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Patient Interpersonal and Cognitive Changes in Relation to Outcome in Interpersonal  
Psychotherapy for Depression

A Thesis Presented

by

SAMANTHA L. BERNECKER

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## ABSTRACT

### PATIENT INTERPERSONAL AND COGNITIVE CHANGES IN RELATION TO OUTCOME IN INTERPERSONAL PSYCHOTHERAPY FOR DEPRESSION

FEBRUARY 2013

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Despite evidence for the efficacy of interpersonal psychotherapy (IPT) for depression, there remains little understanding of its specific change-promoting ingredients. This study aimed to establish candidate change mechanisms by identifying whether patients' interpersonal (theory-specific) and cognitive (theory-nonspecific) characteristics change in an adaptive direction during IPT, and whether such changes differentially relate to depression reduction and improvement in global functioning. The four interpersonal variables and one cognitive variable measured all changed significantly in an adaptive direction, with medium to large effect sizes. Reduced interpersonal problems were marginally associated with self-reported depression reduction ( $\beta = 2.846, p = .062$ ), and greater satisfaction with social support was marginally associated with depression reduction ( $\beta = -1.423, p = .081$ ). Unexpectedly, reduced romantic relationship adjustment was related to depression reduction ( $\beta = 2.028, p = .008$  for self-rated depression and  $\beta = 1.474, p = .022$  for clinician-rated depression), and increased attachment avoidance was marginally related to better clinician-rated global functioning ( $\beta = 1.501, p = .09$ ). Thus, theory-relevant interpersonal variables emerged as candidate change mechanisms, and the findings are discussed with respect to their research and practice implications.

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# CHAPTER 1

## INTRODUCTION

Depressive disorders are the fourth leading cause of public health burden among all diseases worldwide (World Health Organization, 2001). The lifetime prevalence rate of major depressive disorder in the United States alone is approximately 17% and growing (Kessler & Wang, 2009). Fortunately, several depression treatments have demonstrated substantial efficacy, including interpersonal psychotherapy (IPT; Klerman, Weissman, Rounsaville, & Chevron, 1984). The American Psychological Association (APA) has designated IPT as an empirically supported treatment with “strong research support” (APA, 2008; APA Presidential Task Force on Evidence-Based Practice, 2006; Chambless & Hollon, 1998). Further, along with cognitive-behavioral therapy (CBT), IPT is recommended as a first-line psychosocial treatment for depression by several professional and governmental organizations, including the American Psychiatric Association (2000a), the National Institutes of Health (2011), and the United Kingdom’s National Institute for Health and Clinical Excellence (2009).

Randomized controlled trials have repeatedly substantiated IPT’s efficacy. In a meta-analysis of psychotherapies for depression that included only trials with active psychological comparison treatments (i.e., excluding comparisons to placebo, waitlist, medication, etc.), IPT was the only treatment that demonstrated significantly superior outcomes to comparison conditions (Cuijpers, van Straten, Andersson, & van Oppen, 2008). Two other meta-analyses have aggregated the few direct comparisons of IPT to CBT; one found a nonsignificant difference in posttreatment symptomatology that favored IPT (Cuijpers et al., 2011), while the other found that IPT demonstrated

significantly greater depression reduction than CBT, and also had higher, though statistically nonsignificant, remission and retention rates (de Mello, de Jesus Mari, Bacaltchuk, Verdeli, & Neugebauer, 2005).

However, no treatment for depression, IPT included, is completely effective for all patients. Even in this first-line treatment, remission rates range from 20-70%, leaving a substantial proportion of patients clinically depressed following IPT (Frank et al., 2011; Schramm et al., 2011; Shea et al., 1992). Thus, there remains room for improvement. One strategy for refining IPT is to identify and emphasize its specific mechanisms of therapeutic change, while eliminating or substantially revising ineffective or even harmful treatment elements. To date, no such mechanisms of IPT for depression have been identified empirically, leaving a substantial gap in the literature (Bernecker, 2012; Ravitz, Maunder, & McBride, 2008). Consequently, the field has little idea of how one of the most prominent therapies for one of the most prevalent disorders works.

Therapeutic change mechanisms can be understood at two levels: the actions that take place in session that relate to symptom reduction, and the cascade of changes in the patient that facilitate symptom reduction. IPT posits that while interpersonal difficulties are not always part of depression's etiology, they are inevitably involved in its maintenance, and therefore development of social supports and the resolution of relational conflicts are purported to promote symptom reduction (Stuart, Robertson, & O'Hara, 2006; Weissman, Markowitz, & Klerman, 2000). The central theoretical in-session mechanisms, then, are the problem-solving strategies that therapists use with their patients in order to address one or more of IPT's targeted interpersonal problems areas: complicated bereavement, role disputes, role transitions, and interpersonal deficits. The



theoretical patient-level mechanisms are the development of social supports and improved quality of current relationships (Weissman et al., 2000).

However, IPT includes several components that may act through different pathways. Some techniques that are permitted or encouraged in the service of identifying or solving interpersonal problems may act through mechanisms other than the resolution of those problems. For example, change in cognitions may be facilitated during IPT through the induction of the “sick role,” which, as the manual states, “frees the patient from ruminative self-criticism” (Weisman et al., 2000; p. 45) through reframing self-blame, hopelessness, and feelings of dependency as symptoms of a disease and not reality-based. Further, IPT therapists point out to their patients that certain beliefs are depressive distortions (though these beliefs are not systematically examined and challenged, as would be the case in CBT; Weissman et al., 2000). IPT also encourages affective experiencing, which may promote a sense of mastery of one’s emotions and decrease the need for emotional avoidance through rumination. Finally, the development of strategies for addressing conflicts and expanding social circles might mobilize the patient to pursue actively these solutions and thus serve as a type of behavioral activation.

Although no change mechanisms have been identified for IPT, in that no variables have been found to meet the strict criteria for a mechanism (i.e., statistical mediation, temporal precedence, specificity, and so forth; Kazdin, 2009; Nock, 2007), past IPT research has revealed several variables that are correlated with outcome and are therefore potential mechanisms. Among in-session correlates, some are consistent with IPT’s theory. In the Treatment of Depression Collaborative Research Program (TDCRP; Elkin et al., 1989), the frequency of therapists’ IPT technique use was positively associated

with adaptive outcome (Minonne, 2009). The same finding has been demonstrated in maintenance IPT (Frank, Kupfer, Wagner, McEachran, & Cornes, 1991; Spanier, 1998), even when patients evince biological vulnerability to depression (Spanier, Frank, McEachran, Grochocinski, & Kupfer, 1996). In the training phase of the TDCRP, supervisor ratings of therapists' skillful implementation of IPT were associated with reduction in both patient- and clinician-rated depression (O'Malley et al., 1988). "Deconstructing interpersonal patterns" and altering those patterns emerged as common themes that differentiated successful IPT from unsuccessful IPT in a qualitative analysis (Crowe & Luty, 2005). In a maintenance trial of IPT, patients who did not relapse spent more time discussing their children and practical problems, arguably topics consistent with the targets of IPT, than patients who relapsed; patients who relapsed spent more time discussing their mental symptoms, which is more consistent with a cognitive focus, than patients who did not (Jacobson, Deykin, & Prusoff, 1977). In one study, the relation between interpersonal intervention use and outcome was specific to IPT: a therapist's accurately identifying and addressing the patient's interpersonal problems predicted improvement in clinician-rated depression and social adjustment in IPT, but worsening in these domains in CBT (Crits-Christoph, Connolly Gibbons, Temes, Elkin, & Gallop, 2010).

Other in-session correlates of outcome are reflective of the techniques used in IPT (Weissman et al., 2000), but are not necessarily interpersonally focused. In the TDCRP training phase, therapists' use of exploratory techniques related to patient-rated adaptive change and with attenuation of clinician-rated depression symptoms (Rounsaville et al., 1987). In maintenance IPT, patients who did not relapse, relative to those who did,

engaged in more reflective discussion, characterized by problem-solving while maintaining an awareness of one's own behavior and its impact on others (Jacobson et al., 1977). Finally, some in-session correlates suggest the importance of the therapeutic relationship. In a naturalistic study of IPT, patients who rated working alliance higher were more likely to achieve remission (McBride et al., 2010). Patient-rated alliance also was associated with fewer posttreatment symptoms across all treatment arms of the TDCRP (which included IPT, CBT, imipramine, and medication placebo; Krupnick et al., 1996). In a trial comparing IPT, CBT, and pharmacotherapy, the alliance-outcome relation held in all conditions even after controlling for symptom change prior to alliance measurement (Zuroff, Koestner, Moskowitz, Marshall, & Bagby, 2007). Additionally, greater therapist warmth was associated with more patient-rated adaptive change and with improvement in social functioning in another IPT trial (Rounsaville et al., 1987). Given the variety of outcome correlates, a broad set of in-session mechanisms, both theory-specific and nonspecific, may be at play in IPT for depression; on the other hand, it is entirely possible that few or none of these variables act as true mechanisms.

There is even less knowledge of potential patient-level mechanisms of IPT than there is of potential in-session mechanisms. Only one patient variable that relates to outcome has been identified: in IPT for dysthymia, patients' retrospective ratings of having solved interpersonal problems were associated with clinician-rated reduction in depression symptoms (Markowitz, Bleiberg, Christos, & Levitan, 2006).

Although little is known about how changes in patient characteristics relate to outcome, research has identified that some variables do in fact change during IPT to a greater degree than in other treatments. After eight months of maintenance treatment,

IPT patients showed improved work performance, less interpersonal friction, better communication, and less anxious rumination than patients taking amitriptyline (Weissman et al., 1974). Following acute treatment, IPT patients demonstrated greater improvements in social adjustment and self-esteem than patients in an amitriptyline condition (Prusoff, Weissman, Klerman, & Rounsaville, 1980). IPT also increased social functioning more than treatment-as-usual (TAU) when it was adapted to be culturally relevant to low socioeconomic status women (Grote et al., 2009). Also compared to TAU, IPT led to greater decreases in shame and in posttraumatic stress disorder symptoms in depressed women with sexual abuse histories (Talbot et al., 2011). In response to an anxiety-provoking task, depressed mothers in an adapted version of IPT evidenced less physiological reactivity and smaller increases in depressed mood than those in TAU (Cyranowski, Swartz, Hofkens, & Frank, 2009).

Finally, other characteristics have been shown to improve during IPT, but their specificity to IPT has not been investigated. In the same naturalistic trial of IPT from which this study is derived, interpersonal problems, attachment anxiety, and attachment avoidance were all lower at posttreatment relative to baseline (Ravitz et al., 2008). In IPT for postpartum depression, postpartum adjustment, social adjustment, and dyadic adjustment all increased from baseline (O'Hara, Stuart, Gorman, & Wenzel, 2000). Patients' anxiety symptoms have also been shown to attenuate over the course of IPT, but the same happened to a greater degree in pharmacotherapy (Martin, Rai, Richardson, & Royall, 2001). In all conditions of the TDCRP, patients' marital adjustment increased (Whisman, 2001). Although these patient-level changes remain candidate mechanisms through which IPT may lead to depression reduction, they could also be independent,

secondary outcomes of IPT or consequences of decreased depression. For example, in the TDCRP, the effect of time on marital adjustment became nonsignificant when controlling for depression at pre- and posttreatment, suggesting that the effect of treatment on marital adjustment was mediated by symptom improvement (Whisman, 2001).

Thus, despite the importance of identifying mechanisms of change through which IPT reduces depression, there is a dearth of research in this area, particularly at the patient level. The present exploratory study represents a preliminary first step in the search for patient-level IPT mechanisms. Using an archival data set from a naturalistic trial of 16-week, manualized IPT for depression (McBride et al., 2010), this study first examined whether patient characteristics change significantly during treatment. Patient characteristics included several interpersonally oriented variables (dyadic adjustment, attachment style, interpersonal problems, and perceived social support), as well as one cognitive variable (dysfunctional attitudes). As a second step, the study examined whether change on the various patient characteristics were differentially associated with patients' treatment outcome, which was operationalized in four ways: patient-reported depression level, clinician-rated depression level, global level of functioning, and depression remission status.

This step of discovering possible mechanisms is important prior to investigating fully whether a variable meets all criteria for a change mechanism because of the substantial resources required for such investigation (Kraemer, Wilson, Fairburn, & Agras, 2002). Thus, although the present analyses do not meet criteria for demonstrating mediation, let alone causation, they serve as a fundamental first step to indicate what

variables researchers should invest time and money to study more thoroughly. The implications of a research program on change mechanisms are far-reaching, and such research has been characterized as one of the central goals of clinical science for this century (Hyman, 2000). Most obviously, understanding change mechanisms will enable the enhancement of therapies, making them more effective and/or more efficient in time and cost, by allowing therapists to emphasize causal factors (Kazdin, 2009; Kraemer et al., 2002; Nock, 2007). Such work will also provide a theoretical backdrop to guide the development of new therapies and the integration of current techniques (Kraemer et al., 2002), as well help to categorize by their mechanisms the hundreds of currently available therapies (Kazdin, 2009). Studies of change mechanisms also have the potential to settle the debate about the relative importance of common and specific factors (Nock, 2007) and to confirm or disconfirm the theoretical bases of psychotherapies. Finally, understanding what variables change in therapy to promote symptom reduction will reveal that those variables are involved in the maintenance of particular pathologies (Kraemer et al., 2002). This study will serve as a preliminary, though vital, first step in achieving these goals for one of the most empirically well supported therapies for one of the most prevalent, costly, and debilitating disorders.

## CHAPTER 2

### METHOD

Data derived from a naturalistic database of adults treated with IPT at an outpatient mood disorders clinic of a university-affiliated hospital in Southern Ontario (McBride et al., 2010; Ravitz et al., 2008).

#### 2.1 Participants

Patients were 95 adults meeting *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association, 2000b)* criteria for major depressive disorder. To be eligible for inclusion in this novel secondary analysis, patients were required to (a) have a pretreatment Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1996) score of  $\geq 15$ , ensuring that they were at least moderately depressed, and (b) have remained in therapy through session 15 or 16 (i.e., a completer sample). The sociodemographic and clinical/diagnostic characteristics of the sample appear in Table 1. The internal review board of the university-affiliated hospital approved the main study's protocol and all patients provided written consent before study entry.

The 39 therapists in the study each saw between one and seven patients ( $M = 2.8$ ,  $SD = 1.7$ ). Doctoral level staff clinicians treated 77% of the patients, and psychiatry residents and clinical psychology graduate students treated 23%.

#### 2.2 Treatment

Treatment involved 16 sessions of protocol-driven IPT delivered according to the Weissman et al. (2000) manual. IPT is comprised of three phases: (1) the initial phase, which involves psychoeducation around depression and its interpersonal roots, induction

of the “sick role,” identification of interpersonal problem areas, and setting a treatment contract; (2) addressing the primary interpersonal problem, which may fall into the category of complicated grief, role disputes, role transitions, or interpersonal deficits; and (3) preparation for termination.

## **2.3 Measures**

### **2.3.1 Sociodemographic Characteristics**

Patients’ sociodemographic characteristics were assessed with a patient characteristics form, which included age, sex, marital status, and education.

### **2.3.2 Clinical/Diagnostic Characteristics**

The *Structured Clinical Interview for DSM-IV Axis I Disorders* (SCID-I; First, Spitzer, Gibbon, & Williams, 1995) provided information on patients’ age of onset of their first depressive episode, number of prior depressive episodes, the length of their current depressive episode in months, whether or not they were on antidepressant medications, and Axis I diagnostic comorbidity.

### **2.3.3 Outcome Measures**

*Beck Depression Inventory-Second Edition (BDI-II; Beck et al., 1996)*. The BDI-II (see Appendix A) is a widely used 21-item self-report questionnaire that assesses attitudes and symptoms characteristic of depression during the past two weeks. Items, which are rated on a scale from 0 (absence of symptom) to 3 (most severe experience of symptom), are summed, with higher scores reflecting more depression (total score range = 0-63). The BDI-II has repeatedly demonstrated good internal consistency ( $\alpha = .73$  to  $.92$ ; Beck, Steer, & Garbin, 1988) and high test-retest reliability (Pearson’s  $r = .93$ ; Beck



et al., 1996), as well as convergent validity in its correlation with the Hamilton Rating Scale for Depression (Pearson's  $r = .71$ ; Beck et al., 1996).

***Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960, 1967).*** The HRSD (see Appendix B) is a commonly used 17-item measure of depression completed by a clinician after a thorough diagnostic interview (Bagby, Ryder, Schuller, & Marshall, 2004; Williams, 2001). Each item is rated (on variable 3- or 5-point scales) based on its severity as experienced by the patient over the past week. Items are summed, with higher scores reflecting more depression (total score range = 0-52). Psychometric studies have reported adequate internal consistency ( $\alpha \geq .70$ ), interrater reliability (intraclass  $r \geq .60$ ), and test-retest reliability (Pearson's  $r \geq .70$ ; Bagby et al., 2004). The measure has also largely demonstrated adequate convergent and discriminant validity (Bagby et al., 2004).

***Global Assessment of Functioning (GAF; Endicott, Spitzer, Fleiss, & Cohen, 1976).*** The GAF (see Appendix C) is a single clinician rating on a 1-100 scale that captures a patient's overall social, occupational, and mental functioning. A higher score reflects more adaptive global functioning. In this study, GAF ratings were assigned based on patients' responses to the SCID-I. The interrater reliability of GAF ratings is adequately high among trained researchers (intraclass  $r_s = .81$  to  $.94$ ; Aas, 2010; Hilsenroth et al., 2000). In addition to their relation with symptoms, GAF ratings have been shown to correlate with length of inpatient hospitalizations (Hay, Katsikitis, Begg, Da Costa, & Blumenfeld, 2003) and physical and social functioning (Grootenboer et al., 2011).

***Depression remission.*** Posttreatment depression remission was defined as a posttreatment BDI-II score  $\leq 10$  (Constantino, Adams, Pazzaglia, Bernecker, & McBride,

2012; Dimidjian et al., 2006; Hopko et al., 2011). Remission status reflects a more stringent outcome variable than depression level in that it requires one's posttreatment depression score to be within normal limits of a non-clinical sample (i.e., clinically significant change). A BDI-II score of 10 is not only consistent with previous remission definitions based on this instrument, but it is also below Beck et al.'s (1996) recommended clinical cut-point for being asymptomatic (i.e.,  $\leq 13$ ). Moreover, the current cut-point is below the mean (12.6) of the non-clinical normative sample from the original BDI-II validation study (Beck et al., 1996).

#### **2.3.4 Predictor Measures**

These predictors were selected from among those measures collected in the trial based on their theoretical relevance and relatively low levels of missingness and collinearity.

*Dyadic Adjustment Scale (DyAS; Spanier, 1976)*. The DyAS (see Appendix D) is a 32-item self-report measure of the quality of one's marital (or, if not married, most significant) romantic relationship. The total score, which was used in this study, has a theoretical range of 0 to 151, with higher scores reflecting greater adjustment. The DyAS total score has demonstrated good reliability (95% confidence interval for  $\alpha = .906, .922$ ; Graham, Lui, & Jeziorski, 2006), as well as convergent, concurrent, and predictive validity (Kurdek, 1992).

*Experiences in Close Relationships (ECR; Brennan, Clark, & Shaver, 1998)*. The ECR (see Appendix E) is a 36-item self-report measure of attachment style with two orthogonal subscales of anxiety (neediness and fear of loss) and avoidance (distancing behaviors and avoidance of closeness); both subscales were used in the present study.

Participants rate the extent to which each item is descriptive of their feelings in close relationships on a scale ranging from 1 “not at all” to 7 “very much.” Each scale is comprised of 18 summed items (scale score range = 18 to 126). Higher scores on the subscales reflect greater attachment anxiety and avoidance, respectively. The ECR was developed through factor analysis of 60 preexisting attachment measures; items were selected based on the strength of their relations with the anxiety and avoidance factors that emerged, and in this way the ECR represents something of a consensus among attachment measures. There is strong evidence for its reliability, factor structure, and validity across numerous studies (Ravitz, Maunder, Hunter, Sthankiya, & Lancee, 2010).

***Inventory of Interpersonal Problems-Circumplex Version (IIP-64; Horowitz, Alden, Wiggins, & Pincus, 2000).*** The IIP-64 (see Appendix F), which was derived through factor analysis of the original 127-item version (Horowitz, Rosenberg, Baer, Ureño, & Villaseñor, 1988), is a widely used measure of interpersonal problems. Each of the 64 items is rated on a 0 “not at all” to 4 “very much” scale, with higher total scores reflecting greater overall distress from interpersonal problems (range = 0 to 256). Like the original measure, the IIP-64 possesses good psychometric properties, with Cronbach’s alpha of .96 for the total score and a test-retest reliability coefficient of .78 (Horowitz et al., 2000).

***Social Support Questionnaire-Brief (SSQ-B; Sarason, Sarason, Shearin, & Pierce, 1987).*** The SSQ-B (see Appendix G) is a 6-item self-report of perceived social support. Each item reflects a challenging scenario and is rated twice. The first rating is the respondents’ perception of the number of people (from 1 to 9) on whom they can rely in the particular situation (the mean number across the six scenarios is derived). The

second rating is the participants' perceived satisfaction with social support, with each scenario rated on a 1 "very dissatisfied" to 6 "very satisfied" scale (mean satisfaction rating across the six scenarios is derived). The SSQ-B has demonstrated a high correlation with the psychometrically sound longer version (Sarason, Levine, Basham, & Sarason, 1983).

*Dysfunctional Attitude Scale-Form A (Weissman, 1979; Weissman & Beck, 1978).* The DAS-A (see Appendix H) is a self-report measure of maladaptive attitudes that are thought to confer risk for depression. It consists of 40 items rated on a 1 "totally agree" to 7 "totally disagree" Likert scale, with lower total scores indicating more dysfunctional attitudes (range = 40 to 280). The DAS-A has good internal consistency ( $\alpha = .76$  to  $.91$ ) (de Graaf, Roelofs, & Huibers, 2009; Floyd, Scogin, & Chaplin, 2004) and has repeatedly demonstrated validity in predicting depression (Oliver, Murphy, Ferland, & Ross, 2007).

## **2.4 Procedure**

Prior to treatment, a trained graduate assessor administered the SCID-I to determine diagnostic eligibility for the study. The SCID-I included the assessor's GAF rating. For eligible and consenting patients, the assessor then administered the HRSD. These patients also completed at baseline the demographics form, BDI-II, DyAS, IIP-64, SSQ-B, and DAS-A. The BDI-II was administered after every session and at posttreatment. Also at posttreatment, patients again completed the DyAS, IIP-64, SSQ-B, and DAS-A, and they were again interviewed with the HRSD and an abbreviated version of the SCID-I (including GAF rating).

## 2.5 Data Analysis

Though missingness was generally low, some patients were missing item-level data at either pre- or posttreatment; to avoid data loss for these subjects, five item-level datasets were imputed using SPSS Version 20.0's multiple imputation function. Subsequent analyses, described below, were conducted using the HLM6 program (Raudenbush, Bryk, & Congdon, 2004), which addresses multiply imputed datasets by fitting five separate models and then averaging the estimates.

I used hierarchical linear modeling (HLM) to identify whether patient variables changed during therapy (Collins & Sayer, 2001; Raudenbush & Bryk, 2002), fitting a two-wave model of change to each individual's data to obtain the model-based empirical Bayes estimates of each person's change score (i.e., latent difference score) on the predictors of interest. Because only two time points were available, there were too few degrees of freedom to use the standard HLM procedure for estimating change scores; therefore, the known variance procedure was used, in which the error variance for each measure at each time point was calculated using the formula  $(1 - \text{Cronbach's } \alpha) * \text{variance}$ . Rather than allowing the model to estimate the error variances, I constrained the variances to the calculated values. Negative change scores indicate a decrease in a patient characteristic, while positive scores indicate an increase. Additionally, I calculated Cohen's  $d$  for each pre-post change in order to provide a more readily interpretable index of the magnitude of change.

To determine whether change in each variable predicted outcome after controlling for baseline depression, I fit four separate multilevel models with within-therapist (i.e., patient-level) variability at level 1 (L1) and between-therapist variability at level 2 (L2)

predicting variance in each of the four outcome variables. This use of multilevel models controls for nonindependence of the data among the patients treated by the same therapists. The intraclass correlation coefficient (ICC), which provides a measure of this L2 variability, can be calculated from an unconditional model with no predictors at L1 or L2. The percentage of between-therapist variability across the four models ranged from < 1% to 14.5%. Even when the proportion of therapist variability was very small, I retained the multilevel framework for consistent structure across the models.

For the model predicting patient-rated depression (i.e., posttreatment BDI-II), baseline BDI-II was entered as a covariate and the HLM-derived latent difference score for each patient variable was entered as a predictor. Additionally, because it was thought that gender (dummy coded as female = 0 and male = 1) and antidepressant medication status (dummy coded as 0 = not currently on antidepressants and 1 = currently on antidepressants) might relate to the outcome, I entered them as covariates. I fit a second and third model using the same procedures, except that baseline and posttreatment HRSD and GAF scores were substituted for BDI-II scores, respectively. Finally, I fit a fourth model using logistic regression within HLM, with baseline BDI-II as the baseline severity covariate, to predict the dichotomous remission status (i.e., yes or no posttreatment BDI-II  $\leq$  10). Given that the aim of this preliminary study was to identify candidate mechanisms for further research, and given the relatively small sample size for HLM, I interpreted marginally significant findings as worthy of future study.

## CHAPTER 3

### RESULTS

Patients' BDI-II and HRSD scores decreased over the course of treatment, and GAF increased (see Table 2); additionally, 43 (45.3%) patients remitted at posttreatment. All patient characteristic variables changed significantly in an adaptive direction; estimated change score parameters appear in Table 3. Specifically, dyadic adjustment increased ( $d = 0.99$ ), attachment style became more secure both along the anxiety ( $d = -0.71$ ) and avoidance ( $d = -0.84$ ) dimensions, interpersonal problems decreased ( $d = -1.42$ ), perceived number of ( $d = 0.53$ ) and satisfaction with ( $d = 1.18$ ) social supports increased, and dysfunctional attitudes decreased ( $d = 1.03$ ) on average from pre- to posttreatment.

The results of the four HLM models predicting outcome from these changes appear in Tables 4 through 7. All predictors and covariates were entered as fixed effects, as tests of the variance components did not approach significance in any case, which suggests that the effects did not differ across therapists (as is to be expected given the relatively small amount of between-therapist variability). Unsurprisingly, pretreatment severity variables entered as a covariates were associated with posttreatment severity: pretreatment BDI-II was positively associated with posttreatment BDI-II, pretreatment HRSD was positively associated with posttreatment HRSD, pretreatment GAF was positively associated with posttreatment GAF, and pretreatment BDI-II was positively associated with posttreatment remission status.

Additionally, several change scores were associated with outcome: (1) an increase in dyadic adjustment was significantly associated with higher posttreatment self-reported

(BDI-II) and clinician-rated (HRSD) depression, (2) a decrease in attachment avoidance was marginally associated with worse global functioning (GAF), (3) a decrease in interpersonal problems was marginally associated with lower self-reported depression, and (4) an increase in satisfaction with social support was marginally associated with lower self-reported depression.

Finally, the covariates of gender and medication status were associated with outcome in some models. Male gender was associated with higher posttreatment HRSD at a marginally significant level. Those taking antidepressants were significantly less likely to remit and had marginally significantly higher posttreatment BDI scores.



## CHAPTER 4

### DISCUSSION

The goals of this study were to examine whether patient characteristics changed significantly during IPT for depression, and whether changes in these characteristics differentially predicted posttreatment outcome. Regarding the first goal, patient functioning improved significantly in all investigated domains, both interpersonal and cognitive, with medium to large effect sizes. These findings are consistent with O'Hara and colleagues' (2000) and Whisman's (2001) finding that dyadic adjustment increased in IPT, and corroborate Ravitz and colleagues' (2008) findings in this same patient sample (though with different statistical methods for assessing change) that both interpersonal problems and attachment insecurity decreased. These findings add to the literature by demonstrating that perceived social support and dysfunctional attitudes also improved during IPT. The changes in interpersonal domains are consistent with IPT's direct focus, and, as discussed previously, IPT could affect cognitions by classifying dysfunctional thoughts as the product of a disease state rather than as a reflection of reality (Weissman et al., 2000). IPT, then, may foster improvement in all these areas; however, the use of a single treatment group does prevent a definitive conclusion, because effects may be due to history, maturation, regression to the mean, and/or the impact of repeated testing.

Regarding the second goal, changes in several patient characteristics were associated with various outcome variables. Decreased interpersonal problems and increased satisfaction with social support were associated with favorable outcomes, and surprisingly, decreased dyadic adjustment and increased attachment avoidance, both

changes in the less adaptive direction, were also associated with favorable outcome. These findings suggest that it is unlikely that decreasing dysfunctional attitudes, improving dyadic adjustment, increasing attachment security, or gathering more individuals to use as social supports are mechanisms through which IPT leads to symptom improvement (though it does not completely disqualify these as mechanisms, as one cannot prove the null).

Improved interpersonal problems and increased satisfaction with social support, however, did emerge as candidate change mechanisms: both changed adaptively over treatment, and adaptive change in both related to better outcome (albeit to a marginally significant degree). Of course, it is also possible that decreased depression ameliorates interpersonal problems and increases satisfaction with perceived social support, or that a third variable is at work. Therefore, further research with a control group and repeated measures is needed both to establish the temporal sequence of changes in interpersonal problems, social support satisfaction, and depression symptoms, and to test for their statistical mediation of IPT's specific treatment effect. If these variables are demonstrated to be mediators (mechanisms), IPT psychotherapists might target their interventions at tempering those domains of maladaptive interpersonal style that are measured by the IIP-64; that is, tendencies to be overly domineering, submissive, cold, or self-sacrificing. Future studies also could examine whether specific interpersonal problem domains are more associated with symptom change in order to clarify whether interventions should be even more narrowly targeted. Further, psychotherapists might aim to foster patients' effective utilization of their existing social support systems. This might be achieved by focusing on strengthening current relationships rather than seeking

new ones, encouraging patients to spend more time with confidants, teaching self-disclosure and listening skills, and navigating conflicts in these pre-existing close relationships.

That interpersonal variables, but not dysfunctional attitudes, emerged as candidate mechanisms tentatively implies that IPT may work through theorized (i.e., interpersonal) pathways. But it is, of course, premature to draw this conclusion definitely until these candidate mechanisms are confirmed and until other potential theory-nonspecific mechanisms are tested (and either confirmed or ruled out). For example, as discussed previously, it is possible that other theory-nonspecific variables, like self-efficacy/mastery and behavioral activation, might meaningfully foster change in IPT. Additionally, factors common to all psychotherapies may be mechanisms of IPT. Common factors models propose that specific techniques are less relevant than the development of a trusting, collaborative relationship with a therapist and the instillation of a belief that the treatment will be curative (Wampold, 2010). The IPT manual instructs therapists to foster warm and supportive relationships and prescribes various interventions aimed at instilling hope, including explicitly stating that depressed mood is temporary and that treatment is effective, as well as providing a clear rationale for the techniques used (Weissman et al., 2000). Unfortunately, these additional variables were not assessed in the current study.

The unexpected results that better outcomes were associated with reduced dyadic adjustment and increased attachment avoidance require explanation. Because the ECR inquires specifically about attachment to romantic partners, and because insecure attachment style is associated with lower relationship satisfaction and less intimacy,

commitment, and stability (Pietromonaco & Beck, in press), some processes may explain both findings simultaneously, with attachment style and dyadic adjustment reciprocally influencing each other. One possibility is that patients who improved became better able to recognize preexisting problems in their relationships, either through direct re-assessment of the relationship and its maladaptive patterns (perhaps via IPT's techniques), and/or through revision of prior self-blame or other negative self-attributions (e.g., they were able to view the relationship, not themselves, as the main problem). In fact, perhaps some patients expected their relationships to improve along with their depression, and when this failed to occur, they recognized that the issue lay in the relationship. Such decrease in self-reported dyadic adjustment may, then, actually be a positive result of IPT when patients are in unhealthy relationships. Perhaps some patients who experience relationship discord related to improvements in mood cope with that discord by becoming more avoidant in their attachment style. In other words, they may distance themselves and become less reliant on their partners (rather than displaying more anxious attachment, for instance), because their lessening depression enables greater self-sufficiency.

Another possibility is suggested by family systems theory, which posits that symptoms may function to maintain homeostasis in familial interactions. Consequently, any abrupt symptom change, without consideration of the system as a whole, can lead to destabilization of the system and discord in the interrelationships (Jackson, 1957). Thus, as a patient's depression lessens in IPT, marital or romantic relationships may be disrupted. For example, the patient's partner may have felt secure in a caretaking role, and when that role ceases to be necessary, discomfort or disagreements about ideal

interactions between partners could result. Similarly, those patients who endeavor to apply skills in analyzing communications and interacting in novel ways learned in IPT might both improve their own symptoms and cause destabilization of normal communication patterns with partners.

It is important to recall that overall, dyadic adjustment *increased* over the course of treatment. In the mid-20th century, several therapists with psychodynamic or systemic perspectives expressed concern that individual therapy could cause marriages to deteriorate (e.g., Hurvitz, 1967; Kohl, 1962); however, Hunsley and Lee (1995) reviewed empirical work and concluded that while “some temporary disruptions or inconveniences are to be expected,” marital relationships are generally unaffected or improved by one spouse’s individual therapy. This is consistent with this study’s finding of increased dyadic adjustment, as well as with the hypothesis that those patients who change most dramatically might experience mild, temporary discord.

As in the case of demonstrating mechanisms of change, the hypothesis, that the association between decreased dyadic adjustment and improvement in depression is due to depression temporarily disrupting the family system, must be investigated using repeated measurements of the relevant variables in order to establish the sequence of changes within the patient. Measures of communication styles with romantic partners should be included, and partners as well as patients should also respond to measures of dyadic adjustment and attachment style. Additionally, the assumption that dyadic adjustment and attachment avoidance eventually re-stabilize should be tested. If this hypothesis proves accurate, it may be wise for psychotherapists practicing IPT to attend to the effects of patient change on the dyadic, or perhaps even wider familial, systems.

Despite the preliminary nature of the current results, one can still draw tentative recommendations for the practice of IPT from this study. First, therapists may find that focusing on changing maladaptive interpersonal styles and increasing utilization of existing social supports accelerates change. Second, it may be important for therapists to monitor how the changes made in therapy impact the patient's primary romantic relationship, so that any negative effects can be addressed and managed. This study also has implications for research on IPT, both in narrowing down possible mechanisms and in generating hypotheses for further study. Future work that aims to elucidate the mechanisms for IPT can improve upon the limitations of this study by (a) including comparison groups, thereby enabling investigation of whether mechanisms are therapy-specific and eliminating the confounds inherent in designs with no control; (b) repeating measures frequently to allow for better (and more complex) estimates of change than two time points, and to clarify better the temporal sequence of changes among putative mechanisms and symptomatic outcomes; and (c) including not only those variables that are consistent with a treatment's underlying theory, but also those (scientifically more powerful) variables that would falsify or demand revision of the theory. The latter in particular seems to be a widespread problem in the literature: in process studies of IPT, measures of interpersonal variables dominate, while measures of cognitions, behaviors, or other personality traits are absent. Similarly, studies of interpersonal variables in CBT are practically nonexistent, impeding falsification of each theory's claims. Studies of IPT's mechanisms might target interpersonal problems and increased social support, possibly by experimentally manipulating the degree to which psychotherapists address these areas, as well as explore other theory-nonspecific variables. Refining treatment

research in these ways will lead to the discovery and confirmation of change mechanisms, elucidating factors maintaining psychopathology, refining psychotherapy theory, and encouraging the development of more efficient and effective treatments.

Table 1

*Pretreatment Patient Characteristics*

	<i>M</i>	<i>SD</i>	<i>n</i>	%
Age	39.6	11.6	95	
Sex				
Female			71	74.7
Male			24	25.3
Marital status <sup>a</sup>				
Never married			36	37.9
Married			33	34.7
Separated/divorced/widowed			21	22.1
Education <sup>a</sup>				
Graduate school			16	16.8
University			43	45.3
College			20	21.1
High school			13	13.7
Age of onset of first depressive episode	29.1	12.9	88	
# previous depressive episodes	2.25	3.44	87	
Duration current depressive episode (months)	20.6	29.2	91	
On antidepressants? <sup>a</sup>				
Yes			51	53.7
No			31	32.6
Axis I comorbidity				
No comorbidity			54	56.8
Any Axis I disorder <sup>b</sup>			41	43.2
Dysthymia			19	20.0
Anxiety disorder			16	16.8
Substance abuse or dependence			8	8.4
Eating disorder NOS			3	3.2
Impulse-control disorder NOS			1	1.1
Vaginismus			1	1.1

<sup>a</sup>Category totals sum to less than 95 due to unreported or otherwise missing data.

<sup>b</sup>Totals for specific Axis I disorders sum to more than 41 because some patients were diagnosed with more than one comorbid condition.



Table 2

*Pre- to Posttreatment Changes in Depression and Global Functioning*

Measure	Pretreatment		Posttreatment		<i>t</i>	<i>df</i>	<i>p</i>	<i>d</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>				
BDI-II	27.73	8.86	13.38	9.89	13.51	94	<.001	1.53
HRSD	16.42	5.13	8.41	5.90	12.35	94	<.001	1.45
GAF	60.12	5.70	73.14	7.49	-15.42	94	<.001	1.96

*Note.* BDI-II = Beck Depression Inventory-Second Edition (Beck et al., 1996); HRSD = Hamilton Rating Scale for Depression (Hamilton, 1960, 1967); GAF = Global Assessment of Functioning (Endicott et al., 1976)

Table 3

## Estimated Pre- to Posttreatment Change in Patient Characteristics

Measure	$\gamma$	<i>SE</i>	<i>p</i>
DyAS	8.523	1.903	<.001
ECR			
Anxiety	-6.482	1.731	<.001
Avoidance	-7.046	1.775	<.001
IIP-64	-18.989	3.001	<.001
SSQ-B			
Number	0.402	0.194	0.04
Satisfaction	0.523	0.096	<.001
DAS-A	15.398	2.938	<.001

*Note.* DAS-A = Dysfunctional Attitude Scale-Form A (Weissman, 1979; Weissman & Beck, 1978); DyAS = Dyadic Adjustment Scale (Spanier, 1976); ECR = Experiences in Close Relationships (Brennan et al., 1998); IIP-64 = Inventory of Interpersonal Problems-Circumplex Version (Horowitz et al., 2000); SSQ-B = Social Support Questionnaire-Brief (Sarason et al., 1987)

Table 4

*Prediction of Posttreatment BDI-II*

Variable	$\beta$	SE	<i>p</i>
Pretreatment BDI-II	0.535	0.11	<.001
Gender	1.748	1.679	0.304
Medication status	3.825	2.012	0.064
DyAS change	2.028	0.727	0.008
ECR			
Anxiety change	-1.675	1.045	0.117
Avoidance change	-1.519	1.143	0.194
IIP-64 change	2.846	1.482	0.062
SSQ-B			
Number change	0.56	0.772	0.473
Satisfaction change	-1.423	0.795	0.081
DAS-A change	-0.004	0.793	0.996

*Note* . BDI-II = Beck Depression Inventory-Second Edition (Beck et al., 1996); DAS-A = Dysfunctional Attitude Scale-Form A (Weissman, 1979; Weissman & Beck, 1978); DyAS = Dyadic Adjustment Scale (Spanier, 1976); ECR = Experiences in Close Relationships (Brennan et al., 1998); IIP-64 = Inventory of Interpersonal Problems-Circumplex Version (Horowitz et al., 2000); SSQ-B = Social Support Questionnaire-Brief (Sarason et al., 1987)

Table 5

*Prediction of Posttreatment HRSD*

Variable	$\beta$	SE	p
Pretreatment HRSD	0.503	0.114	<.001
Gender	2.482	1.335	0.07
Medication status	1.753	1.232	0.162
DyAS change	1.474	0.618	0.022
ECR			
Anxiety change	0.039	0.691	0.955
Avoidance change	-0.849	0.592	0.152
IIP-64 change	1.081	0.689	0.124
SSQ-B			
Number change	0.286	0.645	0.659
Satisfaction change	-0.153	0.652	0.816
DAS-A change	-0.155	0.584	0.792

*Note.* DAS-A = Dysfunctional Attitude Scale-Form A (Weissman, 1979; Weissman & Beck, 1978); DyAS = Dyadic Adjustment Scale (Spanier, 1976); ECR = Experiences in Close Relationships (Brennan et al., 1998); HRSD = Hamilton Rating Scale for Depression (Hamilton, 1960, 1967); IIP-64 = Inventory of Interpersonal Problems-Circumplex Version (Horowitz et al., 2000); SSQ-B = Social Support Questionnaire-Brief (Sarason et al., 1987)

Table 6

*Prediction of Posttreatment GAF*

Variable	$\beta$	SE	p
Pretreatment GAF	0.37	0.15	0.019
Gender	1.877	1.812	0.306
Medication status	-1.907	1.859	0.311
DyAS change	-0.501	0.741	0.503
ECR			
Anxiety change	0.76	1.011	0.456
Avoidance change	1.501	0.865	0.09
IIP-64 change	-1.256	0.948	0.193
SSQ-B			
Number change	0.787	0.888	0.381
Satisfaction change	-0.037	0.761	0.961
DAS-A change	0.549	0.997	0.585

*Note*. DAS-A = Dysfunctional Attitude Scale-Form A (Weissman, 1979; Weissman & Beck, 1978); DyAS = Dyadic Adjustment Scale (Spanier, 1976); ECR = Experiences in Close Relationships (Brennan et al., 1998); GAF = Global Assessment of Functioning (Endicott et al., 1976); IIP-64 = Inventory of Interpersonal Problems-Circumplex Version (Horowitz et al., 2000); SSQ-B = Social Support Questionnaire-Brief (Sarason et al., 1987)

Table 7

*Prediction of Remission*

Variable	$\beta$	SE	p	OR
Pretreatment BDI-II	-0.098	0.038	0.014	0.907
Gender	0.087	0.496	0.861	1.09
Medication status	-1.235	0.498	0.017	0.291
DyAS change	-0.399	0.279	0.161	0.671
ECR				
Anxiety change	0.125	0.285	0.664	1.133
Avoidance change	0.685	0.452	0.138	1.984
IIP-64 change	-0.92	0.596	0.13	0.399
SSQ-B				
Number change	-0.17	0.3	0.574	0.844
Satisfaction change	0.237	0.25	0.348	1.268
DAS-A change	-0.349	0.32	0.282	0.706

*Note* . BDI-II = Beck Depression Inventory (Beck et al., 1996); DAS-A = Dysfunctional Attitude Scale-Form A (Weissman, 1979; Weissman & Beck, 1978); DyAS = Dyadic Adjustment Scale (Spanier, 1976); ECR = Experiences in Close Relationships (Brennan et al., 1998); GAF = Global Assessment of Functioning (Endicott et al., 1976); IIP-64 = Inventory of Interpersonal Problems-Circumplex Version (Horowitz et al., 2000); SSQ-B = Social Support Questionnaire-Brief (Sarason et al., 1987)

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