confirmed the need to control for comorbidity status in all subsequent analyses.

It was decided, *a priori*, to include therapist level of training as a covariate, given the nature of the training clinic. That is, perceived client complexity, severity, and/or chronicity were intentionally matched to therapist level of training. Surprisingly, baseline analyses did not reveal substantive evidence that therapist level of training was indeed related to baseline markers of severity or complexity. Rather, therapist level of training was only moderately correlated with baseline anxiety symptom severity, but not depressive symptom severity or comorbidity groupings (see Table 3). Despite this, the decision to include therapist level of training as a covariate in all growth models was

Table 1

Baseline Characteristics of the Sample According to Comorbidity Groupings

Analysis	Baseline Characteristic	Total Sample ($N = 222$)	No Axis I Diagnosis ($N = 21$)	Single Diagnosis ($N = 78$)	Comorbid Diagnoses ($N = 131$)
GLM					
	Age	27.85 (11.28)	28.09 (12.29)	27.23 (10.95)	28.19 (11.39)
	Depressive severity	16.96 (8.99)	$13.48\ (9.29)_{a}$	$14.02\ (8.27)_{a}$	$19.41\ (8.68)_{b}$
	Anxiety severity	17.78 (11.07)	$13.86\ (10.43)_{a}$	$14.44\ (8.99)_{a}$	$20.54\ (11.63)_{b}$
	Hopelessness	7.34 (4.92)	$7.71\ (5.78)_{ab}$	$6.20\ (4.28)_{b}$	$7.96\ (5.03)_{a}$
	Precontemplation	1.53 (0.44)	1.53 (0.38)	1.52 (0.46)	1.53 (0.44)
	Contemplation	4.48 (0.40)	4.42 (0.50)	4.46 (0.38)	4.51 (0.40)
	Action	3.92 (0.55)	3.95 (0.52)	3.90 (0.56)	3.93 (0.55)
	Maintenance	3.41 (0.81)	3.62 (0.94)	3.29 (0.71)	3.43 (0.84)
	DAS	141.44 (31.71)	147.45 (33.61)	$132.73\ (27.58)_{b}$	$145.78\ (32.87)_{a}$
	DAS Achievement	47.06 (16.36)	49.05 (18.16)	$42.47\ (13.33)_{a}$	$49.54\ (17.23)_{b}$
	DAS Interpersonal	44.29 (9.87)	45.67 (8.92)	42.58 (9.04)	45.09 (10.45)
	GAF	61.20 (10.28)	$73.50\ (8.31)_{a}$	$64.11\ (9.39)_{b}$	$56.41\ (8.17)_{c}$
χ^2 tests					
	Gender: female	145 (65.32)	15 (71.43)	46 (58.97)	84 (68.29)
	Caucasian	206 (92.79)	20 (100)	70 (89.74)	116 (94.31)
	Asian	4 (1.80)	0	2 (2.56)	2 (1.63)
	Biracial	4 (1.80)	0	2 (2.56)	2 (1.63)
	Other	8 (3.60)	1 (4.76)	6 (7.68)	5 (4.07)
	Student: yes	151 (68.02)	15 (71.43)	53 (67.95)	83 (67.48)
	Suicidality: yes	47 (21.17)	6 (28.57)	15 (19.23)	26 (21.14)
	Medication: yes	87 (40.28)	5 (28.57)	34 (44.74)	48 (40.34)

Note. Mean (*SD*), $^{*}p < .05$. DAS = Dysfunctional Attitudes Scale. GAF = Global Assessment of Functioning. *GLM* = General Linear Model. Post hoc *t*–tests are run when the omnibus test is significant at $p < .05$. Means in the same row that do not share subscripts are significantly different at $p < .05$.

Table 2

Means and Standard Deviations of Depressive and Anxiety Symptom Severity Across Comorbidity Groupings

Session #	No Diagnoses	Single Diagnoses	Comorbid Diagnoses
IDT	13.48 (9.29)$_a$	14.02 (8.27)$_a$	19.41 (8.68)$_b$
S1DT	8.50 (5.87)$_a$	10.03 (7.49)$_a$	14.23 (8.61)$_b$
S2DT	7.38 (4.21)$_a$	9.31 (6.68)$_a$	13.48 (8.83)$_b$
S3DT	8.57 (5.69)$_a$	7.79 (5.87)$_a$	12.38 (7.83)$_b$
S4DT	5.15 (4.83)$_a$	8.49 (6.47)$_a$	12.75 (7.82)$_b$
S5DT	6.67 (4.89)	8.50 (6.61)	10.86 (7.92)
IAT	13.86 (10.43)$_a$	14.44 (8.99)$_a$	20.54 (11.63)$_b$
S1AT	11.25 (8.74)$_a$	10.23 (7.39)$_a$	17.10 (10.24)$_b$
S2AT	11.37 (7.98)$_a$	10.38 (7.95)$_a$	15.41 (9.39)$_b$
S3AT	9.40 (8.23)$_a$	9.04 (7.37)$_a$	14.35 (9.45)$_b$
S4AT	9.15 (9.35)$_a$	8.99 (7.08)$_a$	15.79 (11.17)$_b$
S5AT	8.58 (7.94)$_a$	9.31 (7.47)$_a$	13.81 (10.06)$_b$

Note. I = Intake. DT = Depression Total. AT = Anxiety Total. Means in the same row that do not share subscripts are significantly different at $p < .05$.

Table 3

Bivariate Correlations: Therapist Level of Training, Symptom Severity, Comorbidity

	T_train	BAI	BDI	CG
T_train	1.00	0.22**	0.11	0.089
BAI		1.00	0.40***	0.26***
BDI			1.00	0.28***
CG				1.00

Note. T_train = Therapist Level of Training. BDI = Beck Depression Inventory. BDI = Beck Anxiety Inventory. CG = Comorbidity Grouping.

retained because of the likelihood that therapist level of training was related to a clinically relevant variable, such as chronicity, not captured by our dataset but potentially important in terms of client response.

The final covariate under consideration--medication status (yes/no) at intake--was, however, significantly related to baseline depressive and anxiety symptom severity: reported use of psychotropic medication was related to elevated baseline symptom severity. These significant differences were observed throughout the first five treatment sessions (Table 4). Therefore, medication status was included as a covariate in all subsequent models.

Table 4

Depressive and Anxiety Symptom Severity across Medication Status

	Medication Status	
Variables	Yes (n = 87)	No (n = 129)
IDT	19.78 (8.09)$_a$	14.97 (7.34)$_b$
S1DT	13.82 (7.93)$_a$	11.28 (8.18)$_b$
S2DT	12.44 (7.98)	10.50 (7.91)
S3DT	11.10 (7.51)	9.74 (6.71)
S4DT	11.28 (7.03)	9.95 (7.51)
S5DT	10.67 (7.68)	9.10 (7.24)
IAT	20.44 (12.54)$_a$	15.94 (9.78)$_b$
S1AT	16.64 (11.23)$_a$	12.90 (8.80)$_b$
S2AT	14.66 (10.16)	12.15 (8.29)
S3AT	13.47 (9.78)	11.02 (8.54
S4AT	15.09 (11.60)$_a$	11.47 (9.47)$_b$
S5AT	13.99 (10.48)	10.73 (8.78)

Note. I = Intake. DT = Depression Total. AT = Anxiety Total.

Correlational Analyses. Baseline depressive symptom severity was moderately correlated with anxiety symptom severity and hopelessness. Small, but significant positive correlations were observed between depressive symptom severity and the following pretreatment variables: contemplation stages of change, DAS, the DAS achievement subscale, and age. Both depressive and anxiety symptom severity were

moderately negatively correlated with global functioning. Large significant correlations were observed between the DAS subscales. Moderate positive correlations were observed between the following SOCS subscales: contemplation, action, and maintenance. See Table 5 for a complete listing of bivariate correlations between baseline variables.

Mean Levels of Depressive and Anxiety Symptoms over the First Five Treatment Sessions. Sample means from the first five treatment sessions were plotted to examine trajectories and used to guide subsequent modeling of both BDI and BAI. Figure 1 suggested that both sets of symptom scores (BDI and BAI) followed a roughly linear trajectory indicating that a strict linear slope would indeed serve as an appropriate baseline model for comparison (see Table 6 for means and *SDs*). Each of the first six BDI and BAI total scores (from intake through session 5) were examined to see if multivariate normality as assumed by maximum likelihood estimations was met. Although slight skewness was observed on each of the symptom scores, this deviation from normality was not sufficient to warrant a transformation that would have rendered results less interpretable (Table 7). BDI and BAI over the first five treatment sessions were all moderately to highly correlated (Table 8).

Analytic Step 2.1: Aim 1

Trajectories of Early Change. Latent growth curve modeling (LGCM) was employed in order to characterize the trajectories of client response to treatment over the first five treatment sessions. Specifically, a series of *a priori* specified growth models were examined to identify the best-fitting and most parsimonious model characterizing individual differences in initial status and change in depressive and anxiety symptoms

Table 5

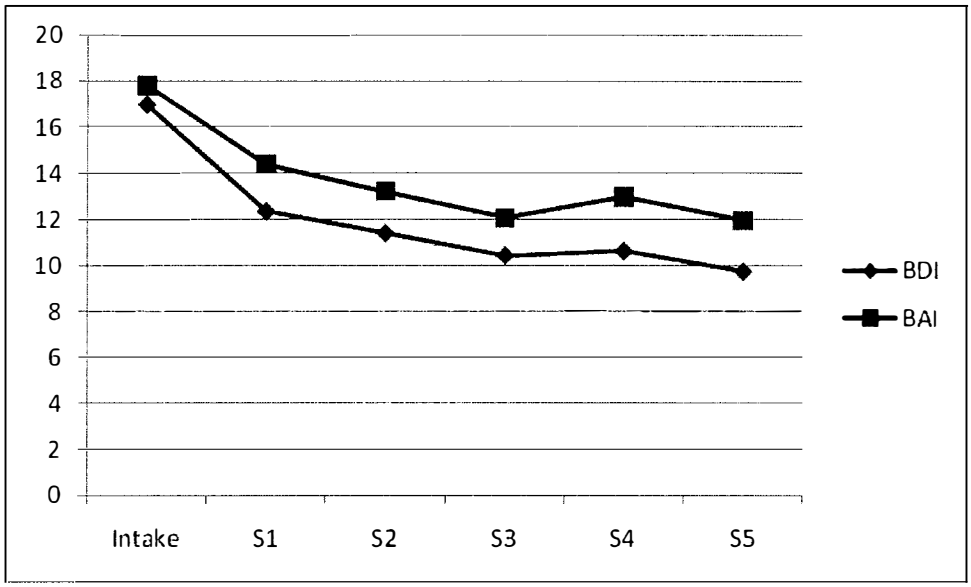
Correlations between Baseline and Outcome Variables

Variables	BDI	BAI	Age	BHS	Pre	Cont	Action	Maint	DAS	DAS Ach.	DAS Int.	GAF
BDI	1.00	.40***	.14*	.33***	-.11	.15*	-.075	.10	.17*	.21**	.10	-.44***
BAI		1.00	-.026	.08	.012	.079	.019	.06	.13+	.13+	.10	-.36***
Age			1.00	.18**	-.091	.022	-.031	.065	-.17*	-.10	-.21**	-.17*
BHS				1.00	-.17*	.05	-.38***	.0015	.42***	.43***	.30***	-.19*
Pre					1.00	-.049	-.082	-.071	.051	.086	-.083	-.043
Cont						1.00	.39***	.27***	.075	.099	.12+	-.03
Action							1.00	.37***	-.21**	-.22**	-.098	.092
Maint								1.00	.11	.10	.11	.022
DAS									1.00	.94***	.84***	-.07
DAS Ach.										1.00	.67***	-.11
DAS Int.											1.00	.0060
GAF												1.00

Note. Mean (*SD*), * $p < .05$, ** $p < .01$, *** $p<.001$. BDI = Beck Depression Inventory. BAI = Beck Anxiety Inventory. BHS = Beck Hopelessness Scale. Pre = Precontemplation Stage of Change. Cont = Contemplation Stage of Change. Action = Action Stage of Change. Main = Maintenance Stage of Change. DAS = Dysfunctional Attitudes. Ach. = DAS Achievement Subscale. Int. = DAS Interpersonal Subscale. GAF = Global Assessment of Functioning.

Figure 1

BDI and BAI Trajectories during the First Five Treatment Sessions



over the first five treatment sessions. The first step specified unconditional two growth factor models. That is, the only parameters included in the modeling were the mean and variance of both the intercept and slope that were allowed to randomly vary within the person (Muthén & Muthén, 1999), no restrictions were imposed, and a test of the assumption that BDI scores followed a strict linear trajectory was conducted. If fit was less than adequate, then linear spline models were estimated and finally, a quadratic growth factor included. Spline models constrain at least two time points in the model and allow the remaining time points to freely vary (Meredith & Tisak, 1990); this method accommodates nonlinearity that may be demonstrated by individuals in the sample. In the current application, intercept loadings were fixed to one and the first two loadings for the slope factor were constrained to zero and one, respectively, with the remaining loadings free to be estimated. Linear spline models allow for flexibility in the linear trajectory by

modeling piecewise curves or crooked lines in which the straight line segments do not necessarily map onto the overall straight line while testing the assumption that a linear trend is the most accurate characterization of the BDI scores. Linear spline models confound both shape (i.e., nonlinear) and slope (i.e., trend upward or downward) within the growth rate factor estimate given the flexibility described above (see Stoolmiller, 1995 for more technical details). The intercept and slope for each growth model were regressed on the following covariates in all modeling estimates: therapist level of training (months since entry to practicum), medication status (yes/no), and comorbidity status (No Diagnosis, Single Diagnosis, Comorbid Diagnosis).

Table 6

BDI and BAI Descriptive Statistics

Measure	Time	N	Mean	(*SD*)	Minimum	Maximum
BDI	Intake	220	16.96	8.99	0	45.5
	Session 1	178	12.36	8.33	0	45.0
	Session 2	172	11.41	8.08	0	49.0
	Session 3	165	10.44	7.34	0	35.5
	Session 4	151	10.63	7.58	0	37.0
	Session 5	144	9.74	7.41	0	35.0
BAI	Intake	219	17.78	11.07	0	59.0
	Session 1	177	14.40	9.81	0	62.0
	Session 2	171	13.24	9.06	0	48.0
	Session 3	162	12.08	9.02	0	48.0
	Session 4	149	12.98	10.35	0	52.0
	Session 5	142	11.97	9.39	0	50.0

Note. BDI = Beck Depression Inventory. BAI = Beck Anxiety Inventory.

Table 7

Skewness and Kurtosis Statistics: BDI and BAI

	BDI		BAI	
Session #	Skewness	Kurtosis	Skewness	Kurtosis
Intake	0.43	-0.10	0.85	0.53
Session 1	0.98	1.69	1.15	2.57
Session 2	1.06	2.23	1.05	1.00
Session 3	0.93	0.77	1.13	1.31
Session 4	0.74	0.16	1.24	1.58
Session 5	0.91	0.54	1.34	1.98

Note. BDI = Beck Depression Inventory. BAI = Beck Anxiety Inventory.

The strict linear and quadratic models were rejected as they fit the data poorly as evidenced by significant Chi Square values, CFI lower than .95, and RMSEA greater than .06. However, the linear spline model in which the residual covariances of the BDI scores were constrained to be equal was chosen as these additional constraints made the model most parsimonious with fit statistics in the desired range: χ^2 (23) = 28.522, p = .19; CFI = 0.994; RMSEA = .033. Figure 2 provides a graphical representation of the final, best-fit linear spline growth curve model. The *R*-square statistics depicted in the figure indicate the proportion of variance in the BDI scores accounted for by the growth factors is relatively high suggesting that the linear spline slope/shape does well in reproducing the observed growth curves.

The model suggests that, on average, the depressive symptom trajectories began at a moderate severity and fairly rapidly decreased over the first five treatment sessions (see Table 9 for coefficient estimates). Significant variability was observed in both initial status and growth rate. The non-significant result of the test of zero covariance between the intercept and slope suggests that the client's initial status of depressive symptom

Table 8

Bivariate Correlations: BDI and BAI

Variables	IDT	S1DT	S2DT	S3DT	S4DT	S5DT	IAT	S1AT	S2AT	S3AT	S4AT	S5AT
IDT	1.00	.69***	.68***	.61***	.63***	.51***	.40***	.37***	.36***	.32***	.32***	.51***
S1DT		1.00	.84***	.76***	.69***	.64***	.38***	.54***	.45***	.40***	.30**	.64***
S2DT			1.00	.85***	.78***	.71***	.30***	.43***	.51***	.46***	.38***	.71***
S3DT				1.00	.84***	.76***	.26**	.40***	.46***	.51***	.44***	.76***
S4DT					1.00	.78***	.32***	.37***	.45***	.44***	.48***	.78***
S5DT						1.00	.18*	.26**	.31**	.33***	.41***	.45***
IAT							1.00	.63***	.64***	.66***	.63***	.62***
S1AT								1.00	.68***	.62***	.60***	.50***
S2AT									1.00	.84***	.73***	.73***
S3AT										1.00	.83***	.76***
S4AT											1.00	.74***
S5AT												1.00

Note. Mean (*SD*), * $p < .05$, ** $p < .01$, *** $p<.001$. DT = Beck Depression Inventory Total Score. AT = Beck Anxiety Inventory Total Score. I = Intake. S = Session.

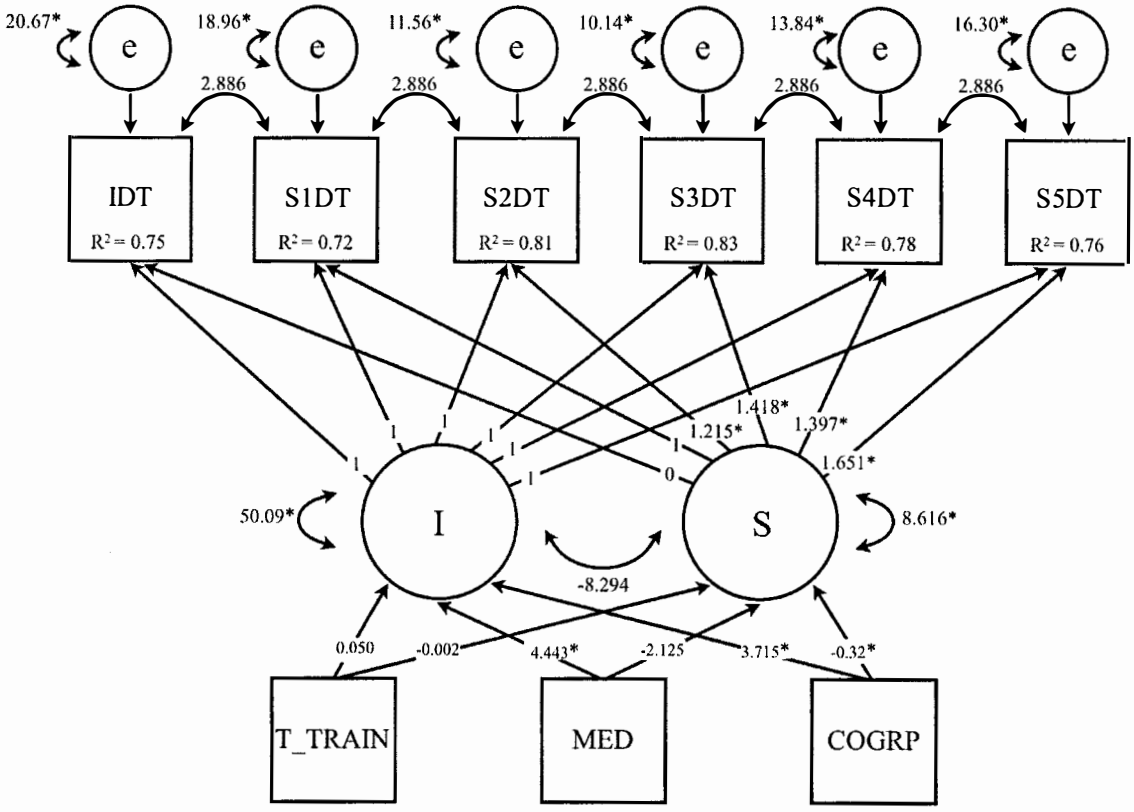
severity has no influence on the rate or direction of change in depressive symptom severity over the next five sessions of treatment. Concomitant psychotropic use and comorbidity were positively associated with baseline levels of depressive symptom severity whereas level of therapist level of training was not, when controlling for all covariates. Only baseline medication status predicted rate of change in BDI symptoms early in therapy: concomitant psychotropic use was related to an accelerated decrease in depressive symptomatology over the first five treatment sessions (see Table 10 for regression coefficients). The *R*-square statistic for the latent curve parameters suggests that the covariates significantly accounted for variation in the intercept ($R^2 = 0.194, p = .001$) but not the slope ($R^2 = 0.119, p = .139$).

Analytic Step 3.1

Pretreatment Predictors of Trajectories of Early Change. We examined whether and to what extent pretreatment variables explained the significant heterogeneity in the initial status (intercept) and growth rate (slope) of depressive symptomatology over the first five treatment sessions. Based on previously identified predictors of psychotherapy outcome for depression and anxiety, 16 pretreatment variables from three categories were explored: (1) Clinical variables: comorbidity, depression symptom severity, anxiety symptom severity, and global functioning; (2) Cognitive variables: hopelessness, readiness to change (i.e., four subscales: precontemplation, contemplation, action, maintenance), and dysfunctional attitudes (overall DAS and its two subscales: achievement and interpersonal); and, (3) Contextual variables: age, gender, student status, and medication status. First, each time-invariant variable was examined in a univariate

Figure 2

Graphical Representation of Best-Fit BDI Model



Note. e = residual variance. IDT = Intake BDI total score. S1DT = Session 1 Depression Total Score and so on. I = Intercept. S = Slope. T_Train = Therapist Level of Training. Med = Medication Status. CoGrp = Comorbidity Grouping. * $p < .05$.

Table 9

Parameter Estimates for BDI Growth Models with Covariates

	Mean of level	Variance of level	Mean of change	Parameter Variance of change	Covariance: level and change
Model	Coeff. (*SE*)	Coeff. (*SE*)	Coeff. (*SE*)	Coeff. (*SE*)	Coeff. (*SE*)
Linear spline with time score residual covariances fixed to equal	8.99*** (1.56)	50.09*** (8.16)	-3.22** (0.95)	8.62* (4.07)	-8.29 (4.99)+

Note. BDI = Beck Depression Inventory. Coeff. = Coefficient. *SE* = Standard Error. *df* = degrees of freedom. *** $p < .0001$. ** $p < .001$, * $p < .05$, + $p < .1$.

Table 10

Covariate Regression Coefficients in Best-Fitting BDI Model

Covariate	Latent Factor	Coefficient (SE)	*p*-value
Therapist Level of Training	Intercept	0.050 (0.056)	.37
Medication Status		4.44 (1.17)	<.0001
Comorbidity Status		3.71 (0.85)	<.0001
Therapist Level of Training	Slope	-0.0002 (-.034)	.96
Medication Status		-2.13 (0.69)	.002
Comorbidity Status		-0.32 (0.51)	.53

Note. SE = Standard Error

growth model. Second, a multivariate analysis including all pretreatment variables was run to examine the extent to which each variable explained variability above and beyond other predictors included in the model. The best-fit linear spline with the inclusion of each regression equation continued to fit the data well as evidenced by fit statistics in the desired range and no significant deterioration in fit as per the Chi-square fitness test.

The univariate analyses (including the three covariates: therapist level of training, medication status, comorbidity status) identified the following variables as predictors of

BDI initial status: comorbidity status, medication status, anxiety symptom severity, student status, global functioning, hopelessness, dysfunctional attitudes, and achievement-oriented dysfunctional attitudes (see Table 11). Specifically, comorbidity (as opposed to having no or a single diagnosis), concomitant psychotropic usage, and community member (as opposed to student) status were all related to greater depressive symptom severity at intake. Similarly, hopelessness, higher dysfunctional attitudes, and specifically, achievement-oriented dysfunctional attitudes were related to greater depressive symptom severity at intake.

Only three pretreatment factors were related to growth rate in these univariate predictor analyses. First, concomitant psychotropic usage predicted a steeper decline in depressive symptomatology over the first five sessions of treatment. Second, higher initial levels of a contemplative orientation to change predicted an accelerated decline in depression symptomatology. Third, having more initial interpersonally-oriented dysfunctional attitudes predicted a steeper decline in growth rate of BDI scores.

Next, a multivariate predictor analysis that included all pretreatment factors was run (see Table 12). This model identified the following as significant predictors of initial status: concomitant psychotropic usage, anxiety symptom severity, community member status, hopelessness, and global functioning. Comorbidity and dysfunctional attitudes dropped out of the multivariate model as predictors of BDI initial status. Concomitant psychotropic usage, student status, and a contemplative orientation to change were identified as significant predictors of an accelerated decline in depressive symptomatology in the multivariate analyses.

Table 11

Parameter Estimates for Time-Invariant Predictors and Covariates in Univariate Analyses with Covariates

Covariate		BDI Linear Spline Estimate (*SE*)	*p*-value	BAI Linear Spline Estimate (*SE*)	*p*-value
Therapist Level of Training	Initial status	0.050 (0.056)	.37	0.17 (0.068)	.014
	Growth rate	-0.002 (0.034)	.96	-0.075 (0.040)	.059
Comorbidity (0 = no, 1 = single disorder, 2 = comorbid)	Initial status	3.71 (0.85)	<.0001	3.95 (1.03)	<.0001
	Growth rate	-0.32 (0.51)	.53	-0.34(0.53)	.51
Medication Status (0=no, 1=yes)	Initial status	4.44 (1.17)	<.0001	3.88 (1.40)	.006
	Growth rate	-2.13 (0.69)	.002	-1.23 (0.74)	.096
Predictor	Regression	Estimate (*SE*)	*p*-value	Estimate (*SE*)	*p*-value
Depressive Severity	Initial status	---	---	0.41 (0.078)	<.0001
	Growth rate	---	---	-0.098 (0.044)	.025
Anxiety Severity	Initial status	0.27 (0.052)	<.0001	---	---
	Growth rate	-0.042 (0.033)	.20	---	---
Gender	Initial status	0.40 (1.18)	.73	1.54 (1.43)	.28
	Growth rate	-0.56 (0.69)	.42	0.062 (0.74)	.93
Age	Initial status	0.078 (0.051)	.12	-0.068 (0.062)	.27
	Growth rate	0.017 (0.031)	.58	0.060 (0.035)	.085
Student status (0=no, 1=yes)	Initial status	-3.42 (1.20)	.004	0.38 (1.47)	.79
	Growth rate	0.55 (0.73)	.45	-0.96 (0.76)	.21
Functioning	Initial status	-0.32 (0.068)	<.0001	-0.28 (0.086)	.001
	Growth rate	0.004 (0.043)	.94	0.015 (0.045)	.74
Hopelessness	Initial status	0.55 (0.12)	<.0001	0.085 (0.15)	.56
	Growth rate	0.007 (0.070)	.92	0.059 (0.075)	.44
Precontemplation	Initial status	-2.32 (1.32)	.08	-0.035 (0.081)	.66
	Growth rate	1.14 (0.78)	.14	0.29 (0.24)	.23
Contemplation	Initial status	2.76 (1.43)	.053	2.27 (1.73)	.19
	Growth rate	-1.89 (0.83)	.024	-2.45 (0.88)	.005
Action	Initial status	-1.18 (1.06)	.26	1.75 (0.29)	.56

Table 11 (Continued)

Covariate		BDI Linear Spline Estimate (*SE*)	*p*-value	BAI Linear Spline Estimate (*SE*)	*p*-value
	Growth rate	0.21 (0.64)	.74	-0.062 (0.66)	.92
Maintenance	Initial status	0.80 (0.72)	.26	0.062 (0.87)	.94
	Growth rate	0.092 (0.42)	.83	0.68 (0.44)	.12
Dysfunctional Attitudes	Initial status	0.042 (0.018)	.023	0.037 (0.023)	.11
	Growth rate	-0.017 (0.011)	.13	0.00(0.012)	.98
DAS: Achievement	Initial status	0.10 (0.035)	.004	0.063 (0.044)	.16
	Growth rate	-0.029 (0.021)	.17	0.013 (0.023)	.58
DAS: Interpersonal	Initial status	0.095 (0.059)	.11	0.12 (0.073)	.10
	Growth rate	-0.075 (0.036)	.036	-0.059 (0.040)	.14

Note. All "univariate" analyses contain therapist level of training (measured in months since start of practicum), client comorbidity status (0=No Diagnosis, 1 = Single Diagnosis, 2 = Comorbid Diagnoses), and medication status (yes/no) as covariates (i.e., predictors of both intercept and slope) in order to control for these effects

Table 12

Parameter Estimates for Time-Invariant Predictors in Multivariate Analyses

Regression	Predictor	BDI Linear Spline Estimate (*SE*)	*p*-value	BAI Linear Spline Estimate (*SE*)	*p*-value
Initial Status	Therapist Level of Training	0.002 (0.051)	.98	0.16 (0.065)	.014
	Medication Status	2.66 (1.03)	.010	2.24 (1.34)	.096
	Comorbidity	1.01 (0.87)	.25	0.80 (1.14)	.48
	Depressive Severity	---	---	0.39 (0.086)	<.0001
	Anxiety Severity	0.21 (0.049)	<.0001	---	---
	Gender	0.10 (1.03)	.92	2.09 (1.33)	.12
	Age	-0.064 (0.058)	.26	-0.098 (0.077)	.20
	Student Status	-3.72 (1.39)	.007	1.06 (1.83)	.56
	Functioning	-0.17 (0.065)	.007	-0.21 (0.087)	.013
	Hopelessness	0.40 (0.13)	.002	-0.16 (0.17)	.35
	Precontemplation	-1.06 (1.40)	.45	0.13 (1.82)	.94
	Contemplation	1.63 (1.64)	.32	0.91 (2.14)	.67
	Action	-0.82 (1.19)	.49	1.45 (1.54)	.35
	Maintenance	0.56 (0.69)	.42	-1.06 (0.90)	.24
	DAS	-0.13 (0.084)	.12	0.087 (0.11)	.43
	DAS: Achievement	0.22 (0.12)	.060	-0.12 (0.15)	.47
	DAS: Interpersonal	0.13 (0.13)	.31	0.013 (0.17)	.94
Growth Rate	Therapist Level of Training	-0.005 (0.035)	.88	-0.097 (0.038)	.011
	Medication Status	-2.19 (0.70)	.002	-1.08 (0.68)	.12
	Comorbidity	-0.21 (0.59)	.72	0.29 (0.56)	.60
	Depressive Severity	---	---	-0.10 (0.045)	.021
	Anxiety Severity	-0.021 (0.034)	.53	---	---
	Gender	-0.61 (0.69)	.38	-0.32 (0.65)	.63
	Age	0.051 (0.039)	.19	0.049 (0.041)	.23
	Student Status	2.27 (0.96)	.018	-0.14 (0.91)	.88
	Functioning	-0.024 (0.045)	.59	0.007 (0.043)	.88
	Hopelessness	0.087 (0.087)	.32	0.068 (0.088)	.44

Table 12 (Continued)

Regression	Predictor	BDI Linear Spline Estimate (*SE*)	*p*-value	BAI Linear Spline Estimate (*SE*)	*p*-value
Growth Rate	Precontemplation	0.19 (0.93)	.83	-0.34 (0.90)	.70
	Contemplation	-2.50 (1.09)	.022	-2.77 (1.10)	.012
	Action	1.13 (0.80)	.16	0.46 (0.77)	.54
	Maintenance	0.40 (0.45)	.38	1.10 (0.45)	.015
	DAS	0.034 (0.056)	.55	0.016 (0.057)	.78
	DAS: Achievement	-0.052 (0.078)	.51	0.048 (0.080)	.54
	DAS: Interpersonal	-0.21 (0.086)	.18	-0.14 (0.090)	.13

Note. DAS = Dysfunctional Attitudes.

In sum, a number of predictors of initial status of depressive symptom severity were identified. When all other pretreatment variables were accounted for, medication status, anxiety symptom severity, student status, global functioning, and hopelessness were significantly related to initial depressive symptom severity. Medication status, student status, and a contemplative orientation to change were found to predict rate of change in BDI. The predictors summarized here were included in the following outcome analyses.

Analytic Step 4.1: Aim 2

Predictors of Outcome: Trajectories of Change vs Pretreatment Factors. Trajectories of early change in depressive symptom severity were further investigated to determine the extent to which initial status and rate of growth predicted outcome in terms of responder status and level of depressive symptom severity at termination. Previously identified predictors of response (e.g., comorbidity, therapist level of training, readiness to change, and dysfunctional attitudes) were also included in the growth modeling framework. Specifically, outcome was regressed on these potential predictor variables concurrently with the latent growth factors to determine which factors "mattered most" in terms of predicting heterogeneity in outcome.

Prior to conducting outcome analyses in the context of growth models, rudimentary pre-post group mean analyses were run. Indeed, change in both depressive and anxiety symptom severity was significant from baseline to termination reflective of large effect sizes (see Table 13 and 14). In addition, nearly half the sample was classified as a "Responder" with an additional 28.38% classified as a "Partial Responder" leaving

less than a quarter in the "Non-responder" class (see Table 15). Almost two-thirds of the sample (65.77%) were considered to have responded as per the BDI whereas 60.81% responded as per BAI scores. The average number of sessions across completers and non-completers of treatment was 12.04 (*SD* = 10.84) whereas when only completers of treatment were considered (*n* = 149) the average number of sessions was 15.88 (*SD* = 10.74) as compared to 4.01 (*SD* = 5.38) for non-completers.

Table 13

Change in BDI from Pre- to Post-Treatment

Group	Intake BDI *M* (*SD*)	Termination BDI *M* (*SD*)	*t*-value	*df*	*p*-value	Effect Sizes
Total Sample	16.95 (8.99)	8.60 (8.07)	14.08	181	<.0001	0.98
No Diagnosis	13.48 (9.29)	5.25 (5.28)	3.56	15	.0028	1.09
Single Diagnosis	14.02 (8.27)	7.61 (6.75)	7.60	60	<.0001	0.85
Comorbid Diagnoses	19.41 (8.68)	9.69 (8.93)	11.36	104	<.0001	1.10

Note. BDI = Beck Depression Inventory.

Table 14

Change in BAI from Pre- to Post-Treatment

Group	Intake BAI *M* (*SD*)	Termination BAI *M* (*SD*)	*t*-value	*df*	*p*-value	Effect Sizes
Total Sample	17.78 (11.07)	9.53 (8.87)	11.51	182	<.0001	0.82
No Diagnosis	13.86 (10.43)	7.06 (9.04)	2.30	16	.035	0.70
Single Diagnosis	14.44 (8.99)	8.51 (8.68)	6.50	60	<.0001	0.67
Comorbid Diagnoses	20.54 (11.63)	10.54 (8.90)	9.51	104	<.0001	0.96

Note. BAI = Beck Anxiety Inventory.

Table 15

Responders, Partial Responders, Non-responders

Group	Responders on BDI *n* (%)	Responders on BAI *n* (%)	Responder Overall *n* (%)	Partial Responders *n* (%)	Non-Responders *n* (%)
Total Sample	146 (65.77)	135 (60.81)	109 (49.10)	63 (28.38)	50 (22.52)
No Diagnosis	14 (66.67)	15 (71.43)	12 (57.14)	5 (23.81)	4 (19.05)
Single Disorder	52 (66.67)	48 (61.54)	37 (47.44)	26 (33.33)	15 (19.23)
Comorbid Disorders	80 (65.04)	72 (58.54)	60 (48.78)	32 (26.02)	31 (25.20)

Note. BDI = Beck Depression Inventory. BAI = Beck Anxiety Inventory

Three outcome variables were then regressed on the intercept and slope of the best-fitting baseline growth model to identify the extent to which heterogeneity in the latent growth parameters predicted distal outcomes: (1) overall responder status (Non-responder, Partial Responder, versus Responder); (2) Non-responder versus Responder on the BDI; and, (3) BDI total score at termination. Given the aforementioned variability in length of treatment, number of sessions was controlled for in all subsequent outcome analyses. These analyses would thus answer the question, "Does initial status of symptom severity or rate of change across the first five treatment sessions predict outcome?" In addition, the inclusion of previously identified predictors of response regressed on the same distal outcomes would answer the questions, "Do pretreatment variables predict outcome over and above latent growth factors? Which factors matter most?"

The first BDI outcome model regressed anxiety symptom severity, student status, hopelessness, and global functioning on the intercept and student status and contemplation scores on the slope, as per the results of the multivariate predictor analyses. The model also controlled for number of sessions in addition to the three

covariates. In order to examine the incremental utility of trajectories of early change and pretreatment variables, all factors were included simultaneously as predictors of the overall trichotomous responder variable. Specifically, responder status was regressed on the intercept and slope in addition to pretreatment variables identified in the literature as predictors of response (i.e., age, gender, student status, global functioning, hopelessness, precontemplation, contemplation, action, and maintenance stages of change, dysfunctional attitudes, achievement-oriented DAS, interpersonally-oriented DAS, anxiety symptom severity, comorbidity, medication status, and therapist level of training) in the growth modeling framework.

This model fit the data relatively well, χ^2 (106) = 123.56, p = .12; CFI = 0.983; RMSEA = .027. Slope predicted to responder status such that an accelerated growth rate predicted higher responder status (e.g., Responder) whereas the intercept was not identified as a significant predictor (see Table 16). Not a single pretreatment variable predicted response over and above latent growth factors.

The second BDI outcome model was identical to the one examined above except that the dichotomous responder variable as per change in BDI scores at termination served as the outcome measure. In addition to regressing the responder status on the intercept and slope, the same pretreatment variables listed above were tested in the growth modeling framework.

The model still adequately fit the data well (χ^2(106) = 120.02, p = .17, CFI = .987, RMSEA = .024). Slope predicted to responder status such that an accelerated growth rate predicted higher responder status (e.g., Responder) whereas the intercept was not

Table 16

Predictors of Distal Outcomes

Model	Outcome Variable	Intercept Coeff (SE)	*p*-value	Slope Coeff (SE)	*p*-value	Pre	*p*-value	Anxiety Severity	*p*-value	R^2
BDI	Responder Status	-0.008 (0.015)	.616	-0.254 (0.111)	.022	---	---	---	---	43.7
	BDI Responder Status	-0.008 (0.009)	.364	-0.165 (0.067)	.014	0.166 (0.083)	.046	---	---	52.3
	BDI Total Score	0.800 (0.105)	<0.001	0.485 (0.110)	<0.001	---	---	-0.142 (0.049)	.004	56.5

Note. Coeff = Coefficient. SE = Standard Error. BDI = Beck Depression Inventory. Pre = Precontemplation SOCS subscale scores.

identified as a significant predictor. The precontemplation stage of change subscale was the only pretreatment variable to predict response when the latent growth factors were included in the model: higher pretreatment precontemplation scores predicted higher responder status (e.g., Responder).

The third and final BDI outcome model included the BDI total scores at termination as the outcome measure. In addition to regressing the termination scores on the intercept and slope, the same pretreatment variables listed above were tested in the growth modeling framework with the exception of student status.

This model fit the data well ($\chi^2(106) = 130.68$, $p = .052$, CFI = .978, RMSEA = .032). Both slope and intercept predicted to responder status such that an accelerated growth rate and greater intake depressive symptom severity predicted lower BDI scores at termination. Greater intake anxiety symptom severity predicted to lower BDI scores at termination. No other pretreatment variables predicted response when the growth factors were simultaneously considered.

In sum, the slope (i.e., rate of early change) of the linear spline growth curve modeling the BDI scores at intake and over the first five treatment sessions predicted outcome on all three outcome measures, whereas the intercept only predicted total BDI scores at termination. These latent growth factors demonstrated predictive validity even when controlling for therapist level of training, comorbidity, and medication status as well as number of sessions and a number of other pretreatment variables.

Precontemplation subscale scores and anxiety symptom severity were the only other pretreatment variables to predict response when included in the growth curve

modeling framework; neither of the identified pretreatment factors consistently predicted response across *all* the outcome measures. These results suggest that rate of early change in depressive symptom severity predicts outcome over and above pretreatment factors.

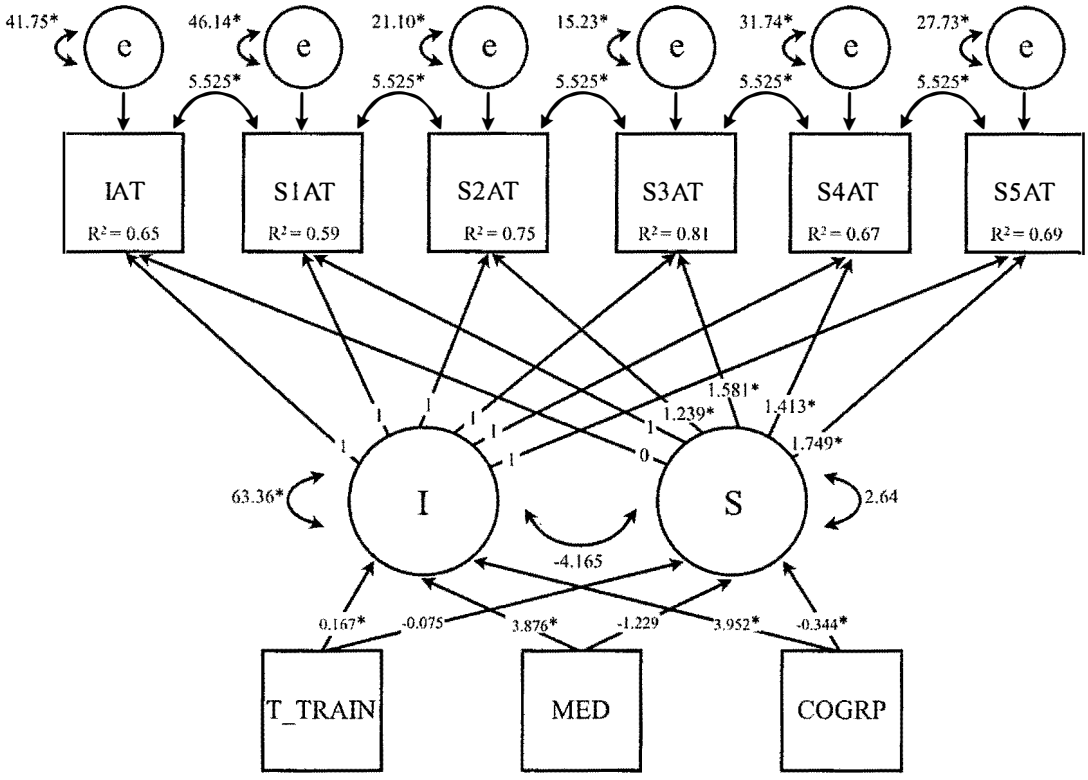
Analytic Step 2.2: Aim 3

Aims 3 and 4 essentially repeated the analytic steps followed above. Aim 3 was to examine trajectories of anxiety symptomatology during the first five treatment sessions and the extent to which pretreatment factors relate to the anxiety trajectories. Aim 4 was to compare how well anxiety symptom trajectories versus pretreatment variables predict ultimate therapy outcome.

Trajectories of Early Change. Similar to the BDI growth modeling, the strict linear and quadratic models were rejected as they fit the data poorly as evidenced by significant Chi Square values, CFI lower than .95, and RMSEA greater than .06. However, the linear spline model in which the BAI score residual covariances were constrained to be equal was chosen as these additional constraints made the model most parsimonious with fit statistics in the desired range: χ^2 (23) = 27.79, p = .22; CFI = 0.994; RMSEA = .031. Figure 3 provides a graphical representation of the final, best-fit model for BAI scores. The *R*-square statistics depicted in the figure indicate the proportion of variance in the time scores accounted for by the growth factors is relatively high suggesting that the linear spline model did well in reproducing the observed growth curves.

Figure 3

Graphical Representation of Best-Fit BAI Model



Note. e = residual variance. IAT = Intake BAI total score. S1AT = Session 1 Anxiety Total Score and so on. I = Intercept. S = Slope. T_Train = Therapist Level of Training. Med = Medication Status. CoGrp = Comorbidity Grouping. * $p < .05$.

The model suggests that, on average, the anxiety symptom trajectories began at a moderate severity and decreased over the first five treatment sessions, though not at as rapid a rate as the BDI scores declined (see Table 17 for coefficient estimates). Significant variability was observed in initial status, however, BAI growth rate was not found to significantly differ among clients. The non-significant result of the test of zero

covariance between the intercept and slope suggests that the client's initial status of anxiety symptom severity has no influence on the rate or direction of change in anxiety symptom severity over the first five treatment sessions. Therapist level of training (more months of training), concomitant psychotropic use, and comorbidity were positively associated with baseline levels of anxiety symptom severity, when all covariates were entered into the model (Table 18). None of the aforementioned covariates predicted rate of change at conventional levels of significance. However, having a therapist with more months of training marginally predicted an accelerated decrease in slope ($\beta = -0.075, p = .059$). The *R*-square statistic for the latent growth curve parameters suggests that the covariates significantly accounted for variation in the intercept ($R = 0.196, p = .002$) but not the slope ($R = 0.307, p = .367$).

Table 17

Parameter Estimates and Fit Indices for BAI Growth Models with Covariates

	Mean of level	Variance of level	Mean of change	Parameter Variance of change	Covariance: level and change
Model	Coeff. (*SE*)	Coeff. (*SE*)	Coeff. (*SE*)	Coeff. (*SE*)	Coeff. (*SE*)
Linear spline with time score residual covariances fixed to equal	8.012*** (1.885)	63.362*** (11.933)	-1.573 (0.989)	2.642 (3.826)	-4.165 (5.994)

Note. BAI = Beck Anxiety Inventory. Coeff. = Coefficient. *SE* = Standard Error. *df* = degrees of freedom. *** $p < .0001$. ** $p < .001$, * $p < .05$, + $p < .1$.

Table 18

Covariate Regression Coefficients in Best-Fitting BAI Model

Covariate	Latent Factor	Coefficient (SE)	*p*-value
Therapist Level of Training	Intercept	0.019 (0.007)	.012
Medication Status		0.44 (0.15)	.005
Comorbidity Status		0.445 (0.112)	<.0001
Therapist Level of Training	Slope	-0.038 (0.025)	.12
Medication Status		-0.63 (0.47)	.19
Comorbidity Status		-0.18 (0.28)	.53

Note. SE = Standard Error.

Analytic Step 3.2

Pretreatment Predictors of Trajectories of Early Change. In this section predictors explaining heterogeneity in initial status (intercept) and growth rate (slope) are reported (see Table 11). The univariate predictor analyses (including the three covariates: therapist level of training, medication status, comorbid status) identified the following variables as predictors of BAI initial status: therapist level of training, medication status, comorbidity status, depressive symptom severity, and global functioning. Specifically, therapists with more months of training, concomitant psychotropic usage, comorbidity, greater depressive symptom severity, and poorer functioning were predictive of greater anxiety symptom severity at intake.

Three variables were identified as predictors of growth rate: therapist level of training, depressive symptom severity, and a contemplative orientation to change. That is, clients paired with therapists with more months of training demonstrated a significant accelerated decline in anxiety symptoms over the first five treatment sessions. And,

greater initial depressive symptom severity and higher pretreatment contemplation scores predicted a steeper decline in anxiety symptomatology.

Next, a multivariate predictor analysis that included all pretreatment factors was run (see Table 12). This model identified the following as significantly related to initial status: therapist level of training, depressive symptom severity, and global functioning. Specifically, greater therapist level of training, greater depressive symptom severity, and lower functioning were associated with greater anxiety symptom severity at intake. In addition, clients matched to therapists with more months of training and those with greater depressive symptom severity, a contemplative orientation to change, and lower maintenance SOC subscale scores predicted an accelerated decline in anxiety symptomatology in the multivariate analyses.

In sum, fewer predictors of initial status of anxiety symptom severity were identified. When all other variables were accounted for, therapist level of training, depressive symptom severity, and global functioning were significantly related to initial anxiety symptom severity. Therapist level of training, depressive symptom severity, a contemplative orientation to change, and lower maintenance subscale scores were found to predict rate of change in BAI. The predictors summarized here were included in the following outcome analyses.

Analytic Step 4.2: Aim 4

Predictors of Outcome: Trajectories of Change vs Pretreatment Factors. Trajectories of early change in severity of anxiety symptoms were further investigated to determine the extent to which initial status and growth rate predicted outcome in terms of

the three measures reviewed above. Previously identified predictors of response (e.g., comorbidity, therapist level of training, readiness to change) were also included in the growth modeling framework. Outcome was regressed on these potential predictor variables concurrently with the latent growth factors to determine which factors "mattered most" in terms of predicting heterogeneity in outcome.

The first BAI outcome model regressed global functioning and depressive symptom severity on the intercept and depressive symptom severity as well as the contemplation and maintenance stages of change subscale scores on the slope, as per the results of the multivariate predictor analyses. The model controlled for number of sessions in addition to the three covariates. In order to examine the incremental utility of trajectories of early change and pretreatment variables all factors were simultaneously included as predictors of the overall trichotomous responder variable. Specifically, responder status was regressed on the intercept and slope in addition to pretreatment variables identified in the literature as predictors of response (i.e., age, gender, student status, global functioning, hopelessness, precontemplation and contemplation stages of change, achievement-oriented DAS, interpersonally-oriented DAS, comorbidity, medication status, and therapist level of training) in the growth modeling framework.

This model fit the data well, χ^2 (96) = 105.13, p = .25; CFI = 0.989; RMSEA = .021. Slope and initial status predicted to responder status such that an accelerated growth rate and lower intake anxiety symptom severity predicted higher responder status (e.g., Responder). See Table 19 for results of the regression analyses. Hopelessness was the

only pretreatment variable to predict outcome. Specifically, higher baseline hopelessness predicted a lower responder status (e.g., non-responder).

The second BAI outcome model was identical to the one examined above except that the dichotomous responder variable as per the change in BAI scores at termination served as the outcome measure. In addition to regressing the responder status on the intercept and slope, the following pretreatment factors were tested in the growth modeling framework: age, student status, hopelessness, global functioning, achievement-oriented DAS, interpersonally-oriented DAS, depressive symptom severity, medication status, and therapist level of training.

Table 19

Predictors of Distal Outcomes

Model	Outcome Variable	Intercept Coeff (SE)	*p*-value	Slope Coeff (SE)	*p*-value	BHS	*p*-value	R^2
BAI	Responder Status	-0.020 (0.010)	.044	-0.199 (0.074)	.007	-0.031 (0.012)	.012	39.4%
	BAI Responder Status	0.001 (0.006)	.86	-0.202 (0.083)	.015	---	---	54.0%
	BAI Total Score	0.750 (0.076)	<.0001	1.673 (1.064)	.116	---	---	55.9%

Note. Coeff = Coefficient. SE = Standard Error. BAI = Beck Anxiety Inventory. BHS = Beck Hopelessness Scale.

This model fit the data well, χ^2 (86) = 101.52, p = .12; CFI = 0.982; RMSEA = .029. The slope predicted to responder status such that an accelerated growth rate predicted higher responder status (e.g., Responder) whereas the intercept was not identified as a significant predictor. Not a single pretreatment variable predicted response over and above latent growth factors.

The third and final BAI outcome model included the BAI total scores at termination as the outcome measure. In addition to regressing the termination scores on the intercept and slope, the following pretreatment variables were tested in the growth modeling framework: age, gender, student status, hopelessness, global functioning, DAS, achievement-oriented DAS, interpersonally-oriented DAS, comorbidity, medication status, and therapist level of training.

This model fit the data well, χ^2 (109) = 120.65, p = .21; CFI = 0.988; RMSEA = .022. The slope did not predict to responder status whereas greater intake anxiety symptom severity predicted higher BAI scores at termination. Not a single pretreatment variable predicted response over and above latent growth factors.

In sum, the intercept *and* slope of the best-fit linear spline growth curve modeling the BAI scores at intake and over the first five treatment sessions predicted outcome on two measures under consideration. These latent growth factors demonstrated predictive validity even when controlling for therapist level of training, comorbidity, and medication status as well as number of sessions and a number of other pretreatment factors. Hopelessness was the only pretreatment variable to predict outcome when the growth factors were simultaneously considered. These results suggest both initial status and rate of early change in anxiety symptom severity predict outcome more so than other pretreatment variables.

Analytic Step 5: Aim 5

Relationship between Depressive and Anxiety Symptoms. Two hypotheses regarding the best characterization of the relationship between depression and anxiety

symptom severity were considered: (1) a distal pathway through latent growth factors (i.e., intercept and slope); (2) a proximal pathway through observed session-by-session BDI and BAI scores. Three models were run to test these hypotheses.

Preliminary analyses indicated a strong relationship between depressive and anxiety symptom severity over the first five treatment sessions. The correlation matrix in Table 8 revealed moderate to high, significant bivariate correlations between BDI and BAI scores across all time points.

Test of Hypothesis One: Parallel Models. Because the parallel growth models were designed to test the hypothesis that BDI and BAI scores were related through their distal latent growth factors, the intercept and slope were entered into a parallel modeling structure. Specifically, the intercepts were specified to covary as were the slopes, and the BDI slope was regressed on the BAI intercept and vice versa. These additional parameters, if significant, would have suggested parallel movement in BDI and BAI time scores through distal latent growth factors was indeed characteristic of the sample.

Even the best-fit model in which the slope for the anxiety growth curve was fixed to zero did not demonstrate adequate fit as the Chi-square test was quite significant, $\chi(76)^2 = 114.67, p = .0028$. This parallel growth model analysis rejected the hypothesis that early change in symptom severity was best characterized by relationships through distal, latent factors (i.e., intercept and slope).

Test of Hypothesis Two: Time-varying Covariates. The second set of tests examined the hypothesis that a more contemporaneous association characterized the relationship between BDI and BAI scores. In order to test this relationship, two additional

models were run. First, the best-fit linear spline depression model was subjected to a test in which the anxiety scores were included as time-varying covariates (TVC). Second, the best-fit linear spline anxiety model was subjected to a test in which the depression scores were included as TVC.

The model in which anxiety symptom scores were introduced as TVC fit less than adequately as per the Chi-square test of significance ($\chi(57)^2 = 84.52$, $p = .01$), however, adequate fit was demonstrated by both the CFI (.974) and the RMSEA (.047). This model suggested that BAI scores at session 1 predicted BDI scores at session 1, and BAI scores at session 2 predicted BDI scores at session 2 and so on. Said differently, the hypothesis that the relationship between depressive and anxiety symptoms is quite proximal was not rejected, however, because the Chi-square index was not significant one final test was run.

Finally, the reverse model whereby depression symptom scores were introduced as the TVC to the overall anxiety linear spline model was run. Though this model still did not fit the data adequately as per the Chi-square test of significance ($\chi(58)^2 = 79.14$, $p = .034$, adequate fit was demonstrated by both the CFI (.978) and the RMSEA (.041). This fit was a significant improvement from the previously reported model as per the Chi-square difference test (nested $\chi^2 = 5.38$, $df = 1$, $p < .05$). This model, in comparison with the previous model, suggests the relationship between depressive and anxiety symptom severity scores is best characterized by depression symptom severity scores directly significantly predicting anxiety symptom severity scores, session-by-session, over time.

CHAPTER IV

DISCUSSION

This study set out to examine trajectories of depressive symptomatology during the first five treatment sessions and the extent to which pretreatment factors relate to the depressive trajectories. A second aim was to compare how well depressive symptom trajectories versus pretreatment variables predict ultimate therapy outcome. Aims 3 and 4 were identical to aims 1 and 2 with the exception that anxiety symptoms were the focus of investigation. Finally, this study was designed, as a fifth aim, to test two hypotheses regarding the relationship between depressive and anxiety symptoms and their change over the course of CBT.

The discussion of the findings begins with those related to trajectories of change, followed by those related to how well pretreatment variables predicted these trajectories of change. Next, the results of the tests of hypotheses related to the relationship between depressive and anxiety symptoms will be discussed. Following this section, the results related to the comparative predictive utility of both rates of early change and pretreatment variables is discussed. Finally, the limitations, methodological and clinical implications, and future directions for this line of research are identified and described.

Trajectories of Early Change

Latent Growth Curve Modeling (LGCM) was employed to characterize the trajectories of early change in depressive and anxiety symptom severity from intake through the first five treatment sessions of CBT. Results revealed 1) significant variability in both depressive and anxiety symptoms at intake; 2) a heterogeneous and steep rate of change in depressive symptoms accounting for 86% of the total improvement in depressive symptomatology; 3) a homogeneous and flatter rate of change in anxiety symptoms accounting for 70.2% of the total improvement in anxiety symptomatology; and, 4) no relationship between initial severity of depressive or anxiety symptoms and rate of change in these symptoms.

That the LGCM revealed no significant correlation between intercept and slope in either the depressive or anxiety symptom profiles suggests that initial severity has no influence on the trajectory of early symptom change. Few studies have reported on this relationship despite its relevance to understanding the process by which symptoms change over the course of therapy. However, Santor and Segal (2001) similarly reported that pretreatment depressive symptom severity was unrelated to rates of early symptom reduction. They highlight the importance of this lack of relationship because it suggests that rapid early symptom reduction is equally achievable in clients with anywhere from mild to severe pretreatment depressive symptom severity (Santor & Segal, 2001).

This is not to say that the initial symptom severity scores were not meaningfully related to trajectories of change or worthy of further discussion. Rather, the largest drop in client depressive and anxiety symptom severity occurred from intake to session one:

56% and 43% of total symptom reduction, respectively. Even so, post hoc analyses indicated the drop in symptom severity from intake to session one did not predict the subsequent rate of symptom change. It is difficult to determine from the current study's data whether clients may have exaggerated the severity of their symptoms to ensure they would be offered treatment. It seems more likely that these scores accurately reflect clients' experience and that the symptom reduction was in response to the validation and hope for improvement engendered by the intake and feedback process. It should be noted that all clients participated in a feedback session in which the therapist talked about the assessment, discussed treatment options and in some cases provided some initial psychoeducation and socialization into treatment and how it would proceed. It is not surprising that clients would experience relief and hope as a result of these activities. Identification of pretreatment variables that do predict rates of early symptom change will be important.

Pretreatment Predictors of Trajectories of Early Change

Pretreatment variables, in addition to initial symptom severity, were investigated to determine the extent to which they were associated with heterogeneity in initial status and predicted rates of early symptom change. Hopelessness, poorer functioning, lower anxiety symptom severity, community member status, and concomitant psychotropic usage were associated with lower depressive symptom severity at intake whereas only poorer functioning, therapist level of training, and greater depressive symptom severity was associated with greater anxiety symptom severity at intake. With respect to rates of change, student status, a contemplative orientation to change, and concomitant

psychotropic usage predicted an accelerated decline in depressive symptoms. Different pretreatment variables predicted rate of change in anxiety symptoms: therapists with more months of training, a contemplative orientation to change, lower maintenance subscale scores, and greater depressive symptom severity predicted an accelerated decline in anxiety symptoms.

Baseline correlates of pretreatment depressive and anxiety symptom severity have been frequently reported in the literature. Indeed, the significant relations observed between pretreatment variables and initial status in the present study, such as hopelessness and depressive symptom severity, replicate much of what is already known. However, there are some unique features of the current sample that were also significantly related to initial symptom severity worth mentioning. For instance, the sample contained a mix of both students and community members, two subpopulations presenting for therapy. Community member status was associated with greater depressive symptom severity at intake and decelerated rates of early depressive symptom change. Perhaps students with *less* severe depression presented *more* frequently because treatment was free whereas community members were required to pay. And, it may be that student status is serving as a proxy for studiousness that manifested as homework compliance/completion--a key ingredient to achieving successful response to CBT (e.g., Bryant, Simons, & Thase, 1999; McEvoy & Nathan, 2005; Persons, Burns, & Perloff, 1988). However, the explanations behind these significant relations lack supportive data particularly with no comparison points, to our knowledge, available in the literature.

Another unique feature of our sample is that therapy occurred in a training clinic where therapists demonstrated significant variability in months of training (range: 1 to 53 months). This range is perhaps more meaningful than a range of 5-10 years, for example, given the differing degree of exposure to clients and CBT from even 1 to 6 months. Indeed, therapist level of training was associated with initial anxiety symptom severity and rate of early anxiety symptom change. That therapist level of training was correlated with initial status may reflect the nature of case assignment in the clinic. Therapists with fewer months of training were typically assigned less severe clients and vice versa. That therapist level of training significantly predicted an accelerated rate of early anxiety symptom change suggests that client anxiety symptoms were more effectively and efficiently reduced through work with a therapist who had more months of training. Similarly, Driscoll, Cukrowicz, Reitzel, Hernandez, Petty, and Joiner (2003) examined therapist level of training also in a training clinic and found that total number of client contact hours was significantly related to outcome. It seems our results replicate those of a growing body of literature that suggests therapist level of training is significantly related to good outcomes (DeRubeis et al., 2005; Grey, Salkovskis, Quigley, Clark, & Ehlers, 2008; Lutz, Leon, Martinovich, Lyons, & Stiles, 2007; Stein & Lambert, 1995; Weertman & Arntz, 2007), but extend the otherwise limited research by identifying its relation to rapid response.

There is also a dearth of research examining the role of readiness to change on treatment outcome, much less on its relation to early symptom change. Recently, however, readiness to change has been identified as a predictor and mediator of treatment

for adolescent depression. Specifically, Lewis, Simons, Silva, et al (2009) found that higher scores on the action subscale were predictive of successful response to CBT, medication, and their combination *and* that increases in action scores were facilitated in CBT conditions which mediated outcome. In the present study, higher contemplation subscale scores predicted an accelerated rate of change in both depressive and anxiety symptomatology. This finding is in accord with Lichtenberg and Hummel's (2000) work with a similarly depressed and anxious adult sample, the difference being that they found higher pretreatment contemplation subscale scores predicted ultimate outcome, rather than rate of early change. Until more studies examine the predictive utility of the SOCS, it is difficult to say if the identification of contemplation scores, as opposed to action scores, as a predictor reflects developmental differences in the way readiness to change influences treatment response. Even so, what is consistent about these findings across developmental stages is that if clients are at least thinking about changing their symptom-related behaviors they are more likely to achieve successful outcomes.

A more widely studied phenomenon is that of medication's effect on rate of depressive symptom improvement. Indeed, one of the desirable features of antidepressants is that they are fast acting, typically more so than psychotherapies (e.g., Hollon, Jarrett, Nierenberg, Thase, Trivedi, & Rush, 2005; Keller et al., 2000). The data presented here are no exception--concomitant psychotropic usage was significantly associated with an accelerated rate of change. Interestingly, although initial symptom severity and rate of change significantly differed according to medication status, in terms of depressive symptom severity, no termination differences were observed. These

findings support the growing literature that medication bolsters the effect of CBT for depression, or vice versa, but that combination treatment does not offer the same benefits in terms of anxiety (Otto, Smits, & Reese, 2005). There is some literature that suggests combining CBT with medication in the treatment for anxiety disorders reduces the overall clinical effectiveness (e.g., Barlow et al., 2000). That anti-anxiety medications, as their name would suggest, minimize the client's experience of physiological symptoms during an exposure is problematic from a CBT perspective. In support of this notion, despite the significant improvement in depressive symptoms in our sample accelerated by medication status, clients concurrently on medication demonstrated significantly higher rates of anxiety symptoms at termination.

One final predictor worth discussing is that of initial depressive symptom severity. Specifically, greater depressive symptom severity at intake predicted an accelerated rate of early anxiety symptom change. Though the direction of this relationship may seem counter to one's expectations, these findings potentially fit with a new line of comorbidity research. Through an innovative experimental design, Craske and colleagues found that individuals with comorbid disorders treated specifically and solely for panic responded significantly better than individuals with comorbidities treated for panic *and* their most severe comorbid disorder. Their interpretation, simply put, is that less is more. That is, it seems focusing on a range of client symptoms was less effective than delivering treatment focused on a single symptom set (Craske, Farchione, Allen, Barrios, Stoyanova, & Rose, 2007; Tsao, Mystkowski, Zucker, & Craske, 2005).

Although there was no experimental manipulation in the present study or adherence data to confirm the following, the findings reported here were obtained in a therapeutic setting whereby CBT was typically delivered consistent with Craske's "less is more" approach. As such, greater initial depressive symptom severity would have suggested that the therapist target depressive symptoms. If the above-summarized research holds, it follows that change in depressive symptom severity would then predict change in anxiety symptom severity. A direct test of this explanation would require simultaneous modeling of depressive and anxiety symptoms. Surprisingly few studies have examined the patterns of change in depressive and anxiety symptoms simultaneously. Persons, Roberts, and Zalecki (2003) used mixed modeling to examine the extent to which depressive symptoms predicted anxiety symptoms and vice versa in a highly comorbid sample. As expected, they observed a strong relationship between symptoms though they reported no differential effect indicating one more strongly predicted the other. It is difficult to say whether Persons and colleagues targeted interventions on a single symptom set or if both depression and anxiety were treated simultaneously.

Relationship between Depressive and Anxiety Symptoms

In an effort to replicate and extend the work of Persons and colleagues (2003), we tested two hypotheses regarding the relationship between depressive and anxiety symptoms: (1) depressive and anxiety symptoms are more distally related through latent constructs; (2) depressive and anxiety symptoms are more proximally related as evidenced by significant time-varying covariate relationships between symptom scores

within each session. The first hypothesis was rejected: the parallel growth modeling did not fit the data well, nor did the expected regression equations and covariance relationships reach significance. However, the second hypothesis, which included symptom scores as time-varying covariates fit the data well. Different from Persons and colleagues, our method of analysis allowed us to conclude, based on comparisons of model fitness, that the relationship between depressive and anxiety symptoms was best characterized as contemporaneous in nature such that depressive symptoms most strongly predicted anxiety symptoms. These results support our interpretation presented above that targeting depressive symptoms leads to change in anxiety symptoms. Nevertheless, given the lack of data regarding specific interventions delivered and/or mechanisms of change, these results are speculative and descriptive at best.

Predictors of Outcome: Trajectories of Change vs Pretreatment Factors

Thus far this discussion has focused on rates of early symptom change and their predictors. However, these findings matter not if there is no relation to outcome. Indeed, the overarching goal of this dissertation was to determine which "mattered most" in predicting treatment outcome: trajectories of early change or pretreatment factors. The current study is the first to simultaneously compare these two approaches to the prediction of acute treatment outcome. Results suggest that the rate of change in depressive symptoms over the first five treatment sessions significantly and consistently predicted outcome over and above the majority of pretreatment variables, except for precontemplation stages of change scores, and initial severity of depression and anxiety symptoms. Similarly, rate of change in anxiety symptoms significantly predicted outcome

on two of the three measures over and above the majority of pretreatment variables, except for hopelessness and initial symptom severity of anxiety.

With respect to depressive symptomatology, the rate of early change in BDI scores was the only predictor that emerged across all three measures of outcome explaining an average of 51% of the variability in response at termination (see Table 16 for R^2 estimates). The consistency with which rates of early depressive symptom change predicted outcome suggests it is indeed a robust predictor as it emerged even when controlling for number of sessions and 16 other pretreatment factors previously identified as predictors of outcome. Two other studies have examined the incremental utility of rates of early symptom change with respect to symptom return at follow-up (Gilboa-Schechtman & Shahar, 2006; Santor & Segal, 2001). Similar to the results reported here, they found that rate of early symptom change (by the 4^{th} or 10^{th} week of treatment) predicted outcome at 3-, 12-, and 18-months since termination over and above pretreatment depressive symptom severity, symptom reduction in other phases of treatment, and remoralizer status. Our study adds to this developing literature specifically that early rate of depressive symptom change can predict to acute outcomes, not simply to follow-up, and that its predictive utility prevails over numerous previously identified pretreatment predictors of outcome.

With respect to anxiety symptomatology, the rate of early change in BAI scores was the only predictor that emerged across two of the three measures of outcome explaining an average of 50% of the variability in response at termination (see Table 19 for R^2 estimates). Although this finding did not reach conventional levels of statistical

significance ($p = .11$), the rate of early change in BAI scores predicted BAI scores at termination (the third outcome measure) in the expected direction. So despite the significantly slower rate of change in anxiety symptoms and its demonstrated homogeneity in the current sample, its predictive utility mirrors that of rate of early change in depressive symptoms. These results, however, are quite preliminary and warrant replication.

Perhaps surprisingly, only four pretreatment variables (i.e., precontemplation subscale scores, hopelessness, depressive and anxiety symptom severity) predicted treatment outcome when rates of early change were simultaneously considered. Specifically, higher precontemplation subscale scores predicted responder status as per change in BDI scores at termination. There is no literature to support the direction of this finding as high pretreatment precontemplation scores are typically associated with treatment dropout. However, it may be that the therapists in our clinic "flagged" these clients with high scores on the precontemplation subscale at intake. There is a strong emphasis in the training clinic to use all assessment materials to inform subsequent treatment decisions. If indeed therapists were keenly aware of this pretreatment prognostic indicator and tailored treatment accordingly by supplementing CBT with Motivational Interviewing, it may have been that these clients who were initially "at risk" for dropping out received a supplemental intervention that improved their rate of response. The only way to test this interpretation with the dataset would be to examine whether precontemplation scores were reduced and contemplation or action subscale

scores were increased over the course of therapy. This meditational analysis is indeed an important future direction for this kind of work.

Higher pretreatment hopelessness scores predicted poorer response on the trichotomous outcome variable: Non-responder, Partial Responder, or Responder. Although it might seem strange that hopelessness emerged as a predictor of response with respect to anxiety symptom modeling, and not the depressive symptom modeling, this result suggests that in individuals with comorbid disorders (as in our sample) both rate of early anxiety symptom change *and* pretreatment hopelessness significantly predicted outcome. That is, hopelessness did not predict outcome with regard to depressive symptom modeling perhaps because it was redundant with depressive symptom severity whereas hopelessness uniquely accounted for variance when anxiety symptom severity was the focus.

Initial depressive and anxiety symptom severity were the only other pretreatment predictors of response that emerged when rates of early symptom change were simultaneously considered. In a review, Hamilton and Dobson (2002) suggest results regarding the predictive utility of initial symptom severity in CBT are equivocal. Even within one study we are unable to report, consistently, whether initial symptom severity "matters" in terms of outcome. One thing is consistent about these predictors—they only emerged when total symptom scores (i.e., BDI or BAI) were the outcome variable under consideration and not when responder status based on clinically significant change indices was the focus. This distinction seems important because it suggests that while

significant differences in total symptom score were observed, initial status had no bearing on clinically significant symptom change over treatment.

Predictors of Outcome: Post Hoc Comparisons

This study was successful in comparing the incremental utility of trajectories of early change and pretreatment variables to the prediction of outcome by simultaneously regressing outcomes on all factors of interest. The typical approach to the identification of outcome predictors includes *either* consideration of rates of early response *or* pretreatment variables. It was thus decided *a posteriori* to explore the extent to which separate examination of these factors would have yielded different results. Indeed, post hoc analyses revealed different predictors of outcome when examining trajectories of change and pretreatment variables separately for both depressive and anxiety symptomatology.

With respect to depressive symptomatology, when the intercept and slope regression equations were dropped from the outcome analyses, hopelessness, global functioning, precontemplation, contemplation, and maintenance subscale scores predicted response. Conversely, when outcome was regressed only on the intercept and slope of the depressive symptom growth model both latent growth factors significantly predicted outcome across all measures.

With respect to anxiety symptomatology, when the intercept and slope regression equations were dropped from the outcome analyses, hopelessness, student status, precontemplation, contemplation, and maintenance stages of change scores predicted response. Conversely, when outcome was regressed only on intercept and slope of the

anxiety symptom growth model both latent growth factors significantly predicted outcome across all measures.

Methodological Implications

This is the first study, to our knowledge, to simultaneously compare trajectories of early symptom change and pretreatment variables to determine which matters most in the prediction of outcome. These results together with Gilboa-Schechtman and Shahar's (2006) and Santor and Segal's (2001) work highlight the importance of simultaneously comparing rates of early symptom change and pretreatment variables. There is now evidence that rates of early symptom change predict acute outcome in addition to outcomes at 3-, 12- and 18-month follow-up over and above pretreatment variables.

The addition of the post hoc analyses presented here are particularly illuminative. Both the intercept and slope of the depressive and anxiety growth models predicted outcome when examined separately. And, numerous pretreatment variables predicted outcome when examined separately. Therefore, the significance of the predictors was contingent solely upon the approach to analyses reaffirming the importance of simultaneously testing the predictive or incremental utility of rates of early symptom change and pretreatment variables. Researchers are thus encouraged to simultaneously test both sets of factors when investigating predictors of outcome in order to best utilize all available information. Investigating pretreatment factors separate from growth rates, or vice versa, may be misleading.

An important piece of our recommendation hinges on one's ability to collect session-by-session data. Investigators of the current study had the fortunate opportunity

to work closely with a computer science technician to merge paper outcome measures with a confidential paperless system. This system facilitated our ability to collect a very rich dataset for the purposes of conducting research. In addition, this system was designed to be user-friendly to meet the needs of both clients and therapists. This system was a success in terms of both research accessibility and clinical utility and is highly recommended.

Clinical Implications

Predictors have long been the focus of treatment outcome research in an effort to identify prognostic indicators to aid in therapist treatment planning. However, few predictors of CBT have been consistently identified (Hamilton & Dobson, 2002) making it difficult for the therapist to make sense of the large body of literature that exists. Conversely, the predictive utility of rapid early response to treatment appears to be a robust finding in the literature. The present study provides additional information suggesting that rates of early symptom change may be our best indicator of successful outcome. Therefore, in terms of clinical implications, it seems important that therapists systematically monitor symptom severity particularly over the first five to ten treatment sessions.

This is not to say that clients who are not demonstrating rapid response should then be terminated. Rather, predictors of rapid rates of response might serve as a guide for treatment planning. For instance, higher scores on the contemplation SOC subscale predicted an accelerated decline in both depressive and anxiety symptom severity. Logic follows that in order to facilitate rapid early symptom change, interventions such as MI

might be important for the therapist to employ early in treatment. As this line of research grows, identification of additional predictors of early symptom change will be important. In sum, this research may help therapists identify individuals who are at risk of not responding to treatment earlier on so as to tailor treatment accordingly.

The majority of the discussion, thus far, has been devoted to highlighting, and making sense of, *significant* predictors of rates of early symptom change and outcome. Switching gears to focus on a non-significant predictor--comorbidity--seems equally important given the role it has already played in this discussion. That comorbidity did *not* predict trajectories of early symptom change *or* outcome is indeed quite important when thinking about the clinical implications of this work. Community therapists all too readily disregard ESTs because the efficacy tradition ruled out clients with comorbidities (Addis, Wade, & Hatgis, 1999; Mahrer, 2005). However, the results from the present study replicate a burgeoning literature that suggests comorbidity does *not* predict outcome (Joormann, Kosfelder, & Schulte, 2005; McLean, Woody, Taylor, & Koch, 1998; Persons, Roberts, & Zalecki, 2003). In the current study, comorbidity did *not* predict rate of early symptom change *or* outcome *or* number of sessions required to reach natural termination. Rather, therapist level of training was identified as a predictor of rapid early symptom change, at least with regard to anxiety symptoms, demonstrating predictive utility over and above that of comorbidity. Indeed, research suggests that the effect of comorbidity may be minimal whereas the competence of the clinician may instead predict outcome. Specifically, although Kuyken and Tsivrikos (2009) replicated the finding that

CBT for depression outcomes were compromised for clients with comorbidity, they found that therapist competence predicted improved outcomes regardless of comorbidity.

Limitations

There are several noteworthy limitations to the present study. First, although therapist level of training was identified as a predictor of initial status and rate of change in anxiety symptomatology, it is unclear what therapist level of training is actually measuring other than simply months since entering practicum. A potentially more informative construct to examine would be actual therapist competence—a likely candidate for explaining the relation between therapist level of training and rapid recovery observed in this study. However, months or years of training may have little to do with the competence of the therapist (Waltz, Addis, Koerner, & Jacobson, 1993). Relatedly, our study lacks the data to elaborate on treatment fidelity. Although it was described above that therapists delivered CBT and its third wave variants through the lens of a cognitive case conceptualization, we do not have data to support this claim. Fidelity measurement and competency ratings are thus both a limitation and a future direction of ours.

Second, although the present study was derived from a rich dataset with 16 potential pretreatment factors included in the predictor analyses, some important variables were omitted from the study. For instance, Hamilton and Dobson (2002) identified the chronicity of depression as a fairly robust predictor of response that was not included in our dataset. Indeed, we hypothesized that chronicity may be the variable most

strongly related to therapist level of training as it was thought to heavily influence client-therapist assignment; however, we were unable to test this hypothesis.

Third, given the range of anxiety disorders represented in the current sample and the limited focus of the BAI on somatic manifestation of anxiety symptoms, a different or an additional measure of anxiety might have yielded greater variability in rate of symptom change. For instance, the Symptom Checklist has been employed by others studying rate of change (e.g., Kopta, Howard, Lowry, & Beutler, 1994). This measure appears to tap into a broader range of anxiety symptoms including, for example, general anxiety, phobias, and obsessive-compulsive symptoms.

Fourth, for a number of reasons, the generalizability of this study might be somewhat limited. Specifically, clients with acute suicidality, psychosis, and concurrent substance dependence/abuse were referred out for more appropriate services. Similarly, the average symptom severity of the sample was mild-moderate suggesting few cases of severe nonpsychotic depression were represented in this sample. Further, the homogenous nature of the clients in terms of ethnicity and culture limits the generalizability. Therefore, the findings presented here cannot be assumed to reflect trajectories of change and predictors of outcome in clients with more severe and persistent mental illness or clients of more diverse cultures. Rather, it might be appropriate to conclude that the clients in the current study represented a subset of those seeking outpatient psychotherapeutic services.

Future Directions

There are numerous potential avenues for future research. First, although the present study collected data on session-by-session rate of change in symptom severity, it would be important to collect additional data on potential mechanisms of change. Because of the robust nature of the predictive utility of the rapid rate of response, researchers have engaged in a debate over whether it is general therapy factors (e.g., working alliance; Ilardi & Craighead, 1999) or therapy specific factors (e.g., change in cognitive distortions; Tang & DeRubeis, 1999), however, the field has yet to resolve what exactly is responsible for this rapid change.

Second, separate subgroup analyses would allow for a more nuanced test of the effect of comorbidity. That is, rather than simply including comorbidity as a pretreatment factor, comorbidity groups could be analyzed separately and compared to determine the extent to which similarities and differences exist. For instance, in the current analysis the clients with a single Axis I diagnosis were all included in the same group. It may be that single anxiety disordered clients responded differently than did single depressive disordered clients. These differences might have implications for treatment planning.

Third, it may be that the average, overall, rate of growth identified in the current sample actually consisted of unobserved subgroups of individuals that could be identified through growth mixture modeling (GMM). GMM relaxes the assumption that individuals in a sample come from the same population. Rather, it assumes that there are subpopulations of individuals that manifest in different patterns of response. GMM then enables one to characterize group membership through the exploration of pretreatment

factors and time-varying covariates. And, within-group differences in terms of rate of change can be investigated in addition to the extent to which group differences predict response. The capabilities of GMM have only recently been applied to treatment outcome research and have yielded very different outcomes than more standard approaches have allowed.

Finally, given the recent push to disseminate CBT and the robust predictive utility of rapid early response it is increasingly important to identify ways in which to maximize the rate of early change. Perhaps Roz Shafran's hypothesis is accurate, that holding sessions twice a week for the first three weeks would achieve this goal. However, an empirical test comparing session dose and early rates of change is in order prior to disseminating this approach to session dose.

Conclusion

In conclusion, the present sought to answer the question "Which clients responded to CBT?" through the merging of two different methodological approaches. Results suggest that the rates of depressive and anxiety symptom change over the first five treatment sessions predicted outcome over and above the majority of pretreatment variables. Pretreatment predictors of rate of early symptom change, such as a contemplative orientation to change and therapist level of training, were identified which may suggest that therapists should target these factors to potentially maximize rapid early symptom change, and in turn outcome.

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