

3-2010

Cognitive and Psychodynamic Mechanisms of Change in Treated and Untreated Depression

Daniel Coleman

Diane Cole

Leslie Wuest

George Fox University, lwuest@georgefox.edu

Follow this and additional works at: http://digitalcommons.georgefox.edu/sw_fac

 Part of the [Psychology Commons](#), and the [Social Work Commons](#)

Recommended Citation

Originally published in *Journal of Clinical Psychology*.

This Article is brought to you for free and open access by the School of Social Work at Digital Commons @ George Fox University. It has been accepted for inclusion in Faculty Publications - School of Social Work by an authorized administrator of Digital Commons @ George Fox University. For more information, please contact arolf@georgefox.edu.

Cognitive and Psychodynamic Mechanisms of Change in Treated and Untreated Depression

▼
Daniel Coleman, Diane Cole, and Leslie Wuest
Portland State University

Two patient-level mechanisms of change, defenses, and cognition were tested over 3 time points in 65 depressed adults, approximately half receiving treatment. Early changes in automatic thoughts and immature defenses were associated with symptom change from time-one to time-three. The directionality of early automatic thought change predicting symptom change was partially supported, but immature defense change occurs simultaneously with, or after, symptom change. Given the convergent evidence of cognitive change as a mediator of depression reduction, all depression therapies should consider how they address depressive cognition. To build a more complete understanding of how to ameliorate depression, future studies should continue to include constructs from multiple theories and have measures of therapy process in addition to patient level mechanisms. © 2009 Wiley Periodicals, Inc. *J Clin Psychol* 66:215–228, 2010.

Keywords: cognitive therapy; psychodynamic therapy; mechanisms of change; depression

Two central psychological constructs to understand the psychopathology and treatment of depression are the cognitive-behavioral and psychodynamic perspectives. One type of evidence for a model of psychopathology is the efficacy and effectiveness of treatment based on that model. Cognitive therapy is well-established as an effective treatment of depression according to several systematic reviews of the literature (Chambless & Ollendick, 2001; Roth & Fonagy, 2005). Psychodynamic therapy is rated as “probably efficacious,” with fewer controlled clinical trials and an absence of replication using the same manual by different research groups (Chambless & Ollendick, 2001; Abbass, Hancock, Henderson, & Kisely, 2006).

One of the next challenges to psychotherapy research is identifying the mechanisms of change, or active ingredients, of effective treatments. At the patient level, mechanism of change research means identifying and studying psychopathological mechanisms that drive symptom change. This study examines two theorized

mechanisms of change (cognition and defenses) and has multiple measurement points to allow testing of temporal order of mechanisms and outcomes.

Theoretical Framework

This study is based in a critical realist epistemology that understands the cognitive and the psychodynamic models as empirically refined maps of depression and its treatment (Wakefield, 1995). The psychodynamic and cognitive explanatory models each illuminate dimensions of psychopathology and treatment, and each developed in traditions of research with little reference to the other. When two explanatory models cover the same territory, it is good science to construct research that examines simultaneously constructs from both traditions. It is possible that more powerful empirically based explanatory models will result from integrative research.

Cognitive Theory and Research

In the tradition of cognitive therapy led by Aaron Beck, a hierarchy of depressogenic cognition is proposed: In-the-moment automatic thoughts can be grouped into patterns of dysfunctional attitudes. Both automatic thoughts and dysfunctional attitudes stem from less directly accessible core beliefs or cognitive schemas. Although cognitive therapy, such as Beck's, includes behavioral interventions, it is held that these interventions work through their effect on changing cognitions (Beck, Rush, Shaw, & Emery, 1979; Beck, 1995). Although the theory proposes that depression is rooted in the underlying core belief or cognitive schema, the depressive cognition can be treated or measured only through the observable automatic thoughts and dysfunctional attitudes (Garratt, Ingram, Rand, & Sawalani, 2007).

A series of studies demonstrate that changes in automatic thoughts or dysfunctional attitudes predict decreased depression. DeRubeis and co-authors (1990) showed that reduction in depression late in treatment was predicted by early change in dysfunctional attitudes but not by automatic thoughts. Kwon and Oei (2003) found that changes in automatic thoughts predicted decreased symptoms, and changes in dysfunctional attitudes had a mediated effect on decreased symptoms through changes in automatic thoughts. Furlong and Oei (2002) demonstrated that change in dysfunctional attitudes was a better predictor than automatic thoughts of reduced depression at termination. In a community mental health sample, Coleman (2005) showed that changes in automatic thoughts were associated with symptom change. Large effect sizes were found in these four studies for the relationship of cognitive change to symptom change.

However, cognition has not consistently proved to be a specific change mechanism of cognitive therapy, with change in cognition associated with symptom change in treatments other than cognitive therapy (Oei & Free, 1995; Whisman, 1999). Garratt et al. (2007) argue that cognitive therapy to pharmacotherapy comparisons provide the best test of specificity, as all psychosocial comparison treatments inevitably directly or indirectly address cognition. Their review found that the majority of studies found similar cognitive change in pharmacotherapy as in cognitive therapy. This result is consistent with an assumption of this study: that depressive cognition is nearly universal in depressed participants and reduction in depressive cognition is likely to be associated with reduced depression.

In comparison to cognitive theory and research on mechanisms of change, psychodynamic theory is less precise about mechanisms of change, and fewer psychodynamic studies include tests of theory-based change mechanisms. There is a long tradition of psychodynamic theorizing about “therapeutic action,” and recent psychodynamic theorists have begun to address mechanisms of change in research findings (Gabbard & Westen, 2003; Jones, 2000).

The psychodynamic theoretical frame in this investigation centers on an ego-psychological perspective that views overall developmental level of defenses and adaptive fit of defensive functioning as intrapsychic processes that contribute to mental health symptoms. Psychological health is found in flexible use of defenses, with predominant use of mature defenses and relatively little use of immature defenses (McWilliams, 1994). The psychotherapy relationship itself, through processes of identification and internalization, stimulates use of more mature defenses, as well as insight generated through interpretative techniques causing a shift to more adaptive defenses (Gabbard, 1994; Gabbard & Westen, 2003). Extensive theoretical discussions of the role of the ego and defenses to therapeutic action can be found in Busch (1995) and Gray (1990).

Most empirical research on defenses and symptoms have not formulated changes in defenses as a mechanism of change, more commonly simply observing the correlation of changes in defenses and symptoms. Two studies using a patient self-report measure of defenses found robust changes in defenses over the course of treatment that were moderately to strongly associated with reduced symptoms (Akkerman, Carr, & Lewin, 1992; Albucher, Abelson, & Nesse, 1998). Bond and Perry (2004) found a similar pattern in long-term psychodynamic therapy, with medium size change in overall defense style that was correlated with symptom change. In Coleman (2005), changes in immature defenses, but not mature defenses, were associated with symptom reduction across two time points in treatment with medium size effects.

Research using projective testing to assess defenses also demonstrated substantial change in defenses across treatment that were associated with outcome (Cramer & Blatt, 1990). One study that used a therapist rating of defenses found that changes in defenses did not predict symptom change, and that the majority of symptom change came earlier in the treatment than defense change (Hersoug, Sexton, & Hoglend, 2002).

Relationship of Cognition and Defenses

Only one study was found that examined the relationship of changes in cognitions and defenses. As previously noted, Coleman (2005) demonstrated that decreases in both automatic thoughts and immature defenses were associated with decreased symptoms. Across two time points in treatment, there was a non-significant trend level correlation of decreases in automatic thoughts with decreases in immature defenses ($r = .33, p = .06$).

In addition to empirical evidence, the relationship of defenses and cognition can be explored theoretically. A defense may be configured by several cognitive patterns working in concert. However, it could be argued that defenses are more about emotional processes than cognitions. Because defenses are at least partially unconscious, they may more resemble deeper cognitive structures such as dysfunctional attitudes, or “core beliefs” (Beck, 1995), than they do the consciously accessible automatic thoughts (Coleman, 2005).

The psychodynamic and cognitive-behavioral perspectives each rank defenses and cognitions by adaptiveness, “cost” to the patient, and distortion of reality. Both psychodynamic and cognitive therapies hope to assist patients to develop more adaptive mechanisms that are better attuned to reality and incur low costs or even benefits to the patient. The theoretical convergence and divergence indicates that some correlation of cognitive and defensive change is expectable, with divergence centering mainly on those areas where defenses are operating at a more unconscious and emotional level.

Summary

The existing literature supports cognition and defenses as intrapsychic correlates of symptom reduction, with better evidence that cognitive change precedes and predicts symptom change (but may not be specific to cognitive therapy). Effect sizes for the association of cognitive change to reduction in depression were generally large, and the association of defense change ranged from medium to large effects, mostly of changes in immature defenses.

The current study advances cognitive, psychodynamic, and trans-theoretical research in several ways. First, this study includes both psychodynamic and cognitive perspectives, allowing further trans-theoretical exploration of these mechanisms of change. Second, three time points are used, permitting a test of full or partial temporal precedence. Third, the study drew depressed participants from the community, allowing examination of intrapsychic mechanisms in untreated depression as well as treated depression. The inclusion of untreated participants allows a test of a non-specificity hypothesis: that change in defenses and cognitions should predict change in mood, regardless of treatment status.

Research Questions and Analyses

Changes in cognition and defenses are hypothesized to be mechanisms related to depressive symptoms, regardless of an individual’s treatment status. Assuming at least a partial common pathway of symptom reduction, the intra-psychic mechanisms of distorted thinking and immature defenses should be present in the natural history of untreated depression as well as treated depression, and in patients receiving different types of treatment. The sample includes both treated and untreated participants, but because those who received treatment received a mix of psychotherapies and medications, this study does not offer conclusions about the effect of specific treatments.

In addition, this sample of lower SES participants with recurrent depression differs from the higher SES, single episode depression participants found in many clinical trials (Morrison, Bradley, & Westen, 2003), making possible a test of the generalizability of clinical trial based mechanism research.

The primary research question is: Does early mechanism change predict later symptom change? The first analysis examines the correlation of changes in time-one to time-two automatic thoughts, immature and mature defenses, to time-two to time-three depression and general psychiatric symptoms. This first test provides full temporal precedence. The second analysis relaxes temporal precedence, correlating time-one to time-two automatic thoughts, immature and mature defenses, to time-one to time-three depression and psychiatric symptoms. The rationale for the relaxed temporal precedence analysis rests in the exploratory, hypothesis generating nature of this study. It is acknowledged in the design of this study that sample size, and the

inclusion of untreated participants who may experience little symptom change, might not allow the power to show a difference in the most rigorous test including full temporal precedence. The results from the full temporal precedence tests should be given more weight than those from relaxed partial temporal precedence analysis.

Follow-up analyses test the directionality of influence by examining the correlation of early symptom change to later defense and automatic thought change, using both full and relaxed temporal precedence models.

Methods

Design

This study utilized a repeated measures survey design, drawing a sample of depressed adults from the community. An initial phone screen with a structured diagnostic interview was followed by three paper and pencil questionnaires spaced approximately 6 weeks apart. Participants were paid \$10 for each paper and pencil survey returned. The Institutional Review Board at Portland State University reviewed and approved the human subject protocol for this study.

Sampling and Procedures

Participants were drawn from the greater Portland Oregon metropolitan area. Recruitment was conducted through posting flyers, advertising in community papers, and posting on local Internet classifieds. Potential participants contacted the researchers via telephone or e-mail. A focused clinical diagnostic interview was conducted in the telephone screening using the depression sections of the Mini International Neuropsychiatric Interview-5 (MINI-5; Lecrubier et al., 1997). Participants were offered participation if they met criteria for a DSM-IV depression diagnosis with current depressive symptoms and, if in treatment of any kind, the treatment had started in the past 2 months.

Telephone screens were conducted with 83 participants, four of which were identified as possibly having subclinical depressive symptoms and one as possibly being an unreliable reporter of symptoms. These five were offered the opportunity to complete the first paper and pencil questionnaire to provide further information for making a decision on inclusion in the study. Four of the five were judged appropriate for continuing in the study. Sixty-five participants completed time-one questionnaires. There were no differences between the 65 responders and the 18 non-responders on any of the variables collected in the phone screen.

There was no attrition at time-two, but six participants did not complete time-three questionnaires. A conservative strategy was used, substituting time-two scores for the missing time-three scores for these six participants, resulting in an analysis sample of 65. The mean lag time between the time-one survey and the time-three survey was 12.6 weeks ($SD = 4$). The lag time was not associated with any of the variables of interest.

Of the 65 participants, 71% were women ($n = 46$) and 77% were white ($n = 50$). The average age was 41.7 years ($SD = 14.4$). Approximately half of the participants (52.3%) reported currently being from a lower income socioeconomic status ($n = 34$) and 60% had some college education ($n = 39$). The vast majority of the sample met criteria for a current major depressive episode ($n = 60$; 92.3%) and two-thirds met criteria for recurrent major depression ($n = 43$; 66%). Twenty-five met criteria for dysthymic disorder (39%), with 23 of these meeting DSM-IV criteria for “double

depression” (lifetime dysthymic disorder preceding first major depressive episode). Forty-one (63%) reported a previous history of diagnosis and treatment for a mood or anxiety disorder.

Out of the 65 participants in the analysis sample, 30 (46%) received some form of treatment for depression during the course of the study (took psychiatric medications or attended at least one therapy session). Sixteen (24%) were prescribed psychiatric medication, and twenty-one (32%) were in psychotherapy. Seven participants (11%) were receiving both therapy and psychiatric medication. Medications were prescribed by a primary care provider for nearly half of those receiving medication ($n = 7$). For those for whom antidepressant prescription data were available, seven participants were receiving an SSRI antidepressant and four an atypical antidepressant (Welbutrin). At time-one, the 30 participants receiving treatment had started this treatment an average of 2.9 weeks earlier ($SD = 3.3$). The time since treatment start was not correlated with time-one BDI or BSI. No further information was available to judge the quality, frequency, or the theoretical orientation of the psychotherapies.

Measures

Automatic thoughts were measured using the Automatic Thoughts Questionnaire (ATQ), a measure of negative automatic thoughts about the self. The scale has excellent internal consistency with an alpha of .97 (Hollon & Kendall, 1980). The six highest loading items in previous published factor analyses were selected for use in this study, achieving very good reliability ($\alpha = .91$). (All of the reliabilities reported for this study are the average Cronbach's α across the three time points).

Mature and immature defenses were measured with the Defense Style Questionnaire (DSQ), a measure of conscious derivatives of unconscious defense mechanisms. The DSQ showed good convergent validity with other defense measures, including observer rated defenses (Bond et al., 1989). Published reliability of the two subscales used in this study were good ($\alpha = .68$ for mature and $\alpha = .80$ for immature; Andrews, Singh, & Bond, 1993). The highest loading items tapping mature and immature defenses were selected from previous published factor analyses, resulting in two six-item scales. The individual defenses in the immature scale were autistic fantasy, displacement, passive-aggression, projection, and somatization. The mature defenses assessed were anticipation, humor, sublimation, and suppression. Adequate to good reliability was observed in this sample ($\alpha = .69$ for mature and $\alpha = .74$ for immature).

General psychiatric symptom severity was measured with the Brief Symptom Inventory (BSI), a 53-item version of the 90-item Symptom Checklist 90 (SCL-90). In both short and long forms, this scale is a widely used outcome measure designed to tap a range of psychiatric symptoms. The published internal reliability coefficients for the nine BSI subscales range from .71 to .85, and test-retest reliability for the whole scale is .90 (Derogatis, 1993). The overall reliability achieved in this study for the 53 BSI items was excellent ($\alpha = .96$).

Depression was measured with the Beck Depression Inventory (BDI). The BDI has extensive reliability and validity evidence (Beck, Steer, & Brown, 1996). Reliability in this study for the BDI was very good ($\alpha = .87$).

Analysis

Power analysis found that this study ($N = 65$) has the power to detect a large association ($r = .50$) of change mechanisms to change in symptoms (Cohen, 1988).

The study was adequately powered to detect the expectable effect of cognitive change to depression and marginally powered to detect the expectable relationship of defense change to depression.

Treated and untreated groups were examined for mean differences on the variables of interest. Regression residualized change score variables were calculated for the BDI, the BSI, automatic thoughts, and immature and mature defenses. Scores were computed for time-one to time-two, time-two to time-three, and time-one to time-three. Residual change scores are superior to subtractive change scores in being less vulnerable to extreme scores at either pre- or post-test and provide better reliability (Cronbach & Furby, 1970). Malgady and Colon-Malgady (1991) provide a formula for calculating the reliability of residualized change scores from pre-and post-test reliabilities and the correlation of the pre- and post-test. The greater the correlation of the pre- and post-test, the greater likelihood that error is concentrated in the change score, attenuating the reliability achieved in the pre- and post-test. The reliability of the change scores was good to very good, with the exception of the reliability for the immature defense change scores ($\alpha = .62$) and poor reliability for the mature defense change scores ($\alpha = .38$). The immature defense change score was retained despite marginal reliability, but the mature defense change score was not used in further analysis due to poor reliability.

The residualized change scores were entered into full and relaxed temporal precedence correlation analyses, as stated in the research questions section. The retention of a marginal reliability immature defense change variable, and the relaxed temporal precedence tests are appropriate to a hypothesis generating study in an emergent area of study. Where meaningful, the statistical significance of differences in correlation coefficients was tested, using the equation for the difference of two dependent correlations (Chen & Popovich, 2002, pp. 23–25). The difference between the association of early mechanism change with symptom change and early symptom change with mechanism change could not be tested due to the variables not matching any known formula for significance testing (correlations of unique pairs of variables on the same sample).

Results

There were no differences in age, gender, or ethnicity of those who received treatment and those who did not. The participants in the treatment group were 1.9 times more likely to have a history of previous treatment ($\chi^2(1, N = 65) = 4.42, p < .05$). The treated and untreated subsamples were compared with time-one, time-three, and change time-one to time-three of automatic thoughts, immature and mature defenses, and BDI and BSI scores. No significant mean differences were found between the treatment group and the no treatment group, except that the no treatment group showed a greater decrease in immature defenses from time-one to time-three ($t(61) = 2.02, p = .04, \text{Cohen's } d = .51$). Because the groups did not differ on the majority of variables of interest, they were aggregated for all further analyses.

The means and standard deviations at each time point are presented in Table 1. The mean BDI score at time-one exceeded the cut-off for severe depression (Beck et al., 1996). The BSI score was also elevated, with the observed mean score one-half of a standard deviation above the mean of 1,002 outpatients (Derogatis, 1993).

A statistically significant mean change was observed in both the BDI and the BSI, with a medium-size half standard deviation change on the BDI and a small one-quarter standard deviation change on the BSI (Cohen's (1988) guidelines for Cohen's d : about .2 is small/weak; about .50 is medium/moderate; about .80 large/

Table 1

Comparison of Time 1 (Baseline), Time 2 (5-Weeks), and Time 3 (10-Weeks) Change Mechanism and Symptom Scores

Variable	Time 1		Time 2		Time 3		Paired <i>t</i> tests	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	T1 vs. T2	T1 vs. T3
Automatic thoughts	3.37	1.06	3.16	1.04	2.94	1.14	2.03* (.20)	3.38** (.39)
Immature defenses	5.27	1.48	4.99	1.59	4.89	4.62	1.65 (.18)	1.58 (.24)
Mature defenses	4.84	1.47	5.19	1.46	5.22	1.37	-2.10* (-.24)	-2.36* (-.27)
Beck Depression Inventory	29.42	11.43	25.62	12.34	23.41	13.06	3.55*** (.32)	5.31*** (.49)
Brief Symptom Inventory	1.67	.66	1.57	.09	1.47	.80	1.42 (.21)	2.60* (.27)

Note. *M* = mean; *SD* = standard deviation; Cohen's *d* statistic is reported in parentheses in the paired *t* test columns. *N* = 65.

p* < .05; *p* < .01; ****p* < .001.

strong). As noted above, the treatment group did not differ from those not receiving treatment in decrease in symptoms across the study.

Ninety-five percent of the sample scored in the clinical range on the BSI and BDI at time-one. At time-three, four of five subjects still scored in the clinical range on at least one measure. Although the majority of subjects continued to have clinical level of symptoms at the end of this study, approximately half of participants had reliable decreases in symptoms. Jacobson and Truax's (1991) Reliable Change Index (RCI) gives the threshold amount of change where we can be confident at the .05 level that the change is not due to measurement error. RCI calculations used followed Barkham et al. (1996), Barkham, Rees, Stiles, Hardy, and Shapiro (2002) for the BDI, and used normative data from Derogatis (1993) for the BSI. Using the RCI, 46% of participants had reliable decrease in symptoms on the BSI and 50% on the BDI.

The mean changes in cognition, immature and mature defenses were all modest across the three time points. The changes were largest for automatic thoughts, followed by mature defenses (both statistically significant). The one-quarter of a standard deviation change in immature defenses was not statistically significant.

Age, sex, ethnicity, medication status, and therapy status had no relationship to the variables of interest. Time-one to time-three changes in automatic thoughts were moderately correlated with changes in immature defenses at ($r = .37, p < .01$). Changes in mature defenses were not related to changes in immature defenses or cognition.

The top section of Table 2 shows the most rigorous test, with early mechanism change predicting later symptom change. No significant relationships were found. The lower part of Table 2 presents the results of the test of change mechanisms and symptom change allowing partial temporal overlap (time-one to time-two change mechanisms association with time-one to time-three symptoms). Change in automatic thoughts was moderately to strongly correlated with decreased symptoms on both the BSI and the BDI. Change in immature defenses was moderately correlated with decreased symptoms on both outcome measures (Cohen's (1988) guidelines for Pearson's *r*: about .10 is weak/small; about .30 is moderate/medium; about .50 is strong/large). Change in mature defenses was not associated with either outcome measure. The difference between the automatic thoughts-BDI correlation ($r = .48$) and the immature defenses-BDI correlation ($r = .27$) was statistically significant ($t(62) = 2.0, p < .05$).

Table 2

Tests of Relationship of Change in Cognition and Defenses to Symptom Change

Most rigorous test—time-one to time-two change mechanisms predicting time-two to time-three symptom scores (temporal precedence of change mechanisms and symptom change)

Independent variables	Dependent variables	
	T2–T3 BDI Pearson's <i>r</i>	T2–T3 BSI Pearson's <i>r</i>
T1–T2 AUTO	.10	.12
T1–T2 IMMA	–.14	–.02

Less rigorous test—time-one to time-two change mechanisms predicting time-one to time-three symptom scores (partially cross-sectional)

Independent variables	Dependent variables	
	T1–T3 BDI Pearson's <i>r</i>	T1–T3 BSI Pearson's <i>r</i>
T1–T2 AUTO	.48**	.41**
T1–T2 IMMA	.27*	.38**

Note. AUTO = automatic thoughts; IMMA = immature defenses; BDI = Beck Depression Inventory; BSI = Brief Symptom Inventory. *N* = 65.

p* < .05; *p* < .01; ****p* < .001.

Table 3

Tests of Relationship of Change in Symptoms to Change in Cognition and Defenses

Most rigorous test—time-one to time-two symptom scores predicting time-two to time-three change mechanisms (temporal precedence)

Independent variables	Dependent variables	
	T2–T3 AUTO Pearson's <i>r</i>	T2–T3 IMMA Pearson's <i>r</i>
T1–T2 BDI	–.08	.08
T1–T2 BSI	.01	.29*

Less rigorous test—time-one to time-two symptom scores predicting time-one to time-three change mechanisms (partially cross-sectional)

Independent variables	Dependent variables	
	T1–T3 AUTO Pearson's <i>r</i>	T1–T3 IMMA Pearson's <i>r</i>
T1–T2 BDI	.27*	.31*
T1–T2 BSI	.28*	.51**

Note. AUTO = automatic thoughts; IMMA = immature defenses; BDI = Beck Depression Inventory; BSI = Brief Symptom Inventory. *N* = 65.

p* < .05; *p* < .01; ****p* < .001.

Table 3 shows the follow-up test of directionality: Does early symptom change predict changes in defenses and automatic thoughts? The most rigorous test found one significant relationship: Early decrease in BSI scores was moderately correlated with subsequent decreases in immature defenses. The test with relaxed temporal

order found that early changes in symptoms had a medium size association with decreased automatic thoughts, and early changes in symptoms had a medium to large association with decreased immature defenses. Early change in symptoms was not associated with change in mature defenses. There was a non-significant trend-level difference between the BSI- immature defenses correlation ($r = .51$) and the BSI- automatic thoughts correlation ($r = .28$); $t(62) = 1.86, p < .10$.

Discussion

The treatment and no treatment groups were comparable in demographics, and no group differences were evident in mental health symptoms over the course of the study. The treatment group in this study is equivalent to a treatment as usual (TAU) comparison group sometimes used in clinical trials- the nature, quality and intensity of treatment is likely highly variable (van de Wiel et al., 2007). The modest, significant decrease in symptoms over the course of the study for both treated and untreated participants likely reflects a number of factors, including regression to the mean and a small treatment effect of the two telephone contacts and completing the questionnaires. This sample largely comprises people with recurrent major depression, with severe symptoms, and from lower SES. The sample is markedly different than participants usually enrolled in clinical trials, offering a test of the generalizability of the existing mechanism of change evidence.

The results indicate that changes in immature defenses occur simultaneously with, or after, symptom change: The only significant relationship found in the full temporal precedence tests was of early BSI change predicting later decrease in immature defenses, and, the correlations of symptom change to defense change were larger than defense change to symptom change (as noted in methods, the difference in these correlations could not be tested).

Change in cognitions is associated with symptom change, with some indication that cognitive change predicts symptom change: The automatic thoughts full temporal precedence tests found no significant relationships, but in the relaxed temporal precedence tests, the correlations were larger for early change in automatic thoughts association with change in symptoms, than those for early change in symptoms association with automatic thoughts.

The different patterns found for cognitive change and defense change was reinforced in that the correlation of cognitive change to symptom change was significantly larger than that of immature defense change to symptom change.

The pattern found for automatic thoughts is consistent with findings from previous studies that showed changes in cognition predicted symptom change (DeRubeis et al., 1990; Kwon & Oei, 2003; Coleman, 2005). The change in cognition from time-one to time-two was relatively small (.2 of a standard deviation), but it had a strong association with symptom change across the three time points. This provides further evidence for the potency of cognitions as a change mechanism: A small early change in cognition has a strong association with larger change in symptoms.

Review articles note the consistent finding of cognitive change associated with decreased symptoms, but also converge in finding that cognitive change is not specific to cognitive therapy (Oei & Free, 1995; Whisman, 1999; Garratt et al., 2007). This current study adds to the evidence that cognitive change predicts symptom change, and that this is not a treatment specific effect. The fact that cognitive change was equally an active ingredient in the untreated group suggests cognition is a factor

in spontaneous amelioration or remission of symptoms, as well as treatment induced remission.

As Kazdin (2007) points out, there is no clear evidence that specific therapy processes, or therapist techniques, are related to changing cognition. It is likely there are numerous direct and indirect pathways of therapy processes to cognitive change. However, it is logical that direct discussion of thoughts (such as in CBT), and exploratory techniques that elicit whole networks of thoughts about the self and others (such as in interpersonal therapy or psychodynamic therapy) will influence cognition.

The Automatic Thoughts Questionnaire, used to measure cognition in this and many other studies, comprises stark, negative phrases about the self and one's life. It is not surprising that thoughts of this kind change in different treatments, and even in untreated depression. For researchers, more sophisticated and subtle means of measuring cognition should be explored. The need to develop, or to transfer from laboratory psychology, measures of cognition that are not self-report based is gaining recognition among cognitive researchers (Garratt et al., 2007; Haubert & Dobson, 2007; Hollon, Thase, & Markowitz, 2002). Some of the methodology of projective psychodynamic techniques could be employed for new cognitive measures. An example is Barton, Morley, Bloxham, Kitson, and Platts (2005) sentence completion test to measure depression from a cognitive perspective. Although the participant provides the sentence completions, the sentences are scored for depressive content by a rater.

The association of decreases in immature defenses with decreases in symptoms was modest and the evidence indicates change in symptoms precedes change in defenses. In contrast to cognitive theory, psychodynamic theory and research does not clearly designate defense change as a mechanism of symptom reduction. Some preceding studies, such as Albucher et al. (1998), made the assumption that symptom change predicts defense change. Hersoug et al. (2002) also found that symptom change preceded change in defenses. One possible pattern for defense change is that use of immature defenses will drop early in treatment as the patient stabilizes from an initial regressive crisis. Although defenses show plasticity with the ebb and flow of depression, they are also a more stable personality level phenomenon. It is logical that characterological or structural change would occur later in treatment. Research with more measurement points may be able to show the interplay of defenses and symptoms over time.

This is only the second study to analyze both measures of cognitions and defenses. The moderate correlation of changes in automatic thoughts and immature defenses was almost identical to that found in Coleman (2005). The earlier study, with only two time points and using the BSI as the outcome variable, found a similar pattern of relationship of changes in automatic thoughts, defenses, and symptom change. The partial independence of immature defense change and cognitive change suggests that the two theories are each picking up something unique in the intrapsychic process of depressed adults. Larger sample studies will be able to provide multivariate tests to partial out the unique effects of defense and cognitive change.

For clinicians, techniques or clinical processes that shift immature defenses or depressive cognitions are supported by these results. In particular, changes in automatic thoughts had strong and broad effects. Clinicians should consider integrating attention to automatic thoughts into treatment. Some process research suggests that psychodynamic clinicians already include work on cognitions. Ablon

and Jones (1998) found that the therapy process of manualized PT was correlated with both ideal, expert-generated PT and CBT process prototypes.

A quote from McWilliams (2005) theoretically integrates psychodynamic and cognitive processes, noting the importance of the emotional working-through grief but also stating: “It is also critical to help them understand the cognitive dimensions of their unconscious experience, namely their childhood conclusion that it was their own badness that provoked their misfortune” (p. 100). This study is a step towards reflecting in research the theoretical and clinical integration of McWilliams’ statement.

The limitations of self-report measurement and few measurement points were noted earlier. In addition, the use of change scores compounds any unreliability in the pre- and post-test scores. This problem is reduced, but not eliminated, by the use of residualized change scores. The mature defense change scores were not reliable, and reliability was problematic for immature defenses, reducing the power to detect relationships between constructs. In addition, this study was adequately powered to find large effects, and the literature suggests medium effects are reasonable to expect for the relation of defense change to symptoms. In the relaxed temporal order analyses, it is impossible to isolate cross-sectional associations from those across time-points. The absence of information about the treatments provided to the treated subsample also limits interpretation of the results.

Strengths of this study are as follows: diagnostic homogeneity determined by structured clinical interview; inclusion of treated and untreated depression; and adequate time-points to model temporal order. The sample provides diversity compared with most clinical trials, confirming the relationship of change mechanisms to symptoms in a different population than earlier studies.

The clinical trial movement in psychotherapy research has done much to demonstrate the efficacy of psychotherapy. Clinical trials, however, often “black box” how or why therapy works. Mechanism of change research gets inside the black box and tests if treatments work as theorized. This study provides further evidence that change in cognition is an active ingredient in reducing symptoms and preliminary evidence that immature defense change is simultaneous to, or follows, symptom change. Other studies that include multiple theory-based change mechanisms into single studies may stimulate the development of research-based hybrid therapies. Multitheoretical research could assist the mental health field to move past partisan theoretical divisions, and research-based hybrid therapies may be one pathway to exceed the efficacy of current treatments.

References

- Abbass, A.A., Hancock, J.T., Henderson, J., & Kisely, S. (2006). Short-term psychodynamic psychotherapies for common mental disorders. *Cochrane Database of Systematic Reviews*, 4. CD004687, 1–47.
- Ablon, J.S., & Jones, E.E. (1998). How expert clinicians’ prototypes of an ideal treatment correlate with outcome in psychodynamic and cognitive-behavioral therapy. *Psychotherapy Research*, 8(1), 71–83.
- Akkerman, K., Carr, V., & Lewin, T. (1992). Changes in ego defenses with recovery from depression. *Journal of Nervous & Mental Disease*, 180(10), 634–638.
- Albucher, R.C., Abelson, J.L., & Nesse, R.M. (1998). Defense mechanism changes in successfully treated patients with obsessive-compulsive disorder. *American Journal of Psychiatry*, 155(4), 558–559.

- Andrews, G., Singh, M., & Bond, M. (1993). The Defense Style questionnaire. *The Journal of Nervous and Mental Disease*, 181(4), 246–256.
- Barkham, M., Rees, A., Stiles, W.B., Hardy, G.E., & Shapiro, D.A. (2002). Dose-effect relations for psychotherapy of mild depression: A quasi-experimental comparison of effects of 2, 8, and 16 sessions. *Psychotherapy Research*, 12(4), 463–474.
- Barkham, M., Rees, A., Stiles, W.B., Shapiro, D.A., Hardy, G.E., & Reynolds, S. (1996). Dose-effect relations in time-limited psychotherapy for depression. *Journal of Consulting & Clinical Psychology*, 64(5), 927–935.
- Barton, S., Morley, S., Bloxham, G., Kitson, C., & Platts, S. (2005). Sentence completion test for depression (SCD): An idiographic measure of depressive thinking. *British Journal of Clinical Psychology*, 44, 29–46.
- Beck, J. (1995). *Cognitive therapy: Basics and beyond*. New York: Guilford.
- Beck, A.T., Rush, A.J., Shaw, B.F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.
- Beck, A.T., Steer, R.A., & Brown, G.K. (1996). *BDI-II, Beck depression inventory manual*. Orlando: Harcourt Brace, Psychological Corporation.
- Bond, M., & Perry, J.C. (2004). Long-term changes in defense styles with psychodynamic psychotherapy for depressive, anxiety, and personality disorders. *American Journal of Psychiatry*, 161(9), 1665–1671.
- Bond, M., Perry, J., Gautier, M., Goldenberg, M., et al. (1989). Validating the self-report of defense styles. *Journal of Personality Disorders*, 3(2), 101–112.
- Busch, F. (1995). *The ego at the center of clinical technique*. Northvale, NJ: Jason Aronson, Inc.
- Chambless, D.L., & Ollendick, T.H. (2001). Empirically supported psychological interventions: Controversies and evidence. *Annual Review of Psychology*, 52, 685–716.
- Chen, P.Y., & Popovich, P.M. (2002). *Correlation: Parametric and non-parametric measures*. Thousand Oaks, CA: Sage.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Lawrence Erlbaum.
- Coleman, D. (2005). Psychodynamic and cognitive mechanisms of change in adult therapy: A pilot study. *Bulletin of the Menninger Clinic*, 69(3), 206–219.
- Cramer, P., & Blatt, S.J. (1990). Use of the TAT to measure change in defense mechanisms following intensive psychotherapy. *Journal of Personality Assessment*, 54(1–2), 236–251.
- Cronbach, L.J., & Furby, L. (1970). How we should measure “change”: Or should we? *Psychological Bulletin*, 74(1), 68–80.
- Derogatis, L.R. (1993). *Brief Symptom Inventory (BSI): Administration, scoring and procedures manual*. Minneapolis, MN: NCS Pearson.
- DeRubeis, R.J., Evans, M.D., Hollon, S.D., Garvey, M.J., et al. (1990). How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *Journal of Consulting & Clinical Psychology*, 58(6), 862–869.
- Furlong, M., & Oei, T.P.S. (2002). Changes to automatic thoughts and dysfunctional attitudes in group CBT for depression. *Behavioural & Cognitive Psychotherapy*, 30(3), 351–360.
- Gabbard, G. (1994). Psychodynamic assessment of the patient. In *Psychodynamic psychiatry* (pp. 65–88). Washington, DC: American Psychiatric Press.
- Gabbard, G., & Westen, D. (2003). Rethinking therapeutic action. *International Journal of Psychoanalysis*, 84(4), 823–841.
- Garratt, G., Ingram, R.E., Rand, K.L., & Sawalani, G. (2007). Cognitive processes in cognitive therapy: Evaluation of the mechanisms of change in the treatment of depression. *Clinical Psychology: Science and Practice*, 14(3), 224–239.

- Gray, P. (1990). The nature of therapeutic action in psychoanalysis. *Journal of the American Psychoanalytic Association*, 38(4), 1083–1097.
- Haubert, L.C., & Dobson, K.S. (2007). Treatment of depression and mechanisms of change: Strengthening the links among theory, research, and practice. *Clinical Psychology: Science and Practice*, 14(3), 247–251.
- Hersoug, A.G., Sexton, H.C., & Hoglend, P. (2002). Contribution of defensive functioning to the quality of working alliance and psychotherapy outcome. *American Journal of Psychotherapy*, 56(4), 539–554.
- Hollon, S.D., & Kendall, P.C. (1980). Cognitive self-statements in depression: Development of an automatic thoughts questionnaire. *Cognitive Therapy and Research*, 4(4), 383–395.
- Hollon, S.D., Thase, M.E., & Markowitz, J.C. (2002). Treatment and prevention of depression. *Psychological Science*, 39–77.
- Jacobson, N.S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting & Clinical Psychology*, 59(1), 12–19.
- Jones, E.E. (2000). *Therapeutic action: A guide to psychoanalytic therapy*. Northvale, NJ: Jason Aronson.
- Kazdin, A.E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Psychology*, 3, 1–27.
- Kwon, S.-M., & Oei, T.P.S. (2003). Cognitive change processes in a group cognitive behavior therapy of depression. *Journal of Behavior Therapy & Experimental Psychiatry*, 34(1), 73–85.
- Leclubier, Y., Sheehan, D.V., Weiller, E., Amorim, P., Bonora, I., & Sheehan, K. (1997). The Mini International Neuropsychiatric Interview (MINI): A short diagnostic. *European Psychiatry*, 12(5), 224–231.
- Malgady, R.G., & Colon-Malgady, G. (1991). Comparing the reliability of difference scores and residuals in analysis of covariance. *Educational & Psychological Measurement*, 51(4), 803–807.
- McWilliams, N. (1994). *Psychoanalytic diagnosis: Understanding personality structure in the clinical process*. New York: The Guilford Press.
- McWilliams, N. (2005). Depressive and masochistic personality organization. *Issues in Psychoanalytic Psychology*, 27(2), 95–117.
- Morrison, K.H., Bradley, R., & Westen, D. (2003). The external validity of controlled clinical trials of psychotherapy for depression and anxiety: A naturalistic study. *Psychology and Psychotherapy: Theory, Research and Practice*, 76(2), 109–132.
- Oei, T.P.S., & Free, M.L. (1995). Do cognitive behaviour therapies validate cognitive models of mood disorders? A review of the empirical evidence. 1995. *International Journal of Psychology*, 30(2), 145–180.
- Roth, A., & Fonagy, P. (2005). *What works for whom? (2nd ed.)*. New York: Guilford.
- van de Wiel, N.M.H., Matthys, W., Cohen-Kettenis, P.T., Maassen, G.H., Lochman, J.E., van Engeland, H., et al. (2007). The effectiveness of an experimental treatment when compared to care as usual depends on the type of care as usual. *Behavior Modification*, 31(3), 298–312.
- Wakefield, J.C. (1995). When an irresistible epistemology meets an immovable ontology. *Social Work Research*, 19(1), 9–17.
- Whisman, M.A. (1999). The importance of the cognitive theory of change in cognitive therapy of depression. *Clinical Psychology: Science & Practice*, 6(3), 300–304.